Rare manifestation of common disease: An adolescent girl with transient ischemic attack (TIA) like presentation in multiple tuberculoma

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Abstract

Central nervous system (CNS) tuberculosis is a life-threatening infection and has a wide variety of presentations in children. We report a case of intracranial tuberculomas with tuberculous cervical lymphadenitis in an adolescent girl with a recurrent transient ischemic attack (TIA) like unusual presentation managed with antitubercular therapy and anti-convulsant.

Keywords: Focal neurological deficits, transient ischemic attack (TIA), tuberculoma

Introduction

Tuberculosis (TB) is a multi-system infectious disease caused by Mycobacterium tuberculosis in humans.¹ It commonly infects the lungs but can spread in form of extrapulmonary TB. Central nervous system (CNS) involvement is one of the most serious forms of TB and can manifest as meningitis, intracranial tuberculoma, or spinal tuberculous meningitis.² Intracranial tuberculomas are an important cause of intracranial space-occupying lesions in endemic countries. They may present with non-specific features with/without pulmonary features, thus making the diagnosis a clinical challenge.¹,³ Though rare in Western countries, intracranial tuberculomas have a high incidence in areas where TB is still endemic such as the Indian subcontinent.⁴ Another form of extrapulmonary TB is tuberculous lymphadenitis which can affect lymph nodes but the involvement of suprasternal lymph node is rare.

We report a case of intracranial tuberculoma with tuberculous lymphadenitis in an adolescent female with unusual neurological presentation successfully managed with antitubercular and anticonvulsant therapy.

Case Description

A 14-year-old girl presented with complaints of multiple episodes of numbness on the right half of her face and right upper limb for one day. These episodes lasted for a duration of 5–10 min and were associated with difficulty in speaking and using the right hand. There was no complaint of headache, vomiting, visual disturbances, unconsciousness, seizures, aura, vertigo, dizziness, or fever. There was no significant past medical and surgical history. Also, there was no family history of TB or contact with a TB patient. She was immunized till 5 years of age as per history from her parents.

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On examination, she was conscious, well-oriented, afebrile, pulse rate 90/min, blood pressure 120/84 mm Hg, respiratory rate 14/min, some pallor, and single enlarged suprasternal lymph node, which was non-tender, mobile, and firm. [Figure 1] No lymphadenopathy was seen in other regions. Systemic examination did not show any abnormality.

Laboratory investigations on admission revealed haemoglobin of 6.3 gm/dL, total leukocyte count 4200/cumm, platelet count 3.63 lakh/cumm, and ESR 60 mm/h. Liver function tests were normal. She was found to be HIV-negative. Her X-ray chest did not reveal any pathology. Plain and contrast-enhanced MRI scan of the brain showed multiple, well defined, ring-enhancing, hyperintense lesions with a hypo-intense centre in the anteromedial, posteromedial portions of the left cerebellar hemisphere, in the posterior part of the left frontoparietal operculum with a thin rim of hyperintensities around these lesions that favoured perilesional oedema. These findings were suggestive of multiple tuberculomas. [Figure 2].

USG guided fine needle aspiration cytology (FNAC) of the single suprasternal lymph node was also performed which revealed extensive caseous necrosis consisting of lymphocytes and neutrophils, multiple tubercular bacilli on AFB staining, and plenty of pus cells. The culture of the specimen showed the growth of Mycobacterium TB which confirmed the diagnosis of tuberculous lymphadenitis.

Based on this diagnosis patient was enrolled under the Revised National Tuberculosis Control Programme (RNTCP) and four-drug antitubercular therapy (ATT) was started along with prophylactic anticonvulsant. TIA-like episodes subsided in 4–5 days after starting this therapy and never recurred throughout the follow-up of 1 year. She completed the ATT course. A subsequent MRI scan was done which was normal. Anticonvulsants were gradually tapered and stopped. She remained asymptomatic during follow-up of further 6 months.

**Discussion**

TB is an endemic disease in India with an estimated incidence of 204/100,000 population according to the Global Tuberculosis Report. It can infect any age group but children, pregnant women, the elderly, and those with other co-morbid conditions are at high risk. CNS TB accounts for only 1% of all TB cases. Haematogenous seeding of bacilli in the CNS after the primary pulmonary infection is one proposed mechanism of CNS involvement in TB. Following this, tubercle formation occurs which is a defence mechanism to limit the spread of infection by forming a fibrous capsule around the foci of infection.

The clinical presentation of tuberculoma depends on their anatomic location in the brain, compression of surrounding structures, obstruction of cerebrospinal fluid pathways, and/or seizures. There is a sub-acute onset of symptoms and signs such as headache, nausea, vomiting, visual disturbances, cranial nerve defects, seizures, depressed level of consciousness, and focal neurological deficits. These non-specific neurological features can lead to difficulty in diagnosis of tuberculoma. Tuberculoma can present with any focal neurological deficit ranging from monoplegia to quadriplegia, facial weakness, and sensory deficit depending on the site of lesion in the brain. The pathogenesis of focal neurological deficit in tuberculoma is the periadventitial inflammation and proliferative subintimal fibrotic lesions consequent to chronic inflammatory processes. There is also ischaemic softening of cerebral tissue causing sensory-motor deficit.

After clinical examination, the next line of investigation includes laboratory tests and radiological imaging. MRI is the preferred investigation as it has a higher resolution and tissue contrast. MRI appearance depends upon the stage of tuberculoma: non-caseating granuloma, caseating granuloma, caseating granuloma with central liquefaction, and calcified granuloma.
In our patient, suprasternal tuberculous lymphadenitis was present. It is an uncommon site for tuberculous lymphadenitis in children.[13] The differential diagnosis of a midline neck swelling includes developmental, inflammatory, infective, and neoplastic pathologies. The first line of investigation in such cases is ultrasound followed by FNAC or excisional biopsy depending upon the nature of the lesion. Tuberculous lymphadenitis commonly presents as swelling and is not associated with constitutional symptoms unless coupled with pulmonary TB. We performed USG guided FNAC of the suprasternal lymph nodes. The AFB stain and culture reports may be false negative at times due to low bacterial load but in our case, both AFB staining and culture showed the presence and growth of mycobacterium TB.

Along with ATT, we started prophylactic anticonvulsant and on day 5 the patient became asymptomatic. It is proven that regardless of the aetiology of seizures, the number of seizures occurring before adequate treatment is a paramount prognostic factor for predicting the patients’ response to AEDs. Therefore, early treatment with AEDs is crucial and may significantly reduce the risk of chronic epilepsy following TB infection.[12‑14]

Excision of cervical lymph nodes in case of tuberculous lymphadenitis is indicated when a single unilateral group is involved and inflammatory changes are seen on the overlying skin. Excision is always supplemented with a full course of ATT as this approach reduces the risk of medical failure and prevents relapse of infection.[9] However, in our case, it was subsided without surgical excision.

In this case, we want to convey some important points for primary care physicians. First is that being an endemic disease TB can present in any form including focal non-specific symptoms and one should not forget to check suprasternal lymph node palpation. Also, always such lymph nodes to be thoroughly investigated and treated.

This is relevant to them because they are the first physicians who come in contact with such patients and they can take decision appropriately whether such patients need outdoor treatment or require hospitalization.

**Conclusion**

In endemic countries such as India, TB can present with non-specific or unusual neurological manifestations. Isolated suprasternal tuberculous lymphadenitis is an uncommon site for tuberculous lymphadenitis. Compliance with treatment is a major deciding factor in TB and must be addressed by clinicians. In cases of CNS TB, anti-convulsant with a full course of antitubercular treatment is the mainstay for the long-term prevention of seizures.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Saleh M, Saeedi AA, Ali Pooran A. Brain tuberculomas: A case report. Jundishapur J Microbiol 2014;7:11252.
2. Sethi P, Treece J, Omweni C, Pai V, Rahman Z, Singh S. The importance of a complete differential: Case report of a tuberculoma in a patient without pulmonary involvement. Cureus 2017;9:e1405.
3. Mohammadian M, Butt S. Symptomatic central nervous system tuberculosis. A Case report in the United States and literature review. IDCases 2019;17:e00582.
4. World Health Organization. A brief history of tuberculosis control in India. 2010. Available from: https://apps.who.int/iris/handle/10665/44408.
5. Marais S, Van Toorn R, Chow FC, Manesh A, Siddiqi OK, Figaji A, et al. Tuberculous meningitis international research consortium. Management of intracranial tuberculous mass lesions: How long should we treat for? Wellcome Open Res 2019;4:158.
6. Garkowska A, Moniuszko A, Ustymowicz A, Panciewicz S, Czupryna P, Krupa W, et al. Tuberculosis of the central nervous system-A Case Report. Przegl Epidemiol 2013;67:23-7.
7. Chatterjee S. Brain tuberculomas, tubercular meningitis, and post-tuberculcular hydrocephalus in children. J Pediatri Neurosci 2011;6(Suppl S1):96-100.
8. Panda BN, Shah RS, Nair R. Tuberculosis of brain: Presenting with recurrent stroke and intracranial haemorrhage: A case report. Med J Armed Forces India 1995;51:139-41.
9. Venter F, Heidari A, Galang K, Vihweg M. An atypical presentation of tuberculosis in an immunocompetent host. J Investig Med High Impact Case Rep 2018;6. doi: 10.1177/2324709618798407.
10. Khatri GD, Krishnan V, Antil N, Saigal G. Magnetic resonance imaging spectrum of intracranial tuberculosis lesions: One disease, many faces. Pol J Radiol 2018;83:e524-35.
11. Vijay V, Vaishya R. Tuberculous suprasternal notch abscess in a child. BMJ Case Rep 2016;2016:10. doi: 10.1136/bcr-2015-214269.
12. Sharma SK, Ryan H, Khaparde S, Sachdeva KS, Singh AD, Mohan A, et al. Index-TB guidelines: Guidelines on extrapolmonary tuberculosis for India. Indian J Med Res 2017;145:448-63.
13. Monitoring during treatment. Treatment of Tuberculosis:
Guidelines. 4th ed. Geneva: World Health Organization; 2010. 4.
14. Abdulaziz AT, Li J, Zhou D. The prevalence, characteristics, and outcome of seizure in tuberculous meningitis. Acta Epileptologica 2020;2:1-8.
15. Weiler Z, Nelly P, Baruchin AM, Oren S. Diagnosis and treatment of cervical tuberculous lymphadenitis. J Oral Maxillofac Surg 2000;58:477-81.