

Case Report

A case of Guillain-Barre syndrome presenting at third trimester of pregnancy complicated with pre-eclampsia and acute kidney injury

Muralidhara Yadiyal¹, Bhushan C. Shetty^{2*}, Shashank M. S.³

¹Department of Medicine, ²Department of Nephrology, Kasturba Medical College Mangalore, Manipal Academy of Higher Education, Karnataka, India

³Institute of Nephrology, Mysore, Karnataka, India

Received: 28 January 2023

Accepted: 06 June 2023

*Correspondence:

Dr. Bhushan C. Shetty,

E-mail: shettycbhushan@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Guillain-Barre syndrome (GBS) is immune mediated injury of the peripheral nerves. This condition can affect any individuals with some trigger. Here the disease affected the lady in her last trimester of pregnancy which was complicated with pre-eclampsia and acute kidney injury. Later she recovered by multimodal treatment approach, with a prolonged intensive care unit stay. Currently she and her baby are stable with frequent follow up. This case highlights the fact that GBS can affect any trimester of pregnancy and her pregnancy could be a trigger. Further, treatment approach involves multiple specialties including internal medicine, nephrologist, neurologist, physiotherapist and psychiatrist.

Keywords: GBS, Acute kidney injury, Pre-eclampsia, Pregnancy

INTRODUCTION

Acute demyelinating polyradiculopathy (AIDP) known as GBS, an immune mediated disease being linked to a number of infectious diseases. Pregnancy has the same incidence as the general population, which is estimated to be 0.75-2:100,000.¹ Despite being uncommon, it poses a considerable risk to mothers, including the requirement for breathing support and greater fatality rates (10%).² GBS typically manifests 2-4 weeks after a respiratory or gastrointestinal illness, with complaints of weakness in the proximal muscles of the lower extremities and finger dysesthesias. Over the course of several hours to days, this weakness could spread to the arms, truncal muscles, cranial nerves, and respiratory muscles.³ GBS can occur in any pregnant trimester and the postpartum period, but it is most common in the third trimester and the first two weeks after delivery.^{4,5}

Here, we provide a case of a GBS patient who came during the third trimester of pregnancy and had pre-eclampsia as well. Given its rarity, this case is reported. It emphasizes the collaborative involvement of a gynecologist, physician, neurologist and a physiotherapist in managing GBS during pregnancy, which if neglected, can have negative effects on both the mother and fetus. We want to inform the medical fraternity that despite technical hurdles, we managed successfully at our government run district hospital

CASE REPORT

A primigravida in her 30's who had been receiving prenatal care, had progressive bilateral upper and lower limb weakness in ascending manner of 2 days duration with no history suggestive of sensory or bladder involvement, arrived at our emergency ward. She was diagnosed with pre-eclampsia during her antenatal visits

and was on tablet labetalol 100 mg twice daily. An abdominal examination revealed that she was between 32-34 weeks pregnant, and was not in labour.

On examination, she was conscious and oriented with intact cranial nerves. She had flaccid quadriparesis in both of her lower and upper limbs with hyporeflexia and mute plantar reflexes. Sensory system was normal. She was intubated in view of labored breathing at emergency ward and connected to mechanical ventilation. Further, she underwent lower segment caesarian section (LSCS) to deliver a baby boy.

Investigations demonstrated iron deficiency anemia, transaminitis and non-oliguric acute kidney injury. Urinary porphyrins, hepatitis B and C results were likewise negative. Urine and blood cultures were sterile. Acute motor axonal polyneuropathy and acute inflammatory demyelinating were both evident in the nerve conduction investigation. In addition to additional supportive treatments, five days of intravenous immunoglobulin were administered. Meticulous physiotherapy was given to her. In view of prolonged intubation, underwent tracheostomy. She stayed in the intensive care unit with supportive care for almost 44 days and showed signs of improvement. On discharge her acute kidney injury recovered and was able to get up from squatting positing. She is on follow up at our hospital and is fine.

DISCUSSION

GBS is a neurological condition that predominantly causes symmetrical muscular paralysis in most cases.⁶ GBS's pathophysiology is not yet fully understood, although there is some evidence to suggest that it is most likely a result of an autoimmune disorder brought on by an aberrant immune response that targets the peripheral nerves. The aberrant response is thought to be caused by molecular mimicry.

In two-thirds of cases, the causing agent can be found. *Cytomegalovirus* and *Campylobacter jejuni* are the two pathogens most frequently linked to GBS in people who are not pregnant (CMV). Pain, numbness, paranesthesia, or varied degrees of muscle weakness in the legs are the typical symptoms of GBS. The arms and upper body were frequently affected by the symmetrical weakness and strange sensations.⁷

A 70% of patients had dysautonomia, with tachycardia being the most prevalent symptom, followed by urine retention, hypertension with hypotension, arrhythmia, and loss of perspiration. Monitoring of blood pressure and heart rate is advised as a result.

During her antenatal follow-ups, our patient was identified as having pre-eclampsia and was receiving therapy. In the third trimester, she experienced flaccid quadriparesis with respiratory muscle involvement. After

being intubated, she began receiving intravenous immunoglobulins (IVIG). Later, she did not advance but instead hit a plateau from which she has not recovered. She had LSCS, and she gave birth to a boy. Pregnancy is hypothesized to temporarily increase the production of systemically active anti-inflammatory Th2 cytokines that are released at the maternal-fetal interface. These substances aid in the establishment of the "fetal allograft" by enhancing humoral immunity and reducing production of proinflammatory Th1 cytokines, which are harmful to pregnancy in women. Rheumatoid arthritis and multiple sclerosis are two examples of chronic autoimmune diseases where proinflammatory cytokines are definitely involved in the pathogenesis.⁴ GBS during pregnancy is treated similarly to GBS in the general population. The majority of the time, a diagnosis can be determined based solely on clinical evidence, however cerebrospinal fluid analysis and electrophysiological investigations can support the diagnosis.⁸ When necessary, ventilator support, plasmapheresis, and intravenous immunoglobulins (IVIG) are all part of the treatment. It has been discovered that immunomodulation using plasmapheresis and IVIG improves treatment results, with 70-80% of patients completing their recovery.⁹

IVIG was used to treat our patient. Additionally, it has been discovered that patients with demyelinating variety as opposed to axonal kind on nerve conduction testing will have a favorable prognosis. Since pregnancy itself carries a higher risk for thromboembolic events, prophylaxis with low molecular weight heparin and supportive stockings for the prevention of deep vein thrombosis are quite important. Up to 30% of patients develop nosocomial infections, including as pneumonia and urinary tract infections¹⁰

CONCLUSION

Any stage of pregnancy can experience GBS. It emphasizes the joint responsibility of gynecologists, physicians, nephrologists and neurologists in the treatment of GBS during pregnancy, which can have negative effects on both the mother and the fetus if ignored. The prognosis for both mother and fetus when GBS complicates pregnancy is improved by early detection and rapid comprehensive multidisciplinary supportive care.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: Not required

REFERENCES

1. Zilberlicht A, Boms-Yonai N, Cohen K, Bardicef M. Gullian- Barre Syndrome in Pregnancy-A Case Report and Review of the Literature. *Gynecol Obstet (Sunnyvale)*. 2016;6:1.

2. Nelson LH, McLean WT Jr. Management of Landry-Guillain-Barré syndrome in pregnancy. *Obstet Gynecol.* 1985;65:25S-9S.
3. Rees JH, Soudain SE, Gregson NA, Hughes RA. *Campylobacter jejuni* infection and Guillain-Barré syndrome. *N Engl J Med.* 1995;333:1374-9.
4. Sharma SR, Sharma N, Masaraf H, Santa A. Guillain-Barré syndrome complicating pregnancy and correlation with maternal and fetal outcome in North Eastern India: A retrospective study. *Ann Indian Acad Neurol.* 2015;18:215-8.
5. Zafar MS, Naqash MM, Bhat TA, Malik GM. Guillain Barre syndrome in pregnancy: An Unusual case. *J Family Med Prim Care.* 2013;2:90-1.
6. Vasudev R, Raina TR. A rare case of Guillain-Barre syndrome in pregnancy treated with plasma exchange. *Asian J Transfus Sci.* 2014;8:59-60.
7. Vijayaraghavan J, Vasudevan D, Sadique N, Rajeswari KS, Pondurangi M, Jayshree A. Rare case of Guillain-Barre syndrome with pregnancy. *J Indian Med Assoc.* 2006;104:269-70.
8. Van den Berg B, Walgaard C, Drenthen J, Fokke C, Jacobs BC, Van Doorn PA. Guillain-Barre syndrome: Pathogenesis, diagnosis, treatment and prognosis. *Nat Rev Neurol.* 2014;10:469-82.
9. Bahadur A, Gupta N, Deka D, Mittal S. Successful maternal and fetal outcome of Guillain-Barre syndrome complicating pregnancy. *Indian J Med Sci.* 2009;63:517-8.
10. Ropper AH. The Guillain-Barré syndrome. *N Engl J Med.* 1992;326:1130-6.

Cite this article as: Yadiyal M, Shetty BC, Shashank MS. A case of Guillain-Barre syndrome presenting at third trimester of pregnancy complicated with pre-eclampsia and acute kidney injury. *Int J Res Med Sci* 2023;11:2702-4.