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Case Report
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Malignant Hypertension-Associated Thrombotic Microangiopathy following Cocaine use

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Abbreviations: WBC: White Blood Cell; CRP: C-reactive protein; HIV: Human Immunodeficiency Virus

Case Report

Cocaine, a natural alkaloid derived from coca plant, is one of the most FREQUENTLY used illicit drug with distribution and consumption throughout the world [1]. According to epidemiologic studies, 25% of chest pains, 25% of moyocardial infarction, and 75% of hypertensive crisis are associated to cocaine use [2]. Cardiovascular dysfunction and renal failure due to rhabdomyolysis are well described following cocaine use. Although hypertension is a well-known manifestation of cocaine use, cocaine-induced malignant hypertension associated with thrombotic microangio pathy has been rarely reported [3]. We report 2 cases of patients who developed malignant hypertension associated with thrombotic microangiopathy after chronic consumption of cocaine.

Case 1

A 25-year-old man presented to our emergency service with a few days history of headaches with blurred vision, dyspnea and reduced urine output. In addition, he had fatigue, palpitations and exertional dyspnea but denied orthopnea. He denied any history of recent travel or exposure to sick contacts. Past medical history was significant for a recent cholecystectomy. His tobacco use is 33 pack-years, and was alcoholic with a habitual use of inhaled cocaine for several years.

Upon physical examination, he was alert, oriented in time and place, afebrile, with blood pressure of 240/130 mmHg a heart rate of 122 bpm, a respiratory rate of 22bpm and a. Oxygen saturation on room air was92%. An oral examination revealed multiple dental caries. A cardiopulmonary examination revealed scattered wheezes and rhonchi. There was no cyanosis or peripheral edema. Urine strips revealed proteinuria and hematuria. Abdominal, neurologic, musculoskeletal and lymph node exams were normal. Ophthalmological exam revealed papillary edema.

An initial laboratory workup showed 2,6mg/l blood urea nitrogen, 145 mg/L serum creatinine, 4.8 mmol/L serum potassium, 1.5 g/24h proteinuria. Blood count revealed elevated white blood cell (WBC) count of 14,700 cells/mm3 with 93% neutrophil predominance, elevated inflammatory markers of erythrocyte sedimentation rate 88 mm/hour and C-reactive protein (CRP) 70 mg/dl, and moderately elevated lactate dehydrogenase. Cardiac enzymes were normal. Serial troponins were negative. Extensive workup with hormonal testing, Human immunodeficiency virus (HIV) and hepatitis serology were negative.

An electrocardiogram was performed revealing sinus tachycardia and electrical left ventricular hypertrophy. An echocardiogram revealed concentric hypertrophy of the left ventricle with normal systolic function. The kidney ultrasound was normal, as well as renal artery Doppler, and the patient underwent kidney biopsy. The renal histological exam revealed that most of the glomeruli had global sclerosis. The rest demonstrated lesions compatible with thrombotic microangiopathy and ischemic lesions, including fibrinoid necrosis, intimal thickening, with microthrombi, and focal interstitial fibrosis.

He was started a continuous infusion of IV nicardipine. Although the patient reported improvement in his symptoms and hypertension was controlled within few hours, renal function did

not improve and the patient required chronic hemodialysis (twice a week).

He was discharged home in a stable condition and was addressed to addictology center.

Case 2

A 50-year-old male presented to cardiology emergency service with a 3 days history of worsening dyspnea, severe headaches and incoercible vomiting. Of note, he underwent a clinical evaluation one month prior that was significant for newly identified hypertension. His tobacco use is 40 pack-years and denied any alcohol or illicit drug use. Initial physical exam was significant for a blood pressure of 230/130 mmHg, a heart rate of 87 beats per minute, and a temperature of 37,8°C. Cardiac, pulmonary, abdominal, neurologic, musculoskeletal and lymph node exams were normal. Electrocardiogram revealed no abnormal feature. Transthoracic echocardiogram was performed which showed concentric left ventricular hypertrophy with normal systolic function.

Computed tomography of the brain was normal. Magnetic resonance imaging revealed focal, symmetric increased signal intensity in white matter and cortex, with occipital lobe involvement, which was compatible with hypertensive encephalopathy.

Full laboratory data was significant for a rapidly progressive renal failure, with a glomerular filtration rate of 30ml/min/1,73m^2 vs 64 two weeks before.

Extensive workup with hormonal dosage showed insignificant rate of aldosterone and Plasma Renin activity. Computed tomography of adrenal glands and renal artery Doppler were normal. Ophthalmological exam revealed stage III hypertensive retinopathy. He was initiated on Nicardipine and Central alpha agonists, with improvement of his tension rates within few hours. His toxicological screen came back with blood traces of cocaine. He was discharged in stable condition after 2 days and was started an addiction treatment.

Discussion

The major pharmacodynamics of cocaine involve the complex relationships of neurotransmitters, and mainly consist of blockage of dopamine transporter protein.

Dopamine transmitter released during neural signaling is normally recycled via the transporter. Cocaine binds tightly at the dopamine transporter forming a complex that blocks the transporter's function. The dopamine transporter can no longer perform its reuptake function, and thus dopamine accumulates in the synaptic cleft. The increased concentration of dopamine in the synapse activates post-synaptic dopamine receptors, which makes the drug rewarding and promotes the compulsive use of cocaine [1].

Regular and chronic use can result in severe arterial hypertension leading, sometimes, to terminal chronic renal failure [4].

Acute renal failure can also happen to patients with acute cocaine intoxication and it is established that the most common renal complication is rhabdomyolysis. The process linking cocaine with rhabdomyolysis remains unclear, but it could involve ischemia due to vasoconstriction and vasospasm due to cocaine's sympathomimetic action. This leads to tissue hypoxia with apoptosis, direct muscle toxicity, hyperthermia and increased muscle activity with sustained trauma due to widely excited activity following consumption [5].

Otherwise, cocaine use is linked to high blood pressure, but malignant hypertension is highly uncommon. To the best of our knowledge, reported cocaine-induced malignant hypertension associated with thrombotic microangiopathy morphological features is uncommon [6].

Cocaine-mediated endothelial injury and platelet activation may play significate pathogenic roles in cocaine users who develop acute renal impairment and malignant hypertension.

Other factors such as chronic hypertension, arteriolosclerosis and low cholinesterase activity can also be involved in some cases [7].

Usually, patients do not report a history of cocaine abuse, thus, it must be suspected by clinicians in case of renal impairment associated with significantly high blood pressure and poor response to conventional treatment. Serum and urine testing for cocaine are advisable in this clinical situation.

The most commonly used drugs are intravenous calcium blockers like nitroprusside, which can quickly and safely reduce blood pressure to target levels [8].

Benzodiazepines can be a part of the treatment of cocaine-induced malignant hypertension, as they minimize the sympathomimetic effects of cocaine on central nervous system.

Conclusion

Although epidemiological studies reported that cocaine use is linked to chronic high blood pressure, these two cases demonstrate that it can lead to acute malignant hypertension. Thus, clinicians should keep in mind this rare feature of cocaine intoxication.

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