Chronic pancreatitis

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Chronic pancreatitis is a progressive inflammatory disease in which there is irreversible destruction of pancreatic tissue. Its clinical course is characterised by severe pain and, in the later stages, exocrine and endocrine pancreatic insu ffi ciency . In the early stages of its evolution, it is frequently complicated by attacks of acute pancreatitis, which are responsible for the recurrent pain that may be the only clinical symptom. The incidence of chronic pancreatitis in several European, North American and Japanese studies ranges from 2 to 10 new cases per 100 /uni00A0 000 population per year, with a prevalence of around 13 cases per 100 /uni00A0 000, although there are suspicions that the prevalence is actually higher. In certain parts of the world, such 100 /uni00A0 000). The disease occurs more frequently in men (male to-female ratio of 4:1) and the mean age of onset is about 40 years. Aetiology and pathology High alcohol consumption is the most frequent cause of chronic pancreatitis, accounting for 60–70% of cases, but only 5–10% of people with alcoholism develop chronic pancreatitis. The exact mechanism of how alcohol causes chronic inflammation in these patients is unclear; genetic and metabolic factors may be at play . Other causes include pancreatic duct obstruction resulting from stricture formation after trauma, after acute pancreatitis or even occlusion of the duct by pancrea tic cancer. Congenital abnormalities, such as pancreas divisum and annular pancreas, if associated with papillary stenosis, are rare causes of chronic pancreatitis. Hereditary pancreatitis, CF , infantile malnutrition and a large unexplained idiopathic group make up the remainder. Normally , if trypsinogen becomes prematurely activated within the pancreas , it is inhibited by SPINK1 and is destroyed. Hereditary pancreatitis is an autosomal dominant disorder with an 80% penetrance; it is associated with a gain-of function mutation in the cationic trypsinogen gene (PRSS1 chromosome 7, which leads to production of a degradation- resistant form of trypsin. A loss-of-function mutation in SPINK1 also predisposes to idiopathic pancreatitis. Some patients with idiopathic chronic pancreatitis have mutations in the CFTR gene. Idiopathic chronic pancreatitis accounts for approximately 30% of cases and has been subdivided into early-onset and late-onset forms. The importance of hereditary pancreatitis and pancreatitis occurring at a young age is that there is a markedly increased risk of developing pancreatic cancer, particularly if the patient smokes tobacco. Hyperlipidaemia and hypercalcaemia can lead to chronic pancreatitis. Tropical pancreatitis is a form of idiopathic pancreatitis that begins at a young age and is associated with a high inci dence of diabetes mellitus and stone formation. This has been described in resource-poor countries in Asia, Africa and cen tral America. Malnutrition, ingestion of cyanogenic glycosides in cassav a and exposure to hydrocarbons released by kerosene or para ffi n lamps have been pr oposed as possible mechanisms for tropical pancreatitis. Autoimmune pancreatitis has been described relatively recently . Features include di ff use enlargement of the pancreas and di ff use and irregular narrowing of the main pancreatic duct. It may occur in associa tion with other autoimmune dis eases, as a multisystem disorder, or may a ff ect the pancreas alone. There may be changes in the biliary tree (autoimmune c holangiopathy) as well. The changes may be confused with neoplasia. Autoantibodies may be present and levels of the immunoglobulin subtype

IgG4 are elevated. At the onset of the disease when symptoms have developed, the pancreas may appear normal. Later, the pancreas enlarges and becomes hard as a result of fibrosis. The ducts become distorted and dilated with areas of both stricture formation and ectasia. Calcified stones weighing from a few milligrams - occluded with a gelatinous proteinaceous fluid and debris and inflammatory cysts may form. Histologically , the lesions a ff ect the lobules, producing ductular metaplasia and atrophy of acini, hyperplasia of duct epithelium and interlobular fibrosis. Clinical features Pain is the outstanding symptom in the majority of patients. The site of pain depends to some extent on the main focus of the disease. If the disease is mainly in the head of the pancreas then epigastric and right subcostal pain is common, whereas if it is limited to the left side of the pancreas left subcostal and back pain are the presenting symptoms. In some patients, the pain is more di ff use. Radiation to the shoulder can occur. Nausea is common during attacks and vomiting may occur. The pain is often dull and gnawing. Severe flare-ups of pain may be superimposed on background discomfort. All the complications of acute pancreatitis can occur with chronic pancreatitis. Weight loss is common because the patient does not feel like eating. The pain prevents sleep and time o ff work is frequent. T he number of hospital admissions for acute exacerbations is a pointer towards the severity of the disease. Analgesic use and abuse are frequent. This, too, gives an indi - - cation of the severity of the disability . The patient’s lifestyle) on is gradually destroyed by pain, analgesic dependence, weight loss and inability to work. Loss of exocrine function leads to steatorrhoea in more than 30% of patients with chronic pan - creatitis. Loss of endocrine function and the development of diabetes are not uncommon, and the incidence increases as the disease progresses. Complications frequently bring the patient to the attention of the surgeon. Infection is not infrequent, possibly related to the diabetes mellitus. Investigations Only in the early stages of the disease will there be a rise in serum amylase. Tests of pancreatic function merely confirm the presence of pancreatic insu ffi ciency or that more than 70% of the gland has been destroyed. - Pancreatic calcifications may be seen on abdominal radio- graphs (Figure 72.18). CT or MRI scan will show the outline - of the gland, the main area of damage and the possibilities for surgical correction (Figure 72.29 ; see also Figure 72.7). Calcification is seen very well on CT but not on MRI. An MRCP will identify the presence of biliary obstruction and the state of the pancreatic duct (Figure 72.30). The use of intravenous secretin during the study may demonstrate a pancreatic duct stricture not apparent on standard MRCP , but a normal-looking pancreas on CT or MRI does not rule - out chronic pancreatitis. ERCP is the most accurate way of elucidating the anatomy of the duct and, in conjunction with the whole organ morphology , can help to determine the type of operation required, if operative intervention is indicated. Histologically proven chronic pancreatitis can, however, occur in the setting of normal findings on pancreatography . Sonographic findings characteristic of chronic pancreatitis include the presence of stones, visible side branches, cysts, lobularity , an irregular main pancreatic duct, hyperechoic foci and strands, dilatation of the main pancreatic duct and hyperechoic margins of the main pancreatic duct. The of chronic pancreatitis. Treatment Most patients can be managed with medical measures. There is no single therapeutic agent that has been shown to relieve symptoms (Summary box 72.9) . Endoscopic, radiological or surgical interventions are indi cated mainly to relieve obstruction of the pancreatic duct, bile duct or the duodenum, or in dealing with complications (e.g. pseudocyst, abscess, fistula, ascites or variceal haemorrhage). Decompressing an obstructed pancreatic duct can provide pain relief in some patients (the assumption is that ductal hypertension causes the pain). Endoscopic pancr eatic sphincterotomy might be bene ficial in patients with papillary stenosis and a high sphincter Charles Frederick Frey , b. 1929, Professor of Surgery , University of California, Davis, CA, USA. Medical treatment of chronic pancreatitis

/uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF - /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF /uni25CF pressure /uni00A0 and pancreatic ductal pressure. Patients with a dom - inant pancreatic duct stricture and upstream dilatation may benefit by placement of a stent across the stricture. The stent should be left in for no more than 4–6 weeks as it will block. The complication rate is high and less than two-thirds of patients experience pain r elief, but those who do get relief may benefit from a surgical bypass. Pancreatic duct stones may be extracted at ERCP; this may sometimes be combined with extracorpo - real shock wave lithotripsy . Pseudocysts may be drained inter - nally under endoscopic ultrasound guidance. Percutaneous or transgastric drainage of pseudocysts under ultrasound or CT guidance may be performed. The role of surgery is to overcome obstruction and remove mass lesions. Some patients ha ve a mass in the head of the pancreas, for which either a pancreatoduodenectomy or a Beger procedure (duodenum-preserving resection of the pan - creatic head) is appropriate. If the duct is markedly dilated, then a longitudinal pancreatojejunostomy or Frey procedure can be of value (Figure 72.31). The natural evolution of the disease may not be altered significantly , but around half the patients get long-term pain relief. The rare patient with disease limited to the tail will be cured by a distal pancreatectomy . Patients with intractable pain and di ff use disease may plead for a total pancreatectomy in the expectation that removing the

Figure 72.29 Computed tomography scan in a patient with chronic pancreatitis. A stone (arrow) is obstructing the main pancreatic duct in the body of the gland. The duct is markedly dilated upstream of the obstruction. Figure 72.30 Magnetic resonance cholangiopancreatography in a patient with chronic pancreatitis, showing a stricture of the pancreatic duct in the body of the gland (arrow), with dilatation upstream. Treat the addiction Help the patient to stop alcohol consumption and tobacco smoking Involve a dependency counsellor or a psychologist Alleviate abdominal pain Eliminate obstructive factors (duodenum, bile duct, pancreatic duct) Escalate analgesia in a stepwise fashion Refer to a pain management specialist For intractable pain, consider CT/EUS-guided coeliac axis block Nutritional and pharmacological measures Diet: low in fat and high in protein and carbohydrates Pancreatic enzyme supplementation with meals Correct malabsorption of the fat-soluble vitamins and vitamin B12 Micronutrient therapy with methionine, vitamins C and E, selenium (may reduce pain and slow disease progression) Steroids (only in autoimmune pancreatitis, for relief of symptoms) Medium-chain triglycerides in patients with severe fat malabsorption (they are directly absorbed by the small intestine without the need for digestion) Reducing gastric secretions may help Treat diabetes mellitus

keep in mind that pancreatic function and quality of life are significantly impaired after this procedure, and the operative mortality rate is not trivial. Moreover, there is no guarantee of pain relief (approximately a third of patients get resolution, a third show some benefit and a third see no benefit at all). Total pancreatectomy and islet autotransplantation have been reported in selected patients, but it is di ffi cult to demonstrate any overall benefit. Prognosis Chronic pancreatitis is a di ffi cult condition to manage. Patients often su ff er a gradual decline in their professional, social and personal lives. The pain may abate after a surgical or percuta neous intervention but tends to return over a period of time. In a proportion of patients, the inflammation may gradually burn out over a period of years , with disappearance of the pain, leaving only the exocrine and endocrine insu ffi ciencies. Development of pancreatic cancer is a risk in those who have had the disease for more than 20 years. New symptoms or a change in the pattern of symptoms should be investigated and malignancy excluded. Sphincter of Oddi dysfunction Sphincter of Oddi dyskinesia

or dysfunction (SOD) is a clinical syndrome in which pain, biochemical abnormalities and dilatation of the bile duct and/or pancreatic duct are attributed to abnormal function of the sphincter of Oddi. The true incidence of SOD is unknown. Females are more commonly a ff ected than males. SOD may result from stenosis of the sphincter or from dysmotility . Scarring or stenosis of the sphincter can result from passage of stones, pancreatitis or prior endoscopic sphincterotomies. is characterised by biliary pain, which may be accompanied by abnormally raised liver enzymes and/or dilatation of the bile duct and/or evidence of delayed emptying on biliary scintigraphy . It may be a cause of persistent postcholecystec - tomy symptoms. A predominance of pancreatic problems, especially recurrent episodes of acute pancreatitis, is known as pancreatic-type SOD. Each type of SOD is further divided into types I, II and III (Table 72.7). T his classification helps to predict the underlying pathology and the likelihood of success - ful treatment. Type I disease is thought to result from a fixed stenosis and responds best to therapy . An episodic dysmotility is the presumed underlying abnormality in the other types and - often does not respond as w ell to treatment. Biliary-type SOD should be considered and excluded /uni00A0 in patients with the postcholecystectomy syndrome. Pancreatic- type SOD should be excluded in patients with recurrent acute pancreatitis of une xplained aetiology . The role of SOD in chronic pancreatitis is unclear. A careful history is essential. CT and MRCP can demonstrate dilatation of the biliary and pancreatic ducts. MRCP with intravenous secretin injection can particularly demonstrate pancreatic duct dilatation due to raised sphincter pressures. EUS may achieve the same end. Quantitative cholescintigraphy (hepatobiliary iminodiacetic acid [HIDA] scan) may demonstrate delayed biliary transit. ERCP with manometry is indicated to confirm the diagnosis if the pain is disabling, non-invasive investigations have not shown structural abnormalities and conservative therapy has

Figure 72.31 Pancreatojejunostomy. The pancreatic duct is opened longitudinally and a loop of jejunum is sutured to the duct. In the Frey procedure, the super /f_i cial part of the head of the pancreas is removed to achieve drainage. TABLE 72.7 Milwaukee classi /f_i cation of sphincter of Oddi (SOD) dysfunction.

1. Biliary-type SOD Type I: Typical biliary-type pain Liver enzymes (AST, ALT or ALP) >2 times normal limit documented on at least two occasions during episodes of pain Dilated CBD >12 /uni00A0 mm in diameter a Prolonged biliary drainage time (>45 min) Type II: Biliary-type pain, and One or two of the above criteria Type III: Biliary-type pain only
2. Pancreatic-type SOD Type I: Pancreatic-type pain Amylase and/or lipase >2 times upper normal limit on at least two occasions during episodes of pain Dilated pancreatic duct (head >6 /uni00A0 mm, body >5 /uni00A0 mm) a Prolonged pancreatic drainage time (>9 min) Type II: Pancreatic-type pain, and one or two of the above criteria Type III: Pancreatic-type pain only AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP , alkaline phosphatase; CBD, common bile duct. a Dif /f_i cult to measure and often eschewed in clinical practice.

is the manometric criterion used to diagnose SOD. Endoscopic sphincterotomy is the treatment of choice for type I SOD. The question of whether dual sphincterotomies (biliary and pancreatic) should be carried out remains unan swered. There is howe ver a particularly high risk of post-ERCP pancreatitis (30% or more), though placement of a pancreatic stent at the time of the procedure appear s to reduce this risk. For patients with type II SOD, manometry should be done before considering sphincterotomy , and the results of sphincterotomy are less consistent. Patients with type III SOD are even more di ffi cult, with response rates to sphincterotomy ranging from 8% to

65%. Medical therapy should be tried before proceed ing to manometry . Proton pump inhibitors, spasmolytic drugs, calcium blockers (nifedipine) and psychotropic agents have all been tried with varying degrees of success. Injection of botu linum toxin (w hich can cause a chemical sphincterotomy for up to 3 months) or placement of a pancreatic stent (these are usually removed after 6 weeks) do not provide lasting relief but can be used to identify patients who may benefit from a sphincterotom y . In a small subgroup of patients who have experienced sig nificant but short-lived relief with sphincterotomy or stenting, surgical transduodenal sphincteroplasty may be considered but the long-term results are often poor. In exceptional cir cumstances, w here the pancreatic head is badly scarred after repeated stenting and numerous attacks of pancreatitis and sphincteroplasty has failed or is unlikely to succeed, one should consider surgical resection of the pancreatic head.

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