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A Rare Case Report of Guillian Barre Syndrome Presenting with Bilateral Facial Nerve Palsy

KEYWORDS: bilateral facial palsy, Guillain-Barre Syndrome, Acute Inflammatory Demyelinating Polyneuropathy (AIDP)

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ABSTRACT *Bilateral paralysis of the facial nerve is a relatively rare presentation and often indicates a serious underlying medical condition. Guillain-Barré syndrome needs to be considered, among others in the differential diagnoses of such presentation. We present here the case of a 40 year old female who presented with bilateral facial nerve paralysis due to the Guillain-Barré syndrome.*

Introduction

Unilateral facial paralysis is a common clinical entity. Majority of these cases are due to idiopathic or Bell's palsy. Bilateral facial paralysis, unlike its unilateral counterpart is an extremely rare presentation. Common causes for bilateral facial palsy include Lyme disease, Guillain-Barre syndrome, Leukaemia, Sarcoidosis, Infectious Mononucleosis and trauma. Only 20% of these cases are due to Idiopathic or Bell's palsy where no evidence of systemic or local disease can be found. We present a case report of bilateral facial paralysis due to Guillain-Barré syndrome which has been successfully managed.

Case presentation

A 40-year-old female initially presented with a 5 day history of both sided facial weakness. There was no other significant past medical history and she was on no regular medications. An otolaryngological examination was unremarkable but for incomplete left lower motor neuron type of facial palsy.

While she did give a history of 'pins and needles' over face. She denied any history of trauma, rashes, or exposure to tick bite. Examination revealed bilateral complete lower motor neuron type of facial palsy. All other cranial nerves were intact and there was no evidence of sensory deficits elsewhere. Lower and upper limb power was normal. Paradoxically, the biceps, triceps and knee reflexes were brisk. Plantar reflexes were flexor throughout. There was no bladder or bowel involvement at any point. Fundoscopy was normal.

Blood tests for full blood counts, urea and electrolytes, liver function tests were within normal. HIV and HbsAg were non-reactive. ESR was 20 mm/1st hr. Chest radiograph was clear. Computed tomography scan of the head was normal.

Nerve conduction studies showed prolonged F-wave and prolonged distal motor latency suggestive of an early Acute Inflammatory Demyelinating Polyradiculoneuropathy motor predominant. Lumbar puncture was then performed revealing an albumin-cytological dissociation (CSF protein of 97 mg/dl and a white cell count of one). CSF glucose was 47 mg/dl (plasma glucose 82 mg/dl). With the characteristic protein-cell count differentiation, a diagnosis of the Guillain-Barre Syndrome (Acute Inflammatory Demyelinating Polyneuropathy) was made. CSF culture was negative.

She was commenced on a course of parenteral steroids on day 5 (1 gm IV Methylprednisolone). Appropriate eye protection measures were taken. After confirmation of AIDP, she was initiated on a three day course of 30 g of intravenous (iv) immunoglobulin infusions from day 8 (fig. shows the patient after 3 days of iv immunoglobulin).

Figure 1:
Day 11: Partial recovery of face after IVIg therapy.

Her facial muscle function gradually improved. She was discharged on day 20, by then she was feeling significantly better and power and reflexes in both the upper and lower limbs were normal.

While both sides the facial nerves had recovered significantly.

Discussion

Bilateral facial nerve palsy has an incidence of only 1 per 5 million populations per year. It may be the presenting feature of a potentially life threatening illness, hence care must be taken to exclude potential metabolic, infectious, vasculitic, traumatic, immunological (eg. multiple sclerosis) and neoplastic causes before diagnosing a bilateral Idiopathic or Bell's palsy.

Guillain-Barré syndrome, also known as an Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is an acute demyelinating polyradiculopathy of uncertain aetiology which

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may present with facial nerve involvement in 27–50% of cases, often bilaterally. In many cases other cranial nerves may also be involved, with the possibilities of coexistent dysphagia and dysarthria. A history of a preceding viral infection is seen in most cases. Facial palsy usually follows limb weakness. Our patient presented rather unusually in that her facial nerve paralysis preceded any significant areflexia in the peripheral limbs, the so called 'descending variant'. Diagnosis was made by cerebrospinal fluid analysis revealing a raised protein content in the absence of an increased cell count. Presenting features are variable and may include significant respiratory muscle paralysis, in which case invasive ventilation may be needed. Hence, early and regular pulmonary function assessments are recommended in all cases. Treatment is usually supportive, with immunoglobulin infusions or plasmapheresis in appropriate cases. Prognosis is generally good with the above measures.

Conclusion

Unilateral facial palsy is usually idiopathic or related to viral illness. On the other hand, bilateral facial nerve palsy is a rare, diagnostically challenging presentation. Emergency physicians should be aware of the various diagnostic possibilities, some of which are life-threatening and potentially fatal.

Management may include ventilatory support, immunoglobulin infusion or plasmapheresis. We reinforce the importance of considering the range of differential diagnosis in all cases presenting with bilateral facial nerve palsy. These patients need thorough assessment and warrant admission and prompt laboratory and radiological investigation for evaluation of the underlying cause and specific further management as relevant.

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