

Guillain – Barré Syndrome in Pregnancy

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Abstract

A case of Guillain Barre Syndrome in pregnancy is presented occurring in 28 years old primigravida. It was treated medically with plasmapheresis. Patient delivered a male baby of 1.1 kg with poor Apgar score. Patients condition improved gradually over three weeks. She has regained full functional capacity in 8 weeks time.

Key Words: Pregnancy, Autoimmune, Paralysis.

Introduction

Guillain – Barré syndrome (GBS) is an *acute inflammatory demyelinating polyneuropathy* (AIDP), an autoimmune disorder affecting the peripheral nervous system, usually triggered by an acute infectious process. It is included in the wider group of peripheral

neuropathies. There are several types of GBS, but unless otherwise stated, GBS refers to the most common form, AIDP. GBS is rare and has an incidence of 1 or 2 people per 100,000.¹ It is frequently severe and usually exhibits as an ascending paralysis noted by weakness in the legs that spreads to the upper limbs and the face along with complete loss of deep tendon reflexes. With prompt treatment by plasmapheresis or intravenous immunoglobulins and supportive care, the majority of patients will regain full functional capacity. However, death may occur if severe pulmonary complications and autonomic nervous system problems are present.² Guillain – Barré is one of the leading causes of non-trauma – induced paralysis in the world.

Case Report

A 28 year old lady, married for 4 years, primigravida resident of Lahore was admitted in labour room through emergency on 21 – 05 – 09. She was known hypertensive for three years. She presented with; H/O gestational amenorrhea of 30.4 wks. Increased B.P for 4 days. B.P record of 180 / 120, one day back at private clinic, Lower limb weakness and urinary incontinence for one day. Her physical examination revealed; a young, obese lady, well oriented in time place and person. Pulse was 88 b / min, B.P was 150 / 100, Temp was 98°F, Pallor, Jaundice and Edema were absent JVP was not raised, Systemic examination was

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unremarkable except for Central nervous system abnormalities : GCS 15/15, POWER : Lower limbs 3/5, Left upper limb 2/5, Right upper limb 3/5. Abdominal examination revealed Symphysis fundal height 28 weeks, Oblique lie, FHR 140 – 146 beats per min. Investigations initially carried out were Blood group B+ve; Hb 13.5 gm/dl; Platelet count 378000 / mm,³ Urine C\E, Albumin +, BSR 141 mg/dl; LFT; SGPT 23 IU/L; SGOT₃₂U/L; S.TB 0.6 mg/dl; ALK. PO₄ 218. RFTs: S. Urea 31 mg/d; S. Creatinine 0.9 mg/dl, HBSAg –ve, Anti HCV –ve. OBS USG; Single alive intrauterine pregnancy. Transverse lie, Placenta fundal, Amniotic fluid adequate, B.P.D 7 cm, F.L 5.6 cm, EF-BW 1.2 kg, Gest. age 29 weeks. Provisional diagnosis included: i Chronic HTN with superimposed pre-eclampsia. ii CVA.

Patient was shifted to medical ICU after consultation with neurologist for further management. In medical ICU, there was progressive deterioration in patients condition with paralysis involving both upper and lower limbs and she was shifted on ventilator because of respiratory compromise. In ICU multidisciplinary care involving, neurologist, pulmonologist and obstetrician was provided. C.T scan brain was carried out which revealed no infarct or hemorrhage. Final diagnosis of GBS (Guillain Barrie syndrome) was made. Further management started on the lines of GBS with supportive care, and plasma pheresis. On 26 – 05 – 09, she went in to spontaneous labour, and delivered a male baby of 1.1 kg with poor Apgar Score and thick paste like meconium. Supportive care and regular sessions of plasma pheresis were continued after delivery. Total 4 sessions were done. Patients condition improved gradually. She was discharged on 08 – 06 – 09 with advice of follow up in medical and Gynae OPD.

Discussion

Pregnancy with Guillain – Barré syndrome is a rare occurrence. The incidence is 1.7 cases per 100,000 of the population.³ The mother will generally improve with treatment but death of the fetus is a risk. The risk of Guillain – Barré syndrome increases after delivery, particularly during the first two weeks postpartum. There is evidence of *Campylobacter jejuni* as an antecedent infection in approximately 26% of disease cases.⁴ However, 60% of cases do not have a known cause; One study suggests that some cases are triggered by the influenza virus, or by an immune reaction to the influenza virus.⁵ Congenital and neonatal Guil-

lain – Barré syndrome have also been reported.⁶ All forms of Guillain – Barré syndrome are due to an immune response to foreign antigens (such as infectious agents) that are mistargeted at host nerve tissues, thought to be gangliosides. The end result of such auto-immune attack on the peripheral nerves is damage to the myelin, the fatty insulating layer of the nerve, and a nerve conduction block, leading to a muscle paralysis that may be accompanied by sensory or autonomic disturbances.

The disorder is characterized by symmetrical weakness which usually affects the lower limbs first, and rapidly progresses in an ascending fashion. Patients generally notice weakness in their legs, with or without dysesthesias (numb-ness or tingling), Facial weakness, oropharyngeal dysphagia and respiratory difficulties. Most patients require hospitalization and about 30% require ventilatory assistance.⁷ Sensory loss, if present, usually takes the form of loss of proprioception (position sense) and areflexia (complete loss of deep tendon reflexes), Bladder dysfunction may occur in severe cases but should be transient.

Diagnostic criteria is: relatively symmetrical weakness of two or more limbs due to neuropathy, areflexia, disorder course < 4 weeks and exclusion of other causes, absence of fever, typical CSF findings obtained from lumbar puncture electrophysiologic evidence of demyelination from electromyogram. Supportive care with monitoring of all vital functions is the cornerstone of successful management in the acute patient. Early intubation should be considered in any patient with a vital capacity (VC) < 20 ml/kg, rapid progression of disorder, or autonomic instability. Once the patient is stabilized, treatment of the underlying condition should be initiated as soon as possible. Either high – dose intravenous immunoglobulins (IVIg) at 400 mg/kg for 5 days or plasmapheresis can be administered,^{8,9} as they are equally effective and a combination of the two is not significantly better than either alone. Following the acute phase, the patient may also need rehabilitation to regain lost functions. Most of the time recovery starts after the fourth week from the onset of the disorder. Approximately 80% of patients have a complete recovery within a few months to a year. About 5 – 10% recovers with severe disability. The death rate among patients with this disorder is still about 2 – 3% even in the best intensive care units. Worldwide, the death rate runs slightly higher (4%), About 5 – 10% of patients have one or more late relapses, in which case they are then classified as having (CIPD) chronic inflammatory demyelinating poly-

neuropathy. Poor prognostic factors include: 1) age > 40 years, 2) history of preceding diarrheal illness, 3) requiring ventilator support, 4) high anti-GM1 titre and 5) poor upper limb muscle strength. Despite the rarity of the situation, early diagnosis and prompt treatment can considerably decrease the morbidity and mortality associated with the disease.

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