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CASE REPORT

Seizures and cardiovascular collapse in a patient at the emergency department

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Abstract

We describe an unknown patient who was left behind at the emergency department with seizures, hypotension, and bradycardia caused by cocaine intoxication due to rupture of one of the packages he was smuggling inside his stomach. He was diagnosed with severe cocaine intoxication, admitted to the ICU and almost all the packages were removed from his stomach by gastronomy. A few days later he was discharged from hospital.

High blood levels of cocaine can produce the opposite clinical features to what you would expect in a patient who took cocaine, due to blockage of the sodium channels in the cardiac myocytes.

Introduction

Psychomotor agitation, tachycardia and high blood pressure are common findings in people who have taken cocaine and these findings are dose-dependent.[1-3] The opposite findings, such as low consciousness, seizures, bradycardia and low blood pressure are also possible in the clinical context of acute cocaine intoxication. These last findings are much more common at very high blood concentrations due to cocaine's reduction of the seizure threshold, Na+ channel blockade in the myocytes which slows or even blocks nerve conduction and its inotropic negative effects.[3,4]

We describe a patient who was 'dumped' at the emergency department while having seizures, low blood pressure, a low heart rate and finally asystole all due, as we found out later, to severe cocaine intoxication as a consequence of the rupture of one of the packets he was carrying inside his stomach (body packer). After a few days on the ICU he recovered completely and was sent to the ward.

Although cocaine intoxications usually present with the characteristic clinical features, it is important to keep in mind that they can also present with the opposite clinical features.

Case Report

A middle-aged looking but unknown male patient was thrown out of a car in front of the emergency room and left behind with seizures. He immediately received clonazepam and we proceeded with a physical examination. He was unconsciousness, hypotensive (BP 73/25 mmHg) with a heart rate of 61 beats/min, saturation of 99% on room air and a blood glucose of 17.3 mmol/l. While doing the physical examination he had two more episodes of tonic-clonic seizures for which he received two doses of clonazepam 2 mg.

Despite fluid challenge he became even more hypotensive and bradycardic and eventually went into asystole. He received cardiopulmonary resuscitation, was intubated, and after eight minutes he regained cardiac output, initially showing a ventricular rhythm with a wide QRS complex and hypotension, which normalised over a short period of time towards a sinus rhythm with a small QRS complex, while he received fluid administration, sodium bicarbonate and norepinephrine.

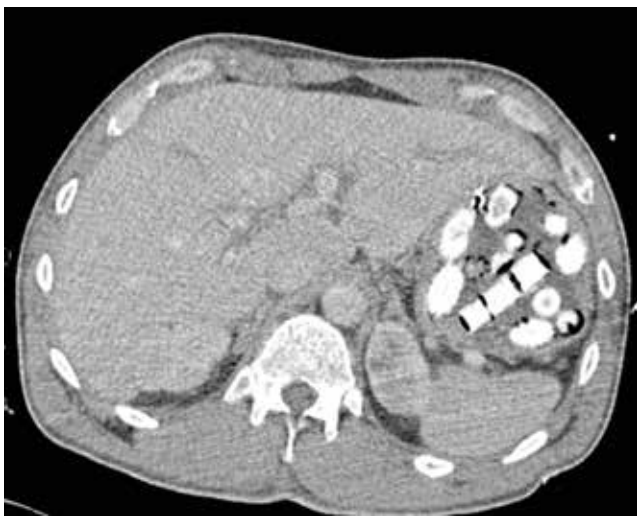

Figure 1. Abdominal CT scan: packages in stomach

A cardiac ultrasound carried out in the emergency department by our cardiologist showed normal cardiac dimensions, normal cardiac pressures and good contractility, and the abdominal ultrasound showed no abnormalities, especially no free fluid. We resumed the physical examination but we did not find any signs of trauma, no signs of intravenous drug abuse, no neck stiffness and only miotic pupils.

As possible causes we thought of intracranial pathology such as intracerebral bleeding, subarachnoid or subdural haemorrhage, cerebrovascular ischaemia or meningitis. Other possibilities were aortic dissection with or without carotid artery dissection, trauma (the patient was thrown out of a car in front of the emergency room and left behind) and of course intoxication and septic shock.

Complete blood count, toxicology screen and blood culture were taken and, once stabilised, he underwent a CT scan where we saw the image shown in *figure 1*.

The abdominal CT scan showed 'packages' in the stomach and one in the duodenum. The toxicology screen came back positive for cocaine and cannabis and with these results the diagnosis of acute cocaine intoxication probably due to rupture of one or more packets was made.

The patient was brought to our ICU where a central catheter and an arterial catheter were inserted and sometime later he was taken to the operating room where the surgeon preformed a gastrotomy.

Thirty-one packets were removed from his stomach, one of which was empty *(figure 2)*. The surgeon was not able to remove the packages that were already in the small intestine.

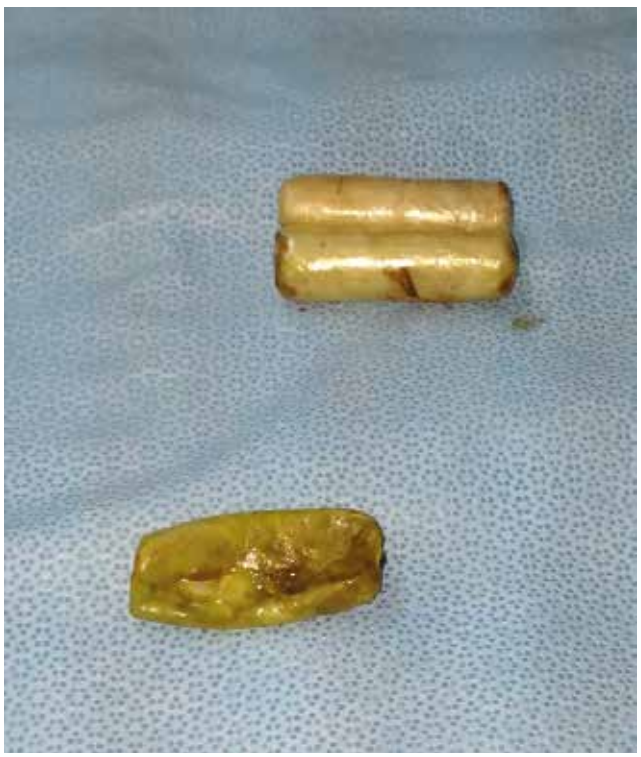

Figure 2. A ruptured package

Two days after the intervention the patient was extubated and he told us he had swallowed 34 packets. We kept the patient on the ICU for observation, due to the high risk of another acute cocaine intoxication; five days after the gastrotomy the patient expelled the other three packets (he received polyethylene glycol-electrolyte lavage solution).

This patient had acute cocaine intoxication due to rupture of one of the cocaine packets. The major symptoms of severe cocaine intoxication include central nervous system effects such as agitation, seizures, psychosis, and cardiovascular effects such as dysrhythmias, myocardial infarction and cardiovascular collapse.[5,6]

Although cocaine toxicity usually causes hypertension, massive toxicity may result in hypotension due to sodium-channel blockade, cardiac dysrhythmias, or cardiac ischaemia.[7]

Discussion

Cocaine is a tropane ester alkaloid found in the leaves of the *Erythroxylum coca* plant that grows in the Andes Mountain region of South America, and its use can lead to addiction and adverse physical effects, such as stroke and cardiac arrest.[5]

Typical cocaine doses are 20 to 100 mg intranasally, 10 to 50 mg intravenously, and 50 to 200 mg smoked. The intended effects include increased energy, alertness, and sociability; elation or euphoria; and decreased fatigue, need for sleep, and appetite.[6-8]

Cocaine stimulates alpha-1, alpha-2, beta-1, and beta-2 adrenergic receptors through increased levels of norepinephrine and, to a lesser extent, epinephrine.[3] The euphoric properties of cocaine derive from the inhibition of neuronal serotonin reuptake in the central nervous system.[9]

Cocaine also slows or blocks nerve conduction and acts as a local anaesthetic by altering the recovery of the neural Na+ channels. Cocaine has similar effects on cardiac Na+ channels and is able to slow the Na+ current in cardiac myocytes. With severe overdose, these cardiac Na+ channel effects manifest on an electrocardiogram as prolongation of the QRS complex, and clinically as negative inotropy.[3]

The seizures he was suffering from are seen in approximately 3-4% of cocaine-related visits to the emergency department and may occur without any underlying seizure focus.[4]

Asymptomatic body packers can be managed expectantly, with close monitoring in an intensive care setting or with whole bowel irrigation with polyethylene glycol/electrolyte lavage solution at a rate of 2 litres per hour and a nasogastric tube is typically required to ensure an adequate rate of administration.[3,10,11]

Whole bowel irrigation helps to speed up the passage of packets and has been used safely in body packers.[12] Use of oil-based laxatives is not recommended because they reduce the tensile strength of the packets.[10]

Body packers who present with cocaine toxicity should receive immediate, emergent surgical evaluation and be taken to the

operating room for surgical decontamination. There is no 'antidote' for cocaine, and therefore no place for conservative management in these patients.[13]

Disclosures

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