Acute Myocardial Dysfunction After Nasal Infiltration With Cocaine

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We report the case of a patient with severe myocardial dysfunction after nasal infiltration of cocaine during septoplasty. Complete recovery of myocardial function was observed in twelve days. Several reports have described chronic cardiomyopathy in long-term cocaine users, but only one case of acute cardiomyopathy. None of these cases were related to the medical use of cocaine.

Key words: *Cocaine. Contractility. Cardiomyopathy.*

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Disfunción miocárdica aguda reversible tras anestesia intranasal con cocaína

Presentamos un caso de disfunción miocárdica aguda tras infiltración nasal con cocaína durante una septoplastia, con recuperación completa de la función miocárdica tras 12 días. En la bibliografía se han publicado casos de miocardiopatía crónica por consumo de cocaína, pero sólo uno de miocardiopatía aguda y ninguno en relación con su uso médico.

Palabras clave: *Cocaína. Contractilidad. Miocardiopatía.*

INTRODUCTION

Cocaine is used in nasal surgery as a local anesthetic and vasoconstrictor. Its use is controversial due to its potential adverse cardiovascular side-effects.[1] However, we are unaware of any published cases of acute, serious myocardial dysfunction after using cocaine as a local anesthetic.

CLINICAL CASE

A 22-year-old woman was admitted for a bilateral breast prosthesis and rhinoplasty for cosmetic reasons. She had used cocaine occasionally, but not within the last 6 months. Electrocardiogram (ECG) and preoperative analysis were normal.

Breast surgery was done without complications using oxygen and nitrogen protoxide (50:50), midazolam (3 mg), perfusion with remifentanyl and propofol, and the topical use of adrenaline (1 mg) and lidocaine. Subsequently, the nasal mucosa was infiltrated with lidocaine (40 mL, 1%), adrenaline (1 mg), and 2 mL of cocaine (40 mg). After 20 min, but before the rhinoplasty had begun, she presented hypoxemia and hypotension requiring orotracheal intubation and perfusion with dopamine. She was immediately transferred to our intensive care unit (ICU) with a blood pressure of 100/70 mm Hg and a heart rate of 112 beats/min, on inotropic support. The analysis showed: troponin T 0.56 ng/mL, CK-MB 15 U/mL, normal total CK, and high concentrations of cocaine in the urine (>600 ng/mL). An electrocardiogram showed nonspecific alterations and chest x-ray showed signs of acute lung edema. The patient required inotropic support to maintain blood pressure. She was extubated 10 h after admission.

At 36 h, a transthoracic echocardiogram showed acute biventricular systolic dysfunction with a left ventricular ejection fraction (LVEF) of 10%. A second echocardiogram done on the fifth day was similar to the first one with an LVEF of 20% (Figure 1).

After 6 days in the ICU she was moved to the cardiology wards. At that time she was hemodynamically stable, without inotropic support and without symptoms. On the 13th day, another echocardiogram showed an LVEF of 60%; all other parameters were normal (Figure 2). The patient was discharged without medical treatment.

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Received 5 September 2002.
Accepted for publication 26 December 2002.

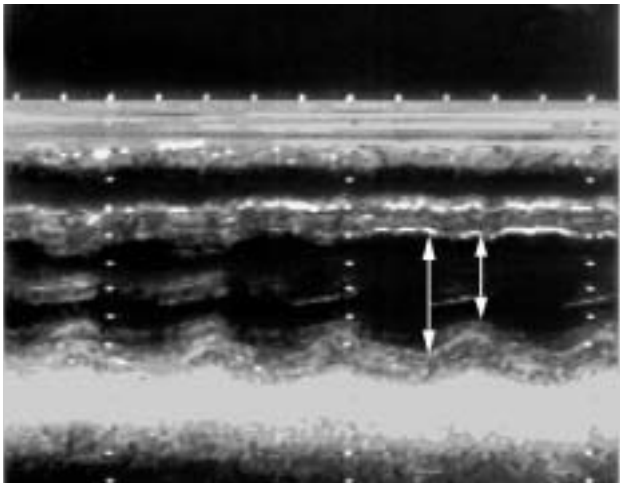

Fig. 1. M-mode echocardiogram done when the patient was in the ICU. Arrows indicate the diameters at end-diastole and end-systole of the left ventricle, which were 45 mm and 35 mm, respectively (shortening fraction 22%). Estimated ventricular function was 20%.

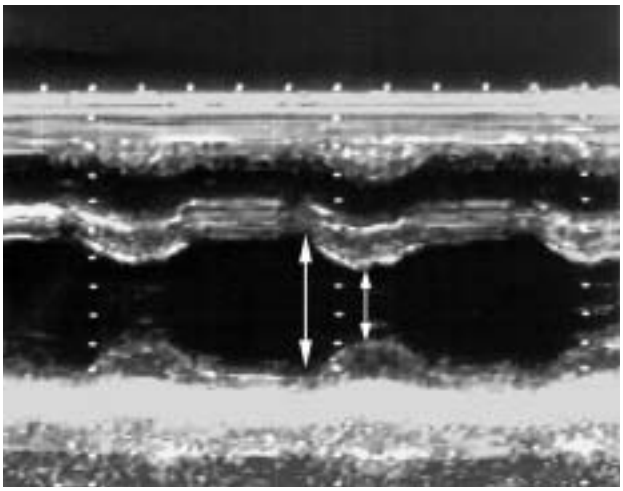

Fig. 2. Before discharge, the echocardiogram revealed full recovery of myocardial contractility (ejection fraction 60%). The end-diastolic and end-systolic diameters (arrows) were 43 mm and 25 mm (shortening fraction 22%).

DISCUSSION

The recommended dose for local anesthesia with cocaine is 1–3 mg/kg, although toxicity has been des-cribed at doses of 30 mg.[1] We used less than 1 mg/kg despite which concentrations in the urine were high. The patient assured us that she had not used cocaine for several months and, and even if she had in the last few days, given the half-life of cocaine, it is unlikely that this would have contributed to the situation. Cocaine users can have depressed asymptomatic myocardial contractility,[2] but this is unlikely in this case as LVEF was normal at discharge. After cocaine is applied intranasally, peak values in plasma are reached within 15-60 min, which coincided with the onset of the cardiotoxicity we report.

Cocaine can produce acute deterioration of systolic and diasystolic function[3] through several mechanisms.[4,5] For example, it induces myocardial ischemia by coronary vasoconstriction, but the relatively few changes in the ECG, as well as her clinical evolution, echocardiography and tests, suggest this was not a determining factor in this patient. Cocaine can also depress myocardial contractility, depleting calcium levels in cardiac myocytes and reducing the response of cardiac myofibrils to calcium. It also blocks the reuptake of catecholamines, which can exert a toxic effect on the myocardium, as seen in pheochromocytoma. A case of acute, severe myocardial dysfunction after crack consumption was reported, where the patient spontaneously recovered after 2 weeks. A case of dilated cardiomyopathy was also reported which resolved 7 mo after cocaine use was stopped. In contrast to these cases, the acute myocardial dysfunction we have described was related to the medical use of cocaine.

In summary, we report a potentially fatal complication, previously undescribed, associated with the use of cocaine as an anesthetic in a healthy person. Thus, we recommend the use of safer and equally efficient alternatives to cocaine, such as lidocaine and tetracaine, as a local anesthetic.[8]

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