being associated with neonatal meningitis and *anophelis* species with nosocomial infection in adults, both in the context of pneumonia and catheter infection, and is considered as an emerging species. It has been increasingly reported to cause life-threatening infections and even outbreaks in humans.[3,4]

Predisposing risk factors for infection are intensive care unit admission, prolonged hospitalisation, use of immunosuppressants, presence of invasive devices, chronic diseases and previous use of antimicrobials.[5]

In our case, it could be a healthcare-associated bacteraemia, perhaps in the context of the patient's recent hospital admission.

The genus often exhibits resistance to multiple antibiotics, both through biofilm formation[3,6] and through the expression of chromosomal beta-lactamases in the periplasmic space, namely an Ambler class A extended-spectrum serine beta-lactamase, which confers resistance to βlactams and class B metallobetalactamases, which hydrolyse carbapenems.[7–9]

In conclusion, *E. anophelis* is a pathogen which should be monitored because of its involvement in nosocomial infections[9,10] and its resistance to multiple antibiotics.

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Tubercular longitudinally extensive transverse myelitis: An unmissable zebra grazing the Indian medical field

Mielitis transversa tuberculosa de gran extensión longitudinal: una cebra imposible de ignorar pastando en el campo médico de la India

Longitudinally extensive transverse myelitis (LETM) is characterized by the involvement of the spinal cord spanning three or more vertebral segments and appearing as hyperintense lesions on T2 weighted-imaging magnetic resonance imaging (MRI).[1–5] The most well-known cause of LETM is primarily neuromyelitis optica spectrum disorders and, recently, the novel severe acute respiratory syndrome coronavirus infection.[1–3]

We herein present a rare case of LETM where a search for the etiology revealed a previously undiagnosed pulmonary tuberculosis.

A 17-year-old male from suburban India was admitted to the emergency department presenting with two weeks of back pain, numbness, and tingling in both lower limbs, accompanied by increased urinary frequency. He complained of headaches and low-grade fever for the past two months and had recently developed a productive cough. His personal history was significant for smoking 3–4 beedis per day.

A neurological exam revealed spastic paraplegia and patchy loss of pain, touch, and temperature sensation below the D4 spinal level. Joint and vibration senses were lost. The patient had difficulty in performing the finger nose test and dysdiadochokinesia. The remaining of the neurological examination was normal.

A diagnosis of non-compressive myelopathy with cerebellar dysfunction was made. Routine laboratory investigations were normal findings except for mild anemia. Digital chest X-ray revealed cavities and infiltrates throughout both lungs, accompanied by enlargement of the hilar lymph nodes (Fig. 1). The sputum sample tested for acid-fast bacilli was negative. However, a subsequent cartridge-based nucleic acid amplification test detected Mycobacterium tuberculosis in the sputum sample. The cerebrospinal fluid (CSF) study results were as follows: cell count was 4 cells per mm^3, the protein level was 60 mg/dl, the glucose level was 74 mg/dl, the adenosine deaminase level was 1.3 U/L, and the acid-fast bacilli Ziehl–Neelsen stain was negative. In addition, the CSF analysis was negative for anti-myelin oligodendrocyte glycoprotein and anti-aquaporin 4 antibodies, as well as herpes simplex virus types 1 and 2. Anti-nuclear antibody (ANA), ANA profile, and autoimmune vasculitis profile were negative; serum C3 and C4 were slightly elevated, with 297 mg/dl and 48 mg/dl, respectively. HIV, HBsAg, anti-HCV, and VDRL were negative. A contrast-enhanced MRI of the brain and spine revealed non-enhancing altered intensity from the D4-D5 level to the distal end of the spinal cord (Fig. 2A). In addition, an altered intensity, perilesional lesion with edema and ring enhancement (granuloma) in the left cerebellar hemisphere was observed (Fig. 2B).

The patient received a three-day regimen of intravenous methylprednisolone at 1 gram daily, which led to significant clin-

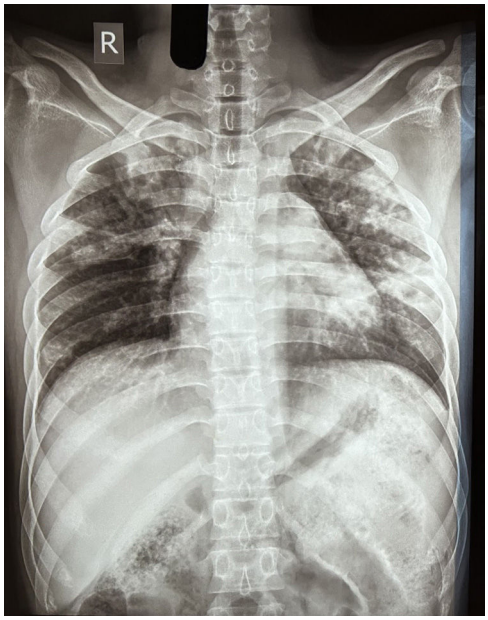

Fig. 1. Digital posteroanterior chest X-ray revealing cavities and infiltrates throughout both lungs, accompanied by enlargement of the hilar lymph nodes.

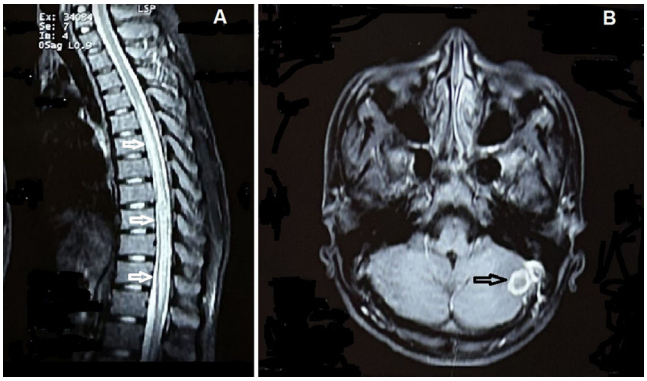

Fig. 2. Mid-sagittal T2-weighted magnetic resonance imaging (MRI) demonstrating changes in signal intensity from the D4–D5 vertebral level to the terminal portion of the spinal cord, indicated by arrows (A). Axial T1-weighted MRI with gadolinium contrast highlights a lesion with altered signal intensity, surrounding edema, and ring enhancement suggestive of a granuloma in the left cerebellar hemisphere, marked by arrow (B).

ical improvement. Concurrently, IV dexamethasone at a dosage of 0.4 mg/kg/day was administered throughout the hospitalization period. Upon discharge, the treatment plan included an oral prednisolone regimen starting at 40 mg daily, gradually tapered over the next two months. Additionally, the patient was initiated on antitubercular therapy in accordance with the guidelines outlined by the National Tuberculosis Elimination Programme (NTEP). Initially, considering the patient's weight of 48 kg, a regimen consisting of three tablets of a fixed drug combination containing isoniazid (75 mg), rifampicin (150 mg), ethambutol (400 mg), and pyrazinamide (275 mg) was administered daily. As the patient's weight surpassed 50 kg, the dosage was adjusted to four tablets of the same fixed drug combination daily, maintained for two months, constituting the intensive phase. This was followed by a continuation phase lasting 16 months, during which four tablets each of isoniazid (75 mg), rifampicin (150 mg), and ethambutol (400 mg) were administered daily.

At the six-month follow-up, the patient demonstrated no notable neurological deficits. Imaging, including an MRI of the brain and spine, showed no abnormalities, and a chest X-ray indicated significant resolution of the lesions previously observed.

Tubercular LETM is an uncommon diagnosis, and all individuals who present with symptoms indicative of transverse myelitis should undergo thorough investigations for a potential underlying tubercular infection, especially in countries like India, where tuberculosis is endemic.[4]

The precise mechanisms by which Mycobacterium tuberculosis leads to LETM are not fully elucidated. It is hypothesized that tuberculous myelitis results from an immune-mediated reaction directed against the antigens from the Mycobacterium cell wall, which subsequently causes inflammatory demyelination of the spinal cord.[4]

Neuroimaging played a critical role in the diagnosis of this case, revealing extensive involvement of the spinal cord. Furthermore, a chest X-ray demonstrated typical features of pulmonary tuberculosis. The analysis of CSF showed elevated protein levels, indicative of inflammation or infection affecting the central nervous system. Additionally, the diagnosis was confirmed by a positive result for Mycobacterium tuberculosis in the sputum sample. These features underscore the complexity of diagnosing tubercular myelitis, highlighting the necessity for a comprehensive approach that integrates clinical, radiological, and laboratory findings to achieve accurate diagnosis and effective treatment.[4]

It is crucial to emphasize that obtaining bacterial confirmation of tuberculosis can be challenging due to the disease's paucibacillary nature, particularly in cases of extrapulmonary tuberculosis.[4] Therefore, a high index of clinical suspicion, supported by corroborative evidence from neuroimaging and laboratory investigations, should suffice to establish a diagnosis of tuberculosis and initiate treatment.[4]

Authors' contributions

All authors contributed significantly to the creation of this manuscript; each fulfilled the criterion established by the ICMJE.

Ethical statement

Written informed consent was obtained from the patient participating in the study (consent for research). We also confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this report is consistent with those guidelines.

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Conflict of interest

Dr. Ila Singh (ilasingh188@gmail.com) reports no relevant disclosures.

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