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Acute Pancreatitis with Normal Amylase and Lipase Enzymes

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Abstract
A 44-year-old non- smoker male with no significant past medical history presented to the Emergency Department with complains of nausea, vomiting, diarrhea, upper abdominal pain and fever. On physical examination, abdomen was notable for tenderness to palpation over epigastrium, with mild guarding, but no rebound or Murphy's sign. Patient was admitted and treated for gastroenteritis but on day third he developed worsening abdominal pain. Abdominal CT was performed which showed extensive pancreatic edema with normal Amylase and Lipase enzymes.

Introduction

Pancreatitis is an inflammatory process in which pancreatic enzymes autodigest the gland. The gland sometimes heals without any impairment of function or any morphologic changes; this process is known as Acute Pancreatitis. Pancreatitis can also recur intermittently, contributing to the functional and morphological loss of gland. Recurrent attacks are referred to as Chronic Pancreatitis.

Case Report

The present case report is about a 44 year-old male with no significant past medical history who presented to the Emergency Department complaining of nausea, vomiting, diarrhea, upper abdominal pain and fever. For the past one week prior to presentation, patient developed pressure-like epigartric pain, radiating to back, worsened with lying down, and associated with non-bloody, non-bilious vomiting, followed by anorexia, nausea and fever to 102F. Patient had not eaten several days prior to arrival to the hospital; hence he was brought in by his family for evaluation. Notably, two months prior to the presentation, patient was evaluated in an outside hospital for abdominal pain similar in quality, but not in intensity, and reportedly had normal blood tests and imaging.

On physical examination, vital signs were significant for hypertension of 150/90, tachycardia to 108, and fever of 101.5; abdomen notable for tenderness to palpation over epigastrium, with mild guarding, but no rebound or Murphy's sign; the rest of the exam, including cardiovascular, pulmonary, integumentary and neurologic exam, unremarkable. Initial laboratory findings were WBC of 10.1, with 81% neutrophils, amylase of 47 (N 28-100 U/L), lipase level of 14 (N 11-82 U/L), and unremarkable basic metabolic panel. Liver function tests were notable normal AST and ALT, elevated *GGT* to 227 (N <50 U/L), LDH: 681 (N 90-225), Total bilirubin 0.9 (N 0-1.5 mg/dL). Lipid panel: Total Cholesterol 201 (N

<50 mg/dL), Triglycerides 80 (N <150 mg/dL), LDL 68 (N <100 mg/dL). Chest X-ray showed a small left sided pleural effusion.

Patient was admitted to medicine service for treatment of gastroenteritis, and was started on intravenous fluids and symptomatic management. On day three of hospitalization, patient developed worsening abdominal pain, associated with inability to tolerate per oral intake secondary to vomiting of food contents, and due to worsening abdominal pain, underwent further workup. CBC revealed leukocytosis with left shift, WBC count of 15.3, with 81.5% neutrophils. Basic metabolic panel notable for sodium of 124, potassium of 3.2, calcium of 7.4, magnesium of 1.7, phosphate of 1.9. Repeat lipase was 67 (N 11-82 U/L). An abdominal CT scan (figure 1) with IV and oral contrast was performed, and showed extensive pancreatic edema, especially involving the pancreatic head and uncinate process, and peripancreatic stranding; these changes deemed consistent with acute pancreatitis; no calcification or pseudocyst were observed on CT. Abdominal ultra sound showed multiple gallbladder stones, however, common bile duct were of normal diameter (2.5 mm), and no intrabiliary duct dilation was noted.

Based on clinical presentation and radiological findings, the diagnosis of acute pancreatitis was made. The patient started on aggressive intravenous fluid hydration, pain management and bowl rest, with good improvement in symptoms. On day five, patient was able to tolerate a regular diet, and noted an almost complete resolution of pain, and therefore was discharged home.

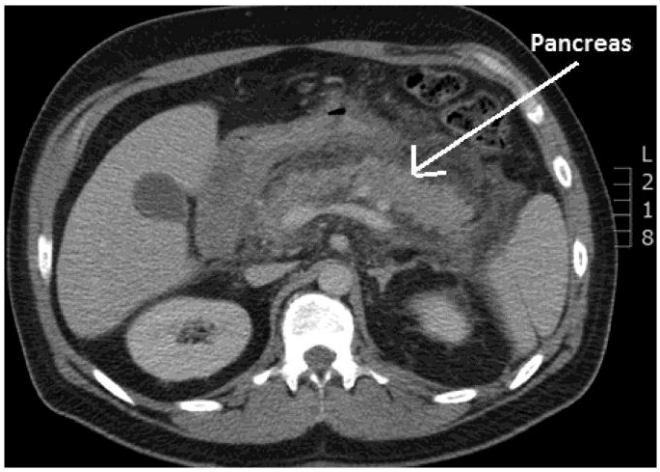

Figure 1: Computerized tomography (CT) of abdomen/Pelvis with IV and oral contrast which demonstrating extensive pancreatic edema, especially involving the pancreatic head and uncinate process, and peripancreatic stranding

Discussion

The cardinal symptom of acute pancreatitis is abdominal pain, which is characteristically dull, boring and steady. Usually, the pain is sudden in onset and gradually intensifies in severity until reachinga constant ache. Most often, it is located in the upper abdomen, usually in the epigastric region, but it may be perceived more on the left or right side, depending on the which portion of pancreas is involved. The pain radiates directly through the abdomen to the back in approximately one half of cases.

Nausea and vomiting often present along with the accompanying anorexia. Diarrhea can also occur. Positioning can be important, because the discomfort frequently improves with the patient in the supine position. The duration of the pain varies, but typically lasts more than a day. It is the intensity and persistence of the pain that usually causes patients to seek medical attention.

Ask the patient about recent operative or other invasive procedures (eg, endoscopic retrograde cholangiopancreatography [ERCP]) or family history of hypertriglyceridemia. Patients frequently have a history of previous biliary colic and binge alcohol consumption, the major causes of acute pancreatitis. On physical examination there may be fever (76%) and tachycardia (65%)

are common abnormal vital signs; hypotension may be noted. Abdominal tenderness, muscular guarding (68%) and distension (65%) are observed in most patients; bowl sounds are often diminished or absent because of gastric and transverse colonic ileus; guarding tends to be more pronounced in the upper abdomen.

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Patient was admitted to medicine service for treatment of gastroenteritis, and was started on intravenous fluids and symptomatic management. On day three of hospitalization, patient developed worsening abdominal pain, associated with inability to tolerate per oral intake secondary to vomiting of food contents, and due to worsening abdominal pain, underwent further workup. Based on clinical presentation and radiological findings, the diagnosis of acute pancreatitis was made. The patient started on aggressive intravenous fluid hydration, pain management and bowl rest, with good improvement in symptoms. On day five, patient was able to tolerate a regular diet, and noted an almost complete resolution of pain, and therefore was discharged home.

Diagnosis

Once a working diagnosis of acute pancreatitis is reached, laboratory tests are obtained to support the clinical impression, eg Serum amylase and lipase, Liver associated enzymes, Blood urea and nitrogen (BUN), creatinine and electrolytes, Blood glucose, Serum cholesterol and triglycerides, Complete blood count (CBC) and hematocrit, C-reactive protein (CRP), Arterial blood gas values, Serum lactic dehydrogenase (LDH), Immunoglobulin G4 (IgG4).

Diagnostic imaging is unnecessary in most cases but may be obtained when the diagnosis is in doubt, when pancreatitis is sever, or when a given study might provide specific information required. Modalities employed include,

1. Abdominal radiography (limited role): Kidney-ureters-bladder (KUB) radio-graphy with the patient upright is primarily performed to detect free air in the abdomen
2. Abdominal ultrasonography (most useful initial test in determining the etiology, and is the technique of choice for detecting gallstones)
3. Endoscopic ultrasonography (EUS) (used mainly for detection of microlithiasis and periampullarylesions not easily revealed by other methods)
4. Abdominal computed tomography (CT) scanning (generally not indicated for patients with mild pancreatitis but always indicated for those with sever acute pancreatitis)
5. Endoscopic retrograde cholangiopancrea-tography (ERCP; to be used with extreme caution in this disease and never as a first-line diagnostic tool)
6. Magnetic resonance cholangiopancreato-graphy (MRCP; not as sensitive as ERCP but safer and noninvasive)

Management

1. Medical management of mild acute pancreatitis is relatively straightforward; however, patients with severe acute pancreatitis require intensive care. Initial supportive care includes; Fluid resuscitation, Nutritional support and Antibiotic therapy. Antibiotics (usually of

imipenem class) should be used in any case of pancreatitis complicated by infected pancreatic necrosis but should not be given routinely for fever, especially early. Antibiotic prophylaxis in severe pancreatitis is controversial; routine use of antibiotic as prophylaxis against infection in severe acute pancreatitis is not recommended.

2. Surgical intervention (open or minimally invasive) is indicated when an anatomic complication amenable to a mechanical solution is present. 1- Gallstone pancreatitis needs cholecystectomy. 2- Pancreatic duct disruption: Image guided percutaneous placement of a drainage tube into the fluid collection. 3-Pseudocysts: None necessary in most cases; for large or symptomatic pseudocysts, percutaneous aspiration. 4-Infected pancreatic necrosis: Image guided aspiration; necrosectomy. 5-Pancreatic abscess: Percutaneous catheter drainage and antibiotics; if no response, surgical debridement and drainage.

Prognosis

The overall mortality in patients with acute pancreatitis is 10-15%. Patients with biliary pancreatitis tend to have a higher mortality then patients with alcoholic pancreatitis. This rate has been falling over the last 2 decades as improvements in supportive care have been initiated. In patients with severe disease (organ failure), who account for about 20% of presentations, mortality is about 30%. This figure has not decreased in last 10 years. In patients with pancreatic necrosis without organ failure, the mortality approaches zero.

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