Bell's palsy: Treatment guidelines

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The most common cause of acute onset unilateral peripheral facial weakness is Bell's palsy. The incidence of Bell's palsy is 20-30 cases for 100,000[1] and accounts for 60-70% of all cases of unilateral peripheral facial palsy.[2] Either sex is affected equally and may occur at any age, the median age is 40 years. The incidence is lowest under 10 years of age and highest in people over the age of 70. Left and right sides are affected equally.[3]

Clinical Characteristics

Bell's palsy is an acute peripheral facial weakness of unknown cause and the diagnosis can be established without difficulty in patients with unexplained unilateral isolated facial weakness. The onset is sudden and symptoms typically peak within a few days. Additional symptoms may include pain in or behind the ear, numbness or tingling in the affected side of the face usually without any objective deficit on neurological examination, hyperacusis and disturbed taste on the ipsilateral anterior part of the tongue.[1] Bilateral idiopathic facial palsy occurs less frequently than unilateral involvement. About 7% of patients with history of Bell's palsy may experience recurrence. The mean interval to first recurrence is reported at 9.8 years after the first episode.

Diagnosis

The first step in the diagnosis is to determine whether facial weakness is central or peripheral. 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.

Bell's palsy is differentiated from other causes of facial palsy 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, by its rapid onset over several hours. Facial palsy secondary to other causes progresses over days to months.

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Diagnostic Workup

Diagnosis of Bell's palsy in a patient with unilateral peripheral facial weakness of unknown cause is purely clinical. However, electrodiagnostic testing done within 14 days of onset may provide prognostic information.

The *nerve excitability test* determines the excitation threshold by recording the minimum electrical stimulus required to produce visible muscle contraction. A difference greater than 3.5 mA between affected and unaffected sides is considered to be significant in terms of poorer outcome. Measuring the peak-to-peak amplitude of the evoked compound action potential of the involved side compared to the normal side has prognostic importance. If there is a 90% or greater reduction in the amplitude of the affected side, the prognosis is poor.[4]

Currently the *trigeminal blink reflex* is the only test to measure intracranial pathway of the facial nerve and also useful test to study various postparalysis sequelae such as synkinesis and hemifacial spasms. With recovery of facial function the ipsilateral R1 latency becomes less prolonged and the amount of initial prolongation of this response correlates with greater loss of facial motor function.

Gadolinium contrast magnetic resonance (MRI) study reveals enhancement of internal acoustic meatal segment on the affected side; however, this is a non-specific finding. MRI should not be done routinely and should be the investigation to look for other possible causes for acute facial paralysis especially if there is little or no recovery of function.

Treatment

The aims of treatment in the acute phase of Bell's palsy include strategies to speed recovery and to prevent corneal complications. Eye care includes eye patching and lubrication, lubricating drops should be applied frequently during the day and a eye ointment should be used at night.[5] Strategies to speed recovery include physical therapy, corticosteroids and antiviral agents [Figure 1].

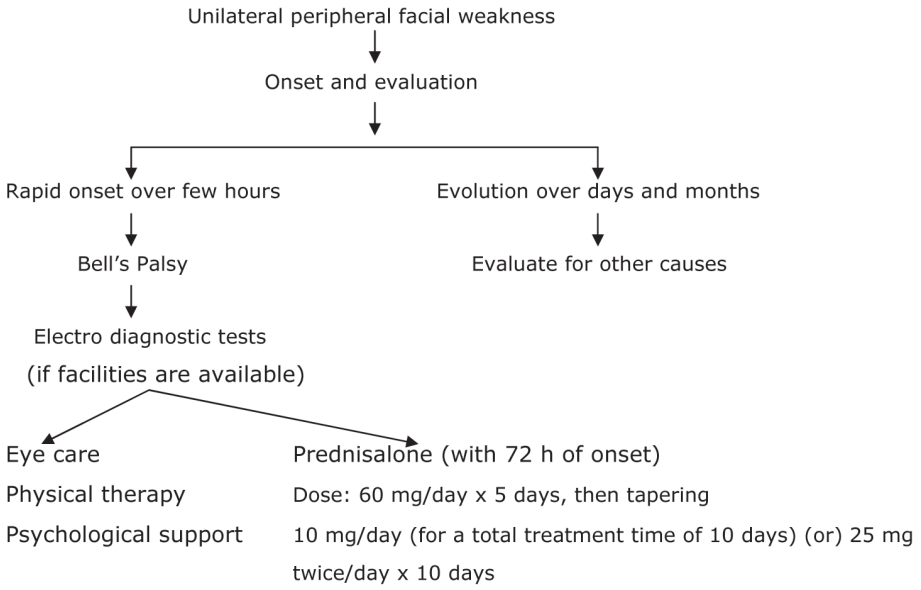

Figure 1: Bell's Palsy: Treatment algorithm

Prednisolone

The rationale for the use of corticosteroids in acute phase of Bell's palsy is that inflammation and edema of the facial nerve are implicated in causing Bell's palsy and corticosteroids have a potent anti-inflammatory action which should minimise nerve damage and thereby improve the outcome.

Randomized, double-blind, placebo-controlled trials have provided compelling evidence that treatment with prednisolone improves outcome in patients with Bell's palsy and shortens the time to complete recovery.[6-8] Prednisolone should be used in all patients with facial palsy of less than 72 h duration who do not have contraindications to steroid therapy. 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)[8] and 50 mg per day (in two divided doses) for 10 days.[7] The reported adverse rates were low. Treatment with prednisolone is likely to be cost-effective.[9]

Antiviral Agents

The rationale for the use of antiviral agents is the evidence that the inflammation of the facial nerve in Bell's palsy might be related to the herpes simplex virus (HSV). In an autopsy study latent HSV type-1 has been isolated from the majority of the geniculate ganglia samples.[10] HSV-1 genome was detected in 79% of facial nerve endoneurial fluid in patients with Bell's palsy, but not in the controls.[11] However, the benefit of acyclovir or valacyclovir, either as single agents or in combination with prednisolone in Bell's palsy has not been definitively established.[6-8,12,13] Thus with the available evidence acyclovir or valacyclovir should not be routine and treatment with acyclovir is highly unlikely to be considered cost-effective.[9]

Physical Therapy

In Bell's palsy various physical therapies, such as exercise, biofeedback, laser, electrotherapy, massage and thermotherapy are used to hasten recovery. However, the evidence for the efficacy any of these therapies, is lacking. Cochrane systemic review of the efficacy of physical therapies, electrostimulation and exercises, on outcome of Bell's palsy concluded that there was no significant benefit or harm from any of these physical therapies for Bell's palsy. There was limited evidence that improvement began earlier in the exercise group.[14] Another systematic review examined the effects of facial exercises associated either with mirror or electromyogram biofeedback with respect to complications of delayed recovery in Bell's palsy and concluded that because of the small number of randomized controlled trials, it was not possible to analyze if the exercises, were effective.[15] However, that the possibility that facial exercise reduces time to recover and sequelae needs confirming with good quality randomised controlled trials.[14]

Prognosis

About 71% of patients with Bell's palsy have motor function recovery completely within 6 months without treatment.[16,17] By 6 months all patients with Bell's palsy should show some improvement.[17] Poor prognostic factors include: old age, hypertension, diabetes mellitus, impairment of taste and complete facial weakness.[18] About one-third of patients may have incomplete recovery and residual effect. Among the residual effects include post-paralytic hemifacial spasm, co-contracting muscles, synkinesis, sweating while eating or during physical exertion. The two most common abnormal regeneration patterns are: 'crocodile tears' - lacrimation of the ipsilateral eye during chewing and 'jaw-winking' - closure of the ipsilateral eyelid when the jaw opens.

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