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(CASE REPORT)

A Silent Smile Restored: Journey Through the Faltering to Graceful Face: A Case Report

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Abstract

This case report details the presentation, diagnostic workup, and management of 57-year-old paramilitary personnel who developed acute onset, left-sided lower motor neuron facial paralysis, diagnosed as Bell's palsy. The patient presented with sudden facial weakness, inability to close the left eye, deviation of mouth, and loss of taste, following a prodromal episode of sore throat and dry cough. Neurological examination confirmed idiopathic lower motor neuron facial nerve palsy, with no abnormalities detected on non-contrast CT head or relevant serologies. A comprehensive treatment approach was adopted incorporating acyclovir, prednisolone with tapering, vitamin B complex, intensive eye care, and physical therapy exercises. Notably, the patient exhibited prompt clinical improvement within 10 days, regaining facial strength, eye closure ability, and taste sensation. The discussion highlights prevailing theories in Bell's palsy pathogenesis, particularly viral reactivation and nerve compression, and supports combination corticosteroid and antiviral therapy for severe cases. The importance of vigilant eye protection and rehabilitative therapy is emphasized to minimize corneal complications and synkinesis. Psychosocial impact is also acknowledged, advocating holistic, multidisciplinary follow-up to support patient recovery and emotional health. This report underscores that early diagnosis, integrated medical management, and tailored rehabilitative strategies are critical for optimal outcomes in Bell's palsy, while ongoing psychosocial support addresses concerns over facial changes and functional impairments during recovery.

Keywords: Bell's palsy; Facial nerve paralysis; Lower motor neuron; Idiopathic; Acyclovir; Physical therapy

1. Introduction

Bell's palsy is an acute peripheral facial neuropathy and represents the most frequent cause of lower motor neuron facial paralysis as well as the most common cranial mononeuropathy. It typically presents with sudden-onset, unilateral, lower motor neuron-type facial weakness, often accompanied by postauricular pain, altered taste sensation, subjective facial sensory changes, and hyperacusis. Although the disorder has been extensively investigated, its precise pathogenesis remains uncertain. Viral infection (particularly herpes simplex virus type 1), nerve compression, and autoimmune mechanisms have all been proposed, but the relative contribution and sequence of these factors are not yet clearly established [1].

It is a condition marked by sudden paralysis of facial muscles supplied by the facial nerve, most often leading to temporary weakness on one side of the face. Although the typical presentation involves unilateral weakness, rare cases of bilateral involvement have been documented. [2].

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This condition accounts for approximately 60-75% of all cases of acute facial paralysis, with an annual incidence of 15-30 cases per 100,000 population [3].

2. Case report

2.1. Presentation

A 57-year-old male, paramilitary personnel, posted at high altitude area became symptomatic with water dripping out from left angle of the mouth while trying to drink water. He developed acute onset inability to close his left eye completely. He rushed to the mirror where he noticed that his mouth seemed to be deviated to the right. Symptoms were acute in onset, with onset to peak symptoms taking around 2 hours to develop. He immediately sought medical attention and rushed to our hospital. Patient had an episode of sore throat with sneezing and dry cough around 01-week prior which had responded to cough suppressant, antihistaminics and other supportive measures including gargling thrice a day with chlorhexidine mouth wash gargle. No rash anywhere in the body. No weakness of any other part of the body.

2.2. On Examination

General examination: Built – Normal. BP – 132/86mmHg Right arm, supine posture with no significant variation in BP measured in both upper limbs. Pulse – 88/min, regular, normal volume, no radio radial delay/ no radiofemoral delay, with all peripheral pulses palpable. Respiratory rate 20/min, regular. Not using accessory muscles of respiration. JVP not raised. Afebrile. No cervical lymphadenopathy.

Systemic Examination: Neurological Examination – Higher Mental Function intact. Speech slurred. Cranial Nerve Examination – Absence of wrinkling of forehead on the left, inability to close left eye completely, inability to make facial expressions on the left, deviation of angle of mouth to the right, inability to hold onto air inside left side of mouth, decreased sensation to taste over anterior two thirds of the tongue – Signifying a lower motor neuron type of palsy of facial nerve on the left (Figure 1)

Figure 1 At Presentation: From left to right: Inability to close left eye completely; Absence of forehead wrinkling on the left side; Deviation of angle of mouth to the right with loss of nasolabial fold on the left

2.2.1. Rest of cranial nerve examination – WNL.

Examination of external auditory canal – Otoscopic examination showed normal tympanic membranes bilaterally with no evidence of vesicles or other pathology in the external auditory canal.

Power in all 4 limbs – Grade 5. Deep Tendon Jerks – B/L Ankle – 1+; B/L Knees 2+; B/L Biceps 2+; B/L Triceps 2+; B/L Supinator 2+. Superficial reflexes normal, B/L Plantar – Flexor. No sensory deficits. No cerebellar signs elicited. Gait Normal. Skull and spine examination normal.

2.2.2. Other system examination – WNL.

Four tier diagnosis after clinical examination:

- Functional – Acute onset flaccid weakness of muscles of facial expression in the left alongwith impaired taste sensation over anterior two thirds of tongue.
- Physiological - Lower motor neuron paralysis of the facial nerve (seventh cranial nerve)
- Anatomical - Intratemporal segment of the left facial nerve likely labyrinthine segment inside the temporal bone (distal to the facial nucleus, before branching of extracranial branches).
- Etiological – To be determined (? Vascular)

2.2.3. Investigations

- NCCT Head – No abnormality detected (Figure 2)
- Blood count including Hb, Total Leucocyte Count and Platelet Count – Within Normal Limit
- Liver Function Test and Renal Function Test – Normal
- Erythrocyte Sedimentation Rate, C Reactive Protein, Procalcitonin - Normal
- HSV serology was negative. Lyme disease serology was negative.

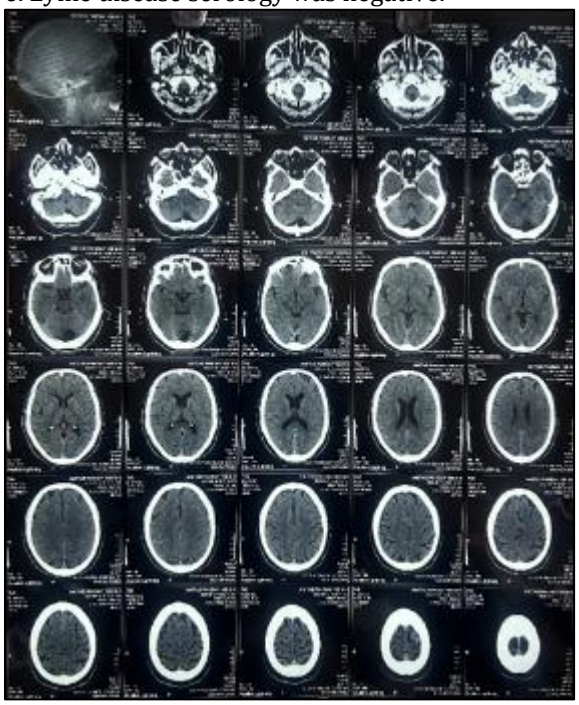

Figure 2 Non-Contrast Computed Tomography of Head – No abnormality detected

Final Diagnosis: Idiopathic left sided Lower motor neuron palsy of Facial Nerve – Bell's Palsy

Treatment: Based on the clinical presentation and diagnostic findings, we initiated a comprehensive treatment plan. The patient received acyclovir 400 mg five times daily for 5 days, along with prednisolone 60 mg daily for 5 days with gradual tapering planned over the subsequent 21 days. Vitamin B complex supplementation was also provided. Eye care included regular application of artificial tears during the day and lubricating ointment at night, with eye patch protection during sleep. Physical therapy was initiated, incorporating facial exercises and massage.

Follow up: Patient responded promptly to treatment, with good response noted within 1st week of treatment. At the end of 10 days, patient was able to close his left eye, make better facial expressions, with return of taste in the tongue. Forehead wrinkles also began to appear on the left side (Figure 3)

Figure 3 10 days into treatment: From left to right: Ability to close left eye completely; Appearance of forehead wrinkling on the left side; Reappearance of nasolabial fold on the left

3. Discussion

This case highlights several key principles in the clinical management of Bell's palsy. Although the precise underlying mechanism remains uncertain, strong evidence from molecular and clinical research supports the hypothesis that reactivation of latent herpes simplex virus (HSV) leads to nerve inflammation and swelling. The facial nerve's passage through the extremely narrow fallopian canal makes it especially prone to compression from inflammatory edema, which accounts for the rapid onset of facial weakness typically seen in Bell's palsy. [1].

The choice to initiate combination therapy with corticosteroids and antiviral agents was guided by current clinical recommendations. Recent meta-analyses have endorsed this treatment strategy, especially for patients presenting with severe facial paralysis or confirmed viral etiology [4]. Corticosteroids like prednisolone help reduce inflammation and swelling within the confined fallopian canal, while antivirals such as acyclovir target the underlying viral cause. Gradual tapering of steroid therapy is employed to minimize rebound inflammation and support the natural healing process.

In managing this patient, comprehensive eye care was prioritized to prevent corneal complications. The inability to fully close the eye (lagophthalmos) significantly increases the risk of exposure keratitis, making regular lubrication and nighttime eye protection essential. Effective management of this aspect frequently involves close collaboration with ophthalmologists to monitor the eye's condition and modify treatment as necessary [5].

Physical therapy's role in recovery from Bell's palsy is still being actively studied. Some research indicates modest improvements from facial exercises and electrical stimulation, while others stress the importance of carefully controlled facial movements to avoid synkinesis. The favorable outcome seen in this patient supports incorporating physical therapy into the overall treatment plan, although the best timing and exercise intensity for maximal benefit are yet to be fully determined [6].

The psychosocial impact of facial paralysis warrants particular attention in the treatment plan. Facial paralysis can profoundly affect social interactions, occupational functioning, and emotional health. Regular follow-up appointments are essential not only to assess physical recovery but also to address psychological issues and provide reassurance. This supportive care is crucial, as concerns about permanent facial changes can significantly influence a patient's quality of life during recovery [7].

Abbreviations

- BP: Blood Pressure
- BL: Bilateral
- CD: Cochrane Database
- CT: Computed Tomography

- Hb: Hemoglobin
- HSV: Herpes Simplex Virus
- JVP: Jugular Venous Pressure
- NCCT: Non-Contrast Computed Tomography
- PubMed: Public/Publisher Medical Database
- WNL: Within Normal Limits

4. Conclusion

This case of acute idiopathic left-sided Bell's palsy in middle-aged paramilitary personnel highlights the typical clinical presentation, diagnostic evaluation, and comprehensive management approach. Despite uncertainties surrounding the exact cause, the presumptive viral reactivation leading to facial nerve inflammation and compression remains the most widely accepted mechanism. Early initiation of combined corticosteroid and antiviral therapy, alongside attentive eye care and rehabilitative physical therapy, contributed to the patient's positive recovery. Additionally, addressing psychosocial impacts through regular follow-up was integral to holistic care. This case underscores the importance of timely diagnosis and a multidisciplinary treatment strategy to optimize outcomes in Bell's palsy patients.

Compliance with ethical standards

*Disclosures of **Conflicts of interest**:*

No conflicts of Interest.

In accordance with the ICMJE uniform disclosure requirements, the authors declare the following:

Payment/services

No financial support was received from any organization for the submitted work.

Financial relationships

The authors report no financial relationships, either current or within the past three years, with organizations that could have an interest in this work.

Other relationships:

The authors also declare no other relationships or activities that could be perceived as influencing the submitted work.

Statement of Ethical Approval

Ethical approval was not applicable for this case report as it does not constitute a research study. Patient informed consent for publication of clinical details and images was obtained.

Statement of informed consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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