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Review Article

Guillain-Barre Syndrome and Pregnancy: About Four Cases

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Abstract

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy that results from an abnormal cell-mediated immune response to peripheral nerves. In about two- thirds of cases, an infectious etiology precedes symptoms. GBS is a rare disease during pregnancy with an incidence of 1.2 and 1.9 cases per 100,000 per year and carries a high maternal risk. We report two cases of GBS, one during pregnancy and the other in the postpartum period.

Both patients recovered with supportive measures and intravenous immunoglobulin (IVIG).

Keywords: guillain-barré syndrome; pregnancy; immunoglobulins; rehabilitation

Introduction

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyradiculoneuropathy. It is characterized by diffuse and symmetrical involvement of the peripheral nervous system, which may extend to the cranial nerves and spinal ganglia.

GBS has motor, sensory, and autonomic repercussions.

The overall incidence of GBS worldwide is 1.1 to 1.8 per 100,000. A similar incidence is observed during pregnancy [1]. Diagnosis depends on clinical, biological, and electrophysiological findings. In around two-thirds of cases, an infectious aetiology precedes the onset of symptoms [2].

Here, we present four cases of GBS: three during pregnancy and one in the postpartum period.

Two of the four patients made a full recovery with supportive measures and intravenous immunoglobulin.

Observation number 1:

The patient is a 23-year-old female with no notable pathological history or GI 0P. The gynecological examination revealed no detectable abnormality. The patient was admitted for tachycardia during pregnancy at 16 weeks and 3 days. A neurological examination revealed a disturbance of balance, a waddling gait, weak muscle tone, normal sensitivity, and a positive Babinski reflex. There was no initial swallowing disorder.

A paraclinical workup was performed with the following results:

- ENMG: consistent with Guillain-Barré syndrome
- LP: Proteinorrachia: 0.65, Glucorrachia: 0.7
- Inf serologies: EBV/CMV+

The patient is on immunoglobulins (3 g/day), IPP (40 mg/day), and a B-blocker (¼ tablet twice daily).

- TSH: 0

- T3L: 4.6

During her stay in the ICU, the patient developed diaphragmatic damage, hence the need for intubation. A tracheotomy was performed, followed by extubation. However, due to the onset of a tonic-clonic seizure, reintubation was performed, followed by a post-seizure workup.

-Brain MRI: No abnormalities

- LP: Biochemical study normal

She was hospitalized for 26 days in the intensive care unit for Guillain-Barré syndrome with diaphragmatic involvement during a pregnancy estimated at 20 weeks and 1 day, which was complicated by two convulsive seizures.

A cesarean section was performed for maternal rescue, resulting in the birth of a male newborn weighing 250 grams.

An extubation attempt was made, and the patient's condition was reassessed:

- Ineffective swallowing; quadriplegic patient.

On day 59 of hospitalization, the patient experienced non-recoverable cardiorespiratory arrest despite resuscitation efforts.

Observation number 2:

The patient was a 37-year-old woman with no notable pathological history and left ventricular volume pressure (LVVP). The gynecological examination showed no detectable abnormality. The patient was admitted with an ascending motor deficit without sensory deficit for 48 hours, accompanied by dysphagia and dysphonia. These symptoms occurred in the context of apyrexia.

Clinical examination revealed an agitated patient with a blood pressure of 150/90 mmHg, no neurological signs of severity, a heart rate of 79 beats per minute, a respiratory rate of 22 cycles per minute, and a negative urine dipstick.

The patient was intubated upon admission. The paraclinical workup showed:

- ENMG: consistent with Guillain-Barré syndrome

- LP: Proteinorrachia: 0.65; Glucorrachia: 0.7.

- Inf serologies: EBV/CMV+

The patient was started on immunoglobulin (3 g/day) and a proton pump inhibitor (PPI) (40 mg/day).

There was no response, so a second course of immunoglobulins was administered.

Plasma exchanges were carried out, but there was no improvement.

The patient went into cardiorespiratory arrest and did not recover despite resuscitation.

Observation number 3:

Patient aged 29 IIIG/IIP, admitted for postpartum preeclampsia, who presented at d18 postpartum of a vaginal delivery (poorly monitored pregnancy) with tingling paresthesias of the lower limbs which progressed to an ascending motor deficit of the lower limbs and extension to the upper limbs, followed by development of right facial paralysis without sphincter disorders. The evolution was marked by the onset of respiratory distress, for which she was admitted to intensive care.

Clinical examination revealed a stable patient with blood pressure of 150/90 mmHg with no neurological signs of severity; heart rate of 75 beats/minute; respiratory rate of 17 cycles/minute; positive urine dipstick with two crosses; apyretic; absence of lower-limb oedema, a good safety globe with, on neurological examination, a picture of sensitivo-motor polyneuropathy in all 4 limbs, with tone tending to be flaccid in the lower limbs (MI) and normal in the upper limbs (MS); muscle strength rated 5/5 in the MS and 4/5 in the MI, more marked proximally; osteotendinous reflexes abolished in the MI, depressed in the MS, and cranial pairs intact. Electroneuromyogram (ENMG) revealed severe axonal damage.

Lumbar puncture (LP) revealed albuminocyte dissociation: WBC <3/mm3, proteinorachy 5.14.

The treatment was immediately started with intravenous immunoglobulin 2 g / kg (i.e., 0.4g/d), which was continued for 5 days. Her recovery was progressive, with improvement in muscle weakness. On the 20th day of her illness, she was able to walk with support and was referred for physiotherapy. Limb strength gradually improved. She had few residual sequelae at 3 months postpartum follow-up. The patient improved progressively and was fully recovered after 6 months.

Observation number 4:

Patient aged 26 IG/IP, admitted for a deficit of the four limbs, during a pregnancy of 26 weeks of unmonitored amenorrhea, evolving since the 4th month of pregnancy, this deficit was progressive in an ascending manner in the form of sensory then motor disorders.

The clinical examination revealed a stable patient with blood pressure at 110/60 mmHg; heart rate at 80 beats/minute; respiratory rate at 18 cycles/minute; negative urine strips; apyretic; absence of edema of the lower limbs. Obstetrically, there was no uterine contraction, the cervix was closed with fetal biometry that corresponded to the gestational age without abnormalities of the fetal adnexa and preserved Doppler

examination shows a picture of sensorimotor polyneuropathy in the 4 limbs made up of a tone which tends to be flaccid in the lower limbs (LM) and upper limbs (UL); muscle strength rated 4/5 in the UL and 3/5 in the LM, more marked proximally, osteotendinous reflexes abolished in the LM, depressed in the UL and intact cranial nerves. The electroneuromyogram (ENMG) showed severe axonal damage.

At lumbar puncture (LP), albumin -cytological dissociation.

The patient was admitted directly to intensive care and her treatment was immediately She started with intravenous immunoglobulins 2 g/kg (i.e., 0.4 g/day), which was continued for 5 days. Her recovery was progressive with improvement in muscle weakness. One month later, she could mobilize her MS and slightly her IM with support. Limb power gradually improved. She had residual sequelae until delivery at 36 weeks for a non-reassuring fetal condition by cesarean section. Postpartum, she improved gradually and she kept amyotrophy of the IM and gait disorder during one year of follow-up with normal brain imaging, then progressive resolution in the 2nd year postpartum with supportive measures.

Discussion

Pregnancy does not appear to offer significant protection against the development of GBS. Some suggest that the incidence of GBS appears to be similar between pregnant women and the general population, i.e., 1.1 to 1.8 / 100,000 [1], others report that the risk may be lower during pregnancy, secondary to the immunological adaptations of pregnancy [3]. Nevertheless, GBS is rare in pregnancy, but when it does occur, the disease is associated with maternal and perinatal morbidity, especially when not properly treated [2].

GBS occurs in all trimesters of pregnancy and in the postpartum period, but is particularly common in the third trimester and the first 2 weeks postpartum [1].

Early symptoms include abnormal sensations such as numbness, paresthesias or similar sensory changes. Paresthesias usually start in the toes and fingertips, then progress upwards, but do not usually extend beyond the wrists or ankles. Pain associated with GBS is most severe in the shoulder girdle, back, buttocks and thighs, and occurs with even the slightest movement [2] [10].

The upward progression is progressive and symmetrical. This stage generally lasts from one to three weeks, requiring hospitalization in an intensive care unit, and is characterized by an increased risk of false routes and respiratory failure, linked to diaphragm paralysis which can be fatal

This is followed by a plateau phase characterized by stabilization of symptoms, which can last from a few days to a few weeks, with the risk of progression to quadriplegia associated with the appearance of signs of autonomic nervous system damage: cardiac rhythm disorders and blood pressure variability. The seriousness of this stage is marked by the thromboembolic risk associated with prolonged bed rest, and the risk of respiratory and urinary infection. Recovery is observed in 85% of cases [10] [11].

Typical GBS was reported to be most frequent during pregnancy and the post-partum period in a study of 47 patients [4].

The precise cause of Guillain-Barré is unknown. However, 60% of cases followed a pulmonary or gastrointestinal infection. Influenza, cytomegalovirus, Epstein-Barr virus infection, mycoplasma pneumonia and HIV are some of the infections associated with GBS. Sometimes,

surgery and anesthesia can trigger the syndrome, and in rare cases, vaccination can increase the risk of GBS [2][5][10].

GBS can lead to life-threatening complications, particularly when the respiratory muscles or autonomic nervous system are involved. Loss of autonomic function is common in severe cases of GBS, manifesting as wide fluctuations in blood pressure with orthostatic hypotension and sinus tachycardia, and even cardiac arrhythmias. In one of our patients, respiratory compromise necessitated her admission to intensive care.

It can sometimes be difficult to distinguish GBS symptoms from other disorders of the brain and nervous system.

The following two tests are generally used to confirm the diagnosis:

a) Nerve conduction studies and electromyography tests, which measure nerve and muscle function,

b) Lumbar puncture shows increased protein levels with normal cell counts [2]. This was the case in our patients.

Neonatal transmission has been reported. This could be due to transplacental passage of maternal blocking antibodies [6].

The treatment of choice for GBS is intravenous immunoglobulin (IVIG) and plasma exchange. However, IVIG is preferable to plasma exchange, as it involves fewer complications. The cost of plasma exchange is much lower than that of IVIG, although both have similar results.

Thromboprophylaxis should be considered, as weakness and immobility may increase the risk of thrombotic events [7].

The outcome is generally good in the majority of GBS patients in terms of motor recovery and independence. GBS, in general, has a mortality incidence of 5%, but post-partum GBS mortality is reported to be higher (10%), underlining the importance of early diagnosis and rehabilitation [8] [9]. In our patients, we observed full recovery after IVIG treatment and rehabilitation after six months' follow-up in one, and IM sequelae in the 2nd patient after 1 year's follow-up resolved within a year.

As clinicians, we need a high index of suspicion and supportive measures for the patient. The cornerstone of GBS management in pregnancy and postpartum is access to the intensive care unit and IVIG therapy.

Conclusion

GBS should be considered in patients developing symmetrical flaccid motor weakness of acute onset during pregnancy or in the postpartum period. Early diagnosis with intensive multidisciplinary supportive care helps improve the prognosis for both mother and fetus.

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