Man With Facial Weakness Hyperbaric Oxygen and other treatments

Michael Vincent Anthony, MS, PA-C

Introduction

A 30-year-old man presented to the emergency department (ED) with limited mobility on the left side of his face and none at all on the right. The patient was also experiencing hyperacusis, a change in his sense of taste, and speech impairment.

This was his third visit to the ED. During his first visit two days earlier, he complained of facial weakness on the right side of his face subsequent to recent recovery from a viral upper respiratory infection. When a physical examination revealed no unusual findings, the patient was given a prescription for acyclovir and discharged with a diagnosis of Bell's palsy.

The patient returned to the ED the next day reporting that the facial weakness had progressed to the left side of his face. Again, physical examination was unremarkable. It was determined that his symptoms were caused by fatigue, and the patient was instructed to get some rest.

During the most recent visit, the patient was examined by the attending neurologist. The differential diagnosis included sarcoidosis and Guillain-Barré syndrome. Blood workups performed included complete blood cell count, HIV screening, and type-specific and antinuclear antibody testing; spinal fluid and urine were also tested. Both posteroanterior and lateral chest x-rays were performed, as was magnetic resonance imaging (MRI) of the head. All laboratory test results and chest x-rays were within normal limits. MRI revealed questionable swelling of cranial nerve VII on the left side of the face; cranial nerve VII on the right was unremarkable.

Shortly after the evaluation, the patient received a 30-minute infusion of methylprednisolone and an antacid (to combat potential gastric effects). Artificial tears were administered to prevent dehydration of the eyes. Three hours later, methylprednisolone (in conjunction with an antacid) was again administered.

The patient was sent home after his 10-hour stay in the ED. He received a prescription for prednisone to be taken for five days and then tapered for one week. He was advised to continue with the acyclovir regimen. A follow-up appointment was made with the consulting neurologist for three weeks following discharge. The diagnosis was bilateral facial paralysis of unknown etiology (ie, bilateral Bell's palsy).

Discussion

Known causes of facial paralysis include stroke, trauma (eg, laceration, fractures), neoplasm (metastatic lesions, parotid tumors), and congenital defects.[1,2] Bell's palsy, however, is an acute idiopathic condition involving damage to the seventh cranial facial nerve. It is the most prevalent form of facial paralysis[1] and usually presents unilaterally. Traditionally thought to be a diagnosis of exclusion, unilateral Bell's palsy can be positively identified based on clinical assessment, without performing expensive tests.[3]

Simultaneous bilateral facial palsies (SBFPs) are extremely uncommon and may indicate a more serious disease. In these cases, it is especially important to immediately rule out other causes.[4] See Table 1[1,5-7] (page 57) for the common differential diagnosis and diagnostic tools.
Some unusual presentations associated with SBFP include HIV[8] and intracranial hypertension.[9] In these cases, research has shown that the etiology was an unknown infectious process.

**Classic Presentation**

Typically, the onset of facial paralysis in patients with Bell's palsy is sudden. Paralysis may be preceded by pain in front of or behind the ears that can last until after paralysis becomes complete, usually between three and 72 hours. Patients may complain that the face feels stiff or pulled to one side but will have no demonstrable sensory loss. Hyperacusis (ie, abnormal acuteness of hearing due to increased irritability of the sensory neural mechanism) on the affected side, drooling, excessive tearing, and a change in the sense of taste can accompany facial paralysis.[2]

Bell's palsy affects approximately 25 persons per 100,000. Although incidence increases with age, peak incidence occurs from ages 10 to 40 years.[1,3] Men are affected at the same rate as women. At risk are those who have had influenza, a cold, or some other upper respiratory infection; persons with hypertension or diabetes; and pregnant women.[10-14] The right side of the face is affected 63% of the time.[2] Human herpesvirus 1 has been implicated as the likely cause of Bell's palsy.[15]

Between 60% and 80% of patients will experience a complete resolution of symptoms, while the remaining cases will have some residual -- or even permanent -- effects.[1,14] Indicators of a favorable prognosis include incomplete paralysis, younger age, and electrodiagnostic tests showing normal nerve excitability.[16]

**Treatment**

Although most Bell's palsy patients do not require specific treatment, a common regimen prescribed for facial paralysis is physical therapy, which can be used alone or in conjunction with surgery. The most serious complication associated with facial paralysis is ulceration of the cornea. This can be averted by using an artificial tear solution or applying a lubricating ointment.[2]

Antiviral agents (if infectious processes are suspected to be the cause of paralysis) and corticosteroids are usually prescribed to treat Bell's palsy. Research has shown that patients who receive such treatment within three days of symptom onset experience higher recovery rates than those who start therapy after four or more days.[17] Steroid treatments have been shown to be of little benefit when initiated more than four days after the onset of paralysis. While research has shown that patients given methylcobalamin with concomitant steroid therapy to treat Bell's palsy experience greater improvement than those treated with steroids alone,[18] use of this combination is not yet widespread. Other nonsurgical treatments to combat the effects of facial paralysis include mime therapy, in which patients measure progress by the symmetry of their smile and judgment of their smile by others[19]; myofeedback[19,20]; Hyperbaric Oxygen Treatments, which have been shown to be more effective than treatment with prednisone[21]; and the use of high-voltage electrical muscle stimulation and chiropractic manipulation.[22]

Surgical intervention to reanimate the face is typically reserved for those cases in which the lesion in the facial nerve has been located.[23] Surgery types include cross facial nerve grafts,[24] rectus abdominis muscle transfers,[25] and nerve decompression.[26] Once general reanimation has been established, other therapies can be used to regain control of movement.

**Psychosocial Implications**
To choose the most appropriate treatment options, clinicians must take into account what their patients consider an acceptable level of functionality, as well as patients' values about their own health and social/moral structures.[27]

The psychosocial impact of facial paralysis can trigger depression, social withdrawal, or both. According to Neely and Neufeld,[28] the importance of the face as a symbol of personal identity and a tool for both verbal and nonverbal communication is often overlooked as part of the psychosocial impact of facial disfigurement. Results from their study show that the quality of patients' smiles influenced how they were perceived by others (see "One Patient's Experience With Bilateral Bell's Palsy," page 58).

Conclusion

Clinical assessment is sufficient to diagnose cases of unilateral Bell's palsy. However, when a patient presents with a complaint of SBFP, a complete and comprehensive examination must be performed. The distinctions among extracranial, intratemporal, infectious, traumatic, and idiopathic origin will dictate what type of treatment is necessary.

A conservative, all-inclusive approach is best. Treatment, whether with physical therapy, drugs, or surgery, should be aimed at what the patient feels is an appropriate recovery goal. Because the face is such an important component of nonverbal communication, the more fully patients can return to their original physical state, the better.

Tables

Table 1. [1,5-7] Differential Diagnosis for Bell's Palsy

<table>
<thead>
<tr>
<th>Disease</th>
<th>Symptoms</th>
<th>Diagnostic tools</th>
</tr>
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<tbody>
<tr>
<td>Melkerson-Rosenthal syndrome</td>
<td>Congenitally fissured tongue, orofacial edema, facial palsy</td>
<td></td>
</tr>
<tr>
<td>Mobius' syndrome</td>
<td>Congenital facial diplegia, ophthalmoplegia</td>
<td></td>
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<tr>
<td>Guillain-Barré syndrome</td>
<td>Ascending motor weakness, usually beginning in legs and possibly leading to respiratory muscle paralysis</td>
<td>Nerve conduction velocity; electromyography</td>
</tr>
<tr>
<td>Facioscapulohumeral muscular dystrophy</td>
<td>Progressive weakness in the face, neck, upper torso, and upper arms</td>
<td>Serum creatine kinase; electromyography</td>
</tr>
<tr>
<td>Myasthenia gravis</td>
<td>Weakness and fatigue, particularly of the extraocular, pharyngeal, facial, cervical, and respiratory musculature</td>
<td>Computed tomography; electrodagnostic studies</td>
</tr>
<tr>
<td>Sarcoidosis</td>
<td>Shortness of breath, cough, skin and ocular lesions; weakness, fatigue, fever, malaise</td>
<td>Imaging studies; bronchoscopy; lymph node biopsy</td>
</tr>
</tbody>
</table>

References


Sidebar: One patient's experience with bilateral bell's palsy
My face was completely paralyzed for more than two weeks after I received a diagnosis of bilateral Bell's palsy. Over the course of more than two years, I gradually regained animation of about 95% of my face, with the greatest gains obtained within the first eight months.

One of the challenges I encountered during that time was not being able to hold anything in my mouth. I had to drink through a straw while using my fingers to hold my lips in place around it and eat only foods that could be broken into small pieces. It took only a couple of bites into my own flesh for me to figure out how to keep my lips and cheeks out of the way while chewing.

Talking was another challenge. I could not pronounce the consonants B, F, M, P, V, W, or Y. After some practice, I resolved this problem by using the fingers on my right hand to manipulate my lips.

But the hardest obstacle to overcome was the inability to express myself nonverbally. It is difficult to joke with people when your face is paralyzed (because you look so "serious" all the time) or to show interest in what they are saying (because all you can do is give expressionless nods).

My condition affected many aspects of ordinary living. Any sound as loud as a dog's bark (including a dog's bark) seemed to pierce my head like a gunshot. Tear replacement drops and use of my hands to blink took care of my dry eyes. My nostrils were so narrow due to the lack of facial tone and gravity that I had to continuously breathe through my mouth. Many mornings I awoke with a dry, sore throat -- something that quickly became tiresome.

I feel fortunate to have recovered as completely as I have. I hope that by reading my story, clinicians can prepare their patients with Bell's palsy for what lies ahead and tell them about the "tricks" that can be used to assist them with everyday living.

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