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Short communication

Cocaine Cardiomyopathy, Physicians should be aware and focused on Management.

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Introduction

More than 14 million people worldwide, mostly within the age range of 15 to 64 years, consume cocaine. Men of 15–35 years represent the majority. Cocaine is a potent sympathomimetic agent associated with the development of possible fatal cardiovascular complications. Hypertension, Dilated cardiomyopathy, Dysrhythmias and Acute myocardial infarction are just some of many cardiovascular effects related to the abuse of cocaine. The management is like other forms of cardiomyopathy; however β-blockers should be avoided. Non-invasive testing should be performed after several months to re-evaluate the treatment response. Dose-dependent tachycardia, hypertension along with increased arousal is the first physiological response to cocaine use. Performance improvement, attentiveness, sense of positive self-image and euphoria often accompany the cumulative use of cocaine. End-organ-damage associated with cocaine can affect almost every organ system. Cocaine results in a gradual addiction due to its vigorous sympathomimetic features with possible destructive cardiovascular effects. Dysrhythmias, acute myocardial infarction, myocarditis, hypertension, endocarditis, hypotensive shock, cerebral vascular accidents and dilated cardiomyopathy are several cardiovascular complications due to cocaine abuse [1]. Cocaine blocks the presynaptic dopamine and catecholamine uptake, resulting in post-synaptic sympathetic stimulation and dopaminergic receptor activation [2]. Peripheral vasoconstriction results in hypertension, tachycardia and an increase in afterload. Arrhythmias are likely to occur due to the altered autonomic action and cardiovascular resistance results in a decreased myocardial blood supply [3]. Negative inotropic events can also occur from cocaine abuse. Hypertrophy of the left ventricle and cardiomyopathy with significant reduction of the ejection fraction has been described in the setting of chronic cocaine consumption. Myocardial hypertrophy is likely to occur secondary to the temporary blood pressure elevation after cocaine use [4]. Smoking and alcohol use exacerbate the cardiotoxic impact of cocaine [5].

Discussion

Cocaine stimulates the sympathetic nervous system by inhibiting catecholamine reuptake at sympathetic nerve terminals, stimulating central sympathetic outflow, and increasing the sensitivity of adrenergic nerve endings to norepinephrine [7-8]. Cocaine also acts like a class I antiarrhythmic agent (local anesthetic) by blocking sodium and potassium channels, which depresses cardiovascular parameters. [9] Of these 2 primary, opposing actions, enhanced sympathetic activity predominates at low cocaine doses, whereas the local anesthetic actions are more Prominent at higher doses. [8] In addition, cocaine stimulates the release of endothelin-1, a potent vasoconstrictor, from endothelial cells [10] and inhibits nitric oxide production, the principal vasodilator produced by endothelial cells. [11] Cocaine promotes thrombosis by activating platelets, [12-13] increasing platelet aggregation, [12,14] increasing platelet α-granule release, [12,15] increasing plasminogen activator inhibitor activity.[16]

Cocaine increases myocardial oxygen demand by increasing both heart rate and blood pressure. [17,18] The influence of cocaine on heart rate and blood pressure is dose dependent and is mediated through α-adrenergic stimulation. [17,18] At the same time, cocaine decreases oxygen supply via coronary vasoconstriction. Cocaine-induced coronary vasoconstriction occurs in normal (nondiseased) coronary artery segments but is more pronounced in atherosclerotic segments. [19] Combining cocaine use with cigarette smoking has additive effects on coronary vasoconstriction while markedly increasing the rate-pressure product. Long-term cocaine users demonstrate coronary endothelial dysfunction. [20,21]. Because endothelial dysfunction increases the sensitivity of a vessel to the constrictor effects of catecholamines, [22] it may be particularly detrimental for cocaine users. Even in the absence of epicardial coronary disease, cocaine causes microvascular disease [23,24] and is associated with thrombosis. [25,26]

Cocaine causes systolic and diastolic dysfunction, arrhythmias, and atherosclerosis. Cocaine decreases myocardial contractility and ejection fraction by blocking sodium and potassium channels within the myocardium. [27] Intracoronary infusion of cocaine decreases left ventricular ejection fraction and increases left ventricular end-diastolic pressure and end-systolic volume.[28] Long-term cocaine use is associated with left ventricular hypertrophy and prolonged deceleration time. [29,30] Cocaine prolongs the PR, QRS, and QT intervals. [31,32] Cocaine is associated with coronary atherosclerosis even in young users with relatively few cardiac risk factors. [33,34] Cocaine causes systolic dysfunction in long-term users and with acute intoxication. In a dog model, acute cocaine intoxication caused left ventricular dilation, decreased contractility, and increased end-diastolic pressure. [35] Rabbits demonstrated regional wall motion abnormalities (mostly anteroseptal) associated with decreased left ventricular fractional shortening and increased systolic dimension in response to acute cocaine intoxication. [36] After 2 weeks of abstinence from cocaine, 6 of 84 (7%) asymptomatic cocaine users (mean age, 36 years) had an ejection fraction <55%.[37] In 33 cocaine-using patients undergoing coronary angiography (indication: chest pain, 28; congestive heart failure, 4), ejection fraction was abnormal in 18 patients (55%) and ≤30% in 6 patients (18%).[33] Moreover, 4 patients had an ejection fraction <30% with global hypokinesis. Dilated cardiomyopathy is more common among cocaine users, [34] but a case of left ventricular apical ballooning (Takotsubo cardiomyopathy) has also been described. [38] In a registry including 83 hospitals nationally, stimulant drug use (96% cocaine, 5% methamphetamine) was self-reported in 594 of 11 258 patients (5.3%) who presented to the emergency department with acute decompensated heart failure. [39] Patients with stimulant drug use were more likely to have ≥3 hospitalizations within 6 months (28% versus 11%) and had lower ejection fractions (median, 23%

versus40%).

Acute cocaine intoxication decreases myocardial contractility and ejection fraction [27,28] and increases left ventricular end-diastolic pressure and end-systolic volume. [28] Long-term cocaine use is associated with left ventricular hypertrophy and prolonged deceleration time. [30] The pathophysiology of cocaine-associated cardiomyopathy, however, remains unclear. Of 18 cocaine users undergoing coronary angiography with an ejection fraction <55%, 12 had coronary artery disease and regional wall motion abnormalities suggesting recent or remote MI; however, 6 did not have coronary artery disease and demonstrated global hypokinesis (4 of 6 with an ejection fraction <30%).[33] Thus, a manifestation of coronary artery disease can explain cocaine-induced ventricular dysfunction in some patients, but cocaine also has a direct toxic effect on cardiac myocytes. Factors contributing to cocaine-induced cardiomyopathy may include the blocking of sodium and potassium channels within the myocardium, alterations in calcium ion handling, [28] myocardial inflammation with necrosis and fibrosis, left ventricular hypertrophy, [29,30] alterations in gene expression, [39] and concomitant alcohol consumption. [33]

Cessation of cocaine is the primary therapeutic goal in cocaine-induced cardiomyopathy. Cocaine-induced heart failure improved dramatically with cessation of cocaine and recurred with resumption of cocaine. [34] As with CACP, medical therapy for cocaine-induced heart failure and cardiomyopathy should follow published guidelines, except all β-blockers should be avoided in the acute setting. Thereafter, β-blockers should be considered for each patient individually, after careful risk-benefit assessment, and maybe after cocaine cessation has been documented. Continued cocaine use precludes eligibility for cardiac transplantation.

Summary

Cocaine is a potent sympathomimetic agent associated with the development of possible fatal cardiovascular complications. Hypertension, Dilated cardiomyopathy, Dysrhythmias and Acute myocardial infarction are just some of many cardiovascular effects related to the abuse of cocaine. The therapy of cocaine-induced cardiomyopathy is similar to the way that other types of cardiomyopathy are managed. Beta-blockers should not be considered initially; benzodiazepine is preferred to counteract the adrenergic effect. In the acute setting the addition of beta-blockers will adversely result in the alpha-adrenergic receptors being unopposed, therefore leading to coronary vasoconstriction, left ventricle wall stress and a hypertensive crisis. As recommended in other types of cardiomyopathy, pharmacological agents such as, diuretics, Angiotensin-Converting-Enzyme Inhibitors, Angiotensin-Receptor Blocker, vasodilators, or digoxin should be initially included. Cessation of cocaine is the primary goal of postdischarge therapy. The use of cocaine should be investigated in patients with cardiovascular disease, especially young patients, because its presence may influence disease diagnosis, management, and therapy. Non-invasive testing should be performed after several months to re-evaluate the treatment response.Cocaine use should be considered if young patients presented with heart failure, mainly without other underlying risk factors. It is important to counsel these patients regarding the deleterious effects of cocaine abuse and the potential reversal of cardiac dysfunction with abstaining from the cocaine use. The unfavorable economic impact and awareness of the possible cardiovascular effects should be considered during the initial evaluation when young adults with heart failure for medical assessment

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