Case Report

Incidental thrombocytosis: Should it concern the anesthesiologist?

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Abstract

Preoperative thrombocytosis, often detected incidentally in surgical patients and inadvertently overlooked, has important implications for the anesthesiologists. The primary form is a chronic clonal myeloproliferative disorder usually affecting adults while the secondary type is a benign reactive disease commonly found in children. Serious perioperative hemostatic complications are reported in primary thrombocytosis and hence, a detailed preoperative evaluation and initiation of therapy to lower the platelet count (PC) is required before undertaking surgery. Patients with reactive thrombocytosis however, usually have complication-free surgeries, and if there is no prior evidence of hemostatic complications and the reactive cause can be identified, no specific perioperative intervention may be required. A thorough preanesthetic checkup and implementation of basic thrombo-prophylaxis measures in all patients with a raised PC is advocated. We present here our experience with three infants diagnosed with high preoperative PC, presumably due to reactive causes, who underwent uneventful neurosurgeries at our institution.

Key words: Anesthesia, essential thrombocythemia, perioperative complications, reactive thrombocytosis, thrombocytosis

Introduction

Thrombocytosis, defined as a platelet count (PC) of more than 450,000/μL, may be a chance preoperative finding during routine complete blood counts (CBC) in patients presenting for surgery. It is a relatively less understood entity among anesthesiologists and is likely to be overlooked; the affected patients may often proceed for surgery without any advice for the raised PC. However, thrombocytosis can have far-reaching perioperative implications due to a potential for hemostatic complications,[1,2] suggesting that its thorough understanding would be helpful when faced in practice.

Case Report

Three patients requiring emergency neurosurgical operations at our institution were incidentally detected to have thrombocytosis. The first patient (case 1), aged 5 months, underwent excision of a ruptured, infected meningomyelocele; his raised platelet count (PC) of 685,000/μL was unwittingly overlooked by us and hence, no perioperative action was taken. In the other two children, aged 2 years (case 2) and 3 years (case 3), both scheduled for endoscopic third ventriculostomy (ETV), high PC values (847,000/μL in case 2 and 1,008,000/μL in case 3) were noticed before surgery. A quick literature search on the subject was done. Significant findings during the preanesthetic check-up (PAC) in case 2 included a history of emergency ventriculoperitoneal shunt performed 2 months back and a current hemoglobin value of 11.1 g%, and in case 3, a recent history of fever and chest infection. Both children had features of raised intracranial pressures. There was no prior or current evidence of thrombosis or bleeding or presence of any prothrombotic risk factors. Based on their clinical profile, a provisional diagnosis of reactive thrombocytosis was made. Further hematology work-up was not done in view of the emergent nature of surgery and absence of thrombotic or hemorrhagic symptoms; all other routine investigations were normal. Informed consent was obtained prior to surgery. Both patients were anesthetized and monitored intraoperatively as per our routine neurosurgical practice which also includes invasive arterial monitoring for management of ETV related hemodynamic complications. The anesthesia protocols however, also incorporated simple prophylactic measures against hemostatic complications which included, ensuring adequate perioperative hydration, minimal surgical blood loss, early postoperative removal of arterial

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cannulas followed by close monitoring of digital pulsations, and encouraging early postoperative patient mobility. All three children had uneventful surgeries. There was no perioperative evidence of thrombotic or bleeding complication. These patients were not investigated further for thrombocytosis and no specific therapy was initiated. The routine complete blood count (CBC) at 6 months of follow-up revealed normal PC in all three children.

Discussion

Thrombocytosis in its mild to moderate forms (PC up to 700,000/μL) may not be an unusual finding in childhood, with an incidence of 3-13%, however, extreme thrombocytosis (PC >1,000,000/μL) is uncommon with an incidence of approximately ≤2%).[3] Based on its etiology, thrombocytosis can be classified as primary or clonal, secondary or reactionary and familial, which is a rare genetic disorder.[1,4] Primary thrombocytosis mostly affects adults and is associated with chronic myeloproliferative or myelodysplastic disorders like essential thrombocythemia (ET), polycythemia vera, chronic myelogenous leukemia, myelofibrosis, etc., which are clonal disorders of the hematopoietic stem cells. ET is characterized by a high PC, abnormalities in bone marrow morphology and platelet structure (giant platelets and dysplastic megakaryocytes) and abnormal platelet function resulting in an increased risk of bleeding as well as arterial and venous thromboembolism. Spontaneous bleeding from the gastrointestinal and genitourinary tracts, hemoptysis, and epistaxis is known; while ischemia of digits progressing to limb-threatening gangrene, cerebrovascular ischemia causing neurological complications, intra-abdominal thrombosis, pulmonary embolism, etc., are among the serious thrombotic complications. Patients with age more than 60 years, PC greater than 1,500,000/μL, prior evidence of thrombosis or hemorrhage, and coexisting cardiovascular risk factors are particularly at risk. The World Health Organization (WHO) diagnostic algorithm for ET is based on clinical evaluation and exclusion of the reactive causes of raised PC and laboratory assessment that includes peripheral blood mutation screening for *JAK2* V617F and bone marrow biopsy.[1] ET patients with symptomatic disease, extreme thrombocytosis, and high risk factors are started on therapy with low-dose aspirin (100-150 mg/day) and platelet-lowering drugs like hydroxyurea, anagrelide, interferon alfa, busulfan, or pipobroman.[1,4]

Secondary thrombocytosis, on the other hand, is a relatively benign condition and is usually associated with modest elevations in PC (up to 700,000/μL), normal platelet structure and function, and a normal bone marrow. It is the most commonly prevailing thrombocytosis (incidence of 6-15% in hospitalized pediatric patients).[3] This reactionary form is usually an acute phase response to infection, chronic inflammation, malignancy, injury, etc. Bacterial and viral respiratory tract infections are the commonest cause (seen in 60-80% of patients), followed by urinary, gastrointestinal tract, central nervous system, skeletal, and other infections.[3] The leading noninfectious cause is iron deficiency anemia; the others being: Hemolytic anemia; bleeding; surgical or functional splenectomy; autoimmune diseases like juvenile rheumatoid arthritis, Henoch-Schonlein purpura, Wegener's granulomatosis, polyarteritis nodosa, inflammatory bowel disease, etc.; solid tumor malignancies; tissue injury in trauma; burns and surgery; and drugs like adrenaline, steroids, miconazole, carbepenams, cephalosporins, etc.[3] Thrombotic complications are uncommon unless additional prothrombotic risk factors are present; a 1.6% incidence of venous thromboembolism has been reported in reactive thrombocytosis associated with prothrombotic risk factors like malignancy and preceding surgery.[1] No antiplatelet or cytoreductive therapy or thromboprophylaxis is required even with PC > 1,000,000/μL unless additional well-defined thrombophilic risk factors are present. The counts normalize spontaneously with resolution of the primary cause and hence, the treatment is directed at the underlying disease and not the PC.[3,4]

It is well-known that surgery in itself enhances perioperative thrombotic risk up to five-fold and concomitant thrombocytosis is expected to further amplify this risk.[1] The main issue for the anesthesiologists while handling surgical patients with a raised PC would be to identify the 'at risk' patients and initiate appropriate perioperative management. Primary thrombocytosis in particular, is reported to be associated with serious perioperative complications like arterial and venous thromboembolism (incidence 5.3 and 1.1%, respectively),[2] bleeding (incidence 10.5%),[2] digital gangrene following radial artery catheterization in two separate reports,[5,6] and postoperative pulmonary thromboembolism.[7]

The perioperative management in patients with high platelet counts is influenced to a large extent by the type of thrombocytosis.[1,4,8,9] As most incidentally detected elevations in PC could be transient, counts should preferably be repeated after a few weeks before further laboratory evaluation.[4] In high risk and symptomatic patients with ET, elective surgery should be deferred, treatment with aspirin and cytoreductive drugs initiated to target PC values to less than 400,000/μL and coexisting cardiovascular risk factors aggressively corrected[1]; preoperative platelet pheresis may be considered if there is evidence of active ischemia.[4,9] In emergency situations where preoperative PC cannot be lowered, use of perioperative thromboprophylactic measures is suggested.[1,4,8,9]

These included maintaining good hydration to decrease the hyperviscosity due to a high PC, ensuring early postoperative ambulation, use of mechanical thromboprophylaxis methods like compression stockings and pneumatic compression devices, and, pharmacological thromboprophylaxis with low molecular weight and unfractionated heparin, warfarin, and aspirin; though pharmacological methods have been used during major surgery in high risk ET patients, it requires caution due to the risk of enhanced bleeding.[2,10] Intra-and postoperative administration of gabexate mesilate, an antiplatelet agent, has also been reported to prevent thrombotic complications.[11] Surgical blood loss and postoperative infection may result in worsening of thrombocytosis and should be promptly managed.[1]

Conversely, patients with reactive thrombocytosis usually have complication-free surgeries even with a high PC if no other prothrombotic risk factors are present and hence, no specific preoperative preparation and perioperative intervention is required; the PC gradually normalizes in the postoperative period.[4] The cause for the reactive disease should however be elicited and treated; in our patients, evidence of infected meningomyelocele in case 1, previous surgery and mild anemia in case 2, and fever and chest infection in case 3 were identified as the possible causes for reactionary thrombocytosis. Clear-cut preoperative differentiation between the primary and secondary disease, though important, may not always be possible, as happened in our patients. Implementation of basic perioperative thromboprophylaxis measures in all surgical patients with a raised PC, irrespective of their distinctive diagnosis, thus appears prudent.

To conclude, preoperative thrombocytosis needs to be acknowledged and appropriately addressed. The elevated PC found incidentally in children is most likely to be due to transient secondary thrombocytosis, especially when the reactive cause can be identified and there are no hemostatic complications. As this form of thrombocytosis is unlikely to complicate the perioperative course and does not require any specific intervention, we feel that postponing surgery till the PC subsides in pediatric surgical patients with reactionary thrombocytosis is not justified. However, if primary thrombocytosis is suspected, thorough preoperative investigation and treatment is necessary, followed by implementation of a definitive perioperative plan.

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