Annals of Medicine and Medical Sciences (AMMS)
Volume 04, 2025, *Page No.: 771 to 778*
Available at: *http://ammspub.com*

Original Article

OPEN ACCESS

Demographic and Clinico-Pathological Profile of Patients with Acute Pancreatitis in a Tertiary Referral Hospital in Eastern India

Pankaj Kumar, AK Maurya*, Ashish Singh, VP Yadav

Department of General Surgery GMC and Super Facility Hospital, Azamgarh, Uttar Pradesh India.

*Corresponding author: Dr. AK Maurya; *dr.akhilbrd@gmail.com*

Abstract

Background: Acute pancreatitis (AP) is an inflammatory condition of the pancreas that is painful and potentially lethal disease. It was 1889 when Fitz first described pancreatitis as a nosdogic disease. In 1901, Opie described the association of gallstones to AP in his common channel hypothesis. ***Methods:*** 100 cases were included in this prospective observational study over a period of 18 months. All patients over 12 years with acute pancreatitis and recurrent cases of acute pancreatitis were included in this study. ***Result:*** Gallstone induced acute pancreatitis was the commonest etiological type (34%) followed by alcohol (24%). In female gallstone induced pancreatitis was more common (56.3%), while alcohol as a causative agent was seen exclusively in males. In children pancreatic divisum and trauma was the cause of acute pancreatitis. Peripancreatic fluid collection was both the commonest imaging finding and local complication noted and was seen in a significantly higher number of cases in either group. ***Conclusion:*** Acute pancreatitis has a wide range of etiology and age group. Gall stone is most common overall followed by alcohol and trauma. Mild cases are usually successfully treated conservatively while severe cases need admission and interventions.

Keywords: ***Acute pancreatitis, pancreatic trauma, pancreas.***

Introduction

It was 1889 when Fitz first described pancreatitis as a nosdogic disease [1]. In 1901, Opie described the association of gallstones to AP in his common channel hypothesis [1]. Clinico-pathologic classification of pancreatitis came in 1963 [1].

Acute pancreatitis (AP) is an inflammatory condition of the pancreas that is painful and potentially lethal disease with wide variation in severity ranging from mild and self-limiting to a rapidly progressing illness leading to multi organ failure. At one end of the spectrum is the mild variety of acute pancreatitis, which invariably results in 'restitutio ad integrum 'or spontaneous resolution of symptoms and requires supportive therapy only [2]. At the other end is the Severe variety which requires aggressive resuscitative and occasionally surgical intervention. In accordance with this wide variation in clinical presentation the treatment of acute pancreatitis requires a multi-disciplinary approach.

Severe acute pancreatitis affecting 10- 15 % of the cases is however associated with severe complication and death. The optimal management of Acute Pancreatitis includes accurate early prediction of the disease severity.

Though the literatures on incidence and prevalence studies of AP are not exhaustive there appears to be much variability between countries and even different regions within one country. Such differences have also been documented in regards to the aetiology and clinico - pathological profile of patients with AP. Some studies have found a definite increase in the number of patients with the diagnosis of AP, this may be attributed either to increased use of newer diagnostic tools or higher alcohol consumption.3 While alcoholic pancreatitis has been found to be the most common form of AP in the western [3].

The true incidence and prevalence of AP in India is not known as is remains an under–diagnosed disease. The data regarding demographic and clinic-pathological profile of patients with acute pancreatitis from eastern India are few, though we find that admission in our surgical wards.

In this prospective study we have sought to document the epidemiological and clinico-pathological profile of patients with acute pancreatitis attending this referral government teaching hospital in eastern India.

Methods

This is a prospective observational study which will be done over one and half years. During this study period all patients admitted in General surgery wards of IPGME&R with the diagnosis of Acute pancreatitis were included.

1. Study area- Department of General surgery, IPGME&R and SSKM hospital Kolkata.
2. Study population- Patients admitted in general surgery wards of IPGME&R and SSKM hospital with diagnosis of Acute pancreatitis.

Received: June 25, 2025; **Revised:** July 23, 2025; **Accepted:** July 28, 2025

Inclusion Criteria

I. Patients admitted in General surgery wards, diagnosed as acute pancreatitis.
II. Patients with a history of acute pancreatitis, presenting with recurrence (not in the background of chronic pancreatitis) were also included.
III. Age of the patients more than 12 years.

Exclusion Criteria

I. Patients below the age of 12 years.
II. Patients not willing take part in the study.
III. Acute episode in patients with chronic pancreatitis

3. Study period-one and half year
4. Sample size-100 cases
5. Study design-prospective observational study
6. Parameters to be studied

- Demographic profile and known risk factors -
  i. Age
  ii. Sex
  iii. Gall stone disease
  iv. Alcohol intake
  v. Metabolic- Hypertriglyceridemia, Hypercalcemia
  vi. History of trauma- post ERCP, blunt abdominal trauma
  vii. Obstruction- Cystic neoplasm of pancreas, pancreatic divisum
  viii. Viral infection- mumps, Hepatitis-B
  ix. Drug intake- furosemide, estrogen, azathioprine etc.

- Complications:

  Systemic

  1. ARDS
  2. Renal failure
  3. GI haemorrhage
  4. DIC
  5. SIRS

  Metabolic

  1. Hypocalcaemia
  2. Hyperglycaemia
  3. Hyper-triglyceridemia

  Local

  1. Acute fluid collection
  2. Pancreatic necrosis
  3. WON
  4. Splenic artery pseudoaneurysm

- Biochemical parameters
  i. WBC
  ii. Serum LDH
  iii. Serum AST
  iv. Serum ALT
  v. Serum creatinine
  vi. PaO2/Fio2

- Imaging findings
  i. Pancreatic edema
  ii. Peripancreatic acute fluid collection
  iii. Pancreatic necrosis
  iv. Pancreatic abscess
  v. Pancreatic pseudocyst
  vi. Splenic artery pseudoaneurysm.

- STUDY TOOLS
  i. Questionnaires.
  ii. Clinical examination.
  iii. Biochemical parameters.
  iv. Imaging finding.

Results and Analysis

Statistical Analysis

Statistical Analysis was performed with help of Epi Info (TM) 3.5.3. EPI INFO is a trademark of the Centers for Disease Control and Prevention (CDC). Using this software, basic cross-tabulation, inferences and associations were performed. Means along with the standard deviations were calculated under descriptive analysis.

Test was used to test the association of different study variables with the study groups. In the cases where one of the cell frequencies were less than 5 corrected Chi-square (χ^2) was used to find the association between variables Z-test (Standard Normal Deviate) was used to test the significant difference between two proportions. t-test was used to compare the means. Odds ratio (OR) with 96% confidence intervals was calculated to find the risk factors. $p < 0.05$ was considered statistically significant.

According to table no. I the mean age (mean ± s.d.) of the patients was 34.04±12.82 years with range 13 -67 years and the median age was 30.0 years.

Test of proportion showed that the proportion of the patients in the age group 20 -39 years (59.0%) were significantly higher than other age group (Z= 5.02; p<0.0001). Thus, the acute pancreatitis prevalence was most prevalent in the age group 20-39 years. Only 4.0% of the patients were in the age group 60-67 years. In table no. II the ratio of male and female (Male: Female) was 2.2: 1.0. Proportion of male (69.0%) was significantly higher than the females (31.0%) (Z=5.37;<,0.001) in our patients' cohort.

In Table no. III corrected Chi-square (χ^2) test showed that there was no significant association between age groups and gender of the patients (p=0.22). Thus, the males and females were more or less equally distributed over age. However, females were more prone to have acute pancreatitis at younger age than females. However, t-test showed that there was no significant difference between mean age of males and females (t98=1. 39; p=0.16).

According to table IV & V GSIAP (34.0%) was found to be the commonest etiological factor in our series and its incidence was significantly higher than other risk factors (Z=4.16; p<0.001). GSAIP was followed by ALC (24%) as the second most common cause of AP in our patient population. Only 6.0% of the patients had no cause attributable to AP (idiopathic).

In table VI corrected Chi-square (χ^2) test showed that there was no significant association between age and status of GSIAP of the patients (p=0.72). The risk of GSIAP was 1.15 times more among the patients with age ≥35 years as compared to the patients with age<35 years but the risk was not significant [OR-1.15 (0.52, 2.54); p=0.72].

In table VII corrected Chi-square (χ^2) test showed that there was significant association between gender and GSIAP of the patients (p<0.00001). The risk of GSIAP was 15.42 times more among the female patients as compared to the male patients and the risk was significant [OR-15.42 (4.79, 49.64); p<0.00001].

According to Table VIII Alcohol as an aetiology of acute pancreatitis was seen exclusively in males (p<0.001)

Table IX describes most of the patients had PPAFC (72.0%) followed by PN (36.0%) WON (31.0%) which were significantly higher than other complications (Z=3.80; p<0.001). Only 5.0% of the patients had PA.

According to Table X as per imaging findings most of the patients had PPAFC (72.0%) followed by PE (60.0%), AS (41.0%) and PEF (38.0%) which were significantly higher than other findings (Z=7.52; p<0.0001). Only 5.0% of the patients had PA.

According to Table XI as per findings of biochemical markers most of the patients had abnormal amylase (96.0%) followed by Lipase (92.0%), WBC (74.0%) and AST (41.0%) which were significantly higher than other findings (Z=8.58; p<0.0001). Only 24.0% of the patients had abnormal LDH.

Table I: Distribution of Age

Age (Years)	Number	%
13 - 19	13	13.00%
20 - 39	59	59.00%
40 - 59	24	24.00%
60 - 67	4	4.00%
Total	100	100.00%
Mean ± s.d	34.04±12.82	
Median	30	
Range	13 -67	

Table II: Gender distribution

Gender	Number	%
Male	69	69.0%
Female	31	31.0%
Total	100	100.0%
Male: Female	2.2:1.0	

Table III: Age and Gender distribution

Age Group (in years)	Gender		TOTAL
	Male (n=69)	Female (n=31)	
13 – 19	11	2	13
Row%	84.62%	15.38%	100.00%
Col%	15.94%	6.45%	13.00%
20 – 39	37	22	59
Row%	62.71%	37.29%	100.00%
Col%	53.62%	70.97%	59.00%
40 – 59	17	7	24
Row%	70.83%	29.17%	100.00%
Col%	24.64%	22.58%	24.00%
60 – 67	4	0	4
Row%	100.00%	0.00%	100.00%
Col%	5.80%	0.00%	4.00%
TOTAL	69	31	100
Row%	69.00%	31.00%	100.00%
Col%	100.00%	100.00%	100.00%
Mean ± s.d	35.04±14.20	31.80±8.81	
Median	30	30	
Range	13 - 67	13 - 48	

χ^2 =4.40; p=0.22 NS- Not Significant

Table IV: Distribution of common risk factors associated with acute pancreatitis

Risk Factors	Number (n=100)	%
GSIAP	34	34.00%
ALC	24	24.00%
ERCP	11	11.00%
HTG	8	8.00%
BLTAB	7	7.00%
IDIOP	6	6.00%
OTHERS	10	10.00%
Total	100	100.00%

Table V: Association aetiology and gender of the patients

Risk Factors	Male		Female		p-value
	Number	%	Number	%	
GSIAP	21	43.8%	27	56.3%	<0.001*
ALC	36	100.0%	0	0.0%	<0.001*
HTG	8	72.7%	3	27.3%	<0.001*
ERCP	7	63.6%	4	36.4%	<0.001*
BLTAB	7	100.0%	0	0.0%	<0.001*
OBST	5	71.4%	2	28.6%	<0.001*
IDIOP	7	100.0%	0	0.0%	<0.001*
HCAL	0	0.0%	3	100.0%	<0.001*
OTHERS	2	66.7%	1	33.3%	<0.001*

Table VI: Association between age and GSIAP

Age Group (in years)	GSIAP		Total
	Present	Absent	
≥35	22	22	44
Row%	50.00%	50.00%	100.00%
Col%	45.83%	42.31%	44.00%
<35	26	30	56
Row%	46.43%	53.57%	100.00%
Col%	54.17%	57.69%	56.00%
TOTAL	48	52	100
Row%	48.00%	52.00%	100.00%
Col%	100.00%	100.00%	100.00%

χ^2 =0.12; p=0.72 NS-Not Significant

Table VII: Association between gender and GSIAP

Gender	GSIAP		Total
	Present	Absent	
Female	27	4	31
Row%	87.10%	12.90%	100.00%
Col%	56.25%	7.69%	31.00%
Male	21	48	69
Row%	30.43%	69.57%	100.00%
Col%	43.75%	92.31%	69.00%
TOTAL	48	52	100
Row%	48.00%	52.00%	100.00%
Col%	100.00%	100.00%	100.00%

χ^2 =27.51; p<0.00001 S-Significant

Table VIII: Association between gender and ALC

Gender	Alcohol	%
Female	0	0.0%
Male	36	100.0%
TOTAL	36	100.0%

Table IX: Distribution of local complications associated with acute pancreatitis

Type of local complications	Number (n=100)	%
PPAFC	72	72.0
PN	36	36.0
WON	31	31.0
PP	19	19.0
SAPA	8	8.0
PA	5	5.0

Table X: Distribution of imaging findings through CECT

Imaging findings	Number (n=100)	%
PPAFC	72	72.0
PE	60	60.0
AS	41	41.0

PEF	38	38.0
PN	36	36.0
WON	31	31.0
PP	19	19.0
SAPA	8	8.0
PA	5	5.0

Table XI: Distribution of findings of biochemical markers

Findings of Biochemical markers	Number (n=100)	%
AMYLASE	96	96.0
LIPASE	92	92.0
WBC	74	74.0
AST	41	41.0
ALT	40	40.0
PAO_2/FIO_2	40	40.0
CREATININE	26	26.0
LDH	24	24.0

Discussion

Acute pancreatitis is an acute inflammatory condition of the pancreas, peripancreatic tissue and/ or remote organ systems. It ranges in severity from mild to severe. Though 80% of the cases run a mild course it is potentially a lethal disease. Globally acute pancreatitis is showing a rising annual incidence and is an increasing burden on the constrained healthcare resources [4]. Gallstones and alcoholism are the most common causes worldwide [5]. The increasing incidence of acute pancreatitis worldwide can be attributable to various factors like increase in the incidence of GSD, increasing alcohol consumption, availability of better diagnostic tools and increasing use of ERCP over the last few decades [6].

This study was conducted in I.P.G.M.E & R / S.S.K.M Hospital, Kolkata over one and half year period. This prospective observational non-randomised study included 100 diagnosed cases of acute pancreatitis admitted mostly in Department of Gastroenterology. The aim of study was to assess the demographic profile of patients presenting with acute pancreatitis and its aetiology.

Historical, biochemical and radiological factors were taken into considerations while arriving at the diagnosis of acute pancreatitis. The diagnosis was made when two of the following three features were present:

1. Acute onset “stabbing” or “knife-like” epigastric pain of varying severity with radiation towards the back or the flanks.
2. Serum amylase or lipase at least three times the upper limit of normal.
3. Characteristic imaging findings on trans-abdominal USG / Endo-USG or CECT abdomen.

Similarly, the aetiology factors responsible for acute pancreatitis were found out from history, biochemical alterations and suggestive imaging findings. In individuals who had more than one known etiological factors present, only the factor which was in all probability primarily responsible for precipitation of acute pancreatitis was considered for statistical analysis. For example, alcohol is said to be the causative factor if the patient had consumed alcohol shortly before the symptoms started and other possible causes of AP have been excluded.

The majority of the patients in our study were in the age group of 21 - 30 years (59%) and this proportion was found to be statistically significant (Z= 5.02; p<0.0001). The mean age of our patients, irrespective of their gender, was 34.04±12.82 years (range= 13 -67 years). Only 4% of the patients were in the >60 years age bracket. More than twice as many males were affected as females. The M: F ratio was 2.2:1 and our patient cohort had a significant male preponderance (Z=5.37; p< 0.001). Thus, males were at higher risk of having acute pancreatitis than females.

Almost similar figures as ours with regards to age group affected were quoted by Lankisch et al.[7] In his study on 602 patients he found 31-40 years to be the most common age group affected by acute pancreatitis. Our findings are similar to that of Gullo et al whose study on 1,068 patients from five European countries also showed male preponderance (64.8%) [8].

There has been no population-based study in India regarding etiology of pancreatitis, but recent small single-centre based studies showed that alcohol is rising as an etiology of acute pancreatitis [9]. Though regional and demographic variation exists, older studies found gallstones to be the predominant cause. Our study also revealed gallstone induced pancreatitis to be the most common cause of acute pancreatitis. It was seen in 34% (n=34) of our patient cohort. This was followed by alcohol in 24% (n=24) as the second most common cause. Collectively these two factors accounted for 58% of the cases. The proportion of patient with GSIAP was significantly higher than other risk factors (Z=4.16; p<0.001). This preponderance of gallstone disease over alcohol as the commonest aetiological factor of acute pancreatitis was also reported Garg et al from AIIMS, Bhat et al from SGPGI and Zargar et al from Srinagar, Kashmir (they also found biliary ascariasis in 22% cases of acute pancreatitis) [10-12]. Similar figure has also been quoted by Jha et al, Nagarjuna et al also from India [13,14]. Some western studies like those Gomez et al in their study on 151 patient and UoMo G et al also found gallstones as the commonest cause of acute pancreatitis, 77.4% and 69.3% respectively [15,16].

We found GSIAP to be 1.15 times more among the patients with age ≥35 years as compared to the patients with age<35 years though the risk was not significant [OR-1.15 (0.52, 2.54); p=0.72]. Bhatia et al also found biliary pancreatitis to be more common amongst older patients [17].

GSIAP was found to be more common in females (56.3%) in our study as because gallstone disease occurs with a higher frequency in females. There was significant association between females’ gender and GSIAP (p<0.001) in our study. we found the risk GSIAP was 15.42 times more among the female patients as compared to the male patients. Only other factor which was significantly higher in females was hypercalcemia (p<0.001).

In males, alcohol was the most common cause (n=36) while gall stone induced pancreatitis was the next (n=21). Alcohol as a causative factor was seen exclusively in males in our series. Pelli et al in their study found alcohol as a cause of acute pancreatitis to be more common in younger patients [18].

In children (<15years), pancreatic divisum and trauma were noted to be the cause of acute pancreatitis in our study. Graham et al and Werlin et al have stated trauma and systemic disease as the most common aetiologies of acute pancreatitis in children in their studies[19,20].

Hypertriglyceridemia (8%), ERCP (11%), blunt trauma abdomen (7%), idiopathic (6%) and 10% "Others" (Pancreatic divisum, cystic neoplasm of pancreas, drug induced, hypercalcemia and viral infection) were the distribution of the rest of the etiological factors amongst our patient's cohort.

For correctly diagnosing the local complications the following standardized definitions were considered [21]

- **Peripancreatic fluid collection:** Fluid that extravasates out of the pancreas during AP into the anterior pararenal spaces and other surrounding areas. It may be associated with pancreatic necrosis as well.
- **Pancreatic necrosis:** diffuse or focal areas of non-viable pancreatic parenchyma which appears as well marginated zones of non-enhanced areas on CECT involving >30% of the pancreas. It may be infected (demonstration of bacteria or fungi in the necrotic tissue) or non-infected.
- **Pancreatic pseudocyst:** collection of clear pancreatic juice without any solid material enclosed by non-epithelialized wall of reactive fibrosis which is usually seen after 4 weeks of onset of AP.
- **Walled-off necrosis:** Necrotic pancreatic or peri-pancreatic tissue surrounded by an enhancing wall (on CECT) of granulation and fibrous tissue, usually identified after 4 weeks of onset of AP.
- **Pancreatic abscess:** it is an infected pancreatic pseudocyst.

We only assessed the local complications in this study and not the systemic complications of AP including organ failure. Peri-pancreatic fluid collection was the most common imaging finding seen in 72 (72%) patients of acute pancreatitis followed by pancreatic edema in 60 (60%) patients and in 41% patients with ascites. The least common imaging finding was pancreatic abscess seen only (5%). The proportion of peri-pancreatic fluid collection as an imaging finding was significantly higher than other findings ($Z=7.52$; $p<0.0001$).

Literatures quote the incidence of peri-pancreatic fluid collection to be around 40-50%, somewhat lower than our figure of 72% [22,23].

There is certain limitation inherent to this study like small sample size and a limited catchment area of the patients enrolled in the study. This study may be further enriched by multi-centric study with a large sample size drawn from wider geographical areas and also by including some other variables like systemic complications and severity assessment.

Summary & Conclusion

This prospective observational non-randomised study, conducted over a period of 18 months included 100 diagnosed cases of acute pancreatitis admitted mostly in Department of Gastroenterology.

The aim of the study was to assess the Demographic and clinico-pathological profile of patients with acute pancreatitis.

The following were our observations at the end of the study period.

1. The mean age of our patients, irrespective of their gender, was 34.04±12.82 years.
2. It was 2.2 times more common in male than female.
3. Gallstone induced acute pancreatitis was the commonest etiological type (34%) followed by alcohol (24%).
4. In female gallstone induced pancreatitis was more common (56.3%), while alcohol as a causative agent was seen exclusively in males.
5. In children pancreatic divisum and trauma was the cause of acute pancreatitis in our study.
6. Peripancreatic fluid collection was both the commonest imaging finding and local complication noted in our study and was seen in a significantly higher number of cases in either group.

Abbreviations

AP: Acute Pancreatitis
GSIAP: Gallstone Induced Acute Pancreatitis
ALC: Alcohol
HTG: Hypertriglyceridaemia
HCAL: Hypercalcaemia
ERCP: Endoscopic Retrograde Cholangio-Pancreaticography
BLNT ABD: Blunt Abdomen
OBST: Obstruction
PD: Pancreatic Divisum
IDIOP: Idiopathic
OTHRS: Others
RF: Renal Failure
GIH: Gastr Ointestinal Haemorrhage
DIC: Disseminated Intravascular Coagulation
SIRS: Systemic Inflammatory Response Syndrome
ARDS: Acute Respiratory Distress Syndrome
AFC: Acute Fluid Collection
PN: Pancreatic Necrosis
AA: Ascites
PA: Pancreatic Abscess
PEF: Pleural Effusion
SAPA: Splenic Artery Pseudoaneurysm
PE: Panceatic Edema
PPAFC: Peripancreatic Acute Fluid Collection
PP: Pancreatic Pseudocyst
WON: World Of Necrosis
WBC: White Blood Count
Sr. LDH: Serum Lactate Dehydrogenase
Sr. AST: Serum Aspartate Transaminase
Sr. ALT: Serum Alanine Transaminase
CREAT: Creatinine

Declarations

Conflict of interest

Authors declares no conflicts of interest.

Funding/ financial support

No funding sources.

Ethical Clearance

The study was approved by the Institutional Ethics Committee.

References

[1] Chávez Rossell M. Historia del páncreas y de la evolución en los conceptos y clasificación de pancreatitis [History of the pancreas and the evolution of concepts and classification of pancreatitis]. Rev Gastroenterol Peru. 2002 Jul-Sep;22(3):243-7. Spanish. PMID: 12378219.

[2] Purschke B, Bolm L, Meyer MN, Sato H. Interventional strategies in infected necrotizing pancreatitis: Indications, timing, and outcomes. World J Gastroenterol. 2022 Jul 21;28(27):3383-3397. doi: 10.3748/wjg.v28.i27.3383. PMID: 36158258; PMCID: PMC9346450.

[3] Hastier, Patrick MD; Buckley, Martin J M MRCPI; Peten, Emmanuel P MD; Demuth, Nicolas MD; Dumas, Remy MD; Demarquay, Jean-Francois MD; Caroli-Bosc, Fancois-Xavier MD; Delmont, Jean-Pierre MD. A New Source of Drug-Induced Acute Pancreatitis: Codeine. American Journal of Gastroenterology 95(11):p 3295-3298, November 2000. | DOI: 10.1111/j.1572-0241.2000.03213.x

[4] Spanier BW, Dijkgraaf MG, Bruno MJ. Epidemiology, aetiology and outcome of acute and chronic pancreatitis: An update. Best Pract Res Clin Gastroenterol. 2008;22(1):45-63. doi: 10.1016/j.bpg.2007.10.007. PMID: 18206812.

[5] Birgisson H, Möller PH, Birgisson S, Thoroddsen A, Asgeirsson KS, Sigurjónsson SV, Magnússon J. Acute pancreatitis: a prospective study of its incidence, aetiology, severity, and mortality in Iceland. Eur J Surg. 2002;168(5):278-82. doi: 10.1002/ejs.46. PMID: 12375609.

[6] Lindkvist B, Appelros S, Manjer J, Borgström A. Trends in incidence of acute pancreatitis in a Swedish population: is there really an increase? Clin Gastroenterol Hepatol. 2004 Sep;2(9):831-7. doi: 10.1016/s1542-3565(04)00355-6. PMID: 15354285.

[7] Lankisch, Paul Georg; Burchard-Reckert, Sabine; Petersen, Mechthild; Lehnick, Dirk*; Schirren, Carl Albrecht†; Stöckmann, Fritz†; Köhler, Heinrich‡. Etiology and Age Have Only a Limited Influence on the Course of Acute Pancreatitis. Pancreas 13(4):p 344-349, November 1996.

[8] Gullo L, Migliori M, Oláh A, Farkas G, Levy P, Arvanitakis C, Lankisch P, Beger H. Acute pancreatitis in five European countries: etiology and mortality. Pancreas. 2002 Apr;24(3):223-7. doi: 10.1097/00006676-200204000-00003. PMID: 11893928.

[9] Thakur S, Kaur R, Bhatia L, Bansal R, Singh A, Singh J. Acute Pancreatitis: Clinical Profile of 60 Patients. Cureus. 2023 Dec 28;15(12):e51234. doi: 10.7759/cureus.51234. PMID: 38288210; PMCID: PMC10823209.

[10] Garg PK, Madan K, Pande GK, Khanna S, Sathyanarayan G, Bohidar NP, Tandon RK. Association of extent and infection of pancreatic necrosis with organ failure and death in acute necrotizing pancreatitis. Clin Gastroenterol Hepatol. 2005 Feb;3(2):159-66. doi: 10.1016/s1542-3565(04)00665-2. PMID: 15704050.

[11] Bhat G, Singh D, Chaudhuri G. Profile of acute pancreatitis at tertiary centre. Indian J Gastroenterol 2007; 25(Suppl 2): A1 13.

[12] Zargar SA, Gupta A, Javid G, et al. Clinical profile of acute pancreatitis in 648 cases: A hospital based study. Indian J Gastroenterol 2007; 26(Suppl 2): A106.

[13] Jha PK, Chandran R, Jaiswal P, Seema K. A clinical study of risk factors of acute pancreatitis in a tertiary care centre in north India. Int Surg J [Internet]. 2017 May 24 [cited 2025 Jun. 15];4(6):1878-83.

[14] Nagarjuna TR, H. L. PHL. The outcome of management of acute pancreatitis. Int J Res Med Sci [Internet]. 2017 Jan. 3 [cited 2025 Jun. 15];4(7):2998-3001.

[15] Gomez D, Addison A, De Rosa A, Brooks A, Cameron IC. Retrospective study of patients with acute pancreatitis: is serum amylase still required? BMJ Open. 2012 Sep 21;2(5):e001471. doi: 10.1136/bmjopen-2012-001471. PMID: 23002153; PMCID: PMC3467606.

[16] Uomo G, Pezzilli R, Gabbrielli A, Castoldi L, Zerbi A, Frulloni L, et al. Members of the ProInf-AISP study group. Diagnostic assessment and outcome of acute pancreatitis in Italy: Results of a prospective multicentre study: ProInf-AISP: Progetto informatizzato pancreatite acuta, Associazione Italiana Studio Pancreas, phase II. Digest Liver Dis. 2007;39(9):829-37.

[17] Bhatia M, Wong FL, Cao Y, Lau HY, Huang J, Puneet P, Chevali L. Pathophysiology of acute pancreatitis. Pancreatology. 2005;5(2-3):132-44. doi: 10.1159/000085265. Epub 2005 Apr 21. PMID: 15849484.

[18] Pelli H, Lappalainen-Lehto R, Piironen A, Sand J, Nordback I. Risk factors for recurrent acute alcohol-associated pancreatitis: a prospective analysis. Scand J Gastroenterol. 2008;43(5):614-21. doi: 10.1080/00365520701843027. PMID: 18415757.

[19] Graham JM, Mattox KL, Jordan GL Jr. Traumatic injuries of the pancreas. Am J Surg. 1978 Dec;136(6):744-8. doi: 10.1016/0002-9610(78)90349-5. PMID: 717659.

[20] Giefer MJ, Lowe ME, Werlin SL, Zimmerman B, Wilschanski M, Troendle D, Schwarzenberg SJ, Pohl JF, Palermo J, Ooi CY, Morinville VD, Lin TK, Husain SZ, Himes R, Heyman MB, Gonska T, Gariepy CE, Freedman SD, Fishman DS, Bellin MD, Barth B, Abu-El-Haija M, Uc A. Early-Onset Acute Recurrent and Chronic Pancreatitis Is Associated with PRSS1 or CTRC Gene Mutations. J Pediatr. 2017 Jul; 186:95-100. doi: 10.1016/j.jpeds.2017.03.063. Epub 2017 May 10. Erratum in: J Pediatr. 2018 Dec; 203:468-469. doi: 10.1016/j.jpeds.2018.08.026. PMID: 28502372; PMCID: PMC5506853.

[21] 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;62:102-111.

[22] Gore Mr, Levive SM. Textbook of Gastroenterological Radiology 3rd ed, Saunders 2008.

[23] Balthazar EJ. Complications of acute pancreatitis: clinical and CT evaluation. Radiol Clin North Am. 2002 Dec;40(6):1211-27. doi: 10.1016/s0033-8389(02)00043-x. PMID: 12479707.