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Case Report

Cocaine nephropathy: A rare cause of abnormal nephrograms ☆

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ABSTRACT

Cocaine use is associated with a variety of renal injuries. Although rhabdomyolysis is the most common cause of cocaine-induced nephropathy, cocaine can also cause renal vasculitis, acute interstitial nephritis, acute tubular necrosis, thrombotic microangiopathy, and renal infarction. We present a rare case of cocaine-induced nephropathy in a 30-year-old male who presented with acute kidney injury and abnormal nephrograms at contrast-enhanced computed tomography. Mechanisms of cocaine-induced renal injury and differential causes of abnormal nephrograms encountered at imaging are discussed. Cocaine-induced nephropathy is a rare but important cause of abnormal nephrograms and should be considered in the differential diagnosis when clinically appropriate.

Introduction

According to the United States National Institute on Drug Abuse, in 2014 approximately 0.6% of the population age 12 and older reported that they had used cocaine recreationally within the prior month [1]. In developed countries worldwide, there is a 1%-3% prevalence of lifetime cocaine use [2]. Cocaine is known to cause a variety of systemic pathologies, including acute kidney injury (AKI). Cocaine-induced AKI is most commonly caused by rhabdomyolysis [3]. Other reported mechanisms of cocaine-induced kidney injury include vasculitis, acute interstitial nephritis (AIN), acute tubular necrosis (ATN), thrombotic microangiopathy, and renal infarction [4].

We present a rare case of cocaine-induced nephropathy in a 30-year-old male who presented with abnormal nephrograms at contrast-enhanced computed tomography (CT).

Case report

A 30-year-old otherwise healthy male presented to the emergency department with bilateral flank pain, nausea, and vomiting following regular cocaine use over the preceding 2-3 weeks. He was taking no medications and had no past medical history. On presentation, he was hypertensive with a blood pressure of 153/95 mmHg. His vital signs were

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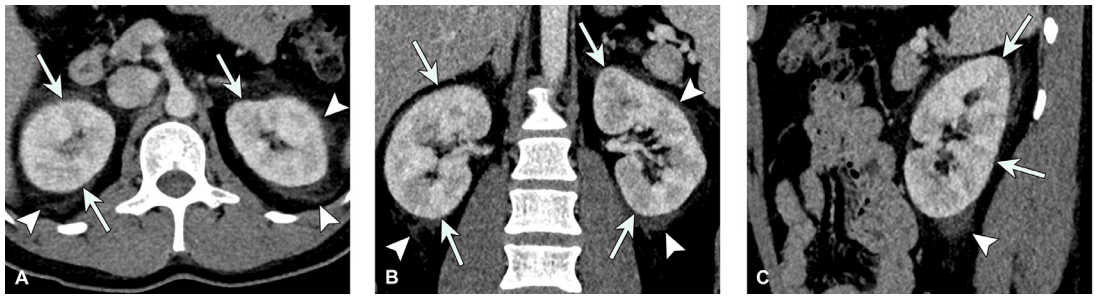

Fig. 1 – Axial (A) and coronal (B) contrast-enhanced CT images of both kidneys and sagittal image of the left kidney (C) show bilateral diffusely mottled nephrograms with scattered cortical striations (arrows) and bilateral mildly increased symmetric stranding of the perinephric fat (arrowheads). Cortical enhancement would be expected to be homogenous in a normal early phase nephrogram.

otherwise within normal limits. On physical examination he had bilateral costovertebral angle tenderness, greater on the right. The remainder of the physical exam was normal. Laboratory tests revealed a slight leukocytosis (12.9 K/uL) and elevated creatinine (1.51 mg/dL) with normal hemoglobin and platelet counts, electrolytes, liver enzymes, and serum lactate levels. The patient was started on intravenous (IV) fluids for treatment of AKI and underwent CT of the abdomen and pelvis for further evaluation. The CT was performed after the uncomplicated intravenous administration of 125 mL of Iopamidol 300 contrast material at an injection rate of 2 mL/s followed by a 30 mL saline flush at 2 mL/s. The scan was performed 65 seconds after contrast injection.

CT showed bilateral heterogeneous, mottled, and striated nephrograms and mild stranding of the surrounding perinephric fat (Fig. 1). There were no urinary calculi and there was no collecting system dilatation. The renal arteries and veins were widely patent and there were no abnormalities of either ureter or the urinary bladder. There were no organized perinephric fluid collections.

The patient was diagnosed with mild AKI attributed to cocaine-induced nephropathy. He subsequently received a total of 2 L of IV fluid in the emergency department. Following treatment, his blood pressure returned to normal and his pain improved. His creatinine then decreased to 1.35 mg/dL and he was discharged home after 9 hours with a plan to follow-up with outpatient nephrology.

Discussion

Cocaine blocks serotonin, norepinephrine, and dopamine reuptake, with resulting anesthetic, CNS stimulatory, and widespread vasoconstrictive effects, including in coronary, cerebral, and renal vasculature [3,5–7]. In addition to vasoconstriction, there are a variety of mechanisms by which cocaine adversely impacts the kidneys. Endothelial dysfunction, increased platelet activation, decreased levels of prostaglandin E2 and prostacyclin, and formation of reactive oxygen species have all been hypothesized to contribute to cocaine-induced AKI [5]. Cocaine has been shown to affect all parts of the nephron both in rats exposed to cocaine [8] and in human autopsy studies [9].

Cocaine-induced nephropathy is a broad term which can be used to describe the variety of renal diseases resulting from cocaine use. Rhabdomyolysis is the most common cause of cocaine-induced AKI [3]. Levamisole, an antihelminthic drug commonly used as a bulking agent in cocaine, has been linked to an ANCA-associated vasculitis [4,10]. Other cocaine-associated causes of AKI are less common, such as vasculitis, atherosclerosis, glomerulonephritis, AIN, renal infarction, and thrombotic microangiopathy [5].

Cocaine-induced nephropathy can produce abnormal renal enhancement on CT, most commonly resulting in striated or mottled nephrograms. A striated nephrogram is a nephrographic pattern in which there are alternating bands of well-enhanced and less well-enhanced renal parenchyma in a radial pattern throughout the cortical and medullary layers. In comparison, a mottled (or spotted) nephrogram is characterized by patchy, amorphous segmental and subsegmental areas of differential enhancement [11,12].

The differential diagnosis of abnormal nephrograms on CT is broad. In general, striated nephrograms are usually caused by edematous nephrons and/or intratubular obstruction. The varying densities between the alternating bands are due to contrast material stasis within the abnormal tubules. A unilateral striated nephrogram can be caused by pyelonephritis, ureteric obstruction, renal contusion, and renal vein thrombosis. Bilateral striated nephrograms can be caused by autosomal recessive polycystic kidney disease, tubular obstruction (such as secondary to ATN), bilateral pyelonephritis, or systemic hypotension [11,12]. Mottled nephrograms are usually caused by differential perfusion of areas of the kidney due to occlusion of small vessels. Most commonly mottled nephrograms result from an intrarenal vasculitis. On occasion, however, mottled nephrograms can occur secondary to embolic disease or severe pyelonephritis [12].

Though both striated and mottled nephrograms can occur due to differential perfusion within the kidney, the location and pattern of the findings (unilateral vs bilateral; focal vs diffuse), clinical history, and laboratory data are all critical for narrowing the differential diagnosis when abnormal nephrograms are encountered at imaging. The renal vasculature and collecting systems should be scrutinized for signs of arterial

thrombosis, venous thrombosis, urinary tract infection, or urinary obstruction when mottled or striated nephrograms are encountered at contrast-enhanced CT.

Perinephric stranding is a nonspecific CT finding that often results from an inflammatory process involving the kidney or collecting system. This finding is characterized by curvilinear areas of soft tissue attenuation that extend radially or parallel to the renal capsule within the perinephric space and is caused by edema and congestion of the surrounding lymphatics. Causes of perinephric stranding are numerous and include acute pyelonephritis, urolithiasis with obstruction, trauma, neoplasm, renal vein thrombosis, renal infarction, AIN, and ATN [13–15]. The prevalence of perinephric stranding related to cocaine nephropathy is unclear, although it has been previously reported in cases of cocaine-induced AIN [16].

In this case, the patient had both a striated and mottled pattern to his nephrograms with bilateral perinephric stranding, findings consistent with a combination of cocaine-induced ATN, intrarenal vasculitis, and/or glomerular ischemia. The inhomogeneous cortical enhancement with multiple wedge-shaped areas of diminished enhancement, diffuse nature of the nephrographic abnormality, and lack of other renal vascular or collecting system pathologies were important clues to the correct diagnosis. Although an episode of severe hypotension could have also produced the imaging findings, this was not consistent with the patient's clinical presentation and would have resulted in additional extrarenal manifestations, such as a "slit-like IVC" and hyperenhancing bowel, findings that were not present in this case. Similarly, if the patient had bilateral pyelonephritis, he should have had additional signs and laboratory evidence of active infection. A creatinine phosphokinase level was not obtained, but there was no suspicion for rhabdomyolysis due to the normal urinalysis and lack of myalgias. In many case reports of cocaine-related AKI, renal biopsy has been used for a definitive diagnosis. However, this is typically performed only in patients with systemic symptoms, abnormal urinalyses, and more pronounced creatinine elevations [4,5]. For this patient with a clear temporal relationship between his symptoms and his cocaine use, and with only mild AKI that quickly improved following IV fluids, the risks of renal biopsy were not felt to be justified.

Conclusion

Cocaine-induced nephropathy is a rare but important cause of abnormal nephrograms at imaging and should be considered in the differential diagnosis when bilateral mottled and/or straited nephrograms are seen in a patient with a recent history of cocaine use.

Patient Consent Statement

In lieu of a patient consent form, note that this case report is free of all 18 types of HIPAA identifiers. The information reported was obtained during a study that was granted a waiver of HIPAA authorization by our institutional review board.

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