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Widespread Tuberculosis Calcification in a 3.5 year old boy: A case report

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INTRODUCTION

Tuberculosis is an infectious disease that has been known for centuries. Meanwhile it still remains as a major cause of morbidity and mortality worldwide and is the most common cause of death from a single infectious disease, particularly in children. Nearly 40 million children are likely to be exposed to the risk of TB and nearly 3-4 million children below age 5 years are estimated to be infected and may progress to disease. [1]

Rarely, the child experiences a clinically significant disease with high fever, cough, malaise, and flu-like symptoms that resolve within a week. Most children who develop tuberculosis disease experience pulmonary manifestations, but approximately 25 to 35 percent of children have an extra pulmonary presentation [2] Tubercle bacilli are disseminated to distant sites in all cases of tuberculosis infection. This dissemination is clinically silent in most cases but can be the origin of miliary tuberculosis or extra pulmonary tuberculosis in the immediate or distant future. Other patients experience protracted hematogenous tuberculosis caused by an intermittent release of tubercle bacilli as a caseous focus erodes through the wall of a blood vessel in the lung.

Although the clinical picture may be acute, more often it is indolent and prolonged, with spiking fevers accompanying the release of organisms into the bloodstream. Multiple organ involvement is common leading to hepatomegaly, splenomegaly, and lymphadenitis in superficial or deep nodes. The most common extra pulmonary form of tuberculosis is lymphatic disease accounting for about two thirds of cases of extra pulmonary tuberculosis. However, the second most common form is meningeal disease occurring in 13% of patients and, historically, occurring in three out of a thousand untreated tuberculosis infections in children < 5 years of age. [3]

CASE PRESENTATION

A 3.5 year old boy from Afghan refugees in Iran who admitted in our centre due to fever, cough and weight loss. His illness started since he was 1 year old with cough and night chills. He diagnosed and partially treated for TB. He was fine until He admitted again due to relapse of pulmonary TB symptoms when he was 2.5 year old.

He was on anti- tuberculosis therapy for 6 month. Since 1 year ago he demonstrated his illness with skin lesions in the face and extremities that remains with scars. Besides he has fever and productive cough since 4 month ago.

The patient father had a history of pulmonary tuberculosis. He had no history of vaccination including BCG for tuberculosis. He had a history of tonic- clonic convulsions one year ago without repetition. In physical exam his weight and height was under 5 percentile. Other positive findings were crackles in both lungs, mild hepatomegaly in the abdomen and erythematous skin plaques in extremities.

Laboratory investigation revealed leukocytosis with shift to the left, anemia, thrombocytosis. CRP was positive and ESR was 56. tuberculin skin test was performed twice. At the time of admission the induration size was 10mm and after 1month of therapy the induration that recorded was 20mm. liver function tests were inclined. Bacteriologic evaluations of gastric washing, urine and wound secretion were done 3 times and were negative for TB. Gastric washing and wound secretion PCR was negative for TB. Serum immunoglobolins and NBT test was in normal range. Skin lesion biopsy report showed a chronic nonspecific inflammation without granuloma.

Chest x-ray and CT scan showed diffuse nodular infiltration and calcified nodules in parenchyma of both lungs and partial collapse of right lung upper lobe. (Figure 1, 2)

Brain CT scan demonstrated focal calcifications in brain parenchyma (figure 3).

In the IVU (Intra Venus Urography) he had calcified nodules in both kidneys. (Figure 4) Abdominal CT scan was done and showed hypo echo, irregular border foci in upper segment right lobe of liver with several calcification. Calcification was seen in spleen too. (Figure 5) Echocardiography was performed for the patient to rule out the probability of TB pericarditis. Thus no evidence was found in the benefit of pericarditis.

Regarding widespread calcifications, tuberculin test conversion, history of contact with his father and positive clinical symptoms in favor of tuberculosis we started anti tuberculosis therapy and the result was remarkable after 1 month of therapy he started to weight gain , skin lesions were healed and his general condition was improved.

DISCUSSION

Our case was a 3.5 year old boy who admitted in our center due to pulmonary tuberculosis following by disseminated tuberculosis. In this child the involvement of skin, CNS and intra abdominal organs was seen after 1 year of pulmonary tuberculosis.

There are other cases with same characters. Khan MQ and colleagues reported an unusual case of widespread abdominal calcifications, in the peri-colic mesentery, liver and spleen. The diagnostic laparoscopy showed them multiple encapsulated calcified hard rounded masses of varying size and shape, with marked adhesions in and around the bowel and mesentery. Histopathology identified them as calcified lymph nodes, but was unable to highlight the pathogenesis of these calcifications. The diagnosis of post tuberculosis calcification of lymph nodes is made on the basis of exclusion. [4]

Another similar report was a 12-year-old girl with clinical evidence of abdominal tuberculosis also had a widespread soft tissue calcification termed calcinosis universalis. Despite anti tuberculosis therapy the patient died and autopsy confirmed tuberculosis peritonitis. [5]

Our patient had a negative result for all the laboratory results such as bacteriology of gastric washing, urine, secretion of skin ulcer, PCR of gastric washing and pathology

of skin ulcer. Calcium, phosphor, parathormone level and immunological tests were in normal range. Tuberculin test was performed and the result was negative. As it is well understood in childhood tuberculosis, even under optimum conditions, three gastric aspirates yield *M. tuberculosis* in < 50% of cases; negative cultures never exclude the diagnosis of tuberculosis in a child.. Unfortunately, the acid-fast stain of either sputum or gastric contents in small children is positive in < 10% of cases. [6, 7]

Our case was an Afghan refugee boy with poor socioeconomical condition. He was malnourished and hasn't been vaccinated for Tuberculosis. All this elements besides to the delay in diagnosis of his illness lead to the severity of the disease. In other words in cases such as this patient early diagnosis is very essential because it can prevent the further consequences of disseminated Tuberculosis. Regarding widespread calcifications, tuberculin test conversion, history of contact with his father and positive clinical symptoms in favor of tuberculosis we started anti tuberculosis therapy. He also treated with steroids in the time of admission because of poor general condition. More over he was on high caloric diet. After 1 month of therapy the result was satisfactory. He started to weight gain, skin lesions were healed and his general conditions remarkably improved and the chest x-ray showed significant improvement.

In conclusion this study is another emphasize on this clue that diagnosis of tuberculosis in children is difficult and confusing and in many cases the child is diagnosed mainly by contact tracing of an adult with tuberculosis. We should denote although the detection of tuberculosis in children is sometimes time consuming but the early diagnosis and treatment can make a prominent difference in the rate of morbidity and mortality of the children.

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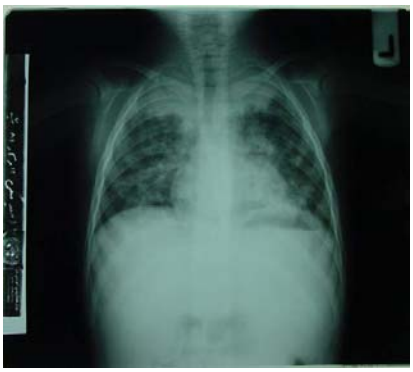

Figure 1- Chest X-ray (Pulmonary TB)

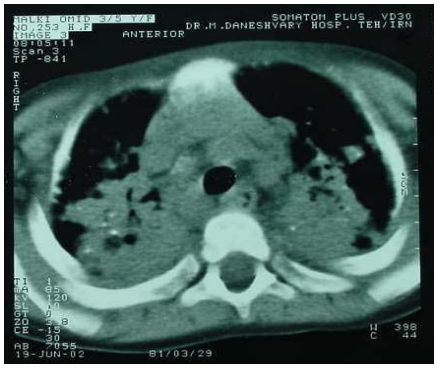

Figure 2- Lung CT scan (pulmonary calcification)

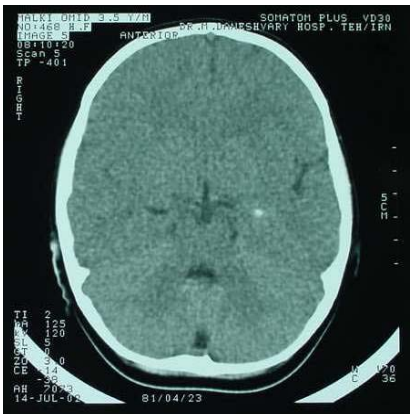

Figure 3- Brain CT scan (CNS involvement)

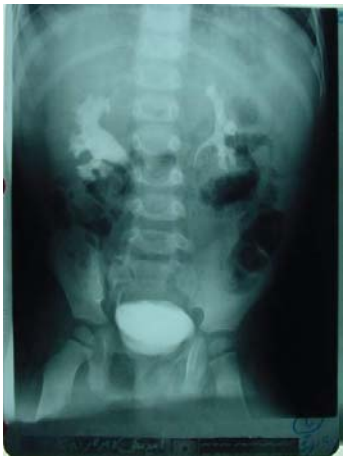

Figure 4- IVU (Renal involvement)

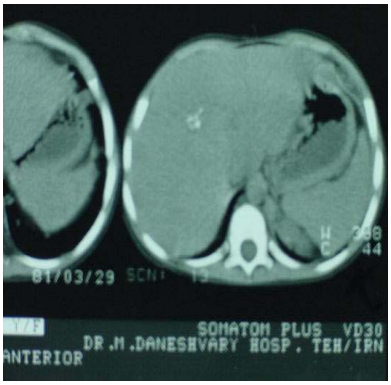

Figure 5-Abdominal CT scan (spleen and liver Calcification)