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Guillain Barre Syndrome in Pregnancy- A Rare Case Report

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Abstract
In pregnancy, Guillain Barre syndrome (GBS) is rare with an estimated incidence of 1.2-1.9 per one lakh annually[1]. It is the most common cause of acute flaccid paralysis affecting all age groups[2]. It can be associated with high maternal and perinatal morbidity if it is not identified and treated promptly. A high index of suspicion, supportive measures, access to intensive care unit and Intravenous immunoglobulin/ Plasmapheresis/ Steroids therapy are cornerstones of management in GBS complicating pregnancy. Surgery and anaesthesia maybe triggers for relapse in association with an overall increase in pro inflammatory cytokines in post partum period. Neurologists and Obstetricians should be aware of the risks of relapsing GBS in the immediate post partum period.
We are reporting a rare case of GBS complicating pregnancy in 2nd trimester. She made a good recovery with steroids and supportive measures.
Keywords: *Guillain Barre syndrome, polyradiculoneuropathy.*

Introduction

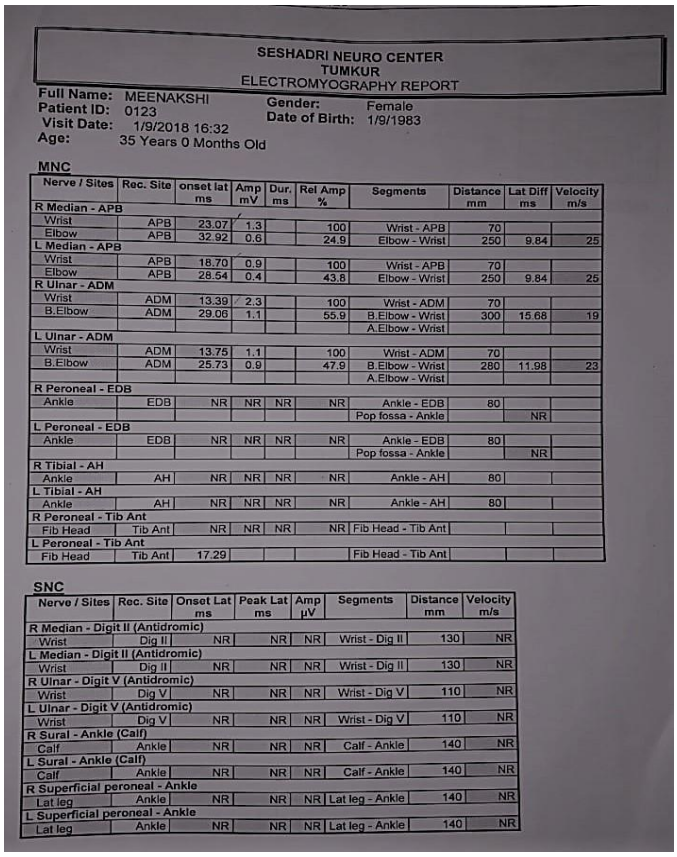
Guillain Barre Syndrome is an acute, frequently severe and fulminant polyradiculoneuropathy that is autoimmune in nature. A feature common to all variants of GBS is a rapidly evolving polyradiculoneuropathy preceded by a triggering event, most often an infection. It manifests as a symmetric motor paralysis, areflexia, with or without sensory, autonomic disturbances and albumin cytological dissociation. The pathogenesis of GB syndrome is unknown, but it thought to be due to molecular mimicry between

epitopes found on cell walls of micro organisms and gangliosides found on Schwann cell membrane[2]. Delayed diagnosis is common in pregnancy or immediate post partum period because the initial non specific symptoms may mimic physiological changes in pregnancy[3]. GBS can be considered in any pregnant patient complaining of muscle weakness, general malaise, tingling of fingers and respiratory difficulty[4,5]. Relapse has been reported to occur in 5.5-6.8% of patients[6].

Case report

This is a case report of a 35 year old female who presented to Department of Medicine at 24 weeks of gestation in her first pregnancy with Complains of weakness of both lower limbs since 1 month& weakness of both upper limbs since 15days. There was no history suggestive of bulbar, respiratory, autonomic or cranial nerve involvement. There was history of fever & loose stools 10days prior to the onset of weakness of lower limbs which had lasted for 2days. She had received immunisation for tetanus in view of pregnancy but otherwise her progress during this pregnancy has been unremarkable. Neurological examination revealed reduced tone, power in both lower limbs. Deep tendon reflexes were absent in both lower limbs, bilaterally plantar reflexes were mute. Sensory system was involved in lower limbs (touch, temperature, vibration). Respiratory examination was within normal limits.

Serum chemistry, hemogram & urine analysis were normal. No atypical cells were seen in peripheral smear. Serological tests for HIV, HbSAg, HCV, VDRL were negative. Thyroid function tests were within normal limits. Toxoplasma, Rubella, Cytomegalovirus &herpes simplex virus antibodies, CSF analysis were not done due to lack of consent. Nerve Conduction Study was done which showed severe Sensori motor axonal polyradiculoneuropathy.



SESHADRI NEURO CENTER
TUMKUR
ELECTROMYOGRAPHY REPORT

Full Name: MEENAKSHI
Patient ID: 0123
Visit Date: 1/9/2018 16:32
Age: 35 Years 0 Months Old
Gender: Female
Date of Birth: 1/9/1983

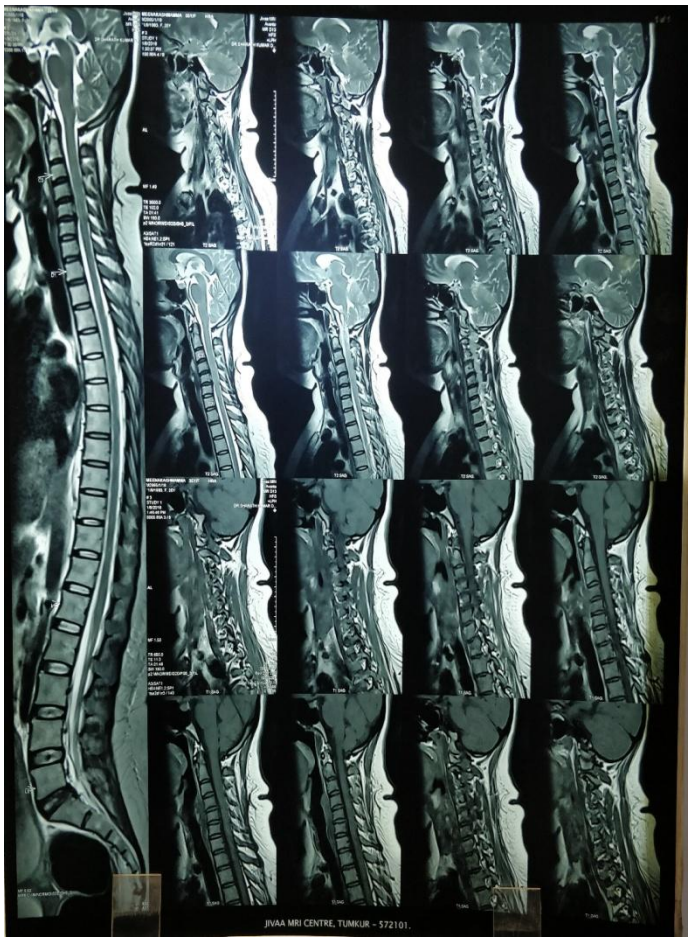
MNC

Nerve / Sites	Rec. Site	onset lat ms	Amp mV	Dur. ms	Rel Amp %	Segments	Distance mm	Lat Diff ms	Velocity m/s
R Median - APB									
Wrist	APB	23.07	1.3		100	Wrist - APB	70		
Elbow	APB	32.92	0.6		24.9	Elbow - Wrist	250	9.84	25
L Median - APB									
Wrist	APB	18.70	0.9		100	Wrist - APB	70		
Elbow	APB	28.54	0.4		43.8	Elbow - Wrist	250	9.84	25
R Ulnar - ADM									
Wrist	ADM	13.39	2.3		100	Wrist - ADM	70		
B.Elbow	ADM	29.06	1.1		55.9	B.Elbow - Wrist	300	15.68	19
						A.Elbow - Wrist			
L Ulnar - ADM									
Wrist	ADM	13.75	1.1		100	Wrist - ADM	70		
B.Elbow	ADM	25.73	0.9		47.9	B.Elbow - Wrist	280	11.98	23
						A.Elbow - Wrist			
R Peroneal - EDB									
Ankle	EDB	NR	NR	NR	NR	Ankle - EDB	80		
						Pop fossa - Ankle		NR	
L Peroneal - EDB									
Ankle	EDB	NR	NR	NR	NR	Ankle - EDB	80		
						Pop fossa - Ankle		NR	
R Tibial - AH									
Ankle	AH	NR	NR	NR	NR	Ankle - AH	80		
L Tibial - AH									
Ankle	AH	NR	NR	NR	NR	Ankle - AH	80		
R Peroneal - Tib Ant									
Fib Head	Tib Ant	NR	NR	NR	NR	Fib Head - Tib Ant			
L Peroneal - Tib Ant									
Fib Head	Tib Ant	17.29				Fib Head - Tib Ant			

SNC

Nerve / Sites	Rec. Site	Onset Lat ms	Peak Lat ms	Amp µV	Segments	Distance mm	Velocity m/s
R Median - Digit II (Antidromic)							
Wrist	Dig II	NR	NR	NR	Wrist - Dig II	130	NR
L Median - Digit II (Antidromic)							
Wrist	Dig II	NR	NR	NR	Wrist - Dig II	130	NR
R Ulnar - Digit V (Antidromic)							
Wrist	Dig V	NR	NR	NR	Wrist - Dig V	110	NR
L Ulnar - Digit V (Antidromic)							
Wrist	Dig V	NR	NR	NR	Wrist - Dig V	110	NR
R Sural - Ankle (Calf)							
Calf	Ankle	NR	NR	NR	Calf - Ankle	140	NR
L Sural - Ankle (Calf)							
Calf	Ankle	NR	NR	NR	Calf - Ankle	140	NR
R Superficial peroneal - Ankle							
Lat leg	Ankle	NR	NR	NR	Lat leg - Ankle	140	NR
L Superficial peroneal - Ankle							
Lat leg	Ankle	NR	NR	NR	Lat leg - Ankle	140	NR

MRI Cervical spine was done which ruled out compression myelopathy.



Weakness did not evolve.

A diagnosis of Guillain Barre syndrome was made & plan of management included Plasmapheresis, IVIg or Steroids. The patients attenders did not consent for the use of Plasmapheresis or intravenous immunoglobulin. After a Glucose challenge test & Anomaly scan which was normal, she was started on Tab. Methyl Prednisone (Category C drug) 1gram for 3 day, Tab. Methyl Prednisone 40mg tapered and stopped over next 7 days. She was undergoing physiotherapy.

She started improving and over next 3 months she was able to walk with support & upper limb power had improved to normal power. She could walk unsupported though with mild unsteadiness, when she was discharged. She remained neurologically stable subsequently & was admitted in her 37th week of gestation with high blood pressure recording. PIH profile was within normal limit. Antepartum fetal surveillance was non reassuring. She was taken up for Caesarean section under spinal anaesthesia. Indication was precious pregnancy with fetal distress.

Intra op period was uneventful. Post partum period was uneventful & she was discharged on post op day 9.She remained neurologically asymptomatic subsequently.

Discussion

GBS is a rare occurrence in pregnancy but can be associated with severe co morbidities if unrecognised, especially respiratory muscle involvement and dysautonomia. GBS is an immune mediated neurological disorder resulting primarily in muscle paralysis, which in most cases is symmetrical. About two thirds of patients have an infection within the previous 4-6weeks, most commonly a flu like illness or gastroenteritis. Implicated infectious agents include Mycoplasma pneumonia, Campylobacter jejuni, Cytomega-lovirus, Epstein Barr virus.

Pregnancy is associated with a decrease in cellular immunity and increase in humoral immunity; this shift is because of production of IL10. Obstetricians should have a high index of suspicion if a pregnant woman complains of muscle weakness or breathlessness in the context of recent diarrhoeal illness or a viral infection. Diagnosis is usually made on clinical grounds supported by CSF examination, serology and nerve conduction studies. Postpartum relapses can occur. The efficacy of IVIg is well established and can be safely given during pregnancy. Judicious use of steroids is also beneficial as seen in this case.

After pregnancy is terminated, this is reversed with an overall increase in pro inflammatory cytokines[7]and this accounts for the increased incidence and worsening of symptoms in the post partum period particularly during the first 2 weeks post partum[8,9]. 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%) [10]. The management of GBS in pregnancy is similar to that in the non pregnant population and includes Intravenous Immunoglobulin, Plasmapheresis, ventilator support wherever required and other supportive measures like identification and treatment of infections, prophylaxis for venous thromboembolism, pain management and management of psychosocial distress resulting from the disease. GBS occurring in pregnancy is associated with an increased need

for ventilator support and an increase in maternal mortality. In cases requiring ventilator support, the risk of premature birth has been noted to be greatly increased. Immunomodulation with IVIg or plasmapheresis has been found to improve treatment outcomes with full recovery in 70-80% of patients.

Most of the patients can be left for spontaneous labour; abortion and Caesarean section is not considered to be indicated.

Poor prognosis in GBS has been associated with rapid onset of illness, severe degree of paralysis, muscle wasting, prolonged period of peak paralysis lasting more than 2 weeks and a delay in onset of recovery lasting more than 3 weeks as well as respiratory involvement.

Conclusion

Early diagnosis, multidisciplinary input and prompt immune modulatory therapy are the cornerstones in management of GBS during pregnancy and post partum to improve outcomes for the mother and the fetus.

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