

Case Report

A case of bilateral facial nerve palsy in a patient with diabetes mellitus

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Introduction

Bilateral facial nerve palsy, with an incidence of 1 per 5,000,000 population, is rare and accounts for less than 2% of all facial palsy cases.^{1,2} The majority of patients with bilateral facial nerve palsy have Guillain-Barre syndrome, sarcoidosis or Lyme disease.¹ Bilateral facial nerve palsy is a sinister sign that warrants prompt investigation for a variety of causes.² We report a case of a 31 year old male who presented to our department with concurrent bilateral lower motor type facial paralysis who was found to have diabetes mellitus which was previously undiagnosed.

Case report

A 31 year old male from Yakkala was admitted to the Neurology Unit of the Colombo North Teaching Hospital with difficulty in closing his eyes which was more prominent on the left. This had developed insidiously, concurrently on both sides, over 3 weeks. He denied paraesthesia or limb weakness and there was no preceding respiratory or gastrointestinal infection. He denied sexual promiscuity or travel but, on direct questioning, revealed polyuria and polydipsia of one month duration. Neurologic examination revealed bilateral asymmetrical lower motor type facial palsy. All other cranial nerves were normal, including hearing and no long tract or cerebellar signs were noted. Physical examination did not reveal any evidence for Hansen's disease, sarcoidosis, neurofibromatosis or Lyme disease.

Laboratory workup revealed a normal FBC, ESR (12mm/1hr), liver and renal functions. Chest X-ray did not show hilar enlargement and serum ionized calcium was normal (1.2mmol/L). HbA1c was 10.5% and CSF studies revealed high sugar (318mg/dl), normal proteins (25mg/dl) and no cells. MRI brain was normal and VDRL, HIV screening and EBV IgM were negative.

Nerve conduction studies and electromyography (EMG) revealed delayed motor latencies with denervation changes in the facial muscles bilaterally, suggestive of lower motor neuron type facial nerve palsy (Bell's palsy). Nerve conduction studies of the limbs, including *f waves*, were normal, ruling out Guillain-Barre syndrome. He was started on acyclovir 800mg five times a day for five days, pre-mixed insulin, prednisolone 40mg/day and physiotherapy and gradually recovered from facial diplegia in two months.

Discussion

Bilateral facial nerve palsy is rare and an association with diabetes mellitus is even rarer, compared to the other common causes like Guillain-Barre syndrome, sarcoidosis, Lyme disease and leprosy. Other uncommon causes are multiple idiopathic cranial neuropathies,

meningitis (neoplastic or infectious), brain stem encephalitis, benign intracranial hypertension, leukemia, Melkersson-Rosenthal syndrome (a rare neurological disorder characterized by facial palsy, granulomatous cheilitis, and fissured tongue), HIV infection, syphilis, infectious mononucleosis, malformations such as Mobius Syndrome, vasculitis and bilateral neurofibromas². Lyme disease is the commonest infective aetiology worldwide³ but is not seen in Sri Lanka.

In a case series of 43 patients with facial diplegia, only one had diabetes. The other causes were idiopathic bilateral Bell's palsy, brain stem tumours, Guillain-Barre syndrome and variants, multiple idiopathic cranial neuropathies and infections, in descending order.² Adour et al⁴ have analyzed 684 patients with Bell's palsy where 64 had recurrent Bell's palsy and 3 had bilateral Bell's palsy. Out of the 67 with recurrent and bilateral Bell's palsy, nineteen (28.4%) had diabetes, but whether those with bilateral facial palsy had diabetes is not specified.

Wakerley et al⁵, Ramakrishnan et al⁶ and Chan et al⁷ describe several patients presenting with facial diplegia who were diagnosed to have bifacial weakness with paresthesia (BFP), a subtype of Guillain-Barré syndrome defined as rapidly progressive bilateral facial weakness in the absence of ataxia or limb weakness. Key features identified in those patients are a preceding history of upper respiratory infection⁵, presence of distal limb paresthesia and diminished or absent tendon reflexes⁴, presence of nerve conduction evidence of facial nerve demyelination^{5,6}, involvement of other cranial nerves⁵, prolonged recovery⁵ and CSF showing albuminocytological dissociation⁴ which were absent in our patient. This case highlights the fact that physicians should be aware of the various diagnostic possibilities of facial diplegia and should thoroughly investigate for all possible causes, some of which are life-threatening and potentially fatal.

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