Subacute Stent Thrombosis in a Patient With Idiopathic Thrombocytopenic Purpura Treated With Intravenous Immunoglobulin

To the Editor:

The use of intravenous immunoglobulin (IVIG) concomitant with platelet transfusion is indicated in patients with idiopathic thrombocytopenic purpura (ITP) in the case of life-threatening severe thrombocytopenia or hemorrhaging, or when therapeutic procedures with some risk of bleeding are performed.1

We describe a 44-year-old man with ITP who underwent a stress test for mixed angina attacks. Early positive results were obtained and, therefore, the patient also underwent coronary angiography. The thrombocyte
The patient's platelet count was 34,000/µL. The patient received 2 units of plasma and 6 of platelet concentrates before the procedure. An ulcerated thrombotic lesion in the proximal anterior descending artery was detected and treated by direct stenting with a drug-eluting stent. In addition, 8000 IU of intravenous heparin sodium and 300 mg of oral clopidogrel were administered; 3 hours later he was prescribed intravenous human immunoglobulin, 1 g/kg, for perfusion over 5 hours. This was repeated 24 hours later. Platelet count rose to 115,000/µL, and treatment with clopidogrel was started (75 mg/day); 5 days later, the patient presented precordial pain with ST elevation in the anterolateral aspect. Emergency coronary angiography showed complete thrombotic stent occlusion (Figure 1) that was treated by thrombectomy with an X-sizer and bare-metal stent (Figure 2). At discharge, oral anticoagulant therapy was prescribed and antiplatelet therapy was discontinued. The patient was asymptomatic 3 years later.

The prophylactic approach we used to prevent bleeding (indicated by the Hematology Department) was probably not the most adequate because the IVIGs were used 3 hours after the procedure, when the bleeding risk was lower, although it is true that clear recommendations in this regard have not been published. The placement of a drug-eluting stent may be criticized. However, the decision had been taken when the propensity of these devices to thrombosis, particularly late thrombosis, was less established. Furthermore, in our patient, the risk for dual antiplatelet therapy conditioned the choice of a bare-metal stent in the second procedure.

In patients with ITP who have undergone a percutaneous coronary intervention, IGIV should be administered only after careful consideration of the risks and benefits, and reserving it for patients at risk of severe hemorrhage, always considering the use of bare-metal stent over drug-eluting stent as the first option.

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REFERENCES


To the Editor:

At present, stent thromboses (ST) are relatively uncommon. Most occur within the first 24 hours (acute ST) or 1 to 30 days after implantation (subacute ST).1 In the case of bare-metal stents, late (>30 days) and very late (>1 year) thromboses are very rare. Drug-eluting stents considerably reduce restenosis and the need for new revascularizations2 and do not increase ST during the first year. However, drug-eluting stents appear to be associated with a slight increase (0.1%-0.2% per year) in the risk of very late ST.3

An association has been described between ST and stress testing, possibly as a result of increased platelet aggregation.4 This association has been described with bare-metal stents,5,6 but not drug-eluting stents, to our knowledge.

We describe a 56-year-old man with hypertension and dyslipidemia, former smoker, who presented an anterior myocardial infarction treated with thrombolysis. A year later, he had an anterior reinfarction, also treated by thrombolysis. Posterior coronary angiography showed severe stenosis in the middle-to-proximal left anterior descending artery. A Taxus stent of 2.75 × 28 mm (16 atm) was directly implanted and found to be successful on angiography (Figure 1). The patient was prescribed aspirin (indefinitely) and clopidogrel (1 year).

Angiographic follow-up at 8 months showed no restenosis. Fifteen months after stent implantation (3 months after clopidogrel was discontinued) with the patient asymptomatic, a symptom-limited stress test was performed with negative results. However, he presented chest pain 3 hours later that disappeared within 5 to 10 minutes; 8 hours later he presented persistent chest pain and went to the hospital after the first 2 hours of symptoms. Anterior reinfarction with criteria for reperfusion was documented and treated with thrombolytics. The patient presented pain and ST elevation.

Letters to the Editor

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