Chronic Pancreatitis in the British Army 1978-1989

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SUMMARY: A retrospective study was made of all 28 soldiers with chronic pancreatitis first diagnosed between 1978 and 1989. All patients were male, alcohol was the aetiological factor in 90%, the mean age at diagnosis was 30 and the commonest mode of presentation was with recurrent painful episodes of pancreatitis. Endocrine and exocrine pancreatic insufficiency occurred in a quarter and a third of patients respectively and one third required surgical intervention.

Introduction

Chronic pancreatitis is an inflammatory process causing permanent irreversible damage to the pancreas. It is most often aetiologically linked with either excessive alcohol consumption or gallstones, but is sometimes seen in association with hyperparathyroidism (1) hyperlipidaemia (2) or congenital anatomical abnormalities, including annular pancreas (3), pancreas divisum (4), duodenal diverticulum (5) and stenosis of the sphincter of Oddi (6). Tropical chronic pancreatitis, linked to a low protein and a very low fat dietary intake and possibly linked to maternal malnutrition, occurs in non alcoholic children and young adults of both sexes in southern India, Indonesia, Zaire, the Ivory Coast and Nigeria (7). There is a strong correlation between the presence of chronic pancreatitis and the consumption of alcohol, incidence increasing linearly with the amount drunk (8). In the past chronic pancreatitis was considered a rare complication of acute pancreatitis but this sequence has been observed more often in the last decade. The prevalence of chronic pancreatitis is increasing (9) and there was a threefold increase in alcoholic chronic pancreatitis in Copenhagen between 1970 and 1979 (10). We describe a series of 28 British Army soldiers in whom chronic pancreatitis was first diagnosed between 1978 and 1989, with particular reference to aetiological factors, means of diagnosis and subsequent complications and surgical therapy.

Methods

Using appropriate ICD 9 diagnostic codes, a search for cases of chronic pancreatitis was carried out with the help of the Medical Statistics Branch of the Ministry of Defence (Med Stats MOD). Once the cases of interest were identified, copies of all relevant documentation were obtained where possible.

Results

Twenty-eight patients with chronic pancreatitis (CP) were included in the study.

Alcohol related CP was present in 25 patients, in one patient gallstones were considered the likely cause and in 2 others there was no definite aetiological factor. One patient with alcoholic CP had a peri-ampullary diverticulum and another had apparent stenosis of the sphincter of Oddi. Eighteen of these patients (72%) had either been diagnosed at a military hospital in BAOR or at a UK hospital following casevac from BAOR. There was no difference in mean age of onset between the two groups (29 + 6.5 years) and the remainder (31 + 10.7). All patients were male; their mean age at the time of diagnosis was 30 years (range: 20-43). At least one previous episode of acute pancreatitis (AP) had been documented in 25; the mean age at the time AP was first diagnosed was 27 years (range: 18-43). The mean interval between the first attack of AP and the diagnosis of CP was 3 years (range: 3 weeks-12 years). The commonest

symptom at the time CP was first diagnosed was abdominal pain (27 patients, in 24 of whom there was a history of recurrent pancreatitis); one patient presented with diabetes mellitus without a clear history of recurring pancreatitis. In addition the patient with CP caused by gallstones was jaundiced at presentation.

Diagnosis

The diagnosis of CP was made on firm clinical grounds in 3 patients. The presence of pancreatic calcification on a plain abdominal X-Ray led to the diagnosis in 3 patients. The diagnosis was made on the basis of abdominal ultrasound scanning in 3 and from CT scan appearances in one patient. CP was diagnosed at endoscopic retrograde cholangiopancreatography (ERCP) in 11 patients although the diagnosis had been suggested from the ultrasound scan in three of these. The diagnosis was made at laparotomy in 6 patients, although it had been considered likely at previous ultrasound scanning in one of these. All six laparotomies were performed before 1982. One patient had been diagnosed at a civilian hospital and it was not possible to be sure of the method of diagnosis.

Complications

Diabetes mellitus occurred in 7 patients, requiring insulin therapy in 4, oral hypoglycaemic agents in one and dietary management alone in a further patient. Diabetes was discovered at the time of diagnosis of CP in 5 of these patients and within a year of diagnosis in another. In one patient diabetes developed 10 years after the diagnosis of CP. One other patient was found to have an abnormal oral glucose tolerance test but did not require treatment. In addition, two patients treated eventually by total pancreatectomy needed insulin therapy afterwards. There was evidence of exocrine pancreatic insufficiency in 8 patients, 3 of whom had steatorrhoea, 2 excessive faecal fat losses and 3 an abnormal pancreolauryl test. One other patient had excessive faecal fat losses and an abnormal pancreolauryl test. One patient developed steatorrhoea after distal pancreatectomy and both patients treated by total pancreatectomy required pancreatic supplements.

Duodenal stenosis was present at diagnosis in one patient.

Nine patients had a past history of other illnesses attributable to alcohol abuse in 6. One patient died from adenocarcinoma of the oesophagus 11 years after the diagnosis of CP.

Of 18 patients with alcohol-related CP for whom details of alcohol consumption were available for the period after the diagnosis of CP, 10 appeared to remain abstinent and 8 continued to drink. It was not possible to determine if the outcome of those who continued to drink was worse than that of the reformed patients as 7 of the 8 who continued to drink left or were discharged from the Army within a short time of diagnosis.

Six patients with alcohol-related CP (24%) were re-

ferred to the alcohol treatment unit (ATU) after CP was diagnosed although two others had been treated there in the past. Two of those treated by the ATU after the diagnosis of CP remained abstinent, three continued to drink and no reliable details were available for the remaining patient.

Surgery

Apart from diagnostic laparotomy in 4 patients, surgical treatment was used in 9 patients, 5 of whom required more than one procedure. Pancreatogastrostomy was used in 2 (repeated in one), surgical sphincterotomy in 2, stricturoplasty in one, distal pancreatectomy in 2, a Whipple's procedure in one and total pancreatectomy in 2 patients. Cholecystectomy with a subsequent choledochojunostomy was used in one patient.

PULHEEMS Grading

Eight patients were medically discharged and 13 medically downgraded (P7: 10; P3: 3) but 7 appeared to remain P2FE, at least 3 of whom certainly left the Army in this category.

Discussion

Alcohol was the aetiological factor in 90% of the patients with CP in this series, in keeping with the general experience in other reports where 50-90% cases were attributable to alcohol (11) (12) (13). Almost three quarters of the patients with alcohol-related CP developed the disease while serving in BAOR. The mean age at presentation in males in Europe is considered to be approximately 40 years (14). A striking feature of this series is the relatively young age at diagnosis and the short interval between the first attack of AP and the development of CP. The premature morbidity involved can be appreciated by observing the incidence of endocrine (25%) and exocrine (29%) pancreatic insufficiency and the numbers requiring surgical therapy (32%).

Most patients presented with recurrent episodes of AP and all but one with abdominal pain. The policy of submitting patients with recurrent AP to ERCP examination is the likely explanation for this preponderance of painful CP.

In addition to 'acute pancreatitis' the original Marseille classification (15) recognised 'acute relapsing pancreatitis' characterised by recurrent attacks of acute inflammation with complete clinical and functional recovery between attacks and no evidence of structural damage. This classification proposed that AP was not a common cause of CP since statistical studies showed that the average age of patients at first presentation with AP (the putative cause of CP) was 13 years more than the mean age of onset of chronic calcifying pancreatitis. As this classification was devised before many of the modern pancreatic imaging techniques, when the only way of assessing pancreatic damage was with a test of exocrine

function, it is no longer ideal. The 1988 Marseille-Rome classification (16) recognised that CP is frequently complicated in the early stages of its evolution by attacks of AP responsible for recurrent pain and often the only clinical symptom. After years there is insufficiency, both exocrine and endocrine and acute attacks decrease and disappear. Most patients in this series presented with recurrent episodes of AP and all but one with abdominal pain. These painful episodes may well have indicated the presence of CP before the formal diagnosis was made. It is quite possible that some patients with painless CP have remained undiagnosed.

A definite or highly probable diagnosis of CP was made in 41% of the patients in this series either by plain abdominal X-Ray, ultrasound or CT scanning. This series was compiled over 12 years during which time marked technological advances were made and it might be reasonable to assume that diagnosis would be made more often now by a non-invasive and economical ultrasound pancreatic scan. However, ERCP resulted in most diagnoses of CP although it was less readily available at the start of this series than in subsequent years.

As exocrine pancreatic insufficiency is a late manifestation of CP, screening for CP by exocrine pancreatic function testing would not seem appropriate. In none of the patients in this series did exocrine pancreatic function testing lead to the diagnosis.

Fewer than a quarter of those patients with alcohol-related CP were referred to an alcoholic treatment unit. This may appear unsatisfactory but there was no great apparent difference between abstinence rates in those treated there and patients treated by the gastroenterologists alone. However it might be reasonable to suggest that patients who continue to drink after the diagnosis of CP be referred to an alcohol treatment unit.

It is difficult in a retrospective study to comment on the appropriateness of the PULHEEMS gradings in these patients. However only 8 were medically discharged and it seems inappropriate that 7 remained P2 FE.

REFERENCES

- HIRSKOVIC T, KEATING F R jr, GROSS J B. Coexistent pancreatitis and hyperparathyroidism. Observations in 15 cases. *Gastroenterology* 1967; **52**: 1093.
- FREDERICKSON D S, LEVY R I, LEES R S. Fat transport in lipoproteins: an integrated approach to mechanisms and disorders. *N Engl J Med* 1967; **276**: 148-156.
- DHARMSATHAPHORN K, BURRELL M, DOBBINS. Diagnosis of annular pancreas with ERCP. *Gastroenterology* 1979; **77**: 1109-1114.
- DELHAYE M, ENGELHOLM L, CREMER M. Pancreas divisum; congenital anatomic variant or anomaly? *Gastroenterology* 1985; **89**: 951-958.
- OSNES M, MYREN J, LOTVEIT T, SWENSEN T. Juxta papillary duodenal diverticula and abnormalities of endoscopic retrograde cholangiopancreatography (ERCP). *Scand J Gastroenterol* 1977; **12**: 347-351.
- TOOULI J, ROBERTS-THOMSON I C, DENT J, LEE. Sphincter of Oddi motility disorders in patients with idiopathic recurrent pancreatitis. *Br J Surg* 1985; **72**: 859-863.
- SARLES H, BERNARD J P, GULLO L. Pathogenesis of Chronic Pancreatitis. *Gut* 1990; **31**: 629-632.
- SARLES H, SAHEL J, STAUB J L, BOURRY J, LAUGIER R. Chronic pancreatitis. In: Howat H T, Sarles H, eds. *The exocrine pancreas*. London: W B Saunders, 1979; 402-439.
- JAMES O, AGNEW J E, BOUCHIER I A D. Chronic pancreatitis in England. A changing picture? *Br Med J* 1974; **ii**: 34-38.
- ANDERSEN B N, PETERSEN N T, SCHEEL J, WORNING H. Incidence of alcoholic chronic pancreatitis in Copenhagen. *Scand J Gastroenterol* 1982; **17**: 252.
- TRAVERSO L W, TOMPKINS R K, URREA F, LONGMUIR W P. Surgical treatment of chronic pancreatitis. *Ann Surg* 1979; **190**: 312-319.
- FREY C F. Clinical Review-role of subtotal pancreatic resection and pancreaticojejunostomy in chronic pancreatitis. *J Surg Res* 1981; **31**: 361-371.
- COOPER M J, WILLIAMSON R C N, BENJAMIN I S, et al. Total pancreatectomy for chronic pancreatitis. *Br J Surg* 1987; **74**: 912-915.
- LENDRUM R. Chronic Pancreatitis. In: Misiewicz J J, Pounder R E, Venables C W, eds. *Diseases of the gut and pancreas*. London: Blackwell, 1987: 511-535.
- SARLES H. Pancreatitis. Symposium in Marseille 1963. Basel: S Karger, 1965.
- SARLES H. Classification et definition des pancreatites Marseille-Rome 1988. *Gastroenterol Clin Biol* 1989; **13**: 857-859.