Cocaine-induced myocardial infarction: not your average acute coronary syndrome

Introduction

Cocaine use in the UK has increased substantially over the past 10 years with a year on year rise in the prevalence of 16–59-year-olds who admitted using the drug (Hoare and Flatley, 2008). The most common reason for those who use cocaine to present to medical services is chest pain; 6% of patients subsequently have a myocardial infarction confirmed by raised cardiac enzymes (Weber et al, 2000). Diagnosis is often difficult as many patients initially deny use of cocaine, and non-specific electrocardiogram (ECG) changes are common, so treatment is often delayed and valuable myocardium lost. This article describes a young woman with a delayed presentation of severe chest pain who initially denied use of illegal substances. As the prevalence of cocaine use continues to increase, physicians must ask about illicit drug use in all cases of chest pain.

Discussion

This case illustrates the difficulties of diagnosis and treatment of cocaine-induced chest pain. Patients are often young, may present late after the onset of pain, and often initially deny using illicit substances. All patients presenting with chest pain should be asked specifically about recreational drug use, in particular if they use cocaine, as this is often overlooked (Wood et al, 2007).

Cocaine prevents presynaptic re-uptake of catecholamines, acting as a powerful cardiac stimulant accentuating the actions of the sympathetic nervous system. In the acute phase cocaine can induce tachycardia, hypertension, arterial vasoconstriction and anxiety, and has been associated with spontaneous aortic and coronary dissection, mesenteric ischaemia, stroke and venous thrombosis. Early onset atherosclerosis and aneurysmal (ectatic) coronary disease is common (Satran et al, 2005) and dilated cardiomyopathy has been described with chronic use (Kloner et al, 1992). Atherosclerosis is accelerated when cigarette smoking is combined with cocaine use (Hollander, 1995).

In addition to its atherogenic properties, cocaine use may precipitate myocardial infarction as it increases blood viscosity and coagulability via elevated levels of von Willebrand factor and plasminogen activator inhibitor (Siegel et al, 1999). Subsequently, a mismatch of oxygen supply and demand in the myocardium, coronary vasospasm, stimulated alpha adrenergic (increased heart rate and systemic arterial pressure) and beta adrenergic receptors (positive inotropy) all contribute to ischaemia.

An abnormal ECG will be recorded in 56–84% of patients with cocaine-induced chest pain and 43% will meet the ST segment ECG criteria for thrombolysis. However, ECG changes are problematic for two reasons. First, many patients are subsequently found to have normal cardiac enzyme levels. Second, what is often taken as ST segment elevation may in fact be an abnormal repolarization pattern caused by repeated cocaine abuse (Hollander et al, 1994). In addition, some individuals (often young and of Afro Caribbean origin) have a normal variant high take off at the J point (where the QRS complex joins the ST segment) which makes interpretation of the ST segment difficult (Hollander et al, 1994). Therefore, the ECG may be less useful in predicting an infarct in those using cocaine.

A positive troponin in the appropriate clinical setting confirms infarction but this is often retrospective and too late for immediate reperfusion therapy. A urine test for illicit substances may be of value, and rapid screening urine tests can confirm the presence or absence of cocaine within 10 minutes (Tomaszewski et al, 2005), but their use is currently not widespread.

Cocaine-induced myocardial infarction requires different management to other acute coronary syndromes. Nitrates and calcium-channel blockers are preferred as

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Case Report

A 31-year-old woman presented to accident and emergency at 03.20 hrs complaining of central chest pain since the preceding evening. She denied any significant medical history or family history and initially denied taking any medication or drugs of abuse. On examination her pulse was 110 beats per minute and her blood pressure 126/82 mmHg but there were no other abnormal physical findings. Her initial electrocardiogram showed multiple ventricular ectopics with periods of idioventricular rhythm but no acute ST segment elevation that met thrombolysis criteria (*Figure 1*).

She was initially treated with aspirin, low molecular weight heparin and intravenous nitrates. Although her symptoms of pain resolved overnight, the electrocardiogram evolved Q waves suggesting a significant anterior myocardial infarction (*Figure 2*). This was confirmed by a raised cardiac troponin T level at 12 hours.

It was not until some 15 hours after admission, and only after direct questioning, that she admitted to being a regular cocaine user, often using up to 3 g per week. She had smoked a pipe containing crack cocaine the preceding evening, and then immediately afterwards injected cocaine into a femoral vein. She described a euphoric rush followed by a non-resolving severe chest pain.

She underwent coronary angiography 36 hours after presentation which revealed a thrombus in the proximal left anterior descending artery (*Figure 3*). The coronary arteries were angiographically normal, with no evidence of angiographic atheromatous disease. She was treated with intravenous heparin and abciximab, and thereafter remained pain free and haemodynamically stable. An echocardiogram showed severe left ventricular impairment (ejection fraction 30%) with gross apical dyskinesis. She was discharged on aspirin and an angiotensin-converting enzyme inhibitor and given strong advice to cease using cocaine. She failed to return for pre-arranged follow up, was not registered with the GP she indicated and could not be traced through the NHS database.

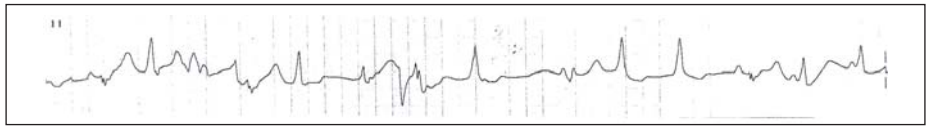

Figure 1. Multifocal ventricular ectopics.

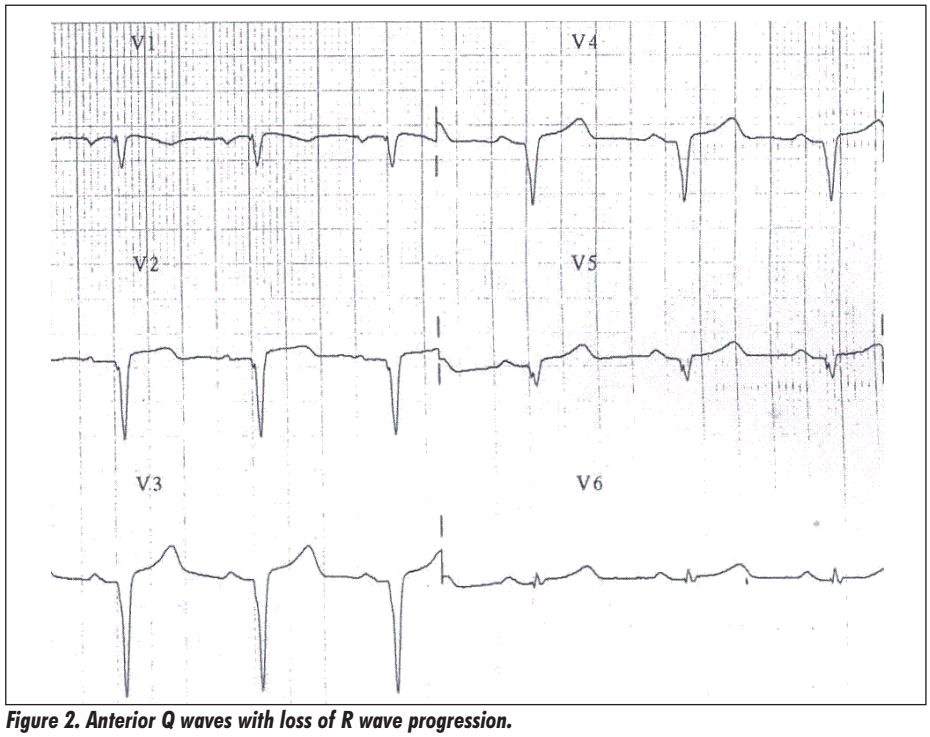

Figure 2. Anterior Q waves with loss of R wave progression.

they reverse cocaine-induced hypertension and coronary artery vasoconstriction, whereas beta-blockers may promote unopposed alpha-adrenergic activity and potentiate cocaine-induced coronary artery vasoconstriction (Lange et al, 1990), increased systemic arterial resistance and hypertension. Diazepam's anxiolytic properties may help reduce sympathetic drive.

A delay in presentation and diagnosis means that the window for thrombolysis is often missed. Anecdotal reports of increased bleeding complications with thrombolysis (Hollander et al, 1996), despite the hypercoagulable state induced by cocaine, mean that many feel percutaneous coronary intervention is safer if symptoms and ECG changes do not respond to initial medical therapy. McKee et al (2007) described an increased risk of stent thrombosis in cocaine abusers, raising doubts about the long-term efficacy of percutaneous coronary intervention.

In one series 12% of patients with cocaine-induced chest pain showed ST segment elevation or depression, elevated levels of cardiac injury markers, or ongoing pain or haemodynamic instability at presentation that required urgent admission; of these, about 50% subsequently had a myocardial infarction confirmed (Weber et al, 2003).

Long-term management involves optimizing cardiac risk factors including strong advice to cease using cocaine. In those with cocaine-induced chest pain but no evidence of infarction or cardiovascular complications in the first 12 hours, the risk of death or myocardial infarct is very low over the following month (Weber et al, 2003).

Conclusions

Cocaine-associated chest pain is an increasingly common presentation to medical services. While the management in most patients is benign, the management of those with suspected myocardial infarction differs from the standard protocol. Patients must be directly questioned and probed about any illicit substance use as they often deny this at first. Furthermore, diagnosis of a suspected cocaine-induced myocardial infarct early on is crucial for optimal medical management and early reperfusion therapy; in this regard a rapid screen urine test may be useful. Strong advice to cease using cocaine must be emphasized: continued use precipitates further attendances to medical services. BJHM

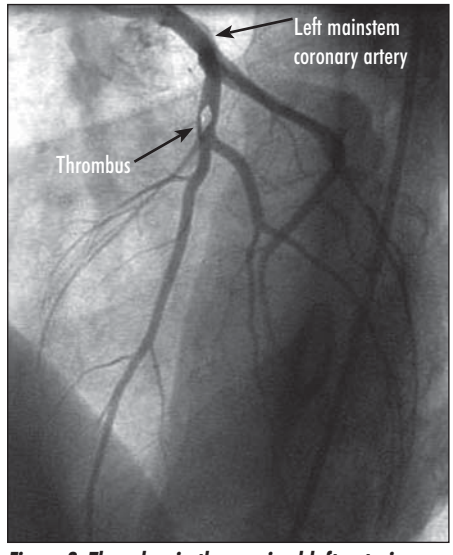

Figure 3. Thrombus in the proximal left anterior descending artery.

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