Original Research Article

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, one patient had acute motor axonal neuropathy, and one patient 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 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 limbsresponded to IVIG treatment and recovered in 2 weeks is available.[6] Congenital GBS is an infrequent occurrence.[2]

The diagnostic evaluation is based upon the clinical presentation,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 the physicians and obstetricians is essential in the management of GBS in pregnant women.Apart from specific treatments such as IVIG or plasmapheresis mentioned earlier, attention should 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 a review of the literature 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 analysed.

Epidemiological data were collected, including the duration of symptoms before admission and the duration of hospital stay. The pattern of involvement of limbs was recorded. The maternal outcome

was analysed 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 in which 8 patients were diagnosed to have 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, and 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, 6patients presented in the 2nd trimester,whiletwo patients 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,and one of them had a bifacial and oropharyngeal weakness. Among these patients, 2(25%) developed lower limb weakness after delivery on postnatal day 2, which improved gradually with conservative management. (Table 2).

In the electro physical study, it was found that 3(37.5%) out of 8 patients had acute motor-sensory axonal neuropathy, one patient had acute motor axonal neuropathy, and one patient 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.

All patients recovered irrespective of treatment. There was no incidence of maternal mortality in thisseries. 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 tothe neonatal intensive care unit. There was no incidence of perinatal mortality or morbidity (Table 3). It was observed that 4(50%) patients delivered by emergency caesarean section for various obstetric indications, and 3(37.5%) delivered vaginally and one (12.5%) was delivered by instrumentation for an obstetric indication. The complications of pregnancy are depicted in (Table 4).

Among 8 patients, 6 patients were discharged with ability to walk unaided. 2 patients were discharged with ability to walk aided.

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. Among these, 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 primary infection.[10,13,14]One patient in the present study had a preceding varicella infection two weeks beforethe onset of weakness of limbs.Studies have showna gap of 4weeks from diagnosis to presenting symptoms with a favourable outcome and rapid recovery.[2]

In 2020 Keyhanian et al. reported COVID-19 associated GBS and the possible mechanism being the neurotropism of the virus and the possible pathways through cranial nerves, especially olfactory nerve and hematogenous pathways.[15] In the present study, one of the patients in the second trimester had a preceding COVID-19 infection with diarrhoea and fever,and one week later presented with bilateral lower limb weakness, which was similar to a case reported by H Zhao et al. 2020.[16]This neurological complication may be due to COVID-19 infection of the enteric nerve plexus,resulting in gastrointestinal symptoms,which served as a pathway for retrograde infection of CNS.[15] A similar study reported neuro-ophthalmological manifestation associated with COVID-19.[17] Although the cause could be direct neuroinvasion of virus also as recently described by Khan et al. but no cerebrospinal fluid analysis has been done.[18] GBS, following meningococcal, poliovirus, influenza virus, and rabies vaccines, is rare.[16] Usually, the disease occurs two-four weeks after initial infection, and if treated within 4 weeks,significant permanent sequelae are 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.[19] The four major subtypes are acute inflammatory demyelinating polyneuropathy, acute motor axonal

neuropathy, acute motor and sensory axonal neuropathy and Miller Fischer syndrome.[20] 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.[21,22] In the present study, five patients presentedwith acute motor and sensory axonal neuropathy and 2 patients presented with acute inflammatory demyelinating neuropathy and 1 patient had acute motor axonal neuropathy. None of our patients had isolated sensory neuropathy or Miller Fischer syndrome.

In a study conducted by Auger et al., rheumatologic disorder and pre-eclampsia due to its immunological component were associated with GBS with 9.84 and 2.62 per 1,00,000 person-years.[23]But none of the patients in the present study had rheumatologic or hypertensive disorder.

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 overlap of symptoms with normal pregnancy, usually there is a delay in the diagnosis and most patients have irreversible consequences. The early diagnosis in our study was mainly based on clinical signs supported by an electrophysiological nerve conduction study to decide on the type of GBS and assess progression, while a few patients were diagnosed based on CSF findings.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.[21] However, these typical features are usually absent inthe 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.[19]Studies have also reported that the maternal and neonatal outcome of GBS in pregnancy is excellent if diagnosed early and managed.[22]

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.[20] Though most of the patients present with prodromal symptoms of respiratory or gastrointestinal tract infections[13] and cranial nerve involvement[24], only 2 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.[25]

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.[26]Treatment usually involves

supportive measures concomitant with disease-specific therapy. Supportive measures commonly instilled are aggressive physiotherapy, enteral nutrition and 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.[27]

Disease-specific therapy includes plasmapheresis or intravenous immunoglobulins, which is equally efficacious both in obstetric and non-obstetric cases is usually started if there is a progression of the disease, especially within 4 weeks of symptoms for any substantial benefit.[28] In the present study, 3 patients received IVIG, 3 patients plasmapheresis, and 2 patients received only supportive care. Hukuimwe M et al. concluded that be IVIG should be considered the treatment of choice as there plasmapheresis treatment requires central venous access and intense monitoring of electrolytes as it can alter blood pressure levels in pregnant patients.[19]In 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.[29]

OBSTETRIC MANAGEMENT:

GBS perse is not an indication for 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,butthere is a lack of maternal bearing down due to muscle weakness. Hence the second stage of labourmay be shortened.[20]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] and Volquind et al.[30] 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, 2 patients had preterm delivery. Studies have hypothesized that immunological mechanism as 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 needed ventilator support.[8]In their study of GBS in pregnancy, Chan et al. reported a neonatal survival rate of 95.7%.[31] However,two patients had intrauterine growth restrictions of unknown cause. Neonatal GBS is rare,but a case report describeda

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, recovery rate is 100%.[32]The risk of GBS is increased postpartum, especially in the initial 2 weeks, which might be due to a delayed-hypersensitivity that flares up the disease, hence proper care and monitoring is essential during this period.[33] 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.[34] and Aabdi et el[35], 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, and improvement is similar to previous treatment. 70-80% of patients recover fully after delivery.[24] In the present study, of the 6 patients diagnosed to have GBS in the antenatal period, 2 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 and concluded that the possible mechanism of relapse might be surgery or anaesthesia, which may trigger proinflammatory cytokines.[24] 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 of thromboembolism, prophylactic anticoagulation should be administered early in GBS pregnant women with poor mobility.[36] In contrast,in the present study, no patient developed pulmonary embolism. Ryabinkina et al. concluded that despite thromboprophylaxis, 52% of patients developed deep vein thrombosis and 12% pulmonary embolism and the significant risk factors including bed rest for more than 3 days, ventilator support, infections, and central venous catheter placement.[25] Further research on preventive strategies of thromboembolism isneeded. Nomani et al. reported that respiratory tract infections are more common in ICU admissions; around 83% of patients having infections with the organisms such as Klebsiella, Acinetobacter and pseudomonas.[37]Hence,early identification and treatment is essential as, in pregnancy, these infections tend to be more severe.[38]

CONCLUSION:

GBS is rare in pregnancy, and prompt treatment is essential to prevent maternal and fetal morbidity. A high index of suspicion is essential, 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:Written Informed consent was obtained from all patients.

Ethical approval: As per International standards or university standard, 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)
Characteristics of GBS	**N=8 (%)**
Gestation at delivery	
Preterm	2(25)
Term	6(75)
Mode of delivery	
Vaginal	3(37.5)
Instrumental	1(12.5)
Caesarean	4(50)

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 2: CHARACTERISTICS OF GUILLAIN BARE SYNDROME:

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)