International Journal of Progressive Sciences and Technologies (IJPSAT)
ISSN: 2509-0119.

Vol. 39 No. 2 July 2023, pp. 291-293

Cocaine& Heart: A Review

Angela Thomas

Tbilisi State Medical University

Abstract – Cocaine use can lead to acute and chronic complications, with one of the most dangerous effects being damage to the cardiovascular system. This review aims to explore cocaine's impact on the heart by examining relevant literature. Cocaine acts on alpha and beta receptors, as well as muscarinic receptors, resulting in hypertrophy of the left ventricle and decreased end-diastolic volume. The study emphasizes the importance of informing the scientific community about this case to raise awareness of the risks associated with cocaine use.

Keywords – Cocaine, Cardiovascular System, Heart Complications, Sympathomimetic, Hypertrophy, Left Ventricle

I. AIM OF THE STUDY

To review the current literature on cocaine's impact on the heart, with a focus on its cardiovascular effects and associated complications.

II. METHODOLOGY

To explore the impact of cocaine on the heart, this review article analysed relevant literature from reputable sources, including medical textbooks, scientific journals, and online databases. The selection criteria included studies that focused on the cardiovascular effects of cocaine use.

III. INTRODUCTION

Chewing the leaves of the Erythroxylon coca bush thousands of years ago gave way to purifying cocaine hydrochloride, used in tonics and elixirs (once in popular cola drinks), insufflating and injecting the fine, white, water-soluble powder form, and finally to a smokeable freebase form known as "crack," which gained popularity in the 1980s.1 2.1 million Americans used cocaine recently in 2007, and 1.6 million of them matched the criteria for cocaine abuse or dependence. Cocaine is the most used illicit drug and the leading cause of drug abuse-related emergency visits and deaths. Cocaine can cause irreversible structural damage to the heart, accelerate cardiovascular disease processes, and trigger arrhythmias and other cardiovascular conditions. Administration routes can be via oral ingestion, snorting, intravenously, or smoking. In a survey on 94 long-term cocaine users (mean regular cocaine use 13.9±9 years), cardiovascular magnetic resonance imaging found that 71% had some form of cardiovascular disease.

Smoking a cheap variety of "crack" cocaine is especially common since it has similar pharmacological characteristics to intravenous administration (Table1). Although snorting cocaine results in vasoconstriction of the nasal mucus membranes, which lowers the drug's bioavailability by more than half, some users prefer snorting due to its incredibly quick start of action.

Corresponding Author: Angela Thomas

Table 1

Cocaine Pharmacokinetics by Administration Route

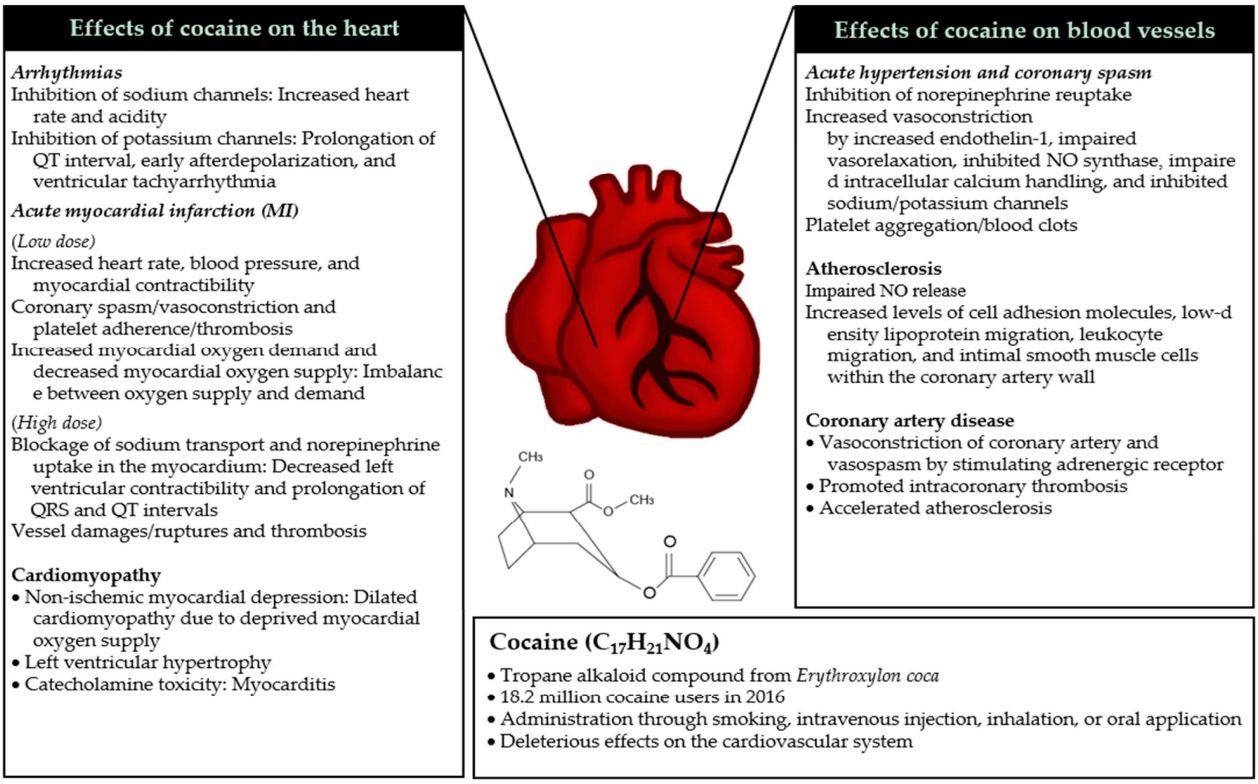
Route of Administration	Onset of Action	Peak Effect	Duration of Action
Inhalation (smoking)	3-5 seconds	1-3 minutes	5-15 minutes
IV	10-60 seconds	3-5 minutes	20-60 minutes
Intranasal	1-5 minutes	15-20 minutes	60-90 minutes

IV. Results

Cocaine inhibits presynaptic reuptake of serotonin and dopamine. Additionally, it increases the release of catecholamines in the central and peripheral nervous systems. Thus, cocaine has a twofold effect because it boosts both sympathetic output and catecholamine production. It causes vasoconstriction, platelet adherence, thrombus formation, and coronary spasm, all of which simultaneously reduce oxygen supply, while also increasing cardiac rate, blood pressure, and contractility, all of which increase myocardial oxygen demand. A myocardial infarction or ischemia may result from this oxygen deficit. In a dose-dependent manner, cocaine can also raise blood pressure and heart rate.

Traditional risk factors and plaque fissure or rupture with plaque haemorrhage, which triggers thrombosis, are the usual causes of MI in non-cocaine-using patients. Contrarily, MI in cocaine users involves intracoronary thrombosis overlaid on fibrous plaques rich in smooth muscle cells without plaque rupture or haemorrhage. Cocaine users frequently consume beer and smoke cigarettes; alcohol intensifies these chronotropic effects and nicotine can worsen pre-existing coronary artery disease and ischemia.

Long-term cocaine use may lead to structural cardiac alterations; this is the physiologic response to cocaine-associated hypertension, which raises vascular resistance and prompts the myocardium, especially the left ventricle, to increase its thickness as a protective mechanism. Symptoms can include chest pain with or without myocardial infarction, heart failure, cardiomyopathies, arrhythmias, aortic dissection, hypertension, myocarditis, and endocarditis. The disease is curable, but others progress to permanent cardiac dysfunction or death. The following figure shows effects of cocaine on heart and blood vessels:



The Approach to Cocaine-Related Chest Pain

The evaluation of the patient's clinical condition, electrocardiographic alterations, and elevation in cardiac troponin are indicated to safely and efficiently distinguish between benign and life-threatening chest discomfort in cocaine users. Accordingly, patients should be referred for an instantaneous coronary angiography, hospitalization, or a 12-hour watch. The mechanical basis for using nitrates, phentolamine, or verapamil (a calcium-channel blocker, to treat cocaine-related chest pain is derived from studies showing that administering each of these medications in the controlled environment of a cardiac catheterization laboratory altered cocaine-induced coronary vasoconstriction.

β-Blockers

The pharmaceuticals related to patients who use cocaine have been the subject of the most in-depth research and the most heated debate. Numerous end outcomes, including mortality, during and after acute MI, and in patients with cardiomyopathy, are benefited by β-blockers in non-cocaine-using patients. However, β- blocking may leave α-stimulation unopposed in cocaine-using patients, leading to significant systemic and coronary vasoconstriction. They may also lower survival and increase the incidence of seizures. The benefit of β -blockers is limited while they raise the risk of hypertension and coronary artery vasoconstriction because cocaine-induced MI seldom results in death. Anecdotal evidence suggests that taking blockers with cocaine carries a risk of severe chest discomfort, cardiac arrest, and death minutes after administration. In the acute environment, all β -blockers should be avoided by patients who use cocaine. Since the majority of patients continue to use cocaine after being released from the hospital, post-discharge β -blocker therapy should only be recommended after a rigorous risk-benefit analysis and may even be postponed until the cessation of cocaine usage has been established. Patients should be made aware of the risks associated with taking blockers and cocaine together.

V. Conclusion

This review highlights the importance of educating the public about the risks associated with cocaine use, particularly its impact on heart health. It emphasizes the need for healthcare professionals to consider cocaine as a potential complicating factor in heart diseases. Cocaine use for an extended period of time may have chronic consequences on the body, such as non-ischemic myocardial depression in the heart and endothelial cell damage and intracoronary thrombosis in the arteries. β- Blockers ought to be avoided in an emergency. Patients who use cocaine should adhere to existing guidelines for post-discharge therapy of MI and cardiomyopathy, with the exception that only a small group of patients should be provided β-blockers.

Additionally, lifestyle and behaviour changes (such as quitting smoking or drinking) are crucial for minimising the negative cardiac effects that these behavioural factors have on cocaine users. By raising awareness and promoting preventive measures, the adverse effects of cocaine on the cardiovascular system can be mitigated.

References

[1]. Brunner & Suddarth Textbook of medical surgical nursing 11th edition,

[2]. 2nd volume, Substance abuse. Table 71-1, 2545

[3]. Finkel JB, Marhefka GD. Rethinking cocaine-associated chest pain and acute coronary syndromes. Mayo Clin Proc. 2011; 86(12): 1198–207. 2. Egred M, Davis GK. Cocaine and the heart. Postgrad Med J. 2005; 81(959): 568–71

[4]. Freeman K, Feldman JA. Cocaine, myocardial infarction, and -blockers: time to rethink the equation? Ann Emerg Med. 2008; 51(2): 130–4.

[5]. Bhargava S, Arora RR. Cocaine and cardiovascular complications. Am J Ther. 2011; 18(4): e95–e100.

[6]. https://pubmed.ncbi.nlm.nih.gov/21690608/

[7]. https://pubmed.ncbi.nlm.nih.gov/21880648/

[8]. https://www.sciencedirect.com/science/article/abs/pii/S1344622322000724

[9]. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6387265/

[10]. https://www.uspharmacist.com/article/cocaine-related-chest-pain-10146/

[11]. https://www.mdpi.com/1422-0067/20/3/584