

CASE REPORT ON GUILLAIN - BARRE SYNDROMEDr Sreelekshmi Vinu¹, Dr Sinchu C Maniangatt^{2*}¹PHARM D, Clinical Pharmacist, PRS Hospital Trivandrum, Kerala, India.²MBBS, DNB(MED), DNB (NEURO) Fellowship in Stroke PRS Hospital, Trivandrum, Kerala India.^{2*}Corresponding Author: Dr. Sinchu C Maniangatt

MBBS, DNB(MED), DNB (NEURO) Fellowship in Stroke, PRS Hospital, Trivandrum, Kerala India.

Article Received on 21/09/2021

Article Revised on 11/10/2021

Article Accepted on 31/10/2021

ABSTRACT

Guillain-Barre Syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy in which the body's immune system attacks the part of peripheral nervous system resulting in progressive weakness and paralysis. Our case report is about a 36 year old female patient with no known co-morbidities who was admitted with weakness of both lower limb and associated with numbness of palms and soles. Diagnosis of GBS was based upon the clinical examination and nerve conduction study (NCS) which showed features of acquired demyelination with conduction block in both upper limb and lower limbs. GBS is a life threatening disorder and need timely treatment and supportive care with intravenous immunoglobulin therapy or plasmapheresis. There is no known cure for GBS, but treatments can help to improve the symptoms of GBS and shorten its duration.

KEYWORDS: Plasmapheresis, GBS, NCS, IvIg.**INTRODUCTION**

Guillain barre syndrome, was first described by Landry in 1859. It's an autoimmune demyelinating condition involving ascending weakness and paralysis. GBS clinical spectrum is heterogeneous and most commonly encountered type is Acute demyelinating polyneuropathy (AIDP). Other types are Acute motor axonal neuropathy (AMAN), Acute motor sensory axonal neuropathy (AMSAN) and Miller Fisher Syndrome.

Cerebrospinal fluid analysis in which increased level in CSF protein without elevation in CSF cell count is the characteristic feature of GBS. Etiology of GBS is unclear but is related with antecedent events such as severe infections like Campylobacter jejuna, Epstein-Barr virus, Cytomegalovirus, due etc. Diagnosis of GBS is difficult and is based on clinical examination and history of patients with electro diagnostic testing, nerve conduction test and cerebrospinal fluid analysis.

CASE PRESENTATION

A 36 year old female patient with no known co-morbidities was admitted with weakness of both lower limb followed by numbness of palms and soles. At the time of admission she had grade 1 power of both upper limb and lower limb and had features of autonomic instability. Nerve conduction study on the day of admission revealed features of acquired demyelination with conduction block in both upper limb and lower limb. She underwent emergency management and was started on IvIg(0.4g/day), as she had all features of Guillain Barre syndrome (AIDP). After the full course of

IvIg(0.4g/day) she had stabilisation of weakness and CSF study revealed albuminocytological dissociation. After stopping IvIg (0.4g/day) for 5 days she had progression of weakness of both upper limb and with involvement of bulbar muscles. Hence she was started on PLEX (Plasmapheresis), she underwent 6 cycles of PLEX on alternate days and found to have stabilisation of weakening of legs and improvement in power of both upper limb. She was intubated and ventilated before the initiation of next PLEX and started to wean off from ventilatory support, after completion of PLEX. As she had recurrence of weakness of both upper limb and bulbar muscles again started on PLEX in view of treatment related fluctuation (TRF). After 2 cycles of PLEX, she had improvement in both upper limb and mild impairment in lower limb. During the course of her hospital stay, she was treated with Inj Methylprednisolone for 5 days. Repeated NCS was done which revealed features of severe demyelinating polyneuropathy and radiculopathy in bilateral upper limbs with inelicitable sensory and motor conduction from all lower limbs bilaterally. She completed 5 cycles of PLEX in second cycle and after that she had no worsening of symptoms and was weaned off oxygen support and tracheostomy. She also developed UTI with Klebsiella and was treated with antibiotics according to culture and sensitivity results.

DISCUSSION

Guillain- Barre syndrome is an inflammatory disorder of peripheral nervous system and most common cause of acute flacid paralysis. This study reports the case of GBS

without any known antecedent history and patient was diagnosed with NCS showing acute demyelinating polyneuropathy with progressive weakening of both upper limb and lower limb and CSF study revealed albuminocytological dissociation. The treatment with Immunoglobulin therapy and plasma exchange was effective in the treatment of Guillain Barre syndrome. During hospital stay she received symptomatic treatment and her condition improved at the time of discharge and she was continued with antibiotics for UTI for 1 week and physiotherapy at home.

Motor System Examination includes evaluation of muscle bulk, tone and strength. It also include assesment of body position, coordination and the presence of involuntary movements. Continuous motor examination through this case helps to identify the improvement in her weakness of both upper limb and lower limb. Not only motor examination but also respiratory status should be were carefully and frequently monitored.

CONCLUSION

Guillain- Barre is a rare serious neurological disorder; because of its abrupt and unanticipated onset. Patient not only face physical difficulties but also emotionally painful periods as well. It is often awful for the patient to accustom to sudden paralysis and depend on others for help with routine daily activities. Prognosis of GBS depend on early diagnosis of the disease condition. Continuous physiotherapy and regular monitoring of sensory and motor system makes a profound effect of patient health style.

ACKNOWLEDGEMENT

I sincerely thankful to our beloved Dr Sinchu C Maniangatt (MBBS, DNB (MED), DNB(NEURO), POST DOCTORAL FELLOWSHIP IN STROKE) (Department of Neurology) for giving this opportunity and immense support for completing this study.

REFERENCES

1. Patel MB, Goyal SK Punnam SR et al. GuillainBarre syndrome with asystole requiring permanent pacemaker: a case report. J of med case rep, 2009; 3: 5.
2. Mishra A, Dave N, Mehta M. Fulminant Guillain barre syndrome with Myocarditis J Family Med Prim Care, 2014; 384-5.
3. Walgard C, Ruts L, Lingsma HF, et al. Prediction of respiratory insufficiency in Guillain-Barre syndrome. Ann Neurol, 2010; 67: 781-7.
4. Ali MI, Fernandez-Perez ER, Pendem S, Brown DR, et Mechanical ventilation in patients with Guillain-Barre syndrome Respiratory Care, 2006; 51: 1403-7.
5. Dhar R, Sti HL, Hahn AF. The morbidity and outcome of patients with Guillain- Barre syndrome admitted to the Intensive Care Unit. J Neurol Sci., 2008; 264: 121-8.
6. Le Guennec L, Brisset M, Viala K et al. Post traumatic stress symptoms in Guillain- Barre syndrome patients after prolonged mechanical ventilation in ICU: a preliminary report .J Peripher Nerv Syst, 2014; 19: 218-23.
7. Saroj Kumar Bhagat, Shrey Sidhant et al. Clinical Profile, Functional Outcome and Mortality of Guillain-Barre syndrome: A Five year Tertiary Care Experience from Nepal. Volume 2019| Article ID 3867946| 5 pages| Doi: 10.1155/2019/3867946. Published date, 02 June 2019.
8. Raphael JC, Chevret S, Hughes RA, et al. Plasma exchange for Guillain Barre Syndrome. Cochrane Database Syst Rev, 2002; 2: CD001798.
9. Sater RA & Rostami A. Treatment of Guillain Barre Syndrome with intravenous immunoglobulin. Neurology, 1998; 51(5): 9-15.
10. Van der Meche FG & van Doorn PA. The current place of high dose immunoglobulins in the treatment of neuromuscular disorders. Muscle Nerve, 1997.