Reflections on Beta-Adrenergic Receptor Blockers and Cocaine Use. A Case in Point

To the Editor:

We present the case of a 41-year-old male, businessman, who went to emergency services due to thoracic pain with anginal symptoms, prolonged (1 h) and triggered at rest. Among his personal antecedents, he presented with smoking and dyslipidemia. He denied consumption of narcotics. An electrocardiogram showed ST segment elevation in II, III, and avF, along with a decrease in V_1 and V_2 (Figure 1). After 5 min in the emergency department, he had an episode of ventricular fibrillation which required electrical defibrillation. A few minutes later, the patient was tachycardic, hypertensive, and sweaty. After 1 minute, 1 mg of intravenous propranolol was administered, and following this, the patient suffered another episode of thoracic pain with higher elevation of the ST segment in II, III, and avF, along with a decrease in V_1 and V_2 (Figure 2). An urgent coronariography was carried out, and it showed a 60% stenosis of the mid circumflex artery. He was later sent to the coronary unit, where as a protocol, a urine drug test was requested, which resulted positive for cocaine (>5000 ng/mL). With this positive result, the patient was asked again about cocaine consumption, which he admitted to have consumed 4 hours before going to emergency department. The patient remained asymptomatic during the rest of his hospital stay, and on the fifth day of admission, he was discharged on treatment with aspirin and statins.
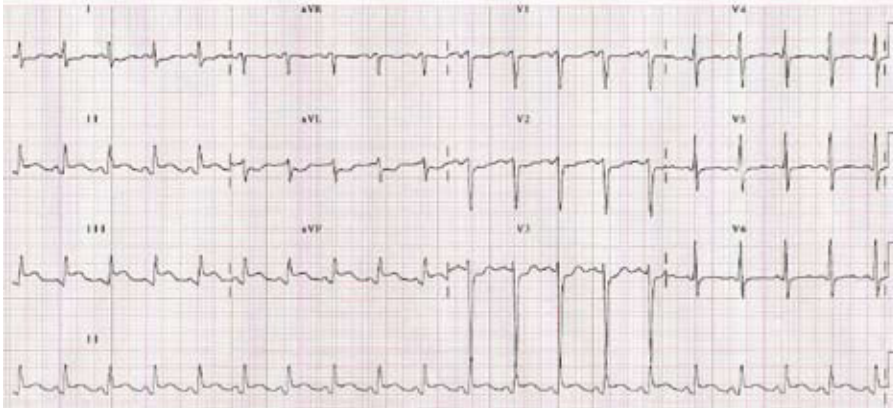
With this case, we have been able to prove the direct effects of intravenous administration of a beta-adrenergic receptor blocker medicine for a patient who goes to emergency services for an acute coronary syndrome with persistent elevation of ST, 4 h after consuming cocaine. Recently, Dattilo et al[1] have published a retrospective study where they recommend using beta-adrenergic receptor blockers after an acute myocardial infarction and cocaine consumption. In that study, they indicate that these could have a protective effect on the cardiac muscle, more important than its capacity for inducing coronary spasm. Additionally, European guidelines practically make no specific reference to the problem, but currently the American Heart Association[2] guidelines recommend not using these medications for patients with an acute myocardial infarction caused by cocaine, because of risk of exacerbating coronary spasm.

All of this puts into perspective the current lack of consensus regarding the management of these patients, and we consider that, given the growing number of similar cases in our emergency services, which is a direct consequence of the increased cocaine consumption towards in Spain, this issue and dealing with these kinds of patients should be examined most exhaustively.[3,4] It is certainly of vital importance to emphasize that acute coronary syndrome patients, especially those under 45 years without other added risk factors, have a well directed anamnesis done or are systematically requested for determination of narcotics in urine at their arrival to emergency services. The purpose of this is to identify cocaine consumers in order to give them more adequate treatment and therefore
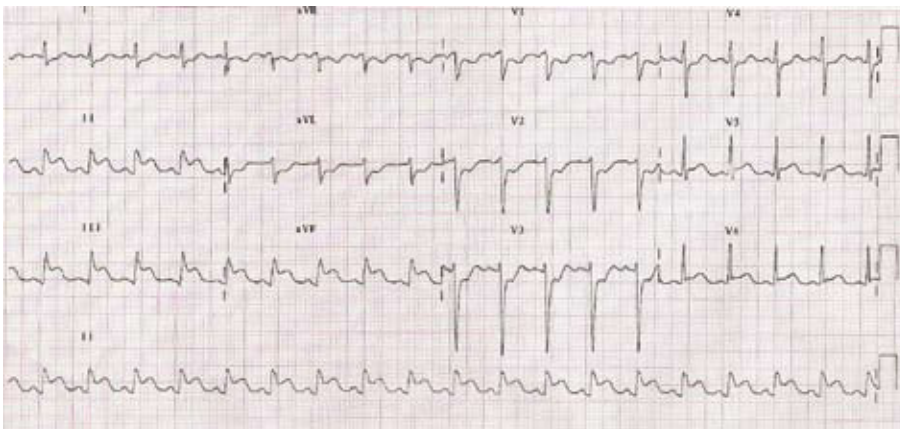
Figure 1. Electrocardiographic tracing with ST segment elevation in II, III, and avF, along with a decrease in V_1 and V_2.

Figure 2. Electrocardiographic tracing after administering intravenous propranolol.

avoid the undesirable effects of beta-adrenergic receptor blocker medicines, which we would usually administer if not suspecting use of this narcotic.

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