Cardiogenic shock due to occlusion of left main coronary in a cocaine user

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To the Editor,

Cocaine use has been related to the occurrence of myocardial infarction in young patients without other coronary risk factors. Acute myocardial infarction (AMI) secondary to the occlusion of the left main coronary artery (LMCA) in a cocaine user is infrequent, with sudden death being the most common form of presentation.

We present the clinical case of a 38-year-old male patient with an ST-segment elevation myocardial infarction complicated by cardiogenic shock because of acute occlusion of the LMCA following cocaine abuse.

He had a history of smoking and had recently used cocaine. He visited the emergency department of another institution with angina lasting for 5 h. The electrocardiogram (ECG) showed a heart rate of 120 bpm, complete RBBB with ST-segment elevation in lead aVR, lead I, aVL, and V2 to V6. The patient evolved with cardiogenic shock requiring mechanical ventilation (MV) and inotropic support. Considering the diagnosis of STEMI complicated with cardiogenic shock, the patient underwent coronary angiography. An intra-aortic balloon pump was placed before the procedure. The coronary angiography demonstrated a total acute thrombotic occlusion of the LMCA, and PPCI was performed. After the predilatation of the total occlusion with a balloon, TIMI-III flow was restored. A stent was successfully implanted.

After the procedure, the patient developed multiorgan failure (acute renal failure, liver failure, respiratory distress). Twenty-four hours later, he presented with ventricular tachycardia–ventricular fibrillation refractory, and the patient died.

In the present case, cocaine was presumed to be instrumental in provoking the AMI (patient with AMI was younger without classic risk factors). Cocaine stimulates the sympathetic nervous system by inhibiting catecholamine reuptake at sympathetic nerve terminals. Among them, include AMI and where the etiology is multifactorial (vasospasm, coronary dissection, atherosclerosis-plaque rupture, increased the determinants of myocardial oxygen consumption) (1).

Secondly, an acute obstruction of the LMCA is encountered at angiography approximately in only 0.5% of AMI cases (2), and it is associated with cardiogenic shock (2-4) as well as sudden death (5).

In patients with cardiogenic shock at admission, mortality was up to 32%–54% (3, 4). In patients with cardiogenic shock and multiorgan failure, mortality was up to 75% (4).

Coronary artery bypass graft surgery (CABG) is the standard revascularization strategy. However, normal blood flow in the infarct-related artery should be restored as rapidly and completely as possible; the high rate of mortality and of postoperative complications in patients with cardiogenic shock makes primary coronary intervention an alternative therapy. Percutaneous coronary intervention allows a rapid reperfusion of the vessel with a survival rate of 89% at 1 year (3, 4).

Among the variables associated with adverse outcomes, our patient presented with cardiogenic shock and underwent reperfusion therapy after 12 hours of symptom onset and multiorgan failure.

In conclusion, the etiology of AMI in patients with cocaine use is multifactorial. The occlusion of the LMCA is associated with high mortality secondary to cardiogenic shock. Survival depends on early reperfusion, and the appropriate strategy should be chosen based on the patient's hemodynamic status.

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DOI:10.5152/AnatolJCardiol.2015.6409