EXTENSIVE CENTRAL NERVOUS SYSTEM TUBERCULOSIS IN AHYPERTENSIVE, IMMUNOCOMPETENTPATIENT: DIAGNOSTIC CHALLENGES AND IMAGING REVIEW. A CASE REPORT

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ABSTRACT

Extensive central nervous system tuberculosis is rare especially in immunocompetent adults. This is a case of a 43-year old female hypertensive, HIV-negative patient, who presented with on and off throbbing headache, dizziness and occasional blurring of six months. No other associated systemic symptoms. General, central nervous and other systemic examinations were remarkably within normal limit. Craniocerebral computed tomography revealed features of widespread central nervous system tuberculoma. Patient was successfully treated with directly observed anti-tuberculosis short course treatment for six months.

Keywords: Hypertension, Central Nervous System Tuberculoma, Craniocerebral Computed Tomography, HIV-Negative, Directly Observed Anti-Tuberculosis Short Course Therapy

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INTRODUCTION

Extra pulmonary TB infections commonly include the lympnodes, pleura, osteoarticular, central nervous system, although any organ can be involved, [1]and its risk also increases with advancing immunosuppression.[1] Central nervous system tuberculosis (CNS TB) is a highly devastating form of tuberculosis, which even in setting of appropriate anti-tuberculous therapy leads to unacceptable levels of morbidity and mortality.[2]Involvement of the CNS is seen in approximately 5% of patients with tuberculosis, however its prevalence is greater in immunocompromised up to 15% in AIDS related TB.[3]

CASE REPORT

A 43yr old female house wife presented with six months' history of on and off throbbing headache, more in the occipital region, that radiated to involve the whole head with no known relieving or aggravating factors. There was history of dizziness which was more on standing often relieved on lying with occasional blurring of vision. No associated history of vomiting, photophobia, seizures, tremors, weakness of the limbs, loss of balance or loss of consciousness. The symptoms did not prevent her from sleeping and her normal activities. No

ear discharge, no history of trauma to head in the past. She admitted history of low grade intermittent fever without chills and rigors initially at the onset of headache, but has stopped. No cough, difficulty in breathing, weight lost, drenching night sweat or history of sustained contact with an adult with chronic cough. No other systemic complains. She has received analgesics for the headache and some other drugs for dizziness and anti-migraine treatment for months in some clinics and our general outpatient clinic without significant improvement before being referred to us.

She was diagnosed hypertensive 3yrs ago and has been on anti-hypertensive drugs. She is not a known diabetic and asthmatic. She is the only wife to her husband with eight children, all alive and well. She has cat as pet for many years.

General, central nervous and other systemic examinations were remarkably within normal limit. Her BP was 130/80 mmHg. Fundoscopy was normal.

Craniocerebral computer tomography (CT) scan revealed*:* wide spread hypo and hyperdense masses of variable sizes in both cerebral and cerebellar hemispheres which showed homogeneous enhancement, other lesions show liquid centre necrotic lesion with ring enhancement (about 10 lesions identified in various locations of the brain).There was associated variable pre-lesional edema with mass effect. No meningeal enhancement noted in the cerebral, cerebella convexities and the basal cisterns, there is mild ventriculomegaly, no area of cerebral infarcts noted (Figure: 1). The CT findings were strongly suggestive of widespread CNS TB particularly tuberculoma. However, other differentials were considered including brain toxoplasmosis and metastatic brain lesions, which added to the diagnostic challenge.Chest and abdominalcomputed tomography, bilateral mammograms did not reveal any possible occult primary malignant source.

Toxoplasma IgG result: [Quantitative =0.71µ/ml (Normal<4.01iµ/ml)
Qualitative = Negative]. Hb=13.3g/dl, WBC: 5.4 X 10^9/L [Differential – Neutrophil – 68.1%, Lymphocytes 25.4%, Others – 6.5%]. ESR =85mm fall in hr. Mantoux test =15mm in diameter (reactive). HIV I & II = Negative.

Diagnosis of CNS TB was then made and patient was commenced on directly observed anti-tuberculosis short course (DOTS) therapy for her age and weight viz: Rifampicin, Isoniazid, Ethambutol, Pyrazinamide with tabs Dexamethasone 4mg twice daily for one month and continued her anti-hypertensive drugs Atenolol 50mg daily and Bendrofluazide 2.5mg daily. One month later, the patient had improved remarkably with remission of her symptoms. Dexamethasone was then stopped and she continued with DOTS treatment and her other medications. After six months, the patient was totally symptom free. Computer tomography scan was repeated and there was total clearance of all observed lesions in the pre-treatment CT with no single lesion identified (Figure: 2).

DISCUSSION

Central nervous system TB results from hematogeneous spread, direct rupture or extension of subependymal or subpial focus and may be located in the meninges, brain or spinal cord.[3]It presents in various forms including tuberculouscerebritis, and military tuberculosis.[3]Clinically adults with tuberculosis meningitis often present with fever, headache, neck stiffness, seizures, behavioral changes and altered consciousness.[3]Whereas those with tuberculoma and abscess depend largely on the locations,they clinically present with signs of raised intracranial pressure viz: headache, seizure, papilloedema, with the pace of

symptoms development measured in weeks to months.[3]Our index patient is middle age female HIV- negative presenting with persistent headache, dizziness ranging for weeks to months, no symptoms or signs of meningitis thereby clinically favoring tuberculoma and abscess as against TBM.

The contrast enhanced craniocerebral computed tomography scan of the patient revealed: were strongly suggestive of widespread CNS TB. Toxoplasmosisand metastatic brain lesions which could havesimilar radiological appearance to tuberculoma were excluded from laboratory investigations. It is well known that, cerebral metastasis occur in 20% of all cancer patients.[4]On cross sectional imaging i.e CT scan, MRI metastatic lesions are commonly seen at grey white matter interface, and they are associated with disproportionately vasogenicperilesional edema considering their size,[4] and there is almost always an identifiable primary source.

Toxoplasmosis usually presents with multiple ring enhancing lesions which can be similar to metastasis and tuberculoma but CNS is only involved in humans with immature immune system (as in fetus) or with compromised immune system as in immunosuppression or AIDS.[4] Toxoplasma antigen was negative in this patient so also non-reactive for HIV I & II.

The hematological parameters of full blood count (FBC), Erythrocyte Sedimentation Rate (ESR) may be nonspecific but increase ESR suggest a chronic disease process, the ESR of 85mm fall/hr, is supportive of chronic disease process and tuberculosis inclusive.

Mantoux test is a sensitive but non-specific in the diagnosis of active tuberculosis. The positive cut-off of 10mm in a person without BCG and 15mm with previous BCG is appropriate.[5]Theinterpretation of Mantoux test needs to be correlated to the patient's clinical context. Mantoux test may have a role in assisting extrapulmonary tuberculosis in children.[5] This patient has a Mantoux test reading of 15mmafter 72hours which in her context of suspected tuberculoma from CT scan is highly suggestive of active TB. Many patients with active TB may have negative tuberculin skin test (Mantoux test),beacause they have a greater number of suppressor monocytes and lymphocytes in their peripheral blood.[6]This fact, however, does not prove that T-cells within tuberculous lesions are suppressed. In the peripheral blood of such patients, mononuclear cells produce transforming growth factor-beta (TGF-β) and IL-10, which are, at least in part, responsible for the immunosuppressive effects on tuberculin sensitivity.[7]

After treatment tuberculoma can completely resolve, however calcification is seen in up to one fourth of cases and is identified most clearly on CT, but in this patient after six months of antituberculous treatment there was complete remission of all identified lesions, not even calcification seen.

CONCLUSION

This case has further broughtto the fore that CNS TB, though rare and devastating, if timely diagnosed and appropriate treatment instituted, can be treated completely without any trace of the disease or its associated sequele. It has also highlighted the critical role of imaging in diagnosis and follows up of such disease entity.

CONFLICT OF INTEREST: None

SOURCE OF SUPPORT: None

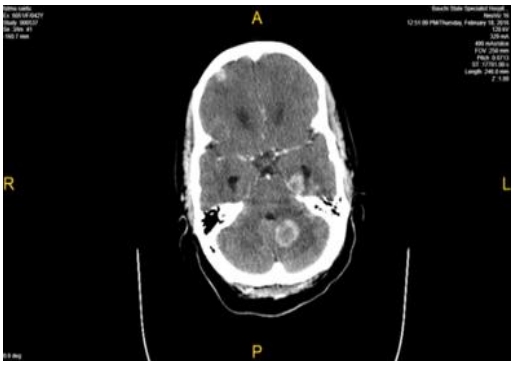

Figure: 1

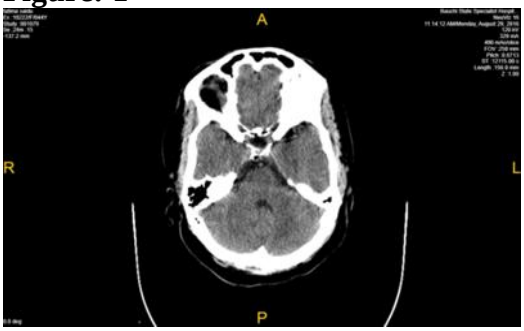

Figure: 2

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