A Case Of Acute Thromboembolic Renal Infarction Associated With Paroxysmal Atrial Fibrillation

Macit Kalcik, MD; Mahmut Yesin, MD; Lutfi Ocal, MD; Taylan Akgun, MD; Nursen Keles, MD; Mustafa Ozan Gursoy, MD; Mehmet Ozkan, MD

Abstract

Infarction of the kidney is an uncommon condition that can result from obstruction or decrease of renal arterial flow. The diagnosis is often delayed because it can mimic many other pathologic states, including pyelonephritis, renal colic, acute abdomen, pancreatitis and more. A high index of suspicion is important for prompt diagnosis. We describe a 20-year-old man presented with abdominal and right flank pain and hematuria. A computed tomography scan with intravenous contrast showed partial infarction of right renal parenchyma and selective renal angiography showed complete occlusion of the right renal artery which was also supplied by an accessory renal artery. Electrocardiography showed normal sinus rhythm. Transthoracic and transesophageal echocardiographic findings were unremarkable except for mild spontaneous echo contrast (SEC) in the left atrial appendage. Subsequent 48-hour holter monitor revealed frequent premature atrial complexes and paroxysmal atrial fibrillation (PAF). Development of thromboembolic renal infarction was attributed to the presence of PAF and concurrent SEC in the left atrial appendage (LAA). Low molecular weight heparin (LMWH) was followed by oral anticoagulant and an electrophysiologic study was planned for the management of PAF after 4 weeks of anticoagulation.

Introduction

Acute renal infarction is an uncommon disease which frequently results in irreversible renal parenchymal damage. High index of suspicion is required for early diagnosis, as timely intervention may prevent loss of kidney function. Common etiologies of renal infarction include thromboembolism, paradoxical embolism, severe blunt trauma, vasculitis, malignancy, endocarditis and cocaine abuse. Major risk factors for renal thromboembolism are atrial fibrillation, previous embolism, hypercoagulopathy, ischemic, congenital and valvular heart disease. Herein, we describe a young patient who presented with right renal thromboembolism without any risk factors.

Case Presentation

A 20-year-old non-diabetic man presented to our emergency department with abdominal and right flank pain that were persistent for 2 days. He complained of urinary symptoms including dysuria, hematuria and difficulty in micturition. His blood pressure, heart rate, temperature and oxygen saturation were within normal limits. Electrocardiography showed normal sinus rhythm. Physical examination was unremarkable except for right costovertebral angle tenderness. Laboratory findings showed a white blood cell count of 10200/µL (74% neutrophils), serum creatinine of 0.9 mg/dL, BUN of 12 mg/dL, serum lactate dehydrogenase (LDH) level of 890 U/dL (0-248 U/dL), international normalized ratio (INR) of 0.9. Urinalysis revealed 3 to 5 red blood cells and 1 to 3 white blood cells per high-power field with no proteinuria or casts. Chest and abdominal X-ray did not provide anything remarkable for the diagnosis. Abdominal ultrasonography was negative for urinary calculi and computerized tomographic (CT) scan with intravenous contrast injection discovered several heterogeneous areas with decreased enhancement of the right kidney which was compatible with renal infarction (Figure 1). Subsequently, he underwent selective renal angiography which revealed normal left renal artery (Figure 2B), but an obstructing thrombus was detected in the middle segment of right renal artery. The suprarenal artery arising from the proximal segment of right renal artery and an accessory right renal artery arising from the aorta were also visualized. (Figure 2A) Transthoracic echocardiography provided normal left and right ventricular systolic functions without any valvular heart disease. Transesophageal echocardiography was performed for suspected any paradoxical embolism in which contrast study with valsalva maneuver was negative for patent foramen ovale(Figure 3A). There was no intracardiac thrombus but a mild SEC in the LAA. (Figure 2B) There was no evidence to suggest a hypercoagulopathic condition according to blood tests. Frequent premature atrial complexes were detected (825) during 24-hours Holter monitoring and 2 PAF episodes were recorded at 48 hour...
renal embolism are characterized by a variety of symptoms including abdominal and flank pain, hematuria, proteinuria, and fever. Acute segmental renal infarction is an even more problematic diagnosis. In evaluating partial renal infarction, a strong clinical suspicion is necessary. A history of dysrhythmia or other cardiac disease, the presence of abdominal or flank pain, fever with an elevated white cell count, and an elevated LDH are clinically significant, and their presence should alert the clinician to the possibility of renal infarction.

Computed tomography plays an important role in evaluation and management of primary renovascular disease. Nonenhanced CT is useful for demonstrating renal hemorrhage, renal parenchymal or vascular calcifications and masses. Contrast-enhanced CT is essential to identify renal abnormalities resulting from the vascular process (eg, renal infarcts, arteriovenous communications). If unenhanced CT is negative for urinary calculi, contrast-enhanced CT of the abdomen may also be needed for early diagnosis of renal infarction or alternatively, to minimize radiation uptake, a combination of ultrasound followed by contrast-enhanced CT might be the initial diagnostic regimen as we did in our case. CT which is noninvasive, sensitive, and capable of identifying segmental lesions appears to be the best method for diagnosis of renal infarction.

In terms of renal infarction, restoration of renal blood flow is important in order to avoid subsequent renal failure. Surgical intervention, percutaneous intervention, thrombolytic therapy and anticoagulant therapy are various methods for management of renal arterial embolism. Since, laboratory findings showed normal renal functions, symptoms started a few days ago, CT scan revealed limited infarct areas and renal angiography showed an accessory right renal artery, we preferred conservative therapy over a percutaneous intervention or thrombolytic therapy in our case.

Discussion

Acute renal artery thromboembolism is an infrequent but important cause of severe renal injury. However, due to its rarity and nonspecific presentation diagnosis is often delayed and occasionally missed. It is primarily associated with cardiac disease and arrhythmia in which atrial fibrillation being the most common. Clinical presentations of

Figure 1: Four different slices of abdominal CT with intravenous contrast injection showing several heterogeneous areas with markedly decreased contrast enhancement of the right kidney consistent with an acute renal infarction and normal contrast uptake by the left kidney. No dilatation of the right ureter or renal pelvis is noted.

Figure 2: Selective right renal angiography (A) revealing right renal artery obstructed by a thrombus in the middle segment. (Arrow number 1) An accessory right renal artery arising from the aorta is supplying inferior pole of the right kidney. (Arrow number 2) The right suprarenal artery arising from the proximal segment of the right renal artery is supplying the right suprarenal gland with the superior pole of the right kidney. (Arrow number 3) Selective left renal angiography (B) revealing normal left renal artery.
heart diseases such as atrial septal defect or patent foramen ovale which may be a cause of paradoxical embolism. Furthermore, any infarction in other organs such as the spleen, bowel and liver should be investigated.

In cases of cryptogenic embolism in a patient with sinusial rhythm and absence of cardiac abnormalities, concurrence of possible PAF should be kept in mind. Holter monitoring may allow identification of occult PAF in patients with cryptogenic embolism. However, optimal duration of monitoring remains unclear. A prolonged holter monitoring may increase the detection rate of patients requiring anticoagulation and may be able to reduce the risk of recurrent embolism. In our case, only supraventricular ectopic activities were detected in the first 24-hours monitorization, but 2 PAF episodes were recorded in the second day. Weber-Krüger et al. reported that excessive supraventricular ectopic activities may be an indicator of PAF. If frequent supraventricular ectopic activities are detected during 24-hour Holter monitoring, the duration of Holter follow-up may be prolonged up to 48 hours in order to detect occult PAFs in cryptogenic embolism patients. Anticoagulation with LMWH should be followed by oral anticoagulation with warfarin sodium in patients with PAF complicated with thromboembolism. Electrophysiologic study or ablation therapy should be delayed after 4 weeks of effective anticoagulation.

**Conclusion:**

PAF should be borne in mind as an etiologic factor of cryptogenic embolism if other causes of intracardiac origin of thromboembolism are excluded.

**References:**