

Case Report:

Bells palsy : a diagnosis of exclusion a case report

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ABSTRACT

Bell's palsy is a neuropathy of the peripheral seventh cranial nerve, usually resulting from traumatic, compressive, infective, inflammatory or metabolic abnormalities. The onset of Bell's palsy may be delayed or it may be sudden onset which often results in panic state. For speedy recovery, correct diagnosis and early treatment are crucial. We report a case of 22-year-old female patient who came to department of oral medicine and radiology with left hemifacial palsy since 7 days. On clinical examination, there were characteristic features indicating facial palsy like lack of movement of the left forehead and eyebrows, inability to close the left eye completely and deviated smile line. Patient was given steroid along with vitamin B which showed improvement in the symptoms.

Keywords: Bells palsy, hemifacial palsy, neuropathy

INTRODUCTION

Bell's palsy, is a termed coined by Sir Charles Bell in 1821, also known as idiopathic facial nerve paralysis⁽¹⁾, is an acute peripheral facial nerve palsy (7th cranial nerve) usually of unknown cause and is typically unilateral, affecting both the sides equally and can be complete or partial.⁽²⁾ According to one study, 68% of acute peripheral facial palsy were classified as Bell's palsy.⁽³⁾ Incidence of Bell's palsy ranges between 11 and 40 cases per 100,000 per year, with 1 in 60 being affected in their lifetime. Bell's palsy affects people of all ages, but most commonly seen in age 15 to 45 years old with slight female prevalence.^(4,5) Conventional studies have mentioned the incidence of Bell's palsy is higher in diabetic and pregnant patients; however, some recent studies challenge this.^(6,7)

Bell's palsy is a clinical diagnosis of exclusion. Although it is considered to be idiopathic in origin, Herpes simplex virus (HSV) is commonly implicated in causing bell's palsy by causing acute inflammation and edema of the facial nerve, thereby entrapment of the nerve in the bony canal (especially in the labyrinthine segment) leading to compression and ischemia. This leads to degeneration of the facial nerve or neuropraxia.⁽⁸⁾ Other infectious causes of Bell's palsy include, other than Lyme disease, herpes zoster, rubella, cytomegalovirus, Epstein-Barr virus, influenza B, adenovirus, mumps, and coxsackievirus.⁽⁹⁾ Other proposed etiologies include physiologic compression of the nerve due to arteriospasm, venous congestion or ischemia, and narrowing of the bony canal.

Facial nerve paralysis is a debilitating condition. Its onset is sudden, with facial muscle weakness progressing over hours to days. Patients with facial nerve disorders are emotionally and psychologically disturbed due to facial disfigurement and the subsequent physical limitations and difficulties associated with speaking, drinking, eating, and facial expression secondary to the disorder. These have a high impact on their social life. Facial nerve paralysis can be unilateral or bilateral depending on the level of neuron affected the clinical picture is accordingly.

The clinical signs of Bell's palsy include widening of the palpebral fissure, flattening of the nasolabial fold, weakened muscle tone of the side involved and drooping of one corner of the mouth when smiling. These signs occur on the same side of the face as the lesion.

CASE REPORT

A 22 year old female patient to the department of oral medicine and radiology with chief complains of asymmetry of face & increased watering from left eye since 7 days. Patient complained of severe sudden, continuous, lancinating pain localized in left ear 15 days back which lasted for 10 days not associated with any other symptoms. She consulted ENT surgeon for same and was prescribed antibiotics & analgesics and was referred to dental department to rule out any dental cause for the same. Patient gave the history of chicken pox infection 4 years back. There was no weakness or any numbness in other part of the body. No familial history of such kind with first of this kind of episode. No history of eruptions, trauma or any surgical procedure associated with this condition. Patient complained of sudden onset of facial asymmetry while smiling, deviated smile line towards right side, inability to spit due to incomplete lip seal, increased watering from left eye, inability to completely close left eye, taste not altered. On extra oral examination there was facial asymmetry with deviated smile line (Fig 1), reduced blinking of left eye, forceful opening of left eye indicating weakness of left eye (Fig 2) decreased wrinkling of forehead in relation to left side (Fig 3), able to completely close both eye deliberately (Fig 4), unable to hold left eye against force (Fig 5) inability to spit and whistle properly due to incomplete lip seal, on blowing deviation of face indicating facial asymmetry, slightly obliterated left nasolabial fold (Fig 6,7), control over upper and lower lip movement, slightly deviated tongue protrusion, (Fig 8) no deviation & tenderness in TMJ region. Intra oral examination was no contributory. Based on history and characteristic clinical features diagnosis of Bells palsy left side was given. Patient was referred to neurologist for treatment of same. She was advised Tab predmet (prednisolone) 8mg TDS 4 days and 4 mg TDS 6 days, Tab felicita (Methylcobamine) OD, Tab Zymoflame D (trypsin) OD, Tab Neksium (proton pump inhibitor) OD for 10 days. Along with physiotherapy to stimulate facial muscles.



Fig 1 Deviated smile line



Fig 2 Weakness in left eye



Fig 3 Decreased wrinkles on left side of
Forehead



Fig 4 Deliberately closed both eyes



Fig 5 Weakness of left eye



Fig 6 Right nasolabial fold



Fig 7 Obliterated Left nasolabial fold

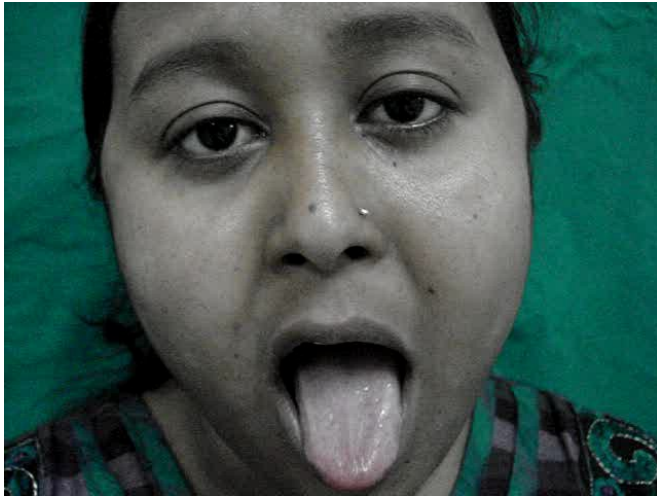


Fig 8 Slight deviation of tongue

DISCUSSION

The term Bell's palsy is used to describe an acute-onset, idiopathic facial paralysis resulting from a dysfunction anywhere along the peripheral part of the facial nerve from the level of the Pons distally. (Martha Ann Keels et al., 1987) Bell's palsy is a peripheral facial nerve palsy involving the lower motor neurons. A lower motor neuron lesion causes weakness of all the muscles of facial expression. The angle of the mouth falls, weakness of frontalis occurs, and eye closure is weak. As was evident in the present case. With an upper motor neuron lesion, the frontalis is spared, normal furrowing of the brow is preserved, and eye closure and blinking are not affected.⁽⁵⁾

The examination is notable for unilateral weakness of the face involving the forehead. There markable reduction in facial creases and obliteration of nasolabial , the forehead unfurrows, and drooping of the corner of the mouth, unable to purse the lips, the eyelids will not close and lower lid sags. On attempted closure of the eyelid, the eye rolls upward (Bell's phenomenon). Tear production decreases; however, the eye may appear to tear excessively because of loss of lid control allowing tears to spill freely from the eye. There may be a positive Hitzelberger sign, a decreased sensation along the external acoustic meatus. Other cranial nerves are normal.⁽¹⁰⁾ 70% of untreated cases shows complete recovery to normal facial function. While 13% cases shows mild degree and 16 % shows major degree of permanently impaired facial function.⁽¹¹⁾ Onset of clinical recovery is nearly always demonstrated within 4 to 6 months of symptom onset, if the hemi-facial tone or movement do not resolve by this time then alternative diagnosis should be considered.

Dental causes have also been reported with facial paralysis such as maxillo-facial surgical procedures (both intra- and extra-oral) which include administration of local anesthesia, tooth extraction, osteotomies, preprosthetic procedures, excision of tumors or cysts, surgery of temporomandibular joint, surgical treatment of facial fractures and cleft lip/palate and orofacial granulomatosis infections .^(12,13,14)Others are Temporal bone fractures and fracture of the mandibular condylar neck.

The main goal of treatment is to improve the function of the facial nerve and reduce neuronal damage. In most of the cases, no treatment is required as it can spontaneously recover by itself . Hence treatment is considered to be controversial . Bell's palsy is normally treated using corticosteroids (prednisolone 1mg/kg) with or without antiviral agent (Acyclovir) and supplemented with vitamin B.⁽¹⁵⁾ Considerable controversy remains over the use of steroids for Bell's palsy in adults. Although many studies states the usefulness of corticosteroids in the

treatment of Bell's palsy. Treatment with corticosteroids should begin within 5 days (earlier if possible) after the onset of palsy and should only be used in the first 7 days. As the etiology is often viral so use of antivirals seems logical in Bell's palsy. The American Academy of Neurology considers acyclovir safe and possibly effective in improving functional outcomes.⁽¹⁶⁾ However, a recent study demonstrated no evidence of a benefit of acyclovir given alone or an additional benefit of acyclovir in combination with prednisolone.⁽¹⁷⁾ Other proposed treatments modalities with little evidence are Methylcobalamin, hyperbaric oxygen, facial retraining, botulinum toxin, transcutaneous electrical stimulation, and acupuncture.

In the present case steroid along with methylcobalamin was given .

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