

## **Chapter 5**

### **Profile of chronic pancreatitis at the PGIMER, Chandigarh**

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## Summary

*In this article, we discuss the profile of chronic pancreatitis as seen at the Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh. Patients presenting with pain and idiopathic pancreatitis formed the commonest group studied. Pancreatic pseudocysts were the commonest complication. In this article, we also present our experience with endotherapy as well as lithotripsy in this setting.*

## Introduction

Chronic pancreatitis refers to the syndrome of a destructive, inflammatory process that leads to long standing pancreatic injury.<sup>1</sup>

The spectrum of chronic pancreatitis in India is variable. There are a number of studies on profile of chronic pancreatitis in south India.<sup>2-5</sup> The clinical spectrum of chronic calcific pancreatitis has been found to be different in northern India.<sup>6</sup> The aim of the present study was to delineate the clinical profile of patients of chronic pancreatitis at a tertiary care center in north India.

## Material and methods

This article summarizes the profile of 103 patients with chronic pancreatitis seen in the Gastroenterology–Unit I services at PGIMER, Chandigarh from June 1999 to June 2004. Patient data was recorded prospectively in a pre-designed proforma. Data included both indoor and outdoor patients. The diagnosis of chronic pancreatitis was based on clinical, biochemical and radiological investigations.

## Results

**Epidemiology:** Our patients hailed from adjoining states of Chandigarh. Many of these patients were referred for possible pancreatic endotherapy. 78/103 patients were male. Mean age of patients was  $36.71 \pm 12.94$  years (Range 7-69 years). Duration of symptoms ranged from 2 weeks to 180 months (Median = 24 months).

**Etiology:** Etiology of chronic pancreatitis is presented in Table 1. Idiopathic pancreatitis formed the commonest etiological group, followed by alcoholism. Pancreas divisum alone was seen in 17 patients and 3 had both alcoholism and pancreas divisum. Pancreatic calcification was evident in 50 /103 cases (48.5%). Twenty two out of 43 cases with idiopathic pancreatitis ( 51.1%) had pancreatic calcification.

**Clinical features:** These are presented in Table 2. Pain was the dominant symptom, present in 95% of cases. Among 5 patients without pain, 2 presented with upper gastrointestinal bleed, 1 each had symptoms of hyperglycemia, pleural effusion and dyspepsia. Overall, GI bleed occurred in 4 patients; 3 had bleeding from gastric varices and 1 had pseudoaneurysm of splenic artery. Jaundice was present in 11 patients. Palpable lump was present in 9 patients and all of them had pancreatic pseudocyst. Other clinical details are summarized in tables 3 and 4.

**Complications:** Various local and systemic complications were recorded as shown in figure 1. Pseudocyst was the commonest local complication (N=32), followed by segmental portal hypertension (11 patients).

Among diabetic patients (N=19), 14 either had symptoms of hyperglycemia, or were already on antidiabetic drugs. The remaining 5 patients were found to have abnormal Glucose Tolerance Test (GTT) or FBS. One patient of calcific pancreatitis presented with diabetic ketoacidosis after pancreatic duct stenting.<sup>7</sup> Seven patients out of 22 (31.8 %) had fecal fat > 7 g /day and 2 patient had symptom of steatorrhea. Three patients had biliary stricture and one developed carcinoma of the pancreas during follow up.

**Management:** Majority (n=78) of symptomatic patients with pain or pseudocysts, and all cases of pancreatic ascites and pleural effusion, were managed by pancreatic endotherapy. Endotherapy was technically successful in 2 out of the 78 patients. Eventually 5 of 19 (26.3%) subjects with diabetes were on diet alone and 10 (52.7%) required insulin. The remaining diabetic subjects were treated with oral hypoglycaemia drugs. Patients were referred for surgery only if there was failure to do endotherapeutic procedure or persistence of symptoms despite pancreatic endotherapy and maximal medical therapy. Overall 18 (17.4%) patients required surgical intervention.

## Discussion

It is often difficult to differentiate recurrent acute pancreatitis from exacerbations of chronic pancreatitis. Even today, in certain situations, the correct diagnosis can often be achieved only on follow up of the patient.<sup>8</sup> In all our patients the diagnosis of chronic pancreatitis was confirmed by imaging studies.

As published in an earlier study from our institute<sup>9</sup>, idiopathic pancreatitis is still the leading etiology (41.8%), followed by alcoholism (34.9%). Alcohol intake is quite common in this region of the country, and all cases of pancreatitis due to alcohol were seen in men. However 37.2 % of idiopathic pancreatitis occurred in females. Calcification was present in 51.1% of the idiopathic group and 38.8% of alcoholic patients.

Majority of patients (95.1%) had pain; however this could reflect selection bias as most patients with persistent pain were referred to our clinic. Diabetes mellitus was significantly more common in calcific pancreatitis group as compared to the non-calcific group. This may reflect that calcification develops in late stages of chronic pancreatitis associated with advanced endocrine deficiency. Patients with alcoholic pancreatitis had significantly shorter duration of symptoms as compared to idiopathic pancreatitis.

Endoscopic retrograde pancreatography followed by pancreatic endotherapy was the primary therapy in most patients. Endotherapy was done via transpapillary route in all these patients with either a pancreatic stent or nasopancreatic drain. Extra corporeal shock wave lithotripsy fragmentation of pancreatic duct calculi in conjunction with endoscopic clearance of the main pancreatic duct is associated with significant improvement in clinical outcomes in most patients with chronic pancreatitis.<sup>10</sup> Pancreatic stone lithotripsy was done in 7 of our patients. Surgery was done only in cases not responding to other means or when endotherapy could not be done.

To conclude idiopathic pancreatitis is the most common form of chronic pancreatitis seen at our center, and in general, the majority of these subjects showed a good response to endotherapy

**Table 1: Etiology of chronic pancreatitis in 103 patients**

<b>Etiology</b>	<b>N (%)</b>
Idiopathic	43 (41.8)
Alcoholism	36 (34.9)
Pancreas Divisum	17 (16.5)
Alcoholism + Pancreas divisum	3 (2.9)
Hyperparathyroidism	4 (3.9)

**Table 2: Presenting features**

<b>Clinical features</b>	<b>N (%)</b>
Pain	98 (95.1)
Diabetes	19 (18.4)
Jaundice	11 (10.7)
Lump	9 (8.7)
Diarrhoea	7 (6.8)
Vomiting	6 (6.8)
G I bleed	4 (3.9)
Hyperparathyroidism	4 (3.9)

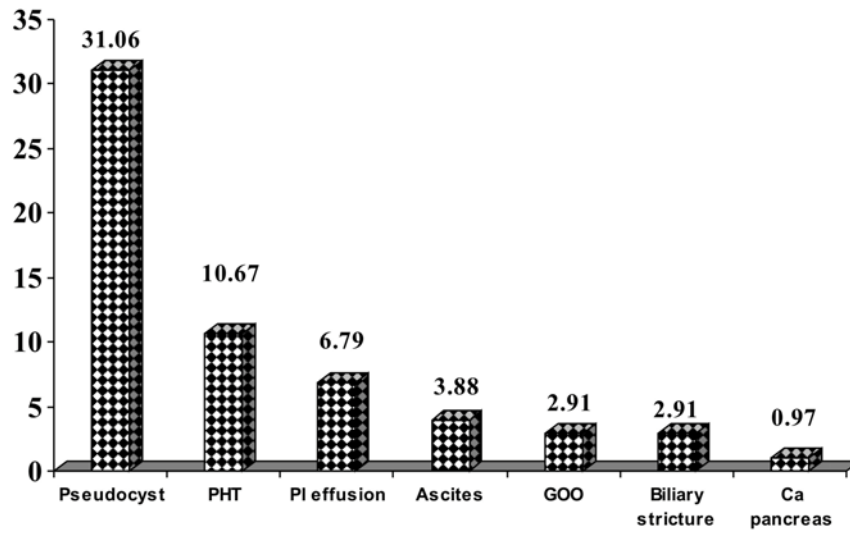
**Table 3: Comparison of calcific and non-calcific chronic pancreatitis**

	<b>Calcific (N=50)</b>	<b>Non calcific (N=53)</b>	<b>p</b>
<b>Age</b>	<b>37.0 ± 12.7</b>	<b>36.4 ± 13.2 yrs</b>	<b>NS</b>
<b>Male: Female</b>	<b>38:12</b>	<b>40: 13</b>	<b>NS</b>
<b>Duration of symptoms (months .Mean±S.D.)</b>	<b>39.07 ± 38.65</b>	<b>40.44 ± 41.45</b>	<b>NS</b>
<b>S.amylase (SU)</b>	<b>205.47 ± 53.42</b>	<b>266.25 ± 170.68</b>	<b>NS</b>
<b>Fecal fat &gt; 7 gm/d</b>	<b>6 (N=13)</b>	<b>1(N=9)</b>	<b>NS</b>
<b>Clinical features</b>			
Pain	47	51	NS
Jaundice	8	3	NS
Diarrhoea	4	3	NS
Lump	3	6	NS
Vomiting	4	2	NS
GI bleed	2	2	NS
Diabetes	12	7	< 0.05
Symptomatic	10	4	0.05
Abn GTT/FBS	2	3	NS
<b>Complications</b>			
Ascites	1	3	NS
Pl effusion	3	4	NS
PHT	5	6	NS
Pseudocyst	15	17	NS
GOO	1	2	NS
<b>Management</b>			
ERCP	36	42	NS
EndoRx	33	38	NS
Surgery	12	6	NS

**Table 4: Comparison of alcoholic and idiopathic chronic pancreatitis**

	<b>Alcoholic (N=36)</b>	<b>Idiopathic (N=43)</b>	<b>p</b>
<b>Age (years) mean+SD</b>	<b>39.3 ± 11.6</b>	<b>34.4 ± 13.3</b>	<b>NS</b>
<b>Male: Female</b>	<b>36:0</b>	<b>27: 16</b>	<b>0.0001</b>
<b>Duration of symptoms (Mean±S.D.) Months</b>	<b>22.55 ± 22.52</b>	<b>50.62±46.71</b>	<b>0.0014</b>
<b>S.amylase (SU) (Mean ± S.D.)</b>	<b>226.41±107.26</b>	<b>260.82±213.64</b>	<b>NS</b>
<b>Fecal fat &gt; 7 gm/d</b>	<b>3 (N=8)</b>	<b>3(N=12)</b>	<b>NS</b>
<b>Clinical features</b>			
Pain	33	41	NS
Jaundice	4	6	NS
Diarrhoea	3	3	NS
Lump	4	2	NS
Vomiting	2	3	NS
GI bleed	1	1	NS
Diabetes	9	8	NS
Symptomatic	5	7	NS
Abn GTT/FBS	4	1	NS
<b>Complications</b>			
Ascites	2	1	NS
Pl effusion	5	2	NS
PHT	5	4	NS
Pseudocyst	14	13	NS
GOO	1	1	NS
<b>Management</b>			
ERCP	25	33	NS
EndoRx	22	32	NS
Surgery	7	8	NS

**Fig. 1: Complications of chronic pancreatitis.**



## References

1. Etemad Bebak and Whitcomb David C. Chronic pancreatitis: diagnosis, classification, and new genetic developments. *Gastroenterology* 2001;120: 682-707.
2. Balaji LN, Tandon RK, Tandon BN, Banks PA. Prevalence and clinical features of chronic pancreatitis in southern India. *Int J Pancreatol* 1994; 15: 29-34.
3. Chari ST, Mohan V, Jayanthi V, Snehalatha C, Malathi S, Viswanathan M, Madanagopalan N. Comparative study of the clinical profiles of alcoholic chronic pancreatitis and tropical chronic pancreatitis in Tamil Nadu, south India. *Pancreas* 1992; 7: 52-8.
4. Balakrishnan V, Saunier JF, Hariharan M, Sarles H. Diet, pancreatic function, and chronic pancreatitis in south India and France. *Pancreas* 1988; 3:30-5.
5. Mori M, Hariharan M, Anandakumar M, Tsutsumi M, Ishikawa O, Konishi Y, Chellam VG, John M, Praseeda I, Priya R, Narendranathan M. A case-control study on risk factors for pancreatic diseases in Kerala, India. *Hepatogastroenterology* 1999; 46: 25-30.
6. Rai RR, Acharya SK, Nundy S, Vashisht S, Tandon RK. Chronic calcific pancreatitis: clinical profile in northern India. *Gastroenterol Jpn* 1988; 23: 195-200.
7. Bhasin DK, Sidhu RS, Bhansali A, Nagi B. Diabetic ketoacidosis in a patient with chronic calcific pancreatitis after minor papilla stent insertion. *Gastrointest Endosc* 2004; 59: 440-3.
8. Talamini G, Falconi M, Bassi C, Mastromauro M, Salvia R. Chronic Pancreatitis: Relation to Acute Pancreatitis and Cancer. *J Pancreas* 2000; 1( Suppl.): 69-76.
9. Kaushik SP, Vohra R, Verma R. Spectrum of pancreatitis at Chandigarh. A ten years experience. *Indian J Gastroenterol* 1983; 2: 9-11.
10. Kozarek RA, Brandabur JJ, Ball TJ, Gluck M, Patterson DJ, Attia F, France R, Traverso LW, Koslowski P, Gibbons RP. Clinical outcomes in patients who undergo extracorporeal shock wave lithotripsy for chronic calcific pancreatitis. *Gastrointest Endosc* 2002; 56: 496-500.