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Severe Cardiogenic Shock Secondary to Acute Cocaine Intoxication Revealing Cocaine-Induced Dilated Cardiomyopathy in a Young Woman: A Case Report

Nabil EL HANAFI SEBTI[1], Youssef EL HAIBA[2], Ayoub SEHBAOUI[3], Hind BAGGAR[4], Sara CHEBBAR[5], Anas MOUNIR[6], Mohammed Aziz BOUHOURI[7]

[1,2,3,4,5,6,7]Surgical Intensive Care Unit P17, Ibn Rochd University Hospital, Casablanca, Morocco

ABSTRACT

Cocaine is a potent sympathomimetic agent responsible for severe cardiovascular complications, including arrhythmias, myocardial ischemia, toxic myocarditis, and, in extreme cases, cardiogenic shock. We report a particularly severe case in a 26-year-old woman with known dilated cardiomyopathy and severely impaired left ventricular ejection fraction, admitted for acute cardiac decompensation following heavy and recent cocaine use. The dramatic hemodynamic deterioration required aggressive vasopressor and inotropic support, as well as multidisciplinary management. This case highlights the extreme danger of cocaine use in patients with pre-existing heart disease and underlines the importance of systematic screening and addiction prevention strategies.

KEYWORDS: Cocaine; Cardiogenic shock; Dilated cardiomyopathy; Acute heart failure; Cocaine-induced cardiotoxicity; Intracavitary thrombus; Intensive care; Toxic myocarditis; Hemodynamic instability; Substance abuse

INTRODUCTION

Cardiovascular toxicity related to cocaine use is one of the leading causes of intensive care unit admission among young adults who use illicit drugs. Cocaine acts by inhibiting catecholamine reuptake, resulting in massive adrenergic stimulation that leads to coronary vasoconstriction, tachycardia, paroxysmal hypertension, or conversely, hemodynamic collapse when myocardial reserves are exhausted. Direct myocardial injury through inflammatory and necrotic mechanisms has also been well documented.

In patients with pre-existing dilated cardiomyopathy, cocaine exposure represents a major trigger for acute decompensation and may lead to fulminant cardiogenic shock. The present case illustrates this deleterious interaction and reflects the potentially life-threatening nature of such situations in the intensive care setting.

CASE PRESENTATION

A 26-year-old woman, followed for two months for dilated cardiomyopathy with a left ventricular ejection fraction estimated at 10–15%, was admitted to the Intensive Care Unit 7 of Ibn Rochd University Hospital with cardiogenic shock. She reported daily cocaine use for four years, associated with regular alcohol consumption and heavy tobacco use. In the days preceding admission, she had self-medicated with a locally available stimulant derivative (“CRC”) and had abruptly discontinued her chronic treatments, which included a loop diuretic, an angiotensin II receptor blocker, an oral anticoagulant, and a beta-blocker.

On admission, the patient presented with profound hemodynamic failure. Blood pressure was 80/40 mmHg despite continuous norepinephrine infusion initiated in the prehospital setting. Heart rate was 135 beats per minute, with mottled skin, a grayish complexion, hypothermia at 35.2 °C, and severe respiratory compromise with oxygen saturation of 87% under a high-concentration oxygen mask. Respiratory examination revealed marked tachypnea and bilateral crackles consistent with cardiogenic pulmonary edema. Oligoanuria indicated severe visceral hypoperfusion.

Transthoracic echocardiography performed at admission revealed a markedly dilated, non-hypertrophied left ventricle with severe global hypokinesia and an ejection fraction of 10–15%. Two intracavitary left ventricular thrombi were identified, measuring 21 × 11 mm and 23 × 6 mm, respectively. The left atrium was markedly dilated. The right ventricle was not dilated but showed longitudinal systolic dysfunction. Additional findings included restrictive mitral regurgitation, a non-collapsible inferior vena cava measuring more than 21 mm, a small pericardial effusion, and large bilateral pleural effusions.

Laboratory investigations confirmed the severity of the condition. Serum creatinine was 1.38 mg/dL, indicating acute kidney injury. C-reactive protein was markedly elevated at 192 mg/L, reflecting a significant inflammatory response. Initial lactate levels exceeded

4 mmol/L. Total bilirubin was elevated at 78 mg/L, likely reflecting hepatic congestion. Subclinical hypothyroidism was noted with a TSH level of 5.8 µIU/mL. Viral serologies, particularly those suggestive of infectious myocarditis, were negative.

The initial course was marked by rapid worsening of hemodynamic instability, requiring escalation of vasopressor support and initiation of inotropic therapy with dobutamine at 10 µg/kg/min. Aggressive diuretic therapy with continuous furosemide infusion, supplemented by bolus doses, was initiated to manage severe fluid overload. A therapeutic pleural puncture drained 500 mL of fluid, resulting in slight improvement in gas exchange.

The patient was managed with continuous invasive hemodynamic monitoring, hourly urine output measurement, and repeated arterial blood gas analyses. Anticoagulation was initiated in view of the intracavitary thrombi, and empirical antibiotic therapy with ceftriaxone was started to treat episodes of fever observed during the first 48 hours.

Gradual stabilization was achieved by the third day of intensive care, following catecholamine support, correction of electrolyte disturbances, and optimized ventilatory management. Lactate levels normalized, blood pressure stabilized under norepinephrine, and urine output improved significantly. Follow-up echocardiography demonstrated modest but definite improvement in left ventricular ejection fraction.

DISCUSSION

Cocaine is widely recognized as one of the illicit substances with the most deleterious effects on the cardiovascular system. Its actions are mediated by intense adrenergic stimulation, promoting coronary vasoconstriction, arrhythmogenesis, and myocardial energy depletion. Several studies have described cocaine-induced toxic myocarditis, characterized by inflammatory infiltration, oxidative stress, and acute myocardial fiber injury. In the present case, the combination of severe dilated cardiomyopathy and massive cocaine consumption likely precipitated fulminant decompensation of an already compromised myocardium.

The presence of intracavitary thrombi, commonly observed in decompensated cardiomyopathy, is further exacerbated by cocaine's prothrombotic effects, including platelet activation and endothelial vasoconstriction. The biventricular systolic dysfunction observed in this patient is consistent with reports showing that cocaine intoxication can induce diffuse myocardial injury rather than segmental involvement.

Management relies primarily on hemodynamic stabilization. Vasopressors are often required, although their use is challenging due to the underlying adrenergic overstimulation induced by cocaine. Inotropes such as dobutamine play a key role in improving cardiac output but must be administered cautiously because of the risk of arrhythmias.

This case also highlights the critical importance of addressing addictive disorders. Abrupt discontinuation of guideline-directed heart failure therapy combined with relapse into cocaine use constituted a major aggravating factor and underscores the challenges of therapeutic adherence in patients with polysubstance abuse.

CONCLUSION

Cocaine-induced cardiogenic shock represents a life-threatening emergency, particularly in patients with pre-existing heart disease. This case, managed in Intensive Care Unit 7 of Ibn Rochd University Hospital, illustrates the severity of such presentations and the need for rapid, intensive, and multidisciplinary management. Cocaine remains a substance with a very high cardiovascular risk, capable of causing dramatic consequences even in young individuals. Prevention, early detection, and long-term addiction support should be integral components of the therapeutic strategy.

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