A 19-year-old man, Ian Grove, presents to the emergency department with severe central abdominal pain with nausea and vomiting. He is rolling around in agony with tears in his eyes. He has never experienced pain like this before.

What is the first thing you want to do?
Ask him if he has any allergies or takes any regular pain medications. Prescribe him a small amount of fast-acting opioid analgesia, such as pethidine or morphine. This will make him more comfortable, enable him to answer your questions and examine him.

What do you want to know about his abdominal pain?

Abdominal pain
There are 10 main features you want to address when asking a patient to describe abdominal pain:
• Character: dull ache, colicky spasms, deep or superficial.
• Location.
• Radiation: to back, loins, groin, chest, shoulder tip or other area of the abdomen.
• Onset: sudden or gradual.
• Progression: is it becoming more severe and intense or constant?
• Intensity: grading scale from 1 to 10, with 10 the worst pain imaginable.
• Frequency: this can be the number of times in a day/week/month/year.
• Duration: when was it first noticed? This may be a prior episode.

• Aggravating and relieving factors: related to posture, eating meals, worsened by movement, inspiration or coughing, better on lying still.
• Associated symptoms.

Associated gastrointestinal symptoms
• Does he have heartburn or reflux? Think peptic ulcer.
• Is there associated nausea and vomiting? Did this precede the pain? What is its frequency and character? Is there haematemesis? Think gallstones, appendicitis and pancreatitis.
• Has he lost his appetite? Is there any weight loss? He may have an inflammatory condition, such as Crohn’s disease with a stricture and obstructive symptoms. He is young and unlikely to have a malignant process.
• Has he had a change in bowel habit? Is there rectal bleeding or melaena? Are there oily stools that are smelly and difficult to flush (steatorrhoea)? Think peptic ulcer and pancreatitis, respectively.
• Does he feel feverish or has he had a rigor? Has he become jaundiced? Is he itching? Think of cholecystitis and cholangitis.

Systemic enquiry
Ask questions to identify evidence of multisystem disease, referred pain or additional pathology.
• Does he have any respiratory symptoms such as a cough, wheeze, sputum and breathlessness? Consider basal pneumonia and pleurisy.
• Does he have cardiac symptoms such as palpitations, chest pain, orthopnoea or syncope? Consider cardiac ischaemia, mesenteric ischaemia or pericarditis causing pain.
• Does he have neurological symptoms such as weakness, parasthesia, spinal pain, gait disturbance or incontinence? Consider spinal lesions or prolapse with radiation of pain.
• Does he have any urological symptoms of frequency, urgency, loin pain or haematuria? Is he in urinary retention? Pain may radiate from renal calculi.
• Does he have a rash? Consider herpes zoster even in the absence of a rash if the pain is dermatomal in distribution.
• Does he have a hernia or testicular problem? Could he have torsion of his testes?
• Has he had a prodromal viral illness? Consider viral or bacterial pancreatitis.

Past history
• Has he had recent surgery? Adhesions could cause intestinal obstruction.
• Has he had recent traumatic injury? Consider delayed splenic rupture.
• Has he had a recent viral infection? Consider pancreatitis due to mumps.
• Does he have a history of gallstone disease or peptic ulcer?

Medications
• Does he take any regular analgesics for pain? Opioid addiction should not be assumed but always considered in the context of prior seeking for opioid drugs.
• Does he take other medications either prescribed or over-the-counter? For example, a side effect of thiopurines (azathioprine, mercaptopurine) is pancreatitis.

Social history
• Does he drink alcohol, and how much? Does alcohol influence the pain? Consider pancreatitis.
• Does he smoke or is he a recent ex-smoker? This is relevant in inflammatory bowel disease.

Family history
• Are there any abdominal complaints that could be inherited? For example, hereditary pancreatitis.

He tells you the pain started suddenly in the central upper abdomen yesterday and has become progressively worse. It is constant and dull. It makes him feel sick and he has vomited food and bilious fluid three to four times. He has loose stools, opening his bowels three times today. He did not look to see if there was blood. He feels warm but does not describe a fever or rigors.

He has no previous medical problems and takes no regular medications. He does not smoke and only drinks at weekends with his friends. He denies binge drinking. He is studying to be a vet and is in his third term at university.

There is no family history.

Examination
General examination
• Vital signs: determine his temperature, pulse, blood pressure, respiratory rate and oxygen saturations to look for haemodynamic compromise.
• Glasgow coma score: determine level of consciousness (beware because opiate analgesia can cause drowsiness and constricted pinpoint pupils).
• Scleral icterus: jaundice may be accompanied by excoriations or scratch marks from itching.
• Cervical lymphadenopathy: suggestive of mesenteric adenitis.

Abdominal examination
• Bluish discoloration around the umbilicus (Cullen’s sign) or reddish-brown discoloration along the flanks (Grey Turner’s sign) from retroperitoneal blood in severe necrotising pancreatitis (see Plate 20.1).
• Scars, distension or visible peristalsis. Consider adhesions if there has been prior surgery.
• Abdominal tenderness, rebound and guarding. Are there signs of peritonism?
• Palpable masses or organomegaly, for example an appendiceal abscess or inflamed gallbladder.
• Bowel sounds may be hyperactive in obstruction and quiet in peritonitis.
• Hernia orifices should be examined for an obstructed hernia.

Digital rectal examination
Tender rectal examination may occur in appendicitis, depending on the position of the inflamed appendix.

Testes examination
A testes exam should be done to exclude torsion.

Systemic examination
• Chest exam: are there chest crepitations indicative of a pneumonia or acute respiratory distress syndrome (ARDS)? Is there a clinical pleural effusion?
• Cardiac exam: pericarditic rub or heart murmur?
• Neurological exam: focal neurology or spinal tenderness with a sensory level?
• Renal angles: loin tenderness?
• Skin exam: rash with vesicles from herpes zoster? Widespread purpuric rash from disseminated intravascular coagulation (DIC)?

He looks unwell on examination. His blood pressure is 80/40mmHg, pulse is 120beats/min and he has a low grade
pyrexia of 37.5°C. His abdomen is tender in the epigastrium and central abdomen with voluntary guarding and hypoactive bowel sounds. He is not peritonitic. Chest sounds are vesicular bilaterally and heart sounds are normal with no rubs or murmurs. There is no musculoskeletal tenderness or focal neurology. His testes are normal. There is no tenderness on DRE.

KEY POINTS

- A careful abdominal pain history is crucial in narrowing the range of differential causes.
- Referred pain from non-abdominal sites should be considered in the differential.
- Never forget the testes and hernia orifices in assessment of abdominal pain.

What is your differential diagnosis?
The differential diagnosis of acute abdominal pain is vast. He is a young man, only 19 years old, and describes an epigastric, dull, constant ache with associated nausea, vomiting and change of bowel habit. Differential diagnoses include:

- Peptic ulcer.
- Gastritis.
- Acute pancreatitis.
- Gallstones.
- Renal stones.
- Mesenteric adenitis.
- Intestinal obstruction.
- Cholecystitis.
- Addison’s crisis.
- Sickle cell crisis.
- Acute intermittent porphyria.

What investigations are appropriate?
There are some general investigations that should be done for all causes of abdominal pain and more specific ones depending on the likely cause.

General investigations
Blood tests
- Full blood count should be taken for evidence of anaemia (inflammatory bowel disease, peptic ulcer) and leucocytosis (inflammatory or infective disease).
- C-reactive protein is elevated in many inflammatory and infective conditions.
- Urea, creatinine and electrolytes may be affected by dehydration, vomiting and diarrhoea.
- Liver function may be abnormal in cholangitis, hepatitis and sepsis of any source.
- Coagulation may be impaired, especially in sepsis with DIC.
- Serum amylase and/or lipase may be markedly raised in acute pancreatitis and minimally elevated in small intestinal obstruction, mesenteric ischaemia, infarcted bowel, perforated peptic ulcer or renal insufficiency.
- Calcium and albumin levels to detect hypercalcaemia, which may cause abdominal pain.
- Blood glucose for pancreatic endocrine dysfunction and in diabetic ketoacidosis.

Urine
Dipstick may show blood and protein in pyelonephritis. A midstream urine specimen should be sent for culture.

Electrocardiogram
This will identify an arrhythmia or ischaemia.

Arterial blood gas
- Metabolic acidosis in sepsis, infarcted bowel and diabetic ketoacidosis. Lactate may be raised.
- Hypoxia may occur in chest pathology.

Chest X-ray
A CXR will look for evidence of air under the diaphragm from a perforated viscus. This will also identify consolidation or an effusion.

Abdominal X-ray
This is used to look for dilated loops of bowel in obstruction, mucosal oedema of an inflamed colon, local ileus (sentinel loop) in pancreatitis or appendicitis. Calculi in the renal system and gallbladder may be seen. Pancreatic calcification may be seen in chronic pancreatitis.

Abdominal ultrasound
This is used to identify a localised abscess, free fluid in the abdomen, gallstone disease and dilated ducts, gallbladder empyema, pancreatic inflammation and oedema.

Specific investigations
- Calcium, cholesterol and triglycerides. These levels are important in the aetiology of pancreatitis (hypercalcaemia, hyperlipidaemia) or complications of pancreatitis (hypocalcaemia).
Mr Grove’s blood tests show a grossly elevated amylase at 2000 U/dL. His liver function and coagulation are normal. His calcium level is normal. Abdominal X-ray and chest X-rays are normal. There is no evidence of free air.

An abdominal ultrasound was booked to look for evidence of gallstones or pancreatic inflammation or cysts. There are no stones. There is fat standing around a poorly visualised pancreas.

What is the most likely diagnosis?
Acute pancreatitis is diagnosed from classic clinical features of severe constant abdominal pain, which may radiate to the back in half of patients, with nausea, vomiting and elevated pancreatic enzymes. Pancreatic enzymes are released that autodigest duct tissue, causing damage and possible necrosis. It is a multisystem disorder.

What are the causes of acute pancreatitis?
According to the British Society of Gastroenterology guidelines the aetiology of acute pancreatitis should be determined in at least 80% of cases (no more than 20% should be classified as idiopathic). Alcohol exposure and biliary tract disease cause most cases.

- Sickling test. This should be done for sickle cell disease.
- Short Synacthen test. This should be done to exclude Addison’s disease. A random cortisol is not sufficient to make the diagnosis.
- Urinary porphobilogens. Consider for acute intermittent porphyria.
- CT abdomen. This may be done to look for abdominal catastrophe, infarcted bowel or a perforated viscus. It may show evidence of pancreatic inflammation, oedema and necrosis or complications such as pseudocysts or fluid collections.
- Upper gastrointestinal endoscopy. It should be done if peptic ulcer disease is suspected.
- Small bowel enema or MRI enterocolysis ± colonoscopy. These should be done to detect small bowel and colonic Crohn's disease or lymphoma.
- Alcohol: this usually affects habitual drinkers although it may develop in those with a binging habit. Alcoholics are usually admitted with an acute exacerbation of chronic pancreatitis.
- Post-ERCP pancreatitis: prospective studies have shown the risk is at least 5%. The risk is increased with an inexperienced endoscopist, sphincter of Oddi dysfunction or manometry on the sphincter.
- Trauma: pancreatic injury occurs more often in penetrating injuries (e.g. from knives, bullets) than in blunt abdominal trauma (e.g. from steering wheels, bicycles).
- Drugs: these cause a mild pancreatitis. Examples include azathioprine, 6-mercaptopurine, sulfonamides, tetracycline, sodium valproate, methyl dopa, oestrogens, frusemide (furosemide), 5-aminosalicylic acid compounds, corticosteroids and octreotide.
- Infection: several infections can cause pancreatitis. Viral causes include mumps, EBV, coxsackie virus, echovirus, varicella-zoster and measles. Bacterial causes include Mycoplasma pneumoniae, Salmonella, Campylobacter and Mycobacterium tuberculosis.
- Autoimmune pancreatitis: IgG4 levels are measured and raised. Typical imaging and histological features and response to steroids help to confirm diagnosis.
- Hereditary: this is due to mutations in PRSSI, CFTR or SPINK-1.
- Hypercalcaemia: causes include hyperparathyroidism, excessive doses of vitamin D, familial hypocalciuric hypercalcaemia and total parenteral nutrition.
- Hypertriglyceridaemia: clinically significant pancreatitis occurs with a serum triglyceride level of over 20 mmol/L. It is associated with type I and type V hyperlipidaemia.
- Malignancy obstructing the pancreatic ductal system.
- Developmental abnormalities of the pancreas: pancreas divisum and annular pancreas.
- Vascular abnormalities: vasculitis can predispose to pancreatic ischaemia, especially in those with polyarteritis nodosa and systemic lupus erythematosus.
- Idiopathic: occult microthiasis is probably responsible for most cases of idiopathic acute pancreatitis.

Box 20.1 Causes of acute pancreatitis

- Gallstones: the most important cause in most developed countries is a small stone passing into the bile duct and becoming lodged at the sphincter of Oddi.

What specific investigations should be ordered?
Tests are ordered to determine the cause and severity of acute pancreatitis.
- Blood tests include pancreatic enzymes in plasma, liver function tests, fasting plasma lipids and fasting plasma calcium.
What are the treatment options for acute pancreatitis?

The management of acute pancreatitis is mainly supportive. The aim is to provide aggressive supportive care, to decrease inflammation, to limit infection or superinfection, and to identify and treat complications as appropriate.

- Oxygenation (to maintain saturations of over 95%).
- Aggressive prompt fluid resuscitation to treat haemodynamic instability and prevent potential systemic complications.
- Fluid balance and urine output should be measured. A central venous catheter should be inserted in high-risk individuals. Urinary catheterisation to monitor urine output may be necessary.
- He should remain nil by mouth initially, to allow inflammation to settle.
- Feeding should be introduced enterally as the patient's anorexia and pain resolves, starting on a low fat diet.
- Analgesics are administered for pain relief.
- Antibiotics are generally not indicated, although in the presence of fever and evidence of systemic sepsis which may suggest necrosis, broad-spectrum antibiotics are often given. There is no consensus in the type of antibiotic used or duration of use. Intravenous co-amoxiclav and metronidazole are often prescribed.
- High dependency care should be offered to all patients with severe acute pancreatitis. They should be managed in a high dependency unit with full monitoring and systems support. Within hours to days a number of complications may develop. These include shock, pulmonary failure, renal failure, gastrointestinal bleeding and multi-organ system failure. Infection of necrosis is the most serious local complication of acute pancreatitis and is associated with a high mortality rate of up to 40%.
- Therapeutic ERCP with sphincterotomy should be performed urgently if an ultrasound shows evidence of gallstones and if the cause of pancreatitis is believed to be biliary, in patients who have severe pancreatitis, and if there is cholangitis, jaundice or a dilated common bile duct.
- Cholecystectomy should be performed during the same hospital admission if gallstone pancreatitis is thought to be the cause. This is the ideal, although some units refer for urgent outpatient cholecystectomy.
- Surgical treatment is not needed for most patients with acute pancreatitis. Those with infected necrosis will require intervention to completely debride all cavities containing necrotic material. The choice of surgical

What are the severity markers of acute pancreatitis?

The Glasgow system is a simple prognostic system that uses the data collected during the first 48h following an admission for pancreatitis (Table 20.1). There is a minimum score of 0 and maximum score of 8. Severe pancreatitis is likely with a score equal or greater than 3.

Patients with persisting organ failure and new organ failure, and in those with persisting pain and signs of sepsis after a week of admission, will require evaluation by dynamic contrast-enhanced CT. CT evidence of necrosis correlates well with the risk of other local and systemic complications.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Finding at any time during initial 48h</th>
<th>Points</th>
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</thead>
<tbody>
<tr>
<td>Age</td>
<td>&gt;55 years</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≤55 years</td>
<td>0</td>
</tr>
<tr>
<td>Serum albumin</td>
<td>&lt;3.2 g/dL</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≥3.2 g/dL</td>
<td>0</td>
</tr>
<tr>
<td>P&lt;sub&gt;o&lt;/sub&gt;2 on room air</td>
<td>&lt;60 mmHg</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≥60 mmHg</td>
<td>0</td>
</tr>
<tr>
<td>Serum calcium</td>
<td>&lt;8 mg/dL</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≥8 mg/dL</td>
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<td>Blood glucose</td>
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<tr>
<td></td>
<td>≤180 mg/dL</td>
<td>0</td>
</tr>
<tr>
<td>Serum LDH</td>
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<td>1</td>
</tr>
<tr>
<td></td>
<td>≤600 U/L</td>
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</tr>
<tr>
<td>Serum urea nitrogen</td>
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<tr>
<td></td>
<td>≤45 mg/dL</td>
<td>0</td>
</tr>
<tr>
<td>WBC count</td>
<td>&gt;15 000 cells/µL</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>≤15 000 cells/µL</td>
<td>0</td>
</tr>
</tbody>
</table>
Mr Grove was admitted to hospital. He had a severity score of 1 in the first 48h. He received oxygen, intravenous fluids and antibiotics. His pain settled over the first 72h and his amylase gradually decreased. On day 5 he was discharged pain free.

He re-presented 4 weeks later with severe upper abdominal pain with an associated rise in amylase level.

What may have happened?
He recovered well from his acute pancreatitis and had a low severity score on the Glasgow criteria. He has presented again with features of acute pancreatitis or a possible complication, such as a pancreatic pseudocyst (Table 20.2).

What tests should be done for recurrent acute idiopathic pancreatitis?
- Repeat abdominal ultrasound to detect gallstones missed on the initial scan.
- Viral antibody titres for viral infections.
- IgG4 for autoimmune pancreatitis in those with no other risk factors.
- Autoantibodies for vasculitis.
- Pancreatic function tests to exclude chronic pancreatitis.
- Genetic analysis in those with a family history or in those that present at a young age.
- Consider an endoscopic ultrasound, ERCP with bile and pancreatic cytology, and sphincter of Oddi manometry in those with a possible biliary source.

What is chronic pancreatitis?
Chronic pancreatitis is the result of recurrent pancreatic inflammation. This can cause endocrine and exocrine deficiency. Abdominal pain may be severe and constant due to damage of sensory nerves and scarring and obstruction of the duct.

Malabsorption develops due to pancreatic enzyme deficiency and diabetes mellitus due to insulin insufficiency. Treatment includes oral pancreatic supplements, insulin if diabetic and analgesia.
He had a repeat abdominal ultrasound which showed multiple, small, fluid-filled pseudocysts (maximum diameter 4 cm) around the pancreatic tail. There were no gallstones or biliary dilatation. A CT abdomen confirmed the pseudocysts and demonstrated an oedematous pancreas (Fig. 20.1).

He had a full screen for recurrent pancreatitis with negative results. He eventually returned to the clinic for genetic screening and was found to be SPINK-1 positive. He was diagnosed with hereditary pancreatitis. There is no definite treatment for this. He has a 40% risk of developing pancreatic carcinoma by the age of 70 years.

**CASE REVIEW**

Acute abdominal pain has a wide list of differential causes. Often, the key to finding the cause is in a careful abdominal pain history and abdominal examination. This should include an examination of the hernia, testes and a digital rectal exam. A systemic search for causes of referred pain is also important. This can be complemented by blood tests and imaging.

This young man had severe central and upper abdominal pain with nausea, vomiting and altered bowel habit. Investigations confirmed an elevated amylase level consistent with acute pancreatitis. Gallstones and alcohol are the most common causes.

Patients with pancreatitis require assessment into the initial severity, evidence or organ failure and complications. Calculation of the Glasgow score on admission helps to predict prognosis. Treatment is predominately supportive and directed at reversal of an underlying cause if found. Recurrent episodes should encourage a repeat exclusion of the common causes and a screen for atypical causes in an effort to limit complications and preserve pancreatic function.

**KEY POINTS**

- Acute abdominal pain has many causes: gastrointestinal, hepatobiliary, urological, gynaecological, vascular, retroperitoneal, musculoskeletal, referred pain and medical causes.
- Symptoms and signs will guide further investigations.
- Acute pancreatitis is diagnosed by classic abdominal pain, nausea and vomiting and markedly elevated pancreatic enzymes.
- Where doubt exists, pancreatic imaging by contrast-enhanced CT scan provides good evidence for the presence or absence of pancreatitis. In the presence of persisting organ failure, signs of sepsis or deterioration in clinical status, a CT scan should also be done.
- Complications of pancreatitis can develop weeks after the initial infection.