Impact of smoking habit on platelet reactivity in a cohort of patients admitted due to an acute coronary syndrome

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Background: Several pharmacodynamic studies have shown the impact of smoking habit on platelet reactivity; with a reduction on platelet aggregation. Wether this inhibition in platelet reactivity is due to tobacco effects in platelet signaling pathways or due to a pharmacodynamic interaction with antiplatelet therapies is not well stablished.

Purpose: Our aim was to study the influence of smoking habit in platelet reactivity and in the response to P2Y12 inhibitors.

Methods: Patients admitted in four tertiary care hospitals due to an acute coronary syndrome that undergone percutaneous coronary intervention (PCI) were consecutively and prospectively recruited. All the patients received dual antiplatelet therapy with aspirin and a P2Y12 inhibitor following current European Guidelines. Platelet function was assessed at day 1 and day 30 post-PCI by VerifyNow P2Y12, VASP (Vasodilator-stimulated phosphoprotein) y MEA (Multiple electrode aggregometry).

Results: A total of 1000 patients were enrolled, of whom 12 had to be excluded due to inaccurate processing of blood samples. 372 patients (37,6%) had smoking habit. Non-smoking patients showed higher prevalence of high blood pressure [423 (68.7%) vs 196 (52.7%)] and diabetes mellitus [213 (34.6%) vs 81 (21.8%)]. Smoking patients were younger [57.3 (9,6) years old vs 68.4 (11.1)], with higher incidence of acute coronary syndrome with ST segment elevation [184 patients (49,5%) vs 241 (39.1%), $p<0,001$]. There were no differences in platelet function at day 1. When analysing platelet function 30 days post-PCI, a lower inhibition of platelet reactivity in non-smoking patients as compared with smoking patients was observed in those treated with clopidogrel, with higher prevalence of clopidogrel-resistance in non-smoking patients (VerifyNow, 51,2% prevalence of high platelet reactivity in non-smoking patients vs 34,9% 30 days after PCI, $p=0,023$). On the other hand, smoking patients that received ticagrelor did not show any differences. Patients with smoking habit treated with prasugrel showed a lower response of borderline statistical significance.

Conclusion: Smoking habit was associated with a lower response to prasugrel of borderline significance, and with higher response to clopidogrel, according with previous studies suggesting a pharmacodynamics interaction between tobacco use and P2Y12 inhibitors.