

Case Report on Guillain Barre Syndrome: Acute Inflammatory Demyelinating Polyneuropathy

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ABSTRACT

Guillain Barre Syndrome (GBS) is an autoimmune disorder which affects the peripheral nervous system. It is a rare disorder affects in 1 per million people in year. It is characterized by symmetrical, progressive limb weakness and tingling.

Case Report: A 53 year old male patient was presented with insidious onset of difficulty in moving right upper and lower limbs as well as gradual weakness of left limbs, and breathing difficulty, known case of diabetics' mellitus and hypertension. Nerve conduction study shows suggest axonopathy; Acute Inflammatory Demyelinating Polyneuropathy (AIDP) is identified, which is a subtype of Guillain Barre Syndrome. Patient gradually develops areflexia, bifacial weakness, and quadriparesis. Patient was treated with IV immunoglobulin and intranasal oxygen therapy. Patient shows slight improvement in his medical condition, shows improvement in the power of lower limbs after one week of therapy. Physiotherapy was suggested.

Keywords: Guillain Barre Syndrome, GBS, Acute Inflammatory Demyelinating Polyneuropathy, AIDP

INTRODUCTION

Guillain Barre Syndrome is an autoimmune disorder which affects the peripheral nervous system. The major types of GBS are Acute Motor Axonal neuropathy (AMAN), Acute Inflammatory Demyelinating Polyneuropathy (AIDP) and

Miller Fischer syndrome. GBS is a very rare disorder, the incidence of this disorder is 1-3/100000 in population ^[1]. Studies suggest that GBS can also occurs after infections caused by various infectious agents such as Camphylobacter jejuni, Cytomegalo virus, Epstein barr virus, and influenza virus. These infection trigger the occurrence of GBS in certain people ^[1,2]. Polyneuropathy of the guillain barre syndrome is due to the interaction between peripheral nerve glycolipid and microorganism. ^[3] GBS is characterized by progressive weakness of limbs, the weakness starts at the distal parts of limbs and severe GBS can results in the weakness of respiratory muscles. The diagnosis of GBS can be done mainly by the assessment of symptoms and also by analyzing the results of nerve conduction studies. Guillain Barre Syndrome is managed by plasmaphoresis and iv immunoglobulin.

CASE STUDY

A 53 year old male patient was admitted with the complaints of difficulty in moving right upper and lower limbs, difficulty in breathing in supine. Patient has no history of dizziness, loss of consciousness, chest pain, slurred speech or neck stiffness. The patient had a previous history of respiratory infection two week prior to the appearance of symptoms. On examination patient shows reduced deep tendon reflexes and decreased muscle tone.

Bilateral lower motor neuron palsy with positive bell's phenomenon. The rest of the cranial nerves were normal. The vitals of the patient were also found to be normal. The patient was sending for further investigation. Brain CT scan appears to be normal, chest CT scan shows bilateral fibrotic pleural thickening due to previous infection. Blood investigation reports were found to be normal. The other investigations were Glycosylated haemoglobin; 8.2%, CRP: 3.6mg/l, ESR: 25mm/hr. Nerve Conduction Studies (NCS) shows abnormal amplitude of M wave suggesting axonopathy. Nerve conduction study shows normal latencies and conduction velocity, suggest myelinopathy of right median and ulnar nerves. The patient condition was diagnosed as Acute Motor Axonal Neuropathy (AMAN), which is a variant of Guillain Barre Syndrome.

The patient was treated with IV fluid 2L/day, subcutaneous enoxaparin 40 mg daily, intranasal oxygen was also provided during first day, IV immunoglobulin 35mg daily was also started for five days, and multivitamins were also provided to the patient. Patient was also treated with pregabalin 75mg every 12 hourly. Since the patient is diabetic daily sugar check was also instructed. From the second day onwards physiotherapy was also started. Patient gradually started to show improvement in symptoms. By the fourth day of treatment patient start to complain about burning pain on his limbs, which was managed using intravenous paracetamol, IV ketorolac 30 mg every eight hourly and oral pregabalin 5 mg every 12 hourly. Patient was on antihypertensive Valsartan 160 mg daily in the morning and Amlodipine 10 mg in the night, since the patient was diabetic he was also taking metformin 1 mg every 12 hourly.

Patient starts to show improvement in his lower limb weakness, but the patient suddenly start to complain about the weakness of his jaw muscles as well as difficulty in speaking during the course of the treatment. For which the patient receives

two extra course of IV immunoglobulin 38 mg daily (2 days). But patient's general condition shows overall improvement. Patient was instructed to continue physiotherapy at least for three months during the discharge.

DISCUSSION

Guillain Barre Syndrome is a rare immune mediated neurological disorder. It is characterized by demyelinating of the neurons. The symptoms of the GBS are rapidly progressive and symmetrical weakness of the lower limbs, which gradually progress to the upper limbs ^[4]. This case the patient shows the weakness of lower limbs. Mainly there are three different forms of GBS, which are Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN) and Miller Fisher Syndrome. Where Acute Inflammatory Demyelinating Polyradiculoneuropathy is the most frequently occurring subtype of GBS, Comparing to AIDP, Acute Motor Axonal Neuropathy is less common and miller fischer syndrome is the uncommon subtype of GBS. AMAN, the subtype of GBS can occur as a post infection complication of a camphylobacter jejuni infection. But the incidence of AMAN after Camphylobacter Jejuni infection is only 1 in 5000 individual. Here this patient AMAN was identified and patient history shows he experiences an acute infectious episode weeks prior to the experiencing of GBS symptoms and patient shows no sensory involvement. Patient mainly shows flaccid quadriplegia and distal limb weakness ^[4]. The incidence of GBS is only 1 per 10,00,000 persons per year. The disease most commonly occurs in older persons above forty years ^[5] and more common among men. This GBS patient is a 53 year old male. The most common clinical characteristics of this neurological disorder includes progressive weakness of limbs, hyporeflexia, parasthesia. ^[6] certain patients, about 20-30% of GBS patients may experience respiratory failure ^[5]. Here, this

patient also experiences respiratory problems due to GBS. The facial pain is also a common manifestation in guillain barre syndrome, this patient also experience facial pain during the course of treatment.

In this case this particular patient shows characteristic features of Guillain barre syndrome. The patient came to the clinic with lower limb weakness which gradually progress into the ascending limbs. This kind of 'ascending pattern' of pain, in which pain begins at legs gradually moves into arms were the characteristics of guillain barre syndrome associated neuropathic pain.^[7] So the preliminary diagnosis was done based on the assessment of patient symptoms. The confirmatory diagnosis can be done by electrophysiological studies such as nerve conduction studies and here the patient shows loss of peripheral nerve axons which shows distal limb weakness that also help to identify axonal motor neuropathy^[7]. The treatment of GBS includes plasma exchange or IV immunoglobulin therapy. Here plasma exchange was the first approved treatment for GBS, usually plasma exchange will be done for 2-5 times depends on patients condition. Due to the reduced access to plasma and increased medical as well as non-medical cost makes plasma exchange less desirable than IV immunoglobulin therapy. The ideal dose of IV immunoglobulin for GBS is 0.4g/kg body weight for 5 days, here for this patient iv immunoglobulin were used^[8].

CONCLUSION

Guillain Barre Syndrome is a rare neurological disorder which results in the paralysis of muscles. The symptoms of GBS are progressive and symmetrical. There are mainly two subtypes of GBS such as Axonal Motor Axonal Neuropathy (AMAN) and Acute Inflammatory Demyelinating Polyneuropathy (AIDP). The standard treatments for GBS are plasma exchange and IV immunoglobulin therapy. In which, IV immunoglobulin is more preferable over the plasma exchange.

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