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COMPARATIVE CLINICAL OUTCOMES OF ALCOHOL-INDUCED AND GALLSTONE-INDUCED ACUTE PANCREATITIS: A PROSPECTIVE OBSERVATIONAL STUDY

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Abstract

Background: Acute pancreatitis (AP) is a common gastrointestinal emergency with alcohol consumption and gallstone disease being the leading causes. Differences in clinical course, complications, and outcomes between these etiologies remain clinically relevant.

Aim: To compare the clinical outcomes of alcohol-induced and gallstone-induced acute pancreatitis.

Methods: This prospective observational study was conducted at RKDF Medical College, Bhopal, India, over 18 months. A total of 152 patients diagnosed with acute pancreatitis were enrolled and divided into two groups: alcohol-induced (n = 101) and gallstone-induced (n = 51). Data were collected on demographics, severity of pancreatitis, complications, and outcomes. Primary outcomes included nil per oral (NPO) duration, length of hospital stay, and mortality. Secondary outcomes included local and systemic complications. Statistical analysis was performed using Chi-square, independent t-test, and Mann-Whitney U test, with significance set at $p < 0.05$.

Results: Alcohol-induced pancreatitis was more common (66.45%) and affected predominantly younger males (mean age 37.8 years, 97% male), while gallstone-induced pancreatitis was more frequent in older females (mean age 46.5 years, 84.3% female; $p < 0.0001$). No mortality was observed in either group. The mean NPO duration (2.49 ± 1.12 vs. 2.75 ± 1.02 days; $p = 0.1656$) and hospital stay (3.55 ± 1.81 vs. 3.41 ± 1.3 days; $p = 0.617$) were comparable. Alcohol-induced pancreatitis showed a higher incidence of acute necrotic collection (21.8% vs. 3.92%), while gallstone-induced cases had more pseudocyst formation (43.1% vs. 27.7%).

Conclusion: Both alcohol-induced and gallstone-induced acute pancreatitis demonstrated favorable short-term outcomes with no mortality when managed appropriately. Alcohol-induced cases were more prone to necrosis, whereas gallstone-induced cases had a higher risk of pseudocysts. Etiology-specific monitoring is recommended to guide early intervention and optimize patient management.

Keywords: Acute pancreatitis; alcohol-induced pancreatitis; gallstone-induced pancreatitis; complications; necrosis; pseudocyst; clinical outcomes; observational study

Introduction

Acute pancreatitis (AP) is an inflammatory condition of the pancreas characterized by sudden onset of abdominal pain and elevated pancreatic enzymes in the blood(1). The pathophysiology involves autodigestion of pancreatic tissue by prematurely activated digestive enzymes, primarily trypsin,

leading to local inflammation and systemic complications(2). This process begins with acinar cell injury, which triggers a cascade of inflammatory responses(2). Globally, the incidence of acute pancreatitis ranges from 5 to 80 cases per 100,000 people annually(2). Approximately 80% of cases resolve without complications, while 20% develop severe pancreatitis, associated with complications like necrosis, organ failure, and mortality rates between 20% and 40%(2). The condition presents most commonly in individuals between the third and sixth decades of life and exhibits variations based on the underlying etiology(3).

Acute pancreatitis is a clinically significant condition due to its potential to cause severe morbidity and mortality(4,5). Clinical severity is broadly categorized into mild, moderate, and severe forms based on the Modified Atlanta Classification(6,7). Mild Acute Pancreatitis: Characterized by the absence of organ failure or local complications. Patients usually recover within a few days with conservative management(6,7). Severe Acute Pancreatitis: Involves persistent organ failure lasting more than 48 hours, along with complications such as necrosis, abscesses, and systemic inflammatory response syndrome (SIRS). Severe cases have a mortality rate as high as 30% to 50%(6,7).

The overall mortality rate for acute pancreatitis ranges from 5% to 20%, depending on severity and the presence of organ failure(8). Alcohol-induced pancreatitis tends to present with higher rates of local complications like pseudocysts and necrosis, while cholelithiasis-induced pancreatitis is associated with a higher risk of biliary sepsis(8).

Acute pancreatitis (AP) is a common gastrointestinal emergency with significant morbidity and mortality, and its two primary etiologies, alcohol consumption and cholelithiasis disease, account for approximately 80% of all cases(5). While both conditions lead to similar pathological outcomes, they differ significantly in patient demographics, clinical course, complications, and prognosis(5). Understanding these differences is crucial for tailoring clinical management strategies and improving patient outcomes.

- Alcohol-Induced Pancreatitis: Typically affects younger males in their third to fifth decades of life. This form of pancreatitis is associated with a chronic inflammatory process and has a higher tendency for local complications like pseudocysts and necrosis(9,10).
- Cholelithiasis-Induced Pancreatitis: More common in older females, typically in their fifth to sixth decades. This form often involves biliary obstruction and can lead to complications like biliary sepsis and the need for early endoscopic or surgical intervention(11,12).

A comparative analysis of these two forms of acute pancreatitis provides valuable insights into the risk factors, clinical presentation, progression, and outcomes associated with each etiology. This comparison helps guide early intervention, improve risk stratification, and optimize treatment protocols for specific patient populations. Differences in the underlying causes of acute pancreatitis influence clinical outcomes, including the severity of the disease, length of hospital stay, incidence of complications, and mortality rates(9,11).

AIM: To evaluate and compare the clinical outcomes of alcohol-induced and cholelithiasis-induced acute pancreatitis.

Material and Methods

❖ **Study Design:** A single centre, hospital-based, prospective observational study designed to compare the clinical outcomes between alcohol-induced and cholelithiasis-induced acute pancreatitis.

❖ **Study Settings:** The study was conducted at the Department of General Surgery, RKDF Medical College, Hospital, and Research Centre, Bhopal, Madhya Pradesh, India.

❖ **Ethical Clearance:** Ethical clearance was granted following the scrutiny of the study protocol, data collection form, and informed consent form. The research protocol, including the methodology, potential risks, and benefits, was reviewed thoroughly by the **Institute's Ethical Committee**.

❖ **Study Duration:** The total duration of the present study was **18 months**,

❖ **Primary Outcomes**:

i. **Length of NPO (Nil Per Oral)**: Measured in days from the time of admission until the resumption of oral intake.

ii. **Length of Hospital Stay**: Measured in days from the date of admission to discharge.

iii. **Mortality**: Recorded as either **survival** or **death** during hospitalization.

❖ **Secondary Outcomes**:

- **Local Complications**: Measured based on imaging findings (e.g., pseudocyst formation, acute peripancreatic fluid collection, walled-off necrosis).
- **Systemic Complications**: Assessed during hospitalization (e.g., acute respiratory distress syndrome, renal failure, cardiovascular complications).

❖ **Follow-Up:** Patients were followed up for the **duration of their hospitalization** until discharge or death.

❖ **Definition of the Exposure:** The exposure assessed was the etiology of pancreatitis (alcohol consumption or cholelithiasis disease) and its impact on clinical outcomes.

❖ **Study Participants:** The participants for the present study were patients diagnosed with **acute pancreatitis** who met the eligibility criteria. These participants were categorized into two groups based on the etiology of their condition: *alcohol-induced and cholelithiasis-induced acute pancreatitis.*

❖ **Inclusion Criteria**

1. Patients diagnosed with acute pancreatitis meeting at least **two or more** of the following criteria:
   - Typical abdominal pain (acute onset, persistent pain in the epigastric region radiating to the back).
   - Serum amylase or lipase levels **> 3 times the upper normal limit**.
   - Imaging findings (USG or CT) consistent with acute pancreatitis.
2. History of:
   - Alcohol consumption within **48 hours** of symptom onset, **or**
   - Cholelithiasiss detected on **USG or CT**.
3. Patients aged **≥18 years**.
4. Patients who provided written informed consent.

❖ **Exclusion Criteria**

1. Patients with acute pancreatitis due to etiologies **other than alcohol or cholelithiasiss** (e.g., hypertriglyceridemia, trauma, drugs, autoimmune, or idiopathic causes).
2. Patients with a history of **chronic pancreatitis**.
3. Patients who did not provide informed consent.
4. Patients with severe comorbidities that could interfere with the study (e.g., advanced malignancy).

❖ **Study Groups:** Participants were divided into two study groups based on the etiology of pancreatitis:

1. **Alcohol-Induced Acute Pancreatitis Group**: Participants with a documented history of alcohol consumption within 48 hours of symptom onset.
2. **Cholelithiasis-Induced Acute Pancreatitis Group**: Participants with cholelithiasiss detected on USG or CT imaging.

❖ **Sample Size:** All eligible participants who attended the study institute during the recruitment period and provided written informed consent were enrolled in the present study. Following this approach, a total of **152 participants** were enrolled in the present study.

❖ **Sampling Methodology:** A **non-probability convenience sampling** method was used for this study.

❖ **Data Collection Procedure:** The data collection process for this study was carried out meticulously to ensure completeness, accuracy, and adherence to ethical standards.

1. Initial Patient Screening: Patients diagnosed with acute pancreatitis were identified upon admission to the Department of General Surgery, RKDF Medical College, Hospital, and Research Centre, Bhopal. Potential participants were identified through the Outpatient Department (OPD), Casualty Department, or transfers from other hospital departments. Each potential participant underwent a preliminary assessment to confirm the diagnosis of acute pancreatitis based on the following criteria:

- Typical abdominal pain.
- Serum amylase or lipase levels **> 3 times** the upper normal limit.
- Imaging findings consistent with acute pancreatitis (USG or CECT).

2. Obtaining Informed Consent: The **Principal Investigator (PI)** explained the purpose, objectives, procedures, risks, and benefits of the study to each potential participant in a language they understood (Hindi or English). Participants were informed of their right to withdraw from the study at any time without affecting their medical care. Bilingual (Hindi and English) informed consent forms were provided to participants. Participants who agreed to take part in the study signed the consent form.

3. Baseline Data Collection: After obtaining informed consent, baseline data were collected systematically:

- **Demographic Data**: Age, gender, socio-economic status, and patient identification number were recorded.
- **Clinical History**:
- **Details of symptoms** (e.g., pain onset, duration, location, radiation).
- **History of alcohol consumption** (type, quantity, and duration of alcohol intake) within the **48 hours prior to symptom onset**.
- **History of cholelithiasis disease** (previous diagnosis or family history of cholelithiasiss).
- **Personal and family medical history** (comorbidities such as diabetes, hypertension, chronic kidney disease).

4. Laboratory and Imaging Data: Relevant laboratory investigations and imaging studies were performed and recorded:

- **Complete Blood Count (CBC)**: Hemoglobin, total leukocyte count (TLC), differential count, and hematocrit.
- **Liver Function Tests (LFT)**: AST, ALT, ALP, bilirubin levels.
- **Renal Function Tests**: Serum creatinine, blood urea nitrogen (BUN).
- **Serum Amylase and Lipase Levels**: Confirmatory tests for acute pancreatitis.
- **Serum Electrolytes**: Sodium, potassium, calcium.
- **CRP Levels**: For assessing inflammation.
- **Arterial Blood Gas (ABG) Analysis**: To evaluate oxygenation and acid-base balance.

6. Monitoring and Follow-Up During Hospitalization: Participants were monitored daily for the duration of their hospital stay:

- **Daily Clinical Assessment**:
- Vital signs and physical examinations.
- Progression or resolution of symptoms.
- Development of new complications.
- **Laboratory Monitoring**:
- Repeat blood tests as required to monitor disease progression and response to treatment.
- **Recording of Complications**:
- Local complications (e.g., pseudocyst, acute peripancreatic fluid collection, necrosis).
- Systemic complications (e.g., respiratory failure, renal failure, sepsis).

- **Outcome Measures**:
  - **Length of NPO** (days until oral intake was resumed).
  - **Length of Hospital Stay** (days from admission to discharge).
  - **Mortality** (recorded as survival or death during hospitalization).

❖ **Statistical Analysis:** All statistical and graphical analyses for this study were undertaken using Stata software version 17.0. Descriptive statistics were used to summarize the demographic and clinical characteristics of the participants. The differences between the two groups (alcohol-induced and cholelithiasis-induced) were analyzed using the Chi-square test for categorical variables. For continuous variables, the independent t-test was used to compare normally distributed data, while the Mann-Whitney U test was employed for non-normally distributed data. The statistical significance level was set at $p < 0.05$.

❖ **Funding:** There was no external funding for this study.

❖ **Conflict of Interest:** The authors declare that there was **no conflict of interest** in the design, implementation, and interpretation of the findings of this study.

Results:

The study population consisted of 152 participants diagnosed with acute pancreatitis. Among these, **101 cases (66.45%)** were attributed to alcohol-induced acute pancreatitis, making it the predominant etiology. Gallstone-induced acute pancreatitis accounted for **51 cases (33.55%)**.

A total of 152 patients with acute pancreatitis were included, of which 101 (66.45%) had alcohol-induced pancreatitis and 51 (33.55%) had gallstone-induced pancreatitis. Alcohol-induced cases were significantly younger (mean age 37.8 ± 12.7 years) compared to gallstone-induced cases (mean age 46.5 ± 13.7 years; $p < 0.001$). Gender distribution differed markedly between groups. Males predominated in alcohol-induced pancreatitis (97%), whereas females were the majority in gallstone-induced cases (84.3%; $p < 0.0001$). Regarding severity, most cases in both groups were moderate (56.4% in alcohol-induced and 58.8% in gallstone-induced). Severe disease was more frequent in alcohol-induced cases (17.8%) compared to gallstone-induced (7.84%), although the difference was not statistically significant ($p = 0.360$).

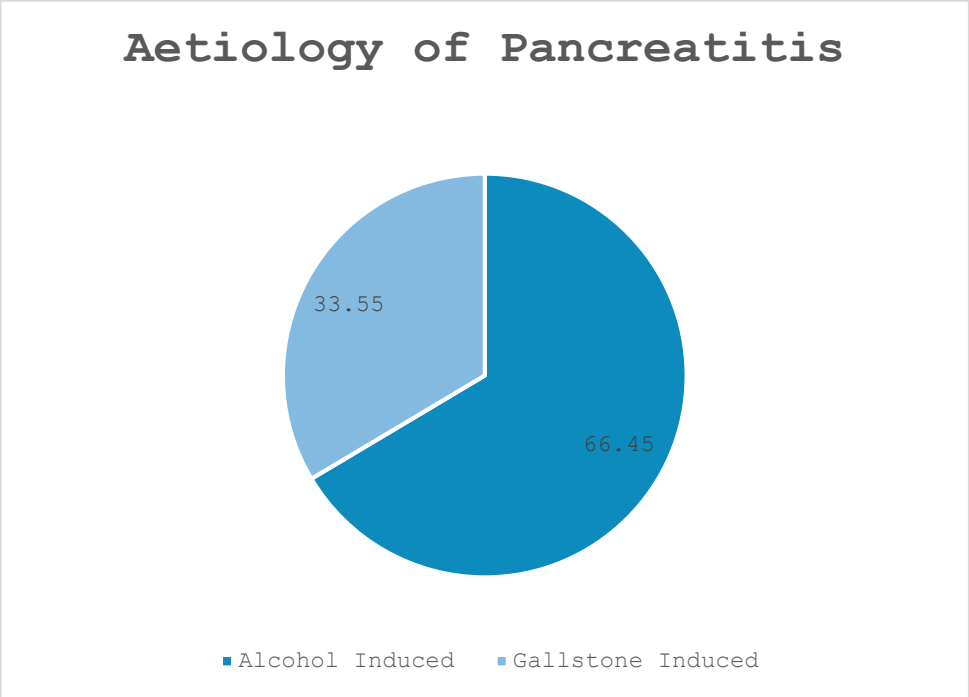

Table 1: Characteristics of Participants					
	Alcohol Induced (n=101)		Gallstone Induced (n=51)		P-value
	n	%	n	%	
Age (Mean, SD)	37.8	12.7	46.5	13.7	< 0.001
Gender					
Female	3	2.97	43	84.3	< 0.0001
Male	98	97	8	15.7	
Severity of Pancreatitis					
Mild	26	25.7	17	33.3	0.360
Moderate	57	56.4	30	58.8	
Severe	18	17.8	4	7.84	

The mean serum amylase levels were similar between alcohol-induced (824.5 ± 287.3 U/L) and gallstone-induced pancreatitis (815.6 ± 456.2 U/L; p = 0.094). However, serum lipase levels were significantly higher in gallstone-induced cases (1,563.4 ± 827.5 U/L) compared to alcohol-induced cases (1,034.2 ± 512.7 U/L; p = 0.026).

Table 2: Serum Amylase and Lipase Levels (U/L)			
Parameter	**Alcohol Induced (n=101)**	**Gallstone Induced (n=51)**	**P-value**
Serum Amylase (Mean, SD)	824.5 ± 287.3	815.6 ± 456.2	0.094
Serum Lipase (Mean, SD)	1,034.2 ± 512.7	1,563.4 ± 827.5	0.026

Table 3: Distribution of Participants based on Associated Complications				
	Alcohol Induced (n=101)		Gallstone Induced (n=51)	
	n	%	n	%
APFC				
No	56	55.4	25	49
Yes	45	44.6	26	51
Pseudocyst				
No	73	72.3	29	56.9
Yes	28	27.7	22	43.1
ANC				
No	79	78.2	49	96.1
Yes	22	21.8	2	3.92
WON				
No	97	96	49	98
Yes	4	3.96	1	2

Complications

Local complications varied by etiology. Acute peripancreatic fluid collection (APFC) was present in 44.6% of alcohol-induced and 51% of gallstone-induced cases. Pseudocyst formation was more frequent in gallstone-induced pancreatitis (43.1%) than in alcohol-induced pancreatitis (27.7%). Acute necrotic collection (ANC) was significantly more common in alcohol-induced cases (21.8%) compared to gallstone-induced cases (3.92%). Walled-off necrosis (WON) was uncommon in both groups but slightly higher in alcohol-induced pancreatitis (3.96% vs. 2%).

Table 4: Distribution of Participants based on Outcome					
	Alcohol Induced (n=101)		**Gallstone Induced (n=51)**		
	n	**%**	**n**	**%**	
Nil per Oral (Mean, SD)	2.49	1.12	2.75	1.02	**0.1656**
Moratality (n, %)	0	0	0	0	**-**
Duration of Hospital Stay	3.55	1.81	3.41	1.3	**0.617**

Clinical Outcomes

The mean duration of nil per oral (NPO) status was 2.49 ± 1.12 days in alcohol-induced pancreatitis and 2.75 ± 1.02 days in gallstone-induced cases, with no significant difference (p = 0.1656). Similarly, the mean hospital stay was comparable between the two groups (3.55 ± 1.81 vs. 3.41 ± 1.3 days; p = 0.617). No mortality was recorded in either group during hospitalization.

Discussion:

In the present study, no mortality was observed in either the alcohol-induced or gallstone-induced acute pancreatitis groups. This finding suggests that early diagnosis, appropriate management, and supportive care significantly improve survival outcomes in both etiologies of acute pancreatitis. The absence of mortality in this study contrasts with prior research, where varying mortality rates have been reported, largely depending on disease severity, presence of organ failure, and complications.

Conversely, Anderson et al. (2007) found no significant difference in mortality between alcohol- and gallstone-induced pancreatitis, with a 30-day mortality rate of 5% and a 1-year mortality rate of 11%, irrespective of etiology(13). Their findings align with the present study in demonstrating that, when managed effectively, mortality rates in acute pancreatitis can be minimized. Additionally, Garcia et al. (2021) reported no significant difference in hospital mortality between alcohol- and gallstone-induced pancreatitis, further supporting the notion that mortality risk may not be inherently linked to etiology but rather to disease severity and timely interventions(14).

In contrast, Cho et al. (2015) reported that alcohol-induced acute pancreatitis had a higher mortality rate than gallstone-induced cases, with deaths occurring exclusively in the alcohol group (P = 0.012)(15). Bhasha et al. (2023) reported a significantly higher mortality rate of 6% in alcohol-induced acute pancreatitis compared to gallstone-induced pancreatitis(16). Similarly, Easler et al. (2016) observed a 7% mortality rate in their cohort, with alcohol-induced cases demonstrating a higher risk of organ failure and pancreatic necrosis, which contributed to increased mortality(17). These findings suggest that settings with a higher burden of severe acute pancreatitis, mortality may be more evident.

The lack of mortality in the present study highlights the effectiveness of current management strategies and early intervention. However, given that previous studies have reported mortality rates ranging from 5% to 11%, continued vigilance in managing severe cases is essential. Future research with long-term follow-up may provide further insights into the long-term survival and recurrence patterns in alcohol- and gallstone-induced acute pancreatitis.

Nil Per Oral (NPO) Duration

The present study found that the mean duration of nil per oral (NPO) status was **2.49 ± 1.12 days** in alcohol-induced acute pancreatitis and **2.75 ± 1.02 days** in gallstone-induced pancreatitis, with no statistically significant difference between the two groups (P = 0.1656). These findings indicate that the overall duration of fasting required before resumption of oral intake is comparable between both etiologies, suggesting that the inflammatory response and gastrointestinal recovery follow a similar

course in both conditions when managed with standard supportive care. Bhasha et al. (2023) also found no significant difference in NPO duration between alcohol- and gallstone-induced acute pancreatitis, supporting the conclusion that both groups require similar periods of bowel rest before resuming oral intake(16). Similarly, Paul et al. (2022) reported that the mean duration of NPO was comparable between the two groups, reinforcing the idea that etiology does not significantly impact the initial conservative management strategy.(18)

In contrast, Garcia et al. (2021) reported a slightly longer NPO duration in gallstone-induced acute pancreatitis compared to alcohol-induced cases, likely due to a higher proportion of patients requiring endoscopic retrograde cholangiopancreatography (ERCP) before resuming oral intake(14). Samanta et al. (2019) also found a marginally longer fasting duration in gallstone-induced cases, particularly in patients who developed biliary obstruction or cholangitis, which necessitated delayed refeeding(19).

However, Cho et al. (2015) reported a shorter NPO duration in alcohol-induced cases, suggesting that these patients may tolerate early oral intake better due to a lower incidence of biliary obstruction(15). This discrepancy may be due to differences in the criteria used for initiating oral feeding, patient comorbidities, or institutional feeding protocols.

The present study analyzed the distribution of acute peripancreatic fluid collection (APFC), pseudocysts, acute necrotic collection (ANC), and walled-off necrosis (WON) between alcohol-induced and gallstone-induced acute pancreatitis. APFC and pseudocyst formation were more frequent in gallstone-induced cases, whereas ANC and WON were more common in alcohol-induced cases.

Acute Peripancreatic Fluid Collection (APFC) was observed in 44.6% of alcohol-induced cases and 51% of gallstone-induced cases, indicating a comparable frequency of early fluid accumulation. This finding aligns with **Paul et al. (2022),** who also reported no significant difference in the occurrence of APFC between the two groups, suggesting that early inflammatory responses and fluid shifts occur similarly regardless of etiology. Similarly, **Garcia et al. (2021)** found that the incidence of APFC did not differ significantly between alcohol- and gallstone-induced cases.

Pseudocysts were observed more frequently in gallstone-induced pancreatitis (43.1%) compared to alcohol-induced cases (27.7%). This is in contrast to findings by **Cho et al. (2015),** who reported that pseudocysts were significantly more common in alcohol-induced pancreatitis (20% vs. 6.6%, $P = 0.023$), likely due to recurrent episodes of inflammation in chronic alcohol users.(15) Likewise, **Samanta et al. (2019)** found a slightly higher incidence of pseudocyst formation in alcohol-induced cases but noted that the overall difference was not statistically significant.(19)

Acute Necrotic Collection (ANC) occurred in 21.8% of alcohol-induced cases compared to only 3.92% of gallstone-induced cases, indicating a higher risk of pancreatic necrosis in alcohol-related pancreatitis. **Easler et al. (2016)** similarly found that alcohol-induced pancreatitis had a significantly higher incidence of pancreatic necrosis (62% vs. 31%, $P = 0.006$), supporting the idea that alcohol-related cases may have a more aggressive disease course.(17) **Kim et al. (2017)** also reported a higher frequency of pancreatic fluid collection in alcohol-induced severe acute pancreatitis ($P = 0.04$), which may contribute to increased necrosis risk. (20)

Walled-Off Necrosis (WON) was slightly more common in alcohol-induced cases (3.96%) compared to gallstone-induced pancreatitis (2%), though the difference was minimal. This finding aligns with Bhasha et al. (2023), who found no significant difference in local complications, including WON, between alcohol- and gallstone-induced cases(16). Similarly, Samanta et al. (2019) found that although necrosis was slightly more common in alcohol-induced pancreatitis ($P = 0.05$), the overall outcomes remained comparable between the two groups.(19)

Overall, these findings suggest that alcohol-induced pancreatitis is more likely to result in necrosis and walled-off necrosis, whereas gallstone-induced pancreatitis may have a higher risk of pseudocyst formation and fluid collections. This underscores the importance of etiology-specific monitoring strategies—patients with alcohol-induced pancreatitis should be closely monitored for necrosis and potential organ failure, while gallstone-induced cases may require early imaging to assess for fluid

collections requiring intervention. Future research should focus on how differences in inflammatory pathways, recurrent episodes, and early interventions influence complication rates in acute pancreatitis.

Conclusion

This study shows that alcohol-induced and gallstone-induced acute pancreatitis differ in patient profile, disease pattern, and complications, but share similar short-term outcomes when managed with timely and appropriate care. Alcohol-induced pancreatitis was more common among younger males and carried a higher risk of necrosis, while gallstone-induced pancreatitis was frequent in older females and more often associated with pseudocyst formation. Despite these differences, there was no mortality in either group, and the duration of nil per oral status and hospital stay were comparable. These findings highlight the importance of early diagnosis, supportive management, and etiology-based monitoring to prevent complications and improve outcomes in acute pancreatitis.

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