

Cocaine Induced Cardiac Arrhythmias

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Abstract

An addictive drug that provides pleasure and reward is more likely to attack various parts of the body and affect health. Dangerous substances are also involved in cocaine processing. Not just the high dosage of cocaine but a lower quantity may also lead to severe health issues, which ultimately cause panic attacks, violent behavior, and tremors. However, long-term use of cocaine may lead to heart attack and is likely to cause sudden death. Cardiac arrhythmia or irregular heartbeat is highly associated with cocaine use. The potential causes of cocaine-associated arrhythmias, including sodium channel blockade, ischemia, and potassium channel blockade, and clinical manifestations that are influenced by acidosis, hyperthermia, hypoxia, and electrolyte abnormalities will be discussed.

Keywords: Cardiac arrhythmias; Cocaine; Coronary artery; Cocaine-associated arrhythmias.

Introduction

What is cocaine?

Cocaine is an addictive drug derived from the leaves of the coca plant, used as an illegal stimulant and sometimes as a local anesthetic. It is a central nervous system stimulant that releases high levels of dopamine, a brain chemical associated with pleasure and reward. Colombia is the largest grower of coca bush, with a 76% increase between 2013 and 2016. The global area under coca bush cultivation was 213,000 hectares, with Colombia occupying 68.5%. In 2016, 1,410

tons of cocaine were produced, a 25% increase from the previous year. Colombia is the main source of cocaine seized in the United States, with 92% of samples seized in 2016 coming from Colombia [1].

Production of cocaine

Several chemicals are involved in the processing of cocaine and the dealers cut the drug with other substances in order to expand batches. Cocaine is a drug containing extracts from the *Erythroxylon* coca plant's leaves and cutting agents like talcum powder, baking soda, sugar, and caffeine.

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coca plant's leaves and cutting agents like talcum powder, baking soda, sugar, and caffeine. These substances, including fentanyl or heroin, are quite detrimental [2].

Cocaine is derived from the leaves of the coca plant, grown in South America. It is made by soaking, filtering, drying, mixing with solvents, pressing, and heating. Crack cocaine is made by dissolving cocaine hydrochloride into a mixture, heating it until an oily substance forms, and excess water and impurities are removed.

Consumption and general side effects

Cocaine is a drug that can be consumed in various forms, including liquid, powder, and crack cocaine. The drug stimulates the brain's dopamine system, causing a high in energy and alertness due to the release of high levels of dopamine [3]. Cocaine increases the activity of dopamine in the brain, which controls movement and rewards. It also enhances norepinephrine, which serves multiple brain functions. Even small doses can lead to euphoria, mental alertness, and increased energy, with hypersensitivity to touch, sound, and sight. Cocaine's impact on dopamine can reinforce drug-taking behavior. After a single dose, effects occur quickly, lasting from a few minutes to an hour. Short-term effects include restlessness, tremors, anxiety, and vertigo. Other potential short-term effects include panic attacks, heart rhythm disturbances, psychosis, and violent behavior. Some effects are more likely due to sensitization or higher doses. However, isolated use can cause sudden death through heart attack, stroke, or seizure [4].

Cocaine use can increase the risk of chronic and acute cardiovascular conditions like acute hypertension, aortic dissection,

coronary spasm, arrhythmia, heart attack, cardiomyopathy, endocarditis, atherosclerosis, and coronary artery disease.

Over time, cocaine use can lead to brain adaptation, leading to tolerance and increased doses. It has high abuse potential, causing dependence and withdrawal symptoms. Long-term cocaine use can increase the risk of conditions like appetite and nutritional changes, gastrointestinal issues, malnutrition, and liver and kidney damage. Additionally, it can cause significant withdrawal symptoms [1].

Cocaine-induced cardiac arrhythmia

Cocaine-induced cardiac arrhythmia refers to irregular heartbeats caused by cocaine use, which can interfere with the heart's electrical system and disrupt the signals that tell each heart to pump in sync. It can lead to various difficult-to-treat cardiac arrhythmias. Cocaine use can lead to serious cardiovascular issues, including the risk of cardiac arrhythmias. This is due to increased heart rate, electrical disturbances, vasoconstriction, increased oxygen demand, and coronary artery spasms. These effects can trigger tachycardia, atrial fibrillation, ischemia, ventricular fibrillation, and other arrhythmias.

The heart's oxygen demand can exceed supply, causing damage to the heart muscle and triggering arrhythmias. Coronary artery spasms can further compromise blood flow to the heart, causing arrhythmias. The immediate and severe effects of cocaine use can result in heart attacks, cardiac arrest, and even sudden death, especially for individuals with pre-existing heart conditions. Seeking help for cocaine addiction is crucial to prevent further health risks [5].

Treatment

Despite extensive animal and basic science research, there is limited data on the incidence or types of arrhythmias in cocaine users. This is largely due to the lack of healthcare access for those who never reach healthcare. In dogs, cocaine can produce various rhythm disturbances, some dose dependent. In human cases, cocaine use is associated with a range of arrhythmias, from benign sinus tachycardia to more severe ones like torsade de pointes, ventricular tachycardia, and ventricular fibrillation. Experimental models show that cocaine and some metabolites interact with cardiac sodium, calcium, and potassium channels. However, potassium channel blockade is not predictable and is likely due to the promiscuous nature of cardiac potassium channels [6].

Cocaine-associated sodium channel blockade and treatment of arrhythmias

Cocaine-induced arrhythmias can be triggered by two mechanisms in intact organisms. Cocaine increases circulating catecholamines, leading to increased heart rate and exacerbated sodium channel blockade. It can cause ischemia and infarction, creating a substrate for arrhythmias. The arrhythmia is related to host factors, dose, co-exposures, acid-base balance, and genetic variability.

Cocaine blocks fast inward sodium channels, resulting in delayed action potential upstroke, and impaired myocardial contractility. Manifestations on surface electrocardiograms range from subtle to overt abnormalities. No study has systematically evaluated electrocardiographic findings in acute cocaine toxicity [7].

Heart rate influences sodium channel blockade in cocaine, but tachycardia is not always linked to sodium channel blockade. Most tachycardia is caused by toxic drugs like tricyclic antidepressants. Tachycardia in cocaine is likely due to central nervous system agitation and increased catecholamines, such as anesthetized animals.

The treatment of wide-complex tachycardia related to cocaine is based on animal experiments and limited human data. It has been observed that cocaine was given to dogs and were likely to develop prolongation of the QRS complex, which is reversed by hypertonic sodium bicarbonate. According to another research, cocaine was given to animals until a significant widening of the QRS complex developed. At that point, either lidocaine, hypertonic sodium bicarbonate, or quinidine was administered.

Both lidocaine and hypertonic sodium bicarbonate improved conduction, while quinidine resulted in further prolongation of the QRS duration. Hypertonic bicarbonate has shown clinical improvement in humans, but it's often accompanied by acidosis and multiple therapies. Lidocaine use in cocaine-toxic patients is controversial due to potential proconvulsant effects. However, clinical experience with lidocaine in cocaine-associated acute coronary syndromes has shown benefits without seizures [8]. Patients with cocaine-related complex tachycardia should be divided into those who appear toxic or not, based on hypertension, tachycardia, diaphoresis, mydriasis, and psychomotor agitation. It is suggested to optimize ventilation and oxygenation and induce rapid cooling. It is also advised to administer hypertonic sodium bicarbonate if the QRS complex persists. Avoid IA and IC anti-

arrhythmic agents and consider electrical cardioversion for unstable or refractory patients.

Cocaine-associated myocardial ischemia and infarction and treatment of arrhythmias

Cocaine-associated myocardial ischemia and infarction is a complex process involving increased demand, vasospasm, enhanced coagulation, impaired vasodilation, thrombolysis, and accelerated atherogenesis. In some cases, ventricular tachycardia, torsades de pointes, and fibrillation are developed in patients. It has been studied that cocaine has catecholaminergic effects that may trigger vulnerable myocardium [9]. Cocaine-induced increase in intracellular calcium could contribute to arrhythmias. Animal models suggest potential therapies for these malignant ventricular rhythms, with nifedipine, verapamil, and diltiazem protecting against ventricular arrhythmias. However, α -adrenergic blockade has shown benefits in reversing acute coronary syndrome and coronary vasoconstriction, but not arrhythmias. Stable patients should be treated with magnesium or lidocaine, followed by hypertonic sodium bicarbonate [9].

Cocaine potassium channel blockade and treatment of arrhythmias

Potassium channel blockade, unlike sodium channel blockade, impairs repolarization and prolongs the QT interval on the surface electrocardiogram. Potassium channel blockade prolongs QT interval, causing atypical T wave morphology. This impairs

repolarization, allowing calcium accumulation, leading to delayed after-depolarizations and ectopic beats. The QT interval was studied in 45 cocaine users with and without chest pain syndromes, and QT prolongation was common [10].

Several cases must be assumed to explain the aberration of reports of consequential torsades de pointes and QT prolongation. Arrhythmias as a result of cocaine-associated potassium channel blockade require therapy, but data is not enough to make specific recommendations. Correct electrolyte abnormalities and treat unstable rhythms with overdrive pacing, magnesium, and lidocaine. Prophylactic magnesium is recommended for stable patients with corrected QT intervals or high heart rates [1].

Case study

32-year-old male presents to the office with palpitation. Patients admit to cocaine use and usually notice the onset of palpitations after consumption. The patient was referred by his primary care to access the patient's palpitations. An EKG was performed and followed by a 24HR Holter monitoring. Cardiac evaluation demonstrated frequent cardiac arrhythmias as noted in the below images.

At the patient's follow-up appointment, the case and findings were discussed with the referring physician and the patient. The patient was advised to stop the use of cocaine consumption specifically to reverse extreme cardiac arrhythmias. The outcome in this case is to be determined with follow-up care and long-term cardiac monitoring.

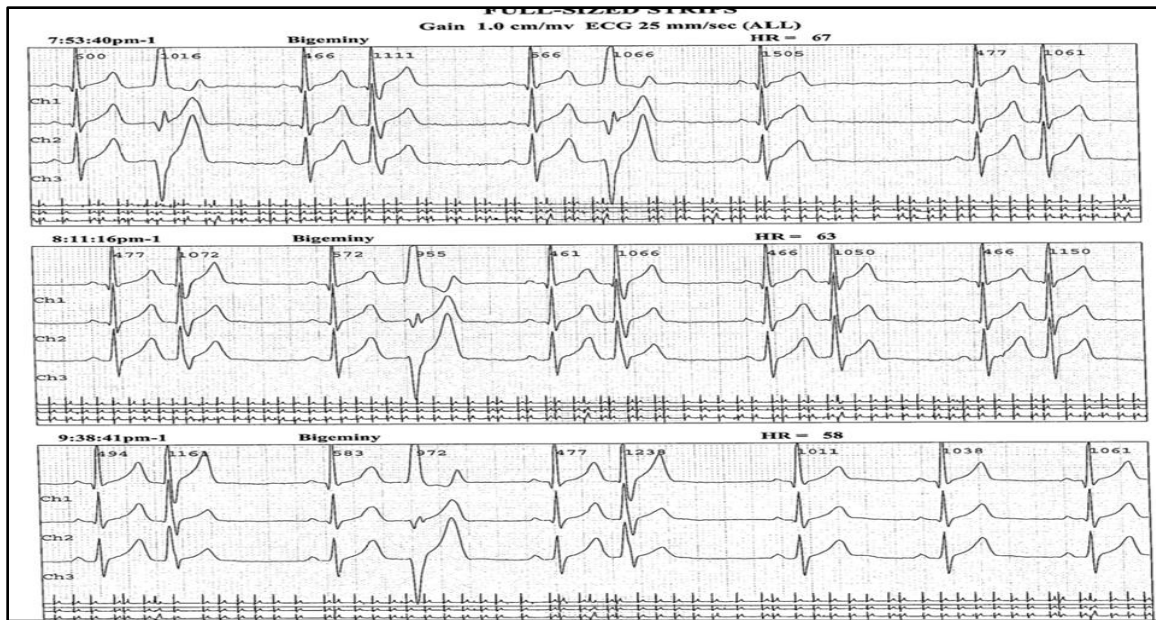


Figure 1: Waveform shows multiform, ventricular, premature contractions with bigeminy.

Conclusion

Arrhythmias in patients with acute cocaine toxicity are caused by complex mechanisms. A safe approach involves supportive care, including sedation, oxygenation, and electrolyte correction. Anti-arrhythmic agents, amiodarone, and hypertonic sodium

bicarbonate are generally used. Lidocaine is safe for re-entrant ventricular rhythms.

Patients should be closely observed in intensive care or a step-down unit until stability is assured. Referral to a detoxification program is crucial to prevent recurrent events.

References

1. Redman M. Cocaine: What is the Crack? A Brief History of the Use of Cocaine as an Anesthetic. *Anesth Pain Med.* 2011;1(2):95. [PubMed](#) | [CrossRef](#)
2. Posada CE. Cocaine Production and Trafficking What Do We Know? *Wrlld Bank Pub*; 2007. [CrossRef](#)
3. Currie IP, Otero-Bahamon S, Uribe S. What is the State Made of? Coca, Roads, and the Materiality of State Formation in the Frontier. *World Dev.* 2021;141:105395. [CrossRef](#)
4. Wei H, Shang L, Zhan CG, Zheng F. Effects of Cebtranopadol on Cocaine-induced Hyperactivity and Cocaine Pharmacokinetics in Rats. *Sci Rep.* 2020;10(1):9254. [PubMed](#) | [CrossRef](#)
5. Dugo E, Barison A, Todiere G, Grigoratos C, Aquaro GD. Cardiac Magnetic Resonance in Cocaine-induced Myocardial Damage: Cocaine, Heart and Magnetic Resonance. *Heart Fail Rev.* 2020;2:1-8. [PubMed](#) | [CrossRef](#)
6. Dominic P, Ahmad J, Awwab H, Bhuiyan MS, Kevil CG, Goeders NE, et al. Stimulant Drugs of Abuse and Cardiac Arrhythmias. *Circ Arrhythm Electrophysiol.* 2022;15(1):e010273. [PubMed](#) | [CrossRef](#)
7. Hoffman RS. Treatment of Patients with Cocaine-induced Arrhythmias: Bringing the Bench to the Bedside. *Br J Clin Pharmacol.* 2010;69(5):448-57. [PubMed](#) | [CrossRef](#)
8. Isenschmid DS. Cocaine. *Princ Forensic Sci.* 2020;371-87. [CrossRef](#)
9. Georgieva E, Karamalakova Y, Miteva R, Abrashev H, Nikolova G. Oxidative Stress and Cocaine Intoxication as Start Points in the Pathology of Cocaine-induced Cardiotoxicity. *Toxics.* 2021;9(12):317. [PubMed](#) | [CrossRef](#)
10. De Rubeis G, Catapano F, Cundari G, Ascione A, Galea N, Catalano C, et al. Cocaine Abuse: An Attack to the Cardiovascular System-insights from Cardiovascular MRI. *Radiol Cardiothorac Imaging.* 2019;1(2):e180031. [PubMed](#) | [CrossRef](#)