To the Editor:

Cocaine use has increased at an alarming rate, with the attendant increase in cardiovascular complications. Acute coronary syndrome associated with cocaine use is becoming more common, with a higher incidence than in the past. Therefore, it is crucial to understand the optimal treatment for such cases. In patients with cocaine-associated acute coronary syndrome, beta-blockers may be contraindicated due to the risk of exacerbating vasospasm and hypertension. Therefore, alternative medications, such as nitrates, calcium-channel blockers, and potentially reversible nitric oxide donors, may be more appropriate in these cases. Further research is needed to better understand the optimal management of cocaine-associated acute coronary syndrome.

References


Response

To the Editor:

Cocaine use has increased at an alarming rate, with the attendant increase in cardiovascular complications. Acute coronary syndrome related to the use of cocaine (or crack) can
no longer be considered incidental in Spain. Although statistics are inadequate and may vary from one area to another, Spanish hospitals with a catchment area of 500,000 inhabitants see about 2 to 4 cases each year. Although the relative incidence continues to be very low, the condition is still a medical problem, and the observation made by Burillo-Putze et al is important from the practical point of view.

Any form of cocaine—inhaled or smoked—inhibits presynaptic catecholamine and dopamine uptake, which increases stimulation of the respective postsynaptic receptors and, consequently, produces vasoconstriction, tachycardia, hypertension, and increased oxygen consumption. Coronary spasm, estimated by the decrease in coronary artery diameters, has been demonstrated in various studies, particularly when cocaine is associated with tobacco use. The use of beta-blockers to treat any of the cardiovascular manifestations of cocaine use (hypertension, arrhythmias, acute coronary syndromes) is controversial. Although beta-blockers can reduce hypertension and tachycardia, they can also induce vasoconstriction. In general, beta-blockers are contraindicated in patients with ischemia secondary to vasoconstriction, as was described in the consensus document of the European Society of Cardiology (page 1348). One of the few clinical trials (perhaps the only one) to address this problem was conducted with 30 volunteers who underwent catheterization and were given cocaine for diagnostic purposes, confirming a decrease in coronary artery diameters; subsequent administration of intracoronary propranolol increased the degree of vasoconstriction. Based on this unique study, the use of beta-blockers in cocaine users is contraindicated, although the study has important limitations: 1) in practice, intracoronary beta-blocker injections are not used, 2) propranolol is not the drug of choice in acute coronary syndromes, 3) other beta-blocker effects can be beneficial, and 4) no comments regarding general clinical data are reported. Another interesting and unanswered question has to do with the relative effect of different beta-blockers. Perhaps beta-blockers that also inhibit alpha receptors (the cause of cocaine-induced vasoconstriction), namely, labetalol, carvedilol, and bucindolol, will have a different effect than propranolol or other beta-blockers without alpha action. Unfortunately, this hypothesis has not been tested in clinical trials.

In short, vasoconstriction or coronary spasm plays an important role in patients with cocaine-induced acute coronary syndromes. This situation may be similar to acute coronary syndromes secondary to vasoconstriction outside the context of cocaine use, although no clinical studies have been done to support this recommendation. Although the European guidelines make practically no specific reference to the problem, a concerned reader can find highly specific recommendations in the most recent guidelines of the American Heart Association/American College of Cardiology. Nevertheless, these recommendations are based on logic, and not on clinical evidence, which is nonexistent. Antiplatelet therapy is essentially the same, and nitroglycerin is the preferred drug to treat acute ischemia or possible hyper tension. Tachycardia is treated preferably with diltiazem or verapamil. When there is persistent pain with ST segment elevation, emergency catheterization is recommended (due to potential coronary occlusion secondary to thrombosis, rather than spasm). Thrombolytic therapy is recommended if catheterization cannot be performed within 90 minutes. Careful use of beta-blockers in patients with hypertension and sinus tachycardia is recommended if the patient is also receiving nitrates or calcium blockers (IIb, evidence C). According to the same reasoning, it is also inadvisable to administer beta-blockers to stable patients who occasionally or regularly use cocaine, regardless of whether they have heart disease.