It is well known that low platelet counts increase bleeding risks. However, high platelet counts sometimes may also lead to bleeding or thrombosis.

We report a case of post hemorrhoidectomy bleeding caused by thrombocytosis induced coagulopathy.

Case Report

A 54 year-old man presented with rectal bleeding and anal pain with bowel movement for a few days. He received bilateral vasectomy 10 years ago and had history of gout for 7 years. Grade IV Hemorrhoids was diagnosed. Laboratory data showed hemoglobin was 18.4 mg/dl, hematocrit was 54.9%, WBC was 13200/µL. Platelet was 865,000/µL. PT and APTT were normal. He underwent closed Ferguson hemorrhoidectomy and was discharged 2 days later. He presented to ER with massive anal bleeding on the third postoperative day. Fever was up to 38 °C and leukocytosis (WBC 16100/µL) was found in ER. Thrombocytosis may be resulted from wound infection and postoperative status. Antibiotics were administered and a Foley balloon tamponade of the rectum was performed. The bleeding was stopped. Twelve days later, he complained of passage of fresh red bloody stool again. Laboratory data showed hemoglobin was 13.0 mg/dl, hematocrit was 37.4%, WBC was 21000/µL, platelet was 1476000/µL, PT was 12.9 with control of 11.0 sec, APTT was 31.0 with control of 30.0 sec, bleeding time was 2’30’’. Bleeding was stopped after Foley balloon tamponade. Abdominal sonography revealed fatty liver. Hematologist was consulted and bone marrow biopsy was performed. Essential thrombocytemia was diagnosed. Hydroxyurea was administered for treatment of essential thrombocytemia. Then, it was shifted to anagrelide due to nausea and malaise. He suffered from small in-
farction of left temporal lobe 15 months later and chest wall subcutaneous hematoma 3 years later.

**Discussion**

A normal platelet count in a healthy adult is between 150,000 and 450,000/μL (150-450 × 10⁹/L) of blood. Thrombocytosis is defined as a platelet count exceeding the top of the normal range.

Thrombocytosis is classified into two types. Primary thrombocytosis is the result of myeloproliferative disorder (autonomous), including polycythemia vera, essential thrombocythemia, chronic myeloid leukemia, myelofibrosis, and myelodysplastic syndromes. Secondary thrombocytosis (reactive thrombocytosis) may be caused by increased release of a number of cytokines in response to infections, inflammation, vasculitis, tissue trauma, and other factors. It is often a transient reaction which resolved when the underlying cause is subsided. It is also known as reactive thrombocytosis (RT).

Secondary thrombocytosis (RT) is much more frequent than primary thrombocytosis in both children and adults. Buss reported a study of 280 consecutive patients with extreme thrombocytosis (platelet count > 1,000,000/μL), RT was diagnosed in 82% and myeloproliferative disorder in 14%, cases of uncertain etiology in 4%. The causes of secondary thrombocytosis included infection (31%), postsplenectomy (19%), malignancy (14%), trauma (14%), noninfectious inflammation (9%), postsurgical status with infection, postsurgical status, and acute blood loss or iron deficiency (6%).

Table 1. Causes of secondary (reactive) thrombocytosis

<table>
<thead>
<tr>
<th>Condition</th>
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<td>Acute blood loss</td>
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<td>Iron deficiency anemia</td>
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<td>Hemolytic anemia</td>
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<tr>
<td>Postsplenectomy</td>
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<tr>
<td>Malignancy</td>
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<tr>
<td>Infection</td>
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<tr>
<td>Chronic inflammations and vasculitis (e.g., rheumatoid arthritis, inflammatory bowel disease)</td>
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<tr>
<td>Tissue damage (postsurgical, burns, trauma, fracture, acute pancreatitis)</td>
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<tr>
<td>Recovery from thrombocytopenia (e.g., bleeding, cancer chemotherapy)</td>
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<tr>
<td>Drug reactions (Vincristine, All-trans-retinoic acid, cytokines, growth factors)</td>
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</tbody>
</table>

There are presently no diagnostic tests that can definitively distinguish between primary and secondary (reactive) thrombocytosis. Once thrombocytosis was found, repeat testing, peripheral blood smear and serum ferritin level should be performed. The risks for developing polycythemia vera, essential thrombocythemia, or associated vascular complications in persons with thrombocytosis were low. The patients with thrombocytosis were only 8 of the 99 patients (8%) on repeat testing at a median interval of eight months (range from 6 to 14 months).

If thrombocytosis is confirmed by repeat testing, further examinations should be undertaken to differentiate whether the high platelet count is secondary or primary. The first step is to take complete history and physical examination. The initial evaluations should exclude many of the common causes of reactive thrombocytosis. Special attention should include recent trauma or surgery, prior splenectomy, local or systemic complaints suggesting infection or inflammation, present and past history of bleeding, thrombosis, iron deficiency, or malignancy.

For the differentiation of secondary from primary thrombocytosis, Messinezy et al found determination of acute-phase reactants (for example, erythrocyte
Blood sedimentation rate (ESR) is most useful. Blood ESR, C-reactive protein (CRP) level, fibrinogen level, factor VIII procoagulant activities, and von Willebrand antigen values are significantly elevated in patients with secondary thrombocytosis, whereas they were normal in patients with primary thrombocytosis.

When persistent thrombocytosis of undetermined cause presents, searching for occult cancer should be performed thorough physical examination, including examination of stool occult blood, chest radiography and the abdominal sonography to find undetected sources of infection, inflammation or malignancy.

When the clinician is confronted with treatment decisions, determining the cause of thrombocytosis becomes especially critical. Patients with RT do not require treatment because their abnormal platelet count does not have risk for bleeding or thrombotic events. Treatment should be directed to the underlying disease.

In primary thrombocytosis with hemorrhage complications, the first step is to discontinue the use of any platelet antiaggregating agent (such as nonsteroidal antiinflammatory drugs). Initial laboratory evaluation should include a workup for disseminated intravascular coagulation and coagulation factor deficiency. Acquired factor V deficiency is sometimes seen in association with primary thrombocytosis and is treated with fresh frozen plasma infusion or platelet concentrates.

In the case of thrombosis with platelet count greater than 800,000/μL, immediate platelet apheresis is recommended. Therapy with a platelet-lowering agent should be started with the goal of keeping the platelet count below 400,000/μL.

**Conclusions**

When thrombocytosis was found in a patient prior to surgery, differentiating between primary and secondary thrombocytosis is important and difficult. Secondary thrombocytosis does not result in hemorrhage or thrombotic complications, but the underlying problems must be identified and treated. In contrast, primary thrombocytosis is associated with thrombotic and bleeding complications. Patients with high risk of complications should receive prophylactic platelet-lowering therapy.

**References**

N. Autonomous megakaryocyte growth in essential thrombocytopenia and idiopathic myelofibrosis is not related to a c-mpl mutation or to an autocrine stimulation by Mpl-L. *Blood* 1999;93:125.


病例報告

因血小板過高引發的痔術後出血

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血小板過高可能發生血栓或出血的併發症。它可分為原發性和繼發性兩種。原發性血小板增多症 (Essential thrombocytosis) 屬骨髓增生性疾病，次發性血小板增多症 (Secondary thrombocytosis) 則是因其他疾病所引起的，包括慢性炎症、出血、鐵質缺乏或是惡性腫瘤等因素。我們經歷一個痔瘡手術前發現有血小板過高的病患，術後發生兩次肛門出血，後來診斷為原發性血小板過多症 (Essential thrombocytemia) 的病例。

關鍵詞  血小板增多、出血、血栓。