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OLGU SUNUMU

Primary pulmonary tuberculosis presenting with mediastinal mass

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Summary

Presentation of pulmonary tuberculosis in the pediatric age group is usually insidious, and the diagnosis is difficult. This case study presents a 9-year-old girl with primary pulmonary mediastinal tuberculosis. Efficacy of computerized tomography in the diagnosis, management and follow-up of the disease is emphasized.

Key words: Computed tomography, lymphadenopathy, mediastinum, tuberculosis

Özet

Mediastinal kitle ile ortaya çıkan primer pulmoner tüberküloz

Pediyatrik yaş grubunda, pulmoner tüberkülozun ortaya çıkması genellikle sinsi ve tanısı zordur. Bu olgu çalışmasında, primer pulmoner mediastinal tüberkülozlu dokuz yaşında bir kız hasta sunulmakta ve bilgisayarlı tomografinin hastalığın tanı, tedavi ve takibindeki etkinliği vurgulanmaktadır.

Anahtar kelimeler: Bilgisayarlı tomografi, lenfadenopati, mediasten, tüberküloz

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Introduction

Primary tuberculosis is the common form of pulmonary tuberculosis. The increase in the prevalence of tuberculosis in both immunocompetent and immunocompromised individuals makes this disease an issue of general concern (1,2). The onset of pulmonary tuberculosis in children is usually insidious and the initial chest radiography is often normal; sputum is usually unavailable, and detection of acid-fast bacilli in sputum and gastric aspirate is possible in only 30% to 40% of patients with primary pulmonary tuberculosis. Computed tomography (CT) can provide valuable information especially in the demonstration of mediastinal lymphadenopaty, which is seen in 92% of primary tuberculosis (3-5). We discuss the efficacy of computerized tomography over conventional radiography in the diagnosis, management and follow-up of primary mediastinal tuberculosis along with other diseases presenting with mediastinal lymphadenopathy.

Case Report

A 9-year-old girl was referred to our clinic for the evaluation of mediastinal mass with the suspicion of lymphoma. She had complaints of fatigue, diminished appetite and weight loss for four months without vomiting or diarrhea. Her grandfather and aunt had died of colon and brain cancer, respectively. Her blood pressure was 120/70 mmHg, pulse rate was 85 beats/minute, temperature was 37 °C and respiration rate was 24 breaths/minute. She also had a 1.0 cm left axillary lymph node. The lower liver margin was palpable two cm below the midcostal margin. The spleen was nonpalpable. Pulmonary examination revealed diminished breath sounds bilaterally. A complete blood count demonstrated hemoglobin, 9.3 g/dL; hematocrit, 29.7 %; white blood cell count, $5100x10^3$/mcL, and platelet count, $424x10^3$/mcL. Peripheral blood smear examination revealed neutrophils 44%, lymphocytes 46%, and monocytes 10%. Sedimentation rate was 62 mm/h. Alfa-fetoprotein and human chorionic gonadotropin levels were both normal. Blood chemistry revealed the following results: serum aspartate aminotransferase, 24 U/L; alanine aminotransferase, 14 U/L; alkaline phosphatase, 126 U/L; creatinine, 0.8 mg/dL; lactic dehydrogenase, 705 U/L; and uric acid, 3.1 mg/dL; potassium, 4.2 mEq/L; sodium, 135 mEq/L. Bone marrow examination findings were normal. The Mantoux test with 5-tuberculin unit (TU) resulted in 20-mm induration. No acid-fast bacillus was detected in gastric material examined in three consecutive mornings.

Gray scale and color Doppler abdominal ultrasonography revealed hepatomegaly. Chest radiography showed mediastinal widening with contour lobulation in addition to left midzone opacification (Figure 1a). CT scanning

with intravenous contrast administration demonstrated conglomerate and enlarged lymph nodes involving whole mediastinum, which displaced the adjacent vasculature (Figures 1b, 1c). Some lymphadenopathies were containing peripheral rim enhancement, calcifications and central necrotic low attenuations consistent with necrosis. CT also showed consolidation and air bronchogram at the lingula of the left lung (Figure 1d). Histopathological findings obtained from mediastinal lymph node biopsy were caseous granulomas with necrosis; however, neither acid-fast stains of the specimen nor PCR technique detected the mycobacteria. Based on chest radiography, a positive purified protein derivative test, CT and histopathological findings, the patient was diagnosed to have primary tuberculosis, and drug therapy was started including isoniazid (INH), rifampicine (RMP) and pyrazinamide. Two months later pyrazinamide was discontinued, and INH and RMP were continued for additional 10 months. The complaints began to disappear from the third week of therapy and the patient was followed up with radiography and CT. Although mediastinal widening and left lung consolidation considerably decreased, some upper mediastinal widening and left lung midzone radioopacity with calcifications persisted on follow-up chest radiographs. After 12 months from the onset of the therapy, contrast enhanced CT scan demonstrated that the number and dimension of lymph nodes decreased significantly, whereas rim enhancement and central necrosis were prominent. In addition, gradual resolution of the left upper lung consolidation was noted (Figure 1e).

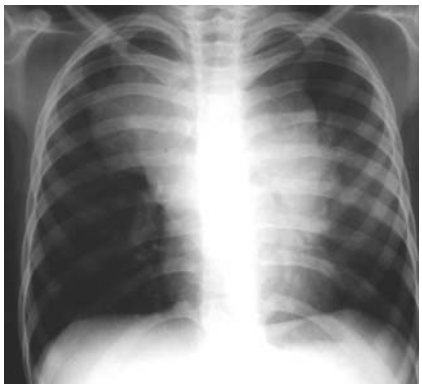

Figure 1a.

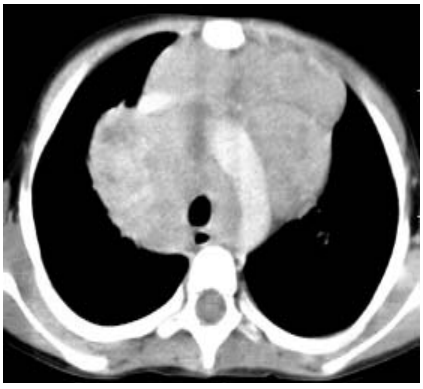

Figure 1b.

Figure 1a. Frontal chest radiography shows a large mediastinal density and left lung midzone consolidation. **b.** Axial contrast material enhanced CT scan shows conglomerate enlarged mediastinal lymph nodes with peripheral enhancement and central areas of low attenuation.

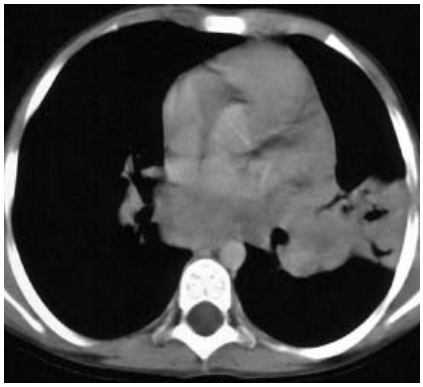

Figure 1c.

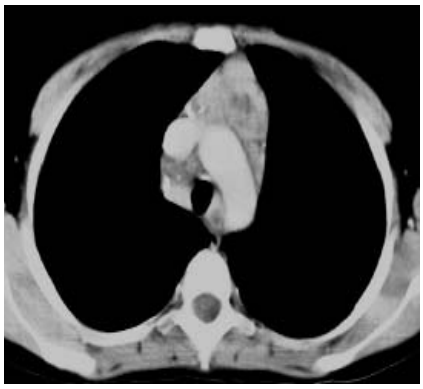

Figure 1d.

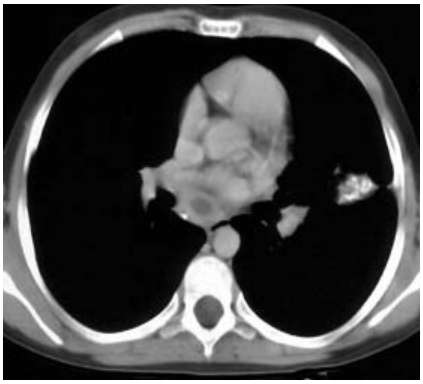

Figure 1e.

Figure 1 c. At a lower level than Figure 1b subcarinal enlarged necrotic lymph nodes and consolidation in the lingula of the left lung with air bronchogram are demonstrated. **d.** Follow-up contrast material enhanced CT scan revealed that the number and dimension of lymph nodes decreased with some calcifications. **e.** Consolidation of the lingula and subcarinal centrally necrotic lymph nodes shrinks and contains some calcifications

Discussion

The diagnosis of pulmonary tuberculosis in children is often based on epidemiological, clinical, radiographic, and skin test information, rather than bacteriological data; however the diagnosis is usually difficult and is only confirmed in less than 40% of the cases. On the radiologic viewpoint, tuberculosis is a greater mimicker, and diagnosis in pediatric patients relies on the demonstration of mediastinal lymphadenopathy, in which CT is considered the modality of choice (1-3).

Although primary tuberculosis has been increasingly encountered in adults, almost all of the cases in children are primary infections; the onset is insidious and the initial radiologic findings are usually normal. Tuberculosis shows a number of clinical and radiologic features depending on the organ site affected and tend to disseminate from its primary site (1). There may be parenchymal infiltrates on CT and the radiological picture may be confused with many other malignant or infectious diseases commonly seen in children. Although positive result of a tuberculin test helps diagnose the cause of an unexpectedly huge hilar enlargement and the area of central low attenuation with peripheral rim enhancement consistent with caseous necrosis on CT, these findings are not specific but helpful for primary tuberculosis (2,3,5). Primary tuberculosis characteristically manifests as dense, homogenous and well-defined airspace consolidation on CT. Lobular consolidation consists of centrally located granulomas that contain caseation necrosis and enveloped by nonspecific inflammation. The prevalence of lobular consolidation in newly diagnosed disease is significantly higher than reactivated disease (1,3). Lymphadenopathy is the radiologic hallmark of primary tuberculosis. Although enlarged nodes occur in up to

92% of pediatric cases, the prevalence of lymphadenopathy decreases with increasing age (1). Similar to the present case, lymphadenopathy is usually seen in association with parenchymal consolidation and bronchial compression (3). Tuberculosis lymphadenopathy typically resolves at a slower rate than the associated parenchymal disease without important radiological sequela (2). CT patterns of tuberculosis range from various degrees of mild homogenous enhancement of lymph nodes with irregular thin or thick enhancing rims to focal/multifocal areas of low attenuation (central caseation and necrosis) (2,3).

The radiologic differential diagnosis of the lymphadenopathy includes metastasis, histoplasmosis, lymphoma, sarcoidosis, pneumoconiosis and hemosiderosis (6-9). Pneumonitis and associated hilar/mediastinal lymphadenopathy are seen at the time of initial infection in most patients with histoplasmosis. Histoplasmosis resolves often with residual parenchymal and nodal calcifications. Mediastinal granuloma in histoplasmosis represents a lobulated mass of caseous mediastinal lymph nodes. On CT examination, a low attenuation mass is usually seen in the right paratracheal and subcarinal region. Similar findings may be seen in tuberculosis. CT scans show hilar and/or paratracheal/subcarinal masses, which frequently contain multiple areas of calcification (7). Sarcoidosis is characterized by nonnecrotizing granulomatous inflammation. Symmetric bilateral hilar lymphadenopathy is the classic radiographic distribution of enlarged intrathoracic lymph nodes in sarcoidosis (8). Intrathoracic disease is seen in over 85% of patients with Hodgkin disease (6,9). In 98% of patients with intrathoracic disease, superior mediastinal lymph node involvement is seen. Therefore, detection of other enlarged mediastinal and/or hilar lymph node groups without associated superior mediastinal lymphadenopathy might suggest an alternative or coincidental disorder; however, lymphomatous masses usually have soft tissue attenuation; there may be moderate enhancement after i.v. contrast injection (9). Significant contrast enhancement rarely occurs. Areas of low attenuation have been demonstrated in about 20% of cases. Calcification is rarely seen before treatment. In a small percentage of cases, non-Hodgkin disease involves the thoracic cavity. In addition superior mediastinal lymph node involvement of non-Hodgkin disease is less than 75% (9).

In conclusion, tuberculosis can mimic a variety of diseases, and one should be familiar with the various radiological features of tuberculosis to establish early and precise diagnosis and effective therapy because it can be devastating if left untreated. Many patients with pulmonary tuberculosis do not need CT in the initial diagnosis of tuberculosis; however, CT has a better accuracy than conventional chest radiography in the diagnosis of primary tuberculosis and can allow prompt and precise diagnosis to start the proper treatment, when the radiographic findings are equivocal and tuberculosis is suspected clinically.

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