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CASE REPORT

Renal infarct: a rare disease due to a rare etiology

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Renal infarction is caused by profound hypoperfusion secondary to embolic/thrombotic occlusion of the renal artery or vasospasm of the renal artery. We present a case of a 54-year-old patient who presented with nausea, vomiting, and vague abdominal pain. He had frequent episodes of migraine headaches and he treated himself with as needed rizatriptan. CT scan of the abdomen showed renal cortical infarction. After extensive investigations, etiology of his renal infarct was deemed to be due to rizatriptan.

Keywords: *renal infarct*; *rizatriptan*; *vasospasm*; *morbidity*; *case report*

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Renal infarction is a rare condition which happens due to embolic/thrombotic occlusion of the renal artery or vasospasm of the renal artery. Bilateral renal infarcts present with acute kidney injury and oliguria/anuria. Unilateral renal infarct often goes unnoticed as it presents with flank pain and nausea with no abnormalities in urinalysis or creatinine. Triptans are well tolerated medications with known side effects of arteriolar vasospasm and end-organ ischemia.

Case report

We present a case of a 54-year-old patient with a 1-week history of nausea, vomiting, and vague abdominal pain. He had a history of recurrent migraines and was receiving daily topiramate for prophylaxis. The patient also used as needed rizatriptan for abortive therapy. Prior to hospitalization, the patient was taking two tablets of rizatriptan per week. He admitted to occasional marijuana use but denied use of tobacco and other illicit drugs such as cocaine. He reported no history of hypertension, diabetes, peripheral vascular disease, and personal or family history of hypercoagulable state. On examination, he appeared dehydrated with a pulse rate of 92 and blood pressure of 115/72 mm of Hg. Examination of all other systems was unremarkable except for tenderness at left costovertebral angle.

Initial investigations, including complete blood counts, basic metabolic panel, liver function tests, lipase and urinalysis, were all within normal limits. Urine drug screen was only positive for cannabinoids. CT scan of abdomen with contrast showed hyperechoic wedge-shaped shadow at the upper pole of left kidney suggestive of renal infarct (Fig. 1). PT and PTT were slightly elevated at 16.9 and 41 s, respectively. ESR and CRP were elevated at 33 and 6.79, respectively. LDH was found to be elevated at 910 U/L. Screening tests for hypercoagulable state and connective tissue disorders – factor V Leiden, homocysteine level, lupus anticoagulant, ANA, ANCA, and rheumatoid factor were all negative. Proteins C and S were within normal limits. EKG and cardiac monitoring for 72 h revealed normal sinus rhythm, and 2D echocardiogram did not show any intracardiac thrombus or valvular vegetations. Renal Doppler ultrasound ruled out renal artery stenosis. After extensive workup, it was deemed that his renal infarct was due to rizatriptan. He was managed conservatively and improved significantly during the course of his hospitalization. He was sent home in a stable condition with recommendations to stop rizatriptan upon discharge and avoid ergot derivatives as well.

Discussion

Renal infarction can be caused by thromboembolism due to atrial fibrillation, cardiac thrombus, aortic atherothrombi, and endocarditis. Atrial fibrillation was found to be the most common cause of this condition (1). Other reasons such as hypercoagulable states and hematological malignancies can also result in *in-situ* thrombosis (2). Non-embolic causes of renal infarct include renal

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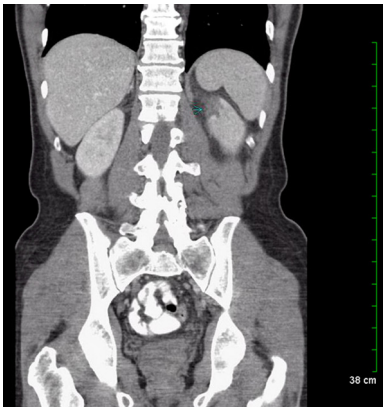

Fig. 1. CT Scan (with contrast): abdomen and pelvis showing left kidney upper pole infarct (pointed by blue arrow).

artery stenosis due to fibromuscular dysplasia, dissection, or vasospasm (3). Drugs like cocaine, tacrolimus, and ergot derivatives that cause vasospasm have also been implicated (4, 5).

Triptans are 5-hydroxytryptamine receptor 1B/1D (5HT-1B/1D) receptor agonists. Through these receptors, triptans cause vasoconstriction of the cerebral vessels thus reversing the abnormal vasodilation and relieving migraine headache (6). Triptans, due to their inherent property of vasoconstriction, can result in myocardial infarction, cerebrovascular ischemia, mesenteric ischemia, spinal cord ischemia, or splenic infarct due to arterial spasm (7–11). A review of literature revealed two cases reported of renal infarction due to triptans (12). We believe the renal infarction in our patient was caused by rizatriptan. The close temporal relationship between the use of the medication and the occurrence of symptoms support this hypothesis. Studies have established the role of 5HT-1 receptors, particularly 5HT-1D receptors in the constriction of renal arteries in rabbits (13). It is emphasized to remember end-organ ischemia as a side effect of triptans, which could add considerable morbidity.

Conclusion

The aim of this report is to stress the potential adverse effects of triptans. Because triptans are commonly used medications, it is important to remember the vasoconstrictive properties and be vigilant about prescribing to patients with history of hypercoagulable/atherothrombotic diseases. We emphasize renal infarction as a rare but serious side effect with triptans. As more cases are recognized and reported, it will be possible to establish a dose–response relationship.

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