

Case report

Tuberculous lymphadenitis associated thrombocytopenic purpura, effectiveness of anti-tuberculous therapy



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Abstract

The association of immune thrombocytopenic purpura and tuberculosis is extremely rare. We presented a case with tuberculous lymphadenopathy and immune-mediated thrombocytopenia, the complete remission with anti-tuberculous therapy, and partial response to immunoglobulin and steroid suggested a causal relationship between the two conditions.

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Introduction

Immune thrombocytopenic purpura is a diagnosis of exclusion, but many disorders like retroviral infections, autoimmune disorders, lymphoproliferative disorders, and primary immunodeficiency have been associated with this idiopathic disease. In children, the presentation is usually acute, but it can run a chronic course in adults (more than six months). Remission is considered if the platelets rise to 150×10^9 , while a response is defined as platelets count above 50×10^9 or if there is a two-fold increase from the initial value [1,2]. Tuberculosis has re-emerged as a major health problem worldwide and can cause various hematological manifestations. Literature review documented few cases with isolated thrombocytopenia associated with tuberculosis [3,4]. We present a case of tuberculous lymphadenopathy with isolated thrombocytopenia.

Patient and observation

A twenty-one-year Indian male shepherd presented to the emergency department with nose and gum bleeding for 2 days and skin rash on lower extremities for one day. The patient also reported painful, gradually increasing swelling on his lateral side of the upper neck during the last month associated with fever, chills, unexplained loss of weight, and sputum with streaks of blood and no bleeding from other sites. Systemic inquiries were unremarkable as were past (no history of hematological, rheumatic, or liver diseases) and family history, he was not taking any medications except for paracetamol. The patient is not consuming alcohol, recreational drugs, and not used to smoke.

The patient looks ill, oriented, and cooperative. Pallor was noted as were bleeding from gum, crust in his nostrils and extensive purpuric rash in his both lower limbs below the knee. There was no jaundice or cyanosis. Vital signs: Pulse 90/minute, blood pressure 126/65, respiratory rate 18/minute, and temperature 37.8. A lymph node 5×4 cm tender, firm, smooth surface, well defined, and mobile was found in the right submandibular region. Other lymph nodes were not palpable as were the liver and spleen.

Admission full blood count showed: platelets of 5000, hemoglobin 9.9 grams/dl, and white cell count of 6.090. Neutrophils 3.41, lymphocytes 1.75, monocytes 0.46, and eosinophils 0.47, MCV 69.3, HCT 33.9, MCH 22.9, MCHC 33, RDWSD 39.2, and RDWCV 16. Investigations including liver (bilirubin, liver enzymes and serum albumin) lactate dehydrogenase (LDH), sputum for Acid Alcohol Fast Bacilli (Aafb), chest X-ray, abdominopelvic ultrasound, Combs' s test, renal functions, hepatitis B surface antigen Hepatitis C serology, HIV serology, antinuclear antibodies, PT, and APTT were all normal. The high sensitive C-Reactive Protein (CRP) was 13.2g/l (0-0.3).

The blood picture suggests peripheral destruction. The patient started on methylprednisolone and immunoglobulin 40mg/kg body weight with partial improvement. Thus bone marrow biopsy was taken and showed: Normal erythrocyte series, leukocytes were normal in morphology and maturation with the blast in reasonable limits and reactive plasma cells, megakaryocytes were adequate with immature forms (Figure 1, Figure 2).

Excisional lymph node biopsy after unremarkable ENT and maxillofacial consultation was taken and revealed non-caseating granuloma diagnostic of tuberculous lymphadenopathy (Figure 3) and the patient was started, isoniazid, ethambutol, pyrazinamide, pyridoxine, and ciprofloxacin in the usual doses, with dramatic

improvement, rifampicin was then introduced in place of ciprofloxacin. The discharge full blood count was: TWBCs 5800, hemoglobin 12g/dl, and platelets of 155000. The patient is now on regular follow up and is off specific treatment for immune thrombocytopenic purpura.

Discussion

Immune thrombocytopenic purpura may require treatment with a steroid, immunoglobulins, and immunosuppressive therapy. Seventy percent of patients respond to treatment while a complete cure is observed in 20% of cases [1]. Tuberculosis can reduce platelets by various mechanisms: Antituberculous medications like rifampicin, ethambutol, and pyrazinamide, bone marrow involvement (granulomatosis, fibrosis, amyloidosis, and necrosis), hemophagocytic syndrome, hypersplenism, thrombotic thrombocytopenic purpura, and intravascular coagulation [5-11]. This patient showed a partial response to prednisolone and immunoglobulin, while he was completely cured with anti-tuberculous therapy and after three months not required specific treatment for ITP or splenectomy. Among the few cases reported in the literature with ITP in patients with complete tuberculosis, a response was observed during the first two months because the mycobacterium can remain alive during this period. Thus a causal relationship had been suggested, but the mechanism remains unknown [12-14].

In this report, the diagnosis of immune-mediated thrombocytopenia was diagnosed based on the following: 1) no previous drug therapy that can cause thrombocytopenia; 2) HIV, hepatitis viruses which can cause thrombocytopenia were excluded by the absence of clinical signs and negative investigations; 3) Lymphoproliferative and immunological disorders were not confirmed clinically together with normal bone marrow and negative anti-nuclear antibodies; 4) The healthy bone marrow also was against tuberculous infiltration and excluded hemophagocytic lymphohistiocytosis that had been previously linked to tuberculosis and can cause thrombocytopenia and anemia due to uncontrolled activation of T cells and macrophages, and an overproduction of inflammatory cytokines [15].

The complete response to anti-tuberculous medication without the need for more specific medications for ITP and only partial response to these drugs confirm the causal relationship between ITP and tuberculosis.

Conclusion

Tuberculosis is a rare, treatable cause of thrombocytopenia that should be sought of especially among challenging to treat thrombocytopenia in the context of the clinical scenario. Relapse during treatment is not uncommon during the first months of treatment. Close monitoring and continuation of the particular therapy are of paramount importance.

Competing interests

The authors declare no competing interests.

Authors' contributions

All the authors contributed equally in the data collection and the drafting of the manuscript. All the authors read and agreed to the final manuscript.

Figures

Figure 1: Bone marrow biopsy of the case report

Figure 2: Bone marrow biopsy 40 magnification

Figure 3: Lymph node tuberculosis of the reported case

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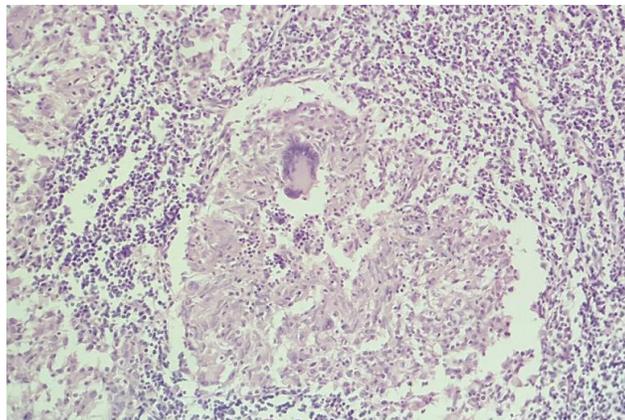


Figure 1: Bone marrow biopsy of the case report

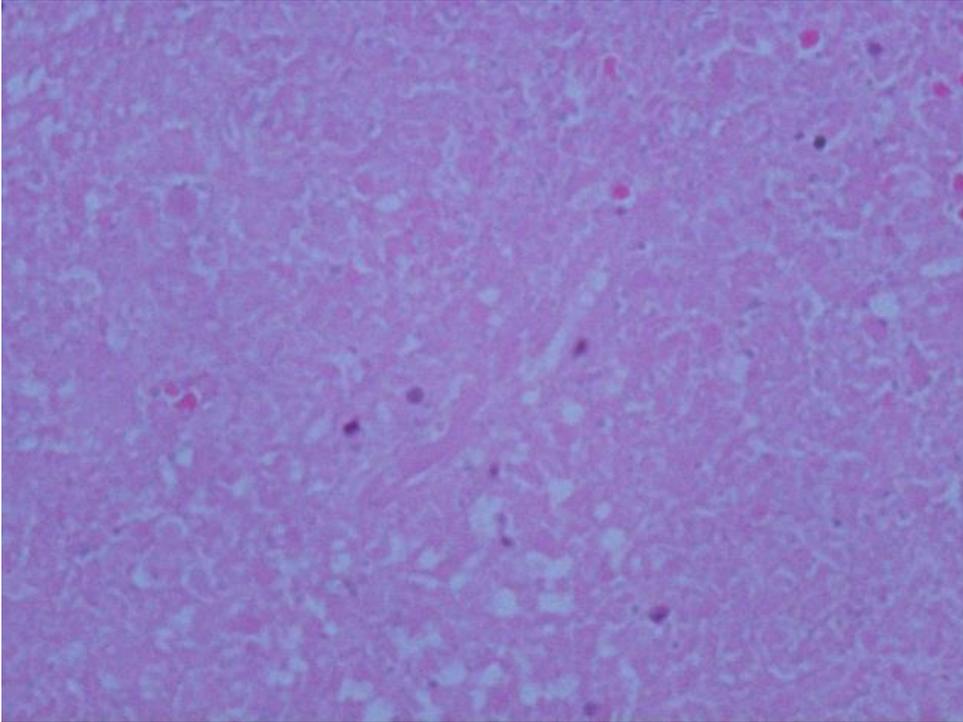


Figure 2: Bone marrow biopsy 40 magnification

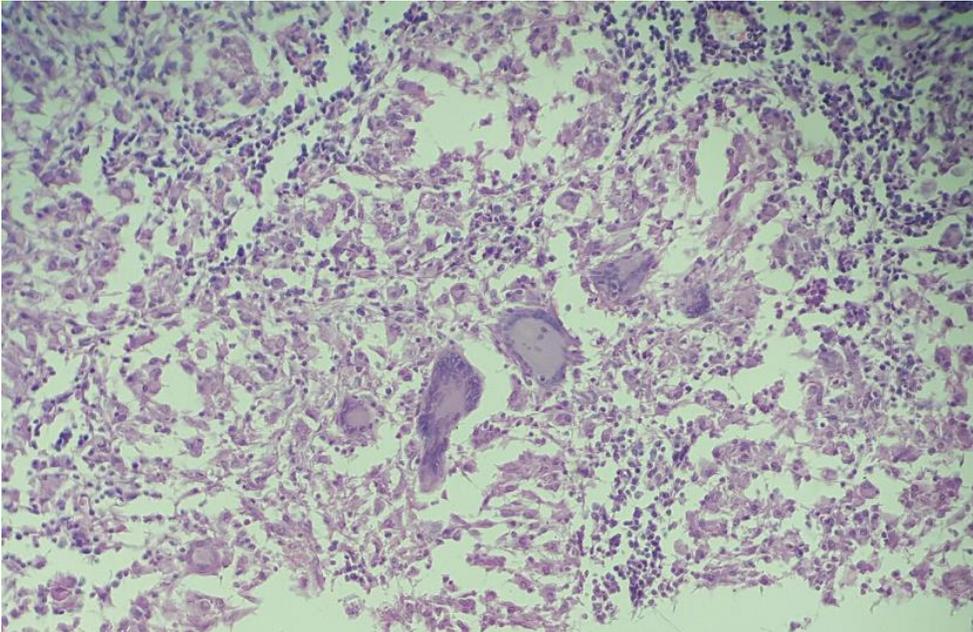


Figure 3: Lymph node tuberculosis of the reported case