

Aetiological Profile of Patients with Acquired Lower Motor Neuron Facial ParalysisKrithika Deepak Mathraden¹, Swathilal S A², Sreejith M K³, Sagesh M⁴¹Senior Resident, Department of ENT, Government Medical College Kannur, Kerala²Associate Professor, Department of ENT, Government Medical College Manjeri, Kerala^{3,4}Assistant Professor, Department of ENT, Government Medical College Kozhikode, Kerala

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Abstract:

Background: Facial nerve paralysis is a disorder that severely affects a patient's quality of life causing severe emotional, social and vocational handicaps. The diverse aetiological factors make it a huge diagnostic challenge for doctors. It is important to understand the cause and nature of the disease to treat and rehabilitate patients with facial nerve paralysis.

Aim: To analyse the aetiological profile of acquired lower motor neuron facial paralysis and to study the socio-demographic characteristics of the patients, grade the disease severity and assess the outcome in disease progression following various treatment modalities.

Methods: A prospective, longitudinal and descriptive study of 18 months duration conducted in 86 patients who presented with LMN facial nerve paralysis. After history taking and clinical examination, including severity grading using House-Brackmann grading system, patients were investigated and treated with appropriate medical and surgical modalities. The recovery status of patients was assessed after 1,3- and 6-month follow-up by grading the improvement with House Brackmann grading system.

Results: Of the 86 patients with acquired lower motor neuron facial paralysis, Bell's palsy was the commonest followed by temporal bone trauma. Most cases presented with House-Brackmann Grade III stage. Majority of the cases were managed conservatively with steroids and physiotherapy. Surgical management included facial nerve decompression, nerve grafting, tumour excision and modified radical mastoidectomy for disease clearance. Most of the cases showed a complete recovery of facial function by six months with Bell's palsy having the best prognosis.

Conclusion: Among the causes of peripheral facial nerve paralysis, Bell's palsy and post-traumatic facial paralysis were the commonest. While most cases can be managed conservatively, some need careful evaluation and early surgical intervention for a complete or partial recovery.

Keywords: Bell's palsy, House-Brackmann grade, lower motor neuron facial paralysis, post-traumatic facial paralysis.

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Introduction

Facial nerve paralysis is a common clinical condition encountered by otorhinolaryngologists. Patients who suffer from facial paralysis experience not only functional consequences; but also, the psychological impact of a change in self-image and impaired communication ability.

Causes of facial nerve paralysis are numerous: congenital, trauma, neurological, infection, metabolic, neoplastic, toxic, autoimmune, iatrogenic, and idiopathic. More than 40 different causes of facial paralysis are known, out of which, 75% are usually due to Bell's palsy or secondary to trauma.[1] The paralysis gradation is important for clinical and post-operative follow-up. Out of the various proposed methods of gradation, the House-

Brackmann Scoring system is the most widely used.[2] Management of facial nerve palsy is individualized and may include observation, medical management with pharmacological agents, surgical interventions, physiotherapy and psychological counselling.[3] In this study, various aetiologies of acquired lower motor neuron (LMN) type of facial paralysis are analysed along with sociodemographic characteristics, grading of the disease severity using the House Brackman grading system and assessment of the disease progression after management with suitable modalities after 1, 3 and 6 months follow up.

Aim: The main objective of this study was to analyse the aetiological profile of patients with

acquired lower motor neuron type of facial paralysis. The other objectives were to analyse the socio-demographic characteristics of the patients, to grade the severity of the disease in patients using the House- Brackmann grading system; and to assess the immediate outcome in disease progression following treatment using different modalities after 1, 3 and 6 months follow up.

Methodology

A longitudinal and descriptive study was conducted at the ENT department in a tertiary care teaching hospital for a period of 18 months from 1st July 2021 to 31st December 2022. All patients presenting with acquired lower motor neuron type of facial nerve paralysis during the initial 12 months of the study period were enrolled for the study. Patients presenting with LMN facial palsy associated with other congenital syndromes and the patients with upper motor neuron type of facial nerve palsy were excluded from the study. A convenience sampling method was adopted and a total of 86 patients were enrolled for the study.

After obtaining approval from the institutional research committee and institutional ethics committee and after getting informed consent, eligible patients were interviewed. Using a semi-structured questionnaire, details were collected including socio-demographic details, a detailed clinical history including the time of onset of symptoms, rapidity of progression and duration of paralysis, previous episodes, family history, associated (auditory, vestibular and neurological) symptoms, medical co-morbidities and history of previous trauma or surgeries. This was followed by a detailed clinical examination and grading of the severity using the House Brackmann grading system. Investigations like baseline blood and urine investigations, and X-ray studies were done in all patients and relevant specific investigations like topodiagnostic tests, CT /MRI imaging and nerve conduction studies were done on an individual basis when required. From these, the various aetiologies of the disease were analysed and broadly classified into idiopathic, traumatic, iatrogenic, infective and neoplastic categories and subdivided into specific causes. Also, disease progression was assessed after management with suitable modalities after 1, 3 and 6 months follow up by clinically grading the improvement in facial paralysis with the help of the House Brackmann (HB) grading system.

Results

In our study, the most common aetiology for LMN facial paralysis was Bell's palsy with 29 cases (30.2%), followed by post-traumatic with 25 cases (26%). Other causes were infections with 16 (18.6%) cases, neoplasms with 9 (10.5%) cases and post-operative/iatrogenic with 7(8.1%) cases. All

25 cases of post-traumatic facial nerve paralysis in our study were due to temporal bone fractures following head injury. Out of them, the most common type of fracture was longitudinal (15 cases; 60%) and the remaining were transverse (5 cases; 20%) and mixed (5 cases; 20%). Most of the patients presented with delayed facial paralysis that developed a few days after the trauma. Only 7 of them developed early facial paralysis on the day of trauma itself. There were no cases of extra-temporal facial nerve injury in our study. Out of the 7 cases of iatrogenic trauma, the most common surgery that resulted in facial paralysis was cerebellopontine angle tumour excision, accounting for 4 cases; that is 57.1% of all the iatrogenic causes. The others were 2 (28.6%) cases of facial paralysis following modified radical mastoidectomy and 1(14.3%) case following cortical mastoidectomy.

The most common cause among infections causing facial paralysis was found to be skull base osteomyelitis, accounting for 9 (56.3%) cases, which was followed by 3 (18.7%) cases each of chronic otitis media squamous and herpes zoster and one (6.3%) case of acute otitis media. The most common tumour that resulted in facial paralysis in our study was paraganglioma affecting 5 cases and accounting for 55.6% of all tumours. Others were 1 (11.1%) case each of middle ear malignancy and malignant parotid tumour and 2 (22.2%) cases of vestibular schwannoma.

The mean age of our study population was 46.55 years with a minimum age of 3 years and a maximum of 78 years with a standard deviation of 14.92. LMN facial palsy was more common in males with 58 cases (67.4%) with a male to female ratio of 2.1:1. The majority of patients belonged to the age group of 41-60 years (45.35%), followed by 21-40 years (34.88%). Most of the patients with Bell's palsy belonged to the age group of 41-60 years, accounting for 16 (55.2%) cases. The next common age group of presentation was 21-40 years, accounting for 9 (31%) cases. In our study, most of the cases with post-traumatic facial paralysis belonged to the age group of 21-40, accounting for 16 (64%) cases. This might be due to the increasing number of road traffic accidents causing head injuries among the younger generation.

Overall, most of our cases had moderate facial dysfunction (HB grade III) at presentation, accounting for 51 (59.3%) cases. 19 (22.1%) cases had only mild facial dysfunction (HB grade I) and the remaining 16 (18.6%) cases had moderately severe facial dysfunction (HB grade IV). There were no cases of severe facial dysfunction (HB grade V) or total facial paralysis (HB grade VI) in our study. 65.5% of Bell's palsy cases in our study presented with an HB Grade of III and the

remaining 34.5% of cases presented with an HB grade of II. Almost 60% of cases with post-traumatic facial paralysis presented with an HB grade III, which was followed by 24% of cases with an HB grade II and 16% of cases with an HB grade IV.

Out of the 86 cases, 66 (76.7%) cases were managed medically with steroids with/without antivirals/antibiotics/anti-inflammatory agents and physiotherapy. These included all cases of Bell's palsy, 20 cases of delayed onset post-traumatic facial paralysis, 13 cases of facial paralysis following infections except chronic otitis media squamous and 4 cases of iatrogenic facial paralysis. The remaining 20 cases were managed by appropriate surgical interventions, followed by post-operative steroid therapy. Surgical excision of the tumour was done in 9 cases of facial paralysis secondary to the tumour with an attempt to repair the facial nerve whenever possible. Out of the 11 cases of surgical interventions for facial paralysis, facial nerve decompression was done in 9 (81.8%) cases -5 cases of post-traumatic facial paralysis, 1 case of post-MRM facial nerve paralysis and 3 cases of chronic otitis media squamous (done along with modified radical mastoidectomy). The remaining 2 (18.2%) cases, which were facial nerve paralysis secondary to paragangliomas, underwent sural nerve grafting along with tumour excision. Only 13 (15.1%) patients with LMN facial paralysis showed complete recovery to normal function at the end of 1 month of follow-up. Thirty-four (39.5%) patients showed improvement in their HB grade, while 36 patients showed no recovery. By the end of three months, 27 (31.4%) patients

had complete recovery of facial nerve functions, 41 (47.7%) patients had partial recovery and 15 (17.4%) patients showed no signs of recovery. Among the 86 patients under study, 44 (51.2%) patients showed complete recovery, 26 (30.2%) patients showed partial recovery while 7 (8.1%) of them showed no signs of recovery at the end of 6 months. Two patients were lost to follow up and 4 of them expired during the course of the study.

In our study, Bell's palsy showed the best prognosis with 26 (89.7%) out of 29 patients showing complete recovery and 2 (6.9%) patients showing partial recovery. Of the 25 cases of post-traumatic facial paralysis, 16 (64%) cases showed complete recovery. All of them were delayed onset paralysis which was managed medically with steroids and anti-inflammatory agents. Seven (28%) cases, including 5 cases managed surgically with facial nerve decompression, showed partial recovery. Out of the 16 cases of facial paralysis following infections, only 2 (12.5%) cases showed complete recovery. Two cases of skull base osteomyelitis showed no signs of recovery and 2 of them expired during the period of follow-up. Out of the 9 cases of facial paralysis secondary to tumours, none of them showed complete recovery. Of the 7 cases of post-operative facial paralysis, 2 cases showed complete recovery within 1 month of follow-up. These were grade II facial paralysis following cortical and modified radical mastoidectomy respectively, which were managed conservatively with steroids. The remaining 5 cases which showed only partial recovery include 1 case of post-modified radical mastoidectomy facial paralysis and 4 cases of post-CPA tumour excision.

Table 1: Recovery status in various aetiological categories at 6-month follow-up

S.No.	Aetiology Category	Complete Recovery		Partial Recovery		No Recovery		Expired/lost follow-up		Total	
		Number	%	Number	%	Number	%	Number	%	Number	%
1	Idiopathic	26	89.7	2	6.9	0	0	1	3.4	29	100
2	Trauma	16	64	7	28	0	0	2	8	25	100
3	Infection	2	12.5	10	62.5	2	12.5	2	12.5	16	100
4	Neoplasm	0	0	3	33.3	5	55.6	1	11.1	9	100
5	Post-operative	2	28.6	5	71.4	0	0	0	0	7	100

Discussion

In the present study, 86 patients with acquired LMN facial paralysis were studied and demographic data, aetiology, the severity of facial paralysis and recovery status after various treatment modalities were assessed. Among the 86 cases of facial paralysis in our study, the most common cause of facial paralysis was idiopathic (Bell's palsy) with 29 cases, accounting for 33.7% of total cases. The second most common cause was trauma with 25 cases (29.1%). In the study by Schiatkin B and May M on 3454 patients with

facial paralysis, Bell's palsy was present in 51.6% of cases, which was followed by 23% of cases of trauma, 5.3 % of tumour, 4% of infection, 7% of Herpes Zoster, 4% of congenital facial paralysis, 4% of hemifacial spasm and 0.8% of central facial paralysis.[1] Mark May conducted a study in 1986 with 1912 cases of facial palsy in which Bell's palsy was the most common cause of facial paralysis with 1082 cases (57%), followed by trauma (both surgical and head injury) accounting for 375 cases (20%).[4]

Other causes were Herpes Zoster (145 cases, 8%), tumours (126 cases, 6%), infection (78 cases, 4%), birth trauma (62 cases, 3%) and others (44 cases, 2%). In another study on aetiologies of facial paralysis conducted by Junior NA et al in 2009, Bell's palsy was found to be the most frequent aetiology (53.7%), followed by traumatic (24%) Ramsay hunt syndrome (9.2%), cholesteatoma (5.5%), malignant otitis externa (3.7%) and acute otitis media (3.7%). [5] All cases of traumatic facial palsy in our study were due to fractures of the temporal bone, with most cases (60%) being longitudinal fractures and less commonly transverse (20%) and mixed (20%) fractures. Hemanth Chopra in his study of 48 cases of traumatic facial paralysis, found that the incidence of longitudinal fracture was 51% and transverse fracture was 19%. [6]

The third most common cause of facial paralysis in our study was infections, accounting for 16 cases (18.6%). In the study by Sudhakaran et al, infections accounted for 17.6% of cases, which included 2 cases of chronic otitis media squamous, 3 cases of malignant otitis externa and 1 case of herpes zoster. [7] Our study included 3 cases (3.5%) of Herpes Zoster. In Mark May's and Junior et al study, Herpes Zoster was the third most common cause with an incidence of 8% and 9.2% respectively. [4,5] In our study, the incidence of facial paralysis following infections except Herpes Zoster was 15.1% of total cases (13 cases) and included 1 case of acute otitis media (1.2%), 3 cases of Chronic otitis media squamous (3.5%) and 9 cases of skull base osteomyelitis (10.5%). In the study by Junior et al, facial paralysis was seen in 2 cases of acute otitis media (3.7%), 3 cases of chronic otitis media squamous (5.5%) and 2 cases of malignant otitis externa (3.7%). [5]

In our study, there were 7 cases of post-operative facial paralysis accounting for 8.1% of all cases. The most common surgery that resulted in facial palsy was cerebellopontine angle tumour excision (4 cases, 57.1 % of post-operative facial palsy), followed by mastoidectomies. In the study by Douglas JG, Clough S and Derald EB involving 22 patients of iatrogenic facial palsy, the most common surgery leading to facial nerve injury was found to be mastoidectomy accounting for 55% of cases. [8] The second most common surgery was the removal of exostosis. In the study by Sudhakaran et al, there were 4 cases of iatrogenic facial palsy, which includes 2 cases following mastoidectomy, one case following cochlear implantation surgery and one case following parotidectomy. [7]

Regarding the severity of facial paralysis, majority of cases (51, 59.3%) presented with a House-Brackmann grade of III, followed by 19 (22.1%) cases with a grade of II and 16 (18.6%) cases with

a grade of IV. A HB grade of III at presentation was seen in 66.5% of Bell's palsy cases and the remaining 34.5% cases had grade II paralysis. In the case of post-traumatic facial paralysis, 60% of cases presented with grade III paralysis, 24% cases with grade II and the remaining 16% cases with grade IV paralysis. In their study, Zohrevandi et al in 121 patients, the most common HB grades of nerve damage were IV and V. [9] In the study by Sudhakaran et al, 47.1% of cases presented with a House-Brackmann grade of IV, followed by 32.4% of cases with a grade of III. [7] Sixty-six (76.7%) cases were managed medically with steroids with or without antivirals/ antibiotics/ anti-inflammatory agents and physiotherapy. The remaining 20 cases were managed by appropriate surgical interventions. Out of the 11 cases of surgical interventions for facial paralysis, facial nerve decompression was done in 9 (81.8%) cases with accompanying modified radical mastoidectomy in three cases of chronic otitis media squamous. The remaining two (18.2%) surgically managed cases underwent sural nerve grafting. Surgical excision of the tumour was done in 9 cases of facial paralysis secondary to tumour.

In the study by Ahmed et al, two-third of the study group received steroids, one-third received anti-viral, one-fourth received antibiotics in combination with steroids and anti-viral drugs. [10] In the study by Sudhakaran et al, conservative medical treatment with steroids, antivirals and physiotherapy was employed in 55.9% of cases and surgical management was employed in 32.4% of cases (those with temporal bone fractures, iatrogenic trauma, cholesteatoma, and a single case of Bell's palsy that did not recover with steroid). [7] Of the surgically managed cases, 9 cases underwent facial nerve decompression and two cases underwent repair by nerve grafting. In a meta-analysis by Quant et al, six trials were included with a total of 1145 patients with Bell's palsy of which 574 patients received steroids alone and 571 patients received steroids and antivirals. [11] The pooled odds ratio for facial muscle recovery showed no benefit of steroids plus antivirals when compared with steroids alone. At the end of 6 months of follow-up in our study, 44 (51.2%) patients showed complete recovery, 26 (30.2%) patients showed partial recovery and 7 (8.1%) patients showed no signs of recovery (Table 1). Two patients did not come for follow-up and 4 of them expired. In the study by Peitersen, out of the 2570 cases of peripheral facial nerve paralysis, facial nerve function returned within 3 weeks in 85% of patients and after 3-5 months in the remaining 15%. [12] In the study by Ahmed et al, 34.9% of patients did not respond to treatment and 65.1% responded to treatment in different grades, where 72% were of good response and 28% were of poor response. [10] In the study by Sudhakaran et

al, at the end of one-year follow-up, 41.2% of cases showed a full recovery, 35.3% of cases showed a partial recovery, 17.6% of cases showed no signs of recovery and 5.9% of cases expired. [7] In our study, 89.7% of patients with Bell's palsy showed a full recovery and 6.9% of patients showed a partial recovery after 6 months of follow-up. Adour et al in his study showed that there was complete recovery in 90% of patients and 10% had partial recovery.[13] In the study by Pietersen, 84% of cases showed complete recovery and the remaining 16% had incomplete recovery.[12] All the 3 cases of facial palsy due to COM Squamous in our study showed partial recovery of facial function. However, in a study by Savic and Djemic, involving 64 patients of facial paralysis due to COM squamous, who underwent modified radical mastoidectomy with facial nerve exploration, 70% of patients had complete recovery of facial function. [14] Out of the 9 cases of facial paralysis secondary to tumours, none of them showed complete recovery. 33.3% of cases showed partial recovery and 55.6% of cases showed no signs of recovery post-tumour excision. Out of the 7 cases of post-operative facial paralysis, 2 (28.6%) cases showed complete recovery within 1 month of follow-up. The remaining 5 (71.4%) cases showed only partial recovery at the end of 6 months.

Conclusions

Bell's palsy and post-traumatic facial paralysis were the most common causes of facial paralysis in this study. Majority of the cases (76.7%) were managed medically with steroids along with physiotherapy. Surgical management included facial nerve decompression, nerve grafting, tumour excision and modified radical mastoidectomy for disease clearance. Majority of the cases showed a complete recovery of facial function at the end of six months of follow-up and a very few of them showed no signs of recovery. Bell's palsy showed the best prognosis with 89.7% of patients showing complete recovery while patients with post-traumatic facial paralysis showed a reasonable recovery. Only 12.5% of cases with facial paralysis following infection and 28.6% of iatrogenic facial paralysis showed complete recovery. No case of facial paralysis secondary to neoplasms showed full recovery.

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References

1. Barry S, Mark M. Disorders of Facial nerve. Scott-Brown's Otolaryngology, 6thed, Kerr AG, Bueth JB. Butterworth-Heinmann, London, 1997: vol 3; 1-38.
2. House JW, Brackmann DE: Facial nerve grading system. Otolaryngol Head Neck Surgery, 1985;93: 146 – 7.
3. Samy RN, Gantz BJ. Surgery of the Facial nerve.in Glasscock-Shambaugh, Surgery of the Ear, 5th ed, Glasscock ME, Gulya AJ, eds. BC Decker, Canada, 2003:615-639.
4. Mark M, Barry M S, Michael P, Jurg U, Erik P, Susan RK, Idiopathic Palsy, Herpes Zoster Cephalicus and other facial nerve disorders of viral origin. The Facial Nerve Second Edition, Thieme, New York 2000: 319 – 338.
5. Junior NA, Jarjura J, Junior J, Gignon VF, Kitice AT, Prado LSA. Facial Nerve Palsy: Incidence of Different etiologies in a Tertiary Ambulatory. International Archives of Otolaryngology 2009; 13(2). 609-614.
6. Hemanth C, Khurana AS, Munjal M, Vanitha C, Anju M, Sobti MK. Facial nerve paralysis in head injury. Indian Journal of Otology, 2002; 8:86-89.
7. Sudhakaran SK, Madayambath S. Facial nerve paralysis- a clinical study. Int J Otorhinolaryngol Head Neck Surg 2019; 5:1309-14.
8. Douglas GJ, Clough S, Derald EB. Iatrogenic Facial nerve injury during otological surgery. Laryngoscope 1994; 104: 114-116.
9. Zohrevandi B, Monsef Kasmaee V, Asadi P, Tajik H. Report of 121 cases of Bell's Palsy referred to the emergency department. Emergency. 2014; 2(2):66-70.
10. Moala H, Ahmed S, Yousif YM (2017) Etiology and Clinical Presentations of lower motor neuron Facial Nerve Palsy in Khartoum, Sudan. J Ear Nose Throat Disord 2 (1):1017.
11. Quant EC, Jeste SS, Muni RH, Cape AV, Bhussar MK, Peleg AY. The benefits of steroids versus steroids plus antivirals for treatment of Bell's palsy: a meta-analysis. BMJ. 2009; 339: b3354
12. Pietersen E. Bell's palsy: the spontaneous course of 2500 peripheral facial nerve palsies of different etiologies. Acta Otolaryngol Suppl.2002; 549:4
13. Adour KK, Byl FM, Hilsinger RL. The true nature of Bell's palsy: Analysis of 1000 consecutive cases. Laryngoscope 1978; 88:787-811.
14. Savic DL, Djemic DR: Facial paralysis in CSOM. Clinical Otolaryngology 1989; 14:515-17.