A CASE OF DISSEMINATED TUBERCULOSIS EMERGING WITH PULMONARY, EYE AND CENTRAL NERVOUS SYSTEM INVOLVEMENT

PULMONER, GÖZ VE SANTRAL SİNİR SİSTEMİ TUTULUMU İLE ORTAYA ÇIKAN DİSSEMİNE TÜBERKÜLOZ OLGUSU

Songül ÖZYURT (0000-0002-9778-1425), **Neslihan ÖZÇELİK** (0000- 0002 -4672- 6179), **Bilge YILMAZ KARA** (0000- 0003- 2690- 4932), **Ünal ŞAHİN** (0000-0001-6389-3393)

Recep Tayyip Erdoğan Üniversitesi, Göğüs Hastalıkları Kliniği, RİZE

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ÖZ

Tüberküloz, gelişmekte olan ülkelerde hala önemli bir halk sağlığı sorunudur. Eğer tedavi edilmezse, dissemine tüberküloz gibi çeşitli komplikasyonlara neden olabilir. İmmün sistemi normal olan hastada yaygın tüberküloz nadir görülen bir durumdur. Bu olgu sunumunda, normal bir bağışıklık sistemi olan, dissemine tüberküloz gelişen 25 yaşında bir olgu sunulmuştur.

ABSTRACT

Tuberculosis is still a major public health problem in developing countries, although it has a complete treatment. If untreated, it may cause various complications such as disseminated tuberculosis. Disseminated tuberculosis in the immunocompetent patient is a rare condition. In this case report, a 25-year-old case with disseminated tuberculosis who had a normal immune system is presented.

INTRODUCTION

Tuberculosis (TB) is still one of the most important health problems in developing countries. It primarily affects the lungs; however, the involvement of extra pulmonary sites, which have high blood flow such as kidneys, bones, meninges and ocular choroids, are also common [1]. Furthermore, the lympho-hematogenous spread of infection from primary focus to several organs may result in miliary tuberculosis which is rarely seen in an immunocompetent person. This paper reports a case of a 25-year-old immunocompetent patient with miliary tuberculosis with pulmonary, ocular and extensive cranial involvement.

CASE REPORT

A twenty-five-year-old male patient was admitted to the emergency department with confusion, agitation, nausea, and vomiting. The patient's history which was taken from his

family indicated that he has prescribed a standard tuberculosis treatment (four drug regimen) for culture-positive pulmonary TB two months ago. After two weeks of treatment, the patient has quit taking his drugs due to ocular pain. Although the patient was provided with direct observed treatment by his mother, they discontinued the treatment because they thought the ocular pain was a side effect. He had poor oral intake over the last week and he started vomiting for the last two days. Upon physical examination, he was unconscious and did not respond to verbal stimuli. He was afebrile, with a heart rate of 72 beats/min, a respiratory rate of 16 breaths/min, and blood pressure of 110/70 mm Hg on initial examination. His arterial oxygen saturation was 96% while breathing room air. Chest auscultation revealed normal breath sounds. Examination of other organ systems was normal. He had no any sistemic disease, no any drug using history and his laboratory tests for HIV was negative, and immunoglobulin levels were normal. Laboratory results were as follows: leucocytes: 7.11 K/uL, C-reactive protein: 1.83 mg/dl, and erythrocyte sedimentation rate: 40 mm/h biochemical tests were normal. Computed tomography of the chest revealed ground-glass opacities and parenchymal nodules that look like each other at the left apex and diffuse miliary nodules in both lung fields (Figure 1). In the emergency department, the patient was evaluated by a neurologist. The cranial magnetic resonance imaging showed multiple contrast-enhanced cerebral and cerebellar lesions with generalized edema (Figure2). A fundus examination was done because of the pain in the eyes. Both eyes had optic nerve involvement while the right eye also had superior temporal arcuate granuloma. With the current findings, the patient was diagnosed with miliary tuberculosis, ocular and cranial involvement and standard tuberculosis treatment was restarted. The patient's unconsciousness resulting from brain edema responded well to mannitol infusion. Forty milligrams of methylprednisolone daily was added to the treatment. The patient's general condition improved and he was discharged after 20 days of hospitalization. Information on Informed Consent was taken from the patient.

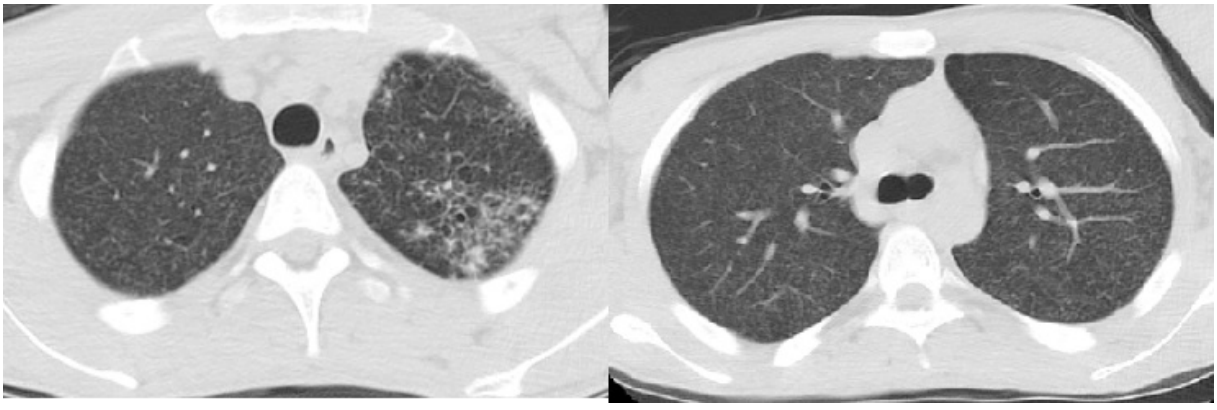

Figure 1. a, b. Chest tomography images show ground-glass opacities and parenchymal nodules that look like each other at the left apex and diffuse miliary nodules in both lung fields.

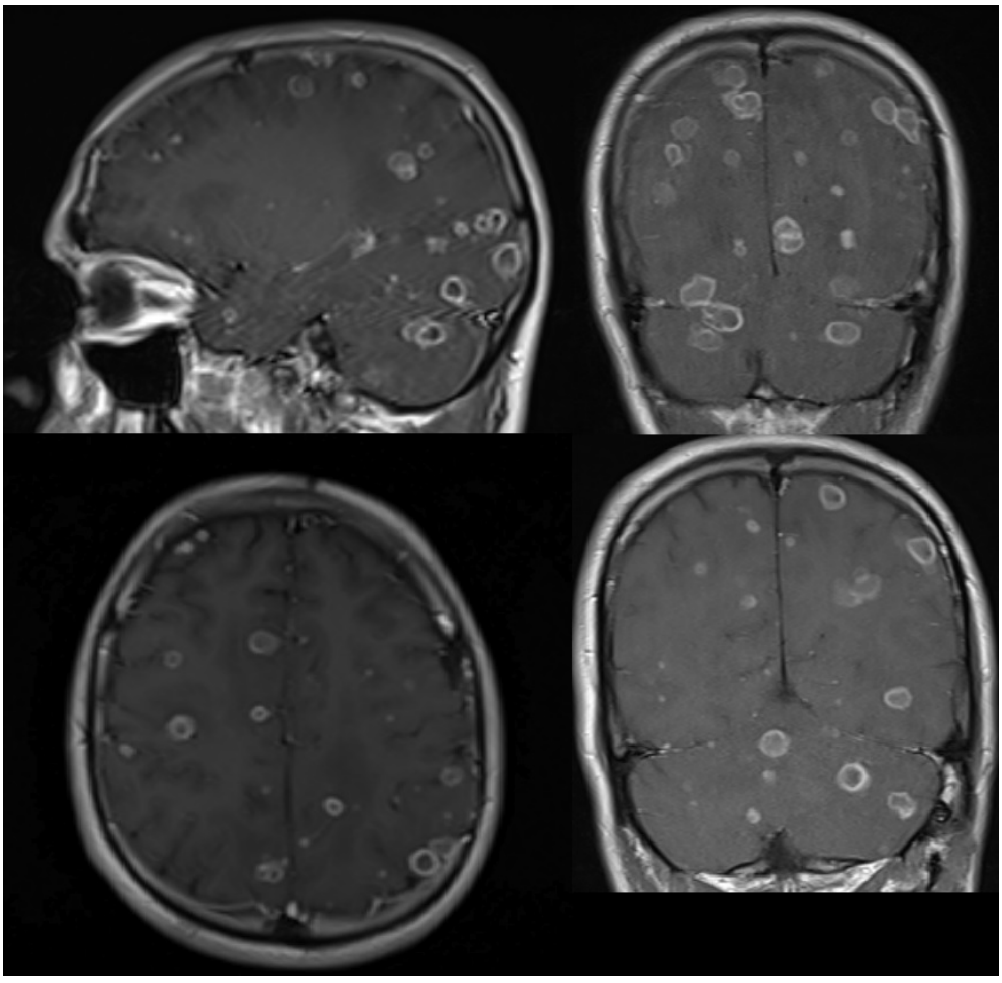

Figure 2. a,b,c,d. Axial (c) and coronal (a,b,d) magnetic resonance images show multiple contrast-enhanced cerebral and cerebellar lesions with generalized edema

DISCUSSION

Miliary tuberculosis is a potentially fatal form of TB caused by the widespread hematogenous dissemination of Mycobacterium tuberculosis bacilli [2]. It may show up during both primary and post-primary tuberculosis. In areas with a high incidence of TB, poor socioeconomic conditions lead to easy dissemination of the disease [2, 3]. In addition, factors causing immune suppression like HIV infections, chronic renal insufficiency and diabetes mellitus can cause miliary tuberculosis. Dissemination is rare in immunocompetent patients [3].

Central nervous system (CNS) involvement may be presented with solitary tuberculomas, meningitis, abscesses, miliary parenchymal disease or infarction [4,5]. Our patient had extensive involvement in the form of cerebral and cerebellar tuberculoma focuses. Intracranial tuberculomas can develop not only due to inadequate treatment but also in a patient with the paradoxical reaction after appropriate and regular therapy [6]. Tuberculomas, often with surrounding edema and contrast enhancement, macroscopically demarcated, often are smaller than 1 cm in size, are gray and hard nodules which may contain acid-fast bacilli in the microbiological examination [6]. If tuberculom opens into

ventricles, bacilli can be detected in the cerebrospinal fluid. Diagnosis of tuberculoma was made with regard to the infiltrative lesions in the left upper lobe, miliary dissemination in both lungs, Mycobacterium tuberculosis growth in the patient's sputum culture and radiological characteristics of the lesions. Histopathologic examination was not necessary since a clinical and radiological consensus over the diagnosis was found. Intracranial tuberculomas may lead to increased intracranial pressure, headache, epileptic attacks and neurological symptoms due to local compression or nonspecific neurological deficits such as confusion, depending on the size and localization [4-6]. The patient's level of consciousness improved after antiedema treatment and medical history which was taken directly from the patient revealed that the patient discontinued taking his medication without consulting a doctor because of headache and eye pain. The fundus examination performed after consciousness improved revealed optic nerve involvement in both eyes and a superior temporal arcuate granuloma in the right eye.

Ocular involvement may occur as a form of extrapulmonary tuberculosis primarily, as well as due to the dissemination of pulmonary tuberculosis by hematogenous spread [7]. The clinical presentations of ocular tuberculosis are anterior uveitis, intermediate uveitis, posterior or panuveitis, retinitis, vasculitis, neuroretinitis, optic neuropathy, endophthalmitis, and panophthalmitis [8]. The choroid plexus which is located in the posterior segment is the most common site of intraocular involvement. In addition, there might be symptoms of choroidal tubercles (small nodules), choroidal tuberculoma (size more than 4 mm), subretinal abscesses, and choroiditis [8]. Choroidal tubercles, which were masquerading as an ocular mass, may arise with tumor symptoms. Diagnosis of ocular TB can be made in a setting when there is a positive tuberculin skin test, active or sequelae lesions specific for tuberculosis on chest radiograph, a growth of Mycobacterium tuberculosis in tissue or fluid cultures from the affected organ, presence of the bacilli in the ocular fluid on direct microscopic examination or detection of mycobacterial DNA with PCR technique. Also, the exclusion of the other uveitis causes is crucial. A positive response to empirical tuberculosis treatment in the presence of supporting evidence is also considered as another approach for diagnosis [9,10]. In addition, fluorescein angiography, indocyanine green angiography, optical coherence tomography, ocular imaging techniques such as orbital ultrasonography and ultrasound biomicroscopy can also provide useful information for the diagnosis of ocular TB [9,10]. After diagnosis, the use of low-dose corticosteroid therapy in addition to standard tuberculosis treatment for 4-6 weeks was shown to be effective in reducing ocular damage resulting from a delayed-type hypersensitivity reaction [9,10].

In summary, disseminated tuberculosis generally affects immunocompromised people, but it should be kept in mind that it may also affect TB patients who have poor medication adherence.

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Yazışma Adresi:

Dr. Neslihan ÖZÇELİK
Recep Tayyip Erdogan University Medical Science, Pulmonary Medicine, Rize
e-mail: neslihan.ozcelik@erdogan.edu.tr