

# Research Journal of Pharmaceutical, Biological and Chemical Sciences

## Acute Motor and Sensory Axonal Neuropathy in Pregnancy: A Rare Case.

N Nikath Nasreen\*, and K Saraswathi.

Sree Balaji Medical College and Hospital, Chrompet, Chennai, 600044, Tamil Nadu, India.

### ABSTRACT

This is a rare case of AMSAN with good prognosis. Guillain-Barré syndrome (GBS) was first described in 1916 (Guillain G, 1916) and is approaching its 100th anniversary. Guillain barre syndrome is rare in pregnancy. incidence is between 1.2 to 1.9 per 100000 persons annually. It is an autoimmune neurological disorder. There are many subtypes acute inflammatory demyelinating polyradiculoneuropathy (AIDP) being most common followed by acute motor axonal neuropathy (AMAN) and much rarer is acute motor and sensory axonal neuropathy (AMSAN). IVIG and plasmapheresis is the mainstay of treatment.

**Keywords:** Guillain barre syndrome, Autoimmune neurological disorder, Acute motor and sensory axonal neuropathy, Plasmapheresis.

*\*Corresponding author*

## INTRODUCTION

Our understanding of the Guillain-Barré syndrome has improved greatly over the last decade with a much clearer idea of the clinical subtypes of the syndrome and the pathogenesis of some of the rarer variants. 2016 will mark the centenary of the original description by Guillain, Barré and Strohl [1]. They described a rapidly progressive motor disorder associated with absent reflexes and a raised CSF protein in the absence of the expected cerebrospinal fluid (CSF) pleocytosis that characterised poliomyelitis. It became clear, over the ensuing years that the syndrome varied in severity so that in its severest form it could lead to respiratory paralysis and death [2]. Acute inflammatory demyelinating polyradiculoneuropathy (AIDP) is the most frequent subtype in the Western world with a primarily demyelinating pathology and various degrees of secondary axonal damage. Acute motor axonal neuropathy (AMAN) [3] is the next most frequent and appears to be a primary axonal disorder affecting just motor nerves. Axonal variants involving both sensory and motor nerves are much rarer Acute Motor and Sensory Axonal Neuropathy (AMSAN) [3].

### Case Report

A 21 year gravida 2 abortion 1 female with gestational age 24 weeks came with complaints of progressive weakness of lower limbs and difficulty in walking for 4 days following an episode of diarrhoea and fever for 1 week. Patient was hypotonia with areflexia and bilateral plantar flexion. ECG showed sinus tachycardia. Electrolyte imbalance suspected and ruled out. Nerve conduction study revealed motor and sensory neuropathy. She was treated with IVIG for 5 days and physiotherapy done. Her condition improved and she started walking with support.

At 38 weeks she came with complaints of decreased fetal movements and as she had fetal distress, she was taken up for emergency LSCS (Lower segment caesarean section). She delivered an alive term 3.1kg male baby with good APGAR. Her post-natal period was uneventful.

## 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 Epstein Bar virus [4]. 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 [5].

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 [6]. 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%) [7].

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 [8].

## 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.

## REFERENCES

- [1] G Guillain, J Barré, and A Strohl. *Bulletins et Memories de la Societe Medicale des Hopitaux de Paris* 1916;40:1462-1470.



- [2] WKJ Haymaker. *Medicine* 1949;28:59–141.
- [3] JW Griffin, CY Li, TW Ho et al. *Brain* 1995;118(3):577–595.
- [4] Hughes RA, Cornblath DR. *Lancet* 2005;366:1653–66.
- [5] Vijayaraghavan J, Vasudevan D, Sadique N, Rajeswari KS, Pondurangi M, Jayshree. *J Indian Med Assoc* 2006;104:269–70.
- [6] Campos da Silva F, de Moraes Paula G, Dos Santos Esteves Automari CV, Mendes de Almeida DS, Ubirajara Cavalcanti Guimarães R. *Gynecol Obstet Invest* 2009;67:236–7.
- [7] Furara S, Maw M, Khan F, Powell K. *Obstetric Med* 2008;1:99–101.
- [8] Elovaara I, Apostolski S, van Doorn P, Gilhus NE, Hietaharju A, Honkaniemi J, et al. *Eur J Neurol* 2008;15:893–90.