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A Case Report: Guillain Barre Syndrome

Rudra Patel[1], Vaishnavi Patel[1], Hardik Jain[1], Dr. Neha Suratiya[2]

[1]Doctor of Pharmacy Students, [2]Assistant Professor, Department of Pharmacy Practice

[1]Department of Pharmacy Practice

Parul Institute of Pharmacy, Parul University, Limda, Waghodia, Gujarat, India – 391760

Abstract:

Background: Guillain barre syndrome (GBS) is an immune mediated polyradiculoneuropathy. It is a disorder in which sensory changes occurs which limits patient mobility. Muscle pain and weakness aggravates in this condition. 31year old male patient was admitted with complaints of fevers without chills, limb weakness and pain. On examination, muscle power was decreased, there was limited shoulder movement and finger grasp was weak. Diagnosis was made on basis of Electromyogram and Nerve Conduction Study it was observed that patient was suggestive of GBS.

Method: the data of report was collected from tertiary care hospital and relevant articles were searched on the databases like "Pubmed" and "Cochrane library".

Result: Plasmapheresis cycles were successful in treating the patient. Physiotherapy and rehabilitation helped in recovery.

Conclusion: Our finding emphasizes the need of knowledge about the pathophysiology of idiopathic GBS in order to improvise the treatment options and targeted therapy.

Keywords: Anti-ganglioside Antibodies, Polyneuropathy, Electromyogram, Nerve Conduction Study, Plasmapheresis

I. INTRODUCTION

Guillain barre syndrome (GBS) is polyradiculoneuropathy with an immunological basis that include symptoms like symmetric muscle weakness, unstable ambulation and hypoflexia or areflexia [1]. GBS is further divided following subtypes: Acute motor and sensory axonal neuropathy (AMSAN), AMAN or acute motor axonal neuropathy, Acute inflammatory demyelinating polyneuropathy (AIDP), and Miller Fisher Syndrome [2]. The estimated yearly incidence per 100,000 people is between 0.6 and 2.4. Men are over 1.5 times prone to develop GBS compared to women. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP), registers 90% of cases in North America and Europe [3]. The latest in-hospital mortality rate is 2.7%, and problems are more likely to occur in people with respiratory muscle weakness, 15% to 20% of individuals experience severe permanent disability [4]. In many cases, this syndrome has been linked to causes like viral infection, surgery, blood transfusions, and mycoplasma infection [5]. Acute GBS begins in the toes or tips of the toes of the foot. After, it occasionally gets worse. Weakness in the arms and legs, trouble walking, and difficulty in climbing stairs [6].

The exact aetiology related to GBS is still unknown, but there are some evidences which have shown that Antecedent infections are the most common cause of Guillain Barre Syndrome. Respiratory problems or Gastrointestinal infections are observed in more than two third of GBS patient during six weeks of appearance of symptoms. This strongly shows that GBS is an immune-mediated, postinfectious illness. The most commonly identified causative organism is Campylobacter jejuni. Various other common pathogens that are likely to precede GBS are Cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae and Zika virus [6,7]. Numerous vaccines have also been thought to as potential GBS triggers, including the influenza

A H1N1, rabies, meningococcal, live-attenuated yellow fever, hepatitis A and B, smallpox, polio, MMR, tetanus-diphtheria, and H. influenzae type B vaccines [7].

GBS is immune driven neuropathic disease. In GBS patients, it is observed that the blood sample contains anti ganglioside- antibodies, inflammatory cytokines and T- lymphocytes. Neuronal membrane contains majority of gangliosides. The pathogenic and homologized antigens contribute in molecular mimicry by triggering a cross-reactive immune response. Two main things must happen in order for a cross-reactive immune response to be triggered against homologized antigens through the molecular mimicry process. The microbial antigen and the homologized antigen must be comparable for immune responses to cross-react [8]. The second prerequisite is that the microbial antigen and the autologous antigen must differ sufficiently. In some patients, anti-ganglioside antibodies have been discovered to be induced by the lipopolysaccharide (LPS) that is present in the walls of C. jejuni cells. The LPS structure suggests that it is in charge of molecular mimicry. Usually, they work in a complement-dependent manner to mediate the disease's course [9].

GBS treatment depends on the patient's disease condition whether it is acute, sever or chronic [10]. Plasmapheresis and Intravenous Immunoglobulin (IVIg) are proven successful treatment and has been used since several years [10,11]. PE and IVIg are most effective when it is used within first few weeks especially in adults and paediatrics patients. The recovery of patient is accelerated with the use of PE and IVIg [12]. Fresh frozen plasma or albumin is infused by replacing the plasma of diagnosed patient. Plasma exchange is most successful when, initiated in primary two weeks of diagnosis [13]. The beneficial regimen consists of five session of plasma exchange over a period of 2 weeks for the patient who are unable to walk [14].

Intravenous immunoglobulins are extracted from reservoirs of purified immunoglobulin from several healthy individuals. Mechanism of IVIg include multiple functions which involves neutralization of activated complement, suppression of proinflammatory cytokines and regulating Fc receptor expression and activity in macrophages [15]. The effective dose of IVIg include 0.4g/kg/day of bodyweight given for 5 consecutive days of treatment [16]. The adverse event associated include renal failure, myocardial infraction, vomiting [17].

Supportive care is needed to prevent complications including respiratory insufficiency, pain, immobility and autonomic dysfunction. Respiratory function can be monitored through measuring vital capacity and maximum expiratory capacity. Endotracheal intubation and mechanical ventilation are needed when vital capacity falls below 15mL/kg. Mortality due to autonomic dysfunction can be controlled by monitoring blood pressure and pulse rate [17].

II. Case:

A 31-year male patient was brought to Tertiary Care Hospital with the clinical presentation of fever without chills, all extremities weakness and pain while movement. Patient suddenly felt loss of sensation and weakness in muscles when he was asleep. He was immediately brought to hospital. Patient had no medical or medication history. On examinations patient had fever (100°F). Muscle power was decreased of both upper limbs and lower limbs. There was no shoulder movement and finger grasp were weak. On examination of deep tendon reflexes (DTR) it was observed that bilateral knee reflex and plantar reflex were low. Neck, hip and trunk flexors were also weak. Laboratory investigation showed Haemoglobin: 13.3gm/dl (13.5 – 18 g/dl), lymphocytes: 11% (20 – 40%), Neutrophils: 82% (30 – 70%), Red blood cells: $4.07*10^{12}$/Litre (4.7 – 6 X $10^6/mm^3$), C. Reactive Protein: 7mg/dL (0.3 to 1.0 mg/dL), Alanine aminotransferase: 79 U/L (4 to 36 U/L) and Serum potassium: 3.0mmol/L (3.5 – 5.5 mmol/l). The Nerve conduction study was performed which was suggestive of generalized motor demyelinating (+Axonal) polyradiculoneuropathy and Guillain-Barre Syndrome. During the course of hospitalization patient was given injection ceftriaxone (1gm once daily), injection pantoprazole (40mg once daily), injection ondansetron (2ml once daily) and injection optineuron (1 ampoule) in normal saline (0.9% at the rate of 50ml/hour). Syrup Potchlor (30ml with half glass of water thrice a day) for first three days of hospitalization. From fifth day, injection pantoprazole and injection ondansetron were replaced by combination on Pantoprazole 40mg+Domperidone30mg (twice a day). 5 cycles of plasmapheresis also known as plex cycles was given to patient during 10 days of hospitalization. Physiotherapy was given along with the plasmapheresis cycles which helped in early recovery of patient. These improved the condition of patient and muscle power was back to normal. On discharge patient was given multivitamin tablet (once daily) for 15 days and was advised to visit back to outpatient department for follow up after 15 days.

III. Discussion:

Guillain barre syndrome is acute demyelinating polyradiculoneuropathy. The characteristic features of this disease are acute symmetric, flaccid paralysis and sensory changes. There are various causes of the disease such as vaccines, viral infections, surgery, blood transfusion or mycoplasma infection. In our patient the cause of GBS was idiopathic. The pathophysiology of idiopathic GBS is still not well studied. Patient had no medical or medication history and arrived hospital with clinical manifestations like fever, muscle weakness, muscular pain and loss of sense in extremities were observed in the patient. The diagnosis was made on the basis of Electromyogram test (EMG) and Nerve conduction study (NCS) which was suggestive of generalized motor demyelinating (axonal) polyradiculoneuropathy and GBS. The effective treatment for the above manifestations was done through plasmapheresis. The patient was given 5 plasmapheresis cycles during the course stay. The cycles were given on Day 01, 04, 06, 08 and 10 days. The mechanism of plasma exchange is not clearly understood, but it helps in removing the neurotoxic antibodies, complement factors and other inflammatory humoral mediators which are responsible in immunopathogenesis of GBS. Fresh frozen plasma is infused by replacing the plasma of diagnosed patient. Each session includes plasma exchange of 50ml/kg of bodyweight.

Along with the plasmapheresis, supportive care measures like patient counselling and rehabilitation techniques can be taken into consideration. Patient's body mass index (BMI) was low. So proper nutritional support should be given to patient to compensate nutritional levels. Patients with GBS develop hyper metabolic or hyper catabolic state. So, they must be advised high protein diet. Continuous monitoring of weight, essential proteins should be checked [18]. Neuropathic pain and reduced flexors caused immobilization in patient. The patient was provided with physiotherapy during the course of hospitalization and post hospitalization. During the hospital stay, physical therapies like Dynamic quads stretch, spot marching, bed side sitting, gripping, static quads, bridging and trunk rotation was given to the patient which helped the patient in faster recovery [19]. Sensory loss being the key finding can be treated by balancing exercises. Walking aid were also used.

IV. CONCLUSION

GBS is a disorder of peripheral nerves and can cause sensory symptoms. Immobilization, numbness and weakness of muscle, occurs mainly in the patients suffering from GBS. The cycles of plasmapheresis successfully treated the symptoms. Continuous physiotherapy sessions were beneficial in-patient recovery. The pathophysiology of idiopathic GBS is still unknown and remains area of concern. Research should be done in this area so that better therapeutic agents can be studied for effective treatment of idiopathic GBS.

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VI. CONFLICT OF INTEREST: There are no conflicts of interest among the authors.

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