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A study of aetiology and associated topodiagnostic and electrodiagnostic tests among subjects with lower motor neuron facial palsy

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***Abstract*---**Background : Lower motor neuron (LMN) facial palsy is a very common clinical occurrence with an estimated incidence of 20 to 30 per 100,000 population. LMN facial palsy is commonly encountered by Otorhinolaryngologists, internal medicine practitioners and Neurologists alike. Being a mixed nerve, it's paralysis can lead to mechanical impairment and emotional, social implications. Objective: To study the topodiagnostic and electrodiagnostic tests associated with Lower Motor Neuron Facial palsy among the study subjects. Methodology : The present cross sectional study conducted by the Department of otorhinolaryngology, neurology, neurosurgery , Pediatric and General Medicine Departments at JSS Medical College and Hospital Mysore from October 2015 to September 2017. A total of 155 study subjects were enrolled for the purpose of the study during the study period who met the inclusion criteria were enrolled for the purpose of the study . Results : Bell's palsy was the most common etiology identified with 101 cases, 65.2%% of the individuals, followed by Ramsay Hunt syndrome with 12 cases, 7.7%, followed by traumatic and delayed facial palsy. Iatrogenic and delayed facial palsy was seen in 5

individuals, 3.2%.Pregnant and post partum patients were 3, 1.9%. In comparison of Bell's palsy vs non-Bell's etiologies, no significant differences were observed between the two in results of Schirmer's test, stapedial reflex on the affected side, blink reflex and Nerve conduction velocities. Statistically significant difference was observed between Bell's and non-Bell's groups in stapedial reflex on the unaffected side due to possible subclinical involvement of the unaffected side in Bell's palsy. Conclusion: Majority of the patients in both Bell's and Non-Bell's groups had topognostic level of lesion at Geniculate and supra geniculate level.But lesions lower down showed paradoxical and confusing results.This could possibly be explained by incomplete and patchy involvement of the facial nerve in Bells palsy

***Keywords*---**lower motor neuron, facial palsy, electrodiagnostic tests**.**

Introduction

Lower motor neuron (LMN) facial palsy is a very common clinical occurrence with an estimated incidence of 20 to 30 per 100,000 population. LMN facial palsy is commonly encountered by Otorhinolaryngologists, internal medicine practitioners and Neurologists alike. Being a mixed nerve, it's paralysis can lead to mechanical impairment and emotional, social implications. Many of the cases are brushed aside as Bell's palsy, though several other causes are likely. This study would help throw light on the incidence, demographic characteristics, and etiologies of peripheral facial palsies in our population.[1,2,3]

Topognostic tests are intended to reveal the site of lesion by use of a simple principle: lesions below the point at which a particular branch leaves the facial nerve trunk will spare the function subserved by that branch.[2] Hence, injury to the terminal branches of the nerve in the face will not affect lacrimation, salivation, taste, or the stapedius reflex. Conversely, the combination of facial paralysis and decreased lacrimation can theoretically be caused only by a lesion in the segment of the nerve in which voluntary motor fibers and parasympathetic fibers to the lacrimal gland run together i.e., from the cerebellopontine angle (CPA to the geniculate ganglion).

In complete focal lesions, as in trauma, topognostic tests are usually reliable but unnecessary, because clinical examination and imaging suffice to describe the site of the lesion. If multiple or extensive lesions of the facial nerve are present, topognostic tests can predict only the most proximal lesion and provide little to no information concerning additional distal facial nerve lesions.[3,4,5] Consequently, many investigators have noted paradoxic and misleading results in applying topognostic testing principles in the evaluation of patients with Bell's palsy. In contrast to traumatic injuries, Bell's palsy is typically a mixed and partial lesion with varying degrees of conduction block and degenerative changes within different fibers and fascicles of the nerve trunk; therefore, topognostic tests are not expected to provide precise information about the level of the lesion.[6,7,8]

Topodiagnostic tests which include schirmer's test, stapedial reflex, taste sensation testing and electrodiagnostic tests, which include nerve conduction velocity and blink reflex were undertaken for the cases presenting to a tertiary care referral hospital with wide catchment area. [9] . The study would help in prognostication of patients and allow comparison of outcomes of different aetiologies.

Objectives

To study the topodiagnostic and electrodiagnostic tests associated with Lower Motor Neuron Facial palsy among the study subjects.

Materials and Methods

The present cross sectional study conducted by the Department of otorhinolaryngology, neurology, neurosurgery , Pediatric and General Medicine Departments at JSS Medical College and Hospital Mysore from October 2015 to September 2017. A total of 155 study subjects were enrolled for the purpose of the study during the study period who met the inclusion criteria were enrolled for the purpose of the study.

Inclusion Criteria

All patients consenting to participate in the study of all ages of either sex, both outpatients and inpatients with a diagnosis of LMN facial palsy during the study period presenting to Otorhinolaryngology, Neurology, Neurosurgery, Pediatrics and General Medicine departments of JSS Hospital, Mysore.

Exclusion Criteria

Patients with Upper Motor Neuron Facial palsy and patients with other cranial nerve palsies clinically.

Method of Collection of Data

Recruitment of subjects and collection of data was spread over a period of two years. Inpatients and outpatients presenting to Otorhinolaryngology, Neurology, Neurosurgery, Pediatrics and General Medicine Departments of JSS Medical College teaching hospital were enrolled over the study period & analyzed with reference to various parameters in order to compile a profile. Patient information was collected after obtaining informed consent. Careful history regarding the onset, causation, duration and progression was taken in a semi structured proforma.

The patients were diagnosed to have Lower Motor Neuron facial palsy if there was weakness of all the muscles of facial expression, the angle of the mouth falls, weakness of frontalis occurs, and eye closure is weak. The patient would be diagnosed to have Upper motor neuron facial palsy if frontalis is spared, normal furrowing of the brow is preserved, and eye closure and blinking are not affected.[1]

Complete ENT evaluation including neurological examination, Schirmer's's test, routine laboratory investigations, audiological evaluation and stapedial reflex, electrodiagnostic tests –nerve conduction velocity and blink reflex were carried out.

Results

A total of 155 LMN facial palsies were enrolled for the study. 25.2% of the patients were from 31-40 years of age followed by 21.9% in 21-30 years. LMN facial palsy was equally distributed at 14.8% in both <20 and 51-60 years and least in >61 years age group. Mean age of presentation was 38 years SD 17 years and showed a normal distribution with 25th percentile at 25 years and 75th percentile at 51 years. Males constituted 61.3% and females 38.7%, 95 and 60 patients respectively.

Table 1 : Distribution of study subjects based on Aetiologies of LMN Facial palsy

		Count	Column N %
ETIOLOGY	Bell's	101	65.2%
	Traumatic and Delayed palsy	11	7.1%
	Pregnancy and postpartum Bell's	3	1.9%
	Ramsay Hunt Syndrome	12	7.7%
	Iatrogenic and Delayed palsy	5	3.2%
	Infective conditions of the ear and parotids	7	4.5%
	Guillain Barre Syndrome	9	5.8%
	other infections	4	2.6%
	Miscellaneous -SLE, Sarcoidosis, Vasculitis	3	1.9%

Bell's palsy was the most common etiology identified with 101 cases, 65.2%% of the individuals, followed by Ramsay Hunt syndrome with 12 cases, 7.7%, followed by traumatic and delayed facial palsy. Iatrogenic and delayed facial palsy was seen in 5 individuals, 3.2%.Pregnant and post partum patients were 3, 1.9%.
Among patients with trauma, patients had a horizontal fracture of the temporal bone, had a vertical fracture of the temporal bone and no fracture was identified in patients.

Infective conditions of the ear and parotids constituted 7 (4.5%) cases of which 3 (1.93%) had Malignant otitis externa, 1 (0.64%)patient had acute suppurative otitis media, 2 had chronic suppurative otitis media-1 (0.64%)with Atticoantral disease and 1(0.64%) with tubotympanic disease, 1(0.64%) patient had parotid abscess as the cause of LMN facial palsy. Among iatrogenic causes, parapharyngeal mass excision 1(0.64%),cortical Mastoidectomy 2(1.28%), radical Mastoidectomy 1(0.64%), were associated with iatrogenic and post reactivation LMN facial palsy. Four patients had infective conditions having LMN facial palsy of which 1 (0.64%)had Hansen's disease and 3 (1.93%) had HIV infection.

Topodiagnostic tests

Table No.2 : Table showing the result of Schirmer's test on the affected and non-affected side

	Mean	Median	Standard Deviation	Percentile 25	Percentile 75
SCHIRMER'S AFFECTED SIDE	13.21	10.00	8.72	7.00	20.00
SCHIRMER'S UNAFFECTED SIDE	16.61	15.00	8.72	10.00	20.00

Mean wetting of schirmer strip was 13.21 mm on the affected side while that on unaffected side was 16.61 mm SD 8.72 mm.

Table No.3: Table showing the percentage of dry eye in the affected and non-affected side

		Count	Column N %
Affected side	Normal	51	32.9
	Dry eye	104	67.1
Unaffected side	Normal	72	46.5
	Dry eye	83	53.5

67.1% of individuals had dry eye (Schirmer strip wetting <15 mm) on the affected side and schirmer test.Results were abnormal in 53.5% of individuals on unaffected side also.

Table No. 4: Table showing comparison of stapedial reflex on affected and unaffected sides

STAPEDIAL REFLEX				
	Affected side		Unaffected side	
	N	%	N	%
Normal	16	10.3	96	61.9
No response	122	78.7	42	27.1
Not done	17	11	17	11
Total	155	100	155	100

Stapedial reflex was abnormal on the affected side in 78.7% individuals. Also, this test was abnormal in 27.1% of individuals on the unaffected side also.

Table No. 5: Table showing abnormal Nerve conduction velocity in the study population

		N	%	% of done
NCV	Normal	38	24.5%	29.85%
	Abnormal	94	60.6%	70.15%

		N	%	% of done
NCV	Normal	38	24.5%	29.85%
	Abnormal	94	60.6%	70.15%
	Not done	23	14.8%	

Of the 155 patients, 132 individuals underwent facial NCV testing. Nerve conduction velocities were abnormal in 70.15% of individuals on the affected side in whom this test was performed.

Table No.6: Table showing distribution of Blink reflex results in the study population

		Count	Column N %
BLINK REFLEX	Normal	25	16.1%
	Abnormal	111	71.6%
	Not done	19	12.3%

Blink reflex results were abnormal in 71.6% individuals on the affected side and this test could not be performed in 12.3% of the individuals.

Table No.7: Composite table showing results of topodiagnostic tests electrodiagnostic tests between Bell's and Non Bell's group of patients

		Non Bell's		Bell's		
		Count	Column N %	Count	Column N %	
Schirmer's test Affected side	Normal	18	32.7	33	33.0	0.9
	Dry eye	37	67.3	67	67.0	
Schirmer's test Unaffected side	Normal	22	40.0	50	50.0	0.2
	Dry eye	33	60.0	50	50.0	
Stapedial reflex affected side	Normal	4	7.3	12	12.0	0.2
	Abnormal	42	76.4	80	80.0	
	Not done	9	16.4	8	8.0	
Stapedial reflex unaffected side	Normal	28	50.9	68	68.0	0.09
	Abnormal	18	32.7	24	24.0	
	Not done	9	16.4	8	8.0	
Blink reflex	Normal	9	16.4	16	16.0	0.9
	Abnormal	39	70.9	72	72.0	
	Not done	7	12.7	12	12.0	
NCV	Normal	10	18.2	28	28.0	0.3
	Abnormal	38	69.1	56	56.0	
	Not done	7	12.7	16	16.0	

In comparison of Bell's palsy vs non-Bell's etiologies, no significant differences were observed between the two in results of Schirmer's test, stapedial reflex on the affected side, blink reflex and Nerve conduction velocities. Statistically significant difference was observed between Bell's and non-Bell's groups in stapedial reflex on the unaffected side due to possible subclinical involvement of the unaffected side in Bell's palsy.

Discussion

Lower motor neuron facial palsy is a common occurrence in the realm of Otorhinolaryngology. There has been a need for study of this kind to investigate the common etiological factors and recovery of the patients to aid in better management of these individuals including the role of surgical exploration of the facial nerve in the selected few.

We investigated the diverse etiologies of lower motor neuron facial palsy in our study. A total of 155 LMN facial palsies were enrolled for the study. 25.2% of the patients were from 31-40 years of age followed by 21.9% in 21-30 years. LMN facial palsy was equally distributed at 14.8% in both <20 and 51-60 years and least in >61 years age group and the mean age of presentation was 38 years ± 17 years and showed a normal distribution with 25th percentile at 25 years and 75th percentile at 51 years. In literature, the incidence of Bell's palsy is greater in patients older than 65 years and lower in children younger than age 13 years[10,11,12] and the male to female ratio for Bell's palsy is approximately equal, except for a predominance in women <20 years and a slight predominance in men older than age 40 years.[10] Peak incidence usually between the ages of 15 and 50 years.[13,14] However, in this present study, Males constituted 61.3% and females 38.7%, 95 and 60 patients respectively.

Sex incidence demonstrated trivial male predominance was observed, paralleling a study by Ayala et al [15] in 2007 which depicted 60% of facial palsy cases as masculine. In the study by Venugopal and others,[16] most common age group developing LMN facial palsy was in 31-40 years. In our study, the most common etiology identified was Bell's palsy (65.16%) which agrees with that described in world literature.[17] But in the study by Venugopal and others,[16]

trauma was the most common etiology while the second common cause identified was Bell's palsy (23.3%) possibly because they had not enrolled cases presenting to other departments like General Medicine and Neurology, which we had enrolled. Compared with Bell's palsy, Ramsay Hunt syndrome has an incidence of 4.5% to 9% of cases, at 130 cases per lakh population.[18] In our study Ramsay Hunt syndrome constituted 12 cases (7.7%),while in the study by Venugopal and others,[16] it was 5%. May and Klein [17] listed the incidence of the same as 7% in patients with facial palsy, which is similar our study.

Trauma and post reactivation constituted 11 cases (7.1%) and iatrogenic and post reactivation constituted 5 cases (3.2%), together totalling to 10.3% of the cases of LMN facial palsy in the present study. Among the 11 patients with trauma and post reactivation, 6 had facial palsy immediately following trauma and 5 had a delayed facial palsy in about 7 days' time. 5 patients had a horizontal fracture of the temporal bone, 3 had a longitudinal fracture of the temporal bone and no fracture was identified in 3 patients. In the study by Venugopal and others,[16] trauma was the most common cause (41.7%) which included iatrogenic (20%) or non-iatrogenic (21.7%) causes and in their study, the explanation for trauma being more common was that there were higher numbers of road traffic accident referrals. In their study, 16.7% cases showed fracture of temporal bone (by and large longitudinal) and 5% showed blunt trauma to temporal region presenting

with late onset palsy. Eftekharian A[19] in 2005 described that while dissecting the cholesteatoma in epitympanum and supratubal recess, the geniculate ganglion and proximal segment of facial nerve are at risk of injury.

Topodiagnostic tests showed majority of Bell's palsy involving supragenicular region (62.38%) in our study, this is in accordance with the reports of Fisch,[20]and Adour[21] and Venugopal and others[16] regarding bottleneck of facial canal. In comparison of Bell's palsy vs non-Bell's etiologies, no significant differences were observed between the two in results of Schirmer's test, stapedial reflex on the affected side, blink reflex and Nerve conduction velocities. Statistically significant difference was observed between Bell's and non-Bell's groups in stapedial reflex on the unaffected side due to possible subclinical involvement of the unaffected side in Bell's palsy.

In our study, 63 out of 101 patients with Bell's palsy (62.37%) had the lesion in the geniculate and supragenicular region. Unfortunately, the accuracy of localization with topodiagnostic testing has been disappointing. This is not a surprising finding in Bell's palsy in which the lesion is a diffuse demyelination throughout the nerve. However, topodiagnostic testing also has been disappointing in tumors in which a sharply defined site of lesion was expected. In complete focal lesions, as in trauma, topognostic tests are usually reliable but unnecessary, because clinical examination and imaging suffice to describe the site of the lesion. If multiple or extensive lesions of the facial nerve are present, topognostic tests can predict only the most proximal lesion and provide little to no information concerning additional distal facial nerve lesions.[3]

Consequently, many investigators have noted paradoxic and misleading results in applying topognostic testing principles in the evaluation of patients with Bell's palsy. In contrast to traumatic injuries, Bell's palsy is typically a mixed and partial lesion with varying degrees of conduction block and degenerative changes within different fibers and fascicles of the nerve trunk; therefore topognostic tests are not expected to provide precise information about the level of the lesion.In our study, we found disparate results with Bell's palsy with respect to topodiagnostic tests where .We attributed these disparate results to patchy involvement of the nerve and partial lesion with varying degrees of conduction block and degenerative changes within different fibers at different level and fascicles of the nerve trunk.

These findings connote a good prognosis for all cases of LMN facial palsy with about 43.9% of all etiologies of LMN facial palsy and about 49% of the Bell's palsy group recovering normal facial nerve functions and 38.7% of all etiologies of LMN facial palsy and about 41.5% of the Bell's palsy group recovering near-normal facial nerve functions by the end of four weeks from the first appearance of symptoms. If we can combine near normal and fully normal facial functions, nearly, 82.6% of individuals of all etiologies of LMN facial palsy and about 90.5 % of the Bell's palsy group recover normal to near normal facial nerve function by the end of 28 days' time. These findings were statistically significant ($p<0.001$). Similar findings by Yeo and Lee in 2007 suggested that 96% of Bell's palsy recovered spontaneously.[269] Hence, the role of facial nerve decompression surgery in LMN facial palsy is possibly limited to the extreme few, especially cases of

trauma related facial palsy and may be unwarranted in the majority as there is an expected spontaneous recovery in most of the individuals.

Conclusion

Majority of the patients in both Bell's and Non-Bell's groups had topognostic level of lesion at Geniculate and supra geniculate level.But lesions lower down showed paradoxical and confusing results.This could possibly be explained by incomplete and patchy involvement of the facial nerve in Bells palsy. Hence, the role of facial nerve decompression surgery in LMN facial palsy is possibly limited to the extreme few, especially cases of trauma related facial palsy and may be unwarranted in the majority as there is an expected spontaneous recovery in most of the individuals.

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