MDCT in Acute Pancreatitis

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Lecture Objectives

- To describe the role of MDCT in the diagnostic work-up of acute pancreatitis
- To discuss the proposed revision of the Atlanta classification of acute pancreatitis and what that means for the radiologist
- To discuss the subtypes of acute necrotizing pancreatitis

Acute Pancreatitis: Etiologies

- Mechanical: choledocho-/microlithiasis, ampullary/peripancreatic obstruction, anatomic variant, trauma, ERCP, surgery
- Toxic-metabolic: alcohol, hyperlipidemia, hypercalcemia
- Infectious: viruses, bacteria, fungi, parasites
- Drug related

Most common: gallstones & alcohol abuse (~70%)

Acute Pancreatitis: Classification

The 1992 Atlanta classification
- Based on clinical criteria: mild and severe AP
- Not universally accepted:
  1. much of controversy over natural course of (peri)pancreatic collections due to lack of prospective data from large patient series
  2. does not provide exact radiological criteria
  3. definitions for AP often used inappropriately

2007 proposed revision of Atlanta classification and definitions of collections associated with AP by Acute Pancreatitis Working Group

Why Revise the Atlanta Classification?

- New insights into pathophysiology
- Improved diagnostic imaging techniques
- New treatment options (ie, minimally invasive surgery, endoscopic necrosectomy, percutaneous drainage) rely on accurate morphological description of complications type of intervention dependent on contents of a collection
- Evolving necrotic collections

Bollen, Pancreas 2007;35:107-113
Bollen, Br J Surg 2008;95:6-21

- Goals: improve patient care and physician communication

Revised Definitions of Acute Pancreatitis

Diagnosis requires two of the three items:
- Abdominal pain suggestive of pancreatic origin
- Serum amylase and/or lipase ≥ 3 times normal
- CT or MRI findings compatible with AP

Defining disease severity:
- First week based on clinical parameters
- Thereafter on morphological parameters
### Revised Definitions of Acute Pancreatitis

<table>
<thead>
<tr>
<th>Old term: mild AP</th>
<th>New term: non-severe AP</th>
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<tbody>
<tr>
<td>80% of all patients with AP</td>
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**Histology**
- Interstitial edema, micronecrosis

**Clinical course**
- No MSOF (multisystem organ failure)
- Improve in 48-72 hrs

**Imaging**
- None
- CECT
- US to evaluate gallstones

### Non-Severe Acute Pancreatitis

Clinical question: ruptured AAA? History: 5 days after laparoscopic cholecystectomy (gallstone causing AP of pancreatic head likely), improve in 48 hrs

### Revised Definitions of Acute Pancreatitis

<table>
<thead>
<tr>
<th>Old term: severe AP</th>
<th>New term: severe AP</th>
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<tbody>
<tr>
<td>20% of all patients with AP</td>
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**Histology**
- Confluent acinar cell & vascular macro-necrosis, ductal disruption, fat necrosis

**Clinical course**
- Predicting severity in AP: various scoring systems (Ranson, APACHE II, CTSI, Marshall) only moderately accurate
- MSOF (renal, pulmonary, cardiovascular) ≥48 hrs = most important determinant of morbidity and mortality

### New Classification: Clinical

- Used IN first week
- Divides severe from non-severe AP based on MSOF ≥48 hrs
- Definition: Onset-beginning of abdominal pain

### New Classification: Morphology on CECT or MRI

- Used AFTER the first week
- Acute interstitial or edematous pancreatitis: normal pancreatic parenchyma enhancement and absence of peripancreatic fluid collections
- Acute necrotizing pancreatitis - 3 subtypes:
  - normal pancreatic parenchyma enhancement with peripancreatic fluid collections (fat necrosis) (1)
  - one or more focal areas of nonenhancing pancreatic parenchyma with peripancreatic fluid collections (2)
  - sterile vs infected necrosis → FNA collection, gas within nonenhancing retroperitoneal tissue

### Acute Necrotizing Pancreatitis, Subtype 1

Etiology: hyperlipidemia

- Slight pancreas enlargement; peripancreatic fluid collections, thickened fascia
Acute Necrotizing Pancreatitis, Subtype 1

ERCP April 20, laparoscopic cholecystectomy April 26 for gallstones and acute cholecystitis

Acute Necrotizing Pancreatitis, Subtype 2

Potential pitfall on CECT: necrosis may not be apparent up to 48 hrs after onset

Acute Necrotizing Pancreatitis, Subtype 2

Miliary tuberculosis, drug (Bactrim® or Isoniazid (INH)?) related pancreatitis

NOMI small bowel & ascending colon

2 weeks later

Acute Necrotizing Pancreatitis, Subtype 3

CT Assessment of Acute Pancreatitis

- CECT = reliable method, widely available
- Balthazair CT Severity Index (CTSI, 10-points severity scale) based on grade of pancreatitis and amount of glandular necrosis
- Accuracy of CTSI higher than for APACHE II (Acute Physiologic and Chronic Health Evaluation)
- CTSI of 7-10 associated with 92% morbidity, 17% mortality
- Pts with CTSI ≤3 can safely be discharged from ICU

Balthazar, Radiology 1994;193:297-306
Balthazar, Radiology 2002;223:603-613
Chen, Eur J Radiol 2006;57:102-107
Leung, World J Gastroenterol 2005;11:4049-4052

CT Severity Index

<table>
<thead>
<tr>
<th>Pancreatitis Grade</th>
<th>0 points</th>
<th>1 point</th>
<th>2 points</th>
<th>3 points</th>
<th>4 points</th>
<th>6 points</th>
</tr>
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<tbody>
<tr>
<td>A (normal)</td>
<td></td>
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<td></td>
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<tr>
<td>B (edema)</td>
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<td></td>
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<tr>
<td>C (peripancreatic inflammation)</td>
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<tr>
<td>D (single fluid collection)</td>
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<td></td>
<td></td>
<td></td>
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<tr>
<td>E (two fluid collections)</td>
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Gland necrosis

<30% 2 points
30-50% 4 points
>50% 6 points
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<thead>
<tr>
<th>MRI Assessment of Acute Pancreatitis</th>
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<tbody>
<tr>
<td>• MRI as efficient as CT in recognizing necrosis and predicting severity (Arvanitakis, Gastroenterology 2004;126:715-723)</td>
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<tr>
<td>• Sensitivity of MRI may surpass CT (minor peri-pancreatic inflammation) on T2w images (Pamuklar, Magn Reson Clin NA 2005;13:313-333)</td>
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<tr>
<td>• Advantages MRI:</td>
</tr>
<tr>
<td>- less nephrotoxicity of contrast medium (potential for extension of necrosis and exacerbation of renal impairment after iv iodine contrast media?) (Carmona-Sánchez, Arch Surg 2000;135:1280-1284)</td>
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<td>- allows evaluation of pancreatic duct &amp; biliary system using secretin MRCP</td>
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<th>Fluid Collections</th>
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<tr>
<td>Old term: acute fluid collections</td>
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<tr>
<td>New term: acute peripancreatic fluid collections</td>
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<tr>
<td>• Enzyme rich pancreatic juice</td>
</tr>
<tr>
<td>• Predominantly adjacent to gland</td>
</tr>
<tr>
<td>• Lacks wall, occurs within 48 hrs in 30-50%</td>
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<tr>
<td>• Majority remain sterile</td>
</tr>
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<td>• Resolves spontaneously within 2-4 weeks</td>
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<table>
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<tr>
<th>Postnecrotic Peripancreatic Fluid Collections</th>
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<tr>
<td>Dec 3: endoscopy &amp; biopsy of papilla Vateri (biliary obstruction?), no malignancy</td>
</tr>
<tr>
<td>Dec 4</td>
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<tr>
<td>CT shortcoming: inability to differentiate fluid collection - extrapancreatic fatty tissue necrosis</td>
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<td>Extension of pancreatitis fluid collections: compression but preservation of fat in posterior pararenal space</td>
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<th>Known biliary stones, minor peripancreatic inflammation</th>
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<td>MRI Assessment of Acute Pancreatitis</td>
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<tr>
<td>Old term: acute fluid collections</td>
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<tr>
<td>New term: postnecrotic peripancreatic/pancreatic fluid collections</td>
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<tr>
<td>• Enzyme rich pancreatic juice containing both fluid and necrotic contents</td>
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<tr>
<td>• Initial necrosis → liquefactive necrosis</td>
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<tr>
<td>• Necrosis plus fluid in same area on CT/MRI</td>
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<tr>
<td>• NOT pseudocyst</td>
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<tr>
<td>• Eventually becomes walled off necrosis in late stage</td>
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**Postnecrotic Peripancreatic Fluid Collections**

- Dec 24
- Jan 1

Heterogeneous enhancement & nonenhancing necrotic areas of pancreas

**Postnecrotic Peripancreatic/Pancreatic Collections**

- History of alcohol abuse

**Walled Off Necrosis**

- Sep 1
- Nov 10
- 2 yrs later

- The longer you wait, the more liquefactive necrosis can occur
- Better demarcation between viable and dead tissue

- CT/MRI show replacement of pancreatic tissue
- Interventions surgical, percutaneous, endo

**Pseudocyst**

- Old term: pancreatic pseudocyst
- New term: pancreatic pseudocyst*

- Collection of pancreatic juice contained by granulation tissue
- Devoid of solid necrotic debris
- Develops over a period of 4-6 weeks after onset of acute episode
- Resolves spontaneously within 6 weeks 40%, 80% if <6 cm
- Communication with pancreatic duct seen in 70%
- *noninfected or infected (suppurative)

**Pseudocyst**

- Adjacent to, occasionally within, an otherwise normal appearing pancreas
- CT: homogeneous low attenuation collection, enhancement of thin wall of fibrous capsule

- Nov 16

- Dec 7

5 weeks later
Multiple Pseudocysts

6 weeks from onset of AP

Pseudocysts and Malignancy

Non-Hodgkin-Lymphoma
Amylase and lipase

AP secondary to tumor compression of pancreatic duct or direct tumor infiltration

Pancreatic Duct Injury: Posttraumatic Pseudocyst

14 yo, f. epigastric pain
2 days after fall during trampoline jumping

Infected Pseudocyst

Old term: pancreatic abscess
New term: infected pseudocyst

• Encapsulated collection of pus, usually in proximity to but outside of pancreas
• Requires 4 weeks to form
• Contains little or no necrosis
• Should be differentiated from infected postnecrotic pancreatic fluid collection and infected walled off necrosis

Infected Pancreatic Necrosis

• Most serious local complication of AP, mortality risk double that for infected pseudocyst
• 30-70% of patients with pancreatic necrosis
• 2nd-3rd week following onset of severe AP
• Typically polymicrobial, E. coli in about 35%; translocation of bacteria directly through colon wall
• Detection of gas bubbles in necrotic tissue indicative of infective necrosis (extraluminal retroperitoneal gas caused by intestinal fistula very rare)
• Surgical strategy: necrosectomy
Infected Pancreatic Necrosis

Gas bubbles in necrotic tissue, postnecrotic peripancreatic fluid collection

Oct 24
Nov 12

Infected Necrosis Pancreatic Head

Infected Pancreatic Necrosis

Nov 11: Liver abscesses segments II & IV
Jan 12: 6 weeks after PTCD & transgastric drainage peripancreatic fluid collection

Infected Pancreatic Necrosis: CT-Guided Drainage

Infected Pancreatic Necrosis

Jan 12
Jan 19

Acute Pancreatitis: Extrapancreatic Complications

- Spontaneous fistulation
- Gastric outlet obstruction
- Intestinal necrosis
- Bleeding pseudoaneurysm
- Splenic vein thrombosis

Acute Pancreatitis: Extrapancreatic Complications

Spontaneous fistulization, oral contrast agent
Pseudoaneurysm of gastroduodenal/gastroepiploic artery (after rupture into preexisting pseudocyst or digestion of arterial wall by enzymes)

Acute Pancreatitis: Extrapancreatic Complications

Non-occlusive mesenteric ischemia (NOMI) and associated intestinal gangrene

Prevalence / mortality (N=120):
NOMI 6.7% / 63%
NOMI-associated intestinal gangrene 4.2% / 100%
(Hirota, Pancreatology 2003)

CDCT in Acute Pancreatitis: Summary

- CECT: primary imaging modality for assessing the severity of AP and for follow-up
- MRI is the best alternative in patients with contraindications for CT
- The Atlanta definitions of severity and local complications of AP are being used inconsistently - a revision of the Atlanta classification has been proposed by the Acute Pancreatitis Working Group