Imaging of Acute Pancreatitis and its Complications

Rachel Sanford, HMS III
Gillian Lieberman, MD
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Learning Objectives

• Overview of pathophysiology and etiologies of acute pancreatitis
• Brief review of pancreatic physiology and anatomy
• Classification of severity of acute pancreatitis
  – Ranson’s Criteria
  – APACHE II
  – CT Severity Index
• Complications of acute pancreatitis, with emphasis on the appearance of local complications on contrast-enhanced CT
• Treatment of acute pancreatitis
Our Patient: E.F.

- 56-year-old man with a history of EtOH abuse
- Presents with acute onset “stabbing, 10/10” epigastric pain that radiates to his back, and nausea and vomiting
- Physical exam reveals tachycardia (P 110), abdomen is moderately distended and tender to palpation with voluntary guarding and without rebounding
- Liver is enlarged
- Remainder of exam wnl
Our patient E.F.'s Lab Data

• Labs reveal:
  – AST 45 (0-40)
  – ALT 16 (0-40)
  – Amylase 268 (0-100)
  – Lipase 143 (0-60)
  – LDH 226 (0-250)
  – WBC 5.4
  – Glu 118
E.F. is presenting with the typical features seen in acute pancreatitis.

Let’s review some basics about the pancreas.
Pathophysiology of Acute Pancreatitis

• Acute inflammation of the pancreas caused by early activation of proteolytic enzymes (trypsin, phospholipase, chymotrypsin, and elastase) that are normally released in zymogen form and only activated once they reach the duodenum

• Leads to auto-digestion of pancreatic tissue, leukocyte chemo-attraction, cytokine release and oxidative stress

• Acute pancreatitis is a clinical diagnosis, however, contrast-enhanced CT is the imaging modality of choice if the diagnosis is unclear and/or to assess disease severity and complications
Pancreatic Anatomy

Pancreatic Physiology

Etiologies of Pancreatitis

Common:

- Alcohol (approx 30% of cases)
- Gallstones (approx 35% of cases)
Etiologies of Pancreatitis

Less Common:

• Metabolic
  • Hypertriglyceridemia (TG must be >1000, usually above 4000)
  • Hypercalcemia

• Obstruction
  • Ampullary or pancreatic tumor
  • Pancreas divisum

• Trauma
  • post-ERCP
  • Blunt abdominal trauma

• Drugs
  • Furosemide, thiazides, sulfa, didanosine, protease inhibitors, estrogen, 6-MP, azathioprine, ACE inhibitors

• Ischemia
  • Vasculitis, cholesterol emboli, hypotension, shock

• Infection
  • Echovirus, coxsackie virus, mumps, rubella, EBV, CMV, HIV, HAV, HBV, ascari

• Familial
  • Mutations in CFTR, PRSS 1, SPINK 1 genes

• Scorpion sting

• Idiopathic
  • Up to 20%
Menu of Tests for Evaluating the Pancreas

• Abdominal CT with contrast
  – Rapid, non-invasive
  – The test of choice

• Abdominal MRI
  – May show additional details of ducts and soft tissues but not always readily available

• Abdominal US
  – Rapid, non-invasive
  – Allows for evaluation of adjacent organs in real time

• ERCP
  – Allows for simultaneous intervention
  – Can cause pancreatitis

• MRCP
  – Non-invasive
  – Not associated with an increased incidence in pancreatitis
Let’s now discuss some methods for assessing the severity of acute pancreatitis.
### Ranson’s Criteria

<table>
<thead>
<tr>
<th>At diagnosis</th>
<th>At 48 hours</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age &gt;55</td>
<td>Hct ↓ by &gt;10%</td>
</tr>
<tr>
<td>WBC &gt;16K</td>
<td>BUN ↑ by &gt; 5 mg/dl</td>
</tr>
<tr>
<td>Glucose &gt; 200 mg/dl</td>
<td>Base deficit &gt; 4 mEq/L</td>
</tr>
<tr>
<td>AST &gt;250 U/L</td>
<td>Ca &lt; 8 mEq/L</td>
</tr>
<tr>
<td>LDH &gt;350 U/L</td>
<td>PaO2 &lt; 60 mmHg</td>
</tr>
</tbody>
</table>

| Fluid sequestration > 6 L |

**Prognosis**

<table>
<thead>
<tr>
<th>Number of Criteria</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤2</td>
<td>&lt;5%</td>
</tr>
<tr>
<td>3-4</td>
<td>15-20%</td>
</tr>
<tr>
<td>5-6</td>
<td>40%</td>
</tr>
<tr>
<td>≥7</td>
<td>&gt;99%</td>
</tr>
</tbody>
</table>

Ranson JH. *Am J Gastroenterol* 1982;77:633
APACHE II

• Used to monitor disease severity in an ICU setting
• Used for a variety of different illnesses
• Good negative predictive value and modest positive predictive value for predicting severe acute pancreatitis

http://www.e-medtools.com/gadgets.html
CT Severity Index for Acute Pancreatitis

<table>
<thead>
<tr>
<th>CT Grade</th>
<th>Description</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Normal pancreas</td>
<td>0</td>
</tr>
<tr>
<td>B</td>
<td>Enlarged pancreas without inflammation</td>
<td>1</td>
</tr>
<tr>
<td>C</td>
<td>Pancreatic or peripancreatic inflammation</td>
<td>2</td>
</tr>
<tr>
<td>D</td>
<td>Single peripancreatic fluid collection</td>
<td>3</td>
</tr>
<tr>
<td>E</td>
<td>$\geq 2$ peripancreatic fluid collections OR gas in the pancreas or retroperitoneum</td>
<td>4</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Necrosis</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 33%</td>
<td>2</td>
</tr>
<tr>
<td>33-50%</td>
<td>4</td>
</tr>
<tr>
<td>&gt;50%</td>
<td>6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Total Points</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-3</td>
<td>3%</td>
</tr>
<tr>
<td>4-6</td>
<td>6%</td>
</tr>
<tr>
<td>7-10</td>
<td>17%</td>
</tr>
</tbody>
</table>

Balthazar EJ. *Radiology* 1990;174:331
Let’s look at some examples of the CT Severity Index.
Companion Patient 1: Grade A Pancreatitis on Abdominal CT

Companion Patient 2: Grade B Pancreatitis on Abdominal CT

Body and tail of pancreas are enlarged

Companion Patient 3: Grade C Pancreatitis on Abdominal CT

Pancreatic swelling and peripancreatic fat stranding

C+ Axial Abdominal CT

Companion Patient 4: Grade D Pancreatitis on Abdominal CT

C+ Axial Abdominal CT

Fluid collection next to tail of pancreas and in pararenal space

Companion Patient 5: Grade E Pancreatitis on Abdominal CT

Several large peri-pancreatic fluid collections

Let’s move on to a discussion of the complications of acute pancreatitis and look at some radiographic examples.
Complications of Pancreatitis

• **Systemic**
  - ARDS
  - Renal failure
  - GI hemorrhage
  - DIC

• **Metabolic**
  - Hypocalcemia
  - Hyperglycemia
  - Hypertriglycerideridemia

• **Local**
  - Acute fluid collection
  - Pseudocyst
  - Pneumoperitoneum
  - Sterile pancreatic necrosis
  - Infected pancreatic necrosis
  - Pancreatic abscess
  - Splenic artery aneurym/pseudoaneurysm, splenic vein thrombosis
  - Scarring of pancreatic duct leading to stricture and chronic pancreatitis
Companion Patient 6: Sterile Pancreatic Necrosis on Abdominal CT

Decreased attenuation in pancreatic neck, consistent with sterile necrosis

Companion Patient 7: Fluid Collection in Lesser Sac on Abdominal CT

A partially loculated fluid collection, such as the one that we see have just seen, may progress to form a pseudocyst if it becomes fully encapsulated. Let’s look at an example.
Companion Patient 8: Pancreatic Pseudocyst on Abdominal CT

Companion Patient 9: Hemorrhagic Duodenal Pseudocyst and Hemoperitoneum on Abdominal CT

Hemorrhagic pseudocyst in wall of duodenum

Blood in peritoneal cavity
Companion Patient 9: Perforation of Duodenal Pseudocyst with Pneumoperitoneum on Abdominal CT

Companion Patient 10: Pancreatic abscess on Abdominal CT

Well-defined fluid collection containing air and debris, surrounded by thick enhancing rim, consistent with abscess.
Companion Patient 11: Infected Pancreatic Necrosis on Abdominal CT

Debris and gas in an enlarged pancreas, consistent with infected necrosis

C+ Axial Abdominal CT

Companion Patient 12: Splenic Artery Pseudoaneurysm on Abdominal CT

Circular density in pancreatic bed with same attenuation as aorta, consistent with splenic artery pseudoaneurysm

C+ Axial Abdominal CT

Companion Patient 13: Splenic Vein Thrombosis on Abdominal CT

Absence of enhancement in splenic vein, consistent with thrombosis

Companion Patient 14: Chronic Pancreatitis on Abdominal CT

Beaded dilatation of pancreatic duct and many small calcifications, consistent with chronic pancreatitis

Treatment of Acute Pancreatitis

- Fluid resuscitation, sometimes requiring large volumes
- NGT to ensure gastric decompression
- Patient kept NPO, with parenteral nutrition if it is anticipated that patient will be NPO for >4 days
- Analgesia with IV meperidine or hydromorphone (avoid morphine due to concern for sphincter of Oddi spasm)
- Prophylactic systemic antibiotics are of unclear benefit and continue to be controversial; may be used in severe necrotizing pancreatitis
- ERCP and sphincterotomy may be helpful in gallstone pancreatitis
- Surgical débridement is reserved for severe infected necrosis and carries significant mortality if performed in the first few days
Our patient E.F.’s Hospital Course

- **Ranson criteria**
  - At admission: 1
  - At 48 hours: 0
- **Supportive care included IV fluids, TPN and IV hydromorphone**
- He was transitioned to PO analgesics on hospital day 5
- His pain improved and he was discharged on hospital day 6
Acknowledgements

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• Maria Levantakis
References


