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CASE REPORTS

Original Article

Snake Bite Induced Coagulopathy: A Study of Clinical Profile and Predictors of Poor Outcome

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Abstract

Background: Snake bite poisoning is known to man since antiquity. Snake bite can result in local and systemic complications. Major systemic complications include acute renal failure, neurologic abnormalities requiring ventilator support and disseminated intravascular coagulation. Disseminated intravascular coagulation can result in serious life threatening systemic complications like hemorrhage, infarction and even death if the treatment is delayed. In tropical countries where snake bite is a serious problem there is very little reliable data on hematological problems of snake envenomation because of inadequate documentation.

Aims: The present study was undertaken to study the clinical profile of the snake bite patients who develop coagulopathy and to study the role of coagulation markers to evaluate the morbidity and mortality of snake bite victims.

Material and Methods: Fifty patients consecutively admitted with history of snakebite were studied from May 2012 to November 2013 in a Kempegowda institute of medical sciences (KIMS), Bangalore, Karnataka, India. The patients were classified into the normal and coagulopathy group based on clinical symptoms and the hematological parameters.

Results: In our study patients who had coagulopathy had prolonged hospital stay and requirement of more blood products transfusion causing increased morbidity. 24 patients had platelets less than 1 lakh and approximately hospitalized for 28 days and they received 102 platelet units. INR was more than 1.5 in 24 patients and hospitalized for 25 days and they received 136 fresh frozen plasma. The case-fatality rate in our study was 4%.

Conclusion: Combined clinical and laboratory parameter evaluation needed to identify the coagulopathy very early to reduce the hospital stay and mortality.

Keywords: Coagulopathy, Snake bite

INTRODUCTION

Bites by snakes represent an important health problem in the tropical world including India. The true incidence of snakebites is difficult to assess and often is underreported. There are approximately 1.2 million and 5.5 million snakebites worldwide each year, with 421,000-1,841,000 envenomations and 20,000-94,000 deaths.[1] Awareness and educating the farmers and labourers is needed to prevent the snake bites.[2]

In tropical countries where snake bite is a serious problem there is very little reliable data because of inadequate documentation. At present very few clinical studies are available on snake envenomation especially on haematological problems of snake envenomation.[3] Many of the toxins in snake venom interact with clotting mechanism and fibrinolytic system and causes coagulopathy. The occurrence of local and systemic snake bite related symptoms is linked to toxins in snake venom.

Snake bite can result in local and systemic complications. Major systemic complications include acute renal failure, neurologic abnormalities requiring ventilator support and disseminated intravascular coagulation.[4,5]

Disseminated intravascular coagulation can result in serious life threatening systemic complications like hemorrhage, infarction and even death if the treatment is delayed.[6]

This study was conducted to evaluate the clinical and laboratory parameters of coagulopathy and evaluation of morbidity and mortality in them.

OBJECTIVES

The primary objective of this study was to describe the clinical profile of the snake bite patients who develop coagulopathy.

The secondary objective is to study the role of coagulation markers to evaluate the morbidity and mortality of snake bite victims

METHODOLOGY

Study population

Fifty patients consecutively admitted with history of snakebite were included in the study after obtaining ethical committee clearance as well as informed consent from all patients. All patients were evaluated with a detailed history and clinical examination. This study was done between May 2012 to November 2013 in a Kempegowda institute of medical sciences (KIMS), Bangalore, Karnataka, India.

Inclusion Criteria

Patients with history of snake bite with signs of envenomation were included in the study after obtaining ethical committee clearance as well as informed consent from all patients.

Exclusion Criteria

Patients with pre-existing coagulopathy, on anticoagulants and antiplatelet drugs, with history of renal diseases. Patients with risk factors like diabetes, hypertension, connective tissue diseases, chronic infection.

Data Collection

Data was collected in a proforma which includes detailed history, clinical examination and appropriate investigations.

Following history and clinical features were found out:

- Snake bite site and snake bite time
- Lapse of time (in hours) after snake bite
- Weather tourniquet applied or not
- Identification of snake and fang marks
- Local swelling at the site of bite and increasing the swelling with time
- Bleeding from the bite sites and bleeding from gums
- History of passing black or brown urine to rule out intravascular hemolysis
- Symptoms like nausea, vomiting, fever, breathlessness and decreased urine output
- Investigations include Haemoglobin (Hb), Total count, Platelet count, Bleeding time, Clotting time, Whole blood clotting test (WBCT), Prothrombin time (PT), Activated partial thromboplatin time (APTT), International normalized ratio (INR), Fibrin degradation products (FDP), Creatine kinase, Blood urea, Serum creatinine, Serum bilirubin, Serum potassium levels
- Number of anti-snake venom serum (AVS) given.

The subjects who were classified into the normal and coagulopathy groups based on clinical symptoms and the hematological parameters like prothrombin time (PT), INR, the fibrin degradation products (FDPs), platelet count tests and whole blood clotting time.[7,8]

RESULTS

Age, gender, the site of bite, tourniquet application, identification snake

58% of the patients aged above 40 years whereas 38% between 18 to 40 years. There were 36 males (72%) and 14 females (28%) out of 50 patients studied. Majority of the snake bites were in lower limbs: Right leg 17 patients (34%), left leg 19 patients (38%) and each right and left upper limb has 7 patients (14%). Tourniquet was applied in just 5 patients (10%). Out of 50 patients studied 35 patients had Viper bite, 6 patients had Cobra bite, 2 patients had Krait bite and in 7 patients snake was not identified.

Symptoms of snake bite patients, Number of ASV vials given and Haemodynamic parameters of the patients

33 patients (66%) had fang marks, 20 patients had bleeding from the bite site (40%), 7 patients had bleeding gums (14%), 20 patients had hematuria (40%), swelling and inflammation of the bite area was present in 45 patients (90%), 17 patients had breathlessness (34%). More than 20 ASV vials were given in 26 patients (52%), less than 10 were given in 11 patients (22%) and 10 to 20 vials were given in 13 patients. 31 patients (62%) had tachycardia (>100 bpm) and 18 patients (36%) had systolic blood pressure less than 100 mmHg at the time of presentation. 13 patients (26%) had Hemoglobin less than 10 gm% and 32 patients (64%) had total leucocyte count more than 11,000. 24 patients (48%) had platelet count less than 1,00,000. 28 patients (56%) had prothrombin time more than 15 seconds. 31 patients (62%) had activated partial thromboplastin time more than 30 seconds. 24 patients (48%) had INR more than 1.5. FDP was positive in 22 patients (44%). WBCT was more than 20 minutes in 30 patients.

Table 1: Hematological parameters in patients with coagulopathy

Hematological parameters	Number of patients	%	Number of hospitalization days	Supportive treatment
Hemoglobin (Hb in gms)				Number of packed red cells
<10.0%	13	26	22	38
Total count				
>11000	32	64	20	
Platelet count				Number of platelets transfusion
<100000	24	48	28	102
Prothrombin time (in secs)				Number of FFP transfusion
>15 sec	28	56	25	136
Activated partial thromboplastin time (APTT in secs)				
>30 sec	31	62	22	
WBCT (Whole blood clotting time) in minutes of patients studied				Number of ASV vials
>20 min	30	60	27	488

Table 2: INR vs number of hospitalization days

INR	Number of patients	Mean±SD	Number of hospitalization days	*P* value
<1.5	26	9.58±6.319	12	0.01
>1.5	24	14.75±7.285	25	0.01

Table 3: End results of snake bite patients studied

End results	No of patients	%
Discharged	47	94
Death	2	4
Chronic kidney disease	1	2

DISCUSSION

Snake venom consist of various enzymes like zinc metalloproteinase haemorrhagins and procoagulant enzymes. Zinc metalloproteinase haemorrhagins lead to vascular endothelium damage. Procoagulant enzymes activate factor X and prothrombin. The toxins in snake venom interact with clotting mechanism and fibrinolytic system and causes "consumption coagulopathy".[3]

In our study vipers constituted for 70% of the total snake bites. We have noticed viper bite causes rapid progression of swelling at the bite site and systemically causes coagulopathy. In our study 6 patients had Cobra bite and 2 patients had Krait bite associated with neurotoxicity manifesting as breathlessness which were managed conservatively without ventilator support.

In the present study, maximum incidence of snake bite was found above the age of 40 years (58%).72% of the snake bite occurred in males attributed mainly to their outdoor activity compared to females.[9] Most of the snake bites were haematotoxic (Viper bite), constituting to 70%. Cobra in 12%, Krait in 4% and snake was not able to identify in 14% of the bites. Snake bite victims had various clinical manifestations; 66% of the victims had fang marks, 90% had swelling of the bite area, 60% had muscle pain, 40% had bleeding from the site and hematuria, 50% had reduced urine output, 34% had breathlessness, 26% had vomiting.[10]

In our study patients who had coagulopathy had prolonged hospital stay and requirement of more blood products transfusion causing increased morbidity. 13 patients had haemoglobin less than 10 g/dl and approximately hospitalized for 22 days and they received 38 packed red cells. 24 patients had platelets less than 1 lakh and approximately hospitalized for 28 days and they received 102 platelet units. INR was more than 1.5 in 24 patients and hospitalized for 25 days and they received 136 fresh frozen plasma. Whole blood clotting time was prolonged more than 20 minutes in 30 patients and approximately hospitalized for 27 days and they received 488 ASV vials.

The case-fatality rate in our study was 4%. Death rate due to snake bites in developing countries like India is more than the developed countries.[10]

Mortality in viper bites commonly secondary to hypovolemia, intravascular haemolysis, a syndrome resembling disseminated intravascular coagulation or venom-induced nephrotoxicity.[11]

The combined clinical manifestations (like gum bleeding and hematuria) and laboratory parameters (like low hemoglobin, thrombocytopenia, raised INR, prolonged WBCT) should be evaluated to identify the coagulopathy very early as it prolongs the hospital stay leading increased morbidity and mortality. These manifestations require prompt treatment to reduce the morbidity and mortality.

CONCLUSION

Haematological manifestations are very common in snake bite. Combined clinical and laboratory parameter evaluation

needed to identify the coagulopathy very early to reduce the hospital stay and mortality.

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Original Article

Role of Nasal Endoscopy in Sinonasal Diseases

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Abstract

Introduction: Nasal endoscopy allows detailed and complete evaluation of intranasal anatomy and identification of pathology that was impossible to see using standard techniques with headlight or head mirror. The following study was undertaken in order to ascertain the efficacy of endoscopy in diagnosing a spectrum of nasal and nasopharyngeal pathology which otherwise remain unrevealed clinically.

Aims and Objectives: To evaluate sinunasal diseases with the help of nasal endoscopy. To study the efficacy of nasal endoscopy in diagnosing nasal pathology over clinical examination. To define medical and surgical (FESS) management according to the type of nasal pathology. To define applications of nasal endoscopy (biopsy, swab, epistaxis control, foreign body removal, rhinolith removal, follow up).

Materials and Methods: Total 100 patients were studied. Patients came with complaints of nasal blocking, nasal discharge, mass in nasal cavity, bleeding etc., included in study. Pre endoscopic assessment was carried out like history, examination, investigation. Endoscopic was done after consent under necessary anaesthesia. Endoscopy was done using 0 degree and 30 degree endoscope with 3 standard passes.

Result: Total 100 patients were studied. Male to female ratio was 1.8:1. Out of 100 patients maximum number of patients had chronic sinusitis on nasal endoscopy examination (22); followed by nasal polyp (27) and deviated nasal septum and epistaxis (10). Nasal endoscopy was an excellent diagnostic aid in condition like epistaxis, nasal mass**,** nasal obstruction, foreign body, nasopharyngeal tumour.

Conclusion: Diagnostic nasal endoscopy offers high diagnostic accuracy in patient with sinonasal complaints. Diagnostic nasal endoscopy is gold standard tool in patient having sinonasal complaints. It has High accuracy due to vision control, has less bleeding, minimal complication, and early post operative recovery. It's a good tool for diagnosing anatomical variation.

Keywords: Anatomical variation, Chronic sinusitis, Nasal endoscopy, Nasal passes

INTRODUCTION

In 1901 Hirschmann[1] first used the modified cystoscope to examine middle meatus. Based on the experience and teaching of Messerklinger, Stammberger and Kennedy[2-4] the diagnosis and treatment of inflammatory sinus disease continue to evolved. Nasal endoscopy allows detailed and complete evaluation of intranasal anatomy and identification of pathology that is impossible to see using standard techniques with headlight or head mirror. With the endoscope, the surgeon gains capacity for precise anatomy identification and angled, illuminated, magnified viewing of the internal nose preoperative, intraoperatively, and postoperatively. As an added benefit, an attached camera can provide a photographic demonstration to the patient or create documentation for the permanent record.[5] Recently combination of diagnostic endoscopy and imaging study has become the corner stone in the evaluation of the paranasal sinus diseases. This is the basis of the new concept of the functional endoscopic sinus surgery (FESS).

The following study was undertaken in order to ascertain the efficacy of endoscopy in diagnosing a spectrum of nasal

and nasopharyngeal pathology which otherwise remain unrevealed clinically.

AIMS AND OBJECTIVES

- To evaluate sinonasal diseases with the help of nasal endoscopy.
- To study efficacy of nasal endoscopy in diagnosing nasal pathology over clinical examination.
- To define medical and surgical (FESS) management according to type of nasal pathology.
- To define applications of nasal endoscopy (biopsy, swab, epistaxis control, foreign body removal, rhinolith removal, follow up).

MATERIALS AND METHODS

It was a prospective study. The study was conducted at Indira Gandhi Govt. Medical College during a period from August 2009 to December 2013. Total 100 patients were studied. Inclusion criteria were patient presenting with nasal complaints like nasal blockage, running nose, bleeding from nose, nasal mass, foul breath, foreign body in nose, and patient above 10 years of age. Exclusion criteria were patient with acute infection of nose and paranasal sinuses, and age less than 10 years. Local ethics committee approval was acquired for this study.

A detailed history and ENT examination was done. Written and informed consent was taken before the diagnostic nasal endoscopy. 0 degree, 30 degree rigid nasal endoscope were used (4 mm). All diagnostic nasal endoscopies were performed under local or general anaesthesia. Nasal cavity was packed with patty of 4% Xylocaine with adrenaline (1:1000) or xylomethazoline/oxymethazoline. A complete examination was successfully accomplished in an organized manner with three mentioned nasal passes of the endoscopy. The findings of nasal endoscopy were recorded in the proforma. Various endoscopic assisted procedures and surgeries were done as and if required. Patients were followed up after medical or surgical management at intervals of 1 week, 1 month, 3 months, and 6 months.

RESULTS

Total 100 patients were studied. The age ranged from 11 years to 80 years. Maximum patients were in 31-40 years of age group, which contribute 26% of total patients. In study male preponderance was 65% and female was 35%, Male to Female ratio was 1.8:1.

In study most common complaint was nasal discharge seen in 32 patients (32%), followed by nasal obstruction in 24 (24%), while least common complaints was foreign body in nose 3 (3%). Many patients came with multiple complaints at a time for particular pathology, most common symptom with which patients presented considered as a primary complaints (Table 1).

Most common finding on anterior rhinoscopy was nasal discharge seen in 46 patients, followed by deviated nasal septum, nasal polyp and inferior turbinate hypertrophy seen in 21 patients. Least common finding was synaechia in 1 patient. Most common finding on nasal endoscopy was middle meatus discharge seen in 40 patients, followed by polyp in 30 patients, followed by Inferior turbinate hypertrophy in 26 cases.

Most common anatomical variation seen in nasal endoscopy was spur it was seen in 10 patients, followed by concha bullosa in 7 patients. Anatomical variation was most commonly associated with chronic sinusitis; Out of 22 patients of chronic sinusitis anatomical variation was seen in 16 patients (72.72%) (Table 2).

Patients were grouped on basis of presenting chief primary complaints and studied.

Patients with Nasal Discharge

32 patients had primary complaint as nasal discharge. Out of 32 patients, 8 patients had ethmoidal polyposis, 15 patients of chronic sinusitis, Antrochoanal polyp seen in 3 patients, 2 patients had deviated nasal septum, Allergic rhinitis in 3 patients. One patient on endoscopy had clear watery fluid discharge from frontal resses. CT showed defect in cribrifom plate and final diagnosis of CSF rhinorrhea was confirmed.

Table 1: Presenting primary complaints of patients

Symptoms	No. of patients	Percentage
Nasal discharge	32	32%
Nasal obstruction	24	24%
Nasal bleeding	15	15%
Nasal mass	15	15%
Foul breath	6	6%
Olfactory disturbance	5	5%
Foreign body	3	3%

Table 2: Anatomical variation

Anatomical variants on nasal endoscopy	No. of patients
Concha bullosa	7
Bulla ethmoidlis	4
Paradoxical turbinate	6
Accessory ostea	1
Spur	10
Total	28

Patients with Nasal Obstruction

24 patients had primary complaint of nasal obstruction. Out of 24, 8 patients had deviated nasal septum. 2 patients were of maxillary malignancy, the biopsy was taken from the mass endoscopically. 3 patients of nasal obstruction had ethmoidal polyposis on nasal endoscopy which was not seen on anterior rhinoscopy. 4 patients had chronic sinusitis. Antrochoanal polyp was seen in 3 patients and 3 had inverted papilloma, patients with inverted papilloma showed polypoidal mass on anterior rhinoscopy, nasal endoscopy was done and biopsy was taken for histopathological examination. One patient had synaechia in right nostril with history of traumatic epistaxis in past, routine anterior rhinoscopy examination not showed any synaechia. These patient underwent endoscopic release of synaechia.

Patients with Nasal Bleeding

15 patients presented as nasal bleed. 6 patients had epistaxis in woodruffs area; in these patients anterior rhinoscopy examination was normal. These patients were managed by endoscopic cauterization. 4 patients had bleeding and congestion in Little's area on anterior rhinoscopy. Nasal endoscopy was done to find out other bleeding site and status of nasal cavity. These patients were also managed by endoscopic cauterization. 2 patients of nasal Angiofibroma presented with history of nasal bleed. Nasal endoscopy was done to locate the size, extension of mass and site of bleeding in operation theatre only with preparation for general anaesthesia if needed, nasal endoscopy was done as non touch technique to avoid epistaxis. Rest 3 patients showed mass in nasal cavity. Endoscopic biopsy was done and histopathological examination report suggestive of squamous cell carcinoma, haemangioma and rhinosporiodosis in each.

Patients with Olfactory Disturbances

5 patients had olfactory disturbance. All these patients were case of atrophic rhinitis. Out of these 5, 1 patient had history of Hansen's disease; other 2 had history of nasal myasis. Diagnostic nasal endoscopy revealed exact pathology; crust was present in all of them. On nasal endoscopy of these, 2 patients had bony and 1 show cartilage perforation over septum. Atrophic rhinitis patients were managed by counseling, nasal douches and regular endoscopic removal of crust.

Patient with Nasal Mass

15 patients with nasal mass were included in study. 4 patients were of ethmoidal polyposis, 6 had antrochoanal polyp, while chronic sinusitis feature was seen in 2 patients. In 2 patients with history of nasal mass endoscopic biopsy was taken and histopathological examination report was suggestive of haemangioma and rhinosporiodosis respectively. 1 patient who presented with nasal mass had Nasal Angiofibroma.

Patient with Foul Breath

6 patients present with such complaint. 1 patient had normal clinical finding, and show foreign body in right nasal cavity on endoscopy and was removed with the help of endoscope. 1 had chronic sinusitis, while 4 patients of foul breath had atrophic rhinitis.

Patient with Foreign Body Nose

3 patients presented with foreign body in nose. In 2 patients there was mucoid discharge in right nasal cavity and 1 had normal clinical finding. It was only by nasal endoscopy that foreign body was identified and removed. In 1 patient foreign body was present posteriorly in nasal cavity.

In this study, out of 100 patients 39 patients had no pathology on routine clinical examination related to particular diseases which was further confirmed after doing nasal endoscopy.

Out of 100 patients maximum number of patients had chronic sinusitis on nasal endoscopy examination (22); followed by nasal polyp (27) and deviated nasal septum and epistaxis (10) (Table 3).

DISCUSSION

The development of modern rigid nasal endoscopy represents a major advance in rhinologic diagnostic capability. The study conducted by Aminnu Bakari et al[6] and Levine et al.[7] had maximum number of patients in between 31 to 40 years with mean age 33.3 and 35.6 respectively. In our study majority of patients was in the age group of 31 to 40 years with total 26 cases (mean 39.1). In the present study

Table 3: Diagnosis of patient on nasal endoscopy

Diagnosis	No. of patients
Chronic sinusitis	22
Ethmoidal polyposis	15
Antrochoanal polyp	12
Deviated nasal septum	10
Epistaxis	10
Atrophic rhinitis	9
Rhinolith	4
Carcinoma maxilla	3
Inverted papilloma	3
Angiofibroma	3
Allergic rhinitis	3
Hemangioma	2
Rhinosporiodosis	2
CSF rhinorrhea	1
Nasal synaechia	1
Total	100

65 patients were male while 35 patients were female with male to female ratio was 1.8:1. In the study conducted by Kirtane et al[8] there were 48 (61.5%) males and 30 (38.4%) females and male to female ratio was 1.6:1. Abtin Tabaee[9] had 39 (63.9%) male and 22 (36%) female with ratio 1.7:1 in his study. Similarly study conducted by Aminnu Bakari et al.[6] showed 42 (55.2%) male and 34 (44.7%) female and had ratio 1.2:1. In the study conducted by Kirtane et al.[8] the commonest complaint was nasal discharge seen in 61% patients, followed by nasal obstruction in 59% patients. In the study conducted by Aminnu Bakari et al[6] the nasal discharge (97.4%) was the most common presenting complaints followed by nasal obstruction (94.7%). Out of 22 patients of sinusitis, 16 (72.72%) patients had associated anatomical variations on diagnostic nasal endoscopy. This was well in agreement with the study done by Lolyd et al.[10] who reported a figure of 62%. Similarly study conducted by Levine et al.[7] showed anatomical variation in 56.6% in his 150 studied patients.

Diagnostic nasal endoscopy was of great significant in patients of epistaxis. It helped in accurate diagnosis of cause of epistaxis and proper management of the same. This measure was better tolerated and less uncomfortable as compared to nasal pack or balloon. This conclusion was consistent with those of McGarry et al.[11]

Diagnostic nasal endoscopy was useful in identifying conductive olfactory loss and associated pathology with it. Clinical examination failed to diagnose pathology in 3 out of 5 (60%) cases of olfactory loss and endoscopy was necessary to make the proper diagnosis. This figure is close to the figure of 51% given by Allen et al.[12]

Rigid endoscopy helped in careful manipulation and removal of nasal foreign bodies and rhinolith under direct vision which were posteriorly placed and were not visible on clinical examination. Also, posterior extent of rhinolith was carefully evaluated. This conclusion was also supported by studies of Keck et al[13] and Hade et al.[14] Nasal endoscopy helps in exact localization and minimizing trauma to surrounding structure and prevents bleeding during foreign body removal.

In this study endoscopic biopsy was taken in 6 patients with sinonasal mass. Nasal endoscopy showed exact site in the region of pathology from where biopsy had to be taken which help in accurate histopathological diagnosis and help to minimize the bias. This conclusion was supported in the study conducted by Abtin Tabaee *et al*[9] who stated that office based nasal endoscopy with biopsy represent a safe and important tool in evaluation of sinonasal neoplasm and this procedure provides diagnostic information that may alter treatment decision.

In this study 5 patient of olfactory disturbance had atrophic rhinitis which was best diagnosed and managed by nasal endoscopy. This conclusion was supported by studies of Sevil Ari et al[15] who managed cases of atrophic rhinitis on regular follow up and endoscopic removal of nasal crust. In our study, anterior rhinoscopy did not reveal pathology and diagnosis in 39 cases (39%) which were diagnosed on Nasal endoscopy. This finding is consistent with Levine et al[7] study showed a figure of 38.7%. Thus, nasal endoscopy is efficient over clinical examination for diagnosing nasal and nasopharyngeal pathologies.

CONCLUSION

The worldwide acceptance of nasal endoscopes as an important diagnostic tool and useful surgical aid has contributed much to the world of Rhinology. It allows an unparalleled vision with brilliant illumination of nose and paranasal sinuses. In our study, we see that nasal endoscopy was an excellent diagnostic aid in many situations like sinusitis, unexplained headache, epistaxis, olfactory disturbances, nasal masses, nasal polyposis, nasal obstruction, nasal foreign bodies, nasal discharge, sinonasal malignancies. Diagnostic nasal endoscopy offers a high diagnostic accuracy in patients with sinonasal complaints. Diagnostic nasal endoscopy is capable of detecting nasal and nasopharyngeal pathologies which would otherwise be missed and this supports diagnostic nasal endoscopy as investigation of gold standard in the field of rhinology. Some nasal fossa pathologies are better defined on Diagnostic Nasal Endoscopy. Endoscopic directed procedures have high accuracy due to vision controlled and incomparable guidance in treatment of nasal and nasopharyngeal pathologies. Clinical examination and Diagnostic nasal endoscopy are complementary in making correct diagnosis. Diagnostic endoscopy must be done prior to any functional endoscopic sinus surgery, as they help in assessing the extent of sinus diseases and to know the variation and vital relation of the paranasal sinuses.

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Original Article

Radiological study of Oral and Craniofacial Findings in β Thalassaemic Children Undergoing Blood Transfusion

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Abstract

Background: Thalassaemia is the single most common gene disorder in the world and represents major health burden. The most common oral and craniofacial manifestations are enlargement of maxilla, bossing of the skull and prominent malar eminences. The aim of the study was to assess the radiological changes of the oral and craniofacial region in β thalassaemic children with in the age group of 12-16 yrs.

Methodology: The study population consisted of 50 diagnosed cases of thalassaemic children attending for regular blood transfusions. In each patient three types ofdigital radiographs were taken, namely intraoral periapical radiograph (IOPAR), Orthopantomograph, and Lateral Cephalogram. The radiographs were interpreted forthinned lamina Dura, short roots, marrow space enlargement, altered trabecular pattern, widened diploic space, Salt and Pepper appearance of the skull, Hair on end appearance, Maxillary prognathism.

Results: IOPAR of mandibular molar teeth region showed 52% thin lamina Dura, 34% cases had short roots, 82% cases showed enlarged Marrow space. OPG revealed 84% of cases with alterations in trabecular pattern. Lateral Cephalogram showed 86% widened diploic space, 84% showed salt and pepper appearance of the skull, 2% of them showed hair-on-end appearance of cranial vault, 50% of them showed Maxillary prognathism.

Conclusion: The characters and the degree of bone changes are often increased markedly, with increase in age of the patient inspite of regular blood transfusion. Early diagnosis, counselling and regular follow up are necessary to reduce the morbidity and to reassure the patient for overall improvement of general and oral health.

Keywords: Altered trabecular pattern, Hair on end appearance, Maxillary prognathism, Short roots, Thalassaemia

INTRODUCTION

Thalassaemia refers to a group of inherited hemolytic anemia involving defects in synthesis of either alpha or beta polypeptide chains of Hemoglobin (alpha-thalassaemia, beta-thalassaemia.[1] The word thalasseamia is derived from Greek term thalas meaning the sea. The term "thalassaemia" was first used by Wimple and Bradford in 1932. The disease manifests as homozygous (thalassaemia major) and heterozygous (thalassaemia minor) form. Thalassaemia minor is mild and usually asymptomatic and blood transfusions are required at a less regular interval. The thalassaemia major exhibits the most severe form of clinical symptoms with marked orofacial deformities, and these children should have regular blood transfusions to survive.[1] Homozygous beta thalassaemia, also known as Cooley's anemia or Mediterranean anemia, is seen chiefly in Mediterranean populations, with prevalence in Greece, Turkey, Cyprus and southern Italy.[2] The onset of symptoms occurs early in infancy (usually at the age of 4-6 months) and the children are severely anemic and have a short life expectancy. Children with most severe form of the disease rarely survive into adulthood because of cardiac failure, chronic anemia and hypoxia.[3] However, with advanced management, the prognosis has improved. The most common oral and facial manifestations are enlargement of the maxilla, bossing of the skull and prominent malar eminences due to the intense compensatory hyperplasia of

the marrow. This leads to expansion of the marrow cavity and a facial appearance known as "chipmunk" face.[4] The maxillary hyperplasia frequently results in proclination of teeth and spacing of maxillary teeth and other degrees of malocclusion.[5,6]

General dental practitioners are less aware of this condition in their daily practices, and are required to be aware of the nature of the disease and its implication on dental care. Reviewing the literature revealed only some case reports regarding the radiological findings in children and very few large studies have been undertaken. Therefore, the aim of this study was to assess the radiological changes of oral and craniofacial region in thalassaemic children ranging from 12-16 years of age.

MATERIALS AND METHODS

This cross sectional study was conducted to evaluate radiologically, the oral and craniofacial manifestations in thalassaemic children in the Department of Pedodontics and preventive dentistry, V. S Dental College and Hospital, Bangalore, Karnataka, India. The study population consisted of 50 diagnosed cases of thalassaemic children attending regular blood transfusions at Indira Gandhi institute of child health, Bangalore, India. Signed written informed consent from all the parents/guardian was obtained. The research protocol was approved by institutional ethical committee. Detailed case histories, clinical examination followed by radiological examination were done. Three types of digital radiographs namely IOPAR, Orthopantomograph, and Lateral Cephalogram with standardization were taken.

IOPAR of mandibular molar teeth were taken by Paralleling Technique, CCX Digital Trophy Trex Group – X-ray machine with specifications of 70 kVp, 8 mA, 16 x/sec (Electronic X-ray timer). IOPAR Films- No. 2 (31 x 41 mm) (Kodak Dental Intra Oral E-Speed Film, Eastman Kodak Company, New York.

Orthopantomogram (OPG) (Odontorama Pc 100 Trophy Radiologie, France) 55-100 kVp, 3-10 mA, 14 seconds of exposure time.

Cephalostat (odontorama pc 100 trophy Radiologie, France**)** 1.20 to 1.60 seconds (Exposure time), 70 to 85 kVp, 8 mA to 10 mA. For Cassette: Rigid (8" x 10") Intensifying Screen (kiran intensifying screen). Lateral cephalographic films 8" x 10" (Kodak tmat g/ra, Eastman kodak company, roechester, New York). The examiner was calibrated prior to interpretation of radiographs. Following features like thinned lamina dura and short roots, marrow space enlargement, altered trabecular pattern, widened diploic space, Salt and Pepper appearance of the skull, Hair on end appearance and maxillary prognathism were assessed. Comprehensive dental treatment was provided to all the patients.

Tracing of Lateral Cephalogram

The lateral cephalogram were traced on acetate paper, after proper orientationof radiograph by attaching one side withcellophane tape. Steiner's analysis was carried out by marking 3 reference points on the radiograph. Then the soft tissue outline was traced.

N –Nasion: Most anterior point midway between frontal and nasal bones onfrontonasal suture.

S-Sella: Geometric centre of pituitary fossa and or sella tursica. It is aconstructed point in mild saggital plane.

Point A - Deepest point in the mid saggital plane between the anteriornasal spine and alveolar crest between the two central incisors. It is also called as subspinale. After registering the cephalometric landmarks SNA angle was calculated.

SNA Angle: Angle formed by the line drawn from sella-nasion to point A. Itrelates the anterior-posterior position of maxilla to the anterior cranial base. The mean SNA angle is around 82°. If it is greater than 82° it indicates forward positioning of the maxilla.

RESULTS

The thalassaemic children in the present study were between the age group of 12-16 years. The mean age was around 14 years, with male predominance (68%). There were 49 cases of β-thalassaemia major and one case of β-thalassaemia intermedia. All the children were under regular blood transfusion therapy and most of the children were under chelation therapy. Out of 50 patients 8 patients had undergone spleenectomy**.** Clinically 5% of them showed characteristic "chipmunk appearance" of the face. Data was statistically analyzed using SPSS software version 15.0. IOPAR of mandibular molar teeth showed 52% of thin lamina dura, 34% cases had short roots, 82% cases showed enlarged Marrow space. 16% of them showed overlapping of above findings (Figure 1, Table 1, Graph 1). OPG revealed 84% of cases with alterations in trabecular pattern (Figure 2). Lateral Cephalogram showed 86% of widened diploic space, 84% showed salt and pepper appearance of the skull, 2% of them showed hair-on-end appearance of cranial vault, 50% of them showed Maxillary prognathism with a mean SNA angle of 84° (Figure 3, 4 and Table 2, Graph 2).

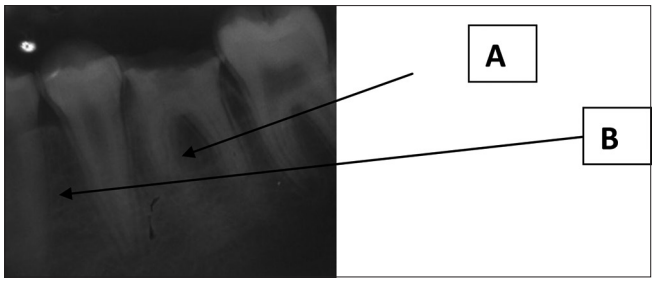


Figure 1: IOPAR showing presence of short roots, thin lamina dura (A) and enlarged marrow spaces (B)

Figure 2: OPG showing presence of alteration of trabecular pattern

Table 1: IOPAR findings

Findings	No.of cases (n)	No.of cases (n%)
Thin lamina dura	26	52
Normal lamina dura	24	48
Short roots	17	34
Normal roots	33	66
Enlarged marrow spaces	41	82

Table 2: Lateral cephalograph findings

Findings	No. of cases (n)	No. of cases (n%)
Widened diploic space	43	86%
Salt and pepper appearance	42	84%
Hair on end appearance	1	2%
Maxillary protrusion	25	50%

DISCUSSION

Thalassaemia is the most common single gene disorder in the world and represents a major health burden. It is a heterogeneous group of recessively inherited disorders of hemoglobin molecule characterized by the deficiency or absence of β or α globin chains. The children with thalassaemia classically present with severe anemia and have transfusion dependent survival. They have bony changes, retardation in growth, Splenomegaly, and iron overload

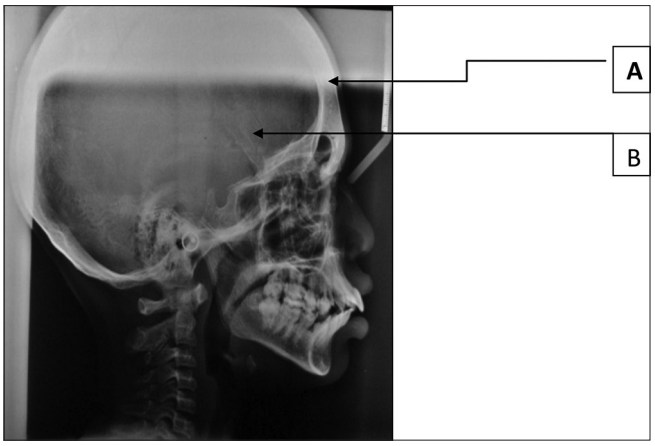


Figure 3: Lateral cephalogram showing, widening of diploic space (A) and salt and pepper appearance of the skull (B)

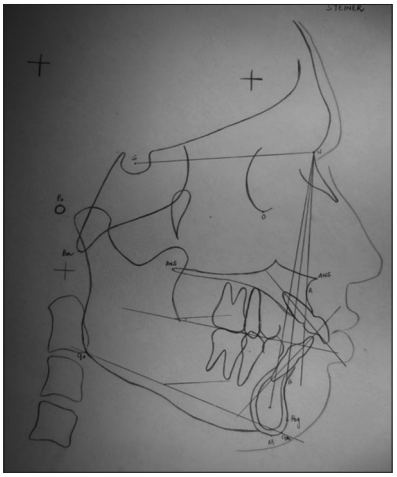
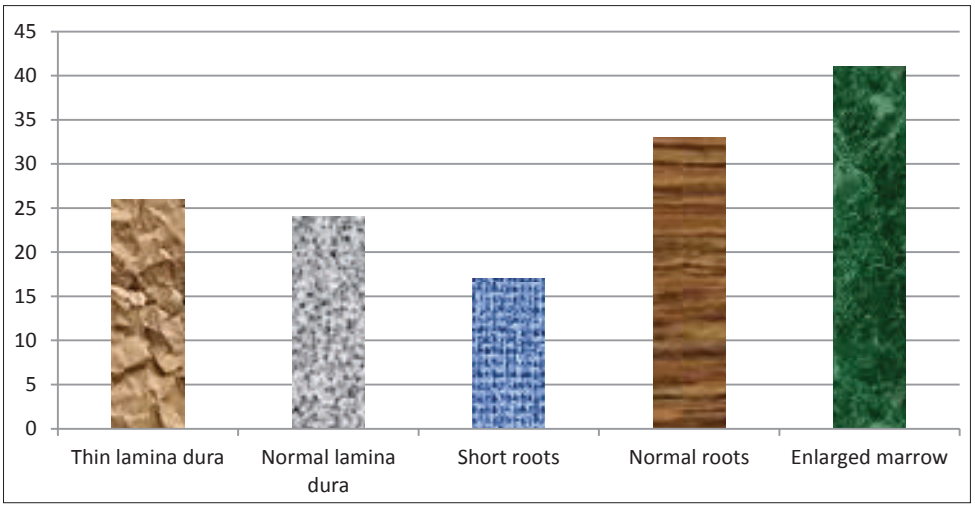
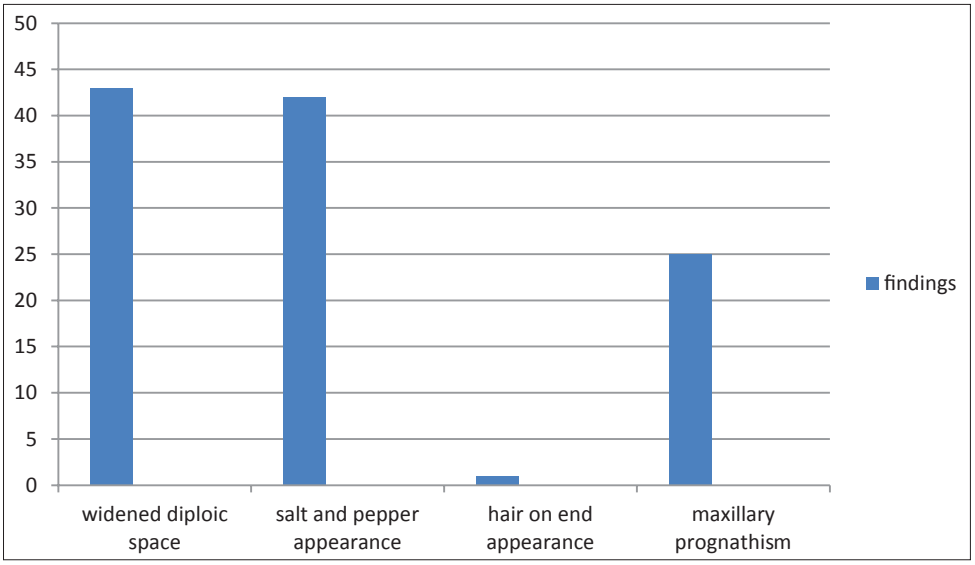


Figure 4: Steiner's cephalometric analysis with increased SNA angle

with consequent deposition in tissues.[7] Clinically 5% of them presented with characteristic 'chipmunk appearance' of the face. In the present study, mandibular molar teeth were considered for demonstrating the presence of short roots. 34% of cases showed short roots in this study. According to Wheeler's, the root lengths of mandibular first and second molars ranges from 13-14 mm. But in case of children with thalassaemia it was varied from 9-12 mm. Children with thalassaemia have generalized growth retardation, which may in turn affect the dimensions of the teeth. It may be due to variety of genetic and environmental factors, such as endocrine dysfunction and somatomedin deficiency which affects tooth size in thalassaemia major. Studies done by Poyton HG et al[8] and Hazza'a AM[9] have also reported cases with short roots. An intact lamina dura is seen as a sign of healthy periodontium. Present study showed 52% cases with thin lamina dura. Lamina dura is nothing but part of the alveolar bone that lines the socket as a thin layer of dense cortical bone. Absence or thinning of lamina dura is also seen in other systemic condition like secondary hyperparathydoism.[10,11] Studies done by



Graph 1: IOPAR findings



Graph 2: Lateral cephalogram findings

Poyton HG et al.[8] and Hazza'a AM et al.[9] reported thin lamina Dura in 46% and 87.5% of thalassaemic patients respectively. Children with thalassaemia have chronic anemia due to ineffective erythropoiesis which damages the red blood cell membrane. The body responds by increasing the production of red blood cells, consequently causing expansion of the bone marrow up to 15-30 times the normal amount.[9] Present study showed 82% cases with enlargement of marrow spaces. Studies done by Kaplan RI[12] and Parkin SF[13] reported enlargement of marrow spaces in 86% and 100% of their patients respectively. Study reported by Poyton HG and Davey KW showed 42% cases with enlarged marrow spaces.[8]

In the present study OPG revealed 84% of cases with alterations in trabecular pattern. These alterations are mainly due to hyperplasia of the bone marrow. Studies reported by Poyton HG[8], Kaplan et al.[12] showed 86% and 87.5% of the cases with similar findings.

Lateral Cephalogram of many children showed significant findings. Majority of the children (86%) showed widened diploic space. Hyperplasia of red bone marrow causes widening of the diploic spaces, which eventually leads to thinning or complete obliteration of the outer table of the skull. Study done by Roy RN et al. reported 73% of the cases with widening of diploic space.[14] Orzincolo et al. reported a case with similar finding.[15] Salt and pepper appearance of the skull is due to presence of osteopenia in thalassaemic patients. Regular blood transfusion of thalassaemic children leads to an iron overload and secondary hemochromatosis. As consequences to iron overload, endocrinopathies like hypogonadotropic hypogonadism may occur, which in turn leads to osteopenia. Present study showed 84% of

cases with salt and pepper appearance. Brandel M[16] and Wisetsin S[17] reported 36.6% of them with salt and pepper appearance of the skull. Orzincolo C et al. reported a case with similar finding.[15]

In our study hair-on-end appearance was observed in only one case (2%). The hair-on-end sign was seen in the diploic space as long and thin vertical striations. This is due to hyperplastic marrow which perforates or destroys the outer table, and new bone spicules are laid down perpendicular to the inner table. It is also more commonly seen in other hemoglobinopathies like sickle cell anemia, less commonly in patients with severe iron deficiency, cyanotic heart disease and also after long-term G-CSF treatment in severe congenital neutropenia.[18-22] Studies done by Wisetsin S[17] reported 8.3% and Roy AN et al.[14] reported 12% of patients with hair-on-end appearance. Parkin SF[13], Orzincolo C et al.[15] reported a case with similar finding. The present study showed 50% cases with maxillary prognathism. Studies done by Abu Alhaija et al.,[6] Bassimitci et al.[23] also reported maxillary prognathism based on cephalometric analysis.

Parkin SF[13], Beard ME et al.[24] reported that if the patient undergoes regular transfusion early in life, it helps to control the changes on the exterior of skull bones and in other bones, which are reflections of extra-medullary hemopoiesis (EMH) later in life. Scutellari PN et al. reported that in skull, the diploic space may become normal, and overgrowth of facial bones moderate, the hair-on-end pattern may disappear completely by regular blood transfusion.[25]

CONCLUSION

As the age increases the characters and the degree of bone changes are often increased markedly, in spite of regular blood transfusion. This is necessary for early diagnosis, counseling and regular follow up in order to reduce the morbidity, reassure the patient and to improve the overall general and oral condition of the patient. However, the findings of this study regarding the global trends in radiological manifestations, severity, patients care, parent's attitude about the disease did not show any greater variations as compared to other studies reported based on similar criteria.

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Original Article

Prevalence of Obesity and Overweight among School Going Children in Rural Areas of Ernakulam District, Kerala State India

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Abstract

Objective: Objective of this study was to assess the prevalence of overweight, obesity among school children in the rural areas of Kochi District.

Methodology: A total of 1098 children from 6-15 years of age were screened from rural school. Overweight and Obese children was determined by the BMI percentile by plotting the BMI number on the appropriate CDC BMI-for-age growth chart

Results: The results of the study exposed the fact that the percentage of overweight and obese children are growing in rural areas of Kerala. The study also showed that obesity was seen more in boys.

Conclusion: Obesity is now the most common disorder affecting children and adolescents, reflecting the current epidemic. Precise causes of this marked increase in prevalence are unclear, but results from both increased intake of energy –dense food and reduced exercise. Energy expenditure has fallen due to an increase in sedentary behaviour. Hence appropriate nutritional intervention programmes involving school children, their parents and school authorities has to be conducted.

Keywords: Kerala, Prevalence, Obesity, Overweight, Rural, School children

INTRODUCTION

Obesity is one of the most serious public health problems.[1] It has become a global pandemic. Obesity implies excess fat and not merely excess weight. Body weight is determined by an interaction between genetic, environmental, psychological factors acting through the physiological mediators of energy intake and expenditure. Management of childhood obesity is challenging with major impetus on life style measures. According to a WHO report, there are 1 billion overweight people in the world, of whom 300 million are obese. Concurrently, a growing prevalence of obesity and its related chronic diseases is being observed in these countries. Increasing obesity is already a major concern in developed countries for pre-school children as well as school children. In developing countries, this rising epidemic along with the persistence of under nutrition and infections typifies the 'Double Burden of Malnutrition' (DBM),[2] which is becoming a great concern for African countries. Indeed, the DBM is a real threat at the population, household and even individual level, and it is now observed among school children. Rural areas of developing countries are generally prioritized as regards nutrition intervention, because under nutrition is more widespread than in urban areas. However, a shift is occurring and children in the cities are at risk of both over-nutrition and under nutrition. The prevalence of child obesity is increasing rapidly worldwide. Childhood obesity has more than tripled in the past 30 years. The prevalence of obesity among children aged 6 to 11 years has increased from 6.5% in 1980 to 19.6% in 2008. The prevalence of obesity among adolescents aged 12 to 19 years has increased from 5.0% to 18.1%.[3,4] Obesity is the result of a caloric imbalance (too few calories expended for the amount of calories consumed) and is mediated by genetic, behavioural, and environmental factors.[5] It is associated with several risk factors for later heart disease and other chronic diseases including hyperlipidaemia, hypertension hyperinsulinemia, and early atherosclerosis. Obesity has become a global health problem, affecting more than 1.3 billion adults in both developed and developing countries.[6]

MATERIALS AND METHODS

The study was a cross-sectional randomized epidemiological study among school students of rural school of Kochi city. A total number of 1098 school children aged 6 to 17 years had participated in this study. Out of them, 537 were boys and 561 were girl. The body weight was measured without shoes using a measuring scale and height to the nearest centimetre was taken. Body Mass Index (BMI) was calculated as weight (in kilograms) divided by height (in meter squared). For children and teens, after BMI is calculated, the BMI number is plotted on the CDC BMI-for-age growth charts[7] (for either girls or boys) to obtain a percentile ranking. Percentiles are the most commonly used indicator to assess the size and growth patterns of individual children in the United States. Percentiles are used for children and teens because the amount of body fat differs between boys and girls and body fat also changes with age. The percentile indicates the relative position of the child's BMI number among children of the same sex and age. Healthy children have a BMI percentile ranging between 5th percentiles to 85th percentile. The children whose weight were more than 85th to less than the 95th percentile were considered as overweight and obese who were equal to or greater than the 95th percentile (WHO 2000). Chi-square-test was used to find out the significance between sex and rural school children with respect to childhood obesity. Odd's ratio indicates that there is strong hazardous association between sex and obesity.

RESULTS

Table 1 gives the age and sex wise distribution of the total number of children screened. A total of 1098 children from 6-17 years of age were screened from a rural school of which 537 were boys and 561 were girls. Figure 1 shows the distribution of the sex in the present study. Table 2 shows the 95th percentile of BMI of boys and girls in the present study. Healthy children have a BMI percentile ranging between 5th percentile to 85th percentile. The children whose weight were more than 85th to less than the 95th percentile were considered as overweight and obese who were equal to or greater than the 95th percentile.

As could be seen in Table 3, from the overall screened sample 3.35 per cent boys were obese, 2.85 per cent girls were obese. The results revealed that 96.9 per centof the children were of normal weight. Figure 2 shows the distribution of obesity by sex. Figure 3 also shows the prevalence of obesity by sex. Table 4 shows the prevalence of obesity by age. Obesity was more among the smaller children of age group 6, and 9 years with 8.79% and 5.67%

Table 1: Age and sex distribution of the study group

Age	Boys	Girls	Total
6	63	28	91
7	28	49	77
8	70	69	139
9	83	58	141
10	67	50	117
11	35	53	88
12	26	66	92
13	26	20	99
14	79	112	172
15	60	56	116
Total	537	561	1098

Table 2: Prevalence of overweight/obese children from rural areas 95th percentile of BMI for Boys and Girls

Age in years	Boys	Girls
6	18.4142	18.8378
7	19.1524	19.6779
8	20.0679	20.6953
9	21.0889	21.8173
10	22.1541	22.9826
11	23.2136	24.1414
12	24.2299	25.2556
13	25.1781	26.2988
14	26.0466	27.256
15	26.8369	28.1237

Table 3: Prevalence of obesity by sex

Sex	Obese	Non obese	Total
Boys	18 (3.35%)	519 (96.65%)	537 (100%)
Girls	16 (2.85%)	545 (97.15%)	561 (100%)
Total	34 (3.10%)	1064 (96.90%)	1098 (100%)

Table 4: Prevalence of obesity by age

Age	Obese	Non obese	Total
6	8 (8.79%)	83 (91.21%)	91 (100%)
7	0 (0.00%)	77 (100%)	77 (100%)
8	5 (3.60%)	134 (96.4%)	139 (100%)
9	8 (5.67%)	133 (94.33%)	141 (100%)
10	3 (2.56%)	114 (97.44%)	117 (100%)
11	3 (3.41%)	85 (96.59%)	88 (100%)
12	4 (4.35%)	88 (95.65%)	92 (100%)
13	0 (0.00%)	99 (100%)	99 (100%)
14	2 (1.16%)	170 (98.84%)	172 (100%)
15	1 (0.86%)	115 (99.14%)	116 (100%)
Total	34 (3.10%)	1064 (96.90%)	1098 (100%)

Table 5: Prevalence of overweight and obesity sex wise

Sex	Overweight	Obese
Boys	32 (5.96%)	18 (3.35%)
Girls	51 (9.09%)	16 (2.85%)
Total	83 (7.56%)	34 (3.10%)

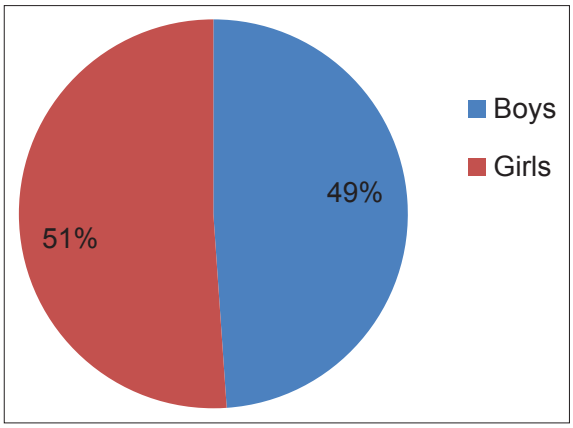


Figure 1: Sex distribution of the study group

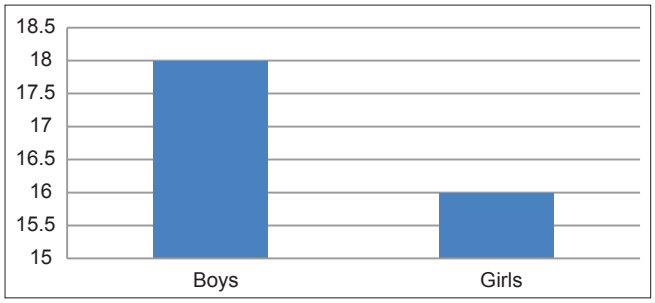


Figure 2: Distribution of obesity by sex

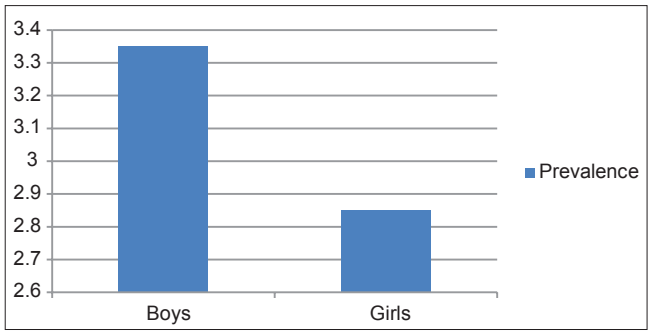
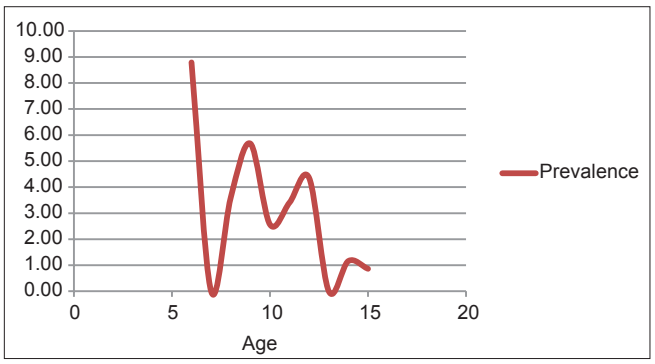


Figure 3: Prevalence of obesity by sex



Graph 1: Prevalence of obesity by age

respectively. This can be seen in Graph -1. When the sex wise comparison of all the boys and girls were made (Table 5), it was noted that out of a total of 1098 children screened, 561 were girls, and 537 were boys. Among the total girls, 9.09 percent were overweight and 2.85 percent were obese. Similarly among total boys 5.96 percent were overweight and 3.35 percent were obese. While obesity seems to be growing in children regardless of sex, it can be noted that there is a sex wise variation in the prevalence of overweight and obesity in children irrespective of the place as revealed in many studies done in India and abroad. The present study also compares the sex wise variation seen in children. The prevalence of obesity among boys were found to be higher than that of girls. But girls were found to be more overweight than boys. Studies by Kapil et al.,[8] also indicated that the prevalence of obesity was lower in girls (6%) as compared to boys (8%). Studies done by Mudur[9] in three major Indian cities found that more girls were overweight and obese than boys. All these studies therefore indicate that the sex of the child has an influence on the prevalence of overweight and obesity. Age wise comparison of boys and girls from both rural were also made. And it was found that 6 & 9 year old rural children had the highest rate of obesity.

Epidemiological Test

Odds ratio = ad/bc = 1.181358 > 1

Odd's ratio indicates that there is strong hazardous association between sex and obesity.

DISCUSSION

In India, very few studies have been carried out to study the overweight/obesity in rural school children and majority of them have been carried out in cities in high income schools. The present study was carried out in a rural school of Kochi district of Kerala. In our study, obesity was found to be more in boys than girls but girls were more overweight than boys. Studies by Kapil et al.[9] also indicated that the prevalence of obesity was lower in girls (6%) as compared to boys (8%). On the contrary, studies done by Mudur[9] in three major Indian cities found that more girls were overweight than boys. All these studies therefore indicate that the sex of the child has an influence on the prevalence of overweight and obesity.

When compared to the prevalence studies done in Kerala, it was found that the rate of underweight is reducing, but at the same time the rate of overweight and obesity is increasing. Studies done by Ramachandra[10] in 1000 adolescent children of Thiruvananthapuram and Geetha[11] on high school girls of Thiruvananthapuram also revealed 5.4 percent and 2.2 percent of obesity respectively. The results of the present study is also

consistent with the above studies revealing that obesity and overweight in children are gradually growing like other countries of the world. Studies reveal that in India, the problem of overweight and obesity; is also growing in other states too. In another obesity study done by Ramnath[12] in 1500 school children of Meerut UP, prevalence was 9 percent. Yet in another study by Popkin[13] in all the five metros of Delhi, Mumbai, Chennai, Hyderabad and Kolkata it had been noticed that one out of every five school children or 20 percent are overweight. The possible risk factors in causing childhood obesity are sedentary lifestyle which makes them stay physically inactive. Giammattei et al. (2003) also reported that children who spent more time watching television had a higher BMI. Often parents are working and unable to concentrate on balanced nutritional food for their children. They find it easier to let their children consume junk and fast foods. Even the burden of school work and academic competitiveness has decreased the participation in sports and other form of physical activities in urban area which leads to high frequency of overweight and obesity.

CONCLUSION

The present findings indicate that prevalence of childhood obesity in Kochi is not as high as the incidence reported by other studies. However, we found higher frequency of obesity in boys as compared to the girls. Obesity is a serious problem, which requires immediate attention, creating awareness program in the schools and parents encouraging their children to be involved in more physical exercises, sports and outdoor activities, thus avoiding the march towards obesity. Prevention of obesity in children is easier than in adults. Thus effective prevention of adult obesity will require the prevention and management of child hood obesity.[14] Thus childhood obesity is an emerging health problem. Hence effective preventive strategies should be developed to halt this epidemic.

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Original Article

Late Onset Shake-Etiology At Stake - A Prospective Study

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Abstract

Background: Late onset seizure is a major cause of morbidity & mortality in the population. Clinical evaluation & etiological analysis paves the way for early and specific treatment.

Objectives: The purpose of this prospective study is to determine the clinical profile of late onset seizure & to determine the etiology of late onset seizure.

Methods: In this descriptive, prospective, cross sectional study, all patients who presented to the department of medicine with one or more episode of seizure with the onset after age 25 yrs were included. Study for a period of 12 months march 2010-march 2011 study population was obtained by random sampling.

Results: Among 50 patients, 16 patients (32%) etiology could not be ascertained. Among the 34 symptomatic patients (68%), 16 patients (47.05%) had post stroke, 1 patient (2.94%) had NCC, 3 patients (8.84%) had tumor, 3 patients (8.84%) had metastasis, 7 patients (20.58%) had metabolic etiology and 4 patients (11.76%) had infective etiology.

Conclusion: In this study of late onset seizure, mean age of onset of seizures was 49.3 & male preponderance was noted. Most common seizures type was GTCS-(64%). Underlying causes were recognized in 68% (i.e., symptomatic seizures). Most common etiology of seizure with onset after 25 years of age was post stroke (16 out of 34 patients accounting for 47%). Between 30 to 60 years, most frequent etiologies were Idiopathic, post stroke and Metabolic.

Keywords: Etiologies, Idiopathic, Metabolic, Post stroke, Seizure

INTRODUCTION

Late onset seizure may be simply defined as seizure beginning in adult life >25 yrs.[1] Much attention has been focused on determining the etiology of late onset seizure. The difference in the emphasis is due to the view that the incidence of idiopathic seizures is greatest during childhood and adolescence. After the age of 25 the risk of developing seizure disorder is low. The importance of late onset seizures is its frequent association with secondary causes. Seizure that begins after age of 25 years may be associated with head trauma, CVA, CNS infection, Brain tumors, congenital CNS abnormality, illicit drug use, metabolic derangement. The current study includes 50 patients with one or more than one seizure with the onset after age 25.[2] The purpose of this study is to know the etiology of seizures after 25 years, since they are due to secondary causes and to find the etiology in our hospital, since they vary according to geographic locations.[3,4]

AIMS OF THE STUDY

- To study the clinical profile of late onset seizure
- To determine the etiology of late onset seizure.

MATERIALS AND METHODS

The current study includes 50 patients with one or more episode of seizure with the onset after age 25. A detail history and clinical evaluation was performed as per the proforma. Basic work up including CBC, RBS, Sodium, potassium, RFT, EEG, Radiological investigation and CSF analysis was performed when it was appropriate.

INCLUSION AND EXCLUSION CRITERIA

Inclusion Criteria

- A diagnosis of seizure was made on the basis of semiology according to the ILAE (International league against Epilepsy) classification scheme revised 1981.

Exclusion Criteria

- Pseudoseizures
- Age of onset before 25 years but who continue to have seizures after 25 years.

RESULTS

Study was done including 50 patients, with 31 males and 19 females. EEG was done in 37 patients, 35 underwent CT scan and 11 underwent MRI & 1 both.

Mean age of onset of seizure was 49.3 ± 28.2 (SD) yrs, youngest patient being 25 yrs and oldest being 86 yrs.

Male to female ratio was 1.63:1.

1. Time of Occurrence of Seizures

Seizures occurred during the day time in 34 patients (68%), 13 patients (26%) seizures occurred at night and 3 patients (6%) had seizures both during day and night.

2. Types of Seizures

Among 50 patients, 32 patients (64%) had GTCS, 5 patients (10%) had SPS, 2 patients (4%) had CPS, 1 patient (4%) had SPS with secondary generalization, 10 patients (20%) had CPS with secondary generalization.

3. Status Epilepticus

Among 50 patients, 7 patients (14%) presented with status epilepticus, of these 4 patients had GTCS & 3 patients had CPS-sec Gen type of seizures, and the etiology among them being Idiopathic in 2 patients, post stroke in 4 patients & in 1 patient due to tumour.

4. Prodrome-11 Patient had Prodromal Symptoms

Among 11 patients who had prodromal symptoms, 8 patients (72.72%) presented with headache, 1 patient (9.09%) had fearfulness & 1 patient (9.09%) had mood changes, and 1 patients (9.09%) had irritability.

5. Aura

Among 50 patients, 10 patients (20%) had elementary aura, 3 patients among elementary aura (30%) had sensory aura, 6 patients (60%) had motor aura & 1 patient (10%) had autonomic aura.

Among 50 patients, 7 patients (14%) had complex aura, 2 patients had complex aura (28.57%) had cognitive aura, 3 patients (42.85%) had affective aura & 2 patients (28.57%) had psych motor/sensory aura.

6. Tongue Bite

Among 10 patients had tongue bite during seizures, of these 7 patients had GTCS and 3 patients had Partial seizures with secondary generalization.

7. Post-ictal Phenomena

Among 50 patients with post ictal phenomenon, 24 patients (48%) had cofusion, 8 patients had (16%) loss of consciousness, 6 patients (12%) had drowsiness 6 patients (12%) had headache, 6 patients (12%) had generalized bodyache.

8. Incontinence

Bladder incontinence was seen in 15 patients (30.0%) & Bowel incontinence was seen in 3 patients (6.0%).

9. Past History

9 patients (18%) had past history of stroke.

10. Family History

6 patients (12%) had family history of seizures.

11. Clinical Examination

Abnormalities on neurological examination identified in 9 patients (18%), among them 2 patients (22.22%) had right hemi paresis, 5 patients (55.55%) had left hemi paresis and 2 patients (22.22%) had Aphasia.

12. EEG Abnormalities

EEG abnormalities were seen in 16 patients (32%), among them 7 patients (14%) had focal EEG abnormalities and 9 patients (18%) had generalized EEG abnormality.

13. ETIOLOGY

Etiology in generlized seizures

Table 2 shows, among 50 patients, 32 patients (64%) had generalized seizures, among them 10 patients (31.25%) etiology could not be ascertained. Among the 22 symptomatic patients (68.75%), 9 patients (28.12%) had post stroke, 1 patient (3.12%) had metastasis, 7 patients (21.82%) had metabolic etiology, 4 patients (12.5%) had infective etiology & 1 patient (3.12%) had tumor intracranial.

Table 1: Etiology in generalized seizures

Generalized	No (n=50)	%
1. Idiopathic	10	31.25
2. Symptomatic		
a) Post stroke	9	28.12
c) Metastasis	1	3.12
d) Metabolic	7	21.82
e) Infection	4	12.5
f) Tumour	1	3.12
Total	32	100

Etiology in partial seizures

Table 2 shows, among 50 patients, 18 patients (36%) had partial seizure; among them 6 patients (33.33%) etiology could not be ascertained. Among the 12 symptomatic patients (66.66%), 7 patients (11.11%) had post stroke, 2 patients (11.11%) had tumor intracranial, 2 patients (11.11%) had metastasis, 1 patient (5.55%) had infective etiology.

Etiology of late onset seizures

Table 3 shows, among 50 patients, 16 patients (32%) etiology could not be ascertained. Among the 34 symptomatic patients (68%), 16 patients (47.05%) had post stroke, 1 patient (2.94%) had NCC, 3 patients (8.84%) had tumor, 3 patients (8.84%) had metastasis, 7 patients (20.58%) had metabolic etiology and 4 patients (11.76%) had infective etiology.

FOLLOW UP

- Patients were followed up for 6 months
- Out of 50 patients 3 expired and 11 dropped out
- Of these 36 patients 7 had recurrence due to non compliance, were on 2 AED (Antiepileptic drugs)
- Remaining were seizure free of which 22 were on 1 AED and 7 were on 2 AED.

DISCUSSION

In this study of clinical profile and etiological analysis of late onset seizures, a total of 50 patients were included over a period of 2 years.

Table 2: Etiology in partial seizures

Partial seizures	No (n=50)	%
1. Idiopathic	6	33.33
2. Symptomatic		
a) Post Stroke	7	38.88
b) Tumor	2	11.11
c) Metastasis	2	11.11
d) NCC	1	5.55
Total	18	100

Table 3: Etiology in the present study

Causes	No (n=50)	%
1. Idiopathic	16	32.0
2. Symptomatic	34	68.0
a) Post stroke	16	47.05
b) NCC	1	2.94
c) Tumour	3	8.84
d) Metastasis	3	8.84
e) Metabolic	7	20.58
f) Infection	4	11.76
Total	50	100

There was male preponderance in this study as quoted by other studies in United States and Europe (Granieri et al. 1983).[3]

It is well known fact that as one enters adult life, partial seizures with or without generalization becomes the predominant seizure type. In current study partial seizures with or without secondary generalization accounted for 36% of the cases.

Simple partial seizures were observed in 6 patients (12%) 4 had motor and 2 had sensory seizures, one of the patient with sensory seizure had no focal neurological deficit but CT scan showed tumor in the temporal region, olfactory and gustatory symptoms are most often associated with temporal lobe involvement (Howe & Gibson 1982).

Simple partial motor seizures seen in 4 patients of this 3 had localized to upper limb & 1 to the face. Such a frequent involvement of hand and face is because of disproportionate involvement of motor cortex in representing hand and face.

Of the 12 patients with complex partial seizures with or without secondary generalization 5 (10%) were idiopathic and 7 symptomatic of this 4 post stroke, 1 Neurocysticercosis & 1 tumor.

A positive family history was noted in 3 (6%) patients in first degree relatives, which was similar to studies in India observed in 5.2% to 8.9% (Koul et al, Rural Kashmir India, Das SK et al. Rural Bengal).[4,5]

Neurological abnormalities were detected in 9 (18%) patients; radiological abnormalities were detected in all patients (100%). In remaining 41 patients neurological examination were normal and radiological abnormalities were noted 15 (30%) patients. These results can be compared to study in Spain 1985 where a total of 250 patients were studied, of these only 41 (16.4%) patients had focal neurological deficit. CT scan abnormalities were found in 92.6% (38 of 41 patients) of the patients with focal neurological findings and 42.5% (89 of 209 patients) with normal neurological examination.

Almost all grey matter conditions can result in seizures and the range of causes is strongly age dependent. In current series 32% were Idiopathic & 68% were symptomatic seizures. Most frequent cause was post stroke seizures (32%), metabolic (14%), Infections (8%), Tumour and metastasis (6%) each.

These Results can be Compared with Few Studies as Follows

In a study of 248 patients by Martinez et al. [13] (1998) Spain, with age of onset after 20 years the most frequent etiologies

were stroke (26.2%), tumors (26.2%), unknown (24.6%) and chronic alcoholic intake (18.5%). Stroke was the most common etiology in patients over 60 yrs of age.

In another study Jimenez et al (1990),[6] etiology was unknown in 51.3% of cases. The most common identified causes were Cerebrovascular disease (20%). Chronic alcoholic abuse (10%), tumors (6.3%), neurocysticercosis (6.3%) and post traumatic (2.5%).

In a study of 250 patients with Late Onset Seizures by perez Lapez et al (1985),[7] Etiology was identified in 201 patients with most frequent cause being chronic alcohol abuse (24.8%) followed by tumor (16.4%), post stroke (13.2%) and post traumatic (11.2%).

In a study in Mexico by medina et al (1990), a total of 100 patients, 50 patients (50%) had neurocysticercosis as the cause.

In a more recent study in Madagascar[8] based on serological analysis of epileptic Vs nonepileptic controls, neurocysticercosis was suggested most important etiological factor for late onset seizures.

The most common cause identified was post stroke which was noted in 16 patients (32%). The mean age at presentation was 58 years with male to female ratio 1.28:1.4 of these patients presented with status epilepticus. Of these patients generalized seizures was observed in 9 patients (56.25%), simple partial seizures in 2 patients (12.5%), 5 patients presented with complex partial seizures with secondary generalization (31%).[9,10] 12 of these patients had scan findings of ischemic infarcts whereas hemorrhagic stroke was noted only in 4 patients. 3 patients with scan evidence of old stroke presented with seizures. Such a presentation has been described in literature. In a care control study by Robert et al.[11] (1998), CT scan evidence of vascular pathology was found in 15 of 132 patients with seizures after the age of 40 years who presented with seizures without any neurological findings. This has also been reported by Shinton et al (1987) who found 8 of their 176 cases (4.5%) had history of seizures before they presented to the hospital with first episode of stroke. These patients were diagnosed to have "Vascular precursor Epilepsy" as coined by Barolin (1982).[12]

Cerebral venous thrombosis was diagnosed in 3 patients with mean age of 36.3 years.

Neurocysticercosis was diagnosed in 1 patient (2%) in this study, the diagnosis was made on radiological grounds & was confirmed by repeating CT scan after 2 months of therapy with albendazole (15 mg/kg for 4 weeks) which showed clearing of the lesion, patient presented with complex partial seizures. The frequency with which it occurs as a cause of late onset seizures varies according to the geographical area. It was the most common cause in Mexico & Madgascar.[8]

Cerebral tumor were noted as cause of seizures in 3 patients (6%) with mean age of presentation of 46.3, male to female ratio 1:2. Of which 2 had simple partial seizures, one presented with GTCS, 1 had meningioma and 1 had Glioma cerebral metastasis seen in 3 patients (6%) with mean age of 43 years and male to female ratio 2:1 of this one presented with simple partial seizures with secondary generalization, one with complex partial seizures with secondary generalization and one with GTCS, of this 2 had primary from lung.

It should be emphasized that despite careful investigation a sizeable proportion of patients (32%) were diagnosed as Idiopathic. More sensitive scanning techniques may help us to further sort out this group of Idiopathic seizure disorder into various etiologies.

CONCLUSION

1. In this study of late onset seizure, The mean age of onset of seizures was 49.3 & male preponderance was noted
2. Most common seizures type was GTCS (64%)
3. Underlying causes were recognized in 68.0% (i.e., symptomatic seizures)
4. Most common etiology of seizure with onset after 25 years of age was post stroke (16 out of 34 patients accounting for 47%).
5. Between 30 to 60 years, most frequent etiologies were Idiopathic, post stroke and Metabolic.

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Original Article

C-Reactive Protein in Ischemic Stroke – An Experimental study

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Abstract

Background: There are many studies which predicts the prognostic significance of C-reactive protein (CRP) in ischemic stroke. Taking values at only one point of time is not important as compared to level its concentration at different perids of hospital stay of patient. Therefore we compared the values of CRP Within 24 hrs, 48-72 hrs & at the time of hospital discharge admission and discharge with 1-year outcome.

Methods: Forty eight patients were included in the study and serum CRP values were measured, within 24 hours after ischemic stroke, within 48 to 72 hours, and at hospital discharge and an association was examined between the CRP levels and the time at which these values were taken and analyzed statistically.

Results: 1.5 mg/dL for CRP at discharge provided optimum specificity for adverse outcome. Markers of worse prognosis were the presence of CHD (HR 1.98, 95% CI 1.21 to 3.38; $P = 0.0081$), PAD (HR 2.66, 95% CI 1.32 to 5.69; $P = 0.0082$), age > 70 years (HR 2.28, 95% CI 1.06 to 3.74; $P = 0.0475$). CRP level at hospital discharge (HR 6.82, 95% CI 2.65 to 20.07; $P = 0.0001$) showed the strongest association.

Conclusions: CRP level is a better prognostic indicator of ischemic stroke at the time of discharge and is of greater utility for risk identification. These findings are strongly and statistically significant ($p < 0.005$) and confirm that raised CRP may predict future outcome in terms of mortality and morbidity.

Keywords: IL-6, Prognosis Proteins ischaemia

INTRODUCTION

C-reactive protein (CRP) is considered as a sensitive predictor of both new-onset and recurrent ischemic events.[1-3] C-reactive protein (CRP), levels are associated with different stroke outcomes and further vascular events. CRP is a potential prognostic marker after vascular events and a potential predictor of future vascular events. Many retrospective studies concerning ischemic stroke indicated that recent infections may increase the possible risk for ischemic stroke.[4,5] Several studies have shown elevated levels of C-reactive protein (CRP), among individuals who are at greater risk of ischemic heart diseases.[6-10] Elevated CRP is more reliable predictor than creatine kinase in MI patients.[11] Medical data relating CRP as a prognostic factor in ischemic stroke is very thin.[12,13] Therefore, this prospective study is performed in patients with first-ever ischemic stroke to further analyze the relationship between CRP values measured immediately and at different times.

MATERIAL AND METHODS

All patients (48) who were admitted in department of medicine of Teerthankar Mahaveer Medical College, Moradabad, India, with a diagnosis of ischemic stroke. To maintain the research protocol, consent was taken from all the patients, institutional ethics committee and research committee. A very strict protocol for screening of patients to be included in this study was maintained and monitored, which included thorough history, systematic examination followed by advanced radiological evaluation with the help of department of radio-diagnosis. Radiological findings were classified into infarcts and lucencies. Great emphasis was put on habit of smoking, alcohol, hypercholesterolemia, hyper-triglyceridemia, hypertension and diabetes mellitus. Routine laboratory investigations were done and we tried to keep the patients, away from hospital acquired infections. Exclusion criteria for the present study included those subjects who had stroke, subarachnoid hemorrhage,

vasculitis, renal, hepatic and malignant diseases within 30 days from the time of starting of the study.

RESULTS

After comprehensive evaluation, 48 patients were included in this prospective study. Among 48 patients mean ± SD age was 69.08 ± 6.17 years. The CRP values, within 24 hours, between 48 to 72 hours, and at hospital discharge were 1.4, 1.0 and 0.7 mg/dl, respectively. CRP levels above normal value (>0.5 mg/dL) at entry were significantly associated with larger infarcts ($P < 0.0003$) and cortical involvement ($P = 0.0001$). At discharge, higher CRP levels were also associated with larger infarcts ($P = 0.0041$). Markers of worse prognosis were the presence of CHD (HR 1.98, 95% CI 1.21 to 3.38; $P = 0.0081$), PAD (HR 2.66, 95% CI 1.32 to 5.69; $P = 0.0082$), age >70 years (HR 2.28, 95% CI 1.06 to 3.74; $P = 0.0475$). CRP level at hospital discharge (HR 6.82, 95% CI 2.65 to 20.07; $P = 0.0001$) showed the strongest independent association with the combined end point at 1 year. There was not a significant association between CRP on admission and death.

DISCUSSION

The aim of the study was to predict the relationship between CRP and prognosis after ischemic stroke. Our data indicate that patients with ischemic stroke who have CRP levels >1.4 mg/dL at discharge have a significantly worse outcome. Several previous studies have reported elevated CRP values in patients with ischemic stroke. [8,9,14-19] Variations in CRP level in ischemic stroke not previously analyzed in detail. According to this study a different prognostic significance can be elucidated: a benign, consisting of either constantly normal or decreasing values from admission through to discharge, and another pattern, represented by those patients with constantly elevated or increasing values from the time of admission to discharge. Constantly elevated levels of CRP represent either an ongoing inflammatory process or the extension of cerebral ischemia.[15] Many previous studies indicate that inflammatory mechanisms contribute to secondary neuronal injury after cerebral ischemia.[4,5,20-24] Rise in CRP levels is not only associated with immediate consequences but also remains elevated in stroke survivors.[10] Many previous studies have also indicated that rise in fibrinogen levels are also associated with higher CRP levels.[25,26] However we didn't find any association between fibrinogen and CRP levels. CRP in structure is protein and its synthesis is controlled at the level of transcription.[3,27,28] and IL-6 is key regulatory factor in this phenomenon.[29] Raised levels of CRP reflect the extent of brain infarction in the form of large sized infarcts. Our findings of large sized infarcts are consistent with previous studies.[15] Patients in whom the CRP levels remain persistently elevated have worst prognosis. The mechanism remains unexplained why the values of CRP at the time of discharge remains lower as compared to entry levels.

CONCLUSION

From above results and discussion it is certain that elevation of CRP is common in ischemic conditions and more so over CRP levels classify stroke patients into high and low-risk groups patients. These observations also raise the possibility that ischaemic patients are at greater risk of subsequent associated complications.

Table 1: Different CRP levels at different times

S.N.	Duration	Values (mg/dl)
1.	Within 24 hrs	1.4
2.	48-72 hrs	1.0
3.	At the time of hospital discharge	0.7

Table 2: Markers of worst prognosis

S.N.	Markers of worse prognosis	HR (Hazard ratio)
1.	CHD	1.98
2.	PAD	2.66
3.	Age>70 yrs	6.82

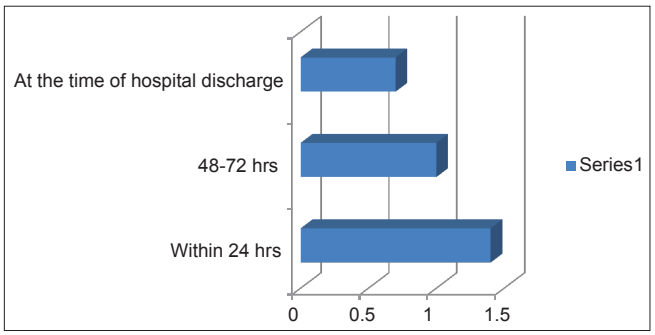

Figure 1: Different CRP levels at different times

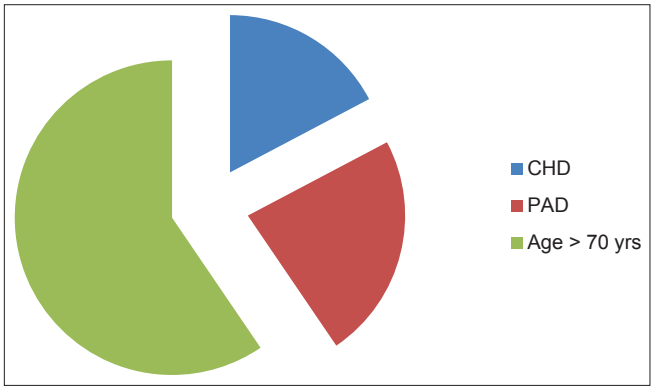

Figure 2: Markers of worst prognosis

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Original Article

Anomalous Branching Pattern of the External Carotid Artery in Cadavers

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Abstract

Background: With increasing use of invasive diagnostic and interventional procedures in cardio-vascular disease, it is important to find out type and frequencies of vascular variations. Variations in the course, branching and distribution of carotid arteries are commonly encountered.

Material & Method: In the present study we have observed variations of the branching pattern of external carotid artery. We examined the 30 cadavers during routine dissection.

Result: One cadaver had common trunk of lingual, facial and superior thyroid artery on one side (i.e thyrolinguofacial trunk) and in four cadavers there were unilateral common trunk of lingual and facial artery i.e linguofacial trunk.

Conclusion: Anatomical knowledge of the origin, course and branching pattern of external carotid artery will be useful during head & neck surgeries. The present study thus provides useful information for clinical application. The clinical importance of this variation is discussed.

Keywords: External carotid artery, Lingualfacial trunk, Thyrolingualfacial trunk

INTRODUCTION

External carotid artery is the chief artery of Head & Neck region in humans. It arises from the common carotid artery, lateral to the upper border of the thyroid cartilage, level with intervertebral disc between the third and forth cervical vertebrae. From its origin it take a slightly curved course, passes upward and forward, and then inclines backward to the space behind the neck of the mandible, where it divides in to the superficial temporal artery and maxillary artery within the parotid gland. It is decreases in size in its course up to the neck, owing to the number and large size of the branches extending from it. It has eight named branches distributed to the head & neck.[1] The facial artery normally arises from external carotid artery, just above the lingual artery, at the level of greater cornu of hyoid bone in the carotid triangle. The reported variation of facial artery includes its intraparotid origin,[2] origin as a common trunk with lingual artery as linguofacial trunk.[3,4] Another variation in branches of external carotid artery are as follows – the lingual artery form a common trunk with the facial (lingofacial trunk) in 10-20 % cases, a rare combination branch of the external carotid artery is a thyrolinguofacial trunk,[5] also reported about the presence of linguofacial trunk, thyrolingual trunk, thyrolingualfacial trunk in human fetus.[6] In the surgical literature, Catell, Phillips and Gorskie[7] had discussed the danger of injury atypically originating large cervical arteries during operation on thyroid gland.

These variations can pose a dangerous situation during surgeries like thyroidectomy, laryngectomy and other neck surgeries, preoperative selective arterial angiograms, in management of head neck tumors. So it is important for surgeons, radiologist to be aware of the variations among these arteries. Surgeons should be able to differentiate between the facial and lingual artery to insure accurate arterial ligation during oral and maxillofacial surgery and radical neck dissection. This knowledge can also help radiologist to understand and interpreted carotid system imaging.[8] The present study was undertaken to know the anatomy of the variation in the branching pattern of external carotid artery as-common liguofacial trunk and rare variation thyro-linguofacial trunk.

MATERIAL AND METHOD

Thirty properly embalmed (sixty sides), formalin preserved cadavers were selected for the study. The present study was carried out during 2006 to 2013 in the department of Anatomy, Muzaffarnagar Medical College, Muzaffarnagar (U.P). This dissection of head and neck carried out according to the instruction by Cunningham's manual of practical anatomy (Vol.3, 15th editions, 130-135). The dissection took place during the year 2006 to 2013. The meticulous dissection of external carotid artery was carried out in the carotid triangle and infratemporal fossa, clearly delineating its origin and all the branches.

RESULT AND OBSERVATIONS

During routine dissection for undergraduate students in the Department of Anatomy, MMC, Muzaffarnagar an unusual branching pattern of external carotid artery was observed in cadavers. Variations in the origin facial, lingual and superior thyroid artery from the external carotid artery on both sides was observed in cadavers. In present study we recorded one of thyrolingual facial trunk (common origin of superior thyroid, lingual artery and facial artery arises from anterior surface of external carotid artery on left side i.e. 3.3%) (Figure 1). The lingual and facial artery were originating (in two cadaver right side and in two cadaver left side i.e. 11.3%) as the common lingual facial trunk from the anterior side of external carotid artery, above the carotid bifurcation (Figures 2 and 3). The facial lingual artery trunk was running medially and upwards, which was crossed by hypoglossal nerve.

DISCUSSION

The location of carotid bifurcation, the branching pattern of external carotid artery and variations of the branch origins are known quite well. The branches of external carotid artery may arise irregularly or alter in number. When increase in number (by two or more), they arises as common stem, or by addition of branches not usually derived from this artery, such as sternomastoid branch of superior thyroid or occipital artery.[5]

There are reports in literature of origin of lingual artery from common carotid artery, lingual facial trunk or thyrolingualfacial trunk from external carotid artery. In present study one such case of thyrolingualfacial trunk was found which originated from anterior surface of external carotid artery (Figure 1) unusual case of origin of superior thyroid, lingual was also described by Arthur Thomson in his notes an Unusual variation[9] Budhiraja and Rastogi reported variable origin of thyrolingual trunk from right and left common carotid artery respectively[10] Ozur et. al.[6] classified the origin of these arteries which were arises from the external carotid artery in four types and reported their incidences. The separate origins of the arteries were defined type 1 (in 90% of the cases), the lingual facial trunk as type 2 (7.5%), thyrolingual trunk as a type 3 (2.5%) and thyrolingualfacial trunk as type 4.[11] Livini[12] observed the origin of superior thyroid artery in common with facial and lingual artery in 1.5 % of cadavers. The Thyrolingual trunk was found in 3.5% of cases by Shintani,[13] in 2% of cases by Gaillard[14] and Md Banna.[15] In present study we recorded one case of the common thyrolingofacial trunk i.e. 3.3% of cases arises from anterior surface of external carotid

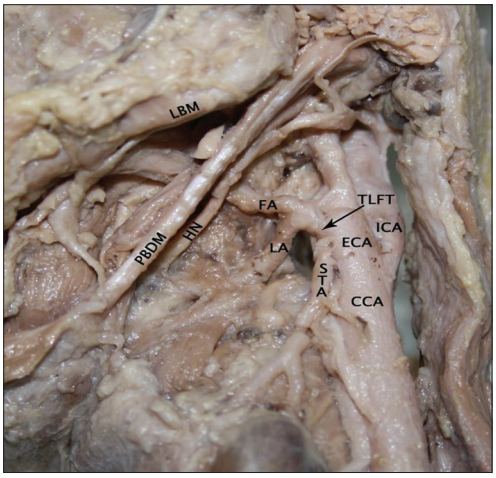

Figure 1: Variant left thyro-linguofacial trunk arising from external carotid artery (TLFT:Thyro linguo facial trunk, CCA: Common carotid artery, ICA: Internal carotid artery, ECA: External carotid artery, STA: Superior thyroid artery, LFT: Linguo facial trunk, LA: Lingual artery, FA: Facial artery, HN: Hypoglossal nerve, PBDM: Posterior Belly of diagastic muscle)

Figure 2: Variant Right linguofacial trunk arising from external carotid artery (CCA: Common carotid artery, ICA: Internal carotid artery, ECA: External carotid artery, STA: Superior thyroid artery, LFT: Linguo facial trunk, LA: Lingual artery, FA: Facial artery, HN: Hypoglossal nerve, PBDM: Posterior Belly of diagastic muscle)

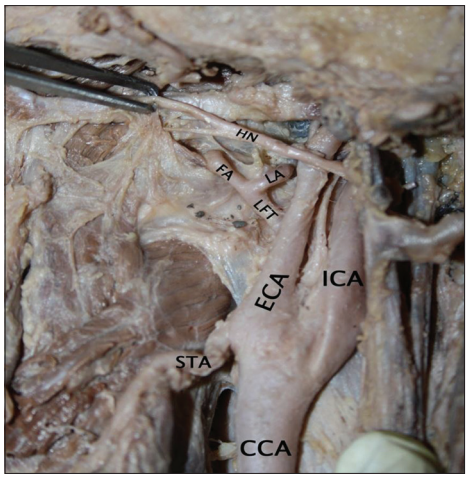

Figure 3: Variant Left linguofacial trunk arising from external carotid artery (CCA: Common carotid artery, ICA: Internal carotid artery, ECA: External carotid artery, STA: Superior thyroid artery, LFT: Linguo facial trunk, LA: Lingual artery, FA: Facial artery, HN: HypoglossaL nerve)

artery on left side. Zumre et al in their study on human fetuses found lingualfacial trunk in 20%, a thyrolingual turnk in 2.5 % and thyrolingualfacial trunk in 2.5% of human fetuses studied.

According to Anil A (2000)[16] the lingual artery arises from a common trunk with the facial as a linguofacial trunk in 10 to 20% of cases. Yildrim et al (2001)[17] also observed the total 6 (15%) linguofacial trunk in 40 neck side (20 adult human cadavers). Lappas et al (2002)[18] also found out linguofacial trunk in 14% cases and in a study conducted by Sanjeev et al,[19] the linguofacial trunk present in 18.92% cases. In present study it was 13.3%. Knowledge of variations of the external carotid artery and its branches and their recognition during diagnostic imaging are also important for vascular surgical procedure in the region, such as carotid endoplasty for the treatment of carotid stenosis[20-22] extra cranial – intracranial arterial bypass for treatment of patients with occlusive cerebrovascular disease, skull base tumors or aneurysms.[23] In our study, we found in four cadavers linguofacial trunk (11.3%) and in one cadaver thyrolinguofacial trunk(3.3%).

This present study to show differences in the branching pattern as compare to the available literature so far, which may be due to racial differences. This implies that these vessels show great variability (as given in Tables 1 & 2).

CONCLUSION

The branches of the external carotid artery are the key land marks for adequate exposure and appropriate placement of cross –clamp on the carotid arteries. The branches of the carotid arteries located in the carotid triangle are also the key land marks for adequate dissection on the carotid arteries and should be identified before cross clamps are placed and an arteriotomy is performed. So awareness of details and topographic anatomy of variations of the external carotid artery may be useful for both radiologists and vascular surgeons to prevent diagnostic error.

Table 1: Comparison of the prevalence of linguofacial trunk in different studies

Name of author	Year	Linguofacial trunk (%)
Ozgur et al.	2008	7.5
Lappas	2002	14
Yildirin et al.	2001	15
Sanjeev et al.	2010	18.92
Lucev	2000	20
Zumre et al.	2005	20 (In faetus)
Shintami	1999	31
Present study	2013	11.3

Table 2: Comparison of the prevalence of thyrolinguofacial trunk in different studies

Name of author	Year	Linguofacial trunk (%)
Jitender Patel	2011	1
Livini	1900	1.5
Takkallapalli Anitha et al.	2011	2
Zumre et al.	2005	2.5 (In faetus)
Present Study	2013	3

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Original Article

Improvement of Protein Energy Malnutrition by Nutritional Intervention with Moringa Oleifera among Anganwadi Children in Rural Area in Bangalore, India

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Abstract

Introduction: Protein energy malnutrition (PEM) is a major public health problem in developing countries.

Aims and Objectives: The study was conducted with the objectives of a) identifying children with Protein Energy Malnutrition, b) to give nutritional intervention in the form of Moringa Oleifera powder to the children for 2 months and c) to reassess the nutritional status after the nutritional intervention at the end of 2 months.

Materials and Methods: A before and after study was conducted in the rural field practice area of Vydehi Institute of Medical Sciences and Research Centre, Bangalore, India on sixty children, thirty in the intervention group and thirty in the control group. Nutritional Intervention was given in the form of Moringa oleifera leaf powder 15 g twice daily for two months. Reassessment of the nutritional status was done following the intervention.

Results: It was found that 70% children with grade II PEM improved to grade I, and 60% children with grade I PEM had shown significant ($P < 0.01$) improvement in their nutritional status.

Conclusion: Moringa Oleifera is a good malnutrition combatant and needs to be promoted in the community.

Keywords: PEM, Nutritional Intervention, Moringa oleifera

INTRODUCTION

Protein energy malnutrition (PEM) develops in children whose consumption of protein and energy is insufficient to satisfy the body's nutritional needs. While pure protein deficiency can occur when a person's diet provides enough energy but lacks the protein, in most cases the deficiency will be dual. PEM may also occur in persons who are unable to absorb vital nutrients or convert them to energy essential for healthy tissue formation and organ function. Malnutrition is a major factor in causing infant mortality in the tropics and sub-tropics. Current treatment for children involves the use of special formulated foods which are either labelled as F-100 or F-75 which is expensive and not sustainable in the long term.[1]

Experts have shown that the drumstick tree (Moringa oleifera) has improved the nutritional status of children with PEM. This tree grows abundantly in developing countries including India especially in the rural areas, where prevalence of malnutrition is high.

For children 1-3 years of age the daily requirements of calcium, 75% iron requirements and half of protein can be obtained in 100grams of fresh Moringa leaves. It is also rich in potassium, copper and B complex vitamins. Studies in (Senegal) as well as Indian medical research have proved the leaf powder to be effective in reducing nutritional deficiency such as vitamin A and protein deficiency.[2,3]

In depth studies regarding the nutrients have clearly shown that Moringa oleifera can be used as a food additive with

multiple purposes for enriching the protein, fatty acid, mineral and vitamins in human feed formulations.

The Moringa leaves are an excellent source of vitamin A, the raw leaves are rich in vitamin C and they also have vitamin B and other minerals. These vitamins and minerals are required for body building, energy as well as blood coagulation and production. The Moringa leaves rank among the best of perennial tropical vegetables as a source of nutrients and vitamins.[6]

Dr. Martin Price in his book *"The Moringa Tree"* reported the results of the administration of Moringa in various developing nations for treating the Protein Energy Malnutrition.

Moringa, added on a daily basis to a child's food, has thoroughly demonstrated its ability to bring about rapid recoveries from moderate malnutrition. While successfully treating malnutrition is good, preventing it is much better.[4-8]

Moringa oleifera tree has probably been one of the most underutilized tropical crops. Leaves of M. oleifera could serve as a valuable source of nutrient for all age groups. In some parts of the world for example Senegal and Haiti, health workers have been treating malnutrition in small children, pregnant and nursing women with Moringa leaf powder (Price, 1985).

In developing tropical countries, Moringa trees have been used to combat malnutrition, especially among infants and nursing mothers. Three non-governmental organizations in particular Trees for Life, Church World Service and Educational Concerns for Hunger Organization advocate Moringa as natural nutrition for the tropics.[9]

As there are few studies in India on nutritional supplementation with moringa oleifera and its effect among children suffering from protein energy malnutrition, the following study was taken up with the objectives of 1) to identify children with Protein Energy Malnutrition, 2) to give nutritional intervention in the form of Moringa Oleifera to the children for 2 months and 3) to reassess the nutritional status after the nutritional intervention at the end of 2 months.

MATERIALS AND METHODS

This study was conducted from 1 June-31 July 2013. Children with grade I and grade II protein energy malnutrition were identified and they participated in the study. Out of them 30 children were categorized as intervention group and 30 as control group. Severely sick children and children with chronic problems like congenital heart disease, asthma or renal problem and children with severe malnutrition of grade III and grade IV were excluded from the study.

All of them were de-wormed with Albendazole at the beginning of the study. Moringa leaves were harvested and dried at a low temperature (not under direct sunlight) ensuring the nutrients remained intact in the leaf tissue. Once dried, Moringa leaves were pulverized into a fine powder-like consistency, making them easy for usage. The Moringa leaf powder was added to salads, steamed vegetables, porridges or included in soups, curry, gravy, chapati, dosa or rice. Twice a day 15 g of Moringa leaf powder was added in the child's diet by the mother for a period of 2 months.

The intervention group was administered 30 grams of dry Moringa leaf powder in their diets every day. Every tenth day the weight was recorded for each of the intervention and control group. The weights of these children at the start of the study were compared with their weights at the end of the study after 2 months. Comparison was made between the recorded weights of the intervention and control group individuals.

Statistical analysis was done based on proportions and McNemar's modified Chi square test to find out if there was any significance in the weights of the children following nutritional intervention with Moringa leaf powder in their diets.

RESULTS

As depicted in Table 1, there was an improvement after the intervention with Moringa leaf powder in the intervention group as compared to the control group.

More than 40% improvement in weight was obtained in 3 children belonging to intervention group though none in the control group showed this much improvement.

30 to 40% improvement in weight was obtained in 9 children belonging to intervention group though none in the control group.

Table 1: Weight improvement between intervention and control group children

	Percentage improvement in weight	
% improvement	Intervention group	Control group
>40%	3	0
30-40%	9	0
20-30%	10	2
10-20%	5	10
<10%	3	18

20 to 30% improvement in weight was obtained in 10 children in intervention group and 2 children belonging to control group.

10 to 20% improvement in weight was obtained in 5 children of the intervention group and 10 children belonging to control group.

Less than 10% gain in weight was seen in 3 children belonging to intervention group and 18 children belonging to control group.

In the intervention group, out of 17 children with grade II PEM, 9 children showed minimum of 28% weight gain when compared to their initial weight at the start of the study. 52% in the intervention group improved from grade II to grade I protein energy malnutrition after intervention with Moringa leaf powder.

9 children out of 13 children identified as having grade I PEM, in the intervention group showed remarkable improvement in weight gain of around 30% after daily consumption of Moringa leaf powder.

In the intervention group, for each of the age group between 2 and 5 years, the average weight improved signigicantly ($P < 0.01$) as seen in Figure 2.

DISCUSSION

Moringa leaves are small, thick and tear-drop shaped. They grow rapidly as the plant matures and are easily available. Moringa leaf powder contains 8 essential amino acids for proper protein synthesis. It is rich in flavonoids, stacked with nutrients, anti oxidants and vital proteins, vitamins and various phenolics. As one of the rare trees whose leaves can be eaten as vegetables, the Moringa's nutrients are easily absorbed and no allergy has been reported.[3,4]

Most of the nutrients of the Moringa tree are in its dry leaves, which can be made into a powder that can be added to the regular diet in order to add essential nutrients.

In this study, the results observed after administration of Moringa leaf powder after 60 days, was that 70% children with grade 2 PEM improved to grade I, and 60% children with grade 1 PEM had shown significant improvement in their nutritional status.

Therefore Moringa leaf powder will be a good supplementation for combating PEM in under 5 children.

As Moringa is accessible to mothers at little or no cost, malnourished children treated with it tend to recover more rapidly than those whose mothers are obliged to follow the "modern" approach which involves purchasing expensive milk powder, cooking oil and sugar.

The major advantage of using Moringa leaves in this study is the fact that it is a local resource. Moringa leaves also are rich in vitamins and minerals such as, B-complex vitamins, vitamin C, calcium, potassium, magnesium, selenium, zinc and amino acids namely arginine and histidine which are especially important for infants.

The present study is akin to the studies conducted in West Africa. In 1996, the Church World Service office in Dakar began studying the potential of the Moringa oleifera tree to combat the problem of malnutrition, on a pilot project, Moringa products added on a daily basis to a child's food had thoroughly demonstrated its ability to bring about rapid recoveries from mild and moderate malnutrition.[9-11]

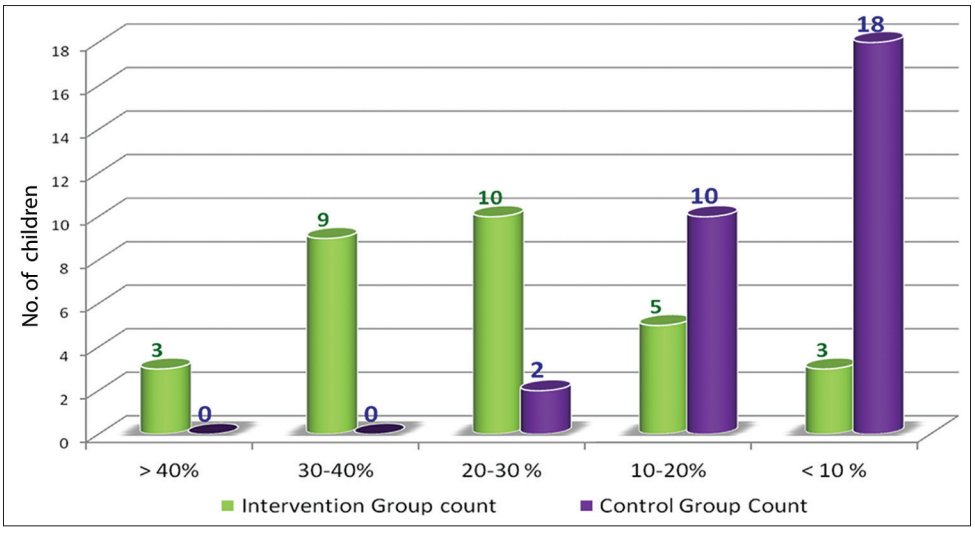

Figure 1: Percentage improvement of weight among intervention and control groups

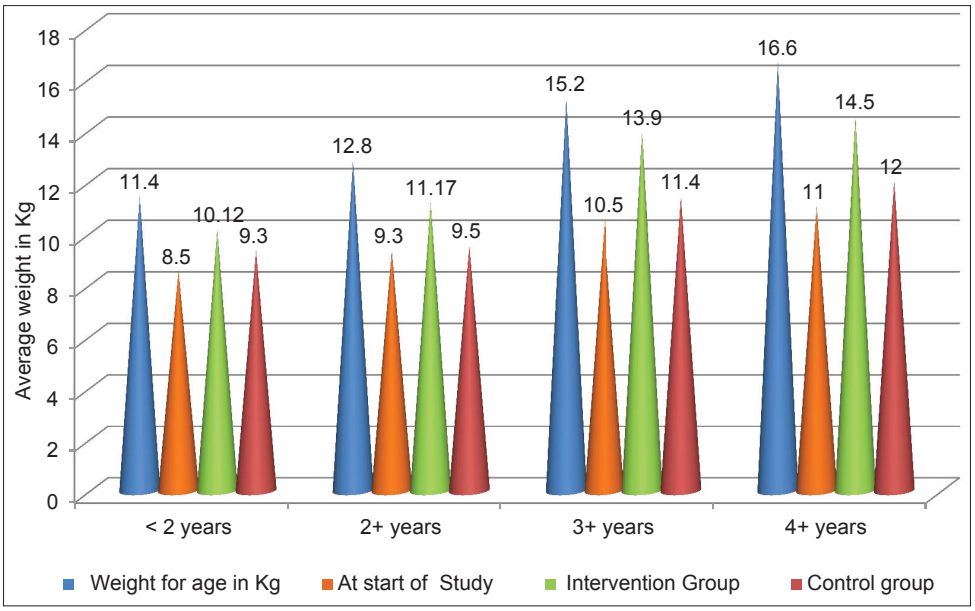

Figure 2: Age wise weight improvement among intervention and control group

CONCLUSION

Nutritional intervention with Moringa oleifera leaf powder showed significant weight gain among children with grade I and grade II protein energy malnutrition.

The Moringa leaf powder can be effectively utilized for treatment of PEM by spreading the awareness about the nutritional value of Moringa oleifera to mothers of children with PEM.

ACKNOWLEDGEMENT

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Case Report

Salvaging an Implant with Abutment Screw Fracture by a Custom Titanium Post and Core Supported Prosthesis - A Novel Technique

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Abstract

Screw loosening and its fracture is one of the most common mechanical complication of implant treatment. Retrieval of the fractured fragment is challenging when the fracture occurs below the head of implant or there is a damage to its internal threads. Many techniques have been described for the removal of the fractured segment from the screw hole. But when all the modalities fail to retrieve the segment or there is a damage to the internal threads during the process, the implant may be rendered useless. The clinician under such conditions might opt for removing the failed implant and replace it with a new one which would be an additional surgical trauma and financial burden to the patient. Thus salvaging the implant by other means appears to be a viable option in such situations. The management of a patient who had reported with fracture of an implant abutment screw by means of a custom cast post and core is presented.

Keywords: Abutment screw retrieval, Custom post and core, Damaged internal threads, Implant screw loosening, Implant screw fracture

INTRODUCTION

Dental implantation is a reliable and predictable treatment for partially and completely edentulous patients and is gaining tremendous popularity and interest amongst patients and dentists alike. With proper diagnosis and treatment planning, appropriate placement, adequate prosthetic design, and proper maintenance, dental implants can achieve a success rate of 97% to 99%.[1,2] The success of dental implants is based primarily on the extent of osseointegration.[3,4] However, many other factors also account for its failure. Amongst them Peri-implantitis and failure of implant-supported restorations are noteworthy. Failures of implant-supported restorations result from technical problems and can be divided into two groups: those related to the implant components, and those relating to the prosthesis.[5,6] Fracture of implant components may occur due to fatigue from biomechanical overload, improper placement techniques, non passive fit of the suprastructures,[7] or manufacturing errors.[8] Abutment failure due to fracture of its retaining screw is generally a challenge for the clinician due to the difficulty of removal of fractured screw fragments. A review of *in vivo* butt-joint implant studies reported abutment screw or prosthesis screw loosening as the most frequent mechanical complication.[9] In most circumstances, the fractured end can be retrieved and replaced by a new abutment screw. When the screw cannot be removed conservatively, rotary instruments can be used to retrieve the fractured screw. But it may damage the internal threads of the screw hole and the implant may be become useless. As a result, clinicians might choose to either remove the implant and replace it with a new one, or abandon the implant and cover it with soft tissue. This article describes a novel technique of salvaging an implant which suffered fracture of its abutment screw and damage of its internal threads.

CASE REPORT

A twenty four year old female patient reported to the Department of Prosthodontics, Govt. Dental College, Thiruvananthapuram with a chief complaint of dislodged crown in relation to an implant placed 5 years back in the upper anterior region (Figure 1). She noticed the mobility of the crown about three months back which gradually increased. But due to her personal commitments was unable to seek treatment on time. This was a typical case of screw loosening which eventually led to screw fracture (Figure 2). Intra oral examination revealed an implant supported crown at 11 with abutment screw fracture below the level of implant head. Radiographs revealed an osseointegrated endosseous screw form implant with the fractured screw fragment locked in the screw hole at around 5mm below the head of implant making its retrieval challenging (Figure 3). The other half of the fractured screw along with the abutment was in the dislodged crown. The fractured screw was visualized under magnification (×2.5). Internal threads of the screw hole appeared damaged under magnification (Figure 4). A fine ultrasonic endodontic tip was placed on the screw and vibrated at a low setting. There was no movement of the screw. Other methods of retrieval of screw fragment[10] was employed which also failed. This can be attributed to the damaged internal threads of the screw hole. The patient was unwilling to undergo extensive procedure of implant removal and placement of new implant and thus it was decided to use the screw hole as a channel for custom fabricated titanium post and core. The internal threads of the impant was eliminated (Figure 5) using a tungsten carbide bur (170 L) in a high speed air rotor handpiece under copious saline irrigation (Figure 6). Coronal fragment of the fractured segment was removed using a 8mm round ended tapered diamond and carbide bur to provide space for sufficient length of post which could resist the torsional forces. An acrylic resin pattern for the post was fabricated directly in the post space with an autopolymerizing acrylic resin and plastic pin using a brush-on technique (Figure 7). Care was taken to prevent locking of the pattern in the screw hole by removing it before it was completely polymerized. After the polymerization is complete, the pattern is replaced and the core portion is fabricated with resin, trimmed, finished and polished to appropriate length and shape (Figure 8). The pattern was sent to the lab for fabrication of titanium cast post and core (Figure 9). The titanium dowel core was tried in the screw hole (Figure 10). It was cemented into the implant using zinc phosphate cement (Phosphate

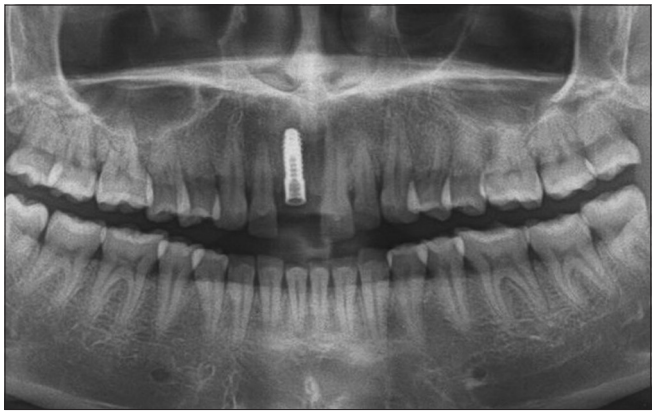

Figure 1: Oral pantamogram of the patient immediately after the placement of implant

Figure 2: Crown with the fractured abutment screw

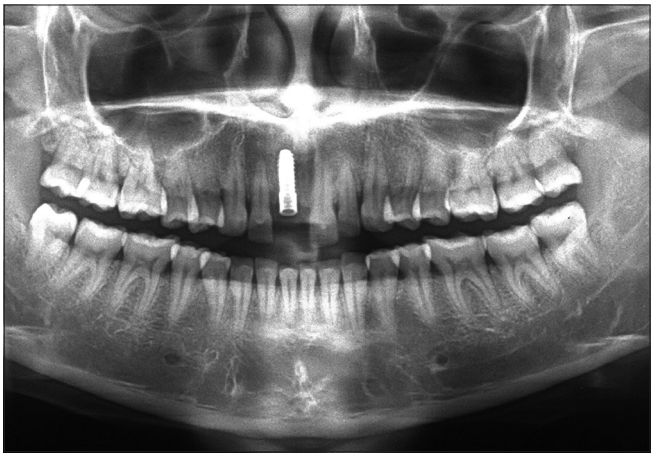

Figure 3: Oral pantamogram of the patient showing abutment screw fracture below thehead of implant

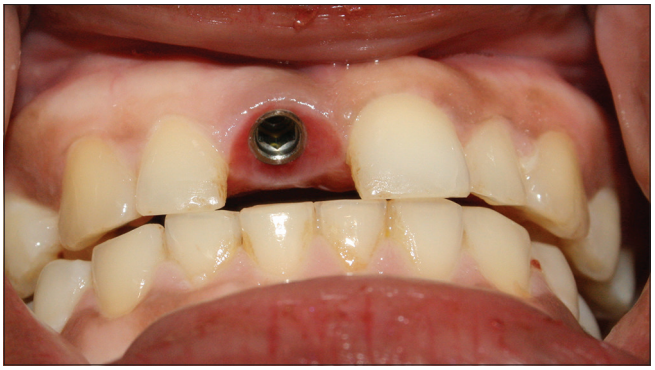

Figure 4: Damaged internal threads of the implant

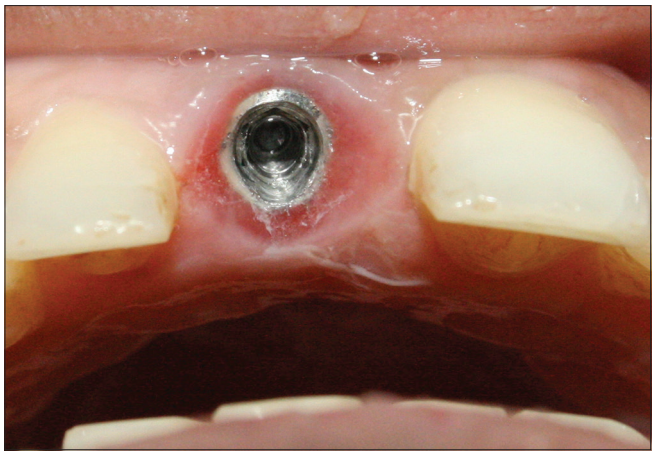

Figure 5: Prepared post space in implant by removing the internal threads

Figure 6: Post space preparation by removing the internal threads using tungsten carbide bur in a high speed air rotor under copious irrigation

Figure 7: Pattern for the custom post and core made using clear autopolymerizing acrylic resin

cement, Heraeus Kulzer, Germany). After cementation of the post and core, metal ceramic crown was fabricated following conventional prosthodontic procedure. The crown was cemented after eliminating the interferences in all protrusive and lateral excursive movements (Figure 11). The patient was followed up after six months with no signs of complications or failure of prosthesis (Figure 12).

Figure 8: Trial of the pattern

Figure 9: Custom post and core

Figure 10: Custom post and core cemented to the implant using zinc phosphate cement

Figure 11: Metal ceramic crown cemented over the cast core. Post operative intra oral view

Figure 12: Post operative extra oral view

DISCUSSION

Abutment screw fracture, although uncommon, occurs in clinical practice[6,11-12] and its removal can be quite challenging for the clinician. If an abutment screw fractures above the head of the implant, hemostats or artery forceps may be used to grasp the broken screw and remove it successfully. If the screw fracture occurs below the head of the implant or is stuck, other methods or systems can be employed to remove the fragment. Most of these systems involve drilling of a hole into the center of the broken screw followed by engaging a removal wedge into the broken screw. Reverse torque is then applied with the removal instrument. But if all the methods fail to retrieve the fractured segment or there is a damage to the internal threads of the implant screw hole, the implant may be rendered useless. In such a scenario a cast post and core supported prosthesis can salvage the near useless implant.

CONCLUSION

In this case report a relatively simple technique of salvaging an implant with abutment screw fracture and damaged internal threads has been described. The technique when executed with ultimate care and precision would provide excellent result. The non-invasive nature of the procedure would also be satisfying to the patient.

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Cystic Lymphangioma of Spleen: A Case Report

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Abstract

Lymphangioma, a very uncommon benign neoplasm, is seen in children and rarely in adults. It most commonly involves the neck (75%) and axilla (20%). It can occur sporadically in mediastinum, retroperitoneum, and internal organs. Splenic lymphangioma is a very rare condition and is usually found incidentally. The rate of malignant transformation is low, its prognosis is good. We report a case of cystic lymphangioma of spleen in a 47 year-old female presenting with abdominal pain. This case emphasis on rarity of the case at this age and the differential diagnosis with other cystic proliferation of spleen in particular hydatid disease.

Keywords: Cystic lymphangioma, Benign neoplasm, Spleen

INTRODUCTION

Cystic lymphangioma is benign neoplasm composed of malformation of lymphatic system.[1] They generally occur under the age of two years with no difference in incidence between males and females.[2] It most commonly involves the neck (75%) and axilla (20%). They can occur sporadically in mediastinum, retroperitoneum, and internal organs. Splenic lymphangioma is a very rare condition and is usually found incidentally.[3] Parasitic cysts are most common cystic proliferations of spleen. Non parasitic cysts are classified as primary or true cysts and pseudocysts. Amongst the true cysts, hemangioma are most common ones.[4] In the majority of cases, lymphangiomas have an asymptomatic course and despite the use of modern imaging techniques, often makes preoperative diagnosis difficult. Lymphangioma of spleen is extremely rare. To prevent complications such as infections, torsion, enlargement etc., total resection of tumor is done. The rate of malignant transformation is low, its prognosis is good. Here, we report a case of 47 year-old female presenting with abdominal pain.

CASE REPORT

A forty seven years old female presented with pain in abdomen since one month. Physical examination revealed enlarged liver till L5 subcostal region. Peripheral blood count, coagulation studies, liver and kidney function tests were all within normal limits. USG abdomen revealed large multilocular cystic mass in spleen. Clinically and radiologically it was diagnosed as hydatid cyst of spleen. A splenectomy was done and sent for histopathological examination.

Grossly, the spleen weighed 500 gm and measured 13 × 8 × 2 cm. Cut section revealed multiple variable sized cystic cavities involving almost the whole spleen. The largest cavity measuring 8 × 6 × 4 cm. The cavities were filled with gelatinous mucoid like material (Figure 1). On microscopic examination, the cysts were lined by endothelial cells and filled with acellular eosinophilic fluid (Figure 2). The cyst wall consisted of fibrous tissue with occasional calcification. Immunohistochemistry revealed D2-40 (Figure 3) and CD 31 positivity in endothelial lined cells and no or weak positivity with CD34. So the final diagnosis of cystic lymphangioma was made. The postoperative course was uneventful and the patient was discharged on the seventh day after the spleenectomy. The patient made complete recovery and free of disease two months postoperatively.

DISCUSSION

In 1885, Frink reported the first lymphangioma in the spleen.[5] Cystic lesions of spleen include parasitic and non parasitic cysts. Among parasitic ones, echinococcal disease represent 50-80% of the cases.[6] Non parasitic cysts are classified as primary or true cysts and pseudocysts. Most

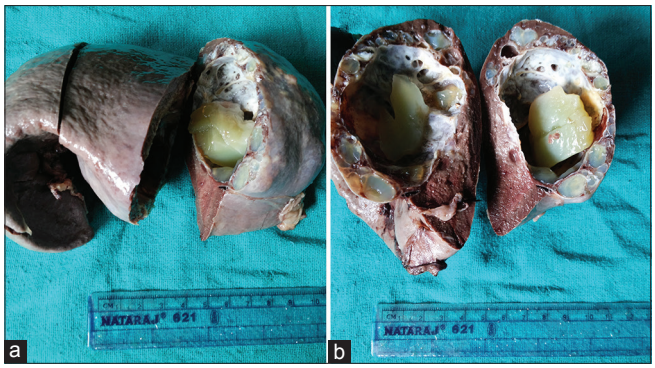

Figure 1: Spleen showing multiple cystic cavities filled with gelatinous mucoid like material

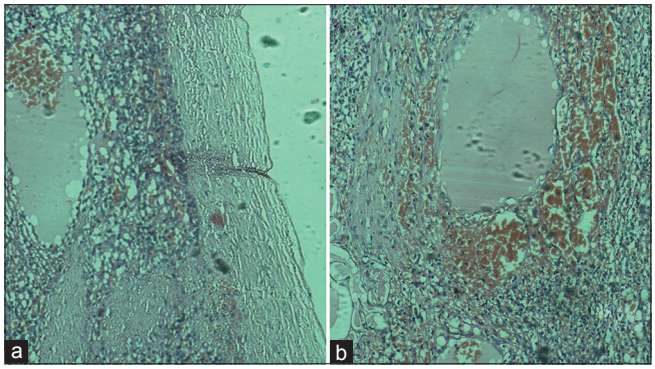

Figure 2: (a) Section showing fibrous splenic capsule over the cystic cavities (H&E, 100X), (b) Section showing cysts lined by flat endothelial cells and filled with acellular proteinaceous fluid (H&E, 200X)

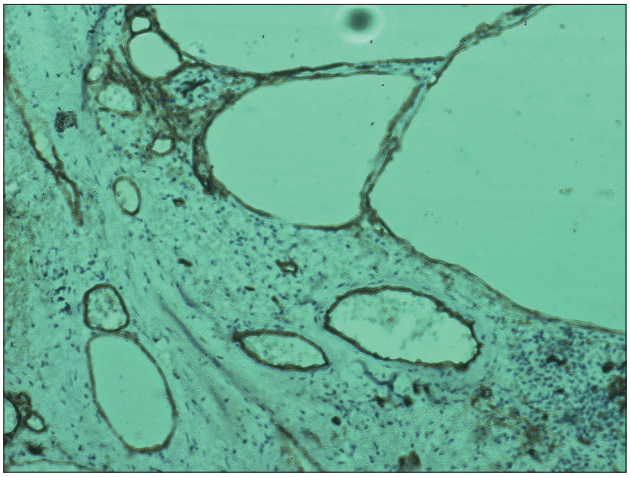

Figure 3: Section showing D2-40 positivity in endothelial lined cells (IHC, 200X)

cysts are post traumatic pseudocysts and true cysts are rare including hemangioma, lymphangioma, epidermoid and dermoid cysts.[4]

Lymphangioma is infrequently seen in mediastinum, adrenal gland, kidney, bone, omentum, gastrointestinal tract, retroperitoneum, spleen, liver and pancreas.[2,5] Opinions regarding histogenesis vary and a conclusive consensus has not been achieved.[7,8] They are considered to be developmental abnormalities due to either obstruction or due to obstruction leading to lymphangiectasias. The cause of obstruction of lymphatic system could also be due to bleeding or inflammation resulting in lymphangioma.[9]

Lymphangioma can be seen in the spleen alone, or it can be associated with multivisceral involvement; when diffuse it is termed systemic cystic angiomatosis.[10] Generally, lymphangioma is divided into capillary, cavernous and cystic types. The cystic type is the most common type.[1]

Patients with splenic lymphangioma present with upper left quadrant pain along with fever, nausea, vomiting and weight loss. Pain in left hypochondriac region was the presenting feature in our patient. Because of similarity of signs and symptoms clinically it is often confused with hydatid disease. Radiological findings are also not conclusive. Hence, histopathological examination is important in making diagnosis.[11] Hemangiomas are also an important differential diagnosis. Lymphangiomas show presence of flat endothelial lined spaces filled with eosinophilic proteinaceous material instead of blood and located in subcapsular area or larger trabeculae of spleen where lymphatics are normally concentrated while random localization is seen in case of hemangiomas. Immunohistochemically, the endothelial cells of lymphatic tissue show positivity for endothelial receptor-1, vascular endothelial growth factor-3, prox-1, and monoclonal antibody D2-40.[3]

Complications of splenic cysts include rupture with peritonitis as well as invasive hemorrhage, infection, abscess formation, pleural effusion or empyema.[4] Splenectomy is choice of treatment. Conservative management like aspiration, drainage and sclerosis are associated with high risk of recurrence,[1] while the prognosis is good.[12]

CONCLUSION

Though cystic lymphangiomas are uncommon entity, they should be considered in differential diagnosis of various types of splenic cystic masses in patients of any age presenting with abdominal lump, pain, nausea and fever.

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Case Report

A Rare Presentation of Ileal Perforation Secondary to Adenocarcinoma of Lung

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Abstract

Ileal perforation due to typhoid or tuberculosis is a well known entity. Ileal perforation secondary to metastasis from a primary is rare. We report a case of perforation in proximal ileum which was later diagnosed as metastatic deposit from a primary in the lung. A 35 yrs old male presented to the casualty with clinical picture of peritonitis, later confirmed as ileal perforation secondary to adenocarcinoma of the lung.

Keywords: Ileal perforation, Lung carcinoma, Metastasis from primary, Peritonitis

INTRODUCTION

Metastatic tumors involving the small bowel are much more common than primary neoplasms. The most common metastasis to the small intestine are those arising from intra-abdominal organs[1] Metastases from extra-abdominal tumors are rare but may be found in patients with adenocarcinoma of breast and carcinoma of the lung.[2] We report an interesting case of carcinoma of the lung with metastasis to the small intestine leading to perforation and present a review of the literature.

CASE REPORT

A 35 years old male patient presented to the casualty with complaints of sudden onset of acute abdominal pain and fever of one day duration. He has a history of smoking of 15 years duration.

On examination, abdomen was distended. There was generalised tenderness all over the abdomen, with local rise of temperature and guarding and rigidity. Obliteration of liver dullness was present. Bowel sounds were absent. X-Ray erect abdomen showed free air under right dome of diaphragm. X-Ray of chest reported as normal study except an evidence of old fracture of third rib on the left side. A provisional diagnosis of hollow viscus perforation was made and exploratory laparotomy was done.

Operative findings were 200 ml of purulent fluid and 0.5 × 0.5 cm perforation on the mesenteric side of proximal ileum approximately 100 cm from ileo-caecal junction. Thickening of ileal wall along with mesentery on either side of perforation was noted (Figure 1). Multiple enlarged lymph nodes were found in the mesentery. Liver and peritoneum were normal. Based on the intraoperative findings a differential diagnosis of carcinoma of small bowel, carcinoid tumor or ruptured gastro-intestinal stromal tumor (GIST) was made. Resection of ileum with 5 cm margin on either side of the perforation, with end to end anastomosis was done. The peritoneal cavity was irrigated with normal saline. The abdomen was closed after placing a pelvic drain.

The histopathological report showed moderately differentiated adenocarcinoma with multiple lymphatic emboli with involvement of serosa and also perforation (Figure 2). Post operative period was uneventful.

Patient was referred to medical oncologist for further management. He came back after 10 days with chest pain. ECG showed normal study. X-ray chest showed a suspicious opacity. CT scan of chest was done which showed lung malignancy – stage III B (Figure 3). A CT guided FNAC was done which showed evidence of adenocarcinoma of the lung. To further characterize the nature of the tumor, we carried out immunostaining of TTF-1 on the resected

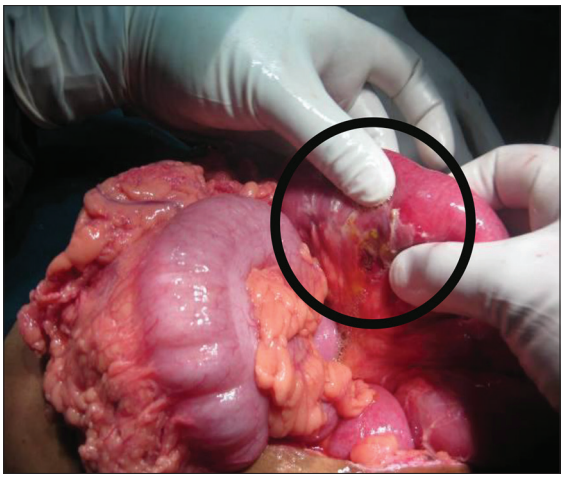

Figure 1: Perforation of ileum along with enlarged lymph nodes in the mesentery

Figure 3: CT Thorax showing lung malignancy in anterior segment of left upper lobe

Figure 2: Multiple secondaries in the small bowel

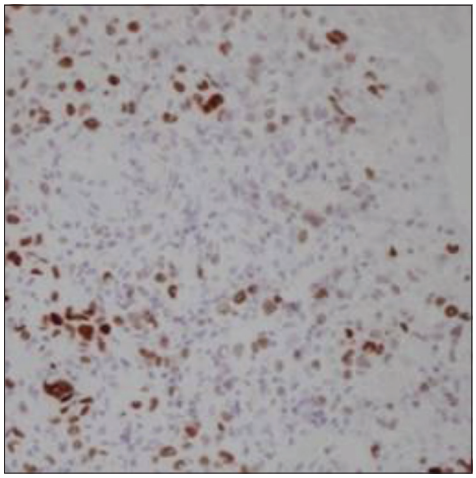

Figure 4: Tumor cells positive for thyroid transcription factor-1

specimen and it was positive (Figure 4). TTF-1 positivity is highly restricted to primary lung carcinoma and thyroid tumor. Since CT revealed lung tumor, we finally diagnosed this tumor as small bowel metastasis from primary lung adenocarcinoma.

DISCUSSION

Primary lung cancer often metastasizes to the brain, liver, adrenal glands and bone.[3] But metastasis to the digestive tract is rare.[4]

Small bowel metastasis from primary lung cancer exhibits symptoms such as abdominal pain and obstruction which are most common and others such as vomiting, melena, weight loss, gastrointestinal perforation, but most cases are asymptomatic.[5]

In normal tissue, TTF-1 is expressed in epithelial cells of thyroid and type II pneumocytes and Clara cells in lung. Carcinomas arising in lung and thyroid show TTF-1 expression frequently.[6] Thus, TTF-1 is a very good marker to determine the lung origin in small bowel metastasis.[7,8]

In one review of literature by Paul McNeil et al. from Virginia University in 1987, autopsy of 431 deaths due to lung cancer was done. 46 cases had deposits in small bowel and they often had lead to perforation.

In another review from world journal of gastroenterology 2005 by Davor Thomas et al. secondaries of small intestine are common than primaries. Small intestine carcinomas, especially when multiple, metastasis from lung should be first excluded, because it seems that they are more common than expected.

CONCLUSION

Small bowel metastasis from primary lung adenocarcinoma is rare and therefore difficult to diagnose. In such cases,

TTF-1 can be a very useful immunohistochemical marker to determine the lung origin.

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Case Report

Alveolar Ridge Augmentation using Autogenous Block Bone Grafts Harvested from Mandibular Ramus to Facilitate Implant Placement: A Case Report

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Abstract

With increased awareness on dental implants, more and more patients are presenting to dental practitioners requesting for fixed solutions to their dilemmas. The placement of endosteal implants requires adequate bone volume for successful osseointegration. When the morphology of the bone does not allow proper implant placement, there are various bone augmentation procedures which aids in reconstruction of the residual alveolar ridge for ideal implant placement. The mandibular ramus can act as an excellent source of autogenous bone for augmentation of alveolar ridge deficiencies. This article describes a case report of localized alveolar ridge augmentation using block bone autografts harvested from the mandibular ramus prior to implant placement.

Keywords: Autogenous bone grafts, Block bone grafts, Dental implants, Mandibular ramus, Ridge augmentation

INTRODUCTION

Successful implant surgery is not merely the achievement of successful osseointegration, but rather the establishment of an ideal foundation for implant-supported prosthetic restorations.[1] A major contraindication for dental implant placement is inadequate bone volume. Osseous defects may occur as a result of trauma, prolonged edentulism, congenital anomalies, periodontal disease and infection.[2] There are minimum dimensions that the remaining alveolar ridge must possess for implants to be placed. Based on clinical experience, the minimum dimensions in the maxilla to insert a dental implant are an alveolar ridge width of 5 mm and a bone height of 10 mm.[3] When these dimensions are not available, it will be necessary to augment the size of the alveolar ridge prior to implant placement using various grafting procedures. Without grafting, the implants may have to be placed in anatomically unfavorable positions or may have adverse angulations. These compromises can lead to unesthetic restorations, mechanical overload and ultimately failure of the implant. Various bone grafting techniques are available for reconstruction of alveolar deficiencies which include autografts, allografts and xenografts.[2] The success rates of grafted bone have been excellent to moderate but have varied more than for conventional implant treatment.[4] Among them, autografts have excellent osteoinductive properties and hence they are considered the gold standard in bone augmentation procedures.[5] This article presents a case report of localized alveolar ridge augmentation using block bone autografts harvested from the mandibular ramus prior to implant placement.

CASE REPORT

An eighteen year old female patient reported to the Department of Prosthodontics, Govt. Dental College, Trivandrum with the chief complaint of missing upper front tooth (Figure 1). She had lost her tooth in an accident 1 year back and was wearing a removable partial denture. All the treatment options were explained to her and she opted for implant supported restorations on 21 and 22.

On clinical evaluation, the gingival biotype was thick with adequate width of attached gingiva and favorable arch position. The clinical and radiological (panoramic and periapical) examinations revealed that the alveolar ridge height was normal, but there was a lack of alveolar ridge width. Labio-palatal atrophy of the edentulous alveolar ridge made it intricate to place implants on 21, 22 region. Hence it was decided to augment the alveolar crest horizontally. The mandibular ramus area was selected as the donor site for bone augmentation.

Pre-operative radiographs and diagnostic casts were prepared (Figure 2). The patient was healthy and had no systemic contraindications for intraoral surgery and implant placement.Surgical procedures were carried out as an outpatient procedure under local anaesthesia (2% lignocaine hydrochloride with epinephrine 1:2,00,000). A full thickness muco-periosteal flap was raised to expose and visualize the size of the defect, and the surface of the bone was released from the remaining muscle and periosteal fibers (Figure 3). Next, the bucco-palatal width and height of the alveolar bone were measured. The alveolar bone height was more than 10 mm. However, the width of the alveolar bone was about 4.1mm. After the extent of bone loss was outlined at the recipient site, we proceeded with the donor site exposure. A surgical marking pen was used to outline area of the vestibular incision at the ramus region. A full-thickness mucoperiosteal incision was made distal to the most posterior tooth in the right mandible continuing to the retromolar pad and ascending ramus. An oblique releasing incision to the depth of the vestibule was given. Three complete osteotomies and one bone groove was prepared using a 702L straight fissure bur before the graft harvest (Figure 4). The order of the osteotomies proceeded as superior cut, followed by anterior, posterior, and the inferior cut. Exposure of the recipient site and the donor site permitted direct measurement of the bony defect and available bone at the donor site. The bone block was carefully loosened and lifted from the donor bed using conventionally designed instruments. Before placing the autogenous graft,

Figure 1: Pre-operative intra oral view

Figure 2: Pre-operative Oral Pantamogram

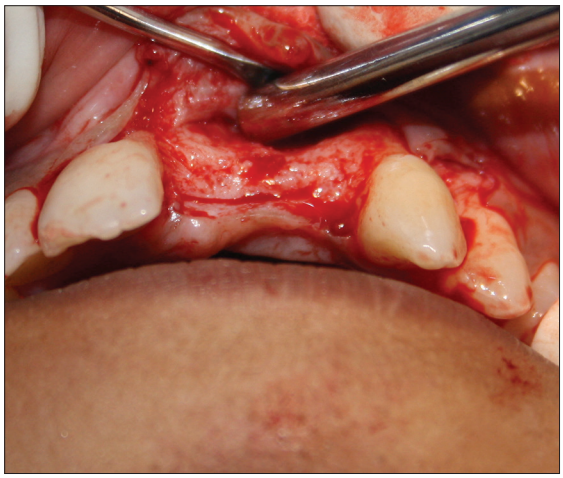

Figure 3: Muco-periosteal flap raised to expose the bone at the recipient site

Figure 4: Muco-periosteal flap raised to expose the bone at the donor site

recipient site was prepared for predictable incorporation of block grafts.[6] The preparation involved decortication and perforation into underlying bone marrow which accelerated revascularization of the graft. The block graft obtained from ramus was also prepared to allow intimate contact with the recipient site to facilitate graft incorporation. Titanium screws of 1.5 mm diameter and 6 mm length were used to stabilize the graft onto the recipient area[4] (Figure 5). A pilot hole was drilled through the graft onto the recipient site and enlarged to allow the placement of a titanium fixation screw without resistance. After fixing the autogenous block bone graft onto the recipient area with a titanium screw, small gaps at the edges of the autogenous bone graft were filled with hydroxyapatite bone grafting material. The graft material was stabilized with an absorbable collagen membrane for guided bone regeneration. Finally the periosteum of the mucoperiosteal flap was relieved at its base to mobilize the flap and allowed to cover the bone graft without any tension. The patient was placed on analgesics, antibiotics, and an antimicrobial mouthrinse for 1 week. Temporarisation were done using a customized fixed composite bridge for esthetics and to aid in adequate graft stability.

The postoperative clinical and radiographic examination showed an increase in the width of alveolar ridge at the grafted site (Figure 6). The site was re-entered after 6 months for removal of the fixation screw and placement of the implants (Figure 7). Under local anesthesia, a mucoperiosteal flap was raised to expose the recipient area. 3.3 × 13 mm implants (Adin) were planned for 21 and 22 regions. It was seen that there was minimal resorption around the screw and the width of the alveolar bone was measured as 6.8 mm. The screw stabilizing the graft was removed with a screw holder and two implants of size 3.3 × 13 mm dimensions were placed in a conventional manner (Figure 8). Cortical perforation caused by the stabilization screw was filled with hydroxyapatite bone grafting material and the graft material was stabilized with an absorbable collagen membrane for guided tissue regeneration. Four months after the second stage surgery, periapical radiographs showed that osseointegration had been completed successfully (Figure 9). During the prosthetic phase, healing abutments were placed to achieve an esthetic soft tissue emergence profile. After stabilization of gingival tissues, implant level impressions were made using open tray impression copings and a master cast was

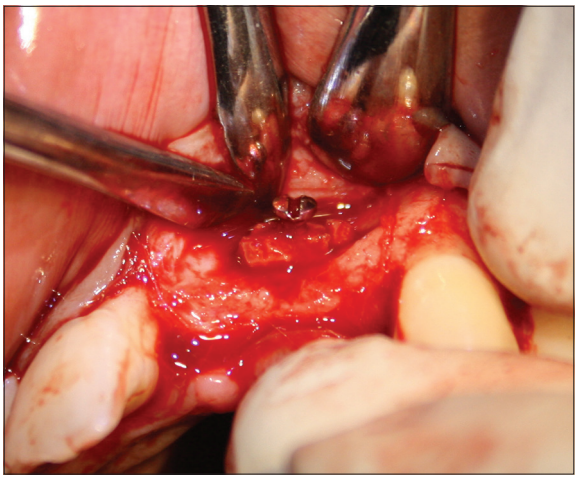

Figure 5: Ramus graft stabilized using titanium screws

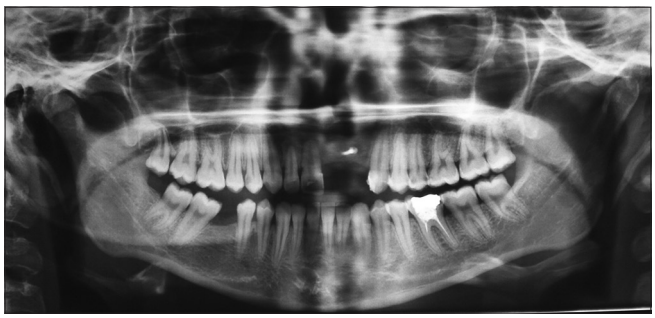

Figure 6: Post-operative oral pantamogram showing titanium screw stabilizing the graft

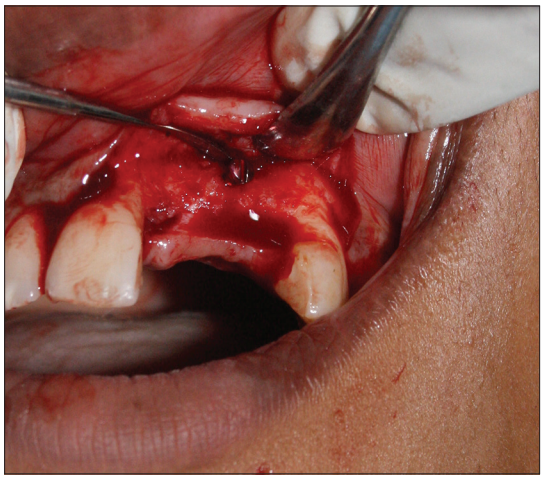

Figure 7: The recipient site re-entered after 6 months

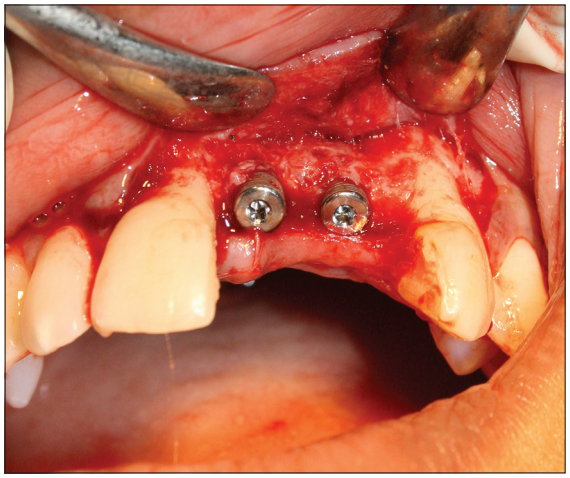

Figure 8: Placement of implant fixtures

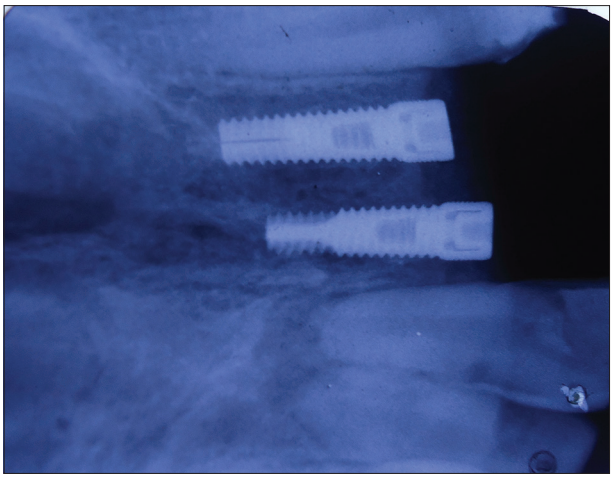

Figure 9: IOPA

Figure 10: Post operative extra oral view

fabricated with implant body analogues. The casts were mounted on an articulator. The abutment preparation was done and the implant crowns were manufactured. The metal porcelain crowns were finished and cemented on to the implants using glass ionomer cement (GC Fugi CEM, GC Corporation, Tokyo, Japan) (Figure 10). Finally, a thorough inspection was performed to ensure that the peri – implant sulcus was free of remaining cement particles hence prevent any foreign body reactions.

DISCUSSION

Esthetic and functional compromises in implant restorations can be prevented by ridge augmentation procedures which results in enhanced emergence profile for an implant supported restoration. A thorough clinical and radiological examination should be done in order to diagnose the exact quantity of bone loss and accordingly plan for various bone augmentation procedures. Autogenous bone grafts are recommended in bone augmentations prior to implant placement because of their osteogenic potential.[7] Intramembranous autogenous osseous grafts including the mandibular ramus, mandibular symphysis, angle of mandible, maxillary tuberosity and intraoral exostoses, are the "gold standard" for improving intraoral osseous volume to facilitate placement of implants.[8] Alveolar defects can be restored by autologous grafting techniques including corticocancellous blocks, compressed particulate cancellous bone and marrow, and cortical grafts. Block grafts are associated with minimal resorption and do not usually require the use of an overlying membrane unless the dimensions of the graft are inadequate. Block grafts take longer to integrate than cancellous bone grafts. When a block graft is used, a staged surgical approach is recommended as opposed to placing the implants in conjunction with the graft.[9] The mandibular ramus is a useful, cortical graft that provides primarily dense cortical bone and high concentration of promoter proteins (eg, bone morphogenetic proteins). In addition, the mandibular ramus donor site is associated with fewer postoperative complications, in comparison to the symphysis region.[6,10-13] Hence they can be successfully used for alveolar ridge augmentation prior to implant placement.

CONCLUSION

This article presents a case of alveolar ridge augmentation in a partially edentulous patient prior to implant placement, using autogenous bone grafts harvested from the mandibular ramus and firmly secured to the recipient site with osteosynthesis screws. The clinical indication for the case described was the lack of sufficient alveolar bone quantity, a situation that could interfere with the esthetics and functional loading of implants. The mandibular ramus block bone grafts gives predictable outcome within a short healing time and provides ideal sites for endosseous implant placement.

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Case Report

Tolosa Hunt Syndrome: Reported From West Bengal, India

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Abstract

Tolosa Hunt Syndrome (THS) is a rare cause of painful ophthalmoplegia. It is caused by nonspecific inflammation of the cavernous sinus or superior orbital fissure. Our article describes a case of a middle aged female who presented with retro orbital pain, diplopia and third, fourth and sixth cranial nerve palsy. She had no other neurodeficit. Her CT & MRI scan was normal. We treated her with systemic steroids because of the suspicion of THS and the patient recovered dramatically. This is probably the first reported case of Tolosa Hunt Syndrome with normal neuroimaging from West Bengal, India.

Keywords: Normal MRI, Painful ophthalmoplegia, Tolosa hunt syndrome

INTRODUCTION

Recurrent painful ophthalmoplegia is a rare condition first described by Tolosa in 1954 in a male patient who had died soon after an operation to explore the sella turcica for left retro-orbital pain and ophthalmoplegia. The autopsy demonstrated nonspecific granulomatous inflammation in the cavernous sinus, surrounding the intracavernous portion of the left internal carotid artery and cranial nerves III, IV, ophthalmic division of V, and VI.[1] In 1961 Hunt et al. reported six cases with similar clinical findings and called this syndrome painful ophthalmoplegia. The authors proposed the clinical criteria for the diagnosis of this condition.[2] In 1966, Smith and Taxdal were the first to apply the eponym "Tolosa-Hunt syndrome" to this entity and they emphasized the dramatic response of the symptoms to systemic steroid therapy.[3] Although newer imaging modalities like CECT and MRI (also MR angiography and venography) with special attention to cavernous sinus, superior orbital fissure and orbital apex can detect several abnormalities in patients with THS but few studies revealed normal imaging finding.[4] In this article we described a patient with clinical features of Tolosa Hunt Syndrome with normal imaging.

CASE REPORT

A 34 years old female patient presented with painful ophthalmoplegia in right eye and diplopia for last 5 days. She had no other complaints or any systemic symptoms. She had a past history of painful diplopia 7 months ago. Those symptoms resolved spontaneously after few weeks without any medication. That time Ophthalmoscopic examination and CT scan was normal.

On examination, she had right sided 3rd, 4th and 6th cranial nerve palsy (Figure 1). Right sided pupil slightly larger in size than the left one. Ophthalmoscopic examination revealed no abnormality. There was no other neurological impairment. She was normotensive. Other systemic examinations were normal.

Laboratory investigations revealed normal CBC, euglycemia, euthyroid. Serum ANA and ACE were negative, ICTC was also nonreactive. CSF study was normal.

Both plain and Contrast Enhanced CT scan were normal. MRI with MR Angiography was also normal (Figures 2 & 3). During MRI study special attention was given to cavernous sinus, superior orbital fissure and orbital apex.

Based on the clinical findings a diagnosis of Tolosa Hunt Syndrome was made and Methylprednisolone at a dose of 32 mg OD for 7 days and then gradual tapering was done. There was dramatic improvement of pain within 3 days. Ptosis and diplopia almost corrected after 2 weeks of therapy (Figure 4).

DISCUSSION

Tolosa Hunt Syndrome (THS) is a rare painful ophthalmoplegia caused by nonspecific inflammation of the cavernous sinus or superior orbital fissure and is responsive to steroid therapy. THS can affect people of age group of 1st to the 8th decades of life, with no sex or side predilection. Uniformly, patients complain of pain, which is a defining symptom. The pain lasts an average of 8 weeks if untreated. Ocular motor cranial nerve palsies may coincide with the onset of pain or follow it within a period of up to 2 weeks.[5] All three ocular motor cranial nerves may be involved, in various combinations. Pupillary reactions may be normal. Since Tolosa[1] described a case of periarteritis of the cavernous carotid artery creating a painful ophthalmoplegia in 1954 there has been considerable interest in THS. In 1961, Hunt et al.[2] outlined six clinical criteria characterizing the syndrome: 1) steady, gnawing or boring retro orbital pain; 2) defects in the IIIrd, IVth, VIth, or 1st branch of the Vth cranial nerve, with less common involvement of the optic nerve or sympathetic fibres around the cavernous carotid artery; 3) symptoms lasting days to weeks; 4) occasional spontaneous remission; 5) recurrent attacks and 6) prompt response to steroid therapy.

Initial radiographic evaluation consisted of carotid artery and superior ophthalmic vein angiography, which often demonstrated narrowing of the carotid siphon or thrombosis of the superior ophthalmic vein and/or cavernous sinus. However, a normal orbital venogram or arteriogram does not exclude THS, and one series found

Figure 1: Patient-before treatment

Figure 2: MRI-Coronal & axial view, showing normal cavernous sinus, superior orbital fissure and orbital apex of the patient

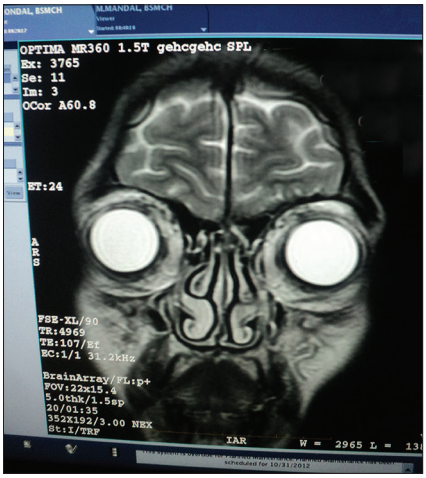

Figure 3: MRI-Coronal & axial view, showing normal cavernous sinus, superior orbital fissure and orbital apex of the patient

Figure 4: Patient-after treatment

no vascular abnormality in 16 of 26 cases.[6] High resolution CT can also demonstrate soft tissue changes in the region of the cavernous sinus/superior orbital fissure, but is less sensitive than MRI. Contrast enhanced MRI with multiple views; particularly coronal sections demonstrated an area of abnormal soft tissue in the region of the cavernous sinus in most, but not all, patients with THS. Typically, the abnormality is seen as intermediate signal intensity on T1 and intermediate weighted images, consistent with an inflammatory process. There is enhancement of the abnormal area after intravenous injection of paramagnetic contrast. With corticosteroid therapy, the abnormal area decreases in volume and signal intensity in most reported cases.[7]

Yousem et al[4] examined 11 patients and reported pathological MRI findings (abnormal signal and/or mass lesions) in the cavernous sinus in nine. Two patients had normal MR studies of the orbit and cavernous sinuses just like our case. In eight cases the abnormality was hypointense relative to fat and isointense with muscle on T1 weighted images; isointense with fat on T2 weighted scans.

The clinical differential diagnosis[8,9] of steroid responsive painful ophthalmoplegia includes metastases, carotid-cavernous fistulae, pituitary adenomas, vasculopathic cranial neuropathy, aspergillus invasion, Wegener's granulomatosis, sarcoidosis, lymphoma and ophthalmoplegic migraine. Meningiomas and aneurysms may rarely cause pain when of sufficient size. While metastases, pituitary adenoma, aspergillus infection, some meningiomas and some cases of lymphoma are often hyper intense relative to fat on long TR images; Sarcoidosis, lymphoma and meningiomas may display hypointensity or isointensity on short TR/TE and long TR/TE sequences as in THS. However, sarcoidosis & lymphoma will often have systemic symptoms and meningiomas will not resolve with steroid therapy. Vascular abnormalities such as arteritides, carotid-cavernous fistulae, ophthalmoplegic migraines and aneurysms are not associated with masses in the cavernous sinus or orbital apex as in THS.

CONCLUSION

In the appropriate clinical setting of painful ophthalmoplegia, MR findings of a cavernous sinus abnormality and a prompt response to steroid therapy, THS need not merely be a diagnosis of exclusion, although other lesions may have similar intensity characteristics, a small percentage of patients with THS may have lesions not detectable with current imaging techniques.

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Case Report

Pleomorphic Adenoma of the Palate: Report of a Case

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Abstract

Pleomorphic adenoma is a most common benign tumor which affects the major salivary glands and infrequently arises from minor salivary glands. It is a mixed tumor of salivary gland origin and has elements from both epithelial and mesenchymal tissues. In this case report we are presenting a case of pleomorphic adenoma of hard palate in a 24 year old female patient who reported to our department with complaint of pain less swelling in the palatal region since one year.

Keywords: Minor salivary gland tumor, Palate, Pleomorphic adenoma

INTRODUCTION

Pleomorphic adenoma (PA) is a benign salivary gland tumor which represents about 3-10% of neoplasm of the head and neck region.[1-3] They are most common salivary gland tumor occurring mainly in parotid and sub mandibular salivary gland.[3-4] As far as intra oral salivary gland are concerned, palate (42.63%) is a most commonly affected site followed by lip (10%), buccal mucosa (5.5%), retromolar area (0.7%) and lastly affecting the floor of the mouth.[1,2,5] It is also called as mixed salivary gland tumor because of its dual origin from the epithelium and myo-epithelial cells.[5] PA usually present as a mobile slow growing painless firm swelling that does not causes ulceration of the overlying mucosa. But these tumors are known to cause underlying bone erosion.[2,3]

CASE PRESENTATION

A 24 yrs. old female patient reported to us with the chief complaint of non painful swelling over the right palatal region since last 1 yr. The swelling was slow growing non tender and do notinterfere with speech mastication or swallowing. Her past medical and family history werenoncontributory. On taking dental history she revealed that she went to a local dentist for the same complain and got her decayed maxillary 1st molar extracted. But as there was no difference in swelling, she reported to institution.

Her intra oral examination revealed a single oval shaped, circumscribed lesion which approximately measures 2 × 3 cms, extending from 5-6 mm from the marginal gingiva in relation to right maxillary second molar till the mid palatine region. The over line mucosa was not ulcerated but was stretched and appears to be more shining in comparison with other aspects of the palate. The lesion was firm and fixed to underline structure (Figure 1). There was no regional lymphadenopathy and nasal examination was within normal limits. The radiography of maxilla by occlusal radiograph and CT Scan (Figure 2) did not show any evidence of bony invasion or perforation.

A differential diagnosis of odontogenic cyst/minor salivary gland tumor were considered. Other lesions like kaposi's sarcoma, syphilitic gumma and intra oral molluscumcontagiosum were also consider. Fine needle aspiration cytology (Figure 3) suggested benign tumor with features characteristic of PA.

TREATMENT & FOLLOW UP

The patient was operated under GA. After nasotracheal intubation, mouth gag was placed in the opposite side of the posterior molars to increase the access for the lesion in the palate. A good visibility and accessibility is the key for complete excision of the lesion. Local anesthetic solution containing 1:200000 adrenaline was infiltrated around the lesion to achieve Vasoconstriction. Mucosa around the lesion was marked & wide excision of the lesion including the periosteum was done (Figure 4) with surgical blade & dissecting scissors. Hemostasis was achieved with electrocautery. The residual site was covered with periodontal pack. Dressing was removed 4 days post operatively. Regular oral irrigation was done with Chlorhexidine to maintain good oral hygiene. In 3-4 week time the donor site granulated & healing was uneventful.

The excised mass (Figure 5) was sent for histopathological examination which further confirmed our diagnosis.

Figure 1: Intra oral view of swelling in the right side of the plate not crossing the midline

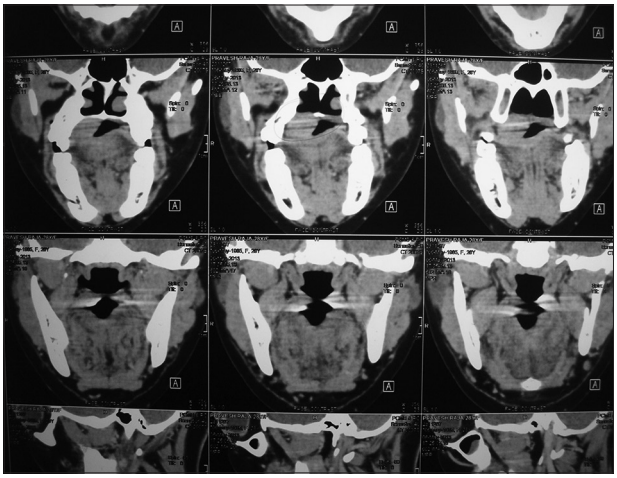

Figure 2: CT Scan reporting no erosion or perforation of palate

Patient was on follow up for one year without any sign of recurrence (Figure 6).

DISCUSSION

There are numerous malignant and benign tumor arises from major and minor salivary gland. PA is a most common benign tumor of salivary gland whereas mucoepidermoid carcinoma is a most common malignant counterpart to be encountered in maxillofacial region.[2]

Spiro RH[6] in his study of 2078 patients with salivary gland neoplasia reported that 20-40% of all salivary gland tumors arise from minor salivary glands. The mixed minor salivary tumors affects mostly patients in their fourth o sixth decades of life.[3,4] Though it has been reported to affect both the sexes, slight predilection for female gender has been reported.[5,8]

Intraoral PA appears as unilateral slow growing non tender firm mass that may become large if untreated. Though it

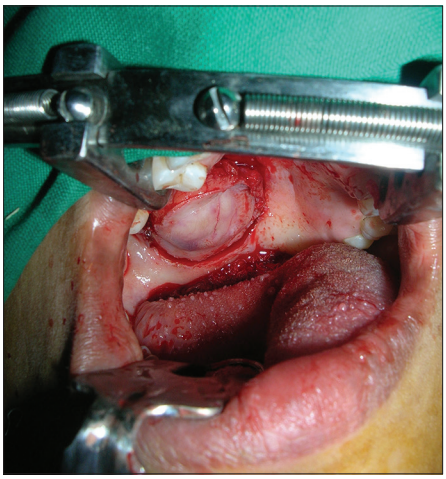

Figure 3: Wide excision of the lesion

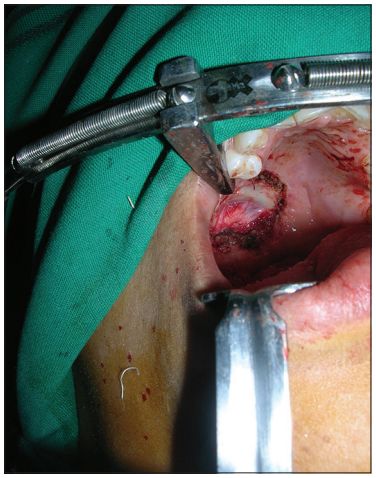

Figure 4: Intra operative view

Figure 5: Excised specimen

Figure 6: Follow up after one year post operatively showing healing

is a benign tumor, it has been reported of having locally aggressive behavior due to lack of the presence of fibrous capsule. These tumors also invade & erode adjacent bone causing radiolucency & mottling on the X-rays of the maxilla.

In the present case the patient also complained of unilateral slow growing non tender swelling in the junction of hard & soft palate.The diagnosis of PA is established on the basis of history, physical and histopathological examination.Plain X-ray and hematological investigation plays no part in the diagnosis of minor salivary gland tumor.[2] Radiograph of maxilla like occlusal view helps by showing the extent of bony erosion or tumor invasion. C.T scan may be helpful in evaluating the erosion of the palate and assess the extension of tumor into the nasal cavity or to the sinus. A histopathological diagnosis is essential for a confirmatory diagnosis.

The differential diagnosis PA includes palatal abscess, odontogenic and non odontogenic cyst, soft tissue tumors like lymphoma, lipoma, fibroma as well as other salivary gland tumors. In the present case, presence of a nonvital maxillary right first molar adjacent to the lesion might be the cause of misdiagnosis by the dentist.

Histopathologically the tumorare composed of island of stellate and spindle cell that are interspersed in a myxoid background.

Simple enucleation of the tumor has been reported with high recurrence. Therefore the treatment of benign minor salivary gland tumors is wide surgical excision[1,2,4,7] with removal of periosteum and under lying bone if found to be involved.[7,8] Many authors had advocated wide surgical excision with curettage of the underlying bone with a surgical curette or bur.[2]

Reconstruction of the palate should be considered for functional and aesthetic point of view.The soft tissue defect of the palate can be left to granulate, whereas the hard tissue defect can be corrected with the help of obturator. In the present case, the patient did not require any reconstruction as the palatal mucosa regenerated without any formation of fistula.

CONCLUSION

Since the majority of minor salivary gland tumors are reported to be malignant, careful history, patient evaluation, histopathological and radio imaging is advised. With adequate surgical excision the tumor usually does not recur, but most recurrences can be attributed to inadequate surgical technique. A long term follow up is warranted because of the recurrence even after several years of initial excision.

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Case Report

Ebstein's Anomaly - A Long Innings

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Abstract

Ebstein's anomaly is a rare disorder with a reported incidence of 0.5% or less among patients with congenital heart disease. Ebstein's anomaly surviving for seven decades patients surviving after 50 years of age is < 5% but without symptoms is rare. Our patient presented with symptoms in the seventh decade only. Surviving till seventh decade is rare but surviving without symptoms is rarest of rare cases.

Keywords: Ebstein's anamoly, Seventh decade, Symptoms

INTRODUCTION

Ebstein's anomaly is a rare disorder with a reported incidence of 0.5% or less among patients with congenital heart disease. Ebstein's anomaly surviving for seven decades patients surviving after 50 years of age is <5%. Anomaly of tricuspid valve very rare, accounting for <1% CHD. Incidence 1:2,00,000 live births. Apical displacement of septal and posterior tricuspid leaflets with dysplasia of valve. Inter atrial communication in 80-95% of patients. Cardinal symptoms being Cyanosis, right heart failure, arrhythmias, sudden cardiac death. Reports of patients surviving to adulthood with symptoms are very few! ECG-Himalayan p-waves, RBBB, Splintered QRS complexes, WPW Syndrome first degree heart block. Chest X Ray Vary from normal to typical globe shaped heart with narrow pedicle, cardiomegaly, normal or low pulmonary vascularity. 2D Echocardiography-Gold standard for diagnosis. Apical displacement of septal leaflet from insertion by atleast 8 mm. Dilation of right atrium, atrialised portion of right ventricle. Pulmonary artery pressure is typically low. Patients surviving after 50 years of age is <5%.[1-4]

CASE HISTORY

A 65 year old male patient, previously asymptomatic, presenting with recent onset of breathlessness and swelling of both lower limbs. Not a known hypertensive or diabetic. He was chronic smoker for 45 years.

GPE: Moderately built and nourished, conscious, oriented. Pulse - 72 beats/min, Regular, Normal Volume. BP - 120/80 mm Hg in upper limbs, 130/90 mm Hg in lower limbs.

Respiratory rate - 22 cycles/min. Grade 2 Clubbing-carpopedal (Figure 1), Jugular Venous Pressure - normal. Bilateral pitting pedal oedema present-upto knee. Central Cyanosis present.

Post-axial polydactyly of right hand present (Figure 1). No Pallor, No Icterus, No Lymphadenopathy.

Height-167 cm, Weight-59 kg, BMI-21.14 kg/m^2.

Cardiovascular system: Apical impulse in left 5th intercostal space, 2.5 cm lateral to midclavicular line, hyperdynamic. Multiple heart sounds in mitral and tricuspid areas with split S1. Pansystolic murmur of grade 3/6 in left parasternal area. Split S2 at pulmonary area, P2 normal.

Respiratory system: Normal vesicular breath sounds heard.

Abdomen: Soft, bowel sounds + No Organomegaly.

Central Nervous System: No neurological deficits.

Routine Investigations

Hb - 14 g%, TLC – 6400 cells/cumm, DC - N^{78}, L^{22}, ESR - 12 mm/hr.

Blood Urea - 39 mg/dl, Serum creatinine - 1.1 mg/dl.

Serum electrolytes - Na - 137, K - 4, Cl - 106.

Urine routine - normal.

Chest X ray PA View - Cardiomegaly with enlarged right atrium, normal vascularity of lung fields.

2D Echocardiography (Figure 2) - Congenital heart disease, Ebstein's anomaly of tricuspid valve, displaced STL by 5 cm, large sail like ATL. Dilated RA and RV with RV dysfunction, hypoplastic pulmonary artery, and severe low pressure TR (PASP-27 mm Hg). Normal left ventricular function.

ECG (Figure 3) - Sinus rhythm with QRS Axis of +60, normal p waves, prolonged PR interval, complete right bundle branch block, splintered QRS Complexes.

Coronary angiogram: Normal.

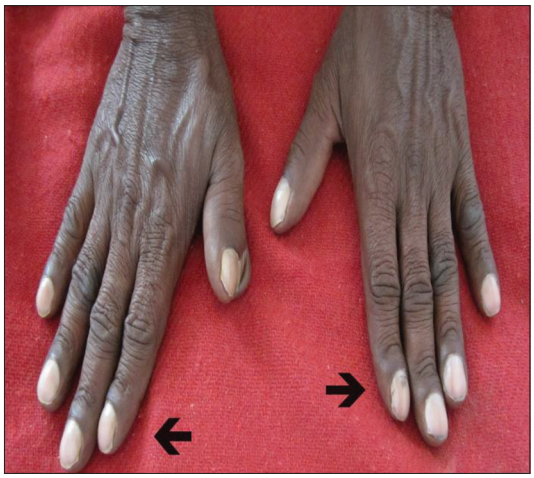

Figure 1: Showing carpel clubbing

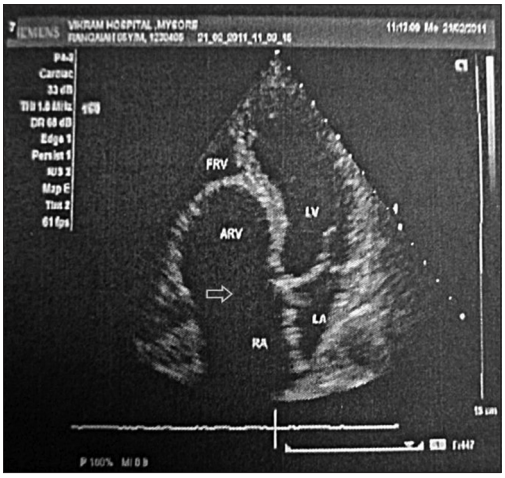

Figure 2: Trans thoracic 2D-Echocardiography showing dilated RA/RV

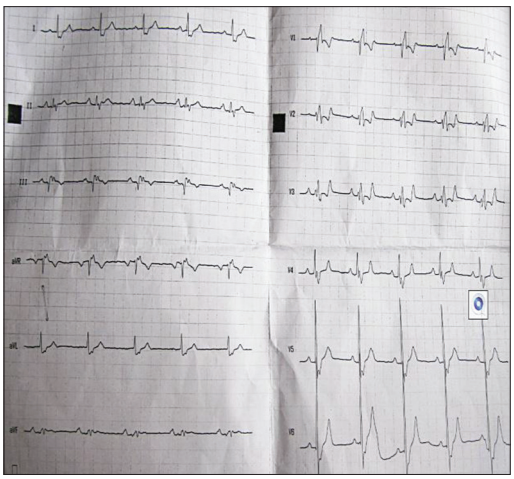

Figure 3: Electrocardiogram showing complete RBBB without himalayan P-waves

DISCUSSION

Ebstein's anomaly of the tricuspid valve is an uncommon developmental abnormality with a reported incidence of less than 1% of all congenital cardiac malformations.[1] The natural history of this disease is variable, and it is believed that early death is often related to diagnostic procedures or thoracotomy.[2] Our case is a 65 year old male who was asymptomatic most of his life but presented with symptoms for the first time. ECG showing Sinus rhythm with QRS Axis of +60, normal p waves, prolonged PR interval, complete right bundle branch block, splintered QRS Complexes.[3] Our case had Normal *P* waves indicate less symptoms in a patient with complete right bundle branch block without WPW syndrome.[4,5]

Though the classical definition of Ebstein's anomaly emphasizes the downward displacement of a part or all of the tricuspid ring and valve, we have in addition, noted a wide range of abnormal features. We believe that dysplasia of valve leaflets is an inherent part of Ebstein's anomaly and this has been stressed by others as well.[6]

The indications for surgical treatment of Ebstein's malformation are not clearly defined and the ideal surgical mode of management remains controversial.[7,8] The dysplastic leaflets and the dilated atrioventricular ring would both contribute significantly to the malfunctioning of the tricuspid valve and resultant cardiac failure. In

this setting, a valve replacement with plication of the thin walled atrialised right ventricle could possibly be the preferable mode of surgical treatment. However, because of the wide spectrum of anatomic variations in the tricuspid valve, the surgical approach to patients with Ebstein's anomaly needs to be individualised according to the specific morphology found at operation. Ebstein's malformation is often associated with other cardiac anomalies but in our case there is no other cardiac abnormality. This explains why our patient has survived without symptoms for this long.[9,10]

CONCLUSION

A rare case of Ebstein's anomaly surviving for seven decades patients surviving after 50 years of age is <5% but without symptoms is rare. Our patient presented with symptoms in the seventh decade only. Surviving till seventh decade is rare but surviving without symptoms is rarest of rare cases.

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Case Report

An Orbital Swelling - Venolymphatic Malformation: A Case Report

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Abstract

Patients presenting with orbital vascular malformations are unusual in ENT outpatient Department. Lymphangioma of an eyelid is recherché though it can occur in the orbit and conjunctiva. Lymphangioma can be traumatic or atraumatic and are the product of sequesteredlymphatic sacs. A comprehensive examination is a must in such cases because unless detected with adequate diagnostic tools its diagnosis could be misleading. CT-Scan imaging is helpful in diagnosing but histopathological examination provides the end result. Vision maybe compromised due to surgical treatment of deep intraorbital vascular malformations intraoperatively and postoperatively. Here is a case of 16 yr old male with a swelling over right eyelid which grew over one and half month, clinically diagnosed as dermoid cyst which wassubsequently diagnosed as venolymphatic malformation (lymphangioma).

Keywords: Dermoid Cyst, Ectatic Blood Vessel, Orbital Swelling, Unilateral proptosis, Venolymphatic Malformation

INTRODUCTION

Sequestered lymphatic sacs not communicating with peripheral draining channels could be its (lymphangioma) point of provenance, is considered. Histologically they are classified as: cystic hygroma, cavernous hemangiomas, capillary hemangiomas and vasculolymphatic malformations. Since they are multi-locular and predominantly cystic masses with both septa and solid components differentiating them with ultrasonography is arduous.[1-2]

The incidence of venous malformation is approximately 1:5,000-10,000.[3]

CASE REPORT

A 16 year old boy came to Otorhinolaryngology OPD with a sudden swelling over right eyelid which was painless, with proptosis and sluggish globe, since one and half month. Eye movement was not restricted. Diplopia on upward gaze was absent. The intraocular pressure of the eye measured by applanation tonometry was within the normal limits. Visual acuity, visual field and pupillary function were normal. Fundoscopic examination results were normal and no bruit or pulsation were noted or felt. On postural changes no variations were detected on proptosis of the eye. On Intra-venous contrast CT-Scan of paranasal sinuses a 3.1 cm × 2.1 cm sized well defined, low intensity mildly enhancing lesion (25hu) (Figures 1 and 2) in the extraconal compartment of right orbit along the supero-medial portion suggestive of lymphangioma. Based upon the clinico-radiological and ct- scan findings, a tentative diagnosis of venolymphatic malformation was made with a differential diagnosis of Dermoid cyst. A surgical excision was done of the lesion under general anaesthesia and the tissue was sent for histopathological examination (Figures 3 and 4). Histopathological microscopy of the lesion revealed cyst lined by atrophic, cuboidal epithelial cells and cyst containing red blood cells with surrounding tissue showing ectatic blood vessels and lymphatic channels.

DISCUSSION

Neoplasms and inflammations are the most common differential diagnosis of unilateral proptosis of eye in children which have acute onset and are not traumatic. Not only are they disfiguring but are also usually associated with complications, such as pain, ulcers, bleeding, and the compression or invasion of adjacent structures. Lymphangiomas can be superficial or deep, and they can involve single or multiple anatomical sites. The cheek, neck, eyelids, lips, tongue, soft palate, parapharyngeal space, and floor of the mouth are most commonly affected sites. The colour of the skin or mucous membrane may be normal or appear blue or dark purple when the entire dermis is involved. The boundary is not clearly defined, and the lesion is soft, compressible and occasionally phlebolith can be palpated.[4]

Pain, swelling, and even bleeding following trauma, secondary infection, abrupt haemorrhage of the lesions, or changes in hormonal level may eventuate. Venous malformations may develop within muscles (such as the temporal muscle, masseter muscle and tongue muscle), which are known as intramuscular venous malformations.

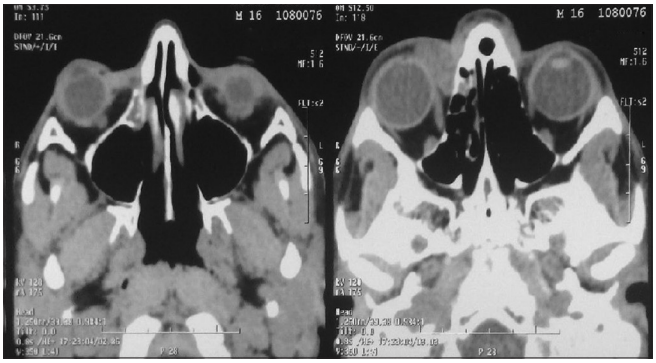

Figure 1: CT-Scan axial

Figure 2: CT-Scan coronal

There is a reasonable debate over the classification of orbital venous malformations, with some authors asserting that the distinction between venous and lymphatic vascular malformation is artificial and prefer to treat according to clinical findings.[5] Lymphangiomas are considered as malformations without flow, but combined venous − lymphatic malformations also exist.[6,7]

Histologically, venous malformations (VMs) may be ectatic or micro-venular. They can be malformed in many varying sizes. Ectasia surges up with advancing age, but the rate at which this takes place is variable. Dystrophic calcification of organizing thrombi can cause formation of phleboliths, as a result of stasis in these low-flow lesions. The thrombus may become infected and cause pain and tenderness.[4]

Diverse treatment modalities are effectual for venous malformation, including surgery, sclerotherapy, laser therapy, cryotherapy, electrocoagulation treatment, and treatment with copper needles.

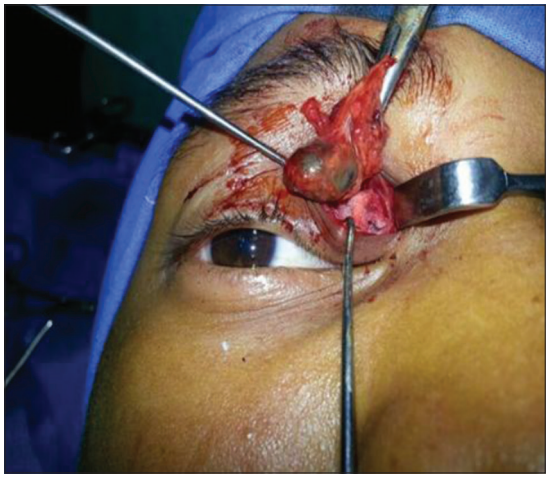

Figure 3: Intra operative

Figure 4: Post operative

Treatment of signs and symptomsfollowing venous malformationis analogousto the site involved and the extent of the venous malformation. Cure may only be obtained in case of small, focal lesions.[8] Multifocal or extensive venous malformations are rarely cured but the symptom and signs can be controlled.

Conservative treatment is primarily suitable for small, isolated, asymptomatic venous malformations. Local compression, anti-infection therapy, pain control can be adopted to suppress symptoms.

In most cases, surgical treatment is considered primarily improving function and appearance.[8] Localized or limited venous malformations can be removed surgically.[9]

CONCLUSION

A diagnosis with thorough clinical examination and imaging technique should be carried out with histopathological co-relation of the specimen. An orbital vascular malformation should be kept in mind before making final diagnosis.

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