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Visual Case Discussion

Cocaine induced chest pain and acute coronary syndrome

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A 59-year-old male with a past medical history of COPD, hypertension, drug abuse and alcoholic pancreatitis presented to the emergency department with chest pain. He reported a one-week history of intermittent, compressive, non-radiating chest pain that was exacerbated following cocaine and alcohol use prior to arrival. He had associated nausea, vomiting, sweating, shortness of breath and dizziness. Apart from mild distress, the physical examination was within normal limits. The patient was treated with aspirin, morphine for pain control, famotidine and ondansetron for nausea and vomiting, and was started on heparin. The EKG done (Fig. 1) showed findings suggestive of an inferior STEMI.[1] He was admitted for percutaneous coronary intervention with the diagnosis of inferior STEMI following cocaine use. On cardiac catheterization, 99% occlusion of the proximal right coronary artery was discovered, which required stenting using 2 bare metal stents. Following the catheterization, his EKG improved significantly (Fig. 2). He was started on eptifibatide, ticagrelor, carvedilol, and atorvastatin after the procedure.[2,3] He recovered well and was discharged from the hospital within 3 days.

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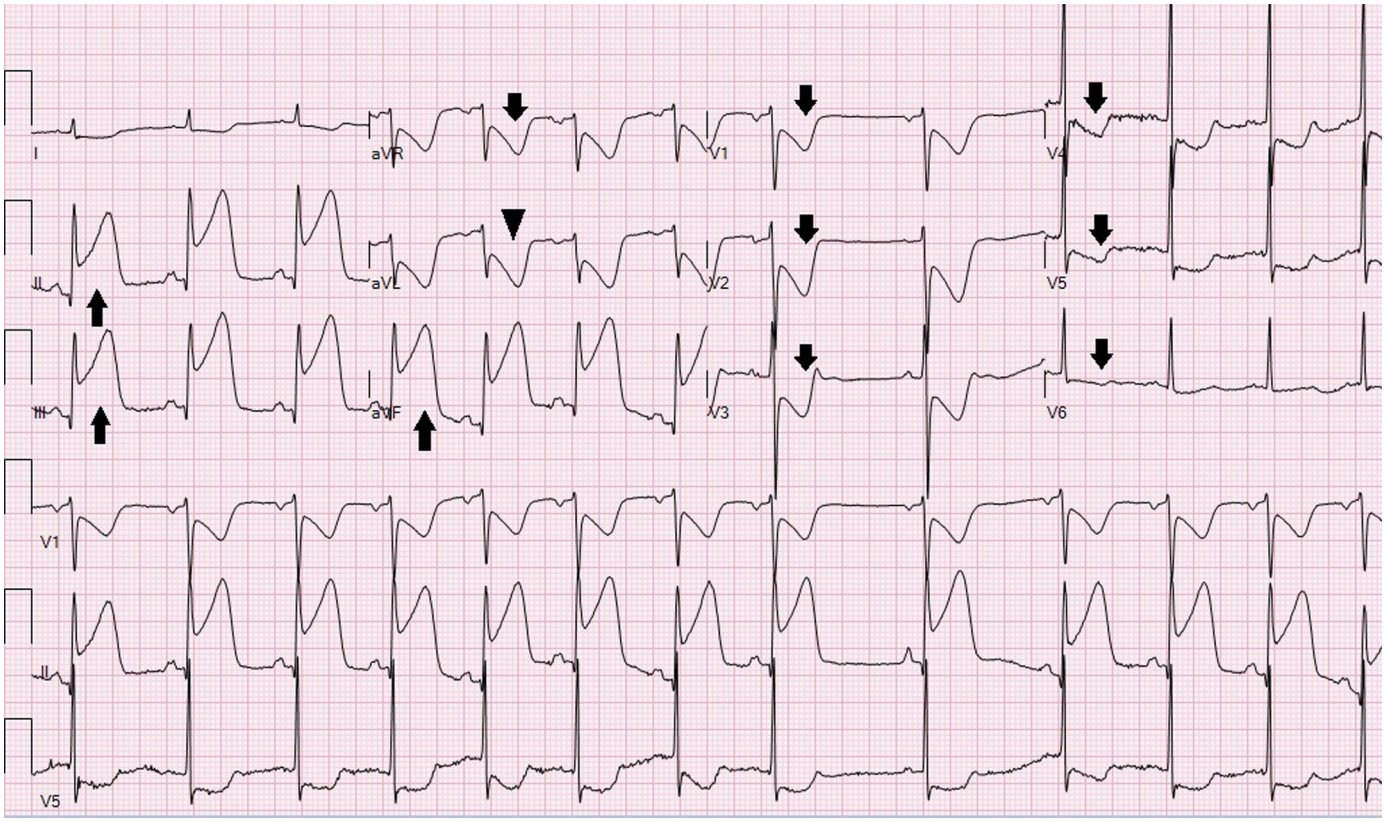

Fig. 1. An image of the patient's EKG on arrival with ST elevations in II, III, avF (arrows up) indicating inferior ischemia and ST segment depressions in avL (arrow head) denoting reciprocal changes, as well as in avR, and v1-6 (arrows down) indicating ischemia.

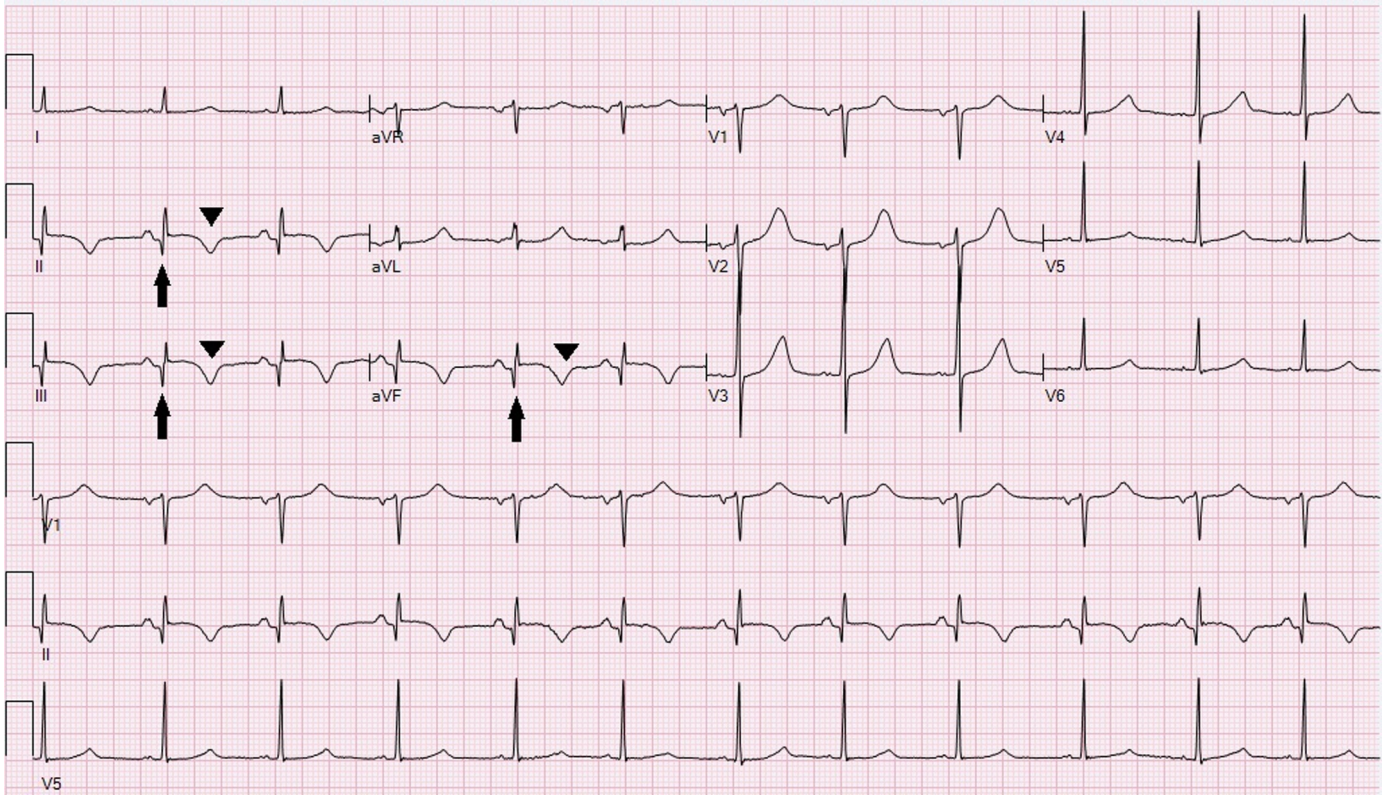

Fig. 2. An EKG after cardiac catheterization and stenting, showing EKG evolution in STEMI with Q waves (arrows) and T wave inversions (arrow heads) in leads II III, avF.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.visj.2019.100589.

References

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Questions

1. Which of the following drugs may be used for a patient with tachycardia not responding to benzodiazepines in the setting of cocaine-induced myocardial infarction?
   a. Non-selective beta blocker
   b. Selective beta blocker
   c. Alpha blocker
   d. Morphine

2. How long after cocaine use can metabolites possibly cause vasospasm and acute myocardial infarct?
   a. 2 h
   b. 12 h
   c. 24 h
   d. 48 h

Answers

1. Non-selective beta blocker. Explanation: β-blockers are a safe and effective treatment option for cocaine abuse with hypertension and tachycardia. Beta-blocker induced “unopposed α-stimulation” is a rare, inconsistent phenomenon. It is safe to use cardioselective beta blockers such as esmolol or metoprolol for patients with acute cocaine toxicity who are not responding to benzodiazepine therapy. Morphine has been classically used as an adjunct in ACS, but does not provide mortality benefit in this patient population. Reference: J.R. Richards, J.E. Hollander, E.A. Ramoska, et al. β-blockers, cocaine, and the unopposed α-Stimulation phenomenon. J Cardiovasc Pharmacol Ther, 2017;22(3):239–249.

2. 48 h. Explanation: Cocaine metabolites can be detected in the blood circulation for up to approximately 48 h after cocaine abuse. This may cause a delayed constriction of coronary arteries and late-onset ischemic symptoms. Clearance of cocaine is dependent on its breakdown to benzoylecgonine and ecgonine methyl ester; these metabolites can cause coronary vasoconstriction, an increase in mean arterial pressure and a widening of QRS, even after cocaine levels in the serum are undetectable. Reference: Chen Y, Ke Q, Xiao YF, et al. Cocaine and catecholamines enhance inflammatory cell retention in the coronary circulation of mice. Am J Physiol Heart Circ Physiol. 2005;288:H2322.