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Review

Evidence based management of Bell's palsy

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Abstract

Bell's palsy (idiopathic facial paralysis) is caused by the acute onset of lower motor neurone weakness of the facial nerve with no detectable cause. With a lifetime risk of 1 in 60 and an annual incidence of 11–40/100,000 population, the condition resolves completely in around 71% of untreated cases. In the remainder facial nerve function will be impaired in the long term. We summarise current published articles regarding early management strategies to maximise recovery of facial nerve function and minimise long-term sequelae in the condition.

Keywords: Bell's palsy; Idiopathic facial paralysis; Corticosteroids; Antiviral therapy; Surgery; Hyperbaric oxygen; Accupuncture; Physical therapy

Introduction

The Scottish surgeon Sir Charles Bell described the facial nerve in 1821[1] and 8 years later presented 3 cases of nerve dysfunction at the Royal Society (2 were idiopathic and the third caused by a parotid tumour). Since then idiopathic facial paralysis has been termed Bell's palsy.

The condition is characterised by sudden onset, and unilateral, lower motor neurone weakness of the facial nerve with no readily identifiable cause. Other features may include retroauricular pain which may extend into the neck and occiput, impaired tolerance of noise, and ipsilateral disturbance of taste.[2] It affects 11–40/100,000 population/annum[3] and there is a lifetime risk of 1 in 60.[4] Most cases will resolve spontaneously. In 71% it will resolve completely and 84% will have near normal function.[5] The remainder will go on to have seventh nerve dysfunction which includes moderate to severe facial weakness, synkinesis, or facial contracture. Facial pain is reported in some cases but it is not universal and the aetiopathogenesis is not clear.[4,6] The severity of weakness at presentation also affects outcome.

When paralysis is incomplete, 94% of patients recover completely within 4 months of onset, but complete paresis is associated with complete recovery in only 61%.[5] A recently published series confirmed that the only general prognostic factor in the disorder was severity of weakness one week after onset.[7]

Management of Bell's palsy therefore aims to achieve complete recovery and reduce negative sequelae in cases that do not resolve spontaneously.

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Aetiology

Based on several lines of evidence, it is thought that the most likely cause is oedema of the facial nerve induced by the herpes virus.[8–12] Swelling of the facial nerve at the stylomastoid foramen has been described at operations that decompress the nerve,[13] and DNA of the herpes simplex (HSV-1)[12] and varicella zoster (VZV)[14,15] viruses has been isolated from endoneural fluid sampled in patients with the condition. Reactivation of VZV is thought to be the cause of Bell's palsy in up to 30% of cases.[16] Other suggested causes include ischaemia, autoimmune inflammatory disorders, and heredity.[2,17,18]

Diagnosis

The clinical history in Bell's palsy is of sudden onset facial weakness, which evolves rapidly. There may also be hyperacusis, altered taste sensation, and impaired lacrimation. A feeling of fullness of the ear and otalgia may precede the onset of facial weakness. Where pain is a marked feature it may imply herpes zoster infection and some of these cases will go on to develop Ramsay Hunt syndrome.

Clinical signs are unilateral weakness of the muscles of facial expression of the entire face and of the platysma in the ipsilateral neck. There is drooping of the brow and angle of the mouth, and loss of the nasolabial fold. Bell's phenomenon, which describes upward rotation of the globe on the affected side on attempted eye closure, is present in only 75% of the population. Sparing of the upper branches suggests an upper motor neurone lesion rather than Bell's palsy, and it occurs because of the crossover of motor tracts. Thorough examination of the head and neck is needed to exclude masses; a lesion in the deep lobe of the parotid may be evident only as oropharyngeal asymmetry. In areas where the condition is endemic Lyme disease should be excluded. It presents with erythema migrans of the limbs or trunk after a tick bite, and serum analysis for the antibody to *Borrelia burgdorferi* will confirm diagnosis. Bell's palsy should therefore be diagnosed only when all other diseases have been excluded.

Management

Most cases of idiopathic facial paralysis resolve spontaneously. Management therefore aims to minimise the possibility of incomplete resolution and reduce the risk of morbid sequelae, which include moderate to severe facial weakness, synkinesis, autonomic dysfunction (hemifacial spasm and crocodile tears), and contracture of the facial tissues.[4]

Measures that need to be taken immediately include protecting the cornea from trauma and drying as a result of incomplete closure of the lid and poor tear flow. Simple eye ointment is prescribed and the globe may need to be protected, particularly at night.

Operation

A Cochrane review[19] has assessed 2 reported studies that described surgical decompression for Bell's palsy. A total of 69 patients met the inclusion criteria. One study considered 403 patients, but included only 44 who were randomised into surgical and non-surgical groups.[20] The other included 25 patients who were randomised for operation or non-surgical treatment.[21] In both the seventh nerve was decompressed through a retroauricular approach. Blinding was not considered possible or attempted, and the Adour group[20] lost 6 patients during follow-up. Nine months after onset, outcome after operation was no better than after non-surgical care in both studies, and one patient in the first study had persistent vertigo and sensorineural hearing loss. Based on the available evidence, operation therefore cannot be recommended.

Acupuncture

Although several papers on the use of acupuncture for Bell's palsy have been published, few are of adequate quality to merit consideration in clinical practice. No evidence of harm has been reported. The recently updated Cochrane review concluded that poor study design and reporting (particularly randomisation, allocation concealment, and blinding) prevented valid, reliable conclusions being drawn.[22]

Physical therapy

A recently updated Cochrane review identified 65 relevant studies of which 12, which included 872 participants, were of adequate quality for consideration.[23] Methods included electrostimulation, exercises, and physical therapy with acupuncture. While meta-analysis was not possible because outcome measure were different, there was no high quality evidence to support benefit. To improve outcome in acute cases there is some low quality evidence to support the use of facial exercises, but it needs to be confirmed in appropriately designed studies.

Hyperbaric oxygen (HBO)

Few reports have been published on the use of HBO. One randomised controlled trial reported that HBO with placebo tablets gave greater benefit than prednisone given orally with sham HBO dives.[24] While randomisation was adequate and medical staff and patients were blinded to the treatment allocated, the outcome assessor was not, which introduced a high risk of bias. Reported recovery at 9 months was 95.2%

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