

A case of Guillain-Barre syndrome presented with bilateral pseudo-internuclear ophthalmoplegia

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ABSTRACT

Guillain-Barre syndrome (GBS) or acute idiopathic polyradiculoneuritis is an acquired immune-mediated inflammatory and mainly demyelinating disorder of the peripheral nervous system. Cranial nerves are affected in over 50% of all cases, with the facial nerves being affected the most. It uncommonly presents as atypical forms such as brachial pharyngeal variant, miller fisher and other restricted forms. Herein, we reported a 44 year old male with GBS who presented with diplopia and bilateral pseudo- internuclear ophthalmoplegia. Initially, the patient was confused as a case of multiple sclerosis but finally diagnosis of GBS was made. Although internuclear ophthalmoplegia is a typical feature of multiple sclerosis, it may be seen as a rare manifestation of GBS as well.

Keywords: Guillain-Barre Syndrome; Internuclear Ophthalmoplegia; Multiple Sclerosis

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INTRODUCTION

Guillain-Barre syndrome (GBS) is an immune-mediated inflammatory and mainly demyelinating disorder of the peripheral nervous system. The classic clinical picture is ascending symmetrical weakness, sensory symptoms and areflexia¹. Cranial nerves are affected in over 50% of all cases and the facial nerves being affected the most. Otherwise oculomotor nerves involvement is uncommon and might occur in about 10% of cases². It uncommonly presents in the atypical forms as brachial pharyngeal variant, miller fisher and other restricted forms¹⁻³. Internuclear Ophthalmoplegia (INO) is a disorder of eye movements caused by a lesion in an area of the brain stem called the medial longitudinal fasciculus. It is a specific gaze abnormality characterized by impaired horizontal eye movements with weak adduction of the affected eye, and abduction nystagmus of the contralateral eye. The most common cause of bilateral INO is multiple sclerosis, a demyelinating disease of the

Central Nervous System (CNS)⁴.

Here, we reported a very rare presentation of GBS which mimicked CNS demyelinating disease.

CASE PRESENTATION

A 44-year-old male employee presented with diplopia since three days prior to admission. The patient was admitted in Namazie Hospital affiliated to Shiraz University of Medical Sciences, Shiraz, Iran for evaluation. He had no history of weakness, vertigo, dysarthria, dysphagia, and headache. Bladder function was normal but he had a history of constipation since the few previous days. He had no history of conserved food consumption. He had no history of fever, upper respiratory infection or diarrhea in the previous one month. His family history was unremarkable.

On examination, the patient was conscious. His blood pressure was 100/70 mmHg. His pulse rate was 70 per minute and regular, respiratory rate was 18 and

was afebrile. In neurological examination, his mental function including speech and language was normal. On cranial nerve examination, on the first day of admission, he had horizontal diplopia during the lateral gaze in both eyes. The patient had limitation in adduction in the right eye and horizontal nystagmus in the left eye on leftward gaze. He also had adduction deficit in the left eye and nystagmus in the right eye on rightward gaze (bilateral INO). Eye movements in upward and downward direction were normal. Pupillary response to light was normal bilaterally and optic fundus was normal. Other cranial nerves were normal. On sensory examination, pain, temperature, pinprick, vibration and position were normal. Romberg test was negative. In motor examination, the tone and power and deep tendon reflexes were normal. Cerebellar exam was normal. Examination in other systems was normal.

With possibility of multiple sclerosis, brain Magnetic Resonance Imaging (MRI) was done; but it was normal. During the hospital course, he developed bilateral peripheral facial palsy and weakness of upper limbs. In motor examination, muscle power in upper limbs was 3/5 and 5/5 in lower limbs. Deep tendon reflexes were decreased in upper limbs. Plantar reflexes were bilaterally downward.

His complete blood count was normal. Blood sugar, urea, creatinine, liver function tests, thyroid function test and serum electrolytes revealed no abnormality. Nerve conduction study was done and showed prolonged distal latency of both facial nerves and prolonged F wave in the median and ulnar nerves. Motor conduction velocity in median and ulnar nerve was decreased significantly and also temporal dispersion in right median was seen, but there was no conduction block. With possibility of atypical form of GBS, lumbar puncture was done. Cerebrospinal Fluid (CSF) cell count was zero and protein was 252 mg/dl and sugar was normal. Then, the patient was managed with intravenous immunoglobulin 0.4 gram per Kg for 5 consecutive days and his facial palsy, diplopia and weakness of upper limbs improved in the hospital course and in follow up completely resolved after 4 months.

DISCUSSION

GBS is an acquired immune-mediated inflammatory disorder of the peripheral nervous system. Clinical hallmarks are symmetrical flaccid paresis and areflexia in the presence of increased cerebrospinal fluid protein content. Also, atypical presentations of GBS can be seen¹⁻³. In 1956, Fisher described three patients with ataxia,

areflexia, and ophthalmoplegia, the classical triad of Miller Fisher syndrome. Mild limb weakness, ptosis, facial palsy, and bulbar palsy may also occur in Miller Fisher syndrome. This accounts for about 5% of patients with GBS⁵.

Internuclear ophthalmoplegia is a specific gaze abnormality characterized with weak adduction of the affected eye, and abduction nystagmus of the contralateral eye. INO may be unilateral or bilateral. Multiple sclerosis is the most common cause of bilateral INO while vascular disease is the most common cause of unilateral INO. Other causes include head trauma, brainstem and fourth ventricular tumors, Arnold-Chiari malformation, infection, hydrocephalus, and lupus erythematosus⁴. The diseases that may mimic INO (pseudo INO) are Myasthenia gravis, Partial cranial third nerve palsy, and long-standing exotropia^{6,7}.

In 1975, Diamond and his colleagues reported a 32 year old patient presented with bilateral pseudo-INO, external ophthalmoplegia and alternate extreme divergent strabismus. The clinical picture was confused with myasthenia gravis, or a demyelinating process, but finally diagnosis of GBS was done⁸. In our report the patient presented with diplopia and eye movement disorder that firstly was confused with INO and multiple sclerosis but there was no evidence of multiple sclerosis in brain MRI. In hospital course the patient developed other symptoms of GBS and the diagnosis was confirmed by clinical course, nerve conduction study and protein cell dissociation in CSF study.

CONCLUSION

GBS may have an atypical presentation and Pseudo INO can be a very rare presentation of it and therefore could be confused with central demyelinating disease.

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