

Bell's Palsy - Revisited

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Pages with reference to book, From 64 To 74

Bell's palsy shows no gender predilection. Female teenagers suffer in large number. It is familial in 10% of the patients. Diabetics and pregnant are predisposed. Infections, immunologic and genetic factors have been suggested to play roles in the aetio-pathogenesis of the disease. Herpes Simplex virus has been demonstrated in the geniculate ganglion by using molecular techniques supported by raised antiviral antibody titres. Entrapment of the nerve at the meatal foramen is the principle pathology. MRI enhancement favours involvement of the labyrinthine, geniculate and the proximal tympanic segments of the facial nerve. Poor vasculature of the labyrinthine segment makes it a vulnerable site.

Evaluation 'of Bells' palsy needs a careful history, head and neck examination. Topographic diagnosis is not very useful. Electrophysiological studies are useful when the paralysis is complete.

Electroneuronography (Enog) informs about the degree of degeneration in facial nerve fibers.

Transcranial magnetic stimulation (TMS) is a new, cheap and non-invasive tool to assess facial nerve's intracranial segment close to the internal meatus. Presence of acoustic reflex is a favourable prognostic indicator. Interferon induced enzyme 2' -5' Oligoadenylate synthetase is a supplementary diagnostic tool. Gadolinium enhanced MM is helpful as a diagnostic and prognostic instrument in selected cases.

Bell's palsy can be a manifestation of neuroborrel iosis, multiple sclerosis, diabetes, HIV infection and Burkitt's lymphoma.

Oral steroids should be started in all the cases of Bell's palsy immediately irrespective of the clinical status. Parenteral steroids, combination of steroids and Acyclovir has produced better results. The role of stellate ganglion block to improve the circulation in Bell's palsy is hypothetical. Middle cranial fossa approach to decompress the facial nerve has proved to be the choicest of the surgical procedures in selected group of refractory Bell's palsy. Care of the eye is very important in Bell's palsy and tarsorrhaphy may be required in cases of corneal abrasions. Old age, post-auricular pain, dysgeusia, hyperacusis, dry eye and delayed onset of recovery affect a bad prognosis. Enog, TMS and eye blink reflex response are the other useful prognostic indices.

Introduction

Bell's palsy has remained a subject of continuing interest for many disciplines of medicine. The neurologist, otolaryngologist and the primary care physicians share the knowledge about the controversies associated with the management of this disease. Burning issues revolve around the aetiopathogenesis, uncertainty about the disease behaviour and the conflicting approaches towards management strategies. With the advent of magnetic resonance imaging, useful data has been compiled about its precise site of the lesion along the course of the facial nerve. Advancements in the immunocytochemical studies have helped to understand the pathogenesis on some sound reasoning.

Bell's palsy is said to be the commonest cause of facial paralysis. Is it still the diagnosis of exclusion? The present article is meant to examine this issue along with literature update on the management.

Epidemiology

Bell's palsy shows no gender predilection. It is said to affect female teenagers more, but after the fourth decade, males are seen to suffer in a larger number. This distribution looks compatible with some relation to menarche and menopause. Pregnant women are more at risk to suffer from Bell's palsy and sometimes it is seen in the postpartum period¹. Suggested explanation for this association include fluid retention, hypertension, compromise of vasa nervorum, infections and the autoimmune process². There

is no left or right-sided inclination for Bell's palsy. Less than 1% of the cases are bilateral. Recurrence rate is about 10% that can present on the same or the contralateral side³ Bell's palsy runs a familial course in 10% of the patients. Data gathered from South America show about 46% family antecedents to suffer from this condition which is surprisingly high supporting a genetic basis⁴ Diabetics are more prone to suffer. In recurrent Bell's palsy abnormal glucose tolerance tests are more frequent⁵. Evidence regarding the seasonality and epidemicity is lacking⁶.

Aetio-pathology

The exact cause of Bell's palsy is still not known. Infections, immunologic and genetic factors have been suggested to play roles in the pathogenesis of the disease. The hypothesis that hypoxia and compression of the facial nerve induced by oedema in the facial canal is widely accepted⁷. Changes are brought about by the Herpes Simplex virus (HSV) infection⁸ In the contemporary times, HSV has been demonstrated in the geniculate ganglion by using molecular techniques⁹. This hypothesis looks more tangible when HSV antibody titres were found to be increased in sufferers of Bell's palsy, when 2-20% of the patients has demonstrated sudden changes in the antibody titres to Herpes group of viruses (Herpes Zoster, Rubella, Cytomegalo, Adeno, Rhino, Mumps and Ebstein Barr). As the patient recovers from a primary HSV infection, the virus subsides in a latency phase in the various cranial and spinal nerve ganglia¹⁰ Through some poorly understood mechanism, the virus is reactivated within the ganglion cells. Circulating antibodies guard against the dissemination of the virus. This promotes local ganglionitis which may manifest clinically as hypofunction. The virus may travel up or down the axon to induce a radiculitis of a nerve plexus. It may reach brain stem to excite a local inflammatory response in terms of meningoencephalitis, which may reflect as an increased protein content in the cerebrospinal fluid³.

The virus may infect the Schwann's cell in the nerve. It acquires a protein coat from the nerve cell as it escapes through the membrane. This operation excites an autoimmune response to the nerve-cell membrane. To follow this phenomenon is the lymphocytic infiltration in the affected nerve fiber, leading to fragmentation of myelin, demyelination and chromatolysis of the facial nucleus³ The cell function is impaired till such time that the distal disease process resolves. Functional muscle reinnervation revive as remyelination starts.

Until recently there was no agreement on the probable location of the facial nerve segment implicated in the Bell's palsy. Magnetic resonance imaging studies procured during the course of Bell's palsy have exhibited enhance in the labyrinthine, geniculate and the proximal tympanic segments of the facial nerve¹¹. Entrapment at the site of meatal foramen as the principle factor has been the subject of debate since long. This happens to be the narrowest point in the fallopian canal. Vulnerable vascular channels, conduction block at this point due to neuropraxic effect, MM enhancement of the segment and above all intra-operative observations favour this hypothesis¹². There are counter evidences to suggest that facial nerve is not tightly contained at the meatal foramen as studied in children. This provides a possible explanation for the relative infrequency of Bell's palsy in the young age¹³. Data accumulated from the cadaver and animal studies points to the fact that labyrinthine segment of the facial nerve contain fewer and smaller intrinsic blood vessels compared to the mastoid and the tympanic segments. This substantiates the contention that labyrinthine segment is the most likely site of lesion in Bell's palsy¹⁴. Autopsy studies in the temporal bone have shown congestion and infiltration of the nerve in the internal auditory meatus. There was compression of the nerve in the proximal portion while demyelination was observed in the tympanic segment¹³. Interestingly histopathological analysis showed a sharp line of demarcation between sclerotic nerve proximal to and necrotic nerve distal to the meatal foramen, favouring this to be the ideal location of the lesion¹⁵.

Diagnosis

Bell's palsy typically presents with an acute unilateral facial weakness, evolving over a period of 24-48 hours. There are certain symptoms, which precede the onset of facial palsy. These include feeling of numbness over face, epiphora, vague feeling of pain, dysgeusia and hyperacusia. The last mentioned symptom is a hallmark of an impending facial palsy³. Incomplete paralysis changes into a complete one over a course of few hours.

The evaluation of Bell's palsy should start with a thorough history, head and neck examination including a full neurological work out. The practical usefulness of detailed topographic diagnosis in cases of Bell's palsy is questioned. The site-of-lesion tests that may indicate about the severity or prognosis of the lesion, is doubtful. However, Schirmer's test for quantification of tearing has practical value to apply eye protection measures effectively, though results are difficult to interpret³.

What is the precise role of electrophysiological studies? They provide useful information about the degree of degeneration in the nerve and carry weight when the paralysis is complete. Testing would be inessential for incomplete cases of Bell's palsy. Good screening tests could be minimal nerve excitability (MNE) and electroneuronography. Enog is a prognostic indicator of favorable return of function when the degree of degeneration is calculated at 90%. Transcranial Magnetic Stimulation (TMS) is a recent development that is a non-invasive method to excite facial nerve in its intracranial segment close to the internal acoustic meatus. This is the most favored location of the lesion in Bell's palsy. TMS allows stimulation of the facial nerve above the stylomastoid foramen, facilitating exposure of a greater tract of the nerve. Compared to Enog motor response with TMS predicts good prognosis of the Bell's palsy at an early stage whereas poor response with Enog predicts less favorable prognosis at a later stage¹⁵. Further, TMS is well tolerated and a cheaper tool and does not need to be repeated serially like Enog¹⁶. Critics of the TMS have examined the validity of this mode and determined that this mode has failed to provide data which can be correlated to the clinical situation observed at the time of study; further TMS does not furnish prognostic data on the clinical evolution of the lesion¹⁷. One parameter that is worth including in the diagnostic protocol of Bell's palsy is the presence or absence of acoustic reflex, particularly in children. Shorter duration and higher percentage of recovery have been found in young sufferers of Bell's palsy with normal acoustic reflex than those with an abnormal or absent reflex¹⁸. Absence of acoustic reflex in Bell's palsy further suggests that this is not a mononeuropathy in the strict sense. It does involve certain of the auditory fibers of the eighth nerve and is worth considering as a polyneuropathy. Stapedius function is important in speech discrimination at higher level of sound intensity¹⁹. However, stapedial reflex findings have demonstrated that this alone is not sufficient to be of prognostic value, but is useful when considered with other clinical parameters and electrical responses²⁰. Central nervous system involvement in Bell's palsy cases has been postulated. Prolonged brain stem conduction time in a significant number of cases with Bell's palsy have been picked up as abnormal auditory brain stem responses to support this hypothesis, independent of the site or severity of the paralysis²¹.

Interferon is produced in response to viral infection and plays an important part in the defense by their antiviral effects. Elevated serum levels of interferon induced enzyme 2' -5' Oligoadenylate synthetase (2' -5' AS) could serve as an effective supplementary diagnostic tool in certain cases of Bell's palsy. Exaggerated levels of 2' -5' AS have been found in cases of complete Bell's palsy²². Elevated serum endothelin levels in cases of Bell's palsy have been a subject of discussion. Endothelin with its potent vasoconstriction action has been found to be implicated in the aetiology of Bell's palsy²³.

Demyelination associated with Bell's palsy depicts an increased level of myelin associated enzyme 2'3' cyclic nucleotide 3 phosphohydrolase (CNP) in the cerebrospinal fluid studies. Association has been reported between antibodies against Herpes Simplex Virus and elevated CNP activity. Serum levels of C4 containing circulating immune complexes in patients with Bell's palsy have been detected using ELISA method²⁴.

The fact that facial nerve in cases of paralysis presents an enhanced picture on magnetic resonance imaging (MRI) is now well established. This has been made possible after intravenous administration of Gadolinium diethylenetriamine. Most of the Bell's palsy cases demonstrate facial nerve enhancement usually in the distal internal auditory canal and labyrinthine/geniculate segments. Gadolinium enhancement occurs regardless of the severity of the paralysis^{25,26}. No difference between the pattern of MRI enhancement in acute or chronic Bell's palsy cases have been observed. Moreover, radiographic resolution appeared to lag behind the clinical improvement²⁷. Gadolinium enhanced MRI has proved its usefulness as a prognostic indicator. The patients with facial nerve enhancement confined to labyrinthine, geniculate and proximal tympanic segments have shown to regain complete facial function. Whereas contrast enhancement in the mastoid segment of the facial nerve have reported a poor outcome regarding complete return of the facial function²⁸. The mechanism of Gadolinium enhancement with MRI in Bell's palsy is a non-specific phenomenon, which is also seen in cases of traumatic facial lesions. MRI study is worthless, if ordered in every case of Bell's palsy. It is recommended in cases of recurrent facial palsy, atypical presentations, failure to elicit any electrical excitability or to exclude a tumour. MRI has its specific application when facial nerve decompression becomes mandatory in intractable cases of Bell's palsy²⁹.

Bell's palsy is said to be an idiopathic lesion having no apparent cause. However, an indistinguishable presentation can sometimes be a manifestation of Neuroborreliosis, multiple sclerosis, diabetes, facial nerve neurinoma and HIV infection³⁰. It has been reported as a manifestation of the Burkitt's lymphoma³¹. A low-grade deep-seated adenocarcinoma of the parotid may present with a picture, which closely mimics Bell's palsy, when facial weakness is the only initial symptom³². Infectious mononucleosis atypically may manifest itself with facial weakness similar to the Bell's palsy³³. Peripheral facial palsy resembling recurrent Bell's palsy has been reported to be precipitated by dental manipulation³⁴.

It has been accepted that Bell's palsy and a number of inner ear disorders may share a common underlying viral aetiology. This could explain as to why some cases who initially presented with Bell's palsy, lately develop vestibular dysfunction with a wide-ranging spectrum³⁵. Galvanic body's way test (GBST) has been accepted as a useful tool in the detection of retrolabyrinthine disorders associated with Bell's palsy. The higher incidence of positive GBST in cases of Bell's palsy is suggestive that 'vestibular neuronitis' may be caused by a similar pathogenesis³⁶. Peripheral facial palsy may be associated with Lyme disease³⁷. Melkersson Rosenthal syndrome is a rare disorder of unknown aetiology characterized by triad of relapsing facial palsy, or facial swelling and fissuring of tongue. The facial paresis is much alike Bell's palsy³⁸. Since the triad is not seen typically, the diagnosis becomes difficult.

MRI findings in the maxillary sinus in established cases of Bell's palsy demonstrate a transient inflammatory picture. This may be taken as an incidental finding, as understanding of the pathophysiology is poorly understood³⁹.

Treatment

The treatment of Bell's palsy has remained controversial. The burning issue is the need for starting treatment in incomplete palsy. It is now generally agreed apart from reassurance and expressions of optimism that oral corticosteroids should be started in all the cases of Bell's palsy as soon as the patient is seen, irrespective of the status of the palsy¹². The recommended dosage is Prednisolone 1 mg/kg/day for 5-10 days followed by a gradual reduction over the next 5 days. This can be rationalized on the fact that it is difficult to predict which patient will progress to the severe or complete form. If the treatment is delayed until the severity is determined, irreversible nerve damage may occur.

In a double-blind study, Bell's palsy was treated with combinations of Acyclovir-Prednisolone and

placeboPrednisolone as a control. The outcome with former was superior to that of the latter⁴⁰. A better return of the volitional muscle motion was observed. Favourable results have been reported with a protocol comprising of the stat intramuscular Prednisolone 60 mg followed by oral steroids. This regimen has accounted for relief in 95% of the sufferers without sequelae^{41,42}. Hyperbaric oxygen as an additional support has shown to enhance the therapeutic benefit in patients with Bell's palsy in terms of complete and shortened recovery⁴³.

Based on the pathophysiology of Bell's palsy that oedema and ischemia leads to compression and hypoxia, Stennert recently introduced parenteral large doses of steroid in the early phase of the Bell's palsy reporting an extremely high rate of 96%⁴¹. This protocol of therapy was not widely accepted because of the hepatic, renal and gastric adverse effects associated with it. Modifications of the Stennert method were employed where hydrocortisone sodium succinate, hydroxymethylated starch and Dmannitol were administered with identical results⁴².

Methylcobalamine when added to the steroid have improved the symptoms of the Bell's palsy significantly by curtailing the recovery period⁴⁴.

Stellate ganglion blockade (SGB) in Bell's palsy has been advocated to increase the microcirculation of the nutrient artery of the facial nerve⁴⁵. A published report contradicts the efficacy of SGB. A patient with traumatic cervical sytidrome having SGB subsequently developed Bell's palsy on the same side challenging the validity of the hypothesis⁴⁶. The role of physical therapy as a supplement to the treatment of Bell's palsy has been discussed. It is generally considered invaluable for maintaining confidence of the patient, but is unmanageable and an expense difficult to justify. Experimental research has shown that electrical stimulation of the denervated muscle retard ingrowth of the neurofibrils to the motor endplates⁴⁷. Contrary to this hypothesis high voltage electrical muscle stimulation has shown benefits in terms of accelerated progress in cases of Bell's palsy⁴⁸.

Surgical decompression of the facial nerve in Bell's palsy is a chronicled controversy. Timings of the decompression, appraoches and sites of the decompression and the imperfect regeneration following decompression have remained the issues of disagreement. The criteria for surgical candidacy include acceptable anaesthetic risk, less than 60 years of age, Enog demonstrating 90% or greater degeneration of nerve fibers within 3 weeks of the onset of paralysis, absence of the evidence of neuropraxia or deblocking on EMG and the informed consent¹⁴ Those fulfilling the requirement should be subjected to surgery only when the ear is free of infection and the proviso that the other ear has servicable hearing. Middle cranial fossa decompression is the procedure of the choice offered to the selected patients who meet the criteria on strict merit. Surgical decompression of the tympanic and mastoid segments are seldom helpful^{12,49}.

Among the supportive measures for treatiing, Bell's palsy care of the eye is of fundamental importance. Corneal abrasions and epithelial defects can lead to dreadful sequel, due to foreign body and drying effect. Dark glasses must be worn during the daytime and application of a bland eye ointment is essential during sleep. An early sign of keratitis calls for packing or tarsorrhaphy. Temporary closure of the eyelid is especially recommended when re-epithelialisation of the cornea becomes problematic. Tube tarsorrhaphy using plastic tubes sutured externally to the upper and lower eyelids is a newly devised technique⁵⁰. Here the eyelid closure is accomplished by tightening a loop of suture passed through the tube.

Prognosis

Predicting prognosis of Bell's palsy is difficult, particularly in the early phase of the disease. It was found that young patients with incomplete paralysis, unaccompanied by post-auricular pain loss of taste sensation, hyperacusis, dry eye and recovery beginning within the 4 weeks of the onset of palsy are likely to make complete recovery. In comparison, older patients with complete palsy, accompanied by postauricular pain, loss of taste, hyperacusia, dry eye and the onset of recovery after the 4 weeks of the

onset of palsy are more likely to have incomplete recovery⁵¹.

Electrophysiological tests are complimentary and when used appropriately can accurately describe the completeness of degeneration⁵². TMS is said to be a better mode of early prediction¹⁵. In a significant number of cases a contralateral early blink reflex response could be elicited on stimulation of the normal side. This indicates synaptic reorganization in the facial nucleus during regeneration and can be used as a prognostic indicator⁵³. Enog estimates the prognosis of the Bell's palsy particularly between the first and the third month of onset by serially testing to determine the end-point of degeneration⁵³. Facial nerve latency test (FNLT) is another useful prognostic marker. When latency is within normal limits the damage to the nerve is slight; if the latency is prolonged the prognosis is usually worse⁵⁴. Personal computers have been successfully employed to gauge the progress of Bell's palsy. Movements of the face were photographed with a video camera and fed continuously into the computer by means of digital image processing technique. Special marks placed on the face were extracted and the movements of these marks were quantitatively analyzed. The rate of improvement process can be evaluated by comparing the ratio of affected to normal side facial movements⁵⁵. MRI studies depict that patients with enhanced segment of the mastoid segment carries a poor prognosis for the complete return of the facial function.

One important issue in the prognostic consideration is the aberrant regeneration of nerve fibers after Bell's palsy. Complications of facial nerve regeneration include contractions, synkinesis (associated facial motion), tics (facial spasm) and gustatory tearing (crocodile tears). These are extremely difficult to treat. Synkinesia can be managed by Botulinum toxin injection into the affected muscle with reasonable results⁵⁶.

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