ejpmr, 2020,7(7), 770-772

SJIF Impact Factor 6.222

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Report

ISSN 2394-3211

EJPMR

A CASE REPORT ON GUILLAIN BARRE SYNDROME

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Article Received on 30/04/2020 **Article Revised on 20/05/2020** **Article Accepted on 10/06/2020**

ABSTRACT

Guillain Barre Syndrome is a rare autoimmune disorder having unknown etiology but with clear pathogenesis of neuronal self damage in peripheral nervous system. Our case report about a male patient having diagnosed with GBS with no history of infection other than trauma (fall from pole) explains that the amalgam of etiologies doesn't vindicate the extant studies on GBS. Poor prognosis of GBS with minimal diagnostic patterns is rarely fatal with respect to their complications but requires extensive studies on it for improvement of patient healthcare.

KEYWORDS: GBS, etiology, poor prognosis, complications, fatal.

INTRODUCTION

Guillain Barre Syndrome or acute polyradiculoneuritis, first described in the year 1916 as a post infectious autoimmune disorder is now considered as a clinical syndrome of different evidenced immunological mechanisms involving motor, sensory and autonomic nerves dysfunctionality; of them acute inflammatory neuropathy is prominent among its subtypes.

According to World Health Organization (WHO) overall incidence of GBS is 0.4 to 4.0 people per 100000 per year across the world.[1] People of all ages can be affected but it is more common in adults and in males.[1] According to Indian Journal of Medical Research 47% male and 19% female and at about 13% as highest mark for 60years or above age group are affected with GBS.[2]

Etiology of GBS is unclear but can be explained with antecedent events such as severe infections (by Campylobacter jejuna and others such as Cytomegalovirus and Epstein Barr virus; due to alcohol; diabetes and poor diet[3]), trauma, immunopathogenesis i.e., antibodies get involved in activation of T cells and it's components as the immune response and damages the axonal nerve terminals of neurons in PNS leading to inflammation and demyelination.

Diagnosis of GBS is difficult and is based on clinical examination & history of patient along with electro diagnostic testing (nerve conduction/ motor conduction tests) and CSF analysis. Acute GBS firstly shows numbness from finger tips of foot and gradually worsens to difficulty in walking, weakness of arms and legs then severity shows paralysis of neuromuscles.

Subtypes of GBS are acute motor axonal neuropathy (AMAN) and acute inflammatory demyelinating polyneuropathy (AIDP), presented with symptoms such as weakness of limbs, areflexia and paralysis. Other subtype is the combination of both AMAN and AIDP known as "Miller Fisher syndrome".

Complications of GBS are determined by the extent of infectious/ inflammatory condition and time taken between onset and its identification. They include autonomic, cardiovascular, respiratory, renal, treatment associated complications.

Effective treatments for GBS are intravenous immunoglobulin and plasma exchange with supportive medical care.

CASE PRESENTATION

A 23 year male patient presented with the chief complaints of weakness in bilateral upper and lower limbs associated with burning sensation since 2 days, high grade fever associated with chills and rigors since 4 days. The patient had history of trauma 2 years back. He had no history of malena /hematuria/burning micturition/pedal edema, no h/o allergies to drugs and no other co-morbidities. The patient was hospitalized for 5 days. At the time of presentation the patient had bradycardia (43-45beats per minute) and motor weakness with the power of left upper limb 2/5, left lower limb 1/5, right upper limb 2/5 and right lower limb 4/5.

Investigations

Examination	Value	Normal Limits
Hemoglobin	12.1	14-18 g/dl
PCV	35.6	40-54%
WBC	8100.	4000-11000cells/L
RBC	5.87	$3.8\text{-}5.8\times10^{12}$/L
Platelets	345×10^{9}	$150\text{-}450\times10^{9}$ cells/L
Blood Urea	3.33	2.49-7.49 mmol/L
Serum creatinine	79.56	61.88-123.76µmol/L
Sodium	140	135-145 mmol/L
Potassium	4.0	3.5-5.0 mmol/L
Chloride	100	95-107 mmol/L
ESR	20	1-15 mm/hr
Total Bilirubin	11.9	3.42-20.52µmol/L
Direct Bilirubin	5.13	1.71-6.84µmol/L
SGPT	0.53	0-0.833 µmol/s·L
SGOT	0.41	0.083-0.833 µmol/s·L
ALP	0.8	0.83-2.5µmol/s·L
Total Proteins	86	55-80 g/L
Albumin	45	35-50 g/L
Globulin	41	28-35 g/L
A/G ratio	1.0	1.7-2.2
2D ECHO	Normal study	
CT Brain Plain	Normal study	
Malaria parasite : V&F	Negative	

Other investigations like viral markers, HIV, HbsAg and HCV were found to be negative, MRI spine cervical was found to be a normal study and ECG shown Brady arrhythmia. The patient was diagnosed with Guillain-Barre syndrome as per Nerve conduction study report i.e., Motor neuropathy of mixed type (Demyelinating & Denervating).

Vitals during the hospital stay: On examination patient was conscious, coherent, oriented. Throughout the stay (day 1-day 4) temperature was consistent 99^{0}F; pulse rate decreased; blood pressure was normal; decreased power of muscle tone; deep tendon reflexes were absent; mildly dehydrated; muscle trunkal weakness is present. On day 2 CSF analysis was advised by the physician and on day 3 CSF sample was collected by lumbar puncture (spinal tap) procedure. The report showed elevated levels of protein in CSF which is suggestive of GBS. On day 5 of the hospital stay the patient had developed complaints of shortness of breath, BP not recordable , RS-B/L crepts present, SpO_2 -85%@ 6 lit O_2. With further fall of saturation patient was intubated with 7.5mm ET tube for Mechanical ventilator. On being treated with Adrenaline, Atropine and Vasopressin the patient had not responded. CPR was done. BP not recordable, pulse not felt, pupils dilated and not reacting to light. ECG shown flat and patient was confirmed dead.

TREATMENT: During the hospital stay patient was treated with Inj.Solumedrol 1gm TID, Inj.Thiamine 1amp BD, Inj.CelrimTz 1.25 gm BD (Antibiotic), Antacids, Anti emetics, Analgesics and physiotherapy assistance was given. IVIg 25 ml with 500ml NS (IV Immunoglobulin) was started on day 4.

DISCUSSION

GBS is an idiopathic complicated neurological disorder with acute and chronic types. Mortality is rare. It is an acquired immune mediated inflammatory disorder which results in demyelination and axonal loss of peripheral nervous system. This may be acute or chronic, permanent or temporary damage. Pathogenesis of GBS involves anterior and posterior junction neurons' root deformities leading to decrease in conduction velocity & deficiency of the blood nerve barrier resulting in myelin sheath abnormalities (and phagocytes release and death in rare cases).

Depending on the extent of demyelination clinical hallmarks such as weakness primarily in lower limbs, ascending paralysis usually symmetrical, loss of cranial nerve conduction are shown. The usual complaints with the above conditions are depressed or absent muscle stretch reflexes, difficulty in walking, running, climbing upstairs, then progresses to upper extremities.

Complications of GBS determine the recovery rate of the patient. They include:

a) Autonomic dysfunction: Unknown physiology.
b) Cardiovascular complications:
   - These are affected by involvement of autonomic dysfunction in 2/3rd of GBS patients.
   - Bradyarrthmias occurred up to 50% in GBS patients and gone to extent of atropine administration in 7-34%.[5] In severe cases irreversible severe asystole was noted.[6]

- Myocardial inflammation or damage triggers cardiac arrest in some patients with prodigal explanation.[5,7]

c) Respiratory complications:
- Functional failure of respiratory muscles in GBS patients is fatal. Studies focusing on prevaricate factors of respiratory failure show demyelinating GBS, delay in hospital admission after onset, facial/bulbar weakness etc.[8]
- The mortality rate in patients with respiratory distress and on mechanical ventilation support is quite less i.e. 6-15%[9] which indicates a high recovery rate of about 74%[10] in GBS patients and even showed no or mild disabilities after recovery.[11]

d) Others :
- Complications of immune response caused post infection
- Short term & long term complications.

e) Treatment associated complications:
- GBS is typically treated with immunotherapy and supportive medical management than with plasmapheresis.

Suggestive treatment method is chosen based on following factors:

Plasmapheresis	Immunotherapy
Not recommended in children	Recommended therapy for all age groups
Cheaper	Costly
Required special equipment	No special equipment is needed
Complications include pneumonia, hypotension, hypocalcaemia etc [12]	Complications include hemolytic anemia in Non O – blood type patients.[13]
Risk in patients with DVT, autonomic cardiovascular instability because of large volume shift	Risk of splenic rupture in patients with cytomegalovirus infection, cerebra vascular diseases etc.

Treatment given in case of this patient is Immunotherapy (IVIg) because the patient is mildly dehydrated and with autonomic cardiovascular instability having bradyarrthmias whose condition is not suitable for plasmapheresis therapy.

CONCLUSION

The mortality rate of GBS is 6.4% as per 2019.[14] Complications resulting from idiopathic and suspecting causes like trauma, delayed hospitalization, lack of awareness regarding onset of symptoms, severe infection may undermine the prognosis of the patient resulting in death which emphasizes us to focus on the patient care for better clinical outcomes.

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