

Guillain-Barré Syndrome and Tuberculosis: a Rare Association. Case Report

Síndrome de Guillain-Barré y tuberculosis: Una rara asociación. Reporte de caso clínico

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ABSTRACT

Background: Guillain-Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by progressive weakness, areflexia, and autonomic or respiratory involvement. It is classically linked to *Campylobacter jejuni* and viral infections, although reports have emerged suggesting an association with tuberculosis.

Case report: We present the case of a 25-year-old incarcerated male patient, who was admitted with paraparesis, areflexia, fever, and weight loss. Cerebrospinal fluid analysis revealed albuminocytologic dissociation, and intravenous immunoglobulin therapy was initiated for suspected GBS. Pulmonary tuberculosis was suspected based on CT findings. The patient experienced neurological deterioration, and brain magnetic resonance imaging (MRI) revealed findings consistent with meningitis. Empirical anti-TB therapy was initiated due to a high clinical suspicion of pulmonary and meningeal tuberculosis, which were subsequently confirmed by cerebrospinal fluid polymerase-chain reaction (PCR) and smear microscopy of the tracheal aspirate. The patient completed the course of anti-TB therapy. The patient currently has persistent neurological sequelae.

Conclusions: Studies suggest that *Mycobacterium tuberculosis* could induce an autoimmune response through molecular mimicry or systemic inflammatory activation.

Key word: tuberculosis, Guillain Barre, meningitis

RESUMEN

Introducción: El síndrome de Guillain-Barré es una polirradiculoneuropatía inflamatoria aguda de causa inmunológica, que se manifiesta por debilidad progresiva, arreflexia y compromiso autonómico o respiratorio. Clásicamente está vinculado a infecciones por *Campylobacter jejuni* y por virus, aunque han surgido informes que sugieren una asociación con la tuberculosis.

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Caso clínico: Se presenta el caso de un paciente varón de 25 años, privado de libertad, que ingresa por paraparesia, arreflexia, fiebre y pérdida ponderal. Se documenta disociación albuminocitológica en el líquido cefalorraquídeo y se inicia tratamiento con gammaglobulina ante la presunción de SGB. Se sospecha tuberculosis pulmonar por hallazgos tomográficos. Evoluciona con deterioro neurológico y se realiza una resonancia cerebral compatible con meningitis. Se inicia tratamiento empírico con antifímicos por la alta probabilidad de tuberculosis pulmonar y meníngea, confirmadas posteriormente mediante reacción en cadena de la polimerasa en el líquido cefalorraquídeo y baciloscopia en muestras de aspirado traqueal. El paciente completa el tratamiento antifímico. Actualmente, presenta secuelas neurológicas persistentes.

Conclusiones: Algunos estudios sugieren que *Mycobacterium tuberculosis* podría inducir una respuesta autoinmune por mimetismo molecular o activación inflamatoria sistémica.

Palabras clave: tuberculosis síndrome de Guillain-Barré meningitis

INTRODUCTION

Guillain-Barré syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy characterized by progressive muscle weakness, areflexia, and, in severe cases, autonomic and respiratory involvement. The pathophysiology of GBS involves molecular mimicry, in which an aberrant immune response to infectious pathogens generates antibodies targeting peripheral nerves, resulting in demyelination and, in some cases, axonal injury.¹ GBS is classified into subtypes according to its predominant electrophysiological pattern, primarily the demyelinating form (acute inflammatory demyelinating polyradiculoneuropathy) and the axonal forms (acute motor axonal neuropathy and acute motor and sensory axonal neuropathy). Diagnosis is supported by standardized clinical criteria, such as those established by the Brighton Collaboration, which categorize the level of diagnostic certainty based on progressive symmetric weakness, hyporeflexia or areflexia, and cerebrospinal fluid and electrophysiological findings.² Classically, GBS has been associated with infections caused by *Campylobacter jejuni* and viruses such as *Cytomegalovirus* and *Epstein-Barr virus*; however, recent reports have described an association between GBS and less common infections, including pulmonary tuberculosis.³

Although the association between GBS and tuberculosis was historically considered anecdotal, an increasing number of reported cases suggests a potential immunopathologic link. It has been

proposed that *Mycobacterium tuberculosis* can induce a cross-reactive autoimmune response directed against myelin and peripheral nerve gangliosides, thereby triggering GBS.⁴ Additionally, the release of proinflammatory mediators during pulmonary infection may act as an additional trigger for the autoimmune cascade.⁵

The clinical presentation in these patients is heterogeneous. Malakar et al⁶ reported in a case series that the classic demyelinating variant was the most common form, although more aggressive axonal variants –associated with an increased need for mechanical ventilation– were also observed. De la Torre et al⁵ described a case involving severe motor axonal involvement concomitant with inflammatory bowel disease and tuberculosis, reinforcing the hypothesis of an underlying autoimmune predisposition in these patients. Similarly, Savin et al⁷ reported a case with a protracted clinical course in a patient with active tuberculosis, significant respiratory impairment, and prolonged neurological recovery.

From a therapeutic standpoint, the coexistence of GBS and tuberculosis represents a clinical challenge. Although intravenous immunoglobulin and plasma exchange remain the first-line therapies for GBS, their use in patients with active tuberculosis requires caution due to the potential risk of exacerbating the infection.³ However, several reports have documented favorable neurological outcomes with early initiation of immunoglobulin therapy, without impairing response to anti-TB therapy.

While some authors argue that the association between GBS and tuberculosis could be a statistical coincidence in endemic regions,⁴ accumulating evidence points to an underrecognized link. Recognizing this association is critical, as prompt diagnosis and timely therapeutic intervention can improve functional outcomes and reduce respiratory complications.

CASE REPORT

25-year-old male patient who has been incarcerated for the past 3 years, with a history of childhood asthma treated with salbutamol as needed, a 15-pack-year smoking history and a history of cannabis use, presented to the emergency department with a one-week duration of self-reported headache and fever. He subsequently developed lower-extremity muscle weakness without pain, which progressed to an inability to stand. He also reported a 15-kg weight loss over the preceding month. On physical examination, the patient was alert and oriented, pupils equal and reactive to light, no signs of meningismus, 2/5 paraparesis, lower-extremity areflexia with preserved sensation. He was breathing sponta-

neously on room air. Initial laboratory evaluation revealed leukocytosis, elevated acute-phase reactants, and hyponatremia (Table 1). A chest CT demonstrated findings consistent with an infectious process suggestive of pulmonary tuberculosis (Figure 1). In view of the tomographic findings, respiratory isolation was initiated. Serological tests were requested, which returned negative. Upon admission to the emergency department, a first lumbar puncture was performed, demonstrating albuminocytologic dissociation (Table 2). The patient was evaluated by the neurology service, who deemed the presentation consistent with probable Guillain-Barré syndrome. In this context, intravenous immunoglobulin was initiated at a dose of 2 g/kg. Three days after initiating intravenous immunoglobulin therapy, the patient developed an altered level of consciousness, non-reactive pupils, and nuchal rigidity. A brain MRI with contrast was performed (Figure 2). Given the suspicion of tuberculous meningitis, anti-TB therapy was initiated. Due to neurological deterioration, the patient was transferred to the ICU, where endotracheal intubation was performed. Tracheal aspirate samples were positive for acid-fast bacilli. A repeat lumbar puncture performed

Table 1. Paraclinical laboratory results at admission

Parameter	Patient value	Reference value	Unit of measure
Hematocrit	41	38-47	%
Hemoglobin	14.2	13-17	g/dL
White blood cells	10750	4000-10000	mm ³
Platelets	457000	150000-350000	Mm ³
PT	97	70-120	%
aPTT	23	23-31	Seg
Erythrocyte sedimentation	90	0-20	Mm/h
Glucose control	109	70-110	Mg/dL
Uremia	15	10-40	Mg/dL
Creatinine	0.78	0.55-1.3	Mg/dL
AST	18	15-37	UI/L
ALP	139	46-116	UI/L
Total bilirubin	0.54	0.1-1	Mg/dL
Direct bilirubin	0.09	0.01-0.2	Mg/dL
Indirect bilirubin	0.45	0.01-0.8	Mg/dL
Na	128.6	135-145	mEq/L
K	4.7	3.5-5	mEq/L
Cl	94	98-106	mEq/L

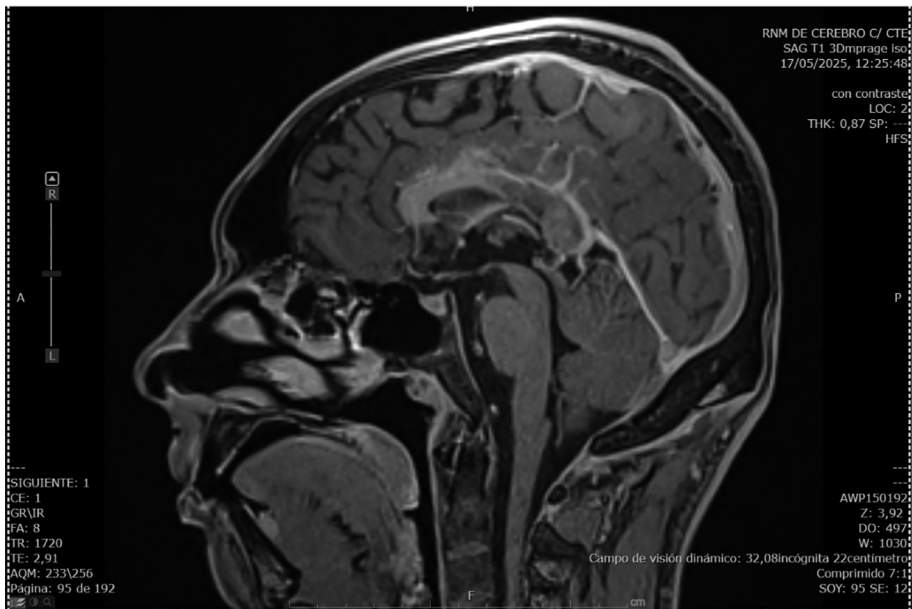
Figure 1. The non-contrast chest CT demonstrated reticulonodular and nodular opacities, some with a tendency toward cavitation, forming linear branching structures with a “tree-in-bud” pattern, involving both lung parenchyma, predominantly the apices.



Table 2. Cerebrospinal fluid analysis

Cerebrospinal fluid analysis	Valor del paciente	Valor de referencia	Unidad de medida
Colour	Light yellow		
Appearance	Clear		
Fibrin clot	Absent		
Red blood cells	2-4 per field		
Cell count	2	10	Mmm ³
Glucose	36	40-55	Mg/dL
Proteins	0.16	0.01-0.04	g/dL
Pandy test	Positive (++)		
Lactic acid	4.12	0.6-2.2	Mmol/L
India ink stain	Negative		

Figure 2. Pachymeningeal and leptomeningeal enhancement, predominantly involving the basal cisterns, cerebellar vermis, right parietal region, and bilateral parasagittal frontal regions following gadolinium administration



9 days after the initial procedure detected *Mycobacterium tuberculosis* DNA via the Xpert MTB/RIF Ultra assay, with no rifampin resistance detected. Mycobacterial cultures from both tracheal aspirates and cerebrospinal fluid (CSF) were negative. The staging workup was limited to the pulmonary and meningeal foci, with no evidence of additional extrapulmonary involvement. Family contact tracing was performed with negative results in all cases; follow-up of incarcerated contacts could not be completed. The patient completed antitubercular therapy with isoniazid, rifampin, pyrazinamide, and ethambutol for 2 months, followed by 7 months of isoniazid and rifampin. Additionally, he received adjunctive systemic corticosteroid therapy and oral pyridoxine (vitamin B6, 25 mg/day) throughout the isoniazid course to prevent drug-induced peripheral neuropathy. Currently, the patient exhibits persistent neurological sequelae, such as memory deficits and impaired balance.

DISCUSSION

We report a case notable for the simultaneous coexistence of Guillain-Barré syndrome (GBS) and pulmonary and meningeal tuberculosis. The diagnosis of GBS was established clinically by the neurology service based on clinical presentation, physical examination, and cerebrospinal fluid findings, without neurophysiological testing (electromyography and/or nerve conduction studies) due to a lack of equipment at the institution. This represents a diagnostic limitation of the present report, whereas tuberculosis was confirmed by the presence of acid-fast bacilli in tracheal aspirate and a positive PCR for *M. tuberculosis* in the CSF.

Notably, the albuminocytologic dissociation observed on the initial lumbar puncture –characteristic of GBS– can biochemically overlap with the typical profile of tuberculous meningitis, which is also characterized by marked protein elevation alongside hypoglycorrhachia (CSF/plasma glucose ratio < 0.5) and elevated lactic acid,

even with paucicellular or minimal pleocytosis in the early stages.⁹ In our patient, the reduced CSF glucose level (36 mg/dL) and elevated lactic acid level (4.12 mmol/L) on the initial lumbar puncture, combined with a low cell count, raise the possibility that these findings already reflected underlying tuberculous meningeal involvement rather than typical albuminocytologic dissociation from pure GBS. This biochemical overlap illustrates the difficulty of differentiating both entities based solely on CSF analysis and reinforces the need for close clinical and microbiological monitoring when both conditions are simultaneously suspected. Nevertheless, the presumptive diagnosis of GBS was maintained based on clinical features and physical examination findings.

This association is extremely rare. Evidence suggests that tuberculosis may act as an immunological trigger for GBS, either through molecular mimicry or via a systemic inflammatory mechanism,^{6,8} which is plausible in this patient.

Given its unusual presentation, this case underscores the imperative need for a high index of suspicion for tuberculosis in immunocompetent patients presenting with atypical neurological symptoms, particularly in endemic regions or high-risk settings such as correctional facilities. Likewise, it highlights the importance of considering GBS as a potential neurological manifestation in patients diagnosed with tuberculosis.

In conclusion, this case report adds to the growing body of evidence on the complex relationship between GBS and tuberculosis. Furthermore, we emphasize the critical importance of early detection and timely treatment of both the neurological involvement and the underlying infection. A multidisciplinary approach is essential to reduce morbidity and mortality and to optimize the quality of life of affected patients.

Conflict of interest

The authors have no conflicts of interest to declare.

REFERENCES

1. Cherian A, Ajitha KC, Iype T, Divya KP. Neurotuberculosis: an update. *Acta Neurol Belg*. 2021;121:11-21. <https://doi.org/10.1007/s13760-020-01575-0>
2. Leonhard SE, Mandarakas MR, de Assis Aquino Gondim F, et al. Guía basada en la evidencia. Diagnóstico y manejo del síndrome de Guillain-Barré en diez pasos. *Medicina (B Aires)*. 2021;81:817-36.
3. Yang GH, Wan TE, Song HP, Song HW. Comorbid Guillain-Barré Syndrome and Tuberculosis: A Case Study and Global Perspective. *Am J Trop Med Hyg*. 2025;112:1072-6. DOI: 10.4269/ajtmh.24-0623. PMID: 39999454[SC2.1].
4. Singh A, Balasubramanian V, Gupta N. The Association of Guillain-Barré Syndrome with Pulmonary Tuberculosis: Chance or Causal? *Neurol India*. 2020;68:209-18. <https://doi.org/10.4103/0028-3886.279657>
5. Gómez de la Torre R, Morís G, Pérez Martínez D, Carrio Montes I. Guillain-Barré syndrome, tuberculosis and inflammatory bowel disease: a multiple association. *Int Arch Med*. 2010;3:15. <https://doi.org/10.1186/1755-7682-3-15>
6. Malakar S, Sharma TD, Raina S, Sharma KN, Kapoor D. Guillain Barre Syndrome with Pulmonary Tuberculosis: A Case Series From a Tertiary Care Hospital. *J Family Med Prim Care*. 2019;8:1794-7. https://doi.org/10.4103/jfmpc.jfmpc_161_19
7. Savin AA, Bugun AV, Tsomaeva EB, Sergeeva SP, Sirotinskaya AY, Savin LA, Khomenko NZ. Guillain-Bare syndrome in a patient with tuberculous. *S.S. Korsakov Journal of Neurology and Psychiatry*. 2023;123(8):129-133. (In Russ.) <https://doi.org/10.17116/jnev-ro2023123081129>
8. Mohta S, Soneja M, Vyas S, Khot W. Tuberculosis and Guillain-Barre syndrome: A chance association? *Intractable Rare Dis Res*. 2017;6:55-7. <https://doi.org/10.5582/irdr.2016.01073>
9. Marais S, Thwaites G, Schoeman JF, Török ME, Misra UK, Prasad K, et al. Tuberculous meningitis: a uniform case definition for use in clinical research. *Lancet Infect Dis*. 2010;10:803-12. [https://doi.org/10.1016/S1473-3099\(10\)70138-9](https://doi.org/10.1016/S1473-3099(10)70138-9)