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Editorial Board

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Contents

CASE REPORTS

Status Epilepticus following a Laparoscopic Surgery in a Patient of Chronic Kidney Disease – A Case Report
Harjinder Kohli, Navjot Kaur, Naresh Anand, Puneet Chopra 1

Bilateral Hirayama Disease: A Case Report
Furqan Mohd. Akram Khan, Mohammed Adnan Sheerazi 5

REVIEW ARTICLE

Seafood Allergy: Causes, Prevention Modalities, and Treatment Guidelines in the Indian Scenario
Mohammed Hidayath Hussain, Hina Afreen, Sayyeda Arshia Jabeen 9

ORIGINAL ARTICLES

Brachial Plexus: Variations in Infraclavicular Part and their Significance
Jyoti, Anshu Sharma 15

Awareness, Perceptions, Attitudes, and Practices among Antenatal Mothers in Goa towards Reproductive Tract Infections/Sexually Transmitted Infections
Manjusha Jindal, Viraj Ramesh Naik, Jagadish Cacodcar, Manojkumar Kulkarni 20

Comparative Study of Hearing Assessment in Diabetic Patients and Non-Diabetic Patients using Otoacoustic Emission and Pure Tone Audiometry at Sree Gokulam Medical College, Trivandrum, Kerala, South India
Mohamed Faris Salam, J Kiranjith, K Sasikumar 25

Lung Function and Health Status in Patients of Chronic Obstructive Pulmonary Disease on Treatment with Tiotropium
Amit Kumar Verma, Naweed Alam, Shiva Narang, Khan Amir Maroof 55

Role of Emergency Response Codes in Handling Hospital Emergencies – An Experience of Protocol Designing, Development, and Implementation in a Large Multispecialty Tertiary Care Teaching Hospital, Mysuru, India
Sathish Raju Nilakantam, M D Ravi, Jayati Bahuguna, M Dayananda 62

International Journal of Scientific Study

April 2020 • Vol 8 • Issue 1

Patterns of Mobile Phone use and Self-reported Health Problems among Adults Visiting a Private Dental Institute
Harish C Jadhav, Arun S Dodamani, Rahul N Deokar, Rahul C Bhandari, Vrushali R Khobragade, Anoli S Agrawal 68

Evaluation of Basic Life Support Knowledge and Skills of Healthcare and Non-healthcare Providers
Dake Rajesh 73

Knowledge, Detection, and Reporting of Domestic Violence among Family Medicine Residents of Eastern Province, Saudi Arabia
Layla M Alghamdi, Fayez Alzahrani 78

Prevalence of Obstructive Sleep Apnea in Surgical Patients Attending at SKIMS Hospital, Kashmir, and its Correlation with Perioperative Morbidity
Rouf Zarger, Umar Jan, A Q Lone 85

Role of Non-stress Test and Vibroacoustic Stimulation Test in Prediction of Perinatal Outcome in High-risk Pregnancies
Kaur Navneet, Upasana, Sharma Sujata 90

Effects of Cardiopulmonary Bypass on Thyroid Function and Need of Prophylactic Low-Dose Thyroxine for Post-operative Cardiac Surgery Patient
K J Vignesvaran, B Karthikeyan, P Rani, Marvin Manoah Baylis 95

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Case Report

Status Epilepticus following a Laparoscopic Surgery in a Patient of Chronic Kidney Disease – A Case Report

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Abstract

Seizures may occur in close relation to surgical procedures or with the use of anesthetic agents in several situations. The causative factors include an interruption of treatment with antiepileptic drugs (AEDs) or inadequate blood concentrations resulting from impaired gastrointestinal absorption. In operative procedures not involving the brain, transient seizures can arise from metabolic derangements or drug neurotoxicity. Other causes include hypoxia, hypotension, and embolic infarction. Seizures occurring shortly after injection of moderate to large amounts of local anesthetic should raise the suspicion of inadvertent intravenous injection. Seizures may also indicate withdrawal from unsuspected chronic use of excessive amounts of alcohol, sedative medications, mood-stabilizing agents, or AEDs. Rarely, sleep deprivation and drugs like flumazenil may precipitate seizures. We discuss the management of status epilepticus following laparoscopic urology procedure in a patient of chronic kidney disease with a history of the previous craniotomy.

Key words: Chronic kidney disease, Laparoscopic urological procedure, Metabolic derangements, Previous craniotomy, Seizures

INTRODUCTION

Peri-operative seizures, though rare, are a recognized complication of anesthetic agents. If prolonged, they can cause brain damage due to hypoxia, apnea, prolonged post-operative mechanical ventilation, and delayed awakening from anesthesia. Management includes maintaining a patent airway with adequate ventilation and protecting the patient from injuries resulting from seizures.

Laparoscopic interventions, if prolonged, in compromised patients, might lead to an increase in the intra-abdominal pressure (IAP) secondary to pneumoperitoneum. This may be detrimental and lead to various metabolic derangements as a result of decreased renal blood flow (RBF).[1,2]

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CASE REPORT AND RESULTS

A 49-year-old, 46 kg, male patient was posted for laparoscopic right ureterolithotomy and a laparoscopic left nephrolithotomy. His previous history included a craniotomy for the posterior cerebellar bleed 2 years back. At that time, he was also detected to have uncontrolled hypertension. Ultrasonography had shown right ureteric calculus and left renal multiple calculi. He had a history of DJ stent placement thrice in the past. His serum creatinine had been around 4.5–5 mg/dl.

A thorough pre-operative evaluation was done and in consultation with a nephrologist, a heparin free dialysis was planned preoperatively. Post-dialysis, hemoglobin of 10 g/dl, urea 48 mg/dl, and creatinine 4.9 mg/dl were reported. The rest of the investigations, including the electrolytes, chest X-ray, and electrocardiograph was within normal limits.

An informed high-risk consent was taken in view of chronic kidney disease (CKD), prolonged laparoscopic surgery, possible hypothermia, and a need for post-operative hemodialysis.

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The patient was induced with propofol 60mg, fentanyl 80 mcg, and atracurium 40 mg with ETT 8 mm. Laparoscopic surgery for the right ureteric stone was undertaken and the patient was positioned with a wedge under right flank for 150 min. Then, the position was changed and a wedge was placed under the left flank for the left renal surgery for about 130 min. IAP was maintained to 10–12 mm Hg, with an end-tidal carbon dioxide ($ETCO_2$) 38–42 mm Hg. Hemodynamic parameters were maintained. Prolonged laparoscopy surgery prompted us to get an arterial blood gas (ABG) after about 180 min into the procedure.

Findings-ABG-pH 7.14, pCO_2 45 mmHg, pO_2 151 mmHg, HCO_3 16.5 mmol/l, Lac 3.2 mmol/l, and glucose 360 mg/dl.

Random blood sugar by glucometer – 326 mg/dl (though the patient was not a diabetic).

Insulin infusion was started. At this stage, the surgeon was informed about the need to cut the procedure short. ABG after further 60 min showed pH 7.124, pCO_2 46.4 mmHg, $P0_2$ 176 mmHg, HCO_3 17.5 mmol/l, Lac 6.4 mmol/l, and glucose 318 mg/dl. In view of metabolic acidosis, we planned to shift him to intensive care units (ICU) on the ventilator and also considered renal replacement therapy (RRT). The patient was turned supine. There were good respiratory efforts and he opened his eyes on verbal commands but seizure activity was observed. Injection midazolam 2 mg followed by injection Levacetam 500 mg was given. The event was documented and the attendants were informed about the same. ICU team took charge of this patient. As the patient was being shifted to ICU, seizure activity was observed again. Injection midazolam 2 mg was repeated. Thereafter, seizure activity increased for which injection thiopentone sodium 200 mg was given. Neurological consultation was sought and electroencephalography (EEG) monitoring was started, which showed continuous seizure activity. Propofol infusion was started (2–10 mg/kg/h, titrated to hemodynamics) and immediate hemodialysis was planned.

ABG after hemodialysis – pH 7.23, P $C0_2$ 44 mmHg, PO_2 193 mmHg, HCO_3 19.4 mmol/l, and lactate 2.2 mmol/l.

However, the patient was kept intubated and was sedated on midazolam-fentanyl infusion in view of continuous seizure activity. He was started on injection Levacetam 500 mg intravenous twice daily, injection phenytoin 100 mg intravenous 8 hourly, and injection carbamazepine 500 mg intravenous twice a day. The seizure activity was controlled over a period of 72 h.

Computed tomography head showed cerebrocerebellar atrophic changes. Gliotic changes were seen in vermis and right cerebellar hemisphere. Cerebrospinal fluid (CSF) was sent for microscopic examination and it showed CSF proteins of 31.9 mg/dl. The glucose of 78 mg dl – inconclusive. The patient was managed on multiple antiepileptic drugs (AEDs) as per clinical response and EEG findings. MRI brain showed chronic right cerebellar hemorrhage with surrounding gliosis, post-operative changes in the right cerebellar hemisphere, and overlying subdural hygroma.

The patient was tracheostomised in view of prolonged ventilator support and a total of five hemodialysis were done during his stay in neuro-ICU. Neurological status was regularly monitored, antiepileptics were gradually tapered, and he was weaned off on the 7th post-operative day and decannulated on the 16th day. The neurological status was intact and he was discharged on the 20th day on two oral antiepileptics (phenytoin and carbamazepine).

DISCUSSION

Post-operative seizures, though rare, are a recognized complication of anesthetics.[3] Patients at risk include those with epilepsy, poor pre-operative control, and those undergoing brain surgeries. Rarely, seizures can occur as an isolated event in previously normal patient and this should prompt search for structural or chemical brain abnormality. Vigil needs to be exercised during post-operative period as numerous risk factors, can be potential cause of seizures.[1-3] These include:

1. Pre-existing seizure disorder
2. Lengthy surgery
3. Pre-existing brain tumor, aneurysm, or scarring
4. Alcoholism and its withdrawal
5. Illicit drug use
6. Local anesthetic systemic toxicity
7. Drug interactions
8. Chemical and electrolyte abnormality.

In the event of persistent seizures, intravenous benzodiazepines should be used (lorazepam or diazepam). If the seizures persist, a second dose of benzodiazepine with phenytoin (20 mg/kg over 30 min) may be used. For refractory seizures, phenobarbital (1.5 mg/kg/min or 100 mg/70 kg/min or a max of 20 mg/kg/min), midazolam (0.1–0.3 mg/kg in 3–5 min followed by an infusion of 0.05–0.4 mg/kg/h), thiopental (5–10 mg/ kg in 10 min followed by infusion of 100–400 mg/h), lidocaine (1.5–2 mg/kg in 2–5 min followed by an infusion of 2–3 mg/kg/h for 12 h), isoflurane (0.5–1.5%), and

ketamine (50–100 mg followed by 50–100 ml/h) can be used.

Due to pneumoperitoneum in laparoscopic surgeries, absorption of CO_2 by the peritoneum leads to increase in the IAP which is usually limited to 12–14 mm Hg. The induced high IAP and its consequences in modifications of organ perfusion and stimulation of major hormonal systems are associated with functional alterations of various organs.[4] Studies have shown the effect of elevated pneumoperitoneum on RBF. Changes in renal function also appear under laparoscopy but are difficult to assess as no reliable markers exist to monitor rapid changes in glomerular filtration rate in clinical practice. Furthermore, the head-up position should be avoided to prevent impeding the venous return further. The duration and intensity of pneumoperitoneum correlate with the risk of acute kidney injury.

Patients with CKD are often afflicted with neurological complications,[5] which include stroke, cognitive dysfunction, encephalopathy, peripheral, and autonomic neuropathies. The encephalopathy is due to the altered brain function induced by an agent or the condition known as posterior reversible encephalopathy syndrome, which can lead to altered mental status and motor disturbances.[6] The direct effects of uremia may be due to metabolites, guanidino compounds, fluid, and electrolyte disturbances. Iatrogenic causes include erythropoietin induced hypertension, polypharmacy, and transplant rejection.

The uremic toxins are likely to contribute to CNS injury either directly or indirectly; these toxins have been investigated for a possible role in direct neurotoxicity in the context of CKD.[7] Indirect effects of uremic milieu include their contribution to systemic inflammation, endothelial dysfunction, and atherosclerosis. Alterations in mental status can be accompanied by motor disturbances, including tremors, fasciculations, asterixis, disorientation, delirium, hallucinations, seizures, and coma.[8]

Fluid and electrolyte disturbances are frequent in patients with CKD and have adverse effects on CNS function. Dialysis disequilibrium syndrome results from rapid changes in urea and other osmolites during dialysis.[9] The osmotic gradient between the blood and brain causes cerebral edema, the presentation of which includes headache, tremor, disturbed consciousness, and convulsions.

In severe acute kidney injury, RRT is the only therapeutic option, which includes intermittent hemodialysis, continuous and intermittent hemofiltration, and hemodiafiltration. Early versus late initiation of RRT needs to be individualized, depending on the associated risk factors, use of clinical scoring system, biochemical markers, and response to drug therapy.[10]

In patients undergoing craniotomy, the risk of seizures is 6% during the 1st post-operative week and 17% over 5 years. Although anecdotal experience suggests that AEDs may stop perioperative seizures, neither valproic acid nor phenytoin given intraoperatively and during the post-operative period has been shown to prevent the development of epilepsy months to years later.[3]

CONCLUSION

Neurological complications are highly prevalent in CKD and are a major cause of morbidity and mortality. Acute encephalopathies may be caused by a variety of metabolic and pharmacological exposures; there may be rapid escalation to seizures or coma, superimposed on chronic conditions such as stroke, cognitive impairment, and dementia. Identification and risk stratification are crucial for the perioperative management of patients with CKD. To improve the clinical outcome, a thorough pre-operative assessment, renal function optimization, maintenance of hemodynamic stability, and avoidance of nephrotoxic drugs are desirable. Information regarding the need of RRT needs to be explained in advance.

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Case Report

Bilateral Hirayama Disease: A Case Report

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Abstract

Hirayama disease is a rare restricted form of motor neuron disease. It commonly affects young males. Patients typically present with the insidious onset of unilateral weakness and atrophy of the hand muscles that often progresses to the forearm. In some cases, the syndrome is bilateral but often asymmetrical. Of note, the brachioradialis muscle is usually spared. The syndrome affects C7–C8–T1 muscles with sparing of the C5–6 muscles. We report a case of a 25-year-old male who presented with 2 years of history of progressive wasting and weakness of muscles of bilateral hands and forearms. Based on clinical features, electrodiagnostic studies and dynamic magnetic resonance imaging cervical spine diagnosis of Hirayama disease were made. The patient was treated conservatively with a cervical collar. Over a period of 8 months follow-up, no progression was seen.

Key words: Hirayama disease, Juvenile non-progressive amyotrophy, Oblique amyotrophy, Snake eye appearance

INTRODUCTION

Hirayama disease also known as monomelic amyotrophy, Sobue disease, juvenile segmental muscular atrophy, or benign focal amyotrophy was first described by Keizo Hirayama as juvenile muscular atrophy of unilateral upper extremity.[1] It is a very rare benign neurological disorder, mainly affecting young males in the second or third decade of life. The onset of this disease usually corresponds to the beginning of the adolescent growth spurt. The distinctive clinical features include insidious onset and slow progression of muscular atrophy with weakness of the forearms and hands, the muscular atrophy reaches a plateau phase after 2–5 years of the onset of disease after which this disease neither improves nor worsens. Sensory deficits are generally absent although some patients may experience paresthesia. It caused by dynamic compression of the lower cervical cord resulting from sustained or repeated neck flexion. The pathologic finding is ischemic changes in the anterior horn cells of the localized lower cervical cord. Dynamic magnetic resonance imaging (MRI) (neutral and flexed) of the cervical spine has become the mainstay for confirming clinical diagnosis. For decades, the disease was thought to be unilateral, later on, bilateral cases were described by many authors as part of short or long case series but it was relatively uncommon compared to unilateral cases. In cases of bilateral diseases, it is often asymmetric and very rarely symmetric.

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CASE REPORT

A 25-year-old male reported with a 2-year history of asymmetric slowly progressive weakness and atrophy of both distal upper limbs that started first in the left hand and the forearm and 6 months later involved the right hand and the forearm. The hand weakness limited several activities of his daily living. He complained of paresthesia in bilateral hands but was able to perceive all sensations normally. There was no history of pain in the neck or radicular symptoms. There was no history of trauma, febrile illness, exposure to toxins, or heavy metals in the past. His past medical history was non-contributory, he denied any addiction, and none of his family members had similar symptoms.

On examination, there was striking muscular atrophy and weakness affecting bilateral hands and forearms left more than the right [Figures 1 and 2] with preserved bulk of brachioradialis giving the characteristic appearance of oblique amyotrophy [Figure 3]. The bulk of arms and shoulders was maintained. The rest of the neurological and general examination was normal.

Results of routine blood analysis were normal, there were negative results for vasculitis screening (rheumatoid factor,

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antinuclear antibody, extractable nuclear antigens, and antiphospholipid antibody) and viral serology: Human immunodeficiency virus, hepatitis B, and hepatitis C.

Serum creatine phosphokinase was mildly raised (210 IU/L), while other markers of muscle injury myoglobin, lactate dehydrogenase, and aspartate aminotransferase were within the normal range.

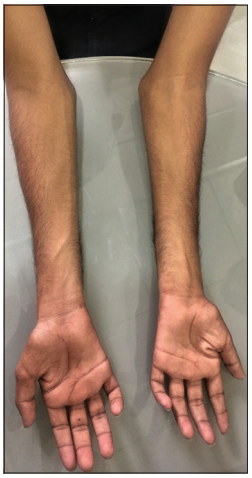

Figure 1: Bilateral wasting of muscles of hands and forearms, left more than right is seen

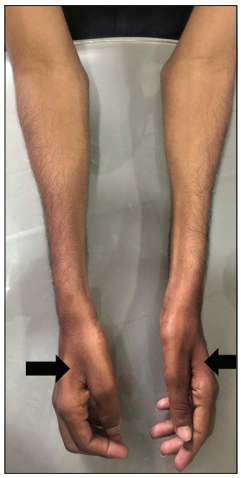

Figure 2: Bilateral wasting of 1st dorsal interossei (arrow heads) is seen along with wasting of other muscles of hands and forearms

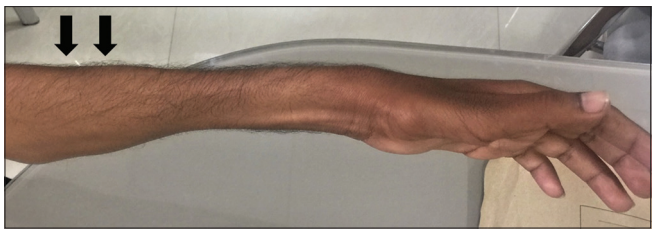

Figure 3: Preserved bulk of brachioradialis (arrow head) with wasting of muscles of hand and forearm is seen, producing a characteristic muscle wasting pattern of the forearm called "oblique amyotrophy"

On electrodiagnostic (EDX) studies, bilateral median and ulnar nerve conduction revealed normal distal motor latencies, low compound motor action potential (CMAP) amplitudes, and conduction velocities. The CMAP amplitudes were relatively lower on the left side. Bilateral median and ulnar nerves sensory onset latencies, sensory nerve action potentials (SNAP) amplitudes, and conduction velocities were normal. Needle electromyography (EMG) exam revealed fibrillations, large-amplitude, and prolonged-duration MUAPs with reduced recruitment suggestive of a chronic axonal neurogenic process in bilateral C8-T1 more than C7 innervated musculature, bilaterally along with the mild active denervation in some of the C8-T1 innervated muscles. The process was more severe on the left side.

The X-ray cervical spine showed a straightening of cervical spine curvature.

Dynamic MRI cervical spine study was done, which revealed the loss of cervical spine lordosis. Localized cervical cord atrophy at the C5-C7 levels was noted, which was asymmetrical on the axial image with a pear-shaped or triangular on axial images [Figure 4]. On sagittal T2-weighted images (T2WI), intramedullary linear hyperintensity extending from C5 to C7 was seen. On axial T2WI bilateral symmetrical small hyperintense foci in the anterior horn cells of the cervical spinal cord extending from C5 to C7 vertebrae were seen, giving the characteristic snake eye appearance [Figure 5]. On flexion study, mild prominence of the posterior epidural space was noted.

Based on clinical, EDX studies and radiological findings, the diagnosis of bilateral Hirayama disease was made.

The patient was managed conservatively with a cervical collar, muscle strengthening exercises, and training in hand-coordination. Over 6 months of follow-up, no worsening is seen.

DISCUSSION

Hirayama disease and the terms benign focal amyotrophy, brachial monomelic amyotrophy, or juvenile segmental muscular atrophy are used to describe a rare disorder characterized by lower motor neuron disease clinically restricted to the distal upper limb. Most cases are sporadic, although a familial form has been reported. The etiology is unknown. Autopsy studies have shown the affected region of spinal cord flattened, the anterior horn markedly atrophied and gliotic, and a reduction in the numbers of both large and small motor neurons. Based on neuroradiological studies, Hirayama, who established the disease entity, has proposed a mechanically induced limited form of ischemic cervical myelopathy, being the

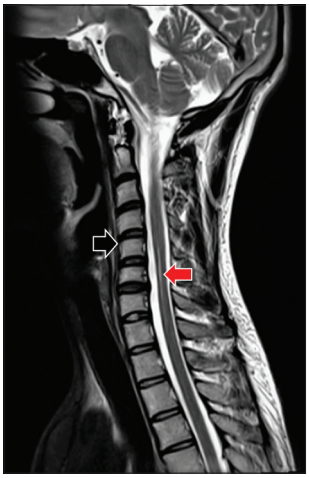

Figure 4: Sagittal T2-weighted magnetic resonance images showing loss of cervical lordosis (white arrow) with focal atrophy and intramedullary hyperintensity and focal atrophy extending from C5 to C7 level (red arrow)

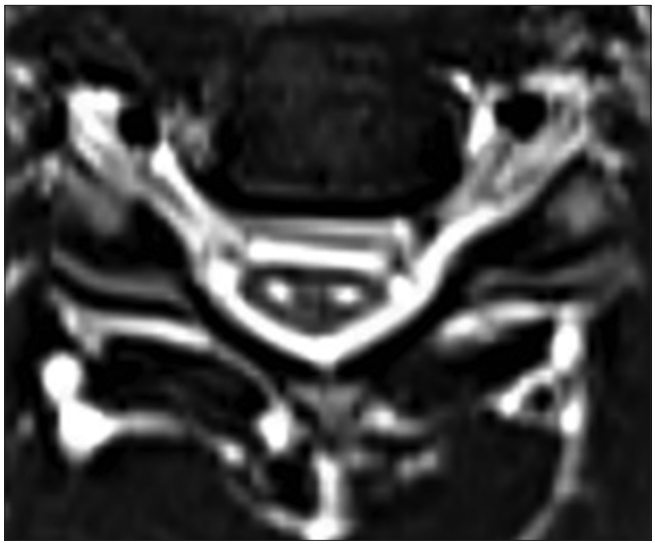

Figure 5: Axial T2-weighted magnetic resonance images at C6 level showing, bilateral symmetrical small hyperintense foci in the anterior horn cells showing characteristic "snake eye appearance" with pear shaped/ triangular anteroposterior cord flattening

result of local compression of the dura and spinal cord against vertebrae during repeated neck flexion/extension, in turn, due to disproportionate growth between the contents of the dural sac and the vertebral column.[2,3] Toma and Shiozawa proposed that the disproportionate shortening of the dural sac is accentuated during the juvenile growth spurt.[4] Another school of thought is that this is a segmental, perhaps genetically determined, spinal muscular atrophy, but the actual cause is still unknown. The disease usually begins in the late teens, but many cases can present in the fourth decade. More than 60% of patients are men. Although originally described in Indian and Japanese patients, the disorder is now recognizable around the world.

The most common presentation is one of an idiopathic, slowly progressive, painless weakness, and atrophy in one hand or forearm. The most common pattern is unilateral atrophy of C7–T1 innervated muscles, with sparing of the brachioradialis referred as the "oblique atrophy" pattern. Muscle stretch reflexes are invariably hypoactive or absent in the muscles innervated by the involved cord segment but are normal elsewhere. UMN signs are not present, and if they are, one should consider the onset of amyotrophic lateral sclerosis (ALS) instead. Approximately 20% have hyperesthesia to pinprick and touch, usually located on the dorsum of the hand. The cranial nerves, pyramidal tracts, and the autonomic nervous system are normal. Weakness and atrophy may progress steadily for the initial 2–3 years, but most patients have stabilized within 5 years. In some patients, there is an aggravation of weakness when exposed to cold, a phenomenon known as cold paresis. Spread may occur to the contralateral limb in about 20% of cases.[5]

No pathognomonic laboratory or EDX tests exist for this condition; their main purpose is to exclude alternative diagnoses.

Motor nerve conduction studies are either normal or may reveal asymmetrically low median or ulnar CMAP amplitudes in the affected hand; normal or a modest reduction in SNAPs occurs in up to one-third of patients. The EMG examination may show some fibrillation and fasciculation potentials, and chronic neurogenic motor unit changes are prominent.

The serum creatine kinase concentration may be modestly elevated, but other routine laboratory test results are normal.

Cervical MRI reveals various findings on neutral and flexion positioning. On neutral MRI, localized lower cervical cord atrophy, asymmetric cord flattening, parenchymal changes in the lower cervical cord, abnormal cervical curvature, loss of attachment between the posterior dural sac, and subjacent lamina have been described.[6] Among these, localized lower cervical cord atrophy, asymmetric cord flattening, and loss of attachment have an accuracy of 80% in the identification of the disease; loss of attachment is the most valuable finding for diagnosing Hirayama disease in the neutral position.[6,7] On flexion MRI, forward migration of the wall of the dura mater is observed with an enlarged posterior epidural space.[2,8,9] A hyperintense, crescentic epidural mass showing curvilinear flow voids, and uniform enhancement after administration of contrast is seen in the posterior epidural space.[9]

Snake-eyes appearance (SEA) is a unique radiological finding characterized as a symmetrical bilateral small high

signal intensity lesion on an axial T2-weighted MRI and is named because of its similar appearance to the eyes of a snake. The description of the SEA was initially given in a computed tomography myelography study of seven cervical spondylotic myelopathy patients in the 1980s.[10] Its pathologic result is cystic necrosis at the junction of the central gray matter and the ventrolateral posterior column and loss of anterior horn cells.[11] SEA is an irreversible lesion, appears late in the course of Hirayama disease, and it signifies a poor prognosis.[12]

Two diseases require distinction from Hirayama disease: ALS, which is almost always a relentlessly progressive terminal disease and MMNCB, which is a treatable peripheral motor neuropathy. A small proportion of ALS presents as Hirayama disease, albeit in an older patient population. It is only with follow-up examination that the more widespread anterior horn cell disorder becomes apparent and upper motor neuron signs appear. Deep tendon reflexes are almost always hyperactive early in the evolution of ALS. Furthermore, the EDX finding of generalized widespread acute and chronic motor neuron loss distinguishes ALS from the segmental motor neuron involvement of benign focal amyotrophy. The slowly progressive focal weakness that is distinctive of benign focal amyotrophy may also be the presenting picture of MMNCB, but detailed motor nerve conduction studies and serum tests for elevated titers of anti-GM1 antibodies can differentiate these two conditions.

Cervical radiculopathy may also appear in a manner somewhat akin to Hirayama disease. However, radicular pains and sensory impairment are typical of radiculopathies. Cervical syringomyelia or a benign tumor involving nerve roots or the spinal cord may also cause progressive weakness in a monomelic fashion. Careful EMG studies and neuroimaging should differentiate these diseases.

Hirayama disease is not life threatening, but it nevertheless severely impairs motor function in the involved extremity, although most patients adapt very well to their disability. Supportive care consists of physical and occupational therapy and effective use of assistive devices such as splinting and braces. Application of a cervical collar is believed to prevent the progression of the disease in early stages.[13] In selected cases, not responding to conservative measures duraplasty, anterior cervical decompression, and reconstructions with tendon transfers have yielded encouraging results.[14] Tendon transfers are a consideration in selected patients with focal weakness in a muscle group whose function is crucial for certain activities of daily living.

CONCLUSION

Hirayama disease is a rare self-limiting disease but disabling. Early diagnosis is important as the use of a simple cervical collar which will prevent neck flexion, has been shown to stop the progression. While dynamic contrast MRI shows characteristic findings, routine MRI has a high predictive value for diagnosis; in all clinically suspected cases of Hirayama disease, dynamic flexion study should be done as it aids diagnosis in patients of focal wasting if routine imaging is normal. All young adolescents with focal upper limb wasting should be evaluated to exclude Hirayama disease, a benign condition amenable to collar therapy.

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Review Article

Seafood Allergy: Causes, Prevention Modalities, and Treatment Guidelines in the Indian Scenario

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Abstract

Seafood allergy is a hypersensitivity disorder with growing prevalence. Allergy to shellfish is among the leading cause of food allergy in adults, and the most common cause of food allergic emergency department visits. Seafood allergy is immunologic response to proteins in these foods and include IgE antibody-mediated allergy. Allergies can occur at any age but are common in adults and adolescents than in children. While figures vary from country to country, approximately 1–2% of the adult population and <1% of children are affected. In most patients tolerance develops to food antigens, however, when tolerance fails to develop, hypersensitivity reaction occurs. Food allergy affects up to 8% of the children below 5 years of age and approximately 3.5% in the general population. Adults with shellfish allergies should be aware of how to use this on themselves or their child if child is suspected shellfish allergy. It is also recommended for such individuals to wear medical alert bracelet necklace or carry USB drive so that health care worker can be aware of their condition in emergency. Effective and accurate diagnostic workup is essential for clinicians and patients. This article summarizes about seafood allergy cause, diagnostic approaches, and management in case of life-threatening emergencies.

Key words: Anaphylaxis, IgE, Inj. EpiPen, Parvalbumin, Seafood allergy, Tropomyosin

INTRODUCTION

Seafood allergy is the common food allergy among the most prevalent food allergies in young children and adults.[1]

The term "seafood" encompasses the following:

- Vertebrate finned fish, such as salmon, tuna, and cod
- Crustaceans, such as shrimp, prawn, crab, lobster, and crawfish
- Mollusks, such as squid, snails, and bivalves.

The term "shellfish," a subset of seafood, includes crustaceans and mollusks.

Seafood allergy is immunologic response to proteins in these foods[2-5] and includes IgE antibody-mediated allergy as well as other allergic syndromes. They are distinct from adverse reactions due to toxins or infectious contaminants, which are not immune based.

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Allergy to seafood is very common. Allergy to sea food is very common, it is estimated to affect 1-2% of adult population and <1% of children.[6-9] Allergy to shellfish is among the leading causes of food allergy in adults, and the most common cause of food allergic emergency department visits.[10]

Adverse food reactions are common and often assumed by patients to be allergic in nature. Food allergies are adverse immune responses toward food proteins.[6]

Sensitization to food allergens may occur in the gastrointestinal tract considered as traditional or Class 1 food allergy or a consequence of allergic sensitization to inhalant allergens considered as Class 2 food allergies.

Several Class 1 and 2 food allergens have been identified. The variety of animal-related allergens appears to be limited in number and cross-reactivity.

PATHOGENESIS

The major allergic proteins in seafood that are responsible are IgE mediated and non-IgE mediated. Persons with seafood allergy may react to these and/or other seafood proteins.

Parvalbumin is the important allergen in fish.[11]

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Tropomyosin is major allergens in shellfish and is also present in other arthropods accounting for cross-reactivity between these groups.[12,13]

In shrimp muscle protein, myosin light chain and sarcoplasmic calcium-binding protein are prominent allergens.[14,15]

The pathogenesis of non-IgE-mediated seafood allergy is unclear. A syndrome that clinically resembles protein-induced enterocolitis has been described in both children and adults.

CLINICAL FEATURES

Three types of reactions have been recorded.

IgE-mediated Reaction

IgE-mediated allergic reactions are the most commonly described type of allergic reactions to seafood ingestion. These may present as generalized reactions, asthmatic reactions in response to occupational or household exposures, or as food-dependent, exercise-induced anaphylaxis.

IgE-mediated reactions are rapid in onset (usually within minutes to an hour after ingestion) and extra-gastrointestinal manifestations, such as urticaria, angioedema, respiratory symptoms, and laryngeal edema, are common. These can range in severity from mild to life-threatening anaphylaxis; severe reactions are not uncommon.[16,17]

Occupational and household exposures involving inhalation of cooking or processing vapors may cause asthma and allergy.

Anaphylaxis

Food-dependent, exercise-induced anaphylaxis to seafood has also been reported.[18] In this condition, the food causes symptoms only if ingestion is followed soon after by exercise or exertion but is tolerated in the absence of exertion. Wheat products and seafood are the two most frequently implicated foods in this disorder.

Severity – IgE-mediated fish and shellfish allergies can vary from mild to severe. In the US prevalence study, 60–70% of respondents experienced urticaria/angioedema, and over one-half reported dyspnea or throat tightness. Consistent with this, approximately one-half of reactions prompted evaluation by a clinician or care in an emergency room.

Non-IgE-mediated Reaction

There are several other reactions to seafood exposure that is not IgE mediated. These include:

Gastrointestinal reactions – food protein-induced enterocolitis[19] has been described in children in response to fish, although there is a paucity of data on this entity. In the largest series of 14 children diagnosed between the ages of 9 and 12 months of age, symptoms consisted of vomiting, diarrhea, or both. Three presented with a sepsis-like picture. Reactions occurred from a few minutes to 6 h after ingestion. Skin prick tests were negative in all patients, and fish-specific serum IgE was positive in just one patient. Nine had the reactions confirmed by oral challenge. Four of these children eventually became tolerant of the causal food.

Seafood may also cause an enterocolitis-like disorder in adults, with delayed onset of nausea, crampy abdominal pain, and protracted vomiting or diarrhea. This appears to be primarily reported in response to mollusks. Adults typically present after having experienced this on several occasions, reporting that they had attributed the first one or two reactions to possible food poisoning.

The etiology of these enterocolitis-like reactions in adults is unclear. Their repeated occurrence in certain individuals suggests either that some persons are more susceptible to toxic components in these foods or that this reaction represents a form of allergy. There are very few studies of the pathogenesis of these reactions, although one study of adults with isolated gastrointestinal symptoms to various types of seafood included six patients who reacted repeatedly to oyster, in whom specific IgE was generally undetectable.[20]

Allergic contact dermatitis – allergic contact dermatitis[21] can result from occupational skin exposure to seafood, in food handlers. Skin barrier disruption has been implicated.

DISCUSSION

All food allergies are caused by an immune system problem. Your immune system identifies certain shellfish proteins as harmful, triggering the production of antibodies to the shellfish protein (allergen). The next time you meet proteins in shellfish, these antibodies recognize them and signal your immune system to release histamine and other chemicals that cause allergy symptoms.

Shellfish/prawns are considered a major cause of food allergic reactions in adults, affecting 4–5% in the Indian population. Variety of seafood has been studied (snail, oysters, prawns, crabs, and lobsters). Shrimp allergens have been extensively studied.

Tropomyosin[11,12] a protein found in the muscle has been identified as a major allergen in shrimp. Invertebrate

tropomyosin is highly homologous and tends to be allergic to those from the crustaceans (shrimp, prawns, crab, crawfish, and lobster), whereas vertebrate tropomyosin tends to be non-allergic.

Histamine and other body chemicals cause a range of allergic signs and symptoms. Histamine is partly responsible for most allergic responses, including running nose, itchy eyes, dry throat, rashes and hives, nausea, diarrhea, difficulty in breathing, and in some cases, anaphylactic shock.

Some people are allergic to only one type of shellfish but can eat others. However, some people with a shellfish allergy must avoid all varieties of shellfish.

Although people of any age can develop a shellfish allergy, it is most common in adults. Among adults, shellfish allergy is more common in women. Among children, shellfish allergy is more common in boys.

Prawn Allergy

Some of the symptoms of prawn allergy that can occur immediately after eating prawn or after few hours are as follows:

- Hives and rash on skin: Prawn allergy rash can occur all over the body, it is associated with immense itching. It can be due to eating prawns or even meeting a prawn
- Sneezing and watering of eyes
- Cough and breathing difficulty
- Swelling over face and other parts of body.

In severe cases, prawn allergy can lead to anaphylaxis, a dangerous allergic reaction marked by a swollen throat (airway constriction), rapid pulse, shock, and dizziness or light headedness. Anaphylaxis can be life threatening.

When you have prawn allergy, you may be at increased risk of anaphylaxis if:

- You have asthma
- You have allergic reactions to very small amounts of shellfish (extreme sensitivity).

SUMMARY

Ingested food represents foreign antigenic load. In most of the patient's, tolerance develops to food antigens; however, when the tolerance fails to develop the immune system respond with hypersensitivity reaction. Food allergies affect up to 8% children below the age of 5 years. and approximately 3.5% in the general population. Inadvertent ingestion of food allergens may provoke symptoms related to gastrointestinal and respiratory system, dermatological symptoms, anaphylaxis, and shock.

PREVENTION IS BETTER THAN CURE

Mayo Clinic Guidelines

If you know you are allergic to shellfish, the only sure way to avoid an allergic reaction is to avoid all shellfish or products that might contain shellfish. Even trace amounts of shellfish can cause a severe reaction in some people. Shellfish are not usually a hidden food ingredient, so it may be easier to avoid than some other allergy causing foods.

The federal Food Allergen Labeling and Consumer Protection Act requires that any packaged food product that contains shellfish as an ingredient must list the name of the specific shellfish on the label. Please be sure to read all product labels carefully before purchasing and consuming any item. Remember, also, that ingredients change from time to time, so check labels every time you shop. If you are still not sure whether a product contains shellfish, call the manufacturer. Always take extra precaution when dining in restaurants or eating foods prepared by others. If you are ever in doubt about any product or dish, do not eat it.

Measures for Avoiding Shellfish

Be cautious when dining out

Eating at restaurants poses the biggest danger of mistakenly eating shellfish. When you eat at restaurants, always check and make sure the same pan, oil or utensils used for shellfish are not also used to prepare other foods. This is called cross-contamination.

Use extra caution at seafood restaurants

Fish and shellfish are biologically distinct, so fish will not cause an allergic reaction if you have a shellfish allergy – unless you are also allergic to fish. However, when eating at a seafood restaurant, there is a higher risk of cross contamination of your food with trace amounts of shellfish. Some people even have allergic reactions to cooking vapors.

Read labels

Cross-contamination can occur in stores where food may be processed or displayed along with shellfish. It also can occur during manufacturing. Be sure to read food labels carefully. Companies are required to clearly label any product that contains even small amounts of shellfish or other foods that often cause allergic reactions.

Keep distance

You may need to completely avoid environments where shellfish are being prepared or processed. Some people even have a reaction after touching shellfish or inhaling steam from cooking shellfish.

Some people mistakenly believe that allergy to iodine or allergy to radiocontrast dye used in some laboratory

procedures can cause reactions in people with a shellfish allergy. Reactions to radiocontrast material or iodine are not related.

POST SCRIPT - A NOTE TO THE ORTHOPAEDICIAN

Glucosamine, a supplement used to prevent and treat arthritis, is made from crab, lobster, or shrimp shells. While it does not appear to cause an allergic reaction in most people who have a shellfish allergy, more studies need to be done to determine whether it is safe for people allergic to shellfish.

MED ALERT BRACELETS, NECKLACES, AND MEDICAL IDENTIFICATION TAGS

The intention is to alert a paramedic, physician, emergency department personnel, or other first responders of the condition even if the wearer is not conscious enough or old enough to explain. Some people prefer to carry a wallet card with the same information.

A new type of medic identification alert is the USB medical alert tag. This is essentially a USB flash drive that contains an individual's emergency information and can carry much more information than the conventional medical ID bracelet. Information such as medications, existing conditions, doctors, and emergency contacts can all be stored on the USB tags.

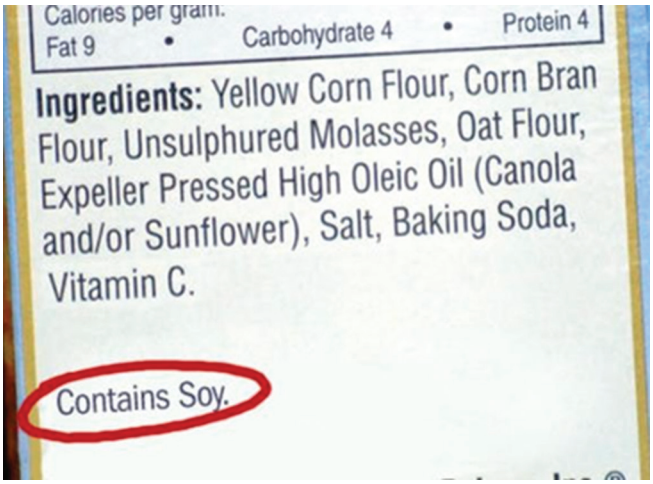
Use of Inj. EpiPen (Epinephrine-Adrenaline)

If you are at risk of a serious allergic reaction, talk with your doctor and relatives about carrying Inj. EpiPen (emergency epinephrine adrenaline).

1. RECOGNIZE the signs and symptoms are life threatening or not
2. REACT quickly activate the emergency care plan
3. REVIEW what caused the reaction and do the plan work.

Recommendations for India

1. All the restaurants who serve the seafood should notify the customers regarding the use of seafood in the menu
2. Warning should be given to customers regarding the allergic reactions
3. All the restaurants who serve the seafood should maintain the EMERGENCY DRUGS as a precautionary measure as emergency can occur
4. Health education must be given to the asthmatic, nasal allergy patients as they have bit higher incidence of seafood allergy
5. Patients who consume wine also should be alerted as fish products can be used as clarifying or fining agents in the manufacture of some wines
6. Medications, various health foods, and cosmetics may have ingredients derived from seafood, and labeling of non-food items should by strictly regulated by the government department and other health organizations to avoid unpleasant reactions.



IS THERE A TREATMENT FOR SEAFOOD ALLERGY

Shellfish are divided into two main groups. The crustaceans include crab, shrimp, and lobster, and the mollusks include scallops, clams, mussels, and oysters. Typically, it is seen that the people who are allergic to a certain kind of seafood will also be allergic to others within the same group. It is not so common to be allergic to all types of seafood. However for the individuals who are allergic to any one seafood, it is recommended to consult a doctor before they attempt to eat any other seafood. Although the best way to prevent any form of allergic reaction is to keep away from all forms of shellfish, there are instances in which one inadvertently consumes them. The best way to treat mild allergic reactions such as itching and rash is to take antihistamines such as loratadine or diphenhydramine. Applying calamine lotion and ice on the hives also helps to reduce the discomfort by shrinking the blood vessels and hence reducing the inflammation. Taking milk of magnesia and peppermint tea also helps to relieve the milder symptoms. In case of a severe allergic reaction like anaphylaxis, it is always required to take an injection of epinephrine or adrenaline and carry injectable epinephrine or an EpiPen with them. Epinephrine needs to be taken as soon as an allergic reaction is suspected. Epinephrine is a very powerful bronchodilator that can combat the effects of the potentially life-threatening anaphylactic shock. Hence, adults with shellfish allergies should be aware of

how to use this on themselves or on their child if the child has a suspected shellfish allergy. It is also recommended for such individuals to wear a medical alert bracelet or necklace so that health care workers may be aware of their condition as emergency. Moreover, we must follow the adage that PREVENTION IS BETTER THAN CURE.

PERSISTENCE, TOLERANCE, AND RECURRENCE

Seafood allergy is considered to be persistent in most cases. In a study evaluating IgE binding to various epitopes of major shrimp allergens, children showed stronger and more diverse IgE binding than adults, implying the allergy may wane with time. American telephone-based survey, only 3–4% of individuals with seafood allergy reported developing tolerance overtime. However, loss of fish allergy during childhood or in adulthood has been reported, although the extent to which this occurs is not well studied. Recurrence of fish allergy after tolerance has also been reported

DEATH RATES

Several deaths from seafood allergy have been recorded. In a registry of food-induced fatal anaphylaxis comprised primarily of children, 1 of 32 deaths was due to fish, and in a report of seven deaths, one was reported to crab and one to fish. In a United Kingdom registry of fatalities from anaphylaxis, 3 of 33 fatalities to a known food were caused by seafood.

THE BOTTOM LINE: BE CAUTIOUS

Always double check labels to be sure you know what you are eating and drinking. Even though a food product may have been safe the last time you purchased or consumed it, it is possible that the ingredients have changed, or the label has been updated. If you have any doubt about food ingredients, contact the manufacturer about whether the food could possibly contain a food allergen. In this era of mobile phones with camera please photograph the labels to see the changes in the label or food constituents and compare to see the changes.

FUTURE STRATEGIES

New strategies may help overcome food allergies.

- Oral immunotherapy: Under close supervision by health-care professionals, patients swallow tiny but gradually increasing amounts of the foods that trigger their allergies, with the idea of building immunity. This method is being tested for peanut, egg, and milk allergies
- Sublingual therapy: Drops containing proteins that trigger allergies are put under the tongue, where they are absorbed into the bloodstream. This method is being tested for various food allergies
- Food allergy herbal formula 2 (FAHF-2): Known as FAHF-2, this pill (not available in stores) is based on a 2000-year-old Chinese remedy. It contains nine botanicals, including ginseng and oil made from cinnamon tree bark. It is being tested for peanut, tree nut, fish, and shellfish allergies.

CONCLUSION

Seafood allergies are most common in young adults and adolescents but can also be seen in children. The allergic reaction is because of hypersensitivity reaction to the proteins present in these foods which are mainly the parvalbumin and tropomyosin which induce IgE-mediated immunologic response and cause release of histamine and other chemical mediators from the mast cells, causing mild to severe allergic reaction and in some cases severe anaphylactic reaction. Thus, the main *per se* treatment is prevention and we must follow the adage that PREVENTION IS BETTER THAN CURE. In cases with mild allergy, symptomatic treatment is given. In severe anaphylaxis, Inj. EpiPen is used as an emergency drug. There are many strategies coming up in future and building tolerance in the children to overcome their food allergies is one of the efforts taken.

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Original Article

Brachial Plexus: Variations in Infraclavicular Part and their Significance

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Abstract

Introduction: Brachial plexus extends from the neck to the axilla and gives motor and sensory nerves to the upper limb. The brachial plexus has two parts supraclavicular and infraclavicular parts.

Material and Methods: The present study was carried out on 15 adult human cadavers (15 specimens of the right and left side of the upper limb) in the Department of Anatomy, Government Medical College and Hospital, Chandigarh. All the cadavers were male.

Observations: Of 30 cases, one specimen (3.34%) had variation in the arrangement of all cords (lateral, medial, and posterior) respective to the 3rd part of axillary artery, i.e., all three t cords lie lateral to 3rd part of axillary artery. In one case (3.34%), it was observed that lateral cord gave separate branch to coracobrachialis muscle. Musculocutaneous nerve passes straight downward placed between biceps brachii and brachialis. Musculocutaneous nerve continues as lateral cutaneous nerve of forearm. In another two cases (6.66%), there is communication between musculocutaneous and median nerve. Five cases (16.66%) had variations in the formation of median nerve. Here, median nerve was formed from three roots of two roots were originating from lateral cord and one root from medial cord. In three cases (10%), there is higher origin of median nerve by joining of its two roots. In one case (3.34%), there are two upper subscapularis nerves from the posterior cord. Other branches of this cord are lower subscapular, nerve to latissimus dorsi, radial, and axillary nerve which is normal in their positions.

Discussion: Development of brachial plexus starts at 34th–35th day of intrauterine life and definitive adult pattern is visible by 46th–48th day of intrauterine life. The growing axons are regulated by chemoattractants and chemorepulsants in a site-specific fashion. Changes in the signaling pattern between mesenchymal cells and neuronal growth cones can lead to variations.

Conclusion: These variations are important for surgeons, neurosurgeons, and plastic surgeons in their respective field in knowing area of loss, loss of muscle power as well as in designing various surgeries.

Key words: Brachial plexus, Infraclavicular, Median nerve, Musculocutaneous nerve

INTRODUCTION

Brachial plexus extends from the neck to the axilla and gives motor and sensory nerves to the upper limb. It is formed by the union of the ventral rami of the lower four cervical and first thoracic spinal nerve (C5, 6, 7, 8, and T1).[1] The brachial plexus has two parts supraclavicular and infraclavicular parts. The supraclavicular part consists of roots, trunks, divisions, and some branches. The infraclavicular part of brachial plexus consists of three cords, lateral, medial, and posterior.[2] Cords of brachial plexus had same relation corresponding to their name with third part of axillary artery. Lateral cord is formed by the anterior division of upper and middle trunks and it lies lateral to the third part of axillary artery. The medial cord is formed by the anterior division of lower trunk and it maintains medial position in respect to third part of axillary artery. The posterior cord is formed by the union of all three posterior divisions of upper, middle, and lower trunks. It lies behind the third part of axillary artery; the branches of the posterior cord lie posterior to the third part of axillary artery.[3]

Each cord of the brachial plexus has its own branches which supply the different compartment muscles or structures of

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the upper limb. The present study was undertaken to observe variations in the cords, branches of each cord, and relation of these cords with the third part of axillary artery. Variations in the branches were observed up to the cubital fossa.

Hence, knowledge of variations is important for diagnosis based on clinical symptoms, during procedures like anesthetic blocks and designing surgical approaches for nerve compressions caused by tumor or trauma. Unawareness of these variations can cause irreversible damage to the structure during surgical procedure.

MATERIALS AND METHODS

The present study was carried out on 15 adult human cadavers (30 specimens of the right and left side of the upper limb) in the Department of Anatomy, Government Medical College and Hospital, Chandigarh. All the cadavers were male. Cadavers with no injury and disease in the neck, axilla, pectoral region, and upper limb were included in the study. Dissection was done according to Cunningham's manual of practical anatomy. The study was done to observe the variations in the infraclavicular part of brachial plexus. The observations were made carefully and recorded. Photographs of dissected parts were taken. The data obtained were compared with those of earlier workers on this topic, and an analysis of the findings was done.

Observations

Normally all three cords (lateral, medial, and posterior) are placed according to their name (lateral, medial, and posterior) to 3rd part of axillary artery.

Of 30 cases, only one specimen (3.34%) [Figure 1], had variation in the arrangement of all cords (lateral, medial, and posterior) respective to 3rd part of axillary artery, i.e., all cords lie lateral to 3rd part of axillary artery.

In the rest of 29 cases (96.6%), all cords were placed in their normal position in relation to 3rd part of axillary artery.

Normally, lateral cord gives three branches lateral pectoral, musculocutaneous, and lateral root of median nerve. Musculocutaneous nerve (C5, C6, and C7) pierces the coracobrachialis muscle and supplies it, passes between biceps brachii and brachialis muscle. Thereafter, it continues as lateral cutaneous nerve of forearm.

In one case (3.34%) [Figure 2], it was observed that lateral cord gave a separate branch to coracobrachialis muscle. Musculocutaneous nerve passes straight downward placed between biceps brachii and brachialis. Musculocutaneous nerve continued as a lateral cutaneous nerve of forearm.

In another two cases (6.66%) Figure 3, there was communication between musculocutaneous and median nerve. Normally, there is no communication between these nerves.

Median nerve is usually formed by the contribution of both lateral and medial cord (C5, C6, C7, C8, and T1) of brachial plexus. Lateral root (C5, C6, and C7) arises from lateral cord and medial root (C8 and T1) arises from medial cord of brachial plexus.

In the present study, five cases (16.66%) [Figure 3] had variations in the formation of median nerve. Here, median

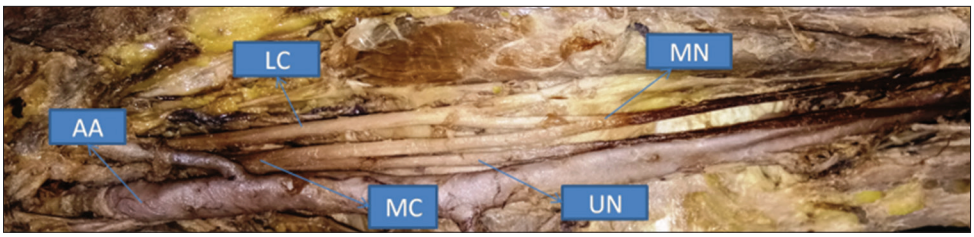

Figure 1: Infraclavicular part of brachial plexus showing all three cords are present lateral to axillary artery. AA: Axillary artery, LC: Lateral cord, MC: Medial cord, MN: Median nerve, UN: Ulnar nerve

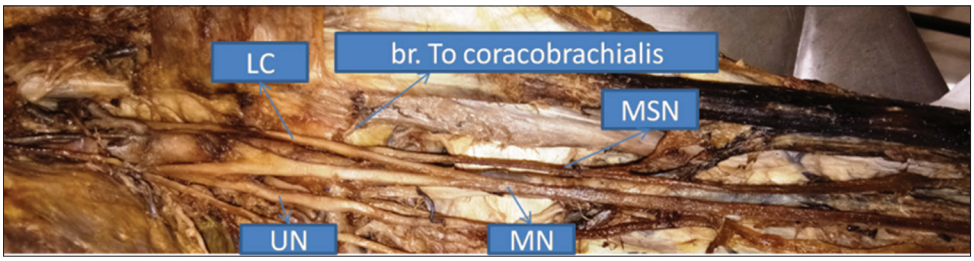

Figure 2: Infraclavicular part of brachial plexus showing separate branch to coracobrachialis muscle. LC: Lateral cord, MSN: Musculocutaneous nerve, UN: Ulnar nerve, MN: Median nerve

nerve was formed from three roots of two roots were originating from lateral cord and one root from medial cord. In these cases, lateral root from lateral cord united with medial root from medial cord to form median nerve. Later some fibers, i.e., extra root from lateral cord united with median nerve at lower level.

In three cases (10%) [Figure 4], there was a higher origin of median nerve by joining of its two roots. Normally, the formation of median nerve was at lower level and lateral to ulnar nerve.

In one case (3.34%) [Figure 5], there were two upper subscapularis nerves from posterior cord. Other branches of this cord were lower subscapular, nerve to latissimus dorsi, radial, and axillary nerve which are normal in their positions.

DISCUSSION

The brachial plexus is formed by ventral rami of C5–T1 spinal nerves and is for the nerve supply of the upper limb structures. This plexus may have a contribution from C4 or T2. Of these, two contributions of T2 spinal nerve are less common. C7–C8 ventral rami are largest and the smallest ventral rami are C5–T1. Three trunks, namely, upper, middle, and lower trunks, are formed by the union of these ventral rami, deep to scalenus anterior. The upper trunk is formed by spinal C5 and C6, the middle trunk is formed by continuation of spinal nerve C7. The lower trunk is formed by the union of C8 and T1. Each trunk further divides into anterior and posterior division. The formation of trunks is fairly consistent; they lie in front of one another rather than side by side, with the subclavian artery passing anteromedially. The phrenic nerve crosses C5 to pass anteromedially on the surface of scalenus anterior. The upper trunk divides above the clavicle and the divisions of middle and lower trunk are present deep to the clavicle. Further three cords are formed the joining of anterior and posterior divisions of three trunks. The lateral cord is formed from the anterior division of the upper and middle trunks, the posterior cord by all three posterior divisions of trunks. The medial cord is formed by continuation of the anterior division of the lower trunk. The part of the brachial plexus from the formation up to its passage into the upper limb through cervicoaxillary canal constitutes supraclavicular part of brachial plexus. Beyond this point,

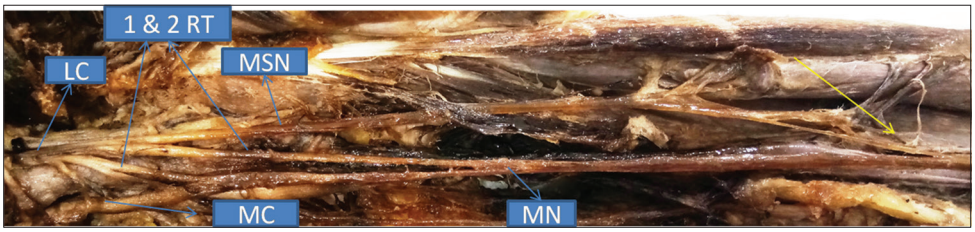

Figure 3: Infraclavicular part of brachial plexus showing three roots of median nerve and yellow arrow showing communication between musculocutaneous and median nerve. LC: Lateral cord, MC: Medial cord, MN: Median nerve, MSN: Musculocutaneous nerve

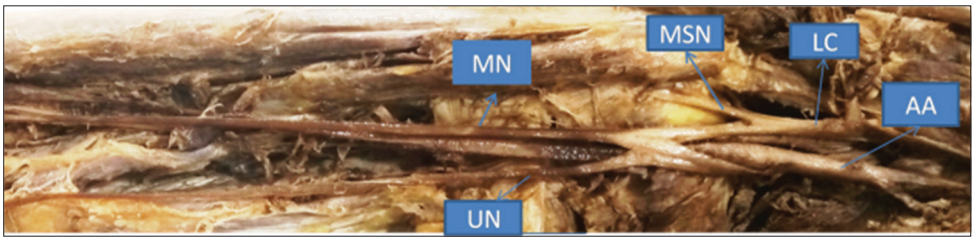

Figure 4: Infraclavicular part of brachial plexus showing higher origin of median nerve. MN: Median nerve, MSN: Musculocutaneous nerve, LC: Lateral cord, AA: Axillary artery, UN: Ulnar nerve

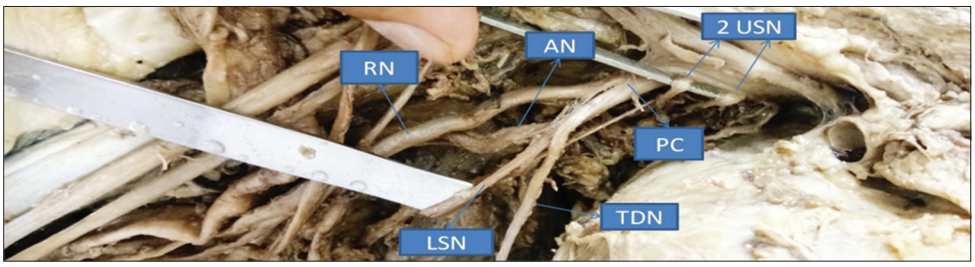

Figure 5: Infraclavicular part of brachial plexus showing two upper subscapular nerves. RN: Radial nerve, AN: Axillary nerve, USN: Upper subscapular nerve, PC: Posterior cord, TDN: Thoracodorsal nerve, LSN: Lower subscapular nerve

the brachial plexus cords and their branches extend as infraclavicular part of brachial plexus.[4]

Formation of the cords from divisions of the trunks is functionally important. Posterior cord formed from posterior divisions of all trunks supply extensor musculature of the upper limb while the lateral and medial cords formed from the anterior divisions innervate flexor musculature of the upper limb. The formation and relations of three cords are important to axillary structures especially axillary artery. Immediately inferior to clavicle, the posterior cord is lateral, the medial cord is posterior, and the lateral cord is anterior, in relation to axillary artery. The cords assume their appropriate relation above the axillary artery deep pectoralis minor. There is considerable variation in this arrangement; most commonly, the axillary artery lies anterior to three cords and median nerve. The branches of the posterior cord and the largest of the three trunks are consistent. In sequence, they are the upper subscapular, thoracodorsal, lower subscapular, axillary, and radial nerve. The branches of medial cord are medial pectoral nerve and medial cutaneous nerve of arm and forearm terminates by division into the medial root of median nerve and the ulnar nerve. The different branches of the lateral cord are lateral pectoral nerve, musculocutaneous nerve, and lateral root of median nerve.

The greatest variation in the formation of trunk nerves is found within the lateral cord. Occasionally, the musculocutaneous nerve arises more distally than usual, springing either directly from the lateral cord as two or three branches or even from the median nerve itself. Sometimes, the highest of these branches enter coracobrachialis no more than 2 or 3 cm below the coracoid process. The lateral root of median nerve may arise as two or three branches, and in some cases, it appears as a branch of musculocutaneous nerve. The ulnar nerve may arise as two or three branches. Like any other nerve plexus variations occur in the branches of this plexus also.[4]

Satyanarayana *et al.* published a case in which all the three cords, namely lateral, medial, and posterior cords of brachial plexus, were noted to be lateral to the third part of axillary artery.[4] Similar variation was found in a case, i.e., all cords are present lateral to the third part of axillary artery [Figure 1].

A case reported by Jamuna and Amudha, only two cords anterior and posterior were formed and lie lateral to the axillary artery instead of developing three cords (medial, lateral, and posterior). Anterior cord is developed by the fusion of medial and lateral cord. Branches of both the cords (medial and lateral) arise from anterior cord.[5]

Singer observed very rare variation that all the roots of plexus fused to form a one cord, he reported that this variation is due to the abnormal formation of axillary artery.[6]

In three cases, Khake *et al.* reported that musculocutaneous nerve gave a separate branch to coracobrachialis muscle before piercing it. After this, it gave muscular branches to supply the biceps brachii and brachialis muscles, a communicating branch was also given to the median nerve. Distally nerve continued as lateral cutaneous nerve of forearm.[7] In the present study, in one case separate branch to coracobrachialis muscle was originated from lateral cord. Musculocutaneous nerve passed straight downward without piercing and giving any branch to coracobrachialis. It was placed between biceps brachii and brachialis where it gave muscular branches to both of them. Nerve continued distally as the lateral cutaneous nerve of forearm [Figure 2].

Chauhan and Roy studied 400 upper limb specimens and observed one case where the communicating branch joined the median nerve below the insertion of coracobrachialis muscle. The communicating branch was joined by a twig from musculocutaneous nerve at level of insertion of coracobrachialis muscle. The musculocutaneous nerve did not pierce the coracobrachialis muscle.[8] We observed in two cases, there is communication between musculocutaneous and median nerve in the arm below the insertion of coracobrachialis [Figure 3].

Arora and Dhingra reported that variation in which median nerve was formed by the contribution of three roots and musculocutaneous nerve was absent.[9]

Budhiraja and Rastogi reported formation of median nerve from three roots, i.e., upper two roots coming from lateral cord and third last root from medial cord.[10] In the present study, similar variation was seen in five cases [Figure 3].

In the present study, in three cases [Figure 4], there is higher origin of median nerve by joining of its two roots. Normally, formation of median nerve is at lower level and lateral to ulnar nerve. We could not found this type of variation in available literature.

Sinha and Chaware observed variation in the branches posterior cord where two upper subscapular nerves were given off instead of one. In the present study, a similar variation is observed in one case[11] [Figure 5].

Miller observed that abnormal formation in the development of trunks, divisions, and cords leads to variations in the branching pattern of brachial plexus.[12] Anatomical variations of brachial plexus can be traced by understanding its normal embryological development. Moore and Persaud noticed that development of brachial plexus starts at

34th–35th day of intrauterine life and definitive adult pattern is visible by 46th–48th day of intrauterine life.[13] Miller observed axillary artery has an important role in orientation of the divisions of the cords.[14]

Hamilton *et al.* explained that the paraxial mesoderm in growing embryo differentiates into myotome, dermatome, and sclerotome. Myotome forms muscles. During development, the cells of myotome usually elongate in a direction parallel to long axis of the embryo. Myotome enlarges rapidly both dorsally, flanking the neural tube and ventrally, where it extends into the somatopleure. At the same time, the fibers of ventral roots of the spinal nerve growing out of the neural tube and come in contact with the cells of appropriate myotome permanently. The myotome becomes divided by slight constriction into a dorsal epaxial portion and ventrolateral portion. Accordingly the ventral root nerve fibers split into a primary dorsal ramus and ventral ramus supplying to corresponding portions of the myotome. During further development, the nerve actually grows to the muscle and follows it during any subsequent migration.[15]

Sannes *et al.* stated that the growing axons are regulated by chemoattractants and chemorepulsants in a site-specific fashion. Changes in the signaling pattern between mesenchymal cells and neuronal growth cones can lead to variations.[16] Result of all these changes is anatomical variations in the formation and branching of brachial plexus. However, little functional significance a variation has the surgeon need to be aware of these while planning surgery and knowing its outcome in the axillary region below in different regions of the upper limb.

CONCLUSION

The present study was conducted with an objective to see variations in the branches of cords. We found many variations, i.e., separate branch to coracobrachialis, two upper subscapularis, three roots of median nerve, and communication between median and musculocutaneous nerve. These variations are important for surgeons, neurosurgeons, and plastic surgeons in their respective field in knowing area of loss, loss of muscle power as well as in designing various surgeries.

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Original Article

Awareness, Perceptions, Attitudes, and Practices among Antenatal Mothers in Goa towards Reproductive Tract Infections/Sexually Transmitted Infections

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Abstract

Introduction: Reproductive tract infections (RTIs) are a universal public health problem among young women in developing countries and occupy the second position in public health problems. RTIs lead to 17% of economic losses in these countries. According to the WHO estimates in 2008, globally, 499 million new cases of RTIs occurred annually among women in the reproductive age group. In India, one among four women in the reproductive age group has any one type of RTIs and the annual incidence of RTI estimated is about 5%. Consequently, the prevalence rate of RTIs in various states of India is 19%–71%. There are about 40% of women estimated to have RTIs/sexually transmitted infections (RTI/STI) at any given point of time, but only 1% complete the full course of treatment of both partners. The present study was conducted to know about the knowledge, attitude, and practices about the RTI/STIs among the antenatal mothers attending outpatient departments in Goa Medical College as part of needs assessment for formulating educational and preventive strategies.

Materials and Methods: This cross-sectional descriptive study was conducted at Goa Medical College, a Tertiary Care Hospital at Bambolim-Goa, from December 2018 until February 2019. Institutional Ethics Committee (IEC) approval was taken from the IEC of the Goa Medical College. Data were collected by interviewing mothers using a semi-structured questionnaire. Additive scores were developed for awareness, knowledge, and perceptions in specific areas of transmission, prevention, and treatment of RTI/STI. The scores were characterized as poor, average, and good. Data were entered in EpiData Manager and analyzed using SPSS 22 version. Categorical variables were expressed in percentages and proportions and quantitative variables in mean ± SD. The association between dependent and independent variables was assessed using bivariate analysis. $P < 0.05$ was taken as statistically significant.

Aims and Objectives: The aims of the study were (1) to study the level of awareness regarding RTIs/STIs among antenatal mothers, (2) to study their knowledge regarding symptoms and modes of transmission of RTI/STI, (3) to study awareness regarding preventive strategies for transmission of RTI/STIs, and (4) to study their attitudes and perceptions toward STIs.

Results: The mean age of the study participants was 27.5 years. The majority of antenatal mothers, i.e., 64% were aware of RTI/STIs in our study. There was poor awareness in 46% of study subjects and good awareness in 28% of study subjects. On bivariate analysis, awareness was significantly related to education level ($P = 0.000$), occupation ($P = 0.002$), socioeconomic status ($P = 0.000$), and location ($P = 0.000$). About 59% antenatal mothers knew whitish discharge per vaginum as the most common symptom of STI/RTI. The other symptoms identified were lower abdominal pain by 56%, itching in perineal region by 40%, weakness by 50%, and loss of weight by 22%. The mode of spread of RTI/STIs was identified as a sexual route by 56% and blood transfusion by 35% and 31% and 30% were of the opinion that unhygienic conditions and unsafe deliveries were the modes of spread of RTI/STIs, respectively. About 50% antenatal mothers were aware that safe sexual practices can prevent STI/RTIs, 36% felt that the use of condoms helps in preventing STI/RTIs, 22% opined that good personal hygiene is the mode of preventing RTI/STIs, and 31% were ignorant about its prevention. Thus, 67% subjects had poor knowledge, 24% average knowledge and good knowledge was present in only 9% study subjects. On bivariate analysis, knowledge was significantly related to age ($P = 0.04$), education ($P = 0.03$), occupation ($P = 0.002$), and location ($P = 0.015$). Socioeconomic status shows significant linear by linear relation trends. About 74% study, subjects felt that RTI should be treated to avoid complications, while 23% did not want to express themselves. Similarly, 20% subjects refused to talk about STIs. Half of the

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study participants (48%) had a poor score with respect to perception about STI/RTI. Only 19% had good scores which were significantly related to education ($P = 0.012$), occupation ($P = 0.001$), and location ($P = 0.000$).

Conclusion: The study findings show that although there is 64% awareness of STI/RTI, there is a lack of requisite knowledge with respect to RTIs. The attitude, knowledge, and perception are significantly related to education, occupation, and location. Therefore, the knowledge has to be spread in society through educational programs so as to increase awareness of this problem, thereby curbing the spread of RTI/ STIs and their complications.

Key words: Antenatal mothers, Awareness, Knowledge, Reproductive tract infection, Sexually transmitted infections

INTRODUCTION

Reproductive tract infections (RTIs) are a universal public health problem among young women in developing countries and occupy the second position in public health problems.[1] RTIs lead to 17% of economic losses in these countries.[1] According to the WHO estimates in 2008, globally, 499 million new cases of RTIs occurred annually among women in the reproductive age group.[2] In India, one among four women in the reproductive age group has any one type of RTIs[3] and the annual incidence of RTI estimated is about 5%.[4] RTIs include three types of infection: Sexually transmitted infections (STIs) such as chlamydia, gonorrhea, chancroid, and human immunodeficiency virus (HIV), endogenous infections caused by overgrowth of organisms normally present in the genital tract of healthy women such as bacterial vaginosis and vulvovaginal candidiasis, and iatrogenic infections which are associated with improperly performed medical procedures such as unsafe abortion or poor delivery practices.[6] Most of the women, who on the outpatient basis, seek care have vaginal infections.[7,8] About 40% of women in India are estimated to have RTI/STI at any given point of time, but only 1% complete the full treatment of both partners.[9] Reproductive ill-health constitutes around 33% of the total burden of disease in women. RTIs, if untreated, can lead to infertility, fetal wastage (10–15%), low birth weight, and prenatal infections (30–50%) as well as congenital infections. STIs, increase the risk of HIV by ten folds[10] and also lead to consequences which include infertility, ectopic pregnancy, post-abortal and puerperal sepsis, stillbirth and perinatal death, cervical cancer, cirrhosis of liver, hepatocellular cancer, chronic pelvic pain, and emotional distress as well as social rejection. Social effects comprise stigmatization, domestic abuse, and even abandonment.[10] In low-income countries, due to lack of knowledge and/or non-availability of health-care facilities, STI is often undiagnosed and untreated. This study was conducted to know about the knowledge, attitude, and practices about the RTI/ STIs among the antenatal mothers attending outpatient departments (OPDs) in Goa Medical College as part of needs assessment for formulating education and prevention strategies.

MATERIALS AND METHODS

A cross-sectional study using a semi-structured interview questionnaire was conducted at Goa Medical College, a Tertiary Care Hospital at Bambolim, Goa. The study duration was 3 months from December 2018 to February 2019. Institutional Ethics Committee (IEC) approval was obtained from the IEC of the Goa Medical College.

Sample Size

Taking the prevalence of RTI/STI (42%) from the previous study by Anjana *et al.*[11] with a margin of 10% error, the sample size was calculated as 97; hence, 100 subjects were recruited for the study.

Sampling Technique

Sampling technique was census method.

Study Participants

All pregnant women registering for antenatal care visits were voluntarily recruited into the study after a careful explanation of the objectives of the study and their consent duly obtained.

Inclusion Criteria

The study subjects were pregnant women attending antenatal clinic for the first time, regardless of gravid status and duration of pregnancy, and willing to participate in the study.

Exclusion Criteria

Women unwilling to participate in the study.

Data Collection and Analysis

The source of data was the semi-structured questionnaire form filled after interview. These interviews included demographic and clinical characteristics, access to media related to RTI/STI, and general knowledge of RTI/STI and attitude and perceptions toward RTI/ STI. The confidentiality was maintained. Additive scores were developed for awareness, knowledge, and perceptions in specific areas of transmission, prevention, and treatment. The scores were characterized as poor, average, and good.

Data was entered in EpiData Manager and analyzed using SPSS 22 version. Categorical variables were expressed in percentages and proportions and quantitative variables in mean ± SD. Association between dependent and independent variables was assessed using bivariate analysis. $P < 0.05$ was taken as statistically significant.

Aims and Objectives

The aims of the study were as follows:

1. To study the level of awareness regarding STIs among antenatal mothers.
2. To study their knowledge regarding symptoms and modes of transmission of STI.
3. To study awareness regarding preventive strategies for transmission of STDs
4. To study their attitudes toward STIs.

RESULTS

A total of 100 antenatal mothers were interviewed in the study.

The mean age of the study participants was 27.5 years. As shown in Table 1, 46% subjects belonged to the age group of 26–30 years and 29% between 21 and 25 years. About were primigravidas and 52% were multigravidas. Education up to secondary and higher secondary was noted in 51% subjects, graduation and above in 22%, 20% secured primary education, and 7% were illiterate. About 71% subjects were homemakers, involvement in clerical job or service was seen in 18% and 9% were salesgirl or professionals. About 20% antenatal mothers belonged to socioeconomic Class I, 26% belonged to Class II, 10% belonged to Class III, 27% belonged to Class IV, and 17% belonged to Class V. About 56% participants were from rural areas and 44% belonged to urban areas. About 63% subjects had access to both private and government health-care facility while 36% had access to a government facility.

Table 1: Sociodemographic details of the study participants

Sociodemographic variable	Total number (*n*=100)	Percentage
Age		
<20 years	4	4
21–25	29	29
26–30	46	46
31–35	15	15
>36	6	6
Parity		
Primi	48	48
Multi	52	52
Education		
Illiterate	7	7
Primary	20	20
Secondary	48	48
Higher secondary	3	3
Graduate	21	21
PG or above	1	1
Occupation		
Housewife	71	71
Clerical/service	18	18
Salesgirl	1	1
Advocate	1	1
Teacher	7	07
Carpenter and others	2	02
Socioeconomic status (modified BG Prasad classification)		
I	20	20
II	26	26
III	10	10
IV	27	27
V	17	17

As shown in Figure 1, 64% antenatal mothers had heard about RTI/ STIs, 29% had not heard about RTI/STIs, and 7% were ignorant of the entity.

The source of information regarding RTI/STI was doctors in 45% study subjects, followed by health workers in 30%, TV and radio (22%), newspapers (5%), and 3% had other sources such as family members, friends, and neighbors, as shown in Figure 2 (total no. exceeds 100).

As per additive scores of awareness, there was poor awareness in 46% of study subjects, and good awareness in 28% of study subjects while 26% had an average level of awareness. On bivariate analysis, awareness was significantly related to education level ($P = 0.000$), occupation ($P = 0.002$), socioeconomic status ($P = 0.000$), and location ($P = 0.000$). There was no significant relation to age, parity, and access to hospital services.

As shown in Table 2, 59% antenatal mothers knew whitish discharge per vaginum as the most common symptom of STI/ RTI. The other symptoms identified were lower abdominal

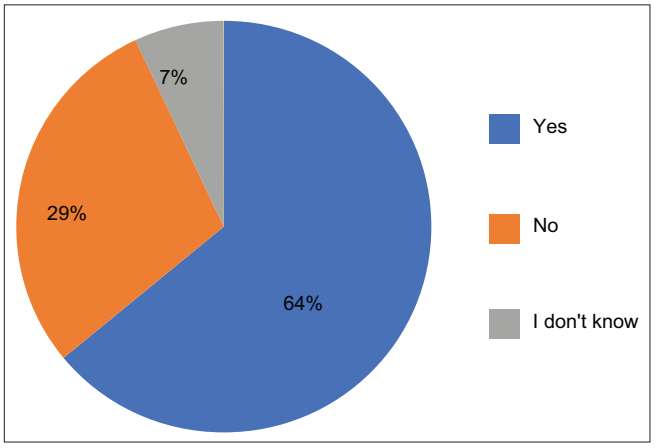

Figure 1: Awareness of antenatal mothers about reproductive tract infection (RTI)/sexually transmitted infections (STIs) – heard of RTI/ STIs

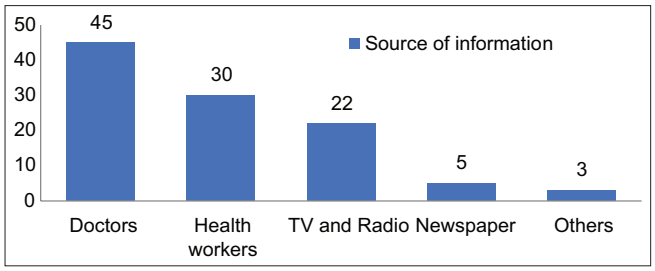

Figure 2: Source of information about reproductive tract infection/sexually transmitted infections

Table 2: Distribution of antenatal mothers based on their knowledge about RTI/STI

Knowledge variable	Number	Percentage
Knowledge of symptoms of RTI/STIs among antenatal mothers*		
Lower abdominal pain	56	56
White discharge per vaginum	59	59
Itching	40	40
Loss of weight	22	22
Weakness	50	50
Do not know	36	36
Knowledge about modes of spread of RTI/STIs*		
Sexual	56	56
Blood transfusion	35	35
Infected needles	22	22
Unsafe delivery	30	30
Unhygienic condition	31	31
Knowledge about methods of preventing RTI/STIs*		
Safe sexual practices	58	58
Good personal hygiene	22	22
Use of condoms	36	36
Do not know	31	31

Total exceeds 100 due to multiple responses. RTI: Reproductive tract infection, / STI: Sexually transmitted infections

pain by 56%, itching in perineal region by 40%, weakness by 50%, and loss of weight by 22%. One third mothers (36%) were ignorant of the symptoms. The mode of spread of RTI/STIs was identified as a sexual route by 56% blood transfusion by 35% and 31% and 30% were of the opinion that unhygienic conditions and unsafe deliveries were the modes of spread of RTI/STIs respectively. About 50% antenatal mothers were aware that safe sexual practices can prevent STI/ RTIs, 36% felt that the use of condoms helps in preventing STI/RTIs, 22% opined that good personal hygiene is mode of preventing RTI/STIs, and 31% were ignorant about its prevention.

As per additives scores of knowledge about STI/RTI, 67% subjects had poor knowledge, 24% average knowledge, and good knowledge was present in only 9% study subjects. On bivariate analysis, knowledge was significantly related to age ($P = 0.04$), education ($P = 0.03$), occupation ($P = 0.002$), and location ($P = 0.015$). Socioeconomic status trends are significant, showing linear by linear relation. Parity and access to hospital services had no significant correlation to knowledge about RTI/STI.

Attitude and Perception

About 74% study, subjects felt that RTI should be treated to avoid complications, while 23% did not want to express themselves. Similarly, 20% subjects refused to talk about STIs while 79% felt that they should be treated but they were unwilling to mix with and take care of persons with STIs. On additive scores, half of the study participants (48%) had a poor score with respect to perception about STI/ RTI. Only 19% had good scores which were significantly related to education ($P = 0.012$), occupation ($P = 0.001$), and location ($P = 0.000$). Age, parity, socioeconomic status, and access to health services had no significant relation to perception about STI/RTI.

DISCUSSION

In our study, 75% antenatal mothers belonged to the young age group between 21 and 30 years which is similar to findings of the study done by Shethwala and Mulla.[12]

In our study, the majority of the antenatal mothers, i.e., 64% were aware of RTI/STIs, which is comparable to the study done by Thekdi *et al.*[13] in Gujarat and Rabiu *et al.*[14] in Nigeria, who reported 60.4% and 77.2%, respectively. This can be attributed to the fairly good level of education in these areas. In our study, we found that educated antenatal mothers (secondary education and above) had a higher level of knowledge than mothers who were illiterate and those who secured primary education and this was found to be statistically significant ($P < 0.05$). Furthermore, we noticed that antenatal mothers belonging to higher socioeconomic status (Class I, II, and III) had a better level of knowledge about STI than mothers belonging to lower socioeconomic status (Classes IV and V). This was found to be statistically significant ($P < 0.05$). Probably, it indirectly reflected access to education material.

In our study, we found that majority of the antenatal mothers, i.e., 45% had gained knowledge about RTI/STIs from doctors, followed by healthcare workers (30%), TV and radio (22%), newspaper (5%), and others such as relatives, neighbors, and friends (3%). Our findings are similar to the findings reported by Rani *et al.*[8]

More than half of the antenatal mothers, i.e., 59% reported vaginal discharge as the most common symptom of RTI/STIs which is comparable to findings in study done by Rabiu *et al.*[14] and Kamini *et al.*[10] In our study, antenatal mothers were aware of multiple symptoms of RTI/STIs such as lower abdominal pain (56%), itching in perineal region (40%), loss of weight (22%), and weakness (50%). This can be attributed to the higher level

of education of the study population. Similar findings are reported by Shetty *et al.*,[15] Thekdi *et al.*,[13] and Rani *et al.*;[8] however, the level of awareness is less as compared to our study.

In our study, the majority of antenatal mothers, i.e., 56% responded that sexual route being the main route of transmission of RTI/STIs, which is similar to findings of the study done by Rani *et al.*[8] and Shetty *et al.*[15] About 1/3rd, i.e. 35% reported transmission through blood transfusion, 22% were of the opinion that route of transmission is through infected needles, and 30% were of the opinion that route of transmission is through unsafe deliveries. Our findings are similar to findings in studies conducted by Rani *et al.*[8] and Prusty and Unisa.[16]

In our study, majority of the antenatal mothers, i.e., 58%, could correctly identify that practicing safe sex can help to prevent the spread of RTI/STIs, 36% opined that using condoms can prevent the spread of RTI/STIs, and 22% felt having good perineal hygiene can help in prevention. Our findings are similar to the findings of studies done by Rizwan *et al.*[17] and Shetty *et al.*[15]

Most of the studies mentioned above are done among married women, among females attending gynecological OPDs, among females in rural field practice areas. However, our study focuses on awareness and knowledge of antenatal mothers about RTI/STIs and we found that the level of awareness of antenatal mothers in Goa is good.

CONCLUSION

It was seen in our study that most of the antenatal mothers were aware of RTI/STIs and the mode of transmission and mode of prevention; however, the knowledge has to be spread deep in the society through educational programs so that even uneducated women become aware of this problem, thereby seeking treatment at the correct time and curbing the spread of RTI/STIs.

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ETHICAL APPROVAL

The study was approved by the Institutional Ethical Committee.

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Original Article

Comparative Study of Hearing Assessment in Diabetic Patients and Non-Diabetic Patients using Otoacoustic Emission and Pure Tone Audiometry at Sree Gokulam Medical College, Trivandrum, Kerala, South India

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Abstract

Introduction: The association between diabetes and hearing loss has been a topic of discussion since many years. Type 2 diabetes mellitus (DM) which is the most predominant form of diabetes worldwide, accounts for 90% of cases globally. DM is a chronic metabolic disorder characterized by high blood glucose level, associated with insulin resistance and relative insulin deficiency. DM is associated with a lot of complications; a lesser known complication is auditory organ dysfunction. The International Diabetes Federation estimated in 2014 that 387 million people have diabetes worldwide and that by 2035 this number will rise to 592 million. Among these, 179 million are undiagnosed. In the United States, the Centers for Disease Control and Prevention estimated in 2014 that 29.1 million people had diabetes and that 8.1 million of them (27.8%) were undiagnosed. These data reflect the late detection of hearing loss due to undiagnosed diabetes. Studies have indicated that vascular changes which include thickening of capillary walls in the stria vascularis, neural changes in the cochlea, and loss of outer hair cells are the causes of hearing changes in DM.

Objective: The objectives are as follows: (1) Primary objective: To assess the prevalence of hearing loss in patients with Type 2 DM when compared to non-diabetic patients and (2) secondary objective: To analyze the effect of age, glycemic control (HbA1c), and duration of diabetes on auditory acuity and to assess whether otoacoustic emission (OAE) can be used as a screening test for hearing assessment in diabetics.

Materials and Methods of Study: The design of this study was hospital based observational prospective study carried out from February 2017 to July 2018. A total of 100 patients who presented to the ENT OP Department Sree Gokulam Medical College and Research Foundation were studied by dividing into two groups; Group A consisted of 50 type 2 diabetic patients and Group B consisted of 50 age- and gender-matched non-diabetic patients. The age group of the patients was between 30 and 50 years. All the patients were subjected to Pure Tone Audiometry for the quantitative and qualitative assessment of hearing loss. Transient OAE (transient evoked OAEs [TEOAE]) was also done to know whether it can be used as a screening test for the early detection of hearing loss. HbA1c was done in diabetic patients to detect any association with the hearing loss. Age and duration of diabetes and its correlation with hearing loss were also assessed.

Results: Our study confirmed the existence of sensorineural hearing loss (SNHL) in type II diabetic patients, mostly bilateral moderate SNHL. Age and duration of diabetics had an association with SNHL. As the age increases, there is increase in the auditory thresholds and amount of hearing loss (AHL). Type 2 diabetic patients with long duration of diabetes also had higher auditory thresholds at all frequencies from 250 to 8000 Hertz (Hz), when compared to non-diabetic patients and the AHL was also higher as the duration of diabetes increased. HbA1c level had no significant correlation with hearing loss. The result of TEOAE was refer, i.e. abnormal in all the patients with hearing loss.

Conclusion: The present study reports SNHL in 68% of type II diabetic subjects and 2% of healthy non-diabetic subjects. The majority of the patients had bilateral moderate SNHL. The diabetic patients had increased hearing threshold at all

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frequencies with gradual increase in hearing loss from 250 Hz to 8000 Hz. Age and duration of diabetes had a positive correlation with hearing loss. As the age increased AHL also increased. Similarly, as duration of diabetes increased, AHL also increased. HbA1c had no relation with auditory threshold and AHL. TEOAE can be used as a screening test for the early detection of hearing loss in Type 2 diabetic patients, as the result was abnormal in all the patients with hearing loss. Since hearing loss can be considered to be a consequence of diabetes, a metabolic assessment may be useful for patients presenting with hearing loss so as to reduce the high rate of undiagnosed diabetes mellitus in the community. The use of audio logical tests to monitor hearing in diabetic patients should be considered as a routine procedure so that quality of life can be improved for long standing diabetics with needed therapeutic interventions for hearing improvement.

Key words: Pure-tone audiometry, Sensorineural hearing loss, Transient evoked otoacoustic emission, Type 2 diabetes mellitus

INTRODUCTION

Hearing loss is a functional disability, in which a person's daily activities are affected in subtle ways. Sensorineural hearing loss (SNHL) is irreversible affecting the inner ear and its central connections. According to literature, the hearing loss in diabetes mellitus (DM) has shown[1-11] to be moderately high, progressive, and bilateral. Microangiopathy of the inner ear has known to be the predominant mechanism of hearing loss in diabetes.[12] The prevalence of hearing loss in diabetic patients has not been extensively studied in India.[13]

The present study is undertaken to assess hearing loss in Type 2 DM patients.

Screening test employed in this study is otoacoustic emission (OAE), which helps in the detection of any cochlear pathology. Pure tone audiometry (PTA) is the diagnostic test that helps for the quantitative and qualitative assessment of hearing. PTA involves the estimation of the threshold of hearing for certain standardized stimuli through the air and bone conduction routes.[14]

Studies have revealed an intimate relationship between DM and hearing loss.[15,16] The clinical manifestations of DM are due to insulin deficiency and relative insulin resistance which leads to defective glucose, lipid, and protein metabolism.[17] In DM, there is increased lipid breakdown and decreased lipid synthesis leading to elevated lipid levels in the blood and formation of atherosclerosis. Hyperglycemia in diabetes contributes to formation of glycated hemoglobin and deposition in the blood vessel walls. As a result, there is injury to the endothelium leading to increased capillary permeability, thickening of basement membrane, and abnormal growth of endothelial cells contributing to reduce lumen size. Thus, there is reduced blood supply, as a result, the nerves become malnourished, and their cellular membrane shows dysplasia and necrotic changes leading to diabetic peripheral neuropathy. DM leads to cochlear microcirculation disorders and hemodynamic changes[18] resulting in neuropathy. Immune system involvement has also been implicated as an etiological factor in DM. Symptoms due to inner ear dysfunction such as dizziness, tinnitus, and hearing loss have also been pointing toward DM pathology. Along with advances in research on DM and its complications, there has been an increasing attention to hearing disorders in patients with DM. Researches in DM and its complications have advanced a lot and now hearing loss as a complication have gained attention. Cochlear vascular and neural changes, i.e., thickening of capillary walls in stria vascularis and loss of outer hair cells (OHC's) have been implicated as pathology of hearing loss in DM.[18] Cochlea has no collateral circulation. Cochlear microcirculation helps in maintaining cochlear hemostasis by providing cochlea with substrates and energy; it also helps to carry away metabolic wastes.

The inner ear function is dependent on cochlear microcirculation. Thus, there is clear evidence that compromised cochlear blood supply is the main etiology of hearing dysfunction in DM.[21-29]

Other proposed mechanisms causing hearing loss include regulation of cellular transduction signals and neural, humoral, and autonomous regulation of microcirculation which are yet to be subjected to research work.

Objective

The objectives are as follows:

1. Primary objective: To assess the prevalence of hearing loss in patients with Type 2 DM when compared to non-diabetic patients
2. Secondary objective: To analyze the effect of age, glycemic control (HbA1c) and duration of diabetes on auditory acuity and to assess whether OAE can be used as a screening test for hearing assessment in diabetics.

REVIEW OF LITERATURE

Inner Ear

The inner ear consists of a central chamber called vestibule, the cochlea, and the three semicircular canals. The function

of semicircular canals is to maintain body balance and proprioception. Cochlea is considered to be the primary auditory organ.

The inner ear or labyrinth is situated in the petrous part of the temporal bone together with its attendant sensory structures, which maintains auditory function and balance.

Membranous labyrinth is seen inside the bony labyrinth and it represents a continuous series of epithelial-lined tubes and spaces of inner ear. It contains endolymph and sense organs of hearing and balance.

Membranous labyrinth has got three parts.
1. Pars superior – The vestibular labyrinth
2. Pars inferior – Cochlea and saccule
3. Endolymphatic duct and sac.

Labyrinthine sense organs have one thing in common, i.e., they contain hair cells with rigid cilia which are innervated by afferent and efferent neurons.[22,23]

Cochlea

The auditory portion of the labyrinth, i.e. the cochlea duct extends approximately 35 mm.[24] It is similar to a snail shell with approximately 2.5–2.75 turns.[25] This long cochlear duct is contained in a small space.

The essential structures in the cochlea are demonstrated by this cross section of a cochlear turn.

The scala media or cochlear duct contains the endolymph and is triangular in shape in cross section. The scala media are separated from scala vestibuli by reissner's membrane. The basilar membrane separates scala media and scala tympani. The scala vestibuli and scala tympani are filled with perilymph. Communication between perilymph of these scale is by helicotrema, situated at the apex of cochlea. The width of the basilar membrane is narrowest at basal end and widest at the apex. Similar morphological gradient is also seen in the spiral ligament and epithelial elements in the organ of corti, which determines the location of maximal stimulation of the basilar membrane and inner hair cells (IHCs) by a given tone or frequency. Thus, high frequencies are located at the base and low frequencies at the apex.

Through the periotic duct by way of cochlear aqueduct, the perilymphatic compartment also communicates with the subarachnoid space, thus allowing exchange of cerebrospinal fluid and perilymph through trabecular meshwork of connective tissue.

It is complex sense organ. It contains inner and OHC's and supporting cells, resting on the basilar membrane. The ciliated ends of hair cells protrude into or near tectorial membrane, a covering structure. Hair cells has got apical portion which are anchored in the cuticular plate.[22] There are stereocilia usually 100–150 per cell which protrudes through the cuticular plate.[22-24]

OHC's has got stereocilia which lie in contact with the tectorial membrane, when compared to stereocilia of IHCs which lie free in endolymphatic space below the tectorial membrane.

The inner and OHC's differ in many ways. IHCs have got a single row of cells whereas there are 3–5 rows of OHC's. The IHCs are flask shaped with tightly surrounded by supporting cells – interphalangeal cells and have got stereocilia which are arranged in a linear fashion. The OHC's are columnar, incompletely surrounded by supporting cells – dieter cells inferiorly and hensen cells laterally.

Innervation of Organ of Corti

The organ of corti has approximately 12,000 OHC's and 3500 IHCs. The hair cells have got innervations by afferent and efferent neurons in a complex and orderly manner.[26] The afferent neurons are bipolar neurons know as spiral ganglion cells, which are located in rosenthal canal of bony modiolus.

Human organ of corti is innervated by approximately 30,000 spiral ganglion cells. About 90–95% of spiral ganglion neurons are Type 1 neurons, rest 5% of them are Type 2. Type 1 neurons are large, myelinated and have a single dendrite which project to an IHC.[27] They also degenerate following injury to dendrite. Type 2 ganglion cells are smaller, unmyelinated and with very thin distal processes and innervate many OHC's. They survive following injury to its dendrite.

Center of organ of corti has got two rows of rod such as bodies, the inner and outer rods of corti, or pillar cells. The rod rests on the basilar membrane and the base is expanded. Inner rods have a single row of IHCs on the medial side. Outer rods have three or four rows of OHC's on the lateral side. Tunnel of corti, also called the inner tunnel of corti, is connected through intercellular crevices with other spaces. They are:
1. Outer tunnel which is seen between outer most hair cell and inner cells of Hensen beneath reticular lamina
2. Space of Neul is seen between outer rod of Corti and OHC. This space is filled with cortilymph.

Hair cells are sensory cells, which bear a cluster of stereocilia on the top and a single kinocilium. They are connected to each other through fine filaments called side

links or tip links, which are thought to be associated with ion channels in hair cell membrane.

Character changes as it progresses from base of cochlea to apex. They are

i. At the base, the partition is approximately 10 times wider than at the apex
ii. The base has more mass because of increase in size and number of supporting cells in the organ of Corti
iii. Base is over 100-fold stiffer than at the apex.

From the base to the apex of the cochlea, there is progressive increase of stereociliary length for both IHC's and OHC's.

Cochlear Micromechanics

Following stimulus an intracellular potential is evoked that consists of a series of deflections and is due to instantaneous displacement of the basilar membrane. An increase in pressure in perilymph following inward movement of footplate is transmitted through the cochlea resulting in an outward bulging of round window.

Von Bekesy[28] showed that basilar membrane vibrations results in a travelling wave starting from the base of cochlea to progress toward the apex, determined by the frequency of stimulating tone. The maximum displacement of basilar membrane is seen in the basal tone of cochlea in high frequency stimulation. In low frequency stimulation, the travelling wave has got maximum amplitude near the apex. The differences in physical characteristics of basilar membrane result the travelling wave to travel from base to apex.

There is release of transmitter from the presynaptic area at the base of hair cells following displacement of stereocilia, this process is known as transduction. The transmitter results in the generation of nerve impulses in the afferent fibers of auditory nerve.

Endocochlear potential is approximately +80 mV, i.e., the resting potential of cochlear duct. It is maintained by active ionic pumps and selective permeable ion channels in stria vascularis. OHC's has a resting potential of −70 mV and IHCs has a resting potential of −40 mV.

A pressure gradient of 120–150 mV exists between inside of hair cells and endolymph. Mechanically gated channels on the upper surface of hair cells are opened following deflection of stereocilia resulting in influx of potassium ions along the electrochemical gradient. This alters the intracellular potential and chemical transmitters are released from presynaptic zone of the lower end of hair cells stimulating the afferent auditory nerve fibers.

OHC's produces cochlear microphonics and summating potential, thus the destruction of the cells results in reduced amplitude of microphonics. OHC's stimulation causes two motile responses;

1. Fast motility or electromotility
   It is due to electrically driven conformational changes in motor proteins densely packed in OHC plasma membrane. Shape changes in microseconds range. There is asymmetric electrical stimulation of motor protein or asymmetric distribution of motor proteins in the plasma membrane of OHC.
2. Slow motility
   It is due to cytoskeletal reorganization which results in change of shape and volume change. Shape changes in milliseconds to seconds range.
   The OHC electromotility is the mechanism involved in spontaneous OAE.

Cochlear Amplifier

It is a positive feedback loop in the cochlea resulting in amplification of travelling wave. It occurs throughout the length of cochlea. Vibrations of basilar membrane are amplified in a cycle by cycle basis. It does not amplify sounds equally at all frequencies. Only at characteristic frequency maximal force is generated.[29] Thus providing sharpening of tuning curve and helps in distinguishing between two tones of nearly same frequency and improves frequency selectivity.

OHC electromotility is due to this unique mechanism. Destruction of OHC thus results in loss of threshold sensitivity of about 50 Db[30] in the cochlea and tuning of 8th cranial nerve CN fibers is lost.

Mechanism of Conduction of Impulses

The tectorial membrane has got anchoring to the limbus medially and is attached to Hensen cells by a fibrous net laterally. Sound energy delivered to the oval window creates a travelling wave which displaces the basilar membrane and tectorial membrane vertically; it results in a shearing action between the tectorial membrane and cuticular plate causing displacement of stereocilia and initiation of electrical event in hair cells.

Auditory Pathway

Classic ascending auditory nervous system

Cochlear nucleus is the first nucleus of the classic ascending auditory pathway, where all auditory nerve fibers are relayed by synaptic transmission. Three main parts of cochlear nucleus include;

1. Anterior ventral cochlear nucleus.
2. Posterior ventral cochlear nucleus
3. Dorsal cochlear nucleus

Through the three acoustic striae and lateral lemniscus, the divisions of cochlea nucleus connect to the inferior colliculus. The inferior colliculus is then projects to thalamic nucleus that is ventral part of medial geniculate body, which finally projects to primary auditory cortex.

Non-classic ascending auditory system

This pathway is an "adjunct" to the classic ascending auditory pathway. When compared to classic ascending auditory pathway, which receives input only from the cochlea, this pathway receives input from other systems also.[31]

In this pathway, the main auditory input is received through connection with the central nucleus of inferior colliculus. From these region, the ascending fibers project to neurons in the medial and dorsal medial geniculate body. These neurons are then projected to association cortex and to structures of limbic system.

This pathway has got subcortical connection to limbic system, thus explaining the importance of this system in tinnitus rather than hearing.

PHYSIOLOGY OF HEARING AND AUDITORY PATHWAY

The hearing mechanism comprises a set of highly specialized structures allowing sound from the environment to activate the brain's auditory response.

The mechanism involves conversion of sound energy collected by the ear to mechanical energy in the middle ear and then to hydraulic and electro-chemical energy in the cochlea, which triggers nerve impulses and transmit it to the brain. The ear, which is concerned with hearing, is divided into three parts: The outer or external ear, the middle ear, and the inner ear. The outer ear consists of pinna or auricle and the external auditory canal (EAC). The tympanic membrane (TM) or eardrum separates the outer ear from the middle ear cavity. There is a significant influence of external ear along with head and body for the sounds that reach the middle ear. External ear produces a gain of 20 dB at 2500 Hertz (Hz), when a sound strikes the external ear. This is called external ear gain, which is a frequency and directionally dependent alteration in the sound pressure at the TM when compared to sound pressure in the environment. When struck by sound waves, the TM vibrates and transmits sound energy to the chain of three small articulated ossicles, the malleus, the incus, and the stapes. There is conversion of air pressure into equivalent mechanical movements and coupling of sound from the EAC to the cochlea.

Areal Ratio

There is a mechanism in the middle ear, which acts as a transformer to increase sound pressure at the footplate of stapes relative to the TM, by decreasing stapes volume velocity relative to TM volume velocity.

Areal ratio is the ratio of TM area to the stapes footplate area. It is one of the major transformer mechanisms in the middle ear. The force gathered on the surface of TM is coupled and transmitted to smaller footplate of stapes. Area of TM is 20 times larger than the area of footplate of stapes. Therefore, if the transformer action of areal ratio is ideal, the sound pressure applied to the inner ear by the stapes footplate will be 20 times or 26 dB larger than sound pressure at TM.

Ossicular Lever

Different lengths of the rotating malleus and incus arms around the axis of rotation of the ossicles results in the ossicular lever action. The length of lever arms in humans is nearly same for malleus and incus. The ratio of these lengths, which is 1.32 results in only a small increase in sound pressure of 2 dB when transmitted by the stapes to the inner ear. Thus, the theoretical middle ear sound pressure gain is about 28 dB (26 dB area ratio + 2 dB ossicular lever) when the transformers acts ideally. However, the measured middle ear gain is less than the ideal anatomic transformer model. This difference between theoretical and measured gain is due to several non-ideal conditions within the middle ear. These are

1. The entire TM moves with the same phase but magnitude varies at low frequencies but at frequencies above 1000 Hz, the patterns of vibration become more complicated, as a result, there is breaking up of vibrations into smaller vibration portions that vibrate with different phases. Thus, the efficacy of TM as a coupler of sound pressure is decreased
2. There is loss of energy when sound pressure is transmitted from the external ear to the cochlea, as part of sound generated by a sound pressure in the EAC is used to move TM and cochlea
3. Some of the pressure increase produced by middle ear transformer is used by middle ear spaces
4. The ossicular system acts as a rigid body according to the anatomic transformer model but at frequencies above 1000–2000 Hz, there is slippage in the ossicular system due to translational movement in the rotational axis at the ossicles or flexion in the ossicular joints.

Mechanism of Injury to the Auditory System in DM

Cochlear physiology is maintained by cochlear microcirculation, which plays a very important role in maintaining inner ear hemostasis.

Metabolic abnormalities in DM cause increased viscosity in the blood and thus contributing to circulatory disorders. Microcirculation disorders mainly involve stria vascularis, affecting the cochlea. Resulting ischemia and hypoxia lead to neural and hair cell damage.

Increased lipogenesis in DM leads to deposition of lipids in cochlear hair cells, damaging cochlear neural cells, resulting in impeded neural transmission. Cochlear functioning is also dependent on glucose as a source of energy which makes cochlea more prone to damage in DM.

The basilar artery or anterior inferior cerebellar artery gives rise to labyrinthine artery. The latter divides into common cochlear artery and vestibular artery, the former then branches into vestibulocochlear artery and spiral modiolar artery that supply the cochlea.[32]

In cochlear microcirculation, there is an autonomic regulatory mechanism which helps in maintaining normal blood flow. In normal circumstances, the lateral cochlear wall blood vessels are not opened fully but they open in altered condition, highlighting action of certain metabolites in blood.[33] L-arginine has found to significantly enhance cochlear microcirculation by increasing the blood flow in cochlea.[34] It has been found that tropomyosin like immune active materials are found near the cells (endothelial and peripheral) in spiral ligament and they help in regulation of cochlear blood flow.

Under anoxic conditions, endothelium releases adenosine triphosphate, acetylcholine, and endothelin which interact with their receptors and produce nitric oxide (endothelium dependent factor) and cause smooth muscle relaxation.[35]

Wang *et al*.[36] in 2006 demonstrated abnormalities in ultrastructure of inner ear capillaries along with thickening of basement membrane in a rat model with DM. Tomisawa[37] in 2002 studied temporal bone stria vascularis sections from ten DM patients with age- and gender-matched 16 non-DM individuals and found thickening of capillary lumen of stria vascularis and came to the conclusion that cochlear microcirculation abnormality is due to atrophy of stria vascularis, determining hearing loss in DM patients.

Studies in rat with DM showed changes in OHC's, spiral ganglion cells and nerve fibers, due to mitochondrial damage.[38] Streptozotocin-induced diabetes in rat models showed increased OHC loss compared to normal controls, when compounded with noise exposure at 95 Db sound pressure level (SPL).[39]

Studies in Type 2 DM middle and old aged mice demonstrated decreased distortion product OAEs (DPOAE) amplitudes when compared to normal controls.[40]

PTA

Whenever an object vibrates in an elastic medium, i.e., air, there is generation of sound. As a result, there is disturbance in the surrounding molecules of air in the form of successive waves of compression and rarefaction, known as sound waves. Frequency is defined as number of sound waves set up in the air in 1 s. For audiological evaluation, frequency is measured in cycles per second or Hz.

PTA is used to determine the hearing thresholds for pure tones. Pure tones are sinusoidal signals with a single defined frequency, amplitude, and phase.

The audiometer is the principle equipment used for deriving the clinical audiogram. The hearing threshold level of subject for pure sounds of various frequencies is plotted graphically, called audiogram. The characteristics and calibration of the equipment used to carry out PTA are based on specific international and national standards, for example, IEC 60645–1:2017[41] and BS EN 60645–1:2017.[42] The procedure for identifying the threshold and the characteristics of the test room are also specified (ISO 8253–1:2017).[43]

Screening, clinical, diagnostic, and computer integrated software applications are four different levels of complexity of functions of audiometer.[41,42]

Pure tone sounds of various frequencies are generated at regular steps of 125, 250, 500, 1000, 1500, 2000, 3000, 4000, 6000, and 8000 by an audio-oscillator. There is an attenuator dial, which attenuates the tone in decibels (dB) and graduated in 5 dB steps from −10 to 100 dB.

Two important terms concerned with PTA are as follows;
1. Threshold
2. Decibel and related terms such as SPL, intensity level (IL), and sensation level (SL).

Hearing Threshold

"The lowest SPL, at which under specified conditions, a person gives a predetermined percentage of correct responses on repeated trials" is defined as hearing threshold by the International Standards Organization. In our clinical practice, the predetermined percentage is 50.

PTA obtains relative thresholds. Relative thresholds are the thresholds which compare the hearing sensitivity of a subject in dB with a fixed ideal or normal hearing (0 dB).

The exact minimum sound pressure which the subject is able to hear just 50% of the number of times that the sound stimulus is presented to the ear is called absolute threshold, which is expressed in dynes/sq.cm.

Decibel

Intensity of sound is measured by the unit called decibel. The average minimum sound just audible to a normal ear is called reference level and is 10–16 watts/sq.cm. IL is the suffix IL used with decibels and it signifies that the reference sound has an intensity of 10–16 watts/sq.cm.

In audiometer, sound is measured with a pressure reference, i.e., SPL. SPL means sound pressure level and it defines the reference sound that has a pressure of 0.00024 dynes/sq.cm. It is expressed in decibels.

The human ear is most sensitive to frequency of 1000–2000 Hz range. At frequency of 250 Hz or 1000 Hz, the ear is less sensitive to sound and the higher intensity of sound will be require for the sound to be audible by the ear.

SL indicates how much sound sensation that a particular subject is actually perceiving. It is also a commonly used suffix with decibel. It is a reference to the auditory threshold of a particular subject.

Procedure of PTA

To the test results to be accurate, the following conditions are mandatory.

1. Calibrating the instrument
   The test equipment needs to be calibrated electronically at least once in every 6 months and a biological calibration should be done each day before the audiometer is used. The ISO–1964 specifications are followed now.
2. Reasonably noiseless test environment
   The American Standards Association has specified certain acceptable level of noise in the audiometry test room for different frequencies required for air and bone conduction tests.
   For air conduction tests, the average is around 25–30 dB (SPL) and for bone conduction tests it is 10–15 dB. Using an instrument called sound level meter, the ambient sound pressure presents in the test room can be measured.
3. Position of headphones
   Threshold variation of 15 dB or even more can occur if the position of headphones is not correct. The headphones diaphragm must be placed on the opening of external auditory meatus.
   Supra-aural headphones are most commonly used but fail to achieve good coupling with the ear because of air leak and as a consequence hearing thresholds below 500 Hz may be overestimated.[44] Circum-aural headphones are the alternative type, which encloses the pinna in its cups. These headphones prevent leakage of the test signal and reduce the amount of physiological noise in the ear.[44]
   Insert earphones are the third type of air conduction transducers. It consists of two probes for right and left ear. These probes are covered by disposable foam tip that is placed in the ear canal for testing.

Advantages of insert earphones

i. Improved attenuation of ambient noise
ii. Less chance of infection transmission, because of single use of inserts[45]
iii. Positioning of insert reduces ear canal from becoming collapsed in soft or elderly ears[45]
iv. Have higher "interaural attenuation" when compared to supra-aural and circum-aural headphones[44,45]
v. When delivering high-level low-frequency tones they reduce vibrotactile stimulation.[46]

But insert earphones are not recommended in the following circumstances;

i. Excessive ear wax in the ear canal
ii. Obstructions and abnormalities affecting ear canal
iii. Infections of the ear.

4. Instructions to the patients
   The procedure should be thoroughly explained to the patient. The patient's cooperation is of great importance, as this is a subjective test. The responsibility of the patient is as much as that of the examiner for getting a proper result.

Technique of Air Conduction Test

Two main techniques include;

i. Hughson-Weslake technique slightly modified by Carhart and Jerger – which is commonly used
ii. American Speech and Hearing Association (ASHA).

Conventional Method

Before the test a detailed clinical history and examination is carried out. The first ear tested is better ear. At 1000 Hz test is begun and then the other frequencies are tested in the following order, 2000, 4000, 8000, and 10,000, then 1000 Hz repeated again, followed by 500–250 Hz. If there is difference in hearing between the octaves of more than 20 dB, half the octaves, i.e. 750, 1500, 3000, and 6000 are tested. The frequency is ascertained in each frequency as follows:

i. The patient is first familiarized with the sound by introducing sound at an arbitrarily presumed supra-threshold level
ii. The patient appreciates the sound, the tone is reduced in step of 10 dB till the patient stops hearing

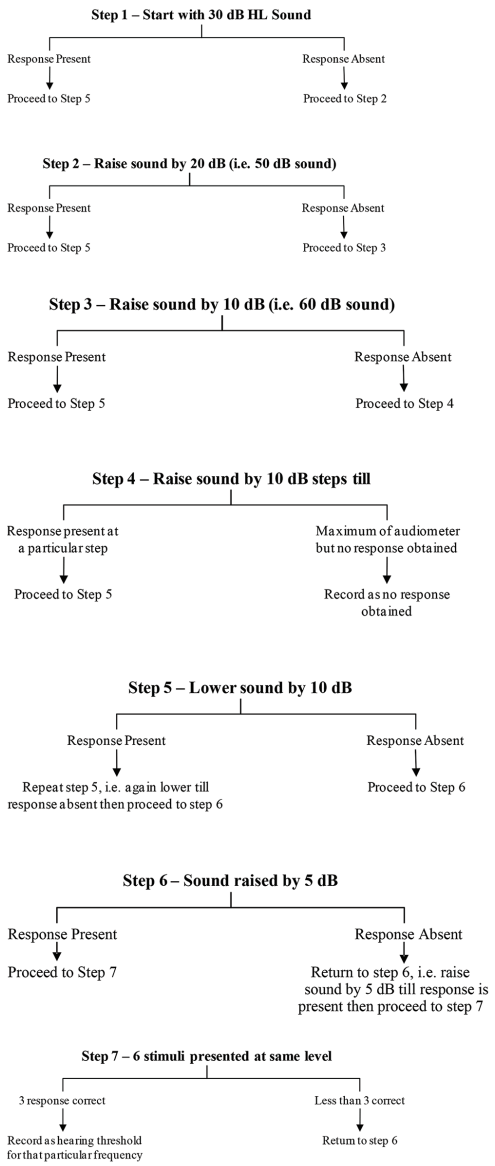
iii. Then, the tone is raised by 5 dB
iv. If the sound is heard then again the tone is decreased by 10 dB
v. If not heard, the tone is again raised by 5 dB

In this way, the actual hearing threshold is obtained, when three out of five responses are correct. The second ear also tested in the same manner, with the last frequency used to test the first ear. This method is called 5 up and 10 down method.

Method of ASHA

This test is also started with 1000 Hz and threshold for various frequencies is obtained in a similar manner as in the conventional method.

The method of obtaining threshold with this technique by minor modification is outlined here.



BC tests

Cochlea is stimulated in three ways, when sound is conducted to the cochlea through the bone.

Compressional/distortional bone conduction

There is alternate compression and expansion of the cochlear shell, when sound reaches the cochlea, resulting in movement of cochlear fluid. Because of the flexibility of the round window membrane and cochlear aqueduct, the expansion is possible. Due to movement of cochlear fluid, there is displacement of the basilar membrane that leads to changes, resulting in the sound to be heard ultimately.

Inertial bone conduction

When the skull is stricken by the sound, it sets the skull into vibration. In the middle ear, due to inertia of the ossicles, the ossicles lag behind and this leads to a relative motion between the foot plate of the stapes and the cochlear fluid deep to the oval window, which causes vibration of the cochlear fluid with movement of basilar membrane and results in the sound to be heard.

Osseotympanic bone conduction tests

In this, sound reaching the skull results in vibration through the column of air in the EAC. The vibration is then partially transmitted to the TM and through the ossicles in the middle ear to cochlear fluid of inner ear and thus sound is heard.

Conditions for bone conduction tests

Similar to air conduction test with minor variations.

1. Calibration of instrument
2. Reasonably noiseless test environment
   Acceptable noise levels for the test environment in bone conduction tests are lesser than air conduction tests for permissible ambient noise. This results as the ears are uncovered during bone conduction test. In air conduction test, a part of the ambient sound is cut down as the ears are covered by earphones.
3. Placement of the bone conduction vibrator
   The placement of the bone conduction transducer is on the mastoid prominence of the worse hearing ear.[47] It may be placed over the forehead also. Each placement has got its own advantages and disadvantages.

Mastoid placement

Here, the bone conduction vibrator, which is attached to a spring metal, is placed over the mastoid bone.

The test ear should invariably be uncovered by the earphones and in the opposite ear; earphone will be placed to deliver the masking sound. There is a false increase in the bone conduction threshold for the lower frequencies due to osseotympanic bone conduction sound, when ears

without any conductive pathology are covered. This is called occlusion effect. There may be around 20–25 dB variability in low frequencies due to occlusion effect.

Frontal placement

The bone conduction vibrator attached with elastic headband can also be placed over frontal bone on the forehead. As the amount of tissue between the bone conduction vibrator and skull bone is less, frontal placement is superior to mastoid placement in terms of consistency of results. However, mastoid placement is more sensitive with the usual bone conduction vibrators and thresholds on the mastoid are about 10–15 dB better than those with frontal placement.

The instructions given to the patient and the technique of bone conduction test are same as that of air conduction audiometry.

Problems during Bone Conduction Audiometry

1. Masking of test tone may occur due to changes of ambient external noise, as the test ear is kept unoccluded
2. Bone conduction test are possible only up to a maximum of 40 dB at 250 Hz, 50 dB at 500 Hz, 60 or 70 dB from 1000 to 4000 Hz
3. Thicker skull bone will result in false elevated hearing thresholds than others
4. Bone vibrator if not placed at the most sensitive point on the mastoid process can result in error
5. At 250–500 Hz and sometimes at 1000 Hz vibrotactile stimulation results in false and erroneous air-bone gap at low frequencies
6. Irrespective of the side of stimulation, the bone conduction vibrator stimulates the cochlear of both ears
7. Errors due to occlusion effect.

Masking

A continuous masking noise is presented to the non-test ear to prevent cross-hearing of the test tone, whenever there is an interaural difference of 40 dB or more when supra or circum-aural headphones are used or 55 dB or more when insert earphones are used.[47]

Minimum amount of masking sound required can be calculated by formula.

For bone conduction

Minimum masking = Bt + (Am–Bm)

Where:
Bt=Bone conduction threshold in the test ear
A_m=Air conduction threshold in the masked ear, and
B_m=Bone conduction threshold in the masked (non-test) ear.

For air conduction

Minimum masking=At–45 + (Am–Bm)

Where:
At=Air conduction threshold in the test ear and 45 is the accepted minimum interaural attenuation for air conducted sound, and
(Am–Bm)=Air bone gap in masked (non-test) ear as in formula above.

The maximum permissible masking sound can be calculated by the formula:

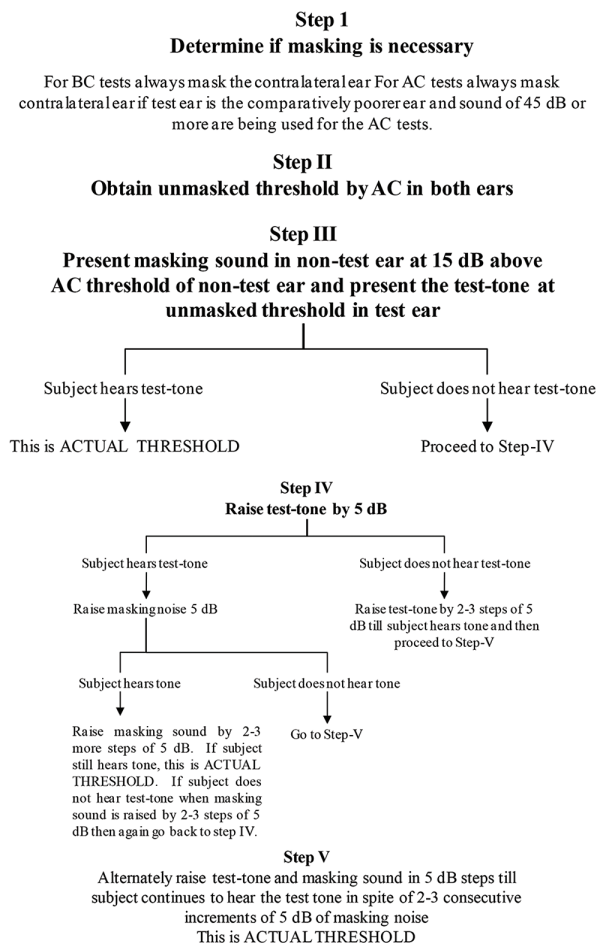
For bone conduction as well as for air conduction

Maximum masking=Bt + 45

Where:
B_t is bone conduction threshold for test ear.
This is so because 45 dB is the interaural attenuation.

The aim is always to keep the intensity of making sound between overmasking and undermasking levels.

Hood's plateau method of masking is followed. The steps are outlined below:



Sounds Used for Masking

Three types of masking sounds usually available are as follows:

- White noise
  It is also known as broadband or wideband noise. Starting from low to very high frequencies, ideally it contains an equal amount of sound for all frequencies.
- Narrow band noise
  It consists of a narrow band of noise centered on the test tone frequency with 100–200 Hz above and below that frequency. It is most effective for masking. Critical band width is the band width which provides the maximum effective masking for a tone of a particular frequency at minimum intensity. The narrow band noise signals used for clinical masking are calibrated as specified in the standard ISO 389–4:1994.[48]
- Complex noise
  This noise is made up of a low frequency fundamental plus the multiplies of that frequency up to 4000 Hz. It is not efficient as narrow band noise and wide noise.

Test-retest Difference

A difference of 10 dB is significant while assessing average change in threshold across two or three frequencies.[49]

The AC transducers have got more test-retest accuracy for absolute thresholds when compared to BC transducers.

The PTA ascertains the following

i. Whether the patients have got any definite hearing loss
ii. The type of hearing loss (THL) – conductive, sensorineural, or mixed
iii. If sensorineural, cochlear, or retrocochlear pathology
iv. The degree of hearing loss.

Interpretation of Audiograms

Classification of the British Society of Audiology[47] based on the average of hearing threshold for 250, 500, 1000, 2000, and 4000 Hz.

Quantitative Information

- 0–20 dB – Normal hearing level for all practical purpose (i.e., no deafness).
- 21–40 dB – Mild deafness
- 41–70 dB – Moderate deafness
- 71–95 dB – Severe deafness
- 96 dB and above – Profound deafness.

These classification originated from need to have an easy way of describing average hearing loss among professionals or from professional to patients.[50]

Limitations and Fallacies of PTA

The limitations of the PTA test are as follows:

1. Audiograms are very often inaccurate
   a. Improper Technique
   b. Improper Test Condition
   c. Improper Test Instrument
   d. Improper Examiner
2. A subjective and time-consuming test
3. The PTA test does not assess the main features of hearing
4. The test does not identify the nature of the pathology
5. The bone conduction test does assess the true sensorineural reserve
6. Many sources of variances in the test result that is not related to hearing.

OAE

Kemp[51] in 1978 demonstrated that very low intensity sounds are emitted by healthy inner ear which could be measured using a sensitive microphone sealed into the ear canal. These responses besides occurring spontaneously, can also be evoked. He came to the conclusion that these low level acoustic signals originated from OHC of the cochlea and resulted from non-linear processing of the healthy inner ear.

OHC's when stimulated showed changing shape, which was demonstrated by Brownell[52] in the early 1980's. In 1990's, Mammo and Ashmore[53] demonstrated motion of the basilar membrane when isolated OHC's are electrically stimulated. Ears with damaged OHC's generally exhibit reduced sensitivity, broader tuning and absent OAE's[30,54-56] due to inherent non-linear nature of cochlear function.

OAEs are of two types

1. Spontaneous emissions.
2. Evoked OAEs
   a. Transient evoked OAEs (TEOAE).
   b. DPOAE.
   c. Stimulus frequency OAEs (SFOAE).[57]

Spontaneous OAE

These are low level acoustic signals which can be recorded in the ear canal without any external stimulation. The signals are recorded only in 50–70% of normal individuals.[58,59] They are recorded more frequently in women than men.[60,61] Spontaneous OAE's can be detected without amplification or sophisticated recording equipment.[62,63] There is no strong correlation between the presence of spontaneous OAE and tinnitus and the frequency of spontaneous OAE varies significantly with time.[64,65]

TEOAE

These are low level acoustic signals which are recorded following a high level click that is a brief acoustic stimulus and elicits oscillatory responses from OHC's of a wide section of cochlea. The local vibrations which are transmitted through the middle ear create low level,

typically inaudible, and sound pressure waves in the ear canal. After 20 ms of onset of the stimulus, TEOAE's are recorded using a microphone in the ear canal.

High frequency components are recorded earlier than low frequency components, as they originate from the base of cochlea and require a shorter travel time. The response of TEOAE provides frequency specific information about the status of the cochlea between approximately 1000 Hz and 4000 Hz.

Signal-to-noise ratio of >6 dB coupled with reproducibility measures of more than 90%, is the typical criteria used to establish the presence or absence of a response within a specific frequency band.

The presence of TEOAE's is suggestive of normal OHC function and indicates normal audiometric sensitivity. The absence of TEOAE's within a specific frequency band is suggestive of audiometric thresholds worse than 25–30 dB HL.[66]

DPOAE

Two continuous sinusoidal signals (F1 and F2) are presented simultaneously into the ear canal and distortion product OAEs are recorded. The responses recorded using a sensitive microphone sealed into the ear canal. The response that is recorded in the ear canal shows evidence of distortion, as the normal cochlea is highly non-linear. The recording includes energy at two primary frequencies, F1 and F2 and also includes energy at numerous other frequencies that are mathematically related to the primaries. These are called distortion products. Most frequently used distortion product to assess auditory function is 2 F1-F2.

Normally functioning OHC's is indicated by the presence of significant amounts of energy at 2 F1-F2 frequency and is suggestive of normal audiometric sensitivity. Loss or damage to OHC's leads to SNHL. Energy at the distortion product frequency decreases and the movement of basilar membrane becomes more linear, when the OHC's are no longer functioning.

The previous studies had come to the inference that most robust are that DPOAE's are recorded when the frequency ratio of F2 to F1 is approximately 1.2.[67,68] The ear identifies normal hearing or impaired hearing most accurately when the level of the two tones is typically set at 65 dB SPL and 55 SPL (L1 and L2).[69]

DPOAE's can be recorded across a wide frequency range from 1000 Hz to approximately 8000 Hz, when the frequency of the two primary tones is varied systematically. Before considering a response to be present, the distortion product OAE amplitudes to noise ratio should be approximately 6 dB. The amplitude of DPOAE is compared with normative values to interpret a response.[55]

Clinical applications:

1. Both DPOAE and TEOAE's assess the cochlear pathology
2. A robust evoked OAE's strongly indicates that the patients have normal or near normal OHC function
3. DPOAE's nor TEOAE's recorded in ears with conductive and mild to moderate degrees of SNHL
4. TEOAE's are more sensitive to very mild amounts of hearing loss than DPOAE's, particularly at 1000 Hz. DPOAE's are slightly more sensitive to mild degrees of hearing loss at frequencies between 4000 and 6000 Hz[66,70]
5. Evoked OAE's can be used as a clinical tool for screening of hearing loss
6. Evoked OAE's are not affected by sleep or sedation. It does not require the application of recording electrodes or active participation.

Stimulus Frequency OAEs (SFOAE)

Sounds are emitted in response to a continuous tone. They provide same information as TEOAE but require more time to record also needs complex calculations to separate stimulus from response.

MATERIALS AND METHODS OF STUDY

Study Design

This was a prospective observational study.

Study Population

Patients were attending the ENT OP Department of Sree Gokulam Medical College and Research Foundation (SGMC and RF).

Inclusion Criteria

The following criteria were included in the study:

- Type II diabetic patients, age 30–50 years
- Non-diabetic patients, age 30–50 years.

Exclusion Criteria

The following criteria were excluded from the study:

- History of any previous ear pathology causing hearing loss
- History of ear surgeries performed in the past
- History of consumption of ototoxic drugs
- History of autoimmune disease (AD)
- History of hypertension (HTN)
- Family history of deafness (FHD).

Sample Size Calculation

The sample size was determined after taking into consideration, The Journal – Auditory Acuity in Type 2 DM.

Int J Diabetes Dev Ctries, 2008 October–December, 28(4):114-120.
doi:10.4103/0973-3930.45270.
PMCID: PMC 2822154.
Author – Pallavi Panchu.

Many published studies on the prevalence of hearing loss in diabetics used as similar sample size between 20 and 45 diabetic subjects.[1,3,6,7,11]

$$Sample\ size\ n = (S1^2 + S2^2)\frac{(Z1 - \frac{\alpha}{2} + Z1 - \beta)^2}{(\overline{X1} - \overline{X2})^2}$$

S1 – Standard deviation of auditory threshold in Group 1= 4.65.
S2 – Standard deviation of auditory threshold in Group 2 = 8.32.
$\overline{X1}$ –mean of auditory threshold in Group 1=19.27

$\overline{X2}$ –Mean of auditory threshold in Group 2=26.59

$$Z1 - \frac{\alpha}{2} = 2.567$$

*Z*2–β=1.282
a=1% *β*=10%
n=25 from each group.

Study Setting

The study was conducted at ENT department of SGMC and RF.

Duration of the Study

The study was 1½ years from February 2017 to July 2018.

Study Variable

1. Hearing loss in diabetic patients
2. Age, duration of diabetes, and HbA1c and its correlation with hearing loss
3. OAE and its usage as screening test in diabetic patients.

Study Instrument and Validation Program

A pro forma based on patient evaluation.

Methodology

All the subjects included in this study are given a prepared pro forma to answer. This pro forma will be assessed and evaluated.

An assessment of hearing was done using Transient OAE (GSI Corti) and PTA (GSI 61 Audiometer). Glycemic control was evaluated with HbA1c level.

PTA

Principle

GSI 61 audiometer was used, which is an electronic device that produces pure tones and the intensity of which can be increased or decreased in 5 dB steps. Air conduction thresholds are measured for tones of 250, 500, 1000, 2000, 4000, and 8000 Hz. Bone conduction thresholds are measured for 250, 500, 1000, 2000, and 4000 Hz. The minimum amount of sound appreciated by the patient is the hearing threshold at that frequency. It is charted in the form of a graph which is called the "audiogram." The audiometer is calibrated such that the hearing of a normal person, both for air and bone conduction are 0 dB and there is no A-B gap.

Methodology of PTA

The method is based on American Society for Speech and Hearing Association (ASHA) 1978 guidelines for manual PTA. If there is a difference of more than 40 dB between air conduction threshold of the test ear and bone conduction threshold of the opposite ear, masking was done. When the air bone gap of the poorer ear under test is more than 10 dB, masking was done.

TEOAE

Principle

GSI corti device was used. A non-linear click stimulus of 64 sec duration, a repetition rate of 50 Hz and an intensity of approximately 80 dB was given. The results were presented for band range 1.5–4 kHz. Mean TEOAE amplitude below 3 dB at band range 1.5–4 kHz was considered as a lack of OAE.

Data Storage

Data are stored as hardcopy in department and softcopy in Microsoft Excel.

Data Management and Analysis

Data will be entered into Microsoft Excel and analyzed using SPSS version 16. Student *t*-test has been used for comparing mean auditory threshold in two groups. Analysis of variance has been used to find the significance of auditory thresholds in different age groups. Qualitative data were compared using Chi-square test [Annexure I-IV].

OBSERVATION AND RESULTS

During the study period from February 2017 to July 2018, 100 patients who presented to the ENT OP Department at SGMC and RF were divided into two groups after

thorough clinical examination. Group A consisted of 50 Type 2 diabetic patients, with diabetes. Group B consisted of 50 age- and gender-matched non-diabetic patients. All of them were subjected to objective tests, i.e., OAE and PTA. Glycemic control was evaluated with HbA1c. Thus, assessment of hearing was done in these two groups. The correlation between age, duration of diabetes, HbA1c, and hearing loss was also assessed.

Gender

Among the 50 patients in each group, 31 (62%) patients were male and 19 (38%) patients were female, Table 1.

Age Distribution

Among the study population, the age group ranged between 30 and 50 years. Out of 50 patients in each group, 20 patients (40%) were in the age group of 30–40 years and 30 patients (60%) were in the age group of 40–50 years, Table 2.

Duration of Diabetes

The diabetic patients were divided into three groups based on the duration of diabetes.

a. Group 1 consisted of patients with duration of diabetes <5 years
b. Group 2 consisted of patients with duration of diabetes between 5 and 10 years
c. Group 3 consisted of patients with duration of diabetes >10 years

Majority of the diabetes patients, i.e., 23 patients (46%) were included in Group 2 when compared to Group 3 which consisted of 14 patients (28%) and Group 1 which consisted of 13 patients (26%), Table 3.

Table 1: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to gender

Gender	Diabetic		Non-diabetic		Total	
	n	%	*n*	%	*n*	%
Male	31	62	31	62	62	62
Female	19	38	19	38	38	38
Total	50	100	50	100	100	100

Table 2: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to age

Age in years	Diabetic		Non diabetic		Total	
	n	%	*n*	%	*n*	%
30–40	20	40	20	40	40	40
41–50	30	60	30	60	60	60
Total	50	100	50	100	100	100

HbA1c Level

In the diabetic patients HbA1c evaluation was done. According to the results, the diabetic patients were divided into two categories.

a. Group 1 – HbA1c value ≤6.5
b. Group 2 – HbA1c value >6.5.

Out of 50 diabetic patients, 14 patients (28%) had HbA1c value ≤6.5 and 36 patients (72%) had HbA1c value >6.5, Table 4.

Hearing Loss

Out of 50 diabetic patients, 34 patients (64%) had hearing loss when compared to non-diabetic patients, in which only one patient (2%) had hearing loss. $P < 0.001$ was shown highly significant relationship between Type 2 DM and hearing loss, Table 5.

PTA – THL

All the 100 patients were subjected to audiological evaluation with PTA. Among Group A; i.e., 50 diabetic patients, 34 (68%) had SNHL and the rest 16 (32%) patients were normal. Among Group B; i.e., 50 non-diabetic patients, only one patient (2%) had SNHL and the rest 49 patients (98%) were normal, Table 6.

PTA – Amount of Hearing Loss (AHL)

Grading of AHL in diabetic patients was done by dividing them into four groups.

a. Group 1 – No hearing loss
b. Group 2 – Mild hearing loss
c. Group 3 – Moderate hearing loss
d. Group 4 – Severe hearing loss

Table 3: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to duration of diabetes

Duration of diabetes	Frequency	Percentage
<5	13	26
5–10	23	46
>10	14	28
Total	50	100

Table 4: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to HbA1c level

HbA1C	Frequency	Percentage
≤6.5	14	28
>6.5	36	72
Total	50	100

Among the 34 diabetic patients with SNHL, majority, i.e., 17 patients (34%) had moderate SNHL while 14 patients (28%) had mild SNHL and three patients (6%) had severe SNHL.

In the non-diabetic group, only one patient (2%) had mild SNHL thus depicting that most of the Type 2 diabetic patients had moderate SNHL, Table 7.

Side of Hearing Loss

Out of 50 diabetic patients, 30 patients (60%) had bilateral SNHL and four patients (8%) had unilateral SNHL. Out of 50 non-diabetic patients, only one patient (2%) had unilateral SNHL, Table 8.

Association of Gender and AHL

According to the Chi-square test and *P* value, there is no significant correlation between gender and AHL, Table 9.

Association of Age and AHL

According to the Chi-square test and *P* value, there is no significant correlation between age and AHL, Table 10.

Association of Duration of Diabetes and AHL

According to the results obtained, as the duration of diabetes increases the AHL also increases, suggestive of a positive correlation. *P* value is less than 0.001, which states a highly significant relationship between the two, Table 11.

Association of HbA1c Level and AHL

According to the Chi-square test and *P* value, there is no significant correlation between age and AHL, Table 12.

The linear correlation between age and AHL was assessed using Pearson correlation product and the results were found to be positive.

Table 5: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to hearing loss

Hearing loss (puretone audiometry)	Diabetic		Non diabetic		Total		χ^2	df	*P*
	n	%	*n*	%	*n*	%			
No	16	32.0	49	98.0	65	65.0	47.868	1	<0.001
Present	34	68.0	1	2.0	35	35.0			
Total	50	100.0	50	100.0	100	100.0			

Table 6: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to type of hearing loss

Type of hearing loss	Diabetic		Non diabetic		Total		χ^2	df	*P*
	n	%	*n*	%	*n*	%			
No hearing loss	16	32	49	98	65	65	47.868	1	<0.001
Sensorineural hearing loss	34	68	1	2	35	35			
Total	50	100	50	100	100	100			

Table 7: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to amount of hearing loss

Amount of hearing loss	Diabetic		Non diabetic		Total		χ^2	df	*P*
	n	%	*n*	%	*n*	%			
No hearing loss	16	32	49	98	65	65	48.021	3	<0.001
Mild	14	28	1	2	15	15			
Moderate	17	34	0		17	17			
Severe	3	6	0		3	3			
Total	50	100	50	100	100	100			

Table 8: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to side of hearing loss

Side of hearing loss	Diabetic		Non diabetic		Total		χ^2	df	*P*
	n	%	*n*	%	*n*	%			
No hearing loss	16	32.0	49	98.0	65	65.0	48.554	2	<0.001
Unilateral hearing loss	4	8.0	1	2.0	5	5.0			
Bilateral hearing loss	30	60.0	0	0.0	30	30.0			
Total	50	100.0	50	100.0	100	100			

Pearson correlation r=0.865 $P < 0.001$.

The linear correlation between duration and AHL was assessed using Pearson correlation product and the results were found to be positive.

Pearson correlation r=0.878 $P < 0.001$.

The linear correlation between HbA1c level and AHL was assessed using Pearson correlation product and the results were found to be negative.

Pearson correlation r=−0.207 $P = 0.149$

Comparison of Auditory Threshold between Diabetic and Non-Diabetic Patients in Various Frequencies

As shown in Table 13, there is a significant difference in the auditory threshold at all frequencies from 250 Hz to 8000 Hz between Type II diabetic patients and non-diabetic patients and all the diabetic patients showed SNHL changes on audiogram.

The cases showed a gradual increase in hearing loss starting at 250 Hz and becoming maximum 8000 Hz. The result is highly significant as $P < 0.001$.

Auditory Thresholds (dB) in Age–Wise Sub Groups of Diabetics

As shown in Table 14, there is highly significant difference between the two age groups in diabetic patients. From

Table 9: Association of gender and amount of hearing loss

Amount of hearing loss	Male		Female		Total		χ^2	df	*P*
	n	%	*n*	%	*n*	%			
No hearing loss	11	35.5	5	26.3	16	32.0	1.549	3	0.671
Mild	8	25.8	6	31.6	14	28.0			
Moderate	11	35.5	6	31.6	17	34.0			
Severe	1	3.2	2	10.5	3	6.0			
Total	31	100.0	19	100.0	50	100.0			

Table 10: Association of age and amount of hearing loss

Amount of hearing loss	Age in years				Total		χ^2	df	*P*
	30–40		41–50						
	n	%	*n*	%	*n*	%			
No hearing loss	16	80.0	0	0.0	16	32.0	38.095	3	<0.001
Mild	4	20.0	10	33.3	14	28.0			
Moderate	0	0.0	17	56.7	17	34.0			
Severe	0	0.0	3	10.0	3	6.0			
Total	20	100.0	30	100.0	50	100.0			

Table 11: Association of duration of diabetes and amount of hearing loss

Amount of hearing loss	Duration of diabetes						Total		χ^2	df	*P*
	<5		5-10		>10						
	n	%	*n*	%	*n*	%	*n*	%			
No hearing loss	13	100.0	3	13.0	0	0.0	16	32.0	61.218	6	<0.001
Mild	0	0.0	14	60.9	0	0.0	14	28.0			
Moderate	0	0.0	5	21.7	12	85.7	17	34.0			
Severe	0	0.0	1	4.3	2	14.3	3	6.0			
Total	13	100.0	23	100.0	14	100.0	50	100.0			

Table 12: Association of HbA1c level and amount of hearing loss

Amount of hearing loss	HbA1c				Total		χ^2	df	*P*
	≤6.5		>6.5						
	n	%	*n*	%	*n*	%			
No hearing loss	3	21.4	13	36.1	16	32.0	5.091	3	0.165
Mild	2	14.3	12	33.3	14	28.0			
Moderate	8	57.1	9	25.0	17	34.0			
Severe	1	7.1	2	5.6	3	6.0			
Total	14	100.0	36	100.0	50	100.0			

250 Hz to 8000 Hz, there is a gradual increase in auditory threshold as the age increases. Age group of 41–50 years has higher auditory threshold at each frequency when compared to age group of 30–40 years.

Auditory Thresholds (dB) in Age–Wise Sub Groups of Non-diabetics

In the non-diabetic patients, there is no significant relationship between age-wise sub group and auditory threshold at various frequencies, supported by the *P* value, which is >0.005.

Auditory Thresholds (dB) in Duration – Wise Sub Groups of Diabetics

As shown in Tables 15 and 16, there is significant difference in the hearing threshold at various frequencies as the duration of diabetes increases. From 250 to 8000 Hz, there is gradual increase in the hearing threshold as the duration of diabetes increases. Duration of diabetes of more than 10 years has got significant increase in the hearing threshold when compared to patients with duration of diabetes <10 years.

Auditory Thresholds (dB) in HbA1C–Wise Sub Groups of Diabetics

As shown in Table 17, there is no significant relationship between HbA1C level and auditory threshold at various frequencies, Table 17.

Correlation between Age and Hearing Threshold at Particular Frequency

A positive relation is seen between age and auditory threshold at various frequencies, according to Pearson correlation. At all frequencies from 250 to 8000 Hz, there is a highly significant relationship between age and hearing thresholds, Table 18.

Table 13: Comparison of auditory threshold between Type 2 diabetic and non-diabetic patients in various frequencies

DM	250 Hz		500 Hz		1000 Hz		2000 Hz		4000 Hz		8000 Hz	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
No	0.02	0.14	12.76	3.95	12.92	3.86	12.26	4.50	11.24	5.31	12.96	5.20
Present	23.94	14.52	29.76	16.70	34.06	20.25	35.64	21.04	38.46	24.14	41.30	25.46
P	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	

DM: Diabetes mellitus

Table 14: Auditory thresholds (dB) in age–wise sub groups of diabetics

Age in DM	250 Hz		500 Hz		1000 Hz		2000 Hz		4000 Hz		8000 Hz	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
30–40	10.35	3.47	14.85	4.65	14.15	4.61	14.35	5.24	14.10	7.47	15.65	7.29
41–50	33.00	11.66	39.70	14.20	47.33	14.94	49.83	14.46	54.70	16.32	58.40	17.45
P	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	

DM: Diabetes mellitus

Table 15: Auditory thresholds (dB) in age–wise sub groups of non-diabetics

Age in non DM	250 Hz		500 Hz		1000 Hz		2000 Hz		4000 Hz		8000 Hz	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
30–40	0.00	0.00	11.65	2.76	11.90	2.43	11.00	2.62	10.40	2.91	11.80	2.55
41–50	0.03	0.18	13.50	4.46	13.60	4.49	13.10	5.29	11.80	6.42	13.73	6.32
P	0.420		0.105		0.129		0.107		0.366		0.021	

DM: Diabetes mellitus

Table 16: Auditory thresholds (dB) in duration–wise sub groups of diabetics

Duration of DM	250 Hz		500 Hz		1000 Hz		2000 Hz		4000 Hz		8000 Hz	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
<5	8.77	2.45	12.92	3.33	12.31	2.06	11.85	2.70	11.00	2.97	12.23	2.62
5–10	24.22	11.18	30.43	13.41	34.70	16.40	37.35	16.62	41.13	19.32	44.09	20.85
>10	37.57	12.11	44.29	15.02	53.21	14.76	54.93	14.93	59.57	17.63	63.71	17.37
P	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	

DM: Diabetes mellitus

Result of OAE in Diabetic and Non-Diabetic Patients

Out of the 50 diabetic patients, 34 patients (68%) had abnormal OAE while only one patient (2%) out of 50 non-diabetic patients had abnormal OAE, Tables 19 and 20.

Association of OAE and Hearing Loss

All the patients with hearing loss, i.e. 34 diabetic patients and one non-diabetic patient had abnormal OAE. This depicts that OAE has 100% sensitivity and specificity and can be used as a screening test for early detection of hearing loss in Type 2 diabetic patients [Figures 1-15 and Graphs 1-16].

DISCUSSION

The aim of the present study is to assess the prevalence of hearing loss in Type 2 diabetic patients when compared to non-diabetic patients.

The present study reports SNHL in 68% of Type 2 diabetic patients and 2% of non-diabetic patients. The majority of them had bilateral moderate SNHL. There was a significant difference in the auditory thresholds at all frequencies from 250 Hz to 8000 Hz between Type 2 diabetic patients and non-diabetic patients. There was a gradual increase in hearing loss from 250 Hz to 8000 Hz for the diabetic patients.

The previous studies in the literature show the prevalence of hearing loss in diabetic patients ranging from 13% to 95%. Out study results are comparable to those of Aggarwal *et al.*[71] (64.86%), Rajendran *et al.* (73.3%), and Harkare *et al.* (74%). The results are higher when compared to those of Somogyi *et al.* (34%) and Saini *et al.* (30%).[72-75]

Researches have started long before as early as 1975, when Friedman *et al.*[1] had compared 20 DM patients with age- and gender-matched normal individual and found that hearing threshold was elevated in at least one frequency of 11 patients (55%).

In 1977, Snashall made a proposal of premature hearing deterioration in DM patients.[76] Kurien *et al.*, in 1989, studied 30 DM patients with age <50 years and found that average hearing threshold was higher when compared to 30 age- and gender-matched non-DM patients.[77] In 1995,[17] a study conducted by Tay, compared 102 DM patients with 102 age- and gender-matched non-DM patients and found that the mid and low frequency thresholds are affected significantly and there was correlation with duration of diabetes but not with stages of retinopathy.

The diabetic patients had more hearing loss in high frequencies (4, 8 kHz) compared to the non-diabetic patients and this loss was more in the chronic diabetic patients in the study conducted by Naini *et al.*15 I`.[78]

Comparative study between DM patients and non-DM patients in 2008 by Loader *et al.* demonstrated higher

Table 17: Auditory thresholds (dB) in HbA1C–wise sub groups of diabetics

HbA1C	250 Hz		500 Hz		1000 Hz		2000 Hz		4000 Hz		8000 Hz	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
≤6.5	29.57	14.62	35.43	15.78	42.79	19.05	44.07	19.84	47.93	23.31	50.57	24.93
>6.5	21.75	14.07	27.56	16.74	30.67	19.93	32.36	20.84	34.78	23.76	37.69	25.08
P	0.087		0.136		0.057		0.077		0.084		0.109	

Table 18: Correlation between age and hearing threshold at particular frequency

Correlation between age and auditory threshold at various frequencies	Pearson correlation r–value	*P* value
250	0.393	<0.001
500	0.501	<0.001
1000	0.521	<0.001
2000	0.525	<0.001
4000	0.504	<0.001
8000	0.521	<0.001

Table 19: Result of otoacoustic emission in diabetic and non-diabetic patients

Otoacoustic emission	Diabetic		Non diabetic		Total		χ^2	df	*P*
	n	%	*n*	%	*n*	%			
Pass	16	32	49	98	65	65	47.868	1	<0.001
Refer	34	68	1	2	35	35			
Total	50	100	50	100	100	100			

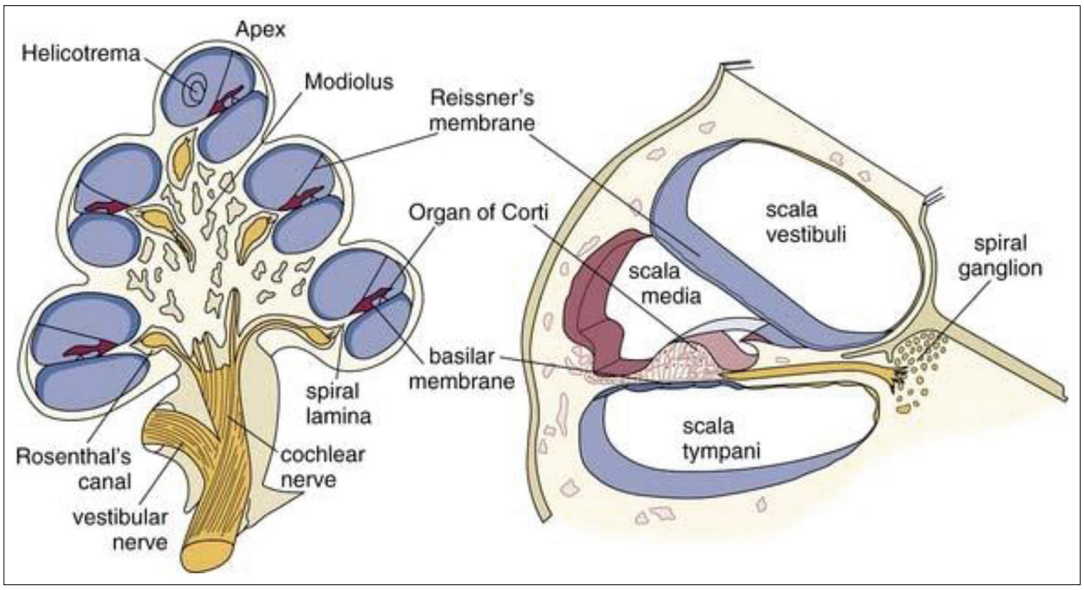


Figure 1: Cross section of the cochlea

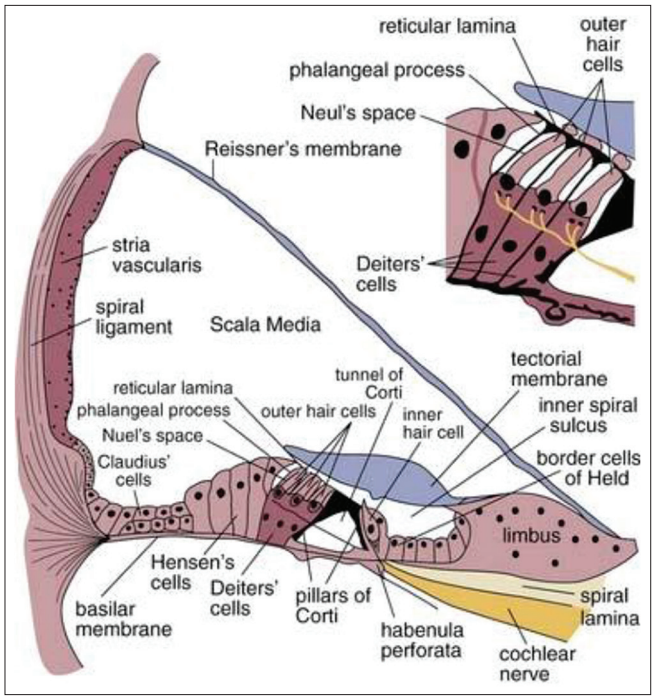


Figure 2: Cross section of the organ of corti

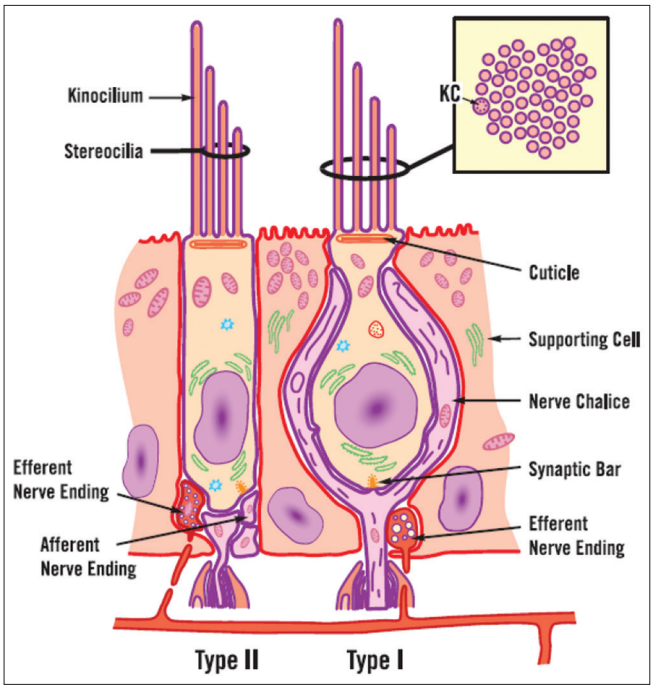


Figure 3: Hair cells

hearing threshold in DM patients at all frequency in PTA, and the mechanism was thought to be cochlear microvascular abnormality.[79]

In our study, TEOAE was done in all patients, both Type 2 diabetic and non-diabetic patients and it was found to be refer, i.e., abnormal in all (34) Type 2 diabetic patients with hearing loss and in one non-diabetic patient with

Table 20: Association of otoacoustic emission and hearing loss

Otoacoustic emission	Hearingloss		Total
	Present	Absent	
Refer	35	0	35
Pass	0	65	65
Total	35	65	100

Sensitivity=100%, Specificity=100%

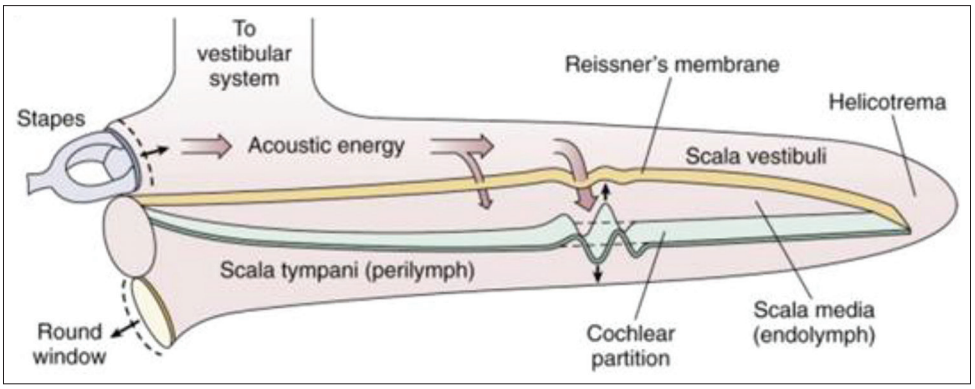


Figure 4: Schematic diagram showing sound propagation in the cochlea

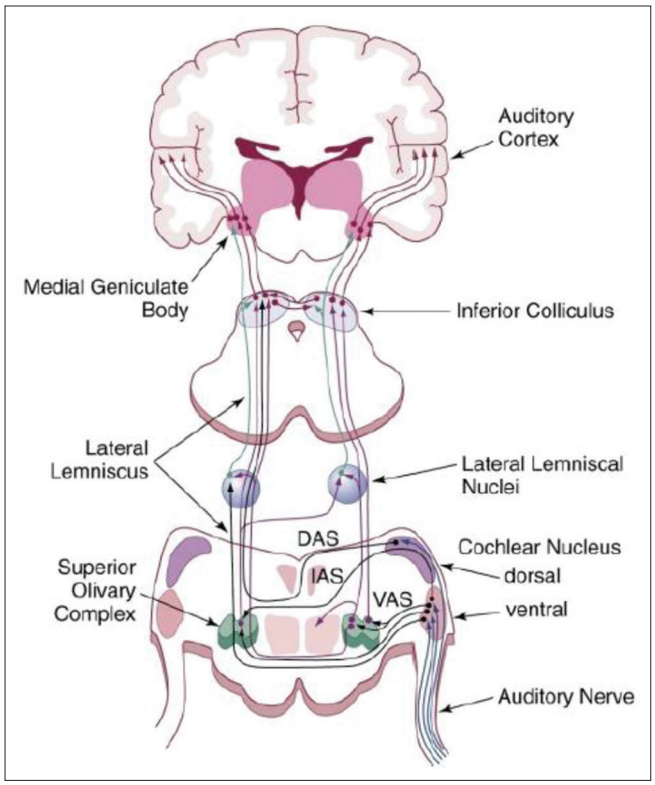


Figure 5: Auditory pathway

Figure 6: Pure tone audiometer

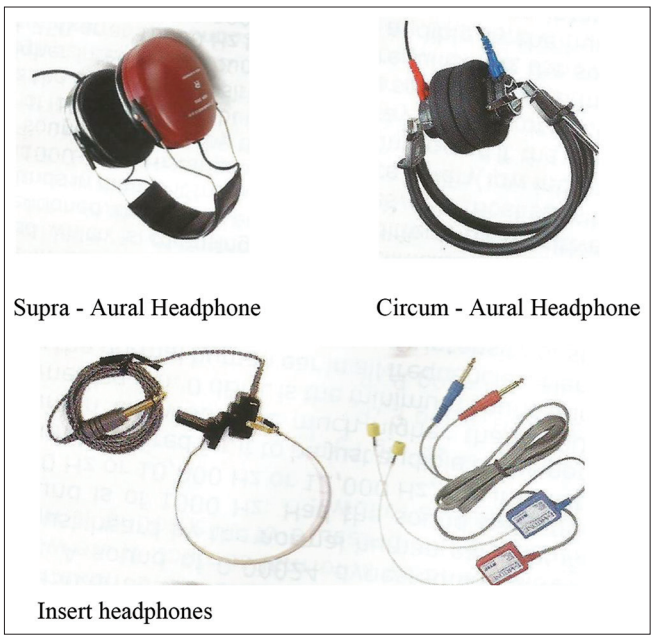


Figure 7: Headphones

hearing loss. Thus highlighting the benefit of TEOAE as a screening tool for early detection of hearing loss in Type 2 diabetic patients.

The amplitude of TEOAE's was reduced across speech frequency (1.5–4 kHz) in all 34 type 2 diabetic patients and one non-diabetic patient with hearing loss.

Wang[80] in 1998 demonstrated abnormal DPOAE's in 19 DM patients when compared to non-DM patients and made an inference of early detection of hearing impairment in DM as DPOAE helps in revealing early cochlear dysfunction. In 1998, Alborch[81] studied 20 Type 1 DM patients and came to a conclusion that cochlear dysfunction in DM patients is related to OHC injury.

Lisowska[82] found reduced DPOAE amplitude in Type 1 DM patients in a comparative study between age- and gender-matched 42 Type 1 DM and 33 non-DM individuals.

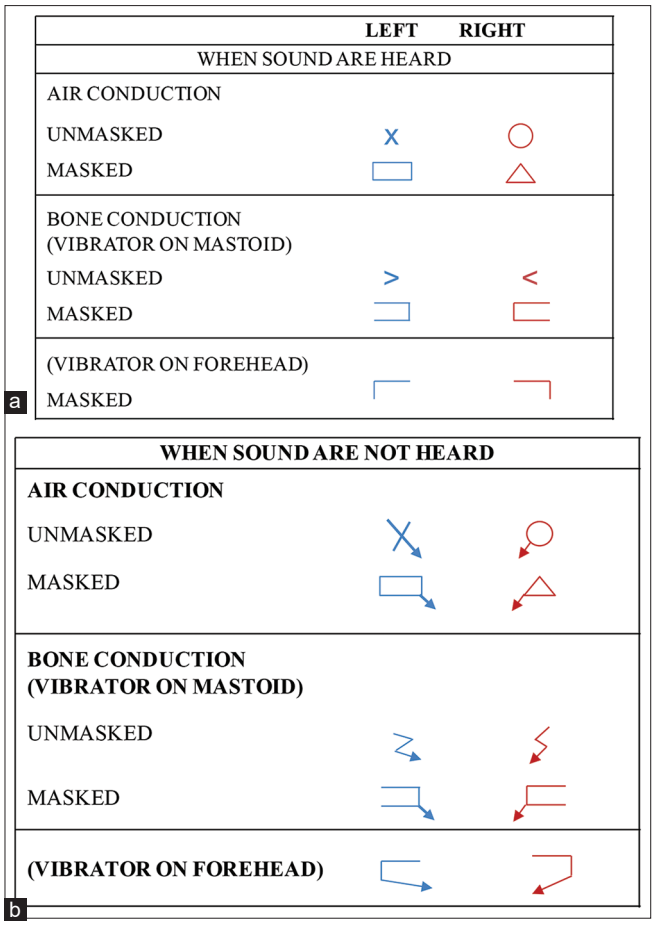


Figure 8: (a and b) Symbols conventionally used for plotting the pure tone audiogram

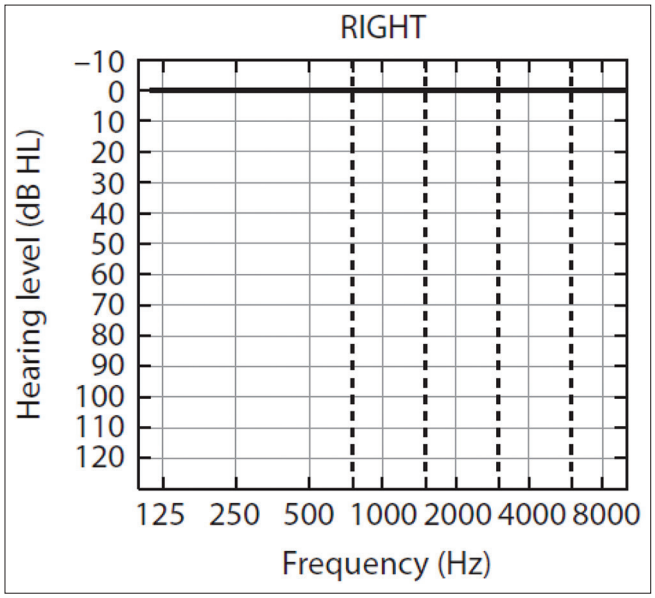


Figure 9: Audiogram chart used in clinical audiometry

Ottaviani[83] conducted a study using OAE in 60 insulin-dependent DM patients and age- and gender-matched 58 non-DM individuals and demonstrated that OAE was

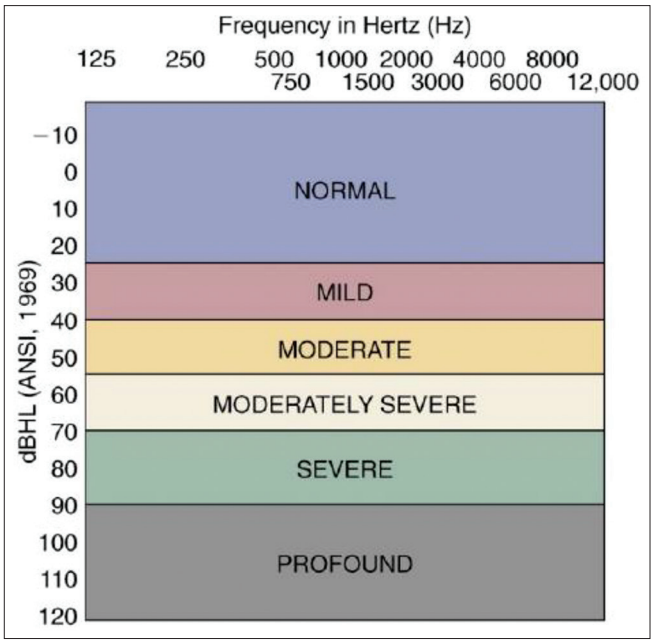


Figure 10: Graphic representation of categories of hearing loss

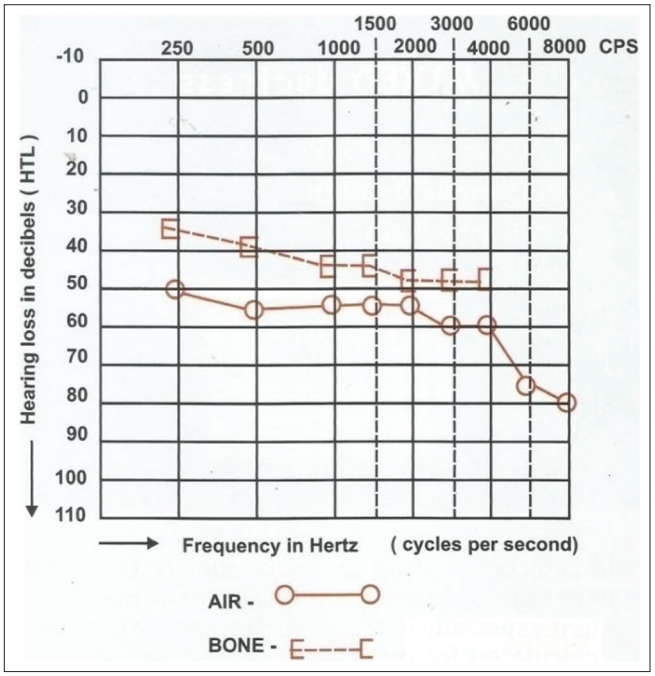


Figure 11: Audiogram showing moderate degree of sensorineural hearing loss

unilaterally or bilaterally abnormal in some Type1 DM patients. He also showed that the magnitude of OAE's to be reduced across speech frequencies (1–4 kHz).

Literature indicates that individuals with significant hearing loss have no measurable OAE's.[84] Simoncelli *et al.*[85] conducted a study, which showed that mean OAE intensity and amplitude by 100 Hz frequency band were significantly lower in diabetic patients when compared to non-diabetic

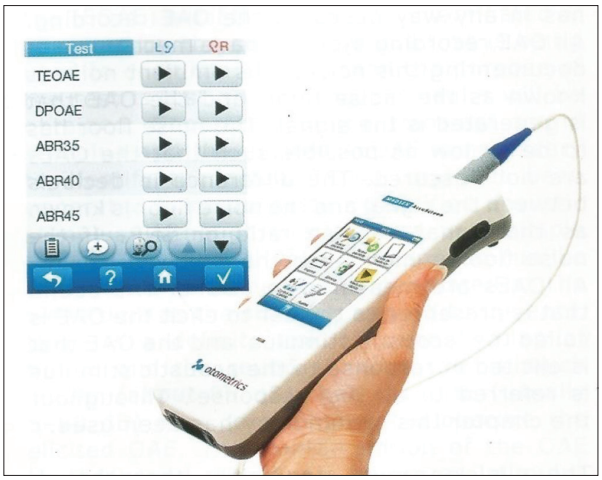


Figure 12: Otometrics portable otoacoustic emission screener

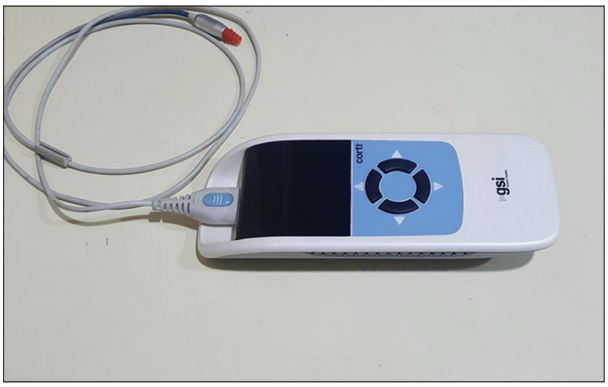


Figure 13: Gsi corti portable otoacoustic emission screener

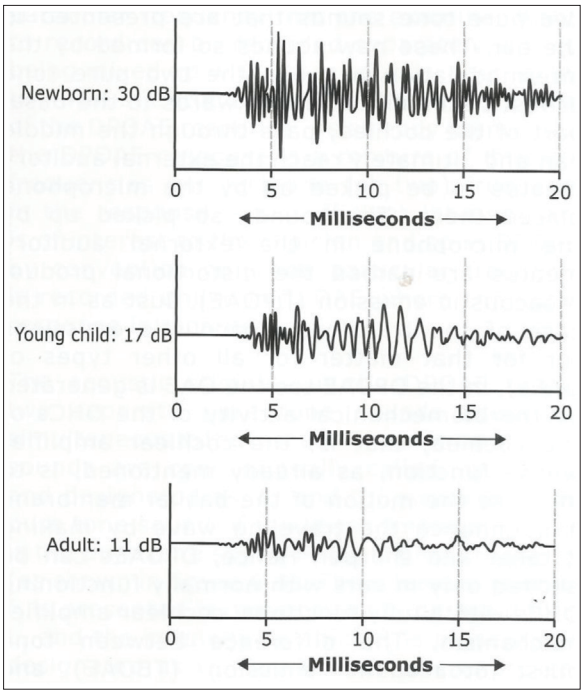


Figure 14: The amplitude of transient evoked otoacoustic emissions in response to a click sound in newborns, young children, and adults

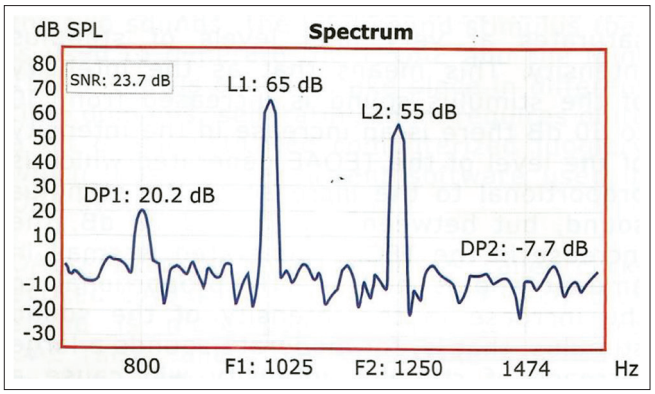
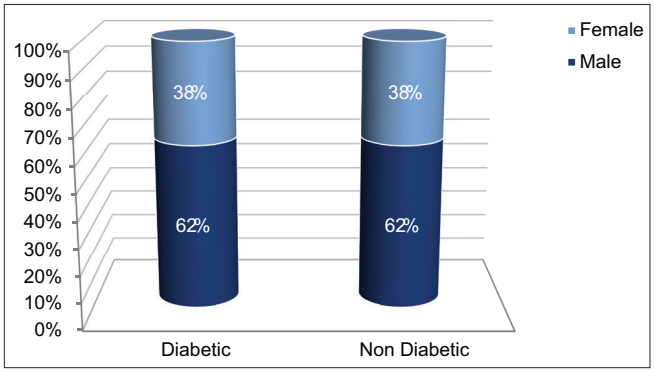
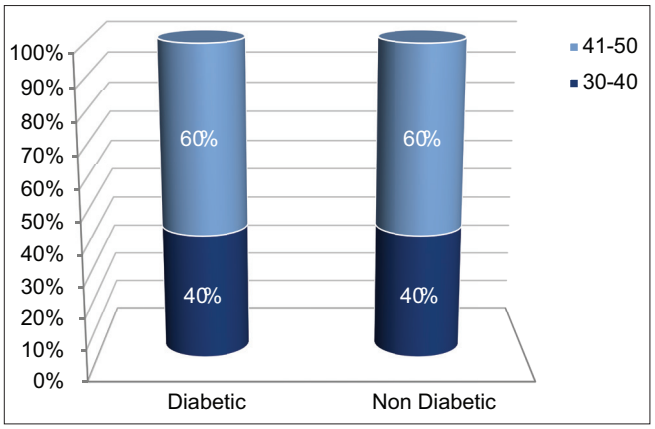


Figure 15: Representation of distortion product otoacoustic emission test



Graph 1: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to gender

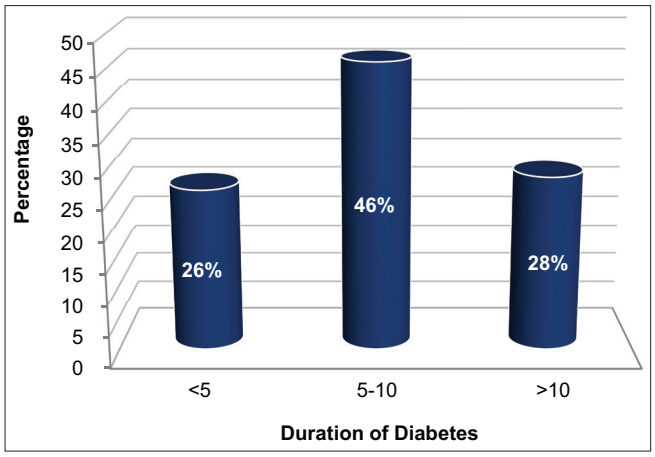


Graph 2: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to age

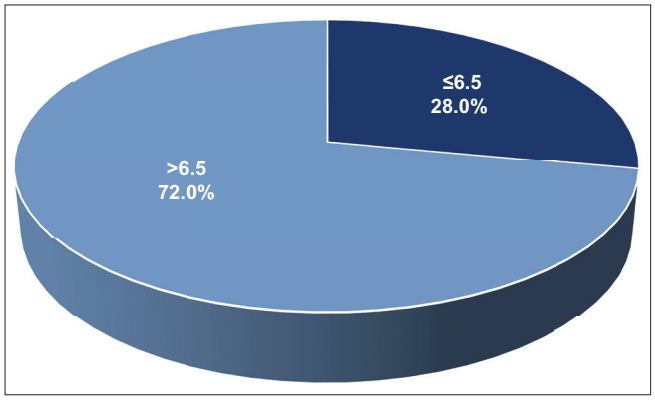
patients, indicating an alteration in cochlear biomechanics in diabetes.

Our study also compared the correlation of age, duration of diabetes, and HbA1c level with hearing loss. Age and duration of diabetes demonstrated a positive correlation with AHL, while HbA1c level had no significant correlation.

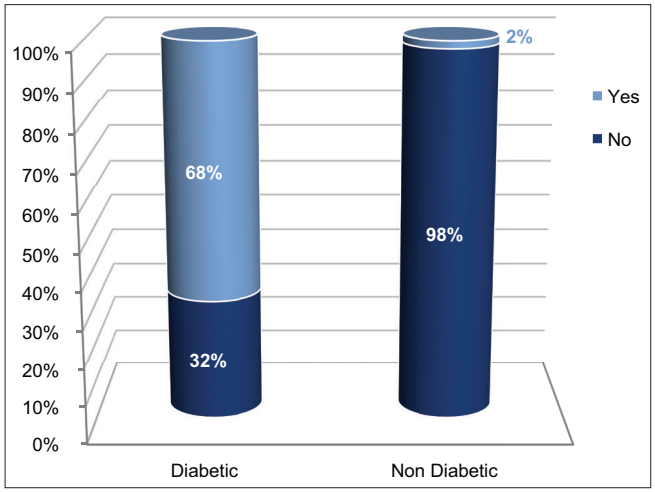
Comparable relationship between duration of diabetes and hearing loss was seen in a study conducted by Lasisi



Graph 3: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to duration of diabetes

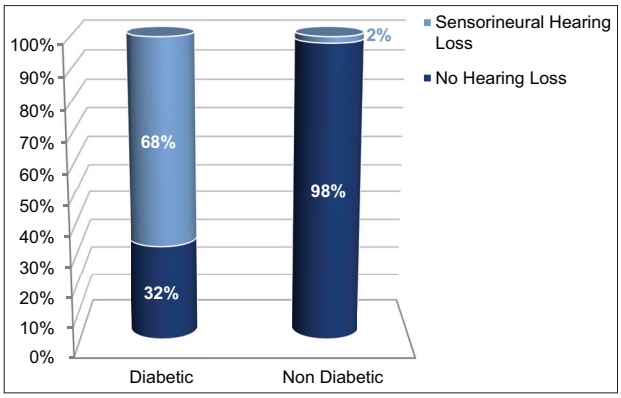


Graph 4: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to HbA1c level

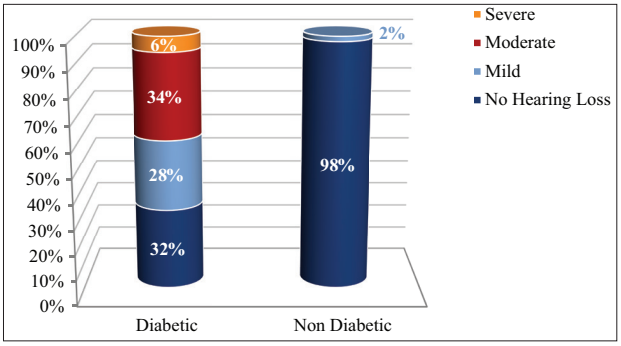


Graph 5: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to hearing loss

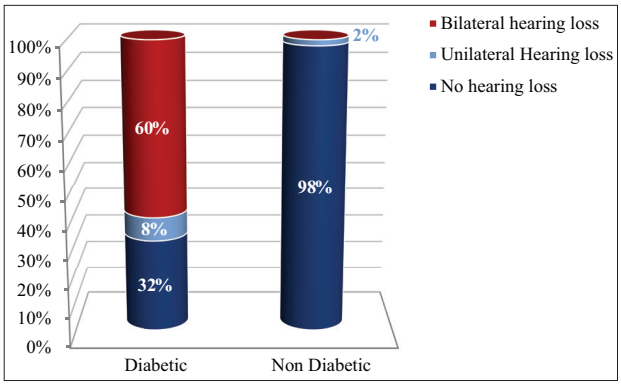
in 2003,[2] which showed a higher bone conduction threshold in 13 DM patients, when compared to non-DM patients, especially in patients with duration of diabetes >10 years. In 2005, Diaz[86] demonstrated hearing loss to increase with age in a study conducted by comparing 92



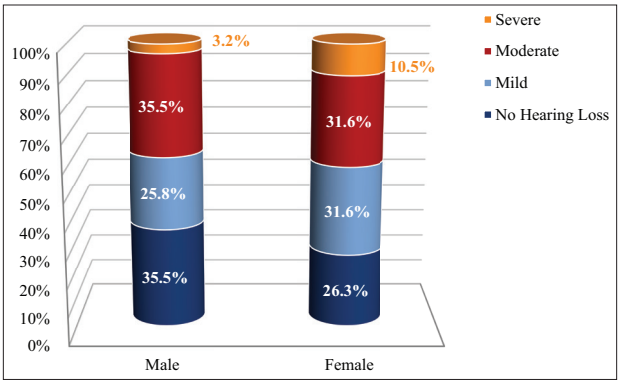
Graph 6: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to type of hearing loss



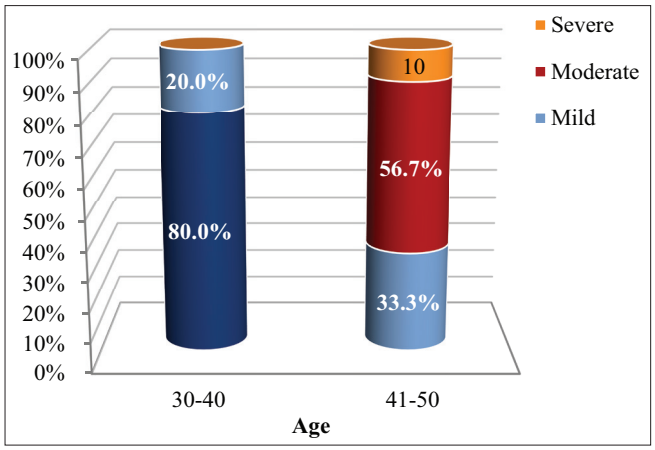
Graph 7: Percentage distribution of the Type 2 diabetic and non-diabetic patients according to amount of hearing loss



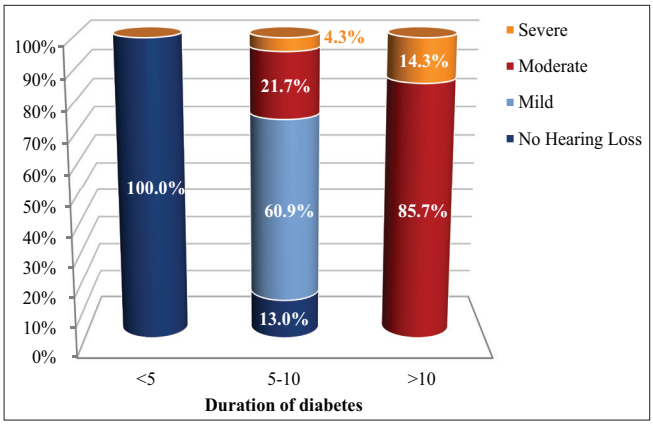
Graph 8: Percentage distribution of the type 2 diabetic and non-diabetic patients according to side of hearing loss



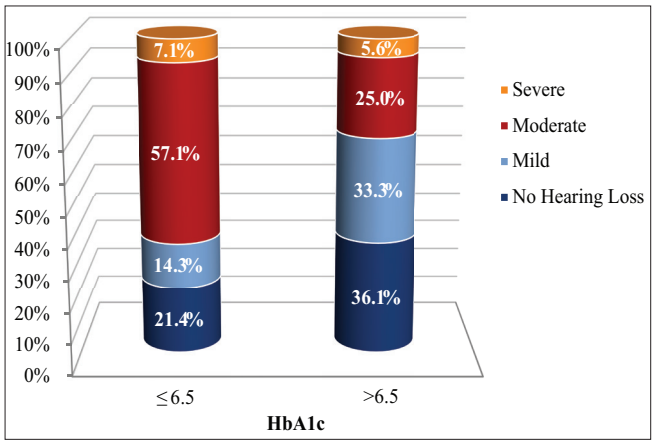
Graph 9: Association of gender and amount of hearing loss



Graph 10: Association of age and amount of hearing loss



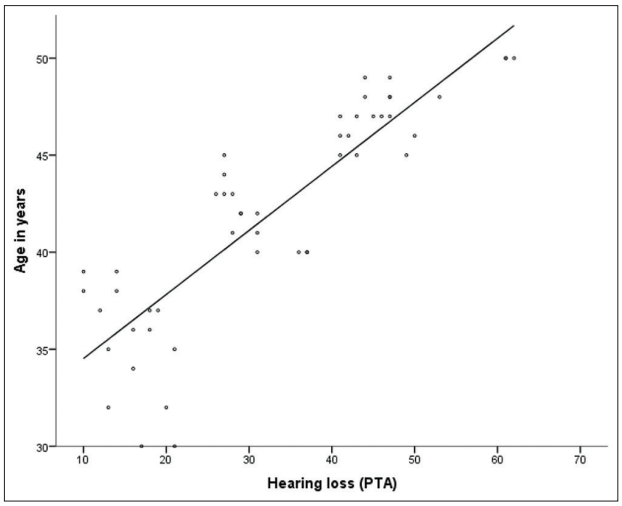
Graph 11: Association of duration of diabetes and amount of hearing loss



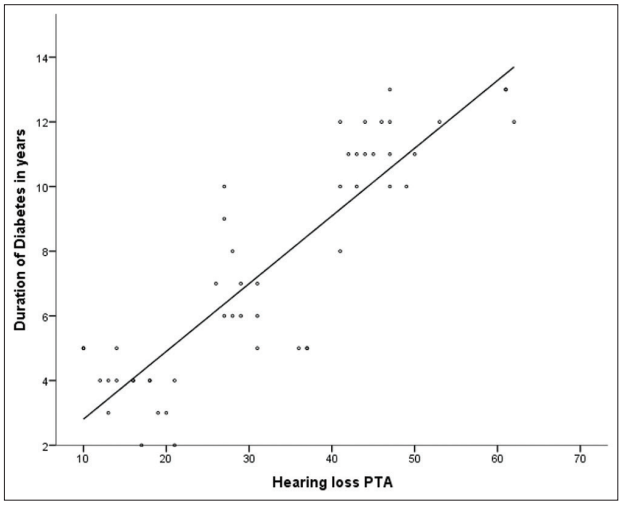
Graph 12: Association of HbA1c level and amount of hearing loss

Type 2 DM patients with age- and gender-matched 92 non-DM patients.

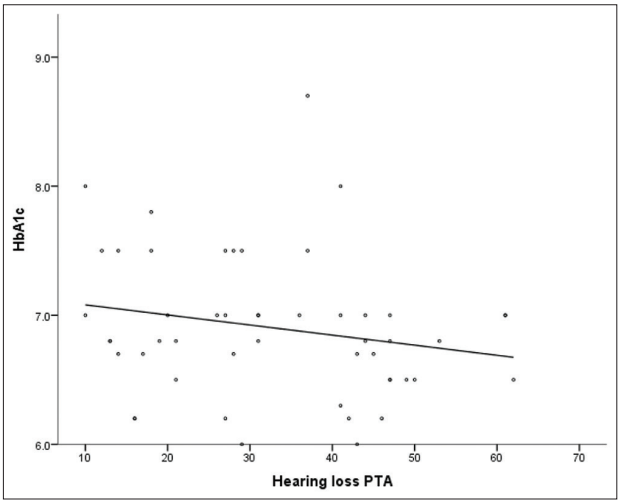
In our study, HbA1c level had no correlation with hearing loss, which is comparable to other study results.



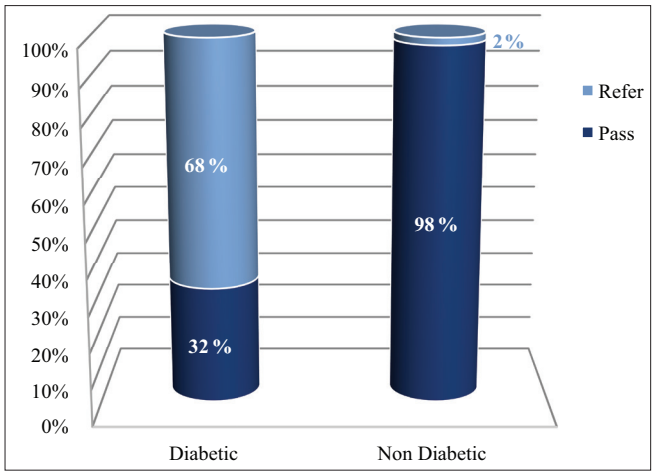
Graph 13: Scatter plot chart showing linear correlation between age and amount of hearing loss



Graph 14: Scatter plot chart showing linear correlation between duration and amount of hearing loss



Graph 15: Scatter plot chart showing linear correlation between HbA1c level and amount of hearing loss



Graph 16: Result of otoacoustic emission in diabetic and non-diabetic patients

Year of study	Authors	Percentage of sensorineural hearing loss
1969	Nagoshi *et al*.[87]	54
1975	Friedman *et al*.[1]	55
1998	Boomsma and Stolk[88]	48
1998	Aggarwal *et al*.[71]	64.86
2002	Rózańska-Kudelska *et al*.[89]	95
2003	Kakarlapudi *et al*.[4]	13.1
2005	Weng *et al*.[16]	44.8
2010	Mozaffari *et al*.[90]	45
2010	Santosh[91]	61.67
2011	Rajendran *et al*.[72]	73.3
2011	Pemmaiah and Srinivas[92]	43.6
2012	Harkare *et al*.[73]	74
2013	Somogyi *et al*.[74]	34
2013	Krishnappa and Naseeruddin[93]	73.58
2013	Bhaskar[94]	78.2
2014	Saini *et al*.[75]	30
2018	Our study	68

CONCLUSION

The present study reports SNHL in 68% of Type II diabetic subjects and 2% of healthy non-diabetic subjects. The majority of the patients had bilateral moderate SNHL.

The diabetic patients had increased hearing threshold at all frequencies with gradual increase in hearing loss from 250 Hz to 8000 Hz.

Age and duration of diabetes had a positive correlation with hearing loss. As the age increased, AHL also increased. Similarly, as duration of diabetes increased, AHL also increased. HbA1c had no relation with auditory threshold and AHL. TEOAE can be used as a screening test for the early detection of hearing loss in Type 2 diabetic patients, as the result was abnormal in all the patients with hearing loss.

Since hearing loss can be considered to be a consequence of diabetes, a metabolic assessment may be useful for patients presenting with hearing loss so as to reduce the high rate of undiagnosed DM in the community. The use of audio logical tests to monitor hearing in diabetic patients should be considered as a routine procedure so that quality of life can be improved for long standing diabetics with needed therapeutic interventions for hearing improvement.

Limitations

1. Sample size was small: Larger sample size with wide geographical area could have yielded more better results
2. PTA used as the diagnostic test needed patient's attention and cooperation, which makes it a subjective test
3. Sub clinical hearing loss could not be assessed.

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ANNEXURE I

Pro Forma

QUESTIONNAIRE

"COMPARATIVE STUDY OF HEARING ASSESSMENT IN DIABETIC AND NON DIABETIC PATIENTS USING OTOACOUSTIC EMISSION AND PURE TONE AUDIOMETRY"

I. PERSONAL DETAILS

1. PROFORMA NO: ☐
2. MRD NO: ☐
3. NAME: ☐
4. AGE: ☐ 30-40 ☐ 41-50 ☐
5. GENDER: Male ☐ Female ☐
6. OCCUPATION: ☐
7. ADDRESS: ☐
8. MOBILE NO: ☐

I. IS THERE ANY HISTORY OF DIABETES Yes ☐ No ☐

IF YES

A. DURATION OF DIABETES <5 Yrs ☐ 5-10 Yrs ☐ >10 Yrs ☐

B. HbA1c LEVEL ≤6.5 ☐ >6.5 ☐

II. IS THERE ANY HISTORY OF EAR PATHOLOGY CAUSING HEARING LOSS Yes ☐ No ☐

V. IS THERE ANY HISTORY OF INTAKE OF OTOTOXIC DRUGS Yes ☐ No ☐

. IS THERE ANY HISTORY OF FREQUENT NOISE EXPOSURE Yes ☐ No ☐

I. IS THERE ANY FAMILY HISTORY OF DEAFNESS Yes ☐ No ☐

II. IS THERE ANY FAMILY HISTORY OF AUTOIMMUNE DISEASE Yes ☐ No ☐

III. IS THERE ANY FAMILY HISTORY OF HYPERTENSION Yes ☐ No ☐

X. IS THERE ANY HISTORY OF EAR SURGERY IN THE PAST Yes ☐ No ☐

. RESULT OF OTOACOUSTIC EMISSION PASS ☐ REFER ☐

I. RESULT OF PURE TONE AUDIOMETRY

A. TYPE OF HEARING LOSS

RIGHT ☐

LEFT ☐

B. AMOUNT OF HEARING LOSS IN DECIBELS

RIGHT ☐ LEFT ☐

Annexure-IIa

Key to Master Chart I

1. Gender
   a. Male–0
   b. Female–1

2. Age group
   a. 30 years–40 years–0
   b. 41 years–50 years–1

3. Type 2 Diabetes Mellitus
   a. No–0
   b. Yes–1

4. DD
   a. <5 years–0
   b. 5 years–10 years–1
   c. >10 years–2

5. HbA1c
   a. ≤6.5–0
   b. >6.5–1

6. History of ear disease
   a. No–0
   b. Yes–1

7. Intake of ototoxic drugs
   a. No–0
   b. Yes–1

8. Frequent noise exposure
   a. No–0
   b. Yes–1

9. FHD
   a. No–0
   b. Yes–1

10. Previous ear surgery
    a. No–0
    b. Yes–11

11. AD
    a. No–0
    b. Yes–1

12. HTN
    a. No–0
    b. Yes–1

13. OAE
    a. Pass–0
    b. Refer–1

14. PTA
    a. THL
       i. No hearing loss–0
       ii. Sensorineural hearing loss–1
       iii. Conductive hearing loss–2
    b. AHL
       i. No hearing loss–0
       ii. Mild–1
       iii. Moderate–2
       iv. Severe–3

Annexure-IIIa

Master Chart 1

S. No	Gender	AG	T2DM	DD	HbA1c	HED	IOD	FNE	FHD	PES	AD	HTN	OAE	PTA				
														THL		AHL		
													R	L	R	L	R	L
1	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
2	1	0	1	1	1	0	0	0	0	0	0	0	1	0	1	0	1	0
3	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
4	1	0	1	1	1	0	0	0	0	0	0	0	1	0	1	0	1	0
5	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
6	0	0	1	1	1	0	0	0	0	0	0	0	1	0	1	0	1	0
7	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
8	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
9	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
10	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
11	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
12	0	0	1	1	1	0	0	0	0	0	0	0	1	0	1	0	1	0
13	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
14	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
15	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
16	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
17	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
18	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
19	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
20	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
21	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	3	3
22	0	1	1	1	0	0	0	0	0	0	0	0	1	1	1	1	2	2
23	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
24	1	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
25	1	1	1	2	1	0	0	0	0	0	0	0	1	1	1	1	2	2
26	1	1	1	2	1	0	0	0	0	0	0	0	1	1	1	1	2	2
27	1	1	1	2	1	0	0	0	0	0	0	0	1	1	1	1	3	3
28	1	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
29	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	2	2
30	1	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
31	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	2	2
32	1	1	1	2	0	0	0	0	0	0	0	0	1	1	1	1	3	3
33	1	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
34	1	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	2	2
35	0	1	1	2	1	0	0	0	0	0	0	0	1	1	1	1	2	2
36	0	1	1	2	1	0	0	0	0	0	0	0	1	1	1	1	2	2
37	0	1	1	2	0	0	0	0	0	0	0	0	1	1	1	1	2	2
38	1	1	1	2	0	0	0	0	0	0	0	0	1	1	1	1	2	2
39	0	1	1	2	0	0	0	0	0	0	0	0	1	1	1	1	2	2
40	1	1	1	2	0	0	0	0	0	0	0	0	1	1	1	1	2	2
41	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
42	1	1	1	2	1	0	0	0	0	0	0	0	1	1	1	1	2	2
43	0	1	1	2	1	0	0	0	0	0	0	0	1	1	1	1	2	2
44	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
45	0	1	1	1	1	0	0	0	0	0	0	0	1	1	1	1	1	1
46	0	1	1	2	0	0	0	0	0	0	0	0	1	1	1	1	2	2
47	0	1	1	1	0	0	0	0	0	0	0	0	1	1	1	1	1	1
48	0	1	1	1	0	0	0	0	0	0	0	0	1	1	1	1	2	2
49	0	1	1	2	0	0	0	0	0	0	0	0	1	1	1	1	2	2
50	0	1	1	1	0	0	0	0	0	0	0	0	1	1	1	1	1	1
51	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
52	1	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
53	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
54	1	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
55	1	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
56	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
57	1	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
58	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0

(Contd...)

Continued...

S. No	Gender	AG	T2DM	DD	HbA1c	HED	IOD	FNE	FHD	PES	AD	HTN	OAE	PTA				
														THL		AHL		
													R	L	R	L	R	L
59	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
60	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
61	1	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
62	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
63	1	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
64	1	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
65	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
66	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
67	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
68	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
69	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
70	0	0	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
71	0	1	0	–	0	0	0	0	0	0	0	0	1	1	1	0	1	0
72	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
73	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
74	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
75	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
76	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
77	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
78	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
79	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
80	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
81	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
82	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
83	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
84	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
85	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
86	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
87	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
88	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
89	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
90	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
91	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
92	1	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
93	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
94	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
95	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
96	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
97	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
98	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
99	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0
100	0	1	0	–	0	0	0	0	0	0	0	0	0	0	0	0	0	0

AG: Age groups, T2DM: Type 2 diabetes mellitus, DD: Duration of diabetes, HED: History of ear disease, IOD: Intake of ototoxic drugs, FNE: Frequent noise exposure, FHD: Family history of deafness, PES: Previous ear surgery, AD: Autoimmune disease, HTN: Hypertension, OAE: Otoacoustic emission, PTA: Pure tone audiometry, THL: Type of hearing loss, AHL: Amount of hearing loss

Annexure-IIb

Key to Master Chart II

1. Age
2. HbA1c level
3. Duration of diabetes

S. No	Age	HbA1c	Duration of diabetes	S. No	Age	HbA1c	Duration of diabetes
1	35	6.8	4	26	48	6.8	12
2	40	7.0	5	27	50	7.0	13
3	36	7.8	4	28	43	7.5	6
4	40	7.5	5	29	45	8.0	8
5	34	6.2	4	30	42	6.8	7
6	40	7.0	5	31	47	7.0	10
7	37	6.8	3	32	50	6.5	12
8	30	6.5	2	33	42	7.5	6
9	32	7.0	3	34	46	7.0	10
10	39	7.5	4	35	47	6.7	11
11	30	6.7	2	36	48	6.8	12
12	40	8.7	5	37	46	6.5	11
13	32	6.8	3	38	47	6.2	12
14	37	7.5	4	39	49	6.5	13
15	37	7.5	4	40	47	6.3	12
16	39	8.0	5	41	41	7.0	6
17	38	6.7	5	42	48	6.8	11
18	35	6.8	4	43	49	7.0	12
19	36	6.2	4	44	45	7.0	10
20	38	7.0	5	45	43	7.5	8
21	50	7.0	13	46	48	6.5	11
22	45	6.5	10	47	44	6.2	9
23	41	6.7	6	48	45	6.0	10
24	43	7.0	7	49	46	6.2	11
25	47	6.7	11	50	42	6.0	7

Annexure-IIIb

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Annexure-IV

List of Abbreviations
AC-Air conduction
ASHA-American speech and hearing association
AVCN-Anterior ventral cochlear fluid
BC-Bone conduction
CSF-Cerebrospinal fluid
dB-Decibel
DCN-Dorsal cochlear nucleus
DM-Diabetes mellitus
DPOAE-Distortion product otoacoustic emission
Hz-Hertz
IHC-Inner hair cells
IL-Intensity level
OAE-Otoacoustic emission
OHC-Outer hair cells
PTA-Pure tone audiometry
PVCN-Posterior ventral cochlear fluid
RWM-Round window membrane
SL-Sensation level
SNHL-Sensorineural hearing loss
SPL-Sound pressure level
TEOAE-Transient evoked otoacoustic emission.

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Original Article

Lung Function and Health Status in Patients of Chronic Obstructive Pulmonary Disease on Treatment with Tiotropium

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Abstract

Introduction: Chronic obstructive pulmonary disease (COPD) is a group of chronic and slowly progressive respiratory disorder characterized by reduced maximum expiratory flow during forced exhalation. Tiotropium, a long-acting antimuscarinic agent, has well-known documented effect on improving lung function and quality of life (QOL). There are many studies globally on tiotropium and its effect on lung function, but limited studies available in our Indian set up. Hence, we planned this study.

Materials and Methods: Patients were recruited from chest clinic and outpatient department from the Department of Medicine of University College of Medical Sciences and GTB Hospital. It was a prospective observational cohort study conducted from November 2017 to April 2019. Tiotropium was given as meter dose inhaler in dose of 18 µg per dose, in schedule as prescribed by the Global Initiative for Chronic Obstructive Lung Disease-2017 guidelines. Patients were followed up for 3 months with periodic assessment of lung functions, Saint George's Respiratory Questionnaire (SGRQ) score, and symptoms assessment.

Results: A total of 65 patients were recruited for study which included 57 (87.7%) males and 8 (12.3%) females. Among the pulmonary function tests measured, there is a significant change in mean forced expiratory volume (FEV_1) at the end of follow-up period compared to FEV_1 at baseline. There is a significant change in mean forced vital capacity at the end of follow-up study compared to start of the study. There was no significant change in mean SGRQ score after 1 month of start of drug, but significant statistical change observed at end of the 3rd month of the study compared to the 1st month that implies SGRQ score decreased and patients health status and QOL improved. There is a significant change in mean SGRQ score at the end of follow-up study compared to baseline. In our study, 16 patients (24.6%) complained of dry mouth, 7 (10.7%) complained of pharyngitis or throat irritation, and 3 (4.6%) patients complained of constipation.

Conclusion: There was a statistically significant change in lung functions and improvement in QOL scores as assessed by SGRQ at the end of the study compared to baseline by use of inhaled tiotropium in COPD patients.

Key words: Chronic obstructive pulmonary disease, Long-acting antimuscarinic agents, Quality of life, Saint George's Respiratory Questionnaire, Tiotropium

INTRODUCTION

Chronic obstructive pulmonary disease (COPD) is an underdiagnosed, progressive, incurable lung disease. Its prevalence is on rise globally as well as in India. COPD is currently the fourth leading cause of death in the world,[1] but its projected to be third leading cause by 2020.[2] The national burden was thus estimated to be 14.84 million. In the past 50 years, COPD has overtaken tuberculosis and pneumonia as the leading cause of death.

Quality of life (QOL) is a much broader multidimensional concept. The World Health Organization defines QOL as the individuals perception of their position in life in the context of culture and value system in which they live and in relation to their goals, expectations, standards, and concerns.

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Health status is defined as perceived heath in descriptive terms of physical and mental symptoms, disability, and social dysfunction related to the health condition.[1] Saint George's Respiratory Questionnaire (SGRQ)[2] and COPD assessment test (CAT) measure health status of COPD patient in recent scenario. Spirometry is required to make diagnosis of COPD, the presence of post-bronchodilator (BD) forced expiratory volume (FEV_1)/forced vital capacity (FVC) <0.7 confirms the presence of persistent airflow limitation.[3] The Global Initiative for Chronic Obstructive Lung Disease (GOLD) assigns patients with COPD into four groups based on degree of airflow restriction, symptom score as per CAT test, and number of exacerbation in 1 year. Group A comprises low risk and less symptoms, Group B comprises low risk and more symptoms, Group C comprises high risk and less symptoms, and Group D comprises high risk and more symptoms.

Tiotropium bromide, a long-acting antimuscarinic agent (LAMA) used in our study, has well-known documented effect on improving lung function and QOL. There are many studies globally on tiotropium and its effect on lung function, but limited studies available in our Indian set up. Tiotropium bromide rapidly dissociates autoinhibitory M2 receptors, but slowly dissociates from M1 and M3 receptors which mediate acetylcholine-mediated bronchoconstriction and mucus secretion. This increased duration of binding at M3 receptor result in prolonged bronchodilation allowing once daily dosage compared to 3–4 times daily dosage of ipratropium. Tiotropium has other effects in body as side effects. Hence, we planned this study to see the changes in lung function and health status of patient on tiotropium bromide and its other side effects.

Aim

This study aims to see changes in lung function and health status in patients of COPD on treatment with tiotropium.

Objectives

The objectives of the study were as follows:

- To compare the changes in pulmonary function of COPD patients on tiotropium before and after 3 months of follow-up period
- To compare health status of COPD patients before and after 3-month follow-up using SGRQ
- To document the side effects of tiotropium use if any.

MATERIALS AND METHODS

It was a prospective observational cohort study done in November 2017–April 2019 at chest clinic and medicine outpatient department (OPD) from the Department of Medicine of University College of Medical Sciences (UCMS) and GTB Hospital, Delhi.

A total of 65 patients with diagnosis of COPD presenting in chest clinic and medicine OPD and receiving inhaled tiotropium bromide drug were recruited for the study.

Inclusion Criteria

The following criteria were included in the study:

- All patients diagnosed with COPD in GOLD-2017 Stages B and C
- Patients between 40 and 70 years of age.

Exclusion Criteria

The following criteria were excluded from the study:

- Known cases of asthma, allergic rhinitis, and atopy
- Patients in Stage A and D GOLD-2017 guidelines
- Patients in acute exacerbation in the past 1 month
- Patients with symptomatic GERD
- Patients on oral corticosteroids.

Methodology

Ethical clearance was taken from the Institutional Ethics Committee and informed written consent was taken from each subject. This study was done on patients recruited from chest clinic and medicine OPD from the Department of Medicine, UCMS and GTB Hospital, patients between 40 and 70 years according to the GOLD COPD guidelines were selected for the study. During the study period, the following medications was prescribed: BDs: LAMA (tiotropium) in dose of 18 μg/dose per day, LABA (salmeterol) 50 μg/dose as twice a day, and LAMA (tiotropium) was given either alone or in combination with LABA (salmeterol). This was as per management guidelines by GOLD-17. Patients were also looked for clinical features suggestive of acute exacerbation of COPD (AECOPD), i.e., sustained worsening of symptoms from stable stage triggered by infections, pollutants that result in addition therapy to the regular medications in a COPD patient. AECOPD is classified as mild, moderate, and severe depending on clinical features and medications needed: Mild (treated with short-acting BDs [SABDs] only), moderate (treated with SABDs plus antibiotics and/or oral corticosteroids), and severe (patient requiring hospitalization or visits the emergency room). Severe exacerbations may also be associated with acute respiratory failure. Patients were managed with BDs (short acting), corticosteroids (i.v or oral), and antibiotics.

Simultaneously, patients was assessed for features suggestive of hospitalization such as sudden worsening of resting dyspnea, high respiratory rate, decreased oxygen saturation, acute respiratory failure, onset of new physical signs, cyanosis, peripheral edema, failure to response to initial medical management, and presence of serious comorbidities. After the onset of these events, patient was hospitalized and managed as per standard protocols.

Patients were followed for 3 months duration, and detailed history and clinical examination were done. Pulmonary function test (PFT) and intraocular pressure (IOP) were measured before prescribing tiotropium. Tiotropium was given as meter dose inhaler in dose of 18 μg per dose, through inhaled route which was available free of cost in our hospital supply. Proper standard inhaler technique was demonstrated to every patient.

PFT and IOP were again assessed at the end of the 1st month and 3 months of follow-up periods. SGRQ was used to assess QOL at baseline, 1st month, and end of the study. Data were obtained from all subjects by

- Details history and clinical examination
- PFT
- QOL patients were assessed using SGRQ
- Statistical analysis.

Detailed History and Clinical Examination

Patients having clinical features suggestive of COPD like history of smoking or smoke exposure, cough and expectoration were evaluated. The diagnosis was confirmed by spirometery.

RESULTS

Demographic Profile of the Study Population

Age distribution

The age of subjects ranged from 40 to 70 years. The mean standard deviation (SD) age of cases was 57.91 (7.89) years.

Age at diagnosis of COPD in the present study

The mean age of diagnosis of COPD patients in this study was 55.26 ± 7.28. The minimum and maximum age of diagnosis of patients was 39–70 years.

Gender distribution

There were 57 males and 8 females in the current study.

Body mass index (BMI)

Out of total 65 patients recruited in our study, BMI ranged from 14.15 to 29.51. Mean BMI of patients was 19.86 ± 3.40 kg/m^2.

Smoking status

Out of 65 COPD patients recruited in our study, 43 were either ever smoker category (current or past), whereas 22 patients belonged to never smoker category.

Clinical profile and history of patients

Out of total recruited patients in our study, 34 patients complained of dyspnea on exertion along with cough without sputum, 15 patients complained of dyspnea on exertion along with cough and sputum, 8 patients complained of dyspnea on exertion only, 6 patients complained of only dry cough, and 2 patients complained of dyspnea while performing day-to-day activities.

On clinical examination (chest auscultation) of patients, 25 patients had decreased air entry bilaterally, 21 patients had wheeze, 16 patients had rhonchi, and 3 patients had crackles along with wheeze.

Laboratory Profile of the Study Population

FEV_1

FEV_1 was measured at 0, 1, and 3 months of study.

In Table 1, it was observed that there is a significant change in mean FEV_1 at the end of 1st month of follow-up period and at the end of the study compared to FEV_1 at baseline.

FVC

FVC was measured at 0, 1, and 3 months of the study.

In Table 2, there was no significant change in FVC after 1 month of start of drug and no significant statistically change observed at the end of 3 months of study compared to the 1st month.

There is a significant change in mean FVC at the end of follow-up study compared to baseline.

Ratio of FEV_1/FVC

FEV1/FVC was calculated at baseline, at the end of 1 month, and end of 3 months of study. In Table 3, there was a significant change in FEV1/FVC after 1 month of start of drug and no significant statistical change observed at the end of 3 months of study compared to the 1st month. There is a significant change in mean FEV1/FVC at the end of follow-up study compared to start of study.

Overall, comparison of lung function changes at baseline, 1 month, and 3 months by repeated measure ANOVA [Table 4].

SGRQ Score

SGRQ score of patient was calculated at 0, 1, and 3 months of study. In Table 5, there was no significant change in SGRQ score after 1 month of start of drug, but significant statistical change observed at end of 3 months of study compared to the 1st month. There is a significant change in mean SGRQ score at the end of follow-up study compared to start of study [Figure 1 and Table 5].

Side Effects Observed with Use of Tiotropium

Out of 65 patients, we followed up in our study some patients complained of side effects such as dry mouth, pharyngitis, URTI, and constipation with use of tiotropium.

Table 1: Comparison of FEV_1

Group-wise comparisons		FEV_1 (as % of predicted value) Mean±SD	Observed difference between two time points	*P*-value*
Baseline value versus 1 month	Baseline value	32.37±9.95	3.45	<0.05#
	Value at 1 month	35.82±10.69		
1 month versus 3 months	Value at 1 month	35.82±10.69	1.55	>0.05
	Value at 3 month	37.37±12.53		
Baseline value versus 3 months	Baseline value	32.37±9.95	5	<0.05#
	Value at 3 month	37.37±12.53		

*Repeated measures ANOVA followed by *post hoc* Tukey's test. #Statistically significant. FEV_1: Forced expiratory volume, SD: Standard deviation

Table 2: Comparison of FVC

Group-wise comparisons		FVC_1 (as % of predicted value) Mean±SD	Observed difference between two time points	*P*-value
Baseline value versus 1 month	Baseline value	56.06±16.60	1.89	>0.05
	Value at 1 month	57.95±16.35		
1 month versus 3 months	Value at 1 month	57.95±16.35	2.56	>0.05
	Value at 3 month	60.51±16.83		
Baseline value versus 3 months	Baseline value	56.06±16.60	4.45	<0.05#
	Value at 3 month	60.51±16.83		

*Repeated measures ANOVA followed by *post hoc* Tukey's test. #Statistically significant. FVC: Forced vital capacity, SD: Standard deviation

Table 3: Comparison of FEV_1/FVC

Group-wise comparison		FEV_1/FVC ratio Mean value±SD	Observed difference between two time points	*P*-value*
Baseline value versus 1 month	Baseline value	58.18±8.09	3.68	<0.05
	Value at 1 month	61.86±9.46		
1 month versus 3 months	Value at 1 month	61.86±9.46	0.03	>0.05
	Value at 3 month	61.89±10.95		
Baseline value versus 3 months	Baseline value	58.18±8.09	3.71	<0.05#
	Value at 3 month	61.89±10.95		

*Repeated measures ANOVA followed by *post hoc* Tukey's test. #Statistically significant, SD: Standard deviation, FVC: Forced vital capacity, FEV_1: Forced expiratory volume

Table 4: Summary of spirometric findings

Pulmonary function	Baseline	1 month	3 months	*P*-value
FEV_1	32.37±9.95	35.82±10.69	37.37±12.53	<0.001
FVC	56.06±16.60	57.95±16.35	60.51±16.86	<0.001
FEV_1/FVC	58.18±8.09	61.86±9.46	61.89±10.95	<0.001

FVC: Forced vital capacity, FEV_1: Forced expiratory volume

Table 5: Comparison of SGRQ score

Group-wise comparison	SGRQ score Mean±SD	Observed difference between two time points	*P*-value*
Baseline value versus 1 month	52.95±5.29 51.55±3.88	1.4	>0.05
1 month versus 3 months	51.55±3.88 48.88±4.27	2.67	<0.05#
Baseline value versus 3 months	52.95±5.29 48.88±4.27	4.07	<0.05#

*Repeated measures ANOVA followed by *post hoc* Tukey's test. #Statistically significant, SD: Standard deviation, SGRQ: Saint George's Respiratory Questionnaire

DISCUSSION

The aim of our study was to see the changes in lung function and health status in patients of COPD on treatment with tiotropium bromide for a period of 3 months. Health status was assessed using SGRQ which was designed to measure impact on overall heath, daily life, and perceived well-being in patients with the disease.

A total of 65 patients were recruited for study which included 57 (87.7%) males and 8 (12.3%) females. The age of subjects ranged from 40 to 70 years. The mean (SD) age of patients recruited was 57.91 (7.89) years. The age at diagnosis of subjects ranged from 39 to 70 years. The mean age at diagnosis of COPD patients in our study was 55.26 ± 7.28 years. BMI of patients ranged from 14.15 to 29.51 kg/ m^2. The mean BMI of patients was 19.86 ± 3.40 kg/ m^2. Out of 65 COPD patients recruited in our study, 43 (66.2%)

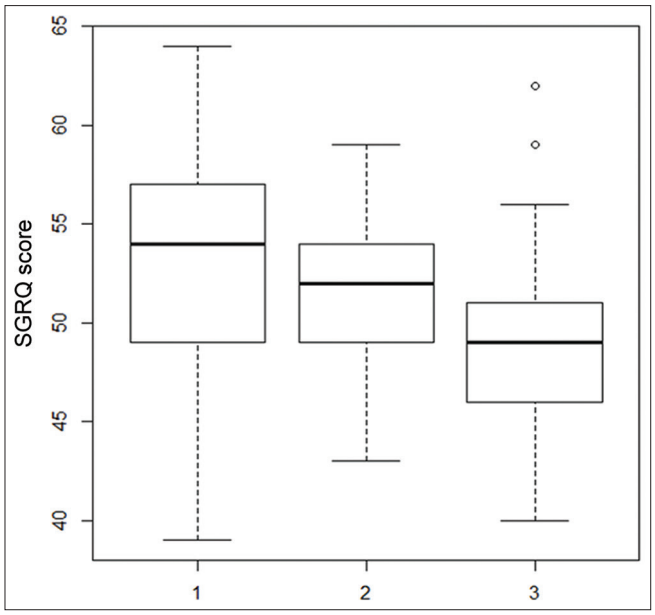

Figure 1: Boxplot depicting Saint George's Respiratory Questionnaire score at baseline, 1 month, and 3 months of follow-up

belonged to smoker category, whereas 22 (33.8%) patients belonged to non-smoker category. Among the pulmonary function tests measured, there is a significant change in mean FEV_1 at the end of follow-up period compared to FEV_1 at baseline (32.37 ± 9.95% of predicted value), but there was no statistically significant change observed in FEV_1 at the 3rd month (37.37 ± 12.53% of predicted value) compare to the 1st month (35.82 ± 10.69% of predicted value). There was no significant change in FVC after 1 month (57.95 ± 16.35% of predicted value) of start of drug and no significant statistical change observed at the end of 3 months (60.51 ± 16.83% of predicted value) of the study compared to the 1st month. There is a significant change in mean FVC at the end of follow-up study compared to baseline (56.06 ± 16.60% of predicted value). There is a significant change in mean FEV_1/FVC ratio at the end of 1 month (61.86 ± 9.46%) and follow-up period of 3 months (61.89 ± 10.95%) compared to the baseline (58.18 ± 8.09%). However, the change in mean FEV_1/FVC ratio of the 1st and 3rd months was not significant statistically. There was no significant change in mean SGRQ score after 1 month (51.55 ± 3.88) of start of drug, but significant statistical change observed at the end of 3rd month (48.88 ± 4.27) of study compared to the 1st month that implies SGRQ score decreased and patients health status and QOL improved. There is a significant change in mean SGRQ score at the end of follow-up study compared to baseline (52.95 ± 5.29). In our study, 16 patients (24.6%) complained of dry mouth, 7 (10.7%) complained of pharyngitis or throat irritation, and 3 (4.6%) patients complained of constipation.

According to the GOLD 2019 guidelines in the past, most studies have reported greater prevalence of COPD among males compared to females, but more recent data from developed countries have reported equal prevalence among males and females. Some studies have also suggested females more susceptible to COPD compared to males. According to the Global Adult Tobacco Survey in India, 19% of males and 2% of females are tobacco smokers so in our Indian setup as smoking is more common in males compared to females; hence, most of the patients were males in our study.[3] COPD is a disease of elderly. Majority of patients are diagnosed after 40 years of age.

Age is often listed as a risk factor for COPD patients. It is unclear if healthy aging as such leads to COPD or if age reflects the sum of cumulative effect of exposure to risk factors throughout life.[4] In a study a 4-year trial of tiotropium in COPD patient by Donald *et al.*, the mean age (years) in tiotropium group was 64.5 ± 8.4 and placebo group was 64.5 ± 8.5, respectively. The percentage of male sex was 75.4% in tiotropium group and 73.9% in placebo group. In another Indian study, the clinicophysiological effect of inhaled tiotropium bromide in severe COPD by Prakash *et al.*[5] mean age (years) was 58.81 ± 9.32, which was very similar to mean age groups of patients recruited in our study.

Nutritional depletion and weight loss are features of COPD patients. There are many studies documented the prognostic value of low body weight in patients of COPD. Patients with low BMI are at increased risk for developing severity of COPD. Low BMI is also an independent negative determinant of survival in patients with COPD. In study on BMI of patients with COPD by Wu *et al*, it was observed that BMI was moderately correlated with pulmonary function positively and exacerbations negatively. BMI might be useful indicator to predict the prognosis of COPD patients and long-term management. In a study of the effect of tiotropium on lung function decline in early stage of COPD patients, Lee *et al.*, mean BMI (kg/m^2) of tiotropium group patients was 22.7 ± 3.43 and control group was 23.0 ± 2.96. Hence, in our study, the observed mean BMI was significantly low compared to other studies.[6]

Smoking is the most studied risk factor in COPD patients, but <50% develop COPD during life time. In a 25-year follow-up study of the general population by Løkke *et al.*, the absolute risk of developing COPD among continuous smokers is at least 25%. The 25-year incidence of moderate and severe COPD was 20.7% and 3.6%, respectively, with no apparent difference in men and women.[7] Smoking cessation, especially early in follow-up, decreases the risk of developing COPD substantially compared with continuous smoking. About 92% of the COPD deaths occurred in subjects who were current smokers at the beginning of follow-up period. In another study, the prevalence and incidence of COPD in smokers and non-smokers by

Terzikhan *et al.*, 21.7% of the study participants were current smokers, 41.7% were former smokers, and 34.2% were never smokers.[8] In smokers, 17.8% had COPD, whereas in non-smokers, the prevalence of COPD was 6.4%. In our study, 43 patients were smokers accounting to 66.2% and 22 were non-smokers accounting 33.8% of the patients. Hence, in our study, majority of patients were smokers.

Pulmonary functions of patients were compared after prescribing tiotropium bromide. They were followed up for 3 months. Baseline pulmonary functions were compared with changes at 1 month and follow-up period of 3 months. Various parameters measured were FEV_1, FVC, and FEV_1/ FVC ratio. A retrospective analysis of the understanding potential long-term impacts on function with tiotropium (UPLIFT) trial data was performed by Halpin *et al.* It graded patients by the 2013 GOLD severity groups.[9] Mean FEV_1 was higher with tiotropium than usual care (control) in all GOLD groups at all post-baseline time points during treatment. In a study called the effect of tiotropium on lung function decline in early stage of COPD patients by Lee *et al.*, out of 587 patients enrolled in the study, 257 took tiotropium following propensity score matching, 404 patients were included in the analysis.[6]

The mean annual rate of post-BD FEV_1 decline was 23.9 (tiotropium) and 22.5 (control) ml/year ($P = 0.31$), respectively. Mean annual rate of post-BD FVC decline was 55.1 (tiotropium) and 43.5 (control) ml/year ($P = 0.33$), corresponding pre-BD values were 37.1 and 33.3 mL/ year ($P = 0.13$).

Therefore, tiotropium does not reduce the rate lung function decline in COPD patients with $FEV_1 \geq 70\%$. In our study, there is a significant increase in mean FEV_1 at 1 month follow-up and at the end of study period of 3 months compared to baseline with the use of tiotropium, although change in mean FEV_1 was not statistically significant between the 1st and 3rd month. In our study also, similar findings were seen as other studies.

In our study, there was statistically significant difference in mean FVC at the end of study period compared to baseline, but there was no significant change observed in FVC at the end of 1st month compared to baseline. Similarly, no significant change was observed in mean FVC of the 1st and 3rd month. In another study Prakash *et al.* compared the effect of tiotropium bromide in severe COPD patients on pulmonary function parameters (PFT), functional exercise capacity (6MWD), exertional and overall dyspnea (visual analogue scale and MRC dyspnea scale), symptom score, drug score, health related QOL (HR QOL) (CRDQ).[5] Thirty-two patients (16 patients each in tiotropium) of severe COPD were followed up for 12 weeks (6 weeks of run in period and 6 weeks of study period). In tiotropium group, there was more improvement in FVC, 6 min walk distance, MRC, CDRQ, symptom score, and drug score. In a study by Adams *et al.*, tiotropium treated patients had significant improvement in FEV_1 and FVC.[10] Significant changes in mean ratio of FEV_1/FVC were observed in our study at the follow-up period of 1 month compared to baseline, but no significant statistical change observed at the end of 3 months of study compared to the 1st month. Hence, overall, there is a significant change in mean FEV_1/ FVC at the end of follow-up study compared to start of study. Health status instruments used in COPD patients are CAT, clinical COPD questionnaire, and SGRQ. In our study, we have used SGRQ score for health status an indicator of QOL. SGRQ has become one of the most widely used instruments for assessing HR QOL in respiratory patients and has been translated into several languages. It is a disease-specific instrument designed to measure impact on overall health, daily life, and perceived well-being in patients with obstructive pulmonary disease. In our study, there was no significant change in SGRQ score after 1 month of start of study, but significant statistical difference observed at the end of 3 months of study compared to 1st month. There is a significant change in mean SGRQ score at the end of follow-up period compared to baseline. In a 4-year trial of tiotropium in COPD by Tashkin *et al.* concluded that the patients in COPD, therapy with tiotropium was associated with improvements in lung function, QOL, and exacerbations during a 4-year period but did not significantly reduce the rate of decline in FEV_1.[11] The mean absolute total score on the SGRQ was improved (lower) in the tiotropium group, as compared with the placebo group, at each point throughout the 4-year period (ranging from 2.3 to 3.3 units, $P < 0.001$). At 4 years and 30 days, tiotropium was associated with a reduction in the risks of exacerbations, related hospitalizations, and respiratory failures. In a study by Vincken *et al.*, tiotropium is effective in improving dyspnea, exacerbations, HR QOL, and lung functions in patients of COPD.[12] The data support use of tiotropium once daily as first-line maintenance treatment in patients with COPD.

In a study role of ipratropium bromide and tiotropium bromide in COPD patients by Khan *et al.*, 57 patients were recruited, tiotropium showed significant reduction in exacerbation and improvement in QOL, health status, and dyspnea compared to ipratropium.[13] In another study, role of tiotropium in the treatment of COPD by Rice *et al.*, tiotropium slowed the rate of decline in FEV_1, reduces lung hyperinflation with associated improvement in exercise capacity, subjective dyspnea, and HRQL scores.[14] It also reduced exacerbations and hospitalization but did not affect overall mortality. Similar results were also seen in our study. The aim of our study

was to see the changes in lung function and health status in patients of COPD on treatment with tiotropium bromide for a period of 3 months. Health status was assessed using SGRQ which was designed to measure impact on overall heath, daily life, and perceived well-being in patients with the disease. We also measured changes in IOP of patient's pre- and post-observation period using Goldmann applanation tonometer and documented the presence of any side effect associated with the use of drug. The most commonly reported adverse effect observed was dryness of mouth, with a total of 10 studies reporting incidence. The cumulative incidence was 7.4% in tiotropium patients, 3.9% in ipratropium patients, and 1.6% in salmeterol patients. The incidence of dry mouth was significantly increased in eight studies which compared tiotropium with placebo. Studies have consistently reported that tiotropium is safe and well tolerated in clinical trials that ranged from 3 months to 48 months. Other adverse events associated with tiotropium include constipation, tachycardia, urinary retention, raised IOP, and angina rarely. In a large long-term clinical trial in COPD patients, tiotropium added to other standard therapies had no effect on cardiovascular risk.

In our study, 16 patients (24.6%) complained of dry mouth, 7 (10.7%) complained of pharyngitis or throat irritation, and 3 (4.6%) patients complained of constipation. Hence, in our study, the most common side effect observed was dry mouth which was also seen in other studies.

Strength of our study is a prospective observational cohort study so it was better than other study which were cross sectional, so patients were recruited and followed up on a regular basis after being diagnosed with COPD or were diagnosed earlier on the basis of history and PFT. Adding SGRQ as a tool for HRQOL is another strength of our study as it provides descriptive heath status of COPD patient.

Limitations of our study, we could not perform randomized control trials, used in UPLIFT study of COPD patients which would have been a better study design due to limited resources, investigators, and time period. We followed patients for a period of 3 months which is a limited time period to see changes in lung function, as certain studies where changes lung function started to occur where of longer period.

CONCLUSION

Thus, we conclude that tiotropium bromide improves lung functions, overall health status, and QOL of COPD patients.

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Original Article

Role of Emergency Response Codes in Handling Hospital Emergencies – An Experience of Protocol Designing, Development, and Implementation in a Large Multispecialty Tertiary Care Teaching Hospital, Mysuru, India

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Abstract

Introduction: "An emergency is defined as any incident, caused by humans or a natural event that requires an effective, responsive action to protect life or property." Therefore, the response to an emergency must be quickly coordinated and well planned. Emergency codes in the hospitals are used worldwide to alert the hospital employees for handling various emergency situations in hospitals. The use of codes is intended to convey essential information quickly with a minimum of misunderstanding to the hospital staff, while preventing stress or panic among visitors of the hospital.

Objectives: The objectives of the study were to prepare standard operating procedures (SOPs) using codes for various emergency situations that arise and to implement the same in the hospital and to make clear all the employees about various codes implemented in the hospital and to provide guidance for the same.

Methodology: (a) Various emergency situations that can occur in the hospital were identified. (b) SOPs were developed for each emergency situation using codes. (c) A training program was planned for all the staff members about their roles and responsibilities for effective functioning of hospital emergency response codes before implementation.

Conclusion: Emergency response codes are color-coded indicators used in hospitals to alert all staff members for emergency issues that may arise. Hence, clear communication is a key element to ensure a quick response to protect patients, visitors, and staff and hospital property.

Key words: Communication, Emergencies, Hospital, Response codes

INTRODUCTION

Hospitals and health-care organizations work to provide a safe, functional, and supportive facility for patients, families, staff, and visitors, but an emergency can arise at any time due to any cause. According to the World Health Organization (WHO), an emergency is a sudden and usually unforeseen event that calls for immediate measures to mitigate impact. An emergency is also defined as any incident, caused by humans or a natural event that requires an effective, responsive action to protect life or property.[1] Therefore, the response to an emergency must be quickly coordinated or communicated and well planned. Standardized emergency codes are required to identify a risk or emergency situation within any facility. An emergency code is a notification of an event that requires an immediate action. Emergency codes

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in the hospitals are used worldwide to alert the hospital employees for handling various emergency situations in hospitals. Hospitals are the most common institutions that use color codes to designate emergencies because hospitals/health-care organizations are house to patients and are vulnerable to mishaps such as fire, earthquake, floods, violence, and epidemic outbreaks.[2] Communication is essential during an emergency situations to convey data and information which supports situational awareness to hospitals and response personnel. The use of codes is intended to convey essential information/communication quickly with a minimum of misunderstanding to the hospital staff, while preventing stress or panic among visitors of the hospital.[3] Emergency response codes allow trained hospital personnel to respond quickly and appropriately to various emergency events. Emergency response codes are necessary to improve the response capability of hospital employees. Hence, hospital/hospital employees must be prepared to deal efficiently and effectively with different emergencies that may arise. Hospital employees, including doctors, should undergo extensive training to respond to each of these events, allowing them to save lives. One of the primary benefits of a code system is that trained hospital employees know to respond to any given emergency without alarming those being treated and hospital visitors. Panicked bystanders can hinder the response efforts of emergency responders. Emergency codes are extremely important for the safety of everyone inside a hospital. They allow doctors and administrative employees to respond quickly and effectively to save lives in emergency situations. As the WHO puts it in its guide to mass casualty management systems, "The most commonly cited problem in disaster management is invariably communications breakdown, with emergency activities and decision-making being seriously affected by vital information being lost or delayed."[4] Thankfully, technology today offers several options that can be deployed to ensure that there is no communications breakdown. From traditional methods of communication such as telephones and on-site alarms to more modern options such as short message service, video conferencing, and virtual receptionists, everyone who needs to be kept informed now can be, and all through their medium of choice. Hence, this project has been taken up for designing, developing, and implementing emergency response codes, policies, and standard operating procedures (SOPs) in this Tertiary care Teaching Hospital in Mysuru.

Objectives

The objectives of the study were as follows:

1. To prepare SOP and policies using codes for various emergency situations that may arise and to implement the same in the hospital.
2. To make clear all the employees about various emergency response codes implemented in the hospital and to train and provide guidance for the same.
3. To make policies available with regard to various emergency response codes which can be followed in a hospital.
4. To explore the experiences of designing and implementation of emergency response codes.

METHODOLOGY FOR IMPLEMENTATION

1. Study of currently available national and international literature on the subject.
2. Various emergency situations that can occur in the hospital were identified.
3. SOP and policies were developed for each emergency situation using emergency response codes.
4. Planning: Steps that are currently required to complete the process like how is it done? Why is it done that way? How will an SOP improve the process? How the performance can be measured?
5. First draft: Emergency events were coded by a color, a code name, and a number. For each code, SOP is structured under following key components: Purpose, policy, team members, roles and responsibilities, process flow/procedure, documentation, training, and education along with the detailed list of the steps to be performed in the order that they can be done. This list is now a draft of the procedure.
6. Review: Inputs from all staff/employees who will perform the procedure have been taken by giving them the first draft and revision of the procedures has been done as necessary.
7. Testing: Procedures for each code has been tested by doing each step exactly written in the SOPs. Revised as necessary.
8. Posting: After preparing a final draft, approval has been taken from the hospital top management, and the same has been uploaded into the quality module of the hospital information system so that it is available to all the stakeholders.
9. Training: Regular training sessions have been planned and organized for all the staff members about their roles and responsibilities for effective functioning of hospital emergency response codes before implementation. Presentations were scheduled with various groups within the hospital such as physicians, nursing staffs, technicians, and security staffs to update on new emergency response codes. Information about new emergency response codes to be implemented was shared at various levels of staff meetings, safety meetings, and all new hospital personnel orientation meetings.
10. After formulating policies and procedures, training ,and education, the same were implemented.

11. A signage poster containing all the details of the emergency response codes and contact information is displayed at all the counters nearest to the telephone. All the employees are periodically trained for the same [Figure 1].
12. Informal oral surveys to determine staff knowledge using management rounding or other existing feedback mechanisms were also conducted.

RESULTS AND DISCUSSION

SOPs are detailed written instructions to achieve uniformity of the performance of a specific function. SOP is a complete reference document or operations manual that describes the purpose of a preferred method of performing a single function or a number of interrelated functions in a uniform manner and provides information about the duration of the operation, the authorities of those involved, and other relevant details.[6] By definition, An SOP or "Standard Operating Procedure" tells in writing about WHAT should be done, WHEN it should be done, WHERE it should be done, and WHO should do it.

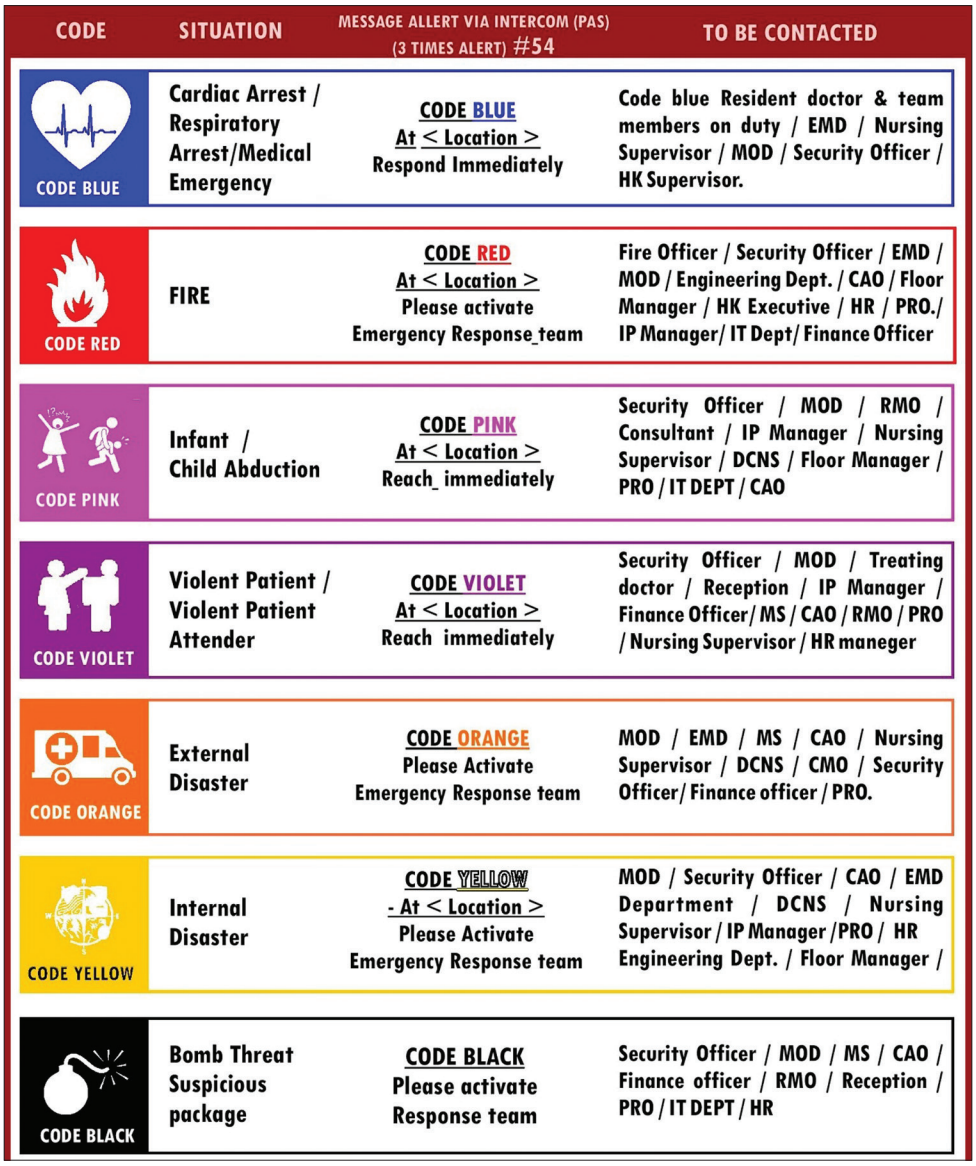
Emergency Response Plan

It can be defined as a set of written procedures that guide emergency actions, facilitate recovery efforts, and reduce the impact of an emergency event.

Emergency Response Codes Policies and Procedures for a Large Multispeciality Tertiary Care Teaching Hospital

Purpose

The purpose of the study was to provide an appropriate response to various emergency situations including hazards



CODE	SITUATION	MESSAGE ALLERT VIA INTERCOM (PAS) (3 TIMES ALERT) #54	TO BE CONTACTED
CODE BLUE	Cardiac Arrest / Respiratory Arrest/Medical Emergency	CODE BLUE At < Location > Respond Immediately	Code blue Resident doctor & team members on duty / EMD / Nursing Supervisor / MOD / Security Officer / HK Supervisor.
CODE RED	FIRE	CODE RED At < Location > Please activate Emergency Response team	Fire Officer / Security Officer / EMD / MOD / Engineering Dept. / CAO / Floor Manager / HK Executive / HR / PRO./ IP Manager/ IT Dept/ Finance Officer
CODE PINK	Infant / Child Abduction	CODE PINK At < Location > Reach_ immediately	Security Officer / MOD / RMO / Consultant / IP Manager / Nursing Supervisor / DCNS / Floor Manager / PRO / IT DEPT / CAO
CODE VIOLET	Violent Patient / Violent Patient Attender	CODE VIOLET At < Location > Reach immediately	Security Officer / MOD / Treating doctor / Reception / IP Manager / Finance Officer/ MS / CAO / RMO / PRO / Nursing Supervisor / HR maneger
CODE ORANGE	External Disaster	CODE ORANGE Please Activate Emergency Response team	MOD / EMD / MS / CAO / Nursing Supervisor / DCNS / CMO / Security Officer/ Finance officer / PRO.
CODE YELLOW	Internal Disaster	CODE YELLOW - At < Location > Please Activate Emergency Response team	MOD / Security Officer / CAO / EMD Department / DCNS / Nursing Supervisor / IP Manager /PRO / HR Engineering Dept. / Floor Manager /
CODE BLACK	Bomb Threat Suspicious package	CODE BLACK Please activate Response team	Security Officer / MOD / MS / CAO / Finance officer / RMO / Reception / PRO / IT DEPT / HR

Figure 1: Poster depicting emergency response codes

and events that may arise in the hospital and may potentially have a significant impact on the normal operations of the hospital.

Policy

All employees have a responsibility to respond quickly to a suspected or actual emergency situation.

Scope

Hospital wide.

Responsibility

All the hospital employees, staff, students, etc.

Emergency response color codes designated for this hospital under study

- Code Blue: Code blue is the most universally recognized emergency code. Code blue means that there is a medical emergency occurring within the hospital. Common reasons for activating a code blue include cardiac arrest like a heart attack or dangerous arrhythmia and respiratory arrest (when someone stops breathing)
- Code Red: A code red denotes the presence or reasonable presumption that fire is occurring in a facility. For instance, a staff member who sees or smells smoke may activate fire codes.
- Code Pink: A code pink denotes a missing child/infant in this hospital.
- Code Violet:A code violet denotes violent patient/patient attender in this hospital. The purpose of this code is to provide an appropriate response to situations involving an aggressive, hostile, combative, or potentially combative persons toward hospital employees.
- Code Orange: A code orange denotes external disaster or a mass causality incident in this hospital. This could mean anything from a major road traffic accident to a natural disaster or act of terrorism or even epidemic disease outbreak. In short, it's any event that results in a high number of casualties that could stretch existing resources to their breaking point. Announcing this code may involve calling in staff on short notice, sourcing additional equipment, repurposing existing equipment, and prioritizing patient treatment so that there is enough physical space available to deal with incoming casualties.
- Code Yellow: A code yellow denotes internal disaster in this hospital.
- Code Black: A code black denotes a bomb threat to the hospital. This may include the identification of an actual bomb within the facility. The purpose of announcing this code is to provide an appropriate response in the event of a bomb threat or physical threat and the discovery of a suspicious device or item.

When to activate an emergency response code

An emergency response code will be initiated when any of the above emergency situations will occur.

Who can activate emergency response code

Any individual, i.e., hospital staff of all categories may call a code.

How to activate emergency response code

1. Step one in organizing an emergency response is notification. An efficient notification service is required to ensure that the right resources are mobilized based on the type of incident.
2. Communication system in the hospital is adequate along with a good public address system, and in fact, each and every telephone in the hospital is connected to public address system so as to communicate to other hospital employees in case of any emergencies.
3. The individual calling the code must dial the designated number to call a code that is applicable to that particular emergency situations.
4. Give the exact location (i.e., block, floor, area, room no., etc.)

What happens when the emergency response code is announced

1. When a code is called, a pre-designated team of physicians, nurses, and other appropriate personnel respond swiftly and efficiently, based on their training.
2. When the emergency response code is announced, the message will be received by the team, who are expected to arrive at the scene and expected response time is 0–3 min.
3. When an emergency response code is called, all team members of that code should respond immediately.
4. The members of the emergency code team must ensure that area/scene is safe before proceeding with their response. This requires rapid assessment of the location and circumstances associated with the emergency response code call.
5. Emergency response code team members function collaboratively during the code with one person identified as code team leader.
6. The code incident report is filled by the team leader at the end of event and submits to the director/medical superintendent/chief administrative officer/committee (CPR committee/safety committee, etc.)

Documentation and reporting

1. Documentation containing information about the activation is reviewed and retained.
2. Reporting of the incident may be completed through an event report/incident report which is designed or any other reporting method.

3. Management will conduct a root cause analysis or similar review of the incident to identify areas for improvement and then implement those improvements

Training and education

1. Training and education ensure that all staffs are aware of potential emergency situations and/or security hazards, how to respond or react and how to protect themselves and their coworkers through established policies and procedures.
2. Specific training should be provided to all the team members as to their specific roles and responsibilities during a variety of scenarios.
3. Training for staff includes what to do when they become a hostage or victim.

CHALLENGES OF IMPLEMENTATION

One of the primary problems of the hospital emergency response code system, in general, is a lack of national standardization. Although some emergency response codes, such as code blue and code red, are universal around the world, many of the other codes are not consistent. Each color can have various meanings across different states or countries. Some hospitals use numbered code systems rather than colors. Each hospital or hospital association is responsible for developing its own emergency codes. When these codes are developed independently by hospitals, there is a variability and overlap between hospitals and a great deal of variance, which can be a source of confusion at critical moments to health-care personnel. This variability may also mitigate the effectiveness and speed of response in critical events. Besides the lack of uniformity in emergency codes, critical events are often defined differently between hospitals, for example, "Fire" is included in "Internal Disaster" in some hospitals, whereas in others, each has its own code. Health care workers move frequently between hospitals and may work at more than one hospital,[5] resulting in an inability to remember and respond to the right code at the right time. Some countries, such as England and Canada, use a nationally standardized set of emergency hospital codes. This means that every hospital uses the same communication terminology to communicate during an emergency situation. However, this standardization has not yet been implemented in India, although it has previously been recommended.

CONCLUSION

Emergency response codes are color-coded indicators used in health-care facilities to alert all relevant hospital staff members for potential issues arising in the hospital. An emergency can happen anytime. Clear communication is a key element to ensure a quick response to protect patients, visitors, and staff and hospital property. Communications technology clearly has a huge role to play in the event of various hospital emergencies and should be an integral part of any emergency handling plan. If you cannot communicate effectively, then your chances of adequately responding to the situation are greatly reduced. The most commonly cited problem in emergency situation management is invariably communications breakdown, with emergency activities and decision-making being seriously affected by vital information being lost or delayed. Hence, the right technology, if implemented correctly, can make a huge difference in emergency situations. The goal is for hospitals to phase in the implementation of the recommended codes so that the required materials and training can be developed and offered at a time best suited for hospital employees. Emergency response codes help hospital personnel understand how to effectively manage various emergency situations. Hospital emergency management is a continuous process requiring the seamless integration of planning and response efforts with local and national programs. The above layout can be useful to assist hospital administrators and emergency managers in responding effectively to the most likely emergency scenarios as well as while planning to implement emergency response plans in other small or large health-care organizations.

Recommendations

1. Continuous training programs should be organized for the hospital employees through regular classes or mock drills.
2. Periodic Corrective Action Preventive Action analysis of the documented events.
3. Periodically reviewing and revising the protocols as per need.
4. Continuous monitoring of the various incidents and staff handling skills because monitoring all aspects of the program provides valuable data to improve the program and further reduce the risks in the hospital.
5. Training exercises can help in raising level of preparedness and ensure that everyone knows what their role should be if an emergency situation arises.
6. Testing: The importance of testing can never be understated. The systems and equipment may be top of the line, but they also need to be easy to work with and ready to go at a moment's notice. An emergency may occur at any time, so you need to be able to launch straight into your emergency procedures. This means that 100% uptime is not just a goal, but a requirement, and to ensure that this is the case, you should carry out regular testing. Your notification system, for example, should have built-in test procedures that enable you to check functionality without actually triggering an alert.

7. To evaluate the adherence to the implemented protocols, the actual training and competence of hospital staff in the use of emergency response codes by conducting surprise mock drills.
8. To evaluate the success of implementation efforts and institutional compliance, a much larger follow-up study to be conducted.
9. It is recommended that the higher authorities and regulatory bodies should bring together local and national experts in the field to develop standard emergency response codes for hospitals in India.
10. To assess the effect of emergency response codes on issues such as the satisfaction of hospital staff and the prevention of in-house complications.

Strengths

1. Our study has several strengths, such as we had developed the policies and procedures in a systematic way, and is validated by the experienced experts in the field.
2. The policies and procedures formulation included individuals who are directly or indirectly related to functioning of those emergency response codes.

Limitations of these Policies

1. The principles and recommendations outlined in these policies are specially designed for this large multispecialty tertiary care teaching hospital only. Hence, the principles and recommendations included in these policies may be used by other hospitals at any level of emergency preparedness only after suitable modifications as per their organizational needs and resources available.
2. Emergency response codes and its activation dial number will vary from one hospital to another. Always follow your organizational policy and procedures for emergency response code activation.
3. Means of announcing these emergency response codes also differ from hospital to hospital depending on the resources available.
4. Depending on each hospital size and level of care, code designations may vary and team composition for various codes is not fixed and may vary from hospital to hospital.

AUTHORS' CONTRIBUTIONS

Dr. Sathish Raju N conceived the concept, developed the structure, and wrote the first draft of the manuscript. Dr. M. D. Ravi made critical revisions and contributed to the writing of the manuscript and approved the final version. Dr. Jayati Bahuguna participated in the design of the study. Dr. (Col) M Dayananda participated in the design of the study and revised the manuscript. All authors read, reviewed, and approved of the final manuscript.

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Original Article

Patterns of Mobile Phone use and Self-reported Health Problems among Adults Visiting a Private Dental Institute

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Abstract

Background: Increased use of mobile phones has led to increase in the prevalence of health problems. Hence, the present study was undertaken to assess the pattern of the use of mobile phone and its association with self-reported health problems among adults.

Materials and Methods: A total of 1520 participants visiting a private dental institute in Dhule, Maharashtra, were interviewed using a pre-tested, structured questionnaire. The questionnaire included variables such as socio-demographic details, use of mobile phone use and its pattern, selected health problems, perceived benefits, or threats of the use of mobile phone. Data obtained were analyzed using SPSS software.

Results: The majority of the participants (87.9%) were using mobile phone for up to 3 h daily. The majority of the participants (98.3%) reported using mobile phone for calling facility followed by internet or social networking purpose (67.2%). Health symptoms such as headache, stressful eyes, tiredness, and painful fingers showed an increased prevalence with increase in the daily usage of mobile phones; whereas hypertension was inversely related to the use of mobile phone. Most of the participants agreed that they start feeling stressed or anxious without their mobile phones.

Conclusion: Selected health problems showed a positive association with increasing mobile phone use. People should be made aware of harmful effects caused due to over-indulgence in the use of mobile phone and should restrict the use of focus only on mobile positive phones to the minimum possible.

Key words: Cell phone, Health, Health promotion, Risk assessment

INTRODUCTION

Mobile phone, a device which was once looked on as a luxury of the classes, has now become a necessity for the classes as well as the masses. Starting from its inception in 1973, mobile phone has undergone tremendous changes.[1] These incredible portable technology boxes have become an integral part of interpersonal communication. From the upsurge of SMS to anywhere, anytime internet connectivity to mobile photography, mobile phones have been the facilitator for cultural and technological changes over the past few decades. A drastic increase in number of mobile phone users has been seen in past decade with more than 7 billion subscribers in the world.[2] Mobile has been primarily used for accessing internet in addition to the calling services. Mobile-broadband subscriptions have grown more than 20% annually in the past 5 years and were expected to reach 4.3 billion globally by the end of 2017.[3]

India, known as the second largest mobile phone-using nation, has the fastest growing telecom network in the world,

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accounting for more than 1.1 billion mobile phone users till April 2017. Mobile phones usage accounts for almost 98% of all telecommunication users and more than 45% of all wireless phone users belong to the rural sector.[4] Indian is set to become the world's largest mobile phone using nation in near future. Mobile phones have become an essential part and have made communication a whole lot easier and convenient; but as they say "every coin has two sides," this technology too has its downsides. Mobile phone usage is associated with few ill-effects on health.[5] These effects depend on many factors such as number of calls per day, length of each call, amount of usage per day, and place where mobile phone is kept. In response to this concern, the World Health Organization started the International Electromagnetic Fields Project in 1996 to evaluate the scientific evidence of potential negative health effects from electromagnetic fields.[6] These health problems or symptoms are termed as electromagnetic hypersensitivity.[7] The symptoms most commonly experienced include dermatological symptoms (redness, tingling, and burning sensations) as well as neurasthenic and vegetative symptoms (fatigue, tiredness, concentration difficulties, dizziness, nausea, heart palpitations, and digestive disturbances).[7]

In spite of the negative impact of mobile phones on health, very few studies have been conducted throughout India in this context.[8-11] Mobile phone use and addiction reported were high among the previously conducted studies. Furthermore, these studies reported positive association of mobile phone use with increased risk of health problems such as headache, earache, anxiety, and eye pain. There is a growing necessity to assess the usage pattern of mobile phones, their impact on health and how the technology can be utilized for the betterment of health. Hence, the present study was conducted to assess the usage of mobile phones, their self-perceived health effects and how they can be utilized for improvement in health.

MATERIALS AND METHODS

Study Design, Study Setting, and Study Participants

The present cross-sectional survey was conducted on a total of 1520 participants visiting a private dental institute in Dhule, Maharashtra, from August 2017 to November 2017. Ethical clearance was obtained from the Institutional Review Board. Only those people who were using a mobile phone were included in the study and non-users were excluded from the study. Informed consent was obtained from the participants after explaining them the purpose of study.

Data Sources/Measurements

All the participants were interviewed using a pre-tested, structured questionnaire. Face validity of the questionnaire was assessed by five expert consensuses. Experts evaluated each item for its wording and grammar, understandability, and relatedness and also for any suggestion. The questionnaires were collected and suggestions were considered. Then, this questionnaire was administered to ten subjects using mobile phones on two different occasions and their responses were analyzed for test-retest reliability. Value of kappa statistics was 0.84. Final version of questionnaire consisted of section on demographic features of participants, section on details of mobile phone use, section on self-reported health problems and section on addiction toward mobile phone. Selected health problems included headache, ear pain, stressful eyes, neck pain, restlessness, sleep disturbances, painful fingers, morning tiredness, and dizziness. Hypertension was considered as blood pressure of 130 mm or above.[12] All the investigators were provided with the training to minimize inter-observer variations.

Statistical Analysis

Data were collected, compiled, and analyzed using SPSS version 17. Results were presented in frequency and percentage. The differences in the prevalence of self-reported health problems and hypertension according to daily usage of mobile (no. of hours of use) were compared using Chi-square test. Level of significance was kept at $P \leq 0.05$.

RESULTS

Table 1 shows the distribution of participants according to age and gender. The majority of the study participants belonged to the age group of 18–50 years. Table 2 presents the detailed information about mobile phone usage among study participants. More than half of study participants (54.5%) reported using mobile phone for <1 h in a day whereas 33.4% of the participants reported using mobile phone for 1–3 h daily and 6.8% of the participants reported using mobile daily for more than 6 h. When asked about the place where mobile phone is kept while sleeping, 45.5% of the participants keep mobile away from the bed while sleeping whereas 29.5% keep mobile phone next to bed while sleeping and 12.5% reported that they keep mobile phone under the pillow while sleeping. Almost all the participants (98.3%) reported using mobile phone for calling purpose. Other reasons for using mobile phones were text messaging (42.7%), internet (67.2%), playing games (40%), for alarm (33.5%), and for listening to music (27.4%).

Table 3 shows the prevalence of self-reported health problems associated with mobile phone use. Health problems reported increased with increase in the daily duration of mobile phone usage. Among the participants

Table 1: Demographic details of the participants

Demographic variable	Frequency (%)
Age (in years)	
18–30	451 (29.6)
31–40	404 (26.6)
41–50	418 (27.5)
51–60	121 (8.0)
61–70	82 (5.4)
>70	44 (2.9)
Gender	
Males	983 (64.7)
Females	537 (35.3)

Table 2: Details of usage of mobile phone among study participants

Details of mobile phone use	Frequency (%)
Daily usage of mobile phones (in hours)	
<1 h	828 (54.5)
1–3 h	508 (33.4)
3–6 h	80 (5.3)
>6 h	104 (6.8)
Place where mobile phone is kept while sleeping	
Under the pillow	184 (12.1)
Next to bed	448 (29.5)
Away from bed	692 (45.5)
Switched off	196 (12.9)
Purpose	
Calling	1494 (98.3)
Text messaging (SMS)	649 (42.7)
Internet/social networking	1021 (67.2)
Playing games	608 (40.0)
Alarm	509 (33.5)
Listening to music	417 (27.4)

using mobile phone for more than 6 h/day, 58% reported to suffer from headache, 35% reported suffering from ear pain, and 49% of participants reported stressful eyes after using mobile for more than 6 h daily. Increase in the daily usage of mobile phone was also associated with painful fingers ($P = 0.001$). Hypertension showed inverse relation with mobile phone use ($P = 0.001$). Sleep disturbances ($P = 0.043$) and morning tiredness ($P = 0.001$) also increased with increase in mobile phone usage.

Table 4 shows assessment of mobile phone addition. When asked about the addiction toward mobile phone, 55.52% of the participants reported that they felt stressed and alone without their mobile phone and 59.21% of the participants reported feeling anxious when their mobile phone did not show good signal strength. Only 10.52% participants reported using mobile phone while driving. When asked for positive benefits of mobile, 74.74% participants stated that excessive usage of mobile phones can have a negative impact on health and on academic or work performance; However, 60.26% also agreed on the fact that mobile phone can be used for safety reasons and as an educational tool for health promotion (62.89%).

Table 3: Prevalence of health problems a/c to duration of usage of mobile phones (in %)

Health problem	No of hours of daily usage				*P* value
	<1 h	1–3 h	3–6 h	>6 h	
Headache	25	46	46	58	0.001*
Ear pain	20	27	32	35	0.037*
Stressful eyes	30	32	46	49	0.012*
Neck pain	19	30	37	38	0.031*
Restlessness	10	12	14	15	0.479
Sleep disturbance	14	20	25	27	0.043*
Painful fingers	10	15	23	27	0.001*
Morning tiredness	13	20	19	20	0.001*
Dizziness	2	5	10	12	0.001*
Hypertension	7	4	2	2	0.018*

Chi-square test; * - indicates significant at $P \leq 0.05$

Table 4: Assessment of addiction and perception of benefits of mobile phones

Criteria	Question	Yes/agree (%)	No/disagree (%)
Assessment of addiction	Do you feel stressed and alone when you don't have your mobile phone? (Y/N)	55.52	44.48
	I cannot relax if my mobile phone does not have good signal strength. (A/D)	59.21	40.79
	Do you use mobile phone while driving? (Y/N)	10.52	89.48
Perception of positive/ negative impact	Excessive usage of mobile phones can have a negative impact on health and/or academic or work performance. (A/D)	74.74	25.26
	Mobile phone is required for safety and security reasons. (A/D)	60.26	39.74
	Mobile phones are used for educational purpose to promote health. (A/D)	62.89	37.11

DISCUSSION

Mobile phones are undeniably the most effective means of communication of the present times. However, this "smart mobile phone revolution" of recent times is not full-proof; it too comes along with its flaws, majority of which are health-related. Hence, the pattern of the use of mobile phones and its associated effect on health needs to be studied. The results show that excessive use of mobile phone leads to addiction and has been associated with risk of developing various health problems.

According to the present study, daily usage of mobile phone for majority of the participants was up to 3 h; whereas small proportion of participants reported using mobile for more than 6 h/day. A study done previously reported usage in terms of more than 4 h (60.5%) and

<4 h (39.5%).[8] The previous studies have evaluated use of mobile phone in terms of daily average talking time, number of years of use, users versus non-users, etc.[9,10,13,14] Out of 1520 participants, 12.1% of the participants reported that they keep mobile phone under the pillow and 29.5% keep mobile phone next to bed while sleeping. These results are similar to a study conducted where 80% of the participants reported keeping their mobile with them while sleeping.[8] Experts recommend that the cell phones should be kept at least three feet away from the body during sleep. This is because the radiation that the mobile phones emit are dangerous and not advisable for any reason and having our cell phone nearby can lead to nightmares, inability to sleep, and waking up several times each night. The majority of the participants reported keeping mobile phone either away from bed or switched off while sleeping. Study participants used calling facility more than SMS facility which was similar to the study conducted by Stalin *et al*.[10] However, a study conducted in Japan in 2012 among 73 high school students showed that the frequency of using SMS facility was more when compared to calling facility.[15] This difference might be attributed to change in reduction in calling charges in recent years and upsurge of social networking sites and messenger applications. The use of mobile phones for accessing internet or social networking sites was reported by 67.2% of the participants. This is in accordance with the report published by the global social media agency which states that mobile social media use has increased by 30% year-over-year to surpass 2.5 billion users globally, with 91% of social media users accessing it from mobile.[16]

Our results show that increase in daily usage of mobile phone is associated with increase in health problems such as headache, ear pain, stressful eyes, neck pain, restlessness, sleep disturbances, painful fingers, morning tiredness, and dizziness. Headache may result from the radiations emitted by mobile phone affecting the part of skull against which it is being held. Continuous staring at any screen, either small or large, may result in eye strain. Continuous use of fingers especially thumb over a small keypad may result in painful fingers. These results are similar to the previously conducted studies[10,13] where cell phone users suffered from health problems when compared with non-users. A study reported increase in fatigue (odds ratio – 1.85) and sleeping problems (odds ratio – 1.25) among children who were using mobile phones for more than 1 year.[14] A study conducted reported that usage of multimedia for more than 1 h/day was associated with increased risk of health problems.[11] Similar results were reported in a study conducted in Sweden where high mobile phone use was associated with sleep disturbances and symptoms of depression for the men and symptoms of depression for the women at 1-year follow-up.[17] However, a study conducted in the United Kingdom reported no association between mobile phone usage and subjective health symptoms.[18] This calls for an extensive, large-scale research in this area to get evident affirmation on the association of mobile phone usage with subjective health symptoms.

In the present study, it was seen that hypertension was inversely proportional to mobile phone use which was similar to study conducted by Stalin *et al*.[10] and Suresh *et al*.[19] It might be attributed to an increase in parasympathetic activity and reduction in sympathetic activity originating in the brainstem because of usage of mobile phone.[20] Another probability is that increased social media usage on mobile phones might help in establishing greater connectedness of the users with their communities and ultimately reduce stress. This has been reported in the previous research where social networks were associated with lower total mortality by reducing deaths from cardiovascular disease.[21]

More than half of the study participants reported that they tend to be stressed or anxious when they did not have their mobile phones with them or their mobile phones did not have good signal strength. This mobile phone separation anxiety (PSA or nomophobia) is all set to become a matter of global concern.[22] PSA can adversely affect not only the mental well-being and social life but also the relations and communication skills in the "real world." 10.52% of the participants reported using mobile phone while driving which can prove to be dangerous and can take a toll on their life. Similar results were observed in a previous study where 67% participants said that they feel unsettled when they forget to carry mobile phone with them and 13% participants used their mobile while driving to receive calls and messages.[8] Another study conducted reported that students felt anxious without their mobile phone and were addicted to its use.[23] The majority of the participants in our study were aware about the adverse sequelae of excessive use of mobile phone on health and academic or work performance. The majority of them also agreed on the use of mobile phones for security and educational purpose.

LIMITATIONS

The present study involved a convenient sample of the local population visiting dental college from in and around Dhule city. Hence, the results of the study cannot be generalized to entire population of Dhule District. Therefore, an extensive research is recommended to reinforce the results of the present study. Furthermore, the present study focused only on mobile users and non-users were excluded from the study. Hence, widespread research involving mobile phone users as well as non-users is recommended

to compare the prevalence of health problems among uses and non-users.

CONCLUSION

There is a growing concern over impact of mobile phone on human health. The present study shows that an alarming proportion of population, practicing excessive use of cell phones, has been inflicted with a wide array of physical as well as mental health problems. These symptoms cannot be ignored and hence require timely measures to curb the excessive use of mobile phones. The simplest and the most effective measure can be to raise the awareness among the population regarding possible health effects of mobile phones and lay down guidelines to use it judiciously, thus minimizing its exposure and avoiding its addiction.

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Original Article

Evaluation of Basic Life Support Knowledge and Skills of Healthcare and Non-healthcare Providers

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Abstract

Aim: Immediate bystander cardiopulmonary resuscitation (CPR) significantly improves survival after a sudden cardiopulmonary collapse. This study assessed the basic life support (BLS) awareness, knowledge, attitude, and performance of healthcare providers (HCP) and non-HCP before and after CPR training.

Materials and Methods: This study included 4625 participants. Participants completed a pre-test to assess their knowledge and 3 h training course that provided a theoretical background on sudden cardiac death and a hands-on CPR tutorial. They were asked to perform BLS on a manikin to simulate an unconscious scenario before the training. Afterward, participants encountered the same scenario and completed a questionnaire of their post-training knowledge.

Results: A total of 4625 participants were included in this study. Of which 56.54% (n = 2615) were HCP and 43.45% (n = 2010) were non-HCP. There is a significant increase in knowledge of BLS among non-HCP which is clearly evident in pre-training and post-training evaluation (written and hands-on). Only 0.62% employees are able to perform BLS in the correct sequence before the training and 76.7% employees after the training. None of the students performed BLS in the correct sequence before the training and 60.85% students performed well after the training. Among HCP, only 12.08% were able to perform BLS in the correct sequence before the training and 94.8% after the training.

Conclusion: Performing BLS and attending BLS training plays a key role in attaining BLS knowledge by both healthcare and non-HCP.

Key words: Basic life support, Cardiac arrest, Cardiopulmonary resuscitation, Evaluation, Rescue breaths, Training

INTRODUCTION

Cardiac arrest is an important acute emergency situation both within and outside the hospital setups and carries a high level of mortality risk. However, if early basic life support (BLS) – cardiopulmonary resuscitation (CPR) is initiated, the survival rate can be substantially improved.[1]

The aim of BLS is to maintain a distribution of oxygen-rich blood through survival organs, especially the brain and heart, through a temporary artificial circulation until normal cardiac activity and breathing are restored.[2-4] The history of BLS goes back many years. The first studies on the standardization of BLS practices started in America in 1974 and studies on BLS standardization and updates were continued and developed by the European Resuscitation Council, founded in 1989, which also updated and published guidelines on BLS and ALS practices every 5 years.[5] Sudden cardiopulmonary arrests in adults outside of a hospital setting occur due to cardiac causes, whereas cardiopulmonary arrests in hospitalized patients are usually caused by the underlying disease. It was reported that of the patients who experienced cardiopulmonary arrest in a hospital, approximately 84% had progressively different clinical findings in the past 8 h and that morbidity and mortality could increase within hospital mostly due to quick diagnosis and flaws in therapeutic approaches. For this reason, the primary approach in preventing cardiopulmonary arrests within a hospital setting is believed to be recognizing the patients that are under risk beforehand and taking care of preparations for their treatment and their early transfer into the intensive care unit. The knowledge and skill levels regarding CPR of healthcare personnel who discovers a patient with a progressive acute condition or

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cardiopulmonary arrest are the most important criteria in terms of providing a fast and accurate intervention.[6-8] BLS training includes exercises on recognizing cardiopulmonary arrest, acute myocardial infarction, seizure, and foreign body airway obstruction, performing CPR, and using an automated external defibrillator.[3] The most important factor in the success of BLS is time management. It is of vital importance to diagnose and start treatment in a timely fashion. In an organism, brain tissues can only tolerate not receiving oxygen for a few minutes. This time can be longer or shorter, depending on the patient's health condition during cardiac arrest. For instance, cerebral damage would occur within a shorter time if the patient had been in a hypoxemic condition previously. If the patient was in a hypothermic condition, the occurrence of cerebral damage would be delayed.[5] Cardiopulmonary arrest is defined as the time when breathing and/or circulation stops suddenly and unexpectedly for some reason. Clinically, an individual shows signs of blackout, absent pulse, and apnoea during the arrest. Circulatory failure that lasts for three or 4 min could cause irreversible cerebral damage. This duration could be shorter if the individual's hypoxemia developed earlier than noticed. Delay in performing BLS could result in a lower probability of a successful result after BLS.[5,9] On the other hand, there is no evidence to prove that the medications used in advanced life support reduce mortality and morbidity, whereas immediate and efficient BLS practices after cardiac arrest are reported to reduce mortality and morbidity.[10-12]

People who suffer from sudden cardiac arrest depend on prompt BLS. Patients who receive bystander CPR have a 2–3 time's higher survival rate (8.2% vs. 2.5% for patients who did not receive CPR).[13] Extensive education of the population in particular countries and regions led to high numbers of bystander CPR in cases of out-of-hospital cardiac arrests (OHCA).[13-15] However, studies show that often less than one-third of out-of-hospital witnessed cardiac arrest victims receive bystander CPR.[16,17] Furthermore, 50–65% of cardiac arrests happen at home.[8] Since these victims are less likely to receive bystander CPR, they have poorer outcomes than those who are subject to OHCA in other non-hospital locations.[18] In these cases, bystanders are usually family members and can include high school-aged students.

The knowledge of BLS is a major determinant in the success of resuscitation and plays a key role in the final outcome of acute emergency situations. Knowledge of BLS is an absolute necessity for medical professionals to face acute medical emergencies in the hospital and for bystanders for out hospital acute emergencies. Healthcare professional skills should be analyzed according to their gap analysis to improve the patient survival rate and patient satisfaction.[19] In the present study, we aimed to assess the awareness, knowledge, and attitude about adult BLS among healthcare and non-healthcare providers (HCP).

MATERIALS AND METHODS

Sample Design

This study was a quasi-experimental study with pre-test and post-test design.

Data Collection

Data were collected by considering the pre- and post-evaluation of written and simulation test conducted before and after the training period.

Inclusion Criteria

All nurses and employees with more than 6 months experience and students of minimum undergraduate were included in this study.

Exclusion Criteria

All nurses and employees with <6 months experience and students of below undergraduate were excluded from this study.

Statistical Analysis

The Kruskal–Wallis test for independent measures was done.

RESULTS

The summary of the results is as follows.

1. A total of 4625 participants were included in this study
2. Of which 56.54% (n = 2615) were HCP and 43.45% (n = 2010) were non-HCP
3. Among those 29.83% (n = 1380) were males and 70.16% (n = 3245) females. Of total participants, 60.75% (n = 2810) were of <25 years age, 23.3% (n = 1078) were of age 25–40 years, and 15.9% (n = 737) were of age more than 40 years
4. Among non-healthcare providers, 55.52% (n = 1116) were employees and 44.47% (n = 894) were students
5. Among HCP, 60.19% (n = 1574) were of <2 years experience, 24.74% (n = 647) were of 2-years experience, and 15.06% (n = 394) were of more than 5 years experience
6. There is a significant increase in knowledge of BLS among non-HCP which is clearly evident in pre-training and post-training evaluation (written and hands-on). Only 0.62% employees are able to perform BLS in the correct sequence before the training and 76.7% employees after the training. None of the

students performed BLS in the correct sequence before the training and 60.85% students performed well after the training

7. Among HCP, only 12.08% were able to perform BLS in the correct sequence before the training and 94.8% after the training.
8. Knowledge of seeking help in case of any medical emergency is 68.8% in employees, 85.23% in students, 75.49% in HCP's before the training, 94.44% in employees, 95.45% in students, and 96.42% in HCP's
9. Knowledge on airway assessment and management while performing BLS such as opening the airway is 5.1% in employees, 1.35% in students, 56.79% in HCP's before the training, 84.7% in employees, 76.6% in students, and 94.26% in HCP's. Moreover, correctly performing ventilation is 2.77% in employees, 1.00% in students, 62.26% in HCP's before the training and 83.24% in employees, 68.57% in students, and 90.4% in HCP's
10. Knowledge on performing cardiac compressions while performing BLS such as hand position during chest compression is 1.52% in employees, 1.79% in students, 66.62% in HCP's before the training and 91.57% in employees, 74.72% in students, and 91.36% in HCP's. Moreover, correctly performing chest compressions is 1.25% in employees, 0.67% in students, 45.62% in HCP's before the training and 89.06% in employees, 67.45% in students, and 89.75% in HCP's.
11. The statistical analysis was performed using the Kruskal–Wallis test for independent measures. The parameters before and after training were tested for significance at $P = 0.05$. The analysis shows that the H statistic before training is 17.6232 (2, $n = 30$) and obtained $P = 0.00001$ for which the test is significant at 0.05. Hence, we accept the alternative hypothesis and reject the null hypothesis. The analysis shows that the H statistic before training is 25.551 (2, $n = 30$) and obtained $P = 0.00001$ for which the test is significant at 0.05. Hence, we accept the alternative hypothesis and reject the null hypothesis.

DISCUSSION

The present study analyzed BLS knowledge and skills among HCP and non-HCP. Each individual is assessed for individual BLS parameters and sequence of parameters which includes checks unresponsiveness, call for help, calls emergency number, assess breathing, opens airway, correctly performs ventilation, hand position, chest compression, combination of chest compression, and ventilation and following algorithm in the correct sequence.

The results depicted above reveal the following:

1. Lack of professional training of BLS was regarded as the most common hindering factor responsible for poor BLS knowledge among non-HCP

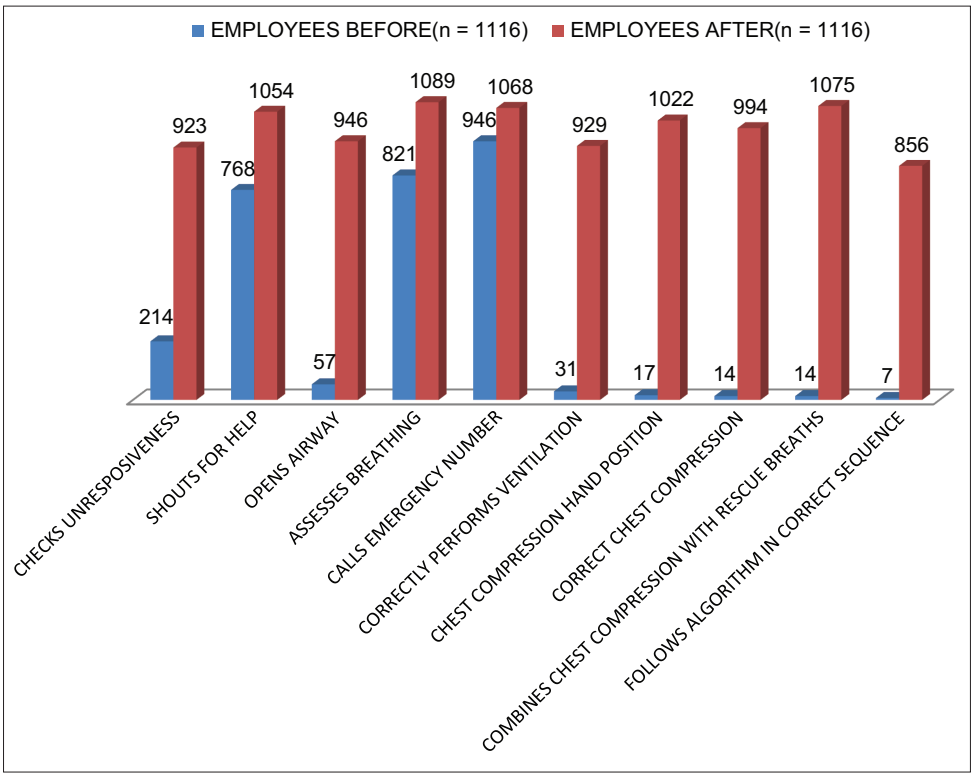

Figure 1: No. of employees and their response to each basic life support parameter

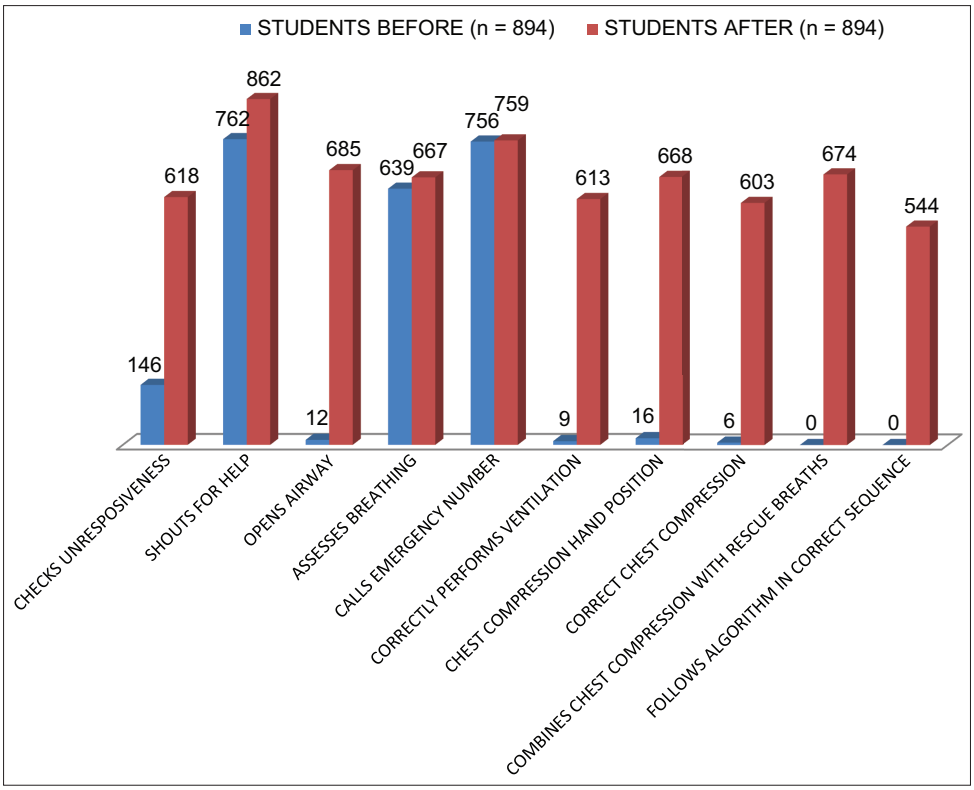

Figure 2: No. of students and their response to each basic life support parameter

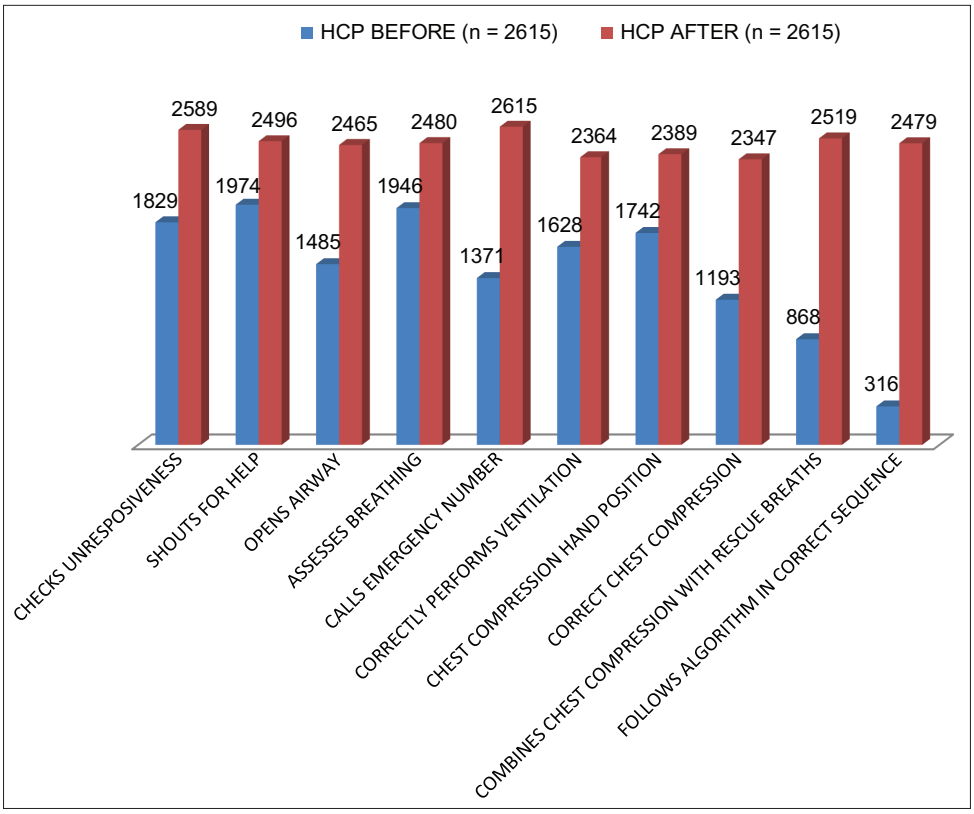

Figure 3: No. of healthcare providers and their response to each basic life support parameter

2. Knowledge on calling for help in case of an emergency is common among HCPs and non-HCPs
3. Knowledge on calling on emergency number in case of OHCA is less among HCPs when compared to non-HCPs, this might be due to HCPs are more concentrating on performing CPR
4. Knowledge on positioning the airway is less in non-HCPs when compared to HCPs
5. There is a complete lack of knowledge on performing compressions among non-HCPs when compared to HCPs
6. Figure 1 depicts there is an increase in knowledge among employees after training
7. Figure 2 depicts there is an increase in knowledge among students after training
8. Figure 3 depicts there is an increase in knowledge among HCP after training.

CONCLUSION

Performing BLS and attending BLS training plays a key role in attaining BLS knowledge by both healthcare and non-HCP. There is a need for structured training of BLS for every individual. This will go a long way in improving the outcome of BLS delivery by healthcare providers and non-HCP, thus immensely benefitting the society and also boosting the morale of the BLS providers. There should be regular refreshing sessions on BLS to all.

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Original Article

Knowledge, Detection, and Reporting of Domestic Violence among Family Medicine Residents of Eastern Province, Saudi Arabia

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Abstract

Introduction: Domestic violence (DV) is a global health-care problem, regardless of the socioeconomic backgrounds and education levels of the nations. Research has always shown that a woman is more likely to be abused by a spousal than by any other person.

Aim: This study aimed to assess the knowledge, detection, and reporting of DV among family medicine resident of Eastern Province, Saudi Arabia.

Materials and Methods: This is a prospective cross-sectional study conducted in the Eastern Province, Saudi Arabia, from November 2018 to September 2019. A modified self-administered questionnaire based on "The Physician Readiness to Manage Intimate Partner Violence Survey" developed and validated in the United States which was distributed among family medicine residents. Both descriptive and inferential statistics were conducted. $P = 0.05$ was considered statistically significant. All data analyses were performed using the SPSS version 20.

Results: There were 166 family medicine residents involved in this study. The mean age was 28.7 years old and majority were female with slightly more were resident 2 (29.5%). In this study, good and poor knowledge were found to be 50.6% and 49.4% of the family residents, respectively. Residents 1 (R1) showed the least knowledge, whereas Residents 2 (R2) showed more knowledge regarding intimate partner violence; however, this result did not differ significantly among the level of knowledge. The most commonly known identified risk factor of DV was alcohol/drugs. The percentage of DV as identified by the residents for the past 6 months was 21.7%. Only around 31.9% of the residents were able to screen new DV patients; however, around one-third (34.3%) of them do not currently screen DV.

Conclusion: There was a moderate level of knowledge regarding DV among the residents. Second level residents showed better knowledge than the other levels while the 1st year level exemplified the least. Alcohol/drugs were the frequently mentioned as the risk factors of DV. On the other hand, residents' practice of screening DV among the patients were found to be low.

Key words: Detection family medicine resident, Domestic violence, Intimate partner violence, Knowledge

INTRODUCTION

Domestic violence (DV) is a global health-care problem, regardless of the socioeconomic backgrounds and education levels of the nations. Research has always shown that a woman is more likely to be abused by a spousal than by any other person.[1] It refers to any violence take place in the home, including violence against children and the elderly.[2]

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Abuse can lead to significant consequences on the health of each member of the society. More than 1.6 million deaths can result worldwide every year. About 90% and more of these occur in low- and middle-income countries, and it is one of the leading causes of death in all parts of the world for persons ages 15–44.[3]

The United Nations in 1993 defines violence against women[4] as "any act of gender-based violence that results in, or is likely to result in, physical, sexual, or mental

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harm or suffering to women, including threats of such acts, coercion, or arbitrary deprivation of liberty, whether occurring in public or in private life."[5] In 1992, Centers for Disease Control and Prevention established the National Center for Injury Prevention and Control (NCIPC) as the primary federal organization for abuse determination. The Division of Violence Prevention is one of three divisions within NCIPC; their target is to prevent harms caused by violence.[6]

International research always determines that a woman is more likely to be assaulted or abused by a current or former partner than by any other person. These studies indicate that a lifetime prevalence of partner violence is estimated between 20 and 50% of women who have ever been partnered.[7] According to the WHO multicountry study on women's health and DV, "lifetime prevalence of physical or sexual spousal abuse, or both, ranged from 15% to 71%, with two locations having a prevalence of less than 25%, seven between 25% and 50%, and six between 50% and 75%."[8] The findings confirm that physical and sexual partner violence against women is widespread.[8]

In Arab, world's studies regarding violence against women were conducted since the past decade. In 2009, a study in Jordan showed that percentages of women facing at least 1 form of control or abuse since marriage were control, 97.2%; psychological maltreatment, 73.4%; physical disorder, 31.2%; and sexual violence, 18.8%.[9] Other study conducted in Egypt showed comparable results, 34% of women in the sample were ever assaulted by their current husband while 16% were beaten in the past year.[1] In Saudi Arabia, different studies have addressed the problem of intimate partner violence (IPV) and another type of DV. In 2016, the prevalence of spousal abuse and its associated risk factors among Saudi female patients attending the primary health-care centers in Western Saudi Arabia concluded that one out of 10 women is a victim of spousal abuse in Taif, KSA. IPV or spousal abuse is significantly associated with a number of psychosocial factors. The detection of which might help to screen for individuals at risk.[10] Other study was conducted in the Eastern Province concluded that DV is as common in Al-Ahsa as in other countries of the world with a lifetime prevalence of 39%.[11] In a cross-sectional study by Fakeeh, about factors associated with IPV "Abused women had more children than non-abused women, and their spouses were significantly older than those of non-abused women. Financially dependent women and those with a high educational status were significantly more likely to report abuse. Assaulted women were also likely to report that their abuser was a smoker and had completed at least primary or secondary education. A significantly lower proportion of abused women said that their male partners were alcohol users. The results of logistic regression found that financially dependent women had about 1.5-fold odds of being physically abused."[12]

The significance of the IPV as a public health problem because of its high prevalence and due to its massive impact not only on the women's health but also the lives inside the womb. It is associated with severe health problems affecting both women and children, including injuries, gynecological disorders, mental health disorders, adverse pregnancy outcomes, and sexually transmitted infections.[7,13]

Today, there are many international institutions against violence. More organizations, service providers, and policy-makers are recognizing that IPV has negative ramification for women's health and the society.[14]

Here, in Saudi Arabia, the council of ministers approved the regulations to protect against abuse in August 2013 by the law of protection from abuse.[15] Hence, it was a great accomplishment regarding this public health problem and reflect the community acknowledgment and awareness of all aspects of health.

Since the family physician is the first contact with the patients in most health-care systems, he/she should be able to detect and report DV according to the laws and regulations available to his country. In many studies, this can be achieved by excellent staff and clinician training in effective patient-centered IPV assessment that ensures a safe and supportive environment for the patient,[16] but unfortunately, many clinicians are unclear about their role in a multiagency response regarding DV.[17,18]

The purpose of this research is to determine how ready are the family medicine residents of the Eastern Province, Saudi Arabia, in the field of detection and reporting DV. Since it is one of the top 50 topics in Saudi board family medicine curriculum,[19] the focus will be on their current knowledge, how to detect and how to report such prevalent cases.

It is also the first study to be conducted in the Eastern Province of Saudi Arabia about the awareness of DV among family medicine resident, to assess their knowledge and clinical practice regarding IPV as the most prevalent type of DV worldwide[14] and their essential role as first-line health-care provider. Hence, the study will evaluate our training progress in this field.

Aim and Objective

This study aims to assess the awareness, knowledge, detection, and reporting of DV among family medicine resident of Eastern Province, Saudi Arabia.

MATERIALS AND METHODS

This is a prospective cross-sectional study conducted in the Eastern Province, Saudi Arabia, from November 2018 to September 2019. A modified self-administered questionnaire based on "The Physician Readiness to Manage IPV Survey (PREMIS)" developed and validated in the United States.[20] It contains five parts; responder profile, background, knowledge, opinions, and practice issues. The questionnaire was modified for use in the study, by deleting culturally sensitive items and the irrelevant items that did not reflect on result objectives. The survey was printed in hard copies and distributed by the primary investigator to the targeted subjects. Each subject needed approximately 25 min to complete the survey.

Study Subjects

All family medicine residents who were in training under Saudi Commission of Health Specialties in the Eastern Province, Saudi Arabia, were eligible in the study. The subjects were recruited when they attend the ½ day release activity for family medicine residents' program by each center on Mondays after permission taken from each program director. A consecutive sampling technique had been applied and the sample size calculated using Raosoft Website with maximum error allowed 5% (common choice), confidence level 95%, target population estimated to be 320, at least response rate 50% (recommended), total sample to be done is 175, since this was almost 50% of target population, we considered all residents at the visit time. Those residents not under training of family medicine program of Saudi Commission for Health Specialties in the Eastern Province were considered as excluded criterion.

Statistical Analysis

Descriptive statistics were presented as counts and proportion (%). The relationship between the level of knowledge and the sociodemographic characteristics of residents had been calculated using Chi-square test. $P < 0.05$ was considered as statistically significant. All statistical analyses were carried out using Statistical Packages for the Software Sciences version 20 (IBM SPSS, Chicago, Illinois, USA).

RESULTS

PREMIS questionnaires were distributed to 175 residents of Family Medicine Program in different levels in the Eastern Province, Saudi Arabia. There were 166

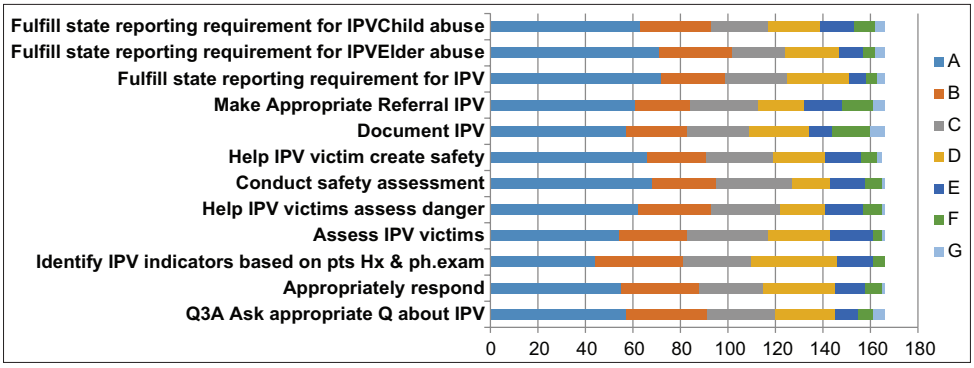

Figure 1: Which best describes how prepared are you to perform the following. A=Not prepared, B=Minimally prepared, C=Slightly prepared, D=Moderately prepared, E=Fairly well prepared, F=Well prepared, G=Quite well prepared

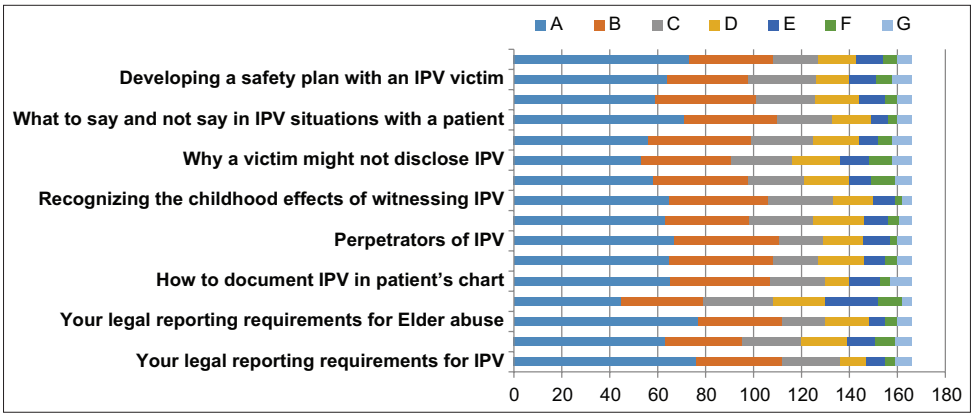

Figure 2: How much you feel you know about. A=Nothing, B=Very little, C=Little, D=Moderate, E=Fair, F=Quite abet, G=Very much

complete (94.8%) response rates, 30.7% were male and 69.3% were female. The mean age was 28.7 years old and the mean practice duration was 3.2 years across all levels [Table 1].

Perceived Preparation to Manage IPV Patients and Perceived Knowledge of IPV

Physician scores for perceived knowledge reflected those for perceived preparation. Levels of perceived preparation were low for all items. On all items across the two scales, there were no significant differences between residency levels.

One-third of the physicians, around 34.34% (57) find themselves not prepared to ask appropriate questions about IPV, where only 3% (5) were well prepared, the rest 64% were minimally to moderately prepared [Figure 1]. Furthermore, around 33% (55) were not prepared to respond to disclosures of abuse or to assess IPV victims, and only around 4.8% (8) think that they were well prepared, the rest 62% were minimally to moderately prepared to do so.

Almost 55.5% (92) responded as not to minimally prepared to help IPV victim to create safety plan, and only 5% (9) think that they were well prepared to do so, the rest 39% were slightly to moderately prepared to help IPV victim to create safety plan [Figure 2].

Many physicians, around 36.7% (61), are not prepared to document IPV or even make appropriate referrals.

Around 45% of the responders report said that they know nothing about the legal reporting requirements for IPV, and only 4.2% (7) know very much. Almost, 27% (45) report said that they know nothing about signs or symptoms of IPV, and only around 17% (29) know little about them. More than 50% do not know how to document IPV in patient charts or referral sources for the victims and only 5% (9) have knowledge about them it.

Actual IPV Knowledge

For R1 level, majority of them (57%) have had poor knowledge, while 42.9% had good results. For R2 level, 40.8% poor, while 59.2% scores scored good. Regarding R3 level, 48.9% had poor knowledge and the rest 51.1% scores scored good results. Finally, for R4 level, 54.5% had poor knowledge and 45.5% had scores good. For items testing actual knowledge about IPV, there were no significant differences in scores between based on residency levels ($P = 0.460$) or gender ($P = 0.106$).

Female scores scored good knowledge around 54.5% more than males who had score scored good 41.2%. Regarding the strongest single risk factor for being an IPV victims, only 27.7% (46) had choose the correct answer (the gender as female), while the majority (47.6%) (79) think that partner abuse alcohol/drugs is the strongest risk factor. Around 39.8% were able to choose the correct response regarding what is true about the batterers as (they use violence as a mean of to control their victim).

The majority of residents had a poor understanding of the medical conditions associated with IPV, such as chronic unexplained pain, anxiety, substance abuse, and depression but most of them, 64% (107), agreed that frequent injures are associated with IPV. About 56% (93) of the residents choose children as the reasons why woman experiencing abuse may feel unable to leave the relationship. However, less than 30% thought that it was appropriate to ask directly

Table 1: Demographic characteristics of the responders

Variables	Frequency	Percentage
Residency level		
R1	28	16.90
R2	49	29.50
R3	45	27.10
R4	44	26.50
Sex, %		
Male	52	30.70
Female	115	69.30
Age		
Years, mean	28.7	
Practice years		
Mean	3.2	
Mean number of estimated patients per week		
<20	9	5.40
20–39	63	38.00
40–59	57	34.30
60+	36	21.70
Total	166	100

Knowledge groups * residency level groups cross tabulation

			Residency level groups				Total
			R1	R2	R3	R4	
Knowledge groups	Poor	Count	16	20	22	24	82
			57.1%	40.8%	48.9%	54.5%	49.4%
	Good	Count	12	29	23	20	84
			42.9%	59.2%	51.1%	45.5%	50.6%
Total	Count	28	49	45	44	166	
		100.0%	100.0%	100.0%	100.0%	100.0%	

if the partner had ever hit or hurt the woman, and the majority 61% (102) prefer indirect asking.

Practice Issues: Detection and Reporting

Identifying abuse only 21.7% (36) of the residents had identified at least one new DV case in the preceding 6 months, and 60.8% (101) never identified DV cases.

Screening for IPV

About 34.3% (57) do not currently screen for DV and around 31.9% (53) screen new patients. Only 2.4% (4) screen pregnant women and female patients periodically. The majority of the responders do not screen teenagers or young adult women, were only 9% (15) screen single or divorced women for IPV. Almost 96.4% (160) of the resident do not screen married women and only 3.6% do screening. Only around 8% (14) of the resident were able to ask about possibility of IPV when seeing patients with injures, chronic pelvic pain, IBS, headache, diarrhea, and anxiety.

How often in the past 6 months have you asked about possibility of IPV when seeing patients with the following

	Never to sometimes	nearly always	Always	N/A
Injuries	117	13	12	21
	72.3%	7.8%	7.2%	12.7%
Chronic pelvic	134	12	2	18
	80.6%	7.2%	1.2%	10.8%
Irritable bowel syndrome	133	12	6	15
	80.1%	7.2%	3.6%	9.0%
Headache	131	14	6	15
	78.9%	8.4%	3.6%	9.0%
Depression anxiety	113	18	14	21
	68%	10.8%	8.4%	12.7%
Hypertension	138	8	3	17
	83%	4.8%	1.8%	10.2%
Eating disorder	129			
77.6%	16	4	17	
		9.6%	2.4%	10.2%

Majority of the responders 54.8% (91) were unsure about the protocol for dealing with adult IPV case at their centers and only 6.6% (11) were knowledgeable and used it, also, most of the residents 78.5% (122) are were not familiar with their institution's policies regarding screening and management of IPV victims, around 60% never document patient's statement of IPV in the patient chart or ever used a body map to document patient injures. Almost 4.2% (7) are able to notify appropriate authorities if IPV victim identified and conducted a safety assessment for them or for their children. About 48.2% (80) were unsure about availability of IPV patient education or resource materials at their practice centers and only 5.4% (9) were sure about them. Furthermore, 15% (26) feel that they have adequate referral resources for the victims, but 46.4% (77) were unsure.

DISCUSSION

This study sought to determine the knowledge, detection, and reporting of DV among family medicine residents of Eastern Province, Saudi Arabia. In this study, half of the residents were having a good knowledge toward DV while the rest had poor knowledge (49.4%). We further observed that among the residency levels, R2 showed better knowledge and it showed mostly among females. However, these results did not differ significantly among the group. The knowledge of clinicians toward DV varies with each region. In neighboring country,[21] researchers reported that although knowledge and attitude score were not statistically significant predictors of DV; nonetheless, those physicians who were screening for violence had significantly higher mean percentage for overall knowledge score which corroborated our study findings. In Israel, Khan *et al*.,[22] when they compared the knowledge and attitudes of family medicine and general practitioners (GPs), they found that family physicians reported more exposure to battered women and had better knowledge of its prevalence and its risk factors. They also showed a greater tendency to view the problem as universal which was not on the line with our results. In the United Kingdom,[20] when they examined the knowledge, attitudes, and clinical practice of selected primary health-care clinicians. They found out that clinicians had basic knowledge about DV but exhibited a positive attitude toward engaging with women experiencing abuse. They further observed that GPs were more prepared and more knowledgeable than practice nurses, to which they can be able to identify a higher number of DV cases. In addition, in the United States,[23] residents showed higher levels of background and knowledge of IPV than medical students and they further observed that being a resident, being female, and being confident about talking to patient regarding IPV was the predictors for increased clinical knowledge about IPV which showed better outcome compared to our study.

In this study, we identified that being a female, being abusive of alcohol/drugs, and frequent injuries are associated with IPV. Alcohol/drugs were also determined to being associated with DV which was reported by Ramsey *et al*.[20] They further indicated that majority of the responders wrongly agreed that alcohol abuse is a leading cause of DV.

Identifying patient who was exposed to DV is necessary to provide the required intervention among the victim. In this study, residents were able to identify 21.7% of at least one new case of DV in the preceding 6 months. In contrast, there were 60.8% of residents never identified DV. This result is not consistent from the paper published by Ramsey *et al*.,[20] among the 272 clinicians, 54% of the clinicians had identified at least one new DV case for the past 6 months which was higher than our study finding. On the other hand, Wenzel *et al*.[24] reported that family medicine residents

were able to identify 5.4% DV cases at some points of their lives with 2.9% of them were self-reported being currently involved in abusive relationships while 2.2% relayed histories of past abuse which was lower than our outcome.

It is important to note that proper action is necessary in case a patient was exposed to DV. In this study, more than a half of the residents were uncertain about the protocol pertaining to DV cases with only 6.6% had sufficient knowledge about it. Moreover, the management and screening among IPV victims were also poorly exercised and documented and a relatively fewer IPV cases (7 cases) were able to refer to appropriate authorities for the necessary assessment and intervention. Likewise, a little below a half of them (48.2%) were not aware of IPV patient education or resource materials at the current health-care setting. These results are not congruent from the study published by Ramsey *et al*.[20] They observed that between 36% and 48% of clinicians reported that they provided information, education, or counseling to the victim while 43% had made a referral to other agencies. Furthermore, the practice of documentation was also better compared to our study since, 70% of the clinicians were practicing documentation of abuse with 30% never or seldom provided referral or resource materials.

The practice of DV screening among injured patients was also poorly achieved in this study. Among the residents of all levels, 34.3% of them do not apply DV screening among injured patients, whereas 31.9% did it otherwise. This result corroborates with the paper of Ramsey *et al*.[20] They accounted that 60.3% of the clinicians do not routinely ask about DV while Sugg *et al*.[25] observed that 39.3% of the primary care providers expressed confidence in asking about physical abused among patients while 45% were never or seldom asked about DV when examining injured patients which was also in line with our study results.

CONCLUSION

There was a moderate level of knowledge regarding DV among the residents. Second level residents showed better knowledge than the other levels while the 1st year level exemplified the least. Alcohol/drugs were the frequently mentioned as the risk factors of DV. On the other hand, residents' practice of screening DV among the patients was found to be low. In consideration with the importance of DV as public health issue, more educations are needed regarding the importance of screening DV among the residents.

Recommendations

1. Improve guidelines regarding screening and management of IPV.
2. Mandate training of family medicine residents to practice the approved IPV screening and management guidelines.
3. Routine courses with contract renewal to ensure the effectiveness of the training.

Actions

1. To distribute the study to program directors and primary care centers chairmen.
2. To distribute appropriate pamphlets/brochures of guidelines summaries and how to notify IPV.

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Original Article

Prevalence of Obstructive Sleep Apnea in Surgical Patients Attending at SKIMS Hospital, Kashmir, and its Correlation with Perioperative Morbidity

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Abstract

Background and Objective: Patients presenting for surgical procedures often get undiagnosed for obstructive sleep apnea (OSA), thus increasing the incidence of perioperative adverse outcomes. Hence, early diagnosis of this disease is important in formulating anesthetic management and specific means which may decrease the complications and improve outcome, and therefore, the study was conducted to evaluate the prevalence of OSA in patients presenting in our institute for surgical procedures.

Materials and Methods: A total of 600 patients of aged >18 years, American Society of Anesthesiologists I-III scheduled for elective surgeries under anesthesia, were randomly enrolled in the study. Their demographic data, anthropometric measurements were noted. They were screened for OSA by STOP-BANG questionnaire and were followed to assess correlation between OSA and perioperative morbidity.

Results: We observed that out of a total of 600 patients, 23 patients had moderate and severe OSA. Hence, the prevalence of moderate-to-severe OSA was 3.8% in our study. Mean age of subjects was 43.1 years and female predominance was seen in this study. Out of a total of 600 patients, 23 patients had moderate and severe OSA. There was a significant correlation between severity of OSA and anthropometric measurements and perioperative morbidity.

Conclusion: Early screening can help in detecting the OSA among patients and thus help in alleviating perioperative morbidity.

Key words: OSA, Perioperative morbidity, Prevalence, Stop-Bang

INTRODUCTION

Sleep disordered breathing includes a spectrum of conditions, the most severe of which is obstructive sleep apnea (OSA) syndrome. It is a potentially disabling condition characterized by disruptive snoring, repeated episodes of complete or partial pharyngeal obstruction during sleep resulting in nocturnal hypoxemia, frequent arousals, and excessive daytime sleepiness.[1]

OSA is a sleep disorder that involves cessation or significant decrease in airflow in spite of a breathing effort. It is the most common form of recurrent episode of upper airway collapse during sleep. These episodes are associated with recurrent oxygen desaturations and arousal from sleep, with excessive daytime sleepiness; it is referred to as "OSA Syndrome."

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Pathophysiologically, the upper airway, which is a tube, collapses during sleep, causing obstruction to airflow either at the level of soft palate (nasopharynx) or tongue (oropharynx). Both anatomic and neuromuscular factors are involved.

Patients with OSA who are undergoing procedures that require sedation, anesthesia, and/or analgesia are at higher risk for complications than patients who do not have OSA. This may be due to upper airway collapse and/or OSA-related comorbidities.

The following perioperative factors increase the frequency and/or duration of upper airway collapse in patients with OSA:

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- Perioperative medications (e.g., sedatives, general anesthetic agents, and narcotic analgesics).
- Upper airway narrowing – post-intubation edema, post-operative edema, nasal packing, nasal tubes, and/or hematomas.
- Supine positioning.
- Sleep deprivation.
- Cessation of continuous positive airway pressure (CPAP) therapy.

Numerous perioperative complications are more common among patients with OSA than among those without OSA. They include difficulty in intubating patients,[2,3] complicated post-extubation course, large blood pressure fluctuations,[4] profound oxyhemoglobin desaturation,[4] myocardial ischemia,[4] cardiac arrhythmias,[4] delirium,[5] post-obstructive pulmonary edema (from breathing against an obstructed upper airway),[6,7] and respiratory arrest.[8,9]

OSA nearly affects 13–19% in India.[10,11] The previous reports suggest that around 3.2–24% number of patients undergoing surgery suffer from OSA depending on the population studied.[12,13]

There is a paucity of data on the prevalence of OSA among patients presenting for surgical procedures. With this background, we conducted a study to evaluate prevalence of OSA in patients presenting in our institute for surgical procedures. We also studied the correlation between demographic profile and anthropometric measurements with the risk of OSA and between severity of OSA and perioperative morbidity.

MATERIALS AND METHODS

The study entitled "Prevalence of OSA in surgical patients attending at Sher-i-Kashmir Institute of Medical Sciences Srinagar in Northern India and its correlation with intraoperative morbidity" was conducted in the Department of Anaesthesiology and Critical Care, Sher-i-Kashmir Institute of Medical Sciences Srinagar, Soura, Jammu and Kashmir, India, after seeking clearance from the Institutional Ethics Committee.

Patients >18 years of age, American Society of Anesthesiologists (ASA) Class I-II scheduled for elective surgical procedures under anesthesia over a period of 6 months (October 2015–March 2016), were enrolled for the study. All patients were explained the purpose of the study and informed consent to participate in the study was taken from all patients.

Children below the age of 18 years, pregnant females, history of substance dependence (except tobacco), emergency surgery, and patients not willing to provide consent were excluded from the study.

Patients were evaluated with the pre-operative STOP BANG questionnaire (Appendix-I) and were classified on basis of score into mild, moderate, and severe OSA. All patients were followed in the perioperative period to see statistical correlation between the severities of OSA with perioperative morbidity. Physical examination of patients was done in the outpatient department (Pre-anesthetic clinic Room).

For the purpose of the study, the following parameters were measured and recorded:

1. Height – using stadiometer.
2. Weight – using a calibrated spring scale.
3. Body mass index (BMI) – calculated using above two variables (1 and 2).
4. Neck circumference – using a non-elastic tape at the level of cricothyroid.

STOP-BANG Sleep Apnea Questionnaire

Chung F et al Anesthesiology 2008 and BJA 2012

STOP		
Do you SNORE loudly (louder than talking or loud enough to be heard through closed doors)?	Yes	No
Do you often feel TIRED, fatigued, or sleepy during daytime?	Yes	No
Has anyone OBSERVED you stop breathing during your sleep?	Yes	No
Do you have or are you being treated for high blood PRESSURE?	Yes	No

BANG		
BMI more than 35kg/m2?	Yes	No
AGE over 50 years old?	Yes	No
NECK circumference > 16 inches (40cm)?	Yes	No
GENDER: Male?	Yes	No

TOTAL SCORE		

High risk of OSA: Yes 5 - 8

Intermediate risk of OSA: Yes 3 - 4

Low risk of OSA: Yes 0 - 2

Statistical Methods

The recorded data were compiled and entered into a spread sheet (Microsoft Excel) and then exported to data editor of SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were summarized in the form of means and standard deviations and categorical variables were expressed as frequencies and percentages. Graphically, the data were presented by bar diagrams. Analysis of variance was employed for comparing continuous variables. Chi-square test was applied for comparing categorical variables. $P < 0.05$ was considered statistically significant. All P values were two tailed.

RESULTS

A total of 600 patients were enrolled in our study during the study period. Out of these 600 patients, 23 patients had OSA (moderate and severe) as per the STOP BANG model. However, 577 patients had mild OSA as per the criteria laid down in the STOP BANG model [Tables 1-9].

Table 1: Prevalence of OSA in studied population

OSA	Number of patients	Prevalence (%)
Yes	23	3.8
No	577	

OSA: Obstructive sleep apnea

Table 2: Age, gender, ASA status, BMI, and severity of OSA in the study patients

Parameters	No. (%)
Age (years)	
18–39	290 (48.3)
40–59	233 (38.8)
≥ 60	77 (12.8)
Total	600 (100)
Gender	
Male	279 (46.5)
Female	321 (53.5)
Total	600 (100)
ASA	
ASA I	403 (67.2)
ASA II	197 (32.8)
Total	600 (100)
BMI (kg/m^2)	
<20	84 (14)
20–24.9	262 (43.7)
25–29.9	183 (30.5)
30–34.9	54 (9)
≥35	17 (2.8)
Total	600 (100)
Severity of OSA	
Mild OSA	577 (96.2)
Moderate OSA	11 (1.8)
Severe OSA	12 (2)
Total	600 (100)

OSA: Obstructive sleep apnea, BMI: Body mass index, ASA: American Society of Anesthesiologists

DISCUSSION

In our study, majority of patients were 18–39 years of age with a mean age of 43.1 ± 13.44 years. Similar demographic profile was seen by Agarwal *et al.* (2013) in their study.[14] They found a mean age of 42.7 years with female preponderance. Our results are consistent with their study as we found 321 (53.5%) female patients in our study out of a total of 600 patients. In our study, 262 (43.7%) patients had a BMI of 20–24.9 (kg/m^2) while as 17 (2.8%) patients had a BMI of > 35(kg/m^2) with a mean BMI of 24.3 ± 3.96 (kg/m^2). Five hundred and seventy-seven (96.2%) patients were found to have mild OSA, 11 (1.8%) patients having moderate OSA, and 12 (2.0%) patients were having severe OSA. Five hundred and seventy-seven patients with mild OSA had a mean age of 42.1 ± 13.24 years, 11 patients with moderate OSA had a mean age of 53.4 ± 8.57 years, and 12 patients with severe OSA had a mean age of 61.5 ± 9.01 years. When BMI was compared with OSA, we observed that 577 patients with mild OSA had a mean BMI of 24.1 ± 3.30 (kg/m^2). Eleven patients of moderate OSA were having 33.5 ± 1.76 (kg/m^2). Twelve patients with severe OSA had a mean BMI of 36.9 ± 2.14 (kg/m^2). Five hundred and seventy-seven patients with mild OSA had a mean neck circumference of 32.4 ± 4.54 cm. Eleven patients of moderate OSA were having mean neck circumference of 37.5 ± 2.54 cm. Twelve patients with severe OSA had a mean neck circumference of 40.7 ± 2.13 cm. When association of severity of OSA with Mallampati grading score was done, it was found that majority of patients with severe OSA, 8 (66.7%) patients belonged to Grade IV, and 4 (33.3%) patients belonged

Table 3: Mean±SD age in patients with OSA

Severity of OSA	Number of patients	Age (years) Mean±SD	Comparison	*P*-value
Mild OSA (I)	577	42.1±13.24	I versus II	<0.001*
Moderate OSA (II)	11	53.4±8.57	II versus III	<0.001*
Severe OSA (III)	12	61.5±9.01	III versus I	<0.001*

OSA: Obstructive sleep apnea

Table 4: Mean±SD, BMI in patients with OSA

Severity of OSA	Number of patients	BMI (kg/m^2) Mean±SD	Comparison	*P*-value
Mild OSA (I)	577	24.1±3.30	I vs. II	<0.001*
Moderate OSA (II)	11	33.5±1.76	II vs. III	0.005*
Severe OSA (III)	12	36.9±2.14	III vs. I	<0.001*

OSA: Obstructive sleep apnea, BMI: Body mass index

Table 5: Mean±SD, neck circumference (cm) in patients with OSA

Severity of OSA	Number of patients	Neck circumference Mean±SD	Comparison	*P*-value
Mild OSA	577	32.4±4.54	I vs. II	<0.001*
Moderate OSA	11	37.5±2.54	II vs. III	0.007*
Severe OSA	12	40.7±2.13	III vs. I	<0.001*

OSA: Obstructive sleep apnea

Table 6: Association of severity of OSA with Mallampati scoring

MP grade	Mild OSA No. (% age)	Moderate OSA No. (% age)	Severe OSA No. (% age)
Grade I	165 (28.6)	0 (0)	0 (0)
Grade II	353 (61.2)	1 (9.1)	0 (0)
Grade III	48 (8.3)	7 (63.6)	4 (33.3)
Grade IV	11 (1.9)	3 (27.3)	8 (66.7)
Total	577 (100)	11 (100)	12 (100)

Chi-square=210.3; *P*<0.001*. OSA: Obstructive sleep apnea

Table 7: Incidence of bougie usage in patients with OSA

Bougie used	Mild OSA No. (% age)	Moderate OSA No. (% age)	Severe OSA No. (% age)
Yes	13 (2.3)	5 (45.5)	9 (75)
No	564 (97.7)	6 (54.5)	3 (25)
Total	577 (100)	11 (100)	12 (100)

OSA: Obstructive sleep apnea

Table 8: Relation of post-operative oxygen saturation with severity of OSA

SpO_2	Mild OSA No. (% age)	Moderate OSA No. (% age)	Severe OSA No. (% age)
<90	13 (2.3)	3 (27.3)	10 (83.3)
90–95	131 (22.7)	7 (63.6)	2 (16.7)
>95	433 (75)	1 (9.1)	0 (0)
Total	577 (100)	11 (100)	12 (100)

OSA: Obstructive sleep apnea

Table 9: Use of CPAP in patients with OSA postoperatively

CPAP used	Mild OSA No. (% age)	Moderate OSA No. (% age)	Severe OSA No. (% age)
Yes	14 (2.4)	4 (36.4)	10 (83.3)
No	563 (97.6)	7 (63.6)	2 (16.7)
Total	577 (100)	11 (100)	12 (100)

Chi-square=198.3; *P*<0.001*, OSA: Obstructive sleep apnea, CPAP: Continuous positive airway pressure

to Grade III. None of the patients in severe OSA had Grade I or Grade II Mallampati grading score. In moderate OSA, majority of patients, 7 (63.6%) patients belonged to Grade III and 3 (27.3%) patients belonged to Grade IV and 1 (9.1%) patient belonged to Grade II. None of the patients were present in Grade I Mallampati grading score. In mild OSA, majority of patients, 353 (61.2%) patients belonged to Grade II and 165 (28.6%) patients belonged to Grade I and 48 (8.3%) patients belonged to Grade III. Eleven (1.3%) patients belonged to Grade IV Mallampati grading score. We never used bougie in majority of patients with mild OSA 564 (97.7%) while as in only 13 (2.3%) patients of mild OSA bougie were used to facilitate intubation. In moderate OSA group, 5 (45.5%) patients used bougie to facilitate intubation while as in 6 (54.5%) patients, bougie was not used to facilitate intubation. In severe OSA group, bougie was used in 9 (75%) patients to facilitate intubation, while as in 3 (25%) patients, bougie was not used to facilitate intubation. In our study, 433 (75%) patients with mild OSA had oxygen saturation of >95, 7 (63.6%) patients with moderate OSA had an oxygen saturation of 90–95, while as 10 (83.3%) patients of severe OSA had oxygen saturation of <90 in the post-operative period. When association of severity of OSA with the usage of CPAP postoperatively was observed, 563 (97.6%) patients with mild OSA never needed CPAP postoperatively. However, 14 (2.4%) patients in mild OSA used CPAP postoperatively. In moderate OSA group, 7 (63.6%) patients never needed CPAP postoperatively while as 4 (36.4%) patients in moderate OSA group needed CPAP postoperatively. In severe OSA group, 10 (83.3%) patients needed CPAP postoperatively, 2 (16.7%) patients in severe OSA group did not need CPAP postoperatively.

In addition, these subjects had higher BMI, larger neck circumference, and abdominal girth. Male predominance was also seen in 72% in patients with moderate and severe OSA. Earlier studies have reported varied prevalence of OSA in surgical patients.[13]

Our results suggested that at least surgical population in India is not different from the Western population with regard to risk of OSA. This is important to recognize considering the potential intraoperative and post-operative complications of OSA.[15] These subjects were fatter than the other group. Male gender and high BMI are known risk factors for OSA and this is why these characteristics have been included in the STOPBANG questionnaire.[15-18]

In the study done by Aggarwal *et al.* (2013),[14] it was seen that the incidence of cardiovascular diseases, diabetes mellitus, and hypothyroidism was higher in patients with OSA. However, we did not study the incidence of cardiovascular diseases, diabetes mellitus, or hypothyroidism in this study. However, on reviewing the literature, these patients are at risk of developing complications related to cardiovascular diseases, diabetes mellitus, and hypothyroidism in the perioperative period. Hence, screening of these patients in the pre-anesthetic clinical evaluation may help to avoid and reduce these complications.[19-24]

CONCLUSION

We found that patients with moderate-to-severe OSA had a prevalence of 3.8%. Most of the patients were in the age group of 18–60 years with a female preponderance. Patients with severe OSA had an increased incidence of poor Mallampati scoring grades, increased use of bougie to facilitate intubation, increased incidence of oxygen desaturation, and increased use of CPAP in the post-operative period.

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Original Article

Role of Non-stress Test and Vibroacoustic Stimulation Test in Prediction of Perinatal Outcome in High-risk Pregnancies

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Abstract

Objectives: The aim of the study was to evaluate the fetal well being by non-stress-test (NST) and vibroacoustic stimulation test (VAST) in high-risk pregnancies, to assess the perinatal outcome, to study the ability of the VAST to convert a false-positive (non-reactive) NST to a reactive one.

Materials and Methods: A total of 100 pregnant women with >32 weeks gestation having certain high-risk factors were subjected to NST and if NST came out to be non-reactive, vibroacoustic stimulation was given with artificial larynx. Perinatal outcome was assessed by various parameters (meconium stained liquor, Apgar score at 5 min, neonatal intensive care units admission). The results were analyzed by Chi-square test to find the association between NST, VAST results, and perinatal outcome.

Results: It was found that VAST reduced the number of false-positive results by 31%. As compared to NST, VAST had less sensitivity (78.05% vs. 80.48%), and better specificity (95.08% vs. 83.61%), better positive predictive valve (91.43% vs. 82.35%) in predicting perinatal outcome.

Conclusion: The addition of vibroacoustic stimulation to the NST reduced significantly the number of non-reactive tests. NST when reactive does represents a satisfactory indicator for fetal well-being but non-reactive test needs further evaluation before any active intervention.

Key words: Non-stress test, Perinatal outcome, Vibroacoustic stimulation test

INTRODUCTION

The main aim of antenatal care is the delivery of a healthy baby without impairing the health of the mother. The assessment of fetal well-being is widely practiced by monitoring the fetal heart rate and its patterns. Fetal morbidity and mortality are greater in high-risk women with Intrauterine growth restriction, hypertension, prolonged pregnancy or other high risk factors.[1] Non-stress test (NST) was introduced to describe fetal heart rate acceleration in response to fetal movement as a sign of fetal health.[2] This test involves the use of Doppler to detect fetal heart rate acceleration which is coincident with fetal movements that are perceived by the mother.

Some pregnancies can be complicated by medical problems in the mother which may have impact on the fetus and pregnancy-specific problems in which fetal health may be affected. In such cases, it becomes all the more important to monitor fetal well-being. The NST is a useful test in the management of high-risk pregnancies. It has high predictive value and a low false-negative rate but has high false-positive results. Attempts have been made to find a suitable stimulant to help decrease non-reactive results as well as to shorten the duration of testing. The vibroacoustic stimulation test (VAST) may have such attributes. The addition of VAST for the fetal assessment in high-risk pregnancies has been proved to be a reliable test due to higher accuracy, ease of performing test and shorter duration of test.[3,4]

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Aim and Objectives

The objective of the study was to compare the VAST with the NST with regard to the ability of detecting a compromised fetus in women with high-risk pregnancies of >32 weeks of gestation, to assess the perinatal outcome and to study the ability of the VAST to convert a false-positive NST to a reactive one.

MATERIALS AND METHODS

This prospective study was conducted on 100 antenatal cases having certain high-risk factors necessitating fetal monitoring in the Department of Obstetrics and Gynaecology, Government Medical College, Amritsar.

After taking a complete history of the patient with special reference to high-risk factors and doing a detailed clinical examination, the women having high-risk pregnancies were recruited for the study after taking informed consent. The risk factor, for which the patient was included, was noted. They underwent NST and vibroacoustic stimulation test in the third trimester of pregnancy. In the study, Hewlett Packard, Avalon Fetal Monitor (FM30) at paper speed of 1 cm/min was used for conducting NST and VAST. For giving vibroacoustic stimulation, an artificial larynx with sound intensity of 85 dB was used.

The NST was performed and recording of fetal heart rate and fetal movements were done. The patient was asked to press the event marker each time she perceived a fetal movement. The trace was designated as reactive, if there were two or more acceleration of ≥15 beats/min lasting for 15 s or more.

If the reactive pattern was not recorded within 20 min period, the fetus was stimulated with artificial larynx and the test continued for another 10 min period. If there was no reactivity in this extended period, only then the trace was deemed non-reactive.

If the NST and VAST were reactive, the tests were repeated weekly or biweekly depending on the risk factor present until the patient went into labor. If the test continued to be non-reactive, then the patient was subjected to further evaluation, i.e., color Doppler and biophysical profile.

The last NST and VAST (within 1 week of delivery) observations were correlated with outcome of pregnancy.

To assess perinatal outcome, the following parameters were taken into account:

- Evidence of fetal distress in labor (meconium stained-liquor)
- Five minutes Apgar score <7 was considered as abnormal
- Perinatal morbidity (neonatal intensive care units [NICU] admission)
- Perinatal mortality.

Analysis of Data

Data from the above-mentioned parameters were compiled and statistically analyzed for their significance. "*P*" < 0.05 and <0.01 were considered significant and highly significant, respectively. The relevance of the results in light of statistical analysis is displayed and discussed.

RESULTS

In the present study, 56% women who presented with high-risk factors were from the unbooked category. The majority of the pregnant women were from rural background (62%). The mean age of the study group was 25.01 years (SD 4.20). Mean gestational age was 38.5 weeks (SD 1.94). The distribution, i.e., the number of patients in the study as per the high-risk factors is shown in Figure 1. The hypertensive disorder of pregnancy comprises the most common high-risk factor in the study as shown below. X-axis shows the high-risk factors and Y-axis shows the number of patients.

The majority of the women having high-risk pregnancies were primigravida (57%). In this study, a total of 213 NSTs were done on 100 women. Two women had twin pregnancy. Hence, the interpretation of NST and VAST of both the fetuses was taken into account. The interpretation of the last NST was correlated with the perinatal outcome. Last NST was non-reactive in 39 (38.2%) women but after VAST, only 27 (26.5%) women have a non-reactive test. Thus, the incidence of false-positive rate of NST was reduced by about 31%. The majority of the women delivered within 24 h of last NST, of which 16% delivered within 6 h of last NST and 68% delivered between 6 and 24 h of test. Regarding mode of delivery, 45% women had normal vaginal delivery. About 53% women underwent caesarean section lower segment

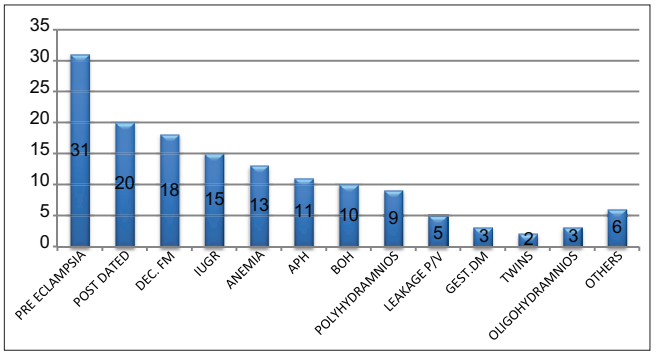

Figure 1: Distribution of patients according to high risk factors

cesarean sections (LSCS) and only 2% of the women had instrumental delivery.

Among the women who underwent LSCS, 39 had emergency section constituting 73.58% of the total LSCS done and only 14 had planned elective section. In our study, when NST was non-reactive, the incidence of emergency cesarean section (71.05%), ($P < 0.001$), was increased as shown in Table 1.

When VAST was also nonreactive, the incidence of emergency cesarean section (79.49%) ($P < 0.001$) was further increased as shown in Table 2.

In the present study, there were six perinatal deaths. The cause of perinatal mortality along with the results of NST and VAST and Apgar score is shown in Table 3.

In our study, when NST was nonreactive, the incidence of thick meconium stained liquor (58.97%) ($P < 0.001$), 5 min Apgar score <7 (33.33%), ($P < 0.001$) and perinatal morbidity ($P < 0.001$) (51.28%) in the form of NICU admissions, need for resuscitation was increased as shown in Table 4.

When VAST was also nonreactive, the incidence of thick meconium stained liquor (70.37%) ($P < 0.001$), 5 min Apgar score <7 (48.15%), and perinatal morbidity (70.37%) ($P < 0.001$) was further increased as shown in the Table 4.

Overall outcome is defined as abnormal outcome if at least one of the predictors of perinatal outcome (namely meconium stained liquor, Apgar score, NICU admission, and perinatal mortality) is abnormal.

The results show that a VAST scores over NST across the board as it is a better predictor of basic parameters of fetal and neonatal prognosis as shown in Table 5.

Table 1: Co-relation of non-stress test with mode of delivery

Mode of delivery	Reactive non-stress test		Non-reactive non-stress test		*P* value
	No.	%	No.	%	
Vaginal delivery	36	58.06	9	23.69	<0.001*
Instrumental	1	1.61	1	2.64	
Lower segment cesarean sections - Emergency	12	19.35	27	71.05	
Lower segment cesarean sections - Elective	13	20.97	1	2.63	
Total (*n*=100)	62	100	38	100	

*$P<0.001$; highly significant

Table 2: Corelation of vast with mode of delivery

Mode of delivery	VAST reactive		VAST nonreactive		*P* value
	No.	%	No.	%	
Vaginal delivery	40	54.05	5	19.23	<0.001*
Instrumental	1	1.35	1	3.85	
Lower segment cesarean sections - Emergency	8	20.51	31	79.49	
Lower segment cesarean sections - Elective	14	27.03	0	0	
Total (*n*=100)	74	100	26	100	

*$P<0.001$; highly significant. VAST: Vibroacoustic stimulation test

Table 3: Perinatal mortality and cause of mortality

S. No.	Case No.	Risk factor	Non-stress test	Vibroacoustic stimulation test	Mode of delivery	Apgar score		Outcome	Cause
						1 min	5 min		
1	8	PIH with ADF	NR	NR	LSCS-E	4	6	Baby died 48 h after birth	Severe IUGR
2	17	Post-term	NR	NR	LSCS-E	6	6	Baby died 3 days after birth	Meconium aspiration
3	18	Twins with abnormal Doppler	NR	NR	LSCS-E	0	0	Fresh still birth	Severe IUGR (discordant twins)
4	36	Abruption	R	R	LSCS-E	1	4	Baby died 3 days after birth	Perinatal asphyxia
5	80	Pre-eclampsia	NR	NR	LSCS-E	2	4	Baby died 4 h after birth	Prematurity and IUGR
6	83	Eclampsia	NR pre-term IUGR	NR	LSCS-E	1	6	Baby died 3 days after birth	Severe asphyxia

LSCS-E: Lower segment cesarean sections-E, IUGR: Intrauterine growth restriction

Table 4: comparison of NST and VAST in relation to perinatal outcome

Indicator of perinatal outcome	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
Meconium stained liquor					
NST	82.14	78.38	58.97	92.06	79.41
VAST	67.86	89.19	70.37	88.00	83.33
APGAR score at 5 min					
NST	81.25	69.77	33.33	95.24	71.57
VAST	81.25	83.27	48.15	96.00	83.33
NICU admission					
NST	71.43	74.32	51.28	87.30	73.53
VAST	67.86	89.19	70.37	88.00	83.33
Perinatal mortality					
NST	83.33	64.58	12.82	98.41	65.69
VAST	83.33	77.08	18.52	98.67	77.45

NST: Non-stress test, VAST: Vibroacoustic stimulation test

Table 5: Comparison of NST and VAST with overall outcome

Overall outcome	NST (%)	VAST (%)
Sensitivity	80.48	78.05
Specificity	83.61	95.08
Positive predictive value	76.74	91.43
Negative predictive value	87.93	86.57
Accuracy	82.35	88.24

NST: Non-stress test, VAST: Vibroacoustic stimulation test

DISCUSSION

A screening test should have a good sensitivity to minimize the number of false-negative results. However, good specificity is also needed to reduce false positives, leading to undue anxiety and the need for further investigations. In this prospective clinical study, the women were followed up until delivery. The perinatal outcome was assessed taking into account the various parameters.

The most common indication of antenatal monitoring was hypertensive disorders accounting for 21.23% of the women. The present study is comparable in this regard to the studies done by TM Batcha and Goonewardene[5] and Xi *et al.*[6] which had similar percentage of the women with hypertensive disorders.

Last NST was reactive in 63 cases (61.8%) and was nonreactive in 39 cases (38.2%). After VAST, 12 cases who were initially nonreactive became reactive. Only 27 cases remained nonreactive after VAST. Thus VAST reduced the number of false-positive results by 31%. The results were almost similar to the study conducted by Goonewardene and Hanwellage,[7] in which VAST reduced the false-positive results by 31%.

Sambarey and Bilagi[8] in their study concluded that VAST is easy to perform adjunct to NST with higher specificity, sensitivity, positive and negative predictive value in predicting perinatal outcome that can be compared to our study.

Gupta *et al.*[9] found that the VAST had higher specificity, sensitivity, positive and negative predictive, value in predicting perinatal outcome.

However, in the study conducted by Batcha and Goonewardene,[5] VAST reduced the number of false-positive results by 48% which was higher than the present study. This may be explained by the fact that every study had women with different risk factors leading to this wide variation in the false-positive rate of NST.

Out of 27 cases which remained nonreactive after VAST, 31 (79.49%) underwent emergency LSCS (1 LSCS in patient with twin gestation), of which 14 babies (70% had Apgar score <7), one patient had forceps delivery for fetal bradycardia and only five had normal vaginal delivery.

Of the 13 cases which became reactive after initial non-reactive NST, only three women underwent emergency LSCS.

One of the objectives of the present study was to evaluate the overall usefulness of NST in relation to overall perinatal outcome. The sensitivity and positive predictive value of the present study is higher than that in similar studies. This may be explained by the fact that in the present study, four parameters of perinatal outcome, i.e. meconium stained liquor, Apgar Score, NICU admission, and perinatal mortality were taken into account whereas in other studies either two or three of the above mentioned parameters were considered. At the same time, the specificity and negative predictive value of NST in the present study is close to that demonstrated by above studies.

The sensitivity of VAST in the present study is comparable to the study conducted by Saracoglu *et al.*[10] but is much

higher than that demonstrated by studies of Khatun,[11] Xi *et al*.[6] The positive predictive value of the present study is much higher and negative predictive value is comparatively less when compared to other studies. As said earlier, this may be due to the difference in number of parameters of perinatal outcome which were taken into account. Thus, the addition of VAST to NST prevents undue intervention by decreasing the false-positive rate of NST.

CONCLUSION

Our study is largely a clinical study where an endeavor has been carried out to critically assess the reliability of NST and vibroacoustic stimulation test for decreasing the fetal morbidity and mortality by appropriate intervention and treatment. From the study, it can be concluded that antenatal NST along with VAST provokes a response in non-hypoxic fetuses who have non-reactive NST, thus significantly reducing the false-positive (non-reactive) NST. It has a good sensitivity, accuracy, and negative predictive value in detecting fetal hypoxia. The VAST complement NST but cannot replace it as it is thought that in some cases it may provoke response in hypoxic fetus who is trying to conserve energy by not moving. They cannot predict an acute asphyxia event. However, it can serve as a useful cost effective and reliable preliminary screening procedure for antepartum fetal monitoring where resources for more sophisticated investigations are not freely available.

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Original Article

Effects of Cardiopulmonary Bypass on Thyroid Function and Need of Prophylactic Low-Dose Thyroxine for Post-operative Cardiac Surgery Patient

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Abstract

Introduction: Cardiopulmonary bypass (CPB) is associated with well-described changes in thyroid hormone levels, consistent with what is described as the euthyroid sick syndrome.

Aim: This study aims to evaluate thyroid hormone changes and their association with post-operative care in low-risk patients undergoing cardiac surgery with CPB.

Materials and Methods: Fifty patients with euthyroid were included; no one received drugs with a known influence on thyroid status at the time of the operation. Eighteen of the patients had coronary artery bypass surgery, 29 had a single valve replaced, and 3 had both valve replacement. Blood samples were collected 6 h after surgery and on the 3rd postoperative days (POD).

Results: Thyroid-stimulating hormone (TSH) levels were raised in 10 patients and free T3 level reduced in 14 patients in the early post-operative period and TSH raised in 4 patients and free T3 reduced in 6 patients during the 3rd POD. Number of the cases with normal TSH is 36, free thyroxine [T4] remained within the normal range in all patients throughout the study.

Conclusion: There is an advantage of prophylactic administration low dose of thyroxine to all cardiac patients, especially female patient who undergoes surgery to improve the general condition of the post-cardiac surgery patients.

Key words: Cardiopulmonary bypass, Euthyroid sick syndrome, Thyroid hormone

INTRODUCTION

Cardiopulmonary bypass (CPB) is associated with well-described changes in thyroid hormone levels, consistent with what is described as the euthyroid sick syndrome.[1-3] The syndrome is characterized by depressed total (TT3) and free (fT3) triiodothyronine levels despite normal concentrations of thyroid-stimulating hormone (TSH) and total (TT4) and free (fT4) thyroxine. Decreased deiodination of T4 to its active compound T3 has been implicated as the central pathophysiologic mechanism, while there is a concomitant rise in the levels of the inactive compound reverse T3.[4] After CPB, some patients have low cardiac output, responding poorly to conventional inotropic stimulation. Recently, it has been claimed that acutely administered triiodothyronine (T3) may benefit cardiac performance by several mechanisms independent of *de novo* protein synthesis. It seems likely that a severely depressed thyroid state at the end of the operation might contribute to acute heart failure. A low T3 syndrome is often seen in patients with serious diseases and those having an operation.[5-8] Very sparse and indeed, conflicting results concerning thyroid hormonal state have been presented in this specific category of patients undergoing CPB.[3,8-11]

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The possible hemodynamic consequences of the respective findings have been discussed only sparsely.

Aim

This study aims to evaluate thyroid hormone changes and their association with post-operative care in low-risk patients undergoing cardiac surgery with CPB and to assess whether free thyroid hormone concentrations are decreased to a degree indicating the need for T3 substitution to restore cardiac performance perioperatively.

MATERIALS AND METHODS

Fifty preoperatively euthyroid patients, 28 men and 22 women, ranging in age from 28 to 64 years (median 48 years), were included in the study after giving written and informed consent. No one received drugs with a known influence on thyroid status at the time of the operation. Eighteen of the patients had coronary artery bypass surgery, 29 had a single valve replaced, and 3 had both valve replacement. The priming solution for the heart-lung machine was composed of 2000 ml Ringer's lactate solution and heparin (5000 IE). Heparin was further administered to the patients before CPB, according to the activated clotting time (about 300 IE/kg body weight). When required during perfusion, bicarbonate was added to the heart-lung machine. Aortic cross-clamp time ranged from 41 to 115 min (62 ± 21 min). Local and universal cooling to 28°C was performed, and blood cardioplegic solution was used and by lowering the temperature in the heart-lung circuit. The flow was kept at 60 ml/kg/min, and blood pressure ranged from 40 to 60 mmHg (by our standard non-pulsatile system). All patients received a single unit of blood and two units of plasma at the end of the procedure. Dobutamine and adrenaline infusion (3 ~g/kg/min) was started at the end of the operation in all patients. Patients have treated with thyroxine 50 µg/day. Blood samples were collected 6 h after surgery and on the 3rd postoperative days (POD).

RESULTS

TSH levels were raised in 10 patients (female 6 and male 4) and free T3 level reduced in 14 patients (female 8 and male 6) in the early post-operative period [Figure 1] and TSH raised in 4 patients (female 1 and male 3) and free T3 reduced in 6 patients (female 3 and male 3) during the 3rd POD [Figure 2]. The number of the case with normal TSH is 36, free thyroxine [T4] remained within the normal range in all patients throughout the study. Total patients with abnormal thyroid levels and treated with tablet thyroxine 50 µg/day through oral are 30 [Figure 3]. Of the 30 patients, 4 develop low cardiac output and high inotropic supports and 3 of them improved from low cardiac output after administration thyroxine.

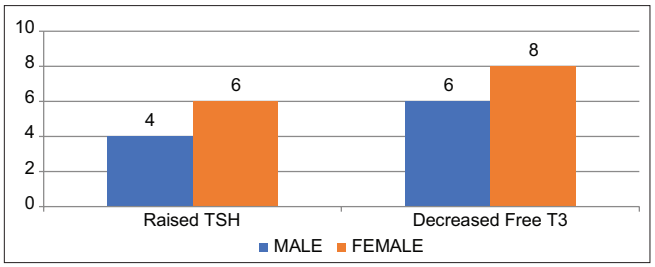

Figure 1: Thyroid status on postoperative days 0

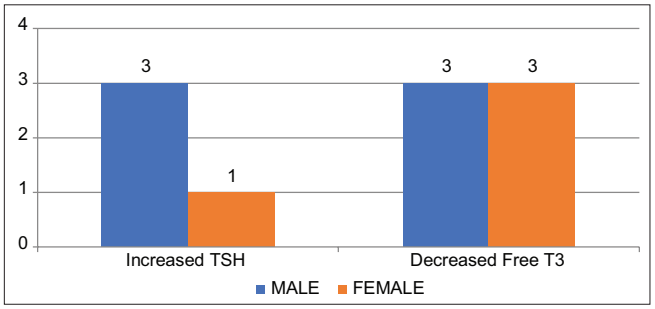

Figure 2: Thyroid status on postoperative days 3

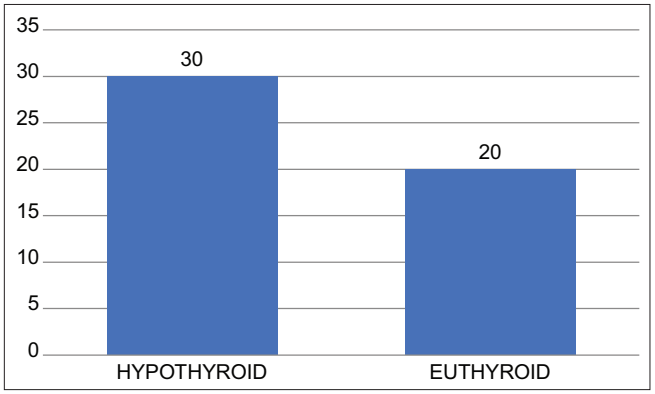

Figure 3: Total patient distribution

DISCUSSION

The study confirms the previous findings of significant thyroid hormone changes after CPB.[1-3,12] The results shows that, a progressive decline in fT3 levels in the early post-operative phase, despite preserved fT4 level. These thyroid hormone changes are consistent with what is known as a euthyroid sick syndrome, which is also characterized by raised levels of the inactive compound reverse T3 (rT3). The previous studies have confirmed the presence of elevated rT3 during and after CPB.

The mechanism responsible for the development of euthyroid sick syndrome involves the regulation of two deiodinases that control T4 metabolism.[2,12] Thyroxine is

3 times less potent than T3 and is entirely secreted by the thyroid gland. Approximately 40% of T4 is deiodinated to the active compound, T3, in the peripheral tissues. Two deiodinases are involved in this process: 5'deiodinase converts T4 to T3 and rT3 to 3,3'T2, and 5-deiodinase converts T4 to rT3 and T3 to 3,3'T2. It is thought that reduced activity of 5'deiodinase results in decreased formation of T3, allowing increased conversion of T4 to rT3 by 5-deiodinase. Moreover, there is concomitantly reduced metabolism of rT3 to 3,3'T2, which contributes to the raised levels of rT3.[4]

There is an obvious difference between euthyroid sick state and primary hypothyroidism, in that the latter is usually characterized by decreased levels of T4 and fT4, raised concentrations of TSH and normal levels of T3 and fT3. Furthermore, primary hypothyroidism is a chronic illness, while euthyroid sick syndrome has been observed as an acute response of the thyroid axis to a variety of insults. These include general surgical operations,[8,9] acute and chronic systemic illnesses, fasting, and major trauma.[13]

It is interesting to note that post-surgical euthyroid sick syndrome occurs in the early post-operative phase, at a time, when the patient is in a catabolic state with increased whole-body oxygen consumption and global oxygen extraction fraction. Similarly, Aun *et al.*[13] observed that in trauma patients, there was a reduction in T3 levels despite increased oxygen extraction by muscular tissue. All this would suggest that the euthyroid sick syndrome represents an adaptive mechanism of the body in an attempt to reduce catabolism,[2,4] rather than a true hypothyroid state.

The documentation of low T3 levels after CPB led to a number of studies that investigated the possible beneficial effect of perioperative T3 administration. Several randomized clinical studies have been undertaken.[5-7,14,15] Despite methodological differences between individual studies, the majority of evidence appears to demonstrate improved hemodynamic performance with T3 administration but no difference in clinical outcome [Table 1].

Hemodilution by the CPB circuit priming volume is unlikely to be a major factor affecting thyroid hormone changes postoperatively since albumin concentrations return to normal by 2 h after CPB, while changes in thyroid hormone levels persist for several days.[1,2,11] Moreover, thyroid hormone changes move in opposite directions during and after CPB, suggesting that despite their structural similarity, they are affected by hemodilution in a different way. Even after correcting for hemodilution, Bremner *et al.* reported thyroid hormone changes of a euthyroid sick response.[12] All this indicates that hemodilution is not a major factor affecting thyroid hormone levels.[2]

The lack of pulsatile flow is perhaps the only factor that has been shown to significantly affect the magnitude of thyroid hormone changes during CPB. Buket *et al.*[3] examined thyroid hormone changes in 30 low-risk patients undergoing CABG with hypothermic (26–30°C) CPB using pulsatile versus non-pulsatile flow. The authors observed the development of euthyroid sick syndrome in both groups. However, total T3 and fT3 concentrations declined significantly less with pulsatile CPB. Immediately relevant to these findings is the pioneering work by Taylor and Bremner *et al.* in the late 1970s. Their group investigated the hypothalamic-pituitary-thyroid axis function during CPB.[12] They found a blunted TSH response to thyrotropin-releasing hormone (TRH) during both the early and late phases of CPB, in contrast to heparinized and non-heparinized patients undergoing major surgery.[12] This led them to repeat the study of TRH administration in 20 patients undergoing normothermic pulsatile versus non-pulsatile CPB. There was a marked difference between the groups, with non-pulsatile patients

Table 1: Summary of T3 administration studies

Author, year	Study design	N	T3 protocol	Main findings
Mullis-Jansson *et al.*, 1999[7]	DB, randomized placebo controlled	170	1 mg/kg bolus+1 mg/kg over 6 h	↓ inotropic use, ↓ myocardial ischemia, ↓ pacemaker dependence, ↓ LV mechanical assistance
Bennett-Guerrero *et al.*, 1996[6]	DB, randomized placebo-controlled positive control (dopamine)	211	0.8 mg/kg bolus+0.12 mg/kg h for 6 h	No differences in hemodynamics or inotropic use
Klemperer *et al.*, 1995[5]	DB, randomized placebo controlled	142	0.8 mg/kg bolus+0.113 mg/kg h for 6 h	↑ CO, ↓ SVR, no difference in outcome or inotropic use
Teiger *et al.*, 1993[15]	DB, randomized placebo controlled	20	Total 0.55 mg/kg in 5 boluses over 20 h	No differences
Novitzky *et al.*, 1989[14]	DB, randomized placebo-controlled LVEF < 30%	24	Total 0.275 mg/kg in 4 boluses over 8 h	↓ inotropic and diuretic requirements, no difference in outcome
	DB, randomized placebo-controlled LVEF > 40%	24	Total 0.55 mg/kg in 5 boluses over 20 h	↑ CO, ↓ SVR, ↓ PVR, no difference in outcome, inotropes, diuretics

*DB: Double blind, LV: Left ventricular, CO: Cardiac output, SVR: Systemic vascular resistance, LVEF: Left ventricular ejection fraction, PVR: Pulmonary vascular resistance

demonstrating the previously reported subnormal response to TRH, while pulsatile CPB resulted in a normal pituitary response to TRH in nine out of 10 patients. These studies provide strong evidence that pulsatile flow during CPB is a major factor contributing to the preservation of a euthyroid hormonal environment.

CONCLUSION

This study has demonstrated the presence of thyroid hormone changes after CPB and consistent with the post-surgical euthyroid sick syndrome. Thyroid function is the result of all effects acting sometimes on the direct production of hormones by the gland and, on the hypothalamic-pituitary-thyroid axis, decreasing TSH, and sometimes on peripheral thyroid hormones metabolism. These manifestations, if transient, determine the presence of euthyroid disease, characterized by reversible but sometimes prolonged thyroid function disorders, particularly in females with extremely relevant post-operative effects such as low cardiac output and high inotropic supports, which are improved well after administration of thyroxine. Through this study, we concluded that there is an advantage of prophylactic administration low dose of thyroxine to all cardiac patients, especially female patient who undergoes surgery to improve the general condition of the post-cardiac surgery patients.

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