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Case Series

Immune cytopenia: an uncommon presentation of a common disease tuberculosis - a case series and literature review

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ABSTRACT

Various haematological abnormalities such as anaemia, leucocytosis, and pancytopenia are described in patients with tuberculosis. These often occur due to non-immunologic mechanisms. Here, we present a series of three paediatric cases with immune–mediated thrombocytopenia or haemolytic anaemia, in the setting of mycobacterial infection. This is an unusual presentation of tuberculosis (TB), with only a few published paediatric case-reports. Three previously healthy children, between 5 to 11 years of age, presented with epistaxis, generalised petechiae, pallor and lymphadenopathy. There was no hepatosplenomegaly or bone tenderness. Laboratory investigations and bone marrow aspiration suggested megakaryocytic thrombocytopenia in all the three patients. Additionally, the first two cases had Coomb's positive haemolytic anaemia. None of them showed a sustained clinical and haematological response to platelet transfusions and steroid therapy. Further work-up revealed histopathological, radiological and microbiological evidence of tuberculosis. The diagnosis of immune cytopenia secondary to TB was thus established. Anti-tubercular treatment (ATT) was started. All patients gradually demonstrated clinical and haematological improvement within four weeks of ATT. Follow-up at 1 year showed complete recovery in each case. Being a treatable cause with a favourable outcome, TB should be included in the etiological investigation of autoimmune cytopenia in childhood, especially in countries with high TB burden.

Keywords: Immune thrombocytopenic purpura, Autoimmune haemolytic anaemia, Tuberculosis, Lymphadenopathy

INTRODUCTION

Tuberculosis with its high mortality and morbidity is a major public health challenge worldwide, particularly in developing countries. According to the World Health Organization statistics, India shares the highest percentage (26%) of global epidemiological burden of tuberculosis, with an estimated 2.6 million newly diagnosed cases in 2019.[1] Principally a disease of the lungs but, in the extra-pulmonary form, it can affect almost any organ in the body. A wide spectrum of haematological abnormalities such as anaemia, leucocytosis, monocytosis, lymphopenia, leukopenia, thrombocytopenia, thrombocytosis, leukemoid reaction and pancytopenia have been described previously in mycobacterial infection.[2,3] These occur via non-immunologic mechanisms secondary to granulomatous infiltration of the bone marrow, hemophagocytic syndrome, hypersplenism, malabsorption, nutritional deficiency, or as a side- effect of anti-tubercular therapy.[3] Immune-mediated cytopenia, in tuberculosis is, however, uncommon; with only a few published paediatric case-reports.[4-20] To the best of our

knowledge, this is the first such paediatric case series from India highlighting an unusual but treatable manifestation of a common disease.

CASE SERIES

This is a case-series study carried out in the department of paediatrics at Guru Teg Bahadur Hospital, a tertiary care centre in North India. Ours is a retrospective study, which included three cases of paediatric tuberculosis (pulmonary or extra-pulmonary) complicated by immune thrombocytopenic purpura (ITP) alone or in combination with autoimmune haemolytic anaemia (AIHA); which resolved with anti-tubercular therapy. The patients presented to our institute between February 2017 and March 2020. Demographic details, medical and laboratory records, radiological and microbiological results were retrieved from the hospital database. These cases were also compared with similar published cases in the literature.

Case 1

A 5-year-old male child, previously healthy, was admitted with six episodes of intermittent spontaneous epistaxis, copious in amount, for past 1 month. There was no history of fever, cough, headache, blurring of vision, jaundice, joint pain, weight loss, bleeding from any other site, and abnormal body movements or weakness. Dietary intake showed a deficit of 500 calories and 4 grams of protein by 24-hour recall method. There was no past history suggestive of haematological or liver disease, or recent medications. Patient was immunized for age, and family history was non-contributory. He weighed 14 kg (10^{th} percentile) and his height was 103 cm (5^{th} percentile) as per Indian academy of paediatrics (IAP) growth charts. Vitals were within normal range. He had pallor, active nasal bleed and petechiae at the time of presentation. There was no icterus, rash, sternal tenderness, lymphadenopathy or hepatosplenomegaly. Systemic examination was normal.

Complete hemogram on day 1 of hospitalization showed severe anaemia (haemoglobin 6 g/dl) and severe thrombocytopenia (platelet count 20×10^9/l) with normal total leucocyte count (TLC) (8×10^9/l, 64% neutrophils, and 36% lymphocytes). Peripheral blood smear depicted paucity of platelets (Figure 1a). Erythrocyte sedimentation rate (ESR) was slightly elevated (30 mm/hour). Coagulation profile (prothrombin time and activated partial thromboplastin time), liver and renal function tests were within normal limits. There was no evidence of bleed elsewhere in the body (normal fundus, urine negative for red blood cells). Chest X-ray on admission showed mediastinal widening. Ultrasound abdomen was normal. We considered the differential diagnosis of aplastic anaemia, megaloblastic anaemia and immune thrombocytopenia. Bone marrow aspiration done on day 2 of hospitalization revealed normal cellularity, normal maturation of myeloid and erythroid precursors, increased megakaryocytes with clusters of both mature and immature forms suggestive of megakaryocytic thrombocytopenia and thus ruling out production defect. In addition, direct Coomb's test was positive, suggesting immune-mediated haemolysis as a cause for anaemia. A repeat complete blood count on day 2 of admission showed hemoglobin of 7 gm/dl and platelet count further decreased to 8×10^9/l. Platelet transfusion was given. Considering the immune - mediated consumption as the underlying cause of bicytopenia, patient was started on oral prednisolone at 4 mg/kg/day. However, intermittent epistaxis continued, fresh petechiae appeared and abnormal blood parameters persisted even after 4 days of steroid therapy. This posed a diagnostic dilemma and further investigations were planned. Anti-nuclear antibodies (ANA) titres, serological tests for hepatitis B virus, hepatitis C virus, HIV, and Epstein-Barr virus were negative. As the chest X ray had depicted mediastinal widening, the patient was subjected to a contrast enhanced computed tomography (CECT) of chest which showed pre-tracheal, right para tracheal and pre-carinal lymphadenopathy. In addition, lymph nodes in right bronchial region were calcified. Bilateral lung fields were unremarkable. Endoscopic ultrasound with bronchoscope-guided fine needle aspiration biopsy (EUS-B-FNA) of right para tracheal lymph node demonstrated large areas of granulomatous reaction. Granulomas were composed of epithelioid cells, with central areas of caseous necrosis in few foci (Figure 1b). Cartridge based nucleic acid amplification test (CB-NAAT, GeneXpert) for tuberculosis was detected positive in the biopsy specimen. Gastric aspirate for acid fast bacilli was negative. Mantoux test was reactive with 18 mm induration. Case 1 was thus diagnosed with tubercular lymphadenitis with secondary immune thrombocytopenia and autoimmune haemolytic anaemia based on laboratory, radiological, histopathological and microbiological findings. Anti-tubercular treatment (ATT) with 4 drugs (isoniazid, rifampicin, pyrazinamide and ethambutol) was initiated for 2 months as daily regimen followed by 4 months of isoniazid, rifampicin and ethambutol. Steroids were tapered and stopped over 1 week. Patient improved symptomatically within 2 weeks of ATT. Haematological parameters nearly normalised after 4 weeks of anti-tubercular therapy (hemoglobin - 8.2 gm/dl and platelet count - 180×10^9/l). The patient remained well at 1-year follow up.

Case 2

An 11-year-old female child, not known to have any chronic medical illness, had bilateral multiple neck swellings, which were painless and gradually progressive for past 1 year. She complained of fatigue and breathlessness on exertion for past 3 months. There was history of low-grade intermittent fever and productive cough with scant expectoration for 15 days. She had one episode of epistaxis, spontaneous and copious, one day prior to admission. There was no other relevant history. She weighed 29 kg (50^{th} percentile) and height was 134 cm (5^{th} percentile). Physical examination showed pallor,

icterus, multiple petechiae and ecchymosis all over body, enlarged (largest 2×2 cm), multiple, discrete, firm and non-tender bilateral cervical lymph nodes. There was no hepatosplenomegaly or bone tenderness. She was afebrile with stable vitals at presentation. Lung auscultation was clear with equal breath sounds bilaterally. Other systemic examination revealed no abnormality.

Complete blood count on day 1 of hospitalization showed a haemoglobin of 6.6 g/dl, TLC 10×10^9/l (65% neutrophils, and 23% lymphocytes) and a significantly low platelet count of 21×10^9/l. Peripheral blood smear examination demonstrated markedly reduced number of platelets, shift to left with toxic granules in neutrophils and evidence of haemolysis in form of polychromasia, spherocytes, moderate anisocytosis, tear drop cells and nucleated RBCs 5/100. The reticulocyte count was 2%. C-reactive protein (13 mg/l, normal <10 mg/l and ESR were elevated (45 mm in first hour). Serum bilirubin was 3.0 mg/dl, predominantly unconjugated fraction. Coagulation profile (prothrombin time and activated partial thromboplastin time), liver enzymes, serum electrolytes, blood urea and serum creatinine were normal. Urine for red blood cells and stool for occult blood were negative. Chest radiograph showed mediastinal widening. Ultrasound abdomen was done to look for additional sites of lymphadenopathy, which revealed enlarged mesenteric lymph nodes, largest being 14 mm in diameter. Bone marrow examination was unremarkable except for increased number of megakaryocytes, compatible with immune mediated thrombocytopenia (Figure 2a and b). In addition, direct Coomb's test was positive, suggesting immune-mediated haemolysis as a cause for anaemia which was out of proportion to clinical bleed. Viral serology and ANA titres were negative. Hemogram on day 2 of admission showed hemogloblin of 4.8 gm/dl and platelet count of 18×10^9/l. Oral prednisolone was started at 4 mg/kg/day. Haemoglobin on day 4 of steroids was 5.8 gm/dl and platelets were persistently low at 18×10^9/l. The patient was transfused one unit of packed red cells. On further evaluation, HRCT chest identified multiple, enlarged (largest was 14 mm in diameter), conglomerated, necrotic cervical and mediastinal lymph nodes with patchy bronchiectasis in apico-posterior segment of left upper lobe (Figure 3). Fine needle aspiration cytology (FNAC) of cervical lymph node showed foci of caseous necrosis. The cytology specimen tested positive for acid fast bacilli. Mantoux was 12 mm induration after 48 hours. The findings led to a diagnosis of pulmonary Koch's with tubercular lymphadenitis with secondary immune thrombocytopenia and autoimmune haemolytic anaemia. After completing the six months of anti-tubercular treatment, general well-being improved, bleeding episodes ceased, blood counts were within normal range with no evidence of haemolysis. Lymph node size decreased significantly during follow-up at 6 months and 1 year.

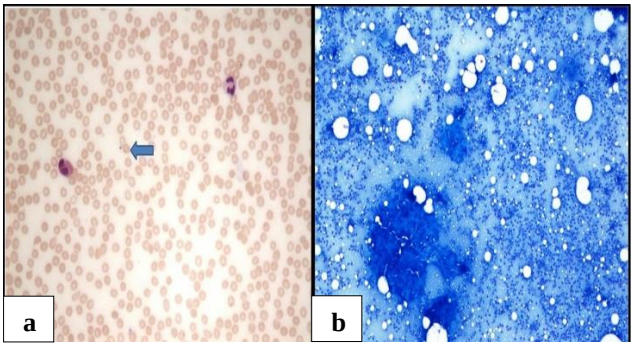

Figure 1: (a) Peripheral blood film (Wright stain 400 X) in case 1 shows reduced number of platelets (blue arrow) and (b): Biopsy mediastinal lymph node (May Grunwald-Giemsa [MGG] 400 X) in case 1 shows multiple epithelioid cell granulomas in a blood mixed lymphoid background..

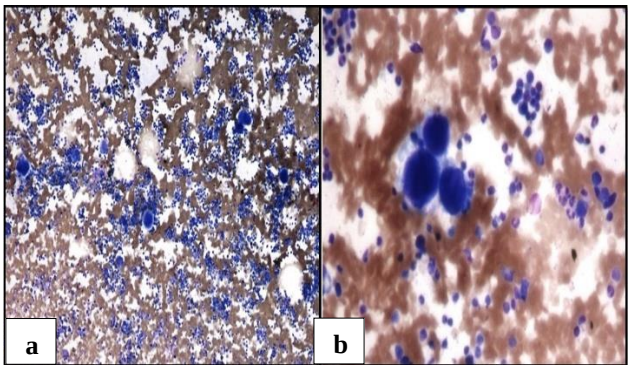

Figure 2: (a) Bone marrow aspirate (Wright's stain 400 X) in case 2, and (b) an immature megakaryocyte in zoom view (Wright's stain 400 X).

Case 3

A 9-year-old female patient, with no past medical history, presented with complaints of left cervical swelling for past 1 year, which was single, painless, insidious in onset and gradually progressive; and 1 episode of spontaneous and profuse epistaxis 1 week back. She had history of fatigue, loss of appetite and malaise for 6 months. There was no history of fever, cough, dysphagia or odynophagia, weight loss, joint pain, jaundice, gum bleed, hematemesis or melena, any recent travel or drug intake. Family history was non-contributory. Physical examination showed a single, enlarged (5×5 cm), firm, non-mobile and non-tender left cervical lymph node. She was afebrile, no evidence of chronic liver disease, no bone tenderness or hepatosplenomegaly. Blood examination on day 1 of hospital admission showed a haemoglobin level of 9.7 g/dl, a white blood cell count of 6,930 cells/mm^3 (65% neutrophils, 30% lymphocytes, 3% monocytes, 2% eosinophils), and a platelet count of 32×10^9/l. Peripheral blood film examination showed thrombocytopenia. Routine investigations including liver and kidney function tests, coagulation profile and chest radiograph were normal. Ultrasound abdomen revealed no abnormality. In

view of thrombocytopenia and peripheral lymphadenopathy, bone marrow aspiration was conducted which demonstrated normal number, maturation and morphology of all cell lines. ANA titres and viral serology for hepatitis B, hepatitis C, and HIV were negative. Considering the diagnosis of immune thrombocytopenia purpura, oral prednisolone was started at a dose of 4 mg/kg/day. A repeat complete blood count on day 4 of steroid therapy showed no improvement in platelet count. Meanwhile, cytological examination (FNAC) reports of left cervical lymph node were available which demonstrated epithelioid granulomatous lesions with foci of caseous necrosis. CB-NAAT for tuberculosis was positive in the cytology specimen. Mantoux test showed 16 mm induration at 48 hours. Final diagnosis of generalised tubercular lymphadenitis with secondary immune thrombocytopenia purpura was thus established. ATT was given for 6 months. Steroids were gradually stopped. Follow-up hemogram at 6 months showed resolution of thrombocytopenia (platelet count - 205×10^9/l). At one-year follow up, cervical lymph node had significantly reduced and patient was doing well.

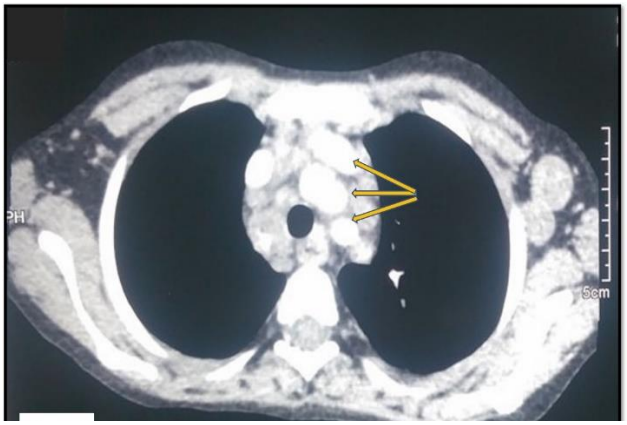

Figure 3: HRCT chest axial view in case 2 shows multiple, enlarged, conglomerated cervical lymph nodes (yellow arrows).

DISCUSSION

Immune-mediated cytopenia represent excessive destruction of normal blood cells by a misfiring immune system.[21] Isolated or combined, these are extremely rare manifestations of tuberculosis.[22] Each of our three cases presented with mucocutaneous bleeding, thrombocytopenia and lymphadenopathy. In addition, the first two cases had severe anaemia. Peripheral blood smear in case 2 showed evidence of haemolysis. Given atypical presentation, bone marrow examination was conducted in each patient which suggested megakaryocytic thrombocytopenia (case 1 and 2) or normocellularity (case 3), ruling out any production defect, lymphoproliferative disorders or hemophagocytic syndrome. Negative viral serology and normal ANA titres excluded other secondary causes of low blood counts like HIV infection and systemic lupus erythematosus (SLE). Normal coagulation profile ruled out disseminated intravascular coagulation (DIC).

Absence of hepatosplenomegaly made sequestration an unlikely cause, and there was no prior drug intake. Direct antiglobulin (Coomb's) test was positive in first two cases. Thus, immune mediated cellular destruction was considered as the underlying mechanism for bicytopenia (Evan's syndrome) in cases 1 and 2 cases and isolated thrombocytopenia in case 3. In view of persistent low counts, steroids were started as a standard therapy. However, patients showed poor clinical and haematological response to steroids. In view of peripheral lymphadenopathy, our patients were simultaneously evaluated for tuberculosis. There was radiological, microbiological and cytological or histopathological evidence of tuberculosis in all our cases. ATT was thus started in each patient, leading to resolution of symptoms, recovery of blood counts and an overall improved general well-being within 4 weeks. These results suggested a causal relationship between tuberculosis and ITP and AIHA in our case series.

There is a paucity of pathophysiological and epidemiological data on TB-associated autoimmune cytopenia. It has been postulated that *Mycobacterium tuberculosis* could stimulate a clone of B lymphocytes that might produce antibodies against autologous platelets or antigens on erythrocyte membrane.[4,22] Another proposed mechanism is some molecular mimicry between tubercular antigens and platelet membrane surface proteins.[4]

Our literature search revealed only 12 paediatric case reports worldwide describing tuberculosis as a cause of isolated immune thrombocytopenia (Table 1). Additionally, only a handful of cases of TB-associated AIHA or Evan's syndrome have been reported in childhood globally (Tables 2 and 3).

Tables 1 to 3 demonstrate that 10 out of 17 identified cases of TB-related immune cytopenia (isolated or combined) in childhood were from India (nearly 60%). It is well known that women are more susceptible to autoimmune diseases compared to men. The literature review showed a clear gender bias with three-fourths of total cases being females, similar to our case series. Most commonly involved age group was 8-16 years, accounting for nearly 2/3rd of the reported cases. The youngest affected patient was a 2-year-old male child from USA diagnosed with isolated ITP secondary to tubercular cervical lymphadenitis.[4] In the published literature, lymph node TB (41%, n=7) was the most frequently reported site of TB, followed by lung (35%, n=6), and disseminated TB (18%, n=3). Among our patients, lymph nodes were the predominantly involved site of TB. The lowest platelet count of 2×10^9/l was seen in a 4-year-old Turkish boy with pulmonary TB.[10] Anti-platelet antibodies to support the immune basis for TB-induced ITP were detected in only 2 case-reports.[4,7] Due to unavailability, we could not assay antiplatelet antibody or

platelet-associated IgG although their absence does not invalidate the diagnosis of immune thrombocytopenia as per American Academy of Haematology guidelines.[23] The lowest haemoglobin of 1.8 g/dl was observed in an 8-year-old female with disseminated TB by Bakshi et al in 2004.[16] ITP and AIHA could be life threatening due to massive bleeding or severe haemolysis respectively. This led majority (13/17, 75%) of authors to initiate treatment with immunomodulatory therapy (IVIG and/or corticosteroids) and/or blood component transfusions (Tables 1-3). However, similar to our patients, all cases identified in literature showed a complete and sustained response only with anti-tuberculosis therapy, and no recurrence was observed after cessation of immune-therapy, thus strongly supporting the etiological role of TB in immune cytopenia.

Table 1: Summary of published paediatric case reports on isolated immune thrombocytopenia associated with tuberculosis.

#	Author/ Year	Country of report	Age (years)/ gender	Site of TB	Nadir platelet count (X 10^9/l)	Treatment
1	Jurak et al, 1983[4]	USA	2/ male	Cervical lymphadenitis	42	ATT only
2	Singh et al, 1986[5]	India	16/female	Cervical lymphadenitis	29	ATT + steroids
3	Bakhshi et al, 2003[6]	India	11/female	Cervical lymphadenitis	29	ATT only
4	Krishnamurthy et al, 2007[7]	India	8/female	Mediastinal lymphadenitis	21	ATT only
5	Verma et al, 2007[8]	India	13/female	Military TB with tubercular meningitis (disseminated)	18	ATT + steroids + platelet and packed RBC transfusion
6	Garrido-Colino et al, 2008[9]	Spain	4/female	Pulmonary	4	ATT + IVIg
7	Akyildiz et al, 2009[10]	Turkey	4/male	Pulmonary	2	ATT + IVIg
8	Borie et al, 2009[11]	France	16/female	Retro-peritoneal lymph node	< 30	ATT + steroids + IVIg
9	Tabarsi et al, 2010[12]	Tehran	17/female	Pulmonary	36	ATT + steroids + IVIg
10	Srividya et al, 2014[13]	India	16/male	Pulmonary	10	ATT + steroids + IVIg
11	Ramachandran et al, 2016[14]	India	10/female	Pulmonary	20	ATT + IVIg
12	Bharadwaj et al, 2018[15]	India	11/female	Cervical lymphadenitis	5	ATT + steroids + IVIg

Table 2: Characteristics of tuberculosis related autoimmune haemolytic anaemia reported in paediatric literature.

#	Author/ year	Country of report	Age (years)/ gender	Site of TB	Direct Coomb's test	Nadir haemoglobin (g/dl)	Treatment other than ATT
1	Bakshi et al, 2004[16]	India	8/female	Disseminated	+	1.8	Steroids, packed RBC transfusion
2	Gupta et al, 2005[17]	India	8/male	Abdominal	+	6	Steroids
3	Safe et al, 2013[18]	Brazil	18/female	Disseminated	+	6.6	Packed RBC transfusion

Table 3: Characteristics of tuberculosis associated immune bicytopenia (Evans syndrome) reported in paediatric literature.

#	Author/ year	Country of report	Age (years)/ gender	Site of TB	Direct Coombs test, anti-platelet antibodies	Nadir Hb (g/dl)	Nadir platelet count (X 10^9/l)	Treatment
1	Khemiri et al, 2008[19]	Tunisia	11/female	Pulmonary	+, -	7.9	5	ATT only
2	Kumari et al, 2019[20]	India	6/female	Cervical lymph node	+, not done	6	34.4	ATT, steroids, packed RBC transfusion

CONCLUSION

The exact pathophysiology of TB related immune cytopenia is unknown. However, in TB endemic regions like India, it is important that we recognize and consider tuberculosis as a treatable secondary cause of new onset ITP/AIHA in childhood. Treatment of the underlying mycobacterial infection alone can correct the abnormal blood counts and the associated manifestations which will not resolve with only immunomodulatory therapies. A high index of suspicion and awareness of the association is required.

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