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Case Study on Guillain Barre Syndrome – A Rare Auto immune Inflammatory De-myelinating neuropathy observed at Tertiary Care Teaching Hospital

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ABSTRACT

Autoimmune cell activation causes damage to different cells of the human body. In rare cases this auto immune cell activation destroys the Myelin sheath which wraps around the long nerve cell bodies and leads to the developmentof extremely rare nerve cell related auto immune disorder known as Guillain-Barre syndrome. The present case of 34 years old male patient who was admitted in the general medicine department of a tertiary care teaching hospital with complaints of arthralgia, generalized weakness mediated with tingling sensation started at feet progressing upwards. Treatment included antibacterial agents, and steroidal auto inflammatory agents. After the continuous treatment for a period of 13 days the patient was found to be normal with no major complaints and so, the pharmacist when performed discharge counselling and patient was discharged with a 2 week drug regimen after discharge.

Key words:
Myelin sheath,
Arthralgia,
Immuno-Modulatory agents.

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INTRODUCTION

Guillain-Barre syndrome is a rare disorder in which the body's immune system attacks and destroys the myelin sheath which wraps around long nerve cell bodies much like insulation around a water pipe[1]. In a subgroup of patients with Guillain-Barre syndrome, serum antibodies are found against gangliosides, which reside at high densities in the axolemma and other components of the peripheral nerves[2]. complement activation, infiltration of macrophages and oedema are typical characteristics of affected peripheral nerves and nerve roots in patients with GBS[3].

EPIDEMOLOGY

Although rare, with an incidence of 0.4 to2 per 100,000 Guillain-Barre syndrome has major effects on the healthcare system. The cost of medical care for a patient with GBS has been estimated at up to $318,966. Overall,the cost of treating patients with GBS has been estimated at $1.7 billion per year. Males are affected at a slightly higher incidence than females. Each year, it is estimated 100,000 patients worldwide would contract GBS[4, 5].

SIGNS AND SYMPTOMS

- Weakness and tingling in your extremities are usually the first symptoms[6].
- Muscle pain or difficulty in moving your legs, arms or face.
- Constipation or difficulty in controlling your urine or bowel movements.
- Blurred vision or dizziness
- Heart palpitations.,
- Dusky, pale or blue finger nails.
- Shortness of breath.
- Fainting or difficulty in thinking.
- Sudden paralysis[7].

TYPES OF GUILLAIN- BARRÉ SYNDROME:

- ACUTE INFLAMMATORY DEMYELINATING POLYNEUROPATHY, the most common sign of third is muscle weakness that starts in the lower part of your body and spreads upwards.
- MILLER FISHER'S SYNDROME, in which the paralysis starts in the eyes.
- ACUTE MOTOR AXONAL NEUROPATHY and ACUTE MOTOR SENSORY AXONAL NEUROPATHY[8].

CAUSES /ETIOLOGY

GBS is a post-infectious neuropathy and known to be triggered by certain infections[9]. Infections Campylobacter jejuni, one of the most common causes of gastroenteritis worldwide causes 30% to 35% of GBS cases. Other infectious triggers include Cytomegalovirus, Mycoplasma pneumonia, Haemophilus Influenza, Epstein-Barr virus and HIV. GBS also occurs in patients with lymphoma, Hodgkin disease, and systemic lupus erythematosus; those cases occur more frequently than can be attributed to chance alone[10].

RISK FACTORS

Infectious diarrhoea caused by Campylobacter jejuni and upper respiratory tract infections are the most important triggers[11]. The risk factors include sex, age, Campylobacter jejuni bacterial infection, Influenza virus, HIV or Epstein-Barr virus, Mycoplasma pneumonia, surgery, Hodgkin's lymphoma, Influenza vaccination or childhood vaccinations[12].

COMPLICATIONS

- Breathing difficulties
- Residual numbness or other sensations

- Heart and blood pressure problems
- Severe nerve pain
- Bowel and bladder function problems
- Blood clots
- Pressure sores
- Relapse[13].

CLINICAL FEATURES OF GUILLAIN- BARRÉ SYNDROME

- MOTOR DYSFUNCTION which includes symmetrical limb weakness, neck muscle weakness, palsies of cranial nerves such as III – VII , IX – XII , Areflexia, wasting of limb muscles.
- SENSORY DYSFUNCTION which includes pain, numbness, paresthesia, loss of joint position sense, vibration, touch, and pain distally and ataxia.
- AUTONOMIC DYSFUNCTION which includes sinus tachycardia and bradycardia, other cardiac arrhythmias, HTN and postural HTN, wide fluctuations of pulse and blood pressure, tonic pupils, hyper salivation, anhydrosis or excessive sweating, urinary sphincter disturbances, constipation, gastric dysmotility, abnormal vasomotor tone causing venous pooling and facial flushing.
- OTHERS includes like papilloedema, etc[14].

CASE STUDY

A 34 year old male patient was admitted in the general medicine department with the complaints of fever not associated with chills since 7 days associated with poly arthralgia , generalized weakness associated with tingling sensation started at feet, progressing upwards. He has a past history of numbness, urinary incoherence, giddiness occasionally. His bowel and bladder habits were regular and takes mixed diet. He is an occasional alcoholic and chyne chewer. His sleep and appetite was normal. There is no involvement of sensory disturbances, burning micturition, pain in abdomen, epilepsy, thyroid abnormalities. No significant family history is noted.

PAST MEDICAL HISTORY

There is no record of TB, asthma, coronary artery disease, epilepsy, thyroid abnormalities, diabetes mellitus, hypertension.

RESULTS AND DISCUSSION

1.BLOOD PRESSURE AND PULSE RATE DATA:

Days	**D1**	**D2**	**D3**	**D4**	**D5**	**D6**	**D7**	**D8**	**D9**	**D10**
BP(mm hg)	150/ 100	150/100	170/100	140/90	130/90	120/80	120/80	130/90	140/90	130/90
PR(bpm)	94	90	84	90	84	90	70	90	80	83

2. COMPLETE BLOOD PICTURE

Haemoglobin(11-16)	15.6g	13.2g
WBC(4000-11000)	13300 cells/cumm	11700 cells/cumm
Polymorphs(40-75%)	65%	70%
Lymphocytes(20-45%)	9%	24%
Eosinophils(1-6%)	5%	4%
Monocytes(2-10%)	-	2%
PCV	42%	39%

3. BIOCHEMICAL INVESTIGATIONS

Serum creatinine(0.6-1.4)	1mg/dl	0.7mg/dl
Blood urea(14-45)	28mg/dl	35.6mg/dl

4. LIVER FUNCTION TESTS

Total bilirubin(upto1)	1.2mg/dl	0.8mg/dl
Direct(upto0.25mg/dl)	0.7mg/dl	-
Indirect	0.5mg/dl	-
Alkaline phosphatase (15-116IU/L)	268IU/L	605.1 IU/L
SGPT(upto65IU/L)	162IU/L	482IU/L
SGOT(upto37 IU/L)	138IU/L	247IU/L
Total protein(6-8g/dl)	8.2g/dl	-

5. LIPID PROFILE TESTS

Total cholesterol(140-250)	166mg/dl	162mg/dl
Triglyceride(25-160)	77mg/dl	121mg/dl
HDL(30-165)	63mg/dl	30mg/dl
LDL(80-180)	83mg/dl	107.8mg/dl
VLDL(5-45)	19mg/dl	24.2mg/dl

6. URINE ANALYSIS

Albumin	Traces	Traces
Sugar	Nil	Nil
Pus cells	4-6/HPF	2-6/HPF
Epithelial cells	2-4/HPF	2-6/HPF

7. SERUM ELECTROLYTES

Serum sodium(135-155)	135meq/L
Serum potassium(3.6-5.5)	3.67meq/L

8. OTHER INVESTIGATIONS

CRP	181mg/dl
Rheumatoid factor	Negative
HIV	Negative
HBsAG	Negative
HCV	Non reactive
Dengue serology	Negative
QBC	Negative
Serum uric acid	3.9 mg/dl
C3	196.7 mg/dl
C4	44.1 mg/dl
Anti ds DNA	9.8 IU/ml
RNA	0.453(negative)
CRP	140.3mg/dl

RENAL ARTERY DOPPLER

This test revealed that the patient has renal parenchymal disease of grade2, faintly visualised congenital muscular dystrophy (CMD) and mild hemodynamic changes are seen.

USG ABDOMEN

This test shows some cystitis changes in patient, and bilateral grade 1 RPD.

DRUG CHART

S No	Trade Name	Generic Name	Dose, Route	Frequ-ency	D-1,2	D-3	D-4,5	D-6,7	D-8	D-9,10	D-11	D-12	D-13
1	Inj ceftriaxone	Ceftriaxone	1g IV	BD	✔		✔	✔					
2	Tab Doxycycline	Doxycycline	100mg PO	BD	✔		✔						
3	Tab Enam	Enalapril	2.5mg PO	BD	✔	✔	✔	✔	✔				
4	Inj Lasix	Furosemide	40mg IV	BD	✔	✔							
5	Tab PCM	Acetamino-phen	500mg PO	TID	✔	✔							
6	Tab pantop	Pantoprazole	40mg PO	OD	✔	✔	✔	✔	✔	✔	✔		
7	Tab Pregabalin	Pregabalin	75mg PO	H/S			✔	✔	✔	✔	✔	✔	✔
8	Inj Methyl prednisolone	Methyl prednisolone	1g IV	OD			✔	✔					
9	Tab Prednisolone	Prednisolone	30mg PO	OD					✔				
10	IV IgG	Immunoglo-bulin	5g IV	OD				✔	✔		✔	✔	✔
11	Inj Albumin	Serum albumin	100ml IV	OD						✔			
12	Tab Enalapril	Enalapril	5mg PO	OD						✔	✔	✔	
13	Tab BC	B Complex	1 tab PO	OD								✔	✔
14	Tab Amitriptyline	Amitriptyline	10mg PO	H/S									✔

DAY-1, 2

Patient was conscious and coherent. The patient developed fever arthralgia, generalized weakness and was admitted in hospital. The therapy was initiated with paracetamol(500mg) to reduce hyperthermia and joint pains. Antibiotics such as Ceftriaxone(1mg) and Doxycycline(100mg).Antihypertensives like Enalapril(ACE inhibitors)2.5mg and Furosemide(Loop diuretics) of dose 40mg were added. Pantoprazole(Proton pump inhibitor)40mg was given to reduce H^+ ion concentration in stomach. Patients temperature was afebrile with normal heart sounds and no vascular breath sounds were felt on respiratory examination. All peripheral pulses were felt.

DAY -3

Patient was conscious and coherent. Complete blood count, liver function tests, Bio-chemical investigations, lipid profile

tests, common urine examinations were advised to the patient. All peripheral pulses are felt with normal heart sounds.

DAY-4,5

Patient was conscious and coherent. The patient developed tingling sensation which started at foot and was progressing upwards. Deep tendon reflex is absent on both sides, plantar reflex is negative in patient. The power of the limbs calculated as 3/5 which indicates when resistance is applied, the muscle is unable to maintain the contraction. Pregabalin of dose 75mg is added to therapy and furosemide is removed due to its potential interaction with Enalapril. Also the drug Methyl prednisolone of dose 1g is added to the therapy. It acts as an anti-inflammatory, immunosuppressive agent.

DAY-6, 7

Patient was conscious and coherent with high mental function.IgG immunoglobulin infection of dose 5g was added to the therapy. Doxycycline is removed as it shows potential interaction with ceftriaxone. The remaining therapy was same as DAY-5.

DAY-8

Patient was conscious and coherent. Here the Methyl prednisolone injection is replaced by tablet prednisolone of dose 30mg and also ceftriaxone is removed from the therapy.

DAY-9, 10

Patient was conscious and coherent, Renal artery Doppler was advised to patient. Plasmapheresis was advised to patient as a part of treatment. Injection albumin is added to the therapy as the plasmapheresis. The vitals of the patient are normal. Deep tendon reflex is absent in both the limbs. The dose of the enalapril is increased from 2.5mg to 5mg which is used to control HTN.

DAY-11

Patient was conscious and coherent. . Complete blood count, liver function tests ,Bio-chemical investigations, lipid profile tests, urine analysis, serum electrolytes, serum uric acid ,CRP, rheumatoid factor test were advised to patient. Albumin injection is stopped and immunoglobulin therapy was again continued to the patient. The remaining therapy is same as DAY-10**.** Physiotherapy is advised to the patient.

DAY-12

Patient was conscious and coherent. The vitals was observed to be normal. The power of the limbs is increased to 4/5 and also the weakness of the limbs was also gradually decreased in patient. His mental function was normal. Pantoprazole was stopped and immunoglobulin therapy is continued. Multivitamin B-complex is added to the therapy.

DAY-13

Patient was conscious and coherent. Immunoglobulin therapy was continued to the patient. Amitriptyline of dose 10µg was added to the therapy. The tingling sensation was slightly reduced.

DAY-14

Patient was conscious and coherent.Patient was asymptomatic and stable. Immunoglobulin therapy was stopped. He relieved from weakness. Discharge medication was prescribed and counselling was performed.

CONCLUSION

Guillain-Barre syndrome is a significant worldwide cause of rapidly progressive muscular paralysis. This disease is causing damage to the people of all ages causing long term compilation. Proper management of the patient often the accurate diagnosis with the help of Immuno modulatory agents and steroidal anti-inflammatory agents could be useful to protect the patient from long term complications. Better disease modifying therapies are still required in Guillain-Barre syndrome as still significant fraction of patients in Asian countries are still suffering from this long term neurological disability.

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