

Case Report**GuillainBarre Syndrome in Pregnancy and Post Partum
- Outcome and Survival**Tilotmma R Parate¹, Pradnya Choudhari³, Nilima Wankhede², Ankita Khatri³, Shailesh Lute³**ABSTRACT**

Guillain–Barre syndrome (GBS) is an acute inflammatory demyelinating polyradiculopathy, characterised by progressive, ascending paralysis and areflexia with or without abnormal sensory and autonomic function. GBS complicating pregnancy is a rare event, with documented incidence of 1–3 per 100,000 people annually. Maternal and perinatal mortality rate of >10% is associated with GBS. Herein, we report a patient successful treated who presented with GBS in the third trimester of pregnancy following acute gastroenteritis.

Background :

Guillain Barre Syndrome (GBS) is an acute inflammatory demyelinating polyradiculopathy, characterised by progressive, ascending paralysis and areflexia with or without abnormal sensory and autonomic function¹⁻³. Symptoms are often preceded by a bacterial or viral infection^{3,4}. In rare instances, vaccination may increase the risk of GBS⁵. GBS complicating pregnancy is a rare event, with documented incidence of 13 per 100,000 people annually^{2,3,6,7}. Maternal and perinatal mortality rate of > 10% is associated with GBS. Maternal mortality is usually due to respiratory complications, and neonatal mortality is due to preterm labour and delivery^{3,4}.

Herein, we report a patient who presented with GBS in the third trimester of pregnancy following acute gastroenteritis. This case is reported considering its rarity and significant complications related to perinatal outcome. It highlights the combined role of physician, gynecologist, psychologist and physiotherapist in the management of GBS during pregnancy, which if missed can be detrimental for the mother and fetus.

Case Report :

A 25 years old primigravida with 28 weeks of intrauterine gestation was brought to Casualty with 5 days history of loose motions and Vomiting, followed by progressive weakness of initially lower limbs followed by upper limb and difficulty of breathing. Patient was initially treated in another hospital and was on mechanical ventilation.

She was relatively alright till 25/7/2019, when she developed multiple episodes of loose motions and vomiting. After 2 days she developed weakness in lower limb, which progressed to upper limbs one day later, and patient was unable to move any of her limbs. Patient was admitted in private Hospital. Electrodiagnostic study was suggestive of Severe Acute axonal Motor Polyneuropathy. Patient received IV Immunoglobulin at the rate of 0.4g/kg/day over 5 days. She was intubated as she developed respiratory failure and was mechanically ventilated. Antibiotics (Piperacillin-Tazobactam) and supportive treatment were started. After 4 days, tracheostomy was done. Patient was brought to IGGMC, Nagpur, on 4/8/2019 with tracheostomy tube in situ, on mechanical ventilation and was admitted to Intensive Critical Care Unit (ICCU).

On admission, her general condition was not satisfactory. She was conscious and alert but, restless and anxious. On cardiovascular system examination, no abnormality was detected. On respiratory examination Crepitations were heard over right mammary, infrascapular and interscapular area. She was on mechanical ventilation with tidal volume of 360 ml, PEEP of 8

¹Associate Professor, ²Assistant Professor, ³Junior Resident, Department of General Medicine, Indira Gandhi Government Medical College, Nagpur.

Address for Correspondence -

Dr. Pradnya Choudhari
E-mail : pradnya196@gmail.com

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mm and FiO₂ of 80%. She had no spontaneous respiratory efforts. On neurological examination there were no signs of cranial nerve involvement. There were no signs of meningism. She had flaccid quadriparesis with grade 0 power in both lower limbs and upper limbs, areflexia and absent plantar reflexes on both sides. Sensory system was normal, and bladder and bowel functions were normal. On obstetric examination, the symphysio-fundal height was compatible with her gestational age of 28 weeks and fetus was viable as assessed by bed side ultrasound. Shielded Chest X ray was done and was suggestive of right hilar and left lower zone haziness with left CP angle blunting s/o Ventilator Associated Pneumonia. Her CBC was Normal. Kidney function tests, Serum K, Ca, Mg and liver function test were normal. Inj Piperacilline and tazobacam 4.5 gm I.V thrice in a day was continued and Tablet Azithromycin 500 mg once daily was added. Low Molecular Weight Heparin was started as prophylaxis of DVT. Regularphysiotherapy was done. Patient developed autonomic dysfunction three days after hospital admission in the form of raised blood pressure and abnormal sweating. Tablet Labetolol 100 mg twice in a day and tablet amlodipine 5 mg once in a day was started to control blood pressure. Tracheostomy tube culture was suggestive of Pseudomonas Aeruginosa growth, that was sensitive to piperacillin / tazobactam. Prenatal vitamins and iron were started. Nebulization and periodic tracheostomy tube care was continued. Her clinical and ventilator parameters were closely observed and monitored. Enteric feeding with Ryle's tube was started. Obstetric examination was done daily. After 15 days, for adequate control of blood pressure, dose of Tablet Labetolol was increased to 200 mg BD and Tablet Nicardipine 10 mg BDs was added. Amlodipine was discontinued. Total Parenteral Nutrition was given. After one month of Hospital admission, her power started improving and was of grade 2. Blood transfusions were given as her hemoglobin levels declined. Electrolytes were monitored and corrections were given. I/V. Fluids were given as per the central venous pressure monitoring.

On 18th September 2019, elective caesarean section was done and preterm female baby with 1.7 kg body weight was delivered. There were no signs of birth asphyxia in the newborn. Baby was monitored at NICCU for 15 days and was discharged with normal parameters. Her post partum period was uneventful. Blood culture was suggestive of Candida albicans growth and was put on inj. Fluconazole for 14 days followed by oral therapy. Meanwhile her VAP had worsened, so her antibiotics were changed to Meropenem, Levofloxacin and Linezolid as per the culture sensitivity report.

Once patient had spontaneous respiratory efforts and VAP was treated, weaning protocol was started. She was shifted from ICCU to general ward on 28/11/2019. From 15/12/2019 onwards, she started maintaining SpO₂ on room air and tracheostomy tube was removed on 29/12/2019. Patient was discharged on nutritional supplements and RT in situ on 13/1/2020. She was advised to continue physiotherapy at home and regular follow up. At the time of discharge, the power was of grade 3/5 in all four limbs. Her single breath count was 24. There was no more neck flexor weakness. Her chest x-ray was clear.

Her follow up was taken every 15 days in Medicine OPD. Her power gradually improved. On follow-up of the patient, there was no residual weakness after 3 months.



Figure 1 : Chest X-ray on admission



Figure 2 : Chest X-ray during hospital stay



Figure 3 : Chest X-ray During Weaning



Figure 4 : Chest X-ray at the time of discharge

Discussion :

GBS is a neurological disorder resulting primarily in ascending muscle paralysis, which in most cases is symmetrical³. It usually preceded by a respiratory or gastrointestinal infection in previous 6 weeks in about 60% of patients¹⁰. Our patient has episode of acute gastroenteritis prior to muscle weakness. The organisms that have been implicated are *C. jejuni*, *Varicella Zoster Virus*, *CMV*, *EpsteinBarr Virus* and *M. pneumoniae*. In Brazil, where the

prevalence of Zika virus infection is high, there has also been an association with high incidence of GBS in pregnancy¹¹. GBS in pregnancy has been reported after **trivalent influenza vaccine** administration¹². However our patient didn't receive any vaccination.

Pathophysiology of GBS is still unclear, yet there is some evidence to suggest that this condition is likely to be a consequence of autoimmune disorder. The mechanism for this is unclear but may be a consequence of molecular mimicry, whereby antibodies or T-cell stimulated by antigenic epitopes on the infecting microbe cross-react with gangliosides or neural epitopes.^{4,5,7,8}

GBS has been reported in all trimesters of pregnancy and postpartum period as well.^{2,4} **Vijayaraghavan et al.**, reported a case of GBS at 16 weeks of pregnancy, **Bahadur et al.** at 21 weeks, **Zafar et al.** and **Vasudev and Raina** at 35 and 36 weeks, respectively and **Campos da Silva et al.** reported a case of GBS at 15 weeks of pregnancy.^{2-4,9} Our patient presented in the third trimester and was managed aggressively with complications like Respiratory Failure and Ventilator Associated Pneumonia. The management of GBS in pregnancy is similar to that of non-pregnant population. Diagnosis can usually be made on clinical grounds, but cerebrospinal fluid analysis and

electrophysiological studies can help to substantiate the diagnosis.^{2,7} Our diagnosis was clinical owing to ascending symmetrical muscle weakness and diminished reflexes following a relatively benign gastrointestinal illness and by Electro diagnostic studies; however CSF was not done.

The management of GBS in pregnancy is mainly supportive, with plasmapheresis or IV Immunoglobulin. About a third of patients with GBS will require mechanical ventilation and most common cause of maternal mortality is respiratory failure³. In addition to mechanical ventilation, supportive management also includes identification and treatment of infections, prophylaxis for venous thromboembolism, pain management and management of the psychosocial distress resulting from the disease¹³. Those with autonomic instability, usually manifesting as tachycardia and hypertension, are managed on labetalol because it preserves uteroplacental blood flow. Rehabilitation should be started during pregnancy focusing on pregnancy-associated change in figure and weight gain and post-delivery with training in movements needed for child care¹⁴. Our patient received IV Immunoglobulin and was managed with mechanical ventilation, prophylactic enoxaparin, antibiotics, labetalol, analgesics and physiotherapy. She did well on supportive treatment as evidenced by the clinical and radiological improvement.

Risk of prematurity is low, but occasionally fetal death may occur. Our patient delivered premature baby with low birth weight of 1.7 kg. However outcome was good after proper neonatal care. Poor prognostic factors are serious deficits in muscular balance, the need for assisted ventilation and reduced amplitude of evoked motor potential.⁴

In Conclusion, though rare, GBS can complicate pregnancy. However early diagnosis along with prompt intensive multi-disciplinary supportive care improves outcomes for the mother and fetus. The diagnosis is usually made on clinical grounds supported by CSF examination, serology and nerve studies. Management of GBS is by a

multidisciplinary team and is supportive including prophylaxis for venous thromboembolism, treatment of infections, pain management; management of autonomic instability and management of psychosocial distress and 60% will require mechanical ventilation. Plasma exchange and/or IVIG are definitive management and are better than supportive treatment alone.

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