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Case Report

CASE REPORT

A 'sniff' away from death: a case of cocaine-induced coronary artery dissection

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Abstract

The multiple, deleterious cardiovascular effects of cocaine abuse are well known. Recent data have shown that cocaine abuse in the UK remains high with an upward trend over the last few years and a significant associated mortality/morbidity. Cocaine-induced coronary artery dissection (CAD) is one of the rare totally preventable manifestations of cocaine abuse. With this case report, we aim to contribute to the limited number of cocaine-induced CAD described in literature and increase public awareness in an attempt to reverse the upward trend of cocaine abuse.

INTRODUCTION

The multiple, deleterious cardiovascular effects of cocaine abuse are well known [1]. Recent data have shown that cocaine abuse in the UK has an upward trend over the last few years. Cocaine-induced coronary artery dissection (CAD) is only a rare and small contributor to the total mortality/morbidity associated with cocaine but it is totally preventable. We report a case of cocaine-induced CAD aiming to increase awareness of the deleterious effects of cocaine in an attempt to educate the public and reverse the increasing trend of cocaine-use.

CASE REPORT

A 38-year-old lady presented with 2 day history of constant, central chest pain radiating down both arms. She was feeling breathless, nauseous and vomited once a few hours later. She was a cocaine-user and had inhaled cocaine on the day that the pain started. She came into the hospital as her pain did not settle despite codeine (included in her previous regular prescriptions). Her past medical history included asthma, anxiety and possibly hypermobility syndrome but there was no formal diagnosis of the latter. She was a smoker and drinking alcohol occasionally.

On arrival to the hospital, she was hemodynamically stable (Blood pressure = 125/75 mmHg and heart rate = 115 bpm). Her electrocardiogram (ECG) showed sinus rhythm with significant ST elevation and Q waves in I, avL, V2-5 (Fig. 1). Troponin T was 4135 ng/l (normal < 14) and 6 h later reduced to 3941 ng/l. In view of the late presentation of her ST Elevation Myocardial Infarction (STEMI) with ECG evidence of completed infarct she was not considered for emergency angiography and percutaneous intervention. She was initially treated with aspirin, clopidogrel, fondaparinux, morphine, diazepam and glyceryl trinitrate infusion. Tirofiban was added later due to chest pain recurrence (BR = 105/73 mmHg, HR = 103 bpm) and urgent coronary angiography was arranged. Her echocardiogram prior to coronary angiography showed mildly dilated left ventricle with severe left ventricular systolic dysfunction and EF < 25%. Most contractility was seen in the basal segments. Coronary angiography showed an extensive spiral CAD from the ostium of the left anterior descending artery to the distal vessel including all diagonal branches (Figs 2–4 and submitted cines). The left main stem, circumflex and right coronary arteries were normal (Fig. 5). Following discussion between

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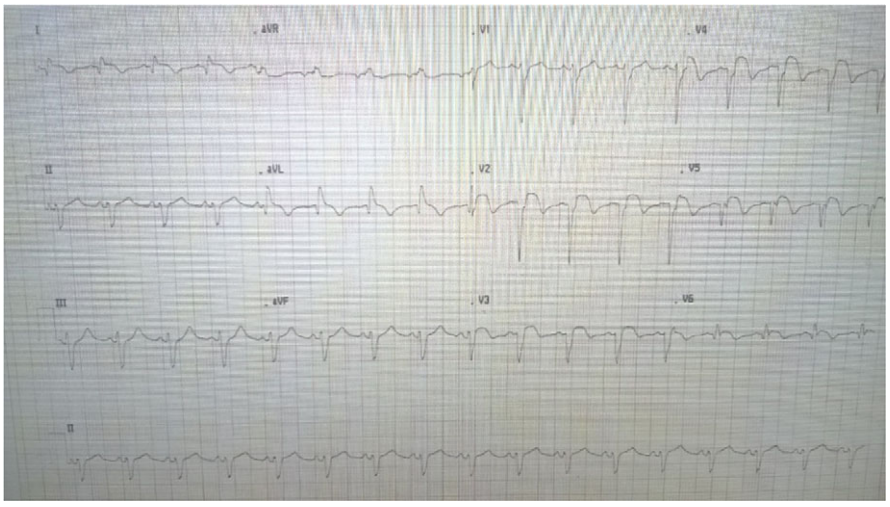

Figure 1: ECG showing anterior ST elevation with deep Q waves.

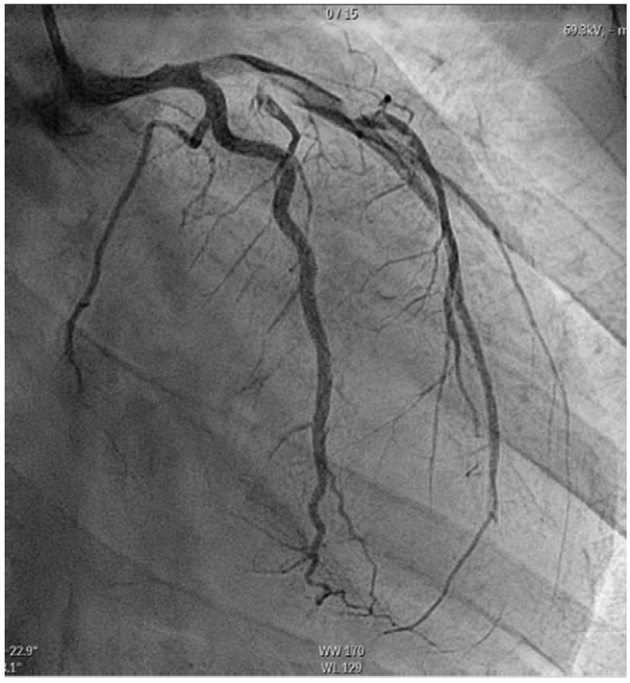

Figure 2: CAD in RAO/Caudal.

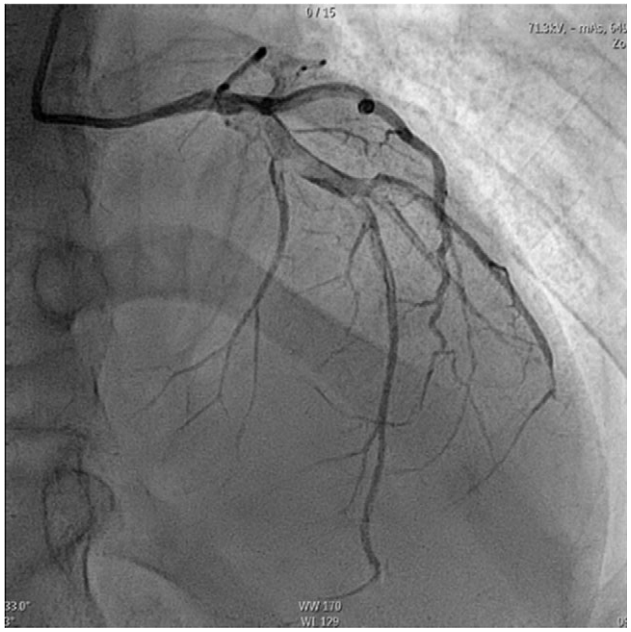

Figure 3: CAD in PA/Cranial.

two Interventional Cardiologists it was felt that there was no percutaneous coronary intervention option and opted for medical management in the first instance. Due to the late presentation of her STEMI and the fact that at the time of coronary angiography the patient was pain-free, it was elected not to pursue urgent coronary artery bypass. Pregnancy was excluded. It was acknowledged that there was a possible but unconfirmed diagnosis of hypermobility syndrome which could potentially increase the risk of CAD. However, there was a temporal relationship of the chest pain with the cocaine abuse which was the most important factor. The patient vomited after the onset of the pain and there was no particular history of emotional upset prior to the onset of the chest pain to suggest possible hypertension spike which might had contributed to the dissection. The patient was started on furosemide, ramipril, eplerenone, ivabradine instead of betablockers due to asthma and she improved over the next few days. Uptitration of her medications proved difficult because of hypotension and she was discharged on Day 9.

DISCUSSION

Cocaine has many deleterious effects to the heart ranging from coronary artery spasm to atherosclerosis. Cocaine-induced CAD is one of the rare but well-recognized causes of acute coronary syndrome especially among young adults. Jaffe *et al.* described the first case of cocaine-induced CAD in 1994, treated with a single coronary artery bypass graft which was occluded 1 year later [2]. In 2001, two further case reports were published. Steinhauer and Caulfield described the first fatal case of cocaine-induced CAD [3]. Gelfand *et al.* described the first cocaine-induced CAD involving the left main stem extending into the left anterior descending and circumflex arteries requiring urgent four vessel

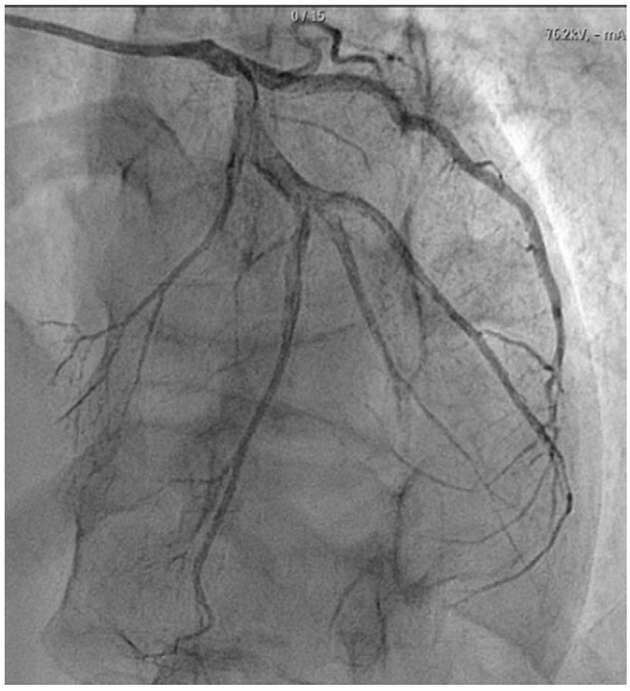

Figure 4: CAD in LAO/Cranial.

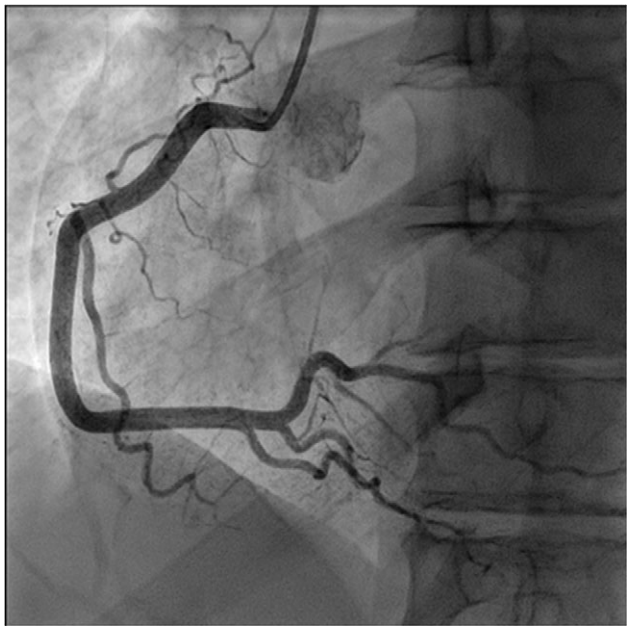

Figure 5: Right coronary artery in LAO.

aorto-coronary bypass [4]. To the best of our knowledge, six more cases have been reported since then. One was diagnosed at post mortem, one was treated with coronary artery bypass, one with percutaneous coronary intervention and three were treated medically [5–10].

It is known since 1986 that even recreational cocaine-use can be temporally associated with cardiac events such as myocardial infarction, ventricular tachycardia/fibrillation or sudden death [11]. The deleterious cardiovascular effects of cocaine can be summarized to (i) sympathetic activation secondary to inhibition of catecholamine re-uptake at the sympathetic nerve terminals, (ii) coronary artery vasospasm due to increased release of the potent vasoconstrictor endothelin-1 and inhibition of nitric oxide production from the endothelial cells and (iii) thrombosis due to platelet aggregation and activation [1]. The exact pathophysiology of cocaine-induced CAD is poorly understood. It is thought to be the result of a complex interaction of the multiple cardiovascular effects of cocaine which cause abrupt sheer wall stress leading to dissection [10].

According to the 2015/16 Crime Survey for England and Wales, cocaine is the second most common (after cannabis) used drug among adults 16–59 year-old. Of note, 2.2% among 16–59 year-old and 4.4% among 16–24 year-old equating to 725 000 and 274 000 people, respectively, used cocaine over the last year. Among 16–24 year-old, cocaine-use peaked in 2008/9 at 6.5% but it followed a downward trend till 2012/13 when it reached the lowest point of 3%. There appears to be an upwards trend since then [12]. According to the Office of National Statistics in 2013, cocaine was involved in 169 of 2995 drug-related deaths registered in England and Wales and in 65 of 526 drug-related deaths registered in Scotland [13]. Judging by the number of cocaine-induced CAD described in literature, this appears to be only a rare and very small contributor to the total mortality and morbidity attributed to cocaine abuse. However, there is definitely a mortality and also significant morbidity associated to cocaine-induced CAD, a condition that tends to affect young adults and it is totally preventable.

Management of patients presenting with acute myocardial infarction in the setting of cocaine abuse should follow the standard guidelines with a few exceptions. It is advised to avoid beta-blockers in the acute phase due to the risk of exacerbating the vasoconstriction from unopposed α-stimulation. Benzodiazepines can also prove to be very helpful, as in our patient, as they relieve the pain and attenuate the hemodynamic effects of the cocaine [1]. Data from patients with spontaneous CADs (SCADs) show that the great majority of the dissections heal spontaneously and patients treated medically have good long-term outcomes if they survive the acute episode [14]. Attempting PCI can be very challenging due to difficulty identifying the true lumen, propagating the dissection during PCI antegradely or retrogradely and requiring extensive stenting with increasing risk of stent restenosis. Furthermore, once the dissection heals and the intramural haematoma gets resorbed the stent might be mal-apposed against the wall increasing the risk of late stent thrombosis [15]. Data have shown that in the setting of SCAD, PCI was technically successful in only ~65%, with long-term durability around 30% [14, 16]. In addition, ~70% of bypass grafts are occluded at follow-up probably as a result of competitive flow after the dissected coronary artery has healed [14, 16]. Given the good long-term outcomes of medical management, provided patients survive the acute event, and the challenges of revascularisation options; medical management is usually the preferred option in the first instance while PCI is reserved for patients with ongoing chest pain/ischaemia and CABG can be considered when the left main stem is involved [15].

With regard to preventable conditions though, the most important part of management is prevention; either before or after the condition has manifested itself. The most recent data indicate that cocaine abuse in the UK is still high and efforts with appropriate campaigns are needed to reverse the upward trend.

SUPPLEMENTARY MATERIAL

Supplementary material is available at *Oxford Medical Case Reports* online.

CONFLICT OF INTEREST STATEMENT

None declared.

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