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Pulmonary tuberculosis associated with multiple intracranial tuberculomas and tubercular brain abscess

Çoklu intrakranial tüberkülomlar ve tüberküler beyin apsesi ile ilişkili akciğer tüberkülozu.

Öz

"Mycobacterium tuberculosis" yaygın bir hastalık olmasına rağmen, miliyer tüberkülozis, özellikle, tüberkülomlar, nadir görülen patolojilerdir. Miliyer tüberkülozun ve diğer organ tüberkülozlarının tanısı oldukça zordur. Bu makalede artmış intrakraniyal basınç ve solunumsal semptomları olan iki olgu bildirilmektedir. Mikobakterium tüberkülozis tanısı konulan olguların biri beyin tüberkülomu ile ilişkili iken, diğer olguda tüberkülomun komplikasyonu sonucu gelişen apse mevcuttu. Apse gelişimi de konvülsiyona neden olmaktaydı. Olgular tüberküloz hastalığının farklı klinik ve radyolojik bulgularına sahip oldukları için sunulmuştur.

Anahtar kelimeler: Tüberküloz, tüberküloma, apse, konvülsiyon.

Abstract

Although mycobacterium tuberculosis is a widespread disease, miliary tuberculosis especially tuberculomas is a rare pathological condition. The diagnosis of miliary tuberculosis and the involvement of other systems by tuberculosis are difficult. We reported two cases with having respiratory and high intracranial pressure symptoms. They diagnosed as mycobacterium tuberculosis associated with brain tuberculomas, and abscess formation developed as a complication of tuberculomas in one of the patients. The development of abscess formation was the cause of convulsion. The cases were presented due to presence of different clinical and radiological findings on tuberculous disease

Keywords: Tuberculosis, tuberculoma, abscess, convulsion

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Introduction

Although tuberculosis (tbc) remains as a major health problem in developing countries, involving of the Central Neural System (CNS) is still uncommon (1). Hematogenous spread of Mycobacterium tbc from a primary focus, characteristically from the lung may result involvement of CNS as tuberculoma (tbm). It is a rare form of extra pulmoner tbc. Only 1% of patients with tbc develop an intracranial tbm (2). There are no clinical signs and having no specific radiological findings in most intracranial tuberculomas (tbms). Therefore, the imaging features of tbm have been a subject of interest. Many publications on this subject are available in literature (3–6). When intracranial tbm changes into abscess formation, neurological signs and symptoms occur with time.

Intracranial tbms can be present in many different clinical and radiological patterns.

If an intra-axial lesion of brain enhances nodular shape, tbm should be considered in the differential diagnosis (7). However, it may be difficult to differentiate the Magnetic Resonance Imaging (MRI) findings of patients with tbm from those of patients with other infective (fungal or bacterial brain abscess, toxoplasmosis, or cysticercosis), inflammatory (sarcoidosis), or malignant (lymphoma, gliomas) lesions (8–10). In this study, we presented imaging features and clinical findings of miliary tbc, intracranial tbm and abscess formation of intracranial tbm.

Case 1

A 63-year-old female patient was admitted to the emergency department with complaints of speech impairment and difficulty in walking, an increase in headache, loss of weight and appetite in her history. Tbc was not noted in her family history and other system examinations were normal. Hemoglobin was 13 g/ 100 ml, blood leukocyte was 8,300/ml, and erythrocytes sedimentation rate was 90 mm within the first hour. Induced sputum, gastric fluid, direct smear, and tuberculin skin test were negative. The chest x-ray (Fig. 1a) and high-resolution thorax computed tomography (Fig. 1b) showed multiple micro nodular lesions in the lung parenchyma. Brain MRI examination was performed because of neurological symptoms. T1-weighted images (Fig. 2a) showed no abnormal signal intensity on the other hand T2-weighted images showed multiple lesions with hypointense peripheries and central cores of varying degrees of brightness surrounded by high intensity edema. After contrast enhancement, brain MRI revealed multiple enhancing nodules in bilateral deep white matter, subcortical area, pons and cerebellum (Fig. 2b). The patient was diagnosed as miliary pulmonary tbc accompany with involvement of CNS as multiple brain tbms. She was treated with a combination of drugs, including INH, 300 mg/d; RMP, 600 mg/d; ethambutol, 1000 mg/d; and pyrazinamide, 1000 mg/d, for 2 months. Metilprednizolon (1 mg/kg per day, 48 mg/per day) was added the treatment for reducing the neurologic symptoms. It was continued with maintenance dosage for 8 weeks. Brain MRI examination was performed in the second month of the treatment. It revealed that significant reduction in the number and size of the lesions in brain (Fig. 2c). It was thought that the treatment was very effective. Therefore, she was taken long-term anti-tbc chemotherapy.

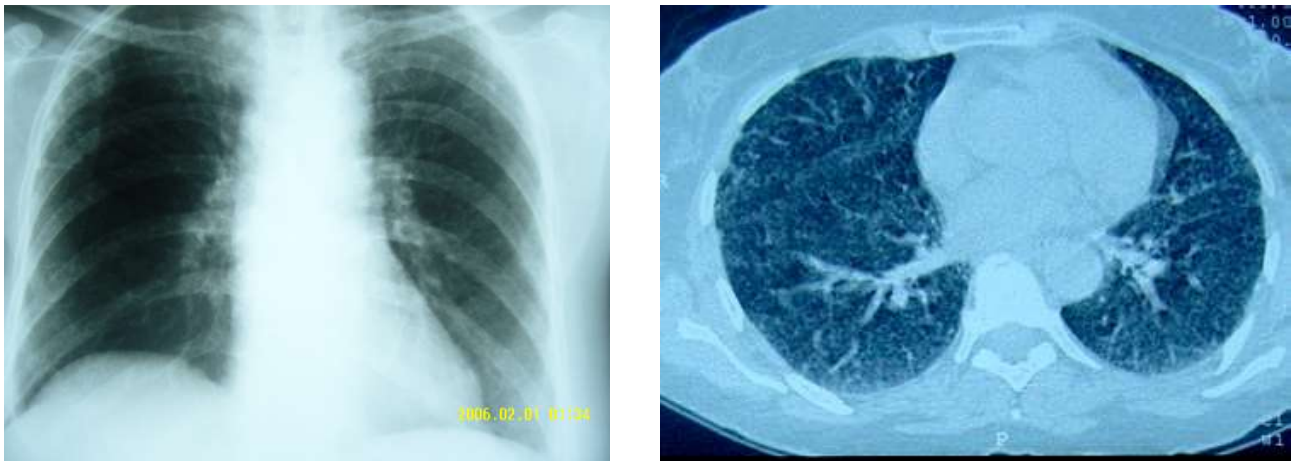

Figure 1. a, b.: Chest X-ray, and high-resolution thorax CT before treatment. Chest X-ray (a) shows evenly distributed, discrete, uniformly sized nodular opacities in both lungs. Similarly High-resolution CT image shows uniform-sized small nodules randomly distributed throughout both lungs, compatible with miliary tuberculosis (b).

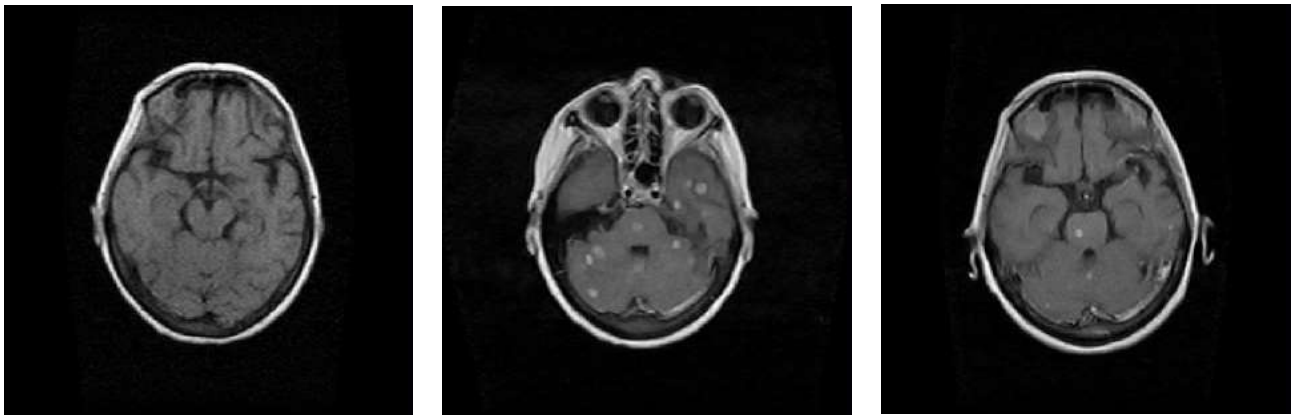

Figure 2. a–c.: Pre-treatment brain axial magnetic resonance images of a 63 year-old-female patient, T1 weighted magnetic resonance image shows anything remarkable (a), contrast-enhanced T1 weighted magnetic resonance image shows multipl small noduler areas of enhancement that are predominantly located at the gray-white matter junction (b). Contrast-enhanced brain MRI taken two months later, number and size of the lesions had a significant decrease (c).

Case 2

A 23-year-old female was admitted to hospital with fever, general weakness, headache, respiratory distress and night sweats lately. Hematological examination revealed an increased sedimentation rate and lymphocytosis. There was no abnormality on physical examination, and induced sputum, gastric fluid, direct smear, and tuberculin skin test were negative. The chest x-ray (Fig. 3a) and high-resolution thorax computed tomography (Fig. 3b) revealed multiple micro nodular lesions in the lung parenchyma. The patient was diagnosed as pulmonary tbc because of radiological findings. The anti-tbc drugs (INH, 300 mg/d; RMP, 600 mg/d; ethambutol, 1000 mg/d; and pyrazinamide, 1000 mg/d, for 2 months) were given. In addition, the oral steroid was added for preventing of pulmonary fibrosis. After treatment protocol was arranged, she was discharged from the hospital. After about two weeks, the patient re-admitted to our hospital by convulsion. She had severe nausea and vomiting in her history when she took oral steroid. It was thought that intolerance of oral steroid might be causing such kind of symptoms. Therefore, oral treatment was changed into parenteral

steroid (dexamethasone 0.5 mg/kg per day). Brain MRI was the best choice for explaining of her convulsion and it showed heterogeneous hypo-isointense on T1-weighted images, and homogeneously hyperintense on-T2 weighted images. Multiple nodular lesions were determined on contrast enhanced MRI examination of the brain. However, one of the lesions had a different character from others. On brain MRI examination, it had significant edema around and a well-enhanced wall on, which were typical for an abscess. Therefore, it was interpreted as the cause of convulsion (Fig. 4). After parenteral treatment of steroid, the patient's seizures decreased gradually and disappeared completely. After six month, it was observed that the abscess were disappeared, and regression in number of tbms on brain MRI.

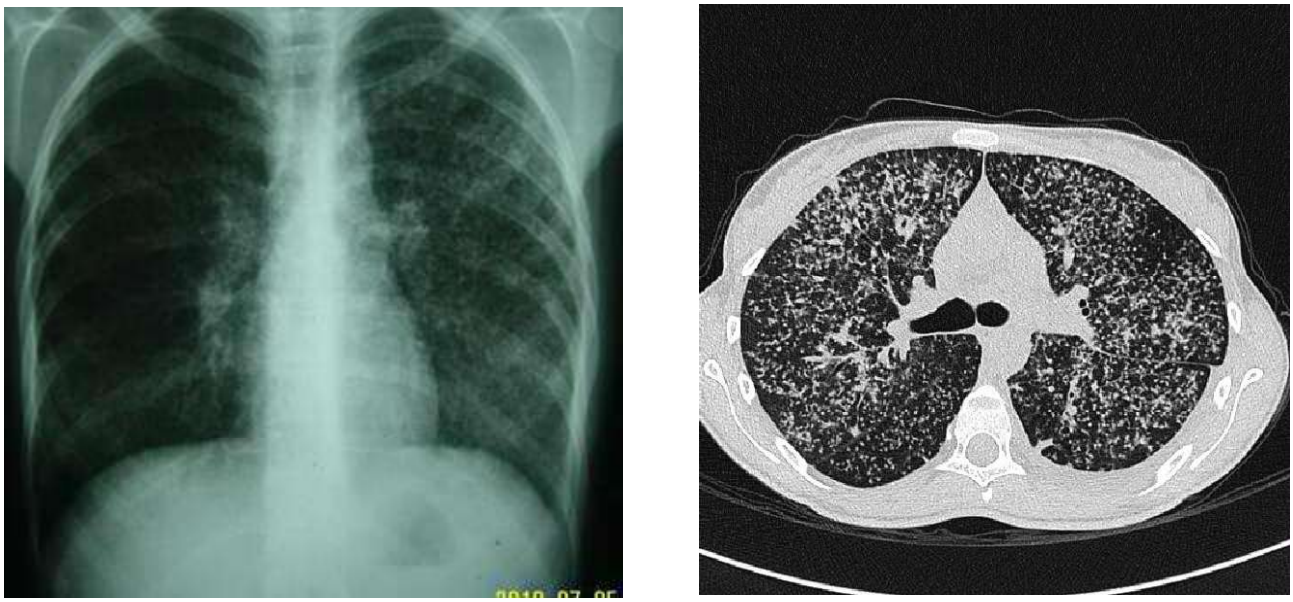

Figure 3. a, b : Posteroanterior chest radiograph shows multifocal masslike airspace consolidation in bilateral lung zones (a). There were wide spread micronoduler hiperdens formations in whole lung in the high-resolution thorax CT (b).

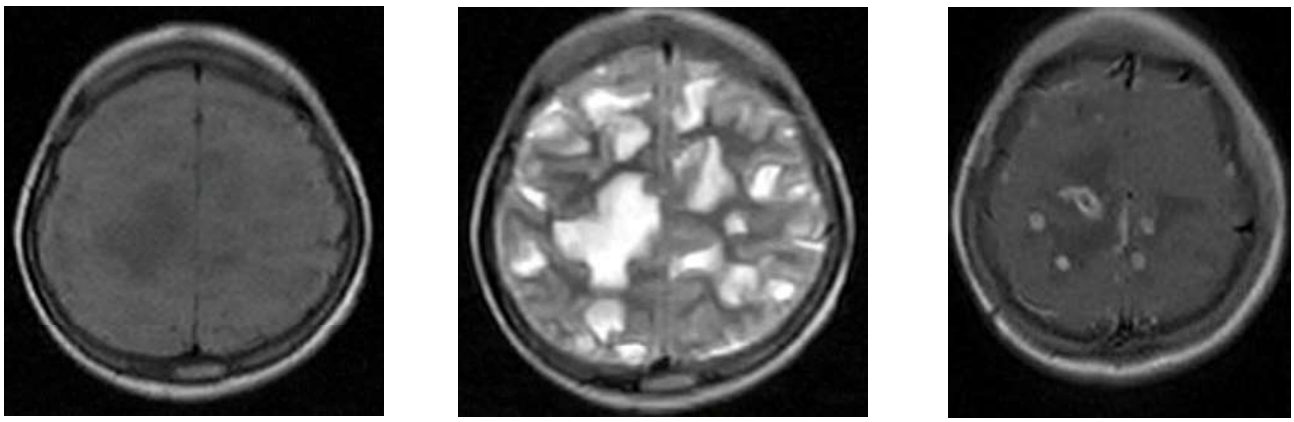

Figure 4. a–c.: Supra-ventricular level, multiple hypointense focus are observed on T1 weighted image without contrast media (a). At the same level, significantly edema observed surrounding area of nodular lesions on T2 weighted image (b), Contrast-enhanced T1weighted brain image, there are multiple contrast-enhancing tuberculoma located in the deep white matter. The lesion in the right cerebral hemisphere, which is typical for tubercum abscess with peripheral contrast enhancement and the presence of peripheral edema (c).

Discussion

Involvement of CNS has frequently been found secondary to tbc elsewhere in the body, particularly the lungs. The presence of tbc elsewhere in the body favors the diagnosis, although its absence does not exclude it (11). The involvement of CNS by tbc occurs often in the form of tbc meningitis or tbm (12). Intracranial tbms are an uncommon form of presentation of the infection by tbc. A non-caseating tbm usually appears hyperintense on T2-weighted and slightly hypointense on T1-weighted images. A caseating tbm appears iso-to hypointense on both T1-weighted and T2-weighted images, with an isointense-to hyperintense rim on T2-weighted images. Tbms on contrast administration appear as nodular or ring-like enhancing lesions (13). The diameter of these enhancing lesions usually ranges from 1 mm to 5 cm. Tbms frequently show various types of enhancement, including irregular shapes, ring-like shapes, open rings and lobular patterns (14). In our cases, all tbms were hyperintense on T2-weighted and slightly hypointense on T1 weighted images. Therefore, brain tbms were interpreted as non-caseos and the diameters of the brain lesions with enhancing were usually smaller than one cm. One of the cerebral lesions in the second case was different from others and it was hypointense on T1-weighted images and hyperintense on T2-weighted images. Peripheral edema was evident around on T2-weighted images. Ring like the one enhancing lesion on axial image was interpreted as an abscess, which was the cause of convulsion.

Involvement of CNS may be seen with pulmonary tbc or not. Even with pulmonary involvement, some patients with CNS involvement may apply to hospital with CNS symptoms or signs without any evidence of pulmonary involvement (7,15). In our two cases, there were either neurological or pulmonary symptoms as fever, cough, loss of weight and appetite, numbness in arms and headache in the early period. However, convulsion occurred during the treatment in our second case.

However, convulsion was occurred as a complication of tbms in the late period of our second case. The early beginning of the therapy is the most important factor affecting the prognosis in brain tbms. The treatment of tbm is primarily medical. Surgery is generally performed only when the diagnosis is in doubt or there is no clinical and/or radiological response after a trial of medical treatment, or in patients with raised intracranial pressure secondary to the lesion or in those who are not responding to medical therapy. The purpose of surgical intervention is to decompress when progressing neurological defects occur clinically (16). Although there is no consensus regarding the duration of treatment, pulmonary and extra pulmonary disease should be treated with the same regimens. However, some experts recommend 9–12 months of treatment for tbc meningitis (17, 18). Many groups have reported effective short-course therapy for intracranial tbms, ranging from 6 to 12 months (19, 20). Our cases were receiving drug therapy for two months. We planned to complete one year of medical treatment. Corticosteroids are routinely used in the management of increased intracranial pressure and of intracranial lesion-associated edema. Moreover, corticosteroids have been shown to have substantial short-term and long-term benefits for patients with tbc meningitis and pericarditis (21). Case reports and series have also shown that corticosteroids may reduce the risk of residual neurological deficits in patients who develop symptomatic intracranial tbms during anti-tbc therapy (22). Steroid is very important for preventing to brain complications during anti-tbc therapy. In our study, steroid therapy and anti-tbc therapy was given in both cases on time. There was no convulsion in the first one. Hoverer, the medical treatment was no effective due to intolerance of oral steroid in the second one. Therefore, oral therapy was changed into parenteral.

Conclusion

It can be conclude that if patient has neurological symptoms, findings in routine chest radiographs can lead us to the diagnosis of tbc. The signal intensity and ring-enhancing pattern of the lesions on brain MR examination may play an important role in distinguishing an intracranial tbm from other similar lesions. Early diagnosis and prompt treatment of tbc without delay can decrease morbidity and mortality rate significantly.

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