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Cocaine Abuse and Acute Heart Failure: Case Report of Rapid Recovery

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Abstract **Case Report**

Previous investigations have repeatedly found that cocaine use or misuse compromises the cardiovascular system. Acute effects include arrhythmia and acute myocardial infarction (MI) as well as chronic disorders such cardiomyopathy and coronary artery disease (CAD) are associated with cocaine. Cardiotoxicity brought on by cocaine can cause rapid death. Our case involves a 37-year-old man who experienced temporary acute left ventricular failure following cocaine and alcohol usage. The initial presentation was marked by an altered state of consciousness. The patient's evolution in the intensive care unit was favorable, with a complete recovery of myocardial function.

Keywords: Cocaine, cardiovascular system, cardiomyopathy, acute effects, chronic effects.

INTRODUCTION

Cocaine ranks second in terms of illicit drug use in Europe, following cannabis. It is estimated that roughly 13 million Europeans, or 3.9% of adults aged 15-64, have used cocaine at least once in their lifetime and is continuously increasing. In the United States, 0.6% of the population, or 1.5 million people, used cocaine at the age of 12 or older. Its usage is associated with acute and chronic effects that can affect any system, with the cardiovascular system being the most common. In fact, the exact incidence of cocaine-induced cardiomyopathy is still unknown and most likely underreported.

The report reveals the patient's quick recovery and demonstrates how important it is to have clear diagnosis criteria and thorough management guidelines for this complicated medical issue.

CASE PRESENTATION

A 37-year-old male was transported to the emergency department after being found unresponsive. He was unable to provide a history that was reliable. In retrospect, the patient reported excessive alcohol consumption accompanied by cocaine intake during the evening hours.

An initial assessment in the emergency department showed a confused and somnolent patient. The blood pressure was normal accompanied by bradypnea and oxygen saturation level while on 15L of oxygen. Chest auscultation revealed bilateral lung crackles. The urine screening was positive for cocaine and methadone. We also performed a brain CT scan, which showed no evidence of acute intracranial pathology.

The electrocardiogram (ECG) indicated a normal sinus rhythm with early repolarization pattern (Figure 1). High-sensitivity (HS) troponin levels were 156 ng/L (reference range: <13 ng/L), and the NTproBNP was 2557 ng/L (normal <450).

The chest X-ray (CXR) upon admission revealed multifocal bilateral lung infiltrates. A transthoracic echocardiogram (TTE) on the day of admission revealed a dilated and non-hypertrophied left ventricle with severe systolic dysfunction, the ejection fraction [EF] was estimated at 26% due to diffuse hypokinesia.

Furthermore, his labs on admission were remarkable for an acute kidney and liver injury: creatinemia of 254 µmol/L with an estimated glomerular filtration rate of 26 ml/min/1.73 m², ASAT: 265 UI/L (< 41), and ALAT: 352 UI/L (< 41). Besides that, his creatine phosphokinase (CPK) level was 2415 U/L. His CRP level was 150 mg/l and his white blood cell (WBC) count was 19.9 × 103/µL. Venous blood gas (VBG) and comprehensive metabolic panel (CMP) suggest lactic acidosis.

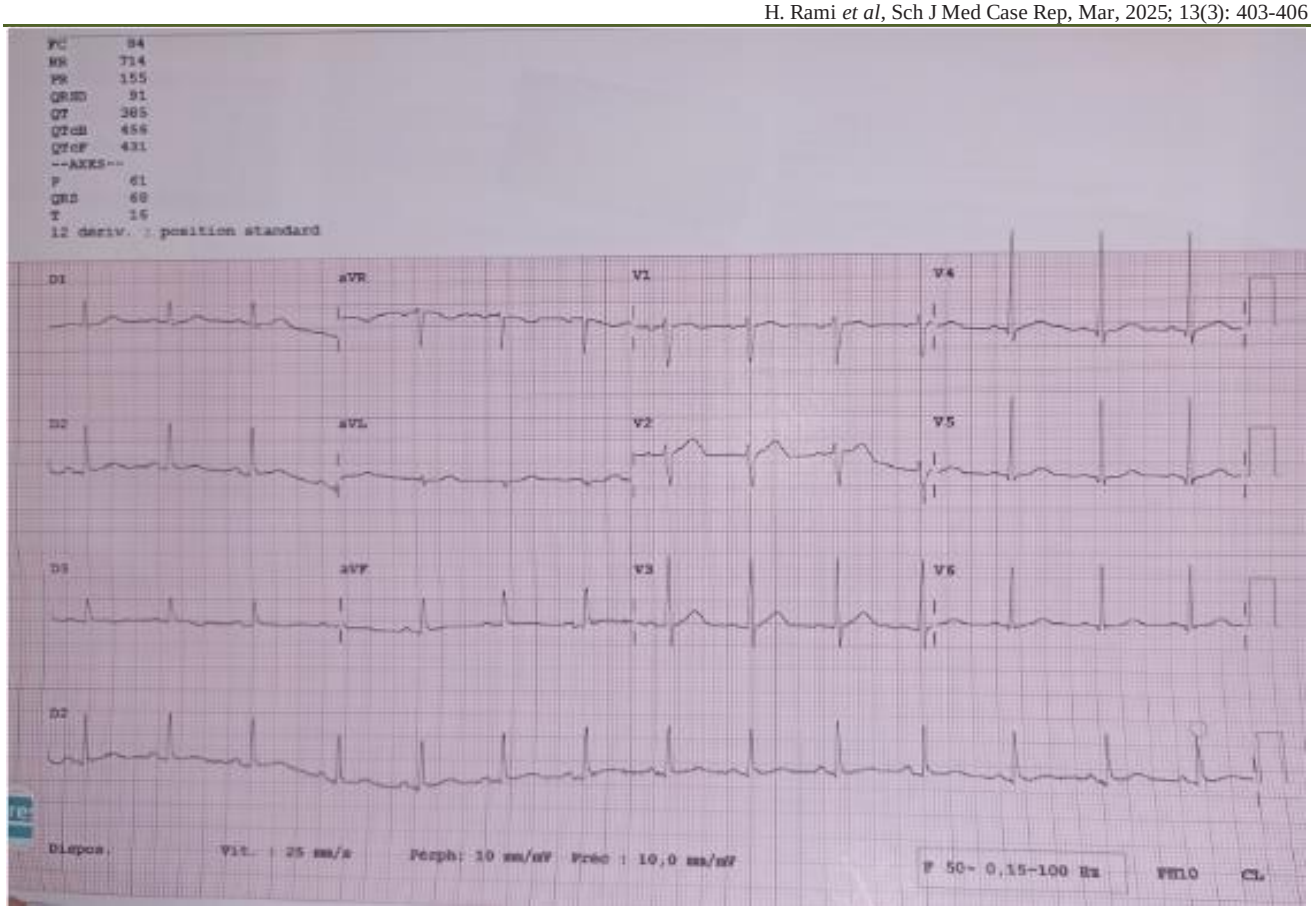

Figure 1: EKG remarkable for sinus tachycardia with early repolarization pattern

Due to the patient's changed mental status and hypoxic respiratory failure, he was admitted to the intensive care unit. The most likely diagnosis for the patient's altered mental status was toxic metabolic encephalopathy. The urine toxicology screening test yeilded positive results for cocaine. The patient's mental status returned to their baseline on the second day of admission,

Considering these observations and the findings from the 2D echocardiography, we made a provisional diagnosis of cocaine-induced cardiomyopathy, coronary exploration was not conducted at first due to the lack of angina symptoms and the absence of electrical and echocardiographic indicators of ischemic etiology. In addition, the patient was treated with amoxicillin for a possible inhalation pneumonia and developed acute kidney injury (AKI) as a result of rhabdomyolysis.

He required oxygen therapy at a flow rate of 6 liters, accompanied by intravenous naloxone at an initial dosage of 0.1 mg/h, administered prior to recovering consciousness and the diagnosis of a cocaine overdose. The subsequent introduction of diuretics occurred and non-invasive ventilation for pulmonary edema. The state of the patient in the intensive care unit was favorable, reflected by decreased oxygen needs and enhanced liver and kidney function.

A subsequent 2D echo revealed a significant enhancement in cardiac function, with a calculated ejection fraction of 47% (Video.1). By day five after admission, the patient's values had returned to normal ranges, confirming his speedy recovery. He was discharged home. One month later, a cardiac MRI verified the complete restoration of myocardial function without any late gadolinium enhancement (LGE) imaging (Figure 2).

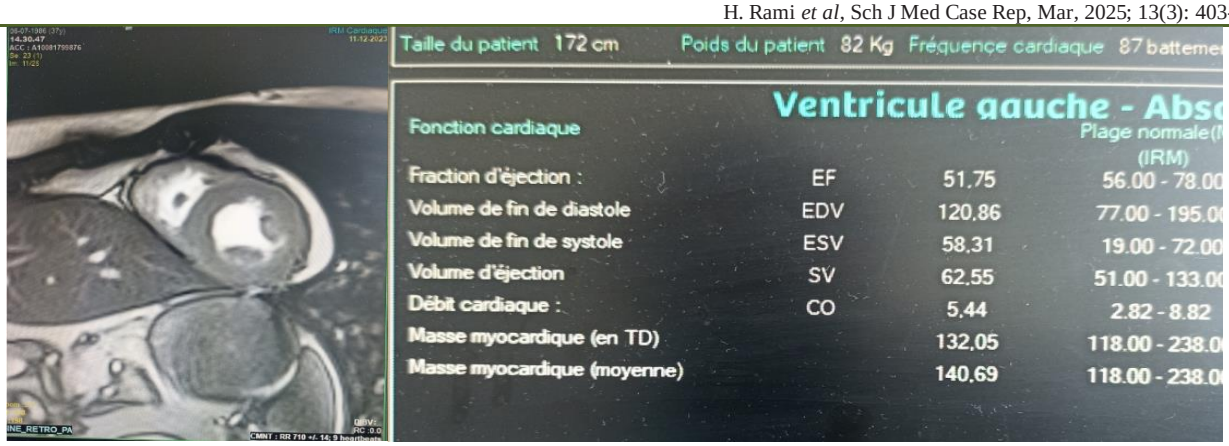

Figure 2: Follow up Cardiac MRI one month later with improved EF of 51 %

DISCUSSION

Tens of thousands of people die globally each year from cocaine usage, which is a serious health concern [1]. This drug can be smoked, snorted, or used intravenously, and it is absorbed readily through all mucosae. Long-term cocaine use, as well as acute cocaine use, is associated with adverse cardiovascular consequences, including arrhythmias, myocardial infarction and heart failure [2]. The cardiotoxicity induced by cocaine is a result of the direct blockage of sodium channels and the indirect suppression of catecholamine absorption, which leads to upregulation of sympathetic activity [4].

Although the exact causes of cocaine-induced cardiomyopathy remain unclear, several factors may play a role: Myofibril destruction, interstitial fibrosis, myocardial dilatation, HF, and possibly an induced hyperadrenergic state. After cocaine administration, acute manifestations of cocaine abuse may manifest within minutes or days; however, the risk is elevated within the initial 60 minutes of consumption [8].

Cardiovascular effects are often worsened by concomitant alcohol consumption; the abuse of these two substances together leads to the metabolism of cocaethylene in the liver, blocking the reuptake of dopamine and extending cocaine cardiovascular action [4]. Cocaethylene is an active metabolite of cocaine and alcohol that increases cardiac toxicity. Its plasma elimination half-life is around 2 hours, which is significantly longer than cocaine's (about 60 minutes). Cocaine plus ethanol users may experience a more intense and prolonged euphoric impact. It has been a subject of scientific interest for a considerable period; however, its metabolic mechanisms remain ambiguous and little understood. The impact on the cardiovascular and hepatic systems is more detrimental than that of the original toxicant [5].

In a retrospective cohort that compared 738 cocaine users with heart failure with reduced ejection fraction (HFrEF) with matched non-cocaine users, cocaine consumption was linked to elevated risks of all-cause mortality, heart failure readmission, and overall readmission [6, 8].

The standard approach for evaluating heart failure in nonusers should be followed when evaluating heart failure in individuals with suspected cocaine consumption. In this evaluation, a comprehensive evaluation is conducted, which includes a medical history, physical examination, 12-lead ECG, chest radiographs, trans-thoracic echocardiography and serial monitoring of cardiac biomarkers. Urine testing for cocaine and its metabolites can verify recent cocaine consumption [7].

At present, there is no specific timeframe that delineates the onset of cocaine-induced cardiac failure. Nevertheless, the diagnostic approach that is currently recommended for reversible factors that contribute to acute decompensated heart failure is adhered to by current guidelines. The ESC Guidelines 2021 recommend quadritherapy for the management of rLVEF, this includes ACE inhibitors or RNAi angiotensin-neprilysin receptor blockers, fixed-combination sacubitril/valsartan, beta-blockers, mineralocorticoid receptor antagonists, and SGLT2 receptor blockers, such as glifozines. However, the guidelines do not specify any specific management for the cocaine-user population. The benefit/risk ratio of these novel treatments with new pharmacological targets must be clarified in this context.

CONCLUSION

The accurate quantification of cases involving cocaine-induced cardiomyopathy and heart failure is still understated. This case highlights that cocaine-induced cardiomyopathy seems to have a rapid course of recovery. Complete cessation of cocaine use remains key for secondary prevention.

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