BELL’S PALSY - A PSYCHOLOGICALLY DISTRESSING CONDITION - OVERVIEW OF LITERATURE

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ABSTRACT

Bell’s palsy is an acute onset facial nerve paralysis of unknown etiology, although, herpes virus has largely been implicated as a causative agent. The muscle weakness is abrupt, unilateral within first day, resulting in numbness and pain around the ear, taste and sound alterations. Diagnosis is usually made by excluding other causes of facial nerve paralysis. Majority of the cases show spontaneous recovery. Corticosteroids and antivirals may be beneficial, although, physical therapy, acupuncture, botulinum toxin and hyperbaric oxygen therapy may also play a role in the management of this psychologically distressing condition.

KEYWORDS: Bells Palsy, facial paralysis, herpes Virus infection, corticosteroids, antiviral therapy.

INTRODUCTION

Bell’s palsy is an acute onset, idiopathic, unilateral, lower motor neuron facial paresis (partial diminished movement), or paralysis (complete loss of movement) with no identifiable aetiologies, such as infection, neoplasm or trauma.1, 2 Bell’s palsy accounts for the most common cause of acute facial nerve (VII cranial nerve) paralysis.3 Dr. Charles Bell in 1821, described the first case of complete facial paralysis after injury of the stylomastoid foramen, and the condition is named after him.4 Etiopathogenesis of Bell’s palsy remains highly controversial. The attributable causes may be a viral infection, vascular ischemia, autoimmune inflammation and heredity.5,6 Herpes simplex virus mediated viral inflammatory immune mechanism has emerged as a major cause, as per observations with the polymerase chain reaction to detect viral DNA.7 Majority of cases are self-limiting,
nonprogressive and spontaneously remitting, although, few patients may present with residual neurologic dysfunction.[8]

**EPIDEMIOLOGY**

Bell’s palsy affects 11–40 persons per 100,000 each year, usually between the ages of 15 and 50 years.[9-11] More than 60,000 cases are diagnosed annually in the United States, [12] with similar incidence rates reported among males and females.[13] Pregnant females during the third trimester and early postpartum periods, have 3 times greater risk of Bell’s palsy than the general population.[14,15] Diabetics, [3,16-18] Elderly individuals, [19-21] and patients with hypothyroidism forms the other affected group.[22]

**ETIOPATHOGENESIS**

The etio pathogenesis of Bell’s palsy remains highly debatable. Acute inflammation and edema of the facial nerve leads to entrapment of the nerve in the bony canal, thus resulting in compression ischemia. Patients with acute Bell’s palsy shows facial nerve enhancement on magnetic resonance imaging (MRI), thus, supporting an inflammatory etiology.[23]

Many viruses, such as HIV, [24] Epstein-Barr virus, [25] and hepatitis B virus [26] have been implicated in initiating this inflammation, but herpes simplex virus (HSV) is the most frequently involved.[27,28] Reactivation and replication of dormant HSV causes inflammation, mainly in the geniculate ganglion cells and in the labyrinthine segment of the facial nerve. Polymerase chain reaction detects HSV in the endoneural fluid, posterior auricular muscle and saliv in patients with Bell’s palsy.

In the past, peripheral neuropathies have been linked with the Influenza vaccines. Although influenza vaccines currently available in the United States have not been associated with Bell’s palsy, [29-31] a recently developed Swiss intranasal vaccine was found to have a very high risk of postvaccine facial nerve palsy and has been withdrawn from use.[32]

The proposed non-infectious causes of Bell’s palsy include autoimmune processes such as Hashimoto’s encephalopathy, [33,34] ischemia from atherosclerosis leading to facial nerve edema, [20-22] and heredity basis, with about 4% to 8% of Bell’s palsy patients reported to have an associated family history.[14]

**CLINICAL MANIFESTATIONS**
Bell’s palsy has a sudden and rapid onset of unilateral facial weakness, often within a few hours.\cite{35} The symptoms may be so pronounced and may mimic a stroke or a serious brain lesion.\cite{10} A preceding viral illness may be noted in up to 60\% of these patients.\cite{36}

Initially, most patients present with partial palsy and maximum facial weakness is often seen within 2 days.\cite{10,35} Patients may also complain of ipsilateral earache as well as numbness of the face, tongue, and ear.\cite{37} Moreover, cases of hyperacusis (possibly from stapedial muscle dysfunction), tinnitus, taste disturbances (most likely from injury to nervus intermedius proximal to geniculate ganglion), and decreased lacrimation have also been reported.\cite{10,14,22} Impaired ipsilateral movement of the affected side of the face, drooping of the corner of the mouth and eyebrow, and the loss of the ipsilateral nasolabial fold may also occur as Bell’s palsy involves the peripheral facial nerve. Bell’s phenomenon – the upward movement of the eye on attempted closure of the lid due to weakness of the orbicularis oculi is a pathognomonic sign.\cite{22,38} [Fig. 1-4]

Fig. 1- Inability to raise the eyebrow on the right side.  Fig. 2- Inability to close the right eye.

Fig. 3- Drooping of the right corner of mouth.  Fig. 4- Inability to blow the right cheek.

Pooling of food and saliva may occur in the affected side of the mouth and may spill out from the corner. Bell’s palsy is almost always unilateral, and both the upper and lower parts of the
face are affected, thus, distinguishing the disorder from a central supranuclear lesion, in which paresis is seen only in the lower facial muscles.[22]

**DIAGNOSIS**

Determination of central or peripheral facial weakness is the first step in the diagnosis. Peripheral facial palsy involves all the facial muscles ipsilateral to the side of facial nerve involvement where as central weakness involves lower facial muscles contralateral to the lesion in the brain stem above pons and cerebral hemisphere.[39]

Bell’s palsy is typically a diagnosis of exclusion, and a thorough history and physical examination is mandatory to rule out other treatable or intracranial lesions.[14]

Onset and progress of paralysis forms an important basis of history taking as gradual onset of more than two weeks’ duration is highly suggestive of a mass lesion. History of recent rashes, arthralgias, or fevers; peripheral nerve palsy; exposure to influenza vaccine or new medications; and exposure to ticks in areas where Lyme disease is endemic should also be excluded.

Careful inspection of the ear canal, tympanic membrane, and oropharynx, evaluation of peripheral nerve function in the extremities and palpation of the parotid gland should be included in the clinical examination. Evaluation of cranial nerve function including all facial muscles must also be considered to assess forehead involvement. Clinical evidence of herpes zoster infection may help aid in the diagnosis of Bell’s palsy. However, vesicular lesions may not be seen in the presence of pre-herpetic neuralgia in a clinical condition termed Zoster sine herpete.[10]

Laboratory and imaging studies are not routine investigations in the diagnosis of Bell’s palsy and are only recommended in patients with recurrence, or if the condition does not resolve after more than 3 weeks of therapy.[40]

Blood glucose monitoring- As more than 10 percent of patients with Bell’s palsy have diabetes mellitus, fasting glucose or A1C testing may be helpful in patients with additional risk factors (e.g., family history, obesity, older than 30 years).[41]

Complete blood count with differential- helps rule out lymphoreticular malignancy, as peripheral facial palsy may be the first presentation of these malignancies.[42]
Serological tests may be essential to rule out Lyme disease in endemic areas. Although, Bell’s palsy is rare in children below 10 years of age, as many as 50% of the reported cases of facial palsy in children are caused by Lyme disease.\cite{14}

Computed tomography or magnetic resonance imaging (MRI) may be performed if the patient does not recover within the expected timeframe. MRI with gadolinium is the test of choice to rule out cerebellopontine angle tumor, stroke, multiple sclerosis, or other structural lesions. CT is recommended in cases of temporal bone fracture.\cite{43}

**DIFFERENTIAL DIAGNOSIS**

Differentiation of Bell’s palsy from other causes of facial paralysis such as diabetes mellitus, human immunodeficiency virus (HIV) infection, Lyme disease, Ramsay Hunt syndrome (peripheral facial palsy with zoster oticus), sarcoidosis, Sjogren's syndrome, parotid-nerve tumors, leprosy, polyarteritis nodosa and amyloidosis is important as Bell’s palsy has a rapid onset over several hours. Facial palsy secondary to other causes progresses over days to months.\cite{39} This has been depicted in Table 1.

**Table 1. Differential Diagnosis for Facial Nerve Palsy**

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>CAUSE</th>
<th>DISTINGUISHING FEATURES</th>
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<tbody>
<tr>
<td>Lyme disease</td>
<td>Spirochete Borrelia burgdorferi</td>
<td>History of tick exposure, rash, or arthralgias; exposure to areas where Lyme disease is endemic</td>
</tr>
<tr>
<td>Otitis media</td>
<td>Bacterial pathogens</td>
<td>Gradual onset; ear pain, fever, and conductive hearing loss</td>
</tr>
<tr>
<td>Ramsay Hunt syndrome</td>
<td>Herpes zoster virus</td>
<td>Pronounced prodrome of pain; vesicular eruption in ear canal or pharynx</td>
</tr>
<tr>
<td>Sarcoidosis or GuillainBarré syndrome</td>
<td>Autoimmune response</td>
<td>More often bilateral</td>
</tr>
<tr>
<td>Tumor</td>
<td>Cholesteatoma, parotid gland</td>
<td>Gradual onset</td>
</tr>
</tbody>
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**SUPRANUCLEAR** (central)  Forehead spared

<table>
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<tbody>
<tr>
<td>Multiple sclerosis</td>
<td>Demyelination</td>
<td>Additional neurologic symptoms</td>
</tr>
<tr>
<td>Stroke</td>
<td>Ischemia, hemorrhage</td>
<td>Extremities on affected side often involved</td>
</tr>
<tr>
<td>Tumor</td>
<td>Metastases, primary brain</td>
<td>Gradual onset; mental status changes; history of cancer</td>
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**GRADING**

Recovery of facial nerve function is an important outcome that guides treatment recommendations. The initial severity of facial weakness provides valuable prognostic
information for facial recovery. Commonly used facial grading instruments (e.g., House–Brackmann and Sunnybrook scales) quantify the severity of facial weakness.\cite{44,45}

**Table 2: House-Brackmann facial nerve grading system with prognosis.**

<table>
<thead>
<tr>
<th>GRADING</th>
<th>CLINICAL FEATURES</th>
<th>PROGNOSIS</th>
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<tbody>
<tr>
<td>I</td>
<td>Normal</td>
<td>Good</td>
</tr>
<tr>
<td>II</td>
<td>Mild dysfunction; slight weakness noticeable only on close inspection</td>
<td>Good</td>
</tr>
<tr>
<td>III</td>
<td>Moderate dysfunction; obvious, but not disfiguring, difference between the two sides</td>
<td>With moderate dysfunction</td>
</tr>
<tr>
<td>IV</td>
<td>Moderately severe dysfunction; obvious weakness and/or disfiguring asymmetry</td>
<td>With moderate dysfunction</td>
</tr>
<tr>
<td>V</td>
<td>Only barely perceptive motion</td>
<td>Poor</td>
</tr>
<tr>
<td>VI</td>
<td>Loss of tone</td>
<td>Poor</td>
</tr>
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**TREATMENT MODALITIES**

Treatment of Bell’s palsy remains controversial due to inadequate number of randomized controlled trials.

**SPONTANEOUS RECOVERY**

Majority of Bell’s palsy patients have a high rate of spontaneous recovery, hence a statistically significant benefit of treatment in placebo-controlled trials is difficult. According to the Copenhagen Facial Nerve Study, out of the 2,570 persons with untreated facial nerve palsy, including 1,701 with idiopathic (Bell’s) palsy and 869 with palsy from other causes; 70 percent had complete paralysis. 85 percent of patients showed recovery of function within three weeks, with 71 percent of these patients showing full function recovery. Among the 29 percent of patients with sequelae, 12 percent marked it slight, 13 percent marked it mild, and 4 percent marked it severe.\cite{46} Because of these findings, some persons have raised queries whether treatment for Bell’s palsy should be routinely indicated; however, patients with incomplete recovery will have obvious cosmetic sequelae and will often be dissatisfied with their outcome.\cite{47}

**CORTICOSTEROIDS**

As inflammation and edema of the facial nerve are implicated in pathogenesis of Bell's palsy and corticosteroids have a potent anti-inflammatory action, thus, minimizing nerve damage and improving the outcome. This forms the basis for the use of corticosteroids in acute phase of Bell's palsy. The American Academy of Neurology [AAN] guidelines 2012 concludes that steroids are highly effective in the recovery of the facial nerve function in acute onset Bell’s
Prednisolone initiated within 48 hours showed significant recovery and between 49 hours -72 hours, showed insignificant difference in the rate of recovery between untreated and treated patients. The prednisolone dose used was 60 mg per day for 5 days then reduced by 10 mg per day (for a total treatment time of 10 days) 50 and 50 mg per day (in two divided doses) for 10 days. Diabetic patients with Bell’s palsy require high-dose steroids (>120 mg/day of prednisone). As corticosteroids are cost-effective,18 clinicians can promote their use for all patients without medical contraindications.

ANTIVIRAL DRUGS
As HSV-1 has been implicated in the etiology of Bell’s palsy, role of antiviral drugs like acyclovir (Zovirax) and valacyclovir (Valtrex) in the treatment of Bell’s palsy has been studied. Acyclovir 400 mg five times per day for seven days or valacyclovir 1 g three times per day for seven days seems beneficial. A 2004 Cochrane review refuted the use of the antivirals alone, although, two recent placebo-controlled trials established full recovery in a higher percentage of patients after combined therapy with an antiviral drug and prednisolone than with prednisolone alone (100 percent versus 91 percent and 95 percent versus 90 percent). However, no benefit was seen when treatment was initiated more than four days after the onset of symptoms (86 percent versus 87 percent).

SURGERY
In the past, surgical decompression within three weeks of onset has been recommended for patients who have persistent loss of function (greater than 90 percent loss on electoneurography) at two weeks. Hearing loss (3%–10% of patients), further damage to the facial nerve (prevalence not known) and cerebrospinal fluid leakage (4%) are the potentially serious risks associated with surgical decompression. The American Academy of Neurology (AAN) currently do not recommend surgical decompression for Bell’s palsy due to evidence of significant risk potential and inadequate data supporting benefits of surgery.

PHYSICAL THERAPIES
Various forms of physical therapy that have been used for Bell’spalsy include Thermal methods, electrotherapy, facial exercises, massage and biofeedback. Facial exercises should be performed while standing in front of a mirror and include trying to raise the eyebrows, opening and closing the eyes, blowing, and whistling. In 2011, Pereira LM et
Hasan et al. reported that facial exercises act as an adjust therapy to improve the facial functionality and recovery in patients with Bell’s Palsy.[62]

ACUPUNCTURE

Acupuncture has been used as a treatment strategy for Bell’s palsy, although, Cochrane review in 2010 conclude that the role of acupuncture in Bell’s palsy has not yet been established. The number and quality of trials are too low to form conclusions.[63]

MISCELLANEOUS THERAPIES

Botulinum toxin type A (Botox) causes muscular paralysis by blocking the uptake of acetylcholine in the muscles. Injectable therapy results in temporary diminution of the unusual contractions and paralysis of the targeted areas of synkinesis.

Role of electrotherapy in the treatment of Bell’s palsy is not yet established, and has not yet been proved to improve coordination of synkinesis or inhibit abnormal movement patterns. On the contrary, it may strengthen abnormal patterns by activating existing hyperactive muscles.[64]

In accordance to a hypothesis, reduction in hypoxic degeneration of the facial nerve may result in improvement of functional recovery. Hyperbaric oxygen therapy (HBOT) is administered as a series of ‘dives’ in which the patient breathes 100% oxygen in a compression chamber. HBOT increases the oxygenation of tissues surrounding hypoxic areas, thus, increasing the oxygen diffusion gradient.[65] Laser phototherapy has been proposed as an adjunct treatment modality in the last decade. Marques et al, reported a case of Bell’s Palsy that was successfully treated by low level infra-red lasers. [66] Low level laser therapy has effectively resulted in regeneration and improved function of the nerves involved, including the facial nerve.[67]

CONCLUSION

Bell’s palsy, also known as idiopathic facial palsy is a common lower motor neuron disorder with an obscure etiopathogenesis, although herpes virus has been commonly implicated as a causative agent. Diagnosis is usually made by exclusion, and treatment of this psychologically distressing condition remains controversial till date.

CONFLICT OF INTEREST

There is no conflict of interest.
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