Complications of Acute Pancreatitis

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Acute Pancreatitis

• “Is a protean disease capable of wide clinical variation, ranging from mild discomfort to ‘apocalyptic prostration’”¹
• While most experience minor episodes with recovery…
  – 1/3 will develop complications
  – Of those who do, 1/4 will die
Complications of Pancreatitis

Systemic
- SIRS / Sepsis
- MODS

Local
- Necrosis
- Fluid Collections
  - Pseudocyst
  - Abscess
- Venous Thrombosis
- Pseudoaneurysms
Case 1

- 58 yo (Bud) presents to ER after 3 d alcohol binge with girlfriend, c/o epigastric pain
- PMH - Several prior episodes of same, never as severe. Otherwise well.
- ER doc ordered labs, Amylase is 1000
- Surgery consulted
Case 1

• O/E:
  – HR 120, Bp 90/60, RR 25, Sats 90%
  – Pale, uncomfortable
  – Abdomen tender, no peritoneal signs

• Bloodwork:
  – WBC 15,000
  – AST 90, ALT 150, LDH 150, ALP 30, Br 15
  – Lytes normal, BUN 11
Case 1

• Management?
  – Admitted, resuscitated with IVF, O$_2$, analgesia
  – US– no gallstones, GB, CBD normal

• Following afternoon BP 80/40; u/o low despite IVF

• On R/A, HR 140, RR 30, Sats 88%

• Diaphoretic, ill-looking

• Abdomen unchanged
Case 1

• What now?
Acute Pancreatitis - Pathogenesis

• Intraacinar activation of enzymes
  – Cathepsin B mediated intra-lysosomal activation of trypsin\(^2,3\)
Acute Pancreatitis - Pathogenesis

Proenzyme

Active Enzyme
Acute Pancreatitis - Pathogenesis

• Intraacinar activation of enzymes
  – Cathepsin B mediated intra-lysosomal activation of trypsin$^{2,3}$
  – Intra-pancreatic release of trypsin
    • → Activation of other enzymes
    • → Activation of complement & coagulation cascades
  – Cycle propagates with autodigestion of tissue
Acute Pancreatitis
Pathogenesis

- Microcirculatory injury⁴
  - Vasoconstriction, capillary stasis, and ischemia initially
  - Subsequent vascular permeability and swelling of gland (edematous or interstitial pancreatitis)
Acute Pancreatitis Pathogenesis

• Leukocyte Chemotaxis
  – Invasion by macrophages and PMNs
  – Proinflammatory cytokines (TNF, ILs, PG, PAF) released
  – Interaction with microcirculation induces thrombosis
    • → further ischemia
    • → pancreatic necrosis
Pathogenesis:

Initiating Events → Common Pathway

OEDEMATOUS PANCREATITIS → NECROTIZING PANCREATITIS

Necrotizing Pancreatitis → Fluid Collection → Abscess → Infected Pseudocyst

Necrotizing Pancreatitis → Sterile Necrosis → Infected Necrosis

SIRS / CARS / MODS

Resolution → Pseudocyst
SIRS / Sepsis

• Etiology:
  – Immunologic dissonance theory
    • Severe extrapancreatic inflammation mediated by cytokines
  – Gut motor theory
    • Damage to mucosal barriers allows translocation of bacteria / their products into systemic circulation
MODS

- Pulmonary insufficiency and renal impairment
- Cardiovascular failure
- Metabolic dysfunction
- Associated with mortality ~50%
Severe Acute Pancreatitis

- Predictors of Severity
  - Ranson’s Criteria
  - Apache II Score
  - CRP
  - Glasgow
  - Banks
  - Leeds
Ranson’s Criteria:

On Admission:
- Age > 55
- WBC > 16,000
- Blood glucose > 11.1 mmol/L
- AST > 250 U/L
- LDH > 350 U/L

48 hours:
- Hct ↑ by 10%
- BUN ↑ by 1.8 mmol/L despite fluids
- Calcium < 2 mmol/L
- pO2 < 60 mmHg
- Base deficit > 4 mEq/L
- Fluid sequestration > 6 L

Scores > 4 associated with significant increases in mortality
<table>
<thead>
<tr>
<th>Physiologic Variable</th>
<th>High Abnormal Range</th>
<th>Low Abnormal Range</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>+4</td>
<td>+3</td>
<td>+2</td>
</tr>
<tr>
<td>Temperature - rectal (°C)</td>
<td>≥41°</td>
<td>39 to 40.9°</td>
<td>38.5 to 38.9°</td>
</tr>
<tr>
<td>Mean Arterial Pressure - mm Hg</td>
<td>≥160</td>
<td>130 to 159</td>
<td>110 to 129</td>
</tr>
<tr>
<td>Heart Rate (ventricular response)</td>
<td>≥180</td>
<td>140 to 179</td>
<td>110 to 139</td>
</tr>
<tr>
<td>Respiratory Rate (non-ventilated or ventilated)</td>
<td>≥50</td>
<td>35 to 49</td>
<td>25 to 34</td>
</tr>
<tr>
<td>Oxygenation: A-aDO2 or PaO2 (mm Hg)</td>
<td>≥500</td>
<td>350 to 499</td>
<td>200 to 349</td>
</tr>
<tr>
<td>a. FIO2 ≥0.5 record A-aDO2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>b. FIO2 &lt;0.5 record PaO2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Arterial pH (preferred)</td>
<td>≥7.7</td>
<td>7.6 to 7.69</td>
<td>7.5 to 7.59</td>
</tr>
<tr>
<td>Serum HCO3 (venous mEq/l) (not preferred, but may use if no ABGs)</td>
<td>≥52</td>
<td>41 to 51.9</td>
<td>32 to 40.9</td>
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<tr>
<td>Serum Sodium (mEq/l)</td>
<td>≥180</td>
<td>160 to 179</td>
<td>155 to 159</td>
</tr>
<tr>
<td>Serum Potassium (mEq/l)</td>
<td>≥7</td>
<td>6 to 6.9</td>
<td>5.5 to 5.9</td>
</tr>
<tr>
<td>Serum Creatinine (mg/dl) Double point score for acute renal failure</td>
<td>≥3.5</td>
<td>2 to 3.4</td>
<td>1.5 to 3.4</td>
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<tr>
<td>Hematocrit (%)</td>
<td>≥60</td>
<td>50 to 59.9</td>
<td>46 to 49.9</td>
</tr>
<tr>
<td>White Blood Count (total/mm3)</td>
<td>≥40</td>
<td>20 to 39.9</td>
<td>15 to 19.9</td>
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<tr>
<td>(in 1000s)</td>
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<tr>
<td>Glasgow Coma Score (GCS) Score = 15 minus actual GCS</td>
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</tbody>
</table>

A. Total Acute Physiology Score (sum of 12 above points)
B. Age points (years) <44=0; 45 to 54=2; 55 to 64=3; 65 to 74=5; >75=6
C. Chronic Health Points (see below)

Total APACHE II Score (add together the points from A+B+C)
CRP

• Acute phase reactants made by liver
• Rises proportionately with severity of pancreatitis
• Level $> 150$ mg/ml at 48 h
## Severe Acute Pancreatitis\(^6,7\)

<table>
<thead>
<tr>
<th></th>
<th>Sn %</th>
<th>Sp %</th>
<th>PPV %</th>
<th>NPV %</th>
<th>Accuracy %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apache II &gt; 8</td>
<td>68–71</td>
<td>48–67</td>
<td>30–40</td>
<td>84–87</td>
<td>53–68</td>
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<tr>
<td>(admission)</td>
<td></td>
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<tr>
<td>Ranson &gt; 3</td>
<td>75–89</td>
<td>54–71</td>
<td>37–49</td>
<td>91–96</td>
<td>62–75</td>
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<tr>
<td>(48 h)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CRP &gt; 150</td>
<td>65</td>
<td>73</td>
<td>37</td>
<td>90</td>
<td>72</td>
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<tr>
<td>(48h)</td>
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\(^{6,7}\)Whitcomb, Larvin
Severe Acute Pancreatitis

• Assessment of Severity
  – (AGA, ACG, BSG Guidelines)\textsuperscript{8-10}

• Predictors:
  – Organ failure
  – APACHE II \geq 8, in first 24 h
  – CRP >150 mg/l after 48 h
Severe Acute Pancreatitis

• Recommended Management Severe / predicted severe disease: (AGA, BSG, ACG)\textsuperscript{8-10}
  – Vigorous fluid resuscitation, O\textsubscript{2}, pain control, correction of metabolic abnormalities
  – Intensive monitoring / triage to ICU
  – CT scan (timing controversial)
Severe Acute Pancreatitis

• Supportive Treatment:
  – Hypoperfusion mechanisms thought to be similar to sepsis
  – Early fluid resuscitation (1st 24h) associated with ↓ rates of organ failure and mortality$^{11,12}$
    • Replacement targets not well studied
      – 250-300 cc/h of IVF for 48h would be required for sequestration of 6L$^{13}$
    • Repeat monitoring of patient response (vitals, U/O, end organ damage) essential
Case 1

• Meanwhile, in ICU…
  – Vasopressors added as pt refractory to fluid resuscitation
  – Intubated for impending respiratory failure
  – CT scan arranged
Pathogenesis:
Necrotizing Pancreatitis

• Occurs in 10-20% of patients
• Develops within 1\textsuperscript{st} 24-48 hours, and in majority, remains stable
• Remains associated with mortality rates of 10-25\%\textsuperscript{14}
  – > 1/3 develop infection
  – With infection, mortality is 40-70\% \textsuperscript{19}
Case 1

- Now has necrotizing pancreatitis, I & V, on vasopressors
- In addition to CV / Respiratory support, other measures / treatment while Bud is in ICU?
Necrotizing Pancreatitis

- Nutritional Support - TPN vs. Enteral feeding?
- Prophylactic systemic antibiotics?
- Gut decontamination with non-absorbable antibiotics?
Necrotizing Pancreatitis

• Nutritional support
  – Numerous RCTs have compared nasojejunal feeds with TPN
    • Most showed enteral nutrition equivalent/ superior re: infection, end-organ failure
    • Recent meta-analyses concluded enteric feeding associated with lower risk of infection and cost\textsuperscript{16,17}
    • Only 1 RCT compared NJ vs NG feeds (similar clinical outcomes)\textsuperscript{18}
Necrotizing Pancreatitis

• ACG, BCG Guidelines:⁸,¹⁰
  – Whenever possible, enteral feeding rather than TPN is suggested for AP patients requiring nutritional support

• AGA Guideline:⁹
  – Nutritional support should be provided to those likely to remain fasting for > 7 days. NJ feeding is preferred to TPN
Necrotizing Pancreatitis

• Prophylactic Systemic Antibiotics
  – Earlier studies did not show benefit
  – More recent RCTs (pre 2004) showed variable improvements in morbidity / mortality with new Abx
  – 2 meta-analyses of these RCTs concluded prophylactic Abx reduce mortality, but advantage limited to patients with SAP\textsuperscript{20,21}
Necrotizing Pancreatitis

• Prophylactic Systemic Antibiotics
  – Since 2004, 2 double-blind multicenter, placebo-controlled trials found no benefit\textsuperscript{22,23}
  – Meta-analysis of 7 trials (including these 2 RCTs) found no reduction in mortality or infection\textsuperscript{24}
Necrotizing Pancreatitis

- Guideline Recommendations
  - ACG: does not recommend prophylactic antibiotics
  - AGA: no recommendation;
    - if antibiotics used, restrict to patients with >30% pancreatic necrosis
  - BCG: no recommendation;
    - if antibiotics used, restrict to 14 days
  - IAP: recommends antibiotics (2002)
Necrotizing Pancreatitis

- Selective Gut Decontamination
  - RCT of 102 SAP patients assigned to standard treatment or gut decontamination (norfloxacin-amphotericin)\textsuperscript{26}
    - Mortality significantly lower in decontamination group
    - Results confounded by use of IV antibiotics in treatment arm
Case 1

- Bud is given NJ feeds
- Over next 2 weeks, continues to deteriorate in ICU
- Intermittently febrile
- Increasing doses of vasopressors
- Develops evidence of ARDS
- Started on HD
- ICU wants to know if he is a candidate for the OR?
Case 1

• Bud undergoes CT guided FNAB
Necrotizing Pancreatitis

- FNAB – best current means of evaluating for infection
  - 90% sensitivity, 90% specificity\textsuperscript{27}
  - Low complication rate\textsuperscript{28}
    - Includes secondary infection
- CT - Rarely, presence of gas in tissues alone confirms infection
Necrotizing Pancreatitis

• Guideline Recommendations:
  – ACG, AGA, IAP:
    • CT-guided FNAB recommended when infected pancreatic necrosis clinically suspected to document infection and help guide therapy
Case 1

• Bud undergoes CT guided FNAB…
  – If FNAB comes back positive?
  – If FNAB comes back negative?
Necrotizing Pancreatitis

• Sterile Necrosis:
  – Majority of patients can be successfully managed non-operatively$^{29-31}$
Necrotizing Pancreatitis

• Sterile Necrosis:
  – Rationale for avoidance of early surgery:
    • Poor demarcation of necrotic vs. viable tissue
    • Potential for extensive bleeding
    • Inflammatory cascade is not easily switched off and is compounded by surgery itself
    • Early surgery may infect sterile necrosis
Necrotizing Pancreatitis

• Sterile Necrosis:
  – When associated with unresolving organ failure, surgery remains controversial
    • In largest series of 172 patients with sterile necrosis, 62% managed surgically\textsuperscript{32}
      – Surgical group had higher Ranson, APACHE II and CRP levels
      – MR not significantly different (13.1% surgical vs 6.2% non-surgical)
    • Smaller series showed similar patients can be managed conservatively\textsuperscript{30}
Necrotizing Pancreatitis

• Guideline Recommendations:
  – ACG:
    • Sterile necrosis is best managed medically during 1st 2-3 weeks. After this, debridement should be considered if abdominal pain persists and prevents oral intake
  – AGA, IAP:
    • Sterile necrosis should be managed conservatively and only undergo intervention in selected cases
Necrotizing Pancreatitis

• Infected Necrosis:
  – Conservative treatment historically associated with mortality rates ~100%
  – Surgical treatment mortality rates 10-30% in select centers²³, ²⁴
  – Timing
    • Only RCT comparing early (<72 h) vs. late (>12 d) debridement in patients with SAP: mortality rates of 73% vs. 29% in infected patients²¹
Necrotizing Pancreatitis

• Guideline Recommendations:
  – BCG/IAP
    • Infected pancreatic necrosis is an indication for intervention
    • *Surgery within 14 days not recommended (IAP)*
  – AGA:
    • Management of infected necrosis depends on how acutely ill the patient is, & consistency of necrotic material. If possible, manage in specialty center
Case 1

• Bud’s FNA comes back positive
• Plans for O.R. made
Necrotizing Pancreatitis

• Necrosectomy:
  – Open necrosectomy with closed continuous lavage of retroperitoneum
  – Staged open necrosectomy with planned re-laparotomies and packing

• Post-op mortality ~15%

• Main complications – bleeding, fistula

• No RCTs to compare techniques
Case 1

- Bud undergoes necrosectomy
- Post-operatively improves
Case 2

• While Bud recovers, you are consulted to see girlfriend, Stella
  – Admitted to internal medicine with milder episode of EtOH pancreatitis
  – Partially recovered after a week of conservative rx, but now has findings on CT
Case 2
Peripancreatic Collections
Pathogenesis:

Initiating Events → Common Pathway →

OEDEMATOUS PANCREATITIS → NECROTIZING PANCREATITIS

Fluid Collection → Abscess → Pseudocyst → Infected Pseudocyst

Resolution → SIRS / CARS / MODS → Sterile Necrosis → Infected Necrosis
Peripancreatic Collections

• Development requires pancreatic duct disruption
• Enzyme leak incites inflammatory reaction of retroperitoneal tissue and serosa of adjacent viscera
• Subsequent events depend on persistence of communication
Atlanta Symposium

- Acute Fluid Collections
- Pancreatic Pseudocyst
- Pancreatic Abscess
Peripancreatic Collections

- Acute Fluid Collections\(^1\)
  - Occur early (48 h) in severe \textit{pancreatitis} in 30-50% of patients, and do not persist > 4 weeks
  - Lack wall of granulation or fibrous tissue
  - Rarely symptomatic
  - Majority regress spontaneously
    - Those that do not regress may evolve into pseudocysts
  - Do not require active treatment
    - Risk of introducing infection with percutaneous drainage
Case 2

• Stella and internal medicine team are reassured
• She is discharged home with outpatient appointment
Case 2

- A month later, Stella appears in clinic
- She is generally well, but notes mild bloating sensation
- Exam non contributory
- CT ordered
Peripancreatic Collections

• Pancreatic Pseudocyst\(^1\)
  – Collections of pancreatic juice that persist > 4-6 weeks\(^1\)
  – Occur in 10% of pancreatitis patients
  – Enclosed by nonepithelialized fibrous wall
  – Usually round/oval in shape
  – *If pus is present, better referred to as a pancreatic abscess*
Pancreatic Pseudocyst

• Management:
  – Historical study showed pseudocysts persisting > 6wk rarely resolve & 50% develop complications if observed\(^{35}\)
  – More recent studies show successful non-operative management in 50-60% of select patients\(^{36,37}\)
    • 10% developed serious complications
    • Smaller cysts in non-op group, however, even 10 cm cysts observed successfully
    • Majority resolved < 6 months
Pancreatic Pseudocyst

• Recommended indications for intervention: \(^{38,39}\)
  – Refractory symptoms
    • Recurrent pain
    • Mass effect causing biliary/bowel obstruction
  – Complications
    • Infection
    • Hemorrhage
  – Diagnosis
    • Exclusion of malignancy
Case 2

• Reassured once again, Stella goes home w plans for f/u visit
• Arrives in ER 3 weeks later with nausea & vomiting for several days, unable to keep any food down
• Repeat CT
Pancreatic Pseudocyst

- Management Options:
  - Open internal surgical drainage
  - Endoscopic transenteric drainage
  - Endoscopic transpapillary drainage
  - Percutaneous drainage
Pancreatic Pseudocyst

• Pre-operative Considerations:
  – Correct diagnosis (pseudoaneurysm, cystic neoplasm)
  – Presence of debris / necrosis
  – Apposition to adjacent viscera
  – Communication with pancreatic duct / duct obstruction
Pancreatic Pseudocyst

• Internal Surgical Drainage:
  – Cystgastrostomy
    • Cysts adherent to posterior stomach
Pancreatic Pseudocyst

• Internal Surgical Drainage:
  – R en Y Cyst jejunostomy
    • Cysts arising from body & tail not adherent to stomach, or under transverse mesocolon
Pancreatic Pseudocyst

• Internal Surgical Drainage:
  – Ren Y Cystjejunostomy
    • Cysts arising from body & tail not adherent to stomach, or under transverse mesocolon
  – Distal pancreatectomy & cystectomy
    • Consider for remote cysts of pancreatic tail
Pancreatic Pseudocyst

• Internal Surgical Drainage
  – Available data based on retrospective series
  – Review of 14 studies (1032 patients): internal drainage associated with mortality rate of 5.8% and complication rate of 24% \(^4^1\)
  – Case series demonstrate feasibility of laparoscopic approach, but no prospective trials \(^4^2,^4^3\)
Pancreatic Pseudocyst

• **Endoscopic Transmural Drainage**
  – Cystgastrostomy or cystduodenostomy
  – Cyst must be close to enteric wall, ideally with bulge
    • EUS may evaluate distance & identify vessels
  – Enterotomy & cystotomy with diathermy needle knife
  – Guidewire passed into cyst; balloon enlarges opening
  – One or two 7-10 F stents inserted, left until resolution

• Disadvantage:
  • Adequate cyst wall biopsy / debridement usually not possible
Pancreatic Pseudocyst

• Endoscopic Transmural Drainage
  – Recent series report long-term success in 88-97%, recurrences in 5-18%\textsuperscript{44,45}
  – Increased failure rates with:
    • Necrosis (can be predicted with EUS)
    • Increased distance from pancreatic head\textsuperscript{46}
  – Complications in 10-34% (hemorrhage, perforation, stent migration, infection) with mortality rate of 1%
Pancreatic Pseudocyst

• **Endoscopic Transpapillary Drainage**
  – Indicated with clearly established communication between duct and pseudocyst as confirmed by ERCP/MRCP
  – Pseudocyst should be minimally loculated, non-viscous
  – Guidewire threaded into PD close to disruption
  – Biliary and pancreatic sphincterotomies performed, and 5-7 F stent passed over wire
  – Stent left in for several months, or until resolution
Pancreatic Pseudocyst

• **Endoscopic Transpapillary Drainage**
  – Studies report successful drainage in 84-93%, with recurrence in 9-20%
    • Improved outcomes with smaller cysts
  – Complications (pancreatitis, infection) in 0-12%\(^{44,47}\)
  – Advantages:
    • Cysts need not be close to organ, enterotomies minimized
  – Disadvantages:
    • Debridement, biopsy not possible
Pancreatic Pseudocyst

- Percutaneous Drainage
  - Reported failures in 25-55% $^{48,49}$
  - Complication rates of 50-75%
    - Specifically, pancreatico–cutaneous fistula $^{48,51}$
  - Indication for percutaneous drainage is often a pancreatic abscess in high-risk surgical candidates
## Pancreatic Pseudocyst

<table>
<thead>
<tr>
<th></th>
<th>Complications</th>
<th>Recurrence</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Open internal drainage</td>
<td>High</td>
<td>Lowest</td>
<td>Most definitive Debridement possible</td>
<td>OR</td>
</tr>
<tr>
<td>Endoscopic Drainage</td>
<td>Low</td>
<td>Low</td>
<td>Minimally invasive Comparable safety to open</td>
<td>Requires expertise Debridement / bx not possible</td>
</tr>
<tr>
<td>Transpapillary Drainage</td>
<td>Low</td>
<td>Low</td>
<td>Least invasive Cyst location / thickness irrelevant</td>
<td>Requires expertise PD communication Debridement / bx not possible</td>
</tr>
<tr>
<td>Percutaneous Drainage</td>
<td>Highest</td>
<td>High</td>
<td>Readily available Avoids OR</td>
<td>Intact PD preferable Risk of fistula Debridement / bx not possible</td>
</tr>
</tbody>
</table>
Case 2

- Stella undergoes endoscopic cyst gastrostomy
- Cyst decreases in size and Stella is able to eat once again
- However, cyst does not resolve completely on CT
- OGD to recheck stents
Case 2
Splenic Vein Thrombosis

• Complication that develops in ~20%, pseudocyst often present
• Most detected incidentally after several weeks\(^{52}\)
Splenic Vein Thrombosis

- Multifactorial Etiology
  - Extrinsic compression of vein
  - Inflammatory reaction
  - Hypercoaguability

- May cause splenomegaly, gastric varices, +/- UGIB
Splenic Vein Thrombosis
Splenic Vein Thrombosis
Splenic Vein Thrombosis

- Multifactorial Etiology
  - Extrinsic compression of vein
  - Inflammatory reaction
  - Hypercoaguability

- May cause splenomegaly, gastric varices, +/- UGIB
  - Risk of variceal bleeding is low\textsuperscript{53}
    - CT identified varices 5%, OGD identified varices 18%
  - Routine splenectomy no longer recommended\textsuperscript{52-54}
Splenic Vein Thrombosis

- **Management:**
  
  - In setting of acute bleeding:
    
    - Control of acute bleeding may be attempted endoscopically
    
    - Recurrence rates > 50% reported\(^{52}\)
    
    - Urgent splenectomy is definitive treatment
Case 2

- Stella is followed as an outpatient
- She begins to c/o increasing fatigue
- Serial Hb is noted to be dropping
- What now?
- Repeat OGD – no sign of bleeding, stent in place
- CT…
Pseudoaneurysm

- Usually in association with a pseudocyst, abscess, or necrosis
- Splenic artery most common (30–50%), followed by GD (15%) and PD (10%)
- Enzyme leak weakens vessel wall, causes aneurysmal dilatation
Pseudoaneurysm

• Presentation & Diagnosis
  – Patients can present in hypovolemic shock or unexplained drop in Hb
  – CT can demonstrate aneurysm on arterial phase
  – Angiography most accurate for diagnosis and localization of pseudoaneurysm\textsuperscript{56}
Pseudoaneurysm

• Management:
  – If patient stable, angiography can enable embolization of pseudoaneurysm
    • Success > 90% in specialized centers\textsuperscript{56,57}
    • Less likely to succeed with bleeding from pancreatic head, and after necrotizing pancreatitis\textsuperscript{56}
    • Complications include splenic abscess, GI tract ischemia and fistulae
Pseudoaneurysm

• Management:
  – If unstable, urgent laparotomy
    • Ligation of bleeding vessel
      – High rebleeding rates
  – Historically, mortality ~20-40%, but recent studies show significant improved survival
Case 2

- Stella undergoes angiographic embolization
Summary:

- Course of Acute Pancreatitis is highly variable
- Complications are associated with significant morbidity and mortality
- Early identification and prevention is key
References

References:


