Cocaine-associated myocardial infarction

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SUMMARY

Myocardial ischaemia and infarction has become a well-recognized sequelae of cocaine use. The possibility of recent cocaine use should be assessed in patients with potential myocardial ischaemia because the treatment of patients with myocardial ischaemia related to cocaine differs from that of patients with myocardial ischaemia unrelated to cocaine. Patients with cocaine-associated myocardial ischaemia should receive initial treatment with benzodiazepines to decrease central adrenergic stimulation. Aspirin should be used to reduce thrombus formation, and nitroglycerin to reverse coronary vasoconstriction. Patients with continued ischaemia can be treated with either low doses of phentolamine, or verapamil. If ischaemia continues after treatment with these agents mechanical reperfusion or thrombolytic therapy should be considered depending upon the clinical circumstances. Patients with myocardial ischaemia secondary to cocaine should *not* receive treatment with beta adrenergic antagonists as these agents enhance coronary vasoconstriction thereby worsening ischaemia.

INTRODUCTION

Myocardial infarction in temporal relation to cocaine use was first reported in 1982[1]. Since that time, emergency physicians and cardiologists have become increasingly familiar with cocaine-associated myocardial ischaemia. The early reports stressed the fact that this entity occurred in patients with normal coronary arteries[2]; coronary artery spasm was the presumed aetiology. With over 250 cases now reported[2–6], we have come to appreciate that cocaine-associated myocardial infarction results from a combination of the acute and chronic pathophysiological effects of cocaine on the cardiovascular and central nervous systems[7,8]. This review will highlight the differences in diagnosis and treatment of patients with myocardial infarction related to and unrelated to cocaine. It is precisely these differences in diagnosis, treatment, and prognosis that make it important for the clinician to discern whether patients have recently used cocaine.

Who sustains cocaine-associated myocardial infarction?

The typical patient with cocaine-associated myocardial infarction is a young male tobacco smoker with few conventional cardiac risk factors who presents to an emergency department with chest pain several hours after cocaine use[2–5]. However, there are many variations on this presentation[8,9].

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When a young patient without cardiac risk factors presents with potentially ischaemic symptoms it is customary to enquire about recent use of cocaine, but this practice should be extended to older patients as well[9]. Myocardial infarction has been associated with cocaine use in patients up to 73 years of age. In one clinical series, urine screening was done in all patients who presented to the emergency department with chest pain at four US hospitals: 18% of 41–50 year olds and 3% of 51–60 year olds had positive assays for cocaine metabolites[9].

PRESENTATION

Patients with cocaine-associated chest pain seem to be at similar risk of infarction to patients with potentially ischaemic chest pain unrelated to cocaine. In clinical series of patients with non-cocaine chest pain, about 10% sustained infarction[10–13]. In younger age groups the proportion is about 4%[11], whereas approximately 6% of patients with cocaine-associated chest pain proceed to myocardial infarction[41]. Are there any characteristic features? Although cocaine alters mentation and may distort perception of events, there is no evidence that these mind-altering properties alter the presentation of cocaine-associated myocardial infarction. Indeed, no patients are reported to have sustained cocaine-associated myocardial infarction in the absence of chest symptoms. The nature of the pain, however, can differ.

In acute myocardial ischaemia due to coronary artery disease (CAD)[10,11,14–17], the pain is seldom sharp, stabbing, or pleuritic; associated symptoms such as dyspnoea, nausea,

and vomiting are common; and the electrocardiogram is the single best clinical predictor of acute myocardial infarction[11]. By contrast, the Prospective Multicenter Evaluation of Cocaine Associated Chest Pain (COCHPA) found that conventional predictors of myocardial infarction perform poorly in patients with cocaine associated chest pain[4]. The pain is often described as sharp or stabbing (43%) or pleuritic (43%), and the electrocardiogram is less useful—in COCHPA, sensitivity for acute infarction was only 35% compared with 50% in non-cocaine chest pain patients. Conversely, abnormal electrocardiograms are more likely to mislead. In the clinical series that addressed this issue, most patients with cocaine-associated chest pain were reported as having abnormal electrocardiograms even though only 0–6% sustained an infarction[4,6,18,19]. These electrocardiograms, often showing early repolarization, may actually be normal variants in the young[20].

SEROLOGICAL MARKERS

The utility of various cardiac markers has also been questioned. Creatine kinase tends to be high in cocaine users, because of enzyme release from skeletal muscle[21]. Thus, a raised creatine kinase on admission for chest pain can be misleading. Even MB creatine kinase, though more specific for myocardial injury, can be above normal in cocaine users without infarction[22,23]. Marker proteins such as troponin I are likely to be more useful. At present, most patients with cocaine-associated chest pain have to be admitted to hospital[4].

PATHOPHYSIOLOGY

The pharmacological effects of cocaine on the cardiovascular system are, at least in part, the result of blockade of the reuptake of adrenaline and noradrenaline, in combination with increased presynaptic release of these excitatory neuromodulators. Centrally, the mechanism is less clear. Cocaine may enhance the release of noradrenaline and/or block the neuronal reuptake of dopamine. Excitatory amino-acids may also play a role.

According to the Goldfrank–Hoffman model of cocaine toxicity (see Figure 1)[7], central nervous system stimulation may result in central nervous system toxicity (such as seizures) as well as exacerbate the peripheral effects of cocaine. Suppression of the central nervous system effects might prevent the peripheral sympathomimetic effects due to central excitation, but blockade of the peripheral manifestations (for example, the cardiovascular effects) might not lessen the central nervous system toxicity.

The pathophysiology of cocaine-associated myocardial ischaemia is complex. The sympathomimetic effects of cocaine (tachycardia and hypertension) increase myocardial oxygen demand[24,25] while reducing coronary artery blood

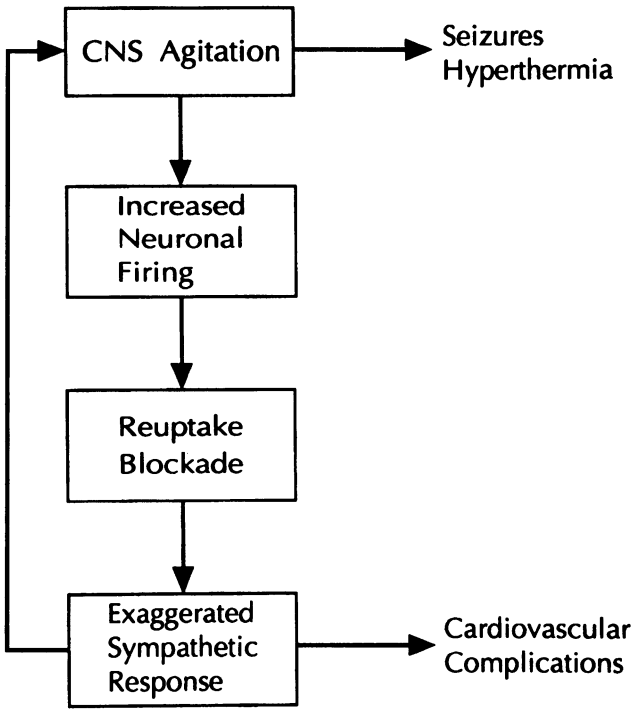

Figure 1 **Goldfrank–Hoffman model of cocaine toxicity**

flow through α-adrenergic mediated coronary artery vasoconstriction[25]. Additionally, cocaine acutely increases thrombogenicity[26], enhances platelet aggregation through adenosine diphosphate, and decreases fibrinolysis by increasing levels of endogenous inhibitor of tissue plasminogen activator[27,28].

In chronic users, left ventricular hypertrophy and premature coronary atherosclerosis may develop[29–35]. The early atherosclerosis is particularly troubling since coronary vasoconstriction secondary to cocaine is enhanced at sites of underlying atherosclerotic disease[36]. Coronary artery dissection and increased carboxyhaemoglobin levels (following 'crack' use) have also been reported[37,38].

HOW SHOULD YOU TREAT PATIENTS WITH COCAINE-ASSOCIATED MYOCARDIAL INFARCTION?

The treatment of cocaine-associated myocardial ischaemia has not been studied in randomized controlled trials. Most current recommendations are based on successful therapies for conventional acute myocardial infarction combined with an understanding of the pathophysiology of cocaine intoxication.

The mainstays of treatment for patients with myocardial infarction *un*related to cocaine include aspirin, heparin, nitroglycerin, beta-adrenergic blockade and thrombolytics. However, in cocaine-associated infarction beta-adrenergic antagonists are contraindicated; and thrombolytics, although possibly safe, may have limited benefit.

In managing the cardiovascular effects one must ensure that the central nervous system effects are not enhanced. Reversal of coronary vasoconstriction, hypertension, tachycardia, and predisposition to thrombus formation are the main methods to decrease myocardial oxygen demand and improve coronary artery perfusion and oxygen delivery; and central nervous system protection and decreased sympathetic outflow can be accomplished with benzodiazepines. Multiple animal experiments and widespread clinical experience support the use of diazepam as the initial agent for management of cocaine-intoxicated patients[39–42]. Diazepam alone will often return vital signs to the normal range. Reduction in hypertension and/or tachycardia (present in over one-third of patients with cocaine-associated chest pain) will decrease myocardial oxygen demand. Attempts to decrease hypercoagulability with aspirin or heparin seem reasonable, though clinical and experimental data are lacking.

Specific anti-ischaemic therapy begins with nitroglycerin. In conventional acute infarction, nitroglycerin reduces infarct size, cardiovascular complications, and mortality[43,44]. In cocaine-induced coronary artery vasocontriction[45] it relieves chest pain[46]. Although benefits in terms of infarct size reduction or mortality have not been assessed, the direct vasodilatory effects, the experimental reversal of coronary vasconstriction, and the clinical relief of chest pain make a case for its use. Sublingual nitroglycerin seems to relieve chest pain most rapidly, and this can be followed with either topical or intravenous administration[45].

Patients with continued myocardial ischaemia despite nitroglycerin should be treated with phentolamine, calcium channel blockers, or thrombolytic agents depending on the clinical circumstances. Phentolamine received a lot of attention before the advent of thrombolytic therapy in the early 1980s, when small clinical trials suggested benefit in conventional acute myocardial infarction, but larger trials were never conducted[47,48]. With respect to cocaine, phentolamine blocks the α-adrenergic effects and reverses the coronary vasoconstrictive effects of cocaine; therefore, it may be useful in these patients[25] (one case report records efficacy in cocaine-associated chest pain[49]). Phentolamine should be used with caution since after benzodiazepine it can precipitate hypotension; low doses (1 mg) may be safe.

Verapamil, a calcium channel antagonist, reverses cocaine-induced coronary artery vasocosntriction[50]. Unfortunately, work in animals points to enhancement of central nervous system cocaine toxicity by concurrent administration of any one of several calcium antagonists (verapamil, diltiazem, and nifedipine[51]). Coupled with their limited success in conventional myocardial infarction, this form of toxicity makes calcium antagonists unattractive in cocaine-associated infarction[52–54].

What of thrombolytic therapy—the standard of care for cocaine-unrelated myocardial infarction? Although initial concerns regarding safety have been tempered, efficacy has not been demonstrated in this patient population[55]. Opponents of the use of thrombolytics raise concerns regarding the diagnostic specificity of the electrocardiogram and the low mortality secondary to cocaine-associated myocardial infarction. From 11% to 43% of patients with cocaine-associated chest pain without myocardial infarction met TIMI criteria for the administration of thrombolytic agents despite the fact they did not infarct[18–20]. The potential administration to patients without infarcts and the very low mortality of cocaine associated myocardial infarction are arguments against use of thrombolytic agents. Patients who have new ST segment elevations (compared with previous electrocardiograms) or echocardiographic wall motion abnormalities may be suitable candidates since 'false positive' ST elevations should then be less of a concern.

The reasons for avoidance of β-adrenergic antagonists in patients who have recently used cocaine are that in several experimental models they increase the central nervous system toxicity[39–41]; that in clinical series they have failed to reverse the hypertensive and tachycardic effects of cocaine[56]; and most convincingly, that they exacerbate cocaine-induced coronary artery vasoconstriction[57,58]. In enhancing the α-adrenergic agonist effects of cocaine[59] labetalol does not seem to offer any advantage over pure β-adrenergic antagonists[8,59].

The treatment of post-infarction arrhythmias in relation to cocaine has not been well studied. Some have speculated that the local anaesthetic and sodium channel blocking effects of cocaine might be worsened by concomitant lignocaine administration, but work in animal models has yielded conflicting results[60–63]. Only one group has studied this issue clinically and no serious arrhythmias or central nervous system toxicity was seen after lignocaine administration[64] (though most patients in that series received lignocaine more than 4 hours after their last cocaine dose). Sodium bicarbonate narrows the QRS in cocaine-toxic animals[65], but its use has not been studied clinically. Sodium bicarbonate may be useful for the early treatment of cocaine-induced arrhythmias, with lignocaine the preferred agent for late arrhythmias.

Diagnostic evaluation after cocaine-associated myocardial infarction

Patients with cocaine-associated myocardial infarction have a very low acute mortality (0–4%)[3,5]. Most of the cocaine-associated cardiovascular complications are identified within a few hours of hospital arrival[5]. Patients who have chest pain without infarction have an even lower risk of complications[4]. One-year mortality is most closely related to continued cocaine use and concomitant medical problems, particularly HIV disease[66].

Patients who sustain a cocaine-associated infarction have a 31–67% prevalence of underlying coronary artery disease[3,67].

As in other young patients with myocardial infarction, those with CAD usually have single vessel disease and seldom left main disease[67–69]. The presence of coronary artery disease in patients with cocaine-associated myocardial infarction is related to age and the number of cardiac risk factors[67]. Patients over age 40 or with two or more cardiac risk factors should probably undergo cardiac catheterization. What of exercise stress testing? These patients do not tolerate high levels of exercise and have difficulty completing the protocols. For a fit young patient with few cardiac risk factors, an exercise stress test would be a reasonable start to evaluation.

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