coronary pretest, and essential thrombocythemia) is the combination of plaque rupture and superimposed thrombosis.

Our patient was young and had no risk factors for coronary disease; however, the OCT revealed mild atherosclerosis and plaque rupture, accounting for the mechanism of the coronary event. Furthermore, this finding allowed treatment optimization at discharge, since it supported the continuation of high-dose statin therapy together with anticoagulation therapy.

Optical coherence tomography is a high-resolution intravascular diagnostic technique. Initially, it was developed for identifying plaque instability, but nowadays it is also used to identify periprocedural complications, correct stent implantation and, as in our case, plaque rupture detection. (6)

This case is reported to consider the possibility of using OCT in this type of patients, not only to get information about the mechanism of thrombosis but also to guide treatment on discharge.

Conflicts of interest

None declared.

(See authors' conflicts of interest forms on the website/Supplementary material).

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Controversial Management of Severe Thrombocytopenia Induced by Abciximab

The three glycoprotein-IIb/IIIa inhibitors currently in clinical use, abciximab, eptifibatide and tirofiban, all share the same therapeutic target, namely blockade of the final common pathway of platelet aggregation and management of acute coronary syndromes. (1) Thrombocytopenia associated with glycoprotein-IIb/IIIa inhibitors occurs in about 1-2% of the patients exposed to this type of drugs. (2)

We report the case of a 50-year old, hypertensive, obese female patient, with type 2 diabetes. She was admitted with non ST-segment elevation acute coronary syndrome and maximum TnI of 0.55 ng/ml. Dual antiplatelet therapy was initiated with aspirin and clopidogrel, and anticoagulation with subcutaneous enoxaparin 1 mg/kg every 12 hours; a coronary angiography was performed. A drug-eluting stent was implanted in the mid-right coronary artery (RCA) with transient ST-segment elevation by microembilization of the acute marginal branch of the RCA. Intracoronary abciximab bolus application resulted in subsequent recanalization of the branch. Another drug-eluting stent was implanted in the most caudal branch of the obtuse marginal artery. Petechiae and ecchymosis in the upper limbs and body were targeted 24 hours after catheterization, progressing to the lower limbs. Control blood count revealed severe thrombocytopenia with 6,000 platelets/μl (199,000 platelets/μl before catheterization). Hematologists ruled out pseudothrombocytopenia, and recommended platelet transfusion if thrombocytopenia was suspected secondary to intracoronary abciximab administration.

The patient was transferred to the Intensive Care Unit (ICU) due to hypotension with SBP of 80 mmHg and anemia (hemoglobin 8.1 g/dl) (on admission, hemoglobin 12.8 g/dl), even though exteriorization of bleeding was not observed. During her stay in ICU, the patient had episodes of self-limited melena and increased cutaneous ecchymoses. Discontinuation of dual antiplatelet therapy was decided, informing her family of the high risk of severe hemorrhage and stent thrombosis. Three platelet concentrates were transfused while in ICU. Stabilization of platelets was achieved 4 days later. Antiplatelet therapy with aspirin was initiated on the 4th day after catheterization (46,000 platelets/μl), and on the 5th day, in view of hemoglobin stabilization and increased platelets (75,000/μl), clopidogrel therapy was restarted and was well tolerated, with no chest pain episodes or ECG abnormalities during follow-up. On discharge, 9 days after catheterization, the patient had platelet count of 230,000 platelets/μl and hemoglobin of 9.7 g/dl.

Patients with drug-induced thrombocytopenia (DIT) typically present with petechiae, ecchymosis and epistaxis caused by acute, and often severe, decrease of platelet production. When thrombocytopenia

is severe (<20,000 platelets/μl), bleeding may occur in the gastrointestinal mucosa or genitourinary tract. In extreme cases, intracranial or pulmonary hemorrhages have been reported, with fatal outcome.

Drug-induced thrombocytopenia diagnosis is often a challenge, particularly in polymedicated patients. It is necessary to consider pseudothrombocytopenia and heparin-induced thrombocytopenia as part of the differential diagnosis, as we did with our patient, since it is an exclusion diagnosis. (3) Pseudothrombocytopenia is the result of in vitro platelet agglutination due to EDTA-dependent anti-platelet antibodies. If a different anticoagulant such as citrate is used, actual platelet count can be performed, as we did in our lab test, confirming that the actual count was 6000 platelets/μl. (3) Type-1 heparin-induced thrombocytopenia (HIT) results from direct interaction of heparin with the platelet membrane, and occurs in up to 10% of patients treated with heparin usually within the first 72 hours (rarely achieving <100,000 platelets/μl), and is not associated with bleeding or increased risk of thrombosis. (3)

Type-2 HIT is caused by the formation of antibodies against heparin-platelet factor 4 (PF4) complex. These antibodies activate platelets and cause release of prothrombotic microparticles, platelet consumption, and thrombocytopenia. The main symptom is the sudden onset of thrombocytopenia involving a drop of platelet count by 50% compared to baseline levels, and/or thrombotic complications some 5 to 14 days after the start of heparin therapy, although it is usually related to long-term, repeated heparin treatments, which did not occur in our case.

In general, venous or arterial thrombotic complications are more common, and platelet count is higher than in thrombocytopenia due to abciximab. Based on clinical criteria, hematologists ruled out HIT, since our patient was treated only with low-molecular weight heparin before and sodium heparin during catheterization, but not after the procedure. Lab demonstration of platelet activation using an antigen or functional assay confirms the clinical diagnosis if considered necessary by hematology. (3, 5)

Abciximab is a chimerical monoclonal antibody (human/mouse) that selectively inhibits GP IIb/IIIa receptor, causing prolonged platelet aggregation blockage. (4) Approximately 1-2% of patients treated with abciximab have acute thrombocytopenia within a few hours after starting treatment. (5) Although its pathophysiological mechanism is partly unknown, it seems that patient antibodies are produced against drugs that recognize murine sequences in the complementary determining region 3 (CDR 3), which is an abciximab region. (5, 6) Some patients with abciximab-induced immune thrombocytopenia have preexisting antibodies in serum, but many healthy people who have never had contact with the drug before have IgG antibodies that react with abciximab bound to platelets. (4, 6)

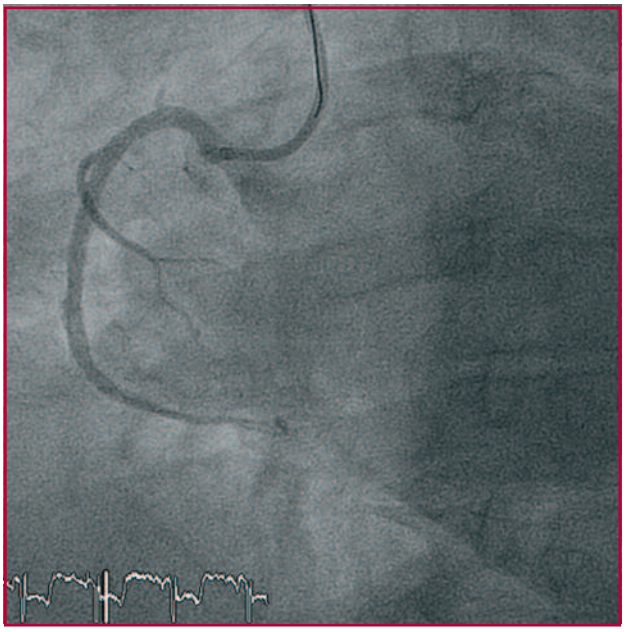

Fig. 1. Coronary angiography showing recanalization of the acute marginal branch of the right coronary artery after intracoronary abciximab bolus administration.

Management of thrombocytopenia secondary to abciximab is controversial, and platelet transfusion and immediate discontinuation of the medication seem to be the most effective measures; (5) in our case, abciximab had been administered as intracoronary bolus and not as continuous perfusion.

Discontinuation of other antiplatelet treatments is also controversial, and there is no consensus among experts. Corticosteroid administration has been researched along with platelet transfusion, with no usefulness evidence. (5) In our experience, we decided to make a balance between the high risk of hemorrhage due to thrombocytopenia and the high thrombotic risk of drug-eluting stents implanted 24 hours before. We found that despite the discontinuation of dual antiplatelet therapy, no acute or subacute thrombosis of the coronary stents occurred (currently 10 months of follow-up free of events), reducing potential hemorrhagic complications. The resolution stage of thrombocytopenia has not been longer than that referred in the literature (3-7 days).

These cases should be reported to the pharmacovigilance committee in each hospital for a more realistic reference of the incidence of thrombocytopenia in daily practice. In some centers, performing a blood count 2 hours after the start of abciximab administration is part of the protocol for an early diagnosis of thrombocytopenia. (5)

All medical professionals should take into account this rare complication in patients undergoing catheterization and treated with glycoprotein-IIb/IIIa inhibitors, for better monitoring and to avoid severe complications in the first hours after the procedure.

Conflicts of interest
None declared.
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Pulmonary Vein Stenosis: A Rare Case of Heart Failure and Pulmonary Hypertension in a Cancer Patient

We report the case of a 55-year-old female patient with no cardiovascular risk factors or cardiac history. The disease began with recurrent facial palsy and left trigeminal neuralgia. Computed tomography scan revealed cervical lymphadenopathies and tumor of the left maxillary sinus. A biopsy of the maxillary sinus was performed, evidencing lymphoproliferative syndrome, which was sent for typification.

A few months later, the patient presented cough and fever together with progressive dyspnea to functional class III in the previous week.

Hospitalization was decided due to febrile syndrome and dyspnea. Presumptive diagnoses were B symptoms due to lymphoma versus right upper lobe pneumonia. The patient had leukocytosis (32,000 WBC) and poor general condition. Culture samples were collected and an empiric broad-spectrum antibiotic therapy was started. A computed tomography scan showed multiple supra- and infra-diaphragmatic enlarged lymph nodes at the mediastinal level, like a large mass that moved the trachea and compressed the superior vena cava entry into the right atrium, and was in close contact with the atria. Bilateral infiltrates in frosted glass (pulmonary edema), compression of the right upper lobe, moderate bilateral pleural effusion with passive atelectasis, and moderate pericardial effusion were also detected (Figure 1A).

Aspiration-biopsy of bone marrow was performed: flow cytometry revealed 37% large cells consistent with large B-cell non-Hodgkin lymphoma (LBCNHL). It was decided to start the pre-stage with glucocorticosteroid therapy.

The patient was transferred to the Coronary Care Unit two days after hospitalization due to FC III dyspnea and pericardial effusion. Given its semiology (bilateral crepitant rales) and X-ray images (venous-capillary hypertension pattern), it was interpreted as left heart failure (Figure 2).

The first transthoracic echocardiography (TTE) showed normal dimensions and thickness, left ventricular preserved ejection fraction (EF) and prolonged LV relaxation pattern (diastolic dysfunction grade I), moderate pericardial effusion, severe pulmonary hypertension (PHT) (60 mmHg), and no valve diseases (Figure 3).

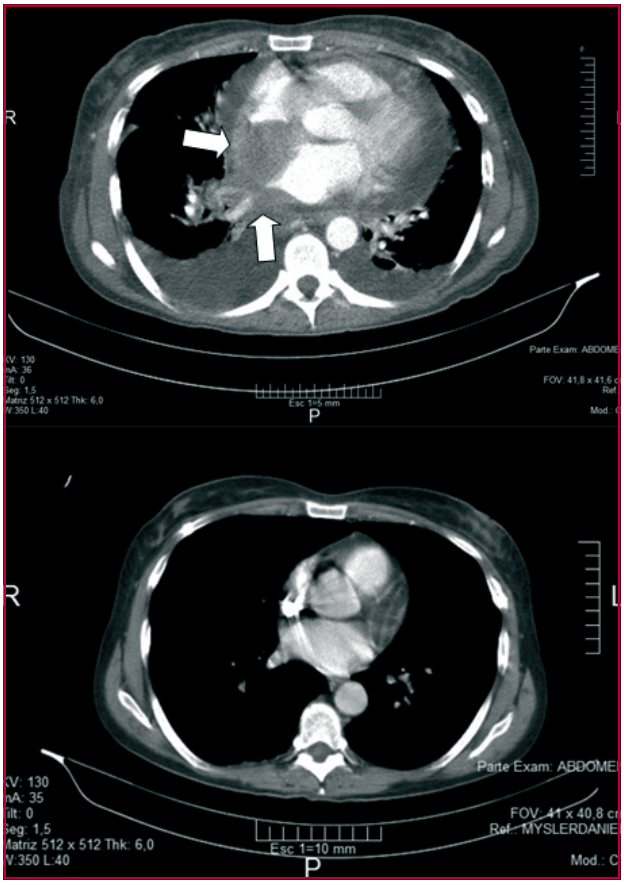

Fig. 1. Pretreatment chest computed tomography (CT) scan (1A top): mediastinal mass in close contact with right to left atria (*arrows*), bilateral pericardial and pleural effusion. Post-treatment chest CT scan (1B bottom): absence of enlarged lymph nodes, pleural or pericardial effusion.