

## Letters to the Editor

## Acute Coronary Syndrome in Patients With Thrombocytopenia



## Síndrome coronario agudo en pacientes con trombocitopenia

## To the Editor,

I read with great interest the article published in *Revista Española de Cardiología* by Bermejo et al.<sup>1</sup> about eltrombopag therapy for primary immune thrombocytopenia (ITP) in a patient with a recent acute coronary syndrome (ACS). I would like to make some additional comments about other scenarios involving thrombocytopenia that may be seen in ACS patients. As Bermejo et al. point out,<sup>1</sup> there is little published data on this subject. Some authors have proposed dual oral antiplatelet therapy in SCA patients with a platelet count of  $> 30 \times 10^9/L$ , and choosing a type of stent that allows shortening the therapy duration with the smallest risk of stent thrombosis.<sup>2</sup> The risk of bleeding in patients with thrombocytopenia depends not only on the platelet count, but also on the condition causing the thrombocytopenia.<sup>3</sup> Patients with PIT and platelet counts between  $20$  to  $30$  and  $50 \times 10^9/L$  generally have a stable clinical course with no bleeding complications.<sup>4</sup> The risk of severe bleeding in these patients is usually associated with platelet counts of  $< 10$  to  $30 \times 10^9/L$ , and patients of advanced age have a higher risk.<sup>4</sup> Therefore, the threshold of  $> 30 \times 10^9/L$  proposed by some authors for ITP patients seems reasonable. Another scenario is thrombocytopenia associated with chronic liver disease. Because of the increased prevalence of metabolic syndrome and the relationship of this condition with both vascular disease and steatohepatitis, which can progress to cirrhosis, chronic liver disease may be a common cause of thrombocytopenia in the future. The perceived bleeding risk in cirrhotic patients with thrombocytopenia and a high international normalized ratio may be higher than the actual risk<sup>5</sup> and lead to less intense antiplatelet therapy, thereby increasing the risk of thrombosis. Patients with liver cirrhosis have a balanced hemostatic status, in which the reduction in procoagulant factors is offset by a parallel decrease in anticoagulant factors. In addition, their increased concentration of von Willebrand factor, the main protein required for platelet adhesion, can compensate for the low platelet count and ensure primary hemostasis.<sup>5</sup> As occurs in relation to ITP, the available data do not suffice to establish firm recommendations on the most appropriate antiplatelet therapy for patients with cirrhosis-associated thrombocytopenia and ACS. Hence, expert recommendations have added value in these cases. In this regard, I had the opportunity to consult Dr Donald Cutlip (Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, Massachusetts) about his recommendations for cirrhotic patients receiving metal stents. For these cases, he proposed dual antiplatelet therapy with aspirin and clopidogrel for 14 days in patients with a platelet count of  $< 20 \times 10^9/L$  and for 14 to 30 days (according to the bleeding history) in those with counts of  $20$  to  $50 \times 10^9/L$ .

Two other scenarios to mention are chemotherapy-related thrombocytopenia in cancer patients and thrombocytopenia associated with myelodysplastic syndromes. Chemotherapy-related thrombocytopenia<sup>6</sup> is transitory and has a predictable

recovery period; there is a low risk of bleeding in patients with platelet counts of  $> 10 \times 10^9/L$ . Patients with thrombocytopenia associated with myelodysplastic syndromes usually have a chronic course, and their bleeding risk is higher than in those with chemotherapy-induced thrombocytopenia.<sup>7</sup> In myelodysplastic syndromes, platelets often express abnormally low concentrations of cell surface procoagulant markers or lack intracellular granules, and bleeding is common even in patients with platelet counts  $> 100 \times 10^9/L$ .<sup>8</sup> As is the case of the other conditions mentioned, there are no recommendations on the most appropriate antiplatelet therapy for patients with myelodysplastic syndrome-related thrombocytopenia who experience an ACS.

In conclusion, the bleeding risk associated with thrombocytopenia may differ depending on the origin of this condition. Over the next few years we will see more patients with thrombocytopenia who could develop an ACS, and these patients will require an appropriate antiplatelet strategy. It is important to develop the recommendations for this population in future clinical practice guidelines.

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