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Case Report

Guillain Barré Syndrome- A Case Report

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Received: May 13, 2019; **Published:** June 21, 2019

Abstract

Guillain Barre Syndrome (GBS) is a rare complicated neurodegenerative disorder which may be acute or chronic in nature, involving peripheral nerves neuropathy. It is a disease where the immune system of host attacks their own neurons, damage the nerve roots, and disable the electrical impulses transmission across brain, spinal cord to muscles resulting in sensory and motor nerve fibers damage and neuropathy. This makes the person unable to walk, making ankle, thighs, feet, legs flaccid and uniformly weak. GBS progresses rapidly within days to weeks and it is to be treated at early stages for better recovery.

Keywords: *Neurodegenerative; Neuropathy; Peripheral Nerves; Electrical Impulses; Sensory; Motor*

Introduction

Guillain Barre Syndrome (GBS) is a rare complicated neurodegenerative disorder which may be acute or chronic in nature, involving peripheral nerves neuropathy. It is a disease where the immune system of host attacks their own neurons, damage the nerve roots, and disable the electrical impulses transmission across brain, spinal cord to muscles resulting in sensory and motor nerve fibers damage and neuropathy. It is an acute inflammatory demyelinating neuropathy, which involves damage to the myelin sheath by the host's immune system, resulting in loss of electrical signals transmission across the neurons. This makes the person unable to walk, making ankle, thighs, and feet, legs flaccid and uniformly weak. GBS progresses rapidly within days to weeks and it is to be treated at early stages for better recovery.

Case Report

A 40 years old male presented with the symptoms of numbness and tingling sensation in both hands, and generalized weakness in both lower limbs. His symptoms began slowly progressing, making him unable to walk, following the development of left lower limb cellulitis. He had a past history of cervical spondylosis from c4-c7.

Examination of this man revealed a healthy person in other wise good condition, developed generalized weakness of the lower extremities, upper extremities. Sensory loss was seen in both hands and feet. A general physical examination gave idea about left leg cellulitis, swelling [1]. His cardiac, respiratory and renal functions were within normal limits.

The patient was referred to his physician and radiology department. Lower limb scan revealed diffuse subcutaneous edema in leg around ankle, cellulitis of left lower limb. Lab reports suggested an increase in the white blood cell count 14,400/Ml, creatinine phosphokinase: 58.6 U/L, creatinine kinase - MB: 30.3/L.

Diagnosis of Guillain-Barré syndrome was made after utilizing the patient's criteria i.e. progression over days to few weeks, relatively symmetrical, coordination problems and unsteadiness, numbness and tingling sensation of hands, feet, pain which get severe at night [1].

Medical management given by his physician included: Intravenous immunoglobulin every 4th hourly for 5 days, Beta-lactam antibiotic and other penicillin antibiotics alternatively along with magnesium sulphate, glycerol to treat cellulitis. This patient slowly improved his condition, which made him able to walk slowly. After 7 days of treatment his leg strength was improved, and his numbness and tingling sensation were reduced.

Discussion

Human nervous system includes brain and spinal cord, referred collectively as central nervous system (CNS), and nerves extending into head, trunk, and limbs which is the peripheral nervous system (PNS). At the point where peripheral nerves emerge from the spinal cord, they are known as nerve roots/radicles. Damage to these nerve roots is called as radiculopathy. Peripheral nerves consist of hundreds of nerve fibres, which include sensory and motor nerve fibers, which transmit electrical impulses across brain to muscles. Peripheral neuropathy also known as polyneuropathy, results from the degeneration of neurons of peripheral nerve either in axon or myelin sheets [2].

Guillain Barré Syndrome (GBS) is an acute peripheral neuropathy, which evolves within days to weeks of onset. GBS is mostly an acute inflammatory demyelinating polyneuropathy (AIDP), in which the immune attack is directed against myelin, leading to the degeneration of myelin sheath, and leading to a short circuit, where the electrical messages cannot travel from brain to periphery parts of body. Even the axonal GBS, is even seen. Where if the axonal GBS attacks motor neurons it results in weakness, and loss of sensation, it results in acute motor axonal neuropathy (AMAN). When both sensory and motor neurons are affected, it is known as acute motor and sensory axonal neuropathy (AMSAN). Occasionally GBS causes paralysis of eye muscles and loss of balance and coordination, resulting in Miller Fisher syndrome (MFS). Hence, GBS is a group of disorders, consisting of AIDP, AMAN, AMSAN, and MFS [2] (Table 1).

Type	Effects
Acute inflammatory demyelinating polyneuropathy (AIDP)	• Primary damage to myelin sheath. • Causes: Acute weakness of limb, ribs, sensory signs (numbness, tingling sensation)
Acute motor axonal neuropathy (AMAN)	• Primary damage to motor axons • Causes acute weakness of limb, ribs.
Acute motor and sensory axonal neuropathy (AMSAN)	• Primary damage to both sensory and motor axons. • Causes acute weakness of limbs, ribs • Causes numbness, tingling.
Miller Fisher syndrome (MFS)	• Primary damage is uncertain • Causes ophthalmoplegia, loss of balance, incoordination, and loss of reflexes.

Table 1: *Associated types of GBS disorder [2].*

Symptoms

Weakness which is caused by the damage to the motor nerves in brain, spinal cord spreading to the muscles is the first clinical feature in GBS. Weakness usually begins in legs, i.e. symmetric which affects both sides of body. In some people muscles in limbs farther from spinal cord (distal muscles) are affected, in others muscles located close to the spinal cord (proximal muscles) are effected developing weakness, hence ankle, thighs, legs, and feet becoming uniformly weak and flaccid. GBS progresses rapidly over days to 4 weeks, it ascends up the body from the legs to arms. This makes the person unable to walk, climb stairs, holding things with hands properly.

The weakness of GBS is typically "pyramidal in distribution" with ankle dorsiflexion and knees and hip flexion are severely affected with shoulder abduction and elbow extension [3]. Facial weakness, head and neck muscles even also become weak. Abnormal sensations called as paresthesias, including pins and needles, or tingling sensations in limbs, spinal cord. Loss of reflexes is most prominent in GBS. Autonomic nerves which connect peripheral nerves in body damage the nerves and develop weakness. Person may develop difficulty in urinating, constipation, lightheadedness and syncope, palpitations, increased sweating, cold body temperature mostly [2].

Triggering factors

Mostly infections lead to development of GBS within 2 - 4 weeks. Infections including cytomegalovirus, *Mycoplasma pneumonia, Campylobacter jejuni*, Epstein Barr virus may act as triggering agents compromising the immune system and initiating infection. This occurs via molecular mimicry, i.e. the antigens of invading organism is similar to the antigens on the surface of myelin sheath, then the antibodies and inflammatory cells will attack and damage the myelin sheath [4]. Inflammation and degeneration of the myelin sheath leads to the leakage of the proteins from the blood into Cerebrospinal fluid (CSF), leading to increased CSF protein concentration in diseased state [2] (Figures 1 and 2).

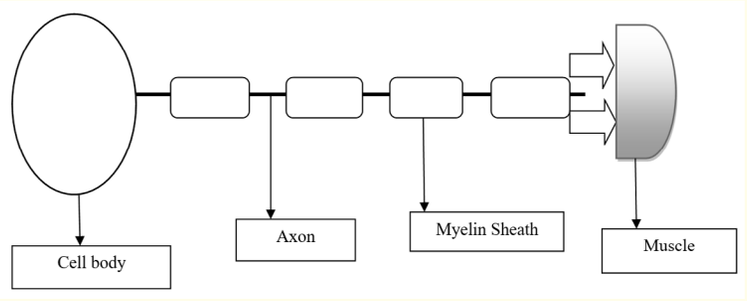

Figure 1: *Normal motor unit [2].*

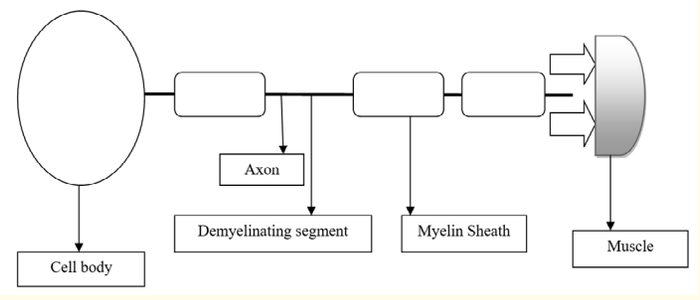

Figure 2: *Demyelinated motor unit seen in GBS [2].*

Treatment

GBS has two main treatment patterns. Firstly, Plasma exchange i.e. plasmapheresis, which involves pathogenic substance, antibodies filter from plasma which where damaging nerves. Secondly, Intravenous administration of Immunoglobulin (IvIg), which is proposed to provide antigen that, blocks antibodies from binding to B lymphocytes, and reducing production of interleukins, and suppressing immune mediated process. It lowers the effectiveness of antibodies by diluting them with non- specific antibodies that bind to harmful antibodies and eliminate them [4].

- Supportive care is important as many complications including paralysis, respiratory failure can occur in GBS, hence close monitoring is needed.
- Vitamin B_{12} which deficiency leads to damage of myelin sheath is essential for GBS patients. Foods sources of vitamin B_{12} include fish, meat, egg, milk. The recommended requirement of vitamin B_{12} is 24 micrograms.
- Vitamin B_6 is essential for GBS patients, but more of it becomes toxic to nerves. Hence recommended dose of 2 milligrams per day is essential. Food sources of vitamin B_6 include Sweet potato, banana, potatoes, avocado, and pistachios.
- Consuming large amounts of frozen fish are related to sufficiently high levels of mercury which is linked to paresthesia, prickling, and burning sensation. Hence limiting the intake of fish is necessary.

- Regular physiotherapy is needed to prevent muscle shortening and improve muscle flexibility and walking with support is needed every 4th hourly is advised.
- Inflatable cuffs can be placed around legs, to provide intermittent compression and prevent blood stagnation in leg veins.

Conclusion

The above discussed case report is regarding a male patient aged 40 years, reported to hospital with numbness, tingling sensation, generalized weakness in both lower limbs, coordination problems and unsteadiness which is relatively symmetrical, and unable to walk which progressed slowly. Based on above symptoms and laboratory reports it was diagnosed as GBS, which is an acute inflammatory demyelinating polyneuropathy, evolving within days to weeks of onset. Resulting in degeneration of myelin sheath, due to host immune system attack, and this leads to loss of neurotransmission in body parts, it may be either motor or sensory. Here the patient was treated with Intravenous immunoglobulins (IvIg) for 7 days continuously, after which the patient was having the strength to walk slowly on his own, with reduced symptoms of weakness.

The patient was checked daily for the development of adverse events associated with IvIg administration, and his vitals and other organs remain unaffected by the pharmacological drug therapy. The patient was counselled here regarding the diet, the vitamins which are essential for nerve strength, and the foods which are to be avoided in large quantities of intake. The patient was counselled on the physiotherapy exercise to make sure that movement is given regularly to limbs regardless of pain, to improve strength of limbs. This case report identifies the rare disease GBS, its manifestations, along with its pharmacological therapy, and the non-pharmacological therapy, importance of physiotherapy, food restrictions, and intake to be followed along with emotional support to be given to the patient and patient care taker.

Acknowledgement

I accept the truth that the case discussed above is a genuine case identified during internship program at multi-speciality hospital located in Warangal, Telangana State, India.

Funding Acknowledgement

This case report received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Conflict of Interest

The authors declare no conflict of interest in preparing this case report.

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Volume 7 Issue 7 July 2019