Case Report

An Unusual/Varied Presentation of Two Cases of Tuberculous Meningitis

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Abstract

Tuberculosis (TB) in India has varied presentation which may manifest as pulmonary as well as extrapulmonary disease leading to difficult diagnosis. Here is the presentation of two cases of tuberculous meningitis (TBM). The first case was of a 35-year-old female having cortical paraparesis along with IX and X cranial nerve palsy. Magnetic resonance imaging of the brain with contrast showed multiple ring-enhancing lesions suggestive of TBM. The second case was of a 17-year-old female with anemia, hyponatremia, and hypochloremia having disseminated TB in the lungs, abdomen, and meninges. TBM presentation maybe varied and may present with disseminated TB.

Keywords: Basal meningitis, disseminated tuberculosis, tubercular meningitis

Introduction

Tuberculosis (TB) can lead to the involvement of any organs, lungs being the most common. Patients may have involvement of lung parenchyma, pleura, meninges, pericardium, kidney, liver, genitourinary system, lymph nodes, eyes, and others.[1] Disseminated TB may present in atypical fashion and may cause diagnostic challenges.[2] This article deals with two case reports of unusual presentation of central nervous system (CNS) TB: first one presented with features of lower cranial nerve palsy and the second patient presented with disseminated TB with tuberculous meningitis (TBM). Imaging plays an important role in substantiating the clinical diagnosis.[3]

Tuberculous granulomatous infection caused by *Mycobacterium tuberculosis* is a major health problem. Efforts are being made globally for the "Stop TB" Partnership program since 2000; however, it is still a public health problem. The WHO data suggest that, in 2017, a total of 10.0 million people developed TB all over the world of which 5.8 million were men, 3.2 million were women, and 1.0 million were children. India contributes 27% of cases of TB and is one of the eight countries which have added two-third of the world TB patients.[4] Indian doctors working with different specialties may come across TB patients in immunocompetent and also in immunocompromised patients.[5]

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As a natural history of disease is variable, the diagnosis of TBM may be difficult at times.[6] We came across two patients in the recent past having TBM: the first one having basal meningitis with lower cranial nerve palsy and another one having disseminated TB.

Case Reports

Case 1

A 35-years-old female patient presented with complains of dull aching, holocephalic headache with heaviness of eyes for the past 1 year. She also had bilateral lower limb weakness in the form of difficulty in walking without support. She did not have any history of vomiting, photophobia, phonophobia, blurring of vision, syncope, or episodes of seizure. She also complained of dysphagia for solids and liquids of recent onset with hoarseness of voice, dry cough, decreased appetite, and weight loss.

On examination, her pulse rate was 82/min and blood pressure (BP) of 96/70 mmHg and she had normal respiratory, cardiovascular, and per abdominal examination. Her detailed

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neurological examination revealed normal higher mental functions, poor gag reflex, deviation of the uvula on the right side, and absent soft palate movement on the left side. Pain, temperature, and tactile sensations of the posterior third of the tongue were affected suggestive of IX nerve involvement. She had Grade IV power in both lower limbs with brisk deep tendon jerks and extensor planters. The patient also had positive meningeal signs. She had hyperemic optic discs suggestive of early papilledema. The patient was immune competent. She had mild iron deficiency anemia without koilonychia. Laryngoscopy showed the left vocal cord with palatal palsy. Her X-ray of the chest was normal. Upper gastrointestinal endoscopy did not show any esophageal pathology. Magnetic resonance imaging (MRI) of the brain with contrast showed multiple ring-enhancing lesions along the falx cerebri, bilateral tentorium cerebelli, within meninges, and posterolateral aspect of the left cerebellar hemisphere [Figure 1]. T2 and fluid-attenuated inversion recovery (FLAIR) images showed hyperintense gyral signal intensity in the bilateral frontal, paramedian, and left occipital region. A possibility of infective etiology, likely TBM, was considered. Cerebrospinal fluid (CSF) was done which was indicative of tubercular etiology. Any form of confirmatory diagnostic tests such as gene expert or culture and sensitivity tests were not done, but the patient was given antitubercular treatment with steroids. On follow-up at the outpatient department, she showed a marked improvement.

Case 2

A 17-year-old unmarried female having altered sensorium of around 24 h was brought to our hospital in a confused and disoriented state. As per relatives, she was unwell for 1 month and had high-grade intermittent fever with chills, cough with scanty mucoid sputum, vague abdominal pain, and anorexia. She had progressive generalized weakness and weight loss.

On examination, her body mass index (BMI) was 19.65, pulse rate was 110/min, BP 102/66 mmHg and had Glasgow Coma Scale of 14/15. She had bilateral reactive pupils of 3 mm, having normal superficial and deep tendon reflexes, bilateral extensor planters as well as the presence of meningeal signs. Her fundus examination showed bilateral papilledema without choroid tubercles. The rest of CNS examination was normal. The patient was immune competent. She had mild anemia, hyponatremia, and hypochloremia (hemoglobin was 9.8 gm%, packed cell volume: 27%, serum sodium: 122 mEq/L, and serum chloride levels of 91 mEq/L). His urine osmolality was 166 mOsm/kg. Chest X-ray showed small nodular opacities in bilateral lung fields suggestive of milliary TB. CSF contained increased protein levels with low glucose and adenosine deaminase levels. Fluid cytology examination confirmed Tuberculosis with lymphocytic predominance. Her contrast-enhanced computed tomography (CECT) of the thorax showed multiple, diffusely distributed, small nodular opacities in both the lung fields. There were "tree-in-bud (TIB)" opacities peripherally. The findings were thus suggestive of pulmonary TB with an endobronchial spread [Figure 2].

The patient's ultrasound of the abdomen showed round and oval enlarged lymph nodes, appearing on CECT of the abdomen as multiple matted conglomerated lymph nodes in the preaortic and para-aortic region with largest measuring 52 mm × 26 mm in the left para-aortic region [Figure 3].

MRI of the brain with contrast showed abnormal leptomeningeal enhancement in the left Sylvian fissure and parietal region with sulcal hyperintensity on FLAIR images, suggestive of meningitis [Figure 4].

The patient was treated for hyponatremia, and antitubercular treatment was given under the RNTCP guidelines. The patient was also given corticosteroids and supportive management. She improved and was discharged from the hospital with the recommendation of follow-up by TB expert at our teaching hospital as per the RNTCP protocol.

Discussion

The first case of TBM presented with acute to subacute progressive neurological illness with central origin paraparesis, signs and symptoms suggestive of IX and X cranial nerve palsy along with the involvement of meninges. TBM can lead to basal meningitis and adhesions which can cause cranial nerve palsies.[6] The affection of the lower cranial nerves

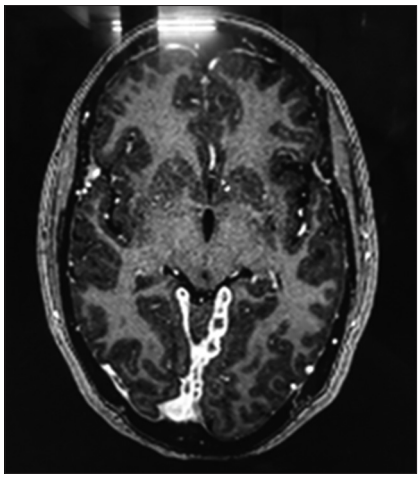

Figure 1: Magnetic resonance imaging of the brain with contrast suggestive of leptomeningeal enhancement and ring-enhancing lesion

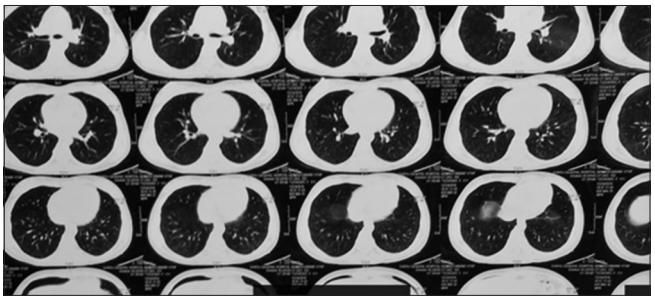

Figure 2: Contrast-enhanced computed tomography of the thorax suggestive of bilateral nodular opacities with a tree in bud appearance

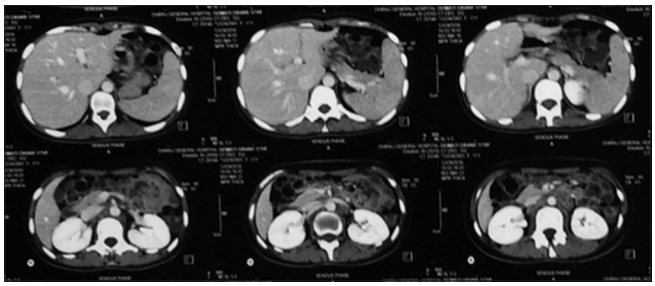

Figure 3: Contrast-enhanced computed tomography of the abdomen suggestive of pre- and para-aortic lymphadenopathy

by tubercular meningitis is rare and when present has poor outcomes.[7] However, our patient had a good recovery. Lower cranial nerves which include IX, X, XI, and XII are important for physiological function such as for taste, swallowing, and speech and also related to heart and intestinal functions.[7] Multiple cranial nerve palsy is reported in leptomeningeal melanomatosis.[8] Chronic infective meningitis such as tuberculous and fungal, sarcoidosis, and other granulomatous meningitis results in basal meningitis and cranial neuropathies. In consecutive series of 979 in patients of multiple cranial palsy in the USA, 102 patients had "infection" as the cause, of which 9 had TBM. Tenth nerve involvement with other cranial nerves involvement is not uncommon, and in this series, it was present in 220 patients with the involvement of other cranial nerves.[9]

The glossopharyngeal nerve (IXth cranial nerve) with vagus (Xth cranial nerve) relates to the upper aerodigestive tract may be affected by a lesion in peripheral nerves or due to the lesion in the brain stem. Sensory inputs and motor outputs of cranial nerves IX and X work together in harmony, and its affection may lead to dysphagia, dysphonia, dyspnea, and loss of gag reflex which was present in this patient.[10] Our patient had the affection of pain, temperature, and touch sensations of the posterior third of the tongue suggestive of IX nerve involvement. Literature is sparse with regard to the IX nerve involvement as it is not routinely documented, and many series of multiple cranial nerve palsy are retrospective.[7,9]

The second case was of disseminated TB in a young female who presented with clinical signs and symptoms of tuberculo-meningoencephelitis with miliary TB and endobronchial spread. Up to 25% of patients with miliary TB may have meningeal involvement.[11] Her CT of the thorax was showing "TIB" pattern, appearance of multiple areas of centrilobular nodules with a linear branching pattern. It may be suggestive of endobronchial spread. The appearance of such a pattern on CT may be due to infections or aspiration, the most common being mycobacterial disease.[12]

She had abdominal TB in the form of tubercular lymphadenitis with the involvement of pre-aortic, para-aortic, retrocaval, and aortocaval lymph nodes. The patient also had mild anemia and hyponatremia. Disseminated TB remains a challenging problem in view of diagnosis as well as management and may report late.[1,2] This young immunocompetent patient had classical symptoms of TB such as cough, fever, weight loss, and loss

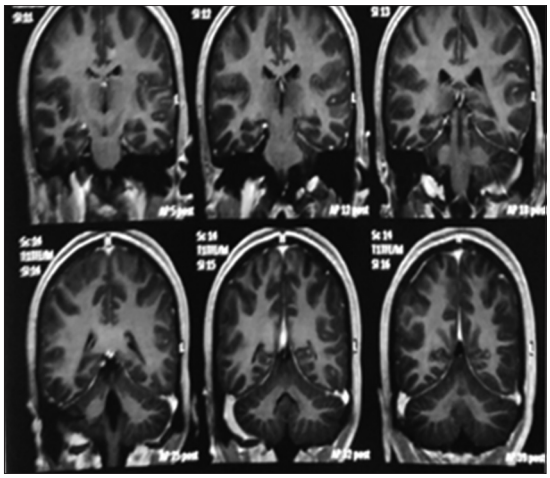

Figure 4: Magnetic resonance imaging brain with contrast suggestive of leptomeningeal enhancement

of appetite. The clinical characteristics and outcome may vary according to the type and number of organs involved. As symptoms remain nonspecific, at times, the diagnosis could be missed. As reported by Talvera *et al*., 33%–80% of such form of TB could be missed antemortem and is detected on autopsy.[13]

Impaired consciousness of this patient was either due to hyponatremia or because of tubercular meningoencephalides. Hyponatremia is considered as a serious metabolic consequence of TBM, especially in children.[14] It may be because of the involvement of the adrenal gland and associated Addison's disease. However, nonAddisonian hyponatremia has also been reported, as the syndrome of inappropriate antidiuretic hormone is reported in tubercular meningitis.[15]

The patient had vague abdominal pain for long time. Diagnosis of abdominal tuberculosis is difficult due to varied presentations. Involvement of peritoneum is the most common form, while a considerable number (27%) have TB in mesenteric lymph nodes, other abdominal structures, and also at multiple sites.[16] Our patient had abdominal lymphadenitis involving pre- and para-aortic as well as retroperitoneal group of lymph nodes. Disseminated and nondisseminated TB may lead to the involvement of omental, mesenteric, anterior pararenal, and upper aortic lymph nodes.[16,17] Lymph nodes in our patient were conglomerated and fused together which is a feature of TB.

Clinical and radiological features of tuberculosis and sarcoidosis are quite overlapping and thus diagnostic dilemma may remain. Sarcoidosis leads to multisystem involvement, lung involvement and multiple cranial nerve palsy like tuberculosis. Constitutional symptoms are also present in one third of the patients.[18] In both described cases, diagnosis of tuberculosis was favored on basis of type of clinical presentation, on positive treatment response, on laboratory investigation and imaging.

All specialists working in India have to make a note that disseminated TB is still prevalent and may present in atypical forms.[19]

Conclusion

Disseminated TB may present in a variety of ways and may lead to critical illness. If diagnosed at the appropriate time, successful treatment is possible.

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.

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