PICTORIAL CME

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Severe Immune Thrombocytopenic Purpura with Life-threatening Bleeds: A Rare Presentation of Disseminated Tuberculosis

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Introduction

Immune Thrombocytopenic Purpura (ITP) is an acquired autoimmune disorder characterised by isolated thrombocytopenia due to accelerated platelet destruction and impaired platelet production. While ITP is often idiopathic, secondary causes such as autoimmune diseases, malignancies, viral infections, and, rarely, tuberculosis (TB) have been reported. Herein, we present a rare case of severe ITP secondary to disseminated tuberculosis causing life-threatening bleeds which showed dramatic improvement following anti-tubercular therapy (ATT) and steroids[1].

Case Report

A 33-year-old male presented to the emergency department with the complaints of multiple rash over the body for 7 days, black coloured stools for 5 days and 1 episode of generalised tonic clonic seizure. There was history of significant weight loss and anorexia. The patient had tachycardia with a pulse rate of 110/min; blood pressure was 96/60 mm of Hg and the patient was restless and agitated. General physical examination revealed pallor and petechial rash over chest (Fig. 1). Blood investigations revealed low haemoglobin 4.5 g/dL, MCV of 64 fl, thrombocytopenia with a platelet count of 3,000 per cu mm and iron deficiency anaemia with a serum iron of 10 micrograms/dL. Peripheral smear revealed microcytic hypochromic anaemia with severe thrombocytopenia. Human immunodeficiency virus (HIV), Hepatitis B surface antigen (HBsAg), Hepatitis C virus antibody test and antinuclear antibody test were negative. Non contrast CT head revealed left frontal intracerebral haemorrhage (Fig. 2). Chest X-ray revealed widened mediastinum with ill-defined bilateral costophrenic angles (Fig. 3). Upper GI endoscopy revealed erosive gastropathy (Fig. 4). CECT chest and whole abdomen revealed "tree-in-bud" appearance with multiple enlarged conglomerated mediastinal, left-sided pleural effusion, periportal and peripancreatic nodes along with moderate ascites and omental thickening (Fig. 5). Ascitic fluid anothers was done which was lymphocyte

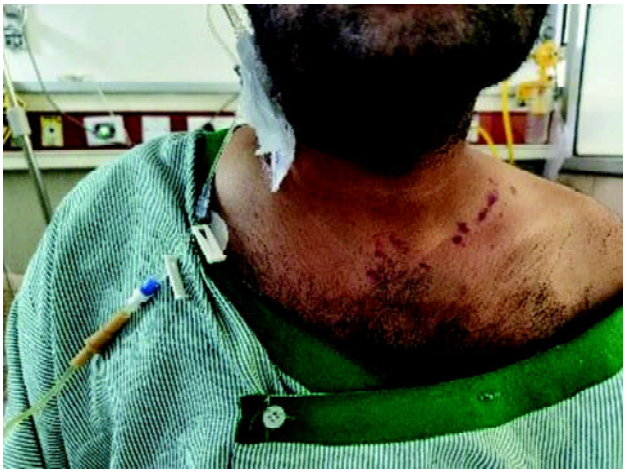

Fig. 1: *Petechial rashes over chest.*

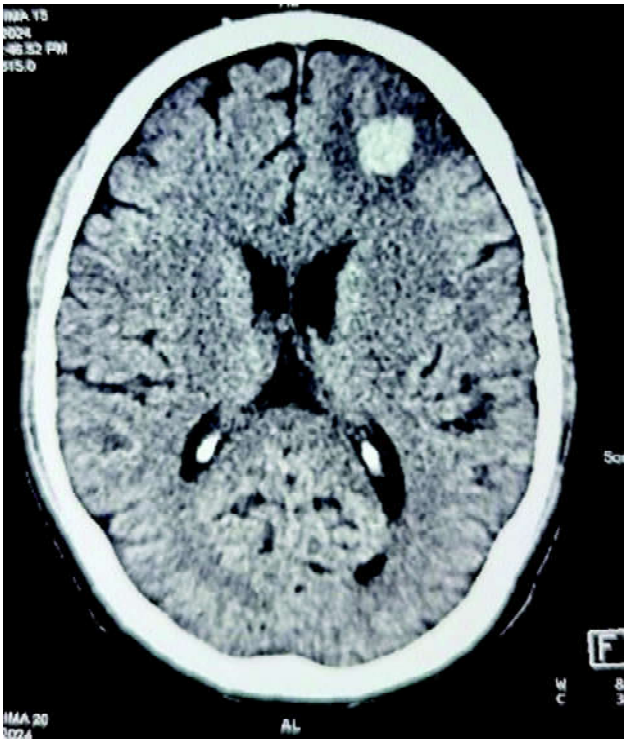

Fig. 2: *CT head showing left frontal ICH.*

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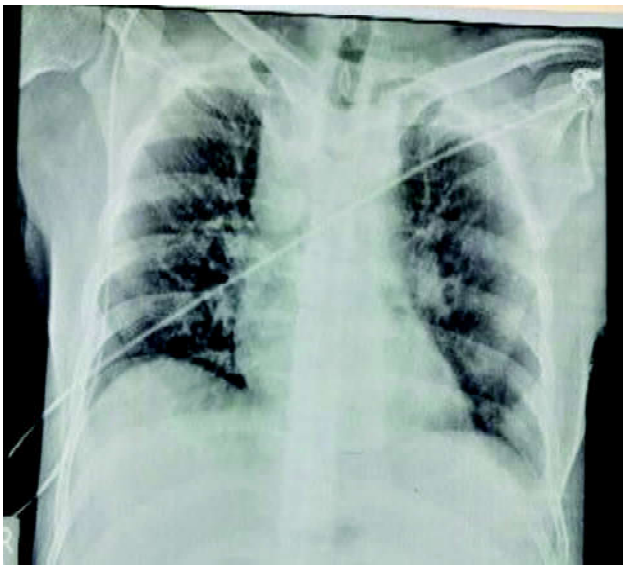

Fig. 3: *CXR showing mediastinum widening.*

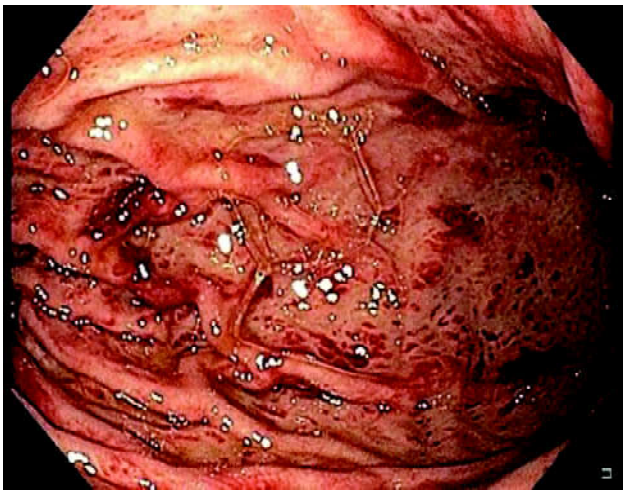

Fig. 4: *Erosive gastropathy on upper GI endoscopy.*

rich exudative fluid with a serum ascites albumin gradient of 0.72 gm/dL with raised adenosine deaminase (ADA) levels of 81.1 U/L. Bone marrow biopsy showed erythroid hyperplasia and was negative for GeneXpert.

A diagnosis of Severe Immune Thrombocytopenia (ITP) secondary to disseminated tuberculosis causing intracerebral haemorrhage and severe gastrointestinal bleed was made. The patient was initially treated with pantoprazole infusion, levetiracetam, IV immunoglobulins 1 gm/kg (for 1 day) along with PRBC and SDP transfusion Intracerebral bleed was managed conservatively. After diagnosis confirmation, weight based anti-tubercular therapy (isoniazid/rifampicin/pyrazinamide/ethambutol) was started along with steroids at 1 mg/kg. On subsequent follow-up visits, platelet count increased and steroids were

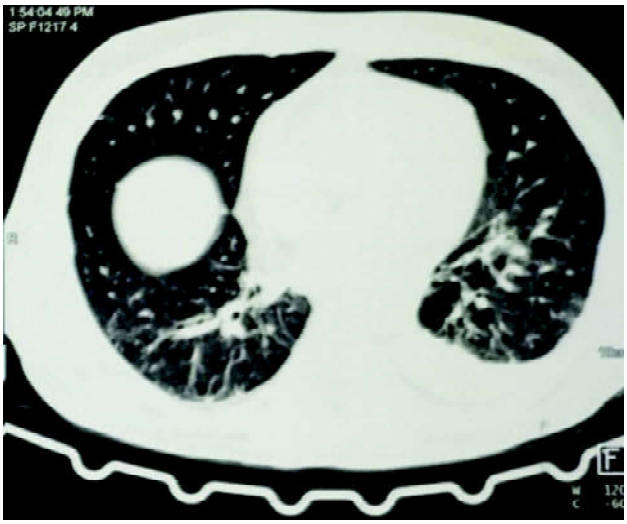

Fig. 5: *CECT Chest showing left pleural effusion.*

tapered over 6 weeks. On 10th day from discharge, the platelet count was 90,000 per cu mm and on 30th day from discharge, the platelets were 2.33 lakh per cu mm. A follow-up scan of chest and brain after 6 months showed resolution of earlier findings with normal platelet count.

Discussion

Tuberculosis-associated ITP (TB-ITP) is uncommon, with only scattered reports in the literature. The pathogenesis remains speculative but is thought to involve immune dysregulation, with the formation of anti-platelet antibodies possibly triggered by *Mycobacterium tuberculosis* antigens[1]. These immune responses may mimic idiopathic ITP but typically occur in the setting of active or latent TB infection. The clinical presentation may range from asymptomatic thrombocytopenia to bleeding manifestations; however, life-threatening bleeding, as seen in our case is very rare[2].

Diagnosing ITP secondary to TB requires careful exclusion of other causes of thrombocytopenia, including marrow infiltration, disseminated intravascular coagulation, drug-induced cytopenias, and hypersplenism. Bone marrow examination in such cases usually shows normal or increased megakaryocytes, ruling out marrow failure[3]. A key differentiating feature is the lack of sustained response to conventional ITP therapies such as corticosteroids or IVIG alone, unless ATT is co-administered[4].

The management of TB-associated ITP involves prompt initiation of ATT, which often leads to spontaneous haematologic recovery. Adjunctive corticosteroids or IVIG may be necessary in cases of severe bleeding or critically low platelet counts[5]. Our patient's platelet counts showed sustained improvement only after introduction of ATT along

with steroids, underscoring the importance of treating the underlying infection.

This case reinforces the need for clinicians to maintain a high index of suspicion for TB in endemic regions when evaluating patients with ITP. Early diagnosis and appropriate therapy can prevent serious complications and unnecessary prolonged immunosuppression.

References

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