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Case Report

Guillain-Barré syndrome complicating pregnancy: a tragic dual loss

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ABSTRACT

Guillain-Barré syndrome (GBS) is a rare, immune-mediated neuropathy characterized by acute, progressive weakness and areflexia. Although its incidence in pregnancy parallels that of the general population, the third trimester and early postpartum period are particularly vulnerable due to immunological alterations. We report the case of a 24-year-old multigravida at 33 weeks gestation who presented with progressive quadriparesis and respiratory distress. Neurological evaluation revealed flaccid limb weakness with preserved sensation, and cerebrospinal fluid analysis demonstrated albuminocytological dissociation. Nerve conduction studies confirmed the acute motor axonal neuropathy (AMAN) variant of GBS. Despite timely administration of intravenous immunoglobulin, ventilatory support, and comprehensive multidisciplinary management, the patient experienced rapid deterioration, leading to maternal and fetal demise. This case underscores the aggressive nature and poor prognosis associated with the AMAN variant of GBS in pregnancy. Prompt recognition of neurological symptoms, early initiation of immunotherapy, and meticulous respiratory monitoring are critical for optimizing maternal and fetal outcomes. Awareness among clinicians regarding this rare but potentially fatal association is essential to facilitate early diagnosis and improve survival.

Keywords: Guillain-Barre syndrome, Pregnancy, AMAN variant, Immunotherapy

INTRODUCTION

Guillain–Barré syndrome (GBS) is a rare neurological disorder characterized by rapid-onset muscle weakness caused by the immune system attacking the peripheral nervous system. Globally, the incidence of GBS varies, with estimates ranging from 0.16 to 4.0 cases per 100,000 individuals annually.

The incidence of GBS in pregnancy is similar to the general population (approximately 1–2 cases per 100,000 people per year). However, some studies suggest that the third trimester of pregnancy and the postpartum period, especially the first two weeks after delivery (due to an increase in delayed type of hypersensitivity) appear to be the most vulnerable times.[1] The maternal mortality rate is 10% and as high as 35% with intensive care unit admission (Table 1).

GBS is characterized by progressive, ascending paralysis and areflexia with or without abnormal sensory function. Symptoms are preceded by an antecedent event in about two-thirds of patients. About one-third of the bacterial and viral infections, systemic diseases, neoplasia, pregnancy, traumatic injury, and organ transplant. GBS has been linked to antecedent infectious agents like Campylobacter jejuni (most common), Epstein-Barr virus, and cytomegalovirus (Figure 1).[2,3]

GBS encompasses several clinical variants, including acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor and sensory axonal neuropathy (AMSAN), and Miller Fischer syndrome (MFS). Among these, the AMAN variant is seen most frequently in Asia, predominantly affects motor axons and often results in more severe paralysis and delayed recovery (Figure 3).

Table 1: Brighton's criteria for diagnosis of classical GBS.

Criteria and level of diagnostic certainty	1	2	3
Bilateral and flaccid weakness of limbs	+	+	+
Decreased or absent DTR	+	+	+
Monophasic pattern of illness	+	+	+
Onset to nadir of illness - 12 hours to 28 days followed by subsequent plateau	+	+	+
Albuminocytological dissociation: CSF protein >45 mg/dl and cells <50 mm^3	+	Either of 2 are positive	-
NCS inconsistent with GBS	+		-
Alternative diagnosis of weakness has been excluded	+		+

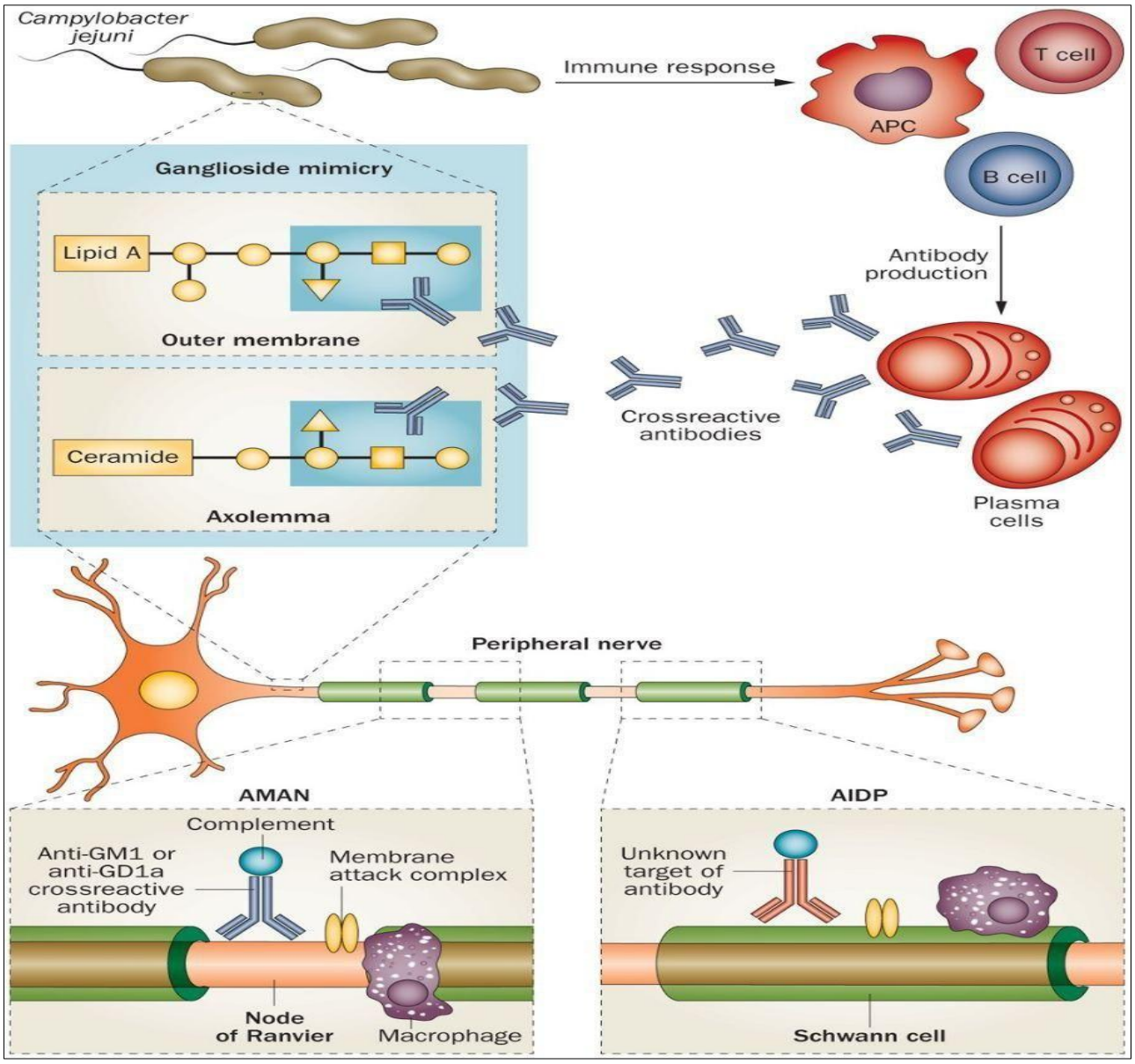

Figure 1: Pathogenesis of GBS.

Disability following GBS is mainly due to neuropathy and pulmonary morbidity due to mechanical ventilation. The most common causes of maternal mortality in GBS are arrhythmia, respiratory failure and pulmonary embolism from deep vein thrombosis. So, termination of pregnancy to prevent morbidity/mortality does not hasten the recovery of maternal disease nor improve maternal outcome. GBS on its own is, therefore, not an indication for termination of pregnancy. Perinatal mortality is mainly due to increased incidence of preterm labour and delivery. There is no increased risk of congenital abnormalities in the baby due to GBS itself. There is no specific therapy for

GBS; However, plasmapheresis and intravenous immunoglobulin (IVIG) administration have been shown to reduce the progression and severity of disease. Here we report a rare and unfortunate case of a multigravida in her third trimester with progressive weakness, developed the AMAN variant of GBS, leading to maternal and fetal mortality despite intensive management.

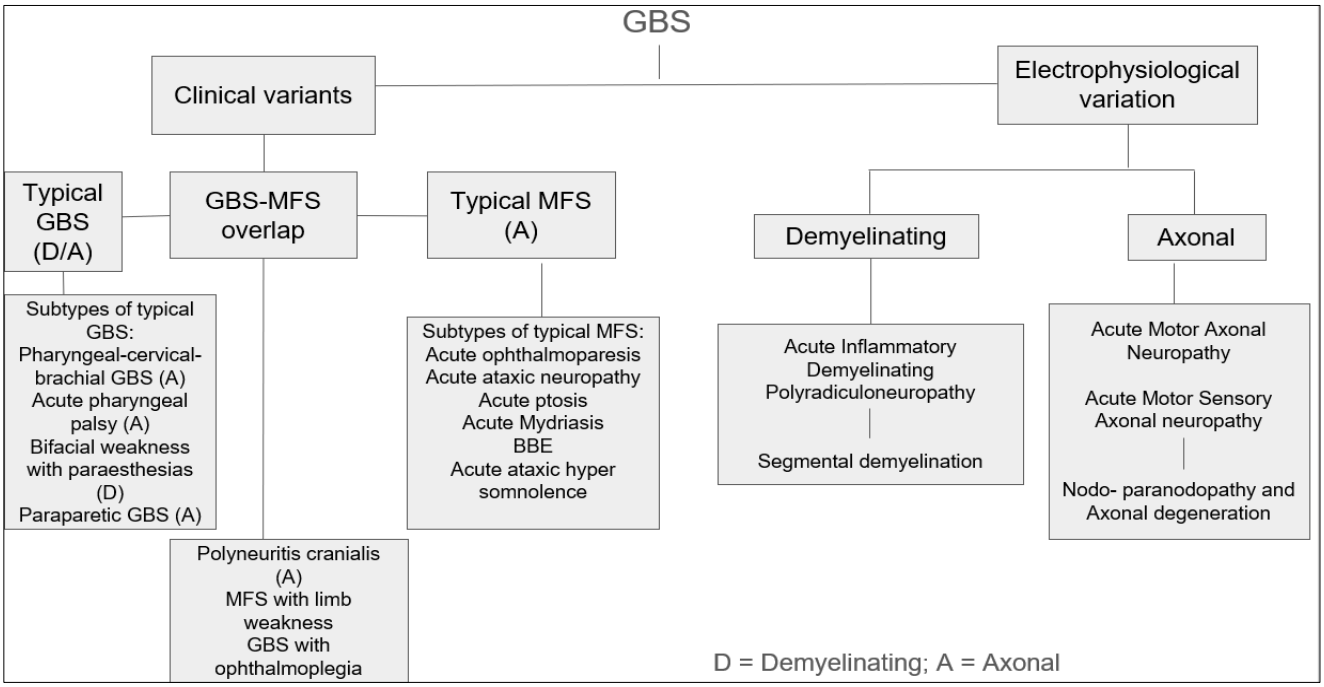

Figure 2: Classification of GBS.

CASE REPORT

A 24-year-old lady, G3P2L2 with 33 weeks of gestation presented to our hospital with complaints of progressive weakness of both upper and lower limbs for the past 1 week, breathing difficulty since past 2 days with a history of fever 1 day ago.

On examination, she was conscious but anxious, had flaccid quadriparesis with power ⅗ in upper and ⅘ in lower limbs and poor neck holding, sluggish deep tendon reflexes with intact sensory system, bowel and bladder and no signs of meningism. She was tachypneic with electrocardiogram showing sinus tachycardia and arterial blood gas analysis indicating carbon dioxide retention thus intubated and was put on mechanical ventilation. Her obstetric examination revealed uterus corresponding to 32 weeks of gestation with reactive non stress test and normal biophysical profile.

Her initial investigations showed normal haematological and biochemical parameters. Magnetic resonance imaging (MRI) brain with whole spine screening was normal. Cerebrospinal fluid analysis revealed albuminocytological dissociation suggestive of GBS. Nerve conduction studies of all 4 limbs confirmed acute motor axonal polyradiculoneuropathy (AMAN) variant of GBS with proximal conduction block.

She was started on Intravenous immunoglobulins and other supportive measures including DVT prophylaxis.[4] However, the patient could not be weaned off ventilatory support due to persistent respiratory muscle weakness. A tracheostomy was performed for prolonged ventilatory management and supportive measures were continued. During her intensive care stay, she developed an episode of atypical seizure activity, followed by sudden cardiac arrest. Despite all resuscitative measures, she could not be revived, leading to the unfortunate demise of both the mother and the fetus due GB syndrome (Figure 3).

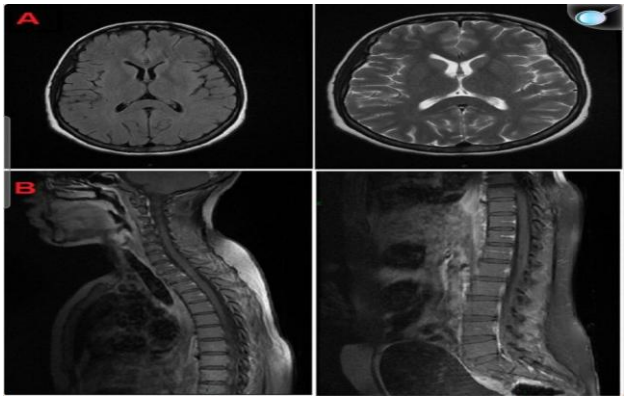

Figure 3 (A and B): Encephalitic and medullary MRI of our patient showing no abnormalities.

DISCUSSION

GBS is rare in pregnancy but carries potentially grave outcomes. The third trimester is the most frequent period of onset or within the first two weeks of puerperium attributed to immune modulation and hormonal changes.[5] The AMAN variant, observed in this patient, is associated with severe motor weakness, rapid progression and poor prognosis.

In GBS, symptoms progress over the first four weeks, followed by a plateau phase lasting for another four weeks. Respiratory issues may arise early, requiring close monitoring for infections, thrombosis, pain and swallowing problems. Respiratory muscle involvement occurs in up to 25% of GBS cases, often necessitating ventilatory support. Autonomic dysfunction, including blood pressure fluctuations and cardiac arrhythmias, further complicates management. Recovery takes about 6-12 months, and around two-thirds of patients recover fully within a year.

Both IVIG and plasmapheresis are proven therapies for GBS and considered safe during pregnancy and show similar efficacy.[6,7] However, outcomes depend greatly on early diagnosis, ICU care and timely respiratory support. In cases requiring ventilatory supporting pregnancy, the risk of premature birth has been noted to be greatly increased.

In a 2020 study conducted by Pakhale et al, patient was diagnosed with GBS at seven weeks of pregnancy, was treated in ICU with plasmapheresis therapy, showing remarkable recovery, but later developed pre-eclampsia with severe features at 32 weeks for which emergency caesarean was done, with no postpartum complications or relapse of GBS. In a literature review analyzing 30 cases of GBS during pregnancy, 22 patients received IVIG or plasmapheresis without maternal or fetal complication.[7]

In a 2013 study by Kim et al, a patient with GBS developed pulmonary embolism after delivery.[8] The other causes of death are lung infection and respiratory failure. In our case, despite appropriate therapy and intensive management, the rapid progression and severity of the AMAN variant led to maternal and fetal mortality.

Early recognition of neurological symptoms in pregnancy is vital. A multidisciplinary team involving neurologists, obstetricians, intensivists and neonatologists is essential for optimizing both maternal and fetal outcomes.

CONCLUSION

GBS in pregnancy, though uncommon, can lead to severe complications and death. The AMAN variant carries a particularly poorer prognosis. Early diagnosis, close monitoring and multidisciplinary management are critical to improve survival. Clinicians should maintain a high index of suspicion when pregnant women present with progressive weakness or respiratory difficulty.

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REFERENCES

1. Goyal V, Misra BK, Singh S, Prasad K, Behari M. Acute inflammatory, demyelinating polyneuropathy in patients with pregnancy. Neurol India. 2004;52:283-4.
2. Hughes RA, Conblath DR. Guillain Barre Syndrome. Lancet. 2005;366:1653-66.
3. Vijayaraghavan J, Vasudevan D, Sadique N, Rajeshwari KS, Pandurangi M, Jayashree. A rare case of Guillain Barre Syndrome with pregnancy. J Indian Med Assoc. 2006;104:269-70.
4. Yerdelen D, Kucukgoz U, Karatas M. Association between acute motor axonal neuropathy and ophthalmoplegia during pregnancy. J Clin Neurosci. 2011;18:999-1000.
5. Mehndiratta MM, Chowdhury D, Goel V, Efficacy and cost effectiveness of current therapies in Guillain Barre Syndrome. J Assoc Physic Ind. 2001;49:459-69.
6. Campos da Silva F, de Moraes Paula G, Dos Santos Esteves Automari CV, Mendes de Almeida DS, Ubirajara Cavalcanti Guimaraes R. Guillain Barre Syndrome: Early diagnosis and treatment is essential for a favourable outcome. Gynaecol Obstet Invest. 2009;67:236-7.
7. Chan LYS, Tsui MHY, Leung TN. Guillain-Barré syndrome in pregnancy. Acts Obstet Gynecol Scand. 2004;83:319-25.
8. Kim H, Ryu J, Hwang JW, Do SH. Anesthetic management for cesarean delivery in a Guillain-Barré syndrome patient - A case report. Korean J Anesthesiol. 2013;64(3):268-71.

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