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A Case Report Of Cns Tuberculoma In A Young Female Presenting As A New-Onset Seizure Disorder

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Abstract
CNS tuberculoma, which is an uncommon clinical manifestation of tuberculosis (TB), still remains a problem of delayed diagnosis and treatment. This case report is supposed to present the comprehensive profile of the diagnosis and treatment of the CNS tuberculoma. The brain or spinal cord CNS tuberculoma is caused by hematogenous spread of Mycobacterium tuberculosis bacteria, that results in the formation of a cluster of granulomatous lesions. The CNS tuberculoma patients can have a wide variety of neurological disorders, including headaches, seizures, focal deficits, a change in the mental status. The case stated shows a 26 year old girl who has got CNS Tuberculoma and suffered a recent seizure.

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I. Introduction

CNS tuberculoma is more rare and can be life-threatening compared to common uncomplicated tuberculosis. The CNS is the target if TB virus gets into the brain or spinal cord.Tuberculomas, which equate to granulomas or nodules that grow after exposure of the central system to Mycobacterium tuberculosis, are the bacteria that cause TB, and they may develop anywhere within the brain or spinal cord varying in size from few millimeters to several centimeters. [1] Tuberculomas are most commonly seen in places where tuberculosis is widely prevalent The regions can harbor tuberculomas accounting up to 50% of intracranial masses. India and parts of Asia are considered as major areas where tuberculomas have been recording. They are seen most often

as single supratentorial lesions in newborns and especially in older children. Different from adults, in adults subtentorial lesions are more common. [2]

Generally, CNS tuberculosis are usually caused by the hematogenous spread of TB from a primary site of the infection at another pulse portion of the body like the lungs. These, however, can also be caused by reactivation of the latent TB infection if that is the case. Typically, CNS tuberculomas can cause a collection of symptoms that vary in their severity according to the size, location, and inflammatory process. A wide array of symptoms is associated with CNS tuberculoma which manifest themselves as a headache, seizures, focal neurologic deficits, mental status changes and symptoms of increased ICP.[3] Diagnosing CNS tuberculoma can be a real challenge because some of its symptoms are similar to signs of other intracranial conditions, such as brain tumors or abscesses. Diagnosis often involves a combination of clinical evaluation, neuroimaging studies (such as magnetic resonance imaging or MRI), cerebrospinal fluid analysis, and sometimes brain biopsy.[4]

II. Case Presentation

26 year old female was brought to the ER with complaints of involuntary movements involving both arms and legs lasting for 2 minutes associated with uprolling of eyes and post-ictal confusion which lasted for about 5 minutes. Patient had history of dry cough for 1 month and weight loss for 2 months. Patient also had history of depressed mood-on and off for the past 2 months. The patient does not have a history of recognized medical conditions such as diabetes, hypertension, TB, thyroid, or seizures. On examination- patient was consious, oriented and afebrile. Patient was vitally stable. During the systemic examination, the following sounds were detected: soft, non-tender abdomen; normal vesicular breath sounds; no additional noises; and normal cardiac sounds (S1 and S2) on the cardiac ultrasound. CNS- GCS- E4V5M6, normal tone on both limbs, bilateral proximal and distal upper limb power –5/5, bilateral proximal and distal lower limb power-5/5 with no cerebellar signs or cranial nerve palsies, plantar-bilateral flexor. Chest X-ray showed bilateral upper and lower zone non-homogenous opacities. CBC and peripheral smear showed features of microcytic hypochromic anemia with neutrophilia, ESR-65mm/hr, CRP-negative and normal LFT, RFT and serum electrolytes reports. MRI Brain with MRA/MRV was done which showed multiple small lesions of varying sizes distributed in bilateral cerebral hemispheres along the cortical surface with significant peri-lesional edema suggestive of active granulomatous infective etiology-possibly tuberculous. Neurologist opinion was taken- advised to start the patient on IV anti-epileptic and to do EEG and MRI Brain with contrast. EEG was found to be within normal limits. MRI Brain with contrast showed multiple (>20) T2 hypointense nodular lesions seen in bilateral cerebral hemispheres and left thalamus (with varying degrees of perilesional edema) with all lesions showing ring-enhancement – suggestive of active tuberculous infection. Neurology review was obtained – advised to start the patient on a 5 day course of IV corticosteroid and Anti-Tubercular drugs. CT Chest was also done which showed nodular patchy opacities seen in right upper lobe, multiple confluent parenchymal nodules seen in bilateral upper lobe, right middle lobe, lingula and bilateral lower lobe – suggestive of active tuberculous infection. Pulmonology opinion was obtained -advised to continue ATT and to follow up regularly. The 5 days of IV corticosteroid therapy was completed following which patient improved symptomatically and was discharged with advise to continue ATT medication and was advised regular follow-up.

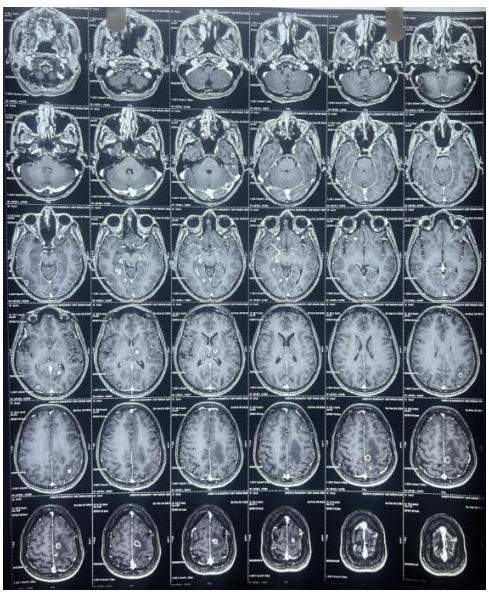

Fig 1- MRI Brain with contrast showing multiple nodular lesions with ring-enhancement

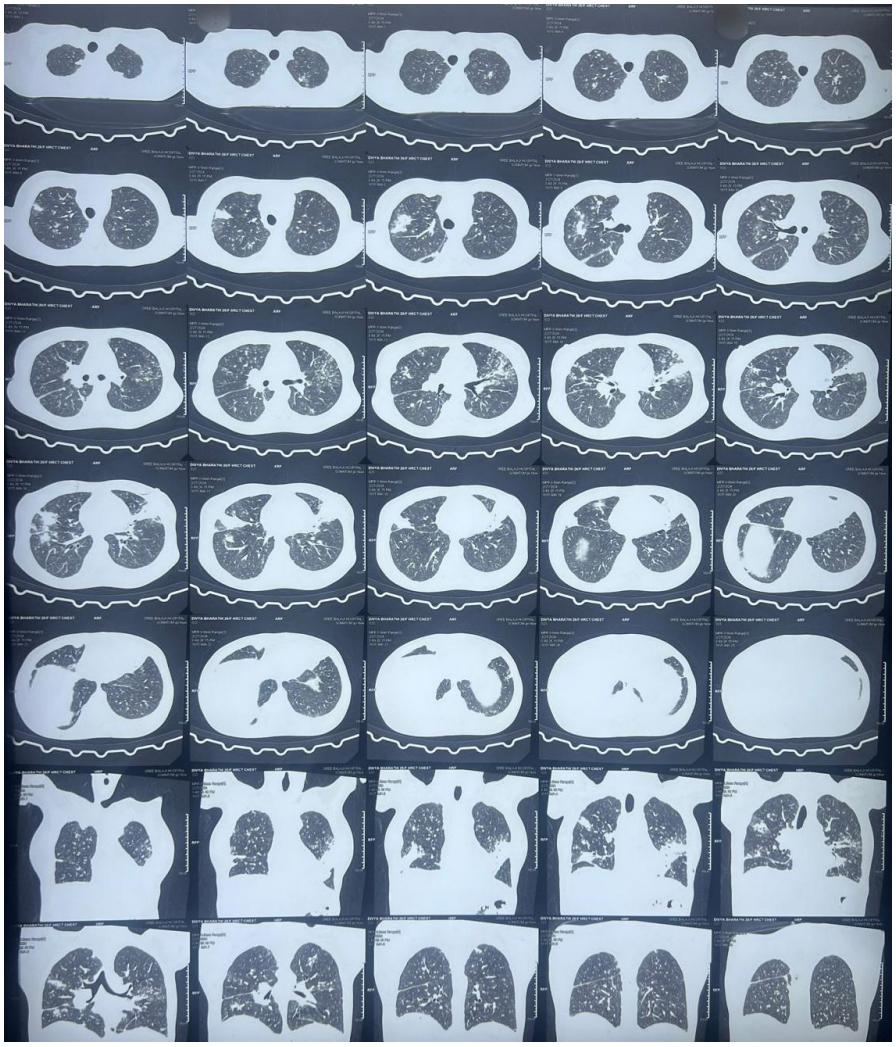

Fig 2- CT Chest showing multiple confluent parenchymal nodules in both lungs

III. Discussion

Mycobacterium tuberculosis may cause meningitis, TB, spinal arachnoiditis, and transverse myelitis, among other infections of the central nervous system (CNS).[5] Multiple tubercular loci (tubercles) are developed within the brain, meninges, or the surrounding bones of the cranium during the bacteraemia that accompany the primary infection or late reactivation tuberculosis (TB).[6] Cumulative granulomatous foci formed by merging tubercles contracted during disseminated bacillemia are known as tuberculomas. Although the spinal cord is another possible site for tuberculomas, the brain is the most typical site of occurrence [7]. Even when there is no inflammation of the meninges, tuberculomas may grow to a large size and cause no symptoms at all. Radiographic imaging may reveal subclinical tuberculomas, either one or several, in cases of tuberculosis meningitis. Even when TB meningitis is not present, tuberculoma may nevertheless cause a noticeable mass lesion in the brain.[8] Headache, seizures, progressive hemiplegia, and/or indications of increased intracranial pressure characterize this presentation, which is more prevalent in endemic regions and usually affects children and young adults. As a consequence of the immune system's reaction to dying Mycobacterium TB germs, tuberculomas may occur even with effective antituberculous treatment.[9] Whether one or many are present, tuberculomas appear on radiographs as distinct, ring-enhancing brain lesions bordered by perilesional edema. If pertinent clinical and epidemiologic factors are present, along with typical radiographic findings, a presumptive diagnosis can be made. The intensive-phase four-drug regimen for adults with central nervous system tuberculosis (CNS TB) that is not known or suspected to be drug resistant consists of isoniazid, rifampin, pyrazinamide, and a fourth agent given daily for two months. During the continuation phase, patients are prescribed isoniazid and rifampin to take daily for a duration of 7 to 10 months.We get follow-up imaging at two weeks, one month, three months, and six months of treatment for patients whose lesion is not in a crucial position and who have little to no persisting neurologic symptoms [10].

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Ethical consent
No ethical issues in this study

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Conflict of interest
There is no potential bias on the part of the authors.

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