AETIOLOGY OF ACQUIRED LOWER MOTOR NEURON TYPE OF FACIAL NERVE PARALYSIS– A DESCRIPTIVE STUDY

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ABSTRACT

BACKGROUND

This study was conducted to evaluate the various aetiologies of acquired lower motor neuron type of facial nerve paralysis, assessment of severity of lesion and outcome through follow up. 47 patients between 15-75 years were studied.

MATERIALS AND METHODS

All patients with acquired LMN facial paralysis who presented to our department were included in the study. They were evaluated with history, clinical examination and investigations. They were treated and followed up for a period of 6 months.

RESULTS

The most common aetiology identified for facial palsy was trauma which was non-iatrogenic. The anatomic level which predominated in our patients was infrastapedial. Maximum number of patients presented with grade 4 facial palsy.

CONCLUSION

Non- iatrogenic trauma was the most common cause among the patients studied and follow up of these patients had a good recovery of 60%.

KEYWORDS

Lower Motor Neuron Type, Facial Nerve Paralysis, House Brackman Grading System, Topodiagnosis, HRCT Temporal Bone, Temporal Bone Fractures.

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BACKGROUND

Facial nerve paralysis causes noticeable disfigurement and emotional distress to those suffering from it. If paralysis is not managed, major complications associated with eye can occur, leading to corneal ulceration and finally blindness.[1] The causes of Facial Nerve Paralysis are numerous which can be congenital, trauma, neurological, infection, metabolic, neoplastic, toxic, autoimmune, iatrogenic and idiopathic.[2] In most cases, no definite aetiology is identified (idiopathic/Bell's palsy). The diagnosis of an idiopathic palsy however can only be made by excluding all other potential causes of facial palsy. Bell's palsy is named after British Physician, Sir Charles Bell– who described onset, physical findings and course of the disease in 1821. The prevalence of facial palsy in the general population is about 1:5000, making it a common-enough clinical entity for all general practitioners to be familiar with.[3] Management varies and depends on a number of variables. It includes observation, administration of pharmacologic agents, surgical intervention, physiotherapy and psychological counselling.[2] This study was conducted to evaluate the various aetiologies of acquired lower motor neuron type of facial nerve paralysis. Attempts were made at systematized topodiagnosis to determine the anatomic level of injury.[4] assessment of severity of lesion and eventual outcome through steroid therapy and regular follow up.[5]

Objectives- Primary Objective: To study the aetiology of acquired lower motor neuron type of facial palsy presenting to otolaryngology department, Government Medical College, Thiruvananthapuram.

Secondary Objectives- Assessment of the severity and the response to treatment of facial paralysis due to various aetiologies using House Brackman grading system.

MATERIALS AND METHODS

This study was conducted over a period of 18 months and comprised of 47 patients of 15-75 years of age and either sex diagnosed as having lower motor neuron type of facial nerve paralysis in ENT department of Govt. Medical College Hospital, Thiruvananthapuram. Patients were included according to inclusion and exclusion criteria.

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Inclusion Criteria

- Patients presenting with acquired LMN type of facial paralysis
- Patients in the age group of 15-75 years.

Exclusion Criteria

- Those not willing to participate in the study.
- Facial palsy associated with congenital syndromes.

All patients were included in the study according to inclusion criteria and after getting informed written consent. The diagnosis of lower motor neuron type of facial palsy was based on history and clinical examination. Patients were evaluated with HRCT of temporal bone to find out the cause. To anatomically localize the site, topodiagnostic tests including examination of taste, Schirmer's test and stapedial reflex were performed. Patients were followed up to a period of 6 months to observe the improvement in severity of palsy.

RESULTS

The sample size in our study was 47 and most common age group was 51-60 years accounting for about 19.1 percent followed by 41-50 years of age.

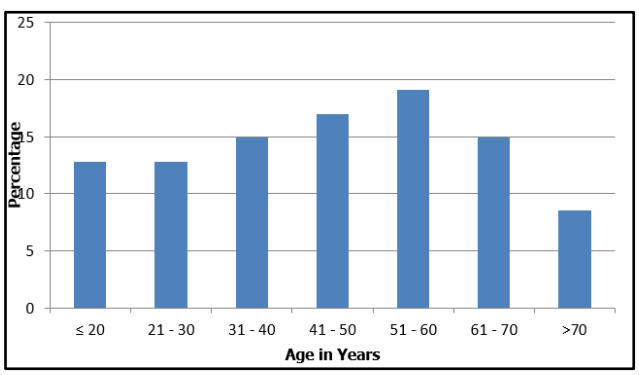

Chart 1. Age Wise Distribution of Cases

According to our study, 79% were males (37) and 21% females (10).

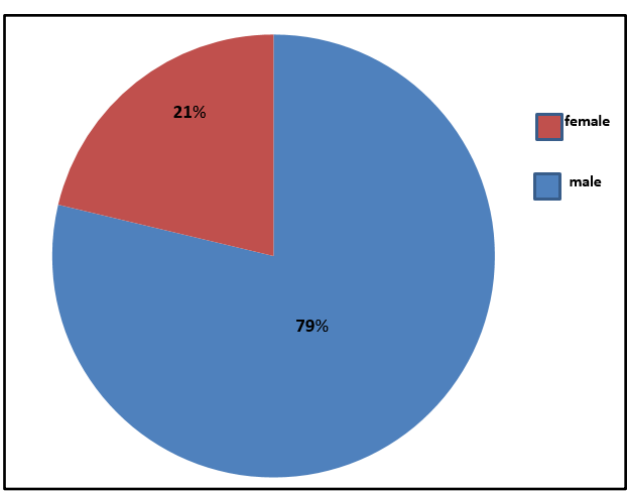

Chart 2. Sex Ratio

Of the 47 cases of facial nerve paralysis, 25 were on left and 22 on right side.

Side of Paralysis	Number of Patients	%
Left	25	53.2
Right	22	46.8
Total	**47**	**100.0**

Table 1. Percentage of side of paralysis

Majority (55.3%) of the patients had grade 4 facial nerve palsy while 15 (31.9%) had grade 3 palsy (House-Brackman grading system).

Grade	Number of Patients	Percentage
2	1	2.1
3	15	31.9
4	26	55.3
5	4	8.5
6	1	2.1
Total	**47**	**100.0**

Table 2. Distribution of Patients According to the Grade of Facial Palsy

Non-iatrogenic trauma in the form of RTA was present in 13 of 47 patients (27%). 10 of them were delayed onset and 3 of them were immediate in onset. History of previous surgery was noted in 3 of our patients. Diabetes as a comorbidity was present in 24 of 47 patients with LMN facial palsy (51%). No significant illness noted in 8 patients (17%).

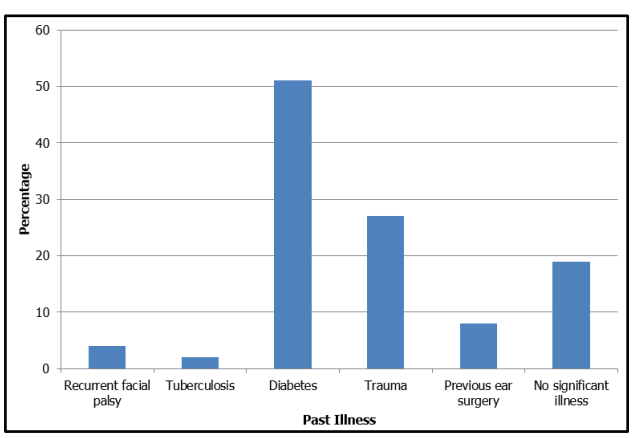

Chart 3. Distribution of Patients According to Past Illness

Lacrimation was absent (Schirmer positive) in 15 of 47 (31.9%) of patients. Dry mouth in 18 patients and ipsilateral loss of taste in 42 patients (89.4%). Absent forehead wrinkling, Loss of nasolabial fold, Inability to blow cheek, Deviation of angle of mouth and positive Bell's phenomenon were present in all cases. The anatomic level to which maximum number of patients fell into was infrastapedial- 21 patients (44.8%), followed by supragenicular (27.7%), suprastapedial (17.%) and extra temporal (10%).

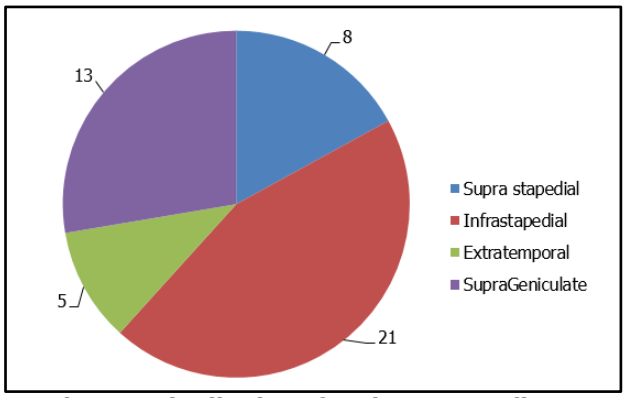

Chart 4. Distribution of Patients According to Anatomic Level of Facial Nerve Involvement

Marginal perforation was seen in 9 patients (19.1%), retracted tympanic membrane was seen in 8 patients and 6 each had haemotympanum, central perforation and traumatic perforation. Congested tympanic membrane was seen in 2 of our patients. Traumatic non-iatrogenic facial palsy was seen in 13 of our patients as aetiology. 2 cases of longitudinal fracture, 8 cases of transverse fracture and 1 case of mixed fracture of temporal bone were identified. Otomastoiditis was seen in HRCT in 9 of our patients (19.14%) and 4 had cholesteatoma (8.5%). In another 7 of our patients, malignant otitis was the finding in HRCT.

HRCT Findings	No. of Patients	%
Transverse fracture	2	4.3
Longitudinal fracture	8	17.0
Mixed fracture	1	2.1
Dehiscent facial canal	9	19.14
Facial canal wall fracture	3	6.4
Otomastoiditis	9	19.14
Cholesteatoma	4	8.5
Skull base osteomyelitis	7	14.9
Not done	11	23.4

Table 3. Distribution of Patients According to HRCT Findings

The aetiology which accounted for facial nerve palsy was most frequently non-iatrogenic trauma in 13 patients, 3 cases of iatrogenic trauma, 9 of them were Idiopathic, 7 cases of malignant otitis externa, 4 cases of Chronic otitis media- active squamous, 2 cases of mucormycosis, 5 cases of chronic otitis media active mucosal disease, 2 cases of acute otitis media, and 1 case of tuberculous otitis media and 1 case of acute necrotizing otitis media. The most common associated symptom that the patient complained of was ear discharge from the affected side.

Aetiology	No. of Patients	%
Non iatrogenic trauma	13	27.6
idiopathic	9	19.1
Malignant otitis	7	14.8
Chronic otitis media active squamous	4	8.5
mucormycosis	2	4.2
Chronic otitis media active mucosal	5	10.6
Iatrogenic trauma	3	6.3
Acute otitis media	2	4.2
Tuberculosis	1	2.1
Acute necrotising otitis	1	2.1

Table 4. Distribution of Patients According to Aetiology of Facial Nerve Palsy

All of our patients were prescribed steroids and physiotherapy exercises. At 6 months, 60% patients had recovery to full extent to grade 1. Ten were lost to follow up.

DISCUSSION

This study included 47 patients with acquired lower motor neuron type of facial palsy, who presented to the Department of Otorhinolaryngology, Medical College, Thiruvananthapuram during the study period of one and half years from January 2015 to June 2016.

In the present study, an attempt was made to identify the etiological factors of patients with acquired lower motor neuron type of facial palsy who were treated in our department. They were followed up for a period of up to 6 months and were assessed for improvement. The results obtained were compared with many studies conducted previously.

In our study, most common age group was 51-60 years accounting for about 19.1 percent followed by 41-50 years of age. Mean age group was 45 and standard deviation of 2.77. In the study by Erik Peitersen.[3] incidence of facial palsy reaches a maximum between the ages of 15 and 45 years.

The gender distribution in our study was 79% males and 21% females. The female gender accounted for 70.71% of cases in the study by Gonçalves-Coêlho TD et al.[6] Laura Sánchez-Chapul et al[7] had 63.7% (160) women and 36.3% (91) men in their study.

The side of palsy was more on left (53.2) compared to right (46.8) in our study. According to the study by Laura Sánchez-Chapul et al,[7] left sided palsy was seen in 54.5% and right 41.5%, comparable to our results.

2 (4%) of our patients had recurrent facial paralysis. It was on the same side. One of them was a male and the other female. The female patient had past history of palsy during pregnancy and recovered completely at that time. The female patient was diabetic while the male was not. Both of the cases were idiopathic. 10.9% were recurrent attacks of peripheral facial palsy in the study conducted by P. Hallmo, H. H. Elverland and I. W. S. Mair.[8] 4-7% of all cases of Bell's palsy have recurrent facial palsy according to study by Pitts DB, Adour KK, Hilsinger RL.[9]

Past history of trauma was present in 13 of our patients and it was the cause for facial nerve palsy in these patients. In 10 of these patients the onset of facial palsy was delayed (21.3%) and 3 of them were immediate in onset. The incidence of delayed facial palsy was only 0.3% after trauma in the study conducted by K. Puvanendran, M. Vitharana, and P. K. Wong.[10] There was a history of previous ear surgery in 3 of our patients. 3 of them were post modified radical mastoidectomy, done for cholesteatoma. Among the post modified radical mastoidectomy patients, 2 of them

were immediate onset facial palsy and one was a delayed onset type.

The anatomic level in which maximum patients fell in our study was infrastapedial (44.8%). In the study by Naoaki Yanagihara et al,[11] infrastapedial lesions were found as maximum, especially in peripheral facial nerve palsy in the sub-acute stage. Supragenicular lesions are responsible for 50% of the facial nerve involvement of several etiologies in the study conducted Bento et al.[12]

At 6 months of follow up, 22 out of 37 patients had grade 1 recovery which is about 60%. It's less compared to the study by I P Tang et al,[13] where after three to six months follow-up, 70 (83.3%) patients fully recovered, 13 (15.4%) patients partially recovered and one (1.2%) patient did not respond to treatment. Still our recovery of 60% is also a fairly good result.

CONCLUSION

The most common age group was 51-60 Years and the majority was male patients. The side of palsy was more on left. The most common associated symptom that the patient complained of was ear discharge from the affected side. Recurrent facial palsy was seen in 2 of our patients. The most common etiology identified for facial palsy was trauma which was non-iatrogenic. Diabetes as a comorbidity was present in a majority. The anatomic level which predominated in our patients was infrastapedial. Maximum number of patients presented with grade 4 facial palsy. All of our patients were prescribed steroids and physiotherapy exercises. Follow up carried up to a period of 6 months showed good recovery of about 60%.

Limitations

The sample size of the study was small only 47 patients presented to our department during the study period. Since it is a tertiary care centre, all cases of facial palsy may not reach us especially idiopathic Bell's palsy which may be managed in the periphery. 10 of our patients were lost to follow up. Time period for our study was limited. Ideally a follow up of up to 1 year is essential for the assessment of recovery of LMN facial palsy which was limited to 6 months in our study.

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