International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Drug Management of Bell's Palsy: A Review

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Abstract: ***Bell's Palsy, also called as Facial Palsy, is a common cranial neuropathy of the 7th cranial nerve that causes acute unilateral motor neuron facial paralysis. Infective, immune and ischaemic mechanisms are all common causes, but the precise cause of Bell's Palsy remains unclear. The condition may occur at any age, the median age being 40 years. It is more common in diabetic patients and has no specific sex predilection. Symptoms include unilateral facial muscle paralysis with no neurologic abnormalities which usually peaks in the first week and then gradually resolve over three weeks to three months. Approximately 70 - 80% patients recover spontaneously without any treatment. However, it is recommended to take a seven day course of acyclovir or valacyclovir and a tapering course of prednisone, initiated within three days of the onset of symptoms.***

Keywords: Bell's Palsy, idiopathic, paralysis, corticosteroids, combination therapy

1. Introduction

The most common peripheral paralysis of the seventh cranial nerve is Bell palsy. The disorder manifests clinically as a rapid onset, unilateral, lower motor neuron - type facial weakness with postauricular pain, dysgeusia, subjective change in facial sensation, and hyperacusis as comorbid symptoms. The anatomical structure of the human facial nerve, specifically its mixed nerve profile containing motor, sensory, and parasympathetic fibres, can explain this clinical presentation. The tendency of the facial nerve to form numerous connections with nearby cranial nerves [1] may also be the reason why certain symptoms, such as altered facial sensation (cranial nerve V), vestibular dysfunction (cranial nerve VIII), or pharyngeal symptoms, are occasionally noticed (cranial nerves IX and X). [2] Reduced lacrimation and salivation may also occur as a result of parasympathetic effects. [2] Maximal disability occurs within the first 48–72 h and the severity of the palsy correlates with the duration of facial dysfunction, the extent of facial recovery and impairment of quality of life.

Etiology

Bell's Palsy is, by definition, idiopathic. A growing body of evidence in the literature demonstrates a variety of potential clinical conditions and pathologies that manifest, at least in part, with a period of unilateral facial paralysis. Several viral illnesses have been highlighted in the literature, including herpes simplex virus, varicella - zoster virus, and Epstein - Barr virus. When a possible etiologic mechanism is present, healthcare providers may ambiguously (and incorrectly) refer to a diagnosis of Bell's Palsy. This can happen, for example, in the context of well - known associations (e. g. Ramsay - Hunt syndrome and Lyme disease). [3]

While there are numerous potential causes of facial nerve palsies, including idiopathic, traumatic, neoplastic, congenital, and autoimmune, Bell's Palsy is diagnosed in approximately 70% of cases.

Clinical Presentation

- Bell's palsy patients frequently complain of weakening or total paralysis of all the muscles on one side of the face.
- The forehead unfurrows, the nasolabial fold and facial creases vanish, and the corner of the mouth droops.
- The lower lid sags and the eyelids won't close; when they do, the eye rolls upward (Bell's phenomenon). Eye irritation is frequently caused by inadequate lubrication and prolonged exposure.
- Tear production reduces, but because the lids can no longer be controlled, tears might run out of the eye easily and the eye may appear to tear excessively.
- Food and saliva can collect in the mouth's affected side and may spill out of the corner of the mouth.
- Patients frequently complain of numbness induced on by their paralysis, yet facial sensation is preserved.

Within three days, and almost invariably within one week, Bell's palsy patients typically proceed from the onset of symptoms to their maximum level of weakness. Diagnosis should be reconsidered if the progression lasts for more than two weeks. If left untreated, 85% of patients will demonstrate at least partial improvement within three weeks of onset of symptoms. [4]

Investigations

The involvement of the chorda tympani, stapedius, posterior auricular, and petrosal nerves suggests that the temporal bone is the location of the malfunction

- Unilateral enhancement of the geniculate, labyrinthine, and meatal segments of the facial nerve in contrast - enhanced MRI studies further supports the localization to the intratemporal facial nerve. This imaging finding is thought to be a result of vascular obstruction around the facial nerve and disruption of the blood - brain barrier.
- The use of VZV and HSV primers on posterior auricular, tear, or face muscle samples in attempts to make a surrogate diagnosis have not been successful in establishing a reliable relationship between viral load and clinical characteristics. [5] Therefore, the efficacy of these tests as diagnostic tools is restricted.

Differential Diagnosis

Facial palsy has a wide differential diagnosis, [6] and misdiagnosis is common. Ramsey Hunt syndrome and Lyme disease should both be ruled out as potential causes of peripheral seventh nerve palsy. Tuberculosis, HIV, trauma, sarcoidosis, vasculitis, and neoplasm are all less common causes of facial palsy. According to one of the authors' (GC) experience in an expert referral setting, the initial consulting clinician misdiagnoses Bell's palsy 10.8% of the time. [7] Tumors (for example, facial nerve schwannoma, parotid malignancy, and, in rare cases, acoustic neuroma), herpes zoster oticus, and granulomatous diseases such as sarcoidosis and granulomatosis with polyangiitis (Wegener's granulomatosis) are all missed diagnoses. In addition, if there are recurrent episodes, clinicians should consider Melkersson - Rosenthal syndrome. This is an uncommon neurocutaneous syndrome characterised by recurrent facial palsy, orofacial edema, and a fissured tongue. Females are more likely to be diagnosed with Melkersson - Rosenthal syndrome.

2. Treatment

Corticosteroids

The American Academy of Neurology [8] (AAN) and the American Academy of Otolaryngology—Head and Neck Surgery Foundation [9] (AAO - HNSF) have just recently released recommendations for the care of Bell's palsy. Starting the oral steroid dose within the first 72 hours of the onset is recommended, and the regimen should be similar to either the Scottish [10] or European [11] randomised controlled trials (RCTs). This is either 50 mg prednisone for 10 days or 60 mg for 5 days, then reducing by 10 mg per day for the next 5 days. Both appear to be effective. [12]Since mild and moderate palsies have a high rate of spontaneous recovery, it has been suggested that the lack of significance shown by combined corticosteroid and antiviral therapy over corticosteroids alone in double - blind RCTs represents a dilution effect of these palsies, which hides any discernible benefit for the severe palsy subgroup. Positive results for combined therapy in non - double - blinded studies are evidence in favour of this. [13], [14]

Antiviral Agents

The use of antiviral agents is justified by evidence that the inflammation of the facial nerve in Bell's palsy is caused by the herpes simplex virus (HSV). Latent HSV type - 1 was isolated from the majority of the geniculate ganglia samples in an autopsy study. [15] The HSV - 1 genome was found in 79% of facial nerve endoneurial fluid from Bell's palsy patients but not in controls. . [16] However, the efficacy of acyclovir or valacyclovir, either alone or in combination with prednisolone, in Bell's palsy has yet to be proven. [10, 17, 18, 19, 20] With the available evidence, acyclovir or valacyclovir should not be used routinely, and acyclovir treatment is highly unlikely to be considered cost - effective. [21]

Physiotherapy

From an evidence - based standpoint, this diverse modality of treatment, which includes heat therapy, electrostimulation, massage, mime therapy, and biofeedback [9], is difficult to assess as a whole. There are numerous treatment regimes, and their timing and variability in implementation complicate their overall utility assessment. Although physiotherapy is not recommended for all Bell's palsy patients, [9] there are subgroups of patients for whom there is evidence to support its use. [22]Patients with incomplete recovery who have developed hypertonia, hyperkinesis, or synkinesis are included in this group, and neuromuscular retraining is tried before considering chemodenervation. [23] In the treatment of synkinesis, physiotherapy and chemodenervation are complementary.

Ophthalmic Treatment

If the corneal risk is low and the healing prognosis is good, applying a strong lubricant and taping or padding the eyelid overnight is usually sufficient. Preservative - free methylcellulose formulations can help if frequent infusions are required, and a mild ointment can be used at night. The extent of rabbit eyeballs and the amount of lubrication required can be reduced by temporarily loading the eyelid with an outer eyelid weight [24]

Injection of botulinum toxin percutaneously through skin folds or subconjunctivally at the superior margin of the eyelid causes complete ptosis and protects the cornea. [25] However, where there is a poor Bell's phenomenon together with a noticeable laxity of the lower lid, the cornea may still be at risk. This procedure also has the disadvantage of impairing the patient's vision, which may provide inadequate protection when levator function is restored.

Another closure option is a temporary tarsorrhaphy (central or lateral). This can be accomplished with simple sutures [26] or cyanoacrylate glue [27] as a temporary means, and for a more permanent effect, surgical approximation of the lateral or medial gray line can be performed. I can do it. The use of lateral blepharoptosis is not recommended as it may reduce the monocular temporal visual field and prevent the eyes from closing properly.

Other Treatments

In the past, individuals with sustained loss of function (more than 90% loss on electroneurography) at two weeks have been advised to have surgical decompression within three weeks after the beginning. The most frequently cited study in favour of this strategy, however, did not use a blinded evaluation of outcome, contained a nonrandomized control group, and only reported results for a total of 34 treated patients at three distinct sites. [28]

Prognosis

The majority of Bell's palsy patients (71%), who are not receiving treatment, fully recover their motor skills within 6 months. [29, 4] All Bell's palsy patients should have made some progress by 6 months. [4] Old age, high blood pressure, diabetes mellitus, taste impairment, and total facial weakness are all indicators of poor prognosis. [30] One third of individuals may experience a partial recovery and residual effects.

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