BILATERAL SIMULTANEOUS LOWER MOTOR NEURON FACIAL NERVE PALSY DUE TO GUILLAIN-BARRE SYNDROME

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Date Received: May 18, 2018
Date Revised: January 21, 2019
Date Accepted: January 29, 2019

ABSTRACT

Bilateral facial nerve palsy (FNP) is a rare, diagnostically perplexing clinical presentation. While idiopathic Bell's palsy is the most frequent etiology of unilateral FNP, multiple etiologies have been implicated in facial diplegia including infectious, neurologic, neoplastic, traumatic or metabolic causes. We report here, a 64-years-old male, who presented with simultaneous bilateral FNP that was attributed to Guillain-Barre syndrome. He made a noticeable recovery after conservative management.

Key Words: Bilateral facial nerve palsy, Guillain-Barre syndrome

This case report may be cited as: **Manzoor F, Ayaz SB, Anjum N. Bilateral simultaneous lower motor neuron facial nerve palsy due to Guillain-Barre syndrome. J Postgrad Med Inst 2019; 33(1): 79-82.**

INTRODUCTION

Facial nerve palsy (FNP) is generally a unilateral entity and it is very uncommon to find a bilateral facial nerve presentation. According to the previous literature, less than 2% of FNP cases present as bilateral involvement[1]. One person from a population of 5,000,000 people suffers from bilateral FNP (BFNP)[1]. BFNP is considered to have a simultaneous onset if one side is involved within thirty days of the involvement of the contralateral side[2]. It is generally a part of the multifarious symptomatology of a systemic disease. Frequent reasons for BFNP incorporate Guillain-Barre syndrome (GBS), sarcoidosis, syphilis, leukemia, Lyme's disease, infectious mononucleosis, and trauma[2,3]. Approximately, in 20% of BFNP cases, an inciting pathology cannot be traced (Bell's palsy)[3]. We present here, report of a case of BFNP due to GBS.

CASE REPORT

A 64-years-old male presented to the emergency department with complaints of acute slurring of speech, bilateral facial weakness and drooling of saliva. He had altered sensation of taste and was unable to close his eyes. The patient was otherwise ambulant and had no peripheral limb weakness. He did not give any history of fever, headache, joint pain, neck rigidity, tinnitus or visual disturbances. There was no history of recent travel abroad, trauma, rashes or exposure to tick bites. Further history revealed that he had an episode of stroke two years ago and had been treated for pulmonary tuberculosis ten years ago. He was an ex-smoker, non-addict and businessman by profession. History of blood transfusion or sexual promiscuity was also absent.

On examination, the patient was in no obvious distress, sitting comfortably on couch with stable vital signs. Otorhinolaryngological examination was unremarkable. Neurological examination revealed BFNP with Grade IV (House-Brackmann classification)[4] facial weakness on right side and Grade V facial weakness on the left side (Figure 1A and 1B). His taste sensation was altered, Bell's phenomenon was present bilaterally and he was unable to purse his lips or smile (Figure 1). Examination of the other cranial nerves was unremarkable. Muscle power, tone, and bulk were normal in all limbs. All deep tendon reflexes were normal and plantars were down-going. Sensory examination did not reveal any superficial or deep sensory loss or hyperesthesia on the face. There were no cerebellar signs. Auscultation of the chest revealed bilaterally equal breath sounds and normal heart sounds. Abdomen was soft and non-tender with no organomegaly and normal bowel sounds.

His complete blood count, blood glucose profile, urine routine examination, renal and liver function tests, serum angiotensin-converting enzyme and serum calcium level were within normal limits. The erythrocyte sedimentation rate (ESR) was 15 mm/hour (normal: <10 mm/hour). His Venereal Disease Research Laboratory test and antibodies against retrovirus, hepatitis B, hepatitis C, human immunodeficiency virus (HIV), varicel-

Figure 1: Bilateral facial weakness and Bell's sign

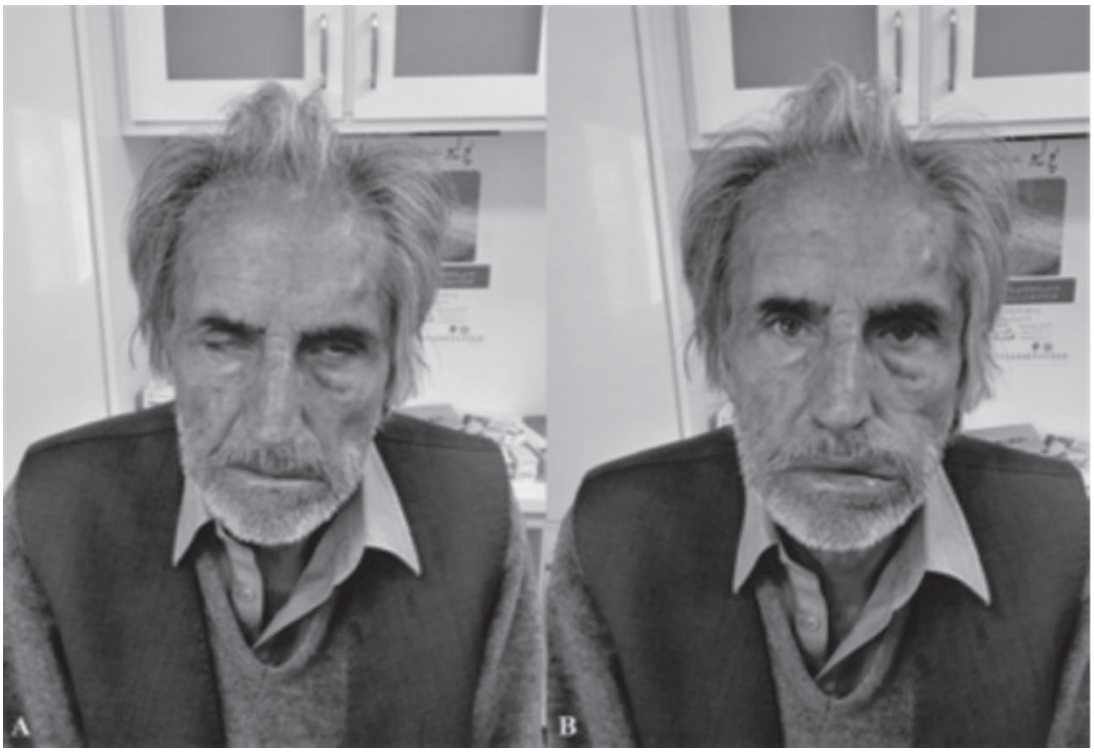
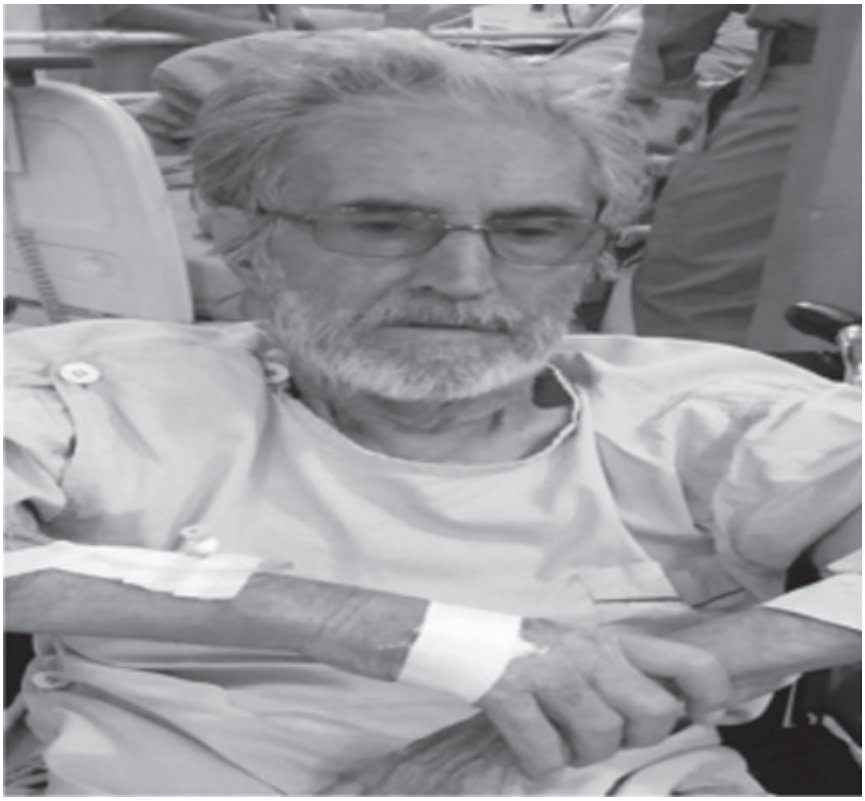


Figure 2: Improvement after two weeks of therapy



la zoster virus and herpes simplex virus were negative. Cerebrospinal fluid examination revealed raised total proteins with hypocellularity denoting characteristic albuminocytologic dissociation associated with GBS. His chest x-ray showed old tuberculous changes and computerized tomographic scan of the brain revealed old infarct in the right posterior cerebral artery region. Nerve conduction studies (NCS) performed on the third day revealed normal amplitudes and conduction velocities of all motor and sensory nerves. There was no spontaneous or voluntary activity in facial muscles bilaterally.

The patient was hospitalized and kept under observation for any progression in the symptoms. He remained ambulatory and no motor weakness developed. Intravenous immunoglobulins or plasmapheresis were not attempted, as the patient remained clinically stable. The patient was educated about the disease, its prognosis and treatment strategies. He was instructed in eye care, facial tapping and massage, mirror exercises, relaxation techniques and specific exercises to regain eye closure. Patient was discharged after five days and reviewed after two weeks when he was readmitted for gastroenteritis. The patient showed improvement up to Grade III of House-Brackmann classification bilaterally (Figure 2).

DISCUSSION

Most cases of BFNP reported in the literature are sequential, but our case is of simultaneous onset. BFNP unlike unilateral palsy is rarely idiopathic. The variety of diseases that may present with BFNP are GBS, sarcoidosis, brain stem encephalitis, Lyme's disease, benign intracranial hypertension, diabetes mellitus, Melkersson-Rosenthal syndrome, HIV infection, leukemia, syphilis, leprosy, infectious mononucleosis, bilateral neurofibromas or trauma[1-3]. Thus, the diagnostic workup for a patient with BFNP depends greatly on a meticulous history and examination.

Symmetrical rapid onset, albuminocytological dissociation and absence of signs as well as supportive investigations suggestive of other etiologies lead us to the diagnosis of GBS. Lyme's disease was excluded as our patient did not give us history of exposure to ticks nor did he visit any area endemic to the disease. The distinguishing bull's-eye shaped rash called erythema migrans, which is present in 70–80% of the infected people, was not observed. The patient also did not show signs of joint swelling or tenderness. Epstein–Barr virus producing infectious mononucleosis and Herpes viruses may also affect the facial nerve but the patient did not report sore throat, fever and body pains. His lymph nodes and liver were not enlarged and screening for herpes simplex and varicella-zoster viruses were negative.

Sarcoidosis, systemic lupus erythematosus[1] and polyarteritis nodosa are other ailments related with BFNP, but with absent respiratory, visual, joint and skin symptoms, a low ESR and a negative auto-immune response screening, they were considered less likely. The individual's signs and symptoms and dearth of supporting findings in the recent magnetic resonance imaging (MRI) scans made central nervous system leukemia, neurofibromas and benign intracranial hypertension improbable. Additional possible etiologies included amyloidosis, syphilis, porphyria[1] and tuberculosis, but bearing in mind their decreased likelihood in our patient's settings and a normal MRI, NCS and laboratory tests, these possibilities were not explored further.

FNP is observed in 27–50% of the cases suffering from GBS, often affecting both sides[5]. As a rule, other cranial nerves may likewise be included, with the potential outcomes of concurrent dysphagia and dysarthria. FNP usually follows limb weakness[6]. Exhibiting features are variable and may incorporate critical respiratory muscle weakness requiring invasive ventilation. Subsequently, timely and regular pulmonary function evaluations are prescribed in all cases. Treatment is typically supportive, with immunoglobulin infusions or plasma exchange for the patients unable to ambulate independently within 2-4 weeks from the onset of symptoms[7]. Both treatments are equally effective, when used alone or in combination[7]. Other elements of the treatment include exercises, electrical stimulation, biofeedback, motor re-education and psychosocial support[8]. Prognosis is commonly favorable with the above measures[5].

CONCLUSION

Diagnosis of BFNP involves an extensive differential diagnostic workup as bilateral disease is generally the manifestation of a systemic disease process. These patients require careful evaluation and deserve indoor monitoring and swift laboratory and radiological assessments for evaluation of the root cause and precise further management as pertinent.

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