Immune Thrombocytopenic Purpura due to Disseminated Tuberculosis

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We read with interest the paper by Al-Majed et al. [1] in a recent issue of this journal. The authors stated that patients with disseminated tuberculosis (TB) rarely develop pure thrombocytopenia and that TB presenting as immune thrombocytopenic purpura (ITP) is exceedingly rare. In relation to this we would like to report a recent case of TB presenting as ITP and who is the single case among 319 patients diagnosed by us with ITP.

A 70-year-old Caucasian man was admitted with a 5-day history of disorientation and mucosal hemorrhages. He had been ill for 1 week with a cough producing small amounts of purulent sputum, fever and rigors. No weight loss or other symptoms were reported, and there was no personal or family history of TB. On examination, he was febrile and had numerous cutaneous pete-chiae on the chest and arms. There was no lymphadenopathy or hepatosplenomegaly present. Scattered inspiratory crackles were heard on both lower parts of the chest. The white and red cell count was normal and the platelet count was $8 \times 10^9/l$ (normal range $140-400 \times 10^9/l$). The chest radiograph showed bilateral patchy opacities. The ESR was 24 mm/h and the coagulation profile was within normal limits. The analysis of arterial blood gases performed while the patient was breathing room air showed severe hypoxia with a p_{aO_2} of 32 mm Hg, p_{aCO_2} of 36 mm Hg, HCO_3^- of 28 mEq/l, pH of 7.32, and he was then transferred to the Intensive Care Unit. Bone marrow aspirate and biopsy were performed and showed an increased cellularity and number of megakaryocytes. With the presumed diagnosis of ITP, the patient received high doses of methylprednisolone (1 g/day $\times$ 3 days) in addition to intravenous immunoglobulins (0.4 g/kg/day $\times$ 5 days), with a poor response of the platelet count ($21 \times 10^9/l$ on day 5 of treatment). On day 10 the trephine bone marrow biopsy was examined and revealed noncaseating granulomas. Standard antituberculous therapy, which consisted of isoniazid 300 mg/day, rifampin 600 mg/day and ethambutol 1,500 mg/day, was then initiated. Two days later, the smears of a bronchial aspirate were found positive for acid fast bacilli (previous ones were negative). The platelet count returned to normal on day 5 of the antituberculous treatment, but the patient developed acute oliguric renal failure, worsening of the hypoxia, persistent hypotension and died 20 days after admission.

In our case, as in some of Al Majed et al. [1], the diagnosis of TB was not entertained initially because severe thrombocytopenia complicating TB is uncommon and makes the early recognition difficult [2]. Ozsoylu [3] has clarified the definition of ITP by emphasizing that

every ITP is an autoimmune disorder, but not every autoimmune thrombocytopenia is ITP. Jurak et al. [4] reported 2 patients with TB, thrombocytopenia and antiplatelet antibodies, speculating that *Mycobacterium tuberculosis* could stimulate a clone of B lymphocytes nonspecifically and the lymphocytes might produce antibodies against autologous platelets. Boots et al. [5], in a case of ITP complicating pulmonary TB, were able to demonstrate platelet surface membrane IgG and in that case, thrombocytopenia improved rapidly with intravenous immunoglobulin treatment. We could not perform immunofluorescence studies to detect platelet autoantibodies but in our case the significant increase in the platelet count after antituberculous treatment was considered as convincing proof of the etiology of TB as the cause of thrombocytopenia. Although the largest increase in annual incidence of ITP is believed to be due to the effect of HIV infection [6], it is still true to say that in western Europe TB is still encountered in older age-groups and represents activation of latent foci. Therefore, it is reasonable to suspect and exclude TB, in order to start an early and specific treatment.

References

- Al-Majed SA, Al-Momen AK, Al-Kassimi FA, Al-Zeer A, Kambal AM, Baaqil H: Tuberculosis presenting as immune thrombocytopenic purpura. *Acta Haematol* 1995;94:135-138.
- Gasser RM, Walker RI, Herion JC, Hill C: The significance of hematological abnormalities in patients with tuberculosis. *Arch Intern Med* 1970;125:691.
- Ozsoylu S: Idiopathic thrombocytopenic purpura. *Acta Haematol* 1994;92:55.
- Jurak SS, Aster R, Sawaf H: Immune thrombocytopenia associated with tuberculosis. *Clin Pediatr* 1983;22:318-319.
- Boots RJ, Roberts AW, McEvoy D: Immune thrombocytopenia complicating pulmonary tuberculosis: Case report and investigation of mechanism. *Thorax* 1992;47:396-397.
- Huxley H, Knox-Macaulay M: TB and the haemopoietic system. *Baillière's Clin Haematol* 1992;5:101-129.

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