Journal of Investigative Medicine High Impact Case Reports
Volume 8: 1–4

DOI: 10.1177/2324709620931649
journals.sagepub.com/home/hic
SAGE

Ascending Paralysis in a 36-Year-Old Woman With Bipolar Disorder and Recent Aspiration Pneumonia

Aditi Vian Varma-Doyle, MD[1,2,*], Kristen Garvie, MD[2,*], Seema Walvekar, MD[1,2], Mae Igi, MD[2], and Radha Mayuri Garikepati, MD[1]

Abstract

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating polyneuropathy affecting both motor and sensory peripheral nerves. Typically presenting after a gastrointestinal or a respiratory tract infection, it manifests as ascending paralysis with concomitant areflexia in patients. Cytoalbuminologic dissociation is a supportive finding on cerebrospinal fluid (CSF) analysis. Due to variability in presentation, misdiagnosis and delay in treatment can occur, and consequently, GBS can become life threatening due to respiratory failure. We report ascending paralysis in a 36-year-old woman with known history of bipolar disorder who recently recovered from aspiration pneumonia following a drug overdose event. Given her psychiatric history, she was initially misdiagnosed as conversion disorder. Intravenous immunoglobulin (IVIG) therapy was initiated at our hospital due to strong suspicion of GBS, based on history and physical examination findings consistent with flaccid quadriparesis and impending respiratory failure. CSF analysis and radiological findings subsequently supported our clinical suspicion and clinical findings. Concurrent IVIG therapy, pain management, aggressive physical and respiratory therapy, and monitoring resulted in symptom improvement. One must have a high index of suspicion for GBS when presented with acute inflammatory demyelinating neuropathies in patients who present with ascending paralysis. Early initiation of therapy is key and can prevent life-threatening complications.

Keywords

Guillain-Barré syndrome, aspiration pneumonia, bipolar disorder, immunoglobulin therapy, early CSF analysis

Case Presentation

A 36-year-old woman with history of bipolar disorder was found unconscious surrounded by multiple mood-stabilizing medications. She was tachycardic, febrile up to 107 °F, with Glasgow Coma Scale score of 3. She was intubated and admitted to medical intensive care unit for further management and cooling protocol. Computed tomography head stat was negative for any acute intracranial event. Arterial blood gas showed respiratory acidosis, and a chest X-ray showed bilateral chest infiltrates. She was resuscitated with ringers' lactate and started on intravenous (IV) antibiotics for possible aspiration pneumonia. After fluid resuscitation and continued IV antibiotic therapy, patient was extubated on the fourth day after admission with recovery of mental status. Examination post intubation was only remarkable for scattered rales at bibasilar lung fields on auscultation. Blood cultures showed no growth, and respiratory cultures showed few gram-positive cocci in clusters and gram-negative rods.

The following week, she presented with insidious onset of bilateral upper and lower extremity weakness. Patient presented initially with falls that progressed to an inability to walk. She also complained of nonradiating lower back pain and requested for analgesics. On examination, she had decreased strength in her lower extremities that were thought to be due to lack of effort. Patient had reflexes in her lower extremities though they were diminished.

Differential diagnoses for patient's presentation initially was a possible conversion disorder. She was transferred to our tertiary care facility for further neurological evaluation.

[1]Louisiana State University Health Sciences Center, New Orleans, LA, USA
[2]Louisiana State University, New Orleans, LA, USA

*Aditi Vian Varma-Doyle and Kristen Garvie are the first authors of this article.

Received February 17, 2020. Revised April 17, 2020. Accepted April 18, 2020.

Corresponding Author:
Seema Walvekar, Section of Hospital Medicine, Louisiana State University, 1542 Tulane Avenue, Box T4 M-2, New Orleans, LA 70112, USA.
Email: swalve@lsuhsc.edu

On transfer to our facility, patient's vitals were stable. On neurological examination, she was alert, oriented ×3 with intact cranial and sensory nervous system examination. Motor examination was significant for weakness with strength of 2/5 bilaterally in all muscle groups of lower extremities. In her upper extremities, finger flexion was minimal at ⅗, with variable strength in her wrist flexion and extension, but declining strength in ascending pattern. Her extremities were hypotonic, with preserved muscle bulk. Areflexia was noted in bilateral lower extremities (Achilles and patellar). Brachioradialis, biceps, and triceps reflexes were 1+ and equal bilaterally. Plantar flexor response was noted bilaterally. Rectal tone was intact. Urinary and bowel incontinence, saddle anesthesia, was not reported.

Respiratory examination revealed no labored breathing but decreased inspiratory effort. Bedside monitoring revealed negative inspiratory force (NIF) of −40 cm of water and forced vital capacity (FVC) of 2.1 L. Lungs were clear to auscultation bilaterally except for some scattered crackles.

Laboratory examination revealed normal white blood cell count, with mildly normocytic anemia (9 g/dL) and thrombocytopenia (125 000/μL). Comprehensive metabolic profile was within normal limits, and urinalysis was unremarkable.

Given the concern of impending respiratory failure, IV immunoglobulin (IVIG) therapy at 0.4 g/kg/day was initiated before other supportive investigations were available.

Cerebrospinal fluid (CSF) analysis subsequently revealed a clear/colorless fluid with elevated protein at 327.2 mg/dL and an elevated CSF white blood cell count of 26 cells/μL (lymphocytes 93%, monocytes 7%). CSF glucose at 68 mg/dL was normal, and CSF red blood cell count of 7 cells/μL and no oligoclonal bands in the CSF. Guillain-Barré syndrome (GBS) diagnosis was supported by CSF analysis with findings consistent for cytoalbuminologic dissociation.

Imaging of her brain was normal. Magnetic resonance imaging of her spine revealed diffuse area of increased enhancement of cauda equina in lumbar spine (Figures 1 and 2). These radiological findings were consistent with acute inflammatory demyelinating polyradiculoneuropathy (AIDP).

Patient received 5 days of IVIG therapy, and a repeat IVIG course the following week. Supportive therapy included pain control, physical, and occupational therapies. Her NIF and FVC scores improved shortly (day 2) after IVIG treatment, and she started showing improvement of her upper extremity weakness, left greater than right. Lower extremity strength did not improve much and stayed at 2/5.

She has oscillations between constipation and diarrhea, but otherwise had no symptoms/signs consistent with dysautonomia and no labilities in cardiovascular function.

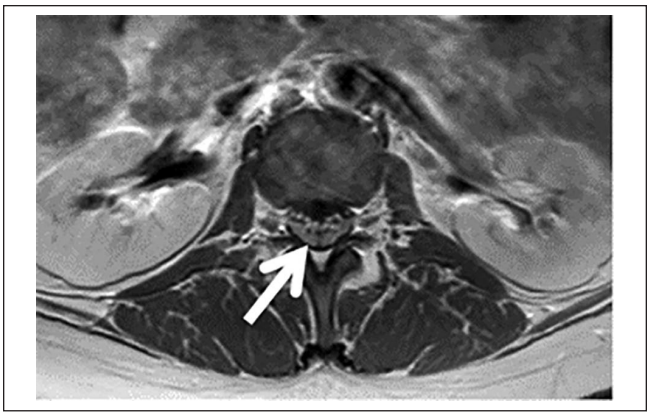

Figure 1. Transverse view at the L3-L4 spinal level. Arrow points to the increased enhancement and abnormally clumped nerve roots of the cauda equina consistent with neural inflammation associated with Guillain-Barré syndrome/acute inflammatory demyelinating polyradiculoneuropathy.

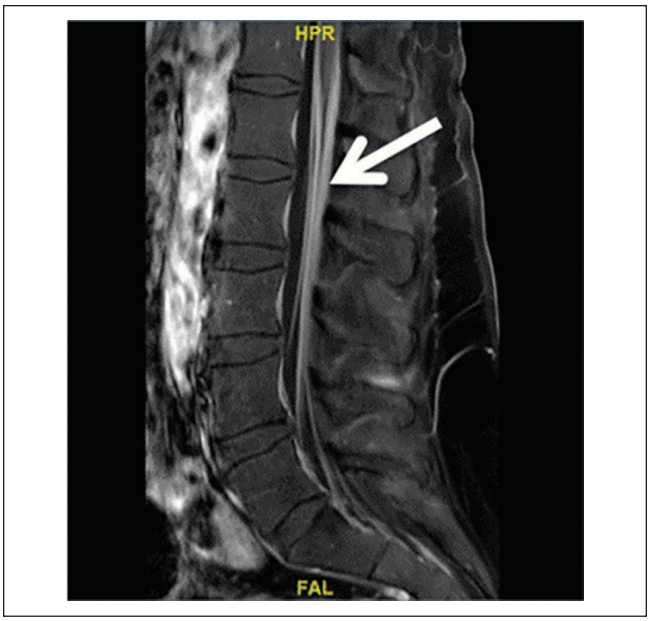

Figure 2. Sagittal view of lumbar spine. Arrow points to cauda equina with diffuse enhancement consistent with neural inflammation associated with Guillain-Barré syndrome and acute inflammatory demyelinating polyradiculoneuropathy.

Discussion

Guillain-Barré syndrome is the most common cause of ascending neuromuscular flaccid paralysis in the developed world,[1,2] with annual incidence of approximately 1 to 2 per 100 000 persons.[3] It is also a frequently misdiagnosed neurological disorder.[4,5]

Being an autoimmune disorder, it has subtypes depending on the type of peripheral nerves involved. AIDP, acute motor and sensory axonal neuropathy, acute motor axonal neuropathy, and the Fisher syndrome variant (ophthalmoplegia, ataxia, and areflexia) are described subtypes of GBS. Our case presented as AIDP subtype, where the immune cells attack the epitopes in Schwann cells resulting in demyelination. AIDP is the most common form of GBS in Europe and North America.[2,3]

The most common antecedent event is an upper respiratory tract or gastrointestinal infection. Gastrointestinal tract infections with *Campylobacter jejuni* are cited as the most frequent causative agent.[4] Respiratory pathogens are typically viruses, such as CMV and Epstein-Barr, and can include bacterial microorganisms including *Mycoplasma pneumoniae* and *Haemophilus influenzae*.

The preceding illness in our case was most likely an aspiration pneumonia following a drug overdose.

Because of her psychiatric comorbidity and recent attempt of self-harm, she was initially misdiagnosed as conversion disorder. This is a common misdiagnosis.[5,6] Given the high incidence of GBS in the developed world, there should be a low threshold for suspecting GBS during diagnostic workup, and a complete neurological workup should be considered in all cases.

Our patient presented with level 2 of diagnostic certainty of Brighton criteria.[7] She had bilateral flaccid paralysis of limbs *and* decreased deep tendon reflexes in upper extremities with absent reflexes in lower extremities. Onset of symptoms was within 28 days of antecedent illness and >12 hours of aspiration pneumonia event. Her CSF white cell count was <50 cells/μL with protein elevation.

Clinical presentation in GBS involves ascending motor paralysis with occasional sensory loss. Symptoms are typically associated with pain in more than 50% of patients. This is important to recognize; patients could be misunderstood as drug seeking and consequently be misdiagnosed, especially, if such a patient has a mood/psychiatric disorder, as seen in our patient. We recommend addressing pain control in GBS patients as this can help them recover and improve their participation in multidisciplinary management of the disease such as participation in Physical, Occupation, and Respiratory Therapy.

Autonomic symptoms such as rhythm abnormalities, blood pressure instability, constipation, and urinary retention can also be seen in more than two thirds of patients and be the initial presentation of the disease.[8] As the disease advances, respiratory muscle paralysis can occur, leading to death. Sometimes paralysis can begin with bulbar symptoms noted frequently as difficulty with tongue protrusion, which in often cases can predict worse respiratory outcome.[1] Tongue weakness may have been initial presentation in our patient leading her to aspirate pills; however, information regarding this event is limited.

Psychiatric manifestations have also been noted in the disease, with visual hallucinations, paranoid delusions, and sleep disorders noted. These were documented in our patient with known mood disorder but can be seen in patients without any psychiatric history.[9]

Laboratory investigations in our patient were relevant for normocytic anemia and thrombocytopenia. Immune thrombocytopenic purpura has been reported in association with GBS.[10] Both these disorders are due to an autoimmune response triggered by molecular mimicry or cross antigenicity of an antecedent infection event.

Guillain-Barré syndrome is a clinical diagnosis. Diagnosis supported by CSF findings of cytoalbuminologic dissociation. In our patient, initiation of treatment was started prior to CSF analysis. This early initiation of therapy, we believe prevented her from developing respiratory compromise. In early GBS, CSF analysis can be normal.[11] Hence, management should be initiated with low threshold for suspicion when diagnosing disease, without relying on CSF analysis.

In GBS, imaging of spine shows demyelinating changes in the form of enhancement mostly found in conus medullaris and cauda equina,[12] a finding that was noted in our patient. Sole enhancement of the anterior roots is strong indicator of GBS.[12]

Intravenous immunoglobulin therapy was initiated in our patient and helped with symptom improvement. It was administered at a dose of 0.4 g/kg/day and repeated after 1 week. IVIG has been found to be more beneficial than plasmapheresis and is also associated with decreased stay in the intensive care unit, earlier weaning from mechanical ventilation and earlier recovery for patients.[13] A recent trial, however, has demonstrated no additional efficacy in a second course of IVIG in treatment of GBS.[14]

In conclusion, clinicians should maintain a high index of suspicion for GBS if a patient presents with progressive ascending paralysis. Since early in the course of the disease, CSF analysis can be normal, history and clinical presentation should be adequate, and there should be a low threshold for early initiation of IVIG therapy that could prevent respiratory failure. In addition, patients who may present with pain should be evaluated without bias of their past medical or psychiatric history and initiated on pain management therapy to help with symptom control and patient participation in supportive therapies.

Authors' Note

This case was presented as an abstract at the Southern Regional Meeting; American Federation of Medical Research Conference; New Orleans, LA; February 21, 2019.

Acknowledgments

Samuel Clarot, DDS, Louisiana State University School of Medicine, New Orleans, and Louisiana State University Oral Maxillofacial Surgery.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethics Approval

Our institution does not require ethical approval for reporting individual cases.

Informed Consent

Written informed consent was obtained from the patient for their anonymized information to be published in this article.

ORCID iD

Aditi Vian Varma-Doyle https://orcid.org/0000-0002-9699-5668

References

1. Nayak R. Practical approach to the patient with acute neuromuscular weakness. *World J Clin Cases*. 2017;5:270-279.
2. McGrogan A, Madle GC, Seaman HE, de Vries CS. The epidemiology of Guillain-Barré syndrome worldwide. A systematic literature review. *Neuroepidemiology*. 2009;32:150-163.
3. Sejvar JJ, Baughman AL, Wise M, Morgan OW. Population incidence of Guillain-Barré syndrome: a systematic review and meta-analysis. *Neuroepidemiology*. 2011;36:123-133.
4. Kuwabara S. Guillain-Barré syndrome: epidemiology, pathophysiology and management. *Drugs*. 2004;64:597-610.
5. Pope JV, Edlow JA. Avoiding misdiagnosis in patients with neurological emergencies. *Emerg Med Int*. 2012;2012:949275.
6. Edelsohn G. Guillain-Barré misdiagnosed as conversion disorder. *Hosp Community Psychiatry*. 1982;33:766-767.
7. Fokke C, Van den Berg B, Drenthen J, Walgaard C, van Doorn PA, Jacobs BC. Diagnosis of Guillain-Barré syndrome and validation of Brighton criteria. *Brain*. 2014;137(pt 1):33-43.
8. Ferraro-Herrera AS, Kern HB, Nagler W. Autonomic dysfunction as the presenting feature of Guillain-Barré syndrome. *Arch Phys Med Rehabil*. 1997;78:777-779.
9. Sangroula D, Durrance R, Bhattarai S, Nandakumar T. Neuropsychiatric debut as a presentation of Guillain-Barré syndrome: an atypical clinical case and literature review. *J Clin Neurosci*. 2017;44:245-249.
10. Kohli RS, Bleibel W, Bleibel H. Concurrent immune thrombocytopenic purpura and Guillain-Barre syndrome in a patient with Hashimoto's thyroiditis. *Am J Hematol*. 2007;82:307-308.
11. DiCapua DB, Lakraj AA, Nowak RJ, Robeson K, Goldstein J, Patwa H. Relationship between cerebrospinal fluid protein levels and electrophysiologic abnormalities in Guillain-Barré syndrome. *J Clin Neuromuscul Dis*. 2015;17:47-51.
12. Byun WM, Park WK, Park BH, Ahn SH, Hwang MS, Chang JC. Guillain-Barré syndrome: MR imaging findings of the spine in eight patients. *Radiology*. 1998;208:137-141.
13. Van der Meché FG, Schmitz PI. A randomized trial comparing intravenous immune globulin and plasma exchange in Guillain-Barré syndrome. Dutch Guillain-Barré Study Group. *N Engl J Med*. 1992;326:1123-1129.
14. Walgaard C, Jacobs BC, Lingsma HF, Steyerberg EW, Cornblath DR, van Doorn PA; Dutch GBS Study Group. Second IVIg course in Guillain-Barré syndrome patients with poor prognosis (SID-GBS trial): protocol for a double-blind randomized, placebo-controlled clinical trial. *J Peripher Nerv Syst*. 2018;23:210-215.