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Type 1 Respiratory Failure in Pharyngeal-Cervical-Brachial Guillain-Barré Syndrome: A Case Report from Tanzania

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Abstract

The Pharyngeal-Cervical-Brachial (PCB) variant of Guillain-Barré Syndrome (GBS) is extremely uncommon. It is characterized by facial diplegia, bulbar symptoms, and rapidly worsening oropharyngeal and cervicobrachial paralysis of the upper extremities. Serial nerve conduction studies suggest that PCB represents a localized subtype of Guillain-Barré syndrome characterized by axonal rather than demyelinating neuropathy. Given that early detection and treatment are essential to preventing fatal bulbar weakness, it should be suspected in any case with acute-onset flaccid symmetrical weakness of the upper extremities. Herein, we report a case of a middle-aged woman who presented with a pharyngeal-cervical-brachial variant of GBS.

Keywords

Pharyngeal-Cervical-Brachial Variant, Guillain-Barré Syndrome, Respiratory Failure, Case-Report

1. Introduction

Guillian-Barré Syndrome (GBS) is an immune-mediated inflammatory, demyelinating polyneuropathy with an immediate onset that is monophasic and frequently develops after an antecedent infection. It progresses to a nadir over a period of up to four weeks and is characterized by sharply ascending weakness, mild sensory loss, and hyporeflexia or areflexia [1].

Patients with the Pharyngeal Cervical Brachial (PCB) variant of Guillain-Barré Syndrome (GBS) usually exhibit areflexia in the upper limbs along with quickly

progressing oropharyngeal and cervicobrachial weakness. Lower limb power usually remains intact or very slightly affected, suggesting that PCB is a localized subtype of GBS [2]. The Pharyngeal, Cervical, and Brachial (PCB) form of GBS is infrequently described. This article describes a rare variant that can lead to acute respiratory failure. Our goal is to present a description of this rare condition with a rare association and a great IVIg response.

2. Case Report

A 37-year-old woman presented at EMD with a three-day history of acutely progressive weakness of both upper and lower limbs. The upper limb weakness was noticed initially as she started experiencing difficulty in holding objects which then progressed to difficulty in lifting both arms. She also complained of neck pain, associated with difficulty in holding her neck, difficulty in swallowing with globus sensation at the level of throat, and slurred speech. There was no history of altered sensorium, loss of consciousness or seizure. There was no bowel and bladder involvement at the onset of the disease. However, she had antecedent symptoms of diarrhea a week before which were treated symptomatically and recovered. The patient had been treated for peripheral arterial disease in the past; otherwise there is no history of chronic illnesses. There is no history of drug exposure. There was no similar history in the family. On examination she was hypertensive with BP of 197/121 mmHg, ventricular tachycardia with HR of 187 b/m, and tachypneic saturating at 99% on 15 L of oxygen therapy by non-rebreather facemask. She had dysarthria, with weak gag reflex suggestive of bulbar palsy and there was difficulty swallowing. She had areflexia in the upper and lower limbs. The neck extensors were stronger than flexors at grade 3− and 3+. Power was 3/5 on the upper limbs and 3/5 on the lower limbs according to Medical Research Council grading. Examination of other systems was unremarkable. Blood counts, liver function tests, renal function tests, thyroid hormone levels, and Serum Vitamin B12, were normal. Arterial carbon dioxide pressure was 36mmhg, and oxygen pressure was 60mmhg. CSF examination showed a protein level of 102 mg/dl, sugar 70 mg/dL. MRI spine revealed degenerative cervical spine disease; CT brain was normal. Nerve conduction test studies revealed prolongation of F-wave latency, and reduced compound muscle action potential of both the upper and lower limbs. Six hours after hospitalization, the patient suffered acute hypoxemic respiratory failure due to secretions and inability to protect airway secondary to pharyngeal paresis. Patient was admitted to ICU and was successfully intubated. Differential diagnosis of space-occupying lesion, PCB variant of GBS and type 1 respiratory failure secondary to GBS was made and she was managed conservatively with IV immunoglobulin at a dose of 30 g od for five days. The patient showed dramatic improvement within 4 days of initiating the treatment. By the end of 2 weeks, patient was extubated, and her arm and leg weakness improved partially. Deep tendon reflexes were absent in bilateral upper and lower limbs. On- follow up visit after 2 weeks, at the neurology clinic, the deep tendon reflexes

were present.

3. Discussion

The primary symptoms we observed in our patient were bilateral weakening of the upper limb with mild weakness in the lower limbs, slurred speech, and dysphagia. This is in keeping with the study done by Ropper et al, where it was described as a PCB variant as patients who develop rapidly progressive oropharyngeal, neck and shoulder weakness but a larger study showed the presence of both arm and leg weakness [3]. Our patient exhibited both upper and lower limb weakness with slight severity in the upper limbs than in the lower limbs with hip flexion weakness. Nagashima et al described patients with PCB symptoms and leg weakness as PCB with GBS overlap; this may explain the reason why our patient had lower-limb weakness which was preceded by upper-limb weakness.

Our patient's clinical, laboratory, and electrophysiological results led to the diagnosis of PCB variant GBS. Though we did not test anti-ganglioside antibodies as it is not required for diagnosis. It has been documented that patients with a lower cranial nerve type of GBS have facial muscle involvement; however, there was no facial weakening in our patient. This is similar to a study done by Nagashima where out of 100 patients with PCB, only 4 patients initially experienced facial weakness. According to a study, antecedent upper respiratory tract infections and diarrhea [4], which are related to GBS, were observed in 70% and 31% of patients, respectively. A study revealed that antecedent infection did not differ between PCB and GBS [5]. Our patient had diarrhea a week before the onset of these symptoms for which she received treatment and recovered well.

Our patient presented with hypertension of 197/121 mmHg, and arrhythmias. This is in parallel to a review study done by Lehman et al, where they reported two-thirds of patients with GBS present with autonomic dysfunction and manifest as hypertension or hypotension, cardiac arrhythmias, abnormal sweating, and/or gastrointestinal dysmotility, urinary retention or constipation [6].

However, in this report, our patient did not have urinary retention or constipation at the time of admission. Patients with the PCB type of GBS may have generalized areflexia or may solely suffer areflexia of the arms. Our patient exhibited generalized areflexia but relatively preserved power of 3/5 in both upper and lower limbs.

From an electrophysiological perspective, PCB is viewed as an AMAN variation continuum exhibiting axonal conduction failure [7]**.** Frequently, individuals who exhibit PCB are initially misdiagnosed as suffering from botulism, myasthenia gravis, or brainstem stroke [5]. Before being referred to our hospital, the private clinic classified our patient as having had a stroke. Initially, we considered a space-occupying lesion; however, to rule out central causes, brain and cervical MRI was done which was found to be normal.

Lumbar punctures should be performed after all other possible causes have been ruled out. Albuminocytologic dissociation is seen in the CSF analysis along with

increased protein levels and a normal WBC count. Although it is not a standard diagnostic procedure, brain/spinal cord imaging with CT/MRI can assist in ruling out alternative diagnoses [8]. Up to 3% of patients have the PCB variant of GBS. Rather than demyelinating, it is an axonal neuropathy. Ptosis, facial, pharyngeal, and neck flexor muscular weakness that extends to the arms with areflexia/hyporeflexia are the initial symptoms [9]. It must be there when there isn't noticeable limb weakness, ataxia, or altered consciousness. While they are much less noticeable, sensory weakness of upper and lower limbs may be present but is much less pronounced.

The clinical course of PCB should be acute and monophasic. Our patient's disease evolved rapidly over days and plateaued at two weeks. Her bulbar symptoms ultimately improved over 14 days of IVIG administration.

The two mainstays of GBS treatment, Intravenous Immunoglobulin (IVIg) and plasma exchange, have both been demonstrated to be equally successful [10] [11]. IVIg is beneficial for treatment when commenced two weeks after the onset of symptoms and plasmapheresis within four weeks [10]. Patients who have rapidly progressing weakness, autonomic dysfunction, bulbar failure, or respiratory insufficiency should be considered for treatment. During five days of IVIg therapy, our patient made a full recovery. Due to bulbar involvement, patients with PCB variant are more likely to require intubation. They also need continuous evaluation of their respiratory effort and bulbar function for guidance on the best way to use nasogastric feeding and ventilator support.

4. Conclusion

GBS is prone to misdiagnosis and may manifest atypically as a PCB variant. In any patient who presents with bulbar palsy, dysphagia, and symmetrical upper limb weakness, one should have a strong index of suspicion for PCB variants. Electrophysiological and CSF analysis are useful in diagnosis, but initial normal values do not rule out the disease. Clinical diagnosis is paramount.

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Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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