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Acute Coronary Syndrome Secondary to Intraoperative Cocaine Use: A Case Study

Abdulaziz Alshamlan[1] Vikram Anumakonda[1*]

[1]The Department of Acute Medicine, Dudley Group NHS Foundation Trust, UK.

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Corresponding Author: Vikram Anumakonda FRCP, FFICM, Consultant Physician, Department of Acute Medicine and Critical Care, The Dudley Group NHS Foundation Trust, UK.

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Abstract

Background

Topical cocaine remains a common choice in ENT surgeries despite known cardiovascular risks, including myocardial infarction (MI). We present a case of acute coronary syndrome following intraoperative cocaine use during a functional endoscopic sinus surgery (FESS).

Case Presentation

A 55-year-old female, with no significant medical history, underwent FESS under general anesthesia. Shortly after the cocaine administration, she developed a profound cardiovascular collapse, necessitating resuscitative measures. Diagnostic workup revealed ST-segment changes, elevated troponin levels, and severe left ventricular dysfunction consistent with stress cardiomyopathy. Subsequent imaging showed no coronary artery disease, supporting the diagnosis of cocaine-induced MI.

Discussion

Cocaine's sympathomimetic effects precipitate MI by increasing myocardial oxygen demand and causing coronary vasoconstriction. Genetic variations affecting cocaine metabolism may influence individual susceptibility to toxicity. Despite guidelines, adverse reactions can occur unpredictably, necessitating vigilance and personalized risk assessment.

Conclusion

This case highlights the serious cardiac risks associated with medical cocaine use and underscores the need for careful patient selection, monitoring, and consideration of alternative agents in sinonasal surgeries. Genetic screening for metabolic variants may aid in optimizing safety. Further research is crucial to refine protocols and enhance safety in cocaine use for medical purposes.

Keywords: Cocaine, myocardial infarction, stress cardiomyopathy, functional endoscopic sinus surgery, genetic variability, personalized medicine.

Introduction

Due to its function as a rapid-acting local anesthetic and vasoconstrictive, topical cocaine is commonly used in ear, nose, and throat (ENT) surgeons in nose surgery **[1,2]**. The use of cocaine in rhinology decreased over the years due to the availability of alternative medications with vasoconstrictive characteristics and the potential toxicities of cocaine, but it is still commonly used in practice **[1]**.

De R et.al. reported that their study aimed to identify the anesthetic and vasoconstrictor preparations used by UK otorhinolaryngologists in rhinological surgery, with a focus on cocaine and adrenaline. A survey of 360 BAO-HNS consultant members revealed that a majority still regularly use peri-operative cocaine, with 66% combining it with adrenaline. Notably, over 40% of these surgeons use cocaine in pediatric patients. While 16% of respondents did not use cocaine, only 11% had encountered cocaine toxicity, and there was just one reported case of mortality **[3]**.

The study found that most surgeons prefer cocaine for its ability to provide a superior operative field and its perceived safety, even when combined with adrenaline. The incidence of adverse reactions to cocaine is low, with serious complications being rarer than those associated with general anesthesia. Thus, cocaine continues to be a valuable tool in rhinological surgeries.

However, Cocaine metabolism in the body involves several key enzymes, each with genetic variations that can significantly influence how the drug is processed and its effects. For instance, the enzyme CYP2D6 helps break down cocaine, but people with certain gene variants may metabolize it either too slowly, leading to increased toxicity, or too quickly, reducing its effectiveness. Similarly, Butyrylcholinesterase (BCHE) is crucial for breaking down cocaine into inactive forms, but some individuals have variants that slow this process, prolonging the drug's effects and increasing the risk of side effects.

Other enzymes like CYP3A4/5 and Carboxylesterase 1 (CES1) also play roles in metabolizing cocaine, with genetic differences affecting their activity. The ABCB1 gene, which encodes the P-glycoprotein transporter, influences how cocaine moves through the body, including crossing the blood-brain barrier. Variations in this gene can alter how much of the drug reaches the brain and how long it stays active.

Case Study

A female patient aged 55 years old with no significant medical history went for a polypectomy by functional endoscopic sinus surgery (FESS) under the care of the ENT team in a Private Hospital. Medical family history includes

the mother passing away due to a cerebrovascular attack (CVA) at the age of 50, and known cardiac issues in different family members.

After Induction of anesthesia with Propofol, Remifentanil, and Rocuronium and on transfer to the operating table, the patient had acute signs of cardiovascular collapse. There was a drop in systolic blood pressure (SBP) from 110 (post-induction) to SBP 70 in the next 10 min. Metaraminol and boluses of IV fluids were given but no significant response was seen. After giving adrenaline SBP raised to SBP 160 with tachycardia.

The surgical procedure was initiated but due to ongoing hemodynamic instability, it was cancelled. SBP was 60 and ECG showed a narrow complex sinus rhythm with a heart rate of 50-60. Given the absence of a peripheral pulse at that moment and drop in EtCO2 to 2.5-2.0 with no pulse oximetry trace CPR commenced and IV adrenaline was also given. There was an immediate return of BP SBP 180-190.

Anaphylaxis protocol was started and CXR was done which showed pulmonary edema. The cause of the cardiovascular collapse was uncertain in her case, with suspicion of an anaphylactic reaction, side effects of rocuronium, cocaine toxicity (used intraoperatively for the procedure), or type 2 myocardial infarction (MI). The patient was later stabilized on adrenaline infusion and transferred to the district general hospital on adrenaline infusion where she was admitted to the intensive care unit (ITU) under the joint care of ITU and ENT teams.

On the next day of admission to ITU, the patient was extubated to continuous positive airway pressure (CPAP) with positive expiratory positive end-expiratory pressure (PEEP) 5 after the throat was deemed clear of clot with GlideScope.

Cardiology team input was sought in view of chest x-ray showing pulmonary edema, ECG showing T wave inversion in leads II, III, and AVF, and ST Depression leads V3-V6.T-waves inversion in inferior leads on electrocardiogram (ECG), and raised troponin levels of 353. Bedside-focused ECHO showed severe systolic left ventricular (LV) dysfunction with left ventricular ejection fraction (LVEF) of 45% +/-5% and a septal bowing into RV. ECHO also showed apical and septal hypokinesia but with no obvious significant stenosis or regurgitation. The patient was started on dobutamine and GTN infusions, in addition to commencing acute coronary syndrome (ACS) treatment. Serial ECGs were done **[figures 1-3]** and discussed with the cardiology team in the tertiary center, agreed that the hemodynamic instability seemed to be likely cardiac in origin and most likely due to cocaine-induced vasospasm. They suggested performing an angiography once the patient is more stable.

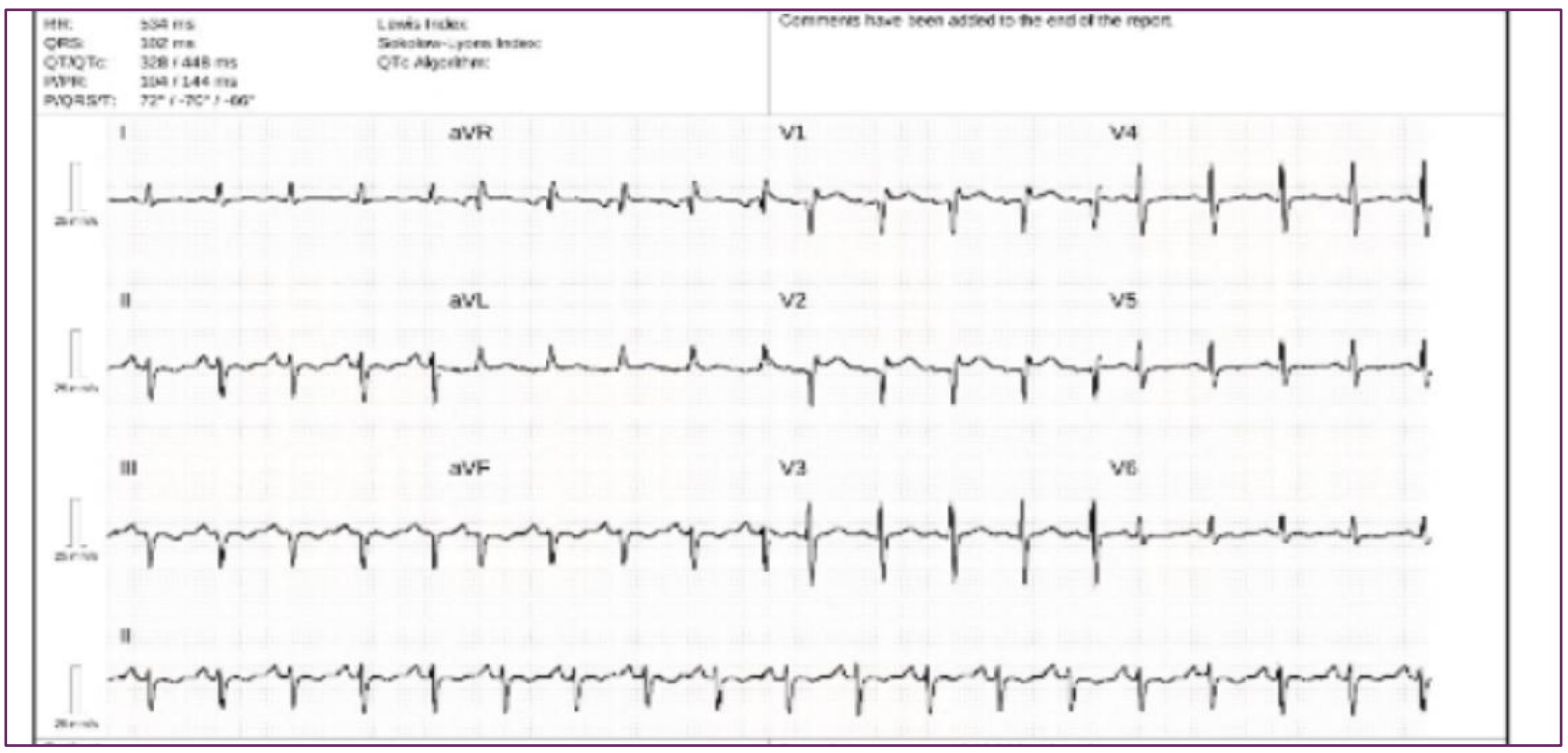

Figure 1: ST elevation in V1, V2, and bifid T wave in inferior leads with poor R wave progression in lateral leads.

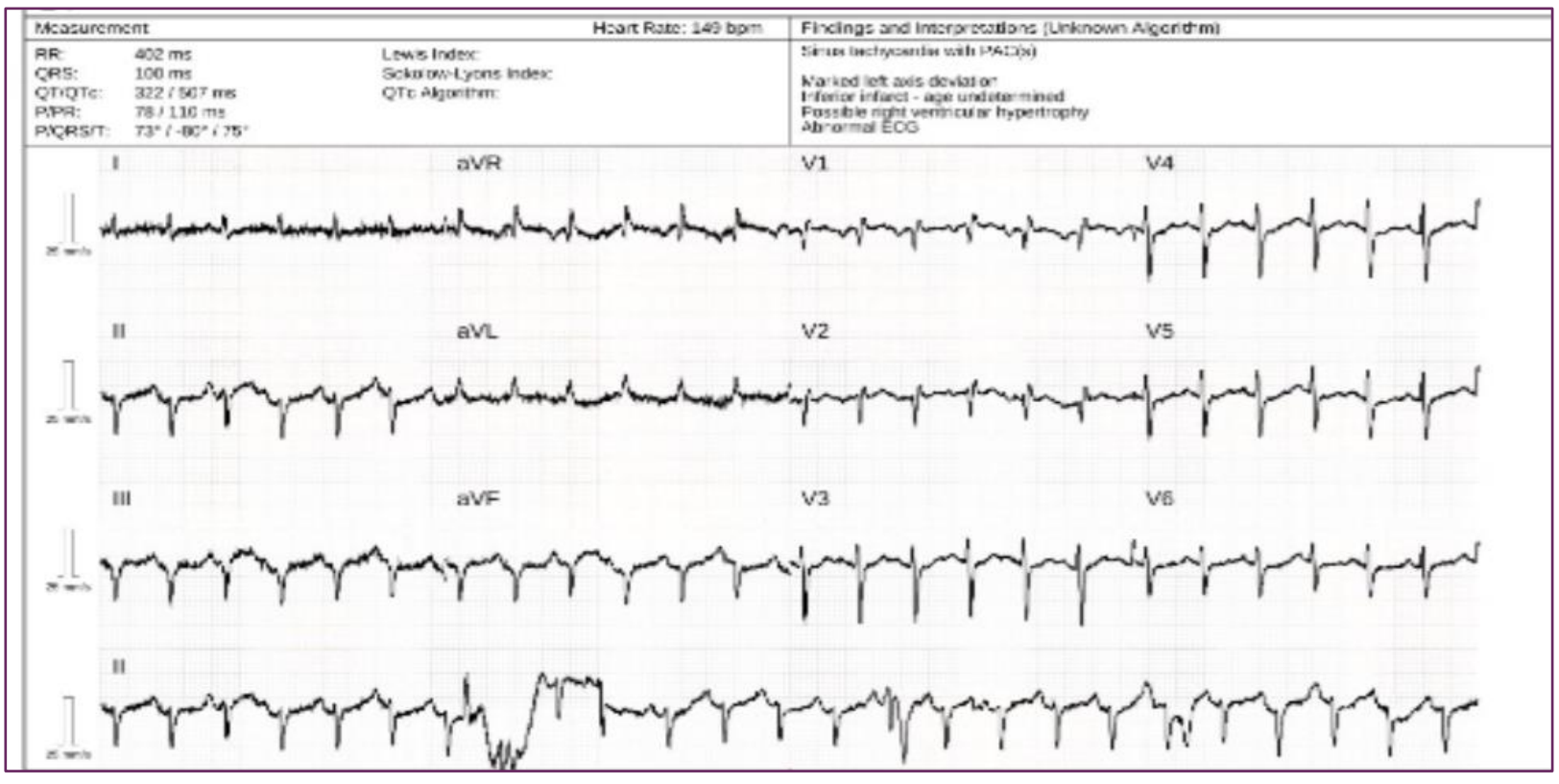

Figure 2: ST elevation in V1, V2 resolving, and subtle ST elevation in inferior leads with poor R wave progression in lateral leads.

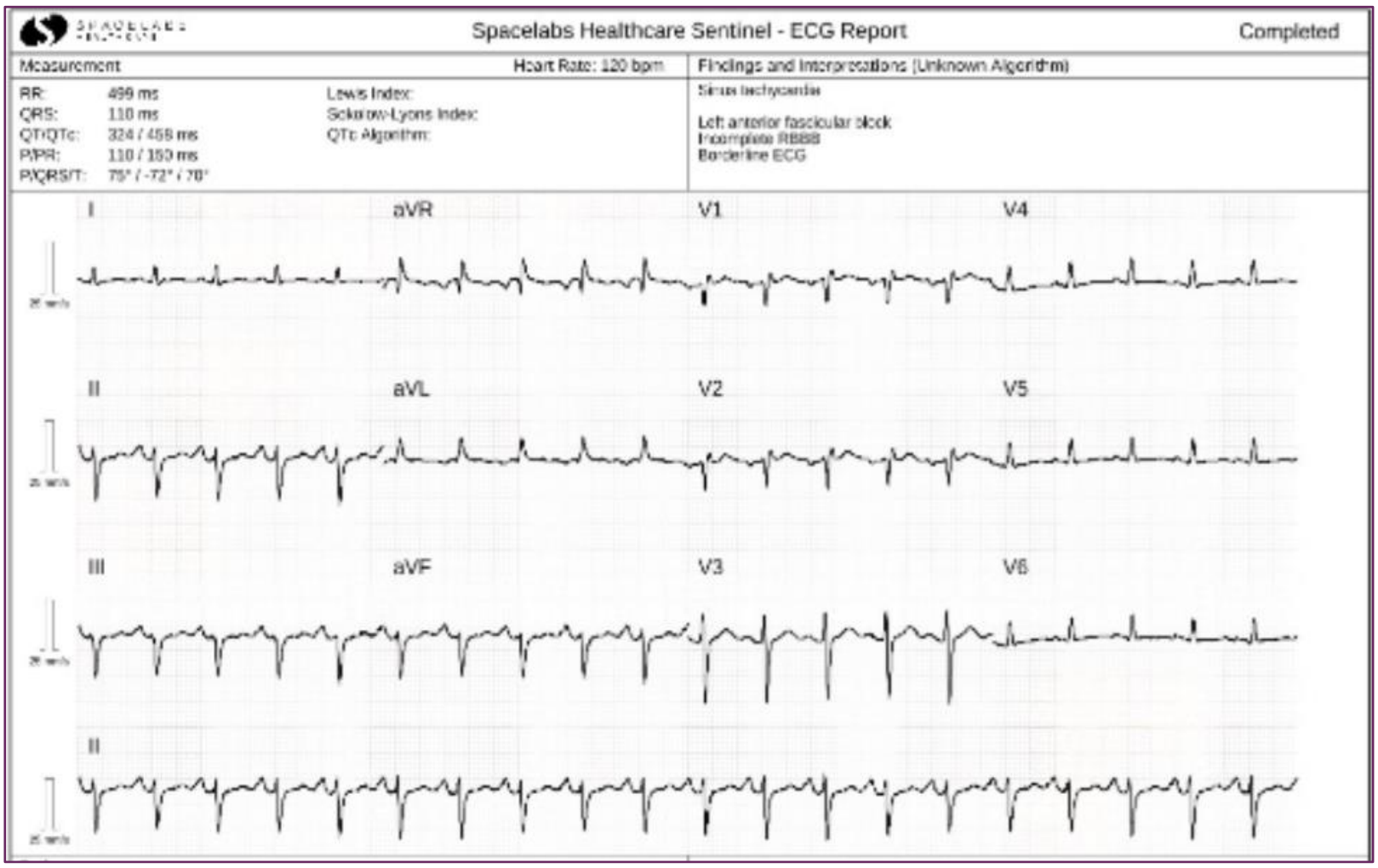

Figure 3: Q waves in V1, V2, and resolved T wave inversion in inferior leads with poor R wave progression in lateral leads.

A few days later, the patient stabilized clinically therefore, a Computed Tomography Coronary Angiogram (CTCA) and a repeat ECHO were requested. Repeat ECHO showed a normal-sized heart with hyperdynamic LV systolic function. The visual LVEF was 70% +/-5%, an improved function compared to the previous ECHO. CTCA showed a normal coronary artery, no coronary stenosis or plaques, and a calcium score of 0, which raised the likelihood the patient had Stress Cardiomyopathy (Takatsubo).

The patient was deemed medically stable for discharge. A follow-up in the cardiology outpatient clinic was arranged along with a cardiac MRI. Another out-patient follow-up with the ENT team has been also arranged.

Discussion

Cocaine is a tropane alkaloid that blocks the reuptake of amines, it acts as a sympathomimetic agent, central nervous system (CNS) stimulant, local anesthetic, and vasoconstrictive agent **[4,5]**.

Systemic side effects of the administration of cocaine are well-known, such as arrhythmias, cardiac ischemia, cerebral ischemia, convulsions, hypertension, hyperthermia, loss of consciousness, renal failure, respiratory depression, and rhabdomyolysis **[6-8]**. In our patient, she experienced a severe cardiovascular collapse shortly after the use of cocaine intranasally.

Acute coronary syndrome (ACS) or MI are well-known complication of cocaine use, however, most of the reported cardiac complications of cocaine were secondary to the recreational use of cocaine until the 1980s when cocaine started being used as a topical anaesthetic more cases of MI secondary to medical use have been published **[9]**. Even with the current common use of cocaine in sinonasal surgeries, cardiac events secondary to the medical use of cocaine are significantly lower than cardiac complications due to recreational use **[7]**. However, our patient was unfortunate to suffer from clear cardiac ischemic changes along with severe LV dysfunction leading to hemodynamic instability.

Cocaine increases the risk of acute coronary syndrome (ACS) in multiple ways. Firstly, due to its sympathomimetic effects leading to increased heart rate and blood pressure, cocaine increases the myocardial oxygen demand **[5,10]**. Secondly, due to its alpha-adrenergic effects, cocaine causes coronary vasoconstriction and therefore decreases the myocardial oxygen supply **[5,10]**.

It was widely reported that 200 mg is the maximum safe dose of topical intranasal cocaine **[2]**. However, there are multiple reports of significant adverse reactions and even deaths due to the administration of doses as low as 10 mg of cocaine, which raises the suspicion of the possibility of cocaine toxicity being dose-independent **[2]**. Recent studies suggest that topical cocaine doses should not exceed 100 mg or 1.5 mg/kg **[7]**. The highest concentration of cocaine in the bloodstream following nasal administration typically occurs within 15 to 60 minutes after application **[11]**. This is the same period during which our patient shows signs of hemodynamic deterioration.

The evidence of cardiac complications of intraoperative topical cocaine use is mainly based on case reports. However, a population study in 2020 studied the difference in perioperative cardiac complications between patients who have sinonasal surgery in institutions that use cocaine versus patients who have their surgeries in institutions that use alternative agents in sinonasal surgeries. They found no statistically significant difference in the rate of major cardiac events or death between patients treated with topical cocaine and those treated with alternatives **[12]**. Moreover, the overall rate of cardiac events and mortality within 48 hours of use was very low, ≤0.2% with no statistically significant difference in comparison to alternative agents.

The metabolism of cocaine is complex and varies significantly across different populations due to genetic differences. These genetic variations can impact the efficacy and safety of cocaine when used medicinally. Cocaine is

primarily metabolized by the cytochrome P450 (CYP) enzyme system, particularly CYP3A4 and CYP2D6 **[13]**. Variations in the genes coding for these enzymes can lead to differences in how individuals process the drug.

Genetic polymorphisms in CYP enzymes are distributed differently among racial and ethnic groups. For instance, certain variants of CYP2D6 that lead to poor metabolism are more prevalent in Caucasians compared to Asians and Africans **[14]**. This means that individuals with these variants may have higher plasma concentrations of cocaine, increasing the risk of adverse effects.

Some populations have a higher prevalence of enzyme deficiencies that can affect cocaine metabolism. For example, pseudocholinesterase deficiency, which can slow the breakdown of cocaine, is more common in certain ethnic groups. This deficiency can lead to prolonged effects and increased toxicity **[15]**.

Conclusion

This article aims to highlight a severe cardiovascular event, likely due to cocaine-induced myocardial infarction and stress cardiomyopathy, following the use of medical cocaine during surgery. It underscores the risks associated with cocaine, even in controlled settings, and the importance of personalised medicine, including genetic screening, to identify patients at higher risk of adverse reactions. The study advocates for cautious use of cocaine, considering safer alternatives in sinonasal surgeries, and calls for further research to better understand cocaine toxicity. Understanding the genetic factors is vital for personalised medicine, as it can help tailor treatments to minimize risks and optimize the safe use of cocaine in medical contexts. It emphasizes the need for thorough patient screening and ongoing vigilance to manage potential complications.

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