

### Case Report

## TWO UNUSUAL CASES OF RECURRENT GBS PRESENTING WITH RECURRENT SUBACUTE INTESTINAL OBSTRUCTION: CASE REPORT

**\*Hibu Habung, Shri Ram Sharma and Masaraf Hussain**

*Department of Neurology, NEIGRIHMS-Shillong*

*\*Author for Correspondence*

### ABSTRACT

Guillain-Barre Syndrome is an immune poly radiculoneuropathy. Guillain-Barre Syndrome can recur in 1–6% of patients, though it has been reported to occur in 1–10% of patients after asymptomatic period of several months to several years. Autonomic neuropathy is an important and common complication of Guillain-Barre syndrome which can be manifested in cardiovascular, sudomotor, gastrointestinal and other systems involving both sympathetic and parasympathetic fibers. Here we report 2 unusual cases of Guillain-Barre Syndrome who had recurrent episodes associated with subacute intestinal obstruction in each episode.

**Keywords:** *Guillain Barre Syndrome, Miller Fisher Syndrome, Autonomic Neuropathy, Intestinal Obstruction*

### INTRODUCTION

Guillain-Barre Syndrome (GBS) is an immune polyradiculoneuropathy that presents with ascending bilateral lower extremity weakness and are flexia and that affects all age groups with a slight male predisposition. Recurrent Guillain-Barre Syndrome (RGS) can recur in 1–6% of patients, though it has been reported to occur in 1–10% of patients after asymptomatic period of several months to several years (Dy *et al.*, 2013). Risk factors for RGS include age less than 30, milder symptoms, and history of Miller Fisher Syndrome variant (Mossberg *et al.*, 2012). There appears to be no significant difference between RGS and GBS episodes with respect to similar clinical symptoms and similar or different triggering events. The episode appears to be shorter with half of the patients accumulating deficits (Dy *et al.*, 2013). Autonomic dysfunction is present in up to two-thirds of GBS patients (Lichtenfeld, 1971). Autonomic neuropathy is an important and common complication of Guillain-Barre syndrome (GBS). Manifestations may be present in cardiovascular, sudomotor, gastrointestinal and other systems involving both sympathetic and parasympathetic fibers (Zochodne, 1994). Paralytic ileus occurs in GBS due to the involvement of sympathetic and parasympathetic fibres (Burns *et al.*, 2001).

### CASES

Here we report 2 cases of GBS who had recurrent episodes and each episodes were associated with autonomic dysfunction in the form of paralytic ileus leading to subacute intestinal obstruction.

**Case 1:** 26 years male presented with complain of sudden onset symmetrical, ascending weakness of all limbs associated with tingling sensation since 11 days. There was no history of altered sensorium and no bladder dysfunction.

On 8<sup>th</sup> day he developed pain and distention of abdomen with episodes of vomiting and inability to pass stool and flatus. Clinically he had areflexic quadriparesis with flexor plantar responses and no significant sensory findings. His abdomen was distended with tympanitic note on percussion. Relevant findings in investigation showed mildly elevated SGOT/SGPT, albuminocytological dissociation in CSF and absent H reflex with conduction blocks in Nerve Conduction studies. X-ray erect abdomen showed dilated gut loops with fluid levels (figure 1). It was diagnosed as subacute intestinal obstruction by surgeons, which improved with conservative measures. He also gave history of similar episode of sudden onset quadriparesis along with inability to pass stool and flatus for 4-5 days with vomiting 3 years back and had completely improved over 3-4 weeks. He was managed conservatively and was discharged with diagnosis

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of recurrent AIDP (acute inflammatory demyelinating polyneuropathy) with subacute intestinal obstruction after he improved.

**Case 2:** A 42 years male was admitted in the surgical ward as a case of subacute intestinal obstruction following pain and distention of abdomen with non passage of stool and flatus and vomiting episodes since 5 days and was being managed conservatively. He was referred to neurology for complain of imbalance in walking and occasional diplopia since past 1 week. There was no history of any drug intake recently and no bladder dysfunction or bulbar weakness. Clinically his higher mental function was normal and no signs of meningeal irritation. Gaze evoked nystagmus was seen bilaterally. He had ataxic gait with areflexia of all limbs. Muscle power of all limbs was normal with bilaterally flexor plantar reflexes. Sensory system was normal. He gave history of similar illness 6 months back when he had imbalance in walking with diplopia and dysarthria along with non passage of stool and flatus with vomiting for 2-3 days. However he improved spontaneously over 3-4 weeks. On investigation relevant findings were mildly elevated SGOT/SGPT, albuminocytological dissociation in CSF and dilated gut loops in X-ray abdomen (figure 2). His Nerve Conduction studies were normal. However, a triad of ataxia, areflexia and ophthalmoplegia was present with albuminocytological dissociation in CSF suggestive of miller fisher syndrome. Anti GQ1b antibodies could not be done as the patient could not afford. He was managed conservatively and improved over 3 weeks after which he was discharged.



Figure 1



Figure 2

### DISCUSSION

GBS is an immune-mediated, acute, polyradiculoneuropathy that usually causes diffuse weakness. Peripheral nervous system pathology is visible by the lack of motor response to peripheral nerve stimulation. This syndrome can occur at any age but is most common between ages 30 and 50. AIDP is the most common form of GBS while Miller Fischer Syndrome forms 5% of GBS subtypes. Recurrence of GBS can occur in 1–6% of patients after asymptomatic period of several months to several years (Dy *et al.*, 2013).

Autonomic neuropathy is an important and common complication of Guillain-Barre syndrome which can be manifested in cardiovascular, sudomotor, gastrointestinal and other systems involving both sympathetic and parasympathetic fibers. Our patients presented with typical history suggestive of GBS and were confirmed clinically and diagnostically. Both of them had recurrence of the episodes and during each episode manifested autonomic dysfunction involving the gastrointestinal system in the form of paralytic ileus leading to subacute intestinal obstruction. Paralytic ileus occurs in GBS due to the

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involvement of sympathetic and parasympathetic fibres (Burns *et al.*, 2001). Dysautonomia in GBS might predict a relatively poor prognosis (Yuan *et al.*, 2000). However, both our patients recovered well spontaneously with conservative measures.

### **Conclusion**

Recurrence of GBS can occur after months or years of asymptomatic period in 1-6% of patients. Autonomic dysfunction is an important and common complication of Guillain-Barre syndrome. Isolated involvement of gastrointestinal system with paralytic ileus leading to intestinal obstruction is very uncommon presentation in GBS. The present cases demonstrate that physicians need to be aware of recurrence of GBS and that dysautonomia may even cause bowel obstruction.

### **REFERENCES**

- Burns TM, Lawn ND, Low PA et al., (2001).** Adynamic ileus in severe Guillain-Barre syndrome. *Muscle and Nerve* **24** 963–5.
- Dy M, Leshner RL and Crawford JR (2013).** An Unusual Case of Recurrent Guillain-Barre Syndrome of a Different Subtype Five Years after Initial Diagnosis. *Case Reports in Neurological Medicine* **2013** 356157. doi: 10.1155/2013/356157.
- Lichtenfeld P (1971).** Autonomic dysfunction in the Guillain-Barre syndrome. *American Journal of Medicine* **50** 772–80.
- Mossberg N, Nordin M, Movitz C, Nilsson S, Hellstrand K, Bergstrom T et al., (2012).** The recurrent Guillain-Barré syndrome: a long-term population-based study. *Acta Neurologica Scandinavica*. **126** 154-61.
- Yuan CL, Wang YJ and Tsai CP (2000).** Miller fisher syndrome: a hospital-based retrospective study. *European Neurology* **44** 79-85.
- Zochodne DW (1994).** Autonomic involvement in Guillain-Barré syndrome: a review. *Muscle and Nerve* **17** 1145-55.