Guillain-Barré Syndrome presenting as facial diplegia

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Guillain-Barré Syndrome (GBS) typically presents as ascending paralysis. GBS presenting as simultaneous bilateral facial palsy is very uncommon. We report a case of GBS presenting as facial diplegia without any limb weakness.

Case report

A 48-year-old housewife presented with complaints of loose motions for 5 days, followed by sudden onset difficulty in chewing along with dribbling of saliva for 3 days. She was not able to smile or blow and close her eyes. There was no history of limb weakness, numbness, bowel or bladder symptoms, earache or discharge, headache, fever, skin lesions, joint pain, vertigo, visual disturbances or altered sensorium.

She had a known case of hypertension on irregular treatment, and had been suffering from impaired hearing for the last 5 years.

On examination, the patient was conscious with a regular pulse of 104/min, BP of 100/70 mm of Hg and respiratory rate of 16/min. CNS examination revealed bilateral infra-nuclear palsy of seventh cranial nerves (Figures 1 & 2a). Rinne test was negative bilaterally, with Weber test lateralised to left ear. Other cranial nerves were found normal on examination. Planters were flexor, while all the deep tendon reflexes were absent. Power was normal in all four limbs, but vibration and joint position sensations were impaired below the knees. Romberg's test was positive. There were no cerebellar or meningeal signs. Fundus was normal.

Figure 1: Inability to close eyes.
Blood counts and biochemistry were normal. Blood sugar values were normal and HbA1c was 5.8%. Stool microscopy was normal and culture did not show any growth. Autoimmune profile, thyroid function tests, CXR and USG-Abdomen were within normal limits. NCCT head was normal. HIV, hepatitis B and C, serology for cytomegalovirus and Epstein-Barr virus were negative. CSF examination showed sugar 67mg/dl (normal range: 40–70), protein 206mg/dl (normal range: 15–50), and no cells (normal range: 0–5 cells/cmm), suggesting albumino-cytological dissociation. Nerve conduction study (NCS) showed that prolonged motor-sensory distal latencies were attained from median nerves with normal amplitude and conduction velocity. Low motor nerve amplitude was attained from peroneal nerves. F-wave was not recordable from right peroneal nerve, while prolonged F-latency was attained from median nerves. NCS findings were suggestive of demyelination and consistent with GBS. ENT consultation confirmed B/L conductive deafness due to wax.

A diagnosis of GBS variant with facial diplegia was made. Patient was administered intravenous immunoglobulin (IVIG) at a dose of 0.4g/kg body weight, daily for 5 days, along with physiotherapy and other supportive treatment. Within a few days, the patient started showing improvements in the facial palsy. She was able to close her eyes and frown (Figure-2b) and drooling of saliva improved. After 6 weeks, she had almost complete recovery of facial weakness.

Discussion

Facial diplegia (bilateral facial paralysis) is a rare clinical finding that can be the presenting feature in a wide range of diseases. Reported aetiologies include Bell's palsy, sarcoidosis, Lyme disease, GBS, diabetes mellitus, brainstem encephalitis, brainstem stroke, Ramsay Hunt/Melkersson-Rosenthal syndrome, leprosy and HIV. Keane, in a 23-year review of 43 patients with predominant bifacial palsy, found that bilateral Bell's palsy (10/43) and GBS (5/43) were the most common causes. Other research has shown that facial diplegia was present in more than half the cases of GBS, but the facial weakness was preceded or accompanied by limb weakness. Isolated facial diplegia, with minimal or absent motor limb weakness, has been described as a GBS variant.

Our case had bilateral facial palsy, areflexia and distal sensory impairment, preceded by diarrhoea. Similar case had been described in literature by Akinori et al, where they showed enhancement of facial nerves on 3D-MRI. In a recent study, predominant facial diplegia has been highlighted as a variant of GBS. This study suggested that facial diplegia could be a regional variant of GBS when accompanied by paraesthesia, albumino-cytological dissociation and NCS abnormalities.

We diagnosed this case as GBS variant on the basis of antecedent diarrhoea, monophasic course, areflexia, albumino-cytological dissociation, evidence of demyelination in NCS and response to IVIG.
REFERENCES:


