Postoperative thrombocytosis: An unusual case report

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Abstract

Thrombocytosis is often an incidental finding seen in 35–50% of cases and the cause determination creates a diagnostic challenge. Extreme thrombocytosis is rare and seen in 2–5.8% patients only. Among the various causes of increased platelet count, surgical procedures have attracted much attention in both experimental and clinical domain. The appearance of thrombocytosis after surgery needs to be diagnosed to establish the type of thrombocytosis (clonal or reactive), as treatment and prognosis are quite different between them. This case report is vital because of two reasons: First, the increase in platelet count is difficult to rationalize than many of the other thrombocytoses, such as those related to primary augmentation of the function of the bone marrow; second, the association of platelets with the clotting process has led to the belief that their increase after a surgical procedure is connected with the occurrence of postoperative thrombosis. This case presents an interesting finding from a patient who has undergone major abdominal surgery and has shown an unexpected perpetual increase in platelet count.

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INTRODUCTION

Thrombocytosis is commonly seen in clinical practice and often the cases reported are incidental. The differential diagnosis for thrombocytosis is broad and the diagnostic process can be challenging. In general, causes of thrombocytosis can be described as spurious, reactive, or clonal in nature. A number of population studies have examined the degree of thrombocytosis as well as the frequency of various etiologies of thrombocytosis when it occurs. Reactive causes are by far the most common etiology of thrombocytosis in these population studies, comprising 88–97% of cases in adults in two large case series^[1,2] and 100% of pediatric cases in a single case series.^[3] Extreme thrombocytosis, defined as a

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platelet count >1,000 × 10^{9} /L is quite rare, as only 2–5.8% of patients demonstrate this degree of thrombocytosis upon presentation. Although often thought to be more common in clonal processes, extreme thrombocytosis can also be due to reactive causes, with 82% of cases of extreme thrombocytosis in one series being reactive in nature.^[4]

CASE REPORT

A 26-year-old male patient was scheduled for surgery of right indirect inguinal hernia with right sided encysted hydrocele of the cord. The preoperative laboratory tests reported normal coagulation and biochemical parameters, haemoglobin values of 15.1 g%, no leukocytosis and a platelet count of 2.60 lakhs/Cumm. Patient was operated, right sided mesh hernioplasty with excision of hydrocele of the cord was done. There were no adverse incidents

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during surgery, the patient was smoothly extubated in the surgery room and transferred to the post anaesthesia care unit.

A routine postoperative analytical blood control was performed, showing high platelets count (1,100,000/Cumm), hemoglobin 11.6 mg/dL and leukocytes 18,400/Cumm, with normal values of coagulation and biochemistry tests. Another blood analysis was extracted to verify these data, which again returned a high platelet count (1,300,000/Cumm). We examine the patient, diagnosed a reactive thrombocytosis secondary to the surgery. We suggested monitoring platelet count throughout the postoperative period by serial analytical, with introduction of thromboembolic prophylaxis and control of bleeding in the face of the probability of some degree of platelet dysfunction.

The immediate period after surgery was uneventful and the patient was discharged to the ward with hemodynamic stability. Later, on the next postoperative day, patient developed huge scrotal haematoma with ecchymosis around the incision line. Scrotal haematoma was explored, clots were drained, but no obvious bleeder could be identified. On 5th postoperative day, patient developed bruises around the right flank. Subsequently, patient developed infection of the wound which settled with antibiotics and dressing.

Postoperative antithrombotic prophylaxis and bleeding monitoring were maintained until the patient was discharged after 2 weeks of admission, to follow up with outpatient treatment. Analytical blood controls showed that the platelet count was down to normal in 20 days.

DISCUSSION

Thrombocytosis is an incidental finding in 35–50% of cases and determination of the cause creates a diagnostic challenge. The causes of thrombocytosis can be described as spurious, reactive, or clonal in nature as shown in Table 1.^[5]

The reactive thrombocytosis, also called secondary thrombocytosis is the most common type and appears after acute inflammatory, infectious, neoplastic and stress processes. In these scenarios the levels of thrombopoietin, interleukin-6 and catecholamines are very high, and are thought to be responsible for the increased number of platelets. Signs and symptoms of the underlying disease usually accompany reactive thrombocytosis. It is always necessary to distinguish between clonal (also known as primary or essential thrombocytosis) and reactive (or secondary) thrombocytosis, because their treatment and prognosis differ. The main features of both types of thrombocytosis are described in Table 2.^[6]

Table 1: Causes of thrombocytosis			
Clonal	Reactive	Spurious	
Essential thrombocythemia	Infection	Microspherocytes	
Polycythemia vera	Inflammation	Cryoglobulinemia	
Primary myelofibrosis	Tissue damage	Neoplastic cell fragments	
Myelodysplasia with del(5q)	Hyposplenism	Schistocytes	
RARS-T	Postoperative	Bacteria	
Chronic myeloid leukemia	Iron deficiency		
Chronic myelomonocytic leukemia	Malignancy		
Atypical chronic myeloid leukemia	Hemolysis		
MDS/MPN-U	Drug effect		
POEMS syndrome	"Rebound" following myelosuppression		

Familial thrombocytosis

RARS-T: Refractory anemia with ringed sideroblasts associated with marked thrombocytosis; MDS: Myelodysplastic; MPN-U: Myeloproliferative neoplasm-unclassifiable

Table 2: Distinguishing clinical features for primary and secondary thrombocytosis

Feature	Primary	Secondary
Underlying systemic disease	No	Clinically
		apparent
Digital or cerbrovascular ischemia	Characteristic	No
Large-vessel arterial or venous	Increased risk	No
thrombosis		
Bleeding complications	Increased risk	No
Spleenomegaly	In about 40% of patients	No
Peripheral blood smear	Giant platelets	Normal
		platelets
Platelet function	May be abnormal	Normal
Bone marrow megakaryocytes		
Number	Increased	Increased
Morphologic features	Giant, dysplastic forms with increased ploidy; large masses of platelet debris	Normal
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Although the diagnostic tests to differentiate essential and reactive thrombocytosis are not easy to perform, laboratory tests that show increased acute phase reactants such as C-reactive protein, fibrinogen, erythrocyte sedimentation rate and interleukin-6 may be useful in the diagnosis of reactive thrombocytosis.^[7] It is accepted that upto 1,000,000 μ L⁻¹ platelets level are a benign condition, although it remains unclear if these findings are associated with an increased postoperative thromboembolic or haemorrhagic risk. Prophylactic treatment with platelet inhibitors in these situations is controversial, although some authors do consider management of low-dose acetylsalicylic acid appropriate.^[8,9]

Reactive thrombocytosis is generally felt thought to be a self-limited process which resolves with resolution of the underlying disorder when possible. The risk of thrombotic complications with reactive thrombocytosis is felt to be low, as 1.6% of patients with reactive thrombocytosis had thrombotic complications in one large case series.^[1] All

of these thrombotic events were venous in location and occurred in patients with other risk factors (postoperative setting or underlying malignancy).^[10] Even in cases of extreme reactive thrombocytosis, the risk of thrombotic complication is relatively low (4–6%).

Conclusion

The appearance of thrombocytosis after surgery needs to be diagnosed to establish the type of thrombocytosis (clonal or reactive), as treatment and prognosis are quite different between them. And at all times, the surgeon must remain vigilant due to the possible risk of bleeding or thromboembolic complications.

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Conflicts of interest

There are no conflicts of interest.

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