MAK HILL Publications International Journal of Tropical Medicine

Research Article
doi: 10.36478/makijtm.2024.2.137.140

OPEN ACCESS

Key Words
Guillain-barré Syndrome, tuberculosis, malnutrition, immunoglobulins

Corresponding Author
Jaladi Paul Rahul Ephraim, Department of Respiratory Medicine, NRI Institute of Medical Sciences, Sanghivalasa, Visakhapatnam, Andhra Pradesh

Author Designation
[1,4,5]Final year Post Graduate
[2]Professor
[3]Assistant Professor
[6,7]Second-year Post Graduate

Received: 18 March 2024
Accepted: 20 May 2024
Published: 25 May 2024

Citation: Jaladi Paul Rahul Ephraim, Viritha Pandu, Anim Roopa, Srilakshmi Maddala, Naveen Sandy Roberts Adepu, Podalakuru Prasanth and Reddy Bhargavi, 2024. TB and GBS: An Intricate Dual Challenge. Int. J. Trop. Med., 19: 137-140, doi: 10.36478/makijtm.2024.2.137.140

TB and GBS: An Intricate Dual Challenge

[1]Jaladi Paul Rahul Ephraim, [2]Viritha Pandu, [3]Anim Roopa, [4]Srilakshmi Maddala, [5]Naveen Sandy Roberts Adepu, [6]Podalakuru Prasanth and [7]Reddy Bhargavi

[1-7]Department of Respiratory Medicine, NRI Institute of Medical Sciences, Sanghivalasa, Visakhapatnam, Andhra Pradesh

Abstract
Guillain-Barre Syndrome (GBS), a severe immune-mediated neuropathy, and tuberculosis (TB), a significant infectious disease, rarely present concurrently. It may be due to malnutrition and antitubercular therapy's neuropathic effects, however it is rare in tubercular infection patients. A 75-year-old male with a history of smoking and alcohol use presented with respiratory symptoms and was diagnosed with active pulmonary TB. The patient had no significant family history of tuberculosis, nor was there any record of contact with a TB patient. The patient developed bilateral limb weakness and sensory disturbances, leading to an acute motor axonal neuropathy variant of GBS. Despite treatment with methylprednisolone, the condition worsened, causing respiratory paralysis and cardiac arrest. The pathogenesis of GBS in TB patients might involve molecular mimicry, where immune responses triggered by TB mistakenly target peripheral nerves. The severity of axonal variants of GBS, which have poorer prognoses than demyelinating types, is highlighted. The case report underscores the importance of suspecting Guillain-Barré Syndrome in tuberculosis patients presenting with neurological manifestations. It suggests that prompt administration of Intravenous Immunoglobulins can significantly improve patient outcomes. The correlation between infectious and immune-mediated neurological conditions warrants further investigation, particularly in high TB prevalence regions like India, where early and aggressive treatment is crucial to recovery.

INTRODUCTION

Guillain–Barre syndrome (GBS) is a potentially fatal condition characterized by an immune-mediated acute inflammatory polyneuropathy. It is linked to different prior infections[1]. The condition manifests at approximately 1-2 cases per 100,000 individuals annually[2]. Guillain-Barre Syndrome (GBS) encompasses autoimmune conditions affecting the peripheral nervous system. The most common form, Acute Inflammatory Demyelinating Polyneuropathy (AIDP), represents about 85% of GBS cases and is characterized by the immune system attacking the myelin sheath of nerves, resulting in widespread muscle weakness and neurological issues. Miller Fisher Syndrome (MFS), accounting for 5-10% of cases, presents with ataxia, eye muscle paralysis and lost reflexes. There are also rarer types, such as Acute Motor Axonal Neuropathy (AMAN) and Acute Motor Sensory Axonal Neuropathy (AMSAN), which mainly involve motor nerves, often following specific bacterial infections and Acute Panautonomic Neuropathy, which disrupts the autonomic functions of the body.

In India, where tuberculosis is widespread, the disease presents in various forms, including neurological complications such as meningitis, tuberculomas, brain abscesses and radiculomyelitis[3]. The co-occurrence of tuberculosis with Guillain-Barre Syndrome (GBS) was first identified in 1966. Since then, only a handful of such cases have been documented, leaving the link between the two conditions not fully understood. This report details the case of a 75-year-old male who arrived with COPD with active pulmonary tuberculosis and was later diagnosed with both GBS and pulmonary tuberculosis.

MATERIALS AND METHODS

A 75-year-old daily laborer residing in Bheemunipatnam presented with a one-month history of breathing difficulty, a two-week history of cough, productive sputum and a week of abdominal discomfort. One month ago, the patient began experiencing shortness of breath, which has escalated from grade 2 to grade 3 on the mMRC dyspnea scale. In addition, he has been suffering from a persistent cough, producing mucoid sputum without traces of blood or associated chest pain. Concurrently, he has observed a significant decrease in appetite, resulting in noticeable weight loss, but without the characteristic evening fever typical of certain infections. Looking back, the patient experienced similar respiratory complaints five years ago, which they managed with formoterol-budesonide rotacaps over six months. Notably, his medical history is void of chronic diseases like hypertension, diabetes, tuberculosis, or cardiovascular issues. He has a known history of long-term tobacco and alcohol use-habits he significantly modified 15 years ago by quitting smoking, a testament to his efforts to improve his health. A review of his family history yields no significant medical conditions, including an absence of tuberculosis, which stands out given its prevalence in certain regions. Upon clinical assessment, the patient presented with a notable pallor but none of the secondary signs, such as icterus, cyanosis, clubbing, lymphadenopathy, or edema, that might typically suggest broader systemic conditions. His vital signs were concerning, with an elevated blood pressure of 140/100 mm Hg, a rapid pulse rate of 112 beats per minute and an increased respiratory rate of 30 per minute, indicating a possible respiratory condition. Despite these signs, he maintained an average temperature of 98°F and oxygen saturation of 95% on room air, indicating adequate oxygenation at the time of examination. Upon physical examination, the chest appeared barrel-shaped and symmetrical, a common sign of chronic obstructive pulmonary conditions. Palpation revealed a shift of the trachea and mediastinum towards the right, consistent with the inspection findings and might indicate lung volume loss or atelectasis on the right. The examination revealed no tenderness upon palpation, thus effectively ruling out any potential pleuritic processes or musculoskel *et al* chest pain. The percussion from the lung fields produced a resonant note throughout, definitively indicating the absence of any consolidation or effusion. Auscultation showed normal vesicular breath sounds in most lung fields. However, it also showed bilateral basal crepitations and quieter breath sounds in the infra axillary and infra scapular areas, which could mean a lung problem, like an infection or too much fluid.

Blood tests show the patient has moderate normocytic normochromic anemia (hemoglobin level

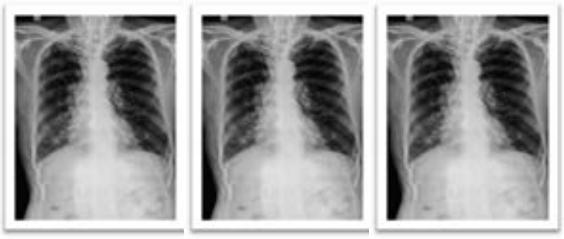

Fig. 1: shows a chest X-ray showing Emphysematous and fibrotic changes in the right upper lobe and bilateral lower lobes, with a tracheal and mediastinal shift towards the right side

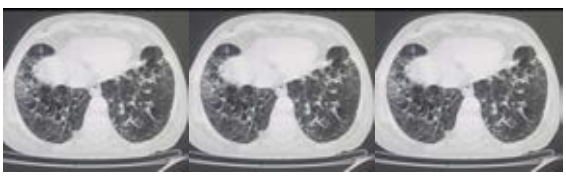

Fig. 2: Shows HRCT chest–diffuse emphysematous and fibrotic changes in bilateral lung fields

Table 1 Summary of reported cases of GBS and tuberculosis[2]

Author and year (ref. No.)	Diagnosis	Treatment	Outcome
Vyravanathan *et al*. 1983[6]	Pulmonary tuberculosis with GBS	ATT and physiotherapy	Recovered completely
Soehardy *et al.* 2005[7]	Pulmonary tuberculosis and GBS (AMSAN variant)	ATT and IVIG	Recovered completely
de la Torre *et al.* 2010[8]	Extra pulmonary tuberculosis (cervical lymph node) with GBS	ATT and IVIG	Recovered (patient developed IBD 2 months later)
Taha *et al.* 2012[9]	Pulmonary tuberculosis with GBS (AIDP)	ATT and IVIG	Recovered completely
Canham *et al.* 2014[10]	Pulmonary tuberculosis with pericardial effusion and GBS (AIDP) restricted ankle	ATT and IVIG	Difficulty walking with restricted ankle movement and constrictive pericarditis
Srikant Mohta *et al.* 2017[2]	Disseminated tuberculosis (Mediastinal and retro peritoneal lymph nodes, pleural effusion, pericardial effusion with thickening) and GBS(AMSAN)	ATT and plasm apheresis	Constrictive pericarditi without functional disability
Present case	COPD with active Pulmonary Tuberculosis and GBS (AMAN)	ATT AND IV Immunoglobulins The recommendation for IV immunoglobulins was not implemented due to economic constraints.	RespiratoryparalysisCardiac arrest and death

of 6.9 g/dL) and leukocytosis (total white blood cell count of 9,810 cells/mm3). The patient's reduced mean corpuscular volume (MCV) of 59.6 fl and regular renal and liver function tests rule out significant impairments. Some crucial findings can be seen on imaging tests, like mild pulmonary arterial hypertension with an RVSP of 44 mmHg on echocardiography and mild splenomegaly with a rough liver echotexture on abdominal ultrasound. The chest X-ray revealed emphysematous and fibrotic changes in the bilateral lower lobes, accompanied by a shift in the tracheal and mediastinal areas towards the right side. HRCT chest revealed diffuse emphysematous and fibrotic changes in bilateral lung fields, which is suggestive of COPD. Sputum CBNAAT exhibited positive with rifampicin sensitivity.

Before beginning the antitubercular treatment, we conducted initial baseline liver function tests. The patient's new-onset symptoms included bilateral lower limb pain and paresthesia, along with weakness in both upper and lower limbs. The severity of the symptoms prompted a request for a neurological evaluation.

Upon motor examination, the patient exhibited a bilateral decrease in hand grip strength, ankle dorsiflexion and neck muscle weakness. We recommended nerve conduction studies for all limbs to assess the patient's condition further. This step is critical for understanding the degree of neurological involvement and tailoring the patient's management plan accordingly. The clinical scenario raises the possibility of drug-induced neuropathy or another neurological complication that is secondary to the tuberculosis infection or treatment. The nerve conduction studies (NCS) suggest axonal Guillain-Barré syndrome (GBS), specifically the acute motor axonal variant.

We started the patient on IV methylprednisolone (1 mg) once daily for five days. Economic constraints prevented the patient from taking the recommended IV immunoglobulins. Later, the patient developed respiratory paralysis, necessitating his continuous mechanical ventilation. We provided the patient with appropriate ventilator care and treatment. Despite our best efforts, the patient's condition did not improve, leading to cardiac arrest that was unrevivable.

RESULTS AND DISCUSSIONS

The current case initially presented with dyspnea that progressed in severity, followed by a productive cough and abdominal discomfort without fever or hemoptysis. His past medical history is significant for a similar respiratory complaint managed with formoterol-budesonide, without any chronic ailments documented. The diagnosis was confirmed by high-resolution computer tomography (HRCT) and a sputum cartridge-based nucleic acid amplification test (CBNAAT)).

On the fifth day of admission, after initiating the treatment, the patient developed pain, paresthesia of bilateral lower limbs and weakness of upper limbs. Neurological examination revealed bilateral decreased hand grip, ankle dorsi flexion and neck weakness. Nerve conduction studies revealed axonal Guillain-Barré syndrome (GBS), specifically acute motor axonal variant (AMAN)[4]. This rare autoimmune disorder can result in rapid-onset paralysis and is particularly severe when respiratory muscles are involved[5].

The association between GBS and TB is not understood clearly. Previously, there have been documented five cases of TB with GBS[6-10]. (Table 1) All previously documented cases showed signs of TB before the onset of weakness. This is the case of the acute motor axonal variant, a rare type of GBS with a prevalence of fewer than 10% of GBS cases[10]. Economic constraints prevented the implementation of IV immunoglobulins in the present case. The patient experienced respiratory paralysis and underwent mechanical ventilation. Despite receiving appropriate ventilator care and treatment, his condition remained unimproved and he went into cardiac arrest, unable to be revived.

The case highlights the challenges of managing life-threatening conditions like GBS amidst chronic diseases like pulmonary TB, emphasizing the

importance of healthcare accessibility and afford ability. It underscores the need for early, aggressive treatment strategies, anticipatory guidance, and planning for advanced care directives in patients with potentially irreversible conditions.

CONCLUSION

This case highlights the rare combination of Guillain-Barré Syndrome (GBS) and tuberculosis (TB) in literature. It emphasizes the need for clinicians in India to diagnose GBS when patients present with motor symptoms alongside TB symptoms. Prompt treatment of GBS with intravenous immunoglobulin can lead to early recovery, even when co-existing with pulmonary tuberculosis, emphasizing the importance of early recognition and treatment.

REFERENCES

1. Nishanth, D., K. Rahul, K. Ashok and C. Sanjay, 2020. Tuberculous meningitis: An unlikely cause of guillain-barre syndrome. Indian J. Tuberc., 67: 139-142.
2. Mohta, S., M. Soneja, S. Vyas and W. Khot, 2017. Tuberculosis and guillain-barre syndrome: A chance association? Intractable Rare Dis. Res., 6: 55-57.
3. Saylor, D., 2021. Neurologic complications of tuberculosis. Continuum. (Minneap Minn)., 27: 992-1017.
4. Mahatoo, A., K. Topiwala, A. Nouh, Y. Zhang, I. Silverman and A. Daniel, 2018. Acute motor axonal neuropathy (Aman) presenting with truncal and appendicular ataxia (p2.453). Neurology, Vol. 90 .10.1212/wnl.90.15_supplement.p2.453.
5. Liu, D.Y., J.R. Hollenbach, J.A. Gregorin and J.H. Wynbrandt, 2020. 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., Vol. 2020 .10.1155/2020/4683507.
6. Vyravanathan, S. and N. Senanayake, 1983. Guillain-barré syndrome associated with tuberculosis. Postgrad. Med. J., 59: 516-517.
7. Soehardy, Z., A. Yuhanisa, S.S. Thein, A.G. Rohana, A.R. Fauzi, M.I. Norlinah, B.B. Hamidon and S.W. Rozaidi, 2005. AMSAN variant of Guillain Barre syndrome progressing to chronic inflammatory demyelinating polyneuropathy in a patient with Marfan's syndrome and pulmonary tuberculosis. Med. J. Malaysia, 60: 655-656.
8. Torre, R.D., G. Morís, D. Martínez and I. Montes, 2010. Guillain-barré syndrome, tuberculosis and inflammatory bowel disease: A multiple association. Int. Arch. Med., Vol. 3 .10.1186/1755-7682-3-15.
9. Taha, A.A.M. and K.H.A. Tee, 2012. Guillain-barré syndrome associated with pulmonary tuberculosis. BMJ Case Rep., Vol. 13 .10.1136/bcr-01-2012-5484.
10. Canham, E.M. and M.D. Iseman, 2014. Guillain-barré syndrome related to pulmonary tuberculosis. Ann. Am. Thoracic. Soc., 11: 855-857