CASE SERIES

Pregnancy-related Guillain-Barré syndrome: A Case Series

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ABSTRACT

Introduction: Pregnancy-related Guillain-Barré syndrome (GBS) is a rare autoimmune disorder that affects pregnant women. With an annual incidence ranging from 0.81 to 1.89 cases per 100,000 population, GBS can occur at any trimester of pregnancy, as well as during the postpartum period are susceptible to GBS. The pillars of managing pregnancy-related GBS to improve outcomes include early diagnosis, prompt immune-modulatory therapy, and multidisciplinary input. **Case Series:** In this study, three case of GBS in pregnancy were reported. The first patient was a 35-year-old woman, G3A1P2 post emergency Transperitoneal Cesarean Section (TPCS), who experienced with lower limb weakness three days before TPCS. After being diagnosed with severe eclampsia and underwent emergency TPCS, her complaint of lower limb weakness worsened. The second patient, a 27-year-old woman, with G2P1A0 experienced weakness in all four limbs. The third patient, a 20-year-old woman with G1P0A0, in the third semester presented with weakness in all four limbs. The electroneurography investigation conducted on these patients supported the diagnosis of GBS, which was subsequently managed with plasma exchange (PE). After the administration of PE, there was observed improvement in the clinical manifestation of GBS. **Conclusion:** The development of GBS in pregnancy is typically preceded by bacterial or viral infection. Preeclampsia was found to be associated with two folds risk of GBS, which was usually diagnosed based on the neurological examinations with supportive studies, including serological tests, cerebrospinal fluid analysis and electroneurography. The management of pregnancy-related GBS included intravenous immunoglobulin, PE, physiotherapy, and supportive therapy, such as ventilator support.
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INTRODUCTION

Guillain-Barré syndrome (GBS) is a neuropathy characterized by deteriorating strength and reduced tendon reflexes. It is an inflammatory condition of the peripheral nervous system (PNS) and is the most frequent cause of acute flaccid paralysis, with an annual incidence of 0.81–1.89 case per 100,000 population (1). Patients with GBS could have clinical manifestation as acute polyneuropathy with inflammation, ascending limb weakness, reduced reflexes, and increased cerebrospinal fluid (CSF) protein level, but with normal cell count (2). The antecedent events that led to GBS have been associated with bacterial and viral infections (mainly upper respiratory tract infections and gastroenteritis), vaccinations, immune inhibitor therapy, pregnancy, organ transplantation, mechanical injury, surgery, and neoplasia (3).

GBS can occur in various trimesters in pregnancy and post-partum (4). A previous study showed that the risk of GBS was lower during pregnancy but increase significantly in post-partum because of increasing delayed type of hypersensitivity (4). Since GBS significantly increased during third trimester of pregnancy, it could develop the risk of prematurity and respiratory complications (5). Furthermore, the demand for mechanical ventilation is higher in GBS with pregnancy, compared to non-pregnancy case, with percentages of 33% and 16%, respectively (5). Another study showed that GBS is associated with more than 10% of maternal and perinatal mortality rates (6). The delayed diagnosis of GBS in pregnancy is common due to the vague early sign and symptoms which could be misinterpreted for psychological issues or changes in the pregnancy (4).

CASE SERIES

Case 1

A 35-year-old woman, G3A1P2 post emergency Transperitoneal Cesarean Section (TPCS) with Gemelli, experienced lower limb weakness for three days before

the procedure. Initially, the patient denied the symptom because it was a mild, but the weakness worsened on examination on 4th day after the procedure. The patient then consulted to the neurology department after the seventh day of treatment. This patient had no history of diabetes mellitus, hypertension, and stroke, but she experienced TPCS for twin delivery in 2019, and her babies weighed 2310 and 2120 grams.

On the physical examination, the patient exhibited a blood pressure of 150/81 mmHg. The examination of cranial nerves was unremarkable, while the muscle strength assessment indicated a score of 4 and 2 for both sides upper and lower extremities, respectively. Furthermore, tendon reflexes were +1 (present but small or only with reinforcement) in all extremities and there were no pathological reflexes.

Electrolytes, serum creatinine, and complete blood count were all within normal ranges. CSF analysis showed cytoalbuminologic dissociation of an increasing protein (70 mg/dL; normal 15-40mg/dL) without increasing number of cells (2/μL; normal 0-5/μL), which supported the diagnosis of GBS (7). In the electroneurography (ENG) study as shown in Table 1, there was a prolonged latency of >125% upper limit of normal in the bilateral deep fibular, bilateral median, bilateral tibial, and left ulnar nerves (8). There was also a decrease in nerve conduction velocity (NCV) of <90% lower limit than normal in the left deep fibular nerve, left median nerve, and right ulnar nerve, as shown in Table I. These values indicated the presence of motor and sensory axonal demyelinating features supporting the diagnosis of GBS based on Shapiro criteria (8). The patient underwent plasma exchange (PE) therapy for two cycles and her symptoms improved, which was marked by an increase to 4 in muscle strength of both lower extremities. An initial 4mg magnesium sulfate (MgSO4) was also administered intravenously, followed by a maintenance dose of 0.5 grams/hour for 24 hours, and 10mg nifedipine t.i.d. for her preeclampsia. One month after she was discharged from the facility, there was no complication or residual symptoms.

Table I: Nerve Conduction Studies of Case 1

Nerve stimulated	Stimulation site	Record site	Latency (ms)	Amplitude (μV)	Velocity (m/s)
Left median (s)	Wrist	2nd Digit	4,0	8,9	35
Right median (s)	Wrist	2nd Digit	5,8	14,8	24
Left sural (s)	Calf	Lateral Malleolus	1,5	30,9	93
Right sural (s)	Calf	Lateral Malleolus	2,6	9,6	54
Left ulnar (s)	Wrist	5th Digit	2,6	3,6	54
Right ulnar (s)	Wrist	5th Digit	2,1	5,2	67
Left fibular (m)	Ankle	Extensor digitorum brevis	NR	NR	NR
Right fibular (m)	Ankle	Extensor digitorum brevis	NR	NR	NR
Left median (m)	Wrist	Abductor pollicis brevis	NR	NR	NR
Right median (m)	Wrist	Abductor pollicis brevis	NR	NR	NR
Left tibial (m)	Ankle	Abductor hallucis brevis	NR	NR	NR
Right tibial (m)	Ankle	Abductor hallucis brevis	NR	NR	NR
Left ulnar (m)	Wrist	Abductor digiti minimi	NR	NR	NR
Right ulnar (m)	Wrist	Abductor digiti minimi	NR	NR	NR

s: sensory, *m:* motoric, *NR:* no response

Case 2

A 27-year-old woman, G2P1A0, 11 weeks pregnant, experienced weakness in all four limbs for 1 day before admission. The weakness was initially felt in both lower limbs, then ascend to the upper limb. This patient was unable to walk unassisted and her hands could not lift any weight. There was also a feeling of reduced sensation in all palms and soles.

In the last four days before admission, this patient could not smile or lift both eyebrows and experienced a double vision from the beginning of facial weakness. However, there was no swallowing difficulty or shortness of breath, but she had a cough a week before the admission. There was no history of recent fever, diarrhea, or weakness.

During physical examination on 5th day after onset of weakness, we observed bilateral paresis of oculomotor nerve, trochlear nerve, abducens nerve, and peripheral facial nerve. Muscle strength were 4 for both right upper and lower extremities and 5 for both left upper and lower extremities. Tendon reflexes were decreased to +1 in all extremities and no pathological reflexes were found. Glove and stocking hypoesthesia was also observed and the GBS disability score of 4 indicated that the patient was confined to chair bound. On obstetrical examination, a positive ballottement was found with crown rump length (CRL) of 4.5 cm, pulsation (+), indicating normal intrauterine pregnancy. The blood test results were within the normal range for serum creatinine, electrolytes, and complete blood count. CSF analysis on the 4th day after onset showed an increased protein (100 mg/dL) without increased number of cells (3/μL), supporting the diagnosis of GBS.

PE was carried out on the 11th day after symptoms onset, requiring 2.912 L of plasma for a body weight of 65 kgs. PE was carried out for 15 cycles with a draw speed, return speed, total input and total output of 50 ml, 80 ml, 2914 ml, and 6523 ml, respectively. After PE, her vital sign was stable, and there was an improvement in muscle strength, tendon reflexes, and dysphonia after undergoing PE. However, oculomotor, trochlear, abducens, and facial nerve palsies were still observed.

The muscle strength increased to 5 for both upper and lower extremities two days after PE. Bicep, triceps,

Table II: Nerve Conduction Studies of Case 2

Nerve stimulated	Stimulation site	Record site	Latency (ms)	Amplitude (μV)	Velocity (m/s)
Left median (s)	Wrist	2nd Digit	4,6	8,5	30
Right median (s)	Wrist	2nd Digit	1,4	2,3	100
Left sural (s)	Calf	Lateral Malleolus	0,7	29,2	200
Right sural (s)	Calf	Lateral Malleolus	0,6	19,1	233
Left ulnar (s)	Wrist	5th Digit	1,7	2,8	82
Right ulnar (s)	Wrist	5th Digit	1,7	2,1	82
Left fibular (m)	Ankle	Extensor digitorum brevis	NR	NR	NR
Right fibular (m)	Ankle	Extensor digitorum brevis	NR	NR	NR
Left median (m)	Wrist	Abductor pollicis brevis	NR	NR	NR
Right median (m)	Wrist	Abductor pollicis brevis	NR	NR	NR
Left tibial (m)	Ankle	Abductor hallucis brevis	NR	NR	NR
Right tibial (m)	Ankle	Abductor hallucis brevis	NR	NR	NR
Left ulnar (m)	Wrist	Abductor digiti minimi	NR	NR	NR
Right ulnar (m)	Wrist	Abductor digiti minimi	NR	NR	NR

s: sensory, *m:* motoric, *NR:* no response

patella, and Achilles reflexes were also improved to (+2) both in the right and left extremities. Furthermore, slight facial nerve palsy was still observed and GBS disability score decreased to 2, indicating that the patient was able to run or walk without a stick but unable to perform manual work. ENG study indicated axonal motor and sensory neuropathy of the left deep fibular and right sural nerves, respectively. It also showed demyelinating sensory neuropathy of the bilateral ulnar nerve, as presented in Table II. This result supported the diagnosis of GBS with the Miller-Fisher variant (8).

Case 3

A 20-year-old woman, G1P0A0, 37 weeks pregnant, experienced weakness in all extremities for 2 days before admission. The weakness was initially felt in the lower extremities, making it difficult to stand up after sitting or squatting. Initially, the patient was able to walk independently, but her weakness was gradually worsened, and ascended to the upper extremities. The patient could not lift her upper extremities, as well as held any object. There was no tingling, numbness, facial muscle weakness, difficulty swallowing, visual disturbances, recent cough, cold, fever, and diarrhea, as well as no history of limb weakness.

A bilateral peripheral facial nerve palsy was observed on the 5th day after the onset of weakness during the physical examination. Muscle strength had a score of 4 for the both right upper and lower extremities and 3 for both the left upper and lower extremities. Tendon reflexes were +1 in all extremities and there were no pathological reflexes observed. GBS disability score was 4, indicating that the patient was confined to chair bound. Obstetrical status examination in the patient found ballottement (+), CRL 4.5 cm, pulsation (+), and an impression of an intrauterine pregnancy in good condition. The total blood count, electrolytes, and serum creatinine results of the blood test were unremarkable. CSF analysis showed an increased protein (88 mg/dL) without an increased number of cells (3/μL), supporting the diagnosis of GBS.

ENG was carried out on the 9th day from the onset, as shown in Table III. The result showed motor demyelinating axonal lesions of the bilateral ulnar and bilateral deep fibular nerves, motor axonal lesions of the left median and bilateral tibial nerves, and sensory demyelinating lesions of the right sural nerve. Furthermore, F wave elongation of the left deep fibular nerve was observed, and conduction block of the right ulnar, bilateral deep fibular, and left tibial nerves were also found. These results were consistent with the diagnosis criteria of acute demyelinating polyneuropathy (8). CSF analysis on the 10th day after onset showed increased protein (109 mg/dL) without an increased number of cells, supporting the diagnosis of GBS.

The patient had a caesarean section and delivered male neonates with a birthweight of 3300 grams, without any

Table III: Nerve Conduction Studies of Case 3

Nerve stimulated	Stimulation site	Record site	Latency (ms)	Amplitude (μV)	Velocity (m/s)
Left median (s)	Wrist	2nd Digit	2,2	101,1	64
Right median (s)	Wrist	2nd Digit	2,3	51,3	61
Left sural (s)	Calf	Lateral Malleolus	3,3	41,5	42
Right sural (s)	Calf	Lateral Malleolus	4,1	9,2	34
Left ulnar (s)	Wrist	5th Digit	2,0	135,6	70
Right ulnar (s)	Wrist	5th Digit	1,8	87,3	78
Left fibular (m)	Ankle	Extensor digitorum brevis	1,3	0,8	36
Right fibular (m)	Ankle	Extensor digitorum brevis	1,6	2,3	NR
Left median (m)	Wrist	Abductor pollicis brevis	2,7	3,9	65
Right median (m)	Wrist	Abductor pollicis brevis	3,0	6,0	70
Left tibial (m)	Ankle	Abductor hallucis brevis	4,8	2,7	48
Right tibial (m)	Ankle	Abductor hallucis brevis	3,8	2,2	47
Left ulnar (m)	Wrist	Abductor digiti minimi	2,0	1,3	51
Right ulnar (m)	Wrist	Abductor digiti minimi	1,1	2,9	47

s: sensory, *m:* motoric, *NR:* no response

worsening symptoms. However, the symptoms of patient of weakness worsened two weeks post-delivery. On physical examination, the upper and lower extremities' strength was 4 and 3, respectively, and the blood test result was within normal range.

PE was carried out on the 27th day after symptoms onset requiring 2.352 L of plasma for a body weight of 60 kg. PE was carried out for 8 cycles with a draw speed of 33 ml, return speed, total input, and total output of 33 ml, 52 ml, 1408 ml, and 3063 ml, respectively. The vital sign of patient was stable after PE and there was an improvement in muscle strength and tendon reflexes.

DISCUSSION

GBS is an uncommon immune-mediated disorder affecting the nerve roots and peripheral nerves. It is often triggered by several antecedent events, such as bacterial or viral infection (9). GBS is also associated with several underlying diseases (10) and its pathophysiology is characterized by repulsive immune cell behavior, which resulting the damage on peripheral nerves and specific gangliosides. The diagnosis of GBS is usually made upon clinical examination, laboratory studies (i.e., CSF and serological test), and neurophysiological studies (11). Furthermore, the clinical signs of GBS include fast-progressing bilateral weakness that begins in the lower extremities and progresses to the upper muscles, absence of evident CNS involvement or other causes, distal paresthesia, and diminished or absent reflexes. (9). In pregnancy-related GBS, these features can be misleading as psychological problems or other neurological disorders that are biased as pregnancy symptoms, resulting in delayed diagnosis and treatment. GBS can occur in any trimester but is most common during the third trimester and first two weeks post-partum.

In the first case, preeclampsia was reported, followed by GBS progression. The patient developed lower limb weakness three days before delivery and ascended to the upper extremities. A longitudinal cohort study by Auger et al. showed that preeclampsia was associated with a 2-fold increased risk of GBS and the highest cumulative incidence of the condition (55.4 per 100 000 women) compared to non-preeclamptic patients (12). Furthermore, preeclampsia reduces perfusion to the placenta due to abnormal trophoblast invasion with poor vascular remodeling of the uterine arteries (13). This process is influenced by an imbalance in the production of autoantibodies and T-cell subtypes (14). The preeclampsia process is also linked to an increase in pro-inflammatory T-cells, autoantibody formation against the angiotensin II receptor, various components of peripheral nerve myelin, and sometimes the axon (14). The first case showed preeclamptic features before GBS progression, while the others showed no increase in the blood pressure. All patients in this study showed the same symptoms, namely weakness in the lower limbs which progressively ascend to the upper limbs. In this case series, an ENG examination was conducted to establish the diagnosis of GBS. The electrophysiologic criteria for acute demyelinating polyneuropathy were used to demonstrate three of the following findings (8). Firstly, prolonged DLs (two or more nerves, not at entrapment sites) DL >115% ULN (for normal CMAP amplitudes) DL >125% ULN (for CMAP amplitudes < LLN). Secondly, CV slowing (two or more nerves, not across entrapment sites) CV <90% LLN (for CMAP amplitudes >50% LLN) CV <80% LLN (for CMAP amplitudes <50% LLN) (Note: CVs are commonly preserved early in the course of acute inflammatory demyelinating polyneuropathy). Thirdly, prolonged late responses: F response and H reflexes (one or more nerves) >125% ULN; or absent F responses (Note: When distal CMAP amplitude is very low, absent F waves may not be abnormal). Lastly, conduction block/temporal dispersion (one or more nerves), including unequivocal conduction block, such as proximal/distal CMAP area ratio <0.50. Others include possible conduction block, namely proximal/distal CMAP amplitude ratio <0.70 and temporal dispersion, such as proximal/distal CMAP duration ratio >1.15.

The hypothesis of the process underlying GBS showed that it is triggered by certain immunizations and prior infections. Among all known pathogens, Cytomegalovirus has been found recently in 26% of patients as the pathogen most frequently linked to GBS in a pregnant population (15). Nevertheless, the second and third case in this study reported no preceding infection or comorbidity, such as preeclampsia. Due to an increase in a delayed kind of hypersensitivity, GBS is exacerbated throughout the postpartum period. Th2 cytokines predominate over Th1 during pregnancy, while Treg activity increases in the first and second trimesters (16). GBS has been attributed to disorders mediated by Th1 cells but some changes cannot be explained by the pathogenic role of Th1 cells and disruption of the Th1/Th2 balance (17). Other inflammatory cytokines, including IFN-λ, TNF-α, IL-6, and IL-1, have been proven to protect against GBS, while the specific mechanism is unknown (18).

The treatment of GBS in pregnancy is not different from non-pregnant patients (6). The managements of GBS in pregnancy involves IVIG, PE, ventilatory support, infection detection and treatment, prevention of venous thromboembolism, pain management, and treatment of the psychosocial stress caused by the illness (4).

IVIG has a proven track record of effectiveness and is safe to use during pregnancy. This method is preferred because it has fewer complications compared to PE (6). However, with full recovery occurring in 70–80% of patients, PE has been shown to improve clinical outcomes (3) and is more affordable compared to IVIG

(19). Some of the serious possible side effects of PE during pregnancy are hemodynamic changes, hepatitis, maternal sepsis, and abnormal coagulation (10), but the patients in this study experienced no complications. The patient had complete remission and showed no relapse on 1 month of follow up and there was no side effect of PE observed on born neonates. Treatment of GBS in pregnancy needs a comprehensive approach by the neurologist, obstetrician, intensivist, radiologist, physiotherapist and pediatrician.

The indicators of poor prognosis for GBS include rapid illness onset, severe paralysis, muscle wasting, a protracted period of peak paralysis, and respiratory involvement. A previous study reported symptoms relapse in 5.6-6.8% of case (12). After one year, up to 20% of patients could become disabled, and a maternal mortality rate of 7% has been reported (20).

CONCLUSION

In conclusion, preeclamptic pregnancy was identified as one of the comorbid factors for GBS, a comorbidity observed during both pregnancy and the postpartum period. The occurrence of GBS needed to be considered when pregnant women experienced a sudden onset of quadriparesis because this condition could worsen rapidly, requiring the implementation of an immediate multidisciplinary. GBS management during pregnancy involved IVIG, PE, ventilator support, infection detection and treatment, venous thromboembolism prevention, pain control, and psychosocial management.

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