Acute pancreatitis

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Incidence Acute pancreatitis accounts for 3% of all cases of abdominal pain among patients admitted to hospital in the UK. The hospital admission rate for acute pancreatitis is 9.8 per year per 100 /uni00A0 000 population in the UK, although worldwide the annual incidence may range from 5 to 50 per 100 /uni00A0 000. The disease may occur at any age, with a peak in young men and older women. Summary box 72.5 Possible causes of acute pancreatitis /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF The two major causes of acute pancreatitis are biliary calculi, which occur in 50–70% of patients, and alcohol abuse, which accounts for 25% of cases. Gallstone pancreatitis is thought to be triggered by the passage of gallstones down the common bile duct. If the biliary and pancreatic ducts join to share a common channel before ending at the ampulla, then obstruc - tion of this passage may lead to reflux of bile or activated pancreatic enzymes into the pancreatic duct. Patients who have small gallstones and a wide cystic duct may be at a higher risk of passing stones . The proposed mechanisms for alcoholic pancreatitis include the e ff ects of diet, malnutrition, direct toxicity of alcohol, concomitant tobacco smoking, hyperse - cretion, duct obstruction or reflux, and hyperlipidaemia. The - remaining cases may be due to rare causes or may be idiopathic. - Among patients who undergo ERCP , 1–3% develop pan - creatitis, pr obably as a consequence of duct disruption and enzyme extravasation. Patients with sphincter of Oddi dys - function or a history of recurrent pancreatitis, and those w ho undergo sphincterotomy or balloon dilatation of the sphinc - ter, carry a higher risk of developing post-ERCP pancreatitis . Patients who have undergone upper abdominal or cardiotho - racic surgery may develop acute pancreatitis in the postoper - ative phase , as may those who have su ff ered blunt abdominal trauma. Hereditary pancreatitis is a rare familial condition associ - ated with mutations of the cationic trypsinogen gene. Patients have a tendency to su ff er acute pancreatitis while in their teens, progress to chronic pancreatitis in the next two decades and have a high risk (possib ly up to 40%) of developing pancreatic cancer by the age of 70 years. Hypertriglyceridaemia should be excluded. Occasionally , tumours at the ampulla of Vater may cause acute pancreatitis. It is important to check the serum calcium level, a fasting lipid profile, autoimmune mar kers and viral titres in patients with so-called idiopathic acute pancreatitis. It is equally important to take a detailed drug history and remem - ber the association of corticosteroids, azathioprine, asparagi - nase and valproic acid with acute pancreatitis. Statins (taken over a long time) and gliptins have been linked with pancre - atitis, but the evidence is slim. It is essential to exclude tiny gallstones. A careful search for the aetiology must be made in all cases, and no more than 20% of cases should fall into the idiopathic category . Summary box 72.6 Aetiology of acute pancreatitis /uni25CF /uni25CF /uni25CF Clinical presentation Pain is the cardinal symptom. It characteristically develops quickly , reaching maximum intensity within minutes rather than hours and persists for hours or even days. The pain is

Gallstones Alcoholism Post ERCP Abdominal trauma Following biliary, upper gastrointestinal or cardiothoracic surgery Ampullary tumour Drugs (corticosteroids, azathioprine, asparaginase, valproic acid, thiazides, oestrogens) Hyperparathyroidism Hypercalcaemia Hypertriglyceridaemia Pancreas divisum Sphincter of Oddi dysfunction Autoimmune pancreatitis Hereditary pancreatitis Viral infections (mumps, coxsackie B) Malnutrition Scorpion bite Idiopathic It is essential to establish the aetiology Investigate thoroughly before labelling it as ‘idiopathic’ If due to gallstones, cholecystectomy is desirable during the same admission

of analgesics. Pain is usually experienced first in the epigas trium but may be localised to either upper quadrant or felt di ff usely throughout the abdomen. There is radiation to the back in about 50% of patients, and some patients may g relief by sitting or leaning forwards. The suddenness of onset may simulate a perforated peptic ulcer, while biliary colic or acute cholecystitis can be mimicked if the pain is maximal in the right upper quadrant. Radiation to the chest can simulate myocardial infarction, pneumonia or pleuritic pain. In fact, acute pancreatitis can mimic most causes of the acute abdo men and should seldom be discounted in di ff erential diagnosis. Nausea, repeated vomiting and retching are usually marked. The retching may persist despite the stomach being kept empty by nasogastric aspiration. Hiccoughs can be trou blesome and may be due to gastric distension or irrita the diaphragm. On examination, the appearance may be that of a patient who is well or, at the other extreme, one who is gravely ill with profound shock, toxicity and confusion. Tachypnoea is com mon, tachycardia is usual and hypotension may be present. The body temperature is often normal or even subnormal, but frequently rises as inflammation develops. It is useful to reiter ate here tha t SIRS is defined by the presence of two or more of the following criteria: heart rate >90/min, core temperature <36°C or >38°C, respirations >20/min or P CO <32 /uni00A0 mmHg, 2 3 and white blood cell count <4000 or >12 /uni00A0 000/mm (see also Chapter 2). Mild icterus can be caused by biliary obstruction in gallstone pancreatitis, and an acute swinging pyrexia sug gests cholangitis. Bleeding into the fascial planes can produce bluish discoloration of the flanks (Grey Turner’s sign) or umbi licus (Cullen’s sign). Subcutaneous fat necr osis may produce small, red, tender nodules on the skin of the legs. Abdominal examination may reveal distension due to ileus or, more rarely , ascites with shifting dullness. A mass can develop in the epig astrium owing to inflammation. There is usually muscle guarding in the upper abdomen, although marked rigidity is unusual. A pleural e ff usion is present in 10–20% of patients. Pulmonary oedema and pneumonitis are also described and may give rise to the di ff erential diagnosis of pneumonia or myocardial infarction. The patient may be con fused and exhibit the signs of metabolic derangement together with hypoxaemia. Investigations Typically , the diagnosis is made on the basis of the clinical presentation and an elevated serum amylase level. A serum amylase level three times above normal is indicative of the disease. A normal serum amylase level does not exclude acute pancreatitis, particularly if there is delay in presentation. The serum lipase level provides a more sensitive and specific test George Grey Turner , 1877–1951, Professor of Surgery , Durham University , Durham (1927–1934), and at the Postgraduate Medical School, Hammersmith, London, UK (1934–1945). Thomas Stephen Cullen , 1870–1953, Professor of Gynecology , Johns Hopkins University , Baltimore, MD, USA. Described bluish discoloration of the perium bilical skin as a sign of ruptured ectopic pregnancy . John HC Ranson , 1938–1995, Professor of Surgery , New Y ork University School of Medicine, New Y ork, NY , USA. J ohn C Marshall , contemporary , trauma surgeon and intensivist, St Michael’s Hospital, Toronto, Canada. - abdomen have to be excluded, contrast-enhanced CT is the best single imaging investigation. ain Summary box 72.7 Investigations in acute pancreatitis should be aimed at answering three questions:

/uni25CF /uni25CF - /uni25CF - Assessment of severity tion of It is important to identify those patients who will develop severe pancreatitis as they require aggressive early manage - ment and possibly transfer to a specialist unit. A severe attack may be heralded by an initial clinical impression of a very ill - patient and a worsening physiological state at 24–48 hours. Various prognostic scoring systems hav e been used, all aimed at predicting persistent organ failure, particularly respiratory , - cardiac and renal. Severity stratification assessments should be performed in patients at 24 hours, 48 hours and 7 days after admission. The Ranson and Glasgow scoring systems are specific for acute pancreatitis, and a score of 3 or more at 48 hours indicates a severe attack (Table 72.3). Several other systems that are used in intensive care units can also be applied. - These include the APACHE, SAPS, SOFA, MODS and modi - fied Marshall scoring systems (the latter has the advantage of - simplicity). Regardless of the system used, persisting organ fail - ure indicates a severe attack. A serum C-reactive protein le vel >150 /uni00A0 mg/L at 48 hours after the onset of symptoms is also an indicator of severity . Patients with a body mass index over 30 are at higher risk of developing complications. A revision in 2013 of the Atlanta classification of acute pancreatitis (1992) recommends that patients with acute pancreatitis be stratified into three groups: /uni25CF Mild acute pancreatitis: /uni25CF - no organ failure; /uni25CF no local or systemic complications. /uni25CF Moderately severe acute pancreatitis: /uni25CF organ failure that resolves within 48 hours (transient organ failure); and/or /uni25CF local or systemic complications without persistent or - gan failure. /uni25CF Severe acute pancreatitis: /uni25CF persistent organ failure (>48 hours); /uni25CF single organ failure; /uni25CF multiple organ failure. -

Is a diagnosis of acute pancreatitis correct? How severe is the attack? What is the aetiology?

Imaging Plain erect chest and abdominal radiographs are not diagnostic of acute pancreatitis but are useful in the di ff erential diagnosis. Non-specific findings in pancreatitis include a generalised or local ileus (sentinel loop), a colon cut-o ff sign and a renal halo sign. Occasionally , calcified gallstones or pancreatic calcifi cation may be seen. A chest radiograph may show a pleural e ff usion and, in severe cases, a di ff use alveolar interstitial shadowing may suggest acute respiratory distress syndrome. Ultrasonography does not estab lish a diagnosis of acute pancreatitis. The swollen pancreas may be seen, but ultraso nography should be performed within 24 hours in all patients to detect gallstones as a potential cause , rule out acute chole cystitis as a di ff erential diagnosis and determine whether the common bile duct is dilated. CT is not necessary for all patients, particularly those deemed to have a mild attack on prognostic criteria. But a contrast-enhanced CT is indica ted in the following situations: /uni25CF If there is diagnostic uncertainty . /uni25CF In patients with severe acute pancreatitis to distinguish in terstitial from necrotising pancreatitis (Figure 72.22 the first 72 hours, CT may underestimate the extent of necrosis. The severity of pancreatitis detected on CT may be staged according to the Balthazar criteria. /uni25CF In patients with organ failure, signs of sepsis or progressive clinical deterioration. /uni25CF When a localised complication is suspected, such as fluid collection, pseudocyst or a pseudoaneurysm. Cross-sectional MRI can yield similar information to that obtained by CT . EUS and MRCP can help in detecting stones in the common bile duct and directly assessing the pancreatic par enchyma but are not widely available. ERCP allows the identification and removal of stones in the common bile duct Emil J Balthazar , contemporary , Professor Emeritus, Department of Radiology , New Y ork University , New Y ork, NY , USA. - x - - -). In in gallstone pancreatitis. In patients with severe acute gallstone pancreatitis and signs of ongoing biliary obstruction and chol - angitis, an urgent ERCP should be sought. The presentation is so variable that sometimes even an experienced clinician

can be mistaken. While this is not desir - able, occasionally the diagnosis is only made at laparotomy . The appearances at laparotomy are characteristic (Figure 72.23).

disease is classi /f_i ed as severe when three or more factors are present. Ranson score On admission Age >55 years 9 White blood cell count >16 × 10 /L Blood glucose >11 /uni00A0 mmol/L (>200 /uni00A0 mg/dL) LDH >350 units/L AST >250 units/L Within 48 hours Haematocrit fall of 10% or greater Blood urea nitrogen rise >5 /uni00A0 mg/dL (1.8 /uni00A0 mmol/L) despite /f_l uids Arterial oxygen saturation (PaO) <8 /uni00A0 kPa (60 /uni00A0 mmHg) 2 Serum calcium <8 /uni00A0 mg/dL (2.0 /uni00A0 mmol/L) Base de /f_i cit >4 /uni00A0 mmol/L Fluid sequestration >6 litres AST, aspartate aminotransferase; LDH, lactate dehydrogenase; PaO Glasgow score Within 48 hours Age >55 years 9 White blood cell count >15 × 10 /L Blood glucose >10 /uni00A0 mmol/L (no history of diabetes) LDH >600 units/L or AST >200 units/L Serum urea >16 /uni00A0 mmol/L (no response to intravenous /f_l uids) Arterial oxygen saturation (PaO) <8 /uni00A0 kPa (60 /uni00A0 mmHg) 2 Serum calcium <2.0 /uni00A0 mmol/L Serum albumin <32 /uni00A0 g/L , arterial oxygen tension. 2 Figure 72.22 Contrast-enhanced computed tomography scan showing acute necrotising pancreatitis. Note the area of reduced enhancement in the pancreas (marked X), the peripancreatic oedema and stranding of the fatty tissues (courtesy of Dr Niall Power).

Management If after initial assessment a patient is considered to have a mild attack of pancreatitis, a conservative approach is indicated with intravenous fluid administration and frequent, but non-invasive, observation. A brief period of fasting may be sensible in a patient who is nauseated and in pain, but there is little physiological justification for keeping patients on a prolonged ‘nil by mouth’ regimen. Antibiotics are not indicated. Apart from analgesics and antiemetics, no drugs or interventions are warranted, and CT scanning is unnecessary unless there is evidence of deterioration. However, if a stable patient meets the prognostic criteria for a severe attack of pancreatitis, then a more aggressive approach is required, with admission to a high-dependency or intensive care unit and invasive monitoring (Table 72.4). Adequate analgesia should be administered. Aggressive fluid resuscitation is important, guided by frequent measure ment of vital signs, urine output and central venous pr Supplemental oxygen should be administered and serial arte rial blood gas analysis performed. The haematocrit, clotting profile, blood glucose and serum levels of calcium and magne sium should be closely monitored. A nasogastric tube is not essential but may be of value in patients with vomiting. Specific tr eatments such as aprotinin, somatostatin analogues, platelet-activating factor inhibitors and selective gut decontamination hav e failed to improve out come in numerous clinical trials. There are no data to support a practice of ‘resting’ the pancreas and feeding only by the parenteral or nasojejunal routes. If nutritional support is felt to be necessary , enteral nutrition (e.g. feeding via a nasogastric tube) should be used. There is some evidence to support the use of prophylactic antibiotics in patients with severe acute pancreatitis but there is no consensus. The rationale is to prevent local and other septic complications. T he regimens used include intravenous cefuroxime, or imipenem, or ciprofloxacin plus metronidazole. The duration of antibiotic prophylaxis should not exceed 14 days. Additional antibiotic use should be guided by microbio - logical cultures. If, however, there is evidence of cholangitis or concomitant respiratory or urinary infection then antibiotics should be given promptly . If gallstones are the cause of an attack of predicted or proven se vere pancreatitis, or if the patient has jaundice, chol - angitis or a dilated common bile duct, ERCP should be carried out within 72 hour s of the onset of symptoms as sphincterot - omy and clearance of the bile duct can

reduce the incidence of - infective complications. In patients with cholangitis, sphincter - essure. otomy should be car ried out or a biliary stent placed to drain - the duct; however, ERCP is an invasiv e procedure and carries a small risk of worsening the pancreatitis . - Systemic complications Pancreatitis may involve all organ systems (Table 72.5) and should be managed by a multidisciplinary team including - intensive care specialists. When there is organ failure, appro - priate supportive therapies may include inotropic support for haemodynamic instability , haemofiltration in the event of renal failure, ventilatory support for respirator y failure and

Figure 72.23 Widespread fat necrosis of the omentum. A test tube has been /f_i lled with blood-stained peritoneal /f_l uid. This specimen was rich in amylase. Fat necroses are dull, opaque, yellow-white areas suggestive of drops of wax. They are most abundant in the vicinity of the pancreas but are widespread in the greater omentum and the mesentery. Fat necroses consist of small islands of saponi /f_i cation caused by the

liberation of lipase, which splits into glycerol and fatty acids. Free fatty acids combine with calcium to form soaps (fatty necrosis) (courtesy of Dr GD Adhia, Mumbai, India). pancreatitis. Admission to HDU/ICU Analgesia Aggressive /f_l uid rehydration Supplemental oxygen Invasive monitoring of vital signs, central venous pressure, urine output, blood gases Frequent monitoring of haematological and biochemical parameters (including liver and renal function, clotting, serum calcium, blood glucose) Nasogastric drainage (only

initially) Antibiotics if cholangitis suspected; prophylactic antibiotics can be considered CT scan essential if organ failure, clinical deterioration or signs of sepsis develop ERCP within 72 hours for severe gallstone pancreatitis or signs of cholangitis Supportive therapy for organ failure if it develops (inotropes, ventilatory support, haemo /f_i ltration, etc.) If nutritional support is required, consider enteral (nasogastric) feeding CT, computed tomography; ERCP , endoscopic retrograde cholangio

pancreatography; HDU, high-dependency unit; ICU, intensive care unit.

role during the initial period of resuscitation and stabilisation and is reserved for the patient who deteriorates following successful stabilisation. Local complications and their management Once the patient survives the acute phase and major organ failure is controlled, local complications become pre-eminent as they carry a significant mortality . A CT scan should be performed if pain persists, signs of sepsis develop, organ dysfunction worsens or there is a further spike in the serum amylase level. The management is conservative with surgery only when conservative management has failed. Definitions are important. Terms such as ‘phlegmon’, which may refer to an abscess or to an inflammatory mass in the pancreas, are best avoided. Acute peripancreatic fluid collection Acute peripancreatic fluid collection (APFC) occurs early in the course of mild pancreatitis without necrosis and is located adjacent to the pancreas. It has no encapsulating wall and is confined within normal fascial planes. The fluid is sterile and most such collections resolve. No intervention is necessary unless a large collection causes symptoms or pressure e ff ects, in which case it can be percutaneously aspirated under ultrasound ultrasound guidance is another option. Sterile and infected pancreatic necrosis The term ‘pancreatic necrosis’ refers to a di ff use or focal area of non-viable parenchyma. This can be identified by an absence of parenchymal enhancement on CT with intravenous contrast. Pancreatic necrosis is typically associated with lysis of peripancreatic fat. This may lead to an acute necrotic collection (ANC). This is typically an intra- or extrapancreatic collection containing fluid and necrotic material, with no definable wall. Gradually , over a period of over 4 weeks, this may develop a well-defined inflammatory capsule and evolve into walled-o ff necrosis (WON). Collections associated with necrotising pancreatitis are sterile to begin with but often become subsequently infected, probably because of translocation of gut bacteria. Infected necrosis is associated with a mortality rate of up to 50%. Ster - ile necrotic ma terial should not be drained or interfered with. However, if the patient shows signs of sepsis, then one should determine whether the collection is infected (Figur e 72.24). Aspiration fluid with a fine needle, percutaneously under CT or ultrasound guidance, can provide the answer. If the aspirate is purulent, drainage of the infected fluid should be carried out. Internal drainage into the stomach under endoscopic ultrasound guidance should be considered first. A plastic or covered metal stent can be used to create a communication between the collection and the gastric lumen. The stent may be left in for weeks if necessary and may need to be changed if blocked. If endoscopic internal drainage is not possible, then percutaneous drainage should be considered. The tube drain inserted should have the widest bore possible. The aspirate should be sent for microbiological assessment and appropriate antibiotic therapy should be commenced as per the sensitivity report. The fluid can be quite viscous with particulate mat - ter, and the drain may need regular flushing with full aseptic

TABLE 72.5 Complications of acute pancreatitis. Systemic Local (More common in the /f_i rst week) (Usually develop after the /f_i rst week) Cardiovascular Peripancreatic /f_l uid collection Shock Sterile pancreatic necrosis Arrhythmias Infected pancreatic necrosis Pulmonary Pancreatic abscess ARDS Pseudocyst Renal failure Pancreatic ascites Haematological Pleural effusion DIC Portal/splenic vein thrombosis Metabolic Pseudoaneurysm Hypocalcaemia Hyperglycaemia Hyperlipidaemia Gastrointestinal Ileus Neurological Visual disturbances Confusion, irritability Encephalopathy Miscellaneous Subcutaneous fat necrosis Arthralgia ARDS, acute respiratory distr ess syndrome; DIC, disseminated intra- vascular coagulation. Figure 72.24 Infected pancreatic necrosis. Note the areas of reduced enhancement in the pancreas and the peripancreatic /f_l uid collection with

pockets of gas within it (arrow). This resolved after percutaneous drainage and antibiotic therapy.

of progressively wider drains is necessary . Pancreatic necrosectomy should be considered if sepsis worsens despite conservative measures. This is a challenging operation that carries a high morbidity and mortality; it is best carried out in a specialist unit and is necessary only in a very small proportion of patients. The surgical approach may be through a midline laparotomy , especially if the area involved is around the pancreatic head. The duodenocolic and gastro colic ligaments should be divided and the lesser sac opened. Thorough debridement of the dead tissue around the pancreas should be carried out. If the body and tail of the gland are primarily involved (Figur e 72.25), a retroperitoneal approach through a left flank incision may be more appropriate. The tissues are inevitably friable, and one should be careful not to precipitate excessive bleeding or inadvertently breach the bowel wall. Blunt dissection is preferable to sharp dissection. A feeding jejunostomy may be a useful adjunct to the procedure. If gallstones are the precipitating factor of the pancreatitis, a cholecystectomy should be included. Some prefer a minimally invasive approach to a formal laparotomy . A rigid laparoscope is inserted into the peripancreatic area through a retroperi toneal approach, and vigorous irrigation and suction is com bined with a gradual nibbling away of the necrotic debris. Once a necrosectomy has been completed, further necrotic tissue may form. There are several possible ways of dealing with this (listed below), none of whic h has been proved to be /uni00A0 more e ff ective than the others. The last two approaches make greater logistic demands as one is committed to a re-exploration every 48–72 hours. /uni25CF Closed continuous lavage. Tube drains are left in and the raw area flushed (Beger) (Figure 72.26). /uni25CF Closed drainage. The incision is closed, but the cavity is packed with gauze-filled Penrose drains and closed suc tion drains. The Penrose drains are brought out through the flank and slowly pulled out and removed after 7 days. /uni25CF Open packing. The incision is left open, and the cavity is packed with the intention of returning to the operat ing room at regular intervals and repacking until there is a clean granulating cavity . /uni25CF Closure and relaparotomy . The incision is closed with drains with the intention of performing a series of planned relaparotomies every 48–72 hours until the raw area granulates (Bradley). There is a subgroup of patients who respond initially to percutaneous treatment but then develop recurrent sepsis that requires repeated insertion of drains and fail to thriv e. Necro sectomy should be considered in these patients, but it can be a di ffi cult judgement call. Patients with peripancreatic sepsis are ill for long periods of time and may require management in an intensive care unit. Nutritional support is essential. The parenteral and nasojeju nal appr oaches are more popular (on the assumption that they Hans Günter Beger , b. 1936, Emeritus Professor of Surgery , Ulm, Germany . Charles Bingham Penrose , 1862–1925, Professor of Gynecology , University of Pennsylvania, Philadelphia, PA, USA. Edward Bradley III , contemporary , Emeritus Professor of Surgery , Florida State University College of Medicine, FL, USA. - - - - - rest the pancreas), although there is little evidence to show that nasogastric feeding, if tolerated, is harmful in any way . Pancreatic abscess This is a circumscribed intra-abdominal collection of pus, usually in proximity to the pancreas. It may be an ANC or a WON that has become infected. The principles of diagnosis and management are as outlined above for infected pancreatic necrosis. Endoscopic internal drainage or, failing that, percu - - taneous drainage with the widest possible drains is the treat - ment, along with appropriate antibiotics and supportive care. Repeated scans may be required depending on the progress of the patient, and drains may need to be flushed, repositioned or reinserted. V ery occasionally , open drainage of the abscess - may be necessary .

Figure 72.25 Necrotic body and tail of the pancreas removed as an intact specimen rather than piecemeal. The patient had suffered severe necrotising gallstone pancreatitis complicated by persistent pancreatic sepsis. Necrosectomy was carried out through a left /f_l ank retroperitoneal approach. Figure 72.26 Continuous postoperative closed lavage of the lesser sac as advised by Beger. Lavage is carried out through several double-lumen and single-lumen catheters. Each time, 1 litre of saline is infused through and then drained over a period of hours, and the process is repeated.

This is a chronic, generalised, peritoneal, enzyme-rich e ff usion usually associated with pancreatic duct disruption. Paracente sis will reveal turbid fluid with a high amylase level. Adequate drainage with wide-bore drains placed under imaging guidance is essential. Measures that can be taken to suppress pancreatic secretion include parenteral or nasojejunal feeding and administration of octreotide. An ERCP may demonstrate duct disruption and allow placement of a pancr eatic stent. Pancreatic effusion This is an encapsulated collection of fluid in the pleural cavity , arising as a consequence of acute pancreatitis. Concomitant pancreatic ascites may be present or there may be a commu nication with an intra-abdominal collection. Percutaneous drainage under imaging guidance is necessary . Haemorrhage Bleeding may occur into the gut, the retroperitoneum or peri toneal cavity . Possible causes include bleeding into a pseudocyst cavity , di ff use bleeding from a large raw surface or a pseudoan eurysm. The last is a false aneurysm of a major peripancreatic vessel confined as a clot by the surrounding tissues and often associa ted with infection. Recurrent bleeding is common, often culminating in fatal haemor rhage. CT , angiography or magnetic resonance angiography helps to make the diagnosis. Treatment involves embolisation or surgery . Portal or splenic vein thrombosis This may develop silently and is identified on a CT scan. A marked rise in the platelet count should raise suspicions. In the context of acute pancreatitis, treatment is usually conservative. The patient should be screened for procoagulant tendencies. If varices or other manifestations of portal hypertension develop, they will require treatment, such as endoscopic injection or banding, β -blockade, etc. Thrombocytosis may mandate the use of aspirin or other antiplatelet drugs for a period. Systemic anticoagulation, if instituted early in the process, may achieve recanalisation of the vein but it is not routinely used as it carries considerable risks in a patient with ongoing pancreatitis. Pseudocyst A pseudocyst is a collection of amylase-rich fluid enclosed in a well-defined wall of fibrous or granulation tissue. Pseudocysts typically arise following an attack of mild acute pancreatitis, lie outside the pancreas and represent an APFC that has not resolved and matured. Formation of a pseudocyst requires 4 /uni00A0 weeks or more from the onset of acute pancreatitis. The term ‘pseudocyst’ is often used more loosely to include sterile WON that has failed to resolve or a collection that has developed in the context of chronic pancreatitis or after pancreatic trauma. (Figure 72.27 ; see also Figure 72.10). More than half have a communication with the main pancreatic duct. Pseudocysts are often single but are occasionally multiple. It is important to di ff erentiate a pseudocyst from an APFC; the clinical scenario and radiological appearances should allow that distinction to be made. Occasionally , a cystic neo plasm may be confused with a chronic pseudocyst. EUS and The fluid should be sent for measurement of carcinoembryonic antigen (CEA) le vels, amylase levels and cytology . Fluid from a - pseudocyst typically has a low CEA level, and levels above 400 /uni00A0 ng/mL are suggestive of a mucinous neoplasm. Pseudo - cyst fluid usually has a high amylase level, but that is not diag - nostic as a tumour that communicates with the duct system may yield similar findings. Cytology typically reveals inflam - matory cells in pseudocyst fluid. If there is no access to EUS, not then

percutaneous FNA is acceptable (just aspiration, percutaneous insertion of a drain). ERCP and MRCP may demonstrate communication of the cyst with the pancreatic duct system, demonstrate ductal anomalies or diagnose chronic pancreatitis, and thus help in planning treatment. - Pseudocysts usually resolve spontaneously , but compli - cations can develop (Table 72.6) . Pseudocysts that are thick walled or large (>6 /uni00A0 cm in diameter), have lasted for a long time (over 12 weeks) or have arisen in the context of chronic pan - creatitis are less likely to resolve spontaneously . Therapeutic - intervention is advised only if the pseudocyst causes symptoms, complications develop or distinction has to be made between a - pseudocyst and a tumour. There are three possible approaches to draining a pseudo - cyst: percutaneous , endoscopic and surgical. Percutaneous drainage to the exterior under radiological guidance should be avoided. It carries a very high likelihood of recurr ence. Moreover, it is not advisable unless one is absolutely certain that the cyst is not neoplastic and that it has no communication with the pancreatic duct (or else a pancreaticocutaneous fistula will develop). A percutaneous transgastric cystgastrostomy can be performed under imaging guidance, and a double-pigtail drain placed with one end in the cyst cavity and the other end in the gastric lumen. In experienced hands, recurrence rates are no more than 15%. Endoscopic drainage usually involves -

Figure 72.27 Barium meal. Pseudocyst displacing the stomach (cour

tesy of Professor VK Kapoor, Lucknow, India).

puncture of the cyst through the stomach or duodenal wall under endoscopic ultrasound guidance, and placement of a tube drain with one end in the cyst cavity and the other end in the gastric lumen. The success rates depend on operator expertise. Occasionally , ERCP and placement of a pancreatic stent across the ampulla may help to drain a pseudocyst that is in communication with the duct. Surgical drainage involves internally draining the cyst into the gastric or jejunal lumen (Figure 72.28). Recurrence rates should be no more than 5%, and this still remains the standard against which the evolving radiological and endoscopic approaches are measured. The approach is conventionally through an open incision but lapa roscopic cystgastrostomy is also feasible. Pseudocysts that have developed complications are best managed surgically . There is a small group of patients who, having su ff ered an attack of necr otising pancreatitis with duct disruption, go on to su ff er repeated complications in the form of recurrent fluid collections, pseudocysts, pleural e ff usions or pancrea tic ascites. V ery often disruption of the main pancreatic duct in the neck, body or tail is compounded by a stricture or a stone in the head that cannot be treated endoscopically . In such patients, some form of surgical resection and/or a drainage procedure – even though it may be technically challenging – may be the only way to achieve lasting resolution. Summary box 72.8 Distinguishing a pseudocyst from a cystic neoplasm /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF William Wayne Babcock , 1872–1963, surgeon, Philadelphia, PA, USA.

pseudocyst. Process Outcomes Infection Abscess Systemic sepsis Rupture Into the gut Gastrointestinal bleeding Internal /f_i stula Into the peritoneum Peritonitis Enlargement Pressure effects Obstructive jaundice from biliary compression Bowel obstruction Pain Erosion into a vessel Haemorrhage into the cyst Haemoperitoneum History Appearance on CT and ultrasonography FNA of /f_l uid, preferably under endoscopic ultrasound guidance: CEA (high level in mucinous tumours) Amylase (level usually high in pseudocysts but occasionally in tumours) Cytology Figure 72.28 Cystgastrostomy for the pancreatic pseudocyst shown in Figure 72.10 . The anterior wall of the stomach has been opened and the edges drawn back, held by Babcock’s forceps. An opening has been made through the posterior wall of the stomach into the pseudocyst and the tips of the dissecting forceps are in the cavity of the pseudocyst, which is lined by slough and granulation tissue. The tip of a nasogastric tube is visible. A running stitch will next be placed along the edges of this opening, suturing the full thickness of the posterior gastric wall to the capsule of the pseudocyst.

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