Lagophthalmos, With Exposure Keratopathy – A Case Report

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Abstract:

Facial nerve paralysis (FNP) is the most common cranial nerve disorders and it results in a characteristic facial distortion that is determined in part by the nerves branches involved. We report a case of 28-year-old female patient who came to ophthalmology department with left hemifacial palsy since 21 days. On clinical examination, there was lack of movement of the left forehead and eyebrows, inability to close the left eye completely. After series of investigations, no definitive etiology could be traced out, hence considered as unilateral bell's palsy of the left side. Patient has been taking vitamin B ,steroid and antiviral , physiotheraphy for three weeks and reported with an improvement of symptoms, hence no other interventions were made to treat this condition. In this article, we discuss the differential diagnosis of facial nerve paralysis, etiology, clinical features and treatment modalities for bell's palsy.

Keywords: *Facial palsy, Bell's Palsy, Facial nerve, Hemifacial paralysis, Unilateral facial paralysis.*

Int. Med. Col. J. 2017; 7(1): 20-24

Introduction:

Facial nerve paralysis is classified as central type or peripheral type, depending on the level of nerve injury. Central type results in paralysis of the lower part of the facial muscles on the opposite side of the lesion. The upper facial muscles are spared due to bilateral cortical connections. The peripheral type (lower motor neuron) lesion produces total facial paralysis on the same side of the lesion. Peripheral lesion produces a more severe type of facial paralysis compared to the central lesion, but central lesion's origin may represent a serious problem in the brain. Facial nerve paralysis can be unilateral or bilateral. The most common cause of facial nerve paralysis in children is Bell's palsy.[1] Bell's palsy is the accepted term to describe unilateral, peripheral facial nerve paralysis which is idiopathic with acute onset.[2]

Bell's palsy, a termed coined by Sir Charles Bell in 1821, also known as idiopathic facial nerve paralysis,[3] 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.[3] Bell's palsy affects people of all ages, but most commonly individuals 15 to 45 years old. It's onset is sudden, with facial muscle weakness progressing over hours to days.

Bell's palsy is diagnosed only by exclusion of all other possible causes. Although etiology is unidentified, 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) which leads to compression and ischemia. This leads to neuropraxia or degeneration of the facial nerve.[4]

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Received: 20 May 2018 **Accepted:** 01 July 2018

Here, we report a case of unilateral facial nerve palsy where after extensive investigations we could not get a definite cause and treated as Bell's palsy.

Review of Facial Nerve Anatomy

The facial (7th cranial) nerve has parasympathetic, motor, and sensory nuclei in the pons, a part of the brain stem located in the posterior cranial fossa. The fibers from the motor nucleus pass below the floor of the 4th ventricle and continue around the nucleus of the abducens (6th cranial) nerve to combine with parasympathetic and sensory fibers to form a common nerve trunk. The nerve trunk exits the brain and continues its lateral course through the facial canal. Where the canal makes an acute bend toward the middle ear cavity, the geniculate ganglion is located. At the level of the geniculate ganglion, the greater superficial petrosal nerve arises and supplies parasympathetic fibers to the lacrimal gland and mucous membrane of the nose and mouth (Fig.-1).[5]

Distal to the geniculate ganglion, the facial nerve gives rise to 2 branches. The first contains the motor nerve to the stapedius muscle, which serves to dampen the oscillations of the ear ossicles. The second is the chorda tympani nerve, which contains sensory fibers for taste from the anterior 2/3rd of the tongue. The chorda tympani nerve also joins with the lingual nerve and provides parasympathetic innervation to the submandibular and sublingual glands. The facial nerve exits the temporal bone through the stylomastoid foramen and subdivides into its terminal branches, which supply the motor innervation to the muscles of facial expression.

Central Versus Peripheral Lesions of the Facial Nerve

The facial nerve primarily innervates the muscles of facial expression. There are 2 types of paralysis affecting the motor function of the facial nerve and they may be classified as a central type or a peripheral type. The central type involves the corticobulbar fibers which convey impulses from the cerebral cortex to the cells of the motor nucleus of the facial nerve. A central lesion interrupting the corticobulbar pathways results in paralysis only of the lower facial muscles on the opposite side of the lesion. This is explained by the fact that the

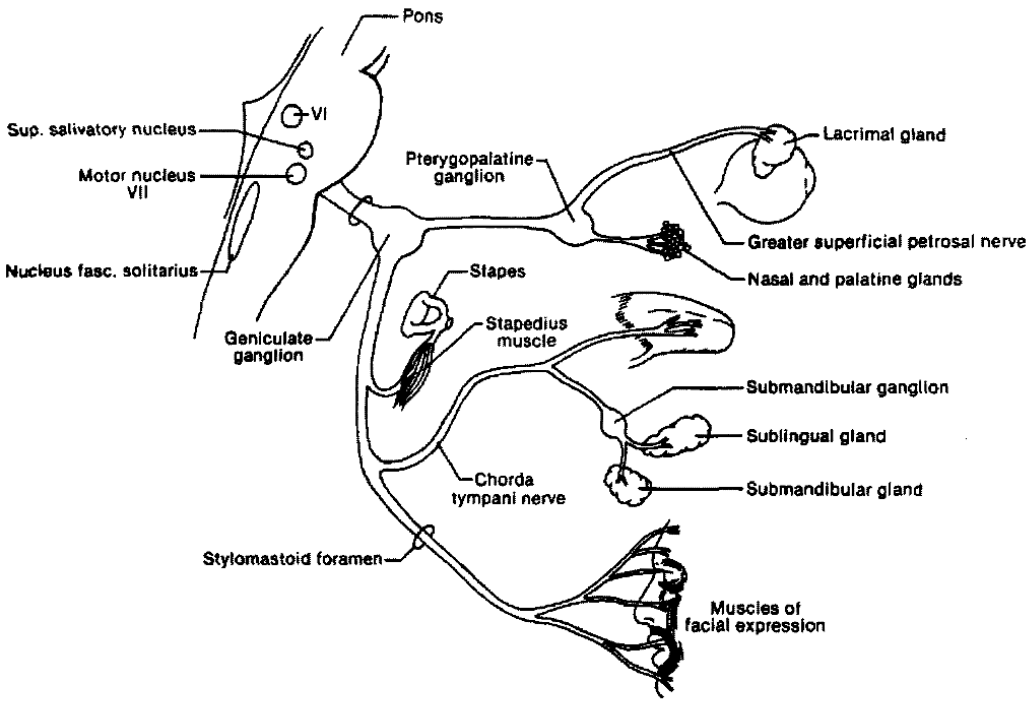


Fig.-1: *Anatomy of the facial nerve (Olsen 1984; Alford et al. 1973; Carpenter 1978; Montgomery 1981).*

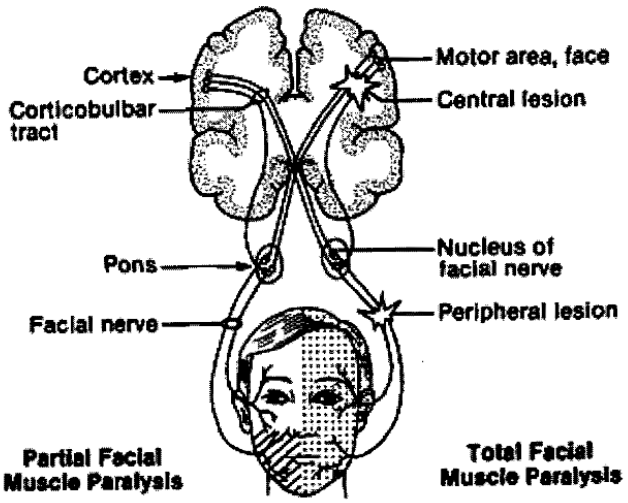


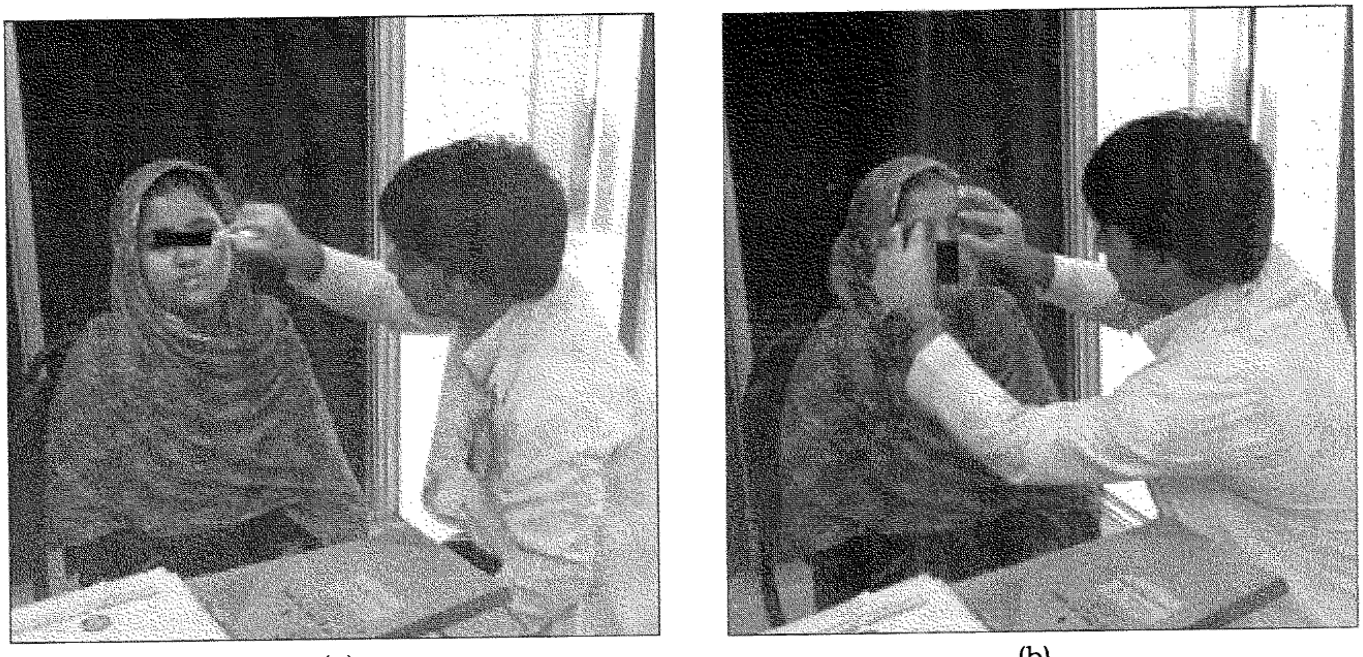
Fig.-2: *Muscle paralysis difference between a central lesion and a peripheral lesion of the facial nerve (Carpenter 1978; Montgomery 1981).*

corticobulbar fibers to the forehead and the upper half of the face are distributed bilaterally; however, the fibers to the lower half of the face are predominantly crossed. The peripheral type lesion of the facial nerve occurs at the level of the pons or anywhere along the distal course of the nerve. This lesion produces total facial paralysis on the same side as the lesion. A central lesion produces a less severe type of facial paralysis compared to the peripheral lesion, but its origin may represent a serious problem in the brain. A simple neurological test to differentiate a central from a peripheral lesion in a patient with facial nerve paralysis is to ask the patient to wrinkle the forehead; if the patient can wrinkle the entire forehead, the lesion is centrally located. If the patient can wrinkle only half the forehead, the lesion is peripherally located.

Case Report:

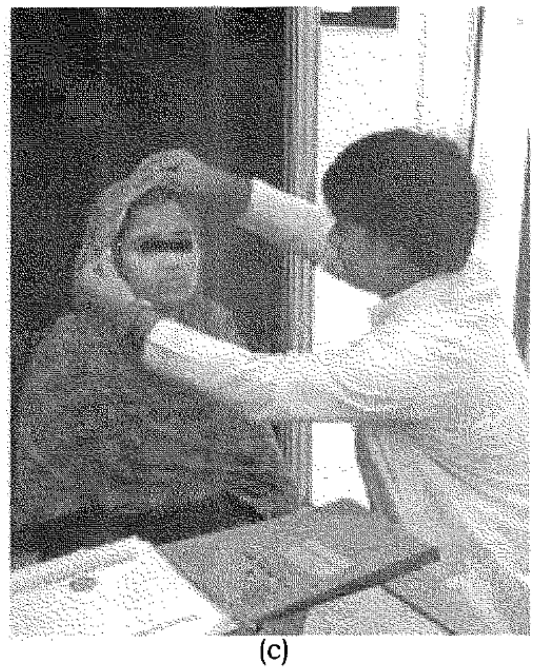
A 28-year-old female reported to International Medical College and Hospital with the complaint of deviation of mouth (right side) for 21 days, history of earache(left) for 7 days, dribbling of water from angle of mouth (left side) for 21 days and redness of left eye for 18 days. Patient's appearance was abnormal and on observation the patient had facial asymmetry. History revealed that duration of palsy was over 21 days. It was of a sudden onset. She had no history of fever, trauma, and prolonged exposure to cold wind. Patient had no history of vesicles in the ears and mouth or oro-facial oedema. Patient was not a known diabetic or hypertensive and there was no other significant medical history and did not have any other systemic illnesses. No history of tobacco, alcohol use or any illicit substances.

She was moderately built, well oriented, and on clinical examination, there was obvious disfiguring difference between two sides, failure

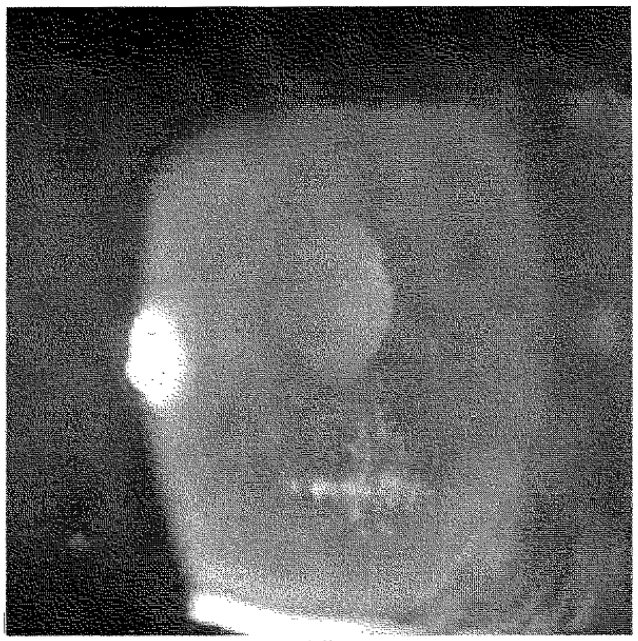


(a) (b)

Fig.-3: *(a) Examining corneal reflex, (b) Examination of orbicularis occuli*



(c)



(d)

Fig.-3: *(c) Performing Hearing test (for hyperacusis), (d) Exposure keratopathy (Fluorescein stain positive)*

of wrinkling of forehead on left side, left eye cannot be closed (on attempting to close, eyeball rolled upward and outward- Bell phenomenon), weakness of left side of face on puffing the cheek, failure to whistle and smile, while showing the teeth lips are drawn to right side, corneal reflex delay on left eye, hyperacusis on left ear, touch (fine and crude) sensation diminished on left side, nasolabial fold absent on left side, taste (salt and sugar) sensation altered on left side, no vesicles (rash) on ear, conjunctiva congested.

Laboratory investigations, CBC, imaging (chest x-ray), SGPT, RBS were done and were within normal limits. Full blood count showed erythrocyte sedimentation rate 56 mm in 1st hour (normal: male: 0-10, female:0-20 mm). X-ray PNS showed frontal sinusitis. On examination other cranial nerves V, VI, IX and X were intact. Patient was diagnosed as having Bell's palsy (unilateral facial nerve paralysis) of the left side (lower motor neuron lesion). Her vision was 6/6 in OD and 6/12 in OS. On slit lamp examination. conjunctiva congested, fluorescein stain positive at lower part of cornea. She was treated with oral prednisolone, anti viral acyclovir and vitamin B complex. She was also treated with artificial tears, antibiotic drop and ointment. She was advised for patching of left eye during sleep. She was advised for physiotherapy also. And on follow up after 3 weeks her condition was improved.

Discussion:

There are many theories about the cause of Bell's palsy but the etiology is unknown[6]. The most popular hypothesis is that it is caused by a virus similar to Herpes simplex or zoster[2]. Other proposed etiologies include physiologic compression of the nerve due to arteriospasm, venous congestion or ischemia, and narrowing of the bony canal and autoimmune disorders[7]. Several case reports support a familial tendency suggesting the inheritance of an aberrant facial canal. Bell's palsy is diagnosed by careful case history, clinical signs and symptoms and evaluation to exclude other possible causes of facial paralysis. The history should include time sequence of onset, prior history of facial paralysis, recent viral or upper respiratory tract infection, recent camping or hiking, ontological symptoms, change in taste, facial numbness, vesicles, or recent immunization. A conclusion of Bell's palsy is arrived usually as a diagnosis of exclusion[8]. The clinical examination should include an accurate testing of the cranial nerves.

Conclusion:

Bell's palsy or idiopathic facial paralysis is the most common cause of unilateral facial paralysis. Since Bell's palsy is a facial paralysis of unknown origin, it is essential to rule out other causes of facial paralysis before making the definitive diagnosis, which implies the intervention. Bell's palsy has been termed as a diagnosis of exclusion. A detailed history, thorough clinical examination, appropriate laboratory investigations and imaging modalities should be carried out in patients with facial palsy to correctly identify its cause which will help in further planning and executing the correct treatment for most promising results. Adequate investigations should be conducted to rule out infections, neoplasm, metabolic and toxic reasons. Misdiagnosis or under-diagnosis would result in worsening of the patient's condition.

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