Pancreatic disease:
surgical aspects

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Chronic pancreatitis

Chronic pancreatitis, that is chronic progressive replacement fibrosis of the pancreas, is a rare disease in this country. I have collected fifty-five cases (thirty-six women and nineteen men) who illustrate some aspects of the disease, but I have had to rely on the literature for most of my information.

The literature on chronic pancreatitis has been very confused and one was never quite sure what disease an author was writing about. It was clarified by the Marseilles symposium in 1963 and I have used Sarles' article in Gut in 1965 extensively in preparing this paper (Sarles et al., 1965). Sarles and his colleagues make the point that chronic alcoholism is the main cause of chronic pancreatitis in France, U.S.A. and South Africa, but that this is not so in Britain, Czechoslovakia, Switzerland or Argentine. Without the alcoholics we are at a great disadvantage in this country when it comes to experience of chronic pancreatitis.

I will follow the classification of the Marseilles workers in their report on 205 cases of pancreatitis. They first of all draw a sharp line between patients with acute pancreatitis and relapsing acute pancreatitis (ninety cases), which with but a single exception did not evolve into chronic pancreatitis, and true chronic pancreatitis with or without calcification.

Their group of acute and relapsing pancreatitis corresponds with our experience in this country. It is with their group of chronic pancreatitis that we find ourselves with so little experience.

Chronic pancreatitis with calcification

In 8 years I have found only six cases of pancreatic calcification, and I am indebted to my colleague Mr Griffin for three of them. The first patient is a woman aged 47 in 1954 when she had a cholecystectomy for gall stones associated with a diffuse enlargement of the pancreas. I saw her in 1959 when she had a diabetic type GTT, normal secretin-pancreozymin test and no abdominal symptoms. In November 1966 she was perfectly well apart from bronchitis and emphysema.

The next patient is a man who was 60 in 1961. He had a long history of rheumatoid arthritis on steroids and presented with mild epigastric pain. Investigations were negative apart from diffuse pancreatic calcification. No treatment was given and in November 1966 he had no abdominal complaints.

The third patient is a man who presented at the age of 45 in 1961 with steatorrhoea without pain. In 1966 he was perfectly well, and is still taking pancreatic. The enormous pancreatic duct calculi are very unusual and have not changed in the last 5 years (see Fig. 1).
The fourth patient is a woman who was 67 in 1960 when she had a cholecystectomy for gall stones. Diffuse pancreatic calcification was seen on X-ray. She was well until 1965 when she developed a pseudopancreatic cyst in the lesser sac and died after cystogastrostomy.

The last slide illustrates the slight pancreatic calcification of a man of 52 whom I investigated last year with upper abdominal pain. All investigations are negative, he has settled down and I will follow his further progress. A year later he was symptom-free.

The sixth patient died 6 years ago of a carcinoma of colon and his X-rays have been destroyed. He had heavy pancreatic calcification without upper abdominal symptoms.

These few cases contrast with the wealth of material from Sarles’ clinic. They report 100 patients with pancreatic calcification. There were ninety-three men and seven women with an average age of 38. Only one patient did not drink, and only five drank less than 50 g of alcohol per day. In 1887 Friedreich (quoted by Howard & Ehrlich, 1961) wrote: ‘I am inclined to believe that general chronic interstitial pancreatitis may result from excessive alcoholism (drunkard’s pancreas’).

The clinical picture is of increasingly frequent recurrences of abdominal pain with progressive deterioration of general health, mental and physical degradation, narcotic addiction, loss of weight, diabetes, steatorrhoea and attacks of jaundice. One patient had cirrhosis of the liver and nine had portal hypertension. Splenography was performed in five and showed portal vein compression in three, thrombosis in one. Pancreatography by direct puncture showed duct dilatation in three-quarters of the cases, with stricturing of the main duct in the head of the pancreas, and in twenty-six patients single or multiple cysts either in or outside the pancreas, usually communicating with the main duct.

The histology of the pancreas was studied in one post mortem, seventeen partial pancreatectomy specimens and twenty-nine biopsies. The striking thing was the irregular distribution of areas of sclerosis, parenchymal atrophy and ductular dilatation and plugging, interspersed with areas of normal pancreas. Calcification is always intraductal and never interstitial.

The conservative surgical treatment of chronic alcoholic pancreatitis is disappointing. Subtotal or total pancreatectomy may be required. Howard and Ehrlich of Philadelphia reported on 127 cases of alcohol pancreatitis in 1961. Of sixty-five patients subjected to such operations as cholecystectomy, cholecystostomy, T-tube drainage, choledochojejunostomy, cholecystojejunostomy, sphincterotomy, caudal pancreateojejunostomy, and thoraco-lumbar sympathectomy, fifty-four were rated as failures.

Du Val & Enquist (1961) followed twenty-eight patients with alcoholic pancreatitis treated by caudal pancreateo-jejunostomy for 8 years and found that only nine were still alive.

**Chronic pancreatitis without calcification** (secondary to obstruction in the duct of Wirsung)

This group according to Sarles has a similar clinical picture to the first group (alcoholic pancreatitis with calcification) and histologically is characterized by a similar picture of parenchymal atrophy and replacement fibrosis. In Sarles’ eight cases the causes of the Wirsung obstruction were:

In three cases, stenosis at the ampulla of Vater.

In three cases, slow-growing carcinoma of the pancreas.

In one case, isthmic sclerosis following an attack of acute pancreatitis.

In one case, accidental ligation of the duct at gastrectomy.

Birnstingl in 1959 found twenty-two patients with a diagnosis of chronic pancreatitis in 16 years at St Bartholomew’s Hospital. In twelve of these the pathological picture of chronic pancreatitis was produced by a carcinoma of the pancreas obstructing the pancreatic duct.

In my series I have three patients with chronic pancreatitis due to carcinoma.

The first patient was a middle-aged man who presented with a pseudo-pancreatic cyst which was drained externally. Abdominal pain and a pancreatic fistula persisted and some months later a second laparotomy showed a diffusely enlarged hard nodular pancreas with an enormously dilated pancreatic duct. Anastomosis of the side of this duct to a Roux loop of jejunum was followed by relief of pain and healing of the fistula, but within a few months the patient had developed obstructive jaundice and metastases in the liver and died.

The second patient was an elderly lady who had had her gall bladder removed many years before. She presented with recurrent severe upper abdominal pain without jaundice and biligrain showed a somewhat dilated common bile duct. At operation I opened her common duct and extracted several stones, encountering a stricture in the intrapancreatic part of the duct corresponding to a firm nodular lesion in the head of the pancreas at this point. Pre-operative cholangiography confirmed this stricture. I opened the
duodenum and did a choledocho-duodenostomy which however lay below the stricture. Post-operatively she did very well, and was perfectly comfortable after her T-tube had been clamped off. A post-operative cholangiogram still showed the stricture and it was my intention to repeat the cholangiogram later. Unfortunately the T-tube fell out and the wound immediately healed. Two months later I found a large fixed mass in the epigastrium, strongly suggestive of carcinoma.

The third patient was a woman of 62 who presented as an emergency with abdominal pain. At operation I found a stone in the gall bladder and a somewhat dilated common bile duct. There were no stones in the bile duct but the pancreas was diffusely enlarged, hard and nodular and the tail of the pancreas was adherent to the back of the stomach. I was doubtful whether this was benign or malignant but decided it was probably a chronic pancreatitis, removed the gall bladder and did a transduodenal sphincteroplasty. Biopsy of the tail of the pancreas showed pancreatic fibrosis infiltrated by a well-differentiated adenocarcinoma.

Chronic pancreatitis (other than the above)

In seven patients with a clinical picture of progressive disease, pain, loss of weight, steatorrhoea and diabetes, Sarles was unable to show calcification or pancreatic duct obstruction. These form a miscellaneous group. They may proceed to common bile-duct obstruction and in this group must be placed the occasional patient whose clinical and operative picture is that of carcinoma of the head of the pancreas with obstructive jaundice but who unexpectedly survives palliative biliary surgery for many years. I know of a middle-aged man who was operated on for obstructive jaundice in 1954. There was an apparently malignant obstruction of the common bile duct in the head of the pancreas and a choledocho-jejunostomy was done. The patient is alive 13 years later.

Acute pancreatitis (and acute recurring pancreatitis)

Sarles has rendered a great service to surgery by separating this group, which is familiar to all of us, from the other groups of chronic pancreatitis. This is the ordinary disease, commoner in middle-aged women with gallstones, in which there is not a strong tendency to recurrence (11% proved recurrences in the Leeds series) (Pollock, 1959). In this country at least it is much commoner than chronic pancreatitis.

Treatment of chronic pancreatitis and acute relapsing pancreatitis

There is an atmosphere of therapeutic nihilism about the treatment of chronic pancreatitis, and this is likely to increase as more surgeons follow the Marseilles classification. It is probable that none of the ordinary operations on the gall bladder, common bile duct, sphincter of Oddi or pancreatic duct will do good to more than a very few patients, and therapy will probably need directing towards the restoration of exocrine and endocrine pancreatic secretions, the avoidance of alcohol, and the symptomatic relief of pain. When complications such as pancreatic duct obstruction, common bile-duct obstruction, or pseudopancreatic cyst occur they may be relieved by operation, and when pain is intractable subtotal or total pancreatectomy may be the only answer. I have only done one total pancreatectomy for chronic pancreatitis with intractable pain. This was a lady of 63 who had had a gastro-jejunostomy for duodenal ulcer 4 years previously. The following year she had recurrent attacks of abdominal pain and laparotomy showed chronic pancreatitis (biopsy). Her gall bladder was removed. Two years later the pain was continuous and she attempted suicide. Splanchnic blocks made little difference to the pain and finally I did a total pancreatectomy from which the patient died 3 weeks later.

On the other hand the treatment of acute pancreatitis is non-surgical, and the treatment of relapsing acute pancreatitis is fairly straightforward. I should like to consider some of the operations which are useful for the relapses of acute pancreatitis and the complications of chronic pancreatitis:

Cholecystectomy

If there are stones in the gall bladder I believe it ought to be removed.

Removal of common bile-duct stones

About the necessity for this there is no doubt, and their removal may necessitate transduodenal division of the sphincter of Oddi.

Sphincterotomy and its modifications

Apart from the removal of stones in the lower common duct, this operation has been favoured because of the theoretical appeal of separating the bile discharge from the pancreatic fluid discharge into the duodenum. There are three complications of this operation, which I have performed twenty-five times.

1. Acute pancreatitis

This occurred no fewer than nine times with
two deaths. In ten cases I did a peroperative pancreatogram at the same time, and five of these got pancreatitis with one death. Believing the pancreatography to be responsible for the complication I gave it up but had four more cases of acute pancreatitis in the next fourteen sphincterotomies with another death. I now realize that any exploration of the pancreatic duct may be dangerous, and the probes and instruments should be confined to the common bile duct.

2. Duodenal leak
This happened three times with two deaths. It may occur through the duodenotomy incision, or by extension of the sphincterotomy incision through the posterior duodenal wall.

3. Post-sphincterotomy stricture (Webb et al., 1964)
Experimentally it is found that the sphincterotomy incision heals with fibrosis which reproduces the sphincteric stricture and returns the common duct and pancreatic pressures to normal within 3 months. One of my patients died 2 years after sphincterotomy due to cholangitis and biliary cirrhosis, with restricting of the sphincter and dilated hepatic ducts.

For this reason many authors have favoured an extended sphincterotomy, through all layers of the duodenal wall and with a formal mucosa-to-mucosa repair. This is known as a sphincteroplasty or a trans-duodenal choledocho-duodenostomy. I have used this operation nine times with three deaths (one from pancreatitis, one from coronary thrombosis and one from bronchopneumonia).

Other methods of diversion of the bile
Cholecyst-enterostomy has not been successful in my experience in preventing recurrent acute pancreatitis, but prolonged T-tube common duct drainage I have used twice with satisfactory results, and formal choledocho-duodenostomy or -jejunostomy is recommended when there is obstruction to the intra-pancreatic common bile duct. Rodney Smith recently published (1965) his method of transluminal T-tube drainage which is a neat way of forming a choledocho-enteric or pancreatico-enteric fistula. In this operation a T-tube is placed in the duct and led to the exterior through either the stomach or a Roux loop of jejunum. I have used this operation once for the relief of obstructive jaundice due to a carcinoma of the head of the pancreas with a satisfactory result.

Methods of draining an obstructed pancreatic duct
There are five ways of doing this:
(a) A sphincteroplasty will give a satisfactory result if the obstruction is at the papilla of Vater.
(b) A Roux loop of jejunum may be brought up and anastomosed to the side of the pancreatic duct.
(c) Puestow's method of unroofing the entire pancreatic duct and anastomosing it to a defunctioned loop of jejunum seems a heroic endeavour and has not found general favour.
(d) Distal pancreatectomy with caudal pancreatico-jejunostomy is probably the best operation when it can be used.
(e) Rodney Smith's transluminal T-tube drainage, forming a pancreatico-gastric fistula.

Treatment of pseudo-pancreatic cysts
I have records of sixteen cysts. Of these all were in the lesser sac but three patients had more than one cyst, the second cyst being in the right iliac fossa in two and the left iliac fossa in one. Nine of these patients were treated by transgastric cysto-gastrostomy with two deaths, and three by external drainage with one death. The cysto-gastrostomy fistula usually closes within a week and the results are good, but in one patient the fistula persisted for more than a month and the patient developed osteomyelitis of the lower ribs.

The remaining four pseudo-pancreatic cysts resolved or discharged internally spontaneously, in one patient with the production of a duodenocolic fistula.

Subtotal and total pancreatectomy
I am sure there is a place for these operations in the management of intractable pain. In the only patient whose chronic pancreatitis I treated this way, it was technically fairly easy to separate the pancreas from the duodenum, and I did this and left the duodenum in place. Unfortunately it sloughed and the patient died. The operation must clearly be a formal pancreatico-duodenectomy.

Conclusion
Chronic pancreatitis must be separated from relapsing acute pancreatitis. It is a rare disease in this country, but in other countries is associated with alcohol and calcification and is very difficult to cure.
References


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