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A Rare Case of Immune Mediated Hemolytic Anemia-Tuberculosis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Tuberculosis is one of the oldest diseases known to mankind. The disease still puzzles us with its varied clinical presentations and complications. Though tuberculosis is known to have many hematological manifestations, auto immune hemolytic anemia is extremely rare in tuberculosis. Here we report aninteresting case of tuberculosis presenting with auto immune hemolytic anemia. The treatment withanti tuberculous therapy is enough for the managing tuberculosis associated auto immune hemolyticanemia.

Keywords: Tuberculosis; haemolytic anemia; pleural effusion.

1. INTRODUCTION

Tuberculosis is one of the oldest diseases known to mankind. With improved treatment and surveillance methods though the incidence of tuberculosis is coming down , we see a lot of atypical presentations of tuberculosis . A variety of hematological abnormalities are reported in tuberculosis commonest being normochromic normocytic anemia [1]. The severity of anemia correlates with the severity of tuberculosis and severe anemia increases the morbidity and

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mortality of tuberculosis [2]. Tuberculosis has varied clinical presentations and complications. Though anemia is a common hematologic manifestation of tuberculosis, auto immune haemolytic anemia is a rare manifestation of tuberculosis. We report a case of tuberculosis which presented with auto immune hemolytic anemia and pneumonia.

2. CASE HISTORY

47 year old female patient with history of systemic hypertension, hypothyroidism and poorly controlled diabetes presented with complaints of cough, modified medical research council(mMRC) grade 3 breathlessness and low grade fever for 2 weeks. The cough was non productive and she complained of left sided chest pain on coughing. She also had easy fatigability and excessive sweating. She had history of abnormal uterine bleeding in past. On examination she was obese with moderate pallor and bilateral pitting pedal edema. Systemic examination was normal except for reduced breath sound on left infrascapular region and mild splenomegaly.Laboratory findings on admission were as follows: hemoglobin 7.3g/dL, total WBC count 11200/mm 3 (81% neutrophils and 9.9% lymphocytes), platelet count 338,000/ mm 3, MCV 64.9FL, MCH 20.6 PG, and reticulocyte 0.8%. ESR was 60mm.Peripheral smear showed microcytic hypochromic with severe anisopoikilocytosis with presence of elliptocytes, tear drop cells, with neutophilia occasional hyper-segmented polymorphs and platelets were adequate. Liver function test and renal function was normalChest X-ray showed left lower zone non-homogenous opacity. High resolution computed tomography chest showed confluent areas of consolidation along with cavitation and few nodular opacities seen in left upper lobar and lingula ,focal areas of consolidation seen in basal segment of left lower lobe, tree in bud nodular opacities in the right lung field, with mild left sided pleural effusion suggestive of tuberculosis. On day 2 of admission complete blood count was repeated which showed a drop in hemoglobin to 5g/dl. Direct Coomb's test was done to rule out autoimmune hemolysis and was positive. Peripheral smear was repeated for schistocytocytes and was negative. Serologic tests for antinuclear antibodies, human immunodeficiency virus, mycoplasma, hepatitis B and C virus were negative, serum LDH was 1145U/. Repeat liver function tests showed unconjugated hyperbilirubinemia. Mantoux test was negative.Ultrasound of abdomen and thorax revealed mild splenomegaly and mild left sided pleural effusion. Diagnostic thoracocentesis showed exudative neutrophil predominant effusion with ADA 22 U/L. No acid bacilli detected in pleural fluid. Bronchoalveolar lavage fluid detected acid fast bacilli and patient was started on antitubercular drugs. On follow up after 1 month with ATT and iron supplementation patients haemoglobin improved to 9.2g/dl, Coomb's test was negative. This case report is to emphasize the effectiveness of anti tuberculosis treatment alone to correct autoimmune hemolytic anemia in tuberculosis.

3. DISCUSSION

Tuberculosis is one of the major public health problem. With the advent of HIV we see a rise in the incidence of tuberculosis cases. As we are aiming for eradication of tuberculosis we come across a lot of atypical presentations. Hematological abnormalities are reported in tuberculosis, though not extensively studied. Pulmonary tuberculosis is more commonly associated with hematological abnormalities than extra pulmonary tuberculosis [3]. The most common one seen is normocytic normochromic anemia, others are leukopenia, leukocytosis, lymphocytopenia, lymphocytosis, monocytopenia, monocytosis, neutropenia [3]. Pancytopenia and thrombocytopenia are seen in disseminated or miliary tuberculosis. Studies say that these hematological abnormalities in tuberculosis have both diagnostic and prognostic significance and they may indicate the complications and response to treatment [2].

The mechanism of anemia in tuberculosis is postulated to be IFN Ɣ, TNFα, IL1, IL6 and other cytokines (from activated lymphocytes and macrophages) mediated diversion of iron into reticulo-endothelial system causing an apparent iron deficiency. They also inhibit erythroid proliferation and production and action of erythropoietin [4]. Other causes are nutritional deficiencies, malabsorption in intestinal tuberculosis, or associated anorexia.

Autoimmune hemolytic anemia occurs when auto antibodies coat and lyse RBC causing acute anemia. It can be warm antibodies, cold antibodies or mixed depending on the temperature at which these antibodies are active [5]. Primary auto immune hemolysis is rare compared to secondary, which occurs secondary

to infections or lympho-proliferative disorders or connective tissue disorders or drugs [5].

4. CONCLUSION

Auto immune hemolytic anemia is extremely rare in tuberculosis [6]. The altered immune response seen in tuberculosis is primarily implicated in pathogenesis [7]. Tuberculosis is reported to be associated with warm or cold or mixed auto antibodies. It is important to recognize secondary auto immune hemolytic anemia as treatment with immunosuppressive agents will worsen the infection and hence anti tuberculous drugs is the definitive treatment [8]. The normalization of clinical and laboratory parameters with anti-tuberculosis treatment gives us an opportunity to list tuberculosis as one of the infectious causes of auto immune hemolytic anemia.

CONSENT AND ETHICAL APPROVAL

As per university standard guideline, participant consent and ethical approval have been collected and preserved by the authors

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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