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Bilateral Renal Infarctions During the Use of Sumatriptan

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INTRODUCTION

Triptans are effective as acute abortive therapy for migraine and cluster headaches by targeting the serotonin 5-$HT_{1B/1D}$ receptors located in the cerebral, coronary, and peripheral arteries. This class of medications inhibits the release of vasoactive peptides and promotes vasoconstriction, combating the pathologic vasodilatation contributing to migraine headaches.[1] Potential complications of these vasoconstrictive properties include myocardial infarction,[2,3] cerebrovascular accident,[4] ischemic colitis,[5] and spinal cord infarction.[6] We present a case of bilateral renal infarctions associated with sumatriptan use.

CASE PRESENTATION

A 45-year-old Caucasian woman with a history of migraine headaches presented to the emergency department with acute left-sided abdominal and lower back pain. She denied symptoms of dysuria or hematuria, and did not report any new medication use. Home medications included sumatriptan 25 mg as needed for migraine headaches, which were used shortly prior to presentation, and Mirena, a intrauterine device. She had no personal or family history of a hypercoagulable state. She was a former smoker but denied the use of illicit drugs, including cocaine. She was diagnosed with presumed pyelonephritis and was discharged with trimethoprim–sulfamethoxazole, a nonsteroidal anti-inflammatory drug, and a proton pump inhibitor.

One week later, she re-presented to the emergency department with persistent left-sided abdominal pain. On arrival, her temperature was 98.8°F, blood pressure was 145/82 mm Hg, heart rate was 70 bpm and regular, and her left upper quadrant and epigastrium were tender to palpation. Laboratory tests were as follows: serum creatinine 0.97 mg/dl (reference range, 0.57–1.00), blood urea nitrogen 8 mg/dl (reference range, 6–24), hemoglobin 12.1 g/dl (reference range, 11.1–15.9), prothrombin time 11.4 seconds (reference range, 12.2–14.2), partial thromboplastin time 31 seconds (reference range, 20.8–34.4), and lactate dehydrogenase 209 IU/l (reference range, 90–190 IU/l), and urinalysis was negative for blood and leukocyte esterase. A computed tomography (CT) examination of the abdomen/pelvis with intravenous contrast showed a patchy wedge-shaped area of decreased enhancement in the lower pole of the left kidney, concerning for a renal infarct (Figure 1a), with thrombosis of a segmental artery to this region (Figure 1b). A transthoracic echocardiogram demonstrated no thrombus or patent foramen ovale, and an electrocardiogram showed normal sinus rhythm. She was discharged with subcutaneous enoxaparin as a bridge to warfarin for management of an acute renal infarction. During her hospitalization, she was treated for a migraine headache with a 1-time dose of sumatriptan.

Five days after discharge, the patient re-presented to the emergency department with nausea, vomiting, and epigastric and right-sided abdominal pain. She denied missing any doses of enoxaparin or oral warfarin, and continued to use sumatriptan for persistent headaches. Repeat urinalysis showed no white blood cells, leukocyte esterase, or blood. Repeat computed tomography showed wedge-shaped areas of hypoattenuation in both kidneys (Figure 2a), consistent with an evolving infarct of the left kidney and a new infarct of the right kidney with thrombosis of a segmental right renal artery branch (Figure 2b). Follow-up magnetic resonance angiography revealed normal aortoiliac, mesenteric, and main renal arteries, with no evidence of dissection or vasculopathy. She was placed on an intravenous

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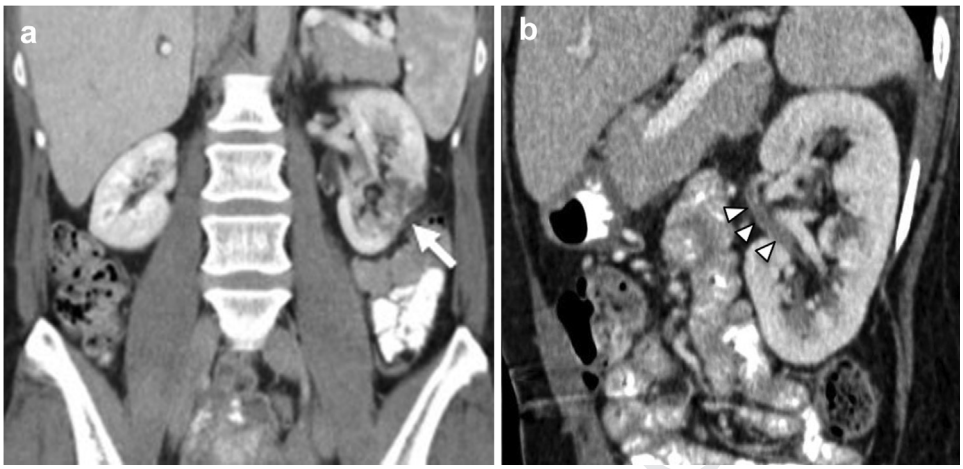

Figure 1. (a) Coronal multiplanar reformatted (MPR) image from a computed tomography examination with i.v. contrast demonstrates a peripheral, wedge-shaped area of hypoenhancement of the lower pole of the left kidney consistent with an infarct (arrow). (b) Oblique MPR image shows thrombosis of segmental artery to the infarcted region (arrowheads).

heparin drip with bridge to dabigatran. A transesophageal echocardiogram showed no cardiac thrombus or valvular vegetations. A hypercoagulable workup revealed a mildly elevated anti-cardiolipin IgM antibody (22 units; reference range, 0−12 units). Given concern for contributors to a hypercoagulable state, her intrauterine device was removed. During her hospitalization, she was treated with 2 doses of sumatriptan for migraine headaches.

Three months later, while still on dabigatran, a hypercoagulable workup was repeated. Again, there was only a mildly positive finding with anti-cardiolipin IgM antibody at 13.5 units. Anti−β2 glycoprotein antibody measurement was within normal limits. Given the negative workup results for cardiovascular, embolic, and hypercoagulable states, her bilateral renal infarctions were attributed to sumatriptan, which was discontinued by her nephrologist. Follow-up renal function after 5 months has remained stable, and she has had no recurrence of symptoms.

DISCUSSION

Renal infarction is a rare diagnosis, resulting from compromised blood flow to the kidney, with the most common causes including thromboembolic etiologies (atrial fibrillation or infective endocarditis), renal artery dissection, or hypercoagulable states.[7] Less common etiologies include fibromuscular dysplasia[8] and vasospasm related to cocaine[9,10] or ergotamine[11] use. In an autopsy series of 14,411 bodies, the incidence of renal infarction was 1.4%.[12] In a retrospective study involving 18,287 patients, idiopathic or spontaneous renal infarction was noted in 0.3% of patients.[13] The incidence of recurrent renal infarction is uncommon. We report a case of recurrent, bilateral renal infarcts

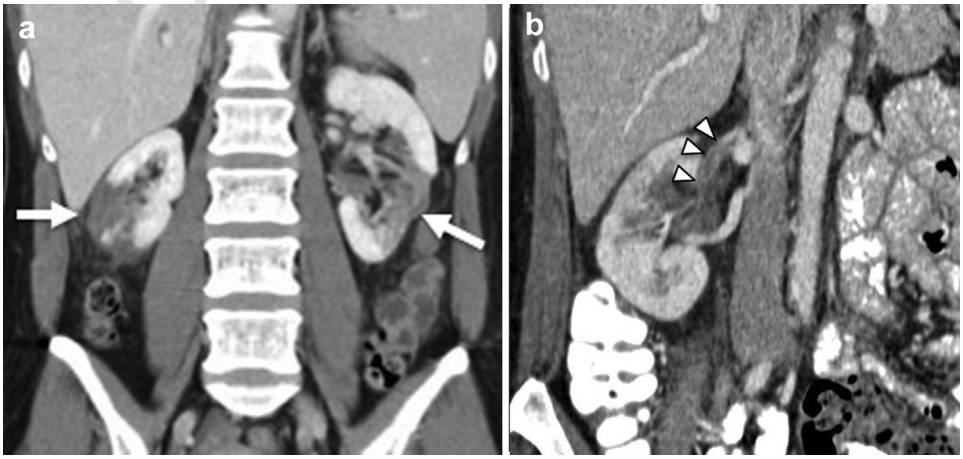

Figure 2. (a) Coronal multiplanar reformatted (MPR) image from a computed tomogram with i.v. contrast identifying areas of infarction in both kidneys (arrows). (b) Oblique MPR image shows thrombosis of segmental artery to the newly infarcted region in the right kidney (arrowheads).

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