

Clinical Problem-Solving

BACK TO THE BASICS

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A 34-year-old man with paraplegia after a snowmobile accident came to the emergency department with a three-day history of fever, malaise, and abnormally colored urine. Moderate abdominal pain and anorexia had developed one day before his visit. The patient did not have headache, neck stiffness, photophobia, shortness of breath, nausea, vomiting, diarrhea, jaundice, or weight loss.

I need to know more about the character of the abdominal pain. This patient's paraplegia might influence the development and localization of his abdominal pain. The fever, malaise, and abnormal urine color immediately raise the possibility of a urinary tract infection. Patients with pyelonephritis may present not only with abdominal pain but also with an acute abdomen, so his symptoms, including the abdominal pain, could be explained by a urinary tract infection. I would like to know whether he has any flank pain or has had previous urinary tract infections, and how long he has had paraplegia.

The patient had a history of catheter-related urinary tract infections and relied on intermittent self-catheterization for urinary drainage. He said that the present episode differed from his previous urinary tract infections in that he had more abdominal discomfort and anorexia this time. He did not have flank pain, nor did he have a history of nephrolithiasis, hepatitis, pancreatitis, or cholecystitis. The snowmobile accident, which had occurred one year earlier, left him with deficits below T7 on the left side of his body and T9 on the right. A tear of the thoracic aorta had been repaired without grafting after the accident. The patient did not use intravenous drugs or drink much alcohol.

His history of urinary tract infections despite the use of catheterization is not surprising. I immediately wonder about two diagnoses; neither is very likely, but both should be considered. First, trauma to the distal urinary collecting system may have led to a urethral tear. The second and more worrisome possibility is bladder perforation. When the bladder is distended with urine, the wall is much thinner and can readily be perforated by catheterization. If the urine was infected when the bladder wall ruptured, acute peritonitis would result. The fact that the patient has had a tear of the thoracic aorta is important, since this must have been repaired immediately. Infections involving the aorta are more likely to occur after emergency surgical intervention than after elective surgery, as is also the case for aneurysm repair. In addition, damage of any kind to the aorta raises the possibility of a subsequent fistula to a nearby organ. At this point, however, I would focus principally on the possibility of a urinary tract infection.

The patient's temperature was 38.8°C (101.9°F), the blood pressure was 126/68 mm Hg, the pulse rate was 95 beats per minute, and the respiratory rate was 16 per minute. He appeared ill. The oropharynx was without exudates, and the neck was supple. The chest was clear on auscultation; the heart sounds were regular, and there was no murmur. The patient's upper abdomen was diffusely tender, with the greatest discomfort in the right upper quadrant. Palpation of the left lower quadrant elicited pain in the right upper quadrant. There was no rebound tenderness, but the patient did have guarding. No organomegaly was apparent, and bowel sounds were present. On percussion of the back, the patient had tenderness in the right axilla that radiated to the flank. His legs were atrophic, but there was no cellulitis or edema. No splinter hemorrhages or Osler's nodes were seen. A rectal examination revealed decreased tone, no blood, and no masses. Sensation and movement were diminished below the level of T7. The skin was not jaundiced, and there were no rashes or decubitus ulcers.

Given the patient's high temperature, I would have expected a faster pulse rate. Although this is a young man who may have a vigorous cardiovascular system and thus a slower heart rate, a temperature-pulse dissociation may occur with any type of enteric fever or atypical pneumonia, such as Q fever, legionellosis, and chlamydial pneumonia. However, the most

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common reasons for a normal heart rate in a febrile patient are the use of beta-blockers and pacemakers, as well as just normal variation. Thus, this finding may not be useful.

It is of interest that palpation of the left lower quadrant elicits pain in the right upper quadrant, and I wonder whether this is a manifestation of impaired innervation of the abdominal wall resulting from the accident. The search for rebound tenderness as a means of assessing whether peritoneal irritation is present should perhaps be discarded, since it causes discomfort to the patient and produces no clinically useful findings. The examiner can learn just as much by a careful evaluation for localized abdominal rigidity. Because of the marked tenderness in the patient's right upper quadrant, I will assume that peritoneal irritation is a possibility. The presence of bowel sounds indicates that peritonitis, if present, is not advanced.

In this patient, the worst-case scenario would be bladder perforation, with exposure to infected urine leading to generalized peritonitis. However, there are several more probable diagnostic considerations. The first is diverticulitis, though it is rather uncommon in patients of this age. The pain in the right upper quadrant raises the possibility of gallbladder disease. This, too, is relatively unusual in a patient of this age. Given the degree of abdominal tenderness, we must not rule out appendicitis, because it is the most common cause of an acute abdomen at any age. In addition, acute pyelonephritis can cause both abdominal pain and an acute abdomen; thus, this diagnosis still needs to be strongly considered. Finally, an endovascular infection is not an unreasonable consideration, given the patient's history of aortic trauma. My chief concern at this point, however, would be whether he has an acute abdomen. I would be interested in seeing the results of a urinalysis, a complete blood count, and an imaging study of the urinary tract.

The patient's white-cell count was 10,300 per cubic millimeter, with a differential count of 90 percent neutrophils, 6 percent lymphocytes, 4 percent monocytes, and no band forms. The hemoglobin level and hematocrit were normal. A urine specimen obtained by catheterization revealed 25 to 50 leukocytes, occasional red cells, and moderate numbers of bacteria per high-power field. Nitrite was absent, whereas leukocyte esterase was present in trace amounts. The serum electrolyte levels were normal. The blood urea nitrogen and serum creatinine, glucose, bilirubin, alkaline phosphatase, aspartate aminotransferase, amylase, and lipase levels were all normal. The findings on a chest film and electrocardiogram were normal.

Even in patients with bladder perforation, it is often possible to retrieve urine from the bladder. The

characteristics of this patient's urine are quite compatible with those of a urinary tract infection. Acute cholecystitis and cholangitis seem much less likely given the normal serum liver-enzyme levels. At this point, I would assume that the patient has a urinary tract infection, and I would obtain blood and urine cultures and treat him accordingly. Although the abdominal pain is compatible with the presence of acute pyelonephritis, I would proceed very cautiously in view of the timing of his symptoms. I would observe him very closely in the hospital — perhaps even in an intensive care unit — for further peritoneal signs or blood loss.

Intravenous ampicillin and gentamicin were begun for empirical treatment of a urinary tract infection, and intermittent bladder catheterization was continued. The patient's urine culture contained more than 100,000 colony-forming units of *Klebsiella pneumoniae*, but blood cultures had no growth. Urinary abnormalities subsequently decreased with antimicrobial therapy, and a culture of urine obtained after two days of antimicrobial therapy revealed no evidence of bacteria. The patient, however, remained ill. His abdominal pain, anorexia, fever, and abdominal guarding persisted. Bowel sounds remained active. A surgical consultation was requested.

Obtaining a surgical consultation is a very wise course of action. The initial urine culture confirms the presence of a urinary tract infection, but in view of the continued abdominal pain and marked tenderness, I wonder whether the patient's symptoms can be entirely explained by the presence of acute pyelonephritis. I think that urinary tract infection, even upper tract disease, has become increasingly unlikely as the sole cause of his problems. Depending on the surgeon's opinion, I think abdominal imaging would be appropriate. In addition, frequent assessments of the hematocrit, white-cell count, and renal function are in order.

During the next three days, the patient's white-cell count, hematocrit, and serum creatinine level remained stable. Abdominal ultrasonography, abdominal spiral computed tomography (CT), and radionuclide imaging of the hepatobiliary system revealed no clear evidence of cholecystitis, nephrolithiasis, appendicitis, or other abdominal abnormalities. In view of the nonspecific findings, the surgical consultant thought that exploratory surgery was unwarranted.

A normal radionuclide scan in the presence of these liver-function findings effectively rules out acute cholecystitis. A contrast-enhanced spiral CT scan is useful in diagnosing periappendiceal and peridiver-

ticular abscesses and might be expected to identify a collection of fluid if it was present in this patient. I would repeat a rectal examination to determine whether there has been any change, given the possibility of a retrocecal collection of fluid due to a ruptured appendix. The normal ultrasonographic findings essentially rule out nephrolithiasis. However, peritonitis could not be excluded on the basis of any of the tests performed. The surgical consultant apparently thought that the physical findings were not consistent with the presence of peritonitis; otherwise, I would have expected him or her to have recommended surgical exploration.

I am still primarily worried about the possibility of peritonitis caused by perforation of a colonic diverticulum or the appendix. In addition, I remain concerned that the previous aortic injury is related to his current symptoms. At this point, I think one of two diagnostic approaches is in order: perform magnetic resonance imaging of the abdomen with gadolinium-enhanced angiographic imaging or go directly to surgical exploration of the abdomen.

On hospital day 5, the patient's white-cell count increased to 11,700 per cubic millimeter. He was taken for exploratory laparoscopy, and a perforated appendix was found. Postoperatively, his fever and abdominal pain resolved.

COMMENTARY

As Silen has commented, "the vast majority of diagnoses of patients with acute abdominal pain are still made on the basis of a careful history and physical examination. The major delays in diagnosis today are those imposed by the need that some feel to obtain special complicated tests and X-ray examinations."¹ Neither the clinicians caring for the patient under discussion nor the discussant relied exclusively on the results of the sophisticated tests performed, all of which were unrevealing. The history of the patient's present illness and findings on the physical examination both suggested a diagnosis other than a catheter-related urinary tract infection. The correct diagnosis was eventually made during surgical exploration, albeit after some delay.

This delay in diagnosis was caused by several factors, the most important of which was perhaps the presence of abnormal findings on urinalysis in the context of frequent urinary-catheter use. Urinary catheters are a leading cause of infection.² This is especially true for patients with spinal cord injuries, in whom infections related to urinary catheters account for approximately half of all life-threatening bacteremias.³ Though several methods, including intermittent self-catheterization, reduce the risk of catheter-related bacteriuria,^{4,5} this complication has substantial consequences.^{3,6-8} Intermittent catheterization is perhaps the best method of urinary drainage in patients with

spinal cord injuries^{6,9,10} and appears to reduce the risk of bacteriuria, as compared with the use of an indwelling catheter.⁷ However, because the incidence of bacteriuria is about 1 to 3 percent per insertion, bacteriuria develops within a few weeks in most patients.² Furthermore, it is difficult to assess whether the microorganisms often found in the urine of a catheterized patient cause any symptoms, because, as the discussant mentioned, patients with spinal cord injuries may have atypical clinical findings.³

Indeed, the findings in this patient fit a predefined pattern: fever, cloudy urine, and an abnormal urinalysis in a man with paraplegia who required intermittent catheterization should be considered to indicate a symptomatic urinary tract infection until proved otherwise. Nonetheless, the clinicians found two clues that militated against an exclusive diagnosis of urinary tract infection. First, the patient said that he had not had abdominal pain or anorexia during previous urinary tract infections. Second, the degree of abdominal pain and tenderness was out of proportion to that encountered in most patients with urinary tract infections.

One could argue that the diagnosis of appendicitis should have been made before perforation occurred, because appendicitis is common and clinicians have been exhorted to have a low threshold for considering it as a cause of abdominal pain.¹¹ However, the diagnosis of appendicitis can be elusive preoperatively, as attested to by the frequency with which the clinical diagnosis is missed.¹² Furthermore, the physical findings can be confusing in patients with and in those without an intact spinal cord. Most patients with paraplegia retain limited abdominal somatic sensation and are often able to report generalized abdominal pain, but they may not be able to specify its location.¹³ In addition, rigidity of the abdominal wall is not a dependable sign of peritonitis in patients with paraplegia.¹⁴ As we saw in this case, the diagnosis of appendicitis becomes even more challenging in the presence of coexisting conditions that may cause abdominal discomfort, such as urinary tract infection.

What, then, can clinicians do to increase their diagnostic accuracy with respect to patients with paraplegia who are suspected of having an acute abdominal process? Abdominal pain accompanied by anorexia, increased spasticity of the legs, or autonomic dysreflexia (e.g., hypertension, headache, and post-gustatory hyperhidrosis) should alert the physician to the presence of serious abdominal abnormalities.¹³⁻¹⁵ Although nondiagnostic in the case under discussion, imaging techniques may provide objective evidence of an acute abdominal process. Ultrasonography, although of value, has been displaced by the more accurate appendiceal CT.¹¹

The delay in diagnosis in this patient led to appendiceal perforation, a condition associated with a greater risk of postoperative complications than sim-

ple appendicitis.^{16,17} Nevertheless, the correct diagnosis was eventually made and the patient did well postoperatively. It is noteworthy that neither the clinicians who cared for the patient nor the discussant fell into the trap of assuming that the patient had only a urinary tract infection. The clinicians aggressively pursued the possibility of a surgical problem by closely observing him in the hospital, performing several imaging studies, and obtaining a surgical consultation. Finally, both the patient's physicians and the discussant relied on a careful history taking and repeated physical examination to help them chart the proper course of action. But lest we feel too proud, let us remember the words of Sir Zachary Cope: "One often, if not always, learns more by analyzing the process of and detecting the fallacy in an incorrect diagnosis than by taking unction to oneself when the diagnosis proves correct."¹

REFERENCES

1. Silen W. Cope's early diagnosis of the acute abdomen. 18th ed. New York: Oxford University Press, 1991.
2. Warren JW. Catheter-associated urinary tract infections. *Infect Dis Clin North Am* 1997;11:609-22.
3. Kamitsuka PF. The pathogenesis, prevention, and management of urinary tract infection in patients with spinal cord injury. *Curr Clin Top Infect Dis* 1993;13:1-25.
4. Saint S, Elmore JG, Sullivan SD, Emerson SS, Koepsell TD. The efficacy of silver alloy-coated urinary catheters in preventing urinary tract infection: a meta-analysis. *Am J Med* 1998;105:236-41.
5. Saint S, Lipsky BA. Preventing catheter-related bacteriuria: should we? Can we? How? *Arch Intern Med* 1999;159:800-8.
6. The prevention and management of urinary tract infections among people with spinal cord injuries: National Institute on Disability and Rehabilitation Research Consensus Statement: January 27-29, 1992. *J Am Paraplegia Soc* 1992;15:194-204.
7. Cardenas DD, Hooton TM. Urinary tract infection in persons with spinal cord injury. *Arch Phys Med Rehabil* 1995;76:272-80.
8. Saint S. The clinical and economic consequences of nosocomial catheter-related bacteriuria. *Am J Infect Control* (in press).
9. Kuhn W, Rist M, Zaech GA. Intermittent urethral self-catheterisation: long term results (bacteriological evolution, continence, acceptance, complications). *Paraplegia* 1991;29:222-32.
10. Lightner DJ. Contemporary urologic management of patients with spinal cord injury. *Mayo Clin Proc* 1998;73:434-8.
11. McColl I. More precision in diagnosing appendicitis. *N Engl J Med* 1998;338:190-1.
12. Rao PM, Rhea JT, Novelline RA, Mostafavi AA, McCabe CJ. Effect of computed tomography of the appendix on treatment of patients and use of hospital resources. *N Engl J Med* 1998;338:141-6.
13. Miller LS, Staas WE Jr, Herbison GJ. Abdominal problems in patients with spinal cord lesions. *Arch Phys Med Rehabil* 1975;56:405-8.
14. Neumayer LA, Bull DA, Mohr JD, Putnam CW. The acutely affected abdomen in paraplegic spinal cord injury patients. *Ann Surg* 1990;212:561-6.
15. Juler GL, Eltorai IM. The acute abdomen in spinal cord injury patients. *Paraplegia* 1985;23:118-23.
16. Calder JD, Gajraj H. Recent advances in the diagnosis and treatment of acute appendicitis. *Br J Hosp Med* 1995;54:129-33.
17. Velanovich V, Satava R. Balancing the normal appendectomy rate with the perforated appendicitis rate: implications for quality assurance. *Am Surg* 1992;58:264-9.

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