Abdominal Aortic Aneurysm in a Patient With Low Back Pain

Low back pain is a common condition routinely managed by physical therapists and accounts for up to 53% of all client visits. It is also the fifth most common reason for all physician visits in the United States, with direct health care costs related to the management of low back pain in the United States estimated to be more than $85 billion annually. Unfortunately, despite increasing medical expenditures, the prevalence of chronic, disabling low back pain continues to increase.

Low back pain is most commonly caused by mechanical dysfunction, which can be defined as symptoms that are related to the musculoskeletal system and vary with movement. However, serious nonmechanical spinal disorders (eg, neoplasms, infection, inflammatory arthritis, fracture) or visceral disease (eg, gastrointestinal, genitourinary, vascular) may be the cause of low back pain in a small percentage of patients. Though mechanical dysfunction accounts for approximately 97% of low back pain cases, nonmechanical spinal disorders and visceral disease account for approximately 1% and 2% of low back pain cases, respectively.

One type of visceral disease that may cause low back pain is an abdominal aortic aneurysm (AAA), which is an abnormal dilation in a weakened or diseased arterial wall. By convention, an infrarenal aorta that is 3 cm in diameter or larger is considered aneurysmal. The diameter of the infrarenal aorta is the strongest known predictor of risk for rupture, with the risk substantially increasing with infrarenal aorta diameters of 5.0 cm or greater.

The etiology of an AAA is not completely understood, but atherosclerosis, degeneration, and chronic inflammation have been identified as contributing to its development. Major risk factors for an AAA include hypertension, an age of greater than 60 years, male sex, past history of smoking, atherosclerosis, coronary artery disease, family history of AAA, and use of statins. Factors that are negatively associated with AAA include female sex, black race, and diabetes.

The purpose of this resident’s case problem was (1) to describe the clinical reasoning and decision making in a patient referred with a diagnosis of mechanical low back pain who had an AAA.
requiring immediate medical referral, and (2) to describe an evidence-based approach to clinical evaluation of patients with suspected AAA.

DIAGNOSIS

Chief Complaint

The patient was a 58-year-old Caucasian man referred to a physical therapist with a primary complaint of low back pain and a secondary complaint of abdominal pain. The patient was employed as a government contractor, and his job consisted primarily of desk work. The patient performed 30 minutes of cardiovascular exercise (elliptical machine) 7 days per week and weight training 6 days per week. He had continued this exercise schedule despite his low back and abdominal pain.

Symptom Location, Description, and Behavior

The patient’s low back pain was located centrally at the L3 through L5 spinal levels. He characterized the pain as a constant, deep, dull ache that varied in intensity, with intermittent throbbing sensations (FIGURE 1). During the physical therapist’s initial encounter, the patient rated his current resting pain as 3 on a verbal numeric pain rating scale ranging from 0 to 10, with 0 as no pain and 10 as the worst pain imaginable. Using the same pain rating scale, the patient rated his pain since the pain first presented as 3 at its lowest level and 7 at its typical maximum level.

The patient also reported abdominal pain that was centrally located, starting in the epigastric region and extending inferiorly to the superior margin of the hypogastric region. He described his abdominal pain as a bloated feeling that progressed to a sharp, intense stabbing pain when aggravated. He currently rated his abdominal pain as 1; he also stated that 1 was the minimum pain he could feel, and the typical maximum pain was 4. The patient expressed that the low back pain and abdominal pain seemed to be related and increased proportionally; that is, when the low back pain increased, the abdominal pain increased as well.

Symptom Behavior

The patient was unable to describe any specific aggravating factors for his low back or abdominal pain. More specifically, he denied any difficulty or increase in symptoms with aerobic exercise, climbing stairs, lumbar flexion/extension activities, or resistance training. He also denied an increase in symptoms with coughing, sneezing, and taking a deep breath. Taking naproxen was the only easing factor the patient identified, and it would reportedly lessen both his low back and abdominal pain temporarily.

He had difficulty falling asleep at night, because the pain in his lower back would increase and be followed by an increase in abdominal pain. Once he fell asleep, he would typically wake at least once each night because of pain in his lower back and abdomen. Position changes did not increase or decrease his symptoms.

### TABLE 1

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Odds Ratio*</th>
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<tbody>
<tr>
<td>Age</td>
<td>1.71 (1.61, 1.82) increase for every 7 y increase</td>
</tr>
<tr>
<td>Male sex†</td>
<td>2.66 (1.69, 4.20)</td>
</tr>
<tr>
<td>Hypertension†</td>
<td>1.44 (1.27, 1.63)</td>
</tr>
<tr>
<td>Smoking history</td>
<td>1.5-2.4 (0.54, 6.75)</td>
</tr>
<tr>
<td>Past history†</td>
<td>6.19-13.72 (2.86, 30.78)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>Not reported in literature</td>
</tr>
<tr>
<td>Atherosclerosis†</td>
<td>1.94 (1.63, 2.32)</td>
</tr>
<tr>
<td>Family history</td>
<td>1.52 (1.37, 1.68)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>3.77 (1.45, 9.81)</td>
</tr>
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*Values in parentheses are 95% confidence interval.†Indicates the risk factors present in this patient.
To resume sleeping, he would typically have to lie in bed and wait 45 minutes to 1 hour until he fell asleep, or take one 500-mg naproxen.

**History of Current Condition**

The patient reported that his pain began 8 days prior to his initial evaluation by the physical therapist. He denied a specific mechanism of injury or trauma that preceded his symptoms. His symptoms came on suddenly while sitting in his hotel room when he was away on a business trip. He described his initial symptoms as a “sudden, severe pain” in his lower back and abdomen that forced him to lie supine for 30 minutes. Over the course of the next 8 days, the pain in his low back and abdomen slowly improved. The patient denied a personal past history of low back or abdominal pain prior to this incident.

One day prior to being seen by the physical therapist, the patient was evaluated by his primary care physician and diagnosed with mechanical low back pain and abdominal pain that was related to the low back pain, both of which were deemed musculoskeletal in nature. Prior to referral to the physical therapist, plain-film radiographs were completed, which revealed mild degenerative changes of the L3 through S1 spinal levels. The physician subsequently prescribed naproxen (500 mg), to be taken twice daily for the next 10 to 14 days, and a physical therapy referral.

**Past Medical History**

The patient had a history of hypertension and atherosclerosis, for which he was currently taking amlodipine (5 mg), votorin (10 mg), hyzaar (12.5 mg), and aspirin (81 mg) once daily. He indicated that he had smoked for 2 years in the early 1970s, amounting to 1 pack-year total, but has not used tobacco products since. He denied numbness and tingling in his upper and lower extremities, recent infection, fatigue, weight changes, fever, malaise, saddle anesthesia, pulsation in his abdomen, and pain with the Valsalva maneuver or coughing. Heartburn, indigestion, changes in appetite, difficulty swallowing, and changes in bowel and bladder habit were also all denied. He denied a family history of hypertension, diabetes mellitus, cancer, coronary artery disease, and AAA.

**Clinical Impression Post–History Intake**

The patient had a primary complaint of low back pain and a secondary complaint of abdominal pain that appeared to be related. When considering the differential diagnosis, initial hypotheses were considered in 3 categories: mechanical (eg, musculoskeletal condition), nonmechanical (eg, neoplasm, infection, inflammatory arthritis, fracture), and visceral (eg, gastrointestinal, genitourinary, or vascular). This approach to differential diagnosis allowed the clinician to determine whether the patient’s condition was appropriate for physical therapist management or an additional physician consult was required.

Mechanical dysfunction accounts for approximately 97% of low back pain cases, suggesting a high probability that the vast majority of patients with low back pain will have pain that is mechanical in nature. Lumbar dysfunction may also cause abdominal pain. From the patient history, however, the physical therapist identified components suggesting that the patient’s symptoms might not be mechanical in origin, such as the patient’s inability to identify a specific mechanism of injury to the sudden onset of symptoms, the inability to identify any aggravating or easing factors for his symptoms, and night pain with an inability to ease his symptoms with changes in position. Additionally, Sparkes et al developed 2 specific clusters of questions to be used in identifying patients with abdominal pain that is musculoskeletal in origin (Table 2). These question clusters have high specificity (96%), suggesting that this tool is most useful for ruling in a musculoskeletal cause of abdominal pain. The patient described in this report could not answer “yes” to either of the first 2 questions of cluster 1, suggesting that he was negative for this question cluster, which decreased the likelihood for a musculoskeletal origin of the patient’s abdominal symptoms. The sensitivity (0.67) and negative likelihood ratio (LR) (0.39) associated with the cluster are relatively weak, suggesting that a musculoskeletal cause of the patient’s symptoms was still high. However, when considered in the context of the other historical findings, the fact that cluster 1 was negative gave the clinician added suspicion that the symptoms could be related to nonmusculoskeletal pathology.
are the most common serious pathology had a spinal fracture. Vertebral fractures patient's low back and abdominal pain of inflammatory arthritis as a cause of the ports of morning stiffness, the likelihood likely. report a fever, fatigue, or malaise, it was cancer was an unlikely cause of low plain-film radiographs, it was believed detecting cancer. Given these factors, in screening for spinal malignancy in patients with low back pain. This review in screening for spinal malignancy in elderly patients), and prolonged use of corticosteroids. When at least 1 of these variables was positive, the positive and negative LRNs were 1.8 and 0.24, respectively. With 2 positive features, the positive and negative LRNs increased to 15.5 and 0.39, respectively; 3 or more positive variables yielded a positive LR of 218.3 and a negative LR of 0.62. In addition to the absence of evidence of spinal fracture on plain-film radiographs, none of these variables were present in this patient, further decreasing the likelihood that a vertebral fracture was causing low back pain.

While mechanical dysfunction of the lumbar spine accounts for the vast majority of symptoms experienced by patients with low back pain, the majority of patients with abdominal pain usually have a visceral cause, and in all patients with abdominal pain, visceral pathology should be ruled out. Furthermore, visceral disease is a more frequent cause of low back pain compared to other nonmechanical spinal disorders. With regard to visceral disorders, initial screening questions for the gastrointestinal (eg, patient denied heartburn, nausea, indigestion, changes in appetite, difficulty swallowing, constipation, and changes in frequency of bowel movements) and genitourinary (patient denied pain with urination or changes in urinary frequency) systems were negative. Although each of these findings does not necessarily rule out the presence of a gastrointestinal or genitourinary disorder, the cluster of negative findings did suggest that the patient's symptoms were most likely not related to these systems. Considering vascular pathology, an AAA is a rare cause of low back pain, and in many cases patients experience no symptoms at all. However, the patient in this case indicated that he had 4 of the 8 risk factors associated with increased prevalence of AAA (TABLE 1).

### Physical Examination

The patient’s height was 188 cm, and he weighed 90.7 kg the day of the examination (body mass index, 25.7 kg/m²). Visual observation revealed a nonantalgic gait. A postural examination revealed a forward head and rounded shoulders, with a decreased lumbar lordosis. The iliac crests, anterior superior iliac spine, posterior superior iliac spine, and greater trochanters were level and in the same horizontal plane bilaterally.

Lumbar active range of motion and associated symptom responses were assessed with the patient in the standing position. The low back and abdominal pain remained unchanged during active range-of-motion testing, and all motions were within normal limits. Because the patient's symptoms remained unchanged with active range-of-motion assessment, overpressure at the end range of active range of motion was applied to lumbar flexion, extension, and bilateral sidebending. The low back and abdominal pain also remained unchanged during overpressure assessment for each movement.

Seated hip active range of motion for flexion, internal rotation, and external rotation was within normal limits and pain free bilaterally. Passive range of motion with overpressure was then applied for hip flexion, internal rotation, and external rotation bilaterally, with the patient lying supine. Passive left hip flexion with overpressure slightly increased the patient's low back pain to 4 on the numeric pain rating scale, but the pain immediately returned to the patient's baseline pain level of 3 when his hip was taken out of the position of left hip flexion with overpressure. The patient did not note a change in his abdominal pain with hip range-of-motion assessment.
With the patient positioned prone, spring testing through posterior-to-anterior pressures over the spinous processes of T10 through L4 suggested mild hypomobility but no increase in the patient's symptoms. However, the patient did report a slight increase in low back pain with spring testing at the L5 level, and there was hypomobility noted at this level as well. A sacral spring test was then performed, which did not change the patient's symptoms. The patient denied any change in his abdominal pain with spring testing from T10 through S1.

The patient was then positioned supine, and an abdominal assessment was performed. Visual inspection of the patient's abdomen did not reveal any skin abnormalities, abdominal masses, or abnormal movement of the abdominal wall. Systematic palpation in each of the 4 abdominal quadrants did not provoke an increase in the patient's symptoms. However, the clinician immediately noticed the presence of a very strong abdominal pulsation that lateralized from the midline bilaterally and was measured at approximately 5 cm in width. The pulsation was palpable from the epigastric to the hypogastric regions. Following this finding, the clinician elected to immediately stop the objective examination and contacted the patient's primary care provider to discuss the findings from the evaluation.

**Clinical Impression Post–Physical Examination**

The patient's low back and abdominal pain was not reproduced with any of the physical examination tests and measures performed, with the exception of a mild, temporary increase in low back pain with overpressure into hip flexion and L5 palpation. In the majority of patients with nonspecific mechanical low back pain, pain is reproduced with lumbar range-of-motion testing and/or lumbar palpation. Therefore, the absence of symptom reproduction during the physical examination further elevated concern for the presence of visceral pathology, which led to a thorough palpation of the abdomen. During abdominal palpation, a very strong, nontender abdominal pulsation at least 5 cm in width was detected, which was concerning for AAA.

**Diagnosis**

After discussing the case, the referring physician immediately ordered an ultrasound imaging study of the abdomen, which demonstrated dilation of the distal abdominal aorta up to 5.5 cm wide (FIGURE 2). The physician subsequently ordered a computed tomography scan confirming the presence of a prominent AAA measuring 5.5 cm in greatest dimension and an 80° angulation (FIGURE 3). The patient was immediately referred to a vascular surgeon. However, after a thorough discussion with the vascular surgeon, the patient decided to forgo immediate surgical intervention, believing that the risks incurred from surgery outweighed the potential benefit. Given this decision, the patient was instructed to return biannually for repeat computed tomography scanning to determine if the AAA was progressing in size. The patient reported that his primary complaint of low back pain and secondary complaint of abdominal pain had completely subsided approximately 3 weeks after the initial onset of symptoms.

Six months following the initial diagnosis of the AAA, repeat computed tomography scanning revealed that the
DISCUSSION

IDENTIFYING INDIVIDUALS WITH AAA is a challenging task for any clinician. There are no established algorithms or clinical prediction rules to determine with reasonable certainty whether an AAA is present in a patient. Additionally, many AAAs are completely asymptomatic, which further complicates the clinical picture. However, there is evidence in the peer-reviewed literature that can inform clinicians in their clinical reasoning and thought process. Knowledge of the risk factors for AAA, systematic application of screening techniques for non-musculoskeletal symptoms, and a basic competence in abdominal palpation and how to interpret findings can help identify AAA in a patient presenting with apparent musculoskeletal complaints.

Understanding the risk factors for AAA can help identify patients who should be referred for further testing. The major risk factors and associated odds ratios for AAA are listed in Table 1. There is discrepancy in the literature as far as when patients should undergo medical screening for AAA. In obese male patients over the age of 60, Ledere et al. recommended ultrasound screening examinations regardless of the physical examination findings. Fleming et al. conducted a systematic review that looked at the benefits and harms of population-based AAA screening. They concluded that screening will reduce age-related mortality in men between the ages of 65 and 75 years. According to Cosford and Leng, an age of greater than 65 years, past history of smoking, male sex, and family history are the most significant AAA risk factors. Therefore, if a male patient in his sixth decade of life has 1 or more additional risk factors for AAA, a referral to the primary care provider with a recommendation for an ultrasound screening examination may be indicated.

In patients with complaint of abdominal pain, the question cluster suggested by Sparks et al. can serve as a valuable clinical decision tool when determining whether a patient has symptoms of musculoskeletal origin. Additionally, the characteristics of abdominal pain can provide insight into the structures responsible for symptoms. Abdominal pain of musculoskeletal origin may present as a deep, sharp, localized, cramping and aching pain. In comparison, pain of non-musculoskeletal origin tends to be dull, continuous, and diffuse. It is important to differentiate between these types of pain as they may require different treatments.

TABLE 3

<table>
<thead>
<tr>
<th>Date</th>
<th>Medical Visits or Procedures</th>
</tr>
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<tbody>
<tr>
<td>Day 1</td>
<td>• Initial physical therapy visit for a primary complaint of low back and a secondary complaint of abdominal pain.</td>
</tr>
<tr>
<td></td>
<td>• Physical therapist informed the patient and the referring provider of the concern of an abdominal aortic aneurysm.</td>
</tr>
<tr>
<td></td>
<td>• Ultrasound imaging and computed tomography scan completed, which confirmed the presence of an abdominal aortic aneurysm.</td>
</tr>
<tr>
<td></td>
<td>• Initial vascular surgery visit.</td>
</tr>
<tr>
<td>Day 17</td>
<td>• Second vascular surgery visit.</td>
</tr>
<tr>
<td>Day 31</td>
<td>• Third vascular surgery visit.</td>
</tr>
<tr>
<td></td>
<td>• Decision made to forego immediate surgical intervention of abdominal aortic aneurysm.</td>
</tr>
<tr>
<td></td>
<td>• Primary complaint of low back pain and secondary complaint of abdominal pain had completely subsided.</td>
</tr>
<tr>
<td></td>
<td>• Patient instructed to complete repeat computed tomography scan in 6 mo.</td>
</tr>
<tr>
<td>Day 204</td>
<td>• Fourth vascular surgery visit.</td>
</tr>
<tr>
<td></td>
<td>• Repeat computed tomography scan results evaluated, which demonstrated interval progression of abdominal aortic aneurysm.</td>
</tr>
<tr>
<td>Day 302</td>
<td>• Open vascular repair of abdominal aortic aneurysm.</td>
</tr>
</tbody>
</table>
arising from visceral tissue is often described as dull, aching, cramping, throbbing, burning, gnawing, or wave-like and is often poorly localized.22

Movement typically alters symptoms associated with musculoskeletal pain.4,5 In contrast, nonmusculoskeletal pain can be characterized as pain that cannot be provoked, alleviated, or eliminated by movement or position changes.4,5,15 If a patient is unable to identify specific aggravating and easing factors and the clinician is unable to reproduce symptoms with provocative testing, then a nonmusculoskeletal origin of pain should be considered and the case discussed with the referring physician.3,32

Abdominal palpation can play an important role in identifying patients with AAA. However, the accuracy of palpation depends on the width of the aneurysm and the patient’s abdominal girth.11 Abdominal palpation has only a moderate overall ability in detecting smaller AAAs, but does appear very useful in identifying aneurysms large enough to warrant elective repairs.11,12,26 The sensitivity of palpation in patients with an abdominal girth less than 100 cm and an AAA 5.0 cm or greater is 100%.11 The patient in this case had a 95-cm waistline and had an AAA greater than 5 cm, resulting in a successful detection of the AAA during the palpation examination. Palpation of an AAA appears to be safe and has not been reported to precipitate rupture.27 If a palpable pulsation of 3.0 cm in width or greater is noticed, physician referral is indicated12; however, it’s important to remember that aneurysms less than 5.0 cm are not as easily identifiable with palpation. Therefore, sole reliance on abdominal palpation is not recommended.11,24

It is important to note that the clinical examination plays a vital role in the recognition of AAAs, but should not be relied on to exclude their presence.29 If a referral to the primary care physician for suspected AAA is indicated, the physician will likely order an abdominal ultrasound, as this can confirm the presence or absence of an AAA (sensitivity, 0.97-1.0; specificity, 0.94-1.0; negative LR, 0-0.025; positive LR, 10.8 to infinity).20,21 Ultrasound screening is not only highly accurate in detecting AAAs but is also low cost and does not expose the patient to radiation.6,22,26 If the presence of an AAA is confirmed with ultrasound imaging, a computed tomography scan is the preferred imaging progression.16,18

While the sensitivity and specificity of computed tomography imaging of the abdomen are lower than that of ultrasound imaging (sensitivity, 0.79-0.90; specificity, 0.77-0.91),26 it can detect additional aneurysms, evaluate the surrounding organs, and provide details on arterial wall characteristics (dissection, rupture, or thickening).16 Computed tomography does have one major disadvantage: it exposes the patient to radiation.16,18 Magnetic resonance angiography yields similar results to those of computed tomography without exposure to ionizing radiation, but high cost and limited availability have prevented widespread adoption of this modality.16,18

CONCLUSION

Although the majority of individuals with AAA are asymptomatic at the time of diagnosis, some patients do present with a history and clinical presentation suggestive of AAA.2 This resident’s case problem described the decision making and clinical reasoning that led a clinician to correctly identify the presence of an AAA in a patient referred from a primary care provider with a diagnosis of mechanical low back pain. Findings from the subjective interview and physical examination led the clinician to suspect AAA as an explanation for the patient’s complaints. This suspicion was confirmed with a series of imaging studies, which led to expedited medical care and eventual surgical intervention.

REFERENCES


