Residents' Clinic

71-Year-Old Woman With Low Back Pain

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A 71-year-old woman presented to the outpatient clinic with a chief complaint of having low back pain for 6 months. The back pain was insidious at onset, nonradiating, moderate in intensity, worse at night, and partially relieved with acetaminophen or aspirin. The patient described the pain as a constant, dull, aching sensation and recalled no trauma. She experienced anorexia and reported a 6.8-kg weight loss and decreased energy over the past several months. She also described occasional night sweats. The patient's history was remarkable for bilateral stage I breast cancer, for which she had a bilateral modified radical mastectomy 7 years previously. She also underwent left carotid artery to left subclavian artery bypass surgery for symptomatic left subclavian artery stenosis 4 years previously. Total hysterectomy was performed many years previously for reasons unrelated to cancer. The patient had hyperlipidemia but was not taking a lipid-lowering agent. Several years previously, microscopic hematuria was detected, and findings on cystoscopy and excretory urography were normal. She denied having gross hematuria. The patient was a current smoker with a 30-pack-year history of cigarette smoking. Her current medications were acetaminophen and aspirin on an as-needed basis.

Physical examination disclosed a pulse rate of 88/min with a regular rhythm and a blood pressure level of 140/88 mm Hg. Examination of the patient's spine showed no deformity, tenderness, or restriction of joint movements. Findings on the neurologic examination, including gait assessment, motor strength, and reflexes, were unremarkable. Examination of her abdomen revealed a midline, pulsatile mass (5 × 10 cm) extending from the epigastric area to the umbilical area. The mass was nontender and fixed. No bruit was audible over the abdominal mass. The patient had been aware of this mass for several months but had not sought medical attention. She denied recent change in the size of the abdominal mass, abdominal pain, change in bowel habits, angina, dyspnea, fatigue, edema, syncope, myocardial infarction, cardiac arrhythmia, or cerebrovascular accident. The cardiovascular examination revealed normal cardiac sounds and symmetrical normal pulses without bruits. Findings on the rest of her examination were normal.

1. Which <u>one</u> of the following is the <u>least likely</u> cause of back pain in this patient?

a. *Spinal malignancy*
b. *Spinal infection*
c. *Abdominal aortic aneurysm (AAA)*
d. *Inflammatory spondylarthritis*
e. *Vertebral compression fracture*

Spinal malignancy is a rare but serious cause of back pain. Our patient's clinical features such as age older than 50 years, pain at night, lack of improvement with conservative treatment, and history of previous cancer are known to be associated with the presence of neoplastic diseases[1]; therefore, spinal malignancy is a possible cause of her back pain. Spinal infection is suspected in a patient with new- and acute-onset back pain who has a history of recent spinal procedure, fever, and severe pain that is focal and unrelieved by rest. Although our patient has new-onset back pain unrelieved by rest, she has not had a spinal procedure and does not have fever or other risk factors such as recent infection, surgery, or immunosuppression. However, lack of fever and other risk factors does not necessarily rule out spinal infection. In a recent series of 41 patients with infectious spondylitis, only 25 patients (61%) had fever, and in 24 patients (59%), no risk factors were identified.[2]

The palpable abdominal mass is most likely an aortic aneurysm in our patient because the sides of the mass could be felt to pulsate in a lateral direction. The presence of associated chronic pain in a smoker raises the suspicion of inflammatory AAA in this patient. The other less common causes of pain related to AAA are compression of adjacent structures, rapid enlargement of the aneurysm, dissection of the arterial wall, or leakage from the aneurysm.

Inflammatory spondylarthritis is insidious at onset, often begins before age 40 years, improves with exercise, persists for at least 3 months, and is associated with morning stiffness. On examination, axial motion is limited in all planes. The patient's back pain began when she was age 70 years and is not associated with morning stiffness, limita-

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See end of article for correct answers to questions.

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tion of spinal movements, or systemic symptoms; thus, inflammatory spondylarthritis appears to be the least likely cause of her back pain.

Vertebral compression fracture causes acute back pain of sudden onset accompanied by marked muscle spasm and is typically severe for several weeks, subsequently diminishing over a few months. Prolonged sitting or standing worsens the pain, and usually percussive tenderness is demonstrated at the affected level. None of these clinical features are present in this patient but osteoporosis is still a possibility because of her race, age, and smoking history.

Complete blood cell counts and serum chemistries, including calcium, phosphate, and alkaline phosphatase, were normal in our patient. Urinalysis revealed 21 to 30 red blood cells on microscopy. There was no evidence of urinary proteinuria, dysmorphic red blood cells, or casts. The erythrocyte sedimentation rate was 45 mm/1 h. A 12-lead electrocardiogram was normal. A spinal radiograph showed only changes of degenerative arthritis in the lower lumbar facet joints with a minimal degree of osteopenia.

2. Which one of the following is the most appropriate initial diagnostic test to order for our patient's evaluation?

a. *Magnetic resonance imaging (MRI) of the spine*
b. *Bone scan*
c. *Abdominal ultrasonography*
d. *Bone mineral densitometry*
e. *Excretory urography*

An MRI of the spine is highly sensitive and specific for diagnosing spinal cancer or infection but is costly.[3] A bone scan is 95% specific and 70% sensitive for diagnosing spinal malignancy or infection.[3] It has the additional advantage of being able to detect extraspinal metastasis. However, malignancy or infection is not the most likely cause of this patient's symptoms. On the basis of her physical examination findings, the pretest probability of AAA is high. Abdominal ultrasonography is inexpensive, noninvasive, and nearly 100% sensitive for the diagnosis of AAA.[4] Another major advantage of ultrasonography is that it does not require the use of a contrast agent. Therefore, abdominal ultrasonography should be the initial test of choice for this patient.

Bone densitometry may be indicated at some point because this patient has risk factors for osteoporosis; however, it is unlikely to detect the cause of her symptoms. She had microscopic hematuria several years previously with a history of negative results on excretory urography and cystoscopy. It is unlikely that repeated excretory urography would provide any diagnostic yield. It can be considered but not as the first diagnostic test.

In our patient, abdominal ultrasonography showed an aneurysm of the distal abdominal aorta measuring $5.7 \times 5.6 \times 10$ cm. Its ultrasonographic appearance suggested an inflammatory AAA.

3. Which one of the following is the most appropriate next step in confirming the suspected diagnosis in this patient?

a. *High-resolution ultrasonography*
b. *Abdominal computed tomography (CT)*
c. *Excretory urography*
d. *Abdominal MRI*
e. *Aortography*

The accuracy of high-resolution ultrasonography in identifying the inflammatory component of an AAA is less than that of an abdominal CT scan.[5,6] Computed tomography has 90% sensitivity in detecting an inflammatory AAA.[5] Computed tomography is the most reliable radiographic technique for detecting aneurysmal wall thickening and perianeurysmal soft tissue changes that suggest inflammatory AAA. It is also useful in diagnosing a leaking aneurysm. Therefore, CT should be performed in our patient.

Excretory urography is used preoperatively to detect ureteral obstruction resulting from perianeurysmal inflammation and fibrosis. Abdominal MRI has been used to diagnose inflammatory AAA.[7] It has an advantage compared with contrast-enhanced CT in patients with renal impairment. However, the cost benefit of MRI compared with abdominal CT and ultrasonography has not been established. Aortography is of no benefit in detecting the inflammatory component of an AAA.[8]

Contrast-enhanced CT of the abdomen showed a 7.5-cm-diameter fusiform infrarenal aortic aneurysm that had a mural thrombus within it and a rind on inflammatory tissue. There was no evidence of leak.

4. Which one of the following is the most appropriate management of this patient?

a. *Nonoperative management with corticosteroids*
b. *Treatment with β-blockers*
c. *Standard surgical repair*
d. *Surgical repair with limited dissection technique*
e. *Endovascular stent graft repair*

The CT findings suggested probable inflammatory AAA in our patient. Clinical trials evaluating the safety and efficacy of corticosteroids for the nonoperative management of inflammatory AAA are not available. However, experts agree that corticosteroids do not alter the long-term course of inflammatory aneurysm.[6,9] On the contrary, corticosteroids may increase the tendency to rupture as a result of a reduction in the periaortic fibrotic reaction and there-

fore are not indicated in this patient. Although β-blockade is an important cardiac protector and is used in nearly all patients undergoing vascular surgery,[10] its role in the treatment of an inflammatory AAA is unclear.

Standard surgical repair is recommended for asymptomatic noninflammatory AAA greater than 5.0 cm in diameter, along with periodic surveillance with CT of aneurysms less than 4.0 cm in diameter.[11] The benefit of surgery for an asymptomatic noninflammatory AAA measuring 4.0 to 5.0 cm in diameter has not been defined. Most noninflammatory AAAs are asymptomatic, and their diameter determines the risk of rupture in the following year.[11] In contrast, most inflammatory AAAs are symptomatic, and their natural history seems to involve enlargement and rupture.[6,9] Therefore, operative repair is the treatment of choice for inflammatory AAAs, irrespective of their size. A modified surgical technique of limited dissection is recommended because of extensive adhesions of perianeurysmal structures in this type of aneurysm. Surgery with a limited dissection technique should be used to repair inflammatory AAA in our patient. Long-term survival in patients with inflammatory AAA has improved with modified operative techniques[6] and is now similar to that of patients with noninflammatory AAA.[12]

Although preliminary data suggest that endovascular stent graft repair may become the treatment of choice for inflammatory AAA, the long-term outcome of endovascular repair is not yet known. Patients at low surgical risk with expected long-term survival are probably better served with an open surgical repair.[13]

Our patient was evaluated by a vascular surgeon who agreed with operative management but asked if she was medically cleared for major noncardiac vascular surgery. Although the patient denied angina with exertion, she was unable to do any substantial exercise because of her back pain.

5. Which one of the following is the most appropriate step in the preoperative cardiovascular risk evaluation of this patient?

a. *Begin β-blockade, proceed with surgery*
b. *No β-blockade necessary, proceed with surgery*
c. *Perform additional risk stratification with a pharmacologic cardiac stress test*
d. *Perform additional risk stratification with coronary angiography*
e. *Perform coronary revascularization to reduce risk*

Coronary artery disease is the most important underlying medical illness contributing to perioperative mortality. In addition, a history of cerebrovascular disease, a preoperative elevated creatinine level greater than 2 mg/dL, insulin treatment for diabetes mellitus, and high-risk (vascular) surgery have all been associated with increased perioperative morbidity.[14]

According to the American Heart Association-American College of Cardiology guidelines,[15] this patient has minor clinical predictors (advanced age) of increased perioperative cardiovascular risk; she has poor exercise tolerance (inability to exercise to 4 metabolic equivalents); and she is to undergo a procedure that is generally associated with a high (>5%) risk of perioperative cardiac events. She has an intermediate (2%-7%) predicted risk for perioperative cardiac events and would benefit from β-blockade.[16] However, the effectiveness of β-blockade alone (without additional risk stratification with cardiac stress testing) to reduce cardiac risk in intermediate-risk patients with poor functional status is unknown. The use of β-blockade alone (without additional risk stratification with cardiac stress testing) reduces the cardiac event rate significantly only in patients with functional status.[16] Thus, beginning β-blockade and proceeding with surgery is not the best choice for this patient. According to recommendations from several sources,[15,17] this patient, who has low exercise tolerance and high surgery-specific risk, would require additional risk stratification using a pharmacologic cardiac stress test before surgery to determine the potential need for additional therapies to reduce risk, eg, coronary revascularization.

Coronary angiography would be indicated only if noninvasive tests suggest the existence of extensive myocardial ischemia or if symptoms of unstable angina are present.[15] Preoperative revascularization with either a coronary artery bypass graft or angioplasty is appropriate for the same indications as would be recommended in the nonpreoperative setting, eg, significant left main coronary artery disease and significant 3-vessel disease. Thus, it would be premature to consider this option before the results of less invasive studies.

In our patient, adenosine sestamibi imaging was done because of her inability to exercise due to back pain, and it did not show myocardial ischemia. She underwent an elective surgical repair of a large juxtarenal inflammatory AAA. The procedure included an aortobilateral external iliac artery bypass graft and an interposition graft to the right internal iliac artery. The results of intraoperative cystoscopy before aneurysm repair were normal. A bilateral retrograde ureteropyelogram showed medial deviation of the ureters without obstruction. Postoperatively, our patient developed no complications and was symptom free at her follow-up visit 3 months after aneurysm repair.

DISCUSSION

Inflammatory AAA is defined as a triad of thickened aneurysmal wall, extensive perianeurysmal and retroperitoneal

fibrosis, and dense adhesions of adjacent abdominal organs.[6,7] Abdominal CT of our patient showed a thickened rind of soft tissue surrounding the aneurysm, a finding consistent with perianeurysmal fibrosis. Perioperatively, the inferior edge of the left renal vein was densely adherent to the aneurysm, as was the duodenum, and the ureters were medially displaced, findings highly suggestive of inflammatory AAA.

Inflammatory AAA represents 3% to 10% of all AAAs.[5-7] The male-to-female ratio ranges from 30:1 to 6:1, depending on the series.[5-7] The mean age at occurrence ranges from 62 to 68 years.[5-7] A significantly higher percentage of patients with inflammatory AAA are active smokers and have a family history of AAA compared with patients with noninflammatory AAA.[5,7] Our patient had no positive family history but was currently smoking.

Only 8% to 18% of patients with noninflammatory AAA are symptomatic.[5,7] In contrast, 65% to 90% of patients with inflammatory AAA are symptomatic.[5-7] Our patient presented with a triad of low back pain, weight loss, and an increased erythrocyte sedimentation rate, which is a common clinical presentation of inflammatory AAA.[10] A tender, pulsatile mass is palpable in 15% to 30% of patients with inflammatory AAA.[6] Arterial occlusive disease seen in our patient has been reported in 10% to 47% of patients with inflammatory AAA.[5,7] Arterial hypertension is another frequently comorbid condition present in these patients; however, our patient had normal blood pressure. Inflammatory adhesions most commonly involve the duodenum, renal veins, and inferior vena cava.[5,7] Entrapment of ureters in the periaortic mass has been reported in 53% of patients with inflammatory AAA.[10] Perioperative and retrograde ureteropyelographic findings in our patient suggested involvement of the duodenum, left renal vein, and ureters in the inflammatory process.

The pathogenesis of inflammatory AAA remains unclear. The earlier belief that the inflammatory response is dependent on the aneurysm itself has been questioned as a result of data showing that only 53% to 68% of patients had complete or partial regression of periaortic inflammatory response after aneurysm repair.[10] The evidence now suggests that inflammatory AAA is not a distinct clinicopathologic entity, as was thought earlier, but rather is an inflammatory variant of well-known atherosclerotic AAA.[10] Only the intensity and the extent of the inflammatory process differ between inflammatory and noninflammatory AAA; otherwise they are the same disease.[6,7] The etiology of inflammatory and noninflammatory AAA appears to be multifactorial. Environmental and genetic factors are important in the pathogenesis.[10] These factors predispose certain individuals to the development of noninflammatory AAA and others to the development of the extreme end of the inflammatory spectrum, inflammatory AAA.

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Correct answers: 1. *d*, 2. *c*, 3. *b*, 4. *d*, 5. *c*