

# Acute Pancreatitis beyond Gallstones and Alcohol

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## ABSTRACT

**Background:** Acute pancreatitis (AP) is a potentially grave abdominal condition where the pancreas gets inflamed and is associated with variable regional and systemic involvement. The inflammatory state is usually characterized by acute abdominal pain with concomitant increase in serum pancreatic enzymes. Investigative workup includes serum amylase, lipase, liver function tests, triglycerides, serum calcium, and parathyroid hormone (PTH) assay; imaging includes ultrasonography, computed tomography (CT) scan, magnetic resonance cholangiopancreatography (MRCP), and/or endoscopic ultrasound in some patients.

**Aim:** To identify the etiology and frequency of nonalcoholic and nonbiliary causes of AP in a hospital scenario, and analyze the severity and outcome of the disease.

**Materials and methods:** A prospective, observational, hospital-based study was conducted on 150 consecutive AP patients in the Department of Gastroenterology at Yashoda Hospitals, Secunderabad, India. A total of 150 consenting patients who were hospitalized consecutively with AP were included, and subjects with chronic pancreatitis were excluded.

**Results:** Overall, 150 patients were included; 117 (72.9%) were men, and 37 (27.1%) women. Alcohol was the most common etiological factor noted in 54 (36%), followed by biliary tract disease 45 (30%), idiopathic 21 (14%), hypertriglyceridemia 7 (4.67%), endoscopic retrograde cholangiopancreatography (ERCP)-related 3 (2%), infection-related 5 (3%), hyperparathyroidism 3 (2%), and drug-induced 8 (5.33%). The most common presentation was abdominal pain (98.6%). Organ failure and mortality were low in the nonalcoholic/nonbiliary cause of pancreatitis.

**Conclusion:** AP in our study reports a significant number of nonalcoholic/nonbiliary causes. The current study showed that the mortality rate in nonalcoholic/nonbiliary cases was low. Despite low mortality in this group, there is a need to identify these causes and treat on an urgent basis for limiting the hospital stay.

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## INTRODUCTION

One of the most frequent causes of acute abdominal pain seen in hospitals is acute pancreatitis (AP), which can have a range of clinical manifestations and consequences, from mild to severe multiorgan failure that can be potentially fatal. The treatment of severe AP and the clinical course of AP are not simple, as there is no specific therapy and there is uncertainty of pathogenesis and outcome.<sup>1</sup> As per the revised Atlanta classification, AP is diagnosed with abdominal pain, serum lipase (or amylase) activity that is at least three times higher than the upper limit of normal, and distinctive pancreatic abnormalities on contrast-enhanced computed tomography (CECT), magnetic resonance imaging, or transabdominal ultrasonography.<sup>2</sup>

A comprehensive summary of studies on AP worldwide over the previous 56 years suggested a consistent upward trend in the incidence of AP in numerous nations.<sup>3</sup> There is a need for multiple observational studies to evaluate the disease burden and etiology of AP in India. A relatively higher incidence

of pancreatitis has been reported in the southern states of India.<sup>4</sup>

The severity levels for AP include mild (no organ failure and no local or systemic complications), moderate (transient organ failure of <48 hours' duration with or without local complications), severe and critical (infected pancreatic necrosis or persistent organ failure lasting longer than 48 hours, involving one or more organs).<sup>2</sup> The two distinct phases of AP include early phase, indicated by systemic inflammatory response syndrome (SIRS) and/or organ failure in the 1st week, while the late phase (lasting longer than 1 week) is indicated by local complications.<sup>5</sup>

The present study aims to identify the etiology, clinical features, and frequency of nonalcoholic and nonbiliary AP and analyze the severity and outcome in relation to the etiology involved.

## MATERIALS AND METHODS

### Study Design

Single-center prospective observational study.

### Sample Size

This study analyzed 150 consecutive, consenting patients of AP from the Department of Gastroenterology, Yashoda Hospitals, Secunderabad.

### Study Site

This is a single-center study conducted at a tertiary care center, Yashoda Hospitals, Secunderabad.

### Study Duration

This study was planned as a hospital-based prospective observational study and carried out from June 2021 to May 2022 in the Department of Gastroenterology, Yashoda Hospitals.

### Inclusion Criteria

The study enrolled male and female participants between 18 and 80 years of age who had a confirmed diagnosis of AP established through clinical, radiological, and biochemical evaluation. A clinical examination was performed for every patient in a routine manner. Patients were enrolled only after providing written and informed consent.

### Exclusion Criteria

The study excluded patients such as cases of recurrent AP, chronic pancreatitis, pregnant and lactating women, AP patients with malignancy, <18 years of age, and patients not consenting for the study.

### Statistical Analysis

The data of categorical values were represented by proportions, and continuous variables were represented as mean  $\pm$  standard deviation. Statistical analyses were conducted using IBM SPSS Statistics, version 29 for Windows. An alpha value of <0.05 (two-tailed) was considered statistically significant. The Chi-squared test was used to assess the

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association of categorical data (gender and age-group of AP, gender and etiology of alcoholic AP, and gender and etiology of gallstone-related AP). The linear correlation between serum amylase and serum lipase levels in AP cases was measured using Pearson correlation coefficient (*r*). One-way analysis of variance (ANOVA) was employed to compare the means of C-reactive protein (CRP), hematocrit (HCT), serum calcium, and serum parathyroid hormone (PTH) levels among mild, moderate, and severe AP.

5 (3.33%), postendoscopic retrograde cholangiopancreatography (post-ERCP) 3 (2%), and pancreatic divisum 2 (1.33%). Alcohol was the prime factor for AP in men, whereas in women, biliary tract-related conditions had a major role. Results also show that 95 cases (63.3%) did not have any noticeable comorbidities, and the remaining 55 cases reported various comorbidities, like diabetes mellitus (11), systemic hypertension (14), diabetes mellitus plus systemic hypertension (18), and other comorbidities (12), such as bronchial asthma,

coronary artery disease, rheumatoid arthritis, metabolic dysfunction-associated steatotic liver disease (MASLD), hypothyroidism, ulcerative colitis, psoriasis, and postatrial septal defect (post-ASD) closure (Table 2).

Patients' clinical presentation was mostly with abdominal pain 147 (98.6%), followed by abdominal distension 57 (38%), vomiting 48 (32%), pleural effusion 12 (8.6%), fever 42 (28%), and jaundice 5 (3.3%) (Fig. 1). As per the diagnostic criteria of the revised Atlanta classification, 129 and 21 cases were diagnosed

## RESULTS

The study comprised a total of 150 patients, of which men had a greater (74.33%) incidence of AP than women (25.7%). The age distribution included 21–80 years, with a mean age of 42.5 years. The maximum percentage of AP was reported in the group of 31–50 years of age, and the least was recorded in <21 years of age in both males and females, which denotes statistical significance ( $p = 0.342$ ) (Table 1).

Gender and etiology of alcoholic- and biliary-related AP were statistically significantly associated ( $p = 0.001$ ). Out of the 150 participants, alcohol was the most common cause of AP 54 (36%), followed by biliary disease 45 (30%), idiopathic 21 (14%), hypertriglyceridemia 7 (4.6%), hyperparathyroidism 3 (2%), drug-induced 8 (5.33%), infectious conditions

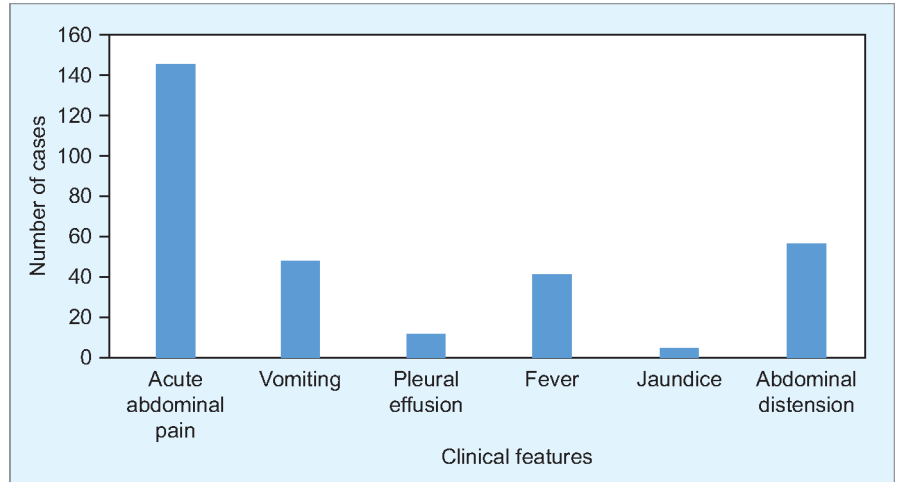


Fig. 1: Clinical features of studied patients

Table 1: Age and gender distribution of AP

Age-group (years)	Male N = 113		Female N = 37		Total	
	N	%	N	%	N	%
<21	7	6.2	1	2.7	8	5.3
21–30	16	14.2	4	10.8	20	13.3
31–40	34	30.1	8	21.6	42	28
41–50	25	22.1	6	16.2	31	20.7
51–60	17	15	10	27	27	18
61–70	8	7.1	6	16.2	14	9.3
71–80	6	5.3	2	5.4	8	5.3

%, percentage of cases; N, number of cases

Table 2: Etiological profile of AP

Etiology	Male	Female	DM	HTN	DM + HTN	Other comorbidities
	N (%)	N (%)	N	N	N	N
Alcohol	51 (34)	3 (2)	2	4	7	2
Biliary-related	15 (10)	30 (20)	4	4	4	3
Idiopathic	14 (9.33)	7 (5)	–	1	3	2
Hypertriglyceridemia	6 (4)	1 (1)	5	1	1	–
Hyperparathyroidism	1 (0.67)	2 (1)	–	–	–	–
Drug-induced	4 (2.27)	4 (3)	–	1	1	3
Infectious	3 (2)	2 (1)	–	2	1	1
Post-ERCP	2 (1.33)	1 (1)	–	–	–	–
Pancreatic divisum	1 (0.67)	1 (1)	–	–	–	1
Hereditary	2 (1.33)	0 (0)	–	1	1	–
Total	99 (66)	51 (34)	11	14	18	12

%, percentage; DM, diabetes mellitus; HTN, systemic hypertension; N, frequency of cases

**Table 3:** Clinical types of AP and modified CTSI of patients

Types of AP	Modified CT severity index		
	Mild	Moderate	Severe
Interstitial edematous pancreatitis	101	28	Nil
Necrotizing pancreatitis	Nil	6	15

**Table 4:** CRP, HCT, calcium, and PTH in AP

	CRP mg/L		HCT %		Calcium mg%		PTH pg/mL	
	N	Mean ± SD	N	Mean ± SD	N	Mean ± SD	N	Mean ± SD
Mild	101	21.87 ± 6.225	101	42.52 ± 3.654	101	8.0 ± 1.34	101	87.9 ± 98.2
Moderate	34	27.71 ± 6.891	34	44.56 ± 2.798	34	7.6 ± 0.89	34	51.4 ± 17.4
Severe	15	40.67 ± 11.690	15	51.13 ± 2.774	15	7.1 ± 0.73	15	56.0 ± 12.0
Total	150	25.07 ± 9.076	150	43.85 ± 4.250	150	7.8 ± 1.2	150	76.2 ± 82.7

as interstitial edematous pancreatitis and necrotizing pancreatitis, respectively (Table 3).

Acute pancreatitis is subgrouped as mild, moderate, and severe. It was observed that out of the 150 cases, 67.3% of the patients were categorized as mild, 22.6% moderate, and 15 (10%) severe AP. Results show that there are no statistically significant differences ( $p = 0.06$ ) of serum calcium level among the mild, moderate, and severe groups of AP. However, there is a statistically significant difference ( $p = 0.05$ ) in the mean levels of the serum PTH (lower values in more severe pancreatitis) between mild and moderate AP. Results also show that the mean difference of CRP and HCT between mild, moderate, and severe AP is significant ( $p = 0.03$ ) (Table 4). Higher values of CRP and HCT suggested a more severe disease pattern.

## DISCUSSION

Our study reports a higher incidence of AP in males. In the past, there has been evidence that suggests excessive alcohol consumption as the etiology of both acute and chronic pancreatitis.<sup>6</sup> In the current study, the next common cause was biliary tract disease. Alcohol and gallstone/biliary-related conditions have been identified as common etiological variables for AP, recurring AP, and alcohol as the cause of chronic pancreatitis in various Indian publications.<sup>7,8</sup>

Our study showed that one-fifth of the biliary-related cases were positive for biliary sludge rather than gallstones among those with biliary-related AP. In fact, AP linked to biliary sludge would be classified as idiopathic if biliary sludge is underrated. Women experience a higher incidence of gallstone-related AP, which may cause a significant increase in mortality and morbidity.<sup>9</sup> According to our research, women are more likely to experience AP associated with gallstones, while men experience alcoholic

AP. In a Chinese study, 35–65% of cases of AP occurred due to acute biliary pancreatitis (ABP), which had 5–20% mortality.<sup>10</sup>

According to a prospective observational study done in a hospital in Lucknow, India, 2.5% of cases had hypertriglyceridemia as the etiological cause of AP.<sup>11</sup> In the current study, 4.67% of AP is attributed to hypertriglyceridemia as the cause of AP. In a different study from Chennai, India, hypertriglyceridemia was not found among the 70 patients with AP.<sup>12</sup> It is important to remember that not every case of severe hypertriglyceridemia results in AP.<sup>13</sup>

The most common drug incriminated in our study was azathioprine (0.1–0.5% of cases of pancreatitis were due to drugs), and most of these cases had mild to moderate pancreatitis.<sup>14</sup> Our study showed drug-induced AP in 5.33% of cases. The majority of studies carried out in India with smaller sample sizes ( $n < 100$ ) revealed a lower incidence of drug-induced AP.<sup>15</sup>

The underlying pathogenic cause of hyperparathyroidism-induced pancreatitis is hypercalcemia, which is due to hypersecretion of PTH. Primary or secondary hyperparathyroidism (PHPT) may be linked to either acute or chronic pancreatitis.<sup>16</sup> In the present study, three cases were due to parathyroid adenoma causing hyperparathyroidism. Population studies on hyperparathyroidism showed that only 1.5% of PHPT patients experienced AP.<sup>17</sup> AP as a post-ERCP complication has been documented in 2% of our study group.

## CONCLUSION

Biliary disease and alcohol are the main etiologies of AP. However, the current study reports a significant number of nonbiliary/nonalcoholic causes. The morbidity and mortality in this group of nonbiliary/nonalcoholic causes seem to

be low. A proper identification of these causes definitely improves the outcome of patients. Various modalities to treat these patients could be insulin infusion, plasmapheresis for hypertriglyceridemia, evaluation for parathyroid adenoma, and early identification of a drug incriminated in the cause of pancreatitis. Hence, there is a need to recognize these causes of AP and treat on an emergency basis for limiting the hospital stay.

## REFERENCES

- Beger HG, Rau BM. Severe acute pancreatitis: clinical course and management. *World J Gastroenterol* 2007;13(38):5043–5051.
- Banks PA, Bollen TL, Dervenis C, et al. Classification of acute pancreatitis—2012: revision of the Atlanta classification and definitions by international consensus. *Gut* 2013;62(1):102–111.
- Iannuzzi JP, King JA, Leong JH, et al. Global incidence of acute pancreatitis is increasing over time: a systematic review and meta-analysis. *Gastroenterology* 2022;162(1):122–134.
- Devi MB, Sampoorna G, Padmalatha P, et al. Study of acute pancreatitis in a tertiary care hospital—assessment of risk factors and outcome. *Renal failure* 2019;22:15–71.
- Busquets J, Fabregat J, Pelaez N, et al. Factors influencing mortality in patients undergoing surgery for acute pancreatitis: importance of peripancreatic tissue and fluid infection. *Pancreas* 2013;42(2):285–292.
- Brock C, Nielsen LM, Lelic D, et al. Pathophysiology of chronic pancreatitis. *World J Gastroenterol* 2013;19(42):7231–7240.
- Mukherjee D, Bhakta S, Lahiry S, et al. Demographic profile of acute pancreatitis in Eastern India: a single centre experience. *Asian J Med Sci* 2017;8(6):24–29.
- Nesvaderani M, Eslick GD, Vagg D, et al. Epidemiology, aetiology and outcomes of acute pancreatitis: a retrospective cohort study. *Int J Surg* 2015;23:68–74.
- Guo X, Li Y, Lin H, et al. A nomogram for clinical estimation of acute biliary pancreatitis risk among patients with symptomatic gallstones: a retrospective case-control study. *Front Cell Infect Microbiol* 2022;12:935927.
- Drake M, Dodwad SJM, Davis J, et al. Sex-related differences of acute and chronic pancreatitis in adults. *J Clin Med* 2021;10(2):300.
- Patel ML, Shyam R, Atam V, et al. Clinical profile, etiology, and outcome of acute pancreatitis: experience at a tertiary care center. *Ann Afr Med* 2022;21(2):118–123.

12. Subramaniam R, Pothapragada VR. A clinical study on epidemiology and etiological factors of acute pancreatitis-hospital based study in South India. *Int Surg J* 2021;8(10):3060–3063.
13. Valdivielso P, Ramirez-Bueno A, Ewald N. Current knowledge of hypertriglyceridemic pancreatitis. *Eur J Intern Med* 2014;25(8):689–894.
14. Jones MR, Hall OM, Kaye AM, et al. Drug-induced acute pancreatitis: a review. *Ochsner J* 2015;15(1):45–51.
15. Alkareemy EAR, Ahmed LAW, El-Masry MA, et al. Etiology, clinical characteristics, and outcomes of acute pancreatitis in patients at Assiut University Hospital. *Egypt J Intern Med* 2020;32:1–6.
16. Sunkara T, Caughey ME, Rawla P, et al. Severe acute pancreatitis as an index clinical manifestation of parathyroid adenoma. *Cureus* 2018;10(4):e2445.
17. Khoo TK, Vege SS, Abu-Lebdeh HS, et al. Acute pancreatitis in primary hyperparathyroidism: a population-based study. *J Clin Endocrinol Metab* 2009;94(6):2115–2118.