CLINICAL CASE OF THE MONTH

A 35 Year Old Woman with Abdominal Pain

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CASE REPORT

A 35 year old woman with past medical history of hypertension presented to the emergency department with chief complaint of severe abdominal pain for one week. The abdominal pain was located in the epigastrium and described as "cramping" and "intermittent". The pain intensity was quantified initially as 6 out of 10 on the pain scale. As the week progressed the pain became constant and radiated to the back. The intensity of the abdominal pain increased to 10 out of 10. The patient reported some relief from her pain while lying in the prone position. Initially the pain was associated with loose stools for several days. The loose stools resolved spontaneously and then the patient began to experience nausea and vomiting. Her medications included lisinopril-hydrochlorothiazide which she had been taking for the past five months. She had no history of alcohol, tobacco or illicit drug use.

On physical examination, the patient's temperature was 99.4°F, blood pressure was 136/67 mmHg, heart rate was 115 beats per minute, and respiration rate was 18 /minute. The patient was five feet and three inches tall, weighed 182 pounds and her BMI was 32.2. On physical exam the patient appeared in distress secondary to pain and was lying in the prone position. Abdominal exam revealed a non-distended abdomen with normal bowel sounds. The patient was tender to palpation in the right upper quadrant and epigastric areas but without rebound or guarding. Periumbilical bruising was present.

Upon admission laboratory data showed: WBC 14.5 103 U/L (4.5-11.0 103U/L) and hemoglobin 12.6 g/dL (12.0-16.0 g/dL), hematocrit 39.8% (35-46%), lipase 4858 U/L (<61 U/L), ALT 317 U/L (<46 U/L), AST 267 U/L (<45 U/L), total bilirubin 3.0 mg/dL (<1.3 mg/dL), Alkaline phosphatase 204 U/L (20-120 U/L), BUN 15 mg/dL (7-25 mg/dL), Creatinine 1.14 mg/dL (0.50-1.10 mg/dL), calcium 9.0 mg/dL (8.4-10.3 mg/dL), and triglycerides 89 mg/dL (< 150mg/dL). An acute hepatitis panel was negative and HIV-1 and HIV- 2 antibody serologies were non-reactive. A CT scan of the abdomen and pelvis with IV contrast noted the pancreas to be severely swollen with decreased enhancement of the head and body secondary to with a large amount of surrounding inflammatory changes (see Figure 1). Abdominal ultrasound showed a decompressed gall bladder with a large gallstone measuring 2.8 x 1.9 cm (see Figure 2). A triple phase CT of the abdomen and pelvis was performed the following day, which revealed acute necrotizing pancreatitis involving 60-70% of the pancreas (see Figure 3).

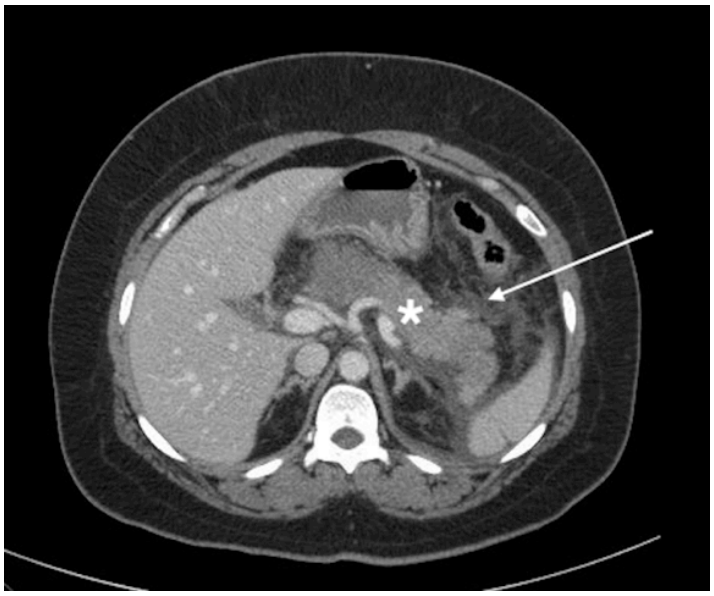

Figure 1. Contrast-enhanced CT of the abdomen upon admission demonstrates swelling of the pancreas (*) with extensive surrounding inflammatory change (white arrow) consistent with pancreatitis.

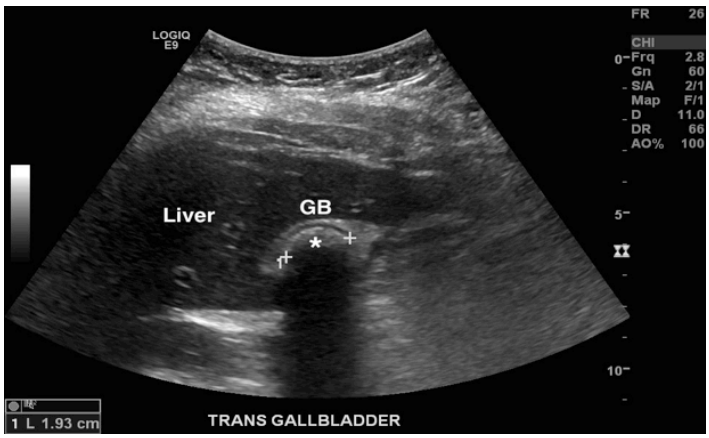

Figure 2. Ultrasound of the right upper quadrant upon admission demonstrates a large shadowing gallstone (*) within the gallbladder (GB) without evidence of acute cholecystitis.

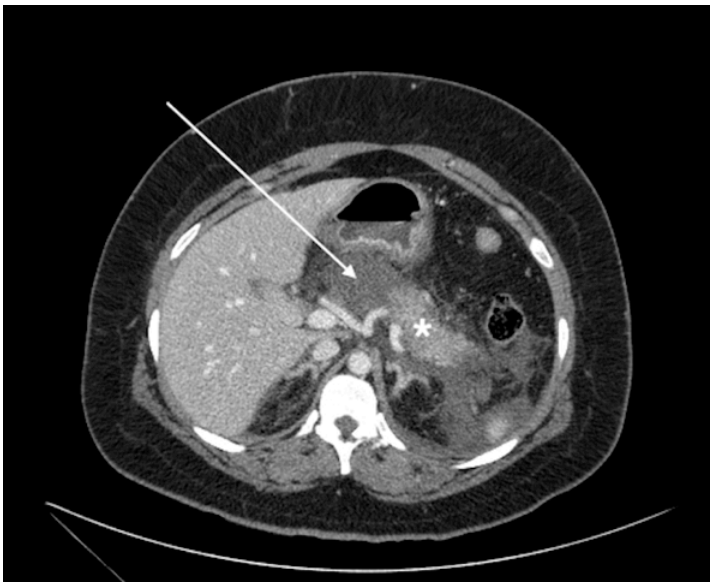

Figure 3. Multiphase CT of the abdomen performed on day 2 of hospitalization demonstrates changes of severe pancreatitis with diminished enhancement of the uncinate process, head, distal body (white arrow), and tail of the gland consistent with necrosis. The proximal body (*) demonstrates normal enhancement as seen in viable tissue.

The patient was treated with IV fluids, analgesics and bowel rest. The patient developed a fever on the second hospital day with persistent tachycardia, worsening leukocytosis to 20.0 X 103U/L (4.5-11.0 X 103 U/L) and persistent abdominal pain. Intravenous imipenem/cilastatin was initiated. Blood and urine cultures remained negative throughout hospitalization. On hospital day 3, a nasojejeunal feeding tube was placed. The patient remained febrile and triple phase CT performed on the sixth hospital day showed stable necrotizing pancreatitis with interval development of a 6.6 cm peri-pancreatic fluid collection likely representing a pseudocyst. Interventional radiology was able to aspirate 130 cc of brown turbid fluid from the pseudocyst (See Figure 4).

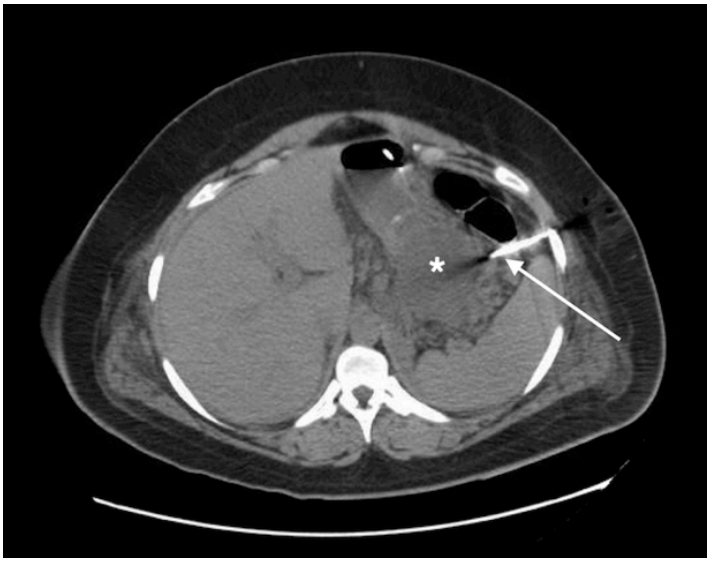

Figure 4. CT-guided drainage performed on day 7 of admission. A 6.6 cm fluid collection (*) superior to the tail of the pancreas was aspirated using an 18 G Chiba needle. The needle is represented by the linear, metallic-density structure inserted percutaneously into the left upper quadrant (white arrow). A total 130 cc of brown turbid fluid was obtained.

No organisms were detected on gram stain of the aspirated fluid and the culture was negative for bacterial growth. Antibiotics were discontinued at this time. The patient began to show clinical improvement following aspiration of peripancreatic fluid. Nasojejunal feeds were discontinued on the tenth hospital day and her diet was advanced. On the fourteenth hospital day the patient underwent cholecystectomy without complication. She was discharged to home a day later in stable condition.

DISCUSSION

Incidence and Epidemiology

Acute pancreatitis is a common cause of hospitalization in the United States, and was listed as the admission diagnosis in 4.6 of every 1000 hospitalization in the United States from 1988-2003 (1). As a discharge diagnosis, it increased 62% between the years 1988 to 2004. In 2004, it was reported to be the 7th most common cause for hospitalization for digestive diseases. The mean length of hospitalization is estimated at 6.9 days with an overall mortality of 2.0%.[2]

ETIOLOGY

The two most common causes of acute pancreatitis (80%) are biliary ductal stones and alcohol use. Hypertriglyceridemia, hypercalcemia, infection, ischemia, toxin/drugs, and trauma account for 10% percent of cases. The final 10% of cases are considered to be idiopathic.[3] Gallstone pancreatitis is more common in patient populations that are elderly, female or Caucasian (non-Hispanic).[4] The mechanism behind gallstone pancreatitis is thought to be the repeated transient obstruction of the ampulla of Vater by migrating gallstones leading to fibrosis of the sphincter of Oddi. This fibrosis may allow duodenal fluid to reflux into the pancreatic duct which over time leads to pancreatitis.[3]

DIAGNOSIS

The diagnosis of acute pancreatitis can be made when two of the following three criteria are met. The first criterion consists of abdominal pain that is suggestive of pancreatitis. Typically, this is described as a steady, sudden-onset pain that radiates to the back, usually associated with nausea and vomiting. The second criterion includes a serum amylase and/or lipase greater than three times the upper limit of normal. The numerical value of these enzymes has no prognostic value and does not reflect the severity of the disease. The third criterion lists the presence of characteristic abdominal imaging findings. The imaging may be either contrast-enhanced computer tomography (CT), magnetic resonance imaging (MRI), or ultrasound (U/S). CT is currently considered the best image modality.[5]

Classification of Pancreatitis – Phase, Category, and Severity:
Pancreatitis can be classified based on the phase of the disease (early or late); the category (necrotizing or not) and the severity (mild, moderate or severe).

The early phase occurs within the first week of onset of the disease[6] and begins with the initiation of abdominal pain. Treatment during this phase is simply supportive.[6] In contrast, the second or late phase is based on morphologic classification and usually starts 2 to 6 weeks from the onset of pain. Resolution of disease in the late phase implies an edematous pancreas without necrosis. Progression of disease is usually slow and may last weeks to months, leading to necrosis of the pancreas. In the second phase, the types of treatment and the need for treatment will be based on morphologic abnormalities of the pancreas and peripancreatic region (i.e., local complications causing bacteremia and/or sepsis).[6]

There are two broad categories of pancreatitis based on radiologic findings: interstitial edematous pancreatitis (IEP) and necrotizing pancreatitis (NP). IEP is found in the majority of patients; it may be diffuse or localized, and generally shows homogeneous enhancement on CT. Morphological local complications of this type of severe AP include acute peripancreatic fluid collection and pancreatic pseudocyst. Necrotizing pancreatitis occurs in approximately 5-10% patients, in either the parenchyma and/or peripancreatic tissues. The most common presentation of necrotizing pancreatitis involves both the peripancreatic tissue and parenchyma. It is less common to find necrotizing pancreatitis in the peripancreatic tissue and rare to find it only in the pancreatic parenchyma. Importantly, necrotizing pancreatitis may not be visualized early with CT. Necrotizing pancreatitis may persist or disappear over time; it may remain solid or liquefy or remain sterile or become infected. Infected pancreatic necrosis is diagnosed by the presence of extra luminal gas in pancreatic and/or peripancreatic tissues on imaging or biopsy showing bacteria and/or fungi on gram stain and culture. Morphological local complications that further define this type of AP include acute necrotic collection and walled-off necrosis.[6]

Severity is classified as mild, moderately-severe or severe based on the 2013 revised Atlanta Criteria.[6] Mild AP includes the absence of organ failure and of local/systemic complications, is self-limiting, and usually only requires brief hospitalization. Moderately severe AP includes transient organ failure (resolving within 48 hours) and/or local or systemic complications without persistent organ failure (>48 hours). Severe AP is defined as persistent organ failure (>48 hours).[6] Organ failure within week 1 that is persistent and worsening (i.e., >48 hours) is associated with increased risk of mortality, ranging as high as 21 to 55%.[7] Transient organ failure (<48 hours) is associated with little to no mortality, as low as 0 to 1.4%.[8]

RISK STRATIFICATION-SCORING SYSTEMS

Historically, in determining the severity of acute pancreatitis, several criteria have been used in the assessment and stratification process. These included traditional multifactorial scoring systems, such as The Ranson scale, the Acute Physiology and Chronic Health Evaluation (APACHE) II scale, the Bedside Index for Severity in Acute Pancreatitis (BISAP) and the Computed Tomography Severity Index (CTSI). The Ranson scale is one of the oldest and most well-known of the prognostic scales. Calculation of the Ranson score involves evaluating criteria upon admission and then 48 hours later. There are 11 total criteria evaluated. Criteria at admission include age > 55 years, WBC > 16,000, glucose > 200mg/dL, AST>250 IU/L, ALT> 350 IU/L. After 48 hours, criteria include a decrease in hematocrit of greater than 10%, an increase in BUN > 5mg/dL, calcium < 8mg/dL, PaO2<60mmHg, base deficit > 4mmol/L and a fluid sequestration of > 6 liters. Each criterion is worth one point. A patient with a score of 11 has a mortality rate of greater than 50%.[11] A meta-analysis in 1999 that reviewed 110 studies reported that the clinical use of the Ranson scale had poor predictive power.[9]

The Acute Physiology and Chronic Health Evaluation (APACHE) II scale gained popularity for its ability to be used daily, also providing good negative predictive value and modest positive predictive value in the prognosis of severe AP.[1] The APACHE II score was originally an ICU predictive scoring system for critically-ill patients. There are 12 measures involved, including rectal temperature, MAP, HR, RR, A-a gradient or PO2, pH or HCO3, sodium, potassium, creatinine, white blood cell count, Glasgow coma scale, age, and presence of chronic diseases.[10] Severe pancreatitis is indicated by an APACHE II score of greater than 8.[11] The Computed Tomography Severity Index—also known as the Balthazar score—is based on radiological findings.[11] It employs a grading system that combines a point system based on appearance of the pancreas on CT with a point system based on the percentage of pancreatic necrosis noted on the CT. The maximum total is ten points. A score of 7 to 10 predicts a 17% mortality rate and a 92% complication rate.[11] The new Bedside Index for Severity in AP (BISAP) has been newly-adopted as an accurate means of risk stratification.[12] The BISAP score includes blood urea nitrogen >25 mg / dl, impaired mental status, SIRS, age > 60 years, and pleural effusions; each criteria counts as a point and a score ≥3 is associated with an increased risk of complications. A BISAP score of 3, 4, or 5 corresponded to a mortality of 5.3%, 12.7 and 22.5%, respectively, in the validation cohort of 18,256 patients.[12] A comparison of the BISAP, Ranson's, APACHE II, and CTSI scores were applied to a prospective cohort of patients in predicting organ failure, complications and mortality, and showed that BISAP's prognostic accuracy is similar to those of other scoring systems and that its criteria are both clinically relevant and easy to obtain.[13]

None of these scoring systems has proven to be better than the others in determining the severity of acute pancreatitis. There are high false positive rates in all of these scoring systems. They are useful, however, in triaging patients into critical care units. The American Gastroenterological Association (AGA) recommends that those with severe disease (based on APACHE II) and those with severe comorbid conditions be admitted into critical care units. In addition, imaging is recommended within the first 48 to 72 hours for those with initial presentation of severe disease (based on APACHE II) and those with worsening status (i.e., organ failure).[14]

TREATMENT

Nutrition

Feedings help to maintain the integrity of the gastrointestinal mucosal lining thus preventing bacterial translocation and possible infection of the pancreatic tissue.[15, 16] Oral feeds can be started immediately in cases of mild pancreatitis. The diet may consist of a soft or solid low fat and low residue diet.[17] In severe acute pancreatitis, enteral feeds should be initiated within forty eight hours.[18] Parenteral feeds should be avoided due to their risk of infection unless the enteral route is not available or not able to meet caloric needs.[17] Enteral nutrition has been shown to improve outcomes, shorten the length of hospitalization, and decrease the rate of infection and surgical intervention.[19] Enteral nutrition may be delivered via a nasogastric tube or a nasojejunal tube. Previously the nasojejunal tube was thought to be safer than the nasogastric tube due to its decreased ability to stimulate gastric secretions. More recent studies have shown nasogastric tube feeds to be comparable in safety. There is a slightly higher risk of aspiration associated with nasogastric tube feeds. Patients receiving tube feeds should be placed on aspiration precautions and fed in the upright position.[17] Nasogastric tubes are more cost conscious as they may be placed at the bedside where as a nasojejunal tubes may require either endoscopy or interventional radiology to ensure appropriate placement.[16, 17]

Fluids

Fluid resuscitation is an important aspect of the treatment of acute pancreatitis. Acute pancreatitis often presents with symptoms of decreased oral intake and vomiting which may lead to hypovolemia. This hypovolemia coupled with inflammation of the pancreas may lead to third spacing of fluids. These complications may decrease perfusion to the pancreas which could lead to pancreatic necrosis. Thus early aggressive fluid rehydration helps to preserve pancreatic micro and macro circulation to prevent pancreatic necrosis.[17, 20] Current recommendations for fluid resuscitation published in the American College of Gastroenterology guidelines are to start isotonic crystalloid solution at 250 to 500ml per hour for the first 12-24 hours of hospitalization unless underlying co morbid conditions will be complicated by such large volume resuscitation.[17] A recent study compared lactated ringers to normal saline, hypothesizing that lactated ringer's would decrease the systemic inflammatory response encountered in patients with acute pancreatitis. SIRS criteria and C-reactive protein were used as markers of systemic inflammation and were monitored during the first 24 hours of fluid resuscitation. The patient's that were volume resuscitated with lactated ringer's solution showed decreased levels of both SIRS criteria and CRP at 24 hours. Also noted in this study was that patients who received the more pH balanced lactated ringer's had less incidence of metabolic acidosis than those treated with normal saline. Studies in canines have shown that acidosis induced by fluid resuscitation can lead to a model of septic shock. This induced acidosis may exacerbate the inflammatory response in acute pancreatitis (20). The current American College of Gastroenterology Guideline: Management of Acute Pancreatitis recommends that lactated ringer's may be the preferred replacement fluid at this time but further research may change the grade of this recommendation.[17]

Pancreatic necrosis and the role of antibiotics

A patient with acute pancreatitis may present with SIRS criteria, and the clinician must distinguish whether these signs of inflammation are attributable to the underlying pancreatitis or infection. If infection is suspected then antibiotics should be initiated while infectious etiologies are sought. Once all cultures have resulted and infection has been ruled out then antibiotics should be discontinued. If the patient is found to have an extra pancreatic infection (examples including but not limited to cholangitis, urinary tract infection, pneumonia, blood stream infection etc.) then antibiotics should be continued and tailored to treating the extra pancreatic infection.[17]

It has been suggested that the infections that often complicate pancreatic necrosis are due to bacterial translocation. This bacterial translocation occurs secondary to motility changes resulting from the underlying pancreatitis. These motility changes increases gut permeability and allow bacterial translocation.[15, 16] Infected pancreatic necrosis occurs in about 30% of cases of necrotizing pancreatitis and usually occurs within two weeks of the onset of acute pancreatitis. Infected pancreatic necrosis should be suspected in patients who show a worsening clinical picture, continued sepsis or those who do not improve after 7 to 10 days of hospitalization. Empiric antibiotic therapy should be initiated to treat possible infected necrosis.[17, 21] Carbapenems, extended spectrum pencillins, and combinations of quinolones,metronadizole, cephalosporins have been shown to have efficacy for AP.[22] CT-guided fine needle aspiration (FNA) of the necrotic tissue can be considered prior to initiation of antibiotics or if there is no clinical improvement following treatment with antibiotics. Organisms that are commonly isolated from infected pancreatic necrosis include: Enterobacter, Enterococcus, Staphylococcus, anaerobes and Candida species.[22] If the gram stain and culture are negative then the tissue can be considered sterile and antibiotics should be discontinued. Supportive care should be continued in the event of sterile necrosis. If the patient does not show clinical improvement then repeat CT guided FNA should be repeated every 5 to 7 days. Recent trials have not shown that antibiotics prevent infection of pancreatic necrosis.[17] Thus, prophylactic antibiotics are not recommended to prevent infected necrosis.[17,23]

Surgical Intervention

Hospitalized patients with mild acute pancreatitis due to gallstones should undergo laparoscopic cholecystectomy prior to discharge to prevent recurrent gallstone pancreatitis. Those with severe gallstone pancreatitis should undergo laparoscopic cholecystectomy following clinical improvement or following drainage or resolution of any peripancreatic fluid collections that have formed.[24] Recent studies have shown that patients

with infected necrosis benefit more from minimally invasive intervention as well as delayed intervention. Patients who underwent percutaneous drainage and then minimally invasive necrosectomy had fewer complications than those who initially had open necrosectomy.[25]

CONCLUSION

Necrotizing pancreatitis does not occur in a high percentage of patients who present with acute pancreatitis. When present, necrotizing pancreatitis is associated with high rates of morbidity and mortality. There are several scales available to assist the physician in estimating which patients are at greatest risk for poor outcomes. Our case is an example of necrotizing pancreatitis secondary to gallstone pancreatitis. Physicians should vigilantly monitor these patients once the diagnosis of acute pancreatitis is made. Our patient's Ranson score was 4 which gave her 15 to 20% mortality at 48 hours. BISAP score was 1 based on the presence of SIRS criteria. Her APACHE II score was 1 on admission. Her CTSI score was 9 based on CT imaging of her pancreas which showed necrotizing pancreatitis with peri pancreatic fluid collection, portending a mortality rate of 17%. This mortality estimation was consistent with her mortality risk associated Ranson score. The patient did well following CT guided drainage of her peripancreatic fluid collection. She did receive IV antibiotics due to her persistently worsening clinical picture. Antibiotics were discontinued following negative gram stain and culture of the fluid collection. Once the patient stabilized and tolerated PO intake she was evaluated for cholecystectomy. The patient underwent laparoscopic cholecystectomy during her index hospitalization and was able to be discharged several days following surgery.

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