CASE REPORT

Cocaine use and delayed myocardial ischaemia and/or infarction

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SUMMARY

A 37-year-old woman was admitted into the coronary care unit following chest pain after using cocaine. She was found to have significant myocardial ischaemia on blood and ECG investigations despite a recent coronary angiogram that had not demonstrated flow-limiting coronary disease. This case report summarises the risks of myocardial ischaemia and/or infarction for patients taking cocaine and the pathophysiology behind it, focusing in particular on the risks of delayed reaction some time after cocaine ingestion.

BACKGROUND

At present, there are no set criteria to guide doctors in safely discharging patients presenting with cocaine-related chest pain.

Around 2.2% of people aged 15–59 in the UK have used cocaine according to the crime survey for England and Wales 2011/2012.¹ A significant proportion of these will develop cocaine-related chest pain and a subset of these present to emergency departments. Often these people are discharged very rapidly after an initial normal troponin result; however, their risk of completed myocardial infarction is in fact significantly raised; cocaine accounts for up to a quarter of cases of myocardial infarction in patients between 18 and 45 years of age and cocaine users have a sevenfold increased lifetime risk of non-fatal myocardial infarction compared with non-users.² The danger lies in discharging a patient too quickly and missing the myocardial infarction.

CASE PRESENTATION

A 37-year-old woman was admitted to hospital with a sudden onset chest pain lasting for 40 min. The nature of the chest pain was similar to that which she had previously had after cocaine ingestion. She did not have any dyspnoea, syncope or palpitations and glyceryl trinitrate (GTN) spray had not relieved her pain.

The patient had a history of cocaine-related chest pain and had undergone coronary angiography which showed clear but spasm-prone arteries (figure 1); this spasm was so significant that during a previous admission it had led to a ventricular fibrillation cardiac arrest. After some discussion, she had had an internal cardiac defibrillator implanted. On this admission, she admitted to taking cocaine 2 days prior to the onset of her chest pain. She was a regular cocaine user, using it every 2 weeks and frequently experiencing chest pain after use. She was pain free on arrival to the hospital and ECG showed sinus rhythm with T wave inversion across leads V1 to V5 with no dynamic changes (figure 2). Routine bloods revealed a small raise in troponin I levels of 141 µg/L at 4 h and 231 µg/L at 8 h (normal levels <40 µg/L). An implantable cardioverter defibrillator interrogation also revealed recurrent episodes of supraventricular tachycardia most likely related to cocaine use.

TREATMENT

The admitting team decided not to repeat cardiac catheterisation on this patient in view of her

Figure 1 Coronary angiogram: right anterior oblique (RAO) view of left coronary system; left anterior oblique view of right coronary artery; RAO view of left coronary system.

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Figure 2 ECG: T wave inversion across V1 to V5.

previously unremarkable angiograms but started her on the local acute coronary syndrome medical treatment protocol of aspirin, ticagrelor and enoxaparin. She was then transferred to a monitored bed on the cardiology ward.

OUTCOME AND FOLLOW-UP

The patient had another episode of chest pain 36 h after admission with anterior ST elevation on the ECG ([figure 3](#)). There was no evidence of further cocaine use prior to this episode. She was given intravenous morphine, GTN infusion, metoclopramide and diazepam before being transferred urgently to the catheterisation laboratory. However, no procedure was undertaken; her pain and ST elevation settled very rapidly and given the previous severe

coronary spasm provoked during angiography ([video 1](#)), it was considered safer not to perform this procedure unless absolutely essential. She made an uneventful recovery during the rest of her time as an inpatient and was discharged soon after.

DISCUSSION

In the COCaine Associated CHest Pain (COCHPA) study, it was demonstrated that 6% of patients who presented to the emergency department with chest pain after using cocaine had developed cocaine-associated myocardial infarction.³

Cocaine users are at a higher risk of developing myocardial ischaemia and/or infarction because of the effects of cocaine on the cardiovascular and haematological system. The risk of a

Figure 3 ECG: anterior ST elevation.

Video 1 Coronary angiogram: right coronary artery.

myocardial infarction is 24 times in excess of the baseline risk for any individual during the 1 h period after using cocaine⁴ and continues to be raised for several hours.⁵ Patients with chest pain associated with cocaine use, but normal cardiac enzymes and troponin and no ECG changes can safely be discharged after a 12 h period of observation.⁵ Patients with higher risk features, however, as was the case with our patient, will require a longer period of monitoring.

Cocaine affects the cardiovascular system primarily in two opposing ways. First, it stimulates the sympathetic system by blocking the reuptake of dopamine and noradrenaline leading to an increase in heart rate, blood pressure and myocardial contractility, thereby increasing myocardial oxygen demand.⁶ Second, cocaine reduces myocardial blood supply because of its vasoconstriction effects. The increased sympathetic output causes vasoconstriction of the coronary arteries by stimulating the alpha-adrenergic receptors in smooth muscle cells in the coronary arteries⁷ and cocaine increases the levels of endothelin-1, a potent vasoconstrictor,⁸ as well as decreasing the levels of nitric oxide, a vasodilator,⁹ resulting in a net vasoconstriction effect. When the increased myocardial oxygen demand exceeds

the reduced myocardial oxygen supply, it leads to myocardial ischaemia and/or infarction.

Cocaine use is also associated with a prothrombotic state as it stimulates platelet aggregation. Longer term, it accelerates atherosclerosis.

Beyond developing myocardial infarction soon after using cocaine, these patients can go on to develop further cardiac complications. Ninety per cent of the complications occur during the first 12 h¹⁰ after seeking medical help.

Continued use of cocaine despite cocaine-related chest pain puts an individual at a much higher risk of cardiovascular complications including myocardial infarction.¹¹

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

- 1 Home Office. *Drug misuse: findings from the 2012 to 2013 Crime Survey for England and Wales*, CSEW. London: Home Office Statistics, 2013.
- 2 Qureshi AI, Suri MF, Guterman LR, *et al.* Cocaine use and the likelihood of nonfatal myocardial infarction and stroke: data from the Third National Health and Nutrition Examination Survey. *Circulation* 2001;103:502–6.
- 3 Hollander JE, Hoffman RS, Gennis P, *et al.* Prospective multicenter evaluation of cocaine-associated chest pain. Cocaine Associated Chest Pain (COCHPA) Study Group. *Acad Emerg Med* 1994;1:330–9.
- 4 Mittleman MA, Mintzer D, Maclure M, *et al.* Triggering of myocardial infarction by cocaine. *Circulation* 1999;99:2737–41.
- 5 Weber JE, Shofer FS, Larkin GL, *et al.* Validation of a brief observation period for patients with cocaine-associated chest pain. *N Engl J Med* 2003;348:510–17.
- 6 Vongpatanasin W, Mansour Y, Chavoshan B, *et al.* Cocaine stimulates the human cardiovascular system via a central mechanism of action. *Circulation* 1999;100:497–502.
- 7 Moliterno DJ, Willard JE, Lange RA, *et al.* Coronary-artery vasoconstriction induced by cocaine, cigarette smoking, or both. *N Engl J Med* 1994;330:454–9.
- 8 Wilbert-Lampen U, Seliger C, Zilker T, *et al.* Cocaine increases the endothelial release of immunoreactive endothelin and its concentrations in human plasma and urine: reversal by incubation with sigma-receptor antagonists. *Circulation* 1998;98:385–90.
- 9 Mo W, Singh AK, Arruda JA, *et al.* Role of nitric oxide in cocaine-induced acute hypertension. *Am J Hypertens* 1998;11:708–14.
- 10 Hsue PY, McManus D, Selby V, *et al.* Cardiac arrest in patients who smoke crack cocaine. *Am J Cardiol* 2007;99:822–4.
- 11 Hollander JE, Hoffman RS, Burstein J, *et al.* Cocaine-associated myocardial infarction: mortality and complications. Cocaine Associated Myocardial Infarction Study Group. *Arch Intern Med* 1995;155:1081–6.
- 12 Amin M, Gabelman G, Karpel J, *et al.* Acute myocardial infarction and chest pain syndromes after cocaine use. *Am J Cardiol* 1990;66:1434–7.
- 13 Hollander JE, Hoffman RS, Gennis P, *et al.* Cocaine-associated chest pain: one-year follow-up. *Acad Emerg Med* 1995;2:179–84.

Learning points

- ▶ Cocaine users have a higher risk of developing myocardial infarction immediately after using cocaine and a higher risk of cardiovascular complications up to 12 h after presentation.
- ▶ Continued use of cocaine for patients with recurrent cocaine-related chest pain further increases risk of cardiovascular complications.
- ▶ Low-risk patients can be discharged after a 12 h period of observation; however, higher risk patients should be admitted and may develop complications several days later; this may be due to persistence of cocaine metabolites.¹²
- ▶ Advising and supporting patients in cessation of cocaine use is key in secondary prevention.¹³
- ▶ This case demonstrates a patient with cocaine-related myocardial ischaemia several days after using cocaine. Her repeated use led to multiple admissions into coronary care unit for myocardial-related complications, despite underlying normal coronary arteries.

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