

Guillain-Barré Syndrome in Pregnancy: An Unusual Case

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Abstract

Guillain-Barré syndrome (GBS) is rare in pregnancy with an estimated incidence between 1.2 and 1.9 cases per 100,000 people annually, and it carries a high maternal risk. We report a 29-year-old primigravida who had pain and progressive heaviness of both lower limbs in her third trimester of pregnancy. The attending gynecologist ascribed these symptoms to ongoing pregnancy. The intrapartum period (lower segment caesarian section) passed uneventfully. On third postpartum day, the patient developed weakness of all the four limbs. A detailed history and physical examination pointed toward GBS although there was no antecedent infective episode. Subsequent nerve conduction velocity studies and cerebrospinal fluid analysis confirmed GBS. All other investigations including electrolytes were normal. The patient improved without the introduction of immunomodulating therapy.

Keywords: GBS, immunomodulating therapy, post-partum period, pregnancy

Introduction

Guillain-Barré syndrome (GBS) represents a heterogeneous group of immune mediated peripheral neuropathies. A feature common to all GBS variants is a rapidly evolving polyradiculoneuropathy preceded by a triggering event, most often an infection. GBS generally manifests as a symmetric motor paralysis with or without sensory and autonomic disturbances. Delayed diagnosis is common in pregnancy or immediate post partum period because the initial non-specific symptoms may mimic changes in pregnancy. GBS should be considered in any pregnant patient complaining of muscle weakness, general malaise, tingling of the fingers, and respiratory difficulty.^[1,2]

We report this case due its rarity and high index of suspicion needed for its diagnosis. It highlights the combined role of gynecologist and physician in the management of GBS during pregnancy, which if missed can be detrimental, both for mother and fetus.

Case Report

A 29-year-old primigravida started with pain and mild heaviness in the both lower limbs distally associated with occasional pins and needles sensation in 35th week of pregnancy. Her symptoms

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were progressive. Over the next 10 days, the patient noticed knee buckling with frequent falls and difficulty in sitting and standing from supine position. At the end of 36th week, an emergency LSCS (lower segment caesarian section) was performed for decreased fetal heart rate. LSCS was uneventful and a healthy baby was born. Second post-partum day, the patient was discharged. On third post-partum day, the patient complained of weakness all the four limbs (in addition to persisting symptoms), more in lower limbs and was not able to maintain the erect posture. In this state, the patient was brought to our accident and emergency department. A detailed history was taken which revealed initiation of symptoms during third trimester of pregnancy without any preceding illness. Subsequent clinical examination revealed sinus tachycardia, fluctuating hypertension, power of 4/5 in upper limbs, 3/5 in lower limbs, hypotonia, areflexia (lower limbs), flexor plantar response, and mild involvement of all the modalities of sensation. A working diagnosis of GBS was made and all other investigations including nerve conduction studies, arterial blood gases, electrolytes, and later cerebrospinal fluid analysis were planned.

Arterial blood gas analysis showed $pO_2 = 89 \text{ mmHg}$, saturation = 94.6%, $pCO_2 = 42 \text{ mmHg}$, and pH = 7.39. Serum potassium was 4.1 meq/L, sodium 136 meq/L and bicarbonate 25 meq/L. Electrocardiography showed sinus tachycardia and routine blood chemistry was normal. Serological tests for campylobacter jejuni, EBV, cytomegalovirus, and mycoplasma pneumonae were negative. Electrophysiological studies revealed

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decreased amplitude of compound action potentials in median and common peronial nerves with decreased conduction velocity and abnormal F waves. After 7 days, lumbar puncture was performed and cerebrospinal fluid chemistry revealed raised protein of 7.8 g/L and normal cell count, confirming the diagnosis. The patient received treatment in the form of physiotherapy and close observation of symptoms and signs to which she rapidly responded with respect to power and sense of well-being over next 72 h, without the intervention of immunomodulating drugs. Sinus tachycardia and episodic hypertension was treated with atenolol. The patient was discharged after 2 weeks of hospital stay with power of 5/5 in upper limbs, 4+/5 in right lower limb, and 4/5 in left lower limb with persisting areflexia in lower limbs.

Discussion

GBS is thought to be immune mediated, but its pathogenesis remains unclear. About two thirds of patients have an infection within the previous 4-6 weeks, most commonly a flu-like illness or gastroenteritis. Implicated infectious agents include Mycoplasma pneumoniae, Campylobacter jejuni, Cytomegalovirus, and Epstin Bar virus.^[1] The preceding infection may cause an autoimmune response against the various components of the peripheral nerve myelin and sometimes the axon. GBS classically presents with pain, numbness, paresthesia, or weakness of the limbs and this can be mistaken for a psychological complaint, leading to delay in diagnosis and treatment.^[2]

GBS can occur in any trimester of pregnancy and post-partum period but specifically in third trimester and first 2 weeks post-partum. GBS is known to worsen in post partum period due to an increase in delayed type of hypersensitivity. da Silva, *et al.* reported a case of GBS, diagnosed at 15 weeks of pregnancy and aggravated postpartum.^[3] Up to 20% of patients are disabled after 1 year and a maternal mortality of 7% has been quoted (non-pregnant GBS has mortality <5%).^[4]

The management of GBS in pregnancy is similar to that in the non-pregnant population and includes intravenous immunoglobulins (IVIG), plasmapheresis, and ventilator support wherever required. Immunomodulation with plasmapheresis and IVIG has been found to improve treatment outcomes with full recovery in 70-80% of patients.^[5] Yerdelen, *et al.* reported a case of GBS in pregnancy (at 34 weeks) with overlapping forms of GBS subtypes, acute motor axonal neuropathy, and ophthalmoplegia. The patient was managed with ventilatory support, IVIG, plasmapheresis, and tracheostomy.^[6] Ventilatory support is required in 25-30% of non-pregnant patients, but respiratory problems may be worse in pregnancy because of splinting of the diaphragm.^[1] In cases requiring ventilatory support in pregnancy, the risk of premature birth has been noted to be greatly increased.^[7] Bahadur, *et al.* reported a 25-year-old, gravida 3, para 2, woman at 21 weeks of pregnancy with successful maternal and fetal outcome.^[8] Goyal, *et al.* have described the management of a primigravida presenting at 26 weeks of gestation with plasmapheresis.^[9] Vijayaraghavan *et al.* have also described its management at 16 weeks of pregnancy.^[2]

In conclusion, a high index of suspicion for early diagnosis and prompt intensive multidisciplinary supportive care in a GBS-complicated pregnancy improve the prognosis for both mother and fetus.

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