Guillain-Barre Syndrome in Pregnancy: Case Series and Review of Literature

ABSTRACT:

This is a case series of a healthy woman diagnosed with Guillain–Barre syndrome (GBS) in pregnancy. GBS has been linked to antecedent infectious agents like Campylobacter jejuni (most common), Epstein-Barr virus, cytomegalovirus, etc. The present study is a 6-year retrospective observational study conducted at our institution from January 2014 to December 2020. Medical records of women in pregnancy and puerperium diagnosed as GBS based on clinical, laboratory, and electro-diagnostic features using Brighton's criteria were selected. In the electro-physical study, it was found that 3(37.5%) out of 8 patients had acute motor-sensory axonal neuropathy, 3(37.5%) patients had acute motor axonal neuropathy, and 2(25%) patients had acute inflammatory demyelinating polyneuropathy. In this case series, 3(37.5%) of patients received intravenous immunoglobulin, 3(37.5%) patients received plasma exchange, and 2(25%) patients received only supportive treatment with neither immunoglobulin nor plasma exchange. Early diagnosis and treatment with intensive supportive care improve the prognosis for both mother and fetus. There must be a multidisciplinary approach with gynaecologists, neurologists, physicians, psychologists, and physiotherapists to manage this disease to prevent poor maternal and perinatal outcomes.

Keywords: cytomegalovirus, Guillain – Barre syndrome, motor-sensory axonal neuropathy, Epstein Barr virus

INTRODUCTION:

Guillain–Barre syndrome (GBS) is a rare condition in pregnancy with an incidence of 1.2-1.9 cases per 100,000 annually.(1) GBS is one the most common cause of acute flaccid paralysis affecting all age groups, with an increased incidence in the third trimester and first two weeks of postpartum.(2) The maternal mortality rate is 10% and as high as 35% with intensive care unit admission.(3)

GBS has been linked to antecedent infectious agents like Campylobacter jejuni (most common), Epstein-Barr virus, cytomegalovirus, etc.

Disability following Guillain barre 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.(4) 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.(5) Perinatal mortality is mainly due to increased incidence of preterm labour and delivery. Only one case report of neonatal GBS resulting from maternal disease presenting as flaccid paralysis of all limbs responded to IVIG treatment and recovered in 2 weeks is available.(6) Congenital GBS is an infrequent occurrence.(2)

The diagnostic evaluation is based on the clinical presentation and laboratory and electrophysiological investigations.(2) A lumbar puncture reveals an elevated cerebrospinal fluid (CSF) protein with normal white blood cell (WBC) counts.(7) Nerve conduction studies (NCS) and electromyography (EMG) show an evolving multifocal demyelinating polyneuropathy.

A multidisciplinary approach involving physicians and obstetricians is essential in managing GBS in pregnant women. Apart from specific treatments such as IVIG or plasmapheresis mentioned earlier, attention is to be paid to identifying and treating infective complications, preventing venous thromboembolism, pain management, and managing psychological distress resulting from the disease and anxiety towards the pregnancy.

The following is a case series of an otherwise healthy woman diagnosed with GBS in pregnancy. Patient presentation, diagnosis, treatment and outcome, and literature review are described below.

MATERIALS AND METHODS:

The present study is a 6-year retrospective observational study conducted at our institution from January 2014 to December 2020. Medical records of women in pregnancy and puerperium diagnosed as GBS based on clinical, laboratory, and electro-diagnostic features using Brighton's criteria were selected. Potential GBS cases during pregnancy and 42 days after birth using the international classification of diseases, 9th revision (ICD 10), were identified. All demographic details, including age, parity, time of presentation of symptoms, and mode of delivery, were analyzed.

Epidemiological data were collected, including the duration of symptoms before admission and hospital stay. The pattern of involvement of limbs was recorded. The maternal outcome was analyzed based on the predisposing factors, time of presentation of symptoms, type of symptoms, stage of presentation, type of GBS, need for ventilator support, treatment with plasmapheresis and intravenous immunoglobulin (IVIG). Perinatal outcomes studied were fetal growth restriction, intrauterine fetal demise (IUFD), and neonatal deaths.

RESULTS:

During the study period, there were 98,952 deliveries, of which eight patients were diagnosed with GBS. The incidence was 1 in 1,00,000 pregnant women per year. The demography characteristics are depicted in Table 1. The mean age of our study population was 25.2 years.

All patients presented as stage 1 progressive phase. One patient developed GBS following Varicella-zoster infection, while another patient had COVID-19 infection two weeks before the onset of symptoms, but in the others, the associated cause was unknown.

The duration of symptoms at the admission was 1–15 days, and the duration of admission to the hospital was 3–24 days. Out of 8 patients, six presented in the 2nd trimester, while two presented in the postnatal period.

All 8 (100%) patients presented with sensory symptoms in the present study, but no patient had a sensory loss on examination. In addition, 7(87.5%) patients had lower limb weakness. At

presentation, examination revealed sinus tachycardia with normal blood pressure in all patients. On neurological examination, a lower limb power of 3/5 was found in 5 patients and a power of 4/5 in 2 patients. All patients had a power of 5/5 in bilateral upper limbs. One of them had a bifacial and oropharyngeal weakness without associated respiratory or cardiovascular symptoms. Among these patients, 2(25%) developed lower limb weakness after delivery on postnatal day 2, which improved gradually with conservative management (Table 2). There was no cranial nerve involvement in any patient.

In the electro-physical study, it was found that 3(37.5%) out of 8 patients had acute motor-sensory axonal neuropathy, 3(37.5%) of patients had acute motor axonal neuropathy, and 2(25%) of patients had acute inflammatory demyelinating polyneuropathy. In NCS, decreased compound muscle action potentials (CMAP) were observed in 3 AMAN patients. Prolonged F-wave latency & conduction block (CB) were noted in 2 AIDP patients. Among three patients with the AMSAN variant, the right peroneal nerve was non-stimulable & decreased CMAP was noted in the left peroneal nerve. Motor nerve conduction velocities were reduced in both the tibial & peroneal nerves for all 3 AMSAN variants. Cerebrospinal fluid analysis was done in only 2 (25%)patients and found to have elevated protein and normal white blood cells.

In this case series, 3(37.5%) of patients received intravenous immunoglobulin with a standard dosage of 2g/kg for five days, 3(37.5%) patients received plasma exchange, and 2(25%) patients received only supportive treatment. The mean time from symptoms to the onset of treatment was ten days. No significant adverse effects were observed with IVIG treatment or plasma exchange.

All patients recovered, irrespective of treatment. There was no incidence of maternal mortality in this series. Two patients (25%) had fetal growth restriction, while 2(25%) presented with preterm premature rupture of membrane. The live birth rate was 100%. Two (25%) babies had poor APGAR scores and were admitted to the neonatal intensive care unit. There was no incidence of perinatal mortality or morbidity (Table 3). It was observed that 4(50%) patients were delivered by emergency caesarean section for various obstetric indications, 3(37.5%) were delivered vaginally, and one (12.5%) was delivered by instrumentation for an obstetric indication. The complications of pregnancy are depicted in (Table 4).

Among eight patients, six patients were discharged with the ability to walk unaided. 2 patients were discharged with the ability to walk aided. No patient had any relapse of GBS.

DISCUSSION:

GBS is an autoimmune condition triggered by an infectious agent leading to damage of peripheral nerves. The incidence of GBS in the general population is around 0.75 to 2 in 1,00,000 per year, with increasing incidence with age.(8). In our study, the incidence of GBS was 1.34 in 1,00,000 pregnant women per year.

CAUSATIVE AGENTS:

GBS is usually preceded commonly by a respiratory or gastrointestinal tract infection. The most common organism, C.*jejuni, accounts for* 30% of cases, especially in the younger age group.(9) The primary mechanism by which *C. jejuni* affects the neurological system is the molecular mimicry of the liposaccharide of C.jejuni with our gangliosides.(10) The causative agents, such as *C.jejuni*, present with a more severe clinical course and delayed recovery.(11,12)

Several studies have shown the incidence of GBS with varicella-zoster as a primary infection.(10,13,14) One patient in the present study had a preceding varicella infection two weeks before the onset of weakness of limbs. Studies have shown a gap of 4 weeks from diagnosis to presenting symptoms with a favourable outcome and rapid recovery.(2)

In 2020, Keyhanian et al. reported COVID-19-associated GBS, the possible mechanism being the neurotropism of the virus and the potential pathways through cranial nerves, especially olfactory nerve and hematogenous pathways.(15) In 2021, Wilson et al. reported a case of GBS with respiratory failure following COVID-19 infection.(16) In the present study, one of the patients in the second trimester had a preceding COVID-19 infection with diarrhoea and fever. One week later presented with bilateral lower limb weakness, similar to a case reported by H Zhao et al. 2020.(17) This neurological complication may be due to COVID-19 infection of the enteric nerve plexus, resulting in gastrointestinal symptoms, which serve as a pathway for retrograde infection of the CNS.(15) A similar study reported neuro-ophthalmological manifestation associated with COVID-19.(18) Although the cause could also be direct neuroinvasion of the virus, as recently described by Khan et al., no cerebrospinal fluid analysis

has been done.(19) Although infectious etiologies most often precede GBS, it has been associated with other antecedent events, including vaccinations. Based on the available literature, cases have been reported on GBS following TdAP vaccination, but it was determined with relative confidence that the TdAP vaccination carries a risk of approximately one additional case per 100,000 doses.(20,21) GBS, following meningococcal, poliovirus, influenza virus, and rabies vaccines, is rare.(17) Usually, the disease occurs two to four weeks following the initial event, and if treated within four weeks, significant permanent sequelae can be prevented.

PATHOGENESIS:

The primary pathogenesis is the molecular mimicry between antibodies produced against the organisms with the epitopes of our own body's gangliosides, resulting in specific subtypes.(22) The four major subtypes are acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, acute motor and sensory axonal neuropathy and Miller-Fischer syndrome.(23) Literature has shown that acute motor and acute inflammatory demyelinating polyneuropathy is a more common type of GBS in pregnancy, and Miller-Fischer syndrome is the rarest.(24,25) In the present study, three patients presented with acute motor and sensory axonal neuropathy(AMAN), three patients had acute motor axonal neuropathy(AMAN) and two patients presented with acute inflammatory demyelinating neuropathy(AIDP). None of our patients had isolated sensory neuropathy or Miller-Fischer syndrome. Although, according to the literature, AIDP is the more common type, in our study, the cases in the other two types, AMAN and AMSAN, are more, attributed to the smaller sample size without statistical significance.

In a study by Auger et al., rheumatologic disorder and preeclampsia due to their immunological component were associated with GBS with 9.84 and 2.62 per 1,00,000 person-years.(26) Another study conducted by Swonger et al. also showed the association of GBS with preeclampsia.(27) None of the patients in the present study had rheumatologic or hypertensive disorders.

DIAGNOSIS:

GBS diagnosis is mainly clinical with support of serological, nerve conduction studies and cerebrospinal fluid analysis. Many recent studies have shown that due to the overlap of

symptoms with normal pregnancy, there is usually a delay in the diagnosis, and most patients have irreversible consequences. The early diagnosis in our study is mainly based on clinical signs supported by an electrophysiological nerve conduction study to decide on the type of GBS and assess progression. The most common nerve conduction study finding is slowing or blocking motor nerve conduction with prolonged distal motor latency and prolonged or absent F wave.(24) However, these typical features are usually absent in the early stage of this disease. Albumin-cytologic dissociation in cerebrospinal fluid with elevated protein content and a normal mononuclear leukocyte count are common findings in 9 out of 10 GBS patients and strongly suggest GBS.(22) Studies have also reported that the maternal and neonatal outcome of GBS in pregnancy is excellent if diagnosed early and managed.(25)

In the present study, the most common clinical presentation was new-onset progressive muscle weakness, in contrast to other studies where patients presented with other manifestations like the inability to void, vague abdominal pain, hyporeflexia/ areflexia in involved limbs, pain and numbness over the limbs.(23) Though most of the patients present with prodromal symptoms of respiratory or gastrointestinal tract infections(13) and cranial nerve involvement(28), only two patients in our study had a history of cough with fever and diarrhoea and none with cranial nerve involvement. A study done by Alia Zaidi et al. reported a case of a GBS patient presenting with respiratory muscle weakness resulting in respiratory failure who had a successful vaginal delivery while on a ventilator.(29)

TREATMENT:

Treatment is similar to that of non-pregnant individuals. Based on severity, a multidisciplinary approach should be initiated. Patients should be closely monitored for disease progression, respiratory muscle involvement and autonomic dysfunction.(30) Treatment usually involves supportive measures concomitant with disease-specific therapy. Supportive measures commonly instilled are aggressive physiotherapy, enteral nutrition, monitoring of progression of muscle weakness, and intermittent antenatal fetal monitoring.

Pain relief is better by narcotic agents or acetaminophen, which are safe in pregnancy(28), and all patients in our study received intravenous Tramadol for pain relief during labour and

postpartum. Careful and intensive monitoring should be done as most patients present with tachycardia/ bradycardia or hypotension/hypertension emergencies.(31)

Disease-specific therapy includes plasmapheresis or intravenous immunoglobulins, which is equally efficacious in obstetric and non-obstetric cases is usually started if there is a progression of the disease, especially within four weeks of symptoms for any substantial benefit.(32) In the present study, three patients received IVIG, three plasmapheresis, and 2 received only supportive care. Hukuimwe M et al. concluded that IVIG should be considered the treatment of choice as plasmapheresis treatment requires central venous access and intense monitoring of electrolytes, which can alter blood pressure levels in pregnant patients.(22) In the present study, three patients underwent plasmapheresis in 2nd trimester and improved following treatment. Liu et al. concluded that Plasma exchange is effective in non-ambulatory GBS as it reduces the extent of demyelination and hastens recovery.(33)

OBSTETRIC MANAGEMENT:

GBS per se is not an indication of cesarean section. Though studies have reported worsening maternal symptoms during labour in the acute phase of GBS, early termination of pregnancy did not impact the prognosis of the patient.(2) Vaginal delivery must be planned with adequate analgesia, but there is a lack of maternal bearing down due to muscle weakness. Hence, the second stage of labourmay be shortened.(23) In the present study, four patients underwent a caesarean section for obstetrics indications under general anaesthesia, while four patients had a spontaneous vaginal delivery, similar to studies done by Rabia et al.(8), Volquind et al.(34) and Fernando et al.(35) Further studies are needed to analyze the benefits of caesarean over vaginal delivery and the safety of various modes of anaesthesia in pregnancy (general or epidural anaesthesia).

FETAL COMPLICATION:

In the present study, two patients had preterm delivery. Studies have hypothesized that immunological mechanisms are a cause of preterm birth. A case report by Bhadur et al. concluded that the risk of preterm birth is higher in GBS patients who need ventilator support.(8) In their study of GBS in pregnancy, Chan et al. reported a neonatal survival rate of 95.7%.(36)

However, two patients had intrauterine growth restrictions of unknown cause. Neonatal GBS is rare, but a case report described a baby born with good APGAR

to a GBS mother developing hypotonia, respiratory distress, and CSF confirmed GBS after 12 days.(6)

MATERNAL COMPLICATION:

The major complication following GBS is respiratory muscle weakness, which may lead to respiratory failure and ventilator support. With vigilant ICU monitoring and IVIG, the recovery rate is 100%.(37) The risk of GBS increases in the postpartum period, especially in the initial two weeks, which might be due to a delayed hypersensitivity that flares up the disease; hence, proper care and monitoring are essential during this period.(38) Two patients in the present study diagnosed with new onset GBS on the second postnatal day showed significant improvement with supportive treatment. However, in the cases reported by Kachuru et al.(39) and Aabdi et al. (40), GBS was diagnosed for the first time in the postpartum period and needed IVIG along with supportive therapy for recovery. Proper counselling sessions are essential for these patients due to separation from their infant and difficulty in feeding, increasing anxiety and depression in postpartum GBS patients.36

Relapse can be managed with repeat IVIG; improvement is similar to previous treatment. 70-80% of patients recover fully after delivery.(28) In the present study, of the six patients diagnosed with GBS in the antenatal period, two patients who had undergone caesarean had worsening in the immediate postpartum period but recovered gradually with supportive care. In a similar study by Meenakshi et al., they reported relapsing GBS in a post-cesarean patient. They concluded that the possible mechanism of relapse might be surgery or anaesthesia, which may trigger proinflammatory cytokines.(28) None of the patients in the present study required disease-specific therapy in the postnatal period.

It is known that the incidence of pulmonary embolism in non-pregnant GBS is 1- 13%. As pregnancy itself is a substantial risk factor for thromboembolism, prophylactic anticoagulation should be administered early in GBS pregnant women with poor mobility.(41) In contrast, no patient developed pulmonary embolism in the present study. Ryabinkina et al. concluded that

despite thromboprophylaxis, 52% of patients developed deep vein thrombosis and 12% pulmonary embolism, and the significant risk factors included bed rest for more than three days, ventilator support, infections, and central venous catheter placement.(29) Further research on preventive strategies of thromboembolism is needed. Nomani et al. reported that respiratory tract infections are more common in ICU admissions; around 83% of patients have infections with organisms such as Klebsiella, Acinetobacter and Pseudomonas.(42) Hence, early identification and treatment is essential as, in pregnancy, these infections tend to be more severe.(43)

CONCLUSION:

GBS is rare in pregnancy, and prompt treatment is essential to prevent maternal and fetal morbidity. A high index of suspicion is necessary, and the obstetrician should be aware of the differential diagnosis of GBS in cases with flaccid paralysis. Early diagnosis and treatment with intensive supportive care improve the prognosis for both mother and fetus. There must be a multidisciplinary approach with gynaecologists, neurologists, physicians, psychologists, and physiotherapists to manage this disease to prevent poor maternal and perinatal outcomes.

Consent: Given that our study was conducted retrospectively on documented patients' records with no specific intervention, implied consent was taken.

Ethical approval: Written ethical approval has been collected and preserved by the author(s).

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TABLE 1: DEMOGRAPHIC CHARACTERISTICS:

Demographic details	**N=8(%)**
Age range(years)	25-30
Parity	
Primiparous	3(37.5)
Multiparous	5(62.5)
Gestation at delivery	
Preterm	2(25)
Term	6(75)
Mode of delivery	
Vaginal	3(37.5)
Instrumental	1(12.5)
Caesarean	4(50)

TABLE 2: CHARACTERISTICS OF GUILLAIN BARE SYNDROME:

Characteristics of GBS	**N=8 (%)**
Onset of symptoms	
Antenatal	6(75)
Puerperium	2(25)
Type of weakness	
Lower limb weakness	7(87.5)
Bifacial and lower limb weakness	1(12.5)
Treatment	
Intravenous immunoglobulin	3(37.5)
Plasmapheresis	3(37.5)
supportive	2(25)
Intensive unit care	
Needed	0
Not needed	8(100)
Ventilatory support	
Needed	0
Not needed	8(100)
The mean duration of symptoms (mean days)	9.2
Duration of hospital stay (Mean days)	12.5
Infectious agent	
Varicella-zoster	1(12.5)
Covid19	1(12.5)
Unknown	6(75)

TABLE 3: MATERNAL AND PERINATAL OUTCOMES:

Outcome	**N=8 (%)**
Maternal outcome	
Recovery	8(100)
Death	0
Perinatal outcome	
Live birth	8(100)
Fetal growth restriction	2(25)
Intrauterine death	0
Low APGAR	2(25)
Neonatal intensive care unit admission	2(25)

TABLE 4: COMPLICATIONS OF PREGNANCY:

Complications	**N=8 (%)**
Premature rupture of membranes	2(25)
Preterm birth	2(25)
Postpartum haemorrhage	1(12.5)