Case Report

Recurrent Paradoxical Tuberculosis with Chest Wall Abscess and Optochiasmatic Tuberculoma

Suresh Kumar, Sumeet R. Dhawan, Lokesh Saini, Paramjeet Singh, Sanjay Verma, Meenu Singh

INTRODUCTION

Paradoxical response is defined as a new-onset clinical or radiological worsening after initiation of antituberculous therapy (ATT) and which is not due to disease relapse or failure. Such response may be seen in 6%–30% of patients receiving ATT. We describe an adolescent with recurrent paradoxical responses, complicating tuberculous meningitis.

CASE HISTORY

A 13-year-old girl was diagnosed as tubercular meningitis (TBM) 2 years back when she had signs and symptoms of chronic meningitis. Her mother had sputum-positive pulmonary tuberculosis and was on irregular ATT. She succumbed to progressive disease (acifast bacilli sensitivity not known). The child had lymphocytic predominant pleocytosis and elevated protein levels in cerebrospinal fluid (CSF) analysis. Her Gene Xpert and CSF culture tests were sterile. Contrast computed tomography (CT) scan showed basal exudates and mild communicating hydrocephalus [Figure 1]. Her chest radiograph and enzyme-linked immunosorbent assay for human immunodeficiency virus were negative. She completed 12 months of ATT. Contrast magnetic resonance imaging (MRI) of brain at 12 months of therapy was unremarkable. She developed chest wall abscess 6 months after stopping ATT. Aspiration from the abscess showed granulomatous inflammation (acid-fast bacilli smear and culture were negative, Gene Xpert was negative). Keeping a clinical possibility of resistant tuberculosis, she was started on Category 2 anti-tubercular therapy. Her collection on chest wall reduced on three months of anti-tubercular therapy. After 5 months of ATT, she presented with unilateral left frontal headache and insidious-onset, gradually progressive, painless loss of vision on the left eye for the last 20 days. She had only perception of light and relative afferent pupillary defect on the left side. Extraocular movements were normal. Her right eye’s best corrected visual acuity was normal. CSF analysis was not suggestive of meningitis (Gene Xpert was negative). Optic nerve compression by tuberculoma and ethambutol-induced optic neuropathy were considered. Contrast MRI showed tuberculoma in the suprasellar cistern abutting the optic chiasma and left optic nerve. The child was treated with 5 days of pulse methylprednisolone following which her vision improved to 6/24 in the left eye. She was discharged on oral steroids while her maintenance ATT was continued. She did not develop any new clinical symptoms after 6 months of follow-up.

DISCUSSION

Recurrent paradoxical tuberculosis is highlighted in the same child. It is paramount to differentiate disease
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relapse in any child with new lesions after stopping treatment (chest wall abscess) or new focal deficits on ATT (vision loss) from paradoxical tuberculosis as in the index case.\[^{1}\] However, the neurological disease had completely improved (as evident by normal MRI after 12 months of ATT) when the child developed chest wall abscesses. Similarly, the chest wall abscesses had improved when the child developed optochiasmatic tuberculoma. Though the child was treated with Category 2 ATT when she developed chest wall abscess, in retrospect, it seems more likely to be a case of paradoxical phenomenon. Both these events suggested drug-responsive tuberculosis in the index case. So the same maintenance ATT was continued when the child developed vision loss because of suspicion of paradoxical response and not drug-resistant tuberculosis. Such late deterioration may be seen in isoniazid-mono-resistant tuberculosis, which was excluded by culture.\[^{1}\]

Paradoxical tuberculosis is a delayed-type hypersensitivity reaction to mycobacterial proteins, which mostly occurs within 3–6 months of initiation of ATT seen in 6%–30% patients receiving ATT.\[^{2,3}\] Recurrent paradoxical tuberculosis is exceedingly rare, and a remote possibility of delayed presentation of monoresistance cannot be entirely ruled out.

Second, this case highlights the importance of early initiation of pulse corticosteroid therapy in preventing the permanent visual loss.\[^{4}\] Vision loss in TBM may be early or late. Early vision

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**Figure 1:** Contrast CT scan (A, B) showing basal exudates (white arrow) and prominent ventricle horns (black arrow). MRI showing contrast-enhancing suprasellar lesion abutting the optic chiasma (sagittal view, C; axial view, D). The lesion is surrounded with perilesional edema as seen on T2-weighted (E) and Fluid-attenuated inversion recovery sequence (F)
loss may be due to optic atrophy secondary to raised intracranial pressure, cortical infarcts, bacterial invasion of optic nerve, compressed optic chiasma because of enlarged third ventricle, and ischemic optic neuropathy. Delayed vision loss as in index case could be due to ethambutol-induced optic neuropathy, paradoxical optochiasmatic arachnoiditis, optochiasmatic tuberculomas, and ventriculoperitoneal shunt blockage. The management of paradoxical tuberculosis includes the continuation of ATT, systemic steroids, and surgical intervention. Decompression of optochiasmatic region may be needed to preserve vision if medical treatment fails.

To conclude, recurrent paradoxical response can rarely complicate childhood tuberculosis, and meticulous clinical analysis of sequence of events in the index case can prevent misdiagnosis of drug-resistant tuberculosis.

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Conflicts of interest
There are no conflicts of interest.

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