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Bilateral Facial Paralysis with Paresthesias, Guillain-Barré Variant: A Case Report

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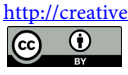

Abstract

A 42-year-old patient with no prior medical history presented with a one-week history of an inability to close his eyelids, swelling of the cheeks, and tingling sensations in the feet. These symptoms were preceded by a flu-like illness two weeks earlier. Clinical examination revealed bilateral facial diplegia with Charles Bell's sign, an inability to puff out the cheeks or whistle, but no facial sensory disturbances. Muscle strength was normal in all four limbs, with absent deep tendon reflexes in the lower limbs. Electroneuromyography showed bilateral facial nerve involvement, with an absent blink reflex and prolonged F waves in the lower limbs. Lumbar puncture revealed albuminocytological dissociation (CSF protein at 0.8 g/L with no pleocytosis). Infectious and immunological workups were negative, and brain MRI was normal. A diagnosis of Guillain-Barré syndrome with predominant facial involvement was made. The patient was treated with prednisolone at 1 mg/kg/day and motor rehabilitation. At three months, partial recovery was observed, with persistent residual facial paresis.

Subject Areas

Neurology

Keywords

Guillain-Barre Syndrome, Facial Diplegia, Paresthesia

1. Introduction

The classic form of Guillain-Barré syndrome presents as ascending sensory-motor involvement of the limbs, but there are specific clinical forms that present differ-

ently. These include facial diplegia with paresthesias (FDPP), defined by rapidly progressive bilateral facial weakness in the absence of other cranial neuropathies, ataxia or muscular weakness of the limbs [1]. We report on a case of PRN in the form of facial diplegia.

2. Case Report

This was a 42-year-old patient with no reported pathological history who was seen in consultation for an inability to close his eyelids, swelling of the cheeks and tingling paresthesias of the feet that had set in a week earlier. He reported having had an influenza-like illness two weeks before the onset of these symptoms. Examination revealed facial diplegia with bilateral Charles Bell's sign, an inability to puff out the cheeks and to whistle, and no sensory disturbance of the face (**Figure 1**). No other cranial nerve abnormalities were found. Muscle strength was normal in all 4 limbs, with abolished osteotendinous reflexes in the lower limbs.

The electroneuromyogram showed decreased motor potential in both facial nerves, with absence of the blink reflex. Neurography and detection were unremarkable in the limbs, apart from prolonged F waves on the SPI and SPE. There was hyperproteinorachia at 0.8 g/l, glycorachia at 0.7 g/l with absence of leukocytes, indicating classic albuminocyte-cytological dissociation. Bacteriological culture and viral PCR (herpes, arbovirus and enterovirus) were negative. The rest of the work-up was normal: blood count and formula, creatinemia, transaminases, serum protein electrophoresis and brain MRI (**Figure 2**). Retroviral and hepatitis serology were negative. Antiganglioside antibodies had not been tested.

Figure 1. Inability to smile with amimic face (A) and bilateral Charles Bell sign (B).

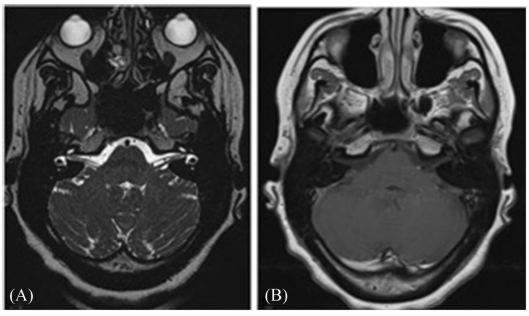

Figure 2. Normal visualisation of the emergence of the two facial nerves at the cerebellopontine angle in T2 (A) and T1 (B) sequences.

He was treated by prednisolone 1 mg/kg/D combined with motor kinsitherapy. Progression of 3 months was marked by partial recovery with persistent facial paresis.

3. Discussion

Guillain-Barré syndrome (GBS) is the leading cause of acute acquired neuropathy, with around 100,000 cases per year worldwide and an incidence rate of 0.8 to 1.9 per 100,000 people per year [2] [3]. Bilateral facial palsy is not considered a common clinical presentation. However, it might appear secondary to systemic diseases such as lupus, sarcoidosis, Kawasaki, infection (mycoplasma, HIV, Lyme, leprosy mononucleosis), pontine glioma or tegmental hemorrhage, systemic lupus erythematosis, and bulbospinal muscular atrophy [4].

The largest series of patients with isolated facial diplegia caused by GBS included 22 cases out of a sample of 8,000 suspected GBS cases referred to a neuroimmunology laboratory in Japan for the detection of antiganglioside antibodies [5]. In contrast to classic GBS, respiratory infections are more frequent than gastrointestinal infections as a triggering factor [1]. It has been shown that, for this sub-type of GBS, an infectious episode of the upper respiratory tract would precede it in two-thirds of cases, and around 30% of patients would have elements in their work-up in favor of cytomegalovirus infection. The anti-ganglioside test is negative for IgG, but may be positive for IgM in 20% of cases [1] [2] [6]. Our patient fulfilled the diagnostic criteria proposed by Wakerley, i.e. bifacial weakness, absence of motor deficits in the limbs, neck and oculomotor muscles, a history of previous infectious disease within 3 days to 6 weeks prior to presentation, and albumin-cytological dissociation [1].

The pathophysiology is not yet fully elucidated, but may be similar to that of Miler Fisher syndrome (MFS) and pharyngo-cervico-brachial palsy (PCB). One example is the ganglioside GQ1b, which is highly expressed in the cranial nerves that innervate extraocular muscles, which could explain why MFS patients with anti-GQ1b antibodies develop ophthalmoplegia. Similar antibody targets have not, however, been demonstrated in the facial nerve, although half of PCB patients and around a third of SMF patients present with facial weakness [3] [5] [7] [8].

The main differential diagnoses are neurosarcoidosis, neuroborreliosis, diabetic neuropathy, vasculitis, infections and cerebrovascular pathologies [4]. To rule out these pathologies, several investigations were carried out, all of which were normal: Lyme serology, fasting blood sugar, CSF analysis (bacteriology, parasitology, etc.) and brain MRI.

Progression of 3 months was marked by partial recovery with persistent facial paresis.

Treatment is identical to that for classical GBS, with immunoglobulins and plasma exchange, while corticosteroids have not been shown to be effective [6].

Prognosis, however, should be considered favorable, and certainly all patients we have seen with BFP have made a full recovery without need for immunother-

apy. In the largest case series11 of 22 BFP patients, 6 (27%) received IVIg, 4 (18%) plasma exchange, and 3 (14%) steroids, but because all but one patient made a full recovery, the benefits of immunotherapy were difficult to assess [1].

4. Conclusion

Facial diplegia with paresthesia is a rare subtype of classic GBS. It is characterized by demyelinating neuropathy and the absence of anti-ganglioside IgG antibodies. The appropriate treatment of this variant remains to be determined, but it is generally treated as GBS.

Conflicts of Interest

The authors declare no conflicts of interest.

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