Acute pancreatitis

Chronic pancreatitis

Pancreatic cancer

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This article discusses the diagnosis and management of the most common and important pancreatic disorders: acute pancreatitis, chronic pancreatitis and pancreatic cancer.

Acute pancreatitis

Pathology and aetiology

ACUTE pancreatitis is the sudden onset of inflammation of the pancreas that may involve, to varying degrees, the surrounding tissues or other organ systems. In Western societies about 80% of the acute pancreatitis cases are caused by gallstones (biliary pancreatitis) or excessive alcohol consumption. The other 20% are caused by rarer causes and idiopathic pancreatitis (see box, next page).

Gallstones

Gallstones are the most common cause of acute pancreatitis in our society. The mechanism is not fully understood. It is thought that gallstones travel down the common bile duct then into the channel that drains the common bile duct and the pancreatic duct, causing transient obstruction of the pancreatic duct. This obstruction leads to the activation of pancreatic pro-enzymes within the acinar cells by intracellular lysosomal enzymes, leading to ‘auto-digestion’ and thus acute pancreatitis. Smaller gallstones are more likely to cause pancreatitis than larger gallstones, as they are more likely to escape the gallbladder through the narrow cystic duct.

Alcohol

Alcohol is the second most common cause of acute pancreatitis and is the most common cause of chronic pancreatitis. The patients are usually young men who consume more than 80g of alcohol a day. However, the aetiology of alcoholic pancreatitis is thought to be multifactorial (genetic, environmental, etc), as there is not a clear dose-dependent relationship as there is for alcoholic liver disease.

The mechanism of how alcohol induces acute pancreatitis is still unclear. It is thought that alcohol increases the sensitivity of acinar cells to cholecystokinin hyperstimulation, continuing on next page.


**HOW TO TREAT**

**Pancreatic disorders**

**from previous page**

resulting in increased intra-
cellular protease activation. However, several other ther-
apiions may also be hypoth-

**Other causes**

Other causes of acute pan-
creatitis are shown in the
box below.

**Clinical presentation**

Patients usually present with
an acute onset of epigastric
pain that radiate to the back
and is relieved by lean-

**Presentation**

On examination, patients
may display varying degrees
of change in vital signs, and
there is tenderness in the
epigastric area. In severe
pancreatitis, Grey–Turner’s
(haemorrhagic discouloura-
tion of the flanks) or Cullen’s
(haemorrhagic discoloura-
tion of the umbili-
cus) signs may be observed.

Patients may sometimes
present with haodynamic
instability or even in a state
of profound shock in extreme
cases.

**Investigations**

**Blood tests**

These may include BFC,
EUC, LFTs, amylase, lipase,
calcium, BSL, CRP, lipid
profile and arterial blood
gases.

They should be drawn early
and/or hip level (higher than
three times normal) support the
diagnosis of acute pancreatitis in
most cases. However, one
should keep in mind that there are
other conditions that can cause mildly ele-

dated amylase and/or lipase
with epigastric pain, such as
peptic ulcer disease or per-
formation, cholecystitis,
appendicitis and ischaemic
bile. If there is doubt
regarding the diagnosis, an
abdominal CT scan should
be performed to rule out
other possible causes.

**Abdominal ultrasound**

All patients with acute pan-
creatitis should have an
abdominal ultrasound within
the first 24 hours to assess
the biliary tree, looking
for any evidence of choledoch-
olithiasis and/or dilatation of
the common bile duct. In the
acute setting, accurate assessment may be difficult
due to overlying bowel gas and abdominal tenderness.

Therefore, abdominal ultra-

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sound should be repeated
twice pancreatitis has

resolved if the initial one is
negative and no other
causes are identified.

Computed tomography
Apart from establishing a
diagnosis and excluding other
causes of abdominal pain, contrast-enhanced CT
may be used to detect com-

plications of pancreatitis
such as pancreatic necrosis
(figure 1). Although less
commonly used in Aus-

tralia, a CEUAP index
derived using the presence of
fluid collection and the
presence or extent of necro-
sis can be used to predict
the severity of pancreatitis.

CT should be performed
at least 48 hours after
the onset of the symptoms, as
early CT can be falsely neg-

ative. CT cholangiography
may also be used to detect
choledocholithiasis.

Other imaging modalities
These include:

- Magnetic resonance
cholangiopancreatogra-
phy (MRCP), as endo-

scopic retrograde cholan-
giopancreatography (ERCP) is now primarily

a therapeutic tool, MRCP
is increasingly being used
to assess choledocholithiasis
and pancreatic duct
anatomy.

- EUS (endoscopic ultra-

sound): this is an emerg-

ing imaging modality increasingly being used
to diagnose microhils, as well as for therapeutic
purposes, such as drainage of peri-pancreatic fluid
collections.

**Severity**

Several prognostic predic-
tors have been developed to
assess the severity of acute pancreatitis and help pre-
dicting patient outcomes.

**C-reactive protein**

CRP is an acute reactive pro-
tin, and patients with CRP
>150mg/L are classified as
having severe acute pan-
creatitis. However, serum
CRP levels do not usually peak
until about 48 hours after
the onset of symptoms, and
its levels usually lags behind
the clinical picture by that
time. Its levels can also be
used to monitor the clinical
course of the disease.

**Definitive management**

**ERCP.** The use of ERCP in acute pancreatitis has also
been much debated. Current best practice is that only
patients with acute biliary
pancreatitis (eg, due to gall-

stones) who also have acute
cholangitis (fear, upper right-

quadrant pain, jaundice, leu-

cocytosis, obstructive pattern
of LFTs) should undergo
urgent ERCP and sphinctero-

try.

Cholecystectomy. The gall-

bladder should be removed to
prevent further attacks of bil-
inary pancreatitis. For patients
with mild acute pancreatitis,
the gallbladder should ideally be
removed during the same
operation. However, if it is not
possible, it should be per-
formed preferably within
the first four weeks after dis-
charge.

**Management of complications**

Complications of acute pan-
creatitis include pancreatic
pseudocyst, necrosis or
obsc, and less commonly, haemorrhage, lateral portal
hypertension and pancreatic duct stricture.

Pancreatic pseudocyst. Peri-
pancreatic fluid collections are
detected on CT in 25% of
patients with acute pancreati-

tis. Most of these collections
require no intervention and
are spontaneously resorbed.
However, if conservative man-
agement fails, they may be
drained percutaneously or
scopically or surgically. The
route of drainage depends on
the contents of the pseudocyst
and whether they are infected.

Pancreatic necrosis. Necro-
sis of the pancreas and peri-
pancreatic tissue may occur
with severe acute pan-
creatitis, uveitis, lew
necrosis is sterile and the
patient is stable, the patient
should be managed conserv-
atively for as long as possi-
ble.

The rationale is that pan-
creatic necrotic tissue becomes
organised as it ‘matures’ and ultimately the
solid necrotic tissue liqui-
fies and becomes a pseudo-
cyst. In other cases, the
pus can be drained
with definitively as above.
This is much simpler
than necrotising.

However, if the patient
has organ failure despite
adequate medical manage-
ment, they may be
bled to be due to infected necro-
sis, necrosectomy may
be necessary. This may be car-
ried out via an open laparo-
tomy or using a minimally invasive (percutaneous or
diagnostic) approach.

**Management of causes of acute pancreatitis**

To avoid further attacks, the
causes of acute pancreatitis
should be treated, for exam-
ple:

- Removal of the gallblad-
ner (see above).
- Avoidance of alcohol.
- Rationalization of medica-
tions.
- Treatment of hyperlipid-
emia.
- Prevention of hyperlipid-
emia.

However, for patients with recurrent pancreatitis
without an identifiable
cause even after CT, MRC/ERCP and EUS, a laparosco-
necronectomy or biliary sphincter-
tomy may be considered.
Chronic pancreatitis

Background
CHRONIC pancreatitis is defined as a continuing chronic inflammatory condition of the pancreas, characterised by irreversible morphological changes of the pancreatic parenchyma, with replacement of glandular tissue by fibrosis. Its incidence is about 13 per 100,000 population in Western societies. The exact mechanism is still uncertain. However, the ‘monosymptomatology-fibrosis’ sequence is currently accepted hypothesis. It involves repeated injury to the pancreatic parenchyma by alcohol, causing in turn recurrent cycles of inflammatory and fibrotic responses. Interestingly, only a small proportion of the general population with alcoholism develop chronic pancreatitis, leading to the hypothesis that other factors contributing to the individual’s sensitivity to alcohol may be present.

Infrequent
About 20% of patients with chronic pancreatitis are thought to have idiopathic disease. However, some of the previously thought idiopathic cases have now been found to have a hereditary cause (see below).

Hereditary
Hereditary chronic pancreatitis is a rare autosomal dominant disease that accounts for about 1% of cases. Mutations in the PRSS1 and/or SPINK1 genes render the enzyme cationic trypsinogen resistant to inactivation, leading to autodigestion of the pancreas. Patients with hereditary chronic pancreatitis are found to have a 50-fold increase in the chance of developing pancreatic cancer.

Another hereditary cause is mutations in the CFTR gene that causes cystic fibrosis. A large number of the CFTR gene mutations have been identified and some of these genotypes result in less severe respiratory disease but are more likely to cause pancreatitis.

Autoimmune
Autoimmune pancreatitis is an uncommon but increasingly recognised form of chronic pancreatitis. It is characterised by enlargement of the pancreas and diffuse irregular narrowing of the pancreatic duct. It is associated with elevated serum IgG level and can also be present with other autoimmune diseases.

Others
Obstructive. Obstruction to the pancreatic duct may also cause pancreatitis. The causes may include pancreatic cancer, intraductal neoplasms, duct disruption from previous attacks, and congenital conditions such as pancreatic divisum.

Tropical. Tropical pancreatitis is rare in Australia but, as the name implies, it is prevalent in some countries near the equator. Nutritional factors such as dietary toxins and macromolecular deficiencies are thought to play a major role in the pathogenesis.

Miscellaneous. Other causes for chronic pancreatitis include hypercalcemia associated with hyperparathyroidism, hypoparathyroidism, and medications.

Clinical presentation
Pain
Pain is the most common and debilitating symptom for patients with chronic pancreatitis. The pain may range from episodic attacks that last for days to weeks with pain-free intervals, to severe and relentless pain. It is usually epigastric, radiates to the back, and may have associated nausea and vomiting. Pain may decrease over time as the pancreas ‘burns out’.

With the improvement in imaging techniques, pancreatic function tests are rarely performed now.

Imaging studies
Plain abdominal X-ray. Diffuse calcification detected in the pancreas on plain abdominal X-ray is considered diagnostic of chronic pancreatitis. However, when these calcifications are absent, abdominal X-ray is not a useful diagnostic test.

CT/MRI. Contrast-enhanced CT performed using a pancreatic protocol can reliably demonstrate features of chronic pancreatitis, which include pancreatic atrophy, calcification and pancreatic duct dilatation for making the diagnosis. However, these calcifications are not always present, and CT or MRI may be used instead of CT in patients with iodine contrast allergy.

ERCP/MRCP. ERCP is an accurate endoscopic test in the diagnosis of chronic pancreatitis (figure 3). However, due to its associated risk of iatrogenic acute pancreatitis, it has virtually been replaced by magnetic resonance cholangiopancreatography (MRCP) as a diagnostic tool, with ERCP now reserved for interventions only.

Endoscopic ultrasound (EUS) is increasingly being used in the diagnosis of chronic pancreatitis, as it can visualise the pancreatic parenchyma and ductal system simultaneously. It can make a reliable diagnosis of chronic pancreatitis, showing fibrosis, pseudocyst, splenic or portal vein thrombosis, pancreatic mass and gastric outlet obstruction. MRI can be used instead of CT in patients with iodine contrast allergy.

Management
The three main treatment goals are the management of pain, malnutrition and complications. Chronic pancreatitis is best managed in high-volume multidisciplinary centres.

Management of pain
Pain is the most debilitating factor in patients with chronic pancreatitis and has a great impact on the quality of life. Patients with alcohol as the aetiology should be advised to abstain from alcohol to reduce the risk of further attacks. NSAIDs and opioid analogues may be used for pain control. The use of pancreatic enzyme replacement may help with pain control but is controversial.

More invasive options may include CT or EUS-guided celiac plexus block and thoracoscopic splanchenecomy. Surgery is sometimes necessary (see below).

Management of malnutrition
Fat malabsorption is the hallmark of exocrine insufficiency of chronic pancreatitis. Steatorrhoea is characterised by excessive fat in faeces (greater than 15g/day). For treatment, oral pancreatic enzyme replacement therapy such as Creon is required (see below).

Acid suppression medications such as proton-pump inhibitors may be necessary for patients who continue to have pancreatic exocrine insufficiency despite pancreatic enzyme dose replacement. This is because the low pH in the stomach may breakdown and inactive the replacement enzymes prematurely. Rarely, removal of the pancreas by resection of the management of pancreatic exocrine insufficiency was published by the Australasian Pancreatic Club recently (see Online resources, page 36).

Diabetes secondary to chronic pancreatitis can be treated by oral hypoglycaemics. Insulin is usually required.

Management of complications/surgery
Complications of chronic pancreatitis may include bile duct strictures, pseudocysts, left-sided portal hypertension secondary to splenic vein thrombosis and pancreatic ascites. Unresolved obstructions may have to be bypassed surgically with a hepaticojejunostomy and gastroenterostomy. Bleeding from gastric varices that form as a consequence of left-sided portal hypertension secondary to splenic vein thrombosis should be treated with a distal pancreatectomy and splenectomy.

Pancreatic pseudocysts that do not settle down with conservative management can be drained into the duodenum or stomach either endoscopically or surgically.

Pain associated with a dilated pancreatic duct or a pancreatic mass can also be managed surgically. However, the degree of pain relief after surgical drainage procedures is variable.
Pancreatic cancer

Background
Pancreatic cancer remains a highly lethal disease despite advances in the treatment of several other cancer types. It is the 11th most common cancer in Australia, with an incidence of 10 per 100,000 population (2181 new cases in 2006).1

However, as the fourth most common cause of cancer-related death, it presents a significant burden on society. The overall five-year survival is poor at about 7%, with almost 90% dying within a year of diagnosis and a consequent death-incidence ratio (the ratio of the number of patients who have died to the number of patients diagnosed with the condition per year) approaching one. This mortality rate has changed very little in almost 50 years.

Surgical resection remains the only chance of cure, with chemotherapy and radiotherapy offering only a modest survival benefit. Unfortunately most patients (>90%) present in an advanced stage that is not amenable to surgery, due largely to the lack of early warning symptoms or any effective screening strategies. Therefore GPs play a crucial role in identifying at-risk populations and recognising subtle patient complaints that may be of pancreatic origin.

Pathogenesis and aetiology
Cancers of the pancreas can be broadly divided into those of endocrine and those of exocrine differentiation. Here we focus on cancer of the exocrine origin, which constitutes most pancreatic cancer (90%).

Pancreatic cancer, like many other cancers, is caused by complex interactions between environmental factors and genetic susceptibility.

Environmental factors
Cigarette smoking is a major risk factor for pancreatic cancer. Smokers have a significantly increased risk (odds ratio 1.77) compared with never smokers.2 However, this risk is normalised 15 years after smoking cessation. Other risk factors include increased BMI, occupational exposure to chemicals such as beta-naphthylamine and benzidine, and chronic pancreatitis.

The relationship between diabetes and pancreatic cancer is less well defined. The association between long-standing diabetes and pancreatic cancer is modest and diminishes with time. On the other hand, there is an association between new-onset diabetes and the development of pancreatic cancer. Therefore, new-onset diabetes may be a manifestation of pancreatic cancer, or it may be linked etiologically to pancreatic cancer, making patients newly diagnosed with diabetes at a risk group.

The value of axial imaging such as CT in every patient with new-onset diabetes to screen for pancreatic cancer is still unclear, although there is some evidence to suggest that such individuals, particularly men, should be included in populations targeted for screening.

Clinical presentation
Clinical presentation may include:
- Weight loss
- Jaundice
- Abdominal or back pain
- Dyspepsia
- Nausea
- Depression

It is important to have a level of clinical suspicion in patients presenting with any non-specific abdominal symptoms that persist.

As noted in the previous section, new onset of adult type 2 diabetes in younger patients may be linked to a diagnosis of pancreatic cancer. Hence the diagnosis of pancreatic cancer should be considered in patients with diabetes with unusual manifestations, such as abdominal symptoms and continuing weight loss.

Diagnosis and staging investigations
Patients should be evaluated initially by CT. The pancreatic cancer CT is the most widely available and best-validated imaging modality for diagnosing and staging patients with pancreatic cancer.

A pancreatic protocol involves triple-phase (arterial, late arterial and portal venous) imaging with thin slices (figure 4).

The late arterial phase provides the greatest difference in contrast enhancement between normal pancreatic parenchyma and pancreatic adenocarcinoma. The various phases also provide crucial information on the relationship between the tumour and surrounding arterial (portal, hepatic and superior mesenteric arteries) and venous (superior mesenteric, splenic and portal veins) structures.

Therefore, in addition to providing a diagnosis, CT also provides staging and information on the suitability for surgical resection if the tumour is localised. However, decisions concerning resectability and further management should only be made in the setting of a multidisciplinary treatment clinic.

Baseline blood tests include:
- FBC
- Electrolytes
- Renal function
- LFTs
- Amylase or lipase
- Coagulation profile
- CA19.9

The tumour marker CA19.9 is the only FDA-approved pancreatic cancer biomarker. However, there are several limitations with CA19.9. First, it is detectable at basal levels in healthy patients, so its value needs to be correlated to radiological findings.

Second, it can be elevated in benign disease such as pancreatic, obstructive jaundice caused by choledocholithiasis, and cirrhosis, and values are falsely elevated in patients with unusual lesions that need to be better characterised.

EUS may be used as an adjunct to CT/MRI for local staging and to assess the resectability if the results of non-invasive imaging modalities are inconclusive. It should also be used in patients with a high clinical suspicion of a lesion that has not been clearly demonstrated using other modalities.

The other advantage of EUS is the ability to perform fine-needle aspiration (FNA), biopsy (figure 6). EUS-FNA should be the biopsy technique of choice in patients in whom a tissue diagnosis or tissue from regional lymph nodes may alter the course of treatment, or if non-surgical treatment (eg, chemoradation before surgery) is contemplated.

The role of PET/CT in pancreatic cancer is still evolving. It is not currently covered under the MBS in Australia. However, PET/CT may potentially be used selectively, for example, when metastatic disease is suspected but has not been demonstrated with other imaging modalities, or if radiotherapy is being considered (to ensure there is no metastatic disease).

Diagnostic laparoscopy is a useful tool in excluding sub-radiological metastatic disease even after a high-quality staging protocol CT scan. The yield varies, so whether it should be performed selectively or routinely is still controversial.

ERCP and biliary stent placement is indicated for the management of obstructive jaundice and pruritus in patients with metastatic disease. Preoperative hilar drainage in jaundiced patients with ERCP may be technically challenging. However, preoperative ERCP and biliary stent placement should be considered in patients who are symptomatic or septic, or in those whom surgical resection is significantly delayed.

Management
Once staged, individuals are directed towards a stage-specific treatment plan (figure 7).

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Sustainable disease
For patients with resectable disease, surgical resection with adjuvant therapy is the only possibility for cure. For tumours of the head of pancreas (60-70% of resectable tumours), a puestagical protocol, for Whipple’s procedure (figure 8, page 36) involves en replacement of the pancreas, duodenum, gall bladder and common bile
Pancreatic disorders

HOW TO TREAT

The treatment of locally advanced, surgically resectable or metastatic disease

Locally advanced/resectable or metastatic disease

The treatment of locally advanced, surgically resectable disease is less uniform across centres. If the patient is considered suitable for surgery, a course of chemoradiation may be given to potentially ‘downstage’ the tumour. The patient will be reassessed for resectability after chemoradiation. Unfortunately, downsizing happens infrequently (<5%).

The treatment for patients with metastatic disease is either chemotherapy or best supportive care, depending on fitness and performance status. Gemcitabine is the current first-line chemotherapy and provides some clinical benefit but only a modest survival advantage. However, these non-randomised trials are controlled very closely, as their clinical status may change very rapidly. Appropriate directed measures should be paramount. Although gemcitabine has only a modest effect overall, there are responsive subgroups who receive significant benefit. Predicting who will benefit before treatment is a current major research focus internationally. The combination of gemcitabine and a new formulation of paclitaxel for metastatic pancreatic cancer is currently in early-stage clinical trials.

Prognosis and follow-up

There is no consensus on how patients should be followed up postoperatively. However, patients should be followed up regularly by their surgeon and GP and the nutritional status and any evidence of recurrence should be monitored.

We follow patients up with a CA19.9 assay and CT scan every 3–6 months in the first two years, then on each visit after that. The overall median survival in the recent ESPAC-3 adjuvant therapy trial was about 21 months. However, the median survival from a community cohort is shorter, as not every patient is suitable for adjuvant chemotherapy.

Recurrence should be monitored very closely, as their clinical status may change. We follow patients up with a CA19.9 assay and CT scan every 3–6 months after surgery. Although 15–20% of patients are suitable for surgery, these individuals have a 25% chance of cure and should be given that option.

References and Further reading

Available on request from julian.mcall.in@healthsouth.com.au

On-line resources


Australian Pancreatic Cancer Network. www.pancreaticcancer.net.au


Summary — Pancreatic cancer

• Pancreatic cancer is the fourth most common cause of cancer-related death in Western societies including Australia.

• Patients usually present at an advanced stage when surgery is not suitable, leading to a poor prognosis (overall five-year survival of <5%).

• Clinical presentation may include, but is not restricted to, weight loss, jaundice, abdominal or back pain, dyspnoea, nausea and vomiting.

• A CT scan using pancreatic protocol should be performed if pancreatic disease is suspected.

• All patients with a pancreatic lesion should be referred to a specialist pancreatic surgeon promptly and be treated in a multidisciplinary setting in a high-volume centre.

• Surgery remains the only chance of cure, with chemotherapy offering modest effect with an overall five-year survival of 20-25% and median survival of 20-25 months in patients who undergo surgery and adjuvant chemotherapy.

• Palliative therapy may include chemoembolisation and endoscopic stenting aiming to improve quality of life.

• Pancreatic cancer is a current focus of research internationally and the Australian Pancreatic Cancer Genome Initiative (APGI) is contributing to the International Cancer Genome Consortium (ICGC) in pancreatic cancer.

• Ensuring complete surgical resections of tumours: the long-term survival of patients depends on the complete macroscopic resection of tumour, as patients with incomplete resection survive less than one year, equivalent to that for patients with locally advanced disease.

• Detection and appropriate management of premalignant lesions in other organs, such as the colon, breast, and cervix, treatment of pre-malignant conditions has resulted in improved outcomes. There is now compelling evidence that pancreatectomy may also be curative for patients with non-invasive precursor lesions. There are three predominant types: pancreatic intraepithelial neoplasia, intraductal papillary mucinous neoplasms, and neoplastic cystic neoplasms.

• With increasing usage of high-resolution imaging techniques, these cystic precursor lesions are being increasingly identified both in symptomatic and asymptomatic individuals. Therefore, early detection and the standardisation of evidence-based treatment of these precursor lesions provide the best opportunity to prevent invasive adenocarcinoma of the pancreas.

The future: personalised cancer care

There is now compelling evidence that the genomic heterogeneity of cancer leads to different molecular phenotypes with disparate sensitivities to therapies in histologically indistinguishable cancers. Therefore, knowledge of the molecular phenotype of an individual’s cancer before treatment allows for the selection and early delivery of the optimal therapeutic regimen to improve overall outcomes and minimise morbidity and costs.

Such genotype-guided therapeutic approaches are already being used in the clinic, such as treating patients with HER-2-positive breast cancer with trastuzumab and patients with HER-2-positive GI stromal tumours with ima-tinib, but not in pancreatic cancer. The Individualised Molecular Pancreatic Cancer Therapy (IMPACT) trial is now underway. The proposed study is an exploratory, open-label, randomised phase II trial comparing standard chemotherapy for pancreatic cancer using genetic sequence data and gene expression profile to direct first-line treatment. The participating patients will be randomised to current standard of care (gemcitabine) or the experimental arm, which is gemcitabine plus a targeted treatment based on the genetic makeup of the tumour.

Conclusion

Pancreatic cancer remains one of the deadliest diseases in our community, with an overall five-year survival of <5%. This has not changed for almost 50 years. This is in part due to the late presentation and diagnosis of patients and a poor response to chemotherapy and radiotherapy.

Medical practitioners should consider pancreatic cancer as a potential diagnosis when elderly patients present with persisting sub-abdominal symptoms, especially if other risk factors are present. Although only 15–20% of patients are suitable for surgery, these individuals have a 25% chance of cure and should be given that option.
HOW TO TREAT
Pancreatic disorders

1. Which TWO statements regarding acute pancreatitis are correct?
   a) Obstruction of the pancreatic duct by gallstones or pancreatic enzymes, resulting in auto-digestion of the pancreas
   b) Larger gallstones are more likely to cause acute than smaller gallstones
   c) Acute pancreatitis typically occurs in young men who consume >80 alcohol a day
   d) There is a clear dose-dependent relationship between alcohol and acute pancreatitis

2. Which TWO drugs are associated with acute pancreatitis?
   a) Thiazide diuretics
   b) Captopril-potassium-antibiotics
   c) Steroids
   d) Calcium-channel blockers

3. Which TWO statements regarding acute pancreatitis are correct?
   a) Patients may sometimes present with haemodynamic instability or even in a state of profound shock
   b) An elevated serum amylase and lipase level is always diagnostic of acute pancreatitis
   c) If there is doubt regarding the diagnosis, an abdominal CT scan should be performed to rule out other possible causes
   d) Abdominal ultrasound is not useful in the first 24-hours after presentation

4. Which TWO statements regarding acute pancreatitis are correct?
   a) CT scan should be performed within 24 hours of the onset of the symptoms to detect complications such as pancreatic necrosis and fluid collections
   b) Serum CRP can be used to monitor the clinical course of acute pancreatitis
   c) Initial management includes the identification and reversal of organ failure, in particular renal and respiratory failure
   d) Pancreatic necrosis and peri-pancreatic fluid collections are usually managed surgically

5. Which TWO statements regarding chronic pancreatitis are correct?
   a) The morphological changes of the pancreatic parenchyma are usually reversible
   b) The most common presentation is abdominal pain and/or loss of exocrine or endocrine functions
   c) Patients with hereditary forms of chronic pancreatitis are at greatly increased risk of pancreatic cancer
   d) Pancreatic exocrine or endocrine insufficiency is an early complication

6. Which TWO statements regarding chronic pancreatitis are correct?
   a) The abdominal pain may decrease over time as the pancreas ‘burns out’
   b) The production of both insulin and glucagon may be reduced
   c) A normal amylase or lipase level excludes the diagnosis
   d) Faecal fat measurements are unhelpful for the diagnosis of exocrine insufficiency

7. Which TWO statements are correct?
   a) Pancreatic calcifications on abdominal X-ray are invariably present in a patient with chronic pancreatitis
   b) Contrast-enhanced CT scan is a highly sensitive and specific test for chronic pancreatitis
   c) Endoscopic retrograde cholangiopancreatography (ERCP) is the recommended imaging modality for the diagnosis of chronic pancreatitis
   d) Endoscopic ultrasound can visualise the pancreatic parenchyma and ductal system and allows a fine-needle aspiration biopsy if necessary

8. Which TWO statements regarding the management of chronic pancreatitis are correct?
   a) A coeliac plexus block and thoracic sympathetic ganglion block may be considered if analgesics fail to control pain
   b) Oral pancreatic enzyme replacement therapy is required with each meal for treatment of exocrine insufficiency
   c) Pancreatic exocrine insufficiency is a major risk factor for pancreatic cancer
   d) Persons with longstanding diabetes are regarded as being at risk of pancreatic cancer

9. Which TWO statements regarding pancreatic cancer are correct?
   a) Smoking, obesity, exposure to chemicals such as beta-naph- toquinone and benzidine, and perhaps diabetes, are said to be risk factors for pancreatic cancer
   b) Which occupations involve such chemicals? Should people with diabetes, especially those newly diagnosed, be having regular ultrasound or MRI to monitor the pancreas?

10. Which TWO statements regarding the treatment of pancreatic cancer are correct?
    a) CA 19.9 is recommended as a screening test
    b) ERCP to insert a biliary stent is indicated for the management of obstructive jaundice and pruritus in patients who cannot undergo surgery
    c) Percutaneous morbidity for curative surgery is about 30%
    d) All patients should be considered for postoperative (adjuvant) chemotherapy

INSTRUCTIONS
Complete this quiz online and fill in the GP examination form to earn 2 CPD or PPD points. We no longer accept quizes by post or fax.

The mark required to obtain points is 80%. Please note that some questions have more than one correct answer.

CPD QUIZ UPDATE
This week’s update is that a brief GP evaluation form is completed with every quiz to obtain 2 CPD or PPD points for the 2011-13 triennium. You can complete this online along with the quiz at www.australiandoc.com.au. Because this is required, we no longer accept the quiz by post or fax. However, we have included the quiz questions here for those who like to prepare the answers before completing the quiz online.

NEXT WEEK
The next How to Treat explores the most common malignancy in young male aged 20-40 – testicular cancer, which has had a threefold increase in incidence over the past 50 years, for reasons not completely understood. The authors are Dr Anthony Linton, medical oncologist Norwest, Concord Repatriation General Hospital, NSW, and Dr Peter Grimes, staff specialist in medical oncology, Royal Prince Alfred Hospital, and clinical senior lecturer, University of Sydney, NSW.