
Reader comment

Postsplenectomy reactive thrombocytosis

We have several concerns related to the article “Postsplenectomy reactive thrombocytosis” published in the January 2009 issue of *Proceedings* (1). The implication of the article is that reactive postsplenectomy thrombocytosis is associated with a high incidence of thrombotic complications and, in select cases, should be treated with systemic chemotherapy. Most series conclude there is not an increase in thromboembolic complications associated with postsplenectomy thrombocytosis or extreme reactive thrombocytosis (2, 3), and experts do not advocate platelet-lowering drugs in reactive, nonclonal thrombocytosis (4). The authors referenced an article by Stamou et al, supporting the association of postsplenectomy thrombocytosis with portal and mesenteric vein thrombosis, but only 7 of 147 patients had such a complication (four of them had an occult malignancy and two had other hematologic disorders) (5). The patients with similar complications they referenced (6, 7) had myeloproliferative disorders and not reactive thrombocytosis. There are no published data that suggest systemic chemotherapy should be used in postsplenectomy reactive thrombocytosis rather than antiplatelet therapy along with standard postoperative anticoagulation.

The pathogenesis of the persistent postsplenectomy thrombocytosis in the case described is not clear. In some cases, a bone marrow biopsy may not be diagnostic of a myeloproliferative disorder in patients who have early essential thrombocythemia. The JAK-2 mutation is abnormal in 50% of patients with essential thrombocythemia, and it is not reported. There is no mention in the case report of whether there was any evidence of extramedullary hematopoiesis in the spleen, as one might anticipate with a myeloproliferative disorder. In select cases when the pathogenesis of thrombocytosis may not be clear, a short interval of myelosuppressive therapy may be warranted until

the diagnosis of a myeloproliferative disorder can be clarified. Yet, this patient was maintained on myelosuppressive therapy with anagrelide for 1 year postoperatively. The authors conclude that the patient probably did have essential thrombocythemia, although the implication of the case report is that this patient had reactive thrombocytosis accounting for thrombotic complications.

The pharmacologic agents listed in Table 3 for management of thrombocytosis include systemic chemotherapy, which is only warranted in patients who have a myeloproliferative disorder. Finally, the procedure to immediately reduce platelet counts in patients with thrombocytosis associated with myeloproliferative disorders is plateletpheresis and not plasmapheresis.

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1. Khan PN, Nair RJ, Olivares J, Tingle LE, Li Z. Postsplenectomy reactive thrombocytosis. *Proc (Bayl Univ Med Cent)* 2009;22(1):9–12.
 2. Boxer MA, Braun J, Ellman L. Thromboembolic risk of postsplenectomy thrombocytosis. *Arch Surg* 1978;113(7):808–809.
 3. Buss DH, Cashell AW, O'Connor ML, Richards F 2nd, Case LD. Occurrence, etiology, and clinical significance of extreme thrombocytosis: a study of 280 cases. *Am J Med* 1994;96(3):247–253.
 4. Schafer AI. Thrombocytosis. *N Engl J Med* 2004;350(12):1211–1219.
 5. Stamou KM, Toutouzas KG, Kekis PB, Nakos S, Gafou A, Manouras A, Krespis E, Katsaragakis S, Bramis J. Prospective study of the incidence and risk factors of postsplenectomy thrombosis of the portal, mesenteric, and splenic veins. *Arch Surg* 2006;141(7):663–669.
 6. Hirsh J, Dacie JV. Persistent post-splenectomy thrombocytosis and thrombo-embolism: a consequence of continuing anaemia. *Br J Haematol* 1966;12(1):44–53.
 7. Daya SK, Gowda RM, Landis WA, Khan IA. Essential thrombocythemia-related acute ST-segment elevation myocardial infarction. A case report and literature review. *Angiology* 2004;55(3):319–323.