A 38-Year-Old Man With Severe Abdominal Pain

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A 38-year-old man presents with several days of ongoing severe abdominal pain in his upper abdomen that radiates to his back. He describes the pain as "boring and unrelenting" and is not crampy or colicky.

History

There is little he can do to ameliorate the pain, although he has found that lying on his side with his legs flexed is least uncomfortable for him. He in unable to keep down any foods or liquids since onset because of increased pain when he tries and emesis. The latter had onset a day or so after the pain started and has been unrelenting with 5 to 10 episodes per day.

When questioned, he relates similar pain but of much milder nature several times in the last year or two, which would resolve in a day or two when he "babied his stomach" with clear liquids only. He related no other symptoms and has not been febrile.

His medical history is noncontributory. His only medications are occasional acetaminophen and ibuprofen for minor muscle and joint symptoms. He works for a landscaper. When he was younger, he sporadically used illicit drugs but has been sober for many years. He is and has been a heavy drinker since he was a teenager, with particularly heavy alcohol intake—both liquor and beer—on weekends.

Physical examination

He is non-icteric but manifests profound dehydration with parched mucosae, has tachycardia with a heart rate of 108 beats/min at rest, and has a blood pressure of 95/60 mm Hg supine. There are no spider telangiectasias or ascites.

The only significant finding was a quiet abdomen with significant guarding but no rigidity. There is exquisite tenderness to any direct palpation to his mid- and upper epigastrium with radiation to his back. There was no tremor or fasciculation of the tongue, and he was oriented to place, person, and time.

Diagnostic testing

Results of STAT basic laboratory testing showed profound hypovolemia with a blood sodium level of 132 mEq/L (reference range, 135-145 mEq/L), creatinine level of 2.1 mg/dL, and a blood urea nitrogen level of 40 mg/dL (reference range, 6-24 mg/dL). His hemoglobin level was 15 g/dL (reference range, 13.5-17.5 g/dL), a white blood cell count of 17,000/µL (reference range, 5000-10,000/µL), and serum lipase level of 670 U/L (reference range, < 160 U/L). An abdominal ultrasound was negative for gallstones and otherwise noncontributory.

After 6 hours of aggressive fluid resuscitation using Ringer's lactate solution, there was improvement in the patient's volume status and metabolic panel values, such that he could safely undergo a thin-slice abdominal computed tomography scan. Results of which demonstrated severe peripancreatic stranding with small areas of pancreatic hypoperfusion (necrosis) and several small areas of fluid collection.

Which of the following is the most accurate early treatment principle in this patient?

A. Broad-spectrum prophylactic antibiotics should be initiated.

B. A procedure to address the fluid collections is required.

C. Early use of parenteral nutrition is required to prevent complications and hasten recovery.

D. Enteral feeding within 24 to 72 hours should be initiated.

E. Oral indomethacin therapy will significantly address and ameliorate the underlying cause of the illness.

Correct Answer: D. Enteral feeding within 24 to 72 hours should be initiated.

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The patient is manifesting findings that are adequate to make the diagnosis of acute pancreatitis, which is a condition caused by inflammation of varying degrees and intensity within the pancreas. He fulfills all 3 clinical criteria of diagnosis: characteristic abdominal pain in the epigastric area that is constant (rather than peristatic, colicky, or crampy); elevated blood levels of the pancreatic enzymes, either amylase or lipase; and imaging abnormalities, which will be described below. Two of these three criteria need to be present to make the clinical diagnosis.

Acute pancreatitis remains common in the United States, perhaps second only to appendicitis, as a cause for admission to hospital care for abdominal pain and is important as mortality rates still approach 5%.[1] The demographics and epidemiology are well known, with by far the 2 main underlying causes being heavy alcohol consumption and gall stone disease. Pathophysiologic theories for causation by the former suggest a direct toxic effect of alcohol on the pancreas, while presumed mechanisms for the latter include obstruction at the ampulla of Vater and sphincter of Oddi causing backflow of enzymes under pressure into the pancreas and/or reflux of bile into the pancreas.[2] A variety of less common causations are more often found on Board examinations than in the clinic, which include severe hyperlipidemia, Types IV and V with very high triglyceride levels, and medications, most common being HIV therapies. Finally, an important iatrogenic etiology, which is actually an adverse event, is post-endoscopic retrograde cholangiopancreatography (ERCP) pancreatitis, which is not surprising given the location of the procedure. These are the historical points that must be probed when encountering a suspected case of acute pancreatitis.

As I have alluded to, the clinical diagnosis involves at least 2 of 3 rather mundane criteria of pain, elevated pancreatic enzyme, and imaging of the presentation. Abdominal pain will almost always be the first, which is usually quite classic in location—epigastric, and nature—constant "boring pain" with radiation to the back. Both the pain and frequently associated emesis will be worsened by oral intake. It should be noted that acute pancreatitis can figuratively behave like an intra-abdominal burn that, when coupled with the emesis and decreased oral intake, results in a profoundly dehydrated patient with many signs of hypovolemia. Easily and quickly obtained laboratory parameters will demonstrate hemoconcentration and elevated blood urea nitrogen/creatinine consistent with hypovolemia. And since just about any abdominal pain visit prompts the testing for serum amylase and lipase in the emergency department or urgent care facility, the second major criterion for the diagnosis is also quickly and easily obtained.

Here are several important literature-based facts concerning serum amylase and lipase. To be considered significant, the values must be at least 3 times the normal limit. Amylase is sensitive but of lower specificity, while lipase levels also remain in plasma longer so are more sensitive and specific in alcohol-related cases and in patients seen later in their illness. Finally, and very important, is that the absolute levels or serial monitoring is not related to either the etiology, severity, or prognosis of cases of acute pancreatitis.[1,3] The third and final diagnostic criteria is a positive imaging study that, unlike enzyme studies, frequently can implicate causation and demonstrate findings predictive of severity and complications. The entry-level noninvasive study is right upper quadrant ultrasonography that can show gallstone disease in a patient with pancreatitis. A better image of the pancreas itself is provided by a thin-slice contrast computed tomography scan, which really enumerates the pancreatic pathology that is present (eg, presence and severity of intrapancreatic edema, necrosis, or pseudocyst formation).[1] Using these 3 clinical pillars for essentially every case of acute pancreatitis can be correctly diagnosed with most having the causative etiology determined and a rough degree of severity, hospital admission requirements, and prognosis being in place.

The questions asked are related to early management and will be addressed below. But a brief discussion of prognosis schemes needs to be had to more accurately answer them. For our purposes, I will mention 2 useful and time-tested schemes: the classical Ranson Criteria for Pancreatitis Mortality[4,5] and the current standard of care, the Revised Atlanta Classification for Acute Pancreatitis.[1] As mentioned above, the mortality rate of pancreatitis is 5%, but we now know there is a bifurcated nature of these deaths—an early peak in the first week, then a second peak 2 to 6 weeks out. The early peak results from acute insult to the body and multiorgan failure, while the second peak usually results from more localized issues in the pancreatic anatomy itself, such as fluid collections, intra-abdominal infections and thromboses.[1,5] The Ranson criteria more represents early morbidity and mortality and, at the 48-hour mark, includes:

- A white blood cell count of more than 18,000/μL
- Age older than 55 years
- A glucose level of more than 200 mg/dL
- An aspartate aminotransferase level of more than 250 U/L
- A lactate dehydrogenase level of more than 350 U/L upon admission
- An hematocrit level drop of more than 10%
- A blood urea nitrogen level increase of more than 5 mg/dL
- A calcium level of less than 8 mg/dL
- A partial pressure of oxygen of less than 60 mm Hg
- A base deficit level of more than 4 mg/dL
- The need for more than 6 L of fluid

One point is assigned for each criterion, and as the total increases, so does the predicted morbidity and mortality.[4] As can be seen, the Ranson criteria is most

powerful in the acute parts of the disease. The Atlanta Classification deals with the acute 48-hour organ failure issues but adds more items that address delayed risk factors, which are now known to heavily involve local complications that can be accurately identified with modern imaging techniques. These include pancreatic necrosis and fluid collections, pseudocyst formation, and intra-abdominal vascular thromboses.[1,5] The Atlanta Classification is the current standard of care.

Just as prognostic classification has evolved over time, so have the therapeutics for acute pancreatitis. The question and pool of possible answers relate to the current therapeutics for acute pancreatitis and will be addressed in detail below. Since gallstones are now one of the 2 major causative epidemiologies, studies on relaxing the sphincter of Oddi were performed. Indeed, there is a role for indomethacin, a sphincter-relaxing agent, in defined circumstances. However, the arena of use for this maneuver is limited to acute pancreatitis with demonstrable gallstone disease or with the use of an ERCP maneuver to treat it, which has up to a 15% incidence of post-ERCP pancreatitis.[6] Further, the literature reports rectal, rather than oral, administration of the indomethacin.[6] So, Answer E is incorrect both in its indication and route of administration aspects.

Another long-term issue in acute pancreatitis is whether to use or not to use prophylactic antibiotics to prevent the very serious and dangerous complication of pancreatic abscess from seeding of a fluid collection or necrotic area. Although the data remain fluid and not totally definitive, prophylactic antibiotics are not recommended in the absence of ascending cholangitis from gallstones or microbial confirmation (eg, needle aspiration) of infected fluid collections[1,2], making Answer A incorrect. Along similar lines, the demonstration of fluid collection necrosis or even pseudocyst formation early in the disease process, which has markedly increased with the advent of imaging technological advances, has not been shown to be a benefit.[7] Most fluid collections resolve with time, and the old clinical saying of waiting 3 to 4 weeks to allow encapsulation to occur if fluid does not resolve before attempting procedure of aspiration or debridement is once again true. Our patient is early in the disease course, and his multiple small fluid collections do not yet require a procedure to address them, making Answer B incorrect.

Answers C and D remain and relate to yet another traditionally debated issue in acute pancreatitis, namely when and how to feed such patients. There is good literature to support the following schema. In milder cases, the traditional clinical points of resolved pain, nausea, and vomiting, as well as the return of bowel sounds, are the indicators that cautious feeding with liquids and low-fat solids can begin and will be tolerated. I was taught, and then did teach similarly on the wards, that when both the bowel sounds and patient were making noise about being hungry, it was then time to feed them. I may have been a bit slow on the trigger and might currently upset the utilization crew, but my patients did not suffer very many relapses and readmissions. In those more difficult patients who cannot tolerate a bona fide oral intake diet, there is broad agreement that enteral methods are superior to parenteral methods, surprisingly because of a lower infection incidence and morbidity more so than actual gastrointestinal issues.[1] It seems that the nasogastric or nasojejunal routes are equivalent. However, again, aggressive maneuvers did not translate into superiority in outcome mortality or complication rates. On the positive side, early enteral feeds seem to cause no excessive harm and may shorten the hospital stay.[8] Most patients will be able to tolerate oral feedings by 72 hours in any event.

In summary and deference to the aforementioned utilization review, the

TAKE-HOME MESSAGE

Acute pancreatitis remains a very common cause for hospital admission for abdominal pain and still carries a significant morbidity burden and even a 5% mortality risk. The 2 most common, by far, epidemiologic causative risk factors are excessive alcohol use and gallstones. Far less common etiologies to be considered are severe hypertriglyceridemia; medications, especially those used to treat HIV; and status post-ERCP. The triad of diagnosis is the presence of typical severe upper abdominal pain with radiation to the back, elevated levels of serum amylase or lipase, and imaging studies demonstrating any of the constellation of pancreatic inflammation, necrosis, and/or fluid collections. A variety of prognostic schemes exist to adjudicate severity/complication and mortality risk, including the older Ranson Criteria and newer Atlanta Classification, which is now currently the standard of care.

Therapeutics involve prompt and aggressive fluid resuscitation to reverse the profound and dangerous hypovolemia in these patients. Early (within 48-72 hours) enteral feedings of varying routes are sager and of mortality benefit compared with parenteral feedings. Further early aggressive maneuvers such as prophylactic antibiotics and performing a procedure on the pancreas to address necrosis (debridement) or fluid collections are neither helpful nor indicated.

order of battle seems to be (1) enteral feedings are essentially always superior to parenteral feedings; (2) early use of enteral feeding is suggested if oral intake is not tolerated to reduce hospital stay; and (3) the goal is an oral regimen by hospital day 3 or 4. Therefore, Answer D is the best option offered here.

Patient Follow-Up

The attending physicians concluded that the patient was at moderate risk for necrosis and elected to start enteral feeding using the nasogastric route on day 2. Meanwhile, fluids and supportive care were continued. His symptoms steadily improved, with the emesis and abdominal pain resolving over the next 48 hours. Cautious low-fat, oral nutrition was initiated and tolerated such that he was discharged on hospital day 6. An appointment at an alcoholic support center, as well as with his medical physician, were arranged.

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