Remote Cocaine Use as a Likely Cause of Cardiogenic Shock after Penetrating Trauma

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ANESTHESIA in the setting of trauma is often complicated by the effects of acute and chronic substance abuse. Recently, cocaine has become one of the most common drugs used by trauma victims.1-3 Despite this all-too-common association of cocaine abuse and trauma, little is known of the impact of acute or chronic cocaine use on the physiologic response to trauma. We present a case in which hypotension after penetrating trauma in a patient with a history of chronic cocaine use is mistakenly assumed to be the result of occult blood loss.

Case Report

A 26-year-old man weighing 75 kg drove himself to the emergency room after sustaining a stab wound to the right flank. The patient had no other injuries and no past medical history of cardiac or pulmonary disease. In the emergency room, the patient was alert and cooperative. His systolic blood pressure was 70 to 80 mmHg and his heart rate was 125 bpm. Cardiac and pulmonary examinations revealed no signs of pneumothorax, heart murmur, or penetrating mediastinal or thoracic wounds. The patient’s abdomen was mildly tender. His initial hematocrit concentration was 41% and a repeated hematocrit test after he received an uncertain volume of intravenous fluid was 38%.

His blood electrolytes were Na⁺ = 145 meq/L, K⁺ = 3.6 meq/L, Cl⁻ = 106 meq/L, HCO₃⁻ = 6 meq/L, blood ura nitrogen = 15 mg/dL, and creatinine = 1.9 mg/dL. In light of his hypotension and tachycardia associated with a stab wound, peritoneal lavage was not performed and he was immediately routed to the operating room for an emergency exploratory laparotomy.

On arrival to the operating room he was awake, alert, and relatively cooperative. His initial blood pressure was 80/46 mmHg, his heart rate was 120 bpm, and his temperature was 36°C. During placement of electrocardiograph leads, he complained of chest pain and shortness of breath and was noted to be diaphoretic and clammy. The electrocardiograph monitor showed sinus tachycardia and ST-segment depression. A 12-lead electrocardiograph confirmed a rate of 120 bpm and 4-mm ST-segment depression in leads II, III, AVF, V4, V5, and V6 (fig. 1).

At this time, drug use was considered in the differential diagnosis of this man’s evident myocardial ischemia. However, he denied recent use of any drug except alcohol and his denial was confirmed by results of a urine toxicology analysis that were negative for cocaine, marijuana, barbiturates, and benzodiazepines. However, he did admit to using cocaine often, with his most recent ingestion occurring during the previous week.

Given that the patient’s hypotension and ischemia were presumed to be the result of intraabdominal blood loss, we felt it necessary to proceed with exploratory laparotomy for definitive treatment. Nonetheless, every effort was made to expeditiously stabilize the patient’s condition to permit safe induction of anesthesia and subsequent surgery. An intraaerterial catheter was inserted and arterial blood gas analysis revealed pH = 7.05, Pco₂ = 17.4 mmHg, PaCO₂ = 25 mmHg, bicarbonate = 7.1 meq/L, and lactate = 9.4 mm/L. A nitroglycerin infusion was begun at 2 µg/kg/min, producing symptomatic relief of chest pain and dyspnea and reducing ST-segment depression to 1.8 mm without a significant change in blood pressure. A dopamine infusion was begun at 8 µg/kg/min and subsequently increased to 10 µg/kg/min with an increase in blood pressure to 100/60 mmHg and a decrease in heart rate to 105 bpm.

With the dopamine and nitroglycerin infusions continuing, the patient was hemodynamically stable during induction with 750 µg fentanyl, 10 mg etomidate, and 110 µg succinylcholine. After tracheal intubation, general anesthesia was maintained with 0.5 to 1.2% isoflurane. A pulmo-
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Fig. 1. A portion of the electrocardiograph demonstrating S-T segment depression in leads V4, V5, and V6.

nary artery catheter was placed via the right internal jugular vein, and initial cardiac parameters were a cardiac output of 10 L/min, a pulmonary capillary wedge pressure of 17 mmHg, and a systemic vascular resistance of 457 dyne × s × cm⁻⁵. A second arterial blood gas analysis revealed pH = 7.32, PaO₂ = 305 mmHg, PaCO₂ = 34 mmHg, bicarbonate = 17 meq/L, and lactate = 5.6 mm/L. The patient’s hematocrit concentration remained stable at 38%.

The patient’s blood pressure continued to improve throughout the 1-h duration of his surgery, and his S-T segment depression resolved completely. The exploratory laparotomy revealed no intraabdominal or retroperitoneal injury. In fact, the stab wound extended only into the very superficial layers of his paraspinal muscle. At completion of surgery, his arterial blood pressure was 120/70 mmHg and his heart rate was 85 bpm on a reduced infusion of dopamine at 6 μg × kg⁻¹ × min⁻¹.

He was transported intubated to the surgical intensive care unit, where his trachea was extubated and all vasoactive drugs were discontinued by 4 h after operation. He had no further hypotensive or ischemic episodes in the surgical intensive care unit. Serial measurements of cardiac enzymes and serial electrocardiographs showed that the patient had not sustained a myocardial infarction. A two-dimensional echocardiogram revealed no valvular or wall motion abnormalities, and an ejection fraction was within normal limits. The patient was discharged 3 days after operation.

Discussion

Although hypovolemia secondary to blood loss was initially considered to be the cause of this patient’s hypotension and myocardial ischemia, his stable hema-
tocrit concentration, negative findings on laparotomy, and the superficial nature of his wound were not consistent with this initial diagnosis. In addition, the fact that his blood pressure did not decrease when the nitroglycerin infusion was started is not consistent with a hypovolemic state. Finally, between the time the nitroglycerin infusion was begun and the pulmonary artery catheter was placed, he received less than 2.1 crystalloid and no blood products. His initial pulmonary capillary wedge pressure was elevated at 17 mmHg, again suggesting that he was not hypovolemic when the nitroglycerin infusion was started.

Thus we believe that this patient’s shock state was cardiogenic in origin and not the result of penetrating trauma and occult blood loss as initially suspected. His anginal symptoms, the concurrent marked S-T segment depression, and the prompt resolution of his symptoms and S-T segment depression after administration of nitroglycerin suggest that his shock state was the result of heart failure caused by myocardial ischemia. We suspect the cause of his ischemia was coronary vasospasm, and the normal two-dimensional echocardiograph and electrocardiograph taken after operation are consistent with this hypothesis.

The cause of his vasospasm is not entirely clear, but we suspect it resulted from his previous cocaine use. The ability of acute cocaine use to cause coronary vasospasm, myocardial ischemia, and even myocardial infarction is well known. Less well known is the fact that even remote cocaine use can result in myocardial ischemia for weeks after the last drug consumption. For example, Nademance and colleagues report that 38% of chronic cocaine users admitted to an inpatient drug rehabilitation program have evidence of spontaneous myocardial ischemia as shown by Holter monitor for as long as 6 weeks after discontinuing cocaine use. These episodes are believed to be caused by coronary vasospasm.

The precise mechanism by which chronic cocaine use predisposes persons to coronary vasospasm even in the absence of the drug has not been completely elucidated; however several important clues offer some insight. Jones and Tackett, using a dog model, showed that chronic cocaine exposure sensitizes the left anterior descending coronary artery to catecholamines, even in the absence of cocaine, resulting in vasoconstriction. Certainly our patient’s endogenous catecholamines were likely to have been increased after his stabbing, and this may have contributed to his presumed vasospasm.
The mechanism responsible for the excessive sensitivity of the coronary vascular bed to catecholamines after chronic cocaine exposure is unknown but may be due in part to a cocaine-induced depletion of dopamine activity. Dopamine produces coronary vasodilation, and reduced dopaminergic activity in the coronary vasculature could predispose patients to vasospasm. We do not know whether chronic cocaine use decreases dopamine activity in the peripheral vasculature, although many animal studies have shown that chronic cocaine exposure does decrease dopamine activity in the central nervous system. In addition, the behavioral depression seen in human chronic cocaine users is also thought to result from central dopamine depletion.

Our treatment of this patient, despite his denial of recent cocaine use, was based on the hypothesis that coronary vasoconstriction related to cocaine abuse was a possible cause of his myocardial ischemia. A nitroglycerin infusion was begun to induce coronary vasodilation because of many reports that it is safe and effective for treating myocardial ischemia in the setting of cocaine use. Similarly, alpha-adrenergic blockade has been shown to be effective in treating cocaine-induced coronary vasoconstriction; however, the hypotension experienced by this patient made alpha blockade less appealing. We avoided use of a beta-adrenergic blocker, despite our patient’s tachycardia, because of evidence that beta blockade worsens coronary vasospasm in the setting of acute cocaine use. Whether beta-adrenergic blockade is also harmful for vasospasm in the setting of chronic cocaine use is not known.

We present a case of hypotension and shock after a minor superficial flank stabbing. The shock episode was the result of heart failure that was caused by myocardial ischemia. The patient’s myocardial ischemia was probably the result of coronary vasospasm related to his history of chronic cocaine use. Anesthesiologists should include the possibility of cocaine use as part of the differential diagnosis of unexplained myocardial ischemia, even if the patient is not currently intoxicated by the drug.

References

8. Jones L, Tackett R. Chronic cocaine treatment enhances the responsiveness of the left anterior descending coronary artery and the femoral artery to vasoactive substances. J Pharmacol Exp Ther 1990; 255:1366-70