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Renal Infarct: Any suggestion – CIRI?

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Abstract
Acute onset flank pain can have various causes. Renal infarction is one such cause. Renal infarcts have multiple causes. Cocaine induced renal infarct (CIRI) is one rare cause of renal infarct. A case of CIRI is presented with review of literature.

Key words: *Flank pain, renal infarct, cocaine*

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I. Introduction

Acute renal infarction is a well-known, although relatively less common cause of flank pain. We present a case of a 35-year old man, complaining of acute severe left flank pain, resistant to common analgesic therapy, who was diagnosed of segmental renal infarctions.

Learning objective:

We report a case of acute renal infarction presenting with abdominal pain. The symptoms were initially thought to be due to renal stone or diverticulitis. It was later concluded that the pain was due to renal infarctions. Detailed history could lead to proper diagnosis. This patient was diagnosed to have Cocaine induced renal infarct (CIRI). However, cardiac examination and ECG should be part of initial evaluation of abdominal pain and renal infarction and other vaso-occlusive events should always be on the list of differential diagnosis.

II. Case:

35 years old male presented to emergency department with excruciating left flank pain of few hours duration. There was no fever, nausea, vomiting, loose motions or past history of renal calculus disease. Basic metabolic profile done in the emergency department was within normal limits. An ultrasound examination of abdomen revealed hypo-echoic area with patchy absence of perfusion on lower pole of left kidney. A post contrast CT scan of abdomen showed two wedge-shaped parenchymal defects involving both the cortex and medulla and extending to the capsular surface (Figure 1, 2). With the diagnosis of acute renal infarct, he was hospitalized for further evaluation and management. Traumatic cause was ruled out as per history, and renal artery occlusion, renal vein thrombosis and aortic dissection were ruled from the CT scan. Thrombo-embolism from heart was ruled out by 2 dimensional echocardiography, however there were few calcific plaques in the in the infra renal aorta on CT scan. Patient was asked for any drug abuse. Except for consumption of ethanol for 5 days every week, he initially denied indulging in substance abuse. Serologic tests for hepatitis B and C viruses, human immunodeficiency virus, antinuclear antibody, and rheumatoid factor were negative. Serum complements, C3 and C4, were within the normal range. Hemoglobin electrophoresis, chest X-ray, electrocardiogram, were unremarkable. Investigations for pro-thrombotic state, including anti-phospholipid antibody IgM and IgG, anti-cardiolipin antibody IgM and IgG, Protein C, Protein S, Anti thrombin III level and Factor V Leiden mutation were all within normal limits. By this time, it was day 4 of admission and severe pain persisted and he developed high grade intermittent fever with chills. CT scan of chest, abdomen and pelvis were already done and had not shown any focus of infection. Two sets of blood cultures were sent (aerobic and anaerobic) along with urine culture, and patient was started on Injection Meropenem. All cultures were negative, and antibiotics were stopped subsequently. On day 5, he was asked about using cocaine prior to admission and he confessed having used it through nasal route with his friend who came to visit him from USA. A urine sample for drug abuse screen was sent immediately, which came negative. However, in view of absence of any other positive test and history of cocaine use prior to onset of severe left flank pain, it was highly suggestive to conclude cause and effect relationship, and hence a diagnosis of CIRI was made.

DTPA renogram showed GFR of 35.48 ml/min on left side and 54.38 ml/min on right side, with delayed aorto-renal transit time on left side with non-uniform tracer uptake, suggesting ischemic areas (Figure 2). On the basis of renogram findings, patient was subjected to renal angiography, which revealed normal left renal artery (Figure 3). His fever and pain had settled by day 7 and he was discharged with normal renal function and blood pressure, but advised to monitor blood pressure regularly and do a follow up DTPA renogram to assess differential GFR.

III. Discussion:

Although major toxic effects of cocaine such as myocardial ischemia, cerebrovascular accidents, mesenteric ischemia and placental infarcts have been well documented in the literature, renal infarction as a complication of cocaine toxicity is rare. In this article, we will attempt to raise awareness of this under reported entity by discussing its pathophysiology, clinical presentation and the current treatment modalities.

Patients presenting to the emergency room with severe colicky pain need immediate diagnosis and pain relief. Renal infarction is underdiagnosed because of the similarity of its presentation to other renal pathology. Left flank pain could be due to renal pathology or due to gastrointestinal causes (diverticulitis, pancreatitis etc.) Renal causes include renal parenchymal (pyelonephritis, abscess, infarction, venous obstruction, arterial dissection, tumor etc.) or non-parenchymal causes (nephrolithiasis, stricture, extrinsic compression, bladder outlet obstruction, intraluminal obstruction). Renal infarction is relatively a rare cause for flank pain, and CIRI is rare. The majority of patients suffering of renal infarction have cardiac diseases, and renal infarction is usually due to acute embolic occlusion of an end-artery vessel. Atrial fibrillation, valvular heart disease, dilated cardiomyopathy, and myocardial infarction are established risk factors for renal infarction[1]. Other causes of renal infarction include hereditary and acquired clotting factor diathesis, vessel anomaly, trauma, cocaine, mucormycosis or idiopathic[2].

It was only when we failed to establish the cause of renal infarct, by ruling out cardiac cause, vasculitis and pro-thrombotic state in this case that we thought of CIRI as a possibility. However, it was day 5 after patient having consumed cocaine, and the urine sample failed to detect cocaine. After last use, cocaine or its metabolites typically can show up on a blood or saliva test for up to 2 days, a urine test for up to 3 days, and a hair test for months to years. A heavy user can test positive on a urine test for up to 2 weeks. Since this patient was not a heavy user, cocaine had disappeared from urine by the time it was tested. The plasma half-life of cocaine is approximately 45 to 90 minutes. Cocaine lasts for 12 hours in plasma. The elimination of cocaine is predominantly controlled by its biotransformation since its renal clearance is only 27 mL/min[3]. But the diagnosis of CIRI was made based on history, clinical examination and course, and radiological findings. We did not do hair test for cocaine. Fever was also considered as part of cocaine toxicity, as all the cultures were negative, and CT scan of chest, abdomen and pelvis did not reveal focus of sepsis. Till date, there are only 15 cases reported on cocaine induced renal infarction [4].

IV. Cocaine:

Cocaine, also known as coke, is a strong stimulant most frequently used as a recreational drug, can pose many serious health risk to users. Cocaine abuse and addiction continues to be a problem that plagues the entire world. It is a potent activator of the sympathetic nervous system leading to intense vasoconstriction, endothelial dysfunction, oxidative stress, platelet activation and decrease in prostaglandins E2 and prostacyclin[5]. Even small doses of cocaine taken via intranasal route have been associated with vasoconstriction. Cocaine can lead to widespread systemic adverse effects such as stroke, myocardial infarction, arterial dissection, vascular thrombosis and rhabdomyolysis.

Cocaine is a sympathomimetic and acts by blocking the uptake of serotonin, norepinephrine and dopamine in presynaptic nerve terminals. It blocks voltage-specific sodium channels which impart a local anesthetic property[6].

Cocaine (benzoyl methylecgonine) is available in two forms: cocaine hydrochloride and the alkaloid cocaine (freebase/crack), which is made by alkanizing the salt, followed by extraction with non-polar solvents. Both forms are well absorbed through most mucous membranes. The pathways for the biotransformation involve plasma and liver cholinesterases that produce benzoyl and ethyl methylecgonine which are water-soluble metabolites excreted in urine[7].

V. Cocaine and kidney:

In human and rat kidneys, cocaine has been associated with glomerular, tubular, vascular and interstitial injury. It is not uncommon to diagnose cocaine-related acute kidney injury (AKI), (due to rhabdomyolysis, vasculitis, infarction, thrombotic microangiopathy and malignant hypertension) and chronic kidney disease[8]. Pathophysiology of renal damage in cocaine users is multifactorial, and involves renal hemodynamic changes, alterations in glomerular matrix synthesis, degradation and oxidative stress, and possible

induction of renal atherogenesis. Cocaine-induced renal damage is well-documented in the literature; however, renal infarction due to cocaine is not a well-known entity[9]. Its pathophysiology includes vasoconstriction and thrombosis mediated by cocaine-induced stimulation of platelet aggregation and thromboxane synthesis. Acute aortic thrombosis, renal artery thrombosis and even dissection in association with cocaine have been described.

It has been postulated that the right kidney was more prone to ischaemia. Left kidney represents an extremely unusual site of cocaine-related renal infarction. The reason for right kidney affection due to CIRI more than left kidney was elegantly explained based on fluid flows as per Poiseuille Equations[9].

There is no consensus on the treatment of renal infarction due to cocaine use. Prior treatment modalities in the literature range from no treatment to anticoagulation, thrombolytic use, aspirin therapy and surgical nephrectomy. Given that no evidence of underlying coagulopathy or thromboembolic events were identified in our patient, we still opted to manage him with low molecular weight heparin followed by oral anticoagulation, (may be for 6 months) besides supportive therapy and pain management. He was successfully discharged on hospital day seven with a serum creatinine of 1.2 mg/dl.

VI. Conclusion

Renal infarction is a rare cause of acute abdominal pain. Incidence is rare and numbers vary according to different studies (0.004-0.007%) and should pay attention to the development of AKI and CKD after renal infarction and follow patients over a long term[10].

It has to be suspected and managed appropriately, especially in patients with risk factors like cardiac arrhythmias (specifically atrial fibrillation). It has to be on the differential diagnosis of the admitting physician for patient presenting with acute onset of severe pain. Proper history can lead to diagnosis of vaso-occlusive disorder like in the present case where the diagnosis of CIRI could be arrived at.

Conflict of Interest: None.

Figure 1 and 2: CT scan showing wedge shaped infarcts in left kidney

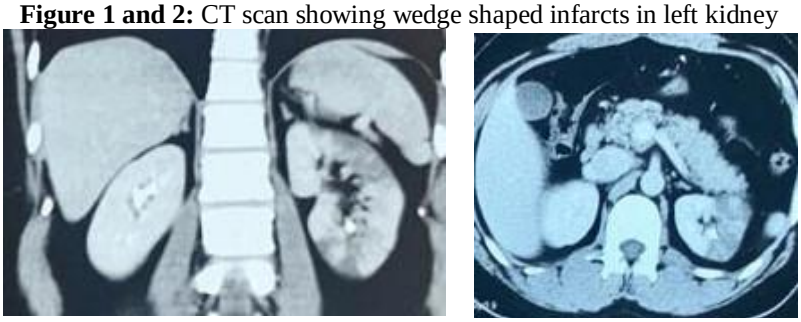


Figure 2: DTPA Renogram showing reduced perfusion and function of left kidney

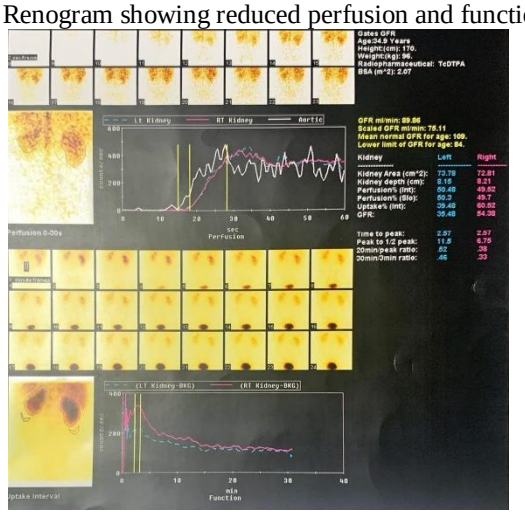
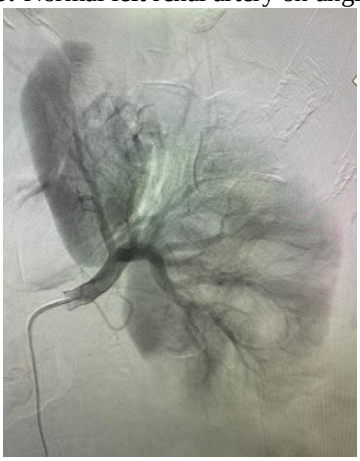


Figure 3: Normal left renal artery on angiography



References:

[1]. Blakely P, Cosby RL, McDonald BR. Nephritic urinary sediment in embolic renal infarction. Clin Nephrol 1994;6:401-403
[2]. Hritani A, Jo-Hoy F, Siddiqui F, Baldeo C, Poenariu A. Acute Renal Infarction Pathogenesis and Atrial Fibrillation: Case Report and Literature Review. World Journal of Nephrology and Urology, 2016;5(1):11-15
[3]. Inaba T: Cocaine: Pharmacokinetics and biotransformation in man. Can J Physiol Pharmacol 1989;67:1154-1157
[4]. Hirachan P, Agarwal R, Wagner B. Cocaine: A Rare Cause of Left-Sided Renal Infarction. J Investig Med High Impact Case Rep. 2015 Mar 16;3(1):2324709615574907. doi: 10.1177/2324709615574907.
[5]. Goel N, Pullman JM, Coco M. Cocaine and kidney injury: a kaleidoscope of pathology. Clin Kidney J. 2014;7(6):513-517
[6]. Schwartz BG, Rezkalla S, Kloner RA. Cardiovascular effects of cocaine. Circulation. 2010;122:2558–2569.
[7]. Chow MJ, Ambre JJ, Ruo TI, Atkinson AJ Jr, Bowsher DJ, Fischman MW. Kinetics of cocaine distribution, elimination, and chronotropic effects. Clin Pharmacol Ther. 1985; 38(3):318-24
[8]. Riezzo I, Fiore C, De Carlo D, Pascale N, Neri M, Turillazzi E, Fineschi V. Side effects of cocaine abuse: multiorgan toxicity and pathological consequences. Curr Med Chem. 2012; 19(33):5624-46
[9]. Bemanian S, Motallebi M, Nosrati SM. Cocaine-induced renal infarction: report of a case and review of the literature. BMC Nephrol. 2005;6:10. Published 2005 Sep 22. doi:10.1186/1471-2369-6-10
[10]. Kwon J, H, Oh B, J, Ha S, O, Kim D, Y, Do H, H: Renal Complications in Patients with Renal Infarction: Prevalence and Risk Factors. Kidney Blood Press Res 2016;41:865-872

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