Turkish Respiratory Journal 2008; 9(1): 51-2

Immune Thrombocytopenic Purpura: Presenting as a Rare Form of Tuberculosis

İsmail Hanta[1], Gülsüm Tezçağırır[1], Sedat Kuleci[1]

[1]Çukurova University, School of Medicine, Department of Chest Diseases, Adana, Turkey

Abstract

We report a 46 year-old male patient who admitted to our clinic with massive hemoptysis, epistaxis, and generalized petechiae. A diagnosis of immune thrombocytopenic purpura was established according to clinical and laboratory findings including bone marrow aspiration. Intravenous immune globulin (IVIg) and methylprednisolone therapy were started. In the folow-up, the patient was found to have clinical, microbiological and radiological evidence of active pulmonary tuberculosis. No platelet response was achieved to IVIG and methylprednisolone therapy and anti-tuberculous drugs were given. By the end of the first week, hemoptysis, epistaxis, and generalized petechiae resolved and platelet counts returned normal levels after four-weeks of treatment.

Key words: Immune thrombocytopenic purpura, treatment, tuberculosis

Received: 25.06.2007 Accepted: 21.11.2007

INTRODUCTION

Tuberculosis has been recognized as a public health burden of increasing proportions worldwide. Hematological manifestations such as anemia, leukocytosis, and pancytopenia have been previously described in tuberculosis [1,2]. When thrombocytopenia occurs, it does so most commonly via non-immunologic means, typically manifesting in the context of pancytopenia that develops secondary to granulomatous infiltration of the bone marrow [3]. However, immune thrombocytopenic purpura (ITP) in a patient with tuberculosis is exceedingly a rare event. There are few published cases with ITP and tuberculosis in English literature [1,2].

CASE REPORT

A 46 years-old healthy smoking male patient had previously cough and sputum for the last month. He admitted to our hospital with the symptoms of hemoptysis, epistaxis, and petechial lesions. In the physical examinations; fever: 36,6 ^{0}C, pulse: 88/min, blood pressure: 115/70 mmHg. He had petechial lesions through out the body and oral cavity. Thoracic examination was normal. He had no lymphadenopathy and organomegaly.

The initial complete blood cell count revealed a white blood cell count 10.7 x 109/l with 87% granulocytes, 7% lymphocytes, 3.4% monocytes, 1.7% eosinhophils, and 0.1% basophils, hemoglobin 10.9 g/dl with a MCV of 81.3, and platelet count 7.0 x 109/l. A peripheral smear was remarkable for a paucity of platelets. Routine biochemical parameters were normal (such as fibrinogen, blood urea nitrogen, creatinin level, aspartat aminotransferase, alanin aminotranferase). In the coagulation parameters; prothrombin time (PT), activated partial thromboplastin time (aPTT), fibrin degradation products (FDP) were normal. Antibody of salmonella, brucella, ANA, rheumatoid factor, HIV, HBsAg, Anti-HCV, repeated blood cultures were negative. A bone marrow biopsy showed hypercellularity of all cell lines with normal maturation of myeloid and erythroid precursors. Megakaryocytes were increased and had normal morphology. No granulomas were detected and culture for tuberculosis was negative.

A chest x-ray demonstrated cavitary and pneumonic lesions in right upper lobe, computerized thorax tomography showed massive pneumonic lesion in right upper lobe (Figure 1).

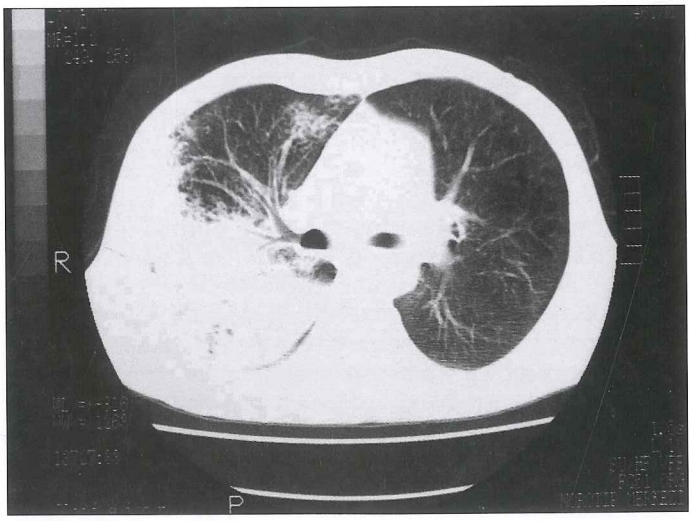

Figure 1. Cavitary and pneumonic lesions in right upper lobe are seen computerized thorax tomography

Corresponding Author: Ismail Hanta, MD, Çukurova University, Faculty of Medicine, Department of Chest Disease, 01330 Balcali, Adana, Turkey Phone: +90 322 338 68 51
E-mail: ihanta@cu.edu.tr

ITP has been thought in the patient due to clinical and laboratory findings. Due to suspicion of lung infection (specific or non-specific) IVIg therapy (0.5 mg/kg/per day) was started previously. Since there was no response to IVIg therapy after 3 days, methyl prednisolone (1 mg/kg/day) was also added later. However the hemoptysis and thrombocytopenia did not recover. As patients' chest x- ray revealed cavitary lesion, asido-resistant bacile examination induced sputum samples was found positive. And also culture positive test result was obtained for tuberculosis bacile. On the fifth day, anti-tuberculosis therapy was started (isoniazid 5 mg/kg/d, rifampin 10 mg/kg/d, ethambutol 25 mg/kg/d, pyrazinamid 30 mg/kg/d). IVIg and methylprednisolone treatment were cessated. Platelets count started to increase on the first week of the anti-tuberculosis therapy, his platelets count were normal by the end of first month (Table 1). Our patient completed a period of six monts for anti tuberculosis, and he has no recurrence of tuberculosis or thrombocytopenia.

Table 1. Treatment algorithm and whole blood cell count of the patient

	Hemoglobin	Hematocrit (%)	White blood cell	Platelet (x 10^9/1)	Treatment
1. Day	10,9	34,0	10,7	7	IVIg
2. Day	10,4	30,6	11,2	6	IVIg
3. Day	9,3	27,	9,5	4	IVIg+Methyl prednisolone
5. Day	9,1	26,7	8,7	9	Anti-TB
7. Day	8,7	25,0	9,2	18	Anti-TB
15. Day	10,2	31,8	9,6	74	Anti-TB
30. Day	12,4	36,6	10,5	158	Anti-TB

DISCUSSION

ITP accompanying to active tuberculosis infection had been reported very rarely. In English literature few cases reported as ITP due to tuberculosis [1,2].

ITP classically occurs in two forms: 1) A childhood variety that is acute, without gender predilection, post-viral and self-limited. 2) Adult form that tends to be chronic, female predominant, and most frequent between the 2nd and 4th decades [4]. Many disorders may lead to secondary ITP such as systemic lupus erythematosus, lymphoproliferative disorders, drugs (including rifampin) but tuberculosis is exceedingly rare condition. The two principal diagnostic criteria for ITP are thrombocytopeina in the context of an otherwise normal blood count and a normal peripheral smear and the exclusion of conditions capable of inducing thrombocytopenia [3]. Similarly in this patient had normal blood count except thrombocytopenia and normal peripheral smear. Clinical and laboratory examinations were also showed no other disorder leading thrombocytopenia.

The association between tuberculosis and ITP is very rare event [3,5,6,7]. An immune basis for tuberculosis induced ITP was supported by the presence of either platelet antigen spesific antibodies or platelet surface membrane IgG [8,9]. It has been postulated that the anti platelet antibodies generated in some cases of tuberculosis induced ITP are secreted by lymphocytes. [10]. In our case, anti-platelet antibody could not detected due to inavailability. Although IVIg and corticosteroid therapy is generally accepted therapy options in the treatment of ITP [4], it is well known that the most important therapy for infection-related thrombocytopenia is that directed to the underlying infection. Corticosteroids are generally undesirable as a treatment for thrombocytopenia in patients with severe infection because of their potential for supressing immune response. It was shown that platelet count return normal range within 2-3 weeks after initiating appropriate antimicrobial therapy in tuberculous and the other infectious disease such as brucellosis [3,11]. In the literature, as in our case, it is reported that there are few cases which could not response to the IVIg and methylprednisolone therapy. However in those cases thrombocytes level returned normal limit with anti-tuberculosis therapy [3,12].

In conclusion, ITP is exceedingly rare presenting manifestation of tuberculous infection. It is thought that clinical suspection of tuberculosis in patients with ITP, early diagnostic procedure following to initiate anti-tuberculosis therapy is a cost-effective and efficient choice.

REFERENCES

1. Mert A, Bilir M, Tabak F, et al. Miliary tuberculosis: Clinical manifestation, diagnosis and outcome in 38 adults. Respirology 2001; 6: 217-24.
2. Glasser RM, Walker RI, Herion JC. The significance of hematologic abnormalities in patients with tuberculosis. Arch Intern Med 1970; 125: 691-5.
3. Ghobrial MW, Albornoz MA. Immune thrombocytopenia: A rare presenting manifestation of tuberculosis. Am J Hematol 2001; 67: 139-43.
4. George JN, Raskob GE. Idiopathic thrombocytopenic purpura. Diagnosis and management. Am J Med Sci 1998; 316: 87-93.
5. Spedini P. Tuberculosis presenting as immune thrombocytopenic purpura. Haematologica 2002; 87: ELT09.
6. Madkaikar M, Ghosh K, Jijina F, et al. Tuberculosis and immune thrombocytopenia. Haematologica 2002; 87: ELT38.
7. Ozkalemkas F, Ali R, Ozkan A, et al. Tuberculosis presenting as immune thrombocytopenic purpura. Ann Clin Microbiol Antimicrob. 2004; 3: 16.
8. Boots RJ, Roberts AW, McEvoy D. Immune thrombocytopenia complicating pulmonary tuberculosis. Case report and Investigation of mechanism. Thorax 1992; 47: 396-7.
9. Jurak SS, Aster R, Sawaf H. Immune thrombocytopenia associated with tuberculosis. Clin Pediatr. 1983; 22: 318-9.
10. Tsuro K, Kojima H, Mitoro A, et al. Immune Thrombocytopenic Purpura associated with Pulmonary Tuberculosis. Intern Med 2006; 45: 739-42.
11. Young E, Tarry A, Genta RM, et al. Thrombocytopenic purpura associated with brucellosis: report of 2 cases and literature review. Clin Infect Dis 2000; 31: 904-9.
12. Perez de Liano LA, Soilan del Cerro JL, Garcia Pais MJ. Immune thrombocytopenic purpura as presenting form of miliary tuberculosis. Arch Broncho-Pneumol 1998; 34: 411-2.