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Case report

Cocaine-induced myocardial injury

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Summary

We report a case of 37-year-old male, who was admitted to the Vilnius University hospital Santaros Klinikos emergency department suffering from acute chest pain. ECG and elevated troponin level confirmed ST-elevation myocardial infarction, although coronary angiogram had not shown hemodynamically significant changes in coronary arteries. Patient admitted after using cocaine a day ago. To conclude, myocardial infarction is rare, but serious consequence of cocaine use. Clinicians should consider interrogating all young patients presented to the emergency department with acute chest pain for cocaine use.

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Keywords: acute myocardial infarction, cocaine, coronary artery disease

Background

In Europe cocaine is the second most widely used illicit substance after cannabis and first most common drug involved in emergency presentations in hospitals. In 2018 more than 18 million European people (5.4% of 15-to 64-year-olds) are reported to have used cocaine at least once in their life, and 3.9 million are estimated to have done so in the last year (1.2% on average) [1]. Among European countries Spain reported the highest number of cocaine-related emergency cases (2 386 in 2011), followed by the United Kingdom (2 247 hospital discharges in 2010–2011) [2]. In 2016 an estimated 48.5 million persons in the U.S. or 18.0% of persons aged 12 years and older, reported use of illicit drugs or misuse of prescription drugs, of which prevalence of cocaine use was 1.9%. In 2016, 2.1 million Americans had recent cocaine use and cocaine was involved in 31% of all visits to the emergency department related to drug misuse or abuse, in 2011 cocaine related emergencies increased to 40.3% [3, 4]. In Lithuania during the five years from 2006–2010 a total of 8.9% (127 of 1 423) intoxications with drugs and psychotropic substances involved cocaine. In 2010 there were four times more males than females registered as hospital inpatients due to poisoning with drugs and psychotropic substances: 204 males (191 in 2009) and 51 females (47 in 2009). Fourteen (5.5%) of these 255 cases were attributed to cocaine (11 males and 3 females). In 2011 heavy intoxications with cocaine increased to 9% (19 of 211) of cases [2, 5].

Case presentation

A 37-year-old male was brought to our emergency department with sudden onset of retrosternal chest pain radiating to the entire chest and lasting for 30 minutes. Paramedics had given him aspirin (500 mg) and diazepam tablets. Upon arrival to hospital patient blood pressure was 116/61 mmHg, heart rate 45 beats/minute and his lung sounds were normal. He had a body mass index 24.7 kg/m^2 and smoked 6–7 cigarettes per day. He was actively exercising all his life and

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had bradycardia and hypotension since young age. His past medical history consisted of migraine diagnosed in 2013 and in the same years he was examined by cardiologist for first degree atrioventricular (AV) block and intermitting second degree AV block. There was no family history of cardiac disease, he was neither diabetic nor hypertensive, but had an increased serum cholesterol level of 6.08 mmol/l and low-density lipoprotein 4.22 mmol/l for which he did not use any treatment. ECG demonstrated ST-segment elevation in inferior and lateral leads (ST elevation above 0.5–1 mm in II, III, aVF and V3-6, V7-9) and patient was admitted directly for cardiac catheterisation. The serum troponin-I level was recorded at 1392.6 ng/L (normal range <14 ng/L for high-sensitivity troponin-I), confirming the diagnosis of myocardial infarction. Coronary artery angiography was performed within 1 h of admission, but no stenosis was found, which could have represented mild coronary artery disease or persistent vasospasm. As there was no evidence of atherosclerotic disease, percutaneous coronary intervention was not performed and patient was transferred to cardiology intensive care unit for comprehensive examination. On further questioning patient confessed, that he had taken cocaine by inhalation a day ago. Blood tests, including haematology, biochemistry and gases were all within normal ranges. Radiological imaging revealed normal chest X-ray. Troponin-I were repeated in the cardiology reanimation and intensive care unit and serum troponin-I concentration increased to 3107.2 ng/L. Transthoracic echocardiography (TE) showed no abnormalities. The patient was treated for acute coronary syndrome with antiplatelets and statins. He was discharged after 4 days. Magnetic resonance imaging (MRI), which was performed after a month, showed normal left ventricular function with ejection fraction of 56%.

Discussion

Cocaine use is definitely the major cardiovascular risk factor. Cocaine users presenting with ST-segment elevation myocardial infarction STEMI have a higher cardiovascular mortality compared to non-cocaine users despite a lower prevalence of the traditional risk factors for coronary artery disease [6]. Regular cocaine use is associated with an increased likelihood of myocardial infarction (MI) in younger patients and moreover regular cocaine use in otherwise healthy subjects is associated with increased systolic blood pressure, aortic vascular stiffness and significantly greater left ventricular mass [7,8].

The cardiac effects of cocaine are complex. Cocaine blocks dopamine and noradrenaline reuptake in peripheral sympathetic nerve terminals, thereby increasing their concentration in the synaptic cleft. This stimulates the sympathetic nervous system and leads to an increase in heart rate, blood pressure and myocardial contractility, which increases myocardial oxygen demand [9,10]. By stimulating the alpha-adrenergic receptors in smooth muscle cells in the coronary arteries and increasing endothelin-1 and decreasing nitric oxide concentrations, cocaine causes vasoconstriction of the coronary arteries. In cases of acute damage, when stress leads to rupture in vascular wall, endothelial damage promotes the increase of fibrinogen and von Willebrand factor leading to platelet aggregation and ultimately the formation of blood clots. Other known mechanisms of cardiotoxicity, include cocaine's blockage of sodium channels and a subsequent increase in calcium flux which critically affects myocardial electrical impulses. By increasing the sympathomimetic stimulation, cocaine produces a progressive reduction in current amplitude for successive pulses. Cocaine creates an elevated inflammatory state with decreased basal anti-inflammatory markers (interleukin-10) and increased pro-inflammatory cytokines (tumor necrosis factor alpha, interleukin 1β) all contributing to endothelial damage and atherosclerosis, which is potentially lethal vascular effect of chronic cocaine use. Although cocaine affects cardiovascular system in a lot of different ways, the main pathophysiological mechanism of cocaine-induced myocardial damage remains increased oxygen demand and decreased supply [11–13].

European Society of Cardiology in the fourth definition of MI, describes myocardial injury due to a mismatch between oxygen supply and demand classified as type 2 MI. Criteria for detection of type 2 MI consist of an elevation of cardiac troponin values with evidence of an imbalance between myocardial oxygen supply and demand unrelated to coronary thrombosis and at least one of the other symptoms such as: clinical symptoms, ischaemic ECG changes, Q waves or imaging based evidence of viable myocardium loss. For MI patients with no angiographic obstructive coronary artery disease (<50% diameter stenosis in a major epicardial vessel) the term MI with non-obstructive coronary arteries (MINOCA) has been applied [14].

The diagnosis of cocaine-induced myocardial ischemia in patients with recent cocaine use by patient history alone is difficult, because of no specific diagnostic algorithms or symptoms and patient unwillingness to admit the fact of co-

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caine use. Diagnosis of cocaine-induced acute MI is made by obtaining a careful history and physical examination of the patient along with appropriate laboratory tests, electrocardiography, cardiac enzymes, echocardiography and coronary angiography. Blood and urine for drug screening may be obtained if there is clinical suspicion of substance abuse as either a cause or a contributor to an acute coronary syndrome, especially in younger patients (aged <50 years) with chest pain of unexplained aetiology [15,16]. Although ECG is one of the criteria for diagnosing type 2 MI, its poor sensitivity and positive predictive value make it unreliable in distinguishing benign cocaine–associated chest pain from actual ischemia. Cocaine Associated Chest Pain Study Group looked at the ECGs of 242 patients with cocaine-associated chest pain and found the sensitivity of ECG for diagnosing MI to be 35.7%, with a specificity of 89.9% and a positive predictive value of 17.9% [17]. Provocative testing for coronary artery spasm should be performed for patients with MINOCA due to vasospasm, except not in acute phase. After initial evaluation of MINOCA patients a routine examination with MRI is recommended.

According to Mayo Clinic recommendations, after initial testing for patient's clinical status, the primary management of cocaine-induced chest pain consists of benzodiazepines and nitrates because many studies had shown this combination therapy more effective than nitroglycerin alone in relieving chest pain. Benzodiazepines because of their anxiolytic properties indirectly combat agitation, hypertension, and tachycardia resulting from the stimulatory effects of cocaine [18].

According to 2017 European Society of Cardiology Working Group position paper (ESC-WGPP) on MINOCA, routine treatment with aspirin, statins and, in cases of vasospasm, calcium channel blockers would be beneficial, because of underlying mechanisms of coronary plaque disruption, coronary spasm, and thromboembolism [19]. American Heart Association recommends MINOCA treatment with statins and angiotensin-converting enzyme inhibitors/angiotensin receptor blockers, and states positive effect of β-blocker treatment, and a neutral effect of dual antiplatelet therapy [20]. American Heart Association and the American College of Cardiology guidelines for the management of cocaine-induced MI released in 2008 predicated that MI associated with cocaine use should be treated similarly to those with traditional acute coronary syndrome. Drug treatment for cocaine associated coronary ischemia includes initial treatment with aspirin, nitrates, and benzodiazepines. Calcium channel blockers may be added for patients, who do not respond to benzodiazepines and nitroglycerin. Because of cocaine-induced vascular endothelial injury and increasing platelet aggregation, aspirin should be routinely administered and unfractionated heparin or low-molecular-weight heparin should be given to patients with cocaine-associated MI. The use of beta-blockers remains unclear [12–16]. Although American Heart Association guidelines states that, beta-blockers should not be administered to patients with STEMI because of coronary vasoconstriction, resulting from their interaction with cocaine, more and more studies shows that beta-blockers are associated with reduction in incidence of MI after cocaine use. The benefit of beta-blockers on myocardial function may offset the risk of coronary artery spasm [21,22]. In our case due to patient's hypotension and bradycardia, neither beta-blockers nor the calcium channel blockers were administered.

Conclusion

Cocaine has a lot of negative effects towards the heart. It not only fastens the development of coronary artery atherosclerosis, but can cause more severe and acute cardiovascular complications such as MI. Although the management of cocaine-abusing patients who present to an emergency room with acute chest pain remains difficult because of conflicting results regarding the incidence of MI, we hope that this case will contribute to raising public awareness about cocaine use and its impact to our health.

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