

Time Since Diabetes Onset as a Determining Factor in Platelet Reactivity. Response

Tiempo de evolución de la diabetes mellitus como factor determinante en la modificación de la reactividad plaquetaria. Respuesta
To the Editor,

We would like to thank de la Hera et al for their interest in our article¹ and their constructive comments based on their experience in the study of patients with coronary artery disease and diabetes mellitus.

Patients with acute coronary syndrome and hyperglycemia have platelet hyperreactivity and a worse prognosis. Although intensive control with insulin reduced platelet reactivity during hospital stay, optimized glycemic control after hospital discharge produced no long-term decrease in platelet reactivity.^{1,2}

We analyzed the percentage of patients with “occult diabetes” in our series. As this was not a specific objective of the study, it was not possible to apply the same criteria as in the report by de la Hera et al.³ Nevertheless, considering “occult diabetes” as that which had not been diagnosed at the time of hospital admission, but was associated with abnormally high glycosylated hemoglobin (HbA_{1c}) levels (according to the American Diabetes Association, HbA_{1c} ≥ 6.5%), in our series, we found a reduced number of patients (n = 10; 4 in the optimized group and 6 in the conventional group). With these figures, we found no significant differences in the baseline characteristics or in platelet reactivity at 12 months.

When we analyzed patients with known diabetes mellitus at the time of admission (n = 65; 36 in the optimized group and 29 in the conventional group; median time since diagnosis, 8.7 years), again, we observed no significant differences (mean [standard deviation] platelet aggregation after stimulation with 20 μM

adenosine diphosphate in the optimized treatment group, 60.4% [15.6%] vs 61.6% [17.3%] in the conventional group).

In summary, like de la Hera et al, we believe that time since diagnosis of diabetes mellitus is undoubtedly an important prognostic factor for cardiovascular events and, as these investigators do well to point out, it may help to explain the lack of improvement in platelet reactivity, despite the optimized glycemic control carried out in our study.

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