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CASE REPORT
Coronary heart disease

Sniff of coke breaks the heart: cocaine-induced coronary vasospasm aggravated by therapeutic hypothermia and vasopressors after aborted sudden cardiac death: a case report

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Introduction Coronary vasospasm and sudden cardiac death are a frequently reported complication of cocaine abuse. We present a case with uniquely severe clinical and angiographic presentation.

Case presentation A 39-year-old patient was presented to the cath lab after out-of-hospital cardiac arrest. Coronary angiography revealed focal coronary vasospasm in the proximal LCx, well responsive for intracoronary nitrates. Accordingly, no coronary intervention was performed and the patient was transferred to the cardiac intensive care unit. There, after systematically cooling sudden haemodynamic deterioration and massive ST-elevation was observed. Repeated coronary angiography revealed subocclusive LAD and LCx vasospasm, which again recovered after intracoronary injection of nitric oxide.

Discussion Coronary-spastic effect of cocaine and its potentially dreadful clinical consequences are well-described phenomena. As novelty this case emphasizes that standard of care, including systematic hypothermia and vasopressor administration after out-of-hospital cardiac arrest can potentiate cocaine-induced coronary spasm with dramatic outcomes.

Keywords Sudden cardiac death • Coronary vasospasm • Cocaine • ST-elevation

Learning point

- Induction of mild hypothermia in post-cardiac arrest patients might deteriorate cocaine-induced coronary spasm, especially when potentiated by administration of vasopressors.

Introduction

Every year, 5 million people worldwide die from sudden cardiac death (SCD). Heart failure and coronary artery disease are the most common aetiologies for SCD, but studies have shown that up to 3% of SCDs are attributed to cocaine.[1,2] With an estimated 12 million consumers in Europe, cocaine is the illicit drug leading to most emergency room presentations. Mechanisms favouring SCD include arrhythmogenicity of the drug *per se* as well as cardiovascular and sympathomimetic effects resulting in increased heart rate, blood pressure, and myocardial oxygen demand.[3–5] Myocardial ischaemia can be due to vasoconstriction in patients with normal coronary arteries.[6] Cocaine-induced myocardial infarction may be the results of coronary artery vasoconstriction, thrombosis, and atherosclerosis.[7]

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Here, we present a case of out-of-hospital cardiac arrest due to massive coronary spasm after cocaine consumption.

Timeline

90 minutes prior to presentation	cardiac arrest, standby CPR, continues CPR by emergency doctor, initial rhythm ventricular fibrillation, defibrillation, ST-elevation in leads V2 and V3
Upon presentation to the cath lab	critical stenosis of the proximal LCx, focal vasospasm, intracoronary administration of nitrate
After transfer to the ICU	induction of mild hypothermia, moderate vasopressor support
Two hours after transfer to ICU	polymorphic ventricular tachycardia, ST-elevation
Second presentation to the cath lab	subtotal occlusion of the entire proximal-middle LAD and LCx, intracoronary administration of nitrate, continuous intravenous administration of nitrate
Third day after presentation	extubation, no neurological deficit
Two months after presentation	discharged from stationary rehabilitation without neurological deficit

Case presentation

A 39-year-old man without history of medical disease and risk factors suffered out-of-hospital cardiac arrest. He was successfully resuscitated first by members of the public and then by paramedics with initial rhythm of ventricular fibrillation. After return of spontaneous circulation (total of 20 min of resuscitation, one successful shock, and GCS 3 after return of spontaneous circulation), the surface electrocardiogram (ECG) showed slight ST-elevation (1 mm) in precordial leads V2 and V3, therefore the patient was transferred sedated and ventilated to the cathlab for primary percutaneous coronary intervention (PCI).

Angiography revealed critical stenosis of ∼9 mm in the proximal left circumflex coronary artery (LCx) (left dominant), however without any typical angiographic sign of a culprit lesion. Intracoronary nitrate was administered, leading to complete disappearance of the stenosis and proving that severe focal spasm was the pathomechanism. Accordingly, no coronary intervention was performed (Supplementary material online, *Videos S1* and *S2*; *Figure 1A ,B*).

In the intensive care unit therapeutic systematic hypothermia was introduced, requiring moderate vasopressor support (norepinephrine 0.025 μg/kg/min), as well. The patient received adequate sedation (remifentanil 0.08 μg/kg/min and midazolam 1.6 μg/kg/min). After 2 h at core temperature of 35°C, the patient developed one episode of non-sustained polymorphic ventricular tachycardia and became haemodynamically unstable, requiring increasing dosage of vasopressor support. ECG showed pronounced ST-elevation in leads I, II, III, aVF, V6, and massive ST-depression in leads V1–V4 (*Figure 2*). Urgent coronary angiography was performed, revealing subtotal occlusion of the entire proximal middle left anterior descending coronary artery (LAD) and LCx with Thrombolysis in Myocardial Infarction (TIMI) 1 flow. (*Figure 1 C,D*, Supplementary material online, *Video S3*). After intracoronary administration of nitrate the vasospasm fully disappeared, followed by reperfusion arrhythmia (ventricular fibrillation) (Supplementary material online, *Video S4*). After successful defibrillation the patient haemodynamically stabilized, ST-elevation resolved and semi-selective angiography proved the resolution of spasm, as well. No indication was for any coronary intervention. Patient was transferred back to the intensive care unit.

Back at the intensive care unit urine toxicology test was performed, being positive for cocaine and tetrahydrocannabinol (THC). The patient's wife confirmed recreational cocaine use (at most once a month) by her husband.

The patient was stabilized by progressively reduced dosage vasopressor and continuous intravenous administration of nitrate (0.4 μg/kg/min). Hypothermia was kept at low-normal level (36°C). Patient was successfully extubated on Day 3. Repeated echocardiograms revealed mild left ventricular concentric hypertrophy, normal left ventricular ejection fraction without wall motion abnormalities. The patient was discharged into stationary rehabilitation with antihypertensive medication (Carvedilol 25 mg b.i.d. and Lisinopril 5 mg b.i.d.), aspirin 100 mg as well as vasodilating agents (Amlodipine 5 mg b.i.d. and Nicorandil 10 mg b.i.d.). Two months after presentation, the patient was discharged from stationary rehabilitation without any neurological deficit.

Discussion

Cocaine may induce SCD via multiple mechanisms including direct arrhythmogenic effect, arrhythmias occurring due to the sympathomimetic effect, coronary vasospasm, thrombosis, or aggravation of pre-existing coronary artery disease.

Cocaine leads to vasoconstriction via stimulation of the alpha-adrenergic receptor of the coronary artery's smooth muscle cells.[8] Vasoconstriction may be further aggravated by increase in endothelin and decrease in nitric oxide production.[9,10] This effect also occurs in recreational doses of cocaine.[7] A study investigating systemic and coronary haemodynamic effects of intravenously administered cocaine in cocaine users presenting to the emergency department revealed increased heart rates, cardiac output, arterial pressures but no changes in coronary artery diameters, coronary vascular resistance, or coronary sinus flow.[11] The authors concluded that recreational doses of cocaine do not cause focal or generalized coronary vasospasm and attributed chest pain in cocaine users to myocardial ischaemia caused by spasm of the small coronary vessels and thrombi. Contrarily to this finding, our patient presented with subtotal vasospasm of the large coronary arteries.

Arrhythmias may occur as a direct effect of cocaine on the myocardium or secondarily to myocardial ischaemia.[3] Cocaine acts as an anaesthetic on the myocardium and inhibits membrane permeability to sodium channels and consequently reduces or blocks the initiation

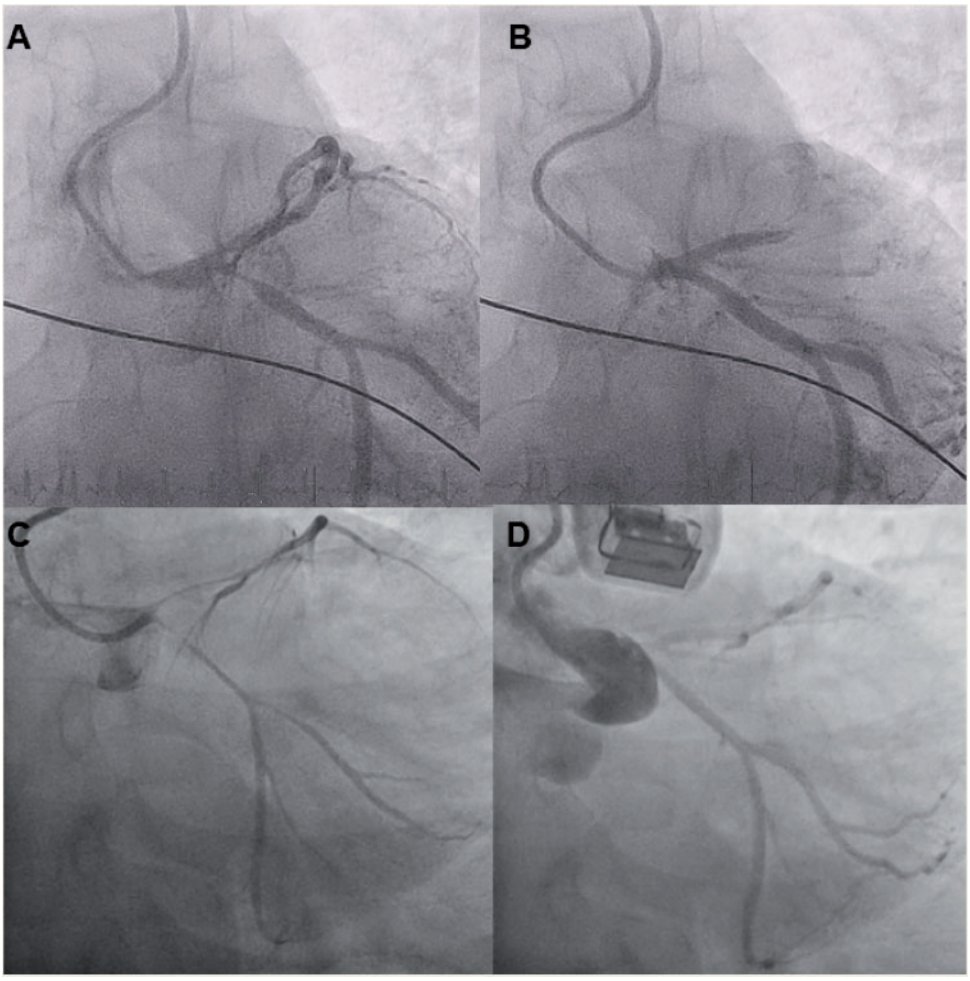

Figure 1 (*A*) Left coronary angiogram showing proximal LCx coronary spasm. (*B*) Resolved spasm after intracoronary administration of nitrate. (*C*) Massive coronary spasms in the second angiography after induction of therapeutic hypothermia. (*D*) Resolved spasm after intracoronary administration of nitrate.

and propagation of electrical signals. The ECG pattern may be similar to that observed in Brugada.[12,13] We suspect that sudden cardiac death may have occurred as a direct effect of cocaine, while ST-elevation occurred at a later point, when the vasoactive effect of cocaine reached a maximum.

Therapeutic options in patients with cocaine-associated myocardial complications include administration of nitroglycerine, verapamil, and phentolamine.[3] There have been reports on the use of benzodiazepines, alpha-adrenoceptor blocking drugs, alpha-2-adrenoceptor agonists, beta blockers, antipsychotics, and morphine, but high-quality evidence for the treatment of cocaine-induced cardiovascular toxicity is limited.[14] In some reports, coronary vasospasm could be reverted by intracoronary injection of nitroglycerine.[15,16] In our patient, massive coronary vasospasm occurred 1 h after nitroglycerine administration, which underlines the importance of continuous administration of vasodilators in this setting.

Data in the literature suggest that hypothermia by itself might not have relevant vasospastic effect.[17,18] However, mild hypothermia may aggravate effects of cocaine.[19] Accordingly, this case underlines that after coronary-spastic acute coronary syndrome caution should be taken with any therapies, such as systematic hypothermia, which might have some vasospastic effect, even if otherwise considered as standard of care after out-of-hospital cardiac arrests. Enhanced by remnant effect of cocaine, hypothermia (or vice versa: cocaine, enhanced by hypothermia) might result in massive and potentially life-threatening coronary spasm, especially when potentiated by administration of vasopressors.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

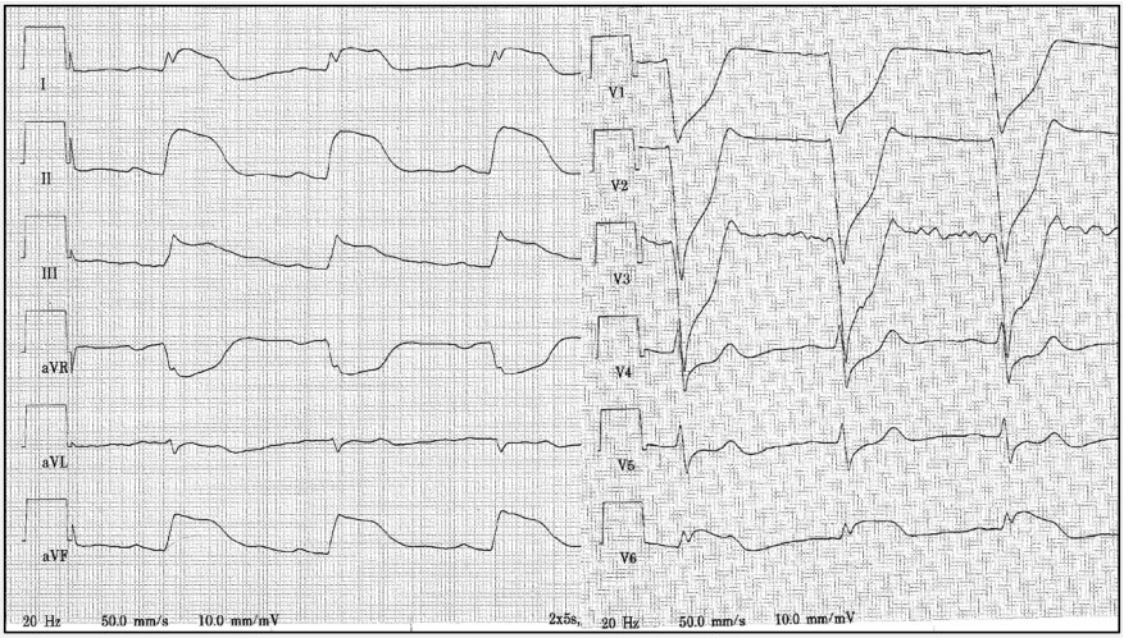

Figure 2 ST-elevation after induction of therapeutic hypothermia.

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