Management in Acute Pancreatitis

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Acute pancreatitis

• Definition

• Etiology
Acute pancreatitis - definition

- Inflammatory disease caused by activation, interstitial liberation and autodigestion of the pancreas by its own enzymes
- A group of reversible lesions characterised by inflammation of the pancreas

Terminology of Acute Pancreatitis

Acute Pancreatitis

- INTERSTITIAL (edematous) 80%
- NECROTIZING 20%

- INFLAMMATORY MASS
- STERILE NECROZIS 30%
- INFECTED NECROZIS 70%

- HEALING
- CHRONIC PSEUDOCYST
- PANCREATIC ABCCESS
Terminology of Acute Pancreatitis

- Acute interstitial pancreatitis (~80%)
- Necrotizing pancreatitis (~20%)
  - Sterile necrosis
  - Infected necrosis
- Pancreatic fluid collection
  - Sterile
  - Infected
- Pancreatic pseudocyst
  - Sterile
  - Infected
- Pancreatic abscess
  - Collection of pus with little or no pancreatic necrosis
- Terms no longer used
  - Hemorrhagic pancreatitis
  - Phlegmon
Acute Pancreatitis – Epidemiology

FIGURE 1. Increases in total hospitalizations for acute pancreatitis and in the population rate of hospitalizations for acute pancreatitis during the study period (p for trend = 0.001 for both).
Famous people who have had pancreatitis

• Alexander the Great
• Ludwig von Beethoven
• Dizzie Gillespie
• Maximilian Schell
• John Ashcroft
Incidence

Male:female ratio is
- 1:3 in those with gallstone and
- 6:1 in those with alcoholism

Sex and age distribution of AP
Etiology

- Non-traumatic (75%)
  - Biliary tract disease
  - Alcohol
  - Viral infection (EBV, CMV, mumps)
  - Drugs (steroid, thiazide, furosemide)
  - Scorpion bites
  - Hyperlipidemia
  - Hyperparathyroidism

- Traumatic (5%)
  - Operative trauma
  - Blunt/penetrating trauma
  - Lab test ERCP/angiography

- Idiopathic (20%)
Etiology

• Gallstones (35%-60%)
  – Gallstone pancreatitis risk is highest among patients with small GS < 5mm and with microlithiasis
  – GS pancreatitis risk is also increased in women > 60 yrs
Etiology

• Alcohol (30-40%)
  – Mechanism not fully understood
  – Not all alcoholics get pancreatitis (only about 15%)
  – This suggests a subset of the population predisposed to pancreatitis, with alcohol acting more as a co-precipitant
Etiology – Trauma

• Blunt Trauma
  – Automobile
  – Bicycle handlebar injuries
  – Abuse

• Iatrogenic – ERCP (1-7%)
  – Likely secondary to contrast but also very operator dependant
  – Risk is also increased with Sphincter of Oddi manometry
Acute Pancreatitis
Etiology

- EtOH: 35%
- Idiopathic: 10%
- Other: 10%
- Gallstones: 45%

Fagenholz H et al. AEP 2007;17: 491-49

Clinical Emergency Hospital of Bucharest, 2009
Etiology - 1

Transabdominal ultrasound should be performed in all patients with acute pancreatitis (strong recommendation, low quality of evidence)

In the absence of gallstones and/or history of alcohol use serum triglyceride should be obtained and considered the etiology if > 1000 mg/dL (conditional recommendation, moderate quality of evidence)

In a patient older than 40 years, a pancreatic tumor should be considered as a possible cause of AP (conditional recommendation, low quality of evidence)
Etiology

Endoscopic investigation in patients with acute idiopathic pancreatitis should be limited, as the risks and benefits of investigation in these patients are unclear (conditional recommendation, low quality of evidence)

Patients with idiopathic pancreatitis should be referred to centers of expertise (conditional recommendation, low quality of evidence)

Genetic testing may be considered in young patients (< 30 years old) if no cause is evident and a family history of pancreatic disease is present (conditional recommendation, low quality of evidence)
Pathogenesis
Pathogenesis of acute pancreatitis

**DUCT OBSTRUCTION**
- Gallstone
- Chronic alcoholism
- Interstitial oedema
- Impaired blood flow
- Ischaemia

**ACINAR CELL INJURY**
- Alcohol, drugs
- Trauma, ischaemia, viruses
- Release of intracellular proenzymes and lysosomal hydrolases
- Activation of enzymes
- Acinar cell injury

**DEFECTIVE INTRACELLULAR TRANSPORT**
- Metabolic injury (experimental)
- Alcohol, duct obstruction
- Delivery of proenzymes to lysosomal compartment
- Intracellular activation of enzymes

**ACTIVATED ENZYMES**
- Interstitial inflammation oedema
- Proteolysis (proteases)
- Fat necrosis (lipase, phospholipase)
- Haemorrhage (elastase)
Acute Pancreatitis

Pathogenesis

Premature enzyme activation

Autodigestion of pancreatic tissue

Local complications

Local vascular insufficiency

Activation of white blood cells

Release of enzymes into the circulation

Distant organ failure
Pathophysiology

Oxidated Phospholipids

Activated Macrophages

TNF

PAF

IL-8

IL-6

Tissue injury

Link between pankreatic inflammation and systemic tissue damage

(from R.Stocker – “Acute pancreatitis nutritional symposium” 2000)
Relationship Between the Systemic and Cellular Immune Responses

Gut I/R Injury or Systemic invasion of bacteria / toxins

Systemic immune response

Cellular defense function
- Degranulation
- Phagocytosis
- Cytotoxicity
- Lymphopoiesis

Mediators
- Eicosanoids (PGE, LTB)
- Cytokines (IL, TNF)
- Nitric oxide (NO)

Substrates
- Smooth vascular muscle
- Smooth bronchial muscle
- Endothelium
- Platelets

Systemic inflammatory response
- Microcirculation
- Ventilation
- Permeability
- Aggregation

Cellular
- Polymorphonuclear neutrophil leucocytes
- Makrophages
- Lymphocytes
- Cellproliferation

Bi-phasic

MODS

Fig.: 1
OFR
Two mortality peaks of severe acute pancreatitis
Diagnosis
Clinical Presentation

• Pain (95%)
  – Acute onset
    • Mid-abdominal or mid-epigastric
    • Radiates to the back (50%)
  – Peak intensity in 30 minutes
    • Lasts for several hours
• Nausea and vomiting (80%)
• Abdominal distention (75%)
• Abdominal guarding and tenderness (50%)
• Restlessness and agitation
Clinical signs

• More severe cases
  – Jaundice
  – Ascites
  – Pleural effusions – generally left-sided
  – *Cullen’s sign* – bluish peri-umbilical discoloration
  – *Grey Turner’s sign* – bluish discoloration of the flanks
Signs

Cullen sign

Grey–Turner sign– discolouration in the flanks
Labs

Amylase

• Elevates within HOURS and can remain elevated for 4-5 days

• Many false positives
Differential diagnosis – Amylase Elevation

- **Pancreatic Source**
  - Biliary obstruction
  - Bowel obstruction
  - Perforated ulcer
  - Appendicitis
  - Mesenteric ischemia
  - Peritonitis

- **Salivary**
  - Parotitis
  - DKA
  - Anorexia
  - Fallopian tube
  - Malignancies

- **Unknown Source**
  - Renal failure
  - Head trauma
  - Burns
  - Postoperative
Amylase

• Elevates within HOURS and can remain elevated for 4-5 days
• Many false positives
• High specificity when using levels >3x normal
• Most specific = pancreatic isoamylase (fractionated amylase)
Labs

Lipase

• The preferred test for diagnosis
• Begins to increase 4-8H after onset of symptoms and peaks at 24H
• Remains elevated for days
• Sensitivity 86-100% and Specificity 60-99%
• >3X normal S&S ~100%
Labs

• Increased amylase and/or lipase
  – >3 times ULN
    • <3 ULN does not rule out diagnosis in right clinical context

• Amylase levels rise w/in 2 to 12 h of SXS
  – Peak w/in first 48 h
  – Remain elevated 3-5 days before return to baseline
  – ↑TGs interferes with assay (false negative)

• Lipase much more specific
  – Causes for <3x elevation
    • Perforated ulcer, mesenteric ischemia, CRF (CrCl<20 ml/min)

• Height of elevation does not correlate with severity
• No utility in following daily levels after the diagnosis
Lab Investigations

- Full blood count: neutrophil, *leucocytosis*
- Electrolyte abnormalities include *hypokalaemia, hipocalcemia*
- Elevated LDH in biliary disease
- *Glycosuria* (10% of cases)
- Blood sugar: *hyperglycaemia* in severe cases
- Ultrasound look for stones diseases
Diagnosis

The diagnosis of AP is most often established by the presence of two of the three following criteria: (I) abdominal pain; (II) serum amylase and/or lipase greater than three times the upper limit of normal, and/or (III) characteristic findings from abdominal imaging (strong recommendation, moderate quality of evidence).

Contrast-enhanced CT and/or MRI of the pancreas should be reserved for patients in whom the diagnosis is unclear or who fail to improve clinically within the first 48-72 h after hospital admission (strong recommendation, low quality of evidence).
Diagnosis – Imaging

• CT
  – Excellent pancreas imaging
  – Recommended in all patients with persisting organ failure, sepsis or deterioration in clinical status (6-10 days after admission)
  – Search for necrosis – will be present at least 4 days after onset of symptoms; if ordered too early it will underestimate severity
  – Follow-up months after presentation as clinically warranted for CT severity index of >3
CT Findings
Severe Pancreatitis

- Tail Indistinct
- Intraperitoneal fluid
- Unenhancing Necrosis
- Peripancreatic edema and inflammation
Balthazar,s score

• **CT Grade**
  - Normal A 0 points
  - Focal or diffuse enlargement B 1 point
  - Intrinsic change or fat stranding C 2 points
  - Single ill-defined fluid collection D 3 points
  - Multiple collections of fluid or gas E 4 points

• **Necrosis Score**
  - None 0 points
  - 1/3 of pancreas 2 points
  - 1/2 of pancreas 4 points
  - > 1/2 of pancreas 6 points

• **Severe = Score > 6 (CT Grade + Necrosis)**

*Balthazar EJ, Robinson DL, Megibow AJ. Radiology 1990; 174(2): 331- 336*
Diagnosis - Imaging

• ERCP / EUS
  – Diagnostic and Therapeutic
  – Can see and treat:
    • Ductal dilatation
    • Strictures
    • Filling defects / GS
    • Masses / Biopsy
Severity
Determining severity

- Clinical criteria
  - Early development/persistence of organ dysfnx
    - Ranson criteria
    - Atlanta criteria
    - POP score
    - BISAP

- Clinical assessment
  - Frequent VS, fluid status/UOP, pulse oximetry

- Radiographic criteria
  - CT severity index
    - Necrosis may not be evident until 48-72h
Ranson Criteria

• **Admission**
  – Age>55 years
  – White blood cells >16.000/mm3
  – Glucose >200 mg/dl
  – LDH >350 UI/l
  – AST >250 UI/l

• **During Initial 48 Hours**
  – Hct decrease of >10mg/dl
  – BUN increase of >5mg/dl
  – Base deficit >4 mEq/l
  – Fluid sequestration >6L
  – Ca++ < 8mg/dl
  – PaO2< 60 mmHg

  • Directly related to fluid resuscitation
  • Independent predictors of mortality

Valid 48h after onset of symptoms and not any other time during the disease

*Ranson JH et all. Surgery Gynecology and Obstetrics-1974; 139(1):69-84*
American College of Gastroenterology Guideline: Management of Acute Pancreatitis

Definitions of severity in acute pancreatitis: comparison of Atlanta and recent revision

<table>
<thead>
<tr>
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<tbody>
<tr>
<td><strong>Mild acute pancreatitis</strong></td>
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<tr>
<td>Absence of organ failure</td>
<td>Absence of organ failure</td>
</tr>
<tr>
<td>Absence of local complications</td>
<td>Absence of local complications</td>
</tr>
<tr>
<td><strong>Severe acute pancreatitis</strong></td>
<td><strong>Moderately severe acute pancreatitis</strong></td>
</tr>
<tr>
<td>1. Local complications <strong>AND/OR</strong></td>
<td>1. Local complications <strong>AND/OR</strong></td>
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<tr>
<td>2. Organ failure</td>
<td>2. Transient organ failure (&lt;48h)</td>
</tr>
<tr>
<td>GI bleeding (&gt;500cc/24hr)</td>
<td><strong>Severe acute pancreatitis</strong></td>
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</table>
| Shock – SBP ≤90 mmHg | Persistent organ failure >48h
| PaO2 ≤60% | |
| Creatinine ≥2 mg/dl | |

*Am J Gastroenterol* 2013; 108:1400–1415; doi:10.1038/ajg.2013.218; published online 30 July 2013
APACHE II Score (Acute Physiology And Chronic Health Evaluation)

Score 0-2: 2% mortality, Score 3-4: 15% mortality
Score 5-6: 40% mortality, Score 7-8: 100% mortality

- Hemorrhagic peritoneal fluid
- Obesity
- Indicators of organ failure
- Hypotension (SPB<90 mmHg) or tachycardia (>130 bpm)
- PO2<60 mmHg
- Oliguria (<50 ml/h) or increasing BUN or creatinine
- Serum calcium < 1.90 mmol/l, <8 mg/dl
- Serum albumine < 33 g/l (<3.2 g/dl)
American College of Gastroenterology Guideline: Management of Acute Pancreatitis

Scott Tenner, MD, MPH, FACG; John Baillie, MB, ChB, FRCP, FACG; John DeWitt, MD, FACG; and Sanhi Swaroop Vege, MD, FACG

**Clinical findings associated with a severe course for initial risk assessment – Intrinsic patient-related risk factors for developing of severe disease**

**Patient characteristics**
- Age >55 years (53,57)
- Obesity (BMI >30 kg/m²) (68)
- Altered mental status (69)
- Comorbid disease (53)

**The systemic inflammatory response syndrome (SIRS) (6,53,54,70,71)**
Presence of >2 of the following criteria:
- Pulse >90 beats/min
- Respiration >20/min or PaCO₂ >32 mm Hg
- Temperature >38°C or <36°C
- WBC count >12,000 or <4,000 cells/mm³ or >10% immature neutrophils (bands)

**Laboratory findings**
- BUN >20 mg/dl (63)
- Rising BUN (63)
- HCT >44% (62)
- Rising HCT (62)
- Elevated creatinine (72)

**Radiology findings**
- Pleural effusions (73)
- Pulmonary infiltrates (53)
- Multiple or extensive extrapancreatic collections (67)

*Am J Gastroenterol* 2013; 108:1400–1415; doi:10.1038/ajg.2013.218; published online 30 July 2013
Treatment in AP
Initial assessment and risk stratification

Risk assessment should be performed to stratify patients into higher and lower risk categories to assist triage, such as admission to ICU (conditional recommendation, moderate quality of evidence).

Patients with organ failure should be admitted to ICU or intermediary care setting whenever possible (strong recommendation, low quality of evidence).
When Do I Transfer to the Intensive Care?

- Severe pancreatitis

- Multi-organ failure
  - Pulmonary
  - Renal

- Consider it if you are placing the patient on antibiotics and/or ordering a CT to evaluate non-improvement
Organ Failure

• Cardiovascular
  – Hypotension
  – Septic physiology
    • ↑HR, CO and ↓SVR

• Respiratory
  – Hypoxemia
  – Pleural effusion

• Renal
  – ATN
  – Oliguria

• Hematologic
  – DIC
  – Thrombocytosis

• Hepatic
  – Encephalopathy
  – ↑T bili (3mg/dl)
  – ↑ AST/ALT 2x nl

• GI
  – Stress ulcer
  – Acalculous cholecystitis
Therapeutical goals

• Ventilatory support
• **Fluid resuscitation**
• Haemodynamic support (vasopresors, inotropes)
• Antibiotherapy
• Sedation
• **Analgesia**
• Early enteral nutrition
• Glycemic and triglycerides control
• Prokinetics
• Stress ulcer prophylaxis
• Thromboprophylaxis
Fluids initial management

Aggressive hydration, defined as 250-500 ml/h of isotonic crystalloid solution should be provided to all patients, unless cardiovascular and/or renal comorbidites exist. Early aggressive iv hydration is most beneficial the first 12-24 h, and may have little benefit beyond (strong recommendation, moderate quality of evidence)

In patients with severe volume depletion (hypotension and tachycardia), more rapid repletion (bolus) may be needed (conditional recommendation, moderate quality of evidence)

Lactated Ringer’s solution may be preferred (conditional recommendation, moderate quality of evidence)

Fluid requirements should be reassessed every 6h for the next 24-48 h. The goal of aggressive hydration should be to decrease the blood urea nitrogen (strong recommendation, moderate quality of evidence)
Recommendations: Fluid Resuscitation

- Fluid resuscitation volume should be carefully monitored to avoid over-resuscitation in patients at risk for IAH/ACS (Grade 1B)
- Hypertonic crystalloid and colloid-based resuscitation should be considered in patients with IAH to decrease the progression to secondary ACS (Grade 1C)

- Fluid resuscitation and “early goal-directed therapy” are cornerstones of critical care management
- Excessive fluid resuscitation is an independent predictor of IAH/ACS and should be avoided
- The use of goal-directed hemodynamic monitoring should be considered to achieve appropriate fluid resuscitation

Graphic Display Of IAP, APP, UOP
How IAP Should Be Measured?

- If two or more risk factors for IAH / ACS are present, a baseline IAP measurement should be obtained (Grade 1B)
- If IAH is present, serial IAP measurements should be performed throughout the patient’s critical illness (Grade 1C)

- Physical examination is insensitive in detecting IAH
- IAP monitoring is a cost-effective, safe, and accurate tool for identifying the presence of IAH and guiding resuscitative therapy for ACS
- Serial IAP measurements are necessary to guide resuscitation of patients with IAH / ACS

Intra-Abdominal Pressure Monitoring Kit

- Kit contains everything you need
- Standardized measurement
- No reproducibility errors
- Ease & simplicity of use
- Time savings: 30 seconds to get data.
- Closed system
- No needles
- No contamination risks
When Do I Start Antibiotics?

• Acute pancreatitis - infection ~10%
  – 30-40% of those with necrosis get infection

• Prophylactic antibiotics
  – Controversial
    • No benefit in mild EtOH pancreatitis
    • Imipenem or meropenem in necrotizing pancreatitis
    • Selective gut decontamination may be beneficial?
    • Abx do not appear to promote fungal infection

• General recommendation for use:
  – Biliary pancreatitis with signs of cholangitis
  – >30% necrosis on CT scan
Antibiotics

• Fever early in the disease process is almost universally secondary to the inflammatory response and NOT an infectious process
The role of antibiotics in acute pancreatitis - 1

Routine use of prophylactic antibiotics in patients with severe AP is not recommended (strong recommendation, moderate quality of evidence)

The use of antibiotics in patients with sterile necrosis to prevent the development of infected necrosis is not recommended (strong recommendation, moderate quality of evidence)

Infected necrosis should be considered in patients with pancreatic or extrapancreatic necrosis who deteriorate or fail to improve after 7-10 days of hospitalization. In these patients, either (I) initial CT-guided fine needle aspiration (FNA) for Gram stain and culture to guide use of appropriate antibiotics or (II) empiric use of antibiotics without CT FNA should be given (strong recommendation, low quality of evidence)
The role of antibiotics in acute pancreatitis - 2

In patients with infected necrosis, antibiotics known to penetrate pancreatic necrosis, such as carbapenems, quinolones, and metronidazole, may be useful (conditional recommendation, low quality of evidence)

Antibiotics should be given for an extrapancreatic infection, such as cholangitis, catheter-acquired infections, bacteremia, urinary tract infections, pneumonia (strong recommendation, high quality of evidence)

Routine administration of antifungal agents is not recommended (conditional recommendation, low quality of evidence)
Guidelines for managing pain in AP

Follow guidelines for the administration of hourly opiates

On the wards

Mild
Pain score = 0-1
Hourly morphine as per guidelines
Paracetamol / N.S.A.I.D (if renal function satisfactory)
Once drinking – regular oral analgesia

Moderate
Pain score = 2-3
3 – 4 Morphine injections
Contact Pain Services Team/
On call anaesthetist to set up P.C.A

Pain continues to be severe
Pain scores = Greater than 3.
Not controlled with P.C.A.

Condition stable
MEWS score less than 5.
Contact Pain Services Team for assessment re Epidural

Condition deteriorating
MEWS score 5 or more.
Contact Outreach Team for assessment and Pain. Services Team re Epidural
Epidural analgesia

- thoracic trauma
  (Bulger EM et al. *Surgery* 2004; 136:426-430)

- cardiac surgery
  (Liu SS et al. *Anesthesiology* 2004, 101:153-161)

- acute pancreatitis

- the effectiveness and safety of epidural analgesia has also been demonstrated in critically ill patients with severe acute pancreatitis
  (Bernhardt A et al. *Anaesthesiol Reanim* 2002, 27:16-22)
Epidural analgesia

- ↓ time to extubation
- ↓ ICU stay
- ↓ incidence of renal failure
- ↓ morphine consumption during the first 24 hours
- ↓ maximal glucose and cortisol blood concentrations
- improves forced vital capacity
  (Guay J. J Anesth 2006, 20:335-340)

- Gold standard - thoracic epidural analgesia (TEA) with a local anaesthetic/opioid infusion
TEA

- ↓sympathetic activity and the stress response (A segmental temporary sympathetic block)
- improved mucosal capillary perfusion

TEA

- accelerated recovery of intestinal function
- The faster resolution of postoperative ileus after major open surgery has been attributed to superior pain therapy, reduced opioid consumption, and sympathetic block
Epidural analgesia – adverse effects

Hypotension

- 3.0% to 10.2%
- Corelate with hypovolemia


Treatment failure

- 22% premature termination of postoperative epidural infusions
  - dislodgement (10%)
  - inadequate analgesia (3.5%)
  - sensory or motor deficit (2.2%)

(Ballantyne JC, McKenna JM & Ryder E. Acute Pain 2003;4: 89–97)

Neurological injury

Epidural abscess
Nutrition

• Mild - moderate pancreatitis
  – Calories from IVF (D5W) are sufficient
  – No benefit from additional nutritional support
  – Oral intake advancing to low fat diet once pain/anorexia resolve
  – NGT decompression
    • If frequent emesis or evidence of ileus on plain films
    • Tube feed if anticipate NPO > 1 week
  – DO NOT follow amylase and lipase levels
Nutrition

• Severe AP
  – Enteral nutrition is preferred
  – Begin nutritional support as early as possible
    • NJ tube preferred
  – however nasogastric feeds have been shown to be effective in 80% of cases
  – NGTs should be used with caution in patients with ACS

– TPN only if:
  • Can’t maintain adequate jejunal access
  • Unable to meet caloric demands enterally for > 5 days
Nutrition in acute pancreatitis

In mild AP oral feeding can be started immediately if there is no nausea and vomiting and no abdominal pain (conditional recommendation, moderate quality of evidence).

In mild AP initiation of feeding with a low-fat solid diet appears as safe as clear liquid diet (conditional recommendation, moderate quality of evidence).

In severe AP enteral nutrition is recommended to prevent infectious complications. Parenteral nutrition should be avoided unless the enteral route is not available, not tolerated or not meeting caloric requirement (strong recommendation, high quality of evidence).

Nasogastric and nasojejunal delivery of enteral feeding appear comparable (strong recommendation, moderate quality of evidence).
Pharmaconutrition

n-3 PUFAs
Glutamine
Arginine

Is immune nutrition the holy grail?

TPN + Glutamine in severe acute pancreatitis

double-blind study

Gln reduces the severity of acute-phase response

Gln supports lymphocyte proliferation
Glutamine-TPN in acute pancreatitis:

- reduced acute-phase response and better lymphocyte proliferation

  *De Beaux, Nutrition 1998*

- reduced length of TPN (10 vs 16 days, p< 0.05)
- reduced length of hospital stay (21 vs 25 days)

  *Ockenga et al, Clin Nutr 2002*
Glutamine-TPN in acute pancreatitis: other RCTs

- less infections and reinterventions
  
  *Fuentes-Orozco et al, JPEN 2008*

- less patients with complications
  
  *Sahin et al, Eur J Clin Nutr 2007*

- lower incidence of complications, prevention of pancreatic infections
  
  *He et al, Clin Nutr Suppl 2004*
ERCP in acute pancreatitis

Patients with acute pancreatitis and concurrent acute cholangitis should undergo ERCP within 24 h of admission (strong recommendation, moderate quality of evidence).

ERCP is not needed in most patients with gallstone pancreatitis who lack laboratory or clinical evidence of ongoing biliary obstruction (strong recommendation, low quality of evidence).

In the absence of cholangitis and/or jaundice, MRCP or endoscopic ultrasound (EUS) rather than diagnostic ERCP should be used to screen for choledocholithiasis if highly suspected (conditional recommendation, low quality of evidence).

Pancreatic duct stents and/or postprocedure rectal nonsteroidal anti-inflammatory drug (NSAID) suppositories should be utilized to prevent severe post-ERCP pancreatitis in high-risk patients (conditional recommendation, moderate quality of evidence).
In patients with mild AP, found to have gallstones in the gallbladder, a cholecystectomy should be performed before discharge to prevent a recurrence of AP (strong recommendation, moderate quality of evidence).

In a patient with necrotizing biliary AP, in order to prevent infection, cholecystectomy is to be deferred until active inflammation subsides and fluid collections resolve or stabilize (strong recommendation, moderate quality of evidence).

The presence of asymptomatic pseudocysts and pancreatic and/or extrapancreatic necrosis do not warrant intervention, regardless of size, location, and/or extension (strong recommendation, moderate quality of evidence).
The role of surgery in acute pancreatitis - 2

In stable patients with infected necrosis, surgical, radiologic, and/or endoscopic drainage should be delayed preferably for more than 4 weeks to allow liquefication of the contents and the development of a fibrous wall around the necrosis (walled-off necrosis) (strong recommendation, low quality of evidence).

In symptomatic patients with infected necrosis, minimally invasive methods of necrosectomy are preferred to open necrosectomy (strong recommendation, low quality of evidence).
Conclusions

• Severe acute pancreatitis should be managed in ICU by a multidisciplinary team (surgeon, intensive care, gastroenterology, radiologist, nutritionist etc.)

• Infected necrosis carries a high mortality

• Antibiotics for suspected infected necrosis

• Tube feedings preferred, post ligament of Treiz

• Always look for the myriad of complications

• Guidelines are useful but not enough