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Navigating the Complexities in Tuberculosis Management: A Case of Transverse Myelitis Masquerade

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ABSTRACT

This case report underscores the intricate nature of diagnosing and treating neurological complications linked with tuberculosis (TB). Initially, a senior male with chronic obstructive pulmonary disease (COPD) was treated for lobar pneumonia, yet failed to show improvement. Further assessment revealed bilateral lower limb weakness and sensory disruptions post Anti Tubercular Therapy (ATT) initiation, suggestive of acute transverse myelitis (ATM). Prompt administration of corticosteroids alongside ATT led to substantial clinical amelioration, despite inconclusive radiological evidence. The case highlights the necessity of considering TB in unconventional neurological presentations, timely initiation of suitable therapy, and the potential benefits of a collaborative approach in enhancing patient outcomes.

INTRODUCTION

The primary treatment of tuberculosis (TB) often involves a Fixed Dosage Combination (FDC) of first-line drugs, including isoniazid (INH), rifampin (RIF), pyrazinamide (PZA), and ethambutol (EMB), tailored to the weight band of the patient. Isoniazid and rifampin play pivotal roles, targeting different stages of the TB bacilli's life cycle, while pyrazinamide aids in shortening treatment duration, and ethambutol helps prevent drug resistance. This combination therapy not only enhances treatment outcomes but also mitigates the emergence of drug-resistant strains.

However, the administration of these anti-tubercular drugs can elicit adverse drug reactions (ADRs). More often, Isoniazid is associated with peripheral neuropathy, hepatotoxicity, gastrointestinal disturbances, rash, and drug-induced lupus, while rifampin can cause flu-like syndrome, orange discoloration of bodily fluids, and thrombocytopenia. Pyrazinamide may lead to hyperuricemia, photosensitivity, and arthralgia, and ethambutol is known to cause optic neuritis and joint pain [1]. Among these, peripheral neuropathy, commonly induced by anti-TB medications, manifests as paresthesia, hypoesthesia, and motor weakness.

Effective management of ADRs is imperative to optimize treatment adherence, efficacy, and overall patient well-being. It involves a multifaceted approach, including anti-tubercular therapy tailored to the specific presentation, adjunctive corticosteroids for TBM, and in some cases, surgical intervention for complications like spinal cord compression. Patients experiencing unpleasant side effects may be less likely to adhere to their medication regimen, leading to suboptimal treatment outcomes and an increased

risk of developing drug-resistant TB strains. Some ADRs associated with ATT such as hepatotoxicity can be potentially life-threatening if unresolved. Thus, addressing ADRs is crucial in preventing serious complications, minimizing neurological sequelae, and ensuring patient safety.

This case underscores the difficulty in diagnosing neurological symptoms in patients with underlying infections such as tuberculosis (TB). Despite thorough radiological investigations, the neurological deficits remained unexplained, highlighting the intricate nature of neurological manifestations in TB patients. In such cases, clinical symptoms may not consistently align with imaging findings, reflecting the involvement of multiple systems. This emphasizes the importance of continued evaluation and interdisciplinary collaboration to achieve optimal management outcomes.

CASE REPORT

A 60-year-old male presented with sub-acute onset of fever, productive cough, and shortness of breath. He has been a known smoker for the past 40 years. He is an active smoker despite being diagnosed with COPD and following the advice of inhalation therapy consisting of long-acting beta-agonists and anticholinergics for 3 months. At admission, the patient's room air saturation maintained a SpO2 of 96%, with other vital signs being stable.

The hematological evaluation revealed an elevated white blood cell count (16,980 cells/mm) with neutrophilia (85%) and lymphopenia (8%), along with other abnormal findings. Hemoglobin levels were measured at 14.58 g/dl. Additionally, elevated Erythrocyte Sedimentation Rate (ESR) at 80 mm/hr and C-reactive protein (CRP) levels at 9.6 mg/L were observed. Liver function tests (LFT) indicated reduced total proteins (6.2 g/dl) alongside elevated Alkaline Phosphatase (ALP) and Gamma-Glutamyl Transferase (GGT). Renal function tests (RFT) and serum electrolytes were within normal limits. Glucose Random Blood Sugar (GRBS) levels were recorded at 141 mg/dl. Sputum smear for acid-fast bacilli was negative, Gram stain showed mixed flora, and Pseudomonas aeruginosa was isolated with a moderate colony count. Arterial blood gas analysis was normal.

Chest X-ray (CXR) Posteroanterior (PA) view (Fig 1) Left upper zone opacity with bilateral hyper-inflated lung fields seen at presentation. High-resolution computed Tomography (HRCT) of the thorax depicted severe diffuse emphysematous changes (of the para-septal type) in bilateral lung fields. Notably, a large consolidation was observed in the left upper lobe with partial sparing of the superior lingular segment, indicative of Lobar Pneumonitis. Additionally, a few small cavities were discernible in the apical region within the consolidation, and scattered patchy areas of ground-glass opacities were noted throughout the bilateral lung parenchyma, suggestive of Bronchopneumonitis.

He was treated with broad-spectrum antibiotics initially for lobar pneumonia. With not much clinical or radiological improvement, diagnostic reconsideration was done. Then based on clinical and radiological findings he was initiated on Anti Tubercular Therapy (ATT). Three days post-ATT initiation, the patient developed bilateral lower limb weakness, urinary and bowel retention, and sensory disturbances.

The neurologist's assessment revealed symmetrically reduced power (2/5) in both lower limbs, accompanied by hypotonia and diminished deep tendon reflexes and sensory level was identified at T10, along with lower motor neuron type bowel and bladder involvement. Notably, no signs of autonomic dysfunction were observed.

To further investigate, a plain MRI of the dorso-lumbar spine was conducted, indicating degenerative changes in the dorsal spine and a disc bulge at the L4-L5 level, resulting in indentation of the thecal sac

and compression of the nerve roots. However, no evidence of a spinal lesion explaining the observed deficits was found. Due to acute kidney injury, a post-contrast MRI was not performed.

Considering the available resources and information, the clinical possibility of transverse myelitis was entertained, prompting the administration of methylprednisolone pulse therapy for five days after a thorough discussion of the risks and benefits. Additionally, limb physiotherapy was initiated. Subsequently, the patient demonstrated gradual improvement with the prescribed treatment regimen. A follow-up CXR PA (Fig 2) view displayed partial resolution of the left upper zone opacity, indicating a positive response to treatment initiation. Upon achieving the ability to walk with support, the patient was discharged. Per the recommendation of the urologist, long-term urinary catheterization was advised.

During a follow-up visit three months later, a contrast MRI of the whole spine (Fig 3) and brain was conducted, revealing no evidence of spinal cord lesions. Furthermore, the patient did not experience any recurrence of neurological deficits, indicating favorable treatment outcomes.

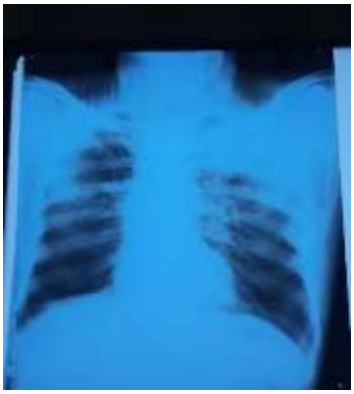

Fig 1

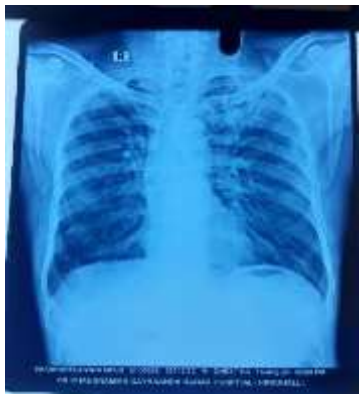

Fig 2

Fig 3

DISCUSSION

The neurological manifestations commonly associated with tuberculosis encompass a spectrum ranging from hemiplegia, paraplegia, sensory impairment, cranial nerve involvement, TB meningitis, Pott's disease, to Tuberculomas.

Acute transverse myelitis (ATM) characterizes a neurological syndrome stemming from inflammation affecting one or more segments of the spinal cord. The term "transverse" denotes the involvement of the entire cross-section of the spinal cord, including the posterior columns, spinothalamic tracts, and pyramidal tracts, yet devoid of clinical or laboratory evidence indicating spinal cord compression. Clinically, ATM presents with an abrupt onset of bilateral sensory and motor deficits alongside impaired sphincter function. Symptoms typically progress over hours to weeks, with approximately 45% of patients experiencing deterioration within 24 hours. Predominant symptoms include lower limb paraesthesia (80%-95%), partial leg movement impairment (paraparesis 50%), sensory level alterations (80%), and nearly universal bladder symptoms. Acute transverse myelitis is a relatively rare occurrence, affecting 1.2–4.6 individuals per million annually [2].

Longitudinally extensive transverse myelitis (LETM) stands apart from ATM, spanning three or more spinal segments. Transverse myelitis is frequently associated with viral agents, autoimmune disorders, or adverse drug reactions, with an immunologic reaction being implicated as the primary driver of demyelinating inflammation [3].

Tuberculous radiculomyelopathy emerges as the most prevalent complication (38.7%) linked with tuberculous meningitis, characterized by subacute areflexic paraparesis, root pain, paraesthesia, bladder disturbance, and muscle wasting. Paraparesis stands out as the most common manifestation, affecting 58.8% of patients. In the lower limbs, hypotonia is evident, accompanied by the absence of deep tendon reflexes. An extensor plantar response may also be observed. Tuberculous exudates surrounding the

lumbosacral segments of the spinal cord and lumbosacral nerve roots create a clinical picture akin to that of cauda equina syndrome [4].

Spinal cord complications, including myelopathy and radiculopathy, are integral components of the symptom profile associated with tuberculous meningitis (TBM). However, diagnosing TBM presents a considerable challenge due to its potential to manifest with unusual or atypical symptoms that may overlap with other disease processes. Diagnostic tests often yield inconclusive results due to their low sensitivity and specificity [5]. Consequently, patients with TBM may exhibit symptoms such as acute paraparesis and urinary retention despite negative TB screening results. In such instances, where there is a strong suspicion of TBM, initiating a trial of ATT is recommended to promptly alleviate symptoms, with CT and MRI scans serving as primary investigative tools. Numerous studies have highlighted the beneficial effects of ATT on patient outcomes [6]. This case highlights the importance of considering tuberculosis as a differential diagnosis in cases of acute non-compressive myelopathy, such as transverse myelitis, to ensure appropriate management and optimize patient care.

This case underscores the complexity of clinical presentations, where multiple medical conditions intertwine, posing a challenging diagnostic and therapeutic scenario. The convergence of lobar pneumonia, COPD, and neurological deficits highlights the necessity of adopting a broad differential diagnosis approach and conducting a thorough evaluation. Despite inconclusive findings from plain MRI scans, Transverse Myelitis was suspected based on clinical symptoms. Initiation of corticosteroid therapy led to noticeable improvement.

Corticosteroids have been shown to significantly reduce mortality and residual neurological deficits among survivors. The use of anti-tuberculous therapy (ATT) in conjunction with high-dose, short-course steroids yielded a remarkable therapeutic response, albeit being a rare occurrence [7]. Consequently, there are currently no consensus guidelines for its management. Follow-up MRI revealed no spinal lesions or recurrence of symptoms, underscoring the efficacy of corticosteroids in this particular case.

The successful outcome of this case emphasizes the necessity of considering TB in the differential diagnosis of atypical neurological presentations, especially in patients with underlying respiratory conditions like chronic obstructive pulmonary disease (COPD). Early recognition and intervention, coupled with comprehensive management, including ATT and corticosteroids, can mitigate the morbidity associated with TB-related neurological complications. It highlights the importance of considering atypical neurological presentations in patients with pulmonary tuberculosis. Despite inconclusive radiological findings, the clinical suspicion of transverse myelitis (TM) prompted the initiation of corticosteroid therapy alongside Anti Tubercular Therapy (ATT), resulting in significant improvement and resolution of symptoms. It underscores the favorable management outcomes achievable through comprehensive evaluation and interdisciplinary management.

CONCLUSION

TB-related spinal complications, such as tuberculous radiculomyelopathy, often present with paraparesis, root pain, bladder disturbances, and muscle wasting. Diagnosing TB-related spinal complications can be challenging due to overlapping symptoms with other conditions and inconclusive diagnostic tests. In such cases, initiating ATT promptly is crucial, as it can significantly influence patient outcomes. Further research and clinical studies are warranted to elucidate optimal management strategies for TB-associated neurological manifestations, given the complexity of diagnostic challenges and varied clinical presentations. Nonetheless, this case underscores the potential efficacy of a

multidisciplinary approach in achieving favorable outcomes and improving patient quality of life in TB-related neurological disorders.

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