ACUTE PANCREATITIS IN A PERIPHERAL HOSPITAL

by

J. G. CAMBLIN

Surgical Registrar Lurgan Hospital

NOW: Orthopaedic Registrar in Musgrave Park Hospital

PANCREATITIS is a disease of variable aetiology and the acute form usually presents as an acute abdominal pain. The incidence in the general community has been estimated as five cases per hundred thousand per year. (Trapnell, 1968). In hospital the incidence has been put at one in six hundred admissions (Robbins, 1967) or at one hundred cases in fifteen hundred acute abdominal emergencies over a period of six years (Walker, 1972). This account is a review of the cases of acute pancreatitis seen in one surgical unit in a peripheral hospital in one year.

CASE HISTORY

The data on all cases is set out below.

Case 1

This was a 36 year old male with a six year history of indigestion and was prone to drink a bottle of spirits and some beer daily. He was a publican by trade. He was admitted with a steady epigastric pain which radiated through to the back and was associated with vomiting. The abdomen was soft and the only clinical finding was tenderness in the epigastrium. A serum amylase of 788 Street Close units was found and the initial diagnosis of perforated duodenal ulcer was corrected to one of acute pancreatitis.

He was treated conservatively by naso-gastric suction and intravenous fluids. Trasylol 200,000 units were given stat. I.V. followed by 200,000 units four hourly via the I.V. drip for three days. In addition D.F. 118, 50 mg., atropine 0.6 mg., and ampicillin 250 mg. were given six hourly. Nine days after admission he was symptom free but later became jaundiced. The amylase at this time was 48 S.C. units and the liver function tests which had been normal until now showed a bilirubin of 0.86 mg. per 100 ml. This was associated with a pyrexia of 101°F and settled over the next four days.

One week later he had a similar attack with a pyrexia of 103°F and the full I.V. regime restarted but without Trasylol in the drip. He settled and was discharged 43 days after admission.

Three months later he was re-admitted with acute abdominal pain and a pyrexia of 103°F and at this time a pancreatic pseudocyst was diagnosed and confirmed on barium meal. This was later drained surgically via a tube drain. A sinogram via the drain site demonstrated a communication to the small bowel. He was discharged symptom free and an amylase of 18 S.C. units. The drain site fistula had closed at this time.

Case 2

This 52 year old female was admitted via the cardiac ambulance complaining of severe epigastric pain and vomiting. There was shortness of breath but no shock. The
pain moved to the left lumbar region and a diagnosis of diverticular disease was made. On examination there was marked tenderness and rebound tenderness in the L.I.F. The E.C.G. was normal and the amylase 350 S.C. units. She was treated by conservative methods and settled by the sixth day. Barium meal and cholecystogram showed a poorly functioning gallbladder and no evidence of peptic ulceration. She was discharged one week after admission and her name placed on the waiting list for cholecystectomy.

Case 3

This 44 year old female was admitted with a two day history of epigastric pain and vomiting. She had had a similar attack three months before and denied any fatty food intolerance. She had a pyrexia of 99°F and a mass could be palpated in the left flank. Rebound tenderness was present. The serum amylase was 50 S.C. units and X-ray did not reveal any pathology. However, at laparotomy a swollen pancreas and massive fat necrosis was found and confirmed by omental biopsy. Post-operatively full conservative treatment was started including Trasylol. On discharge the amylase was 29 units and the convalescent mumps anti-body was slightly elevated.

Case 4

A 78 year old female admitted with a severe pain in the left flank with shortness of breath, a cough and sputum. On examination she was dehydrated, cyanosed and shocked with a B.P. 80/50 mm. Hg. There was generalised rhonchi and crepitations in both lung bases. The abdomen was distended, rigid and tender in the left half. X-ray showed consolidation of the left lower lobe. She was treated as for intestinal obstruction and given antibiotics for her chest infection. She deteriorated over a period of one week and died in renal failure without laparotomy being carried out. The diagnosis of pancreatitis was made at post mortem.

Case 5

This 73 year old man was admitted with a two day history of pain in the L.I.F. and vomiting. There was generalised abdominal tenderness, distension, and rebound tenderness. Slight jaundice was present. At laparotomy a diagnosis of acute haemorrhagic pancreatitis was made. There was marked fat necrosis. He collapsed and died shortly after the operation.

Case 6

The patient was a 76 year old female with a four day history of epigastric pain radiating through to the back. This was associated with vomiting. She had a past history of hypertension and heart disease and an initial diagnosis of mesenteric embolism was made. The amylase was 1080 S.C. units and she was treated by conservative measures including Trasylol. She deteriorated and died within 24 hours.

Case 7

This 70 year old lady had a one week history of crampy upper abdominal pain with slight jaundice and vomiting. On examination she was fat with coexistent obstructive jaundice and a pyrexia of 100°F. She was diagnosed as cholecystitis with bile duct obstruction. Serum amylase was 173 S.C. units and the gall bladder failed to concentrate the dye on two cholecystograms. Six days after admission pancreatitis was found at operation and at the same time cholecystectomy was carried out. She was discharged two weeks after admission and at follow up was symptom free and the convalescent serology showed a 10 unit rise in mumps antibody.

Case 8

A 66 year old male was admitted via the cardiac ambulance complaining of epigastric pain radiating to the chest and left arm. He was short of breath but there were no
abnormal clinical findings. The E.C.G. was normal and the amylase 143.6 S.C. units. All other blood tests were normal. He was initially treated as a myocardial infarction and settled and was discharged one week later. The diagnosis was mild acute pancreatitis and the amylase on discharge was 21 S.C. units.

Case 9

This 50 year old male who had never had any history of heart disease or ulcers was admitted via the cardiac ambulance with epigastric pain and nausea. He had marked epigastric tenderness and his E.C.G. was normal. The amylase was 210 S.C. units and a diagnosis of acute pancreatitis was made. He was treated conservatively including Trasylol and settled within a week. The amylase had fallen to 28 units. The only complication was an initial pyrexia of 100.4°F. There was a slight rise in the Coxackie B4 antibody level and no evidence of ulceration was found on barium meal.

Case 10

This 35 year old obese female was admitted with a sudden onset of severe epigastric pain and vomiting. There was epigastric tenderness and a diagnosis of acute pancreatitis was confirmed by an amylase of 588 S.C. units. She was treated by the full conservative regime until the pain settled five days after admission. Cholecystogram showed a non-functioning gall bladder and on discharge after three weeks the amylase was 27 units. She was re-admitted three weeks later with more pain and a routine cholecystectomy was carried out.

Case 11

This 47 year old female was admitted with severe upper abdominal pain and vomiting. The pain moved into the back and was eased by sitting up. On examination there was marked epigastric tenderness. The initial diagnosis of duodenal ulceration was changed to acute pancreatitis and confirmed by a serum amylase of 1300 S.C. units. She was treated by the full conservative regime and gradually improved so that at three weeks on discharge the amylase was 21 units. A non-functioning gall bladder was noted and removed four months later. Viral studies showed a 20 unit rise in the mumps antibody.

**DISCUSSION**

It can clearly be seen that the diagnosis of acute pancreatitis is not often made correctly, and that there was only one correct diagnosis in this series of eleven cases. The general practitioner who sees less pancreatitis per year than hospital staff diagnosed myocardial infarction in three cases, the remainder being sent direct to the surgical unit with a diagnosis of “acute abdomen”. House officers dealing with the cases on arrival in the hospital medical unit changed the diagnosis to pancreatitis in one of three cases brought in by cardiac ambulance. Surgical advise was sought in all cases. On the surgical side the house officer made only one correct diagnosis in the remaining seven cases. The main confusion was with peptic ulceration, gall bladder disease and diverticular disease. The diagnosis of other acute abdominal conditions also being made in four cases.

**AETIOLOGY**

As already stated the aetiology is varied but there is a large school of thought that puts forward pancreatic duct obstruction as the most important factor (Pollock, 1959; Bailie, 1962; Robbins, 1967). This has been shown in dogs but not sub-
stantiated in man. Obstruction may be produced by biliary calculi or by increased tone in the sphincter of Oddi as can be induced by opiate drugs or by alcohol. Other factors include pancreatic calculi, epithelial debris and pancreatic oedema. Alcohol intake has been often stated as a classical cause of acute pancreatitis but in a large series of cases in Los Angeles no such cause was found (Pollock, 1962). Theoretically alcohol induces pancreatitis by causing the stomach to produce secretin which promotes pancreatic activity and simultaneously directly increases sphincter tone. Infection of the gland is not essential but is an important factor, as shown experimentally by Banks (1971). He introduced E. coli into pancreatic ducts of germ free animals to cause experimental pancreatitis and also found that this organism was involved in cases of high small bowel obstruction.

In this series only one case was noted who admitted to a heavy alcohol intake. Several other cases were associated with gall-bladder disease as is often reported in the literature. In some cases a viral aetiology was suspected, especially in those cases occurring during the month of July as there was a peak incidence at this time. No cases were noted due to any of the other rarer causes suggested, such as an hereditary disposition which is associated with diabetes, pancreatic cysts and an increased tendency to pancreatic carcinoma.

**Pathology**

During the course of the disease amylase and other enzymes are liberated into the circulation. The amylase level rises in the first twenty-four hours and usually settles by the fifth day and almost always by the tenth. This rise is paralleled by the urinary amylase (Edmondson, 1952). The serum lipase level also rises but its estimation is not accurate. The serum amylase level is the most accurate laboratory test but the level also rises in other disease processes, but not to the same extent. The associated shock in acute pancreatitis is said to be due to kallikrein. This substance is present in the plasma and in granulocytes and is maintained inactive by circulating inhibitors. On activation by the release of pancreatic enzymes kallikrein is converted to a decapeptide, kallidin 10, and thence to bradykinin. This acts as a histamine and causes peripheral arteriolar dilation and can result in a loss of thirty per cent of the circulating blood volume (Ganong, 1967; Havard, 1972).

**Clinical**

Acute pancreatitis is said to attack the over forty year old age group and mainly those between the ages of fifty and sixty years. One series in a large hospital found that the majority of cases were between ages of 60 and 70 years old (Pollock, 1959; Ogilvie, 1962). In this present series only two were under 40 years, two in the 40-50 age group and four were over 70 years old. The sex distribution has been reported as 70 per cent female and 30 per cent male and of the eleven cases now reported the split was 60:40 female to male. The thin person is said to be more at risk as compared to the fat as in the case of gall-bladder disease (Bailie, 1962). This is disputed by other authors who point out the strong association with gall-bladder disease in females (Pollock, 1959). In the study group there were
seven female and four male patients with an age distribution of 35-78 years. The range for males was 36-73 years and 35-78 for females. Of these, two males were over 60 years old and three females were over 70 years of age.

During the year a total of 793 acute abdominal emergency cases were admitted to the unit and comprised all forms of acute surgical conditions of the abdomen. Of the 793 cases 91 had gall-bladder disease which was a slightly lower incidence than the 101 cases of gall-bladder disease noted in the same year in another regional hospital.

Epigastric pain and vomiting were the most common presenting symptoms as confirmed by other reports (Trapnell, 1968). Of the eleven cases nine had epigastric pain and of these only one had pain which radiated through to the back and which was eased by sitting up as in the classical description. Seven of the nine cases had associated vomiting. None presented or developed any of the more uncommon signs or symptoms of acute pancreatitis. As with previous accounts the diagnosis was most often confused with duodenal ulceration, gall-bladder disease and with myocardial infarction. With regard to the obesity of the patient only two of the eleven cases were described as fat. Shock was not a prominent feature and was found in only one case. In ten cases intravenous fluids were used as part of the initial treatment and thus may have prevented its onset.

As a diagnostic aid the serum amylase was most useful. Early radiology was of very limited value, its use being limited to the exclusion of other pathology. Radiology was of more value in the later stages of the disease in determining gall-bladder disease in four cases. Barium meal investigation was used to exclude peptic ulceration and also in one case to define pancreatic pseudocyst. The literature confirms the serum amylase as the most useful test (Gurd, 1970; Banks, 1971), and also states that the renal clearance of amylase correlated with the renal creatinine clearance may be a most accurate test and would compensate for the effects of renal disease if the urinary amylase alone were measured (Edmondson, 1952; Gurd, 1970; Banks, 1971). One can also carry out more exotic tests, e.g. methaemoglobin estimations, but these are of no real value. Serum calcium levels may be of use in the 5 per cent that develop tetany and in these cases the disease is usually fatal. Of the eleven cases in this series the serum amylase was positive in eight, negative in one and not estimated in another two.

On looking at the spread of cases throughout the year no cases were found in the first two or the last three months of the year. There were two cases early in the year, one in March and one in April. The next six cases were all in the month of July followed by one case in August and two others in September. The sudden peak in July suggested the possibility of a common factor and this was thought to be viral. Serological studies were made, and of the six July cases three had slight elevation of mumps antibody and the case in April had a slight rise in antibody to Coxackie B4 virus. No other common factor was found. In the literature it was noted that mumps can cause a slight elevation of the serum amylase. In three cases the amylase was elevated from the normal range of 18-30 S.C. units to four times this level and in one case to 1300 S.C. units.

51
TREATMENT

In this series conservative treatment was by naso-gastric suction, intravenous fluids, antibiotics, antispasmodics and by analgesics. Treatment included Trasylol in six and only one died. This was a 76 year old female who died within the first twenty-four hours and her amylase level was 1080 S.C. units. Of the five cases not treated with Trasylol two died, one at 24 hours and one after a week. One was 78 years old and the other had a laparotomy. Of those that did not receive Trasylol only one had an amylase level of over 200 units, and of those that did only one member had a level below 200 units. One had a level of over 800 units and two of more than 1,000 units. In acute pancreatitis there is a low incidence of hyperlipidaemia and a risk of fat embolism (Edmondson, 1952). In this uncommon situation it would appear that Trasylol may be of use as a prophylaxis as it has been shown to be of value in established fat embolism following trauma (Chapman, 1959). Trasylol is very good experimentally in the treatment of acute pancreatitis but less so clinically. This may be due to the delay in starting treatment as the patient rarely presents within the first 24 hours of onset of symptoms. In dogs an 8 hour delay, and a 20 hour delay in man, lowers the effectiveness of the treatment (Trapnell, 1968; Banks, 1971). Trasylol also is said to block kallidin 10 formation and thus prevent shock. It has been suggested that a similar agent to inhibit elamase may be of more use but this has been less widely tried (Banks, 1971). Steroids are also recommended to counter shock (Cook, 1959; Trapnell, 1968).

All are in agreement that intravenous fluids are most important and that intestinal rest is beneficial. There is divergence of opinion as to the value of antibiotics and antispasmodics although it is recognised that antibiotics do help to reduce infection as a complication (Pollock, 1959; Trapnell, 1968). There is also agreement that gall-bladder investigation and follow-up cholecystectomy, if required, is an important procedure. The recommended time for operation being one month after the initial symptoms have settled. In the present series three had a laparotomy as an aid to diagnosis and one a cholecystectomy one week after admission. All told, five cases had a follow-up cholecystectomy. In only one case was there any long term complication requiring surgical intervention. This was the development of a pancreatic pseudocyst that had to be drained some months after the original admission.

The length of stay in hospital varied from one to nine weeks. This was measured by the total number of days of inpatient treatment required and not including time between admissions. Two patients died within the first 24 hours and one in the first week. Two were allowed home after one week, two at the end of the second week, two at three weeks and two after a stay of four weeks. When taking subsequent treatment into account one was in hospital for 9 weeks, three for 4 weeks, two for two weeks and two for only 1 week.

PROGNOSIS

It is expected that the elderly would have a higher mortality due to their inability to withstand shock and to other pathology. It is also expected that the
more serious the disease the worse the outlook. Those who died were all over 70 years old. Acute pancreatitis is reported to have a 26 per cent mortality (Trapnell, 1968) and it has been put as high as 50 per cent in the haemorrhagic variety and as low as 13 per cent in other forms (Banks, 1971). The onset of complications has a serious prognostic significance especially in those with a serum calcium level of below 5.6 mg. 100 ml. in which there is almost a hundred per cent mortality (Banks, 1971). When associated with trauma acute pancreatitis is 80 per cent fatal. Those that do not develop complications settle within the first week in almost all cases.

**Conclusions**

In conclusion acute pancreatitis is difficult to diagnose, especially for the general practitioner and for the junior hospital doctor. At a more senior level six cases were correctly diagnosed and five were not. Of these two had pain in the left iliac fossa and one died shortly after admission. The clinical picture plus a serum amylase estimation on arrival in hospital gives an accurate diagnosis in 80 per cent of cases. The series illustrates the association with gall-bladder disease and it is noted that in these cases cholecystectomy should be carried out at a later date. Intravenous fluids are very important and additional support by other measures of some use.

**Summary**

A series of eleven cases of acute pancreatitis in a peripheral hospital during a one year period have been reported. The difficulty in diagnosis, the value of initial laboratory investigation and the current clinical treatment have been illustrated.

I am grateful to Mr. J. Balmer, Mr. W. Graham and Mr. P. Allen, Consultant Surgeons of the Craigavon Area Hospital for allowing access to their clinical material, and to the Staff of the Records Department for their assistance.

**References**

Bailie, H. and Love, McN. (1962). *Short Practice of Surgery.* 14th Ed. London: Lewis. 846.
Banks, P. A. (1971). *Gastroenterology.* 61. 1. 382.
Chapman, R. (1959). *British Medical Journal.* 2. 602.
Cook, W. T. and Meynell, M. J. (1959). *British Medical Journal.* 1. 233.
Edmondson, H. A., Berne, C. J., Homann Jr., R. E. and Wertman, M. (1952). *American Journal of Medicine.* 12. 34.
Ganong, W. F. (1971). *Review of Medical Physiology.* 3rd Ed. Los Altos. Lange. 354.
Gurd, A. R. (1970). *Journal of Bone and Joint Surgery.* 52B. 732.
Havard, C. W. (1972). *Hospital Medicine.* 7. 443.
Ogilvie, R. (1962). *Histopathology.* 6th Ed. London: Livingstone. 274.
Pollock, A. V. (1959). *British Medical Journal.* 1. 6.
Robbins, S. L. (1967). *Pathology.* 3rd Ed. Philadelphia: Saunders & Co.
Trapnell, J. E. (1968). *Hospital Medicine.* 1. 181.
Walker, R. and Mallinson, C. (1972). *Medicine.* 8. 598.