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Immune Thrombocytopenia Secondary to Pleural Tuberculosis: A Case Report

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Abstract

Immune thrombocytopenia associated with tuberculosis is a rare clinical entity. We report immune thrombocytopenia associated with pleural tuberculosis in a 22-year-old Ethiopian previously healthy man who presented to the emergency department with one-month history of fever, night sweats and productive cough. Chest x-ray showed right pleural effusion and pleural biopsy was performed. During his stay in the hospital, his platelets count dropped persistently until reach 16000/µL. pleural biopsy results were consistent with pleural tuberculosis. The patient was diagnosed with pleural tuberculosis associated with immune thrombocytopenia. The patient received antituberculosis treatment, immunoglobulins, and steroid. After completion of antituberculosis treatment, the patient was seen in the clinic he was doing well, and his platelets count was 276000/µL.

Keywords: Immune Thrombocytopenia, Pleural Tuberculosis, Intravenous Immunoglobulin

ملخص الدراسة

قلة الصفيحات المناعية المرتبطة بالسل هي كيان سريري نادر. لقد استعرضنا في هذا التقرير قلة الصفيحات المناعية المرتبطة بالسل الجنبي في رجل إثيوبي يبلغ من العمر 22 عامًا كان يتمتع بصحة جيدة في السابق والذي قدم إلى قسم الطوارئ يشكو من الحمى و السعال المنتج مصحوب بتعرق ليلي لمدة شهر واحد. أظهرت الأشعة السينية للصدر انصبابًا جنبيًا في الجهة اليمنى وتم إجراء خزعة من الجنب. أثناء إقامته في المستشفى ، انخفض عدد الصفائح الدموية باستمرار حتى وصل إلى 16000 / ميكرولتر. كانت نتائج الخزعة الجنبية متسقة مع مرض السل الجنبي و تم تشخيص إصابة المريض بالسل الجنبي المرتبط بنقص الصفيحات المناعي. تلقى المريض العلاج المضاد للسل والغلوبولين المناعي والستيرويد. بعد الانتهاء من العلاج المضاد للسل ، تمت معاينة المريض في العيادة وهو بحالة جيدة ، وكان عدد الصفائح الدموية لديه 276000 / ميكرولتر.

الكلمات المفتاحية: قلة الصفيحات المناعية ، السل الجنبي ، الغلوبولين المناعي الوريدي

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Introduction

When platelets are covered with autoantibodies to platelet membrane antigens, which cause spleen sequestration and phagocytosis by mononuclear macrophages, the condition is known as immune thrombocytopenia (ITP). The resulting shortened lifespan of platelets in the circulation, together with incomplete compensation due to increased platelet production by megakaryocytes of the bone marrow, lead to thrombocytopenia. In more than half of all ITP cases, an underlying cause is not found. The most common secondary causes of ITP include systemic lupus erythematosus, lymphoproliferative diseases, particularly chronic lymphocytic leukemia and Hodgkin lymphoma, HIV infection, and medications. [1] A growing body of reports from different countries suggests that Mycobacterium tuberculosis (MTB) is the etiology of some cases of acute ITP. [1-8] The purpose of this report is to increase clinicians' awareness of this rare association between the two conditions because early diagnosis and treatment are important to avoid an extreme outcome.

Case history

A 22-year-old Ethiopian previously healthy man presented to the emergency department with one-month history of fever, night sweats and productive cough. His medical history was unremarkable, and he denied loss of weight, anorexia or contact with sick person. His family history was unremarkable.

On physical examination, he was febrile but conscious and fully oriented. His temperature was 38.2ºC, blood pressure 104/67 mm Hg, pulse rate was 96/minute and respiratory rate was 17/minute.

Chest examination showed reduced chest expansion on the right side with decreased air entry and dullness on percussion. The rest of his examination was unremarkable.

Initial investigation showed hemoglobin of 12.2 gm/dl; leucocytes, 7800//µL and platelets of 155,000/µL. blood chemistry and renal function tests were within normal limit. Chest x-ray showed right pleural effusion. The patient was admitted in a respiratory isolation room to rule out tuberculous infection. Thoracentesis was carried out and analysis of the pleural fluid revealed: pH, 7.60; glucose, 4.7 mmol/L; protein, 4.5 g/L; LDH, 858 units/L and leucocytes, 10340 cells/µL (88% lymphocytes). Pleural fluid and two samples of sputum were sent to microbiology laboratory for acid-fast bacilli (AFB) smear, TB polymerase chain reaction (PCR) and TB culture. Pleural fluid and the two sputum samples were negative for AFB and PCR, while TB culture results were pending.

A pleural biopsy was obtained through thoracoscopy and samples were sent for AFB smear, TB PCR and TB culture. Other sample was sent for histopathology study. On the third day of admission the patient developed purpuric rash on his trunk and lower limbs. Cell blood count revealed hemoglobin of 12.5 gm/dl;

leucocytes, 8800//µL and platelets of 66000/µL. Peripheral blood smear confirmed thrombocytopenia otherwise it was normal. The patient's hospital course was complicated by a persistently declining platelet count. Within four days platelets count was 16000/µL. Antinuclear antibody was negative and no findings indicative of disseminated intravascular coagulation (DIC) were detected. On the following days pleural biopsy demonstrated positive AFB on smear and necrotizing granulomatous inflammation.

Based on these findings, the patient was diagnosed with ITP secondary to pleural TB. The patient was started on 4-drug antituberculosis treatment (ATT) and pulse steroid therapy in the form of methylprednisolone 1 g intravenously daily for 3 days followed by prednisone 1 mg/kg PO once daily along with intravenous immunoglobulin (IVIG) 1 g/kg/day for 3 days. After 5 days, platelet count rose to 26000/µL, which then gradually increased to 95000/µL on Day 10, and the patient was discharged. On follow up, the platelet counts improved further, and steroids were tapered till stopped. After completion of ATT therapy, the patient was seen in the clinic he was doing well and his platelets count was 276000/µL.

Discussion

Tuberculosis (TB) is one of the most common communicable illnesses in developing nations, owing to a wide range of well-documented predisposing factors. The clinical manifestations of active TB are variable, and the laboratory findings showed a wide spectrum of hematological manifestations such as anemia, leukocytosis, monocytosis, lymphopenia, leucopenia, thrombocytopenia, thrombocytosis, have been observed, although an association with ITP is rare. [1,7]
The occurrence of thrombocytopenia in tuberculosis could be due to a variety of causes such as granulomatous infiltration of bone marrow causing pancytopenia, thrombotic thrombocytopenic purpura, Disseminated Intravascular Coagulation (DIC) or hemophagocytic lymphohistiocytosis. [9] ITP can be diagnosed by presence of isolated thrombocytopenia with a normal peripheral smear and exclusion of other secondary causes capable of thrombocytopenia. [1] Although not considered necessary, platelet antigen-specific antibodies or platelet surface membrane IgG can support the diagnosis. [1,10] In our patient, common secondary causes of thrombocytopenia such as medications, viral infection, systemic lupus erythematosus and leukemia had been excluded through history and negative investigations. Antiplatelet antibodies were not done as this test is not available in our hospital. Despite these findings, the association between TB and ITP in this patient may be coincidental and has no immunological basis. Nonetheless, in our patient, the response to steroids and intravenous immunoglobulin suggests the immunological basis of thrombocytopenia, while normalization of platelet counts and the absence of recurrence of thrombocytopenia after completion of ATT suggests TB as the etiology of thrombocytopenia.

The pathogenesis of TB associated ITP is unclear, however, it is thought to be the generation of antiplatelet antibodies by lymphocytes borne as a result of clonal proliferation due to host's immune response to the MTB, [10] while some authors suggested that TB may share antigen with platelets leading to antiplatelet antibody. [11]

In conclusion, TB associated ITP is a rare clinical entity. A high index of suspicion for TB is essential and should be considered as one of the secondary causes of ITP especially in patients from high endemicity areas. Initiation of ATT, steroids and immunoglobulins is crucial for recovery.

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