ACUTE PANCREATITIS

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Acute pancreatitis is an uncommon abdominal emergency. It is known by a variety of descriptive terms which are dependent upon the gross pathological features revealed at operation—"acute haemorrhagic pancreatitis," "acute pancreatic necrosis," "acute suppurative pancreatitis," "acute glassy oedema of the pancreas"—but all these conditions represent essentially the same disease.

Aetiology

Acute pancreatitis is almost always associated with chronic disease of the biliary tract: usually calculous cholecystitis, with or without stones in the common bile duct, but sometimes a chronically inflamed gall bladder without calculus formation. In view of this association with gall bladder disease it would be expected that pancreatitis would be commoner in women than men: actually it occurs with at least equal frequency in men, and some statistics show a greater incidence in the male sex. Apart from its occurrence as a complication of mumps, and occasionally of typhoid fever, it is a disease of later life, commonest in the fifth and sixth decades.

The primary affection of the pancreas is probably an auto-digestion due to activation of the pancreatic enzymes trypsinogen, diastase and lipase. The factors responsible for this enzyme activation may be:—

- (a) Regurgitation of normal bile along the pancreatic duct.
- (b) Regurgitation of *infected* bile along the duct.
- (c) Infection of the pancreas alone, without any element of bile regurgitation.

Regurgitation of bile along the pancreatic duct may occur with a stone impacted in the ampulla of Vater, but this is a rare operative finding: it is more likely to be due to spasm of the sphincter of Oddi. Experimental evidence shows that a distended gall bladder is the most potent factor giving rise to sphincter spasm. In either case it would seem essential that the anatomical feature of a common opening of the bile and pancreatic ducts into the ampulla must be present if regurgitation along the pancreatic duct is to occur, but cases of pancreatitis have been described in which this anatomical arrangement is absent. From physiological considerations there would seem to be no reason why regurgitation of normal bile should not activate the pancreatic ferments, but it is commonly held that infected bile is more likely to do so.

Infection of the pancreas alone, without bile regurgitation, is unlikely to be responsible for the development of the *fulminating* cases of acute pancreatitis. Thickening due to *chronic* inflammation of the head of the pancreas is not infrequently found in association with chronic cholecystitis, and lymphatic connections between the gall bladder and the pancreas are known to be present. The dramatic and sudden onset of the fulminating cases, however, would appear to exclude a primary lymphogenic infection and favour biliary regurgitation as the aetiological factor. That infection alone *can* cause pancreatitis is shown by its occurrence as a complication of mumps, but in such cases the disease is usually subacute in type and of a more gradual onset.

Pathology

Activation of the pancreatic enzymes, however it occurs, leads to auto-digestion of pancreatic tissue. The pancreas becomes swollen and oedematous ("acute glassy oedema") behind the posterior parietal peritoneum, and there is an associated peritoneal effusion into the lesser sac, this fluid ultimately escaping through the foramen of Winslow into the general peritoneal cavity. The liberated lipase causes saponification of fatty tissues, first in the root of the transverse mesocolon, later in the omentum and the entire peritoneal cavity: this is the process known as "fat necrosis," and is characterised by the appearance of white or yellowish-white calcified nodules in the affected tissues.

In mild cases the pathological changes may progress no further and resolution may occur: occasionally, if the foramen of Winslow becomes occluded by adhesions, the end result is the slow formation of a collection of fluid in the lesser sac, a "pseudo-pancreatic cyst." Commonly,

however, secondary changes occur in the pancreas, leading to a progressive deterioration in the patient's condition, and frequently, in spite of operation, to a fatal issue. Involvement of the pancreatic vessels in the digestive process is accompanied by haemorrhage ("acute haemorrhagic pancreatitis"), and in these cases there may be massive retroperitoneal haemorrhage with the peritoneal fluid heavily blood-stained. Often sloughing of the pancreas occurs ("acute pancreatic necrosis"), and this is inevitably followed by infection and suppuration ("acute suppurative pancreatitis"): local abscess formation or general peritonitis will then ensue.

CLINICAL FEATURES

Acute pancreatitis is a recognised complication of mumps, and as such may occur in children or young adults. Apart from mumps, however, the disease is rarely seen below the age of forty, and is commonest over fifty: it affects men as frequently as women. Two clinical varieties are encountered, fulminating and subacute: in both cases a previous history suggesting gall bladder disease is commonly obtained—flatulent dyspepsia, attacks of biliary colic with or without jaundice, or previous attacks of acute cholecystitis.

Fulminating cases.

These cases are characterised by the *sudden* onset of *severe* upper abdominal pain, often associated with collapse and sometimes with temporary loss of consciousness. The pain of acute pancreatitis is accepted as probably the most severe of all abdominal pains. It is felt first in the epigastrium, later becoming generalised. Pain in the back, due to stretching of the posterior parietal peritoneum, is also a common feature. The patient may find that to sit forward affords slight relief of the pain, but frequently no position of ease can be found, and the victim lies groaning and restless in bed. The pain is constant, not colicky, but appears to vary in intensity from time to time. Persistent vomiting and retching are regularly present.

From the onset the patient appears desperately ill. There is extreme pallor, sometimes sweating, associated with an appearance of anxious apprehension and agonising pain. The term "shock" is often used to describe this state, but in the early stages the pulse-rate and blood pressure are usually normal. During observation the pulse-rate shows a progressive rise, with a falling tension, and this occurrence forms an indication for exploratory laparotomy in doubtful cases. In the early stages the temperature is normal and the tongue clean, but as infection becomes established the temperature rises.

Examination of the abdomen shows respiratory excursion to be poor, and there is fullness in the epigastrium. Palpation reveals generalised abdominal tenderness, maximal in the upper quadrants, associated with muscular guarding and some degree of involuntary spasm, but board-like muscular rigidity is seldom present. Tenderness may also be present posteriorly in the flanks, particularly on the left side. Rectal examination may show tenderness in the pouch of Douglas. After some hours free fluid may be demonstrable in the peritoneal cavity, but there is no diminution of liver dullness. Diminished peristaltic sounds are noted on auscultation.

The urine sometimes contains sugar, but this is by no means constant: if laboratory facilities are available the urinary diastase is invariably shown to be raised. Dilatation of the pupil after injection of adrenaline into the conjunctival sac (Loewi's test) is unreliable as a diagnostic criterion.

Textbooks describe a peculiar cyanosis, either generalised or localised to the skin of the abdomen, as a feature of acute pancreatitis, but this unexplained phenomenon has not been observed in the writer's cases. In late cases, bruising in the loins has been described. Mild jaundice may also occur.

In untreated cases the general condition of the patient usually deteriorates rapidly, and signs of general peritonitis become evident within 18 or 24 hours. Sometimes the infection becomes localised, and the clinical picture becomes that of an intra-peritoneal abscess, with localised abdominal pain, tenderness and rigidity, and pyrexia of a swinging type.

Subacute cases.

Some cases of pancreatitis are of a milder nature and more gradual in onset. The pain is still quite severe, and vomiting and retching are present, but the patient is not collapsed or desperately ill. Such cases, if treated conservatively, usually get well. There is no rise in pulse-rate or deterioration of general condition during the period of observation, and the pain

and vomiting begin to subside after two to four days. At about this time it is possible to feel a vague mass lying transversely across the epigastrium: this lump is formed by oedematous omentum (showing fat necrosis) and is similar to the rolled mass of omentum sometimes palpable in tuberculous peritonitis. Resolution of this lump usually occurs in a few days, associated with further improvement in the patient's condition, and at this time sugar (if present) disappears from the urine and the diastatic index returns to normal. Occasionally, even in these subacute cases, infection occurs, and a localised abscess is formed which will ultimately require drainage. Rarely, increasing swelling of the abdomen takes place weeks or months after the original attack of pain: this is due to the formation of a large cystic swelling, a pseudo-pancreatic cyst, or collection of fluid in the lesser sac of peritoneum.

DIFFERENTIAL DIAGNOSIS

In the fulminating forms of acute pancreatitis it is seldom possible to establish a confident clinical diagnosis: the true state of affairs is usually discovered at operation performed on a provisional diagnosis of perforated peptic ulcer. Other conditions, as outlined below, may also simulate acute pancreatitis. With the less severe (subacute) forms of the disease clinical diagnosis is again difficult in the early stages: in this type of case, however, the indications for immediate laparotomy are usually absent and accessory investigations may then allow the diagnosis to be made with greater accuracy.

Fulminating forms.

The conditions which may closely simulate the severe forms of pancreatitis are:—

- Perforated peptic ulcer
- Biliary colic
- Acute intestinal obstruction
- Coronary thrombosis
- Dissecting aneurysm of the abdominal aorta.

Perforation of a peptic ulcer is the condition most likely to be simulated by acute pancreatitis: difficulty is particularly likely to arise in patients who have been ill for many hours, this being the stage at which the board-like rigidity characteristic of an early perforation tends to pass off and the condition of the patient rapidly to worsen. In those cases of suspected perforation proven at laparotomy to be pancreatitis it is usual to find, on retrospect consideration, that some features atypical of perforation were present before operation.

Both pancreatitis and perforation show sudden onset of severe epigastric pain, but whereas a patient with a perforated ulcer seldom vomits more than once, persistent vomiting and retching are common in pancreatitis. The patient's reaction to the pain tends to differ somewhat in the two cases: the peptic ulcer patient lies perfectly still, wears an anxious expression, and shows a grunting type of respiration; in pancreatitis the patient is more restless, groaning in agony, and the intensity of his pain varies from time to time. Shoulder-tip pain is not so constant in pancreatitis as in perforation, whilst in pancreatitis pain is often referred to the loins.

The previous history may be helpful in differentiating the two conditions. In pancreatitis it is common to find preceding gall-bladder symptoms: when perforation occurs the symptoms of ulcer-dyspepsia usually precede the acute catastrophe, although this is not always the case.

The board-like rigidity with scaphoid retraction of the upper abdomen, typical of an early perforation, is absent in pancreatitis: there is tenderness and guarding, but true rigidity is usually slight, and the upper abdomen appears full rather than retracted. After twelve hours or more the rigidity associated with perforation tends to pass off, and, with the onset of peritonitis, distension of the abdomen occurs: at this stage it is impossible to differentiate the two conditions. It is at this stage also, in both diseases, that elevation of the temperature and increased pulse-rate occur.

As previously stated, the cyanosis described in association with pancreatitis is by no means always present, and Loewi's test is unreliable. Sugar is infrequently present in the urine in pancreatitis, and estimation of the urinary diastase may not be practicable in an emergency. If the diagnosis of perforation cannot be positively excluded, laparotomy must be performed on these grounds alone.

An attack of **biliary colic** may also closely resemble acute pancreatitis. In both cases a previous history of gall-bladder disease is likely to be obtained, and the sudden acute upper abdominal pain may be felt also in the inter-scapular region. In biliary colic the patient has a constant severe pain with definite colicky spasms which cause her to double up or roll about in agony, clutching the abdomen: this type of reaction is not usual in pancreatitis.

Abdominal rigidity is absent in an attack of biliary colic, but there is guarding and tenderness in the right upper quadrant: in pancreatitis these signs are not so localised and occur across the whole epigastrium and even in the flanks. Pyrexia is absent in biliary colic, and there is no progressive rise in the pulse-rate during the period of observation. After some hours, if a stone be present in the common bile duct, jaundice may appear, with bile pigments in the urine.

Until a diagnosis is definitely established it is unsafe to prescribe morphia and atropine, but inhalation of a capsule of amyl nitrite may be tried: this drug is said to relieve an attack of biliary colic, but in the writer's experience this effect cannot be guaranteed.

Acute intestinal obstruction may be suggested by the onset of *severe* abdominal pain associated with persistent vomiting. Search for a cause of the obstruction will show no abnormality at any of the hernial orifices and no abdominal tumour; if there has been a previous abdominal operation adhesions may be suspected, or failing this an internal hernia or volvulus; if a previous history of gall-bladder disease is present gallstone ileus will be considered.

The pain of pancreatitis, however, is not truly intermittent and spasmodic as in intestinal colic, and in the early stages the patient is much more ill than would be the case with intestinal obstruction. In the later stages, although vomiting persists, it does not become faeculent as in intestinal obstruction. In pancreatitis, although there may be some abdominal distension, this feature is not so marked as in bowel obstruction, and auscultation of the abdomen shows diminished peristaltic activity instead of the increased sounds of a mechanical obstruction. Persistent constipation is not a feature of pancreatitis, and if the two-enema diagnostic test is applied results will be obtained from the enemata and flatus passed.

In some cases of **coronary thrombosis** the pain is felt principally in the upper abdomen. It is usual, however, for pain to occur in the chest as well as the abdomen, and almost invariably the attack of acute pain has been precipitated by exercise. This feature is most useful in distinguishing pain of cardiac origin from that due to primary abdominal disease.

On examination the patient is ill and in obvious distress. Dyspnoea and cyanosis may be present, and cyanosis may lead to confusion in diagnosis if one considers this to be a regular feature of pancreatitis. In coronary thrombosis there is no abdominal distension, but tenderness and guarding may be present in the upper quadrants: vomiting may occur once or twice, but it is not persistent as in pancreatitis. The cardiac signs may not be very striking: occasionally the heart sounds may be muffled or distant in character, but no murmurs are present, and the blood pressure may be low. After some days a pericardial rub may develop and a moderate leucocytosis occur.

A rare condition which may simulate acute pancreatitis is a **dissecting aneurysm of the abdominal aorta**; rupture of this aneurysm may occur, giving rise to massive retroperitoneal haemorrhage with death of the patient within a few hours. It is of course almost impossible more than to suspect the existence of such a condition, and the diagnosis is only made in the post-mortem room. The patient is extremely ill and pale, complaining of severe abdominal pain, and vomiting may occur; the abdomen is diffusely tender. Usually it is possible to determine that the condition does not demand operation, and the patient gets rapidly worse and dies.

Subacute forms.

The subacute forms of pancreatitis do not show the dramatic onset, with collapse, which occurs in the fulminating cases: although commonly sudden, the onset of symptoms may be gradual, and the patient is not as a rule seriously ill. In these circumstances the clinical diagnosis will frequently be in doubt at first, and the conditions with which it may be confused are:—

- Subacute perforation ("leaking" peptic ulcer)
- Penetrating gastric ulcer involving the pancreas
- Acute cholecystitis
- Biliary colic
- Intestinal obstruction.

In **subacute perforation of a peptic ulcer** leakage of gastric or duodenal contents occurs through a small opening which becomes rapidly sealed by a fibrin plug or by omentum: this phenomenon is commoner with duodenal than with gastric ulcers. A previous history of ulcer-dyspepsia is usually obtained, together with a recent sudden onset of a more severe type of pain; associated shoulder-tip pain is of frequent occurrence. The vomiting of pancreatitis is not present. On examination there is tenderness, guarding and rigidity in one or both upper quadrants of the abdomen: although this rigidity is not diffuse and board-like as in an acute perforation it is more marked than that which occurs with pancreatitis. Liver dullness is sometimes diminished, and in doubtful cases a straight radiograph of the abdomen will often show a thin layer of gas below the diaphragm (pneumoperitoneum), which confirms the diagnosis.

A **penetrating gastric ulcer**, eroding the pancreas, gives rise to a severe type of upper abdominal pain, which is referred to the back in the inter-scapular region. Again, unlike pancreatitis, there is usually a long history of ulcer-dyspepsia, and the severe pain associated with erosion of the pancreas is more gradual in onset than that of pancreatic necrosis. Vomiting may be present, but is not usually a prominent feature. Abdominal rigidity is absent, but tenderness may be elicited. The pain is alleviated by rest in bed, alkalis, and hourly feeds of milk.

Acute cholecystitis is of gradual or sudden onset in a patient who presents a longstanding history of chronic cholecystitis or biliary colic. Unlike pancreatitis, the pain, tenderness and rigidity are localised to the right upper quadrant of the abdomen: occasionally the gall bladder is palpably enlarged. Vomiting occurs, but again does not tend to be a prominent feature of the case. The pulse-rate is raised and pyrexia is present from the first, whereas these signs are usually delayed in subacute pancreatitis.

Biliary colic and intestinal obstruction may be differentiated from subacute pancreatitis by the features outlined above in the differential diagnosis of the fulminating forms of the disease.

TREATMENT

With all acute abdominal emergencies the clinician must attempt to answer two questions:—

- (1) What is the diagnosis?
- (2) Is the immediate treatment operative or expectant?

In acute pancreatitis it is unusual to be able to answer the first question positively: the disease may be suspected, but a confident diagnosis of such a rare condition can seldom be made. The answer to the second question depends upon the severity of the disease. In fulminating cases early laparotomy is called for—either because a perforated peptic ulcer is suspected or because the condition of the patient shows deterioration, with a rise in pulse-rate, during a short period of observation. In subacute cases there is usually no indication for immediate operation, and an expectant attitude is adopted: many such cases never require surgical intervention, but later operation for localised abscess formation or for a pseudopancreatic cyst is needed in some. Between the extremes of the fulminating and subacute types of the disease cases occur in which the clinician may spend many anxious hours wondering if his judgment has been correct in delaying operation, or alternatively he may regret performing an unnecessary laparotomy.

Operative procedures.

When laparotomy is deemed necessary attention is directed to two points:—

- (a) Drainage of the lesser sac.
- (b) Drainage of the biliary tract.

It has been said that as experience with cases of pancreatitis increases, less attention will be paid to drainage of the peritoneal cavity and more to drainage of the biliary tract: but it would be a brave (or should one say foolish?) surgeon who would dispense with drainage of the lesser sac in these cases, for even when the pancreas shows only swelling and oedema, necrosis and suppuration may occur later.

(a) Drainage of the lesser sac.

In the writer's opinion drainage of the lesser sac should be provided in all cases of pancreatitis for which laparotomy is performed. In hyposthenic patients drainage is most readily achieved through the lesser omentum: in hypersthenic types the most direct channel is below the stomach through the greater omentum, either above or below the transverse colon according to the anatomical conditions present. Additional dependent drainage may be provided by a stab incision in the left loin, but unless this can be performed without turning the patient it should not be done at all in one who is extremely ill, and who will not tolerate prolonged operative interference.

As well as supplying drainage of the lesser sac, it must be ensured that the pancreas itself is relieved of tension. If the peritoneum covering the pancreas is intact, incisions should be made into it to allow the swollen pancreas to expand; if there is gross haemorrhage, sloughing or suppuration the incisions should be carried into the substance of the gland, parallel to its long axis in order to avoid damage to the ducts.

(b) Drainage of the biliary tract.

In the majority of cases of acute pancreatitis chronic disease of the biliary tract is present. The gall bladder is examined, and if cholecystitis or calculi are found cholecystostomy should be performed. The common bile duct should also be palpated. If a stone is present it should be removed and the duct drained; in the rare cases of impaction at the ampulla of Vater it may prove impossible to dislodge the stone, and the patient is not fit enough to withstand prolonged procedures like transduodenal choledochostomy. Simple drainage of the duct should be performed in such an emergency, leaving removal of the calculus for a later operation. If the common bile duct is dilated without a calculus being palpable, aspiration should be carried out with a hypodermic needle and choledochostomy performed if there is evidence of infection.

Cholecystectomy should never be performed in the presence of acute pancreatitis. The patient is too ill for anything but minimal operative interference, and it is possible that the gall bladder may be needed later for the operation of cholecystgastrostomy should the fibrosis of healing in the pancreas lead to constriction of the common bile duct and the development of obstructive jaundice.

OIL ACNE

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Occupational skin disorders are an important cause of morbidity in industry and oil dermatitis is very common at the present time (MacKenzie 1943). The disorders resulting from the use of cutting oils have received considerable attention (Bridge 1918, Schwartz and Peck 1943, Whitwell 1943), whereas the equally common troubles of those using oil in the repair of mechanical vehicles have received little notice.

Action of Oil on the Skin.

Oil produces dermatogosis (Prosser White 1928) of two kinds—oil acne and oil dermatitis. The former is a true folliculitis with comedo formation; the latter is an eczematoid reaction. Schwartz (1941) states:—"All oils may plug up pores of the skin and form comedones." Thus, like Bridge (1918), he believes the acne is primarily mechanical in origin. On the other hand