Guillain-Barré Syndrome Variant presenting as Acute Descending Paralysis in Diabetes

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ABSTRACT

Guillain-Barré Syndrome (GBS) is an acute monophasic immune-mediated polyradiculopathy. Neuropathic or radicular pain and dysautonomia are common features in all forms of GBS. Phenotypic variants are hypothesized to be mediated by molecular mimicry targeting peripheral nerve motor axons, with resulting weakness deviating from the classic symmetric “ascending” pattern. Weakness can range from mild to severe flaccid quadriplegia and respiratory failure within days of onset. Urinary retention and gastrointestinal dysfunction are seen in approximately 5% of variant cases. A spectrum of clinical features necessitates the use of laboratory testing and diagnostic modalities to exclude mimics and confirm a diagnosis of GBS. A high clinical suspicion must exist as GBS responds to treatment with plasmapheresis or IVIG. Our patient embodied both a diagnostically challenging presentation and clinical response consistent with variant GBS.

Key Words: GBS, Variant, Descending

CASE PRESENTATION

49 year old male, known diabetic patient presented with numbness and weakness of both upper limb. He had history of acute gastroenteritis 2 weeks ago.

On examination, there was total areflexia and plantar was flexor bilateral. MRI imaging of the brain and cervical spine was unremarkable. 12 hours later he developed weakness of both lower limbs. There was rapid development of descending areflexic quadriplegia. There was no dysphagia, dyspnoea or bladder or sensory symptoms. There were no signs and symptoms suggestive of cranial nerve involvement.

A CSF study was done which showed the classic pattern of albuminocytologic dissociation. CSF analysis was negative for anti-GM1 and anti-GQ1B antibodies. Nerve conduction study revealed absent F-wave latencies and prolonged H-reflexes suggestive of early AIDP. Initial EMG suggested axonal loss and segmental demyelination. A diagnosis of variant GBS with descending paralysis was made and the patient was started on IV Immunoglobulin at a dose of 2gm/kg was administered over a period of five days. EMG after treatment showed a recovering motor response, confirming the diagnosis. There was total recovery of motor function of both upper and lower limbs.

DISCUSSION

Determination of GBS variants is based on the types of nerve fibers involved, predominant mode of fiber injury and evidence or absence of alteration in consciousness. The pharyngeal-cervical-brachial motor variant manifests with a descending paralysis, mimicking botulism. Treatment strategies vary depending on the
breadth and severity of weakness and often involve general supportive care. Indications for plasmapheresis or Intravenous immunoglobulin include weakness impairing function or respiratory involvement.

CONCLUSION

In patients who present with rapid progression of descending paralysis, variant GBS should be considered amongst the differential.

REFERENCES