Guillain-Barré syndrome in pregnancy: a case report and review of the literature

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Guillain-Barré syndrome represents a heterogeneous group of immune-mediated peripheral neuropathies that are characterized by various clinical manifestations. Reporting this clinical case emphasizes the rarity of Guillain-Barré syndrome, the diagnostic challenges faced by healthcare providers, and the risk of delayed diagnosis for both the mother and fetus. A 34-year-old pregnant woman at 33 weeks of gestation presented to the inpatient ward complaining of paresthesia in the lower and upper limbs, muscle pain, balance disturbances, moderate headache, nausea and vertigo, general weakness, and pronounced fatigue. The patient had experienced an acute viral respiratory infection 4 weeks before presenting to the hospital. The patient was admitted to the intensive care unit with a preliminary diagnosis of acute viral respiratory infection and nasopharyngitis. The patient's condition worsened dynamically, manifesting bulbar syndrome (swallowing problems), paresthesia of the anterior abdominal wall, reduced perception of fetal movements, numbness of the tongue, and low fever (37.2°C). A diagnosis of acute inflammatory demyelinating polyradiculopathy (Guillain-Barré syndrome) was established. Despite treatment, the neurologic symptoms worsened. The paravertebral radicular type pains were difficult to manage with administered analgesic therapy, and there was a progression of the bulbar syndrome. Treatment with intravenous immunoglobulin was initiated. Consequently, it was recommended by the multidisciplinary council to perform an emergency cesarean delivery, in the interest of the mother and fetus. Guillain-Barré syndrome is a rare condition that occurs during pregnancy and requires thorough evaluation, prompt multidisciplinary assessment, and individualized management of delivery to improve maternal and fetal prognosis.

Key words: cesarean delivery, Guillain-Barré syndrome, intravenous immunoglobulin, pregnancy

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Written informed consent was obtained from the patient for publication of the current study.

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

The authors report no conflict of interest.

The authors report that no funding was received for this study.

Patient consent is not required because no personal detail is included.

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2666-5778/$36.00

http://dx.doi.org/10.1016/j.xagr.2024.100396

Introduction

Guillain-Barré syndrome (GBS) represents a heterogeneous group of immune-mediated peripheral neuropathies, typically involving the myelin sheath of nerve fibers and presenting various clinical manifestations.[1] A common feature of all GBS variants is a rapidly evolving polyradiculoneuropathy, often preceded by a triggering event, such as a respiratory or intestinal infection. *Mycoplasma pneumoniae*, *Campylobacter jejuni*, cytomegalovirus, and Epstein-Barr virus are common infections preceding GBS.[2] GBS generally presents with symmetrical motor paralysis often accompanied by sensory and autonomic disturbances.[3] GBS can occur in any trimester of pregnancy and the postpartum period. However, it is most common in the third trimester of pregnancy and within the first 2 weeks after delivery.[4–7] Pregnancy may delay the diagnosis of GBS, as the initial symptoms are nonspecific and may mimic normal pregnancy changes or other somatic conditions occurring during pregnancy. GBS should be considered in any pregnant woman who presents with muscle weakness, general malaise, tingling in the fingers, and difficulty in breathing.

By reporting this clinical case, this study aimed to emphasize the rarity of GBS and the diagnostic challenges faced by obstetricians, neurologists, and anesthesiologists-reanimatologists. In addition, we highlight the risk of delayed diagnosis for both the mother and fetus.

Case presentation

A 34-year-old woman at 33 weeks of gestation presented to the inpatient unit of a tertiary-level hospital with complaints of paresthesia in the lower and upper limbs, muscle pain, balance disturbances, moderate headache in the occipital and temporal regions, nausea and vertigo, general weakness, and pronounced fatigue. On the previous day, the patient's blood pressure (BP) values increased up to 150/90 mm Hg for the first time during this pregnancy. The patient's obstetrical history included fourth pregnancy, 1 delivery, and complications by 2 spontaneous abortions in the first trimester of pregnancy. Furthermore, the patient was diagnosed with chronic pyelonephritis, moderate myopia corrected with laser surgery,

and iron deficiency anemia during this pregnancy. Normal development of the fetus was confirmed through ultrasound examinations performed during pregnancy. According to the patient's medical history, the patient had an acute viral respiratory infection (AVRI) 4 weeks before the appearance of the abovementioned symptoms. The patient consulted her general practitioner, complaining of difficulty in breathing, hoarse voice, dry cough, ear discomfort, low-grade fever of 37.4°C, and diarrhea. At-home treatment was recommended, during which the patient initially reported limitations in active movements of the legs and hands; pronounced spinal pain, especially in the lumbar region; difficulty in breathing; and epistaxis. In the subsequent 4 weeks, the patient was monitored by a general practitioner.

The patient was admitted to the intensive care unit (ICU) of the hospital. Objective examination revealed the following: the patient was conscious, was oriented to time and space, answered questions adequately and without delay, and had a Glasgow Coma Scale score of 15 points. The patient had difficulty with nasal breathing because of a hyperemic pharynx. However, pulmonary vesicular breathing was normal, and rales were absent. The patient's BP was 160/100 mm Hg, pulse rate was 105 beats/min, and oxygen saturation was 98%. Moderate peripheral edema was observed in the lower limbs. The patient underwent clinical and paraclinical investigations (a complete blood count showed anemia; there was no fractional protein in the urine; and biochemical blood analysis, coagulogram, and general urine analysis revealed no particularity). Electrocardiogram revealed sinus tachycardia. Ultrasound examination confirmed the normal functional state of the fetus. The diagnosis was established as follows: gestational age of 33 weeks, parity 4, birth 1, complicated obstetrical history (2 miscarriages), AVRI, nasopharyngitis, pregnancy-induced hypertension, chronic pyelonephritis, moderate myopia corrected by laser surgery, and iron deficiency anemia.

Hence, interdisciplinary management was recommended following the existing local guidelines.

On the second day of admission, the patient's general condition was stable, with persistent periodic paresthesia in the lower and upper limbs. However, on the third day of admission, the patient exhibited negative characteristics, including visual disturbances, such as diplopia, while maintaining a BP of 120/80 mm Hg. A neurologist consultation and an electrophysiological investigation (neurography) were recommended. The results showed an increase in the distal latent on the bilateral peroneus nerve fibers, an increase in the latent F wave on the bilateral tibial nerve fibers, and an F-wave present in 20%. These findings were predominantly characteristic of acute demyelinating polyneuropathy. The diagnosis was reevaluated and was determined to be acute inflammatory demyelinating polyradiculopathy (GBS), moderate lower flaccid paraparesis, and neuropathic algic syndrome.

Following the multidisciplinary council, it was recommended to initiate plasmapheresis treatment. Therefore, an expectant management of pregnancy was performed with monitoring of the fetus' well-being. Given the potential risk of premature birth related to GBS, prophylaxis of respiratory distress syndrome (RDS) in the fetus is recommended.

On the fourth day of admission, the patient's condition worsened significantly, marked by the onset of bulbar syndrome (swallowing problems), paresthesia of the anterior abdominal wall, difficulty in perception of fetal movements, numbness of the tongue, and low-grade fever (37.2°C). Ultrasound examination was performed to confirm fetal well-being. Laboratory data revealed a mild increase in liver function tests (alanine aminotransferase: 55.6 mmol/L; aspartate aminotransferase: 60.8 mmol/L). Within the multidisciplinary council, it was decided to cancel RDS prophylaxis because of the negative influence of corticosteroids on GBS progression. Pain relief was provided by administering nonsteroidal anti-inflammatory drugs (NSAIDs). Ibuprofen 400 mg every 12 hours was administered for 48 hours. Fetal effects were considered, and the duration of NSAID therapy was minimized, with careful monitoring for potential fetal effects.

Despite the administration of 2 courses of plasmapheresis, the neurologic symptoms worsened. The paravertebral radicular-type pains responded with difficulty to the administered analgesic therapy, with progression of bulbar syndrome (the patient experiencing choking when drinking water).

Therefore, intravenous immunoglobulin (IVIG) was initiated. Thus, following a multidisciplinary council that included obstetricians, an anesthesiologist-reanimatologist, and a neurologist, an emergency cesarean delivery (CD) was recommended in the interest of the mother and fetus. During CD, a female fetus was delivered, weighing 2300 g, with an Apgar score of 7/8/8 points. General anesthesia with muscle relaxation and mechanical breathing was administered during CD. General anesthesia was selected to avoid the risks associated with a regional block in the context of GBS and autonomic dysfunction.

The postoperative period progressed without obstetrical complications. Postoperative management included the following medication: antibiotics (cefuroxime and metronidazole), anticholinesterase drugs (ipidacrine 15 mg), and IVIG. The respiratory and swallowing functions of the patient were monitored. On the second postoperative day, a neurologist's assessment revealed symmetrical palpebral slits; equal pupils; full motility of the eyeballs; direct and indirect photoreactions present; diplopia; disordered swallowing and phonation; hand muscles with strength of 5/5 bilaterally; decreased muscle strength in the distal portions of the hands and proximal legs (2/5) and distal legs (2−3/5); absent Achillean, patellar, and carporadial osteotendinous reflexes; reduced biceps reflex; low muscle tone; diminishing superficial and deep sensitivity "socks" and "gloves" pattern; Neri sign (pain in the lumbar

region and the lower limb with head anteflexion); positive Lasegue sign (pain caused by flexing the thigh on the pelvis, the leg being held in extension by the examiner's hand); and Glasgow Coma Scale score of 15 points. Neurography revealed data characteristic of acute demyelinating neuropathy. In the following days, the swallowing reflex reappeared. There was relief of pain syndrome and increased strength in the upper limbs (strength: 4/5) and in the lower proximal part (strength: 2/5 on the right and 2−3/5 on the left) and distal part (strength: 3/5 bilaterally), the Neri sign disappeared, and muscle palpation became less painful.

The patient was admitted to the ICU for 7 days after delivery and received treatment with centrally acting analgesics. On the eighth day after delivery, after the patient stabilized, the patient was referred to the rehabilitation department of the Institute of Neurology and Neurosurgery for specialized management.

Discussions

The incidence of GBS in the general population ranges from 0.75 to 2.00 in 100,000 per year, with the incidence increasing with age.[8] According to the literature, GBS occurs rarely in pregnancy (1.2−1.9 cases per 100,000 annually).[9] However, Cheng et al[6] found no significant difference in the incidence of GBS in pregnant women compared with the general population, with a ratio of 0.86 (95% confidence interval, 0.52−1.53), depending on age. GBS can occur in any trimester of pregnancy and the postpartum period. However, if GBS occurs, it is more common in the third trimester of pregnancy and the first 2 weeks after delivery. GBS is known to worsen in the postpartum period because of the rapid increase in delayed-type hypersensitivity.[6] In a literature review based on 30 cases of GBS in pregnancy, only 4 women (13%) had onset in the first trimester of pregnancy, 14 women (47%) had onset in the second trimester of pregnancy, and 12 women (40%) had onset in the third trimester of pregnancy.[5] In 2009, Campos da Silva et al[4] reported a case of GBS diagnosed at 15 weeks of gestation, which worsened after delivery.

The cause of GBS remains unknown. However, two-thirds of patients typically report a history of AVRI (40%) or gastrointestinal infection (20%), which occurs 4 to 6 weeks before the onset of symptoms.[8] The etiologic factors can include systemic diseases, certain malignancies, surgical interventions, pregnancy, and trauma. Some data indicate that the development of the GBS after cytomegalovirus infection is characterized by initial severity, pronounced respiratory difficulties, delayed recovery, and severe sensory loss. Other clinical manifestations of GBS include motor and sensory dysfunctions. Vegetative dysfunction occurs in approximately two-thirds of cases, manifested by vegetative dysautonomy, with changes in BP or pulse.

The diagnosis of GBS is based on clinical, paraclinical, and laboratory criteria[10] and requires differential diagnosis with several conditions, such as myasthenia gravis; transverse myelitis; other acute myelopathies; botulism; poliomyelitis caused by the poliovirus; West Nile fever; acute toxic neuropathies (with arsenic, lead); neurologic form of Lyme disease; and carcinomatous meningitis. Patients with GBS typically present with progressive symmetrical weakness that ascends from the lower extremities, along with areflexia, paresthesia, or neuropathic pain. Other clinical symptoms include sensory and cranial nerve damage (most commonly facial paralysis), autonomic dysfunction leading to altered BP and pulse, and respiratory failure, which is a major cause of morbidity and mortality.[10,11]

Laboratory testing can be useful to diagnose GBS in all patients, including pregnant women. However, these tests are often nonspecific. The necessary laboratory investigations to be performed include hemogram and blood biochemistry (glucose, direct and indirect bilirubin, alanine aminotransferase, aspartate aminotransferase, creatinine, urea, total protein, Na+, K+, and Ca+) to perform differential diagnosis. A lumbar puncture is performed to obtain cerebrospinal fluid (CSF) for the analysis of electrolyte, protein, and glucose contents.[12] Examination of CSF may detect a high protein level of ≤10 mononuclear cells/mm^3. Albuminocytological dissociation is nonspecific and can be found in infections, neoplasias, and vascular or inflammatory diseases of the brain and spinal cord, with which it is necessary to perform differential diagnosis. The CSF protein concentration may be normal during the first week of the condition. Of note, 10% to 12% of patients may have ≥5 cells/mm^3. CSF pleocytosis (≥50 mononuclear cells/mm^3) can be observed in HIV-positive patients with GBS. Nerve conduction tests, antibody screening, and electroencephalography, which reveal multifocal demyelinating polyneuropathy, are considered important to diagnose GBS.[8] Electrophysiological examination may reveal demyelination in acute inflammatory demyelinating polyradiculoneuropathy and predominance of axonal involvement in acute axonal motor neuropathy or acute axonal sensory motor neuropathy. During the initial days of the condition, examination results may be within the normal range. Another recommended investigation is serum GQ1b antibody testing. In addition, nuclear magnetic resonance is used for diagnostic purposes. However, the results are often nonspecific.[13]

The literature describes the Brighton criteria for the diagnosis and staging of GBS, which include bilateral flaccid limb weakness, decreased or absent deep tendon reflexes in the affected limbs, a monophasic course and onset period of 12 hours to 28 days, CSF cell count of <50/μL, CSF protein concentration above normal value, results of nerve conduction studies consistent with one of the subtypes of GBS, and absence of alternative diagnosis for weakness. Each abovementioned criterion can be scored from level 1 (the highest level of certainty of the diagnosis) to level 4 (the lowest level of certainty of the diagnosis) (Table).[3,14]

Literature data suggest that GBS does not increase the risk of miscarriage or fetal death. At the same time, it does not affect fetal development.[7]

TABLE
The Brighton criteria

Diagnostic criteria	1	2	3	4
Bilateral flaccid weakness of the limbs	+	+	+	+/−
Decreased or absent deep tendon reflexes in the affected limbs	+	+	+	+/−
Monophasic course and onset period of 12 h to 28 d	+	+	+	+/−
Cell count in CSF of <50/μL	+	+[a]	−	+/−
CSF protein concentration greater than normal value	+	+/−[a]	−	+/−
NCS findings consistent with one of the subtypes of GBS	+	+/−	−	+/−
Absence of an alternative diagnosis for weakness	+	+	+	+

The "+" symbol indicates present, the "−" symbol indicates absent, and the "+/−" symbol indicates present or absent.

CSF, cerebrospinal fluid; *GBS*, Guillain-Barré syndrome; *NCS*, nerve conduction study.

[a] If CSF is not collected or data are not available, nerve electrophysiology data should be consistent with a diagnosis of Guillain-Barré syndrome.

Iliadi-Tulbure. Guillain-Barré syndrome in pregnancy. Am J Obstet Gynecol Glob Rep 2024.

The management of GBS during pregnancy is similar to that of the general population and requires a multidisciplinary approach involving obstetricians, neurologists, and anesthesiologists-reanimatologists. The management aims to reduce the immune response. The specific treatment consists of performing plasmapheresis and administering IVIG, with both treatments showing similar efficacy. According to randomized clinical trials, immunomodulation with plasmapheresis and IVIG have been found to improve treatment outcomes, achieving complete recovery in 70% to 80% of patients compared with conservative management.[14] Satisfactory results are particularly noted when plasmapheresis is initiated within the first 2 weeks after the onset of the disease.[7] It has been observed that patients receiving IVIG or plasmapheresis have a reduced need for ventilatory support. The potential risks of plasmapheresis include hypotension, fluid overload, septicemia, and coagulation changes.[15] Studies have shown that the complications of plasmapheresis are similar in pregnant and nonpregnant women.[5,15] In a literature review analyzing 30 cases of GBS during pregnancy, 22 patients received IVIG or plasmapheresis without maternal or fetal complication.[5]

No method immediately improves the condition of the patients. In some cases, clinical manifestations may even progress after the initiation of specific treatment. After initial stabilization, a relapse is observed in approximately 10% of patients, necessitating a new course of plasmapheresis or IVIG. Corticosteroid treatment of GBS was studied in a double-blind, placebo-controlled, multicenter trial involving 242 patients who were randomized to receive high-dose intravenous methylprednisolone (500 mg/d for 5 days over 2 weeks from onset) or placebo. The data showed no significant difference between the groups.[12]

Respiratory failure is common in GBS, and 15% to 30% of patients require ventilatory support. Frequent monitoring of lung function, including measurement of vital capacity, should be performed in most cases. Predictive factors for the need for mechanical ventilation include forced vital capacity of ≤20 mL/kg, maximum inspiratory pressure of ≤30 cm H_2O, and maximum expiratory pressure of ≤40 cm H_2O. Other predictive factors of respiratory dysfunction include time since pathology onset of <7 days; inability to cough, raise eyebrows, raise the head, and stand; and increased liver transaminases. If ≥4 of these signs are present, mechanical ventilation will be required in 85% of cases. The need for mechanical ventilation is assessed using the Erasmus GBS Respiratory Insufficiency Score.

Ventilator support may be required in 25% to 30% of cases, but respiratory problems may be more severe during pregnancy because of partial ascension and immobility of the diaphragm, requiring complex monitoring.[16] Some studies have found that if ventilatory support is required during pregnancy, the incidence of preterm birth is also increased.[17,18]

Hypotensive episodes in GBS can be treated by administering fluids or small doses of phenylephrine. Episodes of arterial hypertension can be managed by administering urapidil or nitroprusiato. Cardiac arrhythmias usually do not require treatment. However, severe cardiac arrhythmias that endanger the patient's life, such as atrioventricular blocks or asystole, in the context of dysautonomy, require intervention. The most common sphincteric disorders in GBS are adynamic ileus and urinary retention. Neostigmine or laxatives can be used for the treatment of ileus. Intercurrent infections will be treated depending on the pathogen, and NSAIDs can be used for pain management in GBS. However, they usually do not provide adequate pain control and are contraindicated in the third trimester of pregnancy.

The timing and method of delivery are determined by the maternal condition because of GBS progression and the fetal well-being. Clinical cases with altered hemodynamic status may require premature termination of pregnancy. Therefore, fetal RDS prophylaxis is indicated. The obstetrician should assess the maternal risks of GBS and prematurity. However, other studies suggest that GBS itself does not represent the indication for CD, requiring individual management based on maternal and/or fetal conditions.[5]

The choice of analgesia during labor or CD presents a challenge in pregnant women with GBS. Both regional and general anesthesia carry additional potential risks in these cases. Most studies report the use of general or epidural anesthesia techniques.[1] A major concern regarding general anesthesia in GBS is the use of succinylcholine, which can lead to hyperkalemia. Hence, in 2004, Chan et al[5] reported a case of cardiac arrest in a similar clinical situation.

Patients with GBS typically experience disease progression within the first 4 weeks after onset, after which the plateau phase is established, generally lasting for another 4 weeks. Respiratory dysfunction and disorders can have an emergent onset and develop within the first 4 weeks. During this period, follow-up is necessary for the prevention and treatment of infectious complications, venous thrombosis, pain control, and swallowing disorders. During the plateau and convalescence phase, patients should be monitored to prevent the development of intercurrent infections. The recovery period lasts for 6 to 12 months. The prognosis of GBS is dependent on several factors. In general, after a year, approximately two-thirds of patients can completely recover.

However, 1 year after the onset of the disease, 18% of patients cannot run, 9% of patients cannot walk without assistance, and 4% of patients are bedridden.

GBS is associated with an increased rate of maternal and perinatal mortality (7%−10%) compared with the general population (<5%) because of respiratory complications and prematurity.[10,19] Mortality rates vary among different groups, reaching 13%. Immunotherapy has not been shown to reduce the mortality in GBS, which is due to secondary complications arising from the prolonged disease course. Approximately 5% of patients die in the ICU. Approximately 25% of deaths occur during the first week, and approximately 50% of deaths occur during the first month of the disease. Cardiac arrest resulting from vegetative dysfunction is the most common cause of death (20%−30%). In a 2013 study by Kim et al,[20] a patient with GBS developed pulmonary embolism after delivery. The other causes of death are lung infection and respiratory failure.

However, in the context of continuing appropriate management and physical therapy, positive outcomes can be achieved.

Conclusions

GBS is a rare condition in pregnancy. When GBS occurs, it is associated with an increased risk of maternal and/or perinatal morbidity. Early diagnosis and active management using IVIG or plasmapheresis, along with the prevention of maternal and/or perinatal complications, are key to the successful outcome of GBS in pregnancy. GBS alone is not an indication for CD. The decision to perform fetal extraction should be based on maternal and/or fetal conditions related to GBS progression. Pathology requires thorough assessment and proper multidisciplinary management to improve maternal and fetal prognosis.

Glossary

AVRI	acute viral respiratory infection
BP	blood pressure
CD	cesarean delivery
CSF	cerebrospinal fluid
GBS	Guillain-Barré syndrome
ICU	intensive care unit
IVIG	intravenous immunoglobulin
NCS	nerve conduction study
NSAID	nonsteroidal anti-inflammatory drug
RDS	respiratory distress syndrome

CRediT authorship contribution statement

Corina Iliadi-Tulbure: Writing − review & editing, Writing − original draft, Validation, Supervision, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Maria Cemortan:** Writing − review & editing, Validation, Software, Data curation. **Svetlana Jubirca:** Validation, Methodology, Investigation, Conceptualization. **Viorica Cospormac:** Writing − review & editing, Writing − original draft, Supervision, Project administration, Methodology, Formal analysis, Data curation, Conceptualization. **Cristina Bubulici:** Writing − review & editing, Writing − original draft, Methodology, Data curation. **Maria-Magdalena Vicol:** Writing − review & editing, Writing − original draft, Software, Investigation.

REFERENCES

1. Karnad DR, Guntupalli KK. Neurologic disorders in pregnancy. Crit Care Med 2005;33 (Suppl):S362–71.
2. Hadden RD, Gregson NA. Guillain-Barre syndrome and Campylobacter jejuni infection. J Appl Microbiol 2001;90:145–54.
3. Fokke C, van den Berg B, Drenthen J, Walgaard C, Van Doorn PA, Jacobs BC. Diagnosis of GuillainBarré syndrome and validation of Brighton criteria. Brain 2014;137:33–43.
4. Campos da Silva F, De Moraes Paula G, Dos Santos Esteves Automari CV, Mendes de Almeida DS, Ubirajara Cavalcanti Guimarães R. Guillain-Barre syndrome in pregnancy: early diagnosis and treatment is essential for a favourable outcome. Gynecol Obstet Invest 2009;67:236–7.
5. Chan LYS, Tsui MHY, Leung TN. Guillain-Barré syndrome in pregnancy. Acta Obstet Gynecol Scand 2004;83:319–25.
6. Cheng Q, Jiang GX, Fredrikson S, Link H, De Pedro-Cuesta J. Increased incidence of Guillain-Barre syndrome postpartum. Epidemiology 1998;9:601–4.
7. Jallouli O, Kammoun I, Kammoun R, et al. Guillain–Barre syndrome and pregnancy: a case report. Neurophysiol Clin 2018;48:232.
8. National clinic guideline PCN −346. Guillain-Barre sindrom. Ministerul Sănătății al Republicii Moldova. Available at: https://msmps.gov.md/wp-content/uploads/2021/03/Protocol-clinic-na%C5%A3ional-%E2%80%9ESindromul-Guillain-Barre%E2%80%9D.pdf. Accessed September 5, 2023.
9. Sharma SR, Sharma N, Masaraf H, Singh SA. Guillain-Barre 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.
10. Zafar MSH, Naqash MM, Bhat TA, Malik GM. Guillain-Barré syndrome in pregnancy: an unusual case. J Fam Med Prim Care 2013;2:90–1.
11. Vijayaraghavan J, Vasudevan D, Sadique N, Rajeswari KS, Pondurangi M, Jayshree. A rare case of GuillainBarré syndrome with pregnancy. J Indian Med Assoc 2006;104:269–70.
12. Fortuin AW, Van der Kooi ALW, Mulder LJMM, Van der Avoort IAM. Guillain-Barré syndrome complicating first trimester pregnancy: a case report and review of literature. Obstet Gynaecol Cases Rev 2021;8:194.
13. Seneviratne U. Guillain Barre syndrome. Postgrad Med J 2000;76:774–82.
14. Elovaara I, Apostolski S, Van Doorn P, et al. EFNS guidelines for the use of intravenous immunoglobulin in treatment of neurological diseases: EFNS task force on the use of intravenous immunoglobulin in treatment of neurological diseases. Eur J Neurol 2008;15:893–908.
15. Branch DW, Porter TF, Paidas MJ, Belfort MA, Gonik B. Obstetrics uses of intravenous immunoglobulin: successes, failures, and promises. J Allergy Clin Immunol 2001;108 (Suppl):S133–8.
16. Hughes RAC, Cornblath DR. Guillain-Barre syndrome. Lancet 2005;366:1653–66.

17. 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.
18. Vaduva C, De Seze J, Volatron AC, et al. Severe Guillain-Barré syndrome and pregnancy: two cases with rapid improvement post-partum. Rev Neurol (Paris) 2006;162: 358–62.
19. Furara S, Maw M, Khan F, Powell K. Weakness in pregnancyexpect the unexpected. Obstet Med 2008;1:99–101.
20. Kim H, Ryu J, Hwang JW, Do SH. Anesthetic management for cesarean delivery in a Guillain-Barre syndrome patient – a case report. Korean J Anesthesiol 2013;64: 268–71.