IAEM Clinical Guideline

Acute Pancreatitis

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DISCLAIMER

IAEM recognises that patients, their situations, Emergency Departments and staff all vary. These guidelines cannot cover all clinical scenarios. The ultimate responsibility for the interpretation and application of these guidelines, the use of current information and a patient's overall care and wellbeing resides with the treating clinician.

Revision History

Date	Version	Section	Summary of changes	Author
January 2026	1.0	All	Final version	SK/RS/AH/ FH/TMacM

CONTENTS

GLOSSARY OF TERMS

AKI	Acute kidney injury
AP	Acute pancreatitis
APACHE	Acute Physiology and Chronic Health Evaluation
AST	Aspartate aminotransferase
BISAP	Bedside Index of Severity in Acute Pancreatitis
CRP	C-reactive protein
CT	Computed Tomography
ED	Emergency Department
HDU	High dependency unit
IAH	Intra-abdominal Hypertension
IAP	Intra-abdominal Pressure
ICU	Intensive care unit
LDH	Lactate dehydrogenase
MAP	Mean arterial pressure
NG	Nasogastric
NJ	Nasojejunal
NSAID	Non-steroidal anti-inflammatory drug
pO_2	Partial pressure of oxygen
PCT	Procalcitonin
SIRS	Systemic inflammatory response syndrome
WBC	White blood cells

Acute Pancreatitis

INTRODUCTION

Acute pancreatitis (AP) is a common presentation to Emergency Departments (EDs). In Ireland, available data demonstrates an incidence of 17-24 cases per 100,000 population.[1] The United States shares a similar incidence rate,[2] accounting for approximately 300,000 ED visits per annum.[32] [1] In addition to overall ED workload implications, patients with moderate to severe AP have significant associated morbidity and mortality, requiring resuscitation, critical care resources and longer hospital lengths of stay.

PARAMETERS

Target audience:	Healthcare professionals involved in the initial assessment and management of acute pancreatitis in the ED, including emergency medicine clinicians, surgeons and intensivists
Patient population:	Adult patients with suspected or known acute pancreatitis
Exclusion criteria:	Clinical judgement is required in the assessment of abdominal pain, as the differential diagnoses are extensive. This evidence summary is limited solely to acute pancreatitis.

AIMS

To provide an evidence-based guide to the assessment and management of acute pancreatitis in the ED.

DIAGNOSIS

Acute pancreatitis (AP) is most commonly caused by gallstone disease (approximately 50% of cases), then alcohol consumption (25% of cases).[4–6] Less common aetiologies include hypertriglyceridaemia, medications, and benign or malignant masses which should be considered as potential causes in the absence of demonstrable gallstones or alcohol intake.

The diagnosis of AP is established by the presence of **two out of three of the following criteria**:

1. Abdominal pain consistent with the disease.
2. Serum amylase or lipase greater than three times the upper limit of normal.
3. Characteristic findings on abdominal imaging.

Abdominal Pain

Clinicians must keep an open mind during the initial assessment of any patient with severe acute abdominal pain and consider the full differential list, including aortic aneurysmal rupture or myocardial infarction for instance, while avoiding prematurely anchoring on a single diagnosis.

The abdominal pain of AP is typically described as a severe, constant, epigastric or left upper quadrant pain, which can radiate to the back, chest or flanks. Intensity and exact location of pain do not correlate with disease severity.

Examination findings traditionally associated with AP, such as Cullen's or Grey-Turner's signs (umbilical or flank discolouration respectively) are uncommon and, if present, usually found in the later stages of AP due to intraperitoneal or retroperitoneal haemorrhage.

Serum Amylase & Lipase

Serum amylase and lipase are the two most commonly employed serum biomarkers used in the diagnosis of AP.

At the onset of symptoms, serum amylase levels will rise within a few hours and return to baseline within 3-7 days. However, serum amylase may be elevated in alternative conditions, including renal disease, acute appendicitis, and cholecystitis. This limited specificity means that an isolated elevated serum amylase, in the absence of other symptoms indicative of AP, cannot be used reliably for the diagnosis of AP.[7] Conversely, a normal serum amylase does not exclude AP should the patient present very early in their disease course or delay their presentation until after amylase levels have returned to baseline. In acute-on-chronic pancreatitis, serum amylase levels may also remain near normal due to the lack of functional pancreatic tissue.

Urinary amylase is a potentially useful diagnostic adjunct but has lower sensitivity and specificity for AP than serum amylase.[8,9] There is no diagnostic cut-off value defined in the literature beyond which someone with characteristic abdominal pain is considered to have AP, rendering its role as a reliable biomarker for the diagnosis of AP uncertain.[10]

Serum lipase appears to be more specific than amylase for the diagnosis of AP and remains elevated for a longer period in the disease course (Figure 1).[10] Nevertheless, similar limitations exist regarding its use, including elevation in non-pancreatic disease and higher levels of elevation required for diagnosis in certain patient populations (e.g. patients with diabetes).[11] More fundamentally, serum lipase analysis is unavailable in many Irish hospital laboratories.

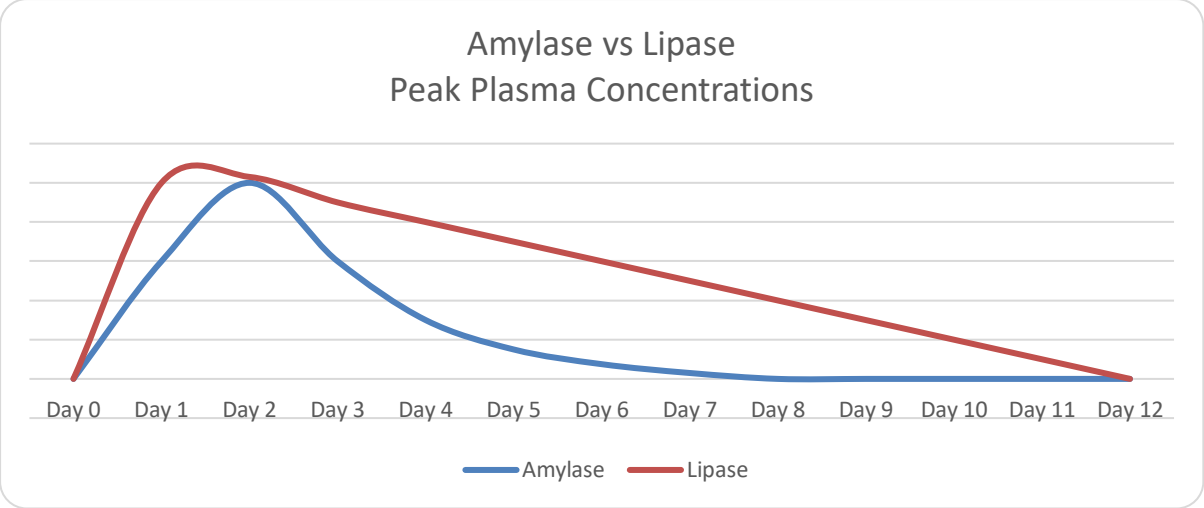

Figure 1: Relative amylase and lipase peak plasma concentrations over time.

Abdominal Imaging

Abdominal imaging is not a requirement for the diagnosis of AP and may be unnecessary in the majority of patients. Although computed tomography (CT) is able to diagnose AP with more than 90% sensitivity and specificity,[12] it does not modify initial clinical management and should be considered only in cases where the diagnosis is uncertain in the ED. Imaging may be required if the patient fails to improve at 48-72 hours, arranged by the relevant inpatient team following admission.[13]

DISEASE SEVERITY

Patients with moderate or severe AP (20% of presentations) develop either transient (<48 hrs) or persistent organ failure and have a mortality rate between 13-35%.[14] It is important to identify these patients early and refer them promptly to high dependency or intensive care units (ICUs).[15]

Predicting Severity

No single laboratory value has been shown to consistently correlate with AP severity or outcome.[16,17] Instead, persistent organ failure 48 hours after admission is the most robust marker of disease severity. This is captured by a variety of scoring systems such as APACHE, Glasgow-Imrie (Table 1) and Ranson (Table 2), each of which has limitations.[18] As a result, AP severity stratification cannot be reliably performed in the ED.[10,19]

Glasgow-Imrie Criteria for Severity of Acute Pancreatitis	**Yes**	**No**
Arterial pO_2 < 7.9 kPa	1	0
Age > 55 years	1	0
WBC > 15 x 10^3/µL	1	0
Calcium < 2mmol/L	1	0
Serum Urea > 16 mmol/L	1	0
LDH > 600 IU/L	1	0
Albumin < 32 g/L	1	0
Glucose > 10 mmol/L	1	0

Table 1: Glasgow-Imrie Criteria for Severity of Acute Pancreatitis.

Higher scores, calculated at 48 hours post admission, correlate with increased severity.[20]

Ranson's Criteria for Pancreatitis Mortality		
On Admission	**Yes**	**No**
WBC > 16 x 10^3/µL	1	0
Age > 55 years	1	0
Glucose > 11.1 mmol/L	1	0
AST > 250 IU/L	1	0
LDH > 250 IU/L	1	0
48 Hours Into Admission		
Haematocrit decrease > 10%	1	0
Urea increase ≥ 1.8 mmol/L	1	0
Calcium < 2 mmol/L	1	0
Arterial pO_2 < 8 kPa	1	0
Base Deficit > 4	1	0
Fluid requirement > 6L	1	0

Table 2: Ranson's Criteria for Pancreatitis Mortality.

A higher score, requiring values at both admission and 48 hours post admission, correlate with increased mortality.[21]

The Bedside Index of Severity in Acute Pancreatitis (BISAP) score (Table 3) has been proposed as a tool that may be helpful in identifying low risk AP in the ED and can be performed in the early stages of disease; however it has not been widely validated and may have poor sensitivity (reported as 64.8%).[22]

Bedside Index of Severity in Acute Pancreatitis (BISAP)	**Yes**	**No**
B: Blood urea nitrogen > 8.9mmol/L	1	0
I: Impaired mental status	1	0
S: Systemic inflammatory response syndrome present	1	0
A: Age > 60 years	1	0
P: Pleural effusion on radiography	1	0

Table 3: BISAP

A score of ≥2 is indicative of severe AP, organ failure and mortality.[23]

Alternative markers for disease severity have been suggested. These include elevated or rising urea or haematocrit (as markers of hypovolaemia and pancreatic hypoperfusion), the presence of systemic inflammatory response syndrome (SIRS), or patient co-morbidities or high-risk features including obesity, age, or altered mental status.[11]

Patients with AP can deteriorate rapidly, so vigilance, supported by serial re-evaluation of clinical findings and laboratory trends, is required to identify patients on a worsening trajectory, regardless of severity score.

EMERGENCY MANAGEMENT

Please refer to Figure 2 for emergency management of acute pancreatitis.

Fluid Resuscitation

In acute pancreatitis, systemically increased vascular permeability from endothelial cell injury leads to fluid losses into the peritoneum and interstitial spaces (third-spacing). This decreased intravascular volume is compounded by systemic hypovolaemia from reduced oral intake, vomiting, and sweating. The resulting pancreatic hypoperfusion results in cellular death, necrosis, and continued activation of digestive enzymes within the pancreas itself.[11,24,25] By supporting pancreatic perfusion with fluid resuscitation, it is hoped to lessen disease severity and avoid complications.

Isotonic crystalloids should be used for fluid resuscitation of patients with AP, with Hartmann's solution (Ringer's Lactate) preferred to normal saline. Hartmann's solution has shown improved outcomes in resuscitation scenarios, reducing pancreatic inflammation and subsequent injury in AP.[26,27] It also is associated with decreased disease severity, and ICU admissions, as well as reducing the risk of hyperchloraemic metabolic acidosis and acute kidney injury (AKI) associated with normal saline.[28–30]

Traditionally, AP was treated with aggressive large volume fluid resuscitation,[30] however the WATERFALL study suggested that this strategy led to fluid overload, increased symptom intensity, longer hospital stay, and a higher incidence of pancreatic necrosis and abdominal compartment syndrome.[31] A subsequent meta-analysis suggests that aggressive fluid resuscitation increases mortality in severe AP.[32] Instead of fixed fluid infusion rates or volumes, there is emerging evidence suggesting that goal-directed therapy is preferable. Several variables have been suggested as possible goal-directed targets, including urine output, serum lactate clearance, urea, and haematocrit levels, as well as mean arterial pressure (MAP).

In the initial phase of resuscitation in the ED, more moderate fluid resuscitation, aiming for 2.5-4L in the first 24 hours, appears to be beneficial to most patients with AP.[2,11,30] A suggested infusion rate based on available literature is 1.5-3mL/kg/hr with initial fluid boluses of 10mL/kg in hypotensive patients to restore an adequate MAP.[11,30]

Analgesia

All patients with AP should be prescribed analgesia as per local practices. This is often in the form of opiate analgesia such as fentanyl and morphine.[33] Due to their association with AKI, non-steroidal anti-inflammatory drugs (NSAIDs) should be avoided.[13]

Antibiotics

There is no evidence which supports the routine use of prophylactic intravenous antibiotics in the management of acute AP.[34–36]

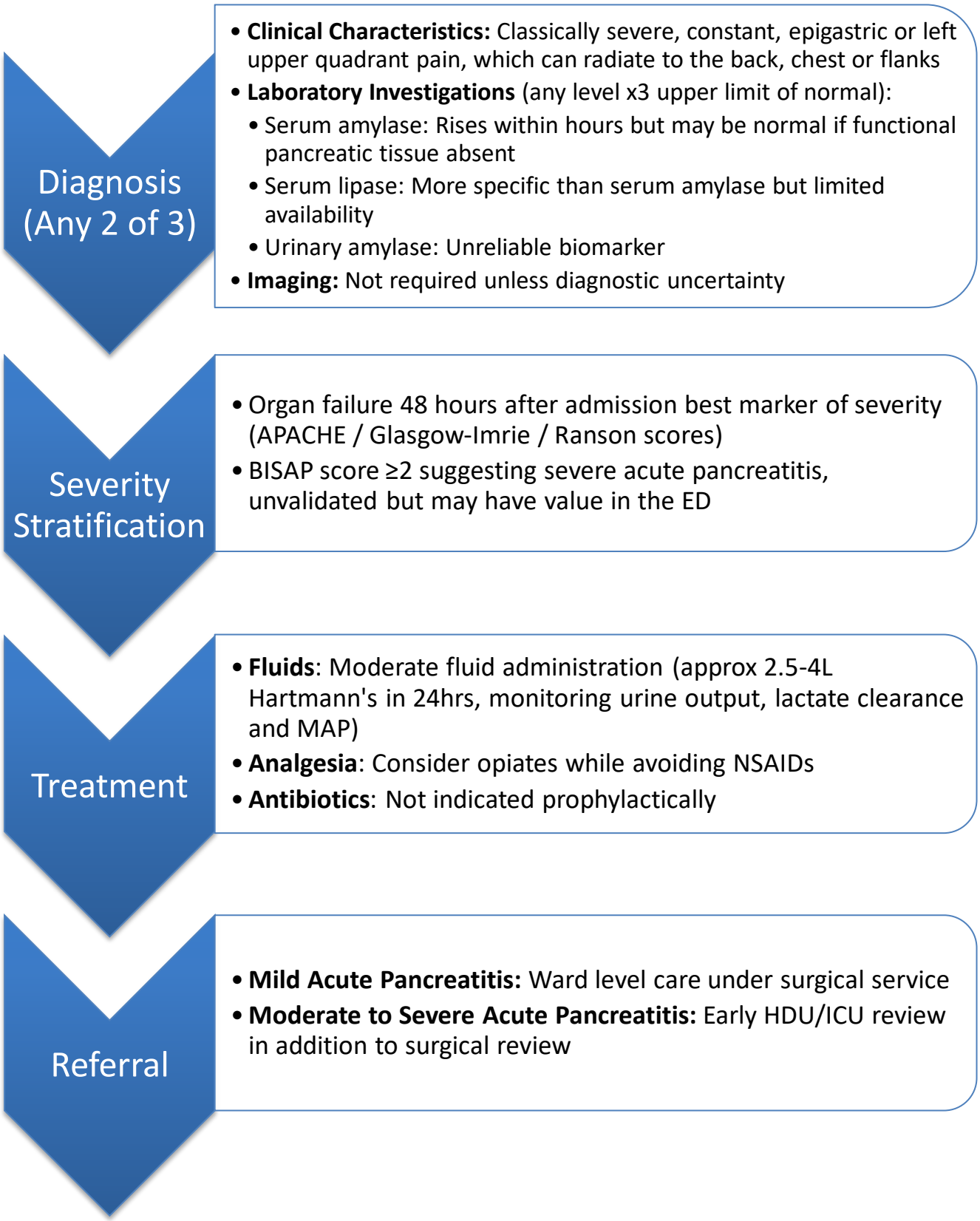

Figure 2: Emergency Department Management of Acute Pancreatitis.

NUTRITION

Historically, patients with AP were kept strictly fasting, until pain and laboratory markers began to normalise. Avoiding food-induced stimulation of exocrine function was thought to allow the pancreas 'rest' and assist in reducing inflammation. However, emerging evidence suggests that early feeding in the cases of mild to moderately severe AP is both safe and beneficial.

Oral intake assists in maintaining the integrity of the intestinal lumen, thereby limiting potential infiltration of vulnerable, inflamed pancreatic tissue by harmful gut flora. Multiple studies have demonstrated the safety of early oral feeding in cases of mild to moderately severe AP, with no increase in adverse outcomes.[11,37]

In cases of severe AP, enteral feeding is preferred to parenteral and may reduce the rate of infectious complications.[38] Nasojejunal (NJ) tubes will bypass gastric stimulation and limit exocrine stimulation, however, their placement requires endoscopic or radiological input whereas nasogastric (NG) tubes are far easier to place and were found, in at least one study, to be non-inferior to NJ tubes.[39] However, gastric outlet obstruction from peripancreatic collections or increased intra-abdominal pressure (IAP) may limit establishing enteral nutrition.

Timing of recommencing oral diet in severe AP remains controversial. Initial studies suggested that an earlier return reduced complication rates, however more recent studies failed to prove benefit from early initiation of feeds (<24 hours).[40]

CRITICAL CARE

Critical care treatment priorities include instituting aetiology-specific and supportive therapies and managing systemic and local complications.

Aetiology-specific treatment

Treatments and potential complications can differ significantly based on the underlying cause of AP.

Severe AP secondary to hypertriglyceridaemia has a higher mortality rate than AP from other causes, and when first-line medical treatment (with lipid lowering agents, heparin, and insulin) fails, therapeutic plasma exchange may be required to rapidly reduce triglyceride levels.

In patients with severe AP **and** cholangitis or persistent cholestasis, ERCP improves outcomes. A conservative strategy for gallstone pancreatitis without these features confers equivalent outcomes to an urgent intervention.[41]

Respiratory

To date there are no studies assessing the impact of non-invasive ventilation vs high-flow nasal oxygenation vs invasive ventilation in improving patient outcomes in severe AP. As a result, management should be tailored to each clinical scenario.

If intubated, these patients can exhibit altered respiratory mechanics causing challenging ventilation. Diaphragm excursion can be limited by increased abdominal pressures, while pleural effusions may impair ventilation. Severe AP may also in itself cause acute respiratory distress syndrome.

Standard lung protective parameters should be followed, namely: limiting plateau pressures to <30cmH_2O; tidal volume breaths of 6-8ml/kg predicted body weight; optimised positive end-expiratory pressure and permissive hypercarbia. Tracheostomy may ultimately be required to

facilitate what can be a slow ventilatory wean due to a combination of both pulmonary pathology and critical illness myopathy.

Cardiovascular

In patients with cardiovascular sequelae, maintaining adequate blood pressures in order to avoid tissue hypoperfusion and end organ damage is a primary goal.

Vasopressor support is often needed to maintain blood pressure in severe AP in addition to a goal-directed fluid resuscitation strategy, which avoids overly liberal fluid administration.[31]

Whether steroids may specifically modify the inflammatory response syndrome that drives shock in severe AP is the focus of ongoing research,[42] but their established role as a haemodynamic adjunct for septic shock means that steroids are frequently prescribed in severe AP.[43]

Gastrointestinal

There are multiple gastrointestinal complications that can arise from severe AP. Intra-abdominal hypertension (IAH), a sustained IAP ≥12mmHg, often accompanies severe pancreatitis and is associated with significantly higher mortality rates at both day 28 & day 90 than those patients without IAH.[44] Serial measurements of IAPs are recommended for this group.

Renal & Metabolic

Acute kidney injury (AKI) is a common sequela of severe AP, and the need for renal replacement therapy is associated with increased mortality rates.[45]

The pathogenesis of AKI in this cohort is multifactorial and includes hypovolemia, toxic inflammatory mediators from the inflamed pancreas, and perfusion limitations arising from raised IAP.

Electrolyte derangement is prevalent in severe AP. Particular considerations include the risk of hypocalcaemia (which forms part of both Glasgow-Imrie and Ranson's criteria), and which can cause neurological and cardiovascular complications, as well as hypomagnesaemia (given the association with alcohol excess and poor nutrition).

Microbiological

Diagnosing infection in this group is challenging, as inflammatory markers may be high in the setting of sterile inflammation. Procalcitonin (PCT) may be of value in guiding antibiotic use.[46]

LOCAL COMPLICATIONS

Pancreatic necrosis is a serious and potentially fatal complication associated with severe pancreatitis. Typically arising at a later phase (3-4 weeks into disease trajectory), diagnosis of pancreatic necrosis is made by a combination of CT imaging plus biochemical markers such as C-reactive protein (CRP) and PCT. If infected necrosis develops, consideration should be given to targeting resistant organisms as these patients have often had prolonged hospital stays. Therapy is ideally tailored to the specific organism isolated during culturing of fluid extracted during either surgical or radiological drainage.

Thrombotic complications associated with severe AP include splenic vein and portal vein thrombosis. This may be asymptomatic or can be clinically expressed by hepatic or mesenteric ischaemia due to occlusive processes or in GI bleeding by rupture of varices caused by portal hypertension.[44] Timely introduction of thromboprophylaxis is an important strategy to minimise such risks.

One of the most feared complications of severe AP is development of pseudoaneurysms – when amylase rich peripancreatic fluid directly damages the walls of adjacent vessels, most commonly the splenic artery. This may cause sudden, life-threatening haemorrhage. Most patients are asymptomatic until rupture of the pseudoaneurysm, which may be fatal without massive transfusion and urgent intervention. The current standard of therapy is endovascular embolisation of the bleeding vessel, which requires timely access to interventional radiologists.

SURGICAL MANAGEMENT

Patients admitted with AP are largely managed conservatively. However, in cases of mild AP with gallstone aetiology, a laparoscopic cholecystectomy should be performed on the index admission to avoid further attacks.[47] In non-gallstone related AP, or those with more severe disease, surgical interventions are reserved for those that develop sequelae, most commonly infected pancreatic necrosis or infected pancreatic pseudocysts. In these circumstances, options include necrosectomy or drainage of collections.

When surgery is required for these conditions, there is a shifting preference towards more minimally invasive techniques. Where infected pancreatic necrosis develops, the PANTER trial advocates a minimally invasive step-up approach to necrosectomy with a 43% reduction in the development of complications or death when compared to an open necrosectomy.[48] Furthermore, a step-up endoscopic approach with transenteric drainage of infected pancreatic necrosis via plastic or metal stents has shown equivalent efficacy to traditional step-up surgery with reduced pancreatic fistula rate and reduced length of hospital stay.[49] A similar method can be adopted with infected pancreatic pseudocysts.

In stable patients, consideration should be given to delaying drainage of peripancreatic collections until a rim is visible on cross-sectional imaging, indicating the collection has matured or “walled off”.[50]

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