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Original Article

A Clinical Analysis of Facial Nerve Paralysis due to Inflammatory Diseases of the Middle Ear and the Role of Early Decompression

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Abstract

Background: Facial nerve (FN) paralysis is one of the most common intratemporal complications of inflammatory diseases of the middle ear. The pathology of FN paralysis in inflammatory diseases of the middle ear is an erosion of the Fallopius canal is either by erosion by suppuration and its pressure effects, cholesteatoma/granulation tissue or osteonecrosis or osteolysis. Surgical decompression remains the choice of treatment.

Aim of the Study: The aim of the study was to analyze the different types of the pathology of FN paralysis in inflammatory diseases of the middle ear intraoperatively and to study the results of FN decompression in a series of patients.

Materials and Methods: A total of 52 patients with inflammatory diseases of the middle ear with facial paralysis attending the Department of ENT of Gandhi Medical College Hospital, Secunderabad, Telangana, were included in the study. A thorough ENT examination was done to know the type of inflammatory disease of the middle ear in all the patients. Patients were subjected to topographic diagnosis. Facial weakness was classified according to House–Brackmann's classification. FN decompression under general anesthesia with canal wall down mastoidectomy was undertaken. Site of the lesion, type of pathology, and post-operative recovery grading were done.

Observations and Results: There were 52 patients with facial paralysis in this study; 39 males and 13 female a ratio of 1.3:1. The mean age was 34.65 ± 6.25 years. The incidence was 19/52 (36.53%) in 40–50 years and 19/52 in 20–30 age group. The lesion was at Genu in 6/52 (11.53%) of patients, at tympanic segment in 15/52 (28.84%), vertical segment in 30/52 (57.69%), and Stylomastoid foramen level in 1/52 (01.92%) patients. Cholesteatoma was cause in 13/52 (25%) patients, granulation tissue in 7/52 (13.46%), dehiscence in 3/52 (05.76%) patients, osteitis in 4/52 (07.69%) patients, edema in 11/52 (21.15%) patients, external compression in 9/52 (17.30%), and inflammation of the nerve in 5//52 (09.61%) patients. Following decompression FN palsy of 4 weeks duration, the recovery was in 49/52 (94.23%) patients.

Conclusions: Early topographic diagnosis of FN paralysis timely decompression of the nerve exposing it from genu to the stylomastoid foramen results in total recovery of the facial muscle power in >94% of the patients. The pathology causing the paralysis and the site of lesion play an important role in the management as well as its prognosis. Paralysis of longer duration has poor prognosis.

Key words: Decompression and topographic diagnosis, Facial expression, Facial muscles, Facial nerve, Nerve monitoring, paralysis

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INTRODUCTION

Chronic suppurative otitis media (CSOM) account for 3.1% of all cases of facial palsy in ENT practice.[1] All types of CSOM can cause facial paralysis which may be acute or insidious in nature. The common causes are acute suppurative otitis media (ASOM), ASOM with, ASOM

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with Mastoid abscess, and CSOM with cholesteatoma. The prognosis in each of this type is varied due to different pathology is the cause of facial palsy in suppurative lesions of the middle ear.[2] Facial paralysis also results from different etiologies which includes infectious, neurologic, congenital, neoplastic, traumatic, systemic, and iatrogenic causes.[3] The management of facial paralysis in all these cases is complex requiring a multidisciplinary approach in its diagnosis, evaluation, and surgical intervention due to wide variation in its potential for regeneration and lack of reliable prognostic indicators for spontaneous recovery.[4] Nonsurgical methods of the treatment of facial paralysis consist of a combination of pharmacologic therapy, physical therapy for facial neuromuscular retraining.[5] Surgical intervention is directed against the nerve and in the failed cases dynamic and static techniques for facial reanimation.[6] The importance of management of facial paralysis lies in the fact that the facial nerve (FN) innervates all the muscles of facial expression which is the most valued possession human beings.[7] As paralysis causes severe disfigurement of facial expression in affected persons, it causes psychological and emotional trauma. Hence, its treatment should be energetic and with concern for the possible outcomes at the very outset[7] FN has a long course (3.75 cm) within the temporal bone and more likely to get entrapped due to edema or erosion of bone;[8] ost common one to be paralyzed. The present study was conducted with an aim to analyze the different causes of the pathology of FN paralysis in CSOM intraoperatively and to study the results of FN decompression in a series of patients.

Type of Study

A combined retrospective and prospective cross-sectional analytical study.

Institution of Study

This study was conducted at the Department of ENT, Gandhi Medical College, Secunderabad, Telangana.

Period of Study

This study was from November 2016 to October 2018.

MATERIALS AND METHODS

A total of 52 patients with inflammatory diseases of the middle ear with facial paralysis attending the Department of ENT of Gandhi Medical College Hospital, Secunderabad, Telangana, were included in the study. An Ethical Committee Clearance was obtained before commencing the study. An Ethical Committee cleared consent letter was used for the study. The retrospective part of the study was conducted between November 2016 and October 2017 and the prospective part of the study was conducted from November 2017 to October 2018 (2 years). The study data for the retrospective study were collected from the medical records section of the hospital.

Inclusion Criteria

1. Patients aged between 10 years and 70 years were included in the study.
2. Patients of both genders were included.
3. Patients with ASOM, CSOM, coalescent mastoid abscess, and CSOM with cholesteatoma were included.
4. Patients with systemic diseases such as tuberculosis, diabetes mellitus, and immunosuppressive diseases were included.
5. Patients with FN palsy developing within 4 weeks were included.
6. Patients with iatrogenic facial paralysis were included.

Exclusion Criteria

1. Patients aged <10 years and >70 years were excluded.
2. Patients with previous mastoid surgery were excluded.
3. Patients with temporal bone fractures, Bell's palsy were excluded.

Demographic data of the patients were elicited. A thorough ENT examination was done to know the type of inflammatory disease of the middle ear in all the patients. All the patients were subjected to a battery of tests of topographic diagnosis to know the site of the lesion. Facial weakness was classified according to House–Brackmann's classification[9] [Annexure 1]. All the patients were subjected to FN decompression under general anesthesia. Canal wall down mastoidectomy was undertaken by the standard method in all patients. During surgery, the course of the FN has been identified in all cases. In cases of erosion by cholesteatoma, the nerve was decompressed a few mm proximal and distal to the site of erosion. Where the site of injury could not be made out, the entire mastoid and tympanic portion were decompressed. Patient's age, sex, clinical presentation, diagnostic evaluation, management, and outcome have been analyzed. The recovery of facial palsy was assessed after 8 weeks and at the end of 12 months. Recovery was grouped as good (HB1), satisfactory (HB-2–3), and unsatisfactory (HB-4–6). The pathology causing the facial paralysis intraoperatively was noted. All the patients were followed up to 2 years. All the data were analyzed using standard statistical methods.

OBSERVATIONS AND RESULTS

Among the 52 patients, there were 39 male patients and 13 were female patients with a female to male ratio of 1.3:1. The youngest patient was aged 10 years, and the eldest one was 68 years with a mean age of 34.65 ± 6.25 years. The

incidence of FN paralysis in the study group was observed and found that there were 19/52 (36.53%) patients in the age group of 40–50 years. 19/52 patients were in the age group of 20–30 years and 7/52 patients were in the age group of 10–20 years [Table 1].

The presenting clinical symptoms and House–Brackmann's grading of facial paralysis were observed and found that a deviation of angle of mouth was observed in 49/52 (94.23%), inability to close the eye in 45/52 (86.53%), ear discharge 52/52 (100%), and decrease hearing in 33/52 (78.84%) of the patients. Facial muscles power was assessed using House–Brackmann's grading, and it was found that in 4/52 patients Grade-II weakness was observed (7.69%), Grade III in 4/52 (7.69%), Grade IV in 8/532 (%), Grade V in 17/52 (32.69%), and Grade VI in 19/52 (36.53%) patients [Table 2].

FN damage was observed at the level of Genu in 6/52 (11.53%) of patients, at tympanic segment in 15/52 (28.84%), at vertical segment in 30/52 (57.69%), and stylomastoid foramen level in 1/52 (01.92%) patients. Intraoperative findings were analyzed in this study and were found that cholesteatoma eroding the facial canal was seen in 13/52 (25%) patients, granulation tissue eroding the Fallopius canal was seen in 7/52 (13.46%), dehiscence of FN canal was observed in 3/52 (05.76%) patients, osteitis in 4/52 (07.69%) patients, edema in 11/52 (21.15%) patients, external compression in 9/52 (17.30%), and inflammation of the nerve in 5//52 (9.61%) patients [Table 3].

The post-operative results in the present study showed varied results depending on the grade of pre-operative status. Grades IV, V, and VI to Grade I was observed ranging from 21.05%, 35.29%, and 87.50%, respectively. Similarly, from Grades VI, V, and VI to Grade II was observed ranging from 73.68%, 52.94%, and 12.05%, respectively. From Grade II to Grade I was observed in 4/4 (100%) of the patients. From Grade III to Grade I was observed in 3/4 patients with one patient showing synkinesis. Synkinesis was observed in 2 patients out of 52 patients (3.84%) and no recovery in 2/52 (3.84%) of patients [Table 4].

DISCUSSION

Prevalence of FN paralysis based on the total number of ear discharge patients attending this general hospital and it was 0.24% (the number of ear discharge cases was 21500). However, as this study is a cross-sectional hospital, based study may not reflect the actual figure in the community. Other authors reported the incidence of facial paralysis in inflammatory diseases of the middle ear with a very

Table 1: The age group incidence of the patients (*n*-52)

Age in groups	Number of patients (%)
10–20	7 (13.46)
20–30	10 (19.23)
30–40	13 (25)
40–50	19 (36.53)
50–60	2 (03.84)
60–70	1 (01.92)

Table 2: The clinical symptoms and signs of facial paralysis in the study (*n*-52)

Clinical symptom/sign	*n* (%)
Deviation of the angle of mouth	49 (94.23)
Inability to close the eye	45 (86.53)
Ear discharge	52 (100)
Pain in the ear	33 (63.46)
Decreased hearing	41 (78.84)
House–Brackmann's grading	
Grade II	4 (7.69)
Grade III	4 (7.69)
Grade IV	8 (15.38)
Grade V	17 (32.69)
Grade VI	19 (36.53)

Table 3: The incidence of site of nerve lesion and the different types of pathologies in the study (n-52)

Intra-operative findings	*n* (%)
Site of facial nerve lesion	
Geniculate ganglion	6 (11.53)
Tympanic segment	15 (28.84)
Vertical segment	30 (57.69)
Stylomastoid foramina	1 (01.92)
Type of pathology	
Erosion by cholesteatoma	13 (25)
Granulation tissue	7 (13.46)
Facial nerve dehiscence	3 (05.76)
Osteitis	4 (07.69
Edema	11 (21.15)
External compression	9 (17.30)
Inflammation of the nerve	5 (09.61)

Table 4: The post-operative results of facial decompression (*n*-48)

Post-operative results of facial nerve decompression	*n* (%)
From Grade VI to Grade I	4/19 (21.05)
From Grade V to Grade I	6/17 (35.29)
From Grade IV to Grade I	7/8 (87.50)
From Grade VI to Grade II	14/19 (73.68)
From Grade V to Grade II	9/17 (52.94)
From Grade IV to Grade II	1/8 (12.05)
Grade III to Grade I	3/4 (75)
Grade II to Grade I	4/4 (100)
From Grade III to synkinesis	1/4 (25)
From Grade VI to synkinesis	1/19 (5.26)
No recovery	2/17 (11.76)

wide range; Pollock and Brown[2] reported it to be 0.16% (2/1250), Kangsanarak *et al.*[10] reported it to be 0.29% in 13948 patients with CSOM, and Savić and Djeric[11] reported it as 5.1% in 1261 patients. The incidence of facial paralysis in relation to the only cholesteatoma is around 0.04–0.16%.[12] This figure is decreasing ever since the use of diagnostic tools and the invention of micro ear surgery. One of the anatomical facts about FN is that the nerve is surrounded by connective tissue and blood vessels to the extent of 65–35% in addition to the bulk of the nerve, not leaving any empty space in the bony canal.[13] Congenital dehiscence of the FN is thought to be one of the major causes of FN paralysis in inflammatory diseases of the middle ear.[11] If the dehiscence is on the vestibular surface of the Fallopius canal it would be hidden by the surgeon's view.[11] In the present study, it was observed that cholesteatoma eroding the facial canal was seen in 13/52 (25%) patients, granulation tissue eroding the Fallopius canal was seen in 7/52 (1S3.46%), dehiscence of FN canal was observed in 3/52 (05.76%) patients, osteitis in 4/52 (7.69%) patients, edema in 11/52 (21.15%) patients, external compression in 9/52 (17.30%), and inflammation of the nerve in 5/52 (09.61%) patients [Table 3]. Waddell and Maw[14] reported transaction of FN by cholesteatoma as a rare case report. The paralysis due to erosion by cholesteatoma occurs at a slow pace when compared to acute onset facial paralysis observed with ASOM or acute mastoid abscess or coalescent mastoiditis.[14] It was also reported that facial paralysis does not become evident clinically as a total paralysis until there is disruption of >50% of its nerve fibres.[15] Even though many authors have reported FN paralysis in inflammatory diseases of the middle ear, there is no definite protocol for its management. Altuntas *et al.*[8] in their study of 20 cases observed that 14 (70%) had cholesteatoma, 3 (15%) had granulation tissue, and 3 (15%) had polypoid tissue in their middle ear. They reported the lesion in the Fallopian canal in 14 (70%) of the patients, 5/14 in the tympanic segment, and 9/14 at the 2nd Genu and/or the vertical segment of the facial canal. Yetiser *et al.*[16] reported from their study, majority of the cases with fallopian canal defect had cholesteatoma, 2 had granulation tissue, and 2 had polypoid tissue in the middle ear. They decompressed the nerve from the first Genu to the stylomastoid foramen and noted the FN to be edematous in 14 cases and normal with no edema in the other 6. In the present study, FN damage was observed at the level of Genu in 6/52 (11.53%) of patients, at tympanic segment in 15/52 (28.84%), at vertical segment in 30/52 (57.69%), and stylomastoid foramen level in 1/52 (1.92%) patients. The post-operative results showed synkinesis in 3.84% of the patients and no recovery also in 3.84% of patients. Yetiser *et al.*[16] from their study reported 75% of their patients with complete recovery without synkinesis and 5 had an incomplete recovery; 14/24 patients had very good recovery within 3 months. 6 had Grade II and 2 patients had Grade III facial paralysis, and 2 patients had Grade II paresis with synkinesis at 1-year follow-up. In the present study, the post-operative results showed varied results depending on the grade of pre-operative status. The post-operative results in the present study showed varied results depending on the grade of pre-operative status. Grades IV, V, and VI to Grade I were observed ranging from 21.05%, 35.29%, and 87.50%, respectively. Similarly, from Grades VI, V, and VI to Grade II were observed ranging from 73.68%, 52.94%, and 12.05%, respectively. From Grade II to Grade I was observed in 4/4 (100%) of the patients. From Grade III to Grade I was observed in 3/4 patients with one patient showing synkinesis. Synkinesis was observed in 2 patients out of 52 patients (3.84%) and no recovery in 2/52 (3.84%) of patients. Jackson recommended from his study removal inflammatory disease from the middle ear and mastoid and decompression of the involved segment of the nerve. In cases of long-standing paralysis, he recommended sectioning and grafting the nerve in attenuated nerves (under 2 years of paralysis).[17] Whenever there was total paralysis of FN with House–Brackmann's Grade of VI or V, it is better to decompress the nerve from genu to stylomastoid foramina especially when the paralysis is acute.[15] Some authors refrain from decompression of FN in the presence of acute infection or granulations and undertake only disease clearance and institute anti-edema measures in the management of FN paralysis.[18] FN decompression performed in palsy cases of longer duration has poorer prognosis and recovery of muscle power may not be complete.[18] In the present study, patients with FN palsy of 4 weeks duration alone were included; hence, the recovery was good in 49/52 (94.23%) patients. In a similar study by Dubey and Larawin[19] showed that 6/9 decompressed FN s had intact fallopian canal and recovered to House–Brackmann's Grade I postoperatively; 3/9 had erosion of the Fallopian canal with granulation tissue over the FN, and post-operatively, their final FN status was House–Brackmann's Grade III.[19]

CONCLUSIONS

Early topographic diagnosis of FN paralysis timely decompression of the nerve exposing it from Genu to the stylomastoid foramen results in total recovery of the facial muscle power in more than 94% of the patients. The pathology causing the paralysis and the site of lesion play an important role in the management as well as its prognosis. Paralysis of longer duration has poor prognosis.

REFERENCES

1. Takahashi H, Nakamura H, Yui M, Mori H. Analysis of fifty cases of facial palsy due to otitis media. Arch Otorhinolaryngol 1985;241:163-8.
2. Pollock RA, Brown LA. Facial paralysis in otitis media. In: House WF, editor. Disorders of the Facial Nerve. New York: Raven Press; 1982. p. 221-4.
3. Melvin TA, Limb CJ. Overview of facial paralysis: Current concepts. Facial Plast Surg 2008;24:155-63.
4. Hadlock TA, Greenfield LJ, Wernick-Robinson M, Cheney ML. Multimodality approach to management of the paralyzed face. Laryngoscope 2006;116:1385-9.
5. Sofferman RA. Facial nerve injury and decompression. In: Nadol JB Jr., Mckenna MJ, editors. Surgery of the Ear and Temporal Bone. Philadelphia, PA: Lippincott Williams and Wilkins; 2005. p. 435-50.
6. Humphrey CD, Kriet JD. Nerve repair and cable grafting for facial paralysis. Facial Plast Surg 2008;24:170-6.
7. Das AK, Sabarigirish K, Kashyap RC. Facial nerve paralysis: A three year retrospective study. Indian J Otolaryngol Head Neck Surg 2006;58:225-8.
8. Altuntas A, Unal A, Aslan A, Ozcan M, Kurkcuoglu S, Nalca Y, *et al*. Facial nerve paralysis in chronic suppurative otitis media: Ankara numune hospital experience. Auris Nasus Larynx 1998;25:169-72.
9. House JW, Brackmann DE. Facial nerve grading system. Otolaryngol Head Neck Surg 1985;93:146-7.
10. Kangsanarak J, Fooanant S, Ruckphaopunt K, Navacharoen N, Teotrakul S. Extracranial and intracranial complications of suppurative otitis media. Report of 102 cases. J Laryngol Otol 1993;107:999-1004.
11. Savić DL, Djerić DR. Facial paralysis in chronic suppurative otitis media. Clin Otolaryngol Allied Sci 1989;14:515-7.
12. Siddiq MA, Hanu-Cernat LM, Irving RM. Facial palsy secondary to cholesteatoma: Analysis of outcome following surgery. J Laryngol Otol 2007;121:114-7.
13. Saito H, Takeda T, Kishimoto S. Facial nerve to facial canal cross-sectional area ratio in children. Laryngoscope 1992;102:1172-6.
14. Waddell A, Maw AR. Cholesteatoma causing facial nerve transection. J Laryngol Otol 2001;115:214-5.
15. Selesnick SH, Jackler RK. Facial paralysis in suppurative ear disease. Oper Tech Otolaryngol Head Neck Surg 1992;3:61-8.
16. Yetiser S, Tosun F, Kazkayas M. Facial nerve paralysis due to chronic otitis media. Otol Neurotol 2002;23:580-8.
17. Jackson GC. Facial Nerve Paralysis; Diagnosis and Treatment of Lower Motor Neuron Facial Nerve Lesions and Facial Paralysis. Washington, Dc: American Academy of Otolaryngology-Head and Neck Surgery Foundation Committee on Continuing Education in Otolaryngology; 1986. p. 235-9
18. Kumar SS, Thakar A. Spectrum of facial paralysis in chronic suppurative otitis media. Indian J Otol 2012;18:92-4.
19. Dubey SP, Larawin V. Complications of chronic suppurative otitis media and their management. Laryngoscope 2007;117:264-7.

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ANNEXURE

Annexure 1: House–Brackmann facial nerve grading system

Grade I - Normal
Normal facial function in all areas
Grade II - Slight dysfunction
Gross: Slight weakness noticeable on close inspection; may have very slight synkinesis
At rest: Normal symmetry and tone
Motion: Forehead - moderate to good function; eye - complete closure with minimum effort; mouth - slight asymmetry.
Grade III - Moderate dysfunction
Gross: Obvious but not disfiguring difference between two sides; noticeable but not severe synkinesis, contracture, and/or hemifacial spasm.
At rest: Normal symmetry and tone
Motion: Forehead - slight to moderate movement; eye - complete closure with effort; mouth - slightly weak with maximum effort.
Grade IV - Moderate severe dysfunction
Gross: Obvious weakness and/or disfiguring asymmetry
At rest: Normal symmetry and tone
Motion: Forehead - none; eye - incomplete closure; mouth - asymmetric with maximum effort.
Grade V - Severe dysfunction
Gross: Only barely perceptible motion
At rest: Asymmetry
Motion: Forehead - none; eye - incomplete closure; mouth - slight movement
Grade VI - Total paralysis
No movement