

Management of Bell's Palsy: A Report of 2 Cases

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SOMMAIRE

La paralysie de Bell est une neuropathie du nerf facial périphérique, qui peut être due à un traumatisme, une compression, une infection, une inflammation ou un trouble métabolique; elle peut aussi être idiopathique. Bien que le VIH, le virus Epstein-Barr et le virus de l'hépatite B soient de présumés agents initiateurs, le virus de l'herpès simplex est l'organisme le plus souvent mis en cause. Ce rapport décrit 2 cas de paralysie de Bell chez des enfants, qui ont été traités par des antiviraux. Un rétablissement complet a été observé chez les 2 patients en moins de 28 jours. Un an plus tard, aucune récurrence n'a été observée et les 2 patients avaient retrouvé des mouvements faciaux normaux. Le diagnostic différentiel est essentiel pour guider le plan de traitement de la paralysie de Bell. Enfin, une attention particulière doit être portée à la prescription de médicaments chez les enfants, car certaines substances peuvent causer d'importants effets secondaires.

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Bell's palsy is a neuropathy of the peripheral seventh cranial nerve, usually resulting from traumatic, compressive, infective, inflammatory or metabolic abnormalities. However, in many cases no etiology is identified and the eventual diagnosis is idiopathic.¹

The condition is named after Dr. Charles Bell, who, in 1821, described complete facial paralysis after injury of the stylomastoid foramen.² Bell's palsy can be defined as acute peripheral facial nerve palsy usually of unknown cause.³ It is typically unilateral and can be complete or partial.⁴ Although there is agreement on the definition, there is no consensus regarding the etiology, diagnostic approach or management of this enigmatic condition.⁵

Bell's palsy is generally a unilateral disease, affecting both sides of the face equally.^{1,5} The pathogenesis of Bell's palsy remains controversial. Acute inflammation and edema of the facial nerve are thought to lead to entrapment of the

nerve in the bony canal (especially in the labyrinthine segment), which leads to compression and ischemia.^{6,7}

An inflammatory process surrounds the nerve fibres. Many viruses, such as HIV,⁸ Epstein-Barr virus⁹ and hepatitis B virus¹⁰ have been suspected in initiating this inflammation, but herpes simplex virus (HSV) is the most frequently implicated.^{11,12}

Increasing evidence is associating HSV with Bell's palsy, and, in time, Bell's palsy may well be reclassified as an HSV mononeuritis of the facial nerve, although designation of this causative agent does not exclude the possibility that other causes may exist or negate the role that entrapment plays in the degeneration of the nerve. According to one hypothesis, HSV, dormant in the geniculate ganglion cells, becomes reactivated and replicates, causing inflammation, primarily in the geniculate ganglion and in the labyrinthine segment of the facial nerve.



Figure 1: Facial aspect of patient in case 1, showing Bell's palsy on the left side. (a) Inability to smile. (b) Incomplete left eye closure. (c) No movement in the upper left eyebrow. (d) Absence of movement in the left portion of the labial orbicular muscle.



Figure 2: Facial aspect of patient in case 1 after recovery of facial movement. (a) Normal, symmetric smile. (b) Complete closure of the left eye. (c) Normal left eyebrow movement. (d) Normal movement of labial orbicular muscle.

These inflammatory events (evident on magnetic resonance imaging) result in entrapment and ischemia, which lead to neurapraxia or degeneration of the facial nerve distal to the meatal foramen.¹³

HSV has been identified in the endoneural fluid, posterior auricular muscle and saliva by polymerase chain reaction in patients with Bell's palsy.¹¹ Increased capillary permeability leads to exudation of fluid, edema and compression of the microcirculation of the nerve, which may be responsible for the vascular ischemia.⁴

Patients generally experience rapid onset of unilateral facial palsy and often describe numbness or stiffness, although no actual sensory loss occurs.^{5,7} Affected patients are usually unable to close their eyes. Facial appearance becomes asymmetric, and saliva dribbles down the angle of the mouth. Depending on the site of the lesion, some patients may complain of noise intolerance or loss of taste sensation.^{1,5} A focused physical examination can help rule out other conditions and narrow the differential diagnosis.⁷

Treatment of Bell's palsy is controversial, because as many as two-thirds of patients recover spontaneously. Corticosteroids alone or associated with antiviral agents have been recommended.⁷ Adour¹⁴ reported that patients with Bell's palsy treated with acyclovir and prednisone experience a more favourable recovery and less neural degeneration than patients treated with placebo plus prednisone. The favourable response to the treatment of Bell's palsy with acyclovir-prednisone supports the theory that reactivated HSV causes a neuritis.

Case Reports

Case 1

A 12-year-old girl with left hemifacial palsy over the past 15 days was referred to the oral and maxillofacial surgery department at Hospital de Base, Bauru, São Paulo, Brazil, by her pediatrician. She denied any type of facial trauma or systemic alteration. Although no diagnosis was made by the doctor, an antibiotic was prescribed and she had been taking



Figure 3: Signs of hemifacial Bell's palsy in case 2. (a) Incomplete smile. (b) Inability to close the right eye. (c) Right eyebrow movement compromised. (d) Incomplete right labial movement.

it for the last 7 days without any improvement. Her mother reported that the girl had been at a school camp for 2 weeks before the first signs of palsy, severe headache, dizziness and fever. A friend of the girl had exhibited the same signs and symptoms after the camping trip.

Clinical examination revealed restricted movement of both the superior and inferior lips and in the left superior palpebra and left eyebrow region, associated with moderate pain around the left ear (Fig. 1). The patient was diagnosed with hemifacial Bell's palsy caused by a viral infection, although no HSV or herpes zoster lesions were present. She was prescribed acyclovir, 200 mg, every 4 hours, vitamin B complex every 12 hours and artificial tears during the day. She was advised to start facial physiotherapy exercises combined with warm water compresses and to keep the left eye closed with tape or a sleep mask during the night to avoid conjunctive dryness. After 28 days of medication, the facial palsy disappeared. The patient was seen weekly during the first month, then monthly for 1 year, and no signs of recurrence were noted (Fig. 2).

Case 2

An 11-year-old girl, who was the roommate of the patient described above during the school camping vacation, exhibited left hemifacial palsy that had been detected 6 days before her first appointment. She described severe pain about 10 mm anterior to her left ear, just above the facial nerve trajectory. During facial examination, the patient demonstrated an inability to close the right eye, to corrugate the left eyebrow or to move her lips (Fig. 3). The patient was prescribed acyclovir, 200 mg every 4 hours and vitamin B complex every 12 hours for 28 days and artificial tears during the day. The patient recovered normal facial function, and after 1 year of follow-up, facial movements were satisfactory.

Discussion

Many pathologies can be included in the differential diagnosis of Bell's palsy: unilateral central facial weakness, Ramsay Hunt syndrome, Lyme neuroborreliosis, tumours, diabetes mellitus, sarcoidosis, weight loss, visual changes, vertigo and weakness or numbness.⁷

Diagnosis of Bell's palsy depends on clinical signs, symptoms and evaluation to exclude other possible causes of facial paralysis.

Laboratory investigations and imaging are carried out to detect the origin of the paralysis: lumbar puncture, IgG and IgM antibody tests and cerebral spinal fluid cell count to detect intracranial pressure and inflammation; magnetic resonance imaging and computed tomography to locate an intracranial lesion or hemorrhage; Lyme titre test to rule out Lyme disease; and acetylcholine-receptor antibody test for myasthenia gravis.¹⁵

IgG and IgM antibody titres were not available for our patients because of the cost and lack of resources. Because no specific laboratory test confirms the diagnosis of Bell's palsy, its assessment remains clinical. Thus, even with IgG and IgM antibody tests, our clinical approach would have been the same. It is important to emphasize the fact that we followed these patients weekly and noted that they were recovering very well.

Patients should be advised to use artificial tears to keep the eyes moist and prevent exposure keratitis. During the day, sunglasses are indicated, and dirty, noxious fumes should be avoided. During sleep, an ophthalmic ointment should be used.⁷

Our patients enjoyed complete recovery after 4 weeks, but clinicians should be aware of possible morbidities. For example, some patients experience lasting facial weakness. Factors associated with a poor prognosis include advanced age, hypertension and impairment of taste and pain other

than in the ear. Bell's palsy does not usually recur; however, if it does, particularly bilaterally, further investigation is required to rule out other causes of facial paralysis such as myasthenia gravis, sarcoidosis and lymphoma.³

Patients who have persistent clinical signs without improvement in facial paresis after 4 weeks, involvement of other cranial nerves or a second episode of palsy require further investigation.⁷ A detailed history and thorough clinical examination should be carried out in those patients. Early recognition of signs and symptoms inconsistent with Bell's palsy is important to avoid misdiagnosis. If the patient does not recover within the expected timeframe, imaging must be performed, such as computed tomography or magnetic resonance imaging. Current imaging techniques may reveal occult lesions of the temporal bone, internal acoustic canal or cerebellopontine angle, for example.¹⁶

Many clinical trials have evaluated acyclovir with or without prednisone for the treatment of Bell's palsy: Adour⁴ and De Diego,¹⁷ for example, using a facial paralysis recovery profile. Studies evaluating the efficacy of antiviral agents for the treatment of Bell's palsy show conflicting results. As each trial has used different treatment modalities, facial nerve recovery scales and doses of acyclovir, and some have involved only a small number of patients, it is difficult to compare their results and verify the effectiveness of acyclovir.³

Although many studies have examined the usefulness of corticosteroids in the treatment of Bell's palsy, they have been limited by small sample size and lack of randomization, controls and blinding. Considerable controversy remains over the use of steroids for Bell's palsy in adults, and there is even less evidence for using steroids to treat Bell's palsy in children.¹⁸ Children are more vulnerable to the side effects of corticosteroids, particularly their effect on growth, immunity and adrenal suppression. A side effect of corticosteroids unique to children is growth suppression, which can be reduced by prescribing the medication on alternate days.¹⁹

Boulloche and others²⁰ carried out a retrospective review of 40 patients aged 1–16 years with acute facial nerve palsy. Patients who received steroids did not have better outcomes than those who did not receive steroids; similar findings were revealed by Dhiravibulya.²¹ Given the natural history of spontaneous recovery in most pediatric patients, steroid therapy is currently not indicated.¹⁸ In the cases described above, we preferred to avoid steroids and prescribe antiviral agents associated with vitamin B.

Differential diagnosis is essential to guide treatment in Bell's palsy. Although its etiology is still unknown, viral infection, vascular ischemia and autoimmune disorders have all been postulated as possible mechanisms. Special attention should be given to children with respect to steroid prescription. Dentists, especially those who deal with children, should be aware of this disorder. ♦

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