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Case Report

Guillain-Barre Syndrome with Bilateral Facial Nerve Palsy: A Case Report

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ABSTRACT

Bilateral simultaneous facial nerve palsy (BFNP) is rarely encountered in clinical practice. It may occur in various infections, as well as neurological, neoplastic and degenerative disorders. We report a case of 30 year female patient presenting with headache, fever and vomiting for 5 days. Next day she developed quadriplegia with areflexia and BFNP. There was evidence of demyelinating neuropathy and axonal degeneration on nerve conduction study (NCS) and albumin- cytological dissociation on cerebrospinal fluid (CSF) analysis. These neurologic findings were suggestive of Guillain-Barre syndrome (GBS). The patient was satisfactorily treated with immunoglobulins.

KEYWORDS: Bilateral facial nerve palsy, Cerebrospinal fluid (CSF), Guillian-Barre syndrome.

INTRODUCTION

Unilateral facial nerve palsy (UFNP) is more common with an incidence of about 25 per 100000 population. The underlying etiology is found in about 20% of cases with majority of them occurring due to Bell's palsy. Bilateral facial nerve palsy (BFNP) is quite rare with an incidence of 1 per 500000 population which may be due to its unusual presentation[1]. GBS mainly involves motor nerves but it may also affect sensory and autonomic nerves. Most patients have a demyelinating type of neuropathy but few patients may have primary axonal degeneration[2]. It is more common in males than females 1.5:1 ratio affecting all age groups[3]. There may be antecedent, usually viral infection triggering inflammation and demyelination. Bulbar involvement may occur in 50% cases while few patients may develop respiratory failure, facial nerve palsy, viral meningitis or meningoencephalitis. BFNP and UFNP have different etiologies. Most common causes of BFNP are GBS, diabetes mellitus, bacterial meningitis, infectious mononucleosis, sarcoidosis and HIV infection while Lyme disease, syphilis and leprosy are rare causes[1]. The main difference in BFNP and UFNP is the diagnostic evaluation of these patients and exclusion of life threatening conditions. CSF studies are important for diagnosis thus showing albumin-cytological dissociation[4,5]. We report a case of 30 years female presenting with quadriplegia, areflexia and BFNP.

CASE REPORT

A 30 year female, non-diabetic, non-smoker, vegetarian presented with headache, fever and vomiting for 5 days. Next day she developed slurring of speech and dribbling from the mouth on eating and weakness of both lower limb muscles. She was unable to close her eyes and mouth. She also complained of altered taste sensation. There was no history of trauma, intramuscular injection, travel abroad, illicit drug use, heavy metal intoxication, recent vaccination, sera use, chemical exposure, inhalation or unprotected sex. On examination she was drowsy with Glasgow coma scale (GCS) 13/15. Vitals were stable with Pulse rate 74 beats/min, BP 130/80 mmHg, respiratory rate 18/min, Temp 38^0C. Kernig's sign and neck rigidity was negative. She had lower motor neuron paresis of bilateral facial nerves, bilateral Bell's phenomenon and inability to purse her lips or smile. All other cranial nerves were normal. In the next 24 hours, weakness progressed to involve both upper limbs with evidence of generalised hypotonia. Power in upper limbs was 3/5 in proximal and distal groups while it was 2/5 in both lower limbs and in all muscle groups. Plantar response was bilaterally flexor. Deep tendon reflexes were absent in both upper and lower limbs. Superficial reflexes were intact. There was no bladder or bowel involvement at any point. Sensory examination did not reveal any hypo or hyperaesthesia. There was no peripheral nerve thickening. Fundus examination was normal. Rest of systemic examination was non-contributory. Laboratory profile revealed Haemoglobin 11.2 g%, Total leucocyte count 9600/mm^3, Differential leucocyte count P 70, L 25, E 3, M 2, platelet count 3 lac/mm^3, ESR 20 mm in first hour. Peripheral blood film was normocytic and normochromic. Malarial parasitic

slide, renal profile, liver profile, serum electrolytes, blood sugar, blood culture, Widal test, Mantoux test, chest X-ray, stool examination, urine examination was normal. Computed tomography of the brain was within normal limits. MRI cervical spine did not show any evidence of cervical myelopathy. CSF analysis revealed protein 95 mg%, sugar 80 mg% (Plasma blood sugar 115 mg%), cells 5/mm^3 depicting albumin-cytological dissociation. Work up for VDRL, autoantibody, hepatitis, serum angiotensin converting enzyme, HIV was negative. The CSF was also negative for syphilis, cysticercosis, tuberculosis, cryptococcus and HIV. Nerve conduction study (NCS) performed on 3rd of admission revealed compound motor action potential (CMAP) in both perioneal and tibial nerves. Distal latencies were markedly prolonged in both median and ulnar nerves with reduced CMAP amplitudes and conduction velocities. F waves were absent in all four limbs. Sensory nerve action potential (SNAP) was slightly reduced in both median, ulnar and sural nerves. The patient was managed with intravenous immunoglobulins 400 mg/kg/day for 5 days along with IV Ceftriaxone. There was marked improvement in the muscle weakness and tone. Patient was able to walk. Facial asymmetry disappeared. She was discharged in a satisfactory condition and now she is on regular follow up.

DISCUSSION

GBS may present initially with muscle pain involving the anterior and posterior thighs, buttocks and lower back. This is followed by symmetric, progressive weakness of the extremities of facial muscles. Patient may develop headache, autonomic dysfunction, ataxia and meningismus. There may be facial nerve involvement which is commonly bilateral and may be seen in 50% of cases[6].Causes of bilateral facial nerve involvement include idiopathic etiology in most of cases followed by GBS, diabetes mellitus, sarcoidosis, leprosy, syphilis, bacterial meningitis and Lyme disease etc[1] . Usually presence of areflexia in such cases helps to differentiate GBS as the underlying etiology. Diagnosis of GBS in the patient was made on the basis of clinical findings, albumino-cytological dissociation in CSF and NCS. The nerve conduction study revealed absent H reflexes and F waves indicating demyelination and axonal degeneration findings typical of early GBS.[7] Severity of GBS varies in different patients after involvement of respiratory muscles making the patients to require ventilator support and immune-modulatory treatment. However mild cases may develop potential side effects of IVIG and plasmaphresis. But depending on the severity of our patient, she responded well to IVIG therapy. Treatment may be unnecessary in ambulatory patients during the second week of illness.[8]

CONCLUSION

Bilateral facial nerve palsy secondary to Guillain-Barre syndrome is rare. GBS primarily involves motor but may involve sensory and autonomic nerves also. It is not hereditary and affects people of all age groups. Timely management is lifesaving.

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