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Case 4-2013: A 50-Year-Old Man with Acute Flank Pain

Anna Greka, M.D., Ph.D., R. Sacha Bhatia, M.D., Sharjeel H. Sabir, M.D., and John P. Dekker, M.D., Ph.D.

From the Departments of Medicine (A.G., R.S.B.), Radiology (S.H.S.), and Pathology (J.P.D.), Massachusetts General Hospital; and the Departments of Medicine (A.G., R.S.B.), Radiology (S.H.S.), and Pathology (J.P.D.), Harvard Medical School — both in Boston.

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PRESENTATION OF CASE

Dr. Emily A. Kendall (Medicine): A 50-year-old man was admitted to this hospital because of acute pain in the left flank.

The patient had been well, with a history of Hodgkin's lymphoma 15 years earlier, until approximately 6 a.m. on the day of admission, when pain in the left flank occurred while he was bicycling. The pain was localized, without radiation, and increased during the next 7 hours from 3 to 8 on a scale of 0 to 10, with 10 indicating the most severe pain. He came to the emergency department of this hospital.

At a routine annual visit 4 days before admission, the patient reported feeling well. Examination revealed nontender varicoceles and prostatic hypertrophy; the remainder of the examination was normal. A tetanus–diphtheria–pertussis vaccine booster was administered. During the next 4 nights, he noted mild nausea and temperatures to 38.1°C, without chills, which he attributed to the vaccination.

Fifteen years earlier, a diagnosis of Hodgkin's lymphoma had been made, and the patient had received six courses of doxorubicin, bleomycin, vinblastine, and dacarbazine, as well as radiation therapy and splenectomy; hypothyroidism had developed after treatment. He also had migraines, hyperlipidemia (type IIA), biapical pulmonary fibrosis, and pulmonary sarcoidosis. A stress echocardiogram 21 months before admission showed normal left ventricular function at rest, a mildly thickened tricuspid aortic valve, mild aortic insufficiency, mild aortic-root dilatation, and trivial regurgitation of the other valves; the stress echocardiogram was negative for ischemia. Medications included calcium citrate, enteric-coated aspirin, simvastatin, levothyroxine, and 25-hydroxyvitamin D (ergocalciferol), and he had received immunization against *Haemophilus influenzae*, pneumococcus, and meningococcus in the past. He was married and worked in an office. He maintained a high-fiber, low-fat diet, and he exercised, drank alcohol in moderation, and did not smoke. His mother had had breast cancer, and she and a maternal aunt had had coronary artery disease.

On examination, the patient was alert and oriented and appeared uncomfortable. The temperature was 37.3°C, the blood pressure 158/72 mm Hg, the pulse 76 beats per minute, the respiratory rate 20 breaths per minute, and the oxygen saturation 100% while he was breathing ambient air. A holosystolic soft murmur, grade 2/6,

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was heard at the apex, with radiation to the axilla, and a crescendo–decrescendo systolic murmur, grade 2/6, also was heard at the base, especially at the left upper sternal border. The abdomen was nondistended, with normal bowel sounds and moderate tenderness to palpation in the left flank below the costophrenic angle, without guarding. Examination for Murphy's sign was negative. The remainder of the examination was normal. The blood levels of electrolytes, calcium, phosphorus, glucose, total protein, albumin, globulin, amylase, and lipase were normal, as were the hematocrit, the hemoglobin level, the platelet count, and urinalysis; tests of coagulation, renal function, and liver function were negative. Other test results are shown in Table 1. Cultures of blood and urine were obtained. An electrocardiogram showed sinus rhythm at a rate of 78 beats per minute, with probable left ventricular hypertrophy.

Intravenous fluids, ondansetron, and narcotic analgesia were administered, as were the patient's usual doses of levothyroxine and simvastatin. There was symptomatic improvement.

Computed tomography (CT) of the abdomen and pelvis after the administration of oral and intravenous contrast material showed layered hyperdense material in the gallbladder, a feature suggestive of gallstones, and a focal wedge-shaped defect in the left inferolateral kidney, with patent renal arteries and veins (Fig. 1). CT of the thorax, performed according to a protocol for the assessment of pulmonary embolism, revealed bilateral posteromedial fibrosis and pulmonary nodules, 2 to 3 mm in diameter, in the right lung, which were stable as compared with previous studies. There was no evidence of filling defects in the pulmonary arteries or of mediastinal, hilar, or axillary lymphadenopathy.

Seven hours after presentation, the patient's temperature rose to 37.8°C. He was admitted to the hospital.

Diagnostic tests were performed.

DIFFERENTIAL DIAGNOSIS

Dr. Anna Greka: This generally healthy 50-year-old man with a remote history of Hodgkin's lymphoma presented with the acute onset of flank pain. The patient provided specific details about the precise time of the onset of pain, which is evidence that the pain began suddenly. The differential diagnosis of acute-onset flank pain must begin with a consideration of renal colic due to a renal calculus, which would classically be manifested as flank pain and hematuria. The fact that the urinalysis was normal would argue against a renal stone, but definitive imaging with a non–contrast-enhanced abdominal and pelvic CT scan is needed to rule out this diagnosis. Pyelonephritis, which classically presents with flank pain and fever, is also an important consideration. Although the patient did indeed present with fever, the abrupt onset of flank pain is not well matched with a diagnosis of pyelonephritis, and the normal urinalysis without evidence of pyuria makes an infection of the kidney unlikely. Flank pain is most commonly related to renal disease; however, intraabdominal processes such as cholecystitis, pancreatitis, and mesenteric ischemia, which may cause referred pain to the flank, should be ruled out. In this case, the physical examination and imaging findings do not support these diagnoses.

ISCHEMIA

Renal ischemia is characterized by the acute onset of pain. Patients with renal ischemia are able to describe the exact time of onset of the pain and its severity. It is important that this patient was aware of the precise moment that he started having flank pain — that is, while riding his

Table 1. Laboratory Data.

Variable	Reference Range, Adults*	On Admission
White-cell count (per mm^3)	4500–11,000	11,400
Differential count (%)		
Neutrophils	40–70	76
Lymphocytes	22–44	14
Monocytes	4–11	8
Eosinophils	0–8	1
Basophils	0–3	1
D-Dimer (ng/ml)	<500	663
Erythrocyte sedimentation rate (mm/hr)	0–11	19
C-reactive protein (mg/liter)	<8.0	59.7
Lipoprotein A (mg/dl)	<3	11

* Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are for adults who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

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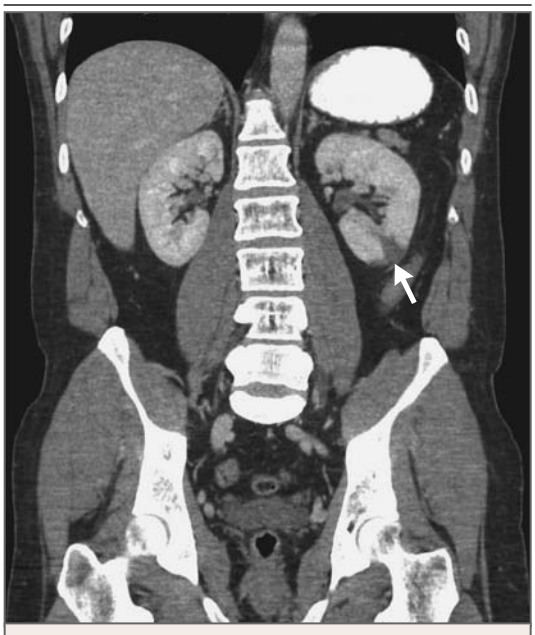

Figure 1. CT of the Abdomen and Pelvis.
A coronal reformatted image from CT of the abdomen and pelvis after the administration of intravenous and oral contrast material shows a wedge-shaped hypodense region (arrow) involving the lower pole of the left kidney, a feature consistent with infarction.

bike. This symptom is most consistent with renal infarction, although the condition is rare.[1,2] The acute onset of severe pain, the normal urinalysis, the development of hypertension in an otherwise normotensive patient (indicating an acute kidney insult[3]), and the presence of fever support the diagnosis of either a thromboembolic event due to clots in the heart or the aorta[4,5] or, more rarely, in situ thrombosis of the renal artery.[6] CT of the abdomen and pelvis with the administration of intravenous contrast material is the test of choice to assess renal infarction.

May we see the imaging studies?

Dr. Sharjeel H. Sabir: CT of the abdomen and pelvis after the administration of intravenous and oral contrast material (Fig. 1) shows a wedge-shaped hypodense region, with its apex at the renal medulla, involving the lower pole of the left kidney. Given the clinical presentation, this appearance is consistent with infarction. Of note, the renal vessels are normal, without evidence of atherosclerotic calcification, stenosis, or aneurysm. There is no other acute intraabdominal process.

A pulmonary-vessel CT angiogram (CTA) shows bilaterally symmetric paramediastinal subpleural reticular opacities in the upper lobes, architectural distortion, volume loss, and traction bronchiolectasis, with relatively straight margins, features that are consistent with pulmonary fibrosis due to previous radiation. A solid subpleural pulmonary nodule, 3 mm in diameter, is visualized in the right middle lobe. Because it has been stable since it was observed on CT of the chest 2 years earlier, this nodule meets the CT criteria for a benign process such as a noncalcified granuloma. Finally, both left and right pulmonary arteries are free from any filling defects. There is no mediastinal or hilar lymphadenopathy.

Dr. Greka: The imaging studies in this case are helpful in narrowing our differential diagnosis. A renal calculus, pyelonephritis, and an intraabdominal process can be ruled out because the images point to a renal infarct as the most likely underlying cause of the patient's symptoms. What could cause the renal infarct?

During a routine visit to his doctor's office just 4 days before presentation, this patient received a scheduled vaccination (tetanus–diphtheria–pertussis), which was associated, or at least temporally correlated, with fever. A low-grade fever is, in itself, not worrisome, but it does cause alarm in this patient because he does not have a spleen. Fever in an asplenic patient carries a specific differential diagnosis that is central to the understanding of this case. Before devoting our attention to the presumed cause of a renal infarct associated with fever, it is worth briefly considering alternative causes. Cancer causing a tumor embolus in the kidney or thromboembolism related to the hypercoagulable state that accompanies many malignant tumors should be considered. However, the patient's history of cancer is remote, and there is no other evidence supporting this diagnosis. In addition, patients with vasculitis (specifically, polyarteritis nodosa, a medium-vessel vasculitis affecting the kidney) can present with pain, fever, and renal infarction, although there is little else in this case to support this diagnosis.

FEVER IN THE ASPLENIC PATIENT

The sinusoids of the spleen filter blood and allow resident mononuclear phagocytes to ingest circulating bacteria.[7] The spleen also contains nearly half of the total B lymphocytes responsible for immunoglobulin production and, therefore, is centrally important for the production of opso-

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nizing antibodies.[7] This function is critical in the clearance of encapsulated organisms.[7] Therefore, an asplenic patient is at risk for postsplenectomy sepsis, a fulminant and often rapidly fatal illness resulting from infections that are normally cleared from the circulation by the spleen. The most common causes of sepsis in asplenic patients relate to bacterial infection with encapsulated organisms, including *Streptococcus pneumoniae*, *H. influenzae*, and *Neisseria meningitidis*; more than 50% of the cases of sepsis and associated deaths are related to *S. pneumoniae*.[8] Appropriately, and to some extent reassuringly, this patient had received immunizations against these organisms in the past, in accordance with the recommendations of the Centers for Disease Control and Prevention (www.cdc.gov; recommendations of the ACIP [Advisory Committee on Immunization Practices]). Since this patient was a bicyclist and spent time outdoors, it is also reasonable to consider other, less common pathogens, such as *Capnocytophaga canimorsus*, a pathogen associated with dog bites or dog scratches,[9] and *Bordetella holmesii*, which is associated with bacteremia and endocarditis in asplenic patients and other immunocompromised hosts.[10] Although bacteria are by far the most common pathogens in asplenic persons, our patient may also have been at risk for parasites that infect red cells, such as babesia, since the spleen removes senescent erythrocytes from the circulation.[7] Presumably, this patient resided in New England and spent time outdoors. Therefore, one should be concerned about babesiosis as the underlying cause of his presentation.[11,12]

ENDOCARDITIS

An important feature of this case was the presence of heart murmurs. An echocardiogram 21 months before presentation showed that the patient had mild insufficiency of all valves. However, on physical examination, a possibly new crescendo–decrescendo murmur was detected that radiated to the left upper sternal border, a feature that is most consistent with aortic stenosis, and a holosystolic murmur was detected that radiated to the axilla, a feature that is most consistent with mitral regurgitation. Although it was not clear whether these murmurs were new, they suggested that further investigation of these clinical signs by echocardiography was indicated. The presence of fever, new heart murmurs, and renal infarction was strongly suggestive of an infectious process, with a specific concern for endocarditis with valvular vegetations[13] and an associated thromboembolic event affecting the kidney.

Careful analysis of available laboratory data was also helpful in this case. The patient had normal blood chemical profiles, normal renal function, and a normal hematocrit with a mildly elevated white-cell count. The absence of a prominent leukocytosis led us away from a diagnosis of sepsis and toward a subacute infectious process, such as subacute endocarditis, a condition that may be associated with renal infarction.

RENAL INFARCTION

Renal infarcts usually present with the abrupt onset of abdominal or flank pain,[3] as in this case, and sometimes they are associated with signs of extrarenal embolization[14] (e.g., neurologic deficits), which were not seen in this case. Renal infarction is often associated with leukocytosis, an elevated creatinine level, and microscopic hematuria or even proteinuria, although these findings are most consistent with large emboli or bilateral disease.[1,2,5,6,14] The most common cause of renal infarction (Table 2) is thromboembolic disease from the heart or aorta due to a left atrial clot in a patient with atrial fibrillation, left ventricular thrombus in a patient with myocardial infarction, or thromboemboli derived from ruptured plaque in the aorta.[1,2,5,6,14] Other sources of thromboemboli can be valvular vegetations from infective endocarditis, which is an important consideration in this case. Tumor or fat emboli or a paradoxical embolus from a deep venous thrombosis in a patient with a patent foramen ovale may also result in emboli to the kidney. However, we have no evidence that this situation occurred in this case.[1,2,5,6,14] In situ thrombosis can also occur; it is most often due to renal-artery occlusion from an aortic dissection, but it can be a complication after either an aortic or a renal endovascular intervention.[15-17] Another consideration is fibromuscular dysplasia,[18-20] which in a young patient with medial fibroplasia could appear on angiography as a renal infarct.[18] Segmental arterial mediolysis, although rare, may also cause renal infarction, but it is usually a sudden manifestation associated with a drop in the hematocrit.[16]

To synthesize all these findings into a final diagnosis, I shall invoke the principle that the simplest answer is most likely the correct one. The most parsimonious explanation for the combination of fever, murmurs, and a wedge-shaped

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Table 2. Causes of Renal Infarction.

Thromboembolic causes, from the heart or aorta
In situ thrombosis
Renal-artery occlusion from aortic dissection
Spontaneous or iatrogenic renal-artery dissection
Fibromuscular dysplasia
Segmental arterial mediolysis
Polyarteritis nodosa
Complication of the antiphospholipid syndrome
Cocaine use

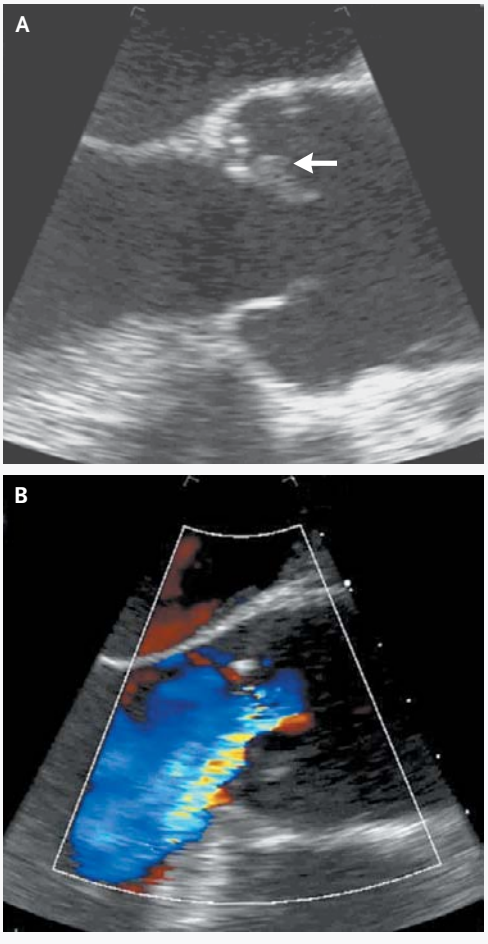

Figure 2. Echocardiographic Images.
A transesophageal echocardiogram at the midesophageal level, long-axis view, shows vegetation on the noncoronary cusp of the aortic valve (Panel A, arrow). There is moderate, centrally directed aortic insufficiency, without evidence of an aortic-root abscess or paravalvular leak (Panel B).

Videos showing transesophageal echocardiography are available at NEJM.org

renal infarct in an asplenic patient is thromboembolic renal infarction due to endocarditis. Although blood cultures may ultimately identify the causative organism in this case, a transthoracic or transesophageal echocardiogram to look for valvular vegetations would be helpful in making the diagnosis of endocarditis.

Dr. Eric S. Rosenberg (Pathology): Dr. Kendall, what was your clinical impression when you saw this patient?

Dr. Kendall: Identifying the heart murmurs on examination led us to think about endocarditis complicated by an embolic event to the kidney. We also considered thromboembolic sources in the heart, including an atrial thrombus in this patient with atrial fibrillation, or a ventricular source, possibly related to a dilated cardiomyopathy caused by previous chemotherapy.

DR. ANNA GREKA'S DIAGNOSIS

Thromboembolic renal infarction due to endocarditis in an asplenic patient.

CLINICAL DIAGNOSIS

Endocarditis complicated by an embolic event in the kidney.

Dr. Rosenberg: Dr. Bhatia, may we see the echocardiogram?

Dr. R. Sacha Bhatia: A transthoracic echocardiogram was obtained, which showed thickening of multiple aortic leaflets but no stenosis. One cannot say definitively that there were vegetations on the aortic valve. There was mild-to-moderate aortic insufficiency. The left ventricular systolic function was normal, with an ejection fraction of 60%. The other valves appeared normal.

Transesophageal echocardiography was performed. An echogenic mass was seen on the noncoronary cusp of the aortic valve, a finding that is consistent with vegetations (Fig. 2A; and Videos 1 and 2, available with the full text of this article at NEJM.org). This echogenic mass was associated with moderate aortic insufficiency. There was no evidence of an aortic-root abscess or a mycotic aneurysm in the aorta (Fig. 2B). The other valves did not have vegetations. There was no evidence of other sources of cardiac embolus, including no clot in the left atrial appendage and no aortic atheroma. No intracardiac shunt was seen.

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PATHOLOGICAL DISCUSSION

Dr. John P. Dekker: Two sets of blood cultures were received by the microbiology laboratory on the day of admission. On the fourth hospital day, one bottle from the initial set turned positive, and a Gram's stain showed chains of gram-positive cocci similar to those shown in Figure 3. After culture on a blood agar plate, the organism was identified as *S. mutans*, a member of the viridans group streptococci. Viridans group streptococci are common commensal components of the human oropharyngeal flora, and *S. mutans* has been identified as a major contributor to tooth decay and dental caries.[21] Streptococcal species (including *S. mutans*) are the most common cause of native-valve endocarditis in adult patients, accounting for 45 to 65% of cases.[13] In total, 15 sets of blood cultures were obtained during the hospital course, of which 11 out of the first 14 bottles grew *S. mutans*. The high-grade nature of the bacteremia was suggestive of an endovascular source of infection and, in conjunction with the clinical and echocardiographic findings, was consistent with bacterial endocarditis and septic embolism to the kidney.

The final blood culture that became positive was received on the fifth hospital day and was reported positive on the seventh hospital day. In addition to growing *S. mutans*, this final positive culture also grew methicillin-resistant *Staphylococcus aureus* (MRSA). It was presumed that the MRSA infection was acquired during the hospital admission. All subsequent blood cultures were negative.

FOLLOW-UP

Dr. Kendall: The patient was treated for streptococcal endocarditis and bacteremia with penicillin while in the hospital. We then switched his antibacterial therapy to ceftriaxone, which is easier to administer on an outpatient basis. He completed a 4-week course of ceftriaxone. For the first 2 weeks, he was also treated with vancomycin and gentamicin for the MRSA bacteremia. A follow-up transthoracic echocardiogram obtained 3 months later showed a persistent mobile density on the aortic valve, although it appeared improved. The patient is now doing well.

Dr. Lloyd Axelrod (Medicine): What can we learn about the physical examination from the cardiac ultrasound study? The patient was said to have two murmurs. One was consistent with aortic stenosis, and the other was more consistent with mitral regurgitation.

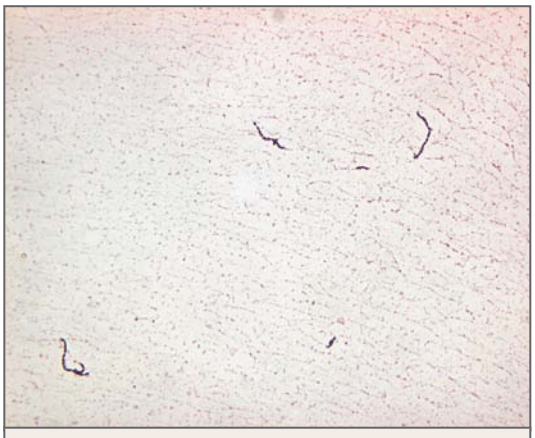

Figure 3. Microbiology.
A representative image from another case shows a Gram's stain prepared from a positive blood culture. Chains of gram-positive cocci and coccobacillary forms are present. (Photograph courtesy of Judith Holden.)

Dr. Bhatia: I suspect the clinical environment makes a huge difference. The initial cardiac examination was performed in the emergency department, which is often a very noisy place. A diastolic murmur is almost always pathological and may be subtle. To appreciate such a murmur, it is best to be in a quiet room; if cardiac disease is suspected, the clinicians should perform maneuvers that may accentuate certain cardiac murmurs. In this case, aortic regurgitation could have been better heard by listening to the heart sounds while the patient was sitting forward at end expiration or by performing a handgrip maneuver. I suspect the clinicians heard a systolic-flow murmur due to increased cardiac output secondary to fever and tachycardia.

ANATOMICAL DIAGNOSIS

Streptococcus mutans bacteremia and endocarditis and methicillin-resistant *Staphylococcus aureus* bacteremia.

This case was presented at the Medical Case Conference.

No potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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