

Rapid Desensitization to Acetylsalicylic Acid in Acute Coronary Syndrome Patients With NSAID Intolerance

To the Editor:

Acetylsalicylic acid (ASA), when used as an antiplatelet agent, plays an important role in reducing ischemic complications from coronary artery disease and percutaneous coronary intervention (PCI). It is recommended to be administered together with ticlopidine or clopidogrel to reduce adverse cardiovascular effects in patients with acute coronary syndrome and in those patients who require placement of a stent.^{1,2} Hypersensitivity or intolerance to ASA, both accepted terms when referring to allergic and pseudoallergic reactions to cyclooxygenase (COX-1) enzyme inhibitors, can manifest as rhinitis or rhinosinusitis and asthma, urticaria-angioedema, or anaphylaxis.³ It occurs in up to 10% of asthmatic patients, while the prevalence of urticaria due to ASA exposure varies between 0.07% and 0.2% in the general population.⁴ There are various reviews of studies on protocols for desensitisation to ASA in patients with cardiovascular disease,⁵⁻⁷ and they all agree on the importance of performing desensitisation once 2 factors appear: coronary artery disease and sensitivity to aspirin.

We present 5 patients between the ages of 53 and 79 years with a history of an adverse reaction after taking ASA in the form of urticaria in 3 of them and facial angioedema in the other 2. None of them had bronchospasm or an anaphylactic reaction. All suffered from acute coronary artery disease that required placement of an intracoronary stent and dual anti-platelet treatment with clopidogrel and ASA.

With the patients' permission and after signing informed consent, a rapid oral desensitisation scheme to ASA was performed, adapted and modified from Wong et al⁸ and Silberman et al,⁹ consisting of the administration of increasing doses at 15-20 minute intervals, beginning with 0.1 mg and ending with 100 mg (0.1, 0.2, 1, 3, 10, 25, 50, and 100 mg), performed in a hospital setting with continuous vital signs monitoring and strict medical oversight without premedication with antihistamines or corticosteroids. In all cases, the ASA desensitisation process was successful, achieving the recommended dose of 100 mg, with a maximum time of 2.20 hours without causing adverse reactions. Currently, the 5 patients tolerate

a daily dose of 100 mg of ASA. The total tolerance time from the moment of performing desensitisation to present varies between 5 and 46 months. ASA intolerance was not confirmed using a controlled oral provocation test when treating patients with ACS since this situation is contraindicated.⁸⁻¹⁰

Hypersensitivity to ASA appears to be a pharmacological process rather than an immune process due to the inhibition of COX-1 and it is dose-dependent. A diagnosis of ASA hypersensitivity normally leads to not prescribing this drug and, given that treatment with ASA reduces mortality in coronary artery disease, desensitisation is indicated in these cases. The published ASA desensitisation protocols propose administering the doses at intervals between 2 and 24 hours, according to the different guidelines. This long interval involves several days to complete desensitisation which is not recommended in patients with unstable coronary artery disease, especially after stent placement. In these cases, a short interval between doses like we propose here (15-20 minutes) facilitates urgent and rapid desensitisation. Unlike the Wong protocol, we did not premedicate with H₁ antihistamines and had good results.

A rapid ASA desensitisation guideline may be safe and effective in patients with a history of urticaria or facial angioedema that require dual anti-platelet treatment due to acute coronary syndrome.

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