CASE SERIES PEER REVIEWED | OPEN ACCESS

Guillain–Barré syndrome post stroke: Two case reports

Tariku Assefa, Wonwossen Tekle, Eskedar Kebede, Fikru Tsehayeneh, Ebenezer Tirsit, Bereket Ethiopia, Yonatan Wudeneh

ABSTRACT

Guillain–Barré syndrome (GBS) is the most common and most severe acute paralytic neuropathy. There are different variants of GBS with distinct clinical and pathological features. Most GBS cases are preceded by infection or other immune stimulation that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots. Molecular mimicry between microbial and nerve antigens is clearly a major driving force behind the development of the disorder, at least in the case of *Campylobacter jejuni* infection. Some patients have been developing GBS either following or concomitant with head trauma, neurosurgical procedures, and rarely following stroke. The exact mechanism is not well understood probably blood–brain barrier damage may play an essential role in triggering the autoimmune activation that leads to post-stroke GBS.

Keywords: Blood–brain barrier, *Campylobacter jejuni*, Guillain–Barré syndrome, Post-stroke GBS

How to cite this article

Assefa T, Tekle W, Kebede E, Tsehayeneh F, Tirsit E, Ethiopia B, Wudeneh Y. Guillain–Barré syndrome post stroke: Two case reports. Int J Case Rep Images 2026;17(1):7–11.

Tariku Assefa[1], Wonwossen Tekle[1], Eskedar Kebede[2], Fikru Tsehayeneh[3], Ebenezer Tirsit[3], Bereket Ethiopia[4], Yonatan Wudeneh[4]

Affiliations: [1]Interventional Neurology and Neuro Critical Care, Axon Stroke and Spine Center, Addis Ababa, Ethiopia; [2]Internal Medicine, Axon Stroke and Spine Center, Addis Ababa, Ethiopia; [3]Neurology and Neurosurgery, Axon Stroke and Spine Center, Addis Ababa, Ethiopia; [4]General practitioner, Axon Stroke and Spine Center, Addis Ababa, Ethiopia.

Corresponding Author: Tariku Assefa Soboka, Interventional Neurology and Neuro Critical Care, Axon Stroke and Spine Center, Addis Ababa, Ethiopia; Email: trkssf4@gmail.com

Received: 26 May 2025
Accepted: 18 September 2025
Published: 31 January 2026

Article ID: 101529Z01TA2026

****

doi: 10.5348/101529Z01TA2026CS

INTRODUCTION

Guillain–Barré syndrome (GBS) is the most common acute inflammatory demyelinating polyneuropathy, with many people developing the disease every year worldwide. Guillain–Barré syndrome is a spectrum disease with different variants with different clinical and pathological features. There are four identifiable forms of GBS which are the acute inflammatory demyelinating polyneuropathy (AIDP), acute motor axonal polyneuropathy (AMAN), acute motor sensory axonal polyneuropathy (AMSAN), and the severe form Miller–Fisher type.

One of the feared complications of GBS is the involvement of bulbar and respiratory muscles resulting in respiratory failure which accounts about 20–30% of GBS cases.

Usually, GBS is preceded by infection mainly gastrointestinal origin or other immune stimulation that induces an autoantibody response targeting peripheral nerves and their spinal roots. The molecular resemblance between microbial and nerve antigens is one of a major factors causingthe development of this disease, like in the case of *Campylobacter jejuni* infection [1].

It is usually an ascending, bilateral, and areflexic condition of the limbs, often with sensory and cranial nerve involvement 1–2 weeks after immune stimulation, and reaching a clinical plateau condition in 2–4 weeks. When patients present with rapidly progressive and ascending paralysis, the diagnosis of Guillain–Barré syndrome needs to be made as soon as possible. Although the diagnosis of typical cases is usually clear, it is better to do clinical judgement and relevant investigations to diagnose the atypical GBS cases [1, 2].

Treatment with intravenous immunoglobulin or plasmapheresis will be the standard of care to hinder the progression of the disease with all supportive medical care [3].

CASE REPORT

Case 1

A 65-year-old male presented with two days history of sudden loss of balance which occurred while he was walking. He was immediately taken to a nearby hospital, and he was told that his blood pressure (BP) was 200 mmHg of systolic BP, but no other problems were identified, and he was sent home with antihypertensive medication.

He then developed double vision, vomiting of ingested matter, and generalized weakness in the next two days which made him come to our center. He was having dry cough, fatigue, and appetite loss for a week.

At our ER, he had high BP and tachypnea. Saturation was normal.

His neurologic exam showed right hemianopia and he has generalized weakness can move all of his limbs.

Few hours after admission, his tachypnea worsened, and his breathing became shallow, so he was taken to the intensive care unit (ICU) to be intubated. Non-contrast brain computed tomography (CT) was normal.

He was put on double antiplatelet therapy, Aspirin 300 mg and Clopidogrel 75 mg, Atrovastatin, mechanical ventilation. Brain MRI which was taken during his stay in ICU showed no acute insults (Figure 1). On the 3rd day of admission during examination, he failed to move all extremities, and all limbs were flaccid and areflexic. Pain and touch sensation were also absent, yet he was alert and could fully comprehend.

The diagnosis of GBS was considered and nerve conduction study (NCS) was done which revealed drop in amplitudes left side ulnar, bilateral peroneal nerve responses with diffusely prolonged late response showing mild axonal motor polyradiculoneuropathy which suggests early AMAN type of Guillain–Barré syndrome.

After the confirmation of the diagnosis, he was started intravenous immunoglobulin (IVIG) and tracheostomy was done. He took 5 doses of IVIG and he started to show improvement with flickering movements of the upper extremity after the 2nd dose. His started movement of lower extremities after about two weeks. He continued to show fast recovery and by fourth week he started to speak and had lower extremities had power of 2+, and he was discharged to continue his rehabilitation at home. When he came for follow-up after a month, his tracheostomy was closed and he could move all 4 limbs with power of 4/5 upon follow-up clinic.

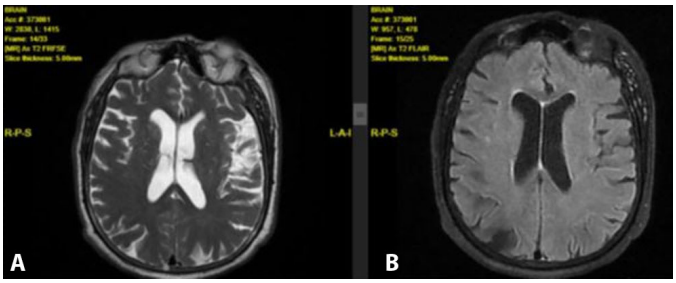

Figure 1: For case 1–(A) Shows multiple small periventricular and subcortical and deep white matter T2 and fluid-attenuated inversion recovery (FLAIR) hyperintensities with no mass effect—likely chronic small ischemia. (B) Right posterior cerebral artery partial occlusion at distal P1 with patent ante grade flow through it. Right vertebral artery is not visualized and presumably occluded.

Case 2

A 65-year-old female patient who was a known hypertensive and Type 2 Diabetes Mellitus but not on medication referred to our center with sudden loss of consciousness of 2 hours duration. Up on arrival to ER, her Glasgow coma scale (GCS) was E2V1M5. Pupils were constricted and sluggish. No other pertinent positive findings in the other systems. She was loaded with mannitol from the center where she was referred from.

She was transferred immediately for non-contrast brain CT which has shown acute cerebellar bleed with intraventricular and subarachnoid extension for which immediate posterior fossa decompressive craniotomy and external ventricular drain (EVD) insertion was done and transferred to ICU. External ventricular drain was fixed at the height of 15 cm and fentanyl sedation was started. Stable vital signs and sedation was stopped on the next day morning. The patient was awake with GCS of E4VTM6. She was moving all her extremities. She was also on minimal ventilator support. She was weaned and extubated. She was stable and breathing comfortably. On the next day the patient started to have difficulty of breathing and desaturating for which she was re intubated.

Then onward, her GCS has dropped to E2VTM1. On motor examination, the power was zero for all extremities and also areflexic. No spontaneous breathing on mechanical ventilator. Arterial blood gas (ABG) of the patient has shown acute respiratory acidosis. Other blood tests including blood and urine culture did not reveal systemic infections. Lumbar puncture has shown albumin cytological dissociation. Brain magnetic resonance imaging (MRI) done after reintubation has

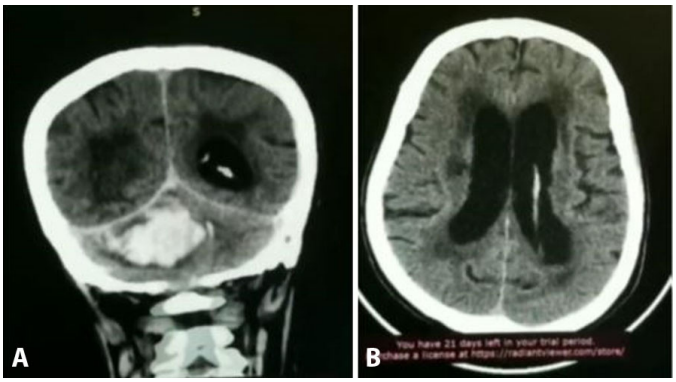

Figure 2: For case 2–(A) Shows acute cerebellar bleed. (B) Left intraventricular bleed.

revealed diffuse white–gray matter hyperintense lesions, suggestive of encephalopathy (Figure 2). Steroid was tried but no response. Tracheostomy was done on 12th day after admission.

Two weeks later nerve conduction study was done. There were drop in amplitudes left side ulnar, bilateral peroneal nerve responses with diffusely prolonged late response showing mild axonal motor polyradiculoneuropathy, which suggests early AMAN type of Guillain–Barré syndrome. Considering the clinical course, the patient was started on plasmapheresis every other day for five sessions. She was improved, weaned from mechanical ventilator and sent home continuing physiotherapy with home care.

DISCUSSION

Guillain–Barré syndrome was first reported by Guillain, Barré, and Strohl in 1916. It has several subtypes including acute inflammatory demyelinating polyradiculoneuropathy (AIDP), acute motor axonal neuropathy (AMAN), acute motor-sensory axonal neuropathy (AMSAN), and Miller–Fisher syndrome (MFS). From the different forms of GBS, AIDP is the most common type with better outcome while AMAN usually affects purely motor nerve damage, especially motor axons with worse outcome. Acute motor sensory axonal polyneuropathy type affects both sensory and motor axonal damage. The most severe form of GBS, MFS usually presents with ophthalmoplegia, ataxia, and areflexia. The pathophysiology has been analyzed for more than a century but the exact mechanism is not yet settled. The disease condition can be preceded by a number of microorganisms' infections, mainly *Campylobacter jejuni* and others like cytomegalovirus, leptospirosis, Epstein–Barr virus, Hepatitis E virus, and Zika virus [3, 4]

We reported two patients with severe GBS. One patient developed it following acute cerebellar bleed and the second one following acute ischemic stroke. Both patients developed acute generalized weakness and areflexia associated with respiratory failure during their recovery time following stroke. The cerebrospinal fluid (CSF) analysis of both patients have showed albumin cytological dissociation, supportive of polyradiculopathy. The brain MRI of both cases showed no evidence of abnormality in the brain stem.

The NCS of first patient has shown drop in amplitudes left side ulnar, bilateral peroneal nerve responses with diffusely prolonged late response showing mild axonal motor polyradiculoneuropathy which suggests early AMAN type of Guillain–Barré syndrome.

For the second patient, NCS has showed absent compound muscle action potentials, sensory nerve action potentials, and F wave latencies, indicating motor and sensory axonal injury, which are common electrophysiological features of AMSAN.

There are cases GBS following head trauma, neurosurgery, or other cerebral hemorrhagic injury. The possible mechanisms of GBS after cerebral hemorrhagic injury are the acute inflammation triggered by hemoglobin infiltration, disturbance of cellular humoral immunity after brain injury, and the stress state after hemorrhage has described that intraventricular hemorrhage can cause a Toll-like receptor 4 (TLR4)- and NF-κB-dependent inflammatory response. In addition, it has been reported that the levels of TLR4 and NF-κB are significantly increased in the CSF of GBS patients. This will promote the secretion of inflammatory molecules, such as TNF-α, IL-6, IL-8, IL-12, IL-23, and IL-1β. These will inhibit Schwann cell proliferation and potentiate Schwann cell apoptosis, increase the antibody affinity to self-ganglioside, induce demyelination, nerve lesions, and axonal degeneration resulting in the development of GBS. One of our patients has developed GBS after acute cerebellar hemorrhage with intraventricular and subarachnoid extension who might have acquired GBS with the above mechanism.

There is a limited number of cases of GBS following ischemic stroke like one of our patients who had acute posterior cerebral artery (PCA) infarction. The mechanism is not well understood. Probably due to the disruption of the blood–brain barrier leading to the release of immunologic components of neuronal debris (including myelin-associated proteins) into the blood, inducing the production of anti-myelin antibodies and subsequent neuropathy [5].

In typical Guillain–Barré syndrome, rapidly progressive bilateral weakness is usually ascending type and starts in the lower extremities, but it can also start more proximally in the legs or arms. The variant which involves the proximal nerves can result in the false clinical impression and can lead to misdiagnosis. So that supportive tests like nerve conduction studies, electromyography and brain imaging may be needed to diagnose earlier and start management on time. A small number of patients present with paraparesis, which can remain during the course of the disease. Others might present with cranial nerve involvement resulting in facial, oculomotor, or bulbar weakness. Patients can have atypical clinical presentations like our cases and we need to have high index of suspicion of GBS for those patients in the post stroke phase and have paralytic and areflexic presentations not explained by stroke.

Several randomized controlled trials (RCTs) studies have shown the effect of IVIG and plasma exchange as effective therapy. However, most of these studies were done in Europe and North America where most patients have the acute inflammatory demyelinating polyneuropathy (AIDP) variant of the disorder. If IVIG or plasma exchange will be started, they should be started as early as possible, before irreversible nerve damage has taken place [6–8]. Our first case was treated with plasmapheresis every other day (1.2–3 L) five sessions and she has improved while the second case was receiving

IVIG, five doses and improved well. For both cases, treatment was started within 24 hours making a diagnosis. As these patients may have atypical presentation, it is clear that diagnosis might made later, after irreversible damage has happened to the nerve [9, 10].

LIMITATIONS

The electromyogram (EMG) and electroencephalogram (EEG) examination were not done because of absence and logistic difficulties at that time. This may play a role for diagnostic certainty.

CONCLUSION

These two cases open our eyes studies in considering GBS as the differential diagnosis for patients developing acute flaccid paralysis and respiratory failure following stroke. Patients may have atypical presentations but better to have high index of GBS suspicion as early diagnosis with initiation of therapy earlier will make a difference in the prognosis and outcome of patient.

REFERENCES

1. Ebrahim Soltani Z, Rahmani F, Rezaei N. Autoimmunity and cytokines in Guillain-Barré syndrome revisited: Review of pathomechanisms with an eye on therapeutic options. Eur Cytokine Netw 2019;30(1):1–14.
2. Huizinga R, van den Berg B, van Rijs W, Tio-Gillen AP, Fokkink WJR, Bakker-Jonges LE, et al. Innate immunity to Campylobacter jejuni in Guillain-Barré syndrome. Ann Neurol 2015;78(3):343–54.
3. Willison HJ, Jacobs BC, van Doorn PA. Guillain-Barré syndrome. Lancet 2016;388(10045):717–27.
4. Chevret S, Hughes RA, Annane D. Plasma exchange for Guillain-Barré syndrome. Cochrane Database Syst Rev 2017;2(2):CD001798.
5. Wu Q, Liu N, Pan C, Bu B, Tang Z. Guillain-Barré syndrome and cerebral hemorrhage: Two cases and literature review. Eur Neurol 2016;76(3–4):182–6.
6. Hughes RA, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain-Barré syndrome. Cochrane Database Syst Rev 2014; 2014(9):CD002063.
7. Hughes RAC, Swan AV, Raphaël JC, Annane D, van Koningsveld R, van Doorn PA. Immunotherapy for Guillain-Barré syndrome: A systematic review. Brain 2007;130(Pt 9): 2245–57.
8. Overell JR, Hsieh ST, Odaka M, Yuki N, Willison HJ. Treatment for Fisher syndrome, Bickerstaff's brainstem encephalitis and related disorders. Cochrane Database Syst Rev 2007;2007(1):CD004761.
9. Hiraga A, Mori M, Ogawara K, Kojima S, Kanesaka T, Misawa S, et al. Recovery patterns and long term prognosis for axonal Guillain-Barré syndrome. J Neurol Neurosurg Psychiatry 2005;76(5):719–22.
10. Liu DY, Hollenbach JR, Gregorin JA, Wynbrandt JH. A case of acute motor sensory axonal neuropathy: A variant of guillain-barré syndrome, with possible syndrome of irreversible lithium-effectuated neurotoxicity. Case Rep Med 2020;2020:4683507.

****

Author Contributions

Tariku Assefa – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Wonwossen Tekle – Design of the work, Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Eskedar Kebede – Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Fikru Tsehayeneh – Acquisition of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Ebenezer Tirsit – Acquisition of data, Analysis of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Bereket Ethiopia – Acquisition of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Yonatan Wudeneh – Acquisition of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission
The corresponding author is the guarantor of submission.

Source of Support
None.

Consent Statement
Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest
Authors declare no conflict of interest.

Data Availability
All relevant data are within the paper and its Supporting Information files.