

Chapter 10

**Etiology and clinical profile of chronic pancreatitis –
the CMC Vellore experience**

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Summary

We describe the etiology, clinical profile and response of pain to therapy of patients with chronic pancreatitis seen at the Christian Medical College, Vellore. A total of 173 patients diagnosed to have chronic pancreatitis between 2000 and 2004 were included in this retrospective analysis. From the results of our study, we conclude that idiopathic chronic pancreatitis is the most common form of chronic pancreatitis seen in Bengal and Tamil Nadu. Cationic trypsinogen gene mutations were not seen in early and late onset chronic pancreatitis. Heterozygous SPINK 1 gene mutation was present in 55% of early onset chronic pancreatitis and 30% of late onset chronic pancreatitis. Homozygous SPINK 1 mutation was present in 14% of early onset of chronic pancreatitis.

Most patients presented in 2nd and 3rd decade of life. Duct disruptive complications were more often seen in alcohol-related chronic pancreatitis as compared to idiopathic chronic pancreatitis. Diabetes mellitus was present in 29% and steatorrhoea in about 10% of patients. About 78% of patients treated with enzyme supplements had partial or complete response of abdominal pain. For pain relief in chronic pancreatitis surgery was better than endotherapy in our experience.

Introduction

Idiopathic Chronic pancreatitis (Tropical chronic pancreatitis) was earlier thought to be seen only in certain areas of India. During the last few years, gastroenterologists have been detecting classical cases of idiopathic chronic pancreatitis from almost all states in the country. The clinical profile, complications and response to therapy may be different in different parts of India. It is therefore important that experiences from different parts of the country be recorded. This will become the baseline for planning future studies in chronic pancreatitis. We have in this paper described the etiology, clinical profile and response of pain to therapy of patients with chronic pancreatitis seen at the Christian Medical College, Vellore.

Methods

Etiology assessment

History: The etiology of chronic pancreatitis was considered alcohol related if significant alcohol (more than 40gm per day) was consumed for a period of more than 5 years. Etiology was considered to be trauma if there was significant trauma to upper abdomen that produced severe abdominal pain suggestive of pancreatitis. None of the patients studied gave history of cassava consumption. **Biochemical:** Fasting serum calcium, lipid profile and antinuclear antibodies were measured. **Genetic studies:** Cationic trypsinogen gene mutations (R22H, N29I) and SPINK 1 gene mutation (N34S) were studied in 52 patients – 22 patients less than 20 years of age (early onset) and 30 patients more than 20 years of age (late onset).

Subjects: One hundred and seventy three patients diagnosed to have chronic pancreatitis between 2000 and 2004 were included in this retrospective analysis. The majority of patients (50 to 60%) were from Bengal and Bihar while the rest were from Tamil Nadu and Kerala. Diagnosis of chronic pancreatitis was made on the basis of a typical history, together with one of the following imaging studies showing evidence of chronic pancreatitis: pancreatic calcification on plain x-ray abdomen, ultrasound abdomen, CT abdomen and ERCP.

Results

Table 1 shows the etiology of chronic pancreatitis in patients studied. Fifty five patients (32%) had alcohol related pancreatitis. The majority of patients (55%) were diagnosed to have idiopathic chronic pancreatitis. Pancreas divisum was seen in 9%, hyperlipidemia in 2%, trauma abdomen in 1% and autoimmune pancreatitis in 1% of the patients studied. Table 1 of 2 shows the age of onset of pain. None of the patients showed cationic trypsinogen gene mutations (table 3). In early chronic pancreatitis, 54.5% showed heterozygous mutation and 13.6% shows homozygous mutation of SPINK 1 gene (N34S). In late chronic pancreatitis, 30% of patients showed heterozygous mutation of SPINK 1 gene (N34S). (The rest of the clinical profile of our subjects is described in a set of tables from 4 to 9.)

Table 1: Etiology of chronic pancreatitis (n = 173)

Alcohol	55 (32%)
Pancreas Divisum	15 (9%)
Hyperlipidemia	3 (2%)
Trauma – Abdomen	2 (1%)
Autoimmune	2 (1%)
Idiopathic	96 (55%)

Table 2: Age of onset among the subjects

	Early onset (< 20y) n = 22	Late onset (> 20y) n = 30
Mean age (y)	15 ± 3.5	34.2 ± 9
Mean age of onset of pain	12.7 ± 0.9	31.4 ± 1.9

Table 3: SPINK 1 gene mutations

SPINK 1 gene mutations (N34S) in early CP 12 / 22 (54.5%) heterozygous 3 / 22 (13.6%) homozygous
SPINK 1 gene mutations (N34S) in late CP 9 / 30 (30%) heterozygous

Note: Cationic Trypsinogen gene mutations (R122H; N29I) were not present in both early and late onset idiopathic chronic pancreatitis.

Table 4: Age and gender distribution of Patients with alcohol related and idiopathic chronic pancreatic

	Age (y)	Sex (M/F)
Alcohol	39.8 (28 – 61)	55 / 0
Idiopathic	35.7 (14 – 58)	63 / 33

Table 5: Symptom profile of Patients with alcohol related and idiopathic chronic pancreatic

	Alcohol (n = 55)	Idiopathic (n = 96)
Pain	55 (100%)	87 (91%)
Vomiting	33 (60%)	42 (44%)
Weight loss	25 (45%)	45 (47%)

Table 6: Complications in subjects with chronic pancreatitis

	Alcohol n = 55	Idiopathic n = 96
Pseudocyst	15 (27%)	5 (5%)
Pancreatic ascites	5 (9%)	3 (3%)
Jaundice	1 (2%)	3 (3%)
Diabetes Mellitus	16 (29%)	28 (29%)
Steatorrhoea	7 (7%)	12 (13%)
Carcinoma pancreas	5 (9%)	6 (6%)

The frequency of diabetes was similar in both alcohol related and idiopathic chronic pancreatitis. Frequency of steatorrhoea was more in idiopathic chronic pancreatitis as compared to alcohol related pancreatitis (13% Vs 7%). Pseudocyst was the most common local complication. Duct disruptive complications like pseudocyst and pancreatic ascites were more common in alcohol related as compared

to idiopathic chronic pancreatitis (Pseudocyst: Alcohol related Vs Idiopathic chronic pancreatitis; 27% Vs 5%; Pancreatic ascites: Alcohol related Vs Idiopathic chronic pancreatitis; 9% Vs 3%). The frequency of carcinoma pancreas was similar in alcohol related and idiopathic chronic pancreatitis. Results of medical therapy, endotherapy and surgery are shown in Tables 7-9. *Medical therapy* : 85 of 173 patients were treated with pancreatic enzyme supplements for abdominal pain and followed for a minimum period of 6 months. Eighteen percent had complete relief of pain, 60% had partial relief and 22% did not respond to therapy. *Endotherapy* : 46 of 173 patients (37%) underwent endotherapy for relief of abdominal pain. Sixty seven percent had complete / partial response and 13% had no response. Twenty percent were lost to follow up. *Surgery* : 34 of 173 patients (20%) underwent surgery for abdominal pain. Twenty four of the operated patients were followed for 1 to 5 years. About 63% had complete relief of pain and 37% had partial response.

Table 7: Results of medical therapy

No response	19 / 85 (22%)
Partial response	51 / 85 (60%)
Complete response	15 / 85 (18%)

Note: 85 patients were followed up for a minimum of 6 months

Table 8: Results of endotherapy

Patients who underwent endotherapy	46 / 173 (27%)
Sphincterotomy + stent	30
Sphinc. /NPD/ESWL/Stone extraction	16
No response	6 / 46 (13%)
Partial / complete response	31 / 46 (67%)
Lost to follow up	9 / 46 (20%)

Table 9: Results of surgical therapy

Patients who underwent surgery	34 / 173 (20%)
Modified pancreatico-jejunostomy	24 / 34
Others	10 / 34
Patients followed up for 1 – 5 y (n=24)	
Partial response	9 / 24 (37%)
Complete response	15 / 24 (63%)

Conclusions

From the results of our study, we conclude that Idiopathic chronic pancreatitis is the most common form of chronic pancreatitis seen in Bengal and Tamil Nadu. Cationic trypsinogen gene mutations were not seen in early and late onset chronic pancreatitis. Heterozygous SPINK 1 gene mutation was present in 55% of early onset chronic pancreatitis and 30% of late onset chronic pancreatitis. Homozygous SPINK 1 mutation was present in 14% of early onset of chronic pancreatitis. Most patients presented in 2nd and 3rd decade of life. Duct disruptive complications were more often seen in alcohol related chronic pancreatitis as compared to idiopathic chronic pancreatitis. Diabetes mellitus was present in 29% and steatorrhoea in about 10% of patients. About 78% of patients treated with enzyme supplements had partial or complete response of abdominal pain. Surgery was better than endotherapy for pain relief in chronic pancreatitis.

