Short answer question case series: management of cocaine-associated chest pain

CASE VIGNETTE

A 35-year-old man presents to the emergency department complaining of chest tightness, shortness of breath and left arm heaviness that began 2 h after using crack cocaine and has been continuous for 4 h. He had one prior episode of chest pain (CP) associated with cocaine use and was told he may have had a 'baby heart attack', but he was not admitted and did not undergo cardiac catheterisation. An ECG reveals a mild sinus tachycardia of 105 beats/min, but is otherwise normal. He is given aspirin and morphine on arrival with complete resolution of his symptoms. Physical examination does not reveal any findings concerning for congestive heart failure.

KEY QUESTIONS

1. How does cocaine mediate its effects?
2. What is the risk of acute myocardial infarction (AMI) with cocaine-associated chest pain?
3. What medications should be considered in cocaine-associated chest pain?
4. What is the appropriate disposition for this patient?

DISCUSSION

1. Cocaine has many effects and points of action. It possesses sympathomimetic properties that are responsible for its desired euphoric effect but also causes tachycardia, hypertension and vasoconstriction due to both α and β adrenergic stimulation. It is also a slow sodium channel blocker that can cause PR and QT prolongation and lead to monomorphic ventricular tachycardia and idioventricular rhythms. In cases of acute ingestion, cocaine's adrenergic effects cause peripheral and coronary artery vasospasm and it acts as a procoagulant via its effects on the von Willebrand factor and other platelet aggregating cofactors. Additionally, chronic use of cocaine is responsible for non-ischaemic cardiomyopathy, endothelial wall stress predisposing to atherosclerosis and increased risk for myocarditis and endocarditis.[1]

2. The risk of AMI with cocaine-associated CP ranges from 1% to 6% depending on the study, with the true incidence being around 4–5% using an elevated troponin level as the diagnostic criteria. A subgroup analysis of the acute cardiac ischemia time-insensitive predictive instrument (ACI-TIPI) trial showed the incidence of AMI among patients with cocaine-associated CP at only 0.7%, with an acute coronary syndrome rate of only 1.4%.[2] However, several other studies reported an incidence closer to 6%. Unfortunately, very little angiographic data from cardiac catheterisation exists on these patients and it is unclear whether these 'troponin leaks' meeting strict definitions of AMI are truly significant with regard to long-term morbidity and mortality. The emergency physician's job is made more difficult by the fact that users of cocaine often engage in other high-risk behaviours, such as smoking, that increase the baseline risk for coronary artery disease. Therefore, the thrombolysis in myocardial infarction risk score (or another validated risk stratification tool) may still be useful and the use of cocaine may be considered additive with regard to risk. The emergency physician should bear in mind that none of these risk scores were applied to isolated cocaine-associated CP patients, so the RR of its use is not known.

Given its pharmacokinetics, most cases of AMI due to cocaine will present shortly after cocaine use[3] with a rapid reduction in risk thereafter. In the setting of a normal ECG, the risk of AMI is about 1.5% and only reaches 10% in the presence of ECG changes consistent with ischaemia.[4] Furthermore, the mortality rate from AMI due to cocaine is <1%.

The main complications from cocaine-associated AMI are dysrhythmias, with bradyarrhythmias and ventricular tachycardia being the most common. These usually occur within the first 12 h after use, but have been reported later due to co-ingestions and toxins as well as withdrawal.

3. Two studies among patients presenting to the emergency department with cocaine-associated CP found that the use of nitroglycerin, as well as benzodiazepines, is preferred to treat the CP and improve symptoms.[5 6] Benzodiazepines have been shown to be useful in reducing myocardial oxygen demand by decreasing the double product of heart rate times blood pressure. As in standard AMI, nitroglycerin improves perfusion via coronary vasodilatation. These two agents should be considered in all cases of cocaine-associated CP, with aspirin and heparin considered to be 'probably effective' in its treatment.[7]

The pure α-antagonist phentolamine, while traditionally used in patients with pheochromocytoma to reverse vasoconstriction, has been shown to be useful in cocaine-associated CP. The initial dose is 1 mg intravenously given slowly to avoid precipitation of hypotension and should be considered second-line treatment for these patients.[7]

In the face of persistent tachycardia and presumed ongoing myocardial injury, several options exist. Historically, β-blockers have been avoided due to the risk of extreme hypertension from unopposed α-agonism. While recent studies have suggested that in the subacute period of intoxication, more than 12 h, it may provide some benefit to those with CP, in the acutely intoxicated individual with CP it should probably be avoided in favour of safer alternatives.[8] Thus, some favour verapamil in cases of persistent tachycardia due to cocaine. It is the only calcium channel blocker that has been studied in this setting and it was found to be effective in improving mean arterial pressure and heart rate.

4. Because of the pharmacokinetics of cocaine and its associated risks, clinicians historically admitted patients with cocaine-associated CP. However, since the morbidity and mortality of cocaine-associated AMI is much lower than with coronary artery disease-associated AMI, many have questioned the cost-effectiveness of such a treatment approach. One multicentre trial found that patients with a normal ECG and negative serial enzymes could be discharged with very few risks (only 1.6% going on to develop mild AMI defined by enzyme increases).[9] Thus, many centres today admit these patients to a CP centre for a 12-hour observation period and enzymatic rule out.

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