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Cardiac Effects of Cocaine Use

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Abstract

Cocaine use can precipitate as well as worsen underlying cardiovascular disease. Its impact ranges from a persistent elevation of blood pressure to catastrophic aortic dissection or ventricular tachyarrhythmias. Cocaine's physiologic effects are primarily driven by its sympathomimetic characteristics through its action on alpha - and beta-adrenergic receptors causing elevations in inotropy, heart rate and systemic blood pressure.

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Introduction

The illicit use of cocaine is a world-wide problem. In the United States, cocaine use has been considered a public health challenge for many years, with millions of Americans falling victim to the drug. Cocaine use has recently increased in the United States [1]. This has become a burden on our healthcare system due to increasing costs and associated morbidity and mortality. Cocaine overdose accounted for 14,666 deaths in the United States in 2018 [2]. Additionally, there were morbidity and societal costs on our communities and the healthcare system.

Cocaine adversely affects several different systems of the human body, the most common being the cardiovascular system. It is the most common illegal substance to precipitate an emergency department visit, and in most of these cases the presenting symptom is chest pain, frequently referred to with the acronym CCP or cocaine-associated chest pain. It is estimated that there are over 500,000 visits to the emergency department annually in the United States due to cocaine use [3].

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Case Presentation

A 61-year-old man presented to the Emergency Department with chest pain. He stated that the onset of his chest discomfort was about 3 hours ago after he had smoked about $60 worth of crack cocaine. The pain was substernal, about 2/10 in intensity and did not radiate. It came and went spontaneously without any relationship with exertion, lasting a couple of minutes each time. He had mild diaphoresis but denied dyspnea or dizziness. The patient had a history of hypertension and hyperlipidemia. He smoked a pack of cigarettes daily for 40 years. He generally consumed a 6-pack of beer daily. He was using cocaine for the last 22 years. In the ED the patient had a blood pressure of 190/80 mm Hg, heart rate of 50 beats per minute, respiratory rate of 17 per minute and he was afebrile. His physical examination was otherwise unremarkable. His laboratory test demonstrated a mildly elevated serum glucose level of 125 mg/dL. The serum troponin level was 0.205 ng/ml and peaked at 1.216 ng/ml after 4 hours. The drug screen was positive for cocaine metabolites. His 12-lead electrocardiogram showed sinus bradycardia at 52 beats per minute, first degree AV block and mild ST depression suggestive of ischemia. Later ECGs showed partial normalization of the ST segment. The chest x-ray was normal. The patient was admitted for follow-up. Since the patient had presented with chest pain and he had an abnormal ECG with an elevated troponin level, he was referred for cardiac catheterization. Coronary angiography demonstrated a 90% stenosis of the mid segment of the right coronary artery. The left anterior descending and circumflex coronary arteries were normal. The patient underwent stenting of the high-grade stenosis with the placement of a 2.5/38 mm drug eluting stent with an excellent outcome and no residual stenosis. He was discharged the next day. His discharge medications were aspirin, clopidogrel, atorvastatin and lisinopril. He was offered substance abuse treatment but declined. He was provided with information on substance abuse treatment resources and community support programs prior to discharge.

Discussion

Cocaine is easily absorbed through any mucus membrane and a high blood concentration can be achieved by numerous routes of administration [4]. Common urine drug tests utilized by emergency departments will test for cocaine metabolites that are detectable for 24-36 hours [5]. A positive test in a patient means that consideration for cocaine induced cardiovascular complications need to be entertained despite what a patient may say during an interview. The pathological effects of cocaine are primarily driven by its powerful sympathomimetic characteristics through its action on alpha- and beta-adrenergic receptors causing increased inotropy, heart rate and systemic blood pressure. All of this results in elevated myocardial oxygen demand [6,7,8]. There is coronary artery constriction along with increased thrombus formation via platelet activation and potentiation of thromboxane. This significantly enhances the risk of injury to the myocardium. The risk of myocardial infarction increases by a factor of 24 during the initial 60 minutes after use [9]. There is also concern for delayed vasoconstriction when active metabolites build up as the drug is metabolized [10]. Nearly 6 percent of patients with cocaine induced chest discomfort will manifest enzymatic evidence of myocardial infarction, but surprisingly 35% of these cases with subsequent angiography will demonstrate absence of significant atherosclerotic coronary artery disease [11,12,13]. Further, cocaine use can cause ST elevation on the electrocardiogram in 43 percent of cases without enzymatic changes [14], again posing a management challenge.

Other cardiovascular complications of cocaine use include aortic dissection, coronary artery aneurysms, myocarditis, cardiomyopathy, arrhythmias, and stroke. Aortic dissection is a direct result of sudden elevation of the systemic arterial pressure and was responsible for 1.8% of all aortic dissections from 1996 to 2012 [15]. Angiography reveals coronary artery aneurysms in approximately 30% of cocaine users as compared to 1.5 % of non-users [16,17,18]. The mechanism for aneurysm formation is likely episodic hypertension with vasoconstriction causing direct endothelial damage. Cardiomyopathy results from a combination of cocaine induced ischemia or infarction, repetitive sympathetic “Storms”, contaminants in the cocaine causing myocarditis and cytokine alteration inducing transcription of genes that alter composition of myocardial collagen and myosin [19,20,21,22,23]. Dysrhythmias from cocaine often result from a combination of cocaine’s sodium-channel-blocking properties and its sympathomimetic effects. These dysrhythmias are generally not life threatening in a patient without abnormal myocardial substrate but can become lethal when previous ischemia, infarct or hypertrophy has occurred [24]. The risk of stroke is about 7 times higher in cocaine users and is the result of prothrombotic effects as well as vasoconstriction occurring through the mechanisms mentioned above [25,26].

The findings in our patient suggest that he had asymptomatic pre-existent coronary artery disease, but the hypertensive crisis induced by his cocaine use resulted in a non-ST elevation myocardial infarction with troponin release. Fortunately, he received timely management for his condition.

Conclusion

Cocaine use poses a worldwide public health problem with a major cost burden for society. The detrimental effects on the cardiovascular system has been well established and remains a major factor related to its morbidity and mortality. For the clinician, the cardiovascular effects of cocaine use necessitates rapid recognition and prompt treatment as use can result in pathological conditions like coronary artery dissection and aortic dissection with a notable high rate of morbidity and mortality.

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