

## Acute Coronary Syndrome in a Young Woman Treated With Sibutramine

**To the Editor,**

In Spain, pharmacologic treatment of obesity depends on 2 authorized drugs, orlistat and

sibutramine, which have different mechanisms of action and different secondary effects profiles.

Sibutramine is a selective inhibitor of monoamine re-uptake, especially of serotonin and noradrenaline (and, to a lesser extent, dopamine). It reduces food ingestion by increasing the sensation of satiety and attenuating the fall in the metabolic rate that usually occurs with weight reduction, probably by stimulating thermogenesis.<sup>1</sup> Bearing in mind its mechanism of action, one of its possible adverse effects may, logically, be increases in blood pressure and heart rate due to the inhibition of peripheral re-uptake of noradrenaline. This has led physicians to contraindicate sibutramine or recommend caution in its use in patients at high cardiovascular risk.

The SCOUT study (Sibutramine Cardiovascular Morbidity/Mortality Outcomes Trial) is currently under way. It aims to determine the effects of treatment with sibutramine or a placebo, in combination with lifestyle changes, on incidence of non-fatal myocardial infarction, non-fatal stroke, reanimation after cardiac arrest and cardiovascular death, in a population of obese patients with high cardiovascular risk.<sup>2</sup>

Reports that relate sibutramine use with acute coronary syndrome are few and far between.<sup>3,4</sup> We present the case of a young and otherwise healthy woman who presented acute myocardial infarction associated with sibutramine use.

This 39-year-old woman with high blood pressure, in treatment with candesartan, and presenting obesity, had been taking sibutramine for 12 days. From the outset, she presented higher blood pressure and palpitations. On admission, she presented intense retrosternal pain at rest, irradiating to the upper left arm, and perspiration lasting 15 min, which remitted after administration of sublingual nitroglycerine. She was asymptomatic on arrival at the emergency room. Her maximum creatine kinase and troponin T serum values were 388 IU/L (normal, <140) and 0.23 ng/mL (normal, <0.035), respectively, with an enzyme curve typical of acute myocardial infarction. Electrocardiograms performed without chest pain were normal throughout hospitalization and showed no alterations in contractility. Coronary angiography showed normal coronary arteries.

Our patient underwent computerized tomography of the chest and a study of hypercoagulability and urine toxins to rule out other possible causes of chest pain and elevated cardiac markers such as cocaine, viral myocarditis, aortic dissection, pulmonary thromboembolism, states of hypercoagulability, and autoimmune vasculitis.

Although it is practically impossible to demonstrate a causal relation, the patient's age, the fact that high blood pressure was her only cardiovascular risk factor, and the negative results of the other studies,

together with the coincidence between the start of drug treatment for obesity, lead us to conclude sibutramine use may have caused her myocardial infarction.

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