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Case Report .

Immune Thrombocytopenia: An Exceptional Manifestation of Tuberculosis

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ABSTRACT

Association of immune thrombocytopenic purpura and tuberculosis is an exceptional condition. We report a case of cervical & retroperitoneal lymph node tuberculosis who presented with ITP. A 17 year old male was admitted to our hospital with oral mucosal bleed and petechial lesions all over body. A diagnosis of immune thrombocytopenia (ITP) was established and patient was treated with Intravenous (IV) Methyl- prednisolone therapy followed by oral steroids, however it was futile to bring forth any sustained platelet response. CT scan abdomen revealed multiple retroperitoneal lymph node masses .Fine Needle Aspiration (FNA) of these lymph nodes revealed caseating lymphadenitis suggestive of tuberculosis. After 2 weeks of anti-tubercular therapy, the platelet counts returned to normal and patient was not on any treatment for ITP thus signifying likely association between tuberculosis and immune thrombocytopenia.

Key Words: ITP, Tuberculosis, Lymph nodes, FNA.

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INTRODUCTION

Tuberculosis is an enormous community health predicament with a variety of manifestations. Diverse hematologic abnormalities such as anemia, leucocytosis, monocytosis, lymphopenia, leucopenia, thrombocytopenia, thrombocytosis, leukemoid reactions and pancytopenia have been described in tuberculosis[1] but immune thrombocytopenia (ITP) as the only presenting feature of tuberculosis is rare with few published reports.[2,3] Immune thrombocytopenia is characterized by a low platelet count associated with the presence of platelet auto antibodies. The diagnosis of ITP remains a diagnosis of exclusion, and a bone marrow examination should be performed in patients with atypical features. Tuberculosis is a rare but curative cause of immune thrombocytopenia.[4,5] Here, we report a patient with lymph node tuberculosis presented as immune thrombocytopenic purpura (ITP).

CASE PRESENTATION

17 years male admitted with complaints of fever of three weeks duration associated with loose stools, oral mucous bleed and bleeding from nose and petechial rashes all over body. On examination, patient was conscious, no pallor , icterus, or pedal edema was seen; BP was 130/80 mmHg, ecchymotic patches were seen inside mouth (Fig 1), diffuse petechial rash along with ecchymotic patches all over the body (Fig 2), cervical lymph nodes were matted, immobile& adherent to skin; liver &spleen ware not palpable; other systemic examination was unremarkable. On Investigations, Hemoglobin (Hb) was 11.4 gm/dl, Total leukocyte count (TLC) was 8200/mm^3, Platelet count 5,000/mm^3, MCV 80fl, MCHC 33%, retic count 2%, ESR 37mm/isthr, Peripheral Blood Smear (PBS) revealed platelets markedly reduced in smear, Red Blood Cells (RBC) showed mild anisopoikilocytosis, predominantly norm chromic; prothrombin time was 1.1 (control 14 seconds), renal & liver function tests were within normal limit.; malaria antigen test & dengue NS1 antigen was negative. Lipid profile, serum ferritin was within normal limit. Bone Marrow aspiration & biopsy revealed megakaryocytic thrombocytopenia with normo cellular marrow suggestive of ITP; no evidence of tuberculosis or lymphoma on biopsy. Ultrasound abdomen was suggestive of multiple enlarged lymph nodes in mesentery, pericaval, paraaortic largest 5/3cm in size. CT Abdomen (Fig 3 & 4) was suggestive of heterogeneously enhancing lymph nodal masses seen in abdomen involving peripancreatic, preaortic, right lumbar region, mesentry; liver is enlarged 17cms, diffuse mural thickening involving terminal ileum. Ultrasound guided Fine Needle Aspiration (FNA) from right iliac lymph node as well as direct FNA from cervical lymph node smears showed epitheloid cell granulomas in background of reactive lymphoid cells, stain for AFB was positive in both.

Patient was transfused 8 units of random donor platelets, 2 units of single donor platelets, injection Methyl Prednisolone for 3 days followed by oral prednisolone 1mg/kg body weight but only transient increase in platelet count (5000 to 25000/mm^3) was seen. After cervical FNA report suggestive of TB, first line anti-tubercular therapy (4 drugs) was started; platelet counts steadily increased to normal within two weeks. Patient came for follow-up after two months; his platelet count was 200,000/mm^3.

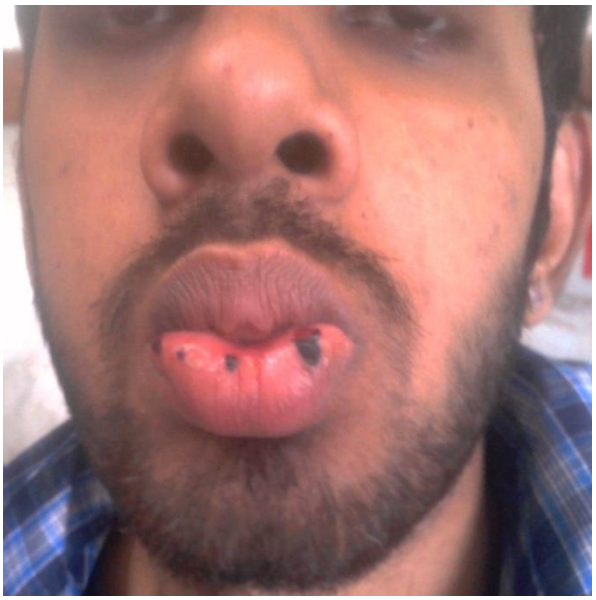


Fig 1: Ecchymotic patches inside the mouth

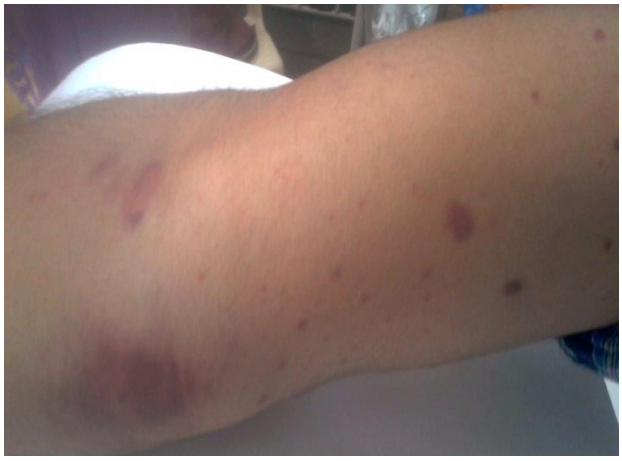
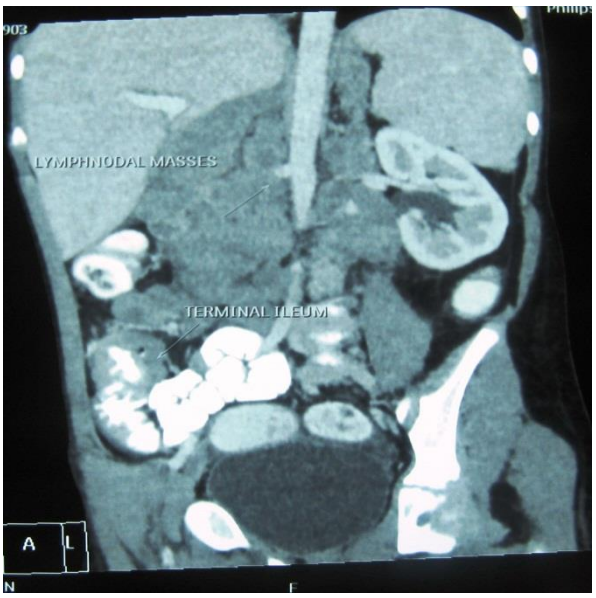


Fig 2: Petechial & ecchymotic rashes over arm



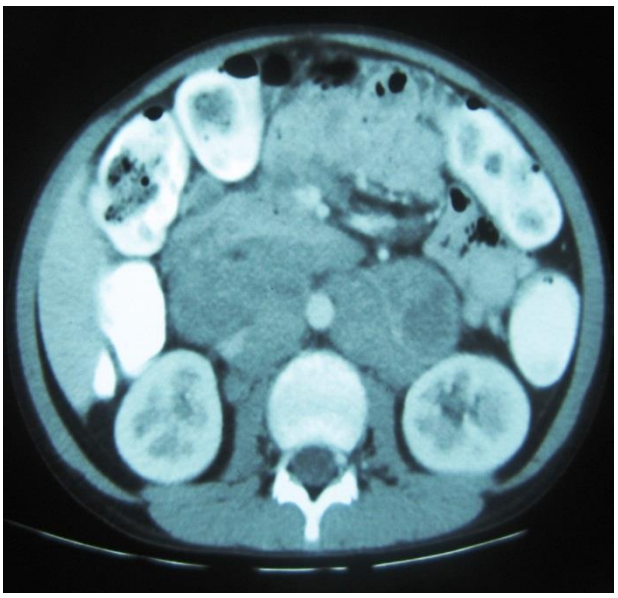


Fig 3 & 4: CT scan Abdomen demonstrating enlarged para-aortic, aorto-caval & retrocaval lymph nodes.

DISCUSSION

ITP is an acquired disorder as there is immune destruction of platelets by inhibition of platelet release from megakaryocytes. In children, ITP typically presents acutely, while in adults it has chronic course. Among secondary causes of ITP include infections (hepatitis C, HIV), drugs (rifampicin)[6] and autoimmune diseases. Patients with pulmonary & extra pulmonary tuberculosis, haemotological abnormalities anaemia, leucocytosis, leucopenia, thrombocytopenia, thrombocytosis & pancytopenia[3], but severe thrombocytopenia due to tuberculosis is rare.[4,5,7,8]

In present case, differential diagnosis of Disseminated Intravascular Coagulopathy (DIC), Thrombotic Thrombocytopenic Purpura (TTP), Evans Syndrome, Henoch Scholein Purpura (HSP) & Haemophagocytic syndrome, Lymphoma were kept as possibilities.

In DIC, there is consumption of procoagulants & platelets; manifested as prolonged coagulation time, thrombocytopenia, raised levels of fibrin degradation products. In our patient PT& APTT (Activated Partial Thromboplastin Time) were normal, so DIC was ruled out. In TTP, presents as anaemia, thrombocytopenia, neurological manifestations with peripheral smears show moderate to severe schistocytosis, but in our patient there was no evidence of microangiopathic haemolysis. In HSP, patient presents with rash, abdominal pain, haematuria, arthritis; but in our patient apart from petechial rashes, no other symptoms were present, so it was ruled out. In Evans Syndrome, there is coexistence of autoimmune hemolytic anemia &immune mediated thrombocytopenia, direct coombs test is positive but in our patient, coombs test is negative & no anemia was documented. In Haemophagocytic Syndrome, there is pancytopenia hypertriglyceridimia, increased ferritin levels with hepato-splenomegaly, however in our patient all these manifestations were not seen so again it was ruled out.

CONCLUSION

ITP is a disorder in which antiplatelet antibodies cause accelerated destruction of platelets, resulting in thrombocytopenia and a varying predisposition for bleeding[9] In addition, it is now acknowledged that these antibodies may also impair platelet production, creating a dual cause of thrombocytopenia.

The diagnostic criteria[9] for ITP are

(1) Isolated thrombocytopenia with otherwise normal peripheral complete blood count and smear,

(2) An absence of hepatosplenomegaly on physical examination and

(3) Platelet response to classic ITP therapy (usually intravenous immunoglobulin, IV anti-D, and possibly steroids).

Our patient met the first two of the above-mentioned criteria for ITP; this supports the diagnosis of ITP in our patient

It has been documented that thrombocytopenia in tuberculosis can occur due to immune mediated platelet destruction, tuberculosis induced Haemophagocytic syndrome, side effect of anti-tuberculosis therapy& defect in platelet production. Our patient was not exposed to anti-tubercular therapy before admission, secondly patient didn't show signs of marrow infiltration with tuberculosis, thirdly Haemophagocytic syndrome has already been ruled out, so most suitable cause is immune mediated platelet destruction which itself is very rare.[10] So to conclude Tuberculosis may manifest as diverse hematological abnormalities including ITP and management of secondary ITP requires identification & treatment of underlying cause.

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