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Reactive thrombocytosis after cancer surgery: a case report

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We describe a female patient, 58 years old, scheduled for bilateral ovarian cancer surgery. Her medical history included penicillin allergy, arterial hypertension and hyperlipidemia treated with ACE inhibitors and Atorvastatin respectively. She was also an ex-smoker of three years. The pre-anaesthetic assessment showed sinus bradycardia (50 b.p.m), with incomplete right branch blockade of the Hiss bundle and mild bilateral pleural effusion with atelectasis in the chest radiograph (secondary to the extended neoplasia). The pre-operative laboratory tests reported normal coagulation and biochemical parameters, haemoglobin values of 13,9 $mg.dL^{-1}$, no leukocytosis and a platelets count of $267.000.\mu L^{-1}$.

The patient was properly monitored in the operating theatre with electrocardiography, pulse oximetry, capnography, invasive arterial pressure, central venous pressure, bi-spectral index and diuresis catheterisation. After antibiotic prophylaxis with Amoxicillin Clavulanic, a total intravenous anaesthesia technique with Propofol and Remifentanil target-controlled infusion was started. Other drugs used were Cisatracurium, morphine hydrochloride and NSAIDs (Dexketoprofen and acetaminophen) for post-operative pain control, and Ondansetron plus dexamethasone to control nausea and vomiting.

Surgery lasted for 7 hours, and a laparotomy was performed, with total hysterectomy, double oophorectomy, pelvic para-aortic radical lymphadenectomy, appendectomy and partial cecum resection. There were no adverse incidents during surgery, with good urine output and 200 ml of estimated bleeding. The patient was smoothly extubated in the surgery room and transferred to the post anaesthesia care unit (PACU).

A routine postoperative analytical blood control was performed in the PACU, showing high platelets count ($1.016.000.\mu L^{-1}$), haemoglobin 11.6 $mg.dL^{-1}$ and leukocytes $18.400.\mu L^{-1}$, with normal values of coagulation and biochemistry tests. Another blood analysis was extracted to verify these data, which again returned a high platelet count ($1.034.000.\mu L^{-1}$). We consulted with the haematologist on call who, after examining the patient, diagnosed a reactive thrombocytosis secondary to both the neoplastic process and the surgery. He suggested monitoring platelet count throughout the post-operative 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.

Table 1. Major causes of reactive thrombocytosis

Transient processes	Acute blood loss Recovery of thrombocytopenia Acute infection and inflammation Intense exercise Surgery
Chronic processes	Iron deficiency Hemolytic anemia Asplenia Cancer Chronic infectious and inflammatory diseases Connective tissue diseases Temporal arteritis Irritable Bowel Syndrome Tuberculosis chronic pneumonitis Drug-induced vincristine retinoic acid cytokines

The immediate period after surgery was uneventful and the patient was discharged to the ward with hemodynamic stability after 6 hours in the PACU. Postoperative antithrombotic prophylaxis and bleeding

Table 2. Main features of essential and reactive thrombocytosis.

Features	Essential thrombocytosis	Reactive thrombocytosis
Underlying disease	No	Yes
Digital ischemia and stroke	Yes	No
Thrombosis of veins and arteries	Yes	No
Bleeding complications	Low risk	High risk
Splenomegaly	40%	No
Peripheral blood smear	Giant platelets	Normal platelets
Platelet function	May be abnormal	Usually normal
Bone marrow	Megakaryocytic hyperplasia, dysplasia, giant form and dysplasia	Increased number of megakaryocytes, normal form

monitoring were maintained until the patient was discharged after 15 days of admission, to follow up with outpatient treatment. Analytical blood controls showed that the platelet count was down to normal levels (350.000.μL^{-1}) in 20 days.

Thrombocytosis is an incidental finding in 35-50% of cases and determination of the cause creates a diagnostic challenge. It can be classified into two types: essential thrombocytosis, a myeloproliferative disorder of the bone marrow, and reactive thrombocytosis, also called secondary thrombocytosis. This latter 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. Major causes of reactive thrombocytosis are described in Table 1.

In lung cancer patients, reactive thrombocytosis has a prevalence as high as 30% and has been linked with tumour extension or metastatic disease, and therefore with a poor prognosis. Reactive thrombocytosis may also appear with incidences of iron deficiency (6-12%), autoimmune disease (4-11%), cancer (1-3%) or drug-induced problems. Unlike in adults, one to two thirds of cases of reactive thrombocytosis in children are caused by viral infections (mainly respiratory, gastrointestinal and urinary viral infections).

It is therefore always necessary to distinguish between essential and reactive thrombocytosis, because their treatment and prognosis differ. The main features of both types of thrombocytosis are described in Table 2.

The reactive thrombocytosis found in patients with systemic inflammatory diseases is not the product of the isolated action of thrombopoietin, but its interaction with other plasma cytokines such as interleukin-6 [1]. 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 [2]. It is accepted that lower levels of platelets 1.000.000. μL^{-1} are a benign condition, although it remains unclear if these findings are associated with an increased post-operative 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 [3,4] appropriate.

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

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Konflikt interesów / Conflict of interest
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