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Case Report

A peculiar case of guillain-barré syndrome in a young female post-partum

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ABSTRACT

Guillain-Barré syndrome (GBS) is a rare autoimmune disorder. GBS is normally caused by demyelination and axonal degeneration resulting in acute polyradiculoneuropathy. The incidence of GBS has been documented as 12–19 per million population with an incidence of 6-24/ one lakh cases reported in pregnancy. The common presentation of GBS is progressive motor weakness and loss of reflexes. Here we report a case of a young female who shows signs of GBS in the post-partum period.

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1. Introduction

GBS is an acute, frequently severe, and fulminant polyneuroradiculopathy that is autoimmune in nature. With males being at a slightly higher risk than females.¹ It is rapidly evolving areflexic motor paralysis with or without sensory disturbance. Subtype of GBS according to the electrodiagnostic and pathological that is most common is Acute inflammatory Demyelinating Polyneuropathy (AIDP). Additionally, there are two other variants which are severe, Acute Motor Axonal Neuropathy (AMAN) and Acute Motor Sensory Axonal Neuropathy (AMSAN).² When seen in pregnancy it is generally seen in the second or third trimester, but sometimes also in the postpartum period. Delayed diagnosis is common in pregnancy or immediate postpartum period because the initial symptoms can be non-specific.³ The relation between GBS and pregnancy is of special interest due to dramatic GBS symptoms in women facing delivery.

2. Case Report

A 24-year-old female delivered four days back, through normal vaginal delivery, was referred to the casualty, with

complaints of fever since two days and lower limb weakness for two days, acute in onset, progressive in nature, the fever was insidious in onset, continually high grade in nature, culminating in the inability to walk. There was no history of vomiting, headache, altered sensorium. On examination, the patient was found to be acutely ill, anemic, febrile, non-icteric, non-cyanotic and hemodynamically stable. The vitals of the patient were as follows pulse rate at 140 beats per minute (sinus tachycardia), blood pressure taken on the right arm was 112/60 mmHg, oxygen saturation was at 96 percentage and the temperature was at 101°F. She was tachypneic with respiratory rate of 24 cycles per minute. On neurological examination, the patient was conscious and restless, with higher mental functions normal. All cranial nerve examinations were satisfactory. On motor examination, she presents flaccid paraparesis, unable to walk with reduced tone and power in the lower limbs as compared to upper limbs. The lower limb power was 3/5 of hip flexion, 4/5 of knee flexion. The superficial plantar reflex of both limbs are flexor with abdominal reflex present. The deep tendon reflexes of biceps, triceps were of grade-I in both the upper limbs and knee jerk and ankle reflexes were absent in both lower limbs. There was no sensory impairment. No meningeal signs present, cerebellar function is normal. Blood investigations complete

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blood count, renal function test, liver function test, random blood sugar were within normal limits. X-ray chest was normal. Magnetic Resonance Imaging (MRI) of brain with spine screening showed no lesions. Over the next two days, the condition of patient worsened with increased weakness of the lower limbs with power of hip flexion falling to 1/5 and knee flexion falling to 3/5. Subsequent CSF analysis showed increased protein levels (153mg/dl), normal cell type and without pleocytosis. Subsequently she was administered Intravenous Immunoglobulin (IvIG), after which her condition improved evidenced by improved power in her lower limbs with the power of hip flexion and knee flexion improving to 4/5 and 4/5 respectively within two weeks.

2.1. Investigations

Cerebrospinal fluid (CSF) (lumbar puncture analysis)

Table 1:

Glucose	94 mg /dl
Adenosine deaminase (ADA)	14.20 U/L
Chlorides	116mmol/L
Protein total	153 mg/dl

Table 2:

Volume	0.5ml
Colour	colorless
Appearance	clear
Cell count	2 cells/mm ³
Gram and zn stain	Negative

3. Discussion

GBS is acute, frequently severe and autoimmune in nature. It is rapidly evolving areflexic motor paralysis with or without sensory disturbance. Subtype of GBS according to the electrodiagnostic and pathological that is most common is Acute inflammatory Demyelinating Polyneuropathy (AIDP). Additionally, there are two other variants which are severe, Acute Motor Axonal Neuropathy (AMAN) and Acute Motor Sensory Axonal Neuropathy (AMSAN).

GBS is generally seen in the second or third trimester, but sometimes also in the postpartum period. The exact etiology is unclear but it has been noticed that it is seen more commonly in women who have undergone LSCS delivery. GBS can occur at any stage of the pregnancy as well as postpartum period, specifically 2 weeks postpartum due to increase in type IV hypersensitivity.

Treatment of GBS during pregnancy and post partum period is similar to that of non pregnant individuals. Plasmapheresis or IVIG to be given at 1g/kg per day for 2 days or 0.4g/kg per day for 5 consecutive days.

Some of these patients may deteriorate even after the proper treatment, with an initial phase of improvement. Retreatment over 5 days is recommended in these patients. High dosage IvIG is said to not have any harmful impact on the course of pregnancy or produce any teratogenicity. The aggressive treatment using IvIG is safe for a lactating mother.

GBS is a rare occurrence in pregnancy but can be associated with severe co-morbidities if unrecognized, especially respiratory muscle involvement and dysautonomia. Clinicians should show a high index of suspicion if a pregnant woman complains of muscle weakness or breathlessness.

4. Conclusion

To conclude early diagnosis and prompt intensive multidisciplinary supportive care - therapy is advantageous in a GBS-complicated early pregnancy, as well as essential for a favorable outcome for both mother and fetus. Sometimes the treatment should continue for a long time. Every case of limb paresis in pregnant women should be considered as a possible GBS case, even in the early stage of pregnancy.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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