GUILLAIN BARRE SYNDROME: A CASE REPORT

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ABSTRACT

Guillain Barre Syndrome is an autoimmune attack on the peripheral nerve myelin. The result is acute and rapid segmental demyelination of peripheral nerves producing ascending weakness with dyskinesia and hyporeflexia. Epidemiology shows overall frequency is 1.9 cases per 100,000 population. In the present report we describe a case of a 19- year old female presented to our hospital with history of quadriparesis, dyspnea and low grade fever for one month. Blood gas studies showed Respiratory acidosis and ECG Showed left ventricular hypertension revealed GBS. Investigation of the disease findings includes CSF and MRI that showed abnormalities. Treatment can be provided such as administering intravenous immunoglobulin (IVIG) for 2-5 days; plasmapheresis steroids and immunosuppressive drugs are alternatives if IVIG is ineffective. Supportive care includes respiratory support and preventing respiratory infections. 3 clinical features predictive of poor outcome were cranial nerve involvement, Intubation, and Maximum disability at the time of presentation.

Keywords: (GBS) Guillain Barre Syndrome, (IVIG) intravenous immunoglobulin, (CSF) Cerebrospinal fluid, (MRI) Magnetic resonance imaging, (ECG) Electrocardiography.

INTRODUCTION

Gullain Barre syndrome is an auto immune attack on the peripheral nerve myelin. The result is acute and rapid segmental demyelination of peripheral nerves & some cranial nerves, producing ascending weakness with dyskinesia, hyporeflexia & paresthesia.

Patients present with ascending type of flaccid weakness. Epidemiology shows overall frequency is 1.9 cases per 100,000 population. Aetiology shows GIT infection, respiratory Infection and Epstein- Barr virus. Clinical manifestation of weakness, Hyporeflexia and areflexia (83%) are the most common occurrences in GBS. Motor deficit occurs in 94% of cases and sensory paraesthesia occur in about 64%, bulbar involvement (50%) and cranial nerve involvement (50%). Investigation findings include CSF analysis showing elevated proteins, Electromyography showing reduced motor conduction velocities and slow sensory nerve conduction time, and MRI Findings showing surface thickening on the conus medullaris and the nerve roots of the cauda equina. [1]

PATIENT INFORMATION

A 19-year old girl got admitted to Medical intensive care unit with the history of quadriparesis, dyspnea, low grade fever, diplopia and dysphagia for one month. She was diagnosed with Guillain Barre Syndrome. There was no family history of autoimmune disorders. There was no relevant past medical and surgical history.

CLINICAL FINDINGS

On physical examination, she was found to have quadriparesis, dyspnea, low grade fever, diplopia and dysphagia.

DIAGNOSTIC EVALUATION

Complete haemogram revealed low level of haemoglobin. Investigation findings included CSF analysis showing elevated proteins, Electromyography showing reduced motor conduction velocities and slow sensory nerve conduction time, and MRI Findings showing surface thickening on the conus medullaris and the nerve roots of the cauda equina.

THERAPEUTIC TREATMENT

The proposed treatment of this condition was, Inj.colistin, Inj.Meropenam 1gm, Inj. Levofloxacin 750 mg, T.Calcium 500mg, Syp. KCL and Neb. Ipravent.

Table - 1: Blood Investigations

Blood parameters	Patient value	Units	Normal value
Complete Haemogram			
Hb	9	g/dl	12-14
W.B.C	8,500	/µL	4000-10000
Neutrophills	54	%	40-70
Lymphocytes	32	%	25-50
Monocytess	8	%	2-10
Eosinophils	5	%	1-7
R.B.C	4.2	10^6/ µL	3.90 – 5.03
Platelet count	2.5	10^3/µL	150-450
Haematocrit	24	%	12-14
ABG Analysis			
Ph	7.30		7.35-7.45
PCO2	62.8	mmHg	35-45
Po2	65	mmHg	75-100
Biochemistry			
Urea	17	g/dl	15-45
Creatinine	0.4	g/dl	0.6 – 1.4
Na	117	mmol/L	135-145
K	3.6	mmol/L	3.5-4.5

DISCUSSION

Gullain Barre syndrome is an autoimmune attack on the peripheral nerve myelin. The result is acute rapid segmental demyelination of peripheral nerves producing ascending weakness with dyskinesia and hyporeflexia. Patients present with ascending type of flaccid weakness. Epidemiology shows overall frequency is 1.9 cases per 100,000 population. Aetiology shows GIT infection, respiratory Infection and Epstein- Barr virus.

Clinical manifestation of weakness, Hyporeflexia and areflexia (83%) are the most common occurrences in GBS. Motor deficit occurs in 94% of cases and sensory paresthesia occurs in about 64%, bulbar involvement (50%), cranial nerve involvement (50%), muscle tenderness and autonomic symptoms.

The diagnostic Investigation of the disease findings includes CSF analysis, Electromyography, serum creatinine kinase, muscle biopsy, nerve biopsy, serological testing for campylobacter infection and MRI to identify the abnormalities.

Treatment for patients with early stages of this acute disease should be admission to the hospital for observation, because the ascending paralysis may rapidly involve respiratory muscles during the next 24 hours. Patients with slow progression may simply be observed for stabilisation and spontaneous remission without treatment. Patient with rapidly progressive ascending paralysis is administered intravenous immunoglobulin (IVIG) for 2-5 days; plasmapheresis steroids and immunosuppressive drugs are alternatives if IVIG is ineffective. Supportive care includes respiratory support and preventing respiratory infections.

Prognosis of this type of disease condition includes spontaneous recovery that begins within 2-3 weeks; most regain normal muscular function; tendon reflexes are usually the last function to recover; bulbar and respiratory muscle

involvement may lead to death if the syndrome is not recognised and treated. 3 clinical features predictive of poor outcome are cranial nerve involvement, Intubation, and Maximum disability at the time of presentation.

CONCLUSION

This case denotes the clinical presentation and diagnosis of the patient with Guillain Barre Syndrome, it is highly risky in children compared to adults. For this condition managing the respiratory needs, prevention of infections and assistance in relief from general weakness and enhancing Nutritional needs of the patient to be cared for. Prolonged paralysis requires intensive rehabilitation. And it is a type of an autoimmune disorder that adds further uniqueness to this condition.

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