



ORIGINAL ARTICLE

Clinico Etiological Profile of Patients with Acute Lower Motor Neuron Facial Palsy and Treatment Outcome of Bell's Palsy: A Prospective Study in a Tertiary Care Centre

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Accepted: 23-January-2026 / Published Online: 3-February-2026

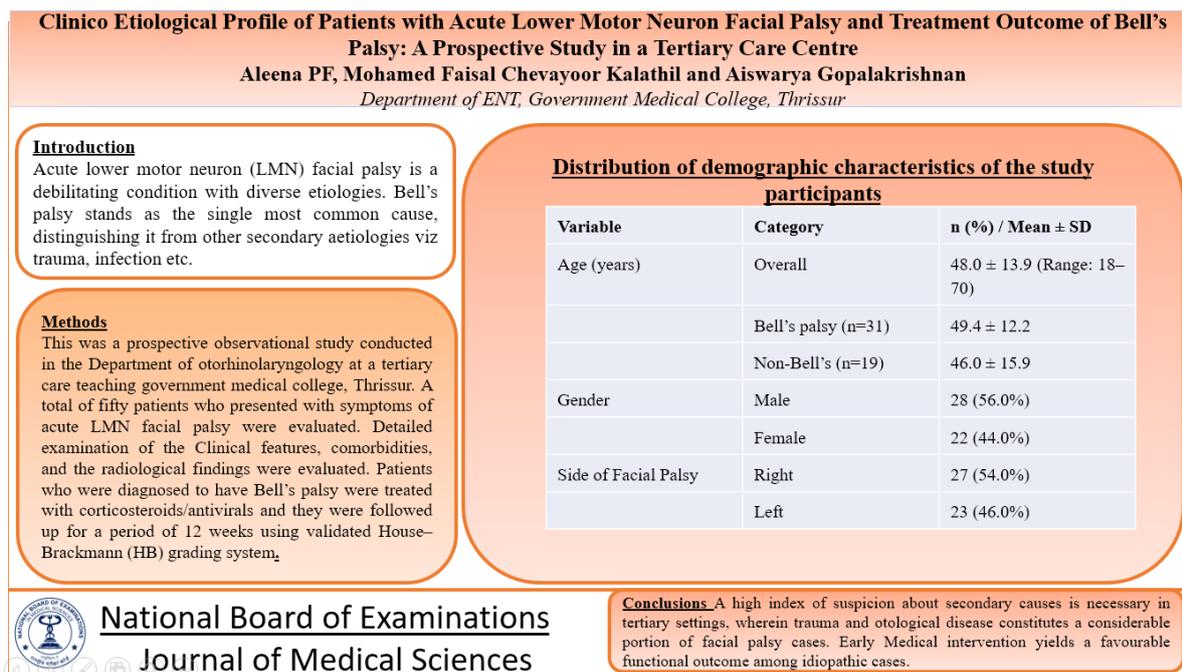
Abstract

Introduction: Acute lower motor neuron (LMN) facial palsy is a debilitating condition with diverse etiologies. Bell's palsy stands as the single most common cause, distinguishing it from other secondary aetiologies viz trauma, infection etc. **Methods:** This was a prospective observational study conducted in the Department of otorhinolaryngology at a tertiary care teaching government medical college, Thrissur. A total of fifty patients who presented with symptoms of acute LMN facial palsy were evaluated. Detailed examination of the Clinical features, comorbidities, and the radiological findings were evaluated. Patients who were diagnosed to have Bell's palsy were treated with corticosteroids/antivirals and they were followed up for a period of 12 weeks using validated House–Brackmann (HB) grading system. **Results:** The mean age of the study cohort was 48.0 ± 13.9 years. Bell's palsy was found to be the predominant etiology (62%), which was followed by temporal bone fracture (18%) and then chronic otitis media (10%). Otalgia was observed as a significant associated symptom (46%). Among the diagnosed Bell's palsy patients (n=31), two thirds (67.7%) achieved good recovery (HB Grade I–II) at the end of 12 weeks. **Conclusion:** A high index of suspicion about secondary causes is necessary in tertiary settings, wherein trauma and otological disease constitutes a considerable portion of facial palsy cases. Early Medical intervention yields a favourable functional outcome among idiopathic cases.

Keywords: Acute facial palsy, Bell's palsy, Lower motor neuron facial nerve paralysis, House–Brackmann grading

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Graphical Abstract



Introduction

Facial nerve is the most commonly paralyzed cranial nerve because its complex anatomical course through the temporal bone. Acute lower motor neuron (LMN) facial palsy is one among the condition that results in significant functional morbidity along with corneal exposure and oral incompetence which caused profound psychological distress [1].

Facial nerve is one of the commonly paralyzed cranial nerves. Bell's palsy (also known as idiopathic facial paralysis) is considered as the most cited cause of LMN facial palsy, which accounts for a total of 60–75% of cases in the global literature [2] However, "Bell's palsy" remains as a diagnosis of exclusion. In a tertiary care setting, a significant proportion of these cases may arise from secondary causes such as the temporal bone trauma, chronic otitis media (COM), herpes zoster oticus (Ramsay Hunt syndrome), and other neoplasms [3] Differentiating these

secondary causes is essential, since the management often requires surgical intervention (e.g., facial nerve decompression or mastoidectomy) rather than medical treatment.

The prognosis is also variable. While Bell's palsy has a generally favorable natural history, evidence suggests that outcomes are optimized with the early administration of systemic corticosteroids and antivirals [4,5]. This study aims to delineate the clinicoetiological profile of LMN facial palsy in a South Indian tertiary care center and evaluate the functional recovery of Bell's palsy patients over a 12-week period.

Methods

Study Design and Ethical Considerations

This prospective observational study was conducted in the Department of ENT at Government Medical College, Thrissur. The study protocol was reviewed and approved by the Institutional Ethics

Committee (Reg. No. EC/NEW/INST/2022/KL/0176) on June 21, 2023. The approved protocol number was IEC/GMCTSR/2023/084. The study was conducted in accordance with the submitted research proposal and we followed all the ethical principles as per Helsinki's declaration.

A total of 50 participants presenting with acute onset LMN facial palsy were recruited. Patients of all ages and genders presenting within 2 weeks of symptom onset were included in the study. Patients with congenital facial palsy, upper motor neuron lesions (sparing the forehead), or recurrent facial palsy were excluded from the study.

A structured study proforma was designed with sociodemographic details, clinical details, comorbidities and clinical examination. Clinical details focussed on onset of the symptoms, progression, associated features like otalgia, vesicles and disturbance in taste. Pure tone audiometry (PTA) and Impedance audiometry was done to assess hearing status and stapedial reflex. Schirmer's Test was conducted to evaluate the greater superficial petrosal nerve function. All study participants with history of trauma, discharge from ear or non recovery underwent High-Resolution Computed Tomography (HRCT) of the temporal bone. MRI was done selectively.

Treatment and Outcome Measures

Patients with Bell's palsy were treated with oral prednisolone (1 mg/kg/day tapered over 10 days) and antivirals (acyclovir/valacyclovir) for 7 days. Eye care (lubricating drops and taping) was instituted for all patients with lagophthalmos. Secondary causes were managed surgically or medically as indicated.

Functional recovery was assessed using the House-Brackmann (HB) grading system [6]. Assessment was conducted at presentation, 6 weeks, and 12 weeks.

Results

Demographic Profile

A total of 50 patients presenting with acute lower motor neuron (LMN) facial palsy were included in the study. The cohort consisted of 31 cases (62%) of Bell's palsy and 19 cases (38%) of secondary facial palsy. The mean age of the overall study population was 48.0 ± 13.9 years (range: 18–70 years). Patients with Bell's palsy were slightly older (mean 49.4 ± 12.2 years) compared to those with secondary facial palsy (mean 46.0 ± 15.9 years). There was a male predominance (56%) and a slight predilection for right-sided involvement (54%). Detailed demographic characteristics are summarized in Table 1.

Clinical Characteristics

Motor asymmetry with deviation of the angle of the mouth was present in all patients. Lagophthalmos was present in 88% of the study participants ($n=44$), followed by otalgia (46%) and dysgeusia (40%). Epiphora was noted in 34% of the study population and 26% had dry eye. Viral Prodrome was noted in 14% of the study population (Table 2).

Table 3 details the Otological and neurological assessment. Otological manifestations were predominantly associated with the secondary etiologies. 26% of the study participants had hearing loss, 18% had tinnitus and 16% had active ear discharge. In 4% of the study participants Vesicular eruptions which is characteristic of the herpes zoster oticus was observed.

Comorbidities and Risk Factors observed are detailed in Table 4. Twenty eight percent of the study population has systemic comorbidities. Diabetes mellitus (14%) and hypertension (12%) were the most common metabolic risk factors. Trauma was a significant specific risk factor, present in 18% of cases (n=9).

Diagnostic Evaluation

Diagnostic findings are summarized in Table 5. Otoscopy was normal in 60% of patients; however, pathological findings such as tympanic membrane perforation, attic erosion, and cholesteatoma were observed exclusively in the secondary facial palsy group. Pure-tone audiometry revealed conductive or mixed hearing loss in 36% of patients. Schirmer's test indicated reduced lacrimation in 32% of the cohort. High-resolution computed tomography (HRCT) of the temporal bone, performed in 21 indicated cases, confirmed temporal bone fractures in nine patients and squamous chronic otitis media in five.

Etiology

Idiopathic (Bell's) palsy was the most common diagnosis (62%). Among secondary causes, temporal bone fractures due to trauma were the leading etiology (18%), followed by chronic otitis media

with squamous disease (10%) and herpes zoster oticus (4%). The complete etiological distribution is provided in Table 6.

Treatment and Management

All patients (n=50) received systemic corticosteroids. Antiviral therapy was administered to 60% of patients, primarily those with Bell's palsy and herpes zoster oticus. Physiotherapy was incorporated into the management plan for 38% of patients. Surgical intervention was required in two cases (4%) to manage underlying temporal bone pathology in the non-Bell's group (Table 7).

Recovery of the facial nerve was assessed using the House Brackmann grading system. In the Bells Palsy group, more than 90% had Grade 3 and above during presentation with symptoms to our hospital. After 12 weeks around 32% only had grade 3 and above. The above observation was statistically significant (Table 8).

Exposure keratitis was observed among 6% of the study participants. Hemifacial spasm or synkinesis was not observed in any of the study participants during the study.

Table 1. Distribution of demographic characteristics of the study participants.

Variable	Category	n (%) / Mean \pm SD
Age (years)	Overall	48.0 \pm 13.9 (Range: 18–70)
	Bell's palsy (n=31)	49.4 \pm 12.2

	Non-Bell's (n=19)	46.0 ± 15.9
Gender	Male	28 (56.0%)
	Female	22 (44.0%)
Side of Facial Palsy	Right	27 (54.0%)
	Left	23 (46.0%)

Table 2. Clinical Features at Presentation (N = 50)

Symptom	Present (n, %)	Absent (n, %)
Difficulty in eye closure	44 (88%)	6 (12%)
Ear ache (Otalgia)	23 (46%)	27 (54%)
Taste abnormality	20 (40%)	30 (60%)
Watering of eye (Epiphora)	17 (34%)	33 (66%)
Dry eye	13 (26%)	37 (74%)
Paraesthesia	7 (14%)	43 (86%)
Viral prodrome	7 (14%)	43 (86%)
Dizziness	2 (4%)	48 (96%)
Diplopia	1 (2%)	49 (98%)

Table 3. Otological and Neurological Findings (N = 50)

Finding	Yes (n, %)	No (n, %)
Hearing loss	13 (26%)	37 (74%)
Tinnitus	9 (18%)	41 (82%)
Ear discharge (Otorrhea)	8 (16%)	42 (84%)
Vesicles (Herpes Zoster Oticus)	2 (4%)	48 (96%)

Table 4. Comorbidities and Risk Factors (N = 50)

Condition	Frequency (n)	Percentage (%)
None	23	46%
Trauma	9	18%
Diabetes mellitus	7	14%
Hypertension	6	12%
Combined (DM/HTN/CAD)	2	4%
Prior ear surgery	2	4%
Pregnancy / Postpartum	1	2%

Table 5. Diagnostic Evaluation Results

Test	Finding	n (%)
Otoscopy	Normal	30 (60%)
	Attic erosion / Cholesteatoma	5 (10%)
	Tympanic Membrane (TM) perforation	2 (4%)
	TM retraction	3 (6%)
	Congestion / Acute Otitis Media	2 (4%)
	Other	3 (6%)
Schirmer Test	Normal Lacrimation	34 (68%)
	Reduced Lacrimation	16 (32%)
Pure Tone Audiometry	Normal	32 (64%)
	Conductive / Mixed Hearing Loss	18 (36%)
Tympanometry	Normal (Type A)	10 (20%)
	Type B	13 (26%)
	Type C	4 (8%)

	Other	2 (4%)
Imaging (HRCT/MRI)	Normal HRCT	5 (10%)
	Temporal bone fracture	9 (18%)
	COM – Squamous disease	5 (10%)
	Other abnormalities	2 (4%)
	MRI Normal (All performed cases)	100%

Table 6. Etiological Distribution of LMN Facial Palsy (N = 50)

Etiology	n	%
Bell's palsy (Idiopathic)	31	62%
Temporal bone fracture	9	18%
COM – Squamous disease	5	10%
Herpes zoster oticus	2	4%
Other causes*	3	6%

- *Includes Acute Otitis Media, Mucosal COM, and Glomus Tympanicum.*

Table 7. Treatment Modalities Administered (N = 50)

Treatment Modality	n (%)	Note
Systemic Corticosteroids	50 (100%)	Administered to all patients
Antivirals	30 (60%)	Primarily for Bell's palsy & HZO
Physiotherapy	19 (38%)	Facial nerve exercises
Surgical Management	2 (4%)	Indicated for non-Bell's cases only

Table 8. House–Brackmann (HB) Facial Nerve Grading Over Time (Bell's Palsy Group, n = 31)

HB Grade	Baseline (n, %)	At 6 Weeks (n, %)	At 12 Weeks (n, %)
Grade I (Normal)	1 (3.2%)	2 (6.5%)	8 (25.8%)
Grade II (Mild)	2 (6.7%)	12 (38.7%)	13 (41.9%)
Grade III (Moderate)	8 (26.7%)	7 (22.6%)	9 (29.0%)
Grade IV (Mod. Severe)	15 (50.0%)	10 (32.3%)	1 (3.2%)
Grade V (Severe)	5 (16.7%)	0 (0.0%)	0 (0.0%)

Discussion

The mean age of the study population was 48 years. This aligns with other studies done. Peitersen's "landmark study" on 2,500 patients reported a peak incidence among the 40–49 age group [7]. The slight male predominance in the study (56%) is observed with Asian studies but some western studies shows female preponderance [1]. Fourteen percent of our study population had Diabetes mellitus and another 12% of the study population had Hypertension. In a study done by Riga et al. the author observed that microangiopathy in diabetes predisposes the facial nerve to ischemic injury, thereby leading to more severe presentations [9].

Bell's palsy constituted to be the predominant etiology in our study (62%); however, there was a relatively high proportion of secondary facial nerve palsy (38%) observed in comparison to the population-based studies. Also, among other secondary causes, the temporal bone trauma accounted for about 18% of study population. This increased incidence in our study can be attributed to the tertiary level care status of our institution, and also that our Institute caters to a large proportion of road traffic accidents and also complex trauma cases. Our study findings also reinforces the importance of performing a high-resolution computed tomography (HRCT) of the temporal bone to all patients with a history of head injury, even when the trauma appears trivial, as identification of a fracture line that directly influences management decisions such as conservative therapy versus surgical decompression [3]. Additionally, squamous chronic otitis media (COM) accounted for 10% of study participants which emphasizes that facial nerve palsy can be an initial manifestation of an unsafe ear mainly due to

cholesteatoma, necessitating an early surgical intervention in the form of mastoidectomy.

Otalgia, a common presenting symptom, was observed in 46% of participants, and was observed in both the conditions - Bell's palsy and secondary facial palsy. In Bell's palsy, otalgia is considered to result from the involvement of sensory fibres in the facial nerve [8]. However, persistent intolerable ear ache should be subjected for prompt evaluation to rule out other serious etiologies such as Herpes Zoster Oticus and malignant otitis externa, which was observed among few cases in our study [10]. Audiological evaluation among the study participants played a crucial diagnostic role, with 36% of the participants demonstrating hearing loss on pure tone audiometry (Table 5). Our observation highlights the fact that facial nerve palsy is often not truly an "isolated," entity and subclinical cochlear involvement may also be present even when the patients do not subjectively perceive the hearing impairment.

The therapeutic approach towards Bell's palsy has evolved over time, with systemic corticosteroid therapy forming the cornerstone of management. In our present study, two thirds of the participants with Bell's palsy achieved House–Brackmann Grade I or II recovery within 12 weeks of initiation of treatment. This aligns with evidence from a Cochrane review, which indicated that corticosteroid therapy significantly reduces the risk of incomplete recovery [4]. Antiviral therapy was administered to 60% of participants, particularly to those who presented with severe facial nerve dysfunction (House–Brackmann Grades IV–V) or a suspected viral prodrome. Although the additional benefit of antivirals when combined with

steroid therapy remains controversial, the current guidelines recommend their use in severe conditions to minimize the risk of synkinesis and extensive nerve degeneration [5,11].

Limitations

Relatively small sample of fifty patients limits the generalisability. Furthermore, the follow-up duration of 12-weeks was inadequate to understand the process of early recovery, to understand the late neurological involvement, the follow period could be continued till one year in future studies [12].

Conclusion

This study confirms that while Bell's palsy is the most common cause of LMN facial palsy, secondary causes—particularly temporal bone trauma and chronic otitis media—constitute a significant proportion of cases in a tertiary care setting. A systematic clinical approach involving otoscopy, audiometry, and selective imaging is indispensable. Standardized treatment with corticosteroids yields favorable functional recovery in the majority of Bell's palsy patients, with nearly 70% achieving near-normal facial function by 12 weeks.

Statements and Declarations

Conflicts of interest

The authors declare that they do not have conflict of interest.

Funding

No funding was received for conducting this study.

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