REVIEW ARTICLE

BELLS PALSY – A REVIEW OF IDIOPATHIC FACIAL PARALYSIS

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Introduction

Facial paralysis is a disfiguring disorder that has a great impact on the patient. Facial nerve paralysis may be congenital or neoplastic or may result from infection, trauma, toxic exposures, or iatrogenic causes. The most common cause of unilateral facial paralysis is Bell's palsy, more appropriately termed 'Idiopathic facial paralysis' (IFP). In Bell's Palsy, the onset is rapid, unilateral and there may be an ache beneath the ear. The weakness worsens over one to two days. Bell palsy is an acute, unilateral, peripheral, lower-motor-neuron facial-nerve paralysis that gradually resolves over time in 80-90% of cases. In an upper motor neuron lesion eg stroke, the forehead is spared since this region is bilaterally represented in the cortex. Looking for 'forehead sparing' is thus a way of differentiating between upper and lower motor neuron causes of facial weakness.

Etiology and Pathogenesis

Controversy surrounds the etiology and treatment of Bell's palsy. The cause of Bell palsy remains unknown, though it appears to be a polyneuritis with possible viral, inflammatory, autoimmune, and ischemic etiologies. According to a study, the pathogenesis of Bell's palsy is attributed to vascular changes because of retrograde epineurial compression edema with ischemia of the facial nerve. Although the etiology is unknown, an attractive theory is vasospasm, from any cause, along any facial nerve branch, with the chorda tympani, perhaps, the usual primary involvement. Retrograde vascular distension and edema, within the epineurium of the bony facial canal, compresses the nerve from outside its perineurial sheath. The compression force may be mild or severe, resulting in varying degrees of reversible or irreversible ischemic degeneration of myelin sheaths and axons, with varying degrees of cellular reaction to myelin breakdown. The edema may be resorbed, leaving reversible or irreversible nerve damage, or may stimulate collagen formation within the epineurium, with persisting fibrous compression (entrapment) neuropathy of the facial nerve. This concept is consistent with the varying results of Bell's palsy, and depends on the severity and duration of edema, and whether fibrosis occurs within the epineurium of the facial canal. Epineurial fibrosis also results in disturbance of metabolic exchange through the epineurial-perineurial-endoneurial tissues, and may ultimately result in obliteration of vascular drainage.[1] Another study provides support for cell-mediated immunopathogenesis in patients with Bell's palsy. Decreased percentages of B cells (CD19) and T helper/inducer (CD4) subsets were found in patients with Bell's palsy as compared with age-matched healthy control patients. This however showed no prognostic significance for the outcome.[2]

Increasing evidence implicates the role of herpes simplex type I and herpes zoster virus reactivation from cranial-nerve ganglia. Thirty-three of 42 affected patients had a positive HSV-1 enzyme-linked immunosorbant assay compared with 16 of 41 controls (P = 0.0003). Ten of 47 affected patients had a positive HSV-1 polymerase chain reaction compared with 4 of 45 of controls (P = 0.08). These findings support an association between HSV-1 infection and Bell palsy in children.[3] In recent studies, detection rates of herpes simplex type 1 (HSV-1) and Varicella-zoster were found to vary strongly which were probably caused by the use of different oral fluid collection devices in combination with molecular assays lacking standardization . But in a recent single-center pilot study, commercially available IVD/CE-labeled molecular assays based on fully automated DNA extraction and real-time PCR were employed, which allowed early and reliable detection of HSV-1 and VZV DNAs in patients with acute idiopathic peripheral facial nerve palsy and may provide a valuable decision support regarding start of antiviral treatment at the first clinical visit.[4]

Bell's palsy is one of the most common neurologic disorders affecting the cranial nerves, and it is the most common cause of facial paralysis worldwide. It is thought to account for approximately 60-75% of cases of acute unilateral facial paralysis. Bell's palsy is more common in adults, in people with diabetes, and in pregnant women. Bell's palsy during pregnancy is significantly associated with severe preeclampsia. Nevertheless, no significant association exists between Bell's palsy and adverse perinatal outcomes.[5] Children with idiopathic facial palsy (Bell's palsy) however have a very good prognosis, while treatment with prednisone does not certainly improve the outcome.

Bilateral facial paralysis is rare. It may be seen in sarcoidosis or the Guillain Barre Syndrome (idiopathic polyneuritis) or posterior cranial fossa tumours. The rare Melkersson-Rosenthal syndrome is a condition comprising tongue fissuring, unilateral facial palsy and facial swelling.

Management

Determining whether facial-nerve paralysis is peripheral or central is a key step in the diagnosis. A lesion involving the central motor neurons above the level of the facial nucleus in the pons causes weakness of the lower face alone. Thorough history taking and examination, including the ears, nose, throat, and cranial nerves, must be performed.

The minimum diagnostic criteria include paralysis or paresis of all muscle groups on one side of the face, sudden onset, and absence of central nervous system disease.

If the clinical findings are doubtful or if paralysis lasts longer than 6-8 weeks, further investigations, including gadolinium-enhanced magnetic resonance imaging of the temporal bones and pons, should be considered. Electrodiagnostic tests (eg, stapedius reflex test, evoked facial-nerve electromyography [EMG], audiography) may help improve the accuracy of prognosis in difficult cases. Further test include blood chemical investigations, cerebro-spinal-fluid-investigations, X-ray of the skull and mastoid, cerebral MRI, or nerve conduction studies.[6] In a study, the initial severity of facial weakness and the electroneurographically detected facial nerve degeneration were found to be important factors in predicting the long-term prognosis of Bell's palsy.[7]

Treatment of Bell's palsy should be conservative and guided by the severity and probable prognosis in each particular case. Studies have shown the benefit of high-dose corticosteroids for acute Bell palsy. Although antiviral treatment has been used in recent years, evidence is now available indicating that it may not be useful.[8-12] Because of acyclovir's relatively poor

bioavailability (15% to 30%), newer drugs in its class are being trialled. Better bioavailability, dosing regimens, and clinical effectiveness in treating shingles have been shown with valaciclovir (prodrug of aciclovir), famciclovir (prodrug of penciclovir), and sorivudine. A recent study showed a significantly better outcome in patients with Bell's palsy treated with valacyclovir and prednisone as compared with patients given no medical treatment. This difference in outcome was especially pronounced among elderly patients.[13]

If presentation is early, most clinicians give prednisolone for 5 days, the aim being to reduce neuronal oedema. Most recent studies therefore advocate prednisolone as the predominant medical treatment especially if begun with in the first 72 hours and less effective after seven days. It has been shown to improve the primary outcome i.e. recovery of facial function as rated on the House- Brackmann scale and secondary outcomes such as the quality of life, appearance and pain.[8,14-16] Additional measures include eye protection, physiotherapy, acupuncture, botulinum toxin, or possibly surgery. Prognosis of Bell's palsy is fair with complete recovery in about 80% of the cases, 15% experience some kind of permanent nerve damage and 5% remain with severe sequelae.[6]

An eye patch is of value to protect the cornea. Topical ocular therapy is useful in most cases, with the exception of those in which the condition is severe or prolonged. In these cases, surgical management is best. Several procedures are aimed at protecting the cornea from exposure and achieving facial symmetry. These procedures reduce the need for constant use of lubrication drops or ointments, may improve cosmesis, and may be needed to preserve vision on the affected side. These include the occlusion of eyelids with tape or patch in case of corneal erosions, External eyelid weights to improve mechanical blink,[17] Injection of hyaluronic acid gel in the prelevator aponeurosis region and/or pretarsal region in patients who are poor surgical candidates and/or as a temporary measure,[18] Tarsorraphy with suture or cynoacylate glue as a temporary / permanent measure,[19] Taping to prevent lower lid ectropion, Botulinum toxin injected at upper border of taisus to produce temporary ptosis,[20] and Botulinum toxin for synkinetic eyelid movements secondary to aberrant regeneration in which there are eye movements such as blinking associated with oral movements.[21,22]

Several physical therapies, including massage and facial exercises, are recommended to patients, but there are few controlled clinical trials of their effectiveness. Some recent evidence supports facial retraining (mime therapy) with biofeedback. A number of studies published in China have suggested acupuncture is beneficial for facial palsy.[23]

A multidisciplinary team approach (general practitioners, otolaryngologists, ophthalmologists, plastic surgeons, dental surgeons, physiotherapists, and psychologists) is essential when there is no prospect of further recovery of facial nerve function. Facial reanimation may be possible by a combination of static and dynamic surgical techniques and may result in functional as well as cosmetic improvements.[24-26]

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