PANCREATITIS
A Retrospective Review of 92 Cases
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The clinical picture of acute pancreatitis has long been recognized and provided the condition is suspected the diagnosis can be confirmed in a few minutes by estimating the serum amylase or urinary diastase.

Chronic relapsing pancreatitis and chronic pancreatitis (Comfort, Gambill and Baggenstoss, 1946) present much greater diagnostic difficulties. Atrophy of the gland results in reduction or absence of enzyme activity, so that in chronic relapsing pancreatitis there is no rise in serum amylase between attacks and with the onset of chronic pancreatitis there may be none even during an attack. Confirmation of the diagnosis, therefore, is often dependent on the onset of steatorrhoea, glycosuria or pancreolithiasis.

Pancreatitis is frequently of unknown aetiology. It is associated with biliary tract disease in roughly 50% of cases (Dragstedt, Haymond and Ellis, 1934; Bockus, Bogoch and Roth, 1953), though evidence of pancreatic duct obstruction is usually present only in a small percentage. Numerous other aetiological factors, such as trauma, septicaemia, mumps, alcoholism, hyperparathyroidism and hyperlipaemia, are well known, but the cause is obscure in about one-third of the total number of diagnosed cases.

Some authors (Smyth, 1940; Probststein, Joshi and Blumenthal, 1957) have drawn attention to the occurrence of pancreatitis in patients with an impaired blood supply to the organ, but this does not seem to have been widely recognized.

With the object of obtaining some practical information about pancreatitis in general and in particular about the relative importance of various aetiological factors and the incidence of undiagnosed cases, we have carried out a retrospective survey of all types seen during the past five years at this hospital. In the past many authors have not defined what they mean by pancreatitis. We regard it as inflammation in the organ, usually due to the escape of enzymes into the intercellular tissue, and for the purpose of the survey we decided to include any cases which conform to one or more of the following criteria:

(i) Serum amylase of over 20 units/ml. (Wohlgeruth).
(ii) Urine amylase over 40,000 units/24 hours.
(iii) Pancreolithiasis visible on abdominal skigrams.
(iv) Fat necrosis seen at operation.
(v) Histological proof from biopsy or from autopsy material.

Material
The series comprises 92 patients seen at the Whittington Hospital in the years 1955-59 inclusive in whom pancreatitis was diagnosed during life or at post-mortem in accordance with the above criteria.

Fig. 1 shows the distribution in decades for age and sex.

Diagnosis
Pancreatitis was diagnosed during life in 51 cases (55%) and at post-mortem in 41 cases (45%). It will be seen from Table 1 that in those diagnosed after death the heaviest incidence is in the seventh, eighth and ninth decades. The evidence for pancreatitis in the 51 cases diagnosed during life was a raised serum or urinary amylase in 30, findings at laparotomy in 20 and radiological evidence of pancreolithiasis in one.

Clinical Classifications
Pancreatitis is usually divided into three main groups of acute, chronic relapsing and chronic, though some authors have adopted a more elaborate classification (Howat, 1952). Some of our cases, in particular those without a characteristic clinical story who were diagnosed only on
histological evidence at post-mortem, did not fall easily into these clinical groups. It is recognized that chronic relapsing pancreatitis may develop insidiously and present with only glycosuria or steatorrhoea (Bartholomew and Comfort, 1956; Gross and Comfort, 1957). Cases diagnosed only at autopsy, in which even in retrospect there was no clinical evidence of pancreatitis, we have classified as silent and divided them into the three clinical groups in accordance with the histological findings: those with acute haemorrhagic pancreatitis and fat necrosis as acute, those with evidence of chronic inflammation and superimposed fat necrosis as chronic relapsing, and those with chronic inflammation and atrophy as chronic.

**Associated Diseases**

Table 2 shows the incidence of associated diseases in the 92 cases.

**Biliary Tract Disease**

The association between gall bladder disease and pancreatitis is well recognized, though opinions differ as to which is the primary condition. It is of interest that in 48 cases out of 92 (52%), in our series, there was no evidence of disease in the biliary tract.

Figure 2 shows the incidence of associated biliary tract disease divided up into age groups.

**Cardio-vascular Disease**

Evidence of cardio-vascular disease was obtained
in 46 of the 92 cases (50%); the following criteria were taken to indicate its presence:

(i) Hypertension (systolic pressure over 160 and diastolic of over 100).
(ii) Cardiac failure.
(iii) Cerebro-vascular accidents.
(iv) Myocardial infarctions (old and new).
(v) Post-mortem evidence of widespread vascular disease and left ventricular hypertrophy.

The distribution of the changes are as follows:

- Hypertension: 28 cases
- Cor pulmonale: 3 cases
- Myocardial ischemia and coronary disease: 8 cases
- Rheumatic heart disease: 1 case

Cerebro-vascular accident: 5 cases
Polyarteritis nodosa: 1 case

Fig. 3 shows the distribution of these changes in the different age groups. As would be expected the incidence is greatest in the older ages.

Fig. 4 shows the degree of overlap with associated biliary tract disease. This is not marked.

**Controls**

Examination of a control series matched for age and sex has shown a very similar incidence of cardio-vascular disease, but a smaller incidence of gall bladder disease (Table 3).
livers. At post-mortem, evidence of liver disease was found in a further 22 cases, but was regarded as secondary to other more important pathology, as seen in the following table:

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive heart failure</td>
<td>12</td>
</tr>
<tr>
<td>Obstructive jaundice with cholangitis</td>
<td>7</td>
</tr>
<tr>
<td>Secondary hepatic carcinoma</td>
<td>3</td>
</tr>
<tr>
<td>Polyarteritis nodosa</td>
<td>1</td>
</tr>
</tbody>
</table>

**Systemic Disease**

The pancreatitis in these cases was regarded as incidental.

**Painless Pancreatitis**

In 18 of our cases (19.6%) no history of pain was obtained. In four of these steatorrhoea, glycosuria or pancreolithiasis was present and they therefore fall into the classical group of chronic pancreatitis (Gross and Comfort, 1956). The remaining 14 were diagnosed only on histological findings, 13 at autopsy and one at laparotomy, and even in retrospect there was no evidence to suggest a clinical diagnosis of pancreatitis. We have, therefore, sub-divided our cases of painless pancreatitis into:

(i) Chronic painless pancreatitis
(ii) Silent pancreatitis

the latter being further classified on histological evidence alone into acute, chronic relapsing and chronic.

**Painless but otherwise Classical Chronic Pancreatitis**

Table 4 summarizes these cases:

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Glycosuria</th>
<th>Steatorrhoea</th>
<th>Pancreolithiasis</th>
<th>P.M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>75</td>
<td>F</td>
<td>2 years</td>
<td>o</td>
<td>+</td>
<td>C.V.D.</td>
</tr>
<tr>
<td>78</td>
<td>F</td>
<td>2 months</td>
<td>o</td>
<td>o</td>
<td>C.V.D.</td>
</tr>
<tr>
<td>81</td>
<td>M</td>
<td>o</td>
<td>o</td>
<td>+</td>
<td>C.V.D.</td>
</tr>
<tr>
<td>43</td>
<td>F</td>
<td>28 years</td>
<td>3 years</td>
<td>o</td>
<td>Atrophy of pancreas</td>
</tr>
</tbody>
</table>

P.M. = Post mortem. C.V.D. = Cardio-vascular degeneration

These are cases of classical chronic pancreatitis which happened to be painless.

**Silent Pancreatitis**

**Acute**

Three cases, two women and one man, had evidence of acute inflammation in the pancreas associated with fat necrosis. Their average age was 73 and all had associated cardio-vascular disease.
Table 5 summarizes these cases and it will be seen that two of them were obviously too ill to give a satisfactory history.

**Table 5**

**Silent Pancreatitis (Three Cases)**

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Clinical Picture</th>
<th>P.M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>78</td>
<td>M</td>
<td>Nephrotic syndrome</td>
<td>Recent thrombosis of renal vein. Acute purulent pancreatitis with fat necrosis</td>
</tr>
<tr>
<td>83</td>
<td>F</td>
<td>Collapse. Died in few hours</td>
<td>Acute pancreatitis with fat necrosis. Bronchopneumonia. Fibrosis left ventricle. L.V.+</td>
</tr>
<tr>
<td>54</td>
<td>F</td>
<td>Diabetic coma</td>
<td>Acute pancreatitis. Fat necrosis. L.V.+</td>
</tr>
</tbody>
</table>

**Chronic Relapsing**

Four cases, all women, had fat necrosis superimposed upon pancreatic calcification, pancreatic atrophy or pancreatic suppurative; their average age was 87. Table 6 summarizes the findings.

**Table 6**

**Silent Chronic Relapsing (Four Cases)**

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Clinical Picture</th>
<th>P.M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>78</td>
<td>F</td>
<td>Appendicitis. Pulmonary infarction</td>
<td>Long-standing suppurrative pancreatitis</td>
</tr>
<tr>
<td>66</td>
<td>F</td>
<td>No history. Admitted in coma</td>
<td>Bronchopneumonia. Chronic inflammation and fat necrosis. Pancreatic atrophy. Fibrosis and atrophy of thyroid</td>
</tr>
<tr>
<td>92</td>
<td>F</td>
<td>Carcinoma stomach. Gastrectomy</td>
<td>Fat necrosis. Calcification in obstructed duct</td>
</tr>
<tr>
<td>92</td>
<td>F</td>
<td>Carcinoma pancreas</td>
<td>Chronic inflammation and fat necrosis</td>
</tr>
</tbody>
</table>

It would appear that these patients had suffered from painless pancreatitis for some time and there was a terminal exacerbation.

**Chronic**

Seven otherwise silent cases had histological evidence of chronic pancreatitis, in one the section was taken after total pancreatectomy for pancreatic carcinoma, the remaining six at autopsy. Table 7 gives a summary of these cases.

**Table 7**

**Silent Chronic Pancreatitis (Seven Cases)**

<table>
<thead>
<tr>
<th>Age</th>
<th>Sex</th>
<th>Clinical Picture</th>
<th>P.M.</th>
</tr>
</thead>
<tbody>
<tr>
<td>72</td>
<td>F</td>
<td>No history</td>
<td>Atrophy of thyroid. Chronic inflammation and fibrosis of pancreas</td>
</tr>
<tr>
<td>67</td>
<td>F</td>
<td>Chronic heart failure</td>
<td>Cor pulmonale. Fibrosis of lungs. Chronic inflammation and fatty atrophy of pancreas</td>
</tr>
<tr>
<td>60</td>
<td>M</td>
<td>Coronary thrombosis</td>
<td>Myocardial infarction. Chronic inflammation and atrophy of pancreas</td>
</tr>
<tr>
<td>36</td>
<td>M</td>
<td>Cirrhosis and haematemesis</td>
<td>Periporal fibrosis. Fibrosis of pancreas</td>
</tr>
<tr>
<td>41</td>
<td>F</td>
<td>Alcoholism. Barbiturate poisoning</td>
<td>Bronchopneumonia. Fatty change liver. Fibrosis and atrophy of pancreas</td>
</tr>
<tr>
<td>59</td>
<td>M</td>
<td>Carcinoma pancreas</td>
<td>Laparotomy. Carcinoma pancreas. Severe fibrosis. Total pancreatectomy</td>
</tr>
<tr>
<td>81</td>
<td>M</td>
<td>Carcinoma prostate</td>
<td>Multiple secondaries in liver. Chronic inflammation of pancreas</td>
</tr>
</tbody>
</table>

It gives a summary of these cases.

**Discussion**

A review of 92 cases of pancreatitis shows a larger incidence in women than in men, the proportion being over two to one. This is in marked contrast to figures from the United States (Gross, 1958).

An association with biliary tract disease is similar to previous published series, for we found it to be present in about half of our cases, and no new information has emerged which would help to indicate whether the pancreatitis precedes the biliary tract disease or vice versa.

We have found a high incidence of cardio-vascular disease in this series without much overlap with those cases occurring in association with gall bladder disease. A control series, however, shows a similar incidence of cardio-vascular disease in the same age and sex groups which suggests that both diseases occur with advancing age.

Joshi, Probst and Blumenthal (1957) showed that the age distribution in 300 cases of pancreatitis was similar to that observed in other cardio-vascular complications. There is considerable evidence to suggest that some cases of pancreatitis are associated with an impaired blood supply to the organ (Probst and others, 1957) and our figures would be consistent with such a possibility. The significance of this observation could be assessed only by taking routine sections of the pancreas at post-mortem on a number of cases having cardio-vascular disease with a control group of the same ages and sex who did not have evidence of cardio-vascular disease.
There were six cases of liver disease, but alcoholism was proved in only two, a remarkable difference from figures published in the United States of America. Eighteen of our cases (19.6%) were painless and 14 were unsuspected until evidence was found at autopsy.

Clearly many cases of pancreatitis are undiagnosed and if this number is to be reduced clinicians must not only be aware that symptoms may be minimal or absent, but must also be prepared to carry out more frequent investigations to make or exclude this diagnosis.

In 1955 the following policy was adopted in this unit:

1. All cases admitted with biliary colic have a routine serum amylase and urinary diastase estimation performed.

2. Pancreatitis is suspected in patients with undiagnosed upper abdominal pain having the following characteristics:

   (i) It is severe.
   (ii) It may radiate to the back.
   (iii) It may be precipitated by taking alcohol or a heavy meal.
   (iv) It is not relieved by alkalis.
   (v) The patient sometimes adopts a characteristic attitude, e.g. curled up in bed or sitting on a chair and sometimes getting on the hands and knees.

In such patients some or all of the following investigations are done, if appropriate:

   (i) Faecal fat estimation.
   (ii) Glucose tolerance curve.
   (iii) Straight x-ray of abdomen for evidence of pancreolithiasis.
   (iv) Are provided with a Winchester bottle in which to collect the urine for 24 hours after an acute attack. The urinary amylase is then estimated.

   (v) Duodenal intubation and secretin test.

The introduction of this procedure has made the medical staff in the unit more aware of the fact that patients with atypical abdominal pain may be suffering from pancreatitis, with the result that 12 cases were diagnosed during the first 11 months of 1960, as compared with three during the whole of 1955.

N.B.—These figures refer to this unit only as compared with the whole hospital from which the 92 cases were collected.

Summary

1. A review of 92 cases of pancreatitis shows that only 55% were diagnosed clinically. The incidence in women was greater than in men, being two to one. This differs from other series.

2. It is concluded that many cases of pancreatitis are undiagnosed, some because the condition may be insidious, but many because they are overlooked.

3. A significant number of cases may be painless.

4. Suggestions are made for clinical aids to the diagnosis of pancreatitis.

5. No new information emerges regarding the etiology of pancreatitis, though 46 (50%) were associated with cardio-vascular disease.

We have to thank our colleagues, both surgical and medical, for permission to include their cases and Dr. S. Robinson and Dr. C. Bryson, of the Morbid Anatomical Department, for the histological information and for their advice and help in preparing this paper.

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Pancreatitis: A Retrospective Review of 92 Cases

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Postgrad Med J 1961 37: 792-797
doi: 10.1136/pgmj.37.434.792

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