

# Resistant Pneumonia In A Child With Non-Hodgkin Lymphoma In Remission: A Case Of M. Tuberculosis

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## Abstract

Respiratory system infections are the most common complications in immunocompromised cancer patients. Pulmonary infections in these patients can also progress rapidly and cause respiratory failure. In this report, we describe a girl who developed pulmonary tuberculosis within two months after completion of chemotherapy for Non-Hodgkin Lymphoma. The diagnosis of tuberculosis was made by bronchoalveolar lavage fluid culture. Sputum and gastric lavage fluid cultures were negative. Pulmonary infiltrates and mediastinal lymph nodes resolved within three months of treatment, and at the end of therapy, tomography was normal except thickening of right major fissure.

## INTRODUCTION

Respiratory system infections are the most common complications in immunocompromised cancer patients. Mucosal desruction, humoral and cellular immunodeficiency secondary to primary disease itself and chemotherapy lessen clearence of aspirated microorganisms and facilitate local infection. In patients having immunosuppressive treatment, unusual pathogens such as mycobacteria, nocardia, aspergillus and chlamidya can cause infections. There are some reports in the literature on patients with malignancy who developed tuberculosis during or after intensive chemotherapy [1,2]. In this report, we describe a girl who developed pulmonary tuberculosis after completion of chemotherapy for Non-Hodgkin Lymphoma.

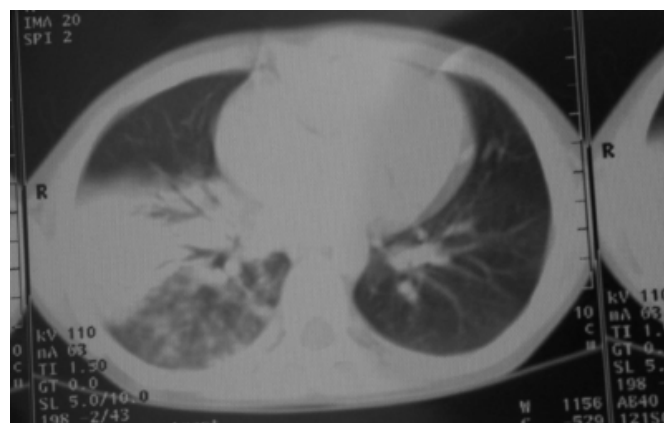
## CASE REPORT

A seven years old girl diagnosed with T cell Non-Hodgkin Lymphoma at age 6 was admitted to our hospital with the chief complaints of fever, persistent cough, and respiratory distress after completion of treatment (NHL-BFM 90 Protocol). On physical examination, fever (38.5 °C, axillary), respiratory distress, intercostal retractions and thin rales in middle and lower regions of right lung were present. Her initial blood count showed a hemoglobin level of 10 g/dl, a hematocrit of 30.8%, a platelet count of  $228 \times 10^9/L$ , and white blood cell and absolute neutrophil counts of  $4.6 \times 10^6/L$  and  $1.0 \times 10^6/L$  respectively. Blood gas analysis showed hypoxemia ( $SO_2$ : 90%). Erythrocyte sedimentation rate (ESR) was 80 mm/h. Liver and kidney function tests were

normal range. Chest roentgenogram showed pneumonic infiltration in middle and lower lobe of right lung. Cefepim, amikacin and teicoplanin were given empirically for the pneumonia. Blood and sputum cultures were negative. PPD test showed no induration and early morning gastric lavage samples on three consecutive days were negative for acid-fast bacteria. On follow-up, antibiotic therapy was changed to combination of piperacilline-tazobactam, amphotericin-B and clarithromycin because of no response. At the same time thorax tomography showed diffuse pneumonic consolidation with right paratracheal precarinal mass image consisting of conglomerated lymph nodes (Figure 1).

### Figure 1

Figure 1: Diffuse pneumonic consolidation with right paratracheal precarinal mass image consisting of conglomerated lymph nodes (Thorax tomography)



For differential diagnosis of this resistant clinical situation,

further investigations as flexible bronchoscopy and broncho-alveolar lavage (BAL) were done in another hospital. BAL fluid cultures revealed *Mycobacterium tuberculosis*. A 9-months regimen consisting of isoniazid, rifampin, and pyrazinamide given for 2 months followed by isoniazid and rifampin for 7 months was started. Three months after initiation of tuberculostatic treatment, a control tomography showed nodular opacity accepted as a sequel of tuberculosis. At the end of therapy, tomography was normal except thickening of right major fissure (Figure 2).

### Figure 2

Figure 2: Normal findings except thickening of right major fissure (Control tomography at the end of therapy)



## DISCUSSION

Mortality and morbidity of patients with acute leukemia are mainly caused by relapses and infections especially febrile neutropenia and lower respiratory infections. There are increased predisposition to infection in patients with malignancies particularly Hodgkin's disease, leukemia and T-cell lymphoma. Adzic [3] reported on 20 adult patients with pulmonary tuberculosis and hematological malignancies of whom 40 % was Non-Hodgkin lymphoma, 25 % chronic lymphocytic leukemia and 2 % Hodgkin's disease. But there is no much reported pulmonary tuberculosis in pediatric cancer patients.

The clinical diagnosis of tuberculosis in immunocompetent children is straightforward and consists of the triad of exposure to an infectious case, a positive tuberculin skin test, and an abnormal chest radiograph. In contrast, clinical symptoms are much less specific among children who are malnourished, immunocompromised, or suffering from HIV or malaria. The gold standard for establishing the diagnosis of tuberculosis in adults is sputum smear microscopy for acid-fast bacilli confirmed by mycobacterial culture. However, because as many as 95 percent of children

younger than 12 years of age have negative sputum smears [4], these diagnostic methods for children are inadequate. Culture of gastric juice or broncho alveolar lavage for tuberculosis bacteria is positive in less than 50 % of the cases [5,6]. False negative TST may occur in children with severe tuberculosis soon after infection, those with debilitating or immunosuppressive illnesses, malnutrition, or other severe infections [7].

Radiological evidence of pulmonary tuberculosis usually includes lymphadenopathy (hilar or mediastinal) and lung parenchymal changes. The most common parenchymal changes are segmental hyperinflation, atelectasis, alveolar consolidation, pleural effusion/empyema and, rarely, a focal mass [7]. Cavitation is rare in young children but is more common in adolescents, who may develop adult-type post-primary disease. Delacourt et al found that in 60 % of children with tuberculous infection and normal findings on chest radiography, enlarged lymph nodes may be seen by CT scan [8]. CT imaging may be helpful in demonstrating pulmonary disease, early cavitation, and bronchiectasis following pulmonary tuberculosis where chest radiographs are normal or unhelpful [7,8].

It is reported that, the role of bronchoscopy and BAL in evaluating children with pulmonary tuberculosis is controversial, the culture yield is usually lower than for three properly obtained gastric aspirates [6], bronchoscopy and BAL may, however, be useful in the diagnosis of endobronchial tuberculosis and excluding other causative agents such as opportunistic infections particularly in immunocompromised children [7]. But our patient presented with a negative TST and gastric lavage samples on three consecutive days specimens, while bronchoalveolar lavage fluid specimen culture were positive for mycobacteria.

The most important means of transmission of tuberculosis is by inhalation of aerosols carrying *Mycobacterium tuberculosis*. But, we couldn't be able to indicate an infectious person who contacted with our patient. She was vaccinated with BCG vaccine against tuberculosis during infancy.

In conclusion, pulmonary infections can complicate the course of cancer treatment in children and generally, it is treated empirically with broad-spectrum antibiotics in most of oncology centers in developing countries. However, for optimal treatment of lung infections in cancer patients, identification of the infectious agent is necessary, as the clinical course in our patient demonstrates. Rare infections

such as tuberculosis should be included in the diagnostic work-up of such immunocompromised children. Because children cannot produce sputum easily, gastric aspirates or bronchoscopic lavage fluid should be obtained at early stages for microbial identification.

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