Brief Report

Renal Emboli and Atrial Septal Aneurysm

Gerard David Spoelhof, MD, and Carol June Farchmin, MD
Duluth, Minnesota

A 53-year-old woman presented with flank pain and hematuria. She was suspected of having a renal stone but instead was found to have a segmental renal infarction. Further evaluation revealed an atrial septal aneurysm, which was presumed to be the source of an embolism. Renal emboli should be considered when more common causes of flank pain are excluded. Atrial septal aneurysm should be considered when embolic events occur without an evident source. Transesophageal echocardiography is the best test to diagnose atrial septal aneurysms.

Key words. Renal circulation; infarction; renal embolism; embolism; aneurysm. (*J Fam Pract 1996; 42:519-522*)

Severe flank pain and microhematuria are usually reliable signs of ureteral colic. The patient in this case taught us to consider another important diagnosis: renal infarction due to an embolism. These emboli usually arise from mural thrombi after myocardial infarction or as a result of atrial fibrillation.[1] Atrial septal aneurysms are also associated with embolic phenomena.[2] They should be considered in young adults with emboli who do not have atherosclerotic or valvular heart disease.

Case Report

A 53-year-old white woman presented to the emergency department with unrelenting pain of sudden onset in the right flank. There were no urinary or bowel symptoms. She had undergone surgery for a ruptured appendix 9 years earlier. She had quit a one-pack-per-day cigarette habit 9 years before the current hospitalization. There was no history of heart or vascular disease. She was taking conjugated estrogen, 0.625 mg, with continuous dose medroxyprogesterone, 2.5 mg.

She was afebrile and had tenderness over the right costovertebral angle. Aside from distress due to pain, results of her physical examination were normal. Urinalysis showed a 2+ positive dipstick test result for blood. Microscopic examination showed 3 to 5 red blood cells per high-power field. Her white blood cell count was 10,200 per mm^3 with a normal differential. An intravenous pyelogram performed in the emergency department was interpreted as normal, with an additional comment indicating that the collecting system on the right side was not as well opacified as the system on the left.

She was admitted to the hospital for analgesia and further evaluation. A blood chemistry profile showed elevations of aspartate aminotransferase at 121 IU/L (normal, 14 to 36) and lactate dehydrogenase (LDH) at 1957 IU/L (normal, 313 to 618). Blood urea nitrogen (BUN) and creatinine were within normal limits at 7 mg/dL and 0.9 mg/dL, respectively. A computed tomographic (CT) scan of the abdomen was performed on the first hospital day, demonstrating a lack of perfusion of the lower one third of the right kidney, consistent with infarction (Figure 1).

Intravenous heparin was administered after the CT scan was completed, and the pain gradually improved. A transesophageal echocardiogram was perfomed on the 2nd hospital day, demonstrating a floppy atrial septum with the appearance of an aneurysm (Figure 2). Right-to-left shunting through the atrial septum was demonstrated by sonicated bubbles. No thrombi were seen.

Ultrasonography of both lower extremities performed on the 3rd hospital day failed to show evidence of deep venous thrombosis. Arteriography perfomed on the 4th hospital day revealed filling defects in the inferior segmental branches of the right renal artery, presumed to be emboli (Figure 3). No other signs of vascular disease were noted.

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From Skyline Family Medical Center, Duluth, Minnesota. Requests for reprints should be addressed to G.D. Spoelhof, MD, 2900 Piedmont Ave, Duluth, MN 55803. E-mail: dspoelho@mail.d.umn.edu

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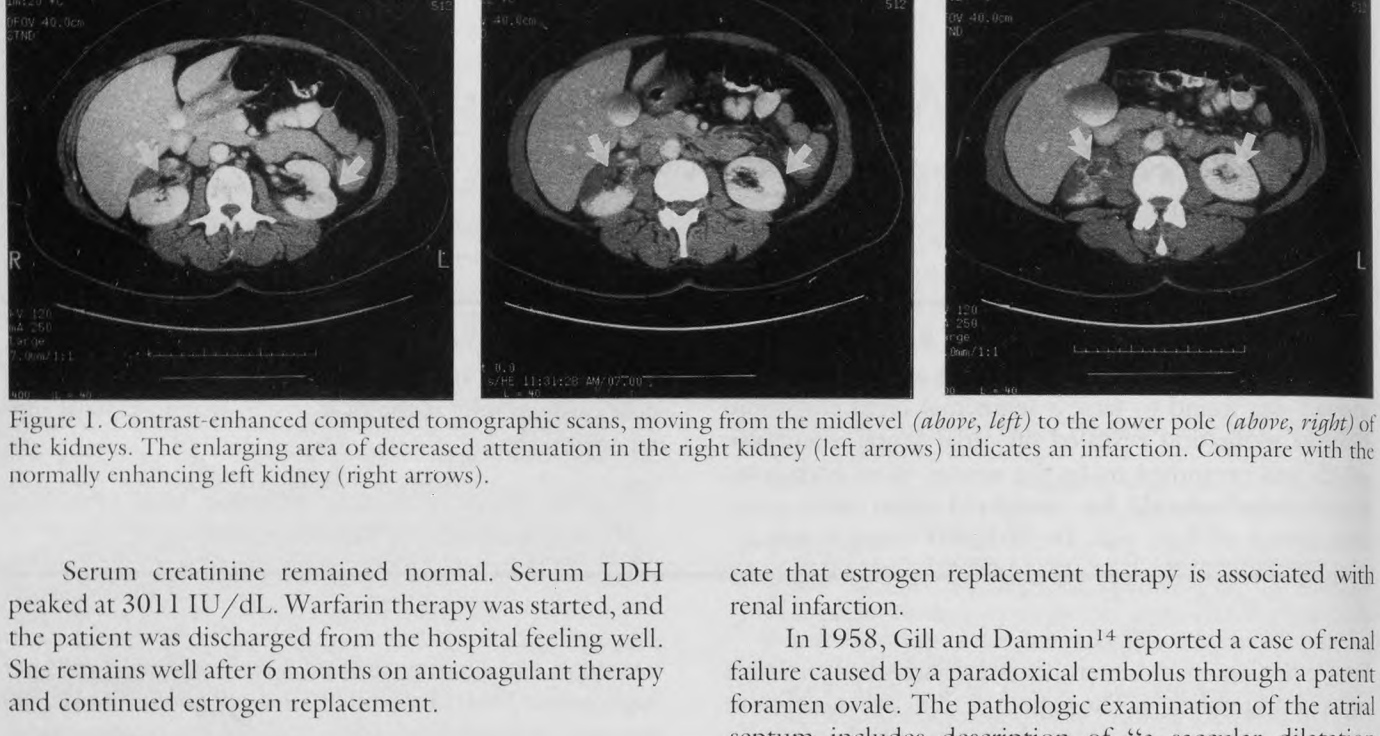

Figure 1. Contrast-enhanced computed tomographic scans, moving from the midlevel *(above, left)* to the lower pole *(above, right)* of the kidneys. The enlarging area of decreased attenuation in the right kidney (left arrows) indicates an infarction. Compare with the normally enhancing left kidney (right arrows).

Serum creatinine remained normal. Serum LDH peaked at 3011 IU/dL. Warfarin therapy was started, and the patient was discharged from the hospital feeling well. She remains well after 6 months on anticoagulant therapy and continued estrogen replacement.

Discussion

Renal Emboli

Although embolization of a kidney usually presents with flank pain similar to ureteral colic, patients may describe a variety of chest and abdominal symptoms. As many as 25% of patients describe no pain. The urinary sediment usually shows microhematuria and proteinuria. Serum LDH is the most sensitive enzyme marker for renal infarction.[3]

Intravenous pyelography may show nonfunction, reduced function, or normal function, depending on the size and number of emboli. Obstructing ureteral lesions may need to be excluded by ultrasonography or retrograde ureterography.[4] A CT scan usually shows areas of decreased density that do not enhance with contrast in the affected kidney.[5] Renal arteriography reliably demonstrates the emboli, but is not required to make a diagnosis.[3]

While most emboli arise from abnormalities of the heart, renal infarction has also been reported in association with sickle cell disease,[6] fibrous dysplasia,[7] cocaine use,[8] neurofibromatosis,[9] trauma,[10] and autoimmune disorders, such as systemic lupus erythematosus,[11] antiphospholipid antibody syndrome,[12] and polyarteritis nodosa.[13] Although thromboembolic disease is a well-known risk of estrogen replacement therapy, the literature does not indicate that estrogen replacement therapy is associated with renal infarction.

In 1958, Gill and Dammin[14] reported a case of renal failure caused by a paradoxical embolus through a patent foramen ovale. The pathologic examination of the atrial septum includes description of "a saccular dilatation which bulged 1.2 cm. into the left atrium," probably representing an aneurysm. There have been subsequent reports linking renal infarction and atrial septal aneurysms (personal communication, Andreas Mugge, October 12, 1995).

Most authorities recommend anticoagulant therapy for renal emboli limited to one renal artery or a segmental branch. Embolectomy has been advocated for patients who have bilateral emboli or emboli to a solitary kidney and who are surgical candidates. Intra-arterial thrombolysis is another option that has not been adequately studied.[15] Even with massive embolization, anticoagulant therapy often results in the eventual return of serviceable renal function.[3]

Atrial Septal Aneurysms

During prenatal life, blood flow between the atria occurs obliquely through the foramen ovale in the septum secundum and the ostium secundum in the septum primum. Postnatally, the fusion of the septa results in a depression seen on the right side of the interatrial septum at the site of the former foramen ovale, referred to as the fossa ovalis. If fusion of the septa occurs with an overlap of the foramen ovale and the ostium secundum, an atrial septal defect occurs. If the tissue at the site of the fossa ovalis is redundant and bulges into either atrium, it is termed an atrial septal aneurysm. Autopsy studies suggest a 1% prev-

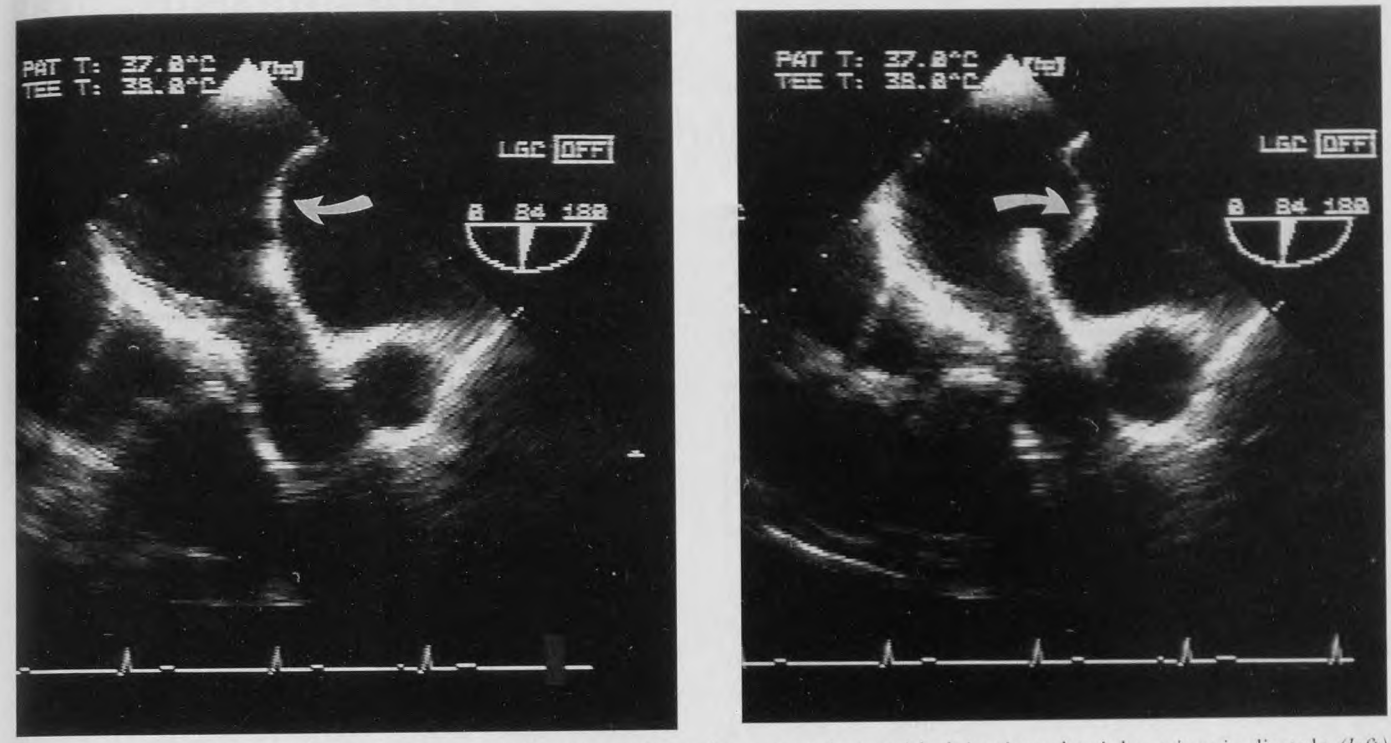

Figure 2. Transesophageal echocardiogram, showing the atrial septal aneurysm (arrows) bulging into the right atrium in diastole *(left)* and the left atrium in systole *(right)*. The circular structure below the left atrium is the aorta.

alence of these aneurysms. Most are associated with right-to-left shunting through a septal defect.[16]

Atrial septal aneurysms have been associated with embolic events, mostly cerebrovascular.[2,17] The mechanism of embolization is uncertain, although most speculation has centered around formation of thrombus at the site of the aneurysm or paradoxical emboli through an atrial septal defect.[16,18] The reported peripheral embolization has most often been to the arteries of the lower extremities.[2,16] Patients who have had a stroke with no other evident source of emboli have a 39% prevalence of atrial septal aneurysms, compared with an 8% prevalence in a control group. Aneurysms that protrude more than 10 mm into the atrial cavity appear to be the higher risk lesions.[17]

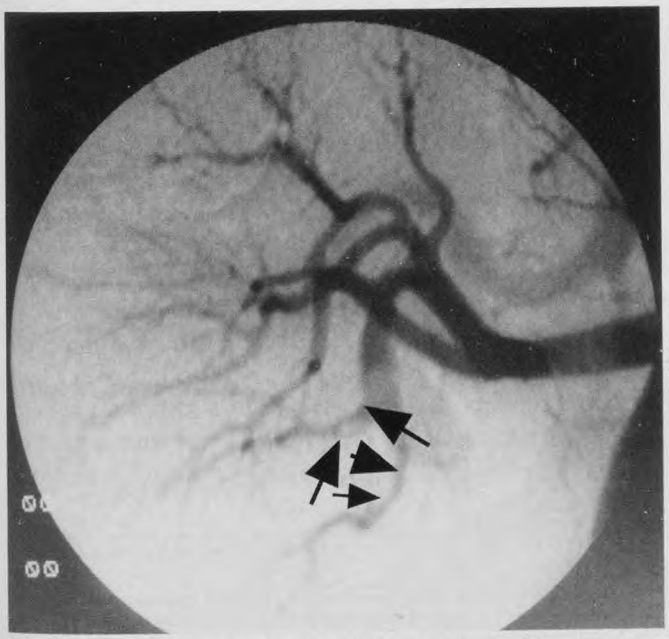

Figure 3. Arteriogram of the right kidney, showing filling defects (arrows) in inferior segmental branches of the right renal artery.

Atrial septal aneurysms are best identified by transesophageal echocardiography. Transthoracic echocardiograms may miss 47% of aneurysms that protrude more than 10 mm.[19] Treatment of patients found incidentally to have atrial septal aneurysms is controversial. One study suggests that aspirin may be an effective prophylaxis against embolic events.[20]

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