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A CASE REPORT ON GUILLAIN BARRE SYNDROME WITH HISTORY OF HIV AND SPINAL TUBERCULOSIS

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ABSTRACT

Background: Guillain-Barre syndrome is a rare autoimmune disorder in which the immune system attacks healthy nerve cells in your peripheral nervous system. Its occurrence throughout the world with a median annual incidence of 1.3 cases per population of 100000, with men being more frequently affected than women. **Case presentation:** A 47 y/o female was brought to the hospital with complaints of weakness, paralysis of both lower limbs since 1 week. The physical findings found were febrile, increased heart rate, weakness and numbness in both lower limbs causing paralysis with areflexia. **Conclusion:** Guillian barre syndrome is a rare autoimmune disorder as it causes weakness, numbness and eventually paralysis of the whole body. Diagnosis can be made by progressive motor weakness and muscle inability to respond to stimulus I.e. areflexia and the history of HIV possess the risk factor in this patient. Current treatment includes plasmapheresis, immunoglobulin therapy and additionally analgesics and anticoagulants in pharmacological therapy. In non pharmacological therapy, physiotherapy is required.

KEYWORDS: Guillian barre syndrome, Human immune deficiency virus, paralysis, areflexia, plasmapheresis, immunoglobulin therapy.

INTRODUCTION

Guillain-Barre syndrome (GBS) syndrome is a rare but serious autoimmune disease triggered by either infectious or non infectious factors in which the immune system attacks healthy

nerve cells mainly peripheral nervous system (PNS) which leads to weakness, numbness, and tingling, and can eventually cause paralysis. The cause of this condition is unknown, but it's typically increased by an infectious illness, such as gastroenteritis (irritation of the stomach or intestines) or a lung infection.[1] The weakness and tingling in the extremities are the first symptoms. These sensations can quickly spread, eventually paralyzing your whole body. In its most severe form Guillain-Barre syndrome is a medical emergency. Most people with the condition gets hospitalized to receive treatment, It is now known to be a heterogeneous syndrome with several variant forms.[2] GBS occurs throughout the world with a median annual incidence of 1.3 cases per population of 100 000, with men being more frequently affected than women, The controlled epidemiological studies have linked it to infection with Campylobacter jejuni in addition to viruses, including cytomegalovirus and Epstein Barr virus.[3] A psoas abscess is defined as a purulent infectious collection within the psoas muscle. The psoas muscle is supplied by venous blood from the lumbar spine and has lymphatics from nearby intraabdominal organs overlying it. Secondary psoas abscesses develop as a result of spread of infection from contiguous structures, such as concurrent vertebral infections.[4] Guillain-Barre syndrome onset is associated with a history of infection, mainly of viral origin. Many patients with GBS describe a febrile illness followed by an ascending paralysis occurring days to weeks later Respiratory infections are the commonest, reported in about 40% of cases within one month before the onset of the disease. About 20% of patients experience gastroenteritis as the antecedent cause. Since 1985, several cases of the disease among HIV-infected patients have been reported, with GBS occurring concomitantly with HIV seroconversion or during the initial phases of infection.[5-12] The co-occurrence of Guillain–Barre syndrome (GBS) and tuberculosis is rare. Even in countries like India, where tuberculosis is common, there is only one case report of co-occurrence of GBS with tuberculosis. We report a case of GBS in association with sputum-positive pulmonary tuberculosis.[13]

THE CASE PRESENTATION

A 47 years old female was brought to the hospital with the complaints of weakness, paralysis of both lower limbs since a week. Patient has no allergies and was non smoker and non alcoholic. Medical history of patient includes HIV, Spinal TB with Psoas abscess. Adequate information of family members is not provided. Upon examination physical findings were found to be weakness, numbness in both lower limbs. HEENT was normal. CVS with S1, S2 (+), no murmurs were noted. Respiratory System with BLAE positive, No signs of wheezing

or crackles were noted. Gastro Intestinal Tract (GIT) was soft with no abdominal distension. In PNS Both lower limb paralysis is seen with areflexia. Vitals were found to be BP 120/90 mmHg, RR 20 bpm, PR 115 bpm, Temp 980F, SpO2 98% Provisional diagnosis was made as Guillain Barre Syndrome. Other blood reports, LFT and RFT is mentioned in Table 1 below.

Table 1: Laboratory data of the patient.

Lab test	Obtained result	Normal value	Inference
Haemoglobin	10.4 g/Dl	13-17 g/dl	Anaemia
RBCs	3.42 mill/cumm	4.5-5.5 mill/cumm	Microcytic hypochromic anaemia
WBCs	9900 cell/cumm	4500-10000 cells/cumm	Normal
Platelets	0.85 lakh/cumm	1.5-4.1 lakh/cumm	Thrombocytopenia.
Neutrophils	76%	40-80 %	Normal
Lymphocytes	39%	20-40%	Normal
Monocytes	01%	02-10%	Low
Eosinophils	07%	01-06%	Relative eosinophilia
ESR	43 mm/hr	<20 mm/hr	Elevated
TT4	12.49 ug/dl	5.5-11.0 ug/dl	Normal
Pus cells	4 hpf	0-3 hpf	Pyuria
Epithelial cells	2 hpf	0-5 hpf	Normal
BUN	11mg/dL	07-20 mg/dL	Normal
S. creatinine	0.4 mg/dL	0.6-1.2 mg/dL	Normal
Direct Bilirubin	0.6 mg/Dl	</=30mg/dl	Normal
SGOT (AST)	60 U/L	5-40 U/L	Elevated
SGPT (ALT)	61 U/L	10-56 U/L	Elevated
Gamma glutamyltransferace	47U/L	<40U/L	Elevated
Folate serum	2.4 ng/ml	1.6-19.5 ng/ml	Normal
PT Test	17.2 sec	11-13.5 sec	Elevated
INR	1.33	<1.1	Elevated
S. Sodium	137mEq/L	135-145 mEq/L	Normal
S. Potassium	3.9mEq/L	3.5-5.1 mEq/L	Normal
S. Chloride	103 mEq/L	98-108 mEq/L	Normal

ECG report: Sinus tachycardia with short PR, Non specific ST and T wave abnormality.

Whole spine screening with contrast: Loss of normal cervical curvature, Diffuse disc bulge at C4/C5 and C5/C6 causing mild to moderate compression over cervical cord.

MRI reports: Altered signal intensity in anterior aspect of D11 vertebrae body appearing T1W and T2W hyperintense healed lesion with fatty degeneration. Altered signal intensity lesion appearing T1W and T2W hyperintense, Superior aspects of L4 vertebral body-hemangioma, Desiccation of D6/D7 and D7/D8 discs noted.

Final diagnosis: Guillain Barre syndrome.

Disease state: Guillain–Barre syndrome (GBS) is an acute monophasic immune-mediated disorder of the peripheral nervous system. The term GBS is a synonymous with acute inflammatory demyelinating polyradiculoneuropathy (AIDP)which is a rare disorder in which the immune system attacks the nerve. Acute bacterial or viral infection may trigger the condition. Symptoms seen are weakness and tingling in the feet and legs that spread to the upper body. Paralysis can occur.

Plan of action: Blood treatments options such as plasma exchange and immunoglobulin therapycan relieve symptoms. Physiotherapy is required.

Nutrition: Nasogastric or gastric tube feeding is instituted early and slowly. High energy (40–45 nonprotein kcal) and high protein diet (2–2.5 g/kg) is recommended so has to reduce muscle wasting and assist respiratory weaning. Continuous enteral feeding is better tolerated than bolus feeding in these patients.

Pharmacological therapy: The detailed treatment was given in table 2 mentioned below.

Table 2: Treatment chart of the Patient.

S.no	Formulation	Brand name	Generic name	Dose	Route	Frequency
1.	Inj.	PANTOP	pantaprazole	40mg	IV	OD
2.	Inj.	ZOFER	ondansetron	4mg	IV	OD
3.	Tab.	GABAPIN NT	Gabapentin, nortriptyline	400mg + 10mg	PO	OD
4.	Tab.	NEUROKEM NT	Pregabalin, nortriptyline	75mg + 10mg	PO	OD
5.	Tab.	FLUMIN-P	fluvoxamine	100mg	PO	BD
6.	Inj.	REJUNEX FORTE	Mecobalamin, niacinamide, pyridoxine hydrochloride	500mcg + 50mg + 50mg (1 amp)	IV	SOS
7.	Tab.	LAMISTAR	Lamivudine, stavudine	150mg + 30mg	PO	OD
8.	Tab.	BENADON	pyridoxine	40mg	PO	OD
9.	Tab.	COMBUTOL	ethambutol	100mg	PO	OD
10.	Tab.	R CINEX	Isoniazid,rifampicin	300mg + 600mg	PO	OD
11.	Tab.	FEBUGET	Febuxostat	40mg	PO	OD
12.	Tab.	THYROX	Liothyronine	75mcg	PO	OD

13.	Tab.	BENFICA FORTE	Alpha lipoic acid, Benfotiamine, Chromium picolinate, Folic acid, Methylcobalamine, myo-inositol, pyridoxine	100mg + 150mg + 200mcg + 1.5mg + 1500mcg + 100mg + 3mg	PO	OD
14.	Inj.	PENTAGLOBN	Intravenous immunoglobulin (IVIG)	10ml in a vial (3vials)	IV	OD(5days)

CASE DISCUSSION

The condition seen in this patient is characterized as a rapidly progressive motor disorder associated with absent reflexes and a raised CSF protein in the absence of the expected cerebrospinal fluid (CSF) pleocytosis that characterized poliomyelitis.[15] It is a rare neurological disorder in which the body's immune system mistakenly attacks part of its peripheral nervous system and the nerves get damaged the brain receive abnormal sensory signals from the rest of the body resulting in unexplained, spontaneous sensations, called paresthesias, that may be experienced as tingling, a sense of insects crawling under the skin called formications and pain in the back or legs.[14] Upon examination the physical findings found were weakness and numbness in both lower limbs causing paralysis with areflexia. Guillain-Barre syndrome (GBS) was first described in 1916.[14] GBS has an incidence of about 1/100,000 across several studies[16,17] in a number of countries. It increases in incidence with age and there is a small predominance of males.[17] Sensory symptoms in the legs usually progress rapidly distal weakness that soon spreads proximally. Lumbar pain is common and may represent inflammation in the nerve roots.[18] Patient upon respiratory system examination, BLAE was positive. No signs of wheezing or crackles. Other systems like CVS and GIT indicated no abnormalities. ECG report has shown Sinus tachycardia with short PR Non specific ST and T wave abnormality. Whole spine screening was done with contrast report revealed Loss of normal cervical curvature. Diffuse disc bulge at C4/C5 and C5/C6 causing mild to moderate compression over cervical cord. MRI reports revealed Altered signal intensity in anterior aspect of D11 vertebrae body appearing T1W and T2W hyperintense healed lesion with fatty degeneration. Altered signal intensity lesion appearing T1W and T2W hyperintense Superior aspects of L4 vertebral body-hemangioma (Hemangiomas are benign tumors that develop from blood vessels).[19] From the above objective evidences the final diagnosis was made as guillain barre syndrome with the history

of HIV and spinal TB with psoas abscess. Generally guillian barre syndrome has no cure. But it can be treated by decreasing the seriousness of the disease. Treatment includes plasmapheresis (plasma exchange) and immunoglobulin therapy. This condition usually worsens progressively for about two weeks. Symptoms reach a plateau within four weeks. Recovery rate is six to 12 months, and for some people it takes as long as three years. Additional drugs include anticoagulants to prevent blood clot and analgesic to relieve pain. Patient with Guillain-Barre syndrome requires physical help and therapy before and during recovery. Movement of arms and legs to help keep your muscles flexible and strong and Physical therapy do help in recovery from fatigue and regain strength and proper movement.

CONCLUSION

Guillian barre syndrome is a rare autoimmune disorder as it causes weakness, numbness and eventually paralysis of the whole body. Current treatment includes plasma exchange, immunoglobulin therapy and additionally analgesics and anticoagulants in pharmacological therapy. In non pharmacological therapy, physiotherapy is required. Diagnosis was made by progressive motor weakness and muscle inability to respond to stimulus called areflexia and the history of HIV possess the risk factor in this patient. Many people with GB syndrome undergo recovery, mortality rate is 4-7%. 40-80% patients are able to walk in about six months. As guillian barre syndrome affects nerves, other complications which can occur include residual numbness, heart and blood pressure problems, developing blood clots and pressure sores as the patient is immobile. A Strong support system of friends and family members, manages the stress of recovery from GB syndrome.

ABBREVATIONS

GBS: Guillian Barre Syndrome

AIDP: Acute Inflammatory Demyelinating Polyradiculoneuropathy

PNS: Peripheral Nervous System

CNS: Central Nervous System

BLAE: Bilateral Air Entry

CVS: Cardiovascular System

SpO2: Oxygen saturation

RS: Respiratory System

GIT: Gastro intestinal tract

HEENT: Head, Eyes, Ears, Nose and Throat.

HIV: Human Immunodeficiency Virus TB : Tuberculosis

C4/C5 and C5/C6: cervical vertebras 4,5,6

T1W and T2W: T1 and T2 weighted image (basic pulse sequences in MRI)

D6/D7 and D7/D8: dorsal vertebra 6,7,8D11 : dorsal spine 11

L4: lumbar vertebra 4

BD: Twice a day

OD: once daily

SOS: when necessary

CSF: cerebrospinal fluid.

ECG : electrocardiogram

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CONFLICTS OF INTEREST

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REFERENCES

1. Wakerley B.R., Yuki N. Infectious and noninfectious triggers in Guillain-Barre syndrome. Expert Rev Clin Immunol, 2013; 9(7): 627–639. PMID: 23899233.
2. Yuki N., Hartung H.P. Guillain-Barré syndrome. N Engl J Med., 2012; 366: 2294.
3. Mazidi M., Imani B., Norouzy A., Rezaei P. Guillain-Barré syndrome: a case report. Int J Hosp Res., 2013; 2(2): 91–93.
4. https://www.sciencedirect.com/topics/medicine-and-dentistry/psoas-abscess
5. L. Hagberg, B. E. Malmvall, L. Svennerholm, K. Alestig, and G. Norkrans, “Guillain-Barré syndrome as an early manifestation of HIV central nervous system infection,” Scandinavian Journal of Infectious Diseases, 1986; 18(6): 591–592. View at: Google Scholar

6. C. A. Thornton, A. S. Latif, and J. C. Emmanuel, “Guillain-Barré syndrome associated with human immunodeficiency virus infection in Zimbabwe,” Neurology, 1991; 41(6): 812–815. View at: Google Scholar
7. T. H. Brannagan III and Y. Zhou, “HIV-associated Guillain-Barré syndrome,” Journal of the Neurological Sciences, 2003; 208(1-2): 39–42. View at: Publisher Site | Google Scholar
8. S. Kumar, M. Alexander, V. Markandeyulu, and C. Gnanamuthu, “Guillain-Barré syndrome presenting in the anti-HIV seroconversion period,” Neurology India, 2003; 51(4): 559. View at: Google Scholar
9. S. H. Ting, J. H. Yeh, and H. C. Chiu, “Guillain-Barré syndrome with HIV infection: a case report,” Acta Neurologica Taiwanica, 2003; 12(3): 139–142. View at: Google Scholar
10. A. Millogo, A. Sawadogo, D. Lankoande, and A. B. Sawadogo, “Guillain-Barré syndrome in HIV-infected patients at Bobo-Dioulasso Hospital (Burkina Faso),” Revue Neurologique, 2004; 160(5): 559–562. View at: Google Scholar
11. H. K. Aggarwal, D. Chakrabarti, N. Nand, Sonia, K. Bharti, and R. P. Verma, “HIV infection presenting as Guillain-Barré syndrome,” Journal, Indian Academy of Clinical Medicine, 2005; 6(4): 341–342. View at: Google Scholar
12. G. de Castro, P. G. Bastos, R. Martinez, and J. F. de Castro Figueiredo, “Episodes of Guillain-Barré syndrome associated with the acute phase of HIV-1 infection and with recurrence of viremia,” Arquivos de Neuro-Psiquiatria, 2006; 64(3): 606–608.
13. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5402507/
14. https://www.ninds.nih.gov/Disorders/Patient-Caregiver-Education/Fact-Sheets/Guillain-Barr%C3%A9-Syndrome-Fact-Sheet#3139 4
15. Haymaker WKJ. The Landry-Guillain-Barré syndrome: a clinicopathologicic report of fifty fatal cases and a critique of the literature. *Medicine,* 1949; 28: 59–141. [PubMed] [Google Scholar]
16. McGrogan A, Madle GC, Seaman HE, De Vries CS. The epidemiology of Guillain- Barré syndrome worldwide: a systematic literaturere review. *Neuroepidemiology,* 2009; 32(2): 150–163.
17. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain- Barré syndrome: a systematic review and meta-analysis. Neuroepidemiology, 2011; 36(2): 123–133.
18. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3910670/

19. https://www.columbiaspine.org/condition/hemangioma/