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Multiple Intracranial Tuberculomas - Diagnostic Dilemmas (Case Series)

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ABSTRACT

Intracranial tuberculoma is the second most common presentation of central nervous system (CNS) involvement in immunocompetent tuberculosis (TB) patients and the major cause of mortality and disability in developing countries. This is a case series of varied age groups presenting with multiple intracranial tuberculoma and their diagnostic challenge. Tuberculosis is endemic in India, Central nervous system tuberculosis (CNS) accounts for approximately 1% among all the CNS infections. [1] In this report, we present to you a series of multiple intracranial tuberculoma patients who presented in varied forms. The diagnosis of intracranial tuberculoma remained elusive in our patients due to a variety of confounding factors such as the lack of definitive tubercular symptoms, altered mental status, unclear meningeal signs among the patients & young and immunocompetent individuals. Our case report attempts to provide a glimpse into the diagnostic suspicion for these patients by highlighting details in the history and severity of symptoms at presentation co-relating with laboratory and radiological findings. Initiation of anti-tubercular therapy immediately after diagnosis led to significant symptomatic improvement. In conclusion, this case series seeks to highlight the wide-ranging presentations of intracranial tuberculosis and the high index of suspicion that must be exercised to suspect and diagnose this entity among all immunocompetent & immunocompromised individuals & a greater emphasis should be made on history and clinical examination by physician for early diagnosis and treatment which results in reduced morbidity and mortality.

Keywords: NIL

INTRODUCTION

Globally the burden of tuberculosis is rising both as primary pulmonary disease and extra pulmonary disease. World health organization states approximately 10 million people were affected by TB and tuberculous Meningitis (TBM) accounted for 5.2% of cases of exclusively extra-pulmonary disease and 0.7% of all reported cases of TB.[2] Recent studies suggests approximately 10% of all patients with tuberculosis caused by mycobacterium tuberculosis develop CNS involvement and increasing the prevalence of tuberculosis within the community. Central nervous system (CNS) involvement of tuberculosis is diagnostic challenge on presentation and treat. It is associated with high risk of morbidity and mortality if not diagnosed and treated adequately early in the disease course, as late presentation of CNS tuberculosis and treatment are associated with poor outcomes. CNS tuberculoma should to be considered in patients even if there is no evident history of Koch's contact.Therefore emphasis on endemicity, detailed history and clinical examination by the physician should be taken in to account with the differential diagnosis.

CASE REPORTS

Case 1

Aims and Objectives

Early Identification and Treatment

A17 year old female, Ms. (A B) presented to Medicine OPD, with chief complaints of headache since 1 month, associated with a low grade fever and multiple episodes of vomiting since 1 week. The headache was insidious in onset, gradually progressive, globally located, associated with vomiting (non-projectile, non-bilious, non-blood stained), and paresthesias over back and bilateral upper and lower limb. The patient also gave history of one episode of syncope. Fever was acute in onset, present since 7 days, intermittent in nature, low grade and associated with an evening rise of temperature, not associated with chills and rigors. Patient also reported history of weight loss of 2 kgs, and associated appetite loss. The patient also reported having sustained a fall 1 month ago, and brief loss of consciousness. In view of mild traumatic brain injury, patient was subjected to a CT scan The patient was hence on a course of anti-epileptic drugs (Phenytoin). Clinical examination revealed a febrile patient, with no focal neurological deficits.. History of consumption of Phenytoin tablets (AED) post TB for a period of 3 months (Completed course 1 month prior to presentation to the OPD). On evaluation, patient was febrile (100F), had a heart rate of 80 bpm, RR of 20 cycles per min, BP 110/80 mm of Hg, Saturation on room air 100%. Systemic examination revealed no abnormal findings. MRI brain features suggestive of hyper intensity noted in bilateral frontal lobes with ex-vacuodilatation of frontal horn of right lateral ventricle suggestive of encephalomalacia. Multiple scattered rounded intra-axial hyper intense lesions noted in bilateral parietal temporal occipital frontal lobe. Bilateral cerebellar hemisphere & left putaman with surrounding vasogenic edema. Post contrast peripheral ring enchancement is noted all around the lesion. cerebrospinal fluid analysis for the patient suggestive of lymphocyte predominance, with low sugar. A cerebrospinal fluid analysis for the patient revealed definitive diagnosis- csf volume 2cc,colourless, clear in appearance, tlc p 55%, L45% csf protein 465 sugars 12. genexpert examination for ruling out mdr resistance, however, Genexpert revealed tuberculosis (low). Patient was started on anti tubercular drugs and was discharged to follow up opd. Regular follow up and 6 months later a repeat MRI Brin was performed which revealed resolution of ring enchanting lesions.

Case 2

A 36 years woman was admitted to MGM hospital, complaining of chronic headache since last two months intermittent and frequent visits to the physician for her complains. The patient had history of intake of painkillers for history of headache. On examination, patient had low-grade fever, blood pressure was 100/70 mmHg and her pulse rate was 80 beats per minute & had neck stiffness, with positive Kernig's sign and Brudzinski's signs and lateral gaze palsy of the right eye. On systemic examination no signs of pleural effusion or added sounds in the chest, no lymphadenoathy on palpation & within normal limits. Blood & CSF nvestigation were as follows

BLOOD	CSF FLUID
Hb -11.8	
TLC 8800	Sugar 14
Plt 2.19	protein 278
ESR 40	Ada 6.2
CRP 160.	Albumin 0.06
Creat 0.8	total cells 330
Na 134	lymphocyte 55
K 4.2	neutrophil 40

MRI brain revealed multiple tuberculomas in frontal, parietal and occipital region of brain parenchyma with perilesional vasogenic edema. Patient was started on Anti tubercular drugs (ATT) and a course of steroids. With the treatment onset there was resolution of her symptoms and continued a course for a period of 10 months inclusive of continuation of intensive and continuation phase of (ATT). Follow up regular 1 monthly to opd with LFT reports was advised and neurological examination was performed and a repeat scan revealed resolution of ring enchancing lesions.

Case 3

40 years old male was bought to MGM hospital with complaints of acute onset of severe headache associated with vomiting early mornings, fatigue and loss of appetite with approximately weight loss of 3kg in last couple of months. History of previous

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admission 3 months ago to a local hospital and diagnosed with right side pleural effusion and thoracentesis was performed, however pleural fluid examination was not done at his local hospital. On examination he appeared febrile, tachycardic, BP 100/70mmhg. On systemic examination, agitated, altered and an episode of seizure was noted on presentation to the hospital. On investigations, his chest X-ray revealed haziness on bilateral lower zones and routine blood investigations were within normal limits. In the pandemic of corona virus, patient was admitted in isolation icu due to fever spike and resulted RTPCR negative and shifted to Medicine ward for further management. CT brain was suggestive of dilation of lateral ventricle. MRI contrast Brain revealed thick ring enhancement & lesion in high parietal region showing conglomerated ring enhancement. Clinical and radiological evidence was giving suspicion of meningitis and ring enhancing lesion may result in various differentials like neurocysticercosis, tuberculoma or toxoplasmosis. Cerebrospinal fluid (CSF) examination was per- formed total cells 30, protein 374.2 mg/dL, CSF sugar/random blood sugar 74.2/108 mg/dL, and acid-fast bacilli (AFB) stain negative, ADA 3.9 and started on anti tubercular management with (ATT) FDC category with steroids and physiotherapy was started.

Discussion

Epidemiology

Nearly 10-15% of majority of cases of tuberculosis involve extrapulmonary tuberculosis without pulmonary involvement. CNS tuberculosis accounts for 1% of all tuberculosis cases. Tuberculoma account for 0.2% of all biopsied brain masses [3]. Tuberculosis involves the central nervous system, a source of morbidity and mortality, forms 5-10% of the disease associated with tuberculosis[1] Central nervous system tuberculosis may present in different forms as follows meningitis, spinal tuberculosis, tuberculoma, abscesses, cerebritis or miliary tuberculosis [6].

Risk factor

Tuberculosis of lung accounts for nearly 40-60% cases of CNS dissemination and result in tuberculoma [3]Immunocompromised patient are highly suspectible to infections like human immunodeficiency virus (HIV) have 5 times greater risk of tuberculosis than a immunocompetant individual. Immune deficiency is a risk factor for acquiring tuberculosis (TB) patients on immunosuppresions, post organ transplant, neoplasms[8].

Clinical features

Tuberculoma can present in a varied presentations with symptoms of acute or chronic headache, seizure, with or without fever, facial weakness and palsies, altered mental status, vomiting which are present in our patients among above mentioned cases[2]. Tuberculoma results in raised intracranial tension causing compression of the surrounding parenchyma.

Diagnostic Features of Multiple Intracranial Tuberculoma & Tubercular Meningitis

• **Cerebrospinal Fluid**	• **Clinical**	• **Clinical**
1) lymphocytosis	1) fever	1) fever
2) Low sugar	2) Headache acute / chronic	2) Headache acute / chronic
3) CSF AFB positive	3) Vomiting	3) Vomiting
4) Increased proteins	4) Altered sensorium or neurological deficit	4) Altered sensorium or neurological deficit
5) Adenosine	5) Seizure	5) Seizure

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Diagnosis

CNS tuberculosis is challenging diagnosis in the patient who shows no clinical signs of systemic involvement other than CNS[10].In a study by Schwartz et al, the most common reason for multiple ring-enhancing lesions was considered neoplastic, however in a developing country like India infec-tious diseases are frequently witnessed [10].

- Radiological investigations like CT and MRI brain contrast showing ring enhancing lesion with intense vasogenic oedema.

-Lumbar puncture showing lymphocytic predominant, acid fast bacillus present on CSF fluid.

-Brain biopsy with histological evaluation.

The differential diagnosis for an intracranial tuberculoma includes primary brain malignancy (high-grade astrocytoma or medulloblastoma), metastatic lesion, sarcoidosis, toxoplasma, granulomatous diseases, and fungal infections.

Treatment

Anti tubercular drugs (ATT) remains the main stay of treatment, ATT i.e.Initiation phase: 2 months

Isoniazid (4-6 mg/kg, 300 mg) Rifampicin (8-12 mg/kg, 600 mg) Pyrazinamide (20-30 mg/kg, 1600 mg) Streptomycin (12-18 mg/kg, 1000 mg) Continuation phase: 4-7 months Isoniazid (4-6 mg/kg, 300 mg) Rifampicin (8-12 mg/kg, 600 mg). World health organisation (WHO) has put cranial tu-berculosis treatment in the type 1 category of the regimen, recommending a initiation phase of 2months and continuation phase of 6 months, studies have revealed a 12 month course helps in pre-venting the residual neurological deficits. Systemic steroids are recommended in the regimen how-ever its role is unproven. Surgery is recommended in cases of large lesions causing compression. Clinical recovery begins within weeks and takes months 9 to 12 months for resolution of mass ef-fect and lesions in brain parenchyma.

Prevention

Bacillus calumet gurine (BCG) is a live vaccine that may cause serious infections in immunocompromised patients, and is not recommend in HIV infection, who are at greatest risk for tuberculosis. BCG vaccination has role in prevention severe disseminated disease in young children.

Conclusion

In our cases, the clinical and radiological features combined with the classical CSF findings weresuggestive of tuberculous etiology. Conservative line of management with ATT and ster-oids is very effective, safe, cheap and gives a good prognosis and resolution of symptoms. New techniques of diagnosis like PCR assay, MR spectroscopy newer techniques for diagnosis.[1] Early diagnosis and treatment is main stay as delay is strongly associated with death and empir-ical ATT should be started as soon as possible[3]

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