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An Unusual Presentation of Guillain -Barre Syndrome: A Case Study

Ananya Vasudhar*

S.S. Institute of Medical Sciences and Research Centre, Bengaluru, Karnataka, India

Abstract

Guillain-Barré syndrome (GBS) is an acute postinfectious polyneuropathy characterized by symmetric and ascending flaccid paralysis. GBS most commonly presents with symmetrical proximal muscle weakness of the lower extremities as the first sign and moves upwards as ascending paralysis. This case presented with asymmetrical descending paralysis and altered sensorium, which made the initial diagnosis quite challenging. CSF reports revealed an albuminocytologic dissociation that supported the diagnosis of post-infectious Guillain-Barré syndrome. Nerve conduction studies revealed an abnormal with increased F wave latency, suggestive of right upper limb sensory and lower limb motor axonal and demyelinating neuropathy. The patient was treated with IVIG and other supportive treatment. The patient showed significant improvement within two weeks.

Background: Guillain-Barré syndrome (GBS) can be described as a collection of clinical syndromes that manifests as an acute inflammatory polyradiculoneuropathy with resultant weakness and diminished reflexes. Rarely does the disease manifest with descending paralysis. We present a case of GBS that on the initial presentation had symptoms of altered sensorium and asymmetrical descending paralysis.

Objective: An unusual presentation

Conclusions: Guillain Barre Syndrome often presents with a wide range of symptoms, although it usually presents with symmetrical ascending paralysis, this case presented with asymmetrical descending paralysis and altered sensorium, which made the initial diagnosis quite challenging.

Keywords: Acute inflammatory demyelinating polyneuropathy; Acute motor axonal neuropathy; Acute paralytic neuropathy; Guillain–Barré syndrome; IVIG; Autoimmune

Introduction

A 56-year-old man who is a known case of hypertension and type 2 diabetes mellitus presented to the department of Internal medicine with complaints of fever, cough, and breathlessness for 1 week. The fever was an intermittent type, moderate grade, and was associated with chills and rigors. He also complained of cough for one week, which was insidious in onset, gradually progressive, associated with sputum production, which was scanty in quantity, whitish in color, and non-blood tinged. He additionally reported breathlessness during that period, which also was insidious in onset, and gradually progressive. Initially, it was grade 2 but progressed to grade 4 according to MMRC classification. On admission, the pulse rate was 80 bpm, bp 130/80, temperature afebrile (97.4 ^{0}F [36.3^{0}C]), he was tachypneic with a respiratory rate of 40 cpm, and saturation of 96% on room air. On auscultation, we found bilateral bronchial breath sounds with bilateral basal crepitations. A chest x-ray was done and revealed bilateral consolidation in the lungs. He was suspected to have COVID 19 infection and was managed in the COVID 19 isolation ward, a throat swab was sent for RT-PCR and was negative. All routine investigations were done, his complete blood panel values showed an elevated WBC count-15,900, hemo globin-14 g/dl, platelets- 2.78, ESR- 30, RBS- 395, Urea- 85, Creatinine- 0.9, sodium- 139 mmol/L, potassium- 5.6 mmol/L, chloride- 102 mmol/L, total bilirubin – 0.78, albumin- 3.64, globulin 3.18, SGOT- 37.97, SGPT- 31.31. The following day, he was drowsy, tachypneic with RR 36 and his saturation decreased to 90% on 10L of oxygen so, he was shifted to the ICU for further management. His ABG reports showed hypoxia - pH – 7.492, pCO2 40.1, pO2 55.5, sodium 124 mmol/L, potassium 2.3 mmol/L and chloride of 102 mmol/L and bicarbonate 30.6 mmol/L. At this time a diagnosis of bilateral pneumonia was considered and he was treated for the same with azithromycin, his GRBS was monitored every 8th hour, and was treated with insugen R accordingly. On day 5 of admission, he developed sudden onset of altered sensorium with pain and weakness initially in his right upper limb which then descended to his right lower limb. On examination, the power on the right upper and lower limb was 1/5, deep tendon reflexes were absent and plantar reflex on the right side was mute. The pain was prevalent in the upper limbs and was aching in nature, and increased on movement. Neurology opinion was taken for the same; he was then suspected to have AIDP. A lumbar puncture was done and CSF reports showed a cell count of 1 cell/cumm, protein 52.8mg/dl, glucose 99.0mg/dl chloride 131.9 mmol/L, suggestive of albuminocytologic dissociation which further supported the diagnosis of post-infectious Guillain-Barré syndrome [1]. Nerve conduction studies were done the next day, the results were abnormal with increased F wave latency, suggestive of right upper limb sensory and lower limb motor axonal and de-myelinating neuropathy. An MRI of the brain and spinal cord was done and was found to be normal. The weakness gradually spread to all the limbs and eventually leads to quadriparesis. All the sensory perception was intact. He was started on IVIG 2mg/kg, a total of 140gm were given [2, 3]. His power began to improve gradually over 5 days. He was also given an injection of vitamin B12.After completing his antibiotics and Iv Ig, he was maintained on Vitamin B12 injections, with considerable improvement in his initial neurological symptoms [4]. Two weeks following his diagnosis, his power improved to 5/5 and 4/5 in his upper and lower limbs respectively.

***Corresponding author:** Ananya Vasudhar, S. S. Institute of Medical Sciences and Research Centre, #15 Byrasandra Main road, Jayanagar 1st block east, Bengaluru, Karnataka, India, Tel: +91 9845022018, **Email:** ananyavasudhar@gmail.com

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	Clinical presentations	Nerve conduction studies	Cerebrospinal fluid (CSF)- Protein levels	Relevant investigations	Diagnosis
1.	Asymmetric weakness and sensory loss; pain	Multiple motor conduction blocks reduced sensory potentials	Normal	Serum cryoglobulin: positive, elevated ESR	Vasculitic neuropathy
2.	Asymmetrical onset, thickened nerves	Demyelinating sensorimotor polyneuropathy, conduction blocks	Elevated Protein	One copy (deletion) of the PMP 22 gene region on chromosome 17	Hereditary neuropathy with liability to pressure palsy (HNPP)
3.	No sensory symptoms, persistent weakness	Short (10 s) exercise test: significant increment in motor potentials	Normal	VGCC antibody-positive, Small cell carcinoma of the lung	Lambert–Eaton myasthenic syndrome (LEMS)
4.	Asymmetric weakness, altered sensorium, respiratory distress	Length dependent axonal sensorimotor polyneuropathy	Normal	Urine: porphobilinogen present	Porphyric neuropathy

Table 1: Differential diagnosis of Post infectious Guillain Barre Syndrome.

Discussion

Guillain- Barre syndrome (GBS) is an acute post-infectious polyneuropathy characterized by symmetric and ascending flaccid paralysis. GBS most commonly presents with symmetrical proximal muscle weakness of the lower extremities as the first sign and moves upwards as an ascending paralysis [5]. The weakness may progress over hours to days to involve the arms, truncal muscles, cranial nerves, and muscles of respiration [6]. In our patient we found the initial presentation to be a weakness of the upper limb which then descended to the lower limbs. The weakness was also asymmetrical in presentation and was more predominant on the right side. Most patients complain of paresthesia, numbness, or similar sensory changes. The diagnosis of GBS poses a challenge for health care providers' especially because the neurological symptoms are so diverse. It has been difficult to develop a definitive criterion for the diagnosis of GBS largely because considerable variations have existed between presentations among different patients. Also, investigative tests including MRI lack specificity of findings. Aside from varied presentations, the ability of GBS to masquerade as a different pathology further contributes to its diagnostic difficulty. The exact cause of GBS is unknown, but 50- 70% of cases appear 1 to 3 weeks after respiratory or gastrointestinal infection or another immune stimulus that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots [7]. In affected patients, cross-reactive auto-antibodies attack the host's axonal antigens, resulting in inflammatory and de-myelinating polyneuropathy [8]. It most commonly manifests as a rapidly evolving are flexic motor paralysis with or without sensory disturbance. Several lines of evidence support an autoimmune basis for AIDP; the most common and best-studied type of GBS, the concept extends to all subtypes of GBS [9]. Both cellular and humoral immune mechanisms likely contribute to tissue damage. Circumstantial evidence suggests that all GBS results from immune response to non-self antigen (infectious agents, vaccine) that misdirect to host nerve tissue through a resemblance of epitope mechanism [10]. The prognosis is usually good with early detection and prompt treatment.

Differential Diagnosis

Guillain Barre Syndrome (GBS) is usually characterized by symmetric and ascending flaccid paralysis. The conditions discussed in Table 1 are conditions that commonly present as ascending weakness and were considered as a differential diagnosis before the CSF analysis supported GBS [11,12].

Conclusion

Guillain Barre Syndrome often presents with a wide range of symptoms, although it usually presents with symmetrical ascending paralysis, our case presented with asymmetrical descending paralysis and altered sensorium, which made the initial diagnosis quite challenging.

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