Hepatobiliary Disease: Inpatient Management

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Case Presentation

- 56-year-old woman presents with epigastric pain, nausea, vomiting, and low-grade fever.
- PMH: Diabetes, hyperlipidemia and obesity
- SH: Drink 1 glass of Wine/day, smokes ½ ppd
- PE: VS are stable, temp: 99F, obese, epigastric tenderness
- CBC: leukocytosis ~15K
- Comprehensive metabolic panel: normal
- Amylase: 2000 IU
- Lipase 2500 IU
- US abdomen: cholecystectomy, CBD 7.5 mm with no stones
- Triglycerides 200mg/dL, Ca 9.5mg/dL
Burden and Cost of Gastrointestinal, Liver, and Pancreatic Diseases in the United States: Update 2018

Anne F. Peery, Seth D. Crockett, Caitlin C. Murphy, Jennifer L. Lund, Evan S. Dellon, J. Lucas Williams, Elizabeth T. Jensen, Nicholas J. Shaheen, Alfred S. Barritt, Sarah R. Lieber, Bharati Kochar, Edward L. Barnes, Y. Claire Fan, Virginia Pate, Joseph Galanko, Todd H. Baron, and Robert S. Sandler

1University of North Carolina School of Medicine, Chapel Hill, North Carolina; 2University of Texas Southwestern Medical Center, Dallas, Texas; 3Gilings School of Global Public Health, Chapel Hill, North Carolina; 4GI Quality Improvement Consortium, Bethesda, Maryland; and 5Wake Forest School of Medicine, Winston-Salem, North Carolina
# Burden of GI Diseases

<table>
<thead>
<tr>
<th>Rank</th>
<th>Principal diagnoses b</th>
<th>Annual no. of admissions</th>
<th>% Δ from 2005</th>
<th>Median LOS, d</th>
<th>Total hospital days</th>
<th>Median costs, US$</th>
<th>Aggregate charges, US$ (&quot;national bill&quot;)</th>
<th>Aggregate cost, US$</th>
<th>In hospital deaths n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>GI hemorrhage</td>
<td>512,925</td>
<td>-0.1</td>
<td>3.0</td>
<td>2,205,578</td>
<td>6901</td>
<td>19,202,312,421</td>
<td>4,992,811,950</td>
<td>10,930 (2.1)</td>
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<td></td>
<td>Upper d</td>
<td>203,460</td>
<td>-18</td>
<td>3.0</td>
<td>895,224</td>
<td>7413</td>
<td>2,143,858,020</td>
<td>4100 (2.0)</td>
<td>4100 (2.0)</td>
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<tr>
<td></td>
<td>Lower d</td>
<td>161,540</td>
<td>+2</td>
<td>3.0</td>
<td>678,468</td>
<td>6376</td>
<td>1,489,914,180</td>
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<td>2280 (1.4)</td>
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<td>2</td>
<td>Choledolithiasis and cholecystitis</td>
<td>347,985</td>
<td>-13</td>
<td>3.0</td>
<td>1,322,343</td>
<td>9609</td>
<td>16,334,942,913</td>
<td>4,083,951,960</td>
<td>1445 (0.4)</td>
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<td>3</td>
<td>Pancreatitis</td>
<td>291,915</td>
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<td>3.0</td>
<td>1,342,809</td>
<td>6240</td>
<td>10,486,824,627</td>
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<td>Acute</td>
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<td>6240</td>
<td>2,640,588,806</td>
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<td>Chronic</td>
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<td>62,573</td>
<td>6202</td>
<td>133,586,970</td>
<td>NE</td>
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<td>4</td>
<td>Intestinal obstruction</td>
<td>266,465</td>
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<td>3.0</td>
<td>1,455,558</td>
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<td>5</td>
<td>Liver disease/viral hepatitis</td>
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<td>4.0</td>
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<td>13,629,065,070</td>
<td>3,545,958,570</td>
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<td>Alcoholic liver disease</td>
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<td>380,934</td>
<td>8552</td>
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<td>3420 (5.3)</td>
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<td>Hepatic encephalopathy g</td>
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<td>4.0</td>
<td>305,168</td>
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<td>+250</td>
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<td>186,670</td>
<td>7388</td>
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<td>1715 (5.1)</td>
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<td>Ascites or SBP d</td>
<td>16,155</td>
<td>+134</td>
<td>3.0</td>
<td>82,391</td>
<td>6073</td>
<td>177,801,930</td>
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<td>595 (3.7)</td>
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<td>10,245</td>
<td>+57</td>
<td>4.0</td>
<td>69,666</td>
<td>9065</td>
<td>205,811,805</td>
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<td>Hepatitis B</td>
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<td>24,072</td>
<td>5134</td>
<td>53,841,040</td>
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<td>6</td>
<td>Diverticulitis</td>
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<td>4.0</td>
<td>977,671</td>
<td>6406</td>
<td>8,052,870,324</td>
<td>2,132,777,795</td>
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<td>7</td>
<td>Appendicitis</td>
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<td>585,990</td>
<td>7724</td>
<td>7,892,884,821</td>
<td>2,036,901,240</td>
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<td>8</td>
<td>Obesity</td>
<td>151,400</td>
<td>+32</td>
<td>2.0</td>
<td>302,800</td>
<td>11,049</td>
<td>7,404,847,404</td>
<td>1,866,913,400</td>
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<td>Noninfectious gastroenteritis/colitis</td>
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<td>5007</td>
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<td>Abdominal pain</td>
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<td>338,372</td>
<td>4794</td>
<td>2,848,489,883</td>
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<td>C difficile infection</td>
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<td>625,008</td>
<td>6675</td>
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<td>1,003,784,400</td>
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<td>13</td>
<td>Functional/motility disorders g</td>
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<td>GI infection h</td>
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<td>330,119</td>
<td>4305</td>
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<td>3.0</td>
<td>526,661</td>
<td>5782</td>
<td>4,063,858,790</td>
<td>1,082,238,870</td>
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<td>Crohn’s disease</td>
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<td>+13</td>
<td>3.0</td>
<td>302,845</td>
<td>5782</td>
<td>631,647,100</td>
<td>130 (0.2)</td>
<td>130 (0.2)</td>
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<tr>
<td></td>
<td>Ulcerative colitis</td>
<td>37,565</td>
<td>+14</td>
<td>4.0</td>
<td>217,877</td>
<td>7443</td>
<td>450,817,565</td>
<td>145 (0.4)</td>
<td>145 (0.4)</td>
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<td>Total</td>
<td></td>
<td>3,010,030</td>
<td></td>
<td></td>
<td>17,709,474</td>
<td></td>
<td>133,134,979,999</td>
<td>30,594,018,855</td>
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</table>

Peery et al. Gastroenterology 2019, Vol. 156, No. 1
Management of acute pancreatitis

- Diagnosis
- Etiology
- Initial assessment: risk for severe disease
- Initial management: early aggressive intravenous hydration
- Role of early ERCP
- Nutrition
- Management of late complications: Pseudocyst and Necrosis
“God put the pancreas in the back because he did not want surgeons messing with it”
Acute Pancreatitis

- Inflammatory condition characterized by:
  
  A. Local injury
  B. Systemic Inflammatory Response Syndrome (SIRS)
  C. Organ failure (respiratory & renal)
Acute Pancreatitis
Clinical/Lab/Rad Diagnosis
2 out of 3 criteria

- Upper abdominal pain
- ↑↑↑Amylase or Lipase ≥ 3 folds
- Cross-sectional images suggestive of AP
  - Edema, fat stranding, fluid collection, necrosis

Common Causes of AP

- Alcohol 30%
- Biliary 40%
- Idiopathic 15%
- Other 15%

Determine Etiology

Biliary Pancreatitis
- History of abdominal colic
- US: Gallstones or sludge
- US is poor for identifying CBD stones
- ↑AST

Alcohol-induced Pancreatitis
- Requires 4-5 drinks daily over > 5 years
- Binge drinking does not count
- Does not depend on type of alcohol
- Smoking increases risk
Don’t Forget…

- NAPS 2 study
  - Cigarette smoking was an *independent, dose-dependent* risk factor for ARP and chronic pancreatitis

Yadav et al. Arch Intern Med 2009
Others (~15%)

- Hypercalcemia, Hypertriglyceridemia, DKA ~2-6%
- IBD, celiac disease
- Intraductal papillary mucinous neoplasms (IPMN)
- Trauma (blunt, post ERCP)
- Drugs (furosemide, HTCZ, azathioprine, sulfa, mesalamine, NRTI, estrogen <5%
- Choledochal cyst
- Duodenal diverticula
- Ampullary tumors
- Autoimmune <1%
- Genetic (familial, sporadic)
- Pancreas divisum
- Infections, Viral (mumps, Coxsackie A, human, immunodeficiency virus), Parasites (Ascaris) <1%
- Venoms (spider, scorpion, Gila monster)
Common Forms of Pancreatitis

- **Acute Pancreatitis (AP)**
  - Acute interstitial edematous pancreatitis (90-95%)
  - Acute necrotizing pancreatitis (5-10%)\(^1\)

- **Chronic Pancreatitis (CP)**

\(^1\)Koutroumpakis E, et al. Pancreatology 2017;17:32–40
Interstitial Edematous Pancreatitis

(Courtesy of K. Barwick, Jacksonville, FL)
Necrotizing Pancreatitis

(Courtesy of K. Barwick, Jacksonville, FL)
Working Party on AP
Revised Atlanta Classification

Clinical Spectrum of acute pancreatitis

- **Mild ~80%**
  - No local / systemic complications

- **Moderate**
  - Transient local / systemic complications <48 hours

- **Severe**
  - Persistent local / systemic complications > 48 hours

Is it possible to PREDICT severity early in the course of acute pancreatitis?

No!
Risk Factors for Developing Severe Disease

- Age >55
- Comorbid disease (obesity)
- Etiology (cholangitis)
- Elevated BMI
- Initial/ongoing fluid status (BUN, Creatinine, Hematocrit)
- Systemic inflammatory response syndrome (SIRS):
  - 2 of the following: Temperature <36, >38, Heart rate >90 BPM, Respiratory rate >20, WBC <4000, >12,000
- Organ dysfunction on admission
- Findings on admission: CXR: pleural effusions or infiltrates
Mortality and acute pancreatitis

- Overall 2%
- Interstitial pancreatitis 1%
- Necrotizing pancreatitis 12%
- Infected necrosis 25%
  - No organ failure 0%
  - Organ failure and necrosis 35%
  - Organ failure >48 hours 25%
  - Organ failure <48 hours 1%
Treatment Goals

- Supportive Care
- Reduce inflammation
- Assess, prevent, and treat complications
Rising hematocrit and BUN, fluid shifts and pancreatitis

Extravasation of Fluid to Peritoneum
Decreased Intravascular Volume
Rise in Hct and BUN

Increased third space loss
Increased “toxic humerus”
TNF, trypsin, PLA2, elastase, etc.

Decreased pancreatic perfusion

Increased Pancreatic necrosis

Halt the cycle with early aggressive intravenous hydration
Fluid Resuscitation

- AGA guidelines 2018: **Goal-directed therapy (GDT)**
- ACG: 250–500 mL/h of isotonic crystalloid solution in the first 12–24 hours
- Goal of hydration to decrease BUN, Hct
- GDT reduces mortality in sepsis\(^1\)
- Ringer’s lactate preferred (esp. in post-ERCP)
- However, no clear benefit of best IVF to use
- Overly aggressive fluid therapy associated with harm (exacerbation of CHF, PE and abdominal compartment syndrome)

Lactated Ringer’s vs Normal Saline

- Both isotonic and crystalloid
- LR has theoretical benefits
  - Avoids non-anion gap hyperchloremic metabolic acidosis associated with infusion of large volumes of normal saline (acid-base disturbances)
  - May stabilize Acinar cells? Low pH leads to premature activation of trypsinogen
  - Clinical trial evidence that LR is more effective in preventing SIRS
  - Indirect evidence that LR associated with less organ failure
Faster rate of initial fluid resuscitation in severe acute pancreatitis diminishes in-hospital mortality

<table>
<thead>
<tr>
<th>Mortality/Intravenous Fluid Resuscitation</th>
<th>Early</th>
<th>Late</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mortality</td>
<td>0/17</td>
<td>5/28</td>
<td>0.03</td>
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<tr>
<td>0-24 hr</td>
<td>4895</td>
<td>1714</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>24-48 hr</td>
<td>4144</td>
<td>3139</td>
<td>NS</td>
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<tr>
<td>72 hr</td>
<td>3,165</td>
<td>2,908</td>
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<tr>
<td>Total</td>
<td>12,190</td>
<td>7660</td>
<td>NS</td>
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</table>

Early Aggressive Hydration Randomized Trial

- 60 patients with mild AP (no SIRS/OF)
- Randomized ~4 hours of diagnosis
  - 20 mL/kg bolus, then 3 mL/kg/hr
  - 10 mL/kg bolus, then 1.5 mL/kg/hr
- Aggressive hydration resulted in:
  - Less SIRS
  - Improved faster

Reduce Inflammation

- Remove impacted gallstone
- Remove proteases
- Inhibit proteases
- Reduce pancreatic secretion
- Inhibit systemic inflammatory response
## When is early ERCP indicated in AP?

**Is this gallstone pancreatitis?**
- Most common cause
- LFTs normal in most patients
- However, if \((\text{AST/ALT}) > 3\) times normal: 95% PPV

**Is a stone in the CBD?**
- Dilation of CBD and/or
- Persistent with rising bilirubin
ERCP and Biliary Pancreatitis

- **Definite benefit**
  - concomitant cholangitis
  - visualized stone in CBD
- **Probable benefit**
  - high suspicion of stone
- **Unlikely benefit**
  - Severe disease without above
- **No benefit**
  - mild disease
  - routine pre lap chole
  - resolved disease
Enteral Nutrition (EN) vs Parenteral Nutrition (PN)

- More physiologic
- Maintains good integrity
- Decreases intestinal permeability
- Maintain less pathogenic intestinal flora
- Large body with evidence, enteral feeding associated with decrease morbidity and mortality
Nutrition in AP

- Enteric feeding (NG or NJ) is clearly preferred to parenteral nutrition in severe acute pancreatitis.
- Early refeeding is not associated with increased risk of complications regardless if mild acute pancreatitis or severe acute pancreatitis.
- Most patients are discharged early.
- In patients with mild disease, restart feeding as pain subsides.
- 2-fold reduction in total and pancreatic infections.
- 2.5-fold reduction in mortality.

Crockett et al, Gastroenterology 2018. Vol. 154, No. 4
Where to place tube? NG vs. ND vs. NJ

- NJ tube stimulates less pancreatic secretion, but tube location makes no difference in outcomes
- No evidence that elemental formulas superior to polymeric
- When to start feeding?
  - Early start is beneficial
  - AGA recommends early oral feeding (~24 hours) instead of keeping patients NPO

Crockett et al, Gastroenterology 2018. Vol. 154, No. 4
Prophylactic Antibiotics

- Not recommended in any guideline
- Not effective in multiple meta-analyses in high quality studies >2002
- No differences in risks of infected pancreatic and peripancreatic necrosis (OR, 0.81; 95% CI, 0.44-1.49) or mortality (OR, 0.85; 95% CI, 0.52-1.8).
- However still widely used (more than 75% in several studies)

Vege S et al., AGA Institute Technical Review. Gastroenterology 2018
Poor Compliance with ACG Guidelines for Nutrition and Antibiotics in the Management of Acute Pancreatitis: A North American Survey of Gastrointestinal Specialists and Primary Care Physicians

Edward Sun, Mathew Tharakan, Sumit Kapoor, Rajarshi Chakravarty, Aladin Salhab, Jonathan M Buscaglia, Satish Nagula

Table 2. Preferred route of nutrition by type of practice and specialty. Other includes surgical jejunostomy tube and percutaneous endoscopic jejunostomy tube.

<table>
<thead>
<tr>
<th></th>
<th>TPN/PPN</th>
<th>NJ tube</th>
<th>NG tube</th>
<th>Other</th>
<th>P value</th>
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<tbody>
<tr>
<td>Overall responses (n=406)</td>
<td>175 (43.1%)</td>
<td>148 (36.5%)</td>
<td>67 (16.5%)</td>
<td>16 (3.9%)</td>
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<td>Practice type ↴</td>
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<td>&lt;0.001</td>
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<tr>
<td>Academic (n=117)</td>
<td>24 (20.5%)</td>
<td>61 (52.1%)</td>
<td>20 (17.1%)</td>
<td>12 (10.3%)</td>
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<td>Private practice (n=161)</td>
<td>113 (70.2%)</td>
<td>32 (19.9%)</td>
<td>15 (9.3%)</td>
<td>1 (0.6%)</td>
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<td>Specialty:</td>
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<td>Internal medicine (n=242)</td>
<td>102 (42.1%)</td>
<td>96 (39.7%)</td>
<td>38 (15.7%)</td>
<td>6 (2.5%)</td>
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<td>Gastroenterology (n=164)</td>
<td>73 (44.5%)</td>
<td>52 (31.7%)</td>
<td>29 (17.7%)</td>
<td>10 (6.1%)</td>
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n-=406 respondents
Why is TPN/PPN initiated in the management of acute pancreatitis over enteral nutrition? why are antibiotics initiated in acute pancreatitis?
**Peripancreatic fluid collection**

<table>
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<tr>
<th>Interstitial Edematous Pancreatitis</th>
<th>Necrotizing Pancreatitis</th>
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<tbody>
<tr>
<td>• &lt; 4 Weeks</td>
<td>• &lt; 4 Weeks</td>
</tr>
<tr>
<td>– APFC</td>
<td>– ANC</td>
</tr>
<tr>
<td>• &gt; 4 Weeks</td>
<td>• &gt; 4 Weeks</td>
</tr>
<tr>
<td>– Pseudocyst</td>
<td>– WON</td>
</tr>
</tbody>
</table>

**Abandoned terms**
- Pancreatic phlegmon
- Pancreatic abscess
- Hemorrhagic pancreatitis

APFC: acute peripancreatic fluid collection  
ANC: acute necrotic collection  
WON: walled-off necrosis
Peripancreatic fluid collection
Interstitial edematous pancreatitis

APFC

Pseudocyst

<4 weeks

>4 weeks
Peripancreatic fluid collection
Acute Necrotizing Pancreatitis

ANC

WON

<4 weeks

>4 weeks
Indicators of infections

- Worsening clinical condition
- Persistent fever and ↑WBC
- Persistent organ failure
Acute Pancreatitis > 4 weeks

**Pseudocyst**
- **Conservative**
- Endoscopic transpapillary drainage if evidence of disconnected pancreatic duct syndrome (DPDS)
- Endoscopic transmural drainage (Cystgastrostomy)

**WOPN**
- **Conservative**
- Endoscopic Necrosectomy
- Surgical necrosectomy
- However, assess for DPDS prior to drainage

**DPDS:** Disconnected pancreatic duct syndrome
Management of pancreatic necrosis

- Develops within first 24-72 hours
- Patients can appear ill, as if they have an infection
- 50% of patients have organ failure
- Prevent infection by limiting IV lines
- Prevent infection by using enteral route of feeding (3-5 weeks) compared to TPN
- Do not use broad-spectrum antibiotics for purpose of preventing infection of necrosis
- No treatment of sterile necrosis unless symptomatic
- Antibiotics only for infected necrosis
- Delay in endoscopic therapy for > 4 weeks, to develop walled-off necrosis. Percutaneous tube drainage to temporize if septic followed by endoscopic therapy

Y. Nemoto et al. / Pancreatology 17 (2017) 663-668
Infected Pancreas Necrosis: >Day 7-14
Change in Symptoms: Suspect Infection

Skip CT aspiration?

Infected Necrosis
Targeted antimicrobial therapy
Pancreatic necrosis penetrating antibiotic

Clinically Stable
Continue antibiotics and observe
Delayed surgical, radiologic or endoscopic debridement
In selected patients, no debridement

Clinically Unstable
Prompt surgical debridement
EUS-guided cystgastrostomy
Peripancreatic necrosectomy
Video-assisted endoscopic retroperitoneal necrosectomy
Summary Management of Acute Pancreatitis

- Diagnosis: Appropriate use of CT
- Etiology: Transabdominal ultrasound to rule out gallstones
- Condition: “Guarded” first 48 hours, evaluate for risk factors for severe disease, consider more monitored bed for patients with multiple risk factors
- Initiate early aggressive intravenous hydration
- Monitor BUN & Hct
- If patient improves (36-72 hours)
  - Early refeeding
  - Prevent recurrence (e.g: cholecystectomy prior to discharge)
Summary Management of acute pancreatitis

- If patient develops organ failure-ICU
- After 48-72 hours, if patient not improving, obtain CT, if necrosis
  - Prevent infection of necrosis
    - Decrease intravenous lines
    - Avoid TPN, consider enteral feeding
    - Do not use prophylactic antibiotics
  - If infected necrosis (Day >7-14):
    - Treat infected necrosis with pancreatic necrosis penetrating antibiotics
  - Drain infectious collection in unstable patients
  - Early 10-30 days—surgically if pancreas involved
  - Later > 30 days: In symptomatic patients: Walled off pancreatic necrosis—pseudocyst (>30 days) (Endoscopic, radiologic, surgical techniques)
- Asymptomatic necrosis (WOPN) and pseudocysts—Conservative
Thank you