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**CASE REPORT** **OPEN ACCESS**

# GUILLAIN-BARRE SYNDROME ASSOCIATED WITH DISSEMINATED TUBERCULOSIS: A CASE REPORT

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**ABSTRACT**

**Background:** Guillain-Barre syndrome (GBS) is a life-threatening, acute, immune-mediated polyneuropathy associated with preceding infections. Tuberculosis (TB), although it has a high incidence rate, is rarely reported to be associated with GBS.
**Case**: We report the case of a 20-year-old female admitted to our hospital with a progressive symmetrical paraparesis, which further developed to paraplegia, along with a month-long history of constitutional symptoms. After investigation, the patient was diagnosed with an acute motor and sensory axonal neuropathy (AMSAN) variant of GBS and disseminated TB. The patient was treated with plasmapheresis, and first-line anti-tuberculosis therapy was initiated. The patient demonstrated significant improvement in muscle strength in response to the treatment. The pathogenesis of GBS in TB is believed to be due to molecular mimicry, leading to nerve damage or direct invasion of the nerve root by tubercular bacilli.
**Discussion**: Guillain-Barre syndrome could be induced by tuberculosis, and treatment for both could improve the outcome. Thus, early diagnosis is critical. Further investigations must be conducted to understand the association of GBS and TB.
**Conclusion**: Guillain-Barre syndrome could be induced by tuberculosis, and treatment for both could improve the outcome. Thus, early diagnosis is critical. Further investigations must be conducted to understand the association of GBS and TB.

**Keywords**: AMSAN, Guillain-Barre syndrome, polyneuropathy, tuberculosis



## Introduction

Guillain-Barre Syndrome (GBS) is an acute polyradiculopathy. In the present systematic review, the incidence rate in the general population ranged from 0.30 cases to 6.08 per 100.000 inhabitants.[1] GBS is usually a monophasic disease in which an aberrant immune response to an infection or other trigger damages the peripheral nerves.[2,3,4] The sensorimotor variant and the demyelinating electrophysiologic subtype were most frequent across all infection groups.[5] The syndrome onset is usually triggered by a history of previous infection in 70% of overall cases, with Campylobacter jejuni as the most associated infection, followed by Cytomegalovirus, Epstein-Barr virus, Mycoplasma pneumoniae, and arbovirus infections (dengue virus, Zika virus, West Nile virus, and Chikungunya virus).[1] Recently, there have been reports about tuberculosis preceding the onset of GBS. While tuberculosis has a high incidence rate and carries a high burden in Indonesia, the case that mentions the association of tuberculosis with GBS in Indonesia is underreported. Diagnosis of sensory-motor or motor GBS is progressive weakness of arms and legs, absent or decreased deep tendon reflexes in affected limbs, and progressive worsening for no more than 4 weeks . 7 Laboratory findings include Cerebrospinal Fluid (CSF) analysis or antibodies, and electrodiagnostic studies are considered helpful to support diagnosis.[3] Effective treatments include plasma exchange and intravenous immunoglobulins. However, ~20% of patients who receive treatment are unable to walk after 6 months, and ~5% die as a consequence of GBS.[2]

Tuberculosis (TB), on the other hand, can present with a broad spectrum of neurological manifestations. These include tuberculous meningitis, radiculomyelitis,

intracranial tuberculomas, brain abscesses, and various combinations of these conditions. In patients presenting with constitutional symptoms such as fever, night sweats, or weight loss, or in those with an established diagnosis of TB, the emergence of neurological symptoms is often readily attributed to direct central nervous system (CNS) involvement by *Mycobacterium tuberculosis*. This may lead clinicians to overlook alternative diagnoses such as Guillain-Barré Syndrome (GBS), especially given the overlapping early neurological presentations. Consequently, GBS may be underrecognized in TB patients, delaying appropriate treatment and increasing the risk of complications. Several case reports have documented the occurrence of GBS in patients with active or recently treated TB, suggesting a possible immunological link between the two conditions. Although rare, these cases highlight the importance of considering GBS as a differential diagnosis when TB patients develop acute flaccid paralysis or other features consistent with peripheral neuropathy. In this article, we report the case of GBS associated with disseminated tuberculosis and the importance of investigating the antecedent infection of GBS, as treating both could improve the patient's outcome.

## Case Report

A 20-year-old female patient was admitted to the emergency department of the hospital with symptoms of progressive symmetric paraparesis and neuropathic pain that developed 2 weeks prior. The symptoms progressed from distal to proximal lower limbs. Gradually, she became bedridden. She lost 16 kg within two months. Three days before hospitalization, the patient suffered from fever and cough. Her mother was diagnosed with pulmonary tuberculosis. She had no dysphagia nor urinary or faecal incontinence.

She had normal mentation on physical examination, and her vital signs were within normal limits. The muscle strength examination showed weakness in four limbs with a Medical Research Council (MRC) scale of 5/5 in the upper extremities and 2/5 in the proximal and 1/5 in the distal regions of the lower extremities. Deep tendon reflexes were reduced. Meningeal irritation signs were negative. Sensory examination found hypoesthesia level S1.

The abnormal laboratory results are anaemia, hypoalbuminemia, and high erythrocyte sedimentation rate. Chest X-ray showed left pulmonary fibroinfiltrate, suspected as pulmonary tuberculosis, and subsequent computed tomography of chest revealed ground glass opacity with centrilobular nodules in a tree-in-bud pattern, bilateral pleural effusion, and compressive atelectasis (Figure 1).

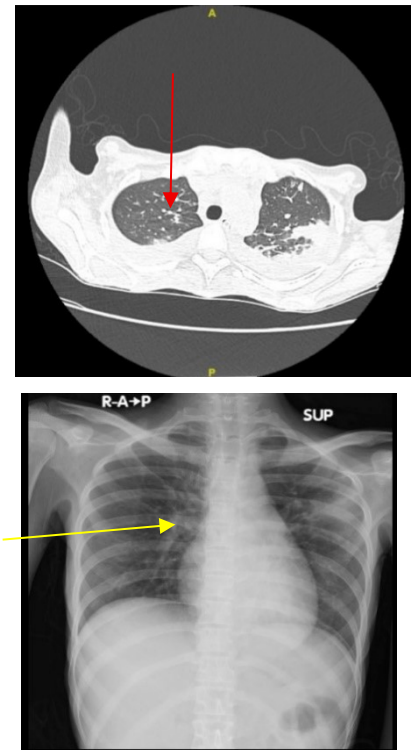


**Figure 1.** Chest X-ray showed left pulmonary fibroinfiltrate, suspected as pulmonary tuberculosis (yellow arrow), and subsequent computed tomography of chest revealed ground glass opacity with centrilobular nodules in bud, bilateral pleural effusion with compressive atelectasis (red arrow)

Whole spine magnetic resonance imaging (MRI) with contrast revealed no abnormalities, indicating that the spinal cord structure appeared normal. However, during the patient's hospital stay, episodes of diarrhea were observed. A fecal occult blood test yielded a positive result, suggesting the presence of blood in the stool. This finding prompted further gastrointestinal investigations through esophagogastroduodenoscopy and colonoscopy, which revealed a gastric ulcer and signs of colitis. To explore the underlying cause of colitis, a colon biopsy was conducted, and a molecular rapid test for tuberculosis returned a positive result, indicating a possible intestinal tuberculosis infection.

Cerebrospinal fluid (CSF) analysis showed no evidence of albuminocytologic dissociation, which typically rules out certain demyelinating neuropathies. Meanwhile, electrodiagnostic studies revealed abnormalities: there was a significant reduction in the amplitude of the compound muscle action potential, and no response was detected in the sensory nerve action potential. These electrophysiological findings strongly support the diagnosis of Acute Motor-Sensory Axonal Neuropathy (AMSAN), a severe variant of Guillain-Barré Syndrome characterized by rapid progression of motor and sensory deficits due to axonal damage.

**Table 1.** Electrodiagnostic Findings of This Patient

| **Sensory Nerve Conduction Studies** | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| Nerve/ Sites | Rec. Site | Onset Lat ms | Peak Lat ms | NP Amp µV | PP Amp µV | Segments | Distance mm | Velocity | |
| **R Median – Digit II (Antidromic)** | | | | | | | | | |
| Wrist | Dig II | 4.64 | 5.16 | 2.6 | 4.7 | Wrist – Dig II | 130 | 28 | |
| **R Ulnar – Digit V (Antidromic)** | | | | | | | | | |
| Wrist | Dig V | 3.80 | 4.48 | 20.2 | 40.0 | Wrist – Dig V | 110 | 29 | |
| **R Sural – Ankle (Calf)** | | | | | | | | | |
| Calf | Ankle | 2.55 | 3.07 | 7.2 | 6.6 | Calf-Ankle | 140 | 55 | |
| **L Sural – Ankle (Calf)** | | | | | | | | | |
| Calf | Ankle | NR | NR | NR | NR | Calf-Ankle | 140 | NR | |
| **R Superficial peroneal - Ankle** | | | | | | | | | |
| Lat leg | Ankle | 5.63 | 6.51 | 9.0 | 6.7 | Left leg - ankle | 140 | 25 | |
| **L Superficial peroneal - Ankle** | | | | | | | | | |
| Lat leg | Ankle | 5.26 | 37.4 | 9.0 | 6.6 | Left leg - ankle | 140 | 39 | |
| **Motor Nerve Conduction Studies** | | | | | | | | | |
| Nerve/ Sites | Muscle | Latency ms | Amplitude mV | Amp % | Duration ms | Segments | Distance | Lat Diff ms | Velocity m/s |
| **R Median – APB** | | | | | | | | | |
| Wrist | APB | 5.05 | 1.7 | 100 | 5.78 | Wrist - APB | 70 | | |
| Elbow | APB | 10.16 | 1.2 | 70.3 | 5.31 | Elbow - Wrist | 200 | 5.10 | 39 |
| **R Ulnar – ADM** | | | | | | | | | |
| Wrist | ADM | 4.53 | 2.4 | 100 | 10.16 | Wrist - ADM | 70 | | |
| B. Elbow | ADM | 9.32 | 2.3 | 92.6 | 10.47 | B. Elbow - wrist | | 4.79 | |
| A.Elbow | ADM | 12.08 | 2.1 | 87.3 | 10.00 | A. Elbow – B. Elbow | | 2.76 | |
| **R Peroneal – EDB** | | | | | | | | | |
| Ankle | EDB | NR | NR | NR | NR | Ankle-EDB | 80 | | |
| Fib Head | EDB | | | | | Fib head - Ankle | | NR | |
| **L Peroneal – EDB** | | | | | | | | | |
| Ankle | EDB | NR | NR | NR | NR | Ankle-EDB | 80 | | |
| Fib Head | EDB | | | | | Fib head - Ankle | | NR | |
| **R Tibial – AH** | | | | | | | | | |
| Ankle | AH | NR | NR | NR | NR | Ankle - AH | 80 | | |
| **L Tibial – AH** | | | | | | | | | |
| Ankle | AH | NR | NR | NR | NR | Ankle - AH | 80 | | |
| **F Wave** | | | | | | | | | |
| **Nerve** | | | **Fmin ms** | **Fmax ms** | **Mean Flat ms** | **% F** | | | |
| R Median - APB | | | 27.40 | 37.29 | 34.1 | 60 | | | |
| R Ulnar - ADM | | | 30.26 | 38.54 | 35.3 | 30 | | | |

APB-Abductor Pollicis Brevis; ADM-Abductor Digiti Minimi; EDB-Extensor Digitorum Brevis; AH-Abductor Hallucis; A. Elbow-Anterior Elbow; B. Elbow-Below Elbow; R-Right; L-Left

The patient was diagnosed with the AMSAN variant of Guillain-Barre Syndrome and disseminated tuberculosis (pulmonary and abdominal). Antituberculosis therapy and plasmapheresis were given. Muscle strength in the lower limbs improved (MRC 3/5) after plasmapheresis. The patient was discharged with ongoing physiotherapy and close outpatient monitoring (Table 1).

## Discussion

Guillain-Barre Syndrome (GBS) is an acute polyradiculopathy resulting in flaccid paralysis in affected muscles. The signs and symptoms can range from muscle weakness to life-threatening respiratory failure.[4] In this case, the patient was admitted to the hospital with a progressive symmetrical paraparesis, along with high-risk contact with a TB patient, weight loss within two months, fever, and cough. Without sufficiently sensitive and specific disease biomarkers, the diagnosis of GBS is based on clinical history and examination. It is supported by ancillary investigations such as CSF examination and electrodiagnostic studies. [4]. We could diagnose this patient with GBS based on clinical history and examination. CSF examination in this patient showed no albumin dissociation, which cannot rule out GBS. A normal CSF profile can be found in 10% of GBS patients throughout the disease, but the pathophysiological mechanism leading to normal values is poorly understood.[9,10] Nerve conduction studies in this patient led to an AMSAN variant of GBS. AMSAN is a newly described variant of GBS characterized by sensory, profound tendon reflexes loss, and distal weakness with an acute onset.[13] AMSAN is usually more severe than another variant of GBS.[11]

In this patient, antituberculosis therapy and plasmapheresis were administered. The patient shows improvement in muscle strength after plasmapheresis. The treatment of GBS should be initiated as early as possible before the injury to the axons becomes irreversible.[12,13] Based on the pathophysiology of GBS, which involves the immune-mediated damage of axons and/or myelin that begins with an immunological trigger, the current immunomodulatory treatment options include plasmapheresis and intravenous immunoglobulin (IVIg).[13] The mechanism of these treatments mainly involves removing the antibodies and membrane attack complex (MAC) in plasma that significantly damage the nerve, aiming to stop additional nerve damage and promote functional recovery.[12,13] Both plasmapheresis and IVIg have shown equal effect and should be started whenever the GBS patient can't walk unaided. Plasmapheresis has only demonstrated benefit within 4 weeks, and IVIg within 2 weeks, from the onset of symptoms.[3]

The patient is still undergoing physiotherapy and getting antituberculosis therapy. The physiotherapy intervention can contribute significantly to patients' recovery and quality of life.[14] Although rare, the association between GBS and TB has been reported. A brief review of ten years prior case reports, including diagnosis, treatment, and outcome (Table 2).

The occurrence of GBS has been reported not only with pulmonary TB but also with extra-pulmonary TB. In several previously reported cases of GBS-associated TB, most patients experienced clinical improvement following ATT in combination with either IVIG or plasmapheresis. In this case, the patient was diagnosed with the acute motor and sensory axonal neuropathy (AMSAN) variant of GBS in the context of disseminated TB. The patient received ATT and underwent plasmapheresis as part of the treatment regimen. While the patient improved muscle strength, independent ambulation has not yet been achieved. This may be explained by the AMSAN subtype of GBS, which is recognized as having the poorest prognosis among GBS variants, often associated with slower recovery and a higher risk of long-term neurological impairment.

The pathogenesis of GBS in TB is believed to involve molecular mimicry, involving both cell-mediated and humoral immune responses, leading to nerve damage or a direct invasion of the nerve root by tubercular bacilli.[19,21,25] Antigens may be produced in response to tuberculosis proteins or cytokines that are generated due to tuberculosis treatment.[22] The proposed mechanism is also based on the fact that in some cases, there is clinical improvement following administration of immune modulatory agents like immunoglobulins [20]

Burden in the world, Indonesia follows India as the second country with the highest burden of tuberculosis worldwide. The estimated number of incident cases in Indonesia is 10% worldwide.[6] Given the high incidence rate of tuberculosis in Indonesia, it is likely that GBS may also be prevalent but is unrecognized or untreated. Furthermore, the fact that tuberculosis is primarily diagnosed at the primary healthcare level means that it becomes crucial for general practitioners to remain highly vigilant when patients present with neurological symptoms along with tuberculosis symptoms.

Guillain-Barre syndrome could be induced by tuberculosis, and treatment for both could improve the outcome. Thus, early diagnosis is critical. Further investigations to understand the association of GBS and TB are also essential.

**Table 2.** Reported Case of GBS and TB

| Author Name | Year/Country | Diagnosis | Treatment | Outcome |
|---|---|---|---|---|
| Huluka et al[15] | 2016/Ethiopia | GBS with pulmonary TB | ATT and ventilator | Died |
| Lakhotia et al[16] | 2017/India | GBS (AMAN) with pulmonary TB | ATT and IVIG | Improvement |
| Mohta et al[17] | 2017/India | GBS (AMSAN) with disseminated TB with mediastinal lymphadenopathy, pericarditis, and pleural effusion. | ATT and plasmapheresis | Completely Recovered |
| Dalai et al[18] | 2019/India | GBS (AMSAN) with TB meningitis | ATT and plasmapheresis | Residual weakness present |
| Malakar et al[19] | 2019/India | GBS (AIPD) with pulmonary TB | ATT and IVIG | Recovery incomplete |
| | | GBS (AIDP) with pulmonary TB | ATT and IVIG | Completely recovered |
| | | GBS (AMAN) with pulmonary TB | ATT | Improving itself |
| | | GBS with pulmonary TB | ATT and IVIG | Improvement |
| Nishanth et al[20] | 2020/India | GBS (AMSAN) with TB meningitis | ATT and IVIG | Improvement |
| Singh et al[21] | 2020/India | GBS (AMSAN) with pulmonary TB | ATT and IVIG | The patient did not survive due to acute respiratory failure |
| Park et al[22] | 2020/South Korea | GBS (MFS) with pulmonary TB | ATT and IVIG | Completely recovered |
| Porey et al[23] | 2023/India | GBS(AMSAN) with pulmonary tuberculosis | ATT and IVIG | Improvement |
| Savin et al[24] | 2023/Rusia | GBS (AMSAN) with tuberculous meningitis and disseminated tuberculosis | ATT and plasmapheresis | Improvement |

ATT-Anti Tuberculosis Therapy, AMAN-Acute Motor Axonal Neuropathy, IVIG-Intravenous Immunoglobulin, AMSAN-Acute Motor and Sensory Axonal Neuropathy

## Conclusion

Guillain-Barre syndrome has the potential to be triggered by tuberculosis (TB), indicating a possible association between the two conditions. Recognizing this connection is essential, as providing appropriate and timely treatment for GBS and TB can significantly enhance the clinical outcomes for affected patients. Therefore, early and accurate diagnosis is critical to effective management and recovery. Moreover, there is a clear need for further in-depth research and clinical investigations to better understand the underlying mechanisms and the extent of the relationship between GBS and TB. Such studies could contribute valuable insights to improve diagnosis, treatment strategies, and overall patient care in cases involving both conditions.

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