

Cocaine Chest Pain

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Introduction:

Cocaine related myocardial ischemia costs an estimated 80 million dollars per year. 40 % of cocaine-related emergency department visits are due to chest pain. Incidence MI in cocaine-related chest pain is thought to be 6%. Given the potential morbidity and mortality in otherwise healthy patients the importance of understanding the presentation and management cannot be underestimated.

Pharmacokinetics:

Cocaine is absorbed from the respiratory tract and gastrointestinal mucosa in both the hydrochloride and base forms. Peak effect occurs in 1-2 minutes for inhalation and intravenous routes. Peaks occur at about 20 minutes for intranasal and 90 minutes for gastrointestinal routes. After inhalation or intravenous administration half-life is about 60 minutes. When via intranasal route half-life prolonged to approximately 2-3 hours.

Pathophysiology:

Cocaine causes hypertension, tachycardia, and vasoconstriction including locally in the coronary arteries. Cocaine is also known to cause thrombogenicity and accelerated atherosclerosis. Additionally it increases oxygen demand. All of these can lead to cardiac ischemia. Amount of cocaine used and the frequency of use are not related to likelihood of MI. Cocaine has also been associated with dissection of the coronary arteries, which can lead to myocardial ischemia and infarction as well.

Presentation:

Most patients develop chest pain and suffer their MI within 3 hours of use. Almost all patients develop chest pain within the first 24 hours (93%). Symptoms have been attributed to cocaine up to 4 days from use. The active metabolite norcocaine may be responsible through enterohepatic recirculation. The location and character of the pain are not predictive of MI and in fact atypical presentations predominate. This in combination with the unusual patient population (for MI) and potential delayed presentation make high index of suspicion paramount.

Workup:

Initial management of cocaine related chest pain should include benzodiazepines and the usual workup for myocardial ischemia including EKG, cardiac enzymes, and rule out of other etiologies for chest pain (dissection for instance). The EKG can be difficult to interpret in a young population with a high prevalence of early repolarization and left

ventricular hypertrophy- changes that can sometimes be difficult to differentiate from acute ischemia. 43% of patients using cocaine without MI in one study had ST elevation of greater than 1 mm in two contiguous leads. Enzymes may need to be checked depending on the situation. In one study, specificity of myoglobin and CK-MB were decreased and that of troponin was not altered.

Management:

Patients should be monitored, placed on oxygen, and have IV access established. Pharmacologic therapy includes benzodiazepines, aspirin, and nitrates. Benzodiazepines should be first line and will often alleviate symptoms on their own. In addition or in place of nitrates, use a calcium channel blocker or an alpha blocker for coronary vasodilatation. There is theoretical concern over the use of beta blockers because of unopposed alpha blockade. Even with short acting agents such as esmolol, exacerbation of hypertension has been noted previously. In terms of observation for sequelae and development of enzyme elevation indicating MI, the shortest observation period validated for cocaine-related chest pain is 12 hours. In a study done by Weber et al those patients with a negative workup and negative stress study at 12 hours had a death rate of 0 percent (95% confidence interval 0-0.99%) and a nonfatal myocardial infarction rate of 1.6% (95% confidence interval (1.1 to 3.1%). Other studies have noted low complication rate of those ruled out for MI. Of those that rule in for MI, 36% in one study developed a complication such as heart failure or a tachyarrhythmia. These complications largely developed in the first 12 hours.

In summary, cocaine-associated chest pain can lead to morbidity and mortality. These patients should be taken seriously, approached aggressively and likely observed for at least 12 hours.

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