Bell's Palsy:

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ABSTRACT: Recent evidence suggests that viral infection is the most likely underlying cause of Bell's palsy. Rapidity of onset is a clue to the diagnosis; unilateral facial weakness or paralysis develops over 24 to 48 hours. Rule out other causes of peripheral facial paralysis, such as Ramsay Hunt syndrome and Lyme disease. Although most cases of Bell's palsy resolve without medical or surgical intervention, older patients and those with abnormal electroneurographic results are less likely to have complete recovery of facial function. Early treatment with an oral corticosteroid is likely to enhance recovery; concurrent use of acyclovir may also be helpful. Surgical decompression has been effective in patients with abnormal electroneurographic results.

In patients with Bell's palsy, the origin of the peripheral facial nerve dysfunction that results in acute unilateral weakness or paralysis is unknown. Possible underlying causes include viral infection, vascular ischemia, autoimmune inflammatory disorder, and inherited susceptibility.

The incidence of Bell's palsy peaks in the sixth decade of life. Predisposing risk factors include diabetes mellitus and pregnancy.

Here I review the clinical features that differentiate Bell's palsy from other causes of unilateral facial weakness or paralysis. I also discuss the effectiveness of the current therapeutic options.

ETIOLOGY
Mounting evidence points to viral infection as the most likely underlying cause of Bell's palsy. Serologic testing in 62 patients with facial palsy revealed that significantly more patients than controls had IgM antibodies to herpes simplex virus and varicella-zoster virus. In another study, herpes simplex virus type 1 DNA was found in the endoneural fluid from the facial nerve of 11 of 14 patients with Bell's palsy.

DIAGNOSIS
A careful history taking and physical examination can help elucidate the cause of the facial paralysis. Note the following in the history:
Date of onset.
Rapidity of onset.
Associated symptoms.
Systemic diseases.
Symptoms. The symptoms of Bell's palsy develop rapidly over a period of 24 to 48 hours. The patient may complain of a sensation of heaviness of the face, but sensory loss should not be evident. Taste is lost over the anterior two thirds of the tongue on the ipsilateral side. Lacrimation in the ipsilateral eye may be reduced. Postauricular pain occurs in about 50% of patients.

To detect unilateral weakness, ask the patient to wrinkle the forehead, wrinkle the nose, close the eyes tightly, blow out the cheeks, purse the lips, grin, and depress the lower lip (Figure). In addition, check the corneal reflex, palpate the parotid glands for tumors, and test the patient's hearing.

Differential diagnosis. Other causes of peripheral facial paralysis must be ruled out (Table 1). Examine the ear for signs of infection and vesicles. Auricular vesicles commonly accompany the acute facial palsy caused by varicella-zoster virus reactivation (Ramsay Hunt syndrome). However, in as many as one third of patients with Ramsay Hunt syndrome, auricular herpetic eruptions develop after the onset of facial palsy. Bell's palsy is frequently misdiagnosed in such patients. Also bear in mind that peripheral facial palsy caused by varicella-zoster virus infection can occur without eruption of vesicles (zoster sine herpete).

A recent history of a rash or a tick bite warrants investigation. In areas where Lyme disease is endemic, the prevalence of peripheral facial palsy attributable to Lyme borreliosis approaches 25%. If the patient lives in or has visited an endemic area, serologic testing for IgM antibodies to Borrelia burgdorferi can be helpful.

Diagnostic studies. If the patient presents with unilateral sensorineural hearing loss and you
suspect a tumor—such as an acoustic or facial neuroma—MRI with an acoustic protocol is recommended. CT is appropriate for patients who have a parotid mass. Electromyography (EMG) can be helpful in patients with severe dysfunction or paralysis.

CONSERVATIVE MANAGEMENT

Most patients recover complete facial function without treatment. A study of 1011 patients with acute, unilateral, idiopathic facial paresis showed that 84% of the patients recovered normal or near-normal function without treatment, while 16% had permanently diminished function. Older patients and those with abnormal electroneurographic results are less likely to recover completely. Eye care is essential for patients with Bell's palsy to prevent ophthalmic complications. Artificial tears and eye lubricants can be used during the day, and viscous ointment and a patch can be applied at bedtime.

DRUG THERAPY AND SURGERY

Many treatments—including corticosteroids, antivirals, and surgical decompression—have been used in patients with Bell's palsy. Studies that have evaluated the effectiveness of these treatments must be interpreted with caution because most patients recover without therapy. In addition, the lack of consistency in the reporting of facial nerve recovery can make it difficult to compare results of trials. The American Academy of Otolaryngology-Head and Neck Surgery recommends that the House-Brackmann facial nerve grading system be used to report the degree of facial nerve recovery (Table 2).

Corticosteroids. The use of corticosteroids to treat Bell's palsy was first proposed in the 1950s. A meta-analysis showed that corticosteroid therapy improves the likelihood of complete facial recovery by 17%.

Corticosteroids are the mainstay of treatment. The recommended regimen is 60 mg/d of prednisone for 4 days, followed by a tapering dosage over the next 10 days. Start treatment within 5 days of the onset of symptoms.

Acyclovir. The focus on a viral origin of Bell's palsy has prompted the study of antiviral agents in the treatment of idiopathic facial paresis. A double-blind, randomized, controlled trial compared the effectiveness of combination acyclovir-prednisone therapy with that of prednisone alone. The study included 99 patients with Bell's palsy; 53 received acyclovir (400 mg 5 times daily for 10 days) and prednisone, and 46 received placebo and prednisone. All patients were given the same dosage of oral prednisone: 30 mg twice daily for 5 days with subsequent tapering during the following 5 days. The results of the study showed that acyclovir-prednisone is superior to prednisone alone in the treatment of Bell's palsy. Patients who received acyclovir-prednisone had improved return of voluntary movement compared with those who received prednisone alone.

Surgical decompression. This operation was a widely accepted intervention for Bell's palsy in the 1930s. Recent evidence that supports the efficacy of surgical decompression has been lacking. In addition, the procedure is associated with serious complications, including permanent unilateral deafness. However, in one study, patients with Bell's palsy who displayed significant abnormalities on electroneurography and EMG clearly benefited from surgical decompression.

Recommendations from the American Academy of Neurology. To determine whether corticosteroids, acyclovir, and surgical decompression are effective in improving facial functional outcomes in patients with Bell's palsy, the Quality Standards Subcommittee of the American Academy of Neurology (AAN) conducted a systematic review and analysis of the literature. The AAN makes the following recommendations based on the strongest available evidence:

Early treatment with oral corticosteroids is probably effective in improving recovery of facial function.

Early treatment with acyclovir in combination with prednisone is possibly effective in improving recovery of facial function.

There is insufficient evidence to recommend facial nerve decompression.

PROGNOSIS

The severity of the palsy at 2 weeks after symptoms occur indicates the likelihood of long-term sequelae. Amelioration of symptoms 1 to 2 weeks after their onset is a good predictor of a complete recovery.
In 85% of patients with Bell's palsy, facial function returns to normal within 3 weeks; in the remaining 15%, normal function generally returns within 3 to 5 months. The most common complications are ophthalmic; these result from corneal drying and abrasions.

References:


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