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Cocaine-Induced Renal Artery Dissection as a Cause of Secondary Hypertension: A Rare Presentation

Authors' Contribution:
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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Conflict of interest: None declared

Patient: Male, 36-year-old
Final Diagnosis: Vertebral and right renal arteries dissection due to cocaine abuse
Symptoms: Abdominal discomfort • headache
Medication: —
Clinical Procedure: Renal artery angioplasty
Specialty: General and Internal Medicine

Objective: Rare disease

Background: Cocaine abuse is a globally recognized problem with great socioeconomic and health impacts on society. We report a case of dissection of vertebral arteries and right renal artery after cocaine abuse that clinically presented as atypical headache and hypertension.

Case Report: A 36-year-old male sought emergency care due to cervical pain after cocaine abuse. The pain was located to the right cervical side with irradiation to the homolateral temporal region. He had no previous comorbidities, except for cocaine abuse on a weekly basis. Angiotomography showed alterations compatible with recent arterial dissection of the right vertebral artery, confirmed on angioresonance. The patient received double antiaggregation and antihypertensive drugs and was discharged. He was readmitted 5 days later due to hypertensive crisis and mild abdominal pain. Abdominal ultrasound with a Doppler of renal arteries showed signs right renal artery stenosis. Magnetic resonance angiography confirmed dissection of the same vessel. The patient underwent arteriography with stent implantation in the right renal artery. During outpatient follow-up, he progressed with gradual reduction of antihypertensive drugs.

Conclusions: There is only 1 case report correlating renal artery dissection with cocaine use and none with concomitant presentation of dissection in the vertebral and renal arterial beds. The scarcity of reports is a consequence of many problems. Therefore, young patients presenting with new-onset hypertension or abdominal pain and cocaine abuse history should raise suspicion for renal artery dissection.

MeSH Keywords: Cocaine • Dissection • Hypertension, Renovascular

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/921565

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Background

Cocaine abuse is a globally recognized problem with great socioeconomic and health impacts on society. Its sporadic or habitual use can lead to a broad spectrum of organic dysfunctions, with varying degrees of severity. In this article, we report a case of dissection of the vertebral arteries and right renal artery after cocaine abuse that clinically presented as atypical headache and a cause of secondary hypertension, followed by a brief literature review.

Case Report

A 36-year-old male sought emergency care with a complaint of pain in the cervical region for 7 days, refractory to the use of simple analgesics and muscle relaxant. He reported that pain was located to the right cervical side with irradiation to the homolateral temporal region, of high intensity, which worsens when lying down or rotating the neck, and improves when standing. At the entrance he was afebrile, heart rate of 80 beats per minute, eupneic and hypertensive (170/130 mmHg). At physical examination, the patient reported pain on palpation of the temporal region, with no other relevant changes. Regarding the antecedents, the patient denied previous hypertension, other diseases or medication of continuous use. He had a moderate amount of alcohol ingestion at social events, 1 to 2 times a month, and smoking habit of 8 pack-year. When actively questioned, he reported being a cocaine user, on a weekly basis, and that he had used it the night preceding the onset of symptoms.

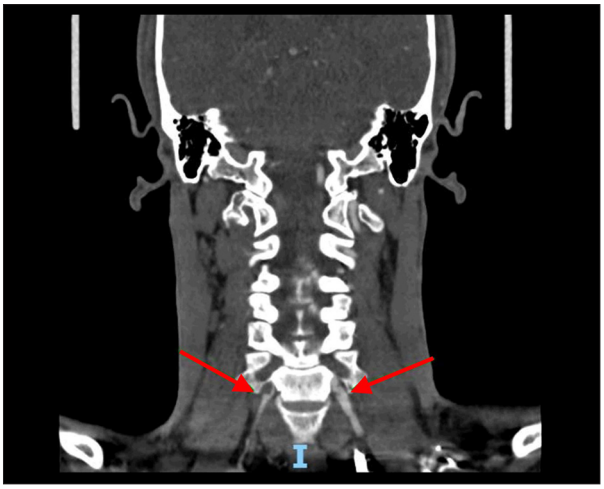

Figure 1. Computed tomography angiography of the neck and brain. Irregularities along with the cervical segments of the vertebral arteries (red arrows), more extensive to the right side where there are signs of subocclusion. The findings are consistent with recent arterial dissection.

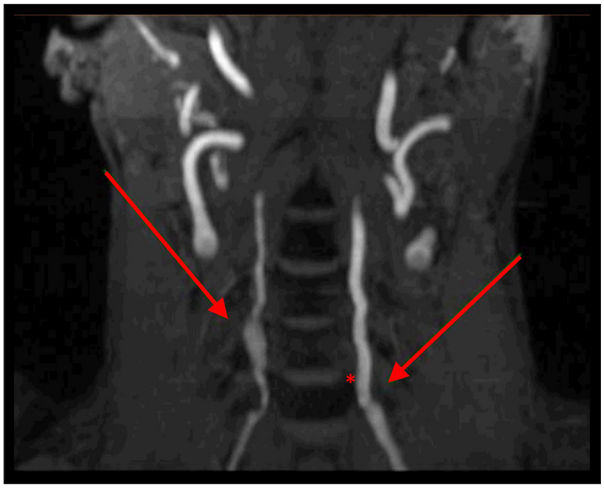

Figure 2. Magnetic resonance angiography of the neck and brain. Signs of recent arterial dissection in the V1/V2 segment of the right vertebral artery (right red arrow), determining a moderate reduction of its lumen. Discrete irregularities in the contours of the V1/V2 and proximal V2 transition in the left vertebral artery (left red arrow), with a small image in addition in its mesial contour (red asterisk), without clear intramural hematoma, determining discrete luminal reductions, and may be related to the sequelae of previous dissection.

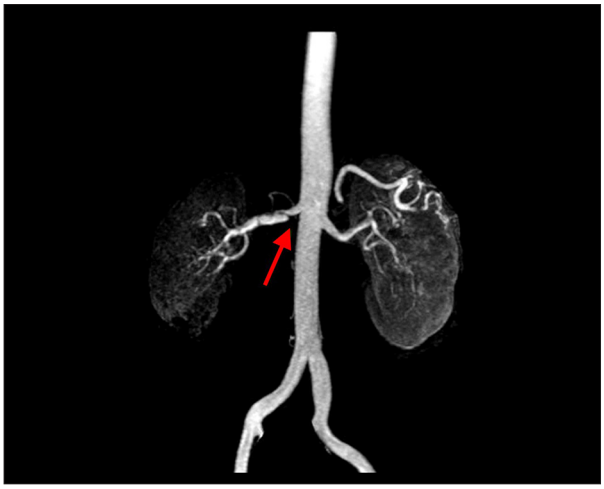

Figure 3. Magnetic resonance angiography of the renal arteries. Single and patent right renal artery, with flap of focal dissection in the proximal third, about 1.5 cm from the emergency, and deficit of contrasting the middle and distal segments (red arrow).

An evaluation of the on-call neurologist was requested, which, considering the atypical and new onset headache, indicated a cranial and cervical angiotomography. In the tomography, irregularities were found along with the cervical segments of the vertebral arteries, more extensive to the right side where there were signs of subocclusion, and the findings were compatible

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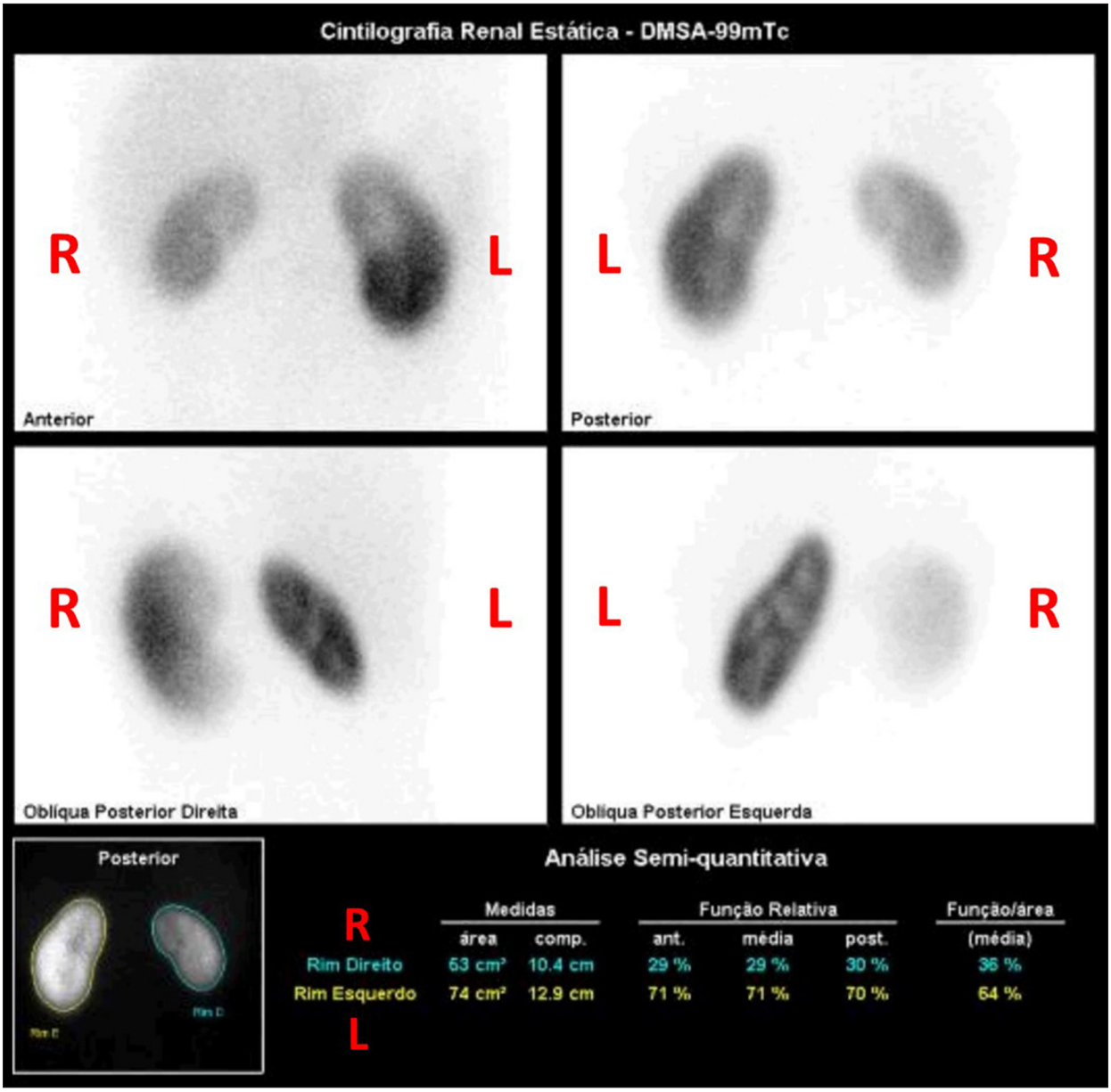

Figure 4. Renal scintigraphy with 99mTc-DMSA. Preserved global renal tubular function. Right kidney (R) with moderate tubular function deficit. Absence of cortical scars. Left kidney (L) with preserved tubular function and no evidence of cortical scarring.

with recent arterial dissection (Figure 1). Subsequently, the diagnosis was confirmed by magnetic resonance angiography of cervical and cranial vessels, performed on the same date (Figure 2). Transcranial doppler ultrasound did not reveal any significant change in brain posterior circulation.

The patient was treated with double anti-aggregation with aspirin 100 mg daily and clopidogrel 75 mg daily, plus losartan 50 mg twice a day and amlodipine 5 mg daily to control blood pressure, being discharged after 2 days for outpatient follow-up. However, 5 days after discharge, the patient returned due to a hypertensive crisis, being hospitalized for further evaluation.

At the time of readmission, the patient's blood pressure was 160/100 mmHg, having denied cocaine use during the period in which he was at home. He also reported mild abdominal pain of low intensity, with no associated symptoms, and no relevant findings on physical examination. Due to the complaint of abdominal pain, an abdominal ultrasound with a Doppler of renal arteries was requested and showed normal-sized kidneys, with velocities of acceleration and pulsatility of the intraparenchymal branches of the right renal artery reduced, indicating probable downstream stenosis.

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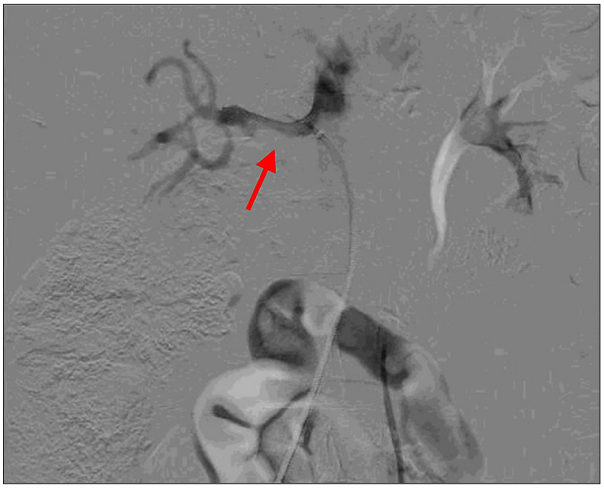

Figure 5. Angiography and stent implantation in the right renal artery. Persistent right renal artery, with signs of extensive dissection in its trunk to the bifurcation of the segmental arteries (red arrow).

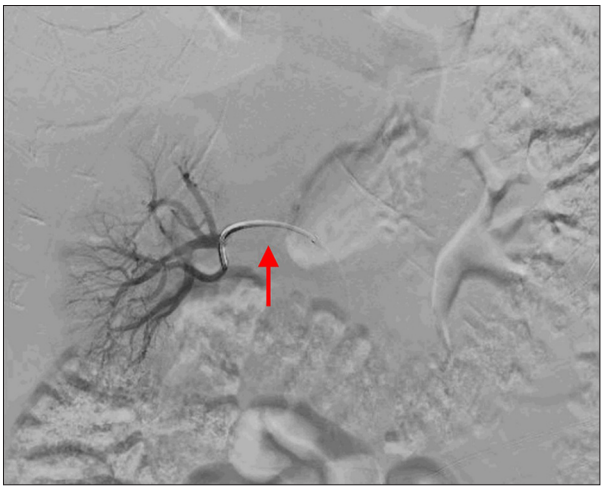

Figure 6. Angiography and stent implantation in the right renal artery. Transposition of the dissection zone was performed, finding the true light followed by self-expanding stent implantation to treat dissection (red arrow).

To complement the investigation, the patient underwent magnetic angio-resonance of renal arteries, which showed the presence of right renal artery dissection (Figure 3). The impact of dissection on renal function was measured by renal scintigraphy with DMSA (dimercapto succinic acid), showing a reduction of the glomerular and tubular function of the right kidney, according to Figure 4. Additionally, the patient underwent continuous blood pressure monitoring for 24 hours, confirming high blood pressure levels, with an average of 152/112 mmHg.

Due to the high blood pressure levels despite antihypertensive treatment intensification and the risk of progression of

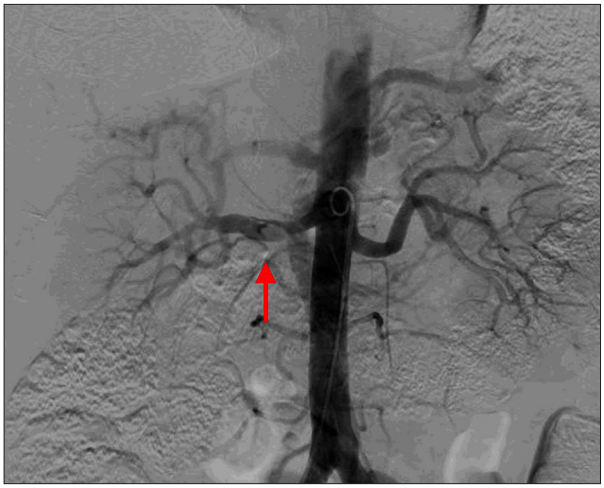

Figure 7. Angiography and stent implantation in the right renal artery. The arteriographic control revealed adequate stent placement as well as renal trunk recanalization (red arrow).

the arterial dissection in the renal bed, we decided to initiate interventional treatment. The patient underwent arteriography by percutaneous approach of the right femoral artery, with self-expanding stent implantation in the right renal artery (Figures 5, 6). The arteriographic control showed improvement of the renal arterial flow and venous return (Figure 7).

He was submitted to a Doppler ultrasonography of renal arteries that demonstrated adequate flow of the right renal artery, with normalization of the spectral pattern and the relationship between the systolic velocities of the aorta and renal arteries, along with the patent stent in the right renal artery. He was discharged on the fifth day of hospitalization, asymptomatic, with a blood pressure of 130×80 mmHg and no changes on physical examination.

At the time of discharge, he was on losartan 50 mg twice daily, amlodipine 5 mg daily, nebivolol 5 mg daily, aspirin 100 mg daily and clopidogrel 75 mg daily. During outpatient follow-up, he progressed with gradual reduction of antihypertensive drugs.

Discussion

To our knowledge, there is only 1 case report published correlating renal artery dissection to cocaine use [1], and no case reports with concomitant dissection of the vertebral and renal arterial beds. Most likely, the scarcity of reports is a consequence of multiple factors, among these factors are the difficulty in diagnosis due to the often non-specific clinical presentation, the difficult causal correlation with cocaine abuse, and the lack of consideration of this diagnostic hypothesis by the physician, being therefore an under reported entity.

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Cocaine is extracted from the leaves of the Erythroxylum coca plant and is now the second most widely consumed drug in the United States of America and Europe, supplanted only by marijuana use. In the United States, in the years 2016 and 2017, 14.4% and 14.9% of the population over 12 years of age, respectively, reported having ever used cocaine throughout their lives [2]. It acts stimulating the sympathetic nervous system, inhibiting the reuptake of noradrenaline and dopamine from the presynaptic slit and increasing its supply at postsynaptic receptors [3]. In addition, as it also blocks sodium and potassium channels, it behaves similarly to class I anti-arrhythmic agents and to local anesthetics [4].

Given its sympathomimetic effects, the initial signs and symptoms result from the stimulation of alpha-1, beta-adrenergic receptors, and cardiomyocytes, which clinically translates into increased heart rate, palpitation, increased blood pressure and myocardial contractility. The latter leads to increased myocardial oxygen consumption, which associated with coronary artery vasoconstriction may result in myocardial ischemia, a more prominent effect in patients with previous coronary artery disease [5].

In the vasculature system, multiple mechanisms participate in tissue ischemia due to the use of cocaine. The most likely is vasospasm due to the increased demand for oxygen in tissues and vessels. However, the formation of arterial thrombi by the activation and aggregation of platelets induced by the consumption of cocaine also participates; the mechanisms involve the increase of the plasma concentration of von Willebrand factor, greater shear stress in the endothelial wall, as well as increase of tissue factor expression by the endothelium [3].

In relation to the dissection of several arterial vascular beds already described, its genesis is multifactorial. Acceleration of the atherosclerotic process of the vessels in cocaine users has been reported, which favors the fragility of the arterial aspect [6]. At the molecular level, cocaine induces apoptosis of the smooth muscle cells of the arterial wall, a phenomenon that occurs in a concentration-dependent manner [7]. Signaling for apoptosis may be the result of increased production of reactive oxygen species-mediated by the drug, which leads to mitochondrial dysfunction and consequent induction of apoptosis [8].

Edmondson et al. [1] described the only case of renal artery dissection published so far in a 40-year-old male cocaine user who presented to the emergency department with severe abdominal pain associated with low back pain occurring 2 months after cocaine abuse had ceased. Dissection of the right renal artery was confirmed by imaging, associated with multiple areas of renal infarction. The therapy consisted of open vascular surgery associated with anticoagulation with warfarin.

Consistent with our report, both are young male patients, evolving with right renal artery dissection, but with different clinical presentations. In our case, the symptomatology was mild, and the main finding was secondary hypertension. In the external case, the clinical presentation was more exuberant, with no report of hypertension and with a greater time gap between last drug consumption and first symptoms, indicating that vascular wall fragility persists for some time even after cessation of cocaine use.

Conclusions

Therefore, whenever a young patient with a history of cocaine abuse reports to a medical center or clinic with abdominal pain or recent onset of hypertension, the on-call physician should raise suspicion for renal artery dissection and further evaluation with image examination must be considered.

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