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CASE STUDY

Simultaneous bilateral facial palsy

Gustavo M. Torres,[a] Alano N. Barcellos,[a] Marco A.R. Santos,[a] Marconi T. Fonseca,[a] Eustáquio C. Santos,[b] and Rubiana F. Sousa[c,*]

[a]*Servicio de ORL, Hospital Socor, Belo Horizonte, MG, Brazil*
[b]*Servicio de Neurología, Hospital Socor, Belo Horizonte, MG, Brazil*
[c]*Student of Medicine, FASEH, Vespasiano, MG, Brazil*

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KEYWORDS
Simultaneous bilateral facial palsy;
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Abstract
Simultaneous bilateral facial palsy (SBFP) is an uncommon disorder that usually results from a systemic disease, with only a few cases diagnosed as Bell's Palsy. The most common causes of SBFP are head injuries, Bell's Palsy, Lyme disease, Guillain-Barré syndrome, sarcoidosis, and meningitis. We present a case of SBFP.

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PALABRAS CLAVE
Parálisis facial bilateral simultánea;
Síndrome de Guillain-Barré;
Parálisis de Bell

Parálisis facial bilateral simultánea

Resumen
La parálisis facial bilateral simultánea (PFBS) es una rara entidad clínica que generalmente surge como una manifestación de enfermedades de carácter sistémico, que se diagnostica como parálisis idiopática de Bell en una pequeña proporción de casos. Entre las causas más comunes de PFBS están los traumatismos craneoencefálicos, parálisis de Bell, enfermedad de Lyme, síndrome de Guillain-Barré, sarcoidosis y meningitis bacteriana. Presentamos el caso de una paciente con cuadro de parálisis de Bell bilateral y simultánea.

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*Corresponding author.
E-mail address: rubiana.sousa@gmail.com (R. Ferreira Sousa).

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Introduction

Facial palsies are classically divided into peripheral and central: they are also classified as unilateral, recurrent ipsilateral, recurrent alternating, and bilateral simultaneous. Facial palsy is a relatively common diagnosis in otolaryngology practice. Simultaneous bilateral facial palsy is much rarer and occurs in 0.3%2%[-3] of cases of facial palsy; it is defined as palsy affecting both hemifaces during a period no longer than 2 weeks.[4]

Case Report

We report the case of a 43-year-old female patient admitted to the emergency room with headache, neck stiffness, neck pain, and vomiting lasting for 3 days. Physical examination on admission revealed cervicobrachial hypoaesthesia on the right side and grade VI peripheral facial palsy on the House-Brackmann scale on the left side. The computerized tomography of the skull, chest radiograph, blood count, and other biochemical tests were normal. In the emergency room a puncture was performed to extract fluid, which turned out to be clear, with mononuclear leukocytes, 1/µL; erythrocytes, 33/µL; glucose, 55 mg/dL; Gram-negative stain; positive Pandy test with 82 mg/dL of proteins. A digital cerebral angiography was also conducted and was normal. She was admitted and treated with prednisone 60 mg/day and aciclovir 2000 mg/day divided into 5 doses.

After 24 hours of hospitalization, she suffered sudden-onset facial palsy in the right hemiface classified as grade III on the House-Brackmann scale. A cranial MRI was performed, which showed only faint, scattered sub-cortical foci of T2 hypersignal, of non-specific appearance. The serologies for the virus of herpes, human immunodeficiency, Epstein-Barr, cytomegalovirus, Toxoplasma, Lyme disease, and VDRL were negative. The serologies for herpes virus and Lyme disease analyzed in the CSF were also negative. The otolaryngology review presented no other abnormalities. The electroneuromyography on the twelfth day of right-side facial palsy showed acute denervation of the frontal muscle on the left side and normal conduction in the nerves of the upper limbs.

Faced with the unfavourable electroneuromyography outcome, the lack of improvement in the grade VI palsy on the left and the lack of a precise aetiology for her condition, we chose to perform surgical decompression of the left pair VII on the fourteenth day after admission. We conducted a closed mastoidectomy with decompression of the geniculate ganglion and the labyrinthine, mastoid, and tympanic segments of the facial nerve on the left side, which presented intense oedema. She was discharged 2 weeks after surgery. She presented a slow and gradual improvement of the left facial paralysis, and continued with grade III paresis on the House-Brackmann scale one year after surgery. The situation of the right hemiface remained unchanged (Figures 1 and 2).

Discussion

Unlike unilateral facial palsy, simultaneous bilateral facial palsy (SBFP) is considered idiopathic in only 20% of cases and usually indicates a more serious alteration.[3] The most important causes of SBFP are brain trauma, infectious diseases (infectious mononucleosis, syphilis, bilateral otitis media, herpes zoster, Lyme disease, meningitis), neurological diseases (multiple sclerosis, neoplasias, or vascular accidents), disorders of undetermined origin (Guillain-Barré syndrome, sarcoidosis, Melkerson-Rosenthal syndrome, leukaemia, Bell's palsy).

There are few case series of SBFP in the literature. In one series of 43 patients, Keane[5] marked Bell's palsy as a common cause, followed by Guillain-Barré syndrome (GBS).

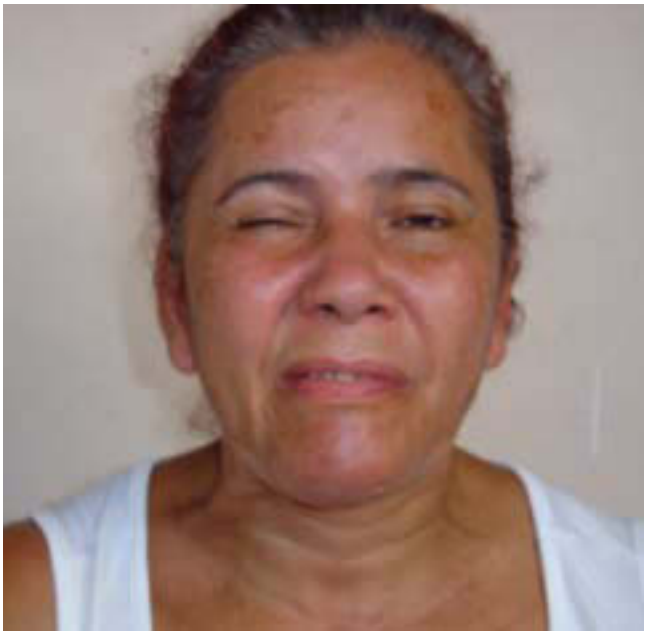

Figure 1 Six months after surgery.

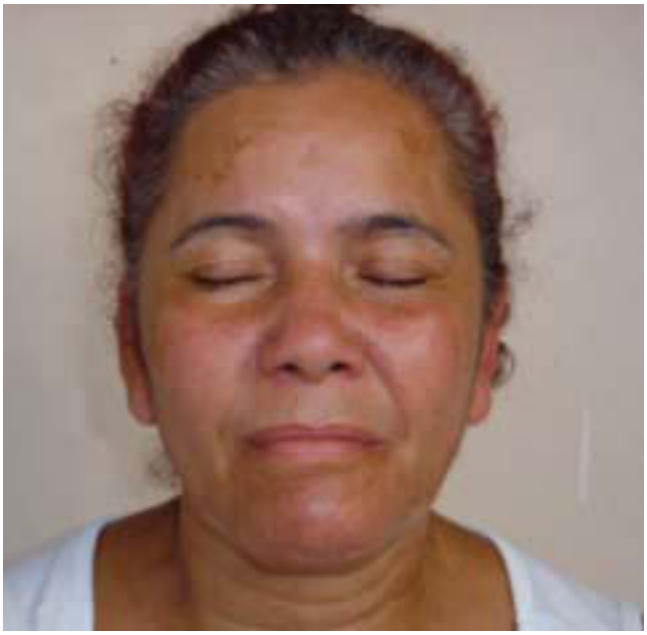

Figure 2 Six months after surgery.

In another series of 24 patients, Wormald et al[6] pointed to Bell's idiopathic palsy as the most common cause, followed by osteosclerosis, craniotubular dysplasia with autosomal recessive transmission, and an increased distribution in a southern African community. In 6 cases of SBFP, May[3] found Guillain-Barré syndrome in 4 patients, leukaemia in 2, and 1 case of pseudo-bulbar palsy. Morales et al[7], in a report of 4 cases, found the following aetiologies for SBFP: leukaemic infiltration, Bell's idiopathic palsy, sarcoidosis and demyelinating mixed polyneuropathy (probably a form of Guillain-Barré syndrome, according to the authors).

In the case we report, we immediately began treatment with prednisone and aciclovir, in line with the protocol in place at our department. This occurred because the initial clinical presentation, sudden palsy, initially unilateral on the left side, without apparent cause and with normal tests, indicated that it was a case of Bell's palsy.

The CSF puncture was performed on admission of the patient because she presented stiffness of the neck and cervicalgia. Examination of the fluid showed an increased number of erythrocytes and a positive Pandy test. This test detects an increase in proteins and is positive in infectious or autoimmune processes of the central nervous system, which may show a false positive when there is blood in the fluid. In this case, the increased number of erythrocytes in the fluid was produced by the trauma of the lumbar puncture, since the digital angiography ruled out intracranial haemorrhage. Therefore, we cannot say that the positive Pandy test actually indicates a case of pathology. Morales et al[7], in their review of cases, mention that in the absence of any cause indicative of SBFP, a CSF puncture should be included in the protocol.

Due to the onset of the contralateral facial paresis and the lack of improvement in the paralysis on the left, the patient underwent electroneuromyography (ENM) of the muscles of the face and upper and lower limbs. This examination revealed bilateral facial palsy, with signs of acute denervation in the left frontal muscle and reduction of muscle recruitment in all the muscles of the face. We found no prolongation of F waves, which are the first changes in GBS. The rest of the examination of sensory and motor conduction in the upper and lower limbs was normal.

The recommendation of surgical decompression of the facial nerve on the left was based on the lack of improvement in the grade VI paralysis in this hemiface and on the ENM results. This result showed, after 12 days of evolution, a marked reduction in motor function on the left side, in addition to the lack of a clear aetiology and diagnosis for this alteration.

Garcia Callejo et al[8] focus on the fact that GBS cannot be ruled out as the cause of SBFP, although this is would be an isolated manifestation of this syndrome. We discarded GBS as a diagnostic hypothesis as the patient did not present in the ENM the most common alterations of this ailment: prolonged distal latencies with a reduction in the amplitude of the compound muscle action potential, decrease in the conduction velocity and abnormalities of the F waves. García Callejo et al[8] found these alterations in all their patients with SBFP secondary to GBS.

We believe that the patient presented an episode of bilateral Bell's palsy with unfavourable evolution despite all therapeutic measures taken.

Conclusions

We believe that in the absence of a prior history explaining the SBFP, the investigation of its possible causes requires a detailed clinical history with laboratory tests, imaging studies, and CSF analysis.

Conflict of interests

The authors have indicated there is no conflict of interest.

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