

Asymptomatic pons tuberculoma in an infant with miliary tuberculosis

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ABSTRACT

Miliary tuberculosis is caused by the hematogenous spread of *Mycobacterium tuberculosis* and consists of 1.5% of all tuberculosis cases. It is seen mostly in infants because of the immature immune system, and central nervous system (CNS) involvement is not rare. Tuberculomas are rarely seen in the localized form of CNS tuberculosis, and only 4% are localized in the brain stem. We report a 4.5-month-old infant who deteriorated during follow-up with the diagnosis of cytomegalovirus pneumonia, and afterwards received the diagnosis of miliary tuberculosis. Although the baby had no neurologic abnormality and cerebrospinal fluid findings were normal, cranial MRI revealed contrast enhanced nodular lesions in pons, cerebellum, and right parietal region. The case is presented to intensify the importance of CNS investigation even if the patient with miliary tuberculosis has no neurologic finding.

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Extrapulmonary tuberculosis is seen most often among infants and young children, and they tend to develop severe forms such as meningitis and miliary tuberculosis.¹⁻³ We report an infant case with miliary tuberculosis with multiple cranial tuberculomas, including pons, to intensify the need for examination of the central nervous system (CNS), especially in infants with no neurologic findings, as the diagnosis of CNS involvement is important in the treatment and follow-up of these patients.

Case Report. A 4.5-month-old male infant presented with the complaints of cough and respiratory difficulty. He was born in the 30th week of gestation weighing 1750 gm. His family history revealed that his mother died because of lung cancer when the baby was one-month-old, and he was adopted when he was 2.5-months-old. He had been hospitalized in a local hospital for one month with the diagnosis of pneumonia, and had received

antibiotic therapy and occasionally steroids in case of respiratory difficulty. However, he had showed no response to therapy, so he was referred to our hospital. On initial examination the patient had a temperature of 36.5°C, heart rate of 144/min, respiratory rate of 48/min, blood pressure of 90/60 mm Hg, weight of 4 kg (<3 percentile), length of 53 cm (<3 percentile). He had retractions, and rhonchi were present on auscultation. Expiration was prolonged. The liver and spleen were palpable 6 cm and 2 cm below the costal margin. Laboratory studies revealed a white blood cell count of 10.3×10^3 cells/mm³ with lymphocyte predominancy, a hemoglobin level of 11.4 gr/dl and a platelet count of 537000/mm³. The erythrocyte sedimentation rate and C-reactive protein concentration gave normal results. His urinalysis, blood gases and biochemical evaluation were in normal ranges. His chest roentgenogram revealed interstitial infiltration. As cytomegalovirus (CMV) IgM was 0.52 mg/dl (normal range [NR]: 0-0.5), IgG 202 mg/dl (NR:

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0-15) and CMV viral load was 38 pg/ml (NR: 0-16.6), ganciclovir therapy was initiated with the diagnosis of CMV pneumonia. However, his clinical condition got worse and respiratory distress increased. His subsequent laboratory studies revealed a negative tuberculin skin test although he had a BCG scar. Gastric aspirates were negative for acid-fast bacilli and tuberculous polymerase chain reaction. Sweat test, 1 antitrypsin level, serum immunoglobulin and complement levels were in normal range. The esophagogastroduodenal roentgenogram and reflux scintigraphy revealed no pathology. Anti HIV antibody was negative. Chest roentgenogram obtained in the third week of hospitalization showed enlarged hilar lymph nodes and reticulonodular infiltration. Hypodense areas resembling bilateral hilar lymphadenopathy, patchy infiltrates in the apex of left lung and consolidation in the posterior segment of upper lobe, and in the superior and basal segments of lower lobe of right lung and reticulonodular densities in both lung parenchyma's were detected in chest tomography. Antituberculous (isoniazid, rifampin, pyrazinamide, streptomycin) and steroid treatment was initiated with the diagnosis of miliary tuberculosis. His clinical condition got better, and the gastric acid aspirate cultures were positive for *Mycobacterium tuberculosis*. It was learned from the repeated family history that his real mother had not died and had been receiving anti tuberculous therapy for approximately 5 months. Although the baby did not have any neurologic findings and cerebrospinal fluid (CSF) findings were normal, cranial MRI revealed contrast enhanced nodular lesions in pons, cerebellum, and right parietal region (**Figures 1a & 1b**). The nutritional status of the baby and auscultatory findings of the lungs got better gradually. He was externalized on the 47th day of

antituberculous therapy. Streptomycin and pyrazinamide therapies were ceased at the end of the 2nd month. On follow-up controls, he had healthy appearance with normal physical examination and weight gain. Cranial MRI obtained in the 7th month of therapy was normal with no tuberculomas.

DISCUSSION. Miliary tuberculosis is seen in 1-3% of all tuberculosis patients. The risk is greater in children younger than one-year-old as their immune system is not fully mature.^{2,3} Our patient deteriorated during follow-up with the diagnosis of CMV pneumonia, and chest roentgenogram showed diffuse reticulonodular infiltration. Prematurity, suppression of the immune system with CMV infection, and steroid therapy given in case of dyspnea might have accelerated the course of miliary tuberculosis. Families usually tend to conceal tuberculous disease.⁴ In our case, the real family of the patient concealed the reality that the mother had tuberculosis. However, repeated interrogation revealed that the mother had been receiving antituberculous therapy. As lower respiratory tract infection findings started when the baby was 3.5 month-old, this infection may have been acquired postnatally.

Miliary tuberculosis often involves more than one organ system. Vallejo et al⁵ observed extrapulmonary tuberculosis in 30% of infants with tuberculosis, and 78.5% of these patients had CNS involvement. In addition, cranial MRI investigation of 7 patients with miliary tuberculosis, who did not have any neurologic symptoms, revealed CNS involvement.⁶ Similarly, our patient also did not have any neurologic finding, and CSF investigations were all normal. Cranial MRI showed lesions

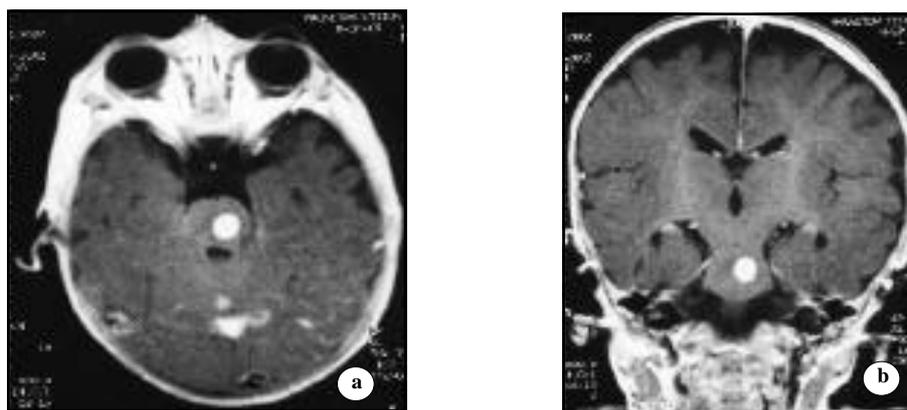


Figure 1 - Contrast enhanced T1-weighted MRI showing: a) axial cranial image with nodular lesions in the centrolateral region of pons and cerebellum. b) coronal image with nodular lesions in the pons.

resembling tuberculomas in pons, cerebellum and right parietal region, and these lesions dissolved in the 7th month of therapy in accordance with the literature.⁷ Tuberculomas are found in 9-27% of cases with CNS tuberculosis, and they can have many signs and symptoms depending on their size and location.⁵⁻⁸ Brainstem tuberculomas, including pons, constitute 2.5-8% of all intracranial tuberculomas. Pontine tuberculomas present mostly with findings such as cranial nerve involvement, paraplegia of the legs and ataxic gait.^{7,9,10} However, as in our case they can be rarely asymptomatic.⁶

The case was presented to intensify the importance of family history in the diagnosis of tuberculosis, the importance of early diagnosis and treatment, and to highlight the necessity of CNS investigation even if the patient with miliary tuberculosis had no neurologic finding.

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