

Chest Pain In a Previously Healthy Young Male: Possible Cocaine-Induced Vasospasm?

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CLINICAL POINTS

- When taking a history of chest pain always enquire about smoking status (pack years), presence/absence of diabetes mellitus, hypercholesterolaemia, hypertension, and whether there is a previous history of cardiac disease
- In any pain history enquire about: site, onset, character, radiation, associated features, timing, relieving/aggravating factors and severity
- When a patient presents with chest pain an ECG, a chest x-ray and cardiac troponins must be ordered
- Always consider the possibility of cocaine use in a young patient presenting with chest pain in the absence of risk factors regardless of professional or social status

Patient X, a 22 year old Caucasian male presented to St James Hospital (SJH), Dublin complaining of 'unbearable' central chest pain that began 1 day prior to presentation.

History of The Presenting Complaint: X described a central, retrosternal pain that was constant, dull and crushing in character with a superimposed, intermittent and stabbing element. The pain began 1 day prior to presentation. It radiated to the upper pectoral region, but not to the jaw or left arm. There was no associated nausea, sweating or vomiting. The pain was aggravated by inspiration and movement, and was not relieved by simple analgesia. X graded the pain as 7/10 but graded the intermittent, stabbing element as 10/10. X denied any previous episodes of similar chest pain. After much initial denial, X eventually admitted to cocaine use on the evening prior to the onset of pain. Following physical exertion on the following afternoon, he developed the chest pain. He thought the pain would resolve spontaneously but when it failed to do so he became concerned and attended the emergency department at SJH.

Past Medical History: X has no previous medical history of note and has had no prior admissions to hospital.

Past Surgical History: X has no surgical history of note.

Medications: X does not take medications regularly and he has no known drug allergies.

Family History: X has a positive family history of cardiac disease.

Social History: X is married and is a banker, working in Dublin. He is a smoker of 15/day for 2 years, he admits to cocaine use once a month, a habit he began 5 months prior to presentation. He is a social drinker (approximately 18units/week).

Review of Systems: Musculoskeletal - X described a feeling of tenderness over the left pectoral region which is exacerbated by movement.

On Examination: X appeared alert and orientated in person, time and place. He was not in any significant discomfort but was notably agitated and anxious. His vital signs were normal (BP 127/85mmHg, Heart-rate 73bpm, temperature 36°C, respiratory rate 12/minute and O₂ saturation 99% on room air).

Respiratory Exam: X complained of pain over the left pectoral region on movement (sitting forward) and on inspiration. His lungs were clear to percussion and auscultation.

Cardiac Exam: The JVP was not elevated and there was no ankle oedema. The apex beat was not displaced and no thrills or heaves were evident on palpation. Heart sounds 1 and 2 were present; with no added sounds and no murmurs.

Gastrointestinal Exam: The abdomen was soft, non tender and not distended. There was no evidence of hepatomegaly or splenomegaly. Bowel sounds were present on auscultation.

Nervous System Exam: The pupils were equal and reactive to light and accommodation. Cranial nerves 2 to 12 were intact. The peripheral nervous system exam was normal.

Musculoskeletal Exam: There was tenderness to palpation over the left pectoral region. This was exacerbated by inspiration and on adduction and internal rotation of the left arm.

Impression:

1. Cocaine-induced coronary vasospasm
2. Musculoskeletal pain
3. Pericarditis

PLAN

1. 12 lead ECG
2. IV access and bloods (full blood count, renal profile, liver function tests, coagulation screen, toxicology and cardiac troponins)
3. Analgesia (2.5mg morphine iv)
4. Aspirin (300mg), clopidogrel (300mg) and sublingual GTN (2 puffs)
5. Chest X ray

RESULTS

1. ECG - Normal sinus rhythm, T wave inversion in V1, no other abnormalities detected.
2. Chest X-ray - normal.
3. Bloods - normal troponin level (<0.01), normal D-dimer (171.3), negative toxicology.

CONCLUSION

X had chest pain suggestive of an acute coronary syndrome in the absence of a definite acute myocardial infarction (MI), ischaemic ECG changes or a positive troponin level, thus he was considered to be a suitable candidate for the chest pain assessment unit (CPAU).

X was admitted to the CPAU 4 hours after presentation, where he was monitored by 12 lead ECG for 24 hours and had 3 CK-MB levels taken during this period. Because there were no abnormalities detected in the ECG or CK-MB levels during this period, X was considered suitable for an exercise stress test (EST). The EST revealed an appropriate heart rate and blood

pressure response to exercise and X managed 13.32 minutes of exercise according to the Bruce Protocol before the test was stopped due to chest discomfort. The chest discomfort resolved 2 minutes into recovery and the EST was considered normal.

X was discharged from the CPAU on the following day and given an appointment to attend the nurse specialist led review clinic 48 hours post discharge.

X attended the review clinic as scheduled. He was strongly advised to discontinue cocaine use and to abstain from cigarette smoking.

X was discharged to the care of his GP and advised to return to the emergency department should any similar episodes of chest pain occur.

DISCUSSION

Cocaine is the second most common illicit drug used and the most frequent cause of drug related deaths in the United States¹. The younger age group, 18 - 25 years, are the most common users and it is estimated that 11% of the population have used cocaine at some point¹. Cocaine may be smoked, inhaled or injected. Its use is associated with both acute and chronic complications that may involve any system, the most common being the cardiovascular system (Table 1)². Cocaine use should be considered as a differential diagnosis in any young adult with a cardiovascular event because of its potential to cause serious cardiovascular and cerebrovascular

Table 1: Cocaine related complications

SYSTEM	COMPLICATIONS
Cardiovascular	myocardial ischaemia, coronary artery spasm, acute MI, atherosclerosis, myocarditis, arrhythmia, hypertension, cardiomyopathy and endocarditis ²
Neurological	intracranial haemorrhage, cerebral infarction, seizures and migraine ³
Vascular	aortic dissection, rupture and vasculitis ⁴
Gastrointestinal	Mesenteric ischaemia, infarction and perforation ⁵
Respiratory	pulmonary oedema, infarction and haemoptysis ⁶
Musculoskeletal and dermatological	rhabdomyolysis, skin ischaemia, superficial/deep venous thrombosis and thrombophlebitis ⁷
Genitourinary and obstetric	renal and testicular infarction, abruptio placentae, spontaneous abortion, prematurity and growth retardation ⁸

and complications. Among the cardiac complications, myocardial ischaemia and infarction have been most commonly reported in clinical and autopsy studies². Complications can follow any route of administration. Pre-existent vascular disease is not a prerequisite for the development of cocaine-related cardiovascular complications².

Pharmacology of Cocaine

Cocaine (benzoylecgonine) is an alkaloid extract from the leaf of the *Erythroxylon coca* plant, which usually grows in South America¹. Cocaine is available in two forms:

1. Hydrochloride salt: This can be taken orally, intranasally or intravenously.
2. Free base: Known as crack cocaine. It can be smoked and is considered to be the most potent and addictive form.

Cocaine is absorbed in both forms from all body mucous membranes. The peak effect ranges from 1 to 90 minutes depending on the route of administration. The half-life ranges from 0 minutes after inhalation to 2-3 hours after gastrointestinal ingestion, with duration of action between 15 minutes by IV or inhalation routes to 3 hours by the gastrointestinal route².

Mechanism of Action

Cocaine acts as a powerful sympathomimetic agent. It blocks the re-uptake of dopamine and noradrenaline producing high levels of these neurotransmitters at the postsynaptic receptors. Cocaine blocks sodium channels, which accounts for its local anaesthetic effects. It also produces a dose dependent increase in blood pressure and heart rate. By blocking the reuptake of dopamine, cocaine causes euphoria; and by blocking noradrenaline re-uptake vasoconstriction results. Coronary artery spasm is exacerbated by beta blockade and antagonized by phentolamine, suggesting that it is mediated through the stimulation of alpha adrenergic receptors².

Cocaine-Related Chest Pain and MI

The commonest cocaine related cardiovascular problem is chest pain. MI after cocaine use involves several mechanisms. It is related to the block of the re-uptake of noradrenaline that leads to alpha and beta adrenergic effects. These include increased heart rate and blood pressure, and simultaneous coronary vasospasm, with reduced myocardial oxygen delivery leading to ischaemia. In addition, there is evidence that cocaine activates platelets, increases platelet aggregability, and potentiates thromboxane production thereby promoting thrombus formation. Acute coronary events and MI can occur minutes after cocaine administration or as late as a few days afterwards. However, the highest risk of coronary events is in the first hour after cocaine use with no relation to the dose or route of administration².

Cocaine-induced MI often occurs in patients with normal coronary arteries, and the typical patient is described as a male in his 30s, with only smoking as a coronary risk factor; 50% of these patients would have experienced chest pain previously. The anterior wall is involved in most cases (77%) of cocaine induced MI².

Chest pain and ECG changes are very common in cocaine users, even in the absence of myocardial ischaemia and MI, and only 6% of cocaine-induced chest pain is attributable to MI. The risk of MI is increased up to 24 times over baseline in the first 60 minutes after cocaine use². Young patients presenting with chest pain and suspected acute coronary syndrome should be questioned about cocaine use. Cocaine-induced MI can be difficult to diagnose accurately, as the ECG is difficult to interpret in young patients. Furthermore, MI can occur with normal ECGs or with only non-specific findings. Serum creatine kinase is not a reliable indicator of myocardial injury and is increased in almost half of cocaine users without MI; this is thought to be attributable to rhabdomyolysis. In contrast, cardiac troponins are more sensitive and specific for myocardial injury and should be used for the diagnosis of MI². Fortunately, there is a low incidence of complications after cocaine-induced MI. This is possibly due to the young age of most patients, who often have normal coronary arteries and these complications mostly occur within 12 hours of presentation. Ventricular arrhythmias occur in 4% to 17%, congestive heart failure in 5% to 7%, and death in less than 2%².

Stroke

The risk of stroke is considerably increased with cocaine use³. The aetiology of cocaine-induced brain ischaemia is multi-factorial:

1. Cocaine stimulates vasospasm, presumably by increasing levels of extracellular monoamines, particularly dopamine.
2. Cocaine may cause thrombus formation in the cerebral vasculature.
3. Long - term cocaine use may cause a cerebral vasculitis that impairs cellular oxygenation by exacerbating non-laminar blood flow and sludging in the vessels, with consequent increase in platelet aggregation and thrombus formation.

Over time, repeated ischaemic episodes and subsequent reperfusion can weaken vessel walls, thereby increasing the likelihood of cerebral haemorrhage³.

Management of Cocaine Related Chest Pain

The cornerstone of treatment is sedation using benzodiazepines, which decrease central sympathetic outflow⁹.

In addition:

1. Hyperthermic patients should be cooled
2. Fluid resuscitation must be initiated to maintain urine output

3. Seizures, if present, should be treated with benzodiazepines
4. An urgent CT brain should be ordered in all cases of seizures (to exclude an intracranial haemorrhage)
5. Acute MI should be excluded using cardiac troponins in patients presenting with chest pain
6. Myocardial ischaemia should be treated with aspirin, benzodiazepines or nitrates, heparin, and opiates
7. Beta blockers alone are absolutely contraindicated (they cause unopposed alpha stimulation which worsens coronary and peripheral vasoconstriction)

CONCLUSION

The recognition of cocaine induced ischaemia or MI is crucial for optimal management. A previously healthy young person presenting with cardiac type chest pain should be asked about cocaine, or any illicit drug, use. Many cocaine users have little or no idea of the risks associated with its abuse. Patients, health care professionals, and the public should be educated about the dangers and the considerable risks of cocaine and other illicit drug use.

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