

Facial Palsy – 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 54-year-old female patient who came to department of oral and Maxillofacial Surgery with right hemifacial palsy since 7 years. On clinical examination, there was lack of movement of the right forehead and eyebrows, involuntary blinking of the right eye, inability to close the right eye completely and hyperkinesia of the right cheek. After series of investigations, no definitive etiology could be traced out, hence considered as unilateral bell's palsy of the right side. Patient has been taking vitamin B complex once daily for the past one year 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

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 is a debilitating condition. Patients with facial nerve disorders are often devastated due to the emotional and psychological impact of facial disfigurement and the subsequent physical limitations and difficulties associated with speaking, drinking, eating, and facial expression secondary to the disorder. Socialization and community participation is extraordinarily limited and difficult for many of these patients. Facial nerve paralysis can be unilateral or bilateral.

Thousands of people each year develop facial paralysis, a relatively common disorder with many different origins (infectious, traumatic, neoplastic, inflammatory, metabolic or idiopathic). Bell's palsy, a term 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]. It is the most common cause of unilateral facial paralysis, accounting for approximately 70% of these cases [3]. Incidence of unilateral facial paralysis is 1,000 cases per 5 million population per year. Bell's palsy is also responsible for approximately 20 per cent of cases of bilateral simultaneous facial nerve palsy [4]. Bell's palsy affects people of all ages, but most commonly individuals 15 to 45 years old. Its 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 [5]. 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.

CASE REPORT

A 54-year-old female reported to Saveetha Dental College, Chennai with a complaint of missing teeth and desired replacement of her missing teeth. Patient's appearance was abnormal and on observation the patient had facial asymmetry, involuntary continuous blinking of the right eye and twitching of the right cheek. Patient was provisionally diagnosed as having right hemifacial palsy and was referred to the Department of Oral and Maxillofacial Surgery for the management.

History revealed that duration of palsy was over 7 years. It was of a sudden onset. She had no history of fever, trauma, and extraction of third molar, or 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 had visited many doctors for the problem but was not cured. One physician prescribed vitamin B complex capsules to the patient, which she has been taking once daily for the past 1 year.

Right side Bell's palsy with mask-like facial appearance**Fig 1. At rest - right side Bell's palsy****Fig 2. Smiling-deviation of corner of mouth****Fig 3. Unable to wrinkle forehead on right side****Fig 4. Able to close eyes voluntarily with effort**

She was moderately built, well oriented, and on clinical examination, there was obvious disfiguring difference between two sides [Figure 1], lack of movement of the right forehead and eyebrows [Figure 3], involuntary blinking (synkinesis) of the right eye and hyperkinesia of the right cheek. At rest normal symmetry and tone of facial muscles were present. Patient was able to voluntarily close eyes completely with effort [Figure 4] and also had reduced vision in her right eye since the onset of the palsy. Reduced movement on forehead was seen on motion. The tip of the nose is drawn to the unaffected side. On smiling, there was decreased activity of the muscles on the right side compared to the left [Figure 2] and there was deviation of angle of the mouth. She had no change in taste sensation or paraesthesia, no xerostomia, no dry eyes or tearing on salivation (crocodile tears), no excessive facial sweating or sweating on salivation. No associated pain over ears and face. Patient had normal hearing and her speech was normal.

Lab investigations, imaging (chest x-ray) were done and were within normal limits. Magnetic resonance imaging (MRI) and contrast enhanced Computed Tomography (CT) of the brain showed normal study. 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 right side (lower motor neuron lesion) with the House-Brackmann facial nerve grading as Grade III - moderate dysfunction.

No treatment was given for the patient as she was already under vitamin B complex therapy which had improved her condition over the past one year. Also, due to the chronic nature of the condition, patient had no queries about the mild facial disfigurement and had adapted well to the situation at hand. Unilateral facial weakness was not bothering the patient and it did not interfere with her day-to-day routine life activities.

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 [7]. 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 [8]. Several case reports support a familial tendency suggesting the inheritance of an aberrant facial canal [6].

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 [9]. The clinical examination should

include an accurate testing of the cranial nerves. Laboratory investigations carried out are complete blood count, a Syphilis and HIV test, fasting glucose, erythrocyte sedimentation rate, Lyme titer, antinuclear antibody level measurement, as well as a lumbar puncture for Cerebrospinal fluid (CSF) cell count and examination after a cerebral CT, IgG, IgM antibody tests, Acetylcholine-receptor antibody test [10]. Imaging modalities like computed tomography, magnetic resonance imaging are done to detect any intracranial lesion. Electromyography has a high predictive value for negative outcome after acute facial palsy and seems to be more sensible to detect signs of defective healing rather than clinical evaluation of facial function.

In a bell's palsy patient the following signs occur on the same side of the face as the lesion:

Rapid onset of unilateral facial weakness, with mask-like appearance. Pain and numbness or stiffness on the affected side of the face, especially in the temple, mastoid area, and along the angle of the mandible without actual sensory loss [11]. Facial appearance becomes asymmetric, patients are unable to wrinkle half of their forehead, corner of the mouth drops while smiling, saliva dribbles down the angle of the mouth, unable to purse the lips, unable to close one eye completely, or to wink, widening of the palpebral fissure is seen with bell's phenomenon. Bell's phenomenon is a classic condition wherein the eye cannot close without a simultaneous movement of the eyeball upward and outward. There may be dry mouth due to decreased salivary secretion, loss of taste sensation over anterior tongue, inability to blow air, inability to clench teeth or grin, speech slurred, obliteration of nasolabial fold and hyperacusis (noise intolerance) [11].

House-Brackmann facial nerve grading system is used for assessing the severity of facial paralysis [12].

Differential diagnosis of Facial nerve palsy:

Idiopathic conditions (Bell's palsy), Iatrogenic problems (parotidectomy, Temporomandibular joint surgery), strokes, acoustic nerve tumors, brain tumours, Parotid neoplasms, cholesteatoma, schwannoma, cerebral infarct, pseudobulbar palsy, uncontrolled diabetes mellitus, multiple sclerosis, Lyme disease, pregnancy, and viral infections including Herpes-Zoster, HIV, Ramsay Hunt syndrome [13]. Other conditions include myasthenia gravis, lymphoma, sarcoidosis, Guillain-Barre syndrome, leukaemia, bacterial meningitis, leprosy, syphilis, infectious mononucleosis, trauma (skull fracture), Pontine lacunar infarct, microcirculatory failure of the vasonervorum, ischemic neuropathy, autoimmune reactions, rhabdomyosarcoma, mucormycosis, polio, tuberculosis, chronic alcoholism, carbon monoxide poisoning [14].

Katz et al. [15] described history of transient facial palsy in 18% of their patients with Burkitt's lymphoma. Cartwright et al. [16] reported 1% incidence of lower motor neurone facial nerve palsy in patients with lympho-proliferative malignancies.

Dental causes have also been reported with facial paralysis such as orofacial granulomatosis, infections, 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 and surgical treatment of facial fractures and cleft lip/palate [17-23]. Others are Temporal bone fractures and fracture of the mandibular condylar neck.

Most sources suggest that if a patient with a sudden onset of facial paralysis diagnosed as Bell's palsy does not show return of facial muscle function from 4 weeks of onset of paralysis to 6 months, further testing (imaging) should be conducted to rule out other origins of the paralysis such as bone tumours. Treatment of cause of palsy will help alleviate the paralysis.

Treatment of Bell's palsy is controversial, because as many as two-thirds of patients recover spontaneously. Bell's palsy is normally treated using corticosteroids (prednisolone 1mg/kg) with or without antiviral agent (Acyclovir) and supplemented with vitamin B [24]. Considerable controversy remains over the use of steroids for Bell's palsy in adults. Although many studies have examined the usefulness of corticosteroids in the treatment of Bell's palsy, they have been limited by small sample size and lack of randomization, controls and blinding. Also there are controversies over the use of antiviral agents for treating bell's palsy. Adour reported that patients with Bell's palsy treated with acyclovir and prednisone experience a more favourable recovery and less neural degeneration than patients treated with placebo plus prednisone [25]. Tazi, Soichot and Perrin [21] report that the treatment of Bell's palsy is still controversial because the benefit of acyclovir has not been definitively established. However, the safety of this anti-herpetic drug combined with prednisone and its possible effectiveness in improving facial functional outcomes in patients with Bell's palsy make most experts favor its use with corticosteroids as soon as possible to treat patients with this disease [25].

Patients are advised to use sunglasses during the day and ophthalmic ointment and night eye patches to prevent corneal abrasions and must avoid dirty noxious fumes. Artificial tears are advised to keep the eyes moist and prevent exposure keratitis [24]. Patients are advised to start facial physiotherapy exercises combined with warm water compresses. Physiotherapy in the form of transcutaneous peripheral nerve stimulation, tarsorrhaphy and sometimes surgical nerve decompression for patients with persistent paralysis are also some of the modalities in the treatment of bell's palsy. Peripheral vasodilator may also be used.

Local superficial heat therapy for 15 minutes per session for the facial muscles prior to electrical stimulation has been recommended as part of physical treatment. Facial massages are also encouraged to improve circulation and reduce occurrence of contracture [26]. Acupuncture as an alternative therapy has emerged but is controversial as there isn't sufficient scientific evidence to support it as an effective therapy [27]. Hyperbaric oxygen therapy can be considered as a treatment option. It is reported to have better recovery than prednisone [28]. For management of hyperkinesis, synkinesis, and hemifacial spasms, local

injection of Botulinum toxin-A is an appropriate therapy, especially in cases where surgery is not indicated [29]. 75% of Bell's palsy cases regress spontaneously with complete recovery. Approximately 15% of the cases have satisfactory recovery with a slightly detectable neurological deficit and 10% of the cases have permanent paralysis with residual weakness, hyperkinesia, synkinesis, or contracture [8]. A favourable prognosis is associated with Bell's palsy which occurs in a single episode, is painless, involves only a partial paralysis of the peripheral part of the facial nerve, and has early signs of recovery. A good prognosis is associated with Bell's palsy seen in children compared to elderly people [8]. Bell's palsy usually do not recur. Bell's palsy reported in our patient did not regress either spontaneously or with the treatment which she received at the time of onset of the facial palsy. At present she had mild neurological deficit with permanent paralysis of facial musculature to which she got adapted well.

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 [30].

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