
Greater Manchester and Cheshire HPB Unit Guidelines for the Assessment & Management of Hepatobiliary and Pancreatic Disease Chapter 10

Contents

10.	<i>Acute Pancreatitis</i>	126
10.1.	Diagnosis	127
10.2.	Initial Management	127
10.3.	Definitions	127
10.4.	Severity Prediction	130
10.5.	Determine and treat the underlying aetiology	130
10.6.	Role of ERCP	130
10.7.	CT Scanning & Ultrasound	130
10.8.	Nutritional Support	131
10.9.	Antibiotics	131
10.10.	Management of gallstones	131
10.11.	Management of alcoholism	132
10.12.	Management of idiopathic acute pancreatitis	132
10.13.	Management of Abdominal Compartment Syndrome	133
10.14.	Management of Pancreatic Necrosis	135
10.15.	Indications for Referral to the specialist centre	136
10.16.	Radiology Guidelines in Acute Pancreatitis	137

10. Acute Pancreatitis

10.1. *Diagnosis*

Typical clinical features of abdominal pain together with a high plasma concentration of amylase in most cases, however:

- Serum amylase analysis concentrations decline quickly over two to three days. Therefore diagnosis should not rely on arbitrary limits of values three times greater than normal, but on values interpreted in light of the onset of abdominal pain
- Amylase levels may be normal in patients with pre-existing chronic pancreatitis
- Hyperamylasaemia is found in several non-pancreatic diseases (e.g. visceral perforation, small bowel obstruction and ischaemia, leaking aortic aneurysm, ectopic pregnancy)
- CT may help make the diagnosis when clinical criteria and biochemical tests are equivocal

10.2. *Initial Management*

The main goal of initial management is adequate fluid resuscitation – Hartmann’s fluid is recommended.

- A urinary catheter ensures that output is accurately measured
- Central venous monitoring may be needed in acute severe pancreatitis
- Supplemental oxygen should be provided to maintain normal arterial oxygen saturation
- All patients with severe acute pancreatitis should be managed in a high dependency unit or intensive therapy unit
- Adequate treatment of pain usually requires opiate analgesia

10.3. *Definitions*

The definitions of the morphological features of acute pancreatitis (Table 10-1) and the severity criteria (Table 10-2) within the 2012 revised Atlanta classification should be used.

Organ failure is defined as a score of 2 or more for one of three organ systems (respiratory, cardiovascular and renal) using the modified Marshall scoring system (Table 10-3).

Table 10-1 Revised Atlanta (2012) definitions of morphological features of acute pancreatitis

1. Interstitial oedematous pancreatitis

Acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognisable tissue necrosis

2. Necrotising pancreatitis

Inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis

3. APFC (acute peripancreatic fluid collection)

Peripancreatic fluid associated with interstitial oedematous pancreatitis with no associated peripancreatic necrosis. This term applies only to areas of peripancreatic fluid seen within the first 4 weeks after onset of interstitial oedematous pancreatitis and without the features of a pseudocyst.

4. Pancreatic pseudocyst

An encapsulated collection of fluid with a well defined inflammatory wall usually outside the pancreas with minimal or no necrosis. This entity usually occurs more than 4 weeks after onset of interstitial oedematous pancreatitis to mature.

5. ANC (acute necrotic collection)

A collection containing variable amounts of both fluid and necrosis associated with necrotising pancreatitis; the necrosis can involve the pancreatic parenchyma and/or the peripancreatic tissues.

6. WON (walled-off necrosis)

A mature, encapsulated collection of pancreatic and/or peripancreatic necrosis that has developed a well defined inflammatory wall. WON usually occurs >4 weeks after onset of necrotising pancreatitis.

Table 10-2 Revised Atlanta (2012) definitions of severity of acute pancreatitis

Mild acute pancreatitis	Moderately severe acute pancreatitis	Severe acute pancreatitis
No organ failure	Organ failure that resolves within 48 hours (transient organ failure) and/or	Persistent organ failure (>48 h) <ul style="list-style-type: none"> - Single organ failure - Multiple organ failure
No local or systemic	Local or systemic complications	

complications	without persistent organ failure	
---------------	----------------------------------	--

Table 10-3 Modified Marshall Scoring System of Organ Dysfunction					
Organ system	Score				
	0	1	2	3	4
Respiratory: (PaO2/FiO2)	>400	301–400	201–300	101–200	≤101
Renal: *					
(serum creatinine, μmol/l)	≤134	134–169	170–310	311–439	>439
(serum creatinine, mg/dl)	<1.4	1.4–1.8	1.9–3.6	3.6–4.9	>4.9
Cardiovascular:	>90	<90, fluid	<90,	<90,	<90,
(systolic blood pressure, mmHg) †		responsive	not fluid	pH<7.3	pH<7.2
			responsive		
For non-ventilated patients, the FiO2 can be estimated from below:					
Supplemental oxygen (l/min)	FiO2 (%)				
Room air	21				
2	25				
4	30				
6 – 8	40				
9 – 10	50				
A score of 2 or more in any system defines the presence of organ failure.					
*A score for patients with pre-existing chronic renal failure depends on the extent of further deterioration of baseline renal function. No formal correction exists for a baseline serum creatinine ≥134 μmol/l or ≥1.4 mg/dl.					
†Off inotropic support.					

10.4. *Severity Prediction*

Scoring systems have limited value in day-to-day patient management due to their lack of sensitivity and specificity.

- The Early Warning Score (EWS) is a good predictor of outcome within 24 hours
- APACHE II >8 on admission predicts severity (although it tends to over-estimate severity in the elderly)
- Other scoring systems, such as the Ranson and Glasgow scores, require 48 hours after the onset of symptoms for accurate prediction
- A cut-off value for C-reactive protein of 150 mg/l at 48 hours predicts a severe attack

10.5. *Determine and treat the underlying aetiology*

Gallstones continue to be the most common cause. Occult gallstones (microlithiasis or biliary sludge) can cause acute pancreatitis too. Other causes include: alcohol, hypertriglyceridaemia, hyperparathyroidism, pancreatic malignancy, ERCP, trauma, infectious agents (increasingly associated with HIV infection), drugs, autoimmunity, and heredity. Suspected but controversial causes include pancreas divisum and sphincter of Oddi dysfunction.

10.6. *Role of ERCP*

Early (within 72 hours) ERCP & ES:

- Acute biliary pancreatitis with cholangitis
- Acute severe biliary pancreatitis with cholestasis (bilirubin >40 µmol/L and/or dilated common bile duct)

Same admission ERCP & ES:

- Acute biliary pancreatitis, unfit for cholecystectomy
- Management of proven bile duct stones (alternatively: laparoscopic bile duct exploration).

10.7. *CT Scanning & Ultrasound*

Perform CT to detect and stage complications of acute severe pancreatitis (especially pancreatic necrosis) in patients with persisting organ failure, signs of sepsis, or clinical deterioration occurring after an initial improvement

- The full extent of pancreatic necrosis cannot be appreciated until at least 4 days after the onset of symptoms
- Should be performed according to the acute pancreatitis protocol

- Ultrasonography shows gallstones and dilated bile ducts and is recommended as an initial investigation in all patients with acute pancreatitis

10.8. *Nutritional Support*

Early enteral nutrition is an important mode of acute treatment and is essential in patients with severe disease

- When the enteral route is not tolerated, additional or total parenteral nutrition may be needed
- Nasogastric feeding may be as effective as nasojejunal feeding and is simpler to use
- No specific type of enteral nutrition or immunonutrition is superior in AP.
- Do not prescribe probiotics

10.9. *Antibiotics*

There is no role for antibiotic prophylaxis in acute pancreatitis; it risks encouraging antibacterial resistance and opportunistic fungal infections leading to even higher mortality rates.

- Antibiotics should be prescribed according to microbiology culture and sensitivity results
- Empirical prescribing or antibiotics “on demand” may be justified if any of the following occur: newly developed sepsis or systemic inflammatory response syndrome, failure of two or more organ systems, or an increase in serum CRP in combination with other evidence supporting the possibility of infection

10.10. *Management of gallstones*

Patients with gallstone pancreatitis should have cholecystectomy with intraoperative cholangiography, ideally during the same admission.

- All patients who have gallstones and acute pancreatitis require imaging of the bile duct (by MRCP, EUS, OTC, or ERCP if therapeutic intervention indicated)
- For patients with severe gallstone pancreatitis, cholecystectomy may be delayed until systemic complications have resolved
- For those unfit to undergo cholecystectomy, ES alone is considered sufficient treatment

10.11. *Management of alcoholism*

Interventions to treat alcoholism reduce the incidence of recurrent acute pancreatitis.

- Contact the Alcohol Liaison Team
- Use chlordiazepoxide 10-40 mg (depending on severity of symptoms) as the first level treatment of alcohol withdrawal syndrome, in a symptom triggered manner.
- Vitamin prophylaxis is needed for all patients with alcohol dependence:
 - o 1-2 pairs of vitamins B+C (Pabrinex) by IV infusion TDS for 3-5 days
 - o Oral vitamins should be started after Pabrinex is complete (Vitamin B Compound Strong 2 tablets TDS and Thiamine (Vitamin B1) 50mg QDS). These should be taken for at least 3 months.

10.12. *Management of idiopathic acute pancreatitis*

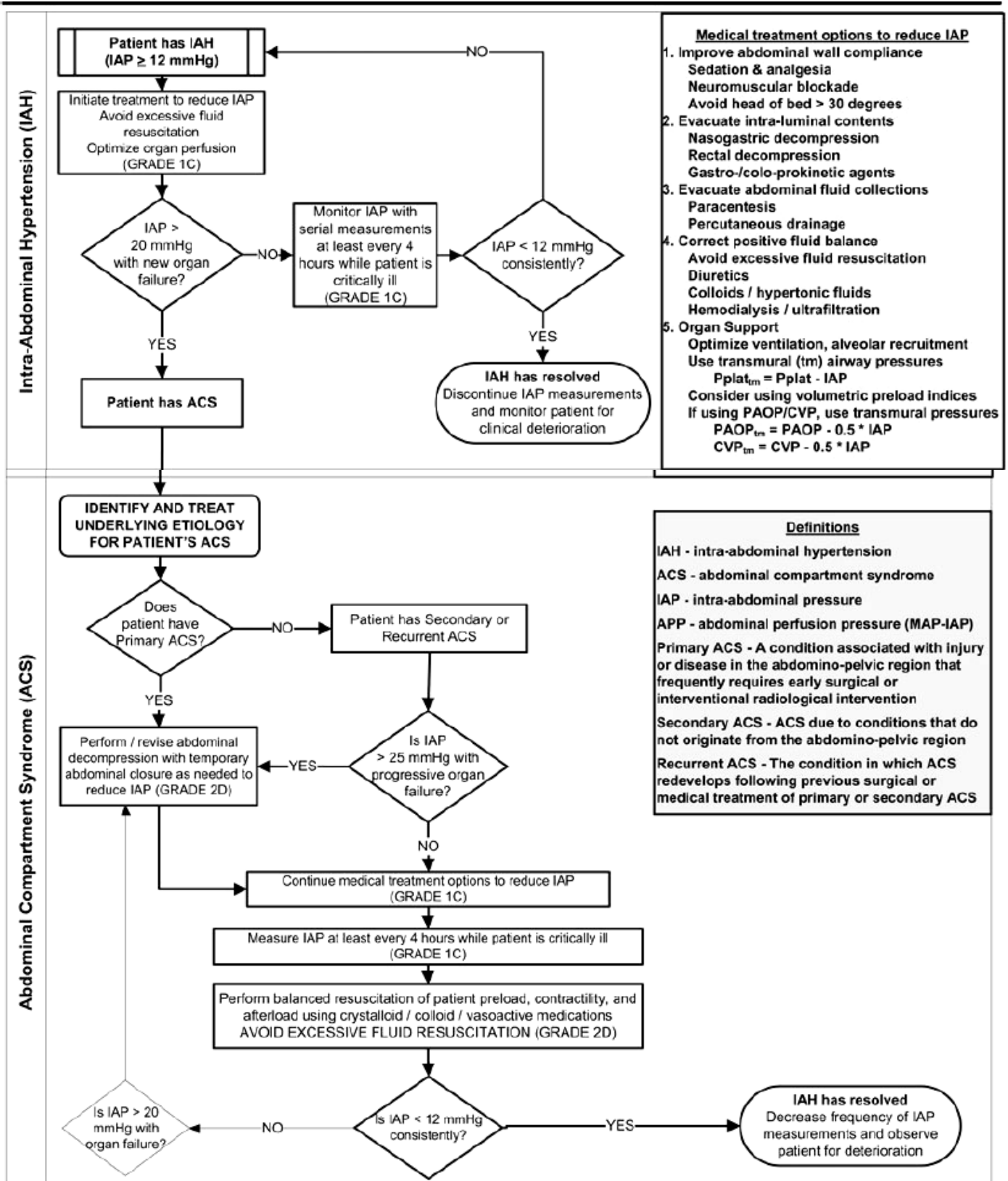
A thorough history and physical examination, liver function tests, and biliary ultrasonography will indicate the correct cause in most cases.

- Retake clinical history with particular emphasis on alcohol, drug and family history (Use EUROPAC referral guidelines)
- Ensure the following blood tests have been performed: LFT's, calcium, fasting triglycerides, IgG4
- EUS to assess for occult microlithiasis
- Consider CT (especially in the elderly) and secretin stimulated MRCP
- Consider empiric cholecystectomy if recurrent attacks after careful patient counselling

10.13. *Management of Abdominal Compartment Syndrome*

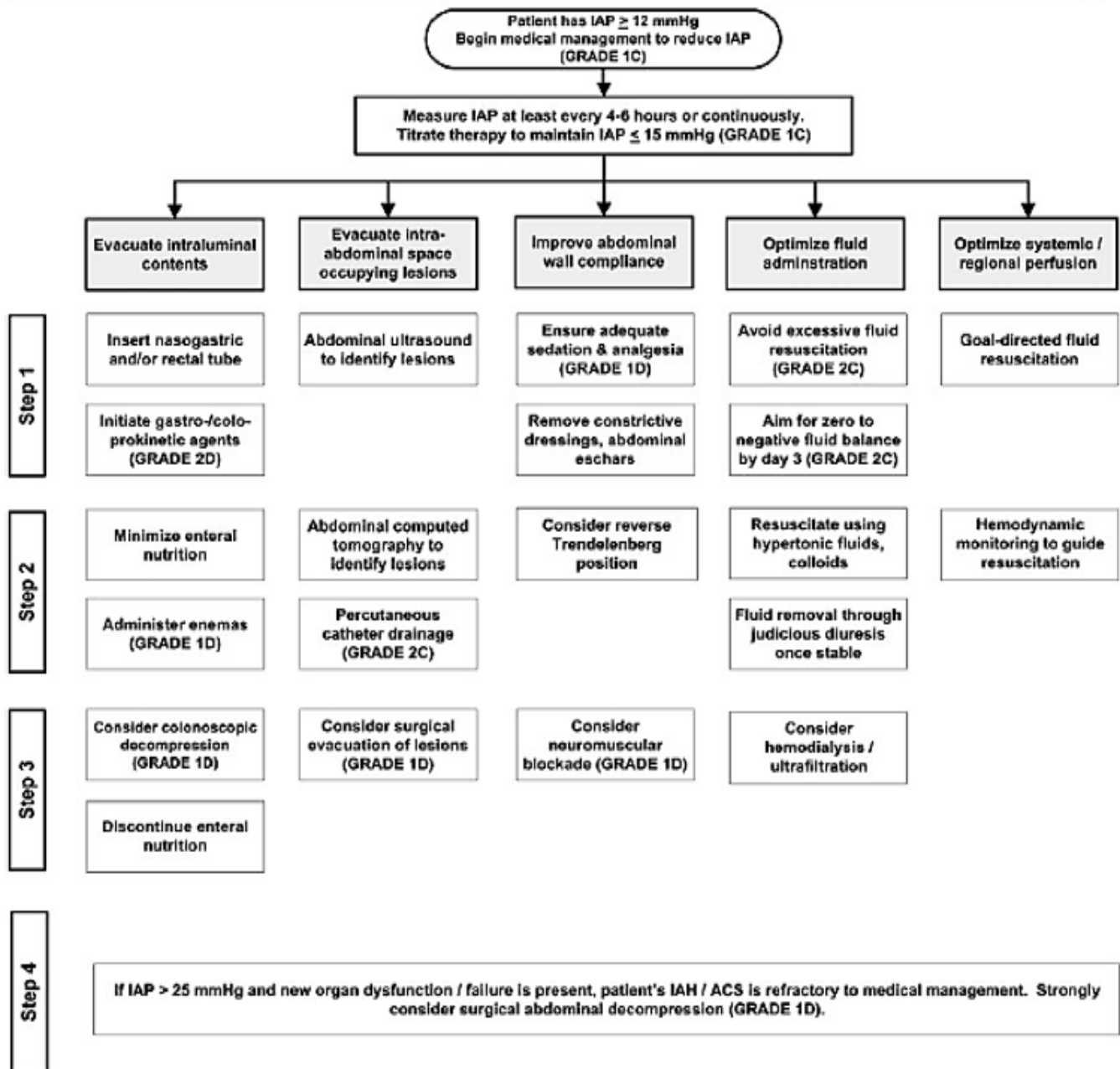
Abdominal compartment syndrome is defined as a sustained intra-abdominal hypertension > 20 mmHg associated with new organ dysfunction/failure.

Follow the World Society of Abdominal Compartment syndrome 2013 international consensus guidelines.



IAH / ACS MEDICAL MANAGEMENT ALGORITHM

- The choice (and success) of the medical management strategies listed below is strongly related to both the etiology of the patient's IAH / ACS and the patient's clinical situation. The appropriateness of each intervention should always be considered prior to implementing these interventions in any individual patient.
- The interventions should be applied in a stepwise fashion until the patient's intra-abdominal pressure (IAP) decreases.
- If there is no response to a particular intervention, therapy should be escalated to the next step in the algorithm.



10.14. *Management of Pancreatic Necrosis*

A conservative and minimally invasive approach to necrotizing pancreatitis improves outcomes.

- Differentiation between sterile and infected necrosis is essential for patients with greater than 30% necrosis on computed tomography and persistent symptoms or those with any degree of necrosis and signs of sepsis.
- This is achieved by fine needle aspiration for bacteriology of pancreatic or peripancreatic necrosis, or the presence of retroperitoneal gas on CT.
- Clinical suspicion of infection combined with clinical deterioration or ongoing organ failure is also an indication for intervention.

Principles of Management:

- Sterile necrosis should largely be managed conservatively.
- Necrosectomy should be delayed for 3-4 weeks after disease onset.
- Use a step-up approach.
- At present there is no evidence showing superiority of one minimally invasive approach over another, and these should be seen as complimentary.
- The aim of intervention is control of sepsis.

Adopt the “Step-Up approach” to infected necrosis until sepsis is controlled.

Figure 10-1 Step-up approach to infected pancreatic necrosis

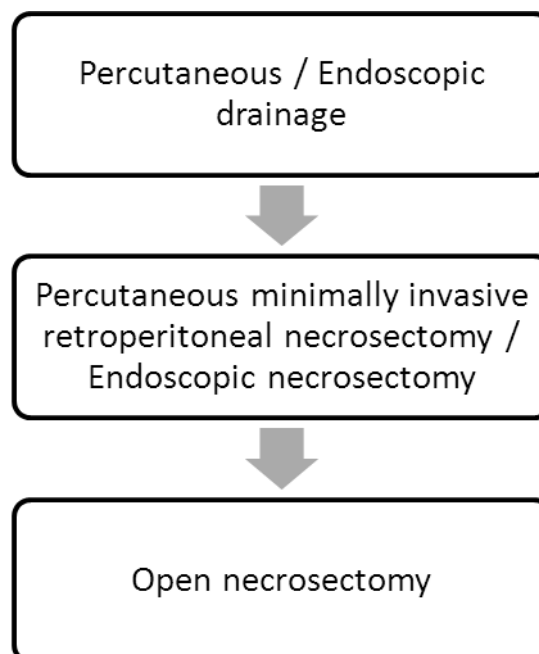


Table 10-4 Management of Percutaneous Necrosectomy

- This is a new technique used in the treatment of infected pancreatic necrosis.
- Patients should have a retroperitoneal percutaneous drain placed under radiological guidance
- A specimen is sent for urgent Gram-stain microscopy, culture & sensitivity
- Patients should be on antibiotics, according to culture & sensitivity results
- Image intensifier needed for first surgical intervention and needs to be booked with X-ray
- After theatre, the patient will have continuous irrigation and drainage of the retroperitoneum. Prescribe 3x1litres of 0.9%NaCl per 24 hours **via the pancreatic drain**.
- Check that the amount of irrigating fluid going in matches that being drained out.
- If bleeding occurs via the pancreatic drain: discontinue irrigation and clamp the drain, resuscitate the patient with colloid and cross-match 6 units of blood. Contact Mr. O'Reilly, via switchboard (or other HPB consultant if he is unavailable). Interventional radiology will usually be required (for angiography and embolisation).
- When the cavity is deemed clear of necrosis, the irrigating fluid is reduced to 50mls of 0.9%NaCl /hour, a tubogram is obtained and if only a small cavity is present, the pancreatic drains are replaced with a 10F NGT. The patient may be discharged and the NGT shortened in the OPD.

10.15. *Indications for Referral to the specialist centre*

Patients with severe acute pancreatitis, potentially in need of interventional radiological, endoscopic or surgical procedures should be referred to a specialist centre.

10.16. *Radiology Guidelines in Acute Pancreatitis*

CT scan protocol acute pancreatitis: multidetector CT with thin collimation and slice thickness (5mm or less), 100–150 cc non-ionic contrast material at a rate of 3mL/s, pancreatic and/or portal venous phase (50-70 seconds delay). During follow up a portal venous phase (monophasic) is generally sufficient.

Table 10-5: Revised Atlanta (2012) definitions of CECT features of acute pancreatitis

1. Interstitial oedematous pancreatitis

- Pancreatic parenchyma enhancement by intravenous contrast agent
- No findings of peripancreatic necrosis

2. Necrotising pancreatitis

- Lack of pancreatic parenchymal enhancement by intravenous contrast agent and/or
- Presence of findings of peripancreatic necrosis (see below—ANC and WON)

3. APFC (acute peripancreatic fluid collection)

- Occurs in the setting of interstitial oedematous pancreatitis
- Homogeneous collection with fluid density
- Confined by normal peripancreatic fascial planes
- No definable wall encapsulating the collection
- Adjacent to pancreas (no intrapancreatic extension)

4. Pancreatic pseudocyst

- Well circumscribed, usually round or oval
- Homogeneous fluid density; no non-liquid component
- Completely encapsulated by a well defined wall
- Maturation usually requires >4 weeks after onset of acute pancreatitis; occurs after interstitial oedematous pancreatitis

5. ANC (acute necrotic collection)

- Occurs only in the setting of acute necrotising pancreatitis
- Heterogeneous and non-liquid density of varying degrees in different locations (some appear homogeneous early in their course)
- No definable wall encapsulating the collection
- Location—intrapancreatic and/or extrapancreatic

6. WON (walled-off necrosis)

- Heterogeneous with liquid and non-liquid density with varying degrees of loculations (some may appear homogeneous)
- Completely encapsulated by a well defined wall
- Location—intrapancreatic and/or extrapancreatic
- Maturation usually requires 4 weeks after onset of acute necrotising pancreatitis