

## Acute pancreatitis in general practice

S. P. COUGHLIN, MB, CH.B, MRCP  
General Practitioner, Great Eccleston, Lancashire

**SUMMARY.** During an eight-year period, 13 patients suffering from acute pancreatitis were seen in a three-doctor rural practice. The incidence was greater than most published hospital series would suggest. With one unusual exception, all the cases were mild, in that there were no complications or fatalities. In some of the patients the clinical features were such that the diagnosis might have been overlooked. Eight of the patients were managed at home. There was a strong association with biliary tract disease.

### Introduction

**A**CUTE pancreatitis is not a common disease. The reported incidence of the condition varies from about 50 per million per year (Bourke, 1975) to 331 per million per year (Graham, 1977). The annual incidence is not steady and wide variations may occur from year to year (Bourke, 1975).

The aetiology of the disease is unknown but there are certain associated factors. Trapnell (1966) recorded that 54 per cent of cases were associated with biliary tract disease and 4.5 per cent with chronic alcoholism. Other factors included mumps, hyperparathyroidism, carcinoma and drugs (notably steroids), but in 34.5 per cent of his patients he found no associated factor. A far greater association with alcoholism has been reported abroad: Opie (1975) found it in 24 per cent of his cases, and in Olsen's (1974) series 78 per cent were alcoholics.

The classical symptoms of acute pancreatitis are acute upper abdominal pain and vomiting, with collapse in severe cases. The usual method of diagnosis is by means of the serum amylase test. Levels greater than 1,000 units/l are generally regarded as diagnostic. However, 5 per cent of cases may have no elevation of serum amylase, even early in the attack (Trapnell, 1972; Ranson, 1979). Others may only have slightly elevated levels (200-1,000 units/l). Unfortunately, elevated serum amylase levels can occur in other acute abdominal conditions, in particular acute cholecystitis, perforated duodenal ulcer, intestinal obstruction, mesenteric embolus and ruptured or dissecting aortic aneurysm, all of

which can present with a similar clinical picture to acute pancreatitis. However, in these conditions the serum amylases are usually in the range of 300-500 units/l. In a severely ill patient with only slight elevation of the serum amylase, a laparotomy may be necessary even though the history might suggest acute pancreatitis.

Complications are reported in about 12 per cent of patients admitted to hospital (Trapnell, 1972). These complications greatly increase the patient's chances of dying and are, in order of frequency, abscess, pseudocyst, haematemesis, duodenal ileus, toxic psychosis and renal failure. Burst abdomen may complicate laparotomy more commonly than usual. Mortality rates of about 20 per cent have been quoted (Pollock, 1959; Trapnell, 1972). If cases in which the diagnosis is made only at necropsy are excluded, then lower mortality rates (around 10 per cent) are quoted (Medical Research Council, 1977).

### Thirteen episodes of acute pancreatitis

In an eight-year period, 13 patients were diagnosed as suffering from acute pancreatitis in a three-doctor rural practice of approximately 5,500 patients.

#### *Clinical features*

The first striking feature of our 13 cases (see Table) is that 10 requested urgent home visits, usually outside normal working hours. With the exception of case 12, who never experienced any pain, all the patients had upper abdominal pain, usually in the epigastrium. The degree of pain was classified as mild, moderate or severe, according to the doctor's observations and the patient's description. Although there was wide variation in the severity of pain when the patients were seen, all who experienced pain had a history of moderate or severe pain when the attack began, even though, in cases 3, 4 and 11, it had subsided by the time the doctor was consulted. The severe phase of pain usually lasted only a short time; most patients had very little pain after a few hours. In two of the cases sent to hospital (cases 2 and 7) the pain lasted for more than 24 hours. Case 13 had pain severe enough to require a second pethidine injection 16 hours after the attack began.

Though vomiting was present in all cases, in some it

Thirteen cases of acute pancreatitis (1973-81).

Case	Sex	Age	Nature of consultation	Pain when seen	Amylase (units/l)	Home or hospital	Cholecystogram	Other features
1	F	36	Urgent call	Severe	4,800	Home	Pathological	—
2	F	69	Urgent call	Severe	2,250	Hospital	Pathological	Vomiting, drip and suction
3	F	79	Urgent call	Mild	4,000	Home	—	Abdominal distension
4	F	84	Routine visit requested	Mild	1,300	Home	—	—
5	M	73	Urgent call	Severe	4,000	Hospital	Pathological	Ileus, drip and suction
6	F	82	Urgent call	Moderate	4,000	Home	Pathological	Vomiting +
7	M	60	Urgent call	Severe	3,000	Hospital	Pathological	Vomiting + aprotinin
8	M	64	Urgent call	Moderate	800	Home	Pathological	Possible recurrent attack. Vomiting +
9	M	72	Urgent call	Severe	4,800	Hospital	Pathological	Ileus, distended abdomen. History of duodenal ulcer. Drip and suction
10	F	67	Urgent call	Moderate	1,067	Home	Pathological	Vomiting +
11	F	47	Routine appointment	Nil	2,400	Home	Pathological	—
12	M	55	Routine appointment	Nil	—	Hospital	Normal biliary tract at post-mortem	Hyperosmolar diabetic ketoacidosis. Later renal failure. Post-mortem diagnosis
13	M	42	Urgent call	Severe	4,130	Home	Normal	Ileus, vomiting +

was quite severe. Another notable feature was that the amount of tenderness was less than one would expect relative to the initial severity of the pain. Cases 5, 9 and 13 had very severe pain and absent bowel sounds, and yet one had to press quite deep into the epigastrium to elicit tenderness. This is a well-known feature of acute pancreatitis.

*Diagnosis: the serum amylase test*

All but one patient showed levels above 1,000 units/l. In case 8 the level was only 800 but, on clinical grounds, this patient was still thought to have acute pancreatitis. Case 12 was a post-mortem diagnosis and no amylase estimation was performed. Three cases with amylases between 300 and 500 were seen during the eight-year period, but have not been included in the series.

The local laboratories performed the tests urgently and telephoned the results. We found that asking one of the patient's relatives to take the sample to the laboratory was usually the quickest way to get it there. If the blood was taken after the laboratory had closed, the amylase estimation was carried out at the earliest opportunity the next morning. The delay in these cases was felt to be reasonable because clinical improvement had been noticed after a period of observation and because amylase remains stable in stored blood for some time—a few hours' standing does not affect the level. Samples collected during office hours were sent for analysis immediately if the clinical picture was severe.

*Management*

Eight patients were treated at home, as their symptoms were not severe when they were seen, or subsided within a few hours. Six of them required an injection of pethidine to relieve the pain. Case 13 had a second injection because, as the effects of the first one wore off, the patient felt that his pain was beginning to get worse, although it never again reached severe levels. For the first 24 hours the patients were allowed only sips of water or tea. Thereafter, there was a gradual return to a normal diet. Ampicillin was usually given to reduce the risk of secondary infection, although there has been some doubt about its efficacy (Howes *et al.*, 1975). All the patients were quickly able to walk about. These patients were all closely followed up because, in a minority of mild cases, unexpected deterioration can occur (Trapnell, 1966). No complications were observed and none of the patients died.

Five patients were admitted to hospital:

Case 2. No details about the reason for admission are available.

Case 5. This man collapsed suddenly about 18.00 with severe epigastric pain. On examination, there was only slight epigastric tenderness, yet an absence of bowel sounds. The clinical diagnosis was confirmed by serum amylase estimation the next morning but, although the pain had eased, there was still evidence of paralytic ileus. He was therefore admitted to hospital, where the

house surgeon duly noted the presence of bowel sounds on arrival.

Case 7. This man had a history of dyspepsia and it was felt that a perforated peptic ulcer was a more likely diagnosis than acute pancreatitis.

Case 9. Acute pancreatitis was considered the most likely diagnosis on clinical grounds. There was, however, a history of duodenal ulcer and, since it was Saturday afternoon, a rapid amylase estimation was not possible. He was therefore admitted to hospital, where the medical staff have the benefit of an on-call technician.

These four patients were treated with intravenous fluids and nasogastric suction. All recovered without complications. One patient was treated with the pancreatic enzyme inhibitor aprotinin (Trasylol).

Case 12. Presented at a normal surgery with a dry, ulcerated mouth. He proved to be a diabetic with hyperosmolar ketoacidosis. Following control of his diabetes in hospital, he became oliguric and uraemic and died. Post-mortem examination revealed acute necrotizing pancreatitis with almost total destruction of that organ. He never experienced abdominal pain.

Nine patients had a cholecystogram performed after the attack. In all but one of these a pathological gall bladder was demonstrated. Case 6 was already known to have biliary tract disease at the time of the attack. Cholecystograms were not performed on two patients, as they were thought to be too old to undergo surgery and they made it clear that they would refuse cholecystectomy if offered. Case 12 was found to have a normal biliary tract at post-mortem. Six patients subsequently underwent cholecystectomy. In none of the patients was alcohol thought to be a precipitating factor. No patient has had a recurrence of pancreatitis, though I have lost contact with one of them and four have died for reasons unrelated to pancreatitis.

## Conclusions

Although this series of 13 is small compared with published hospital series, it represents an incidence of approximately 295 per million per year. This is a high rate compared with other series, which suggest incidence rates of between 50 and 100 per million per year (Bourke, 1975; Imrie, 1977; McMahon, 1977). However, the true incidence of acute pancreatitis in our practice may be even higher than I have reported, as many mild cases may go undiagnosed and so produce artificially low rates. One series has reported a rate of 337 per million per year (Graham, 1977).

Any urgent request for a home visit to a case of acute upper abdominal pain should alert the doctor to the possibility of acute pancreatitis, particularly if vomiting is prominent and tenderness not as marked as one would expect. If the patient does not seem too distressed, then

a history of more severe pain some time earlier should alert one to the diagnosis. The general practitioner should be prepared to use the serum amylase test more liberally.

Some patients can be successfully managed at home. It is reasonable to consider this option if the diagnosis is confirmed, the pain is settling, any ileus is short-lived and the patient seems to be on the road to recovery. If the patient is very ill and the pain and ileus are not settling, or if there is any suspicion that any other severe intra-abdominal emergency is present, then urgent hospital admission will be necessary.

After the attack, the biliary tract should be investigated and any disease of the gallbladder eradicated.

## References

- Bourke, J. B. (1975). Variation in annual incidence of primary acute pancreatitis in Nottingham, 1969-74. *Lancet*, **2**, 967.
- Graham, D. F. (1977). Incidence and mortality of acute pancreatitis. Letter. *British Medical Journal*, **2**, 1603.
- Howes, R., Zuidema, G. D. & Cameron, J. L. (1975). Evaluation of prophylactic antibiotics in acute pancreatitis. *Journal of Surgical Research*, **18**, 197-200.
- Imrie, C. W. (1977). British Society of Gastroenterology Meeting (York).
- McMahon, M. J. (1977). Incidence and mortality of acute pancreatitis. Letter. *British Medical Journal*, **2**, 1350.
- Medical Research Council Multicentre Trial of Glucagon and Aprotinin (1977). Death from acute pancreatitis. *Lancet*, **2**, 632-635.
- Olsen, H. (1974). Pancreatitis: a prospective clinical evaluation of 100 cases and review of the literature. *American Journal of Digestive Diseases*, **19**, 1077-89.
- Opie, J. C. (1975). Acute pancreatitis—ten years' experience in the Waikata district. *New Zealand Medical Journal*, **82**, 126-129.
- Pollock, A. V. (1959). Acute pancreatitis. Analysis of 100 patients. *British Medical Journal*, **1**, 6-14.
- Ranson, J. H. C. (1979). Acute pancreatitis. *Current Problems in Surgery*, **16**, 1-84.
- Trapnell, J. E. (1966). The natural history and prognosis of acute pancreatitis. *Annals of the Royal College of Surgeons*, **38**, 265-287.
- Trapnell, J. E. (1972). The natural history and management of acute pancreatitis. *Clinics in Gastroenterology*, **1**, 147-166.

## Acknowledgements

I wish to thank my partner, Dr J. Mackie, for his valuable help in the preparation of this paper, and Mrs J. Clark for typing the manuscript.

## Address for reprints

Dr S. P. Coughlin, The Health Centre, Raikes Road, Great Eccleston, Preston, Lancs. PR3 0ZA.

---

## Sexual activity in teenage girls

In 1979 there were 33,000 unmarried teenagers who had abortions, with the rate increasing, and 24,000 live births were to unmarried teenagers—this figure is higher than in 1970. Pre-maritally conceived births were half in 1979 what they were in 1970.

Source: Speech by Sir George Young, Junior Health Minister, 27 April 1981.