SCIENTIFIC LETTER

Antiaggregation Strategy post Coronary Angioplasty with Stent in a Patient with Severe Thrombocytopenia

Estrategia de antiagregación post angioplastia coronaria con stent en un paciente con trombocitopenia grave

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Dual antiplatelet therapy is a key element in the pharmacological treatment of coronary heart disease. It consists in the combination of aspirin (ASA) and a P2Y12 platelet inhibitor. This therapy reduces ischemic and stent thrombosis risk, but on the other hand, it increases bleeding risk. The combination of drugs and treatment duration is still today under constant debate. Scores such as the PRECISE DAPT score, the PARIS risk score, or the DAPT score are tools that are used in medical practice as a guide for decision making, although they do not include platelet count. On the other hand, the ARC-HBR (Academic Research Consortium for High Bleeding Risk) score includes as a criterion moderate to severe thrombocytopenia (platelet count less than 100 000/mm^3), which implies a risk of bleeding greater than or equal to 4% in 1 year, and that can sometimes be the greatest limitation of dual antiplatelet therapy. Patients with severe thrombocytopenia are usually excluded from the research studies giving origin to these scores, so decisions in this regard are subject to the experience of the treating medical team.

We present the case of a 52-year-old male patient, with a diagnosis of acute lymphocytic leukemia (ALL) associated with synchronous clear cell renal carcinoma and a history of acute myocardial infarction (AMI) in 2016, which required angioplasty with a bare metal stent (BMS) in the right coronary artery (RCA). He had an ongoing hospitalization for severe pancytopenia due to his oncohematological disease. Admission laboratory tests revealed hematocrit 22%, hemoglobin 7.9 g/dL, platelet count 25 000/mm^3, leukocytes 1190/mm^3, and unremarkable results for the remaining tests. He was asymptomatic for angina or anginal equivalents with an electrocardiogram (ECG) without pathological findings.

Prior to targeted treatment, a myocardial perfusion test with pharmacological stress was requested. At a dose of 30 mcg/kg/min of dobutamine, severe and extensive ischemia was evident in the middle and apical anterior, basal, and middle anteroseptal, basal and middle inferoseptal and inferoapical segments, compatible with the territory of the left anterior descending (LAD) artery. Results showed summed stress score (SSS) 28, summed rest score (SRS) 4, and summed differential score (SDS) 24, with dilation of the left ventricle (LV), radiotracer uptake in the right ventricle (RV) and drop in left ventricular ejection fraction (LVEF) after stress (45% to 32%).

A coronary angiography (CA) was performed with prior platelet infusion, which revealed LAD artery subocclusion and severe in-stent restenosis of the RCA. The case was discussed in an interdisciplinary meeting with Hematology given the high ischemic risk that would have prevented him from receiving onco-specific treatment, as well as facing its eventual complications (infectious, hemorrhagic, among others). As a consequence, revascularization was decided. Percutaneous transluminal coronary angioplasty (PTCA) was performed with insertion of a BMS in the LAD artery, without subsequent antiplatelet therapy given the very high hemorrhagic risk. The patient developed infectious and oncological complications, so he was transferred to the general ward. On the seventh day after stent placement and with 44 000 platelets/mm^3, ASA treatment at a dose of 100 mg per day was initiated. The rest of the hospitalization progressed without cardiovascular events, and without bleeding episodes up to discharge 26 days after admission. Five months after hospitalization, he continues antiplatelet therapy only with ASA, without presenting any intercurrences.

The case presented here raises a controversy that exceeds the guidelines present in the literature. Platelets in myelodysplastic syndromes often have abnormal concentrations or are dysfunctional, with

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high bleeding risk, even with platelet values over 100 000/mm^3. As in other hematological processes, (1) there are no recommendations on antiplatelet treatment for patients with severe thrombocytopenia due to myelodysplastic syndrome, who suffer an acute coronary syndrome or stable coronary disease.

In this type of patients, an angiographic study should be considered as a first measure since this procedure already presents a great challenge. The European cardio-oncology guideline is clear on preventive measures to reduce the risk of bleeding including, among other, platelet transfusion if they are below 20 000/mm^3, radial access, careful hemostasis, and low doses of heparin, between 30-50 IU/kg, (2) all actions that were carried out in our patient.

Regarding double antiplatelet therapy, expert agreements suggest starting it with platelet values above 30 000/mm^3, as well as opting for a type of stent that allows shortening its duration. (3) The European cardio-oncology guideline recommends using aspirin starting at 10 000 platelets/mm^3, and clopidogrel at 30 000 platelets/mm3 (there are experts who recommend cut-off values of 30 000 and 50 000 platelets/mm^3, respectively). (2) Our patient had less than 30 000 platelets/mm^3. In this sense, we have not found any similar reported case of severe thrombocytopenia.

The greatest evidence regarding thrombocytopenia and antiplatelet scenarios is provided by contradictory opinion articles. On the one hand, in patients with chronic coronary syndrome (CCS), they suggest stopping antiplatelet therapy and avoiding angioplasty if there is a platelet count of less than 50 000/mm^3. This consideration was considered in the discussion regarding our patient, but it could not be respected. (4) In patients with platelets between 50 000 and 100 000/mm^3, monotherapy with clopidogrel and proton pump inhibitor (PPI) is suggested, based on randomized studies, which mostly used second-generation drug-eluting stents (DES). Finally, in patients with CCS, symptomatic despite triple antianginal therapy, PTCA is reasonable when evaluating the risk-benefit ratio. If carried out, the suggestion is second-generation DES rather than BMS, and subsequent double antiplatelet therapy with ASA and clopidogrel for one month, and then continuing with clopidogrel as monotherapy, associated with PPI. The indication for second-generation DES arises from the evidence that demonstrates a lower rate of early stent thrombosis compared with BMS, considering dual antiplatelet therapy with the usual recommendations (duration and composition), a situation that is far from the scenario presented in our case, and that, in fact, would not be advisable to follow given the high ischemic risk that would arise if one did not comply with conventional treatment. (4)

On this issue, evidence has shown that DES reduces early restenosis and ischemia associated with the index lesion, when compared with BMS, but has failed to demonstrate superiority with respect to late thrombosis. (5,6) Therefore, BMS are reserved for patients who cannot receive double antiplatelet therapy for more than a month given the risk of bleeding, (6) and, as in our patient, in scenarios in which the standard double antiplatelet therapy cannot be administered. This type of stent presents the challenge of the risk of thrombosis during the first month, but once this period is over, the risk of late thrombosis would be reduced and thus the need for dual antiplatelet therapy, making simple antiplatelet treatment a more reasonable goal in these cases.

In conclusion, the evidence is scarce and divergent, so we sustain that the behavior to be adopted should be individualized, and of multidisciplinary decision, until more studies are developed

Ethical considerations
Not applicable.

Conflicts of interest
None declared.

(See conflicts of interest forms on the website).

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