CASE REPORTS/CLINICAL VIGNETTES

Renal Artery Embolism

A Case Report and Review

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Renal artery embolism was first described in 1940, but it is only recently becoming recognized as a clinically significant entity. Although relatively uncommon, it is clearly responsible for considerable morbidity in patients who experience it. The pathogenesis is typically related to cardiac thrombus formation with subsequent embolization, although other etiologies have been described. The authors present a case report followed by a review of the literature to highlight the clinical characteristics of this phenomena. Presentation, diagnostics, and treatment options will be reviewed with the aim of increasing awareness of renal artery embolism. As clinicians become more familiar with this condition, they will be more likely to consider it as a possible diagnosis in patients with a typical presentation. This will hopefully lead to improved care through prompt diagnosis and treatment, particularly as one treatment option may be time sensitive.

KEY WORDS: renal artery embolism; renal infarction; renal artery obstruction; thromboembolic disease.

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Acute renal artery embolism (RAE) has historically been a poorly described entity. In 1940, Hoxie and Coggin reported a postmortem incidence of 1.4 percent and described a few cases antemortem.¹ Lessman et al.² described 17 patients with renal artery embolism in 1978. A number of case reports of renal infarction secondary to embolus were subsequently described, but it was not until 1999 that another case series was published. In that report, Domanovits et al.³ described the clinical characteristics of 17 patients with renal infarction. The majority of these patients were at increased risk of thromboembolic events. Korzets et al.⁴ followed with a similar series in 2002. Most recently, Hazanov et al.⁵ described 44 cases of renal infarction in the setting of atrial fibrillation in 2004. Given its emergence as a

clinically significant entity, we present a review of the literature on renal artery embolism and highlight its clinical characteristics. We precede this discussion with a case report to illustrate these characteristics. Our goal is to improve patient care by heightening awareness of this uncommon condition. As more clinicians become familiar with RAE, more will include it in their differential when presented with the appropriate clinical scenario. This will hopefully lead to timely testing, diagnosis, and treatment.

CASE REPORT

An 85-year-old white male presented to our Emergency Department complaining of a 1-day history of right upper quadrant and right flank pain. He stated that the pain started suddenly and seemed to originate deep within his right upper quadrant. It extended laterally into his flank. He graded the pain as “severe” and described it as sharp and constant. It had not changed in quality or intensity since its onset. He denied any exacerbating factors such as oral intake or movement. There were no relieving factors and he had not taken any medications for relief. Review of systems was significant for nausea with emesis. He denied recent fevers, trauma, dysuria, hematuria, or change in bowel pattern. He had a past medical history of coronary artery disease, ischemic cardiomyopathy, and chronic atrial fibrillation without any prior history of thromboembolic disease. His medications included aspirin, clopidogrel, lisinopril, metoprolol, and digoxin. He was not on warfarin because of recurrent episodes of epistaxis while anticoagulated. On physical examination, he had a temperature of 35.5°C. Blood pressure was 126/88 mmHg, with a heart rate of 86 beats per minute. Respiratory rate was 22 per minute, with an oxygen saturation of 92% on room air. He appeared restless and in moderate distress secondary to pain. There was an irregularly irregular heart rhythm and tenderness on the right upper and lower quadrants. No CVA tenderness or peritoneal signs were elicited. Screening laboratories, including a complete blood count and chemistry panel, were within normal limits. Urine analysis was positive for microscopic hematuria and an EKG showed atrial fibrillation with a ventricular rate of 86. An enhanced computed tomography (CT) of the abdomen was performed, which revealed a delayed enhancement of the right kidney with an infarction of the upper pole (Fig. 1). Serum LDH was checked and found to be elevated at 1,509 U/L (reference range 307–658 U/L). A diagnosis of acute renal artery embolism was made and interventional radiology was emergently consulted for possible intraarterial thrombolysis. They deferred intervention as the duration of symptoms suggested a prolonged arterial occlusion. The patient was started on intravenous heparin along

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Figure 1. Axial (a) and Coronal (b) enhanced CT images through the level of the right kidney demonstrate absent enhancement of a segment of renal parenchyma in the upper pole. Ill-defined border and perinephric stranding suggest this infarction is acute

with oral warfarin. The following day, his pain had subsided. Repeat laboratories revealed a leukocytosis with a white count of 22.8 k/mcL (reference range 4.4–11.0 k/mcL) and mild renal insufficiency with a serum creatinine of 1.6 mg/dL (reference range 0.6–1.3 mg/dL). A repeat serum LDH was not drawn so no trend could be identified. A transthoracic echocardiogram revealed global hypokinesis with an estimated ejection fraction of 10% and severe mitral regurgitation. No intracardiac thrombus was visualized. He did well for the remainder of his hospital stay without evidence of further embolic events. His serum creatinine peaked at 2.1 mg/dL, but returned to within normal limits by hospital day 8. That same day, he was transferred to a long-term acute care facility. He died 3 days after transfer because of complications of heart failure.

DISCUSSION

Renal artery embolism (RAE) involves the acute occlusion of arterial blood flow to a segment of the renal parenchyma because of an embolus from a distant source. The postmortem incidence of RAE has been reported as 1.4%, although its clinically

Table 1. Risk Factors for Renal Artery Embolism*

Risk Factor	Percent of Patients
Atrial Fibrillation†	62.2
Ischemic Heart Disease	31.1
Cardiomyopathy	8.9
Mitral Valve Disease	26.7
Aortic Valve Disease	4.4
Previous Thromboembolism	26.7

*Compiled using data from Lessman et al.,² Domanovits et al.,³ Korzets et al.,⁴ and Hazanov et al.⁵

† Data from Hazanov et al.⁵ is excluded because AF was an inclusion criteria.

Table 2. Clinical Characteristics of Patients with Renal Artery Embolism*

Characteristic	Percent of Patients
Age (Mean)	66.7
Female	51.7
Pain	91.1
Vomiting	41.1
Fever	48.5
Hematuria	72.3
Leukocytosis	85.6
LDH > 400 U/L	90.8
Creatinine > 1.5 mg/dL	53.9

*Compiled using data from Lessman et al.,² Domanovits et al.,³ Korzets et al.,⁴ and Hazanov et al.⁵

significant incidence is in the order of 0.007% of hospitalized patients or 6.1 patients per million.⁴ It is typically seen in patients with atrial fibrillation. Other significant risk factors include ischemic heart disease, cardiomyopathy, and valvular disease. These conditions predispose patients to cardiac thrombus formation. RAE develops as the cardiac thrombus embolizes peripherally. A history of prior embolic disease is not uncommon in these patients^{2–5} (Table 1). Noncardiac sources of renal artery emboli include thrombi on unstable aortic plaques, valvular vegetations in infective endocarditis, and paradoxical emboli in the setting of a patent foramen ovale.^{6–10}

The clinical presentation of RAE is variable, but most patients are in the sixth decade of life and complain of the acute onset of abdominal, flank, or back pain. The pain is typically constant and without significant modifying factors. Vomiting and fever are common and have been present in about half of the reported cases.^{2–5} Blood pressure may be acutely elevated, and this is presumed to be through a renin-mediated mechanism.^{2,11} Signs of extrarenal embolism may also be present.

In terms of laboratory tests, a leukocytosis is usually present. Serum creatinine may be elevated, but whether this is a direct result of the renal infarction is unclear. Most patients are exposed to contrast during their diagnostic workup, placing them at risk for contrast nephropathy. Some patients have had evidence of renal insufficiency before contrast exposure, suggesting that infarction was the causative event.² Urine analysis may reveal microscopic hematuria, which is seen in approximately two thirds of patients.⁵ The absence of hematuria is associated with worse renal function. It is postulated that this reflects hypoperfusion of the infarcted area with resulting reduction of glomerular filtration and urine production.²

Several serum markers have been suggested as indicative of renal infarction.^{2,12,13} Elevations in alkaline phosphatase, fibrinogen, C-reactive protein (CRP), and aspartate aminotransferases (AST) are all associated with renal infarction, but these are inconsistently elevated.³ Serum lactate dehydrogenase

Table 3. Diagnostic Studies in Renal Artery Embolism*

Study	Sensitivity
Renal Angiogram	100.0
Excretion Urography	100.0
Radioisotope Renogram	95.7
Enhanced CT	80.8
Ultrasound	10.7

*Compiled using data from Lessman et al.,² Domanovits et al.,³ Korzets et al.,⁴ and Hazanov et al.⁵

Table 4. Outcomes in Renal Artery Embolism*

Outcome	Percent of Patients
Normal Renal Function	57.7
Mild Renal Impairment (creatinine < 2 mg/dL)	16.7
Moderate Renal Impairment (creatinine > 2 mg/dL)†	15.4
Severe Renal Impairment/ESRD	10.2
Death within 1 year‡	14.3
Embolitic Disease	50.0
Myocardial Infarction	25.0
Sepsis	25.0

*Compiled using data from Lessman et al.,² Domanovits et al.,³ Korzets et al.,⁴ and Hazanov et al.⁵

† Temporary HD required in 25%

‡ Severe renal dysfunction or ESRD in 58.3%

(LDH) appears to be the most sensitive marker as it has been elevated in most of the cases described. It has poor specificity, however, and other causes of serum LDH elevation must be ruled out. Urinary LDH may be a more specific measure. LDH is too large to be filtered by the kidney, and its excretion would be normal in extrarenal disorders. In renal infarction, there is a direct renal origin for the LDH resulting in increased excretion.¹³ Clinical data are largely lacking in regard to urinary LDH (Table 2).

A number of imaging modalities have been used to aid in the diagnosis of RAE and renal infarction. These include radioisotope renogram, excretion urography, renal angiogram, and contrast-enhanced CT (Table 3). Radioisotope renogram and excretion urography have high sensitivity, but these studies are rarely used today. Renal angiography demonstrates high sensitivity as well, but it is invasive and not employed as a first line study. The sensitivity of enhanced CT is relatively poor, but some of the data predates spiral and multidetector CT technology (MDCT). Other data are based on protocols with a reconstruction thickness of 10 mm.^{4,5} Enhanced CT with submillimeter resolution and current MDCT technology is expected to be more sensitive than that which is found in these studies.

Computed tomography and magnetic resonance (MR) are widely used in the evaluation of renal parenchyma in current practice. Renal infarction is easily demonstrated on enhanced CT and T1-weighted gadolinium-enhanced MR studies as a well-defined, peripheral focus of renal parenchyma that fails to enhance with contrast. Enhanced CT images may also demonstrate a preserved rim of peripheral cortical enhancement.^{14,15} Vascular abnormalities are rarely identified in acute renal infarct, but both CT and MR angiography (CTA and MRA) are well-established methods of evaluating the renal vasculature.¹⁵⁻¹⁷ An appropriate diagnostic strategy for a patient presenting with renal colic may involve obtaining an unenhanced CT to rule out nephrolithiasis followed by an enhanced study to look for infarction.^{5,18,19} If the patient cannot receive contrast because of allergy, a MR could be considered.

The standard treatment strategy for RAE includes anticoagulation with or without thrombolysis. Patients are typically anticoagulated with intravenous heparin and oral warfarin. This helps prevent further embolic events. Perfusion can be reestablished through thrombolysis or surgical revascularization. The former is generally favored as surgery has a higher mortality rate and does not result in better outcomes.²⁰ Both systemic and local intraarterial thrombolysis have been successful in produc-

ing clot lysis. Local infusion minimizes systemic bleeding and is the preferred approach.^{21,22} There are data that suggest thrombolysis does not improve outcomes once the ischemic tolerance of the kidney is exceeded. This is generally felt to be in the range of 180 minutes.²² Conflicting data suggest renal recovery can be accomplished after prolonged occlusion.^{23,24} In this setting, renal viability may be sustained through collateral circulation.²⁵ This has prompted some authors to suggest that blood flow should be reestablished regardless of the time frame.²⁶ There is no consensus regarding this issue, and the exact role of intraarterial thrombolysis in RAE is yet to be determined.

Despite the uncertainty regarding management, the renal outcomes associated with RAE remain favorable. Many patients do develop some degree of renal insufficiency during the acute episode, but a majority return to their baseline renal function. Even when renal function does not return to baseline, it is rarely impaired to the point of requiring hemodialysis. When hemodialysis is necessary, the mortality rate is quite significant. The death in these patients is typically caused by recurrent embolic disease or heart disease rather than renal complications²⁻⁵ (Table 4).

The literature on renal artery embolism remains sparse, although in recent years it has become an increasingly recognized entity. The few case series that have been reported have described the epidemiology and clinical characteristics of this diagnosis. These series have also commented on the utility of various diagnostics, including laboratory tests and imaging modalities. We have presented a review of these topics, as well as treatment options and historical outcomes. Our purpose has been to increase awareness of this condition so that clinicians will be more apt to consider it as a possible diagnosis. This will facilitate prompt diagnosis and treatment. Improved patient care will be the end result, particularly as one treatment option may be time sensitive.

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