Abdominal Pain

Oct 29 - Nov 2, 2007

Otis Walker is a 57 year old AA male patient who presents to your office on Friday afternoon at 1:50 PM. He complains of abdominal pain that is slowly "getting worse." When asked to describe it further, he says his stomach is "just terrible sick," and he just doesn't know how to better describe it.

1) What kind of questions do you want to ask next?

As you direct him to the examination table, you notice that he is limping also. He states his gout is acting up and has been doing so for several days.

Otis, on exam, doesn't point to or complain of any specific areas of pain except that it is definitely not in the lower abdomen. The current pain is constant. No rebound or guarding. Bowel sounds are normal. As you examine him you coax him into giving a little more history. He hasn't felt good "in his stomach" for days. No reflux, but maybe heartburn. No vomiting and no loose stools. He notes that his urine has been strong smelling, and that sometimes he has had left flank pain over the last few days. He also agrees what pain he has had before today has been episodic. When asked again to describe how bad it was, he states today's pain is different and very bad. He reports that his sugar control has been bad the last few days but he doesn't check his accuchecks often because he doesn't like to stick his fingers. He swears that he is taking all of his medications "just as you told me to," and doesn't think he has skipped any meds despite not knowing all of the names. He denies current alcohol use and denies illicit drug use or smoking.

2) What physical exam components are important?

He has subjective epigastric tenderness with left sided predominance, but no reproducible abdominal tenderness to examination - no rebound or guarding – but is visibly uncomfortable. No umbilical, inguinal, or abdominal wall hernias. Lungs have chronic opening sounds c/w history of smoking. CV: RRR with tachycardia, no overt S1 or S2 murmurs, but chronic lung disease and habitus limits the ability to hear S3 and S4 sounds. Has 2 – 3+ lower extremity edema to the upper calves with chronic skin changes. Genital exam is unremarkable. Rectal exam reveals brown stool (heme negative), normal tone, and no pain. You sit down and look at his intake sheet: Temp 98.9^0 F, BP 189/104, pulse 92, RR 20, O2 SAT 95%. The chart shows his medicines are Avandamet, Amlodipine 10mg, Lisinopril 40mg, Clonidine 0.1mg BID, Atenolol 50mg, Allopurinol, Furosemide, and Simvastatin 20mg. He has a history of diabetes mellitus, hypertension, recurrent gout, kidney stones, sleep apnea and morbid obesity. No prior surgeries. Unfortunately, his abdominal pain exam appears non-specific.

3) What are you going to do next?
   a) Draw some labs and send him home?
   b) Give him some PPI samples and send him home?
   c) Admit him to the hospital and observe him?

d) Send him to the ER for further evaluation?

You decide to go ahead and send him to the hospital emergency room based on the ***clinical impression*** that he is sick ***and at risk*** knowing that labs may not be very helpful in establishing the diagnosis and that linear observation is the primary responsibility at this point (while other work-up is performed). Hospital admission is a possibility, but insurance likely will require a higher proven acuity, which can be developed/confirmed in the ER.

Two days later, you are consulted at the hospital by the general surgery service to assist with the difficult to control HTN and diabetes management of Mr. Walker. You find out that he had a number of tests performed: CBC, UA, CMP, EKG, CXR, HA1-c, blood culture (1 set), uric acid, urine culture, CT abdomen with stone protocol, USG abdomen, UDS, amylase, lipase, lipid profile, protime. The computer shows that his WBC count is elevated in the 14.6 to 16.3 range since admit. HCT is noted to be 34.1 dropping to 32.4 today. Other CBC indices are ok. LFT's range 2 – 15 points above normal range. The UA had 20 – 30 RBC's, LE+, nitrite -, Bact 0. Bun was 22, creatinine was 1.6, and the bicarb was 20 on admission but normalized on subsequent labs. The metformin from home was correctly not resumed on the admission orders. Glucose was consistently 285 to 400 on labs and accuchecks, but may be improving today to 210. Uric acid was 10.1 (no surprise). HA1-c at entry was 6.2. Everything else on the lab, and the radiologic tests, were negative.

The patient feels a little better today, but has had orthopnea after the IVF at admit (with resultant subsequent Lasix dosings). His pressure control has not significantly improved since your office visit. The surgery resident has placed him empirically on Levaquin, but all cultures are negative (urine and blood). They theorize urinary tract infection +/- left ureteral calculus, already passed. It would appear to them that the patient is likely not compliant with his meds at home. He is also not eating correctly and his diabetes is thus not under control – after all, we see a lot of patients with this presentation around here. They want to discharge him.

4) Any additional ideas about what is going on?

My purpose with this presentation is to make several points. First is that abdominal pain often cannot be diagnosed easily and a complete history and abdominal exam are the **most** useful parts of the work-up, **not** the labs. The second point is that placing a patient in an environment where they can be protected, serially examined, and have other procedures done at the same time is **not** dependent on labs, and is essential if the patient is at high risk as determined by your interview and exam. Third, this is a two armed cautionary tale – to be careful to keep an open mind and an open differential until the diagnosis is made (and check EVERYTHING you ordered) and to not profile the patient.

5) What do you mean, profile?

(*end of the question area of the sheet*)

Abdominal Pain Answer Sheet

1. This is a difficult subject because there often is no right answer. A lot of what you are trying to accomplish is trying to protect the patient from harm. Here are some high yield questions that delineate increased risk:

    1. ***How old are you?*** Advanced age means increased risk.
    2. ***Which came first—pain or vomiting?*** Pain first is worse (i.e., more likely to be caused by surgical disease).
    3. ***How long have you had the pain?*** Pain for less than 48 hours is worse.
    4. ***Have you ever had abdominal surgery?*** Consider obstruction in patients who report previous abdominal surgery.
    5. ***Is the pain constant or intermittent?*** Constant pain is worse.
    6. ***Have you ever had this before?*** A report of no prior episodes is worse.
    7. ***Do you have a history of cancer, diverticulosis, pancreatitis, kidney failure, gallstones, or inflammatory bowel disease?*** All are suggestive of more serious disease.
    8. ***Do you have human immunodeficiency virus (HIV)?*** Consider occult infection or drug-related pancreatitis.
    9. ***How much alcohol do you drink per day?*** Consider pancreatitis, hepatitis, or cirrhosis.
    10. ***Are you pregnant?*** Test for pregnancy—consider ectopic pregnancy.
    11. ***Are you taking antibiotics or steroids?*** These may mask infection.
    12. ***Did the pain start centrally and migrate to the right lower quadrant?*** High specificity for appendicitis.
    13. ***Do you have a history of vascular or heart disease, hypertension, or atrial fibrillation?*** Consider mesenteric ischemia and abdominal aneurysm.

    From Colucciello SA, Lukens TW, Morgan DL: Abdominal pain: An evidence-based approach. Emerg Med Pract 1:2, 1999.

2. "The objective evaluation begins with measurement of the vital signs. Significant tachycardia and hypotension are indicators that shock may be present. Tachypnea may be an indication of metabolic acidosis from gangrenous viscera or sepsis, hypoxemia from pneumonia, or simply a catecholamine-induced reaction to pain. Elevated temperature is often associated with intraabdominal infections. However, fever does not accurately predict significant abdominal pathology. For example, the temperature is often normal in elderly patients with laparotomy-proven intraperitoneal infections.

The abdomen and pelvis are examined to identify the area of maximal tenderness, anticipating some correspondence with the location of the diseased organ. This can be true, but it is often not the case. Although 80% of suspected appendicitis cases manifest right lower quadrant abdominal tenderness, 20% of patients with proven appendicitis do not.

Rectal examination may have limited use in abdominal pain, except when associated with intraluminal gastrointestinal hemorrhage, prostatitis, and perirectal disease. Its main utility is in the detection of heme-positive stool. Rectal examination has not been shown to increase diagnostic accuracy for appendicitis when added to external physical examination of the abdomen.

The abdominal evaluation should include a pelvic examination in female patients with lower abdominal pain or an otherwise uncertain diagnosis. Male patients should receive a genital examination as well as evaluation for the presence of inguinal or femoral hernias.

Given the evolving nature of abdominal pain, repetitive examinations may be used. This is common practice with respect to suspected appendicitis and has improved the diagnostic accuracy in patients whose presentations were atypical."

Chapter 22 – Abdominal Pain, Marx: Rosen's Emergency Medicine: Concepts and Clinical Practice, 6th ed.

3. You might contemplate answer a), but consider this:

"Complete blood counts are frequently ordered for patients with abdominal pain. Despite elevated white blood cell (WBC) counts being associated with many infectious and inflammatory processes, the WBC count is neither sufficiently sensitive nor specific to be considered a discriminatory test to help establish the cause of the abdominal pain. Even serial WBC counts have failed to differentiate surgical versus nonsurgical conditions. The WBC count is, therefore, not helpful for diagnosis. Serum electrolytes, even in the presence of protracted emesis or diarrhea, are abnormal in less than 1% of patients. These studies are not indicated for most patients in the absence of another indication.

Despite the significant variety of tests available, close to one half of the patients presenting to the emergency department [or office for that matter] with acute abdominal pain have no conclusive diagnosis…" [based on the labs and examination at the time of first evaluation].

Chapter 22 – Abdominal Pain, Marx: Rosen's Emergency Medicine: Concepts and Clinical Practice, 6th ed.

d) should be the answer, and beyond personal opinion, we'll look to some support in the same textbook of emergency medicine:

"Because up to 40% of patients presenting with acute abdominal pain receive the diagnosis of nonspecific abdominal pain, the dispositions of patients with abdominal pain can be as difficult as their diagnoses. Categories for disposition may include surgical versus nonsurgical consultation and management, admission for observation, and discharge to home with follow-up evaluation. The decision to admit a patient to an observation unit or a hospital bed must factor in the

following: 1) information gained from the history, physical examination, and test results, 2) the likelihood of any suspected disease, 3) any potential ramifications, if a known disease progresses or the patient is incorrectly diagnosed or managed, and 4) whether follow-up evaluation can occur in a timely manner if the patient is discharged home.

Clinically stable patients may be discharged from the emergency department [or office] with appropriate follow-up care, possibly to include repeated or additional diagnostic imaging.

In the case of nonspecific abdominal pain that is considered potentially worrisome, it is prudent to have the patient reevaluated after 8 to 12 hours. This can be done through a return visit to the emergency department, an appointment with a primary care physician, or an observation unit protocol."

Chapter 22 – Abdominal Pain, Marx: Rosen's Emergency Medicine: Concepts and Clinical Practice, 6th ed.

4. Did you remember to check the EKG tucked neatly into the cardiology section in the back of the chart? Because there you will find an admission EKG that has "tombstones" all across the anterior leads with an automated computer reading of acute MI. This happens in real life… just like this. And it makes sense retrospectively: intermittent pains over days leading to an early AM much bigger event. Acute gout – which neither causes nor heralds heart problems, but is a common co-morbid condition that tends to flair with elevation of blood pressure, increased purine production, acidosis, and diminishment of renal function. The gout or the AMI can trigger the WBC elevation. Diabetics often have atypical chest pain, and the AMI can trigger hypertension and very difficult to control diabetes. And it all gets better with time (and a little Lasix) if there are no arrhythmias.

5. To some, this case would be a good example of a form of racism. *Or* bias at least.

The medical residents in an inner city training program can close their eyes and profile this patient – overweight, diabetic, hypertensive, gout, older. Then overlay the presumptions of uneducated, non-compliant, using drugs/alcohol/cigarettes, and poorly interactive with follow-up. And these patients very often have dark skin. Although I have been unable to locate the specific source where I have read it, this learned (+ or -) profiling is not limited to Caucasian doctors.

The issues are not simple and straightforward, however. Let's look (with a limited scope) at some of these issues and/or preconceptions in the evaluation and treatment of acute coronary syndromes (ACS) in African Americans (AA). First, AA patients may have a different perception of coronary syndromes as they begin:

"African American patients were significantly more likely to perceive their symptoms at onset as serious (needing medical attention) or life-threatening than were white patients (P = .05). Self-assessment of pain or discomfort, on a 0 (none) to 10 (worst) scale, showed that median discomfort level of African American patients (8, interquartile range 5.5 - 10) was significantly higher than that of white patients (7, interquartile range 4.5-9) (P =

.023). . . Patient attributions of symptoms at onset were categorized into predetermined responses of heart, indigestion (stomach, gas, heartburn, related to food), muscle pain (strain, bursitis, arthritis, injury), fatigue, or other (respiratory, emotional, medication-related). For the purposes of analysis, fatigue and other were combined and recategorized as noncardiac. African American patients were significantly more likely than were white patients to attribute their symptoms to their stomach or a noncardiac origin or were unable to identify cause. White patients were more likely than African American patients to attribute their symptoms to their heart or musculoskeletal pain ($P = .05$). In secondary analysis, attribution was categorized as heart, stomach, or other (combined muscle pain, noncardiac, and other categories) for those having been found to have had MI. African American patients were more likely to attribute their symptoms to the stomach ($P = .05$), and white patients were more likely to attribute their symptoms to the heart ($P = .05$).

Am Heart J. 2002 Jul;144(1):51-9.

This fits pretty well with Mr. Walker's presentation: vague indescribable pain attributed to GI problems. But he still has a multitude of risk factors and many diagnoses needs to be considered. What about the possibility of being uneducated? In a study in the VA system, patients that *knew* they had coronary disease were reviewed in regard to their time to presentation when they developed symptoms of heart problems and "race was not associated with delay in seeking care among patients awaiting coronary angiography." (Med Care. 2002 Jan;40(1 Suppl):I97-105.) There are other articles that speak to the opportunities to better educate AA's as to the presentation of acute coronary syndromes, but once they know, there is no apparent disparity in presentation for this reason. Maybe the fact that he is a poor person will cause delays in presentation. In fact, in a study of AA patients who had a documented MI, "delay was longer for insured patients than for uninsured patients (4.45 vs 0.50 hours)." (Am J Crit Care. 2006 Mar;15(2):149-57.)

When AA patients get to the hospital with a possible ACS, "the first screening test for acute coronary syndrome, the ECG, took longer to obtain for nonwhite patients, regardless of final diagnosis." (Acad Emerg Med. 2006 Aug;13(8):867-72. Epub 2006 Jun 26.) Multiple studies have documented delays to lab evaluation, IV therapies, and angioplasty. "Multivariable analysis showed that patients who presented to the emergency department with acute cardiac ischemia were more likely not to be hospitalized if they were women less than 55 years old (odds ratio for discharge, 6.7; 95 percent confidence interval, 1.4 to 32.5), were nonwhite (odds ratio, 2.2; 1.1 to 4.3), reported shortness of breath as their chief symptom (odds ratio, 2.7; 1.1 to 6.5), or had a normal or nondiagnostic electrocardiogram (odds ratio, 3.3; 1.7 to 6.3). Patients with acute infarction were more likely not to be hospitalized if they were nonwhite (odds ratio for discharge, 4.5; 95 percent confidence interval, 1.8 to 11.8) or had a normal or nondiagnostic electrocardiogram (odds ratio, 7.7; 95 percent confidence interval, 2.9 to 20.2)" (N Engl J Med. 2000 Apr 20;342(16):1163-70.)

What about the perception of non-compliance with medicines and follow-up among AA patients? In a study performed at the University of South Florida, this belief among medical providers was directly tested. "Contrary to our hypotheses, Blacks and

Hispanics engaged in preventive care more frequently than Whites. Whites were less likely to have seen a doctor in the previous year, less likely to have had a foot exam, more likely to smoke, and less likely to have attempted smoking cessation. Persons of lower social class were at greatest risk for not receiving preventive care regardless of race/ethnicity. Persons with no health care coverage were twice as likely to have not visited the doctor in the previous year and twice as likely to have not had an eye exam, 1.5 times more likely to have not had a foot exam or attempted smoking cessation." (BMC Public Health. 2006 Oct 19;6:259.) It would appear that this is more of a socioeconomic problem than one limited to skin melanin content.

These above observations are not made to fully delineate the disparities in treatment of ACS in African Americans. They are presented to begin to challenge your assumptions. At best, a doctor's presumptions about a person because of the "mold" they fit may delay diagnosis. At worst it may cost them their life. Mr. Walker's history told us something was wrong. He told us his sugars were up acutely. He indirectly told us something was making his gout flare. He told us that he was taking all of his medicines correctly (his admission HA1-c was 6.2, and his heart rate was up not because he didn't take his Atenolol), and that he was neither smoking, drinking, nor using drugs. But our preconceptions can help us ignore the real history and findings. Close your eyes and imagine that I started out describing a Billy Smithson, a 57 yo WM . . . would it have changed, *honestly*, any of your presumptions?

As a neat activity to get a better handle on where your intrinsic leanings may skew, whether for race or age or ethnicity, go to www.implicit.havard.edu and participate in their online research - "Project Implicit" – that hopes to further evaluate and educate the general society about their tendencies. Anything you do to think about and work with these issues should make you a better doctor.