

Isolated Brain Stem Tuberculoma: A Case Report and Review of The Literature

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ÖZET

İzole beyin sapı tüberkülozu: Bir olgu sunumu ve literatürün gözden geçirilmesi

Tüberküloz vakalarının yaklaşık %5-10'unda beyin ve sinir sistemi tutulumu görülür. Hematojen yayılıma bağlı merkezi sinir sisteminin tutulduğu tüberküloz nadir değildir. Merkezi sinir sisteminin etkilenmesi menenjit, soliter tüberküloz, apse, infarkt veya milier parankimal hastalık olarak karşımıza çıkabilir. Bu olgu sunumunda, izole pontin tüberkülozu nedeniyle takip ettiğimiz, genetal tüberküloz eşanlı 17 yaşındaki kadın hasta literatür verileri eşliğinde tartışılmıştır.

Anahtar kelimeler: Merkezi sinir sistemi enfeksiyonları, beyin sapı tutulumu, tüberküloz

ABSTRACT

Isolated brain stem tuberculoma: a case report and review of the literature

Approximately 5 to 10% of tuberculosis cases involve brain and central nervous system. Tuberculosis involving central nervous system (CNS) due to haematogenous spread is not a rare entity. CNS manifestations may be meningitis, solitary tuberculomas, abscess, infarct or milier parenchymal disease. In this case report, we discuss a 17-year-old female with isolated pontin tuberculoma and comorbidity of genetal tuberculosis and review the literature.

Key words: Central nervous system infectious, brain stem involvement, tuberculosis

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INTRODUCTION

Tuberculosis is an infectious disease which is caused by the pathogenic microorganism Mycobacterium Tuberculosis and a highly prevalent disease in developing countries; however, due to increasing HIV epidemics and migration from third world countries, it is increasingly threatening public health in developed countries as well (1-4). Approximately at 5-10% of tuberculosis cases, brain and nervous system is involved. Involvement of central nervous system (CNS) can be represented as meningitis, solitary tuberculoma, abscess, infarction or miliary parenchymal disease. Intracranial tuberculomas are tumor-like lesions which tuberculous granulation tissue forms in brain parenchyma. Tuberculomas are relatively rare compared to other tuberculosis types. They may cause neurological deficits due to local mass effect. Tuberculomas generally respond to anti-

tuberculosis treatment in 2-3 months (5). Isolated brain stem tuberculomas are rare and makes up 2.5-8% of all intracranial tuberculomas (3,6,7).

Characteristic features of tuberculomas in brain computerized tomography (CT) and magnetic resonance imaging (MR) are not well known. Diagnosis of tuberculoma can be made by histopathology, clinical manifestations, response of neuroimaging to tuberculosis treatment or presence of systemic tuberculosis (4,8).

In this report, it was aimed to present the clinical and neuroimaging findings of a case with concomitant CNS tuberculoma and genetal tuberculosis and to review the literature.

CASE

Seventeen year old, right-handed, married female patient admitted to our neurology emergency unit



Picture 1: In the initial MR imaging, the lesion caused expansion in basis pontis can be seen.



Picture 2: Shrinking of basal pontine lesion after treatment.

with abrupt onset severe headache which is partially responsive to simple analgesics. Headache was localized to forehead and around left eye and was pulsatile;

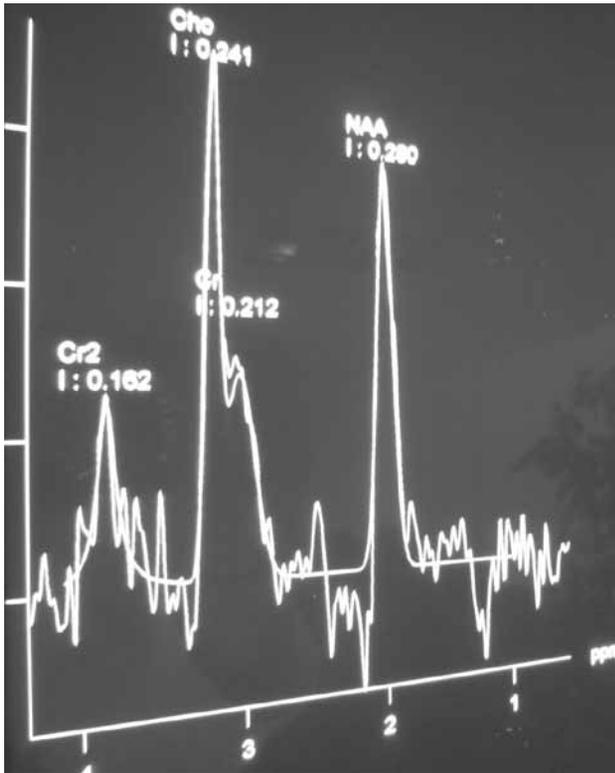
nausea and vomiting, photophobia and phonophobia were not present. In the history, she told that she was evaluated in the gynecology department 2 months ago due to fever, sweating, malaise, abdominal pain and vaginal bleeding and diagnosed as genital tuberculosis, isoniazide and rifampicin was started at that time and her symptoms were significantly relieved. She was from a low socio-economical background and it was learned that her childhood vaccination was incomplete. Her general systemic evaluation was normal. In neurological examination, she was alert and fully cooperated and oriented. No meningeal irritation was found. Eyeballs were in midline, pupillae were isocoric and light reflex was preserved. In left gaze, gaze-oriented nystagmus was present in the left eye and there was medial gaze paresis in the right eye. Fundoscopic examination was normal. Other cranial nerves were intact. Motor functions and cerebellar tests were also normal. Deep tendon reflexes were normoactive. Sensory examination was normal.

Complete blood count, liver and renal function tests, electrolyte levels, erythrocyte sedimentation rate and C-reactive protein were within normal limits. Viral hepatitis markers, VDRL and brucella antibodies were also negative.

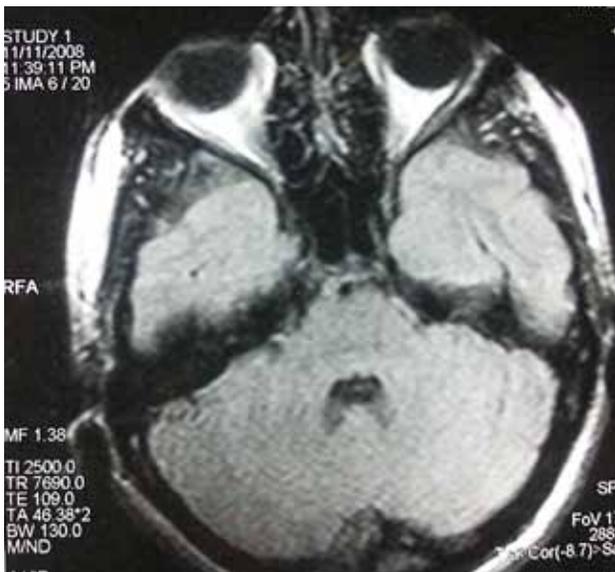
No lesion was found in chest X-ray, thorax and abdominal CT. No lesion could be detected in cranial CT but in cranial MR with contrast, at brain stem in pontine level, a hyperintense solitary mass lesion having peripheral edema and absorbing contrast medium was detected (Picture 1,2).

After spinal tap cerebrospinalfluid (CSF) was collected. CSF pressure was increased and CSF was clear and colorless. No cell was detected. CSF protein level was detected 114 mg/dL and glucose was detected 22 mg/dL (concomitant blood glucose was 85 mg/dL). In Gram and Ziehl-Neelsen staining of CSF, no microorganism was detected. Tuberculosis PCR was found positive but CSF culture was found to be negative.

For the differential diagnosis of the pontine mass, MR spectroscopy was done (Picture 3). Our neuroradiology unit reviewed the scan and due to choline peak, tumoral lesion such as a glioma was



Picture 3: Significantly increased Cho/Cr ratios in MR spectroscopy can be seen



Picture 4: Basal pontine lesion disappeared at MR imaging taken 9 months after the treatment.

thought (Picture 3). Cho/Cr ratio was 3.2 and NAA/Cr ratio was 2. Neurosurgery department evaluated the patient and decided to follow-up the lesion by

imaging. Due to current position of the lesion and risk of complication, biopsy was not thought of.

Patient was followed in our inpatient clinic and diclofenac tablets were prescribed for her headache. Pyrazinamide was added to the current antibiotic treatment. Headache improved in time and cranial MR with contrast taken one month later, pontine mass shrunk and edema was greatly resolved. In neurological examination, no deficit was found except gaze paresis and nystagmus.

Patient was followed by monthly blood counts and chemistry, ophthalmological examination, cranial MR with contrast and gynecological examination. In cranial MR follow-up, dimension of the lesion decreased and 9 months after her first visit mass was completely resolved (Picture 4).

Clinical history of the patient, examination, neuroimaging follow-up and response to anti-tuberculous treatment suggested that mass in the brain stem was a tuberculoma.

DISCUSSION

Brain stem tuberculoma should be considered in patients living in regions which tuberculosis is endemic such as our country and having a space-occupying lesion in the brain stem. Intracranial tuberculomas are tumor-like masses which are formed by tuberculous granulation tissue and relatively rarely encountered compared to other types of tuberculosis. Tuberculomas are generally found as more than one mass and rarely solitary and when they are too big they may show a mass effect. Early diagnosis and appropriate medical treatment is important to prevent mortality and to reduce morbidity (6). When intracranial tuberculoma is seen with meningitis, it is easier to diagnosis. However, isolated intracranial tuberculomas are generally difficult to diagnose. Radiological imaging is important for diagnosis and following the treatment course. However, there is not a pathognomonic radiological test. In isolated central nervous system (CNS) tuberculomas without a primary source, clinical course is better than CNS tuberculosis accompanied by miliary tuberculosis.

Clinical and spontaneous remissions were reported very rarely (7, 13). In cranial CT, it can be seen as a isodense mass and homogenous or ring-like contrast enhancement can be seen (4,9). In MR imaging (MRI) tuberculomas can be viewed in different forms according to pathological stages. MRI findings change with the caseification of the mass. In lesions with solid caseification, there is an iso or hypointense appearance in T1 and T2-weighted images. Caseified lesions having central liquid appear as hypointense in T1 and hyperintense in T2-weighted images and ring-like contrast enhancement is observed. In non-caseous granulomas, lesion is hypointense in T1 and hyperintense in T2-weighted images with homogenous contrast enhancement (4,8). In MR spectroscopy, in hypointense tuberculomas at T2-weighted images choline, creatine and N-acetylaspartate (NAA) is not seen; this is supporting criterium to differentiate from neoplasms. In neoplasms, it is known that there is characteristically higher choline resonance, variable creatine and NAA. Lesion sites with wide choline-variable creatine resonance may mimic neoplasms in MR spectroscopy. Increase in choline peak in MR spectroscopy of the patient suggests space-occupying lesions such as gliomas. In tuberculomas, histopathological examination revealed high cellularity at solid caseification areas. In these kinds of tuberculous granulomas, due to presence of choline tuberculomas were thought to be related with cellular component (11). Neuroimaging of our patient is an example to choline peak seen in tuberculomas.

In the neuroimaging of the female patient with

genital tuberculosis evaluated for headache, nystagmus and gaze paresis, although MR spectroscopy was harmonious with a tumoral mass, both history of systemic tuberculosis and low CSF glucose level and a positive PCR test, lesion was diagnosed as CNS tuberculoma developed concomitantly with an organ infection. Absence of a cellular reaction in CSF can be explained by denaturation of cells between spinal tap and cell counting or atypical CSF findings during the course of CNS tuberculosis (12). Patient was followed-up by monthly MRI scans and lesion regressed under anti-tuberculosis treatment.

Only neurological findings of our patient were nystagmus in left gaze and medial gaze paresis in the right eye and found interesting as the 4th case with brain stem tuberculoma having gaze paresis in the literature (3,10). Current clinical findings can be explained by its location in the right side of medial longitudinal fasciculus. With anti-tuberculosis treatment paresis regressed in cases reported. However, in our case, although the lesion completely disappeared in imaging at 9th month, gaze paresis remained as a sequelae.

CONCLUSION

We wanted to present our case due to its presentation as a brain stem glioma by clinical and radiological findings but evaluated as tuberculosis by CSF serology and well-responded to anti-tuberculous treatment and being an example of atypical presentation of tuberculosis both clinically and radiologically.

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