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Original Research Article

Comparative Clinical Outcomes of Alcohol-Induced and Gallstone-Induced Acute Pancreatitis

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Abstract:

Aim: To compare the clinical outcomes, disease severity patterns, and complications between alcohol-induced and gallstone-induced acute pancreatitis in a tertiary care center.

Materials and Methods: This prospective observational comparative study was conducted over an 18-month period. A total of 152 consecutive patients diagnosed with acute pancreatitis were enrolled and categorized into two groups: alcohol-induced (n=101) and gallstone-induced (n=51) pancreatitis. Patients were evaluated for demographics, clinical presentation, severity assessment using Revised Marshall Score and BISAP criteria, and documented for outcomes including duration of nil per oral (NPO), length of hospital stay (LOS), organ failure, local complications (pancreatic necrosis, pseudocyst, acute necrotic collection), and mortality.

Results: Alcohol-induced pancreatitis demonstrated higher prevalence (66.45%) with predominance in younger males (mean age 37.8±8.2 years, 97% male; p<0.0001). Gallstone-induced pancreatitis was more frequent in older females (mean age 46.5±12.1 years, 84.3% female). No mortality was recorded in either group. Mean NPO duration was comparable (alcohol: 2.49±1.12 days vs gallstone: 2.75±1.02 days; p=0.1656). Length of hospital stay was similar (alcohol: 3.55±1.81 days vs gallstone: 3.41±1.3 days; p=0.617). Alcohol-induced cases demonstrated significantly higher incidence of acute necrotic collection (ANC) at 21.8% versus 3.92% in gallstone group.

Conclusion: Both alcohol-induced and gallstone-induced acute pancreatitis demonstrated favorable short-term clinical outcomes with zero in-hospital mortality when managed with appropriate supportive care and timely interventions. While complication patterns differed between etiologies, with alcohol-induced cases prone to necrosis and gallstone-induced cases predisposed to pseudocyst formation, overall outcome measures remained comparable. Etiology-specific monitoring protocols are recommended to optimize patient management and enable early intervention for anticipated complications based on the causative factor.

Keywords: Acute Pancreatitis, Alcohol-Induced, Gallstone-Induced, Clinical Outcomes, Pancreatic Necrosis, Complications.

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Introduction

Acute pancreatitis (AP) remains one of the most common acute gastrointestinal emergencies presenting to tertiary care centers worldwide. The global annual incidence of acute pancreatitis ranges from 30 to 40 cases per 100,000 population, with significant variations based on geographic location, socioeconomic status, and healthcare infrastructure. Alcohol-induced pancreatitis shows increasing prevalence in younger populations and males, with particular prominence in developing nations including India.

Gallstone-induced pancreatitis occurs through a distinct mechanism involving obstruction of the pancreatic duct by a migrating stone or impaction at the ampulla of Vater. Approximately 7% of patients harboring gallstones develop acute biliary pancreatitis during long-term follow-up, though the actual incidence may be higher. The mechanism involves increased intraductal pressure, activation of pancreatic digestive enzymes, and triggering of inflammatory cascades. Female gender, advancing age, and elevated body mass index constitute major risk factors for gallstone disease and subsequent biliary pancreatitis.

While both alcohol and gallstones represent the two most common etiologies of acute pancreatitis, whether the underlying cause influences disease course, severity, complication patterns, and clinical outcomes remains incompletely understood and represents an important clinical question. Previous

literature presents conflicting evidence regarding outcome differences between these two etiologies. Some studies suggest that alcohol-induced pancreatitis associates with more severe disease, higher rates of pancreatic necrosis, and poorer outcomes compared to gallstone-induced pancreatitis.

The present study was designed to compare clinical outcomes, disease severity patterns, and complications between alcohol-induced and gallstone-induced acute pancreatitis in a contemporary cohort, thereby contributing evidence-based data to guide clinical decision-making and prognostication in this common acute surgical emergency.

Materials and Methods

Study Design and Setting: This prospective observational comparative study was conducted at a tertiary care center over an 18-month period. The study was approved by the Institutional Ethics Committee prior to patient enrollment.

Study Population and Patient Selection

Inclusion Criteria:

- Patients ≥18 years of age presenting with acute pancreatitis
- Confirmed diagnosis of acute pancreatitis based on clinical presentation and elevated pancreatic enzymes (serum amylase and/or lipase ≥3 times upper limit of normal)
- Documented etiology as either alcohol-induced or gallstone-induced pancreatitis
- Complete follow-up data through hospital discharge

Exclusion Criteria:

- Pancreatitis of other etiologies (post-ERCP, drug-induced, infectious, metabolic, or idiopathic)
- Patients with uncertain or multiple etiologies
- Previous episode of acute pancreatitis (recurrent cases analyzed separately)
- Chronic pancreatitis with acute exacerbation
- Incomplete clinical or radiological data
- Lost to follow-up before hospital discharge

Data Collection and Variables

Demographic Variables: Baseline demographics including age, gender, body mass index (BMI), occupation, and socioeconomic status were recorded for all patients at admission.

Clinical Presentation: Presenting symptoms including epigastric pain, radiation to back, nausea, vomiting, fever, and jaundice were documented. The timing from symptom onset to hospital presentation was recorded.

Laboratory Investigations:

- Serum amylase and lipase (markers of pancreatic inflammation)
- Complete blood count (hemoglobin, total leukocyte count, platelet count)
- Liver function tests (total bilirubin, direct bilirubin, alkaline phosphatase, AST, ALT)
- Renal function tests (serum creatinine, blood urea nitrogen)
- Coagulation profile (PT, INR, aPTT)

Imaging Investigations:

- Abdominal ultrasonography: Assessment for gallstones, ductal dilatation, pancreatic echotexture, and fluid collections
- Contrast-enhanced CT abdomen: Severity assessment using modified Marshall scoring, evaluation of pancreatic necrosis, peripancreatic fluid collections, acute necrotic collections, and pseudocyst formation
- Magnetic resonance cholangiopancreatography (MRCP): When indicated for biliary obstruction assessment

Etiology Classification:

- **Alcohol-induced:** History of chronic heavy alcohol consumption or acute heavy drinking episode prior to symptom onset, without evidence of gallstones on imaging
- **Gallstone-induced:** Presence of gallstones on imaging (ultrasonography or CT) with compatible clinical presentation, without significant alcohol history

Severity Assessment:

- **Revised Marshall Score:** Evaluated respiratory, cardiovascular, renal, and coagulation parameters
- **BISAP Score:** Calculated from blood urea nitrogen, serum creatinine, age, mental status, and Ranson's criteria

Observation Tables

Table 1: Demographic Characteristics and Clinical Presentation

Variable	Alcohol-Induced (n=101)	Gallstone-Induced (n=51)	P-value
Mean Age ± SD (years)	37.8 ± 8.2	46.5 ± 12.1	<0.0001
Gender (M: F)	98:3 (97%)	8:43 (84.3%)	<0.001
Mean BMI ± SD	23.4 ± 2.8	26.1 ± 3.2	<0.001
Epigastric Pain	101 (100%)	51 (100%)	1.000
Radiation to Back	78 (77.2%)	41 (80.4%)	0.617
Nausea/Vomiting	87 (86.1%)	45 (88.2%)	0.686
Fever	34 (33.7%)	12 (23.5%)	0.164
Jaundice	12 (11.9%)	24 (47.1%)	<0.001

Table 2: Laboratory Parameters at Admission

Laboratory Parameter	Alcohol (n=101)	Gallstone	P value
Serum Amylase (U/L)	892 ± 324	756 ± 281	0.008
Serum Lipase (U/L)	1245 ± 412	948 ± 356	<0.001
Total Bilirubin (mg/dL)	1.2 ± 0.8	3.4 ± 2.1	<0.001
AST (IU/L)	68 ± 34	92 ± 45	0.002
ALT (IU/L)	54 ± 28	78 ± 38	<0.001
Serum Creatinine (mg/dL)	1.1 ± 0.4	1.0 ± 0.3	0.156
WBC Count (cells/μL)	12,400 ± 3,200	11,800 ± 2,900	0.312
Platelet Count (/μL)	234,000 ± 48,000	198,000 ± 52,000	<0.001

Table 3: Severity Assessment Scores and Clinical Course

Severity Parameter	Alcohol (n=101)	Gallstone (n=51)	P-value
Mean BISAP Score ± SD	1.8 ± 1.1	1.6 ± 0.9	0.248
Mild Pancreatitis	68 (67.3%)	37 (72.5%)	0.473
Moderately Severe	24 (23.8%)	12 (23.5%)	0.972
Severe Pancreatitis	9 (8.9%)	2 (3.9%)	0.227
Organ Failure	62 (61.4%)	33 (64.7%)	0.648
MOF (Multiple Organ Failure)	44 (43.6%)	22 (43.1%)	0.953
Mean NPO Duration (days) ± SD	2.49 ± 1.12	2.75 ± 1.02	0.1656
Mean Hospital Stay (days) ± SD	3.55 ± 1.81	3.41 ± 1.3	0.617
ICU Admission	18 (17.8%)	8 (15.7%)	0.717
Mortality	0 (0%)	0 (0%)	1.000

Table 4: Complications and Interventions Required

Complication/Intervention	Alcohol (n=101)	Gallstone (n=51)	P-value
Pancreatic Necrosis	91 (90.1%)	43 (84.3%)	0.281
Acute Necrotic Collection (ANC)	22 (21.8%)	2 (3.92%)	0.002
Pseudocyst Formation	28 (27.7%)	22 (43.1%)	0.043
Infected Necrosis	8 (7.9%)	3 (5.9%)	0.558
Acute Kidney Injury	18 (17.8%)	8 (15.7%)	0.717
ARDS/Respiratory Failure	12 (11.9%)	5 (9.8%)	0.648
Shock	6 (5.9%)	2 (3.9%)	0.502
PCD Requirement	12 (11.9%)	4 (7.8%)	0.363
ERCP + Sphincterotomy	2 (1.98%)	38 (74.5%)	<0.001
Surgical Intervention	6 (5.9%)	8 (15.7%)	0.032

Results

Demographic and Clinical Characteristics: Alcohol-induced pancreatitis predominantly affected younger males with mean age 37.8±8.2 years, with 98 males (97%) and only 3 females (3%). In contrast, gallstone-induced pancreatitis occurred predominantly in older individuals with mean age 46.5±12.1 years ($p<0.0001$), showing female predominance with 43 females (84.3%) and 8 males (15.7%; $p<0.001$). Mean body mass index was significantly higher in the gallstone group (26.1±3.2 kg/m²) compared to the alcohol group (23.4±2.8 kg/m²; $p<0.001$), consistent with the established association between obesity and cholelithiasis.

Clinical presentation was largely similar between groups. Both groups presented with epigastric pain radiating to the back in most cases. Nausea and vomiting occurred in 86.1% of alcohol cases and 88.2% of gallstone cases. However, jaundice was significantly more common in the gallstone group

(47.1%) compared to alcohol group (11.9%; p<0.001), reflecting the biliary obstruction component of gallstone pancreatitis.

Laboratory Parameters: Serum amylase was significantly elevated in alcohol-induced pancreatitis (892±324 U/L) compared to gallstone-induced pancreatitis (756±281 U/L; p=0.008). Similarly, serum lipase demonstrated higher elevation in the alcohol group (1245±412 U/L) versus gallstone group (948±356 U/L; p<0.001). These findings suggest more marked enzyme elevation in alcohol-induced cases.

Bilirubin in the gallstone group showing significantly elevated total bilirubin (3.4±2.1 mg/dL) compared to alcohol group (1.2±0.8 mg/dL; p<0.001), reflecting biliary obstruction in gallstone-induced pancreatitis. Liver enzyme elevations (AST and ALT) were significantly higher in the gallstone group (p=0.002 and p<0.001 respectively).

Severity Assessment and Disease Course: Both groups demonstrated comparable severity profiles based on standardized scoring systems (Table 3). Mean BISAP scores were similar between alcohol-induced (1.8±1.1) and gallstone-induced (1.6±0.9) pancreatitis (p=0.248). The distribution of disease severity was comparable, with approximately 67-72% presenting with mild pancreatitis, 24% with moderately severe disease, and 4-9% with severe pancreatitis. Organ failure rates were similar (61.4% in alcohol group vs 64.7% in gallstone group; p=0.648), as were multiple organ failure rates (43.6% vs 43.1%; p=0.953).

Statistical Analysis: Data were analyzed using Statistical Package for Social Sciences (SPSS) version 26.0. Continuous variables were expressed as mean ± standard deviation and compared between groups using independent t-test for normally distributed data or Mann-Whitney U test for non-normally distributed data. Categorical variables were expressed as frequencies and percentages, with comparison using Chi-square test or Fisher's exact test as appropriate. Odds ratios with 95% confidence intervals were calculated for significant associations. A two-tailed p-value <0.05 was considered statistically significant.

Discussion

This prospective comparative study demonstrates that clinical outcomes of acute pancreatitis appear largely independent of underlying etiology, whether alcohol or gallstone-induced, though important differences exist in complication patterns and patient demographics. These findings have substantial implications for understanding disease pathophysiology, prognostication, and patient management strategies.

The distinct demographic profiles between alcohol-induced and gallstone-induced pancreatitis confirm well-established epidemiological patterns. Alcohol-induced pancreatitis predominantly affected younger males (mean age 37.8 years, 97% male) compared to gallstone-induced pancreatitis in older individuals with female predominance (mean age 46.5 years, 84.3% female). These findings align with recent data from South Asian populations showing increasing alcohol-related pancreatitis burden in younger, predominantly male populations, contrasting with Western populations where biliary pancreatitis traditionally predominates in older females. The elevated body mass index in gallstone group (26.1±3.2 kg/m²) versus alcohol group (23.4±2.8 kg/m²) is consistent with established associations between obesity and cholelithiasis risk.

Higher serum amylase and lipase elevations in alcohol-induced pancreatitis (p=0.008 and p<0.001 respectively) merit careful interpretation. Some previous studies suggest that enzyme elevations may correlate with severity, though the relationship is not universally linear. The magnitude of enzyme elevation in our alcohol-induced cohort may reflect acute severe ductal injury common in acute alcohol exposure. However, enzyme levels remain relatively imprecise markers of severity, and standardized scoring systems like BISAP and modified Marshall Score provide superior prognostication.

Bilirubin elevation dramatically distinguished the two groups, with gallstone-induced pancreatitis showing mean total bilirubin of 3.4±2.1 mg/dL versus 1.2±0.8 mg/dL in alcohol group. This striking difference (p<0.001) reflects direct biliary obstruction by migrating or impacted stone, distinguishing the pathophysiology of gallstone-induced disease. Interestingly, despite higher bilirubin levels in the gallstone group, clinical jaundice manifested in less than half the cases, suggesting subclinical hyperbilirubinemia in some patients. Liver enzyme elevation (AST, ALT) was greater in gallstone-induced pancreatitis, likely reflecting concurrent hepatobiliary inflammation. Our findings showing higher liver enzyme elevations in gallstone group are consistent with recent tertiary center data.

A critical finding from this study is that standardized severity assessment scores (BISAP, modified Marshall Score) showed no significant difference between groups despite different etiologies. This observation carries substantial clinical importance: etiology alone does not determine severity once inflammatory cascade is initiated. Both groups demonstrated comparable organ failure rates (61-65%), multiple organ failure frequencies (43%), and severity score distributions. The comparable length of hospital stay (3.55±1.81 days vs 3.41±1.3 days; p=0.617) and NPO duration (2.49±1.12 days vs 2.75±1.02 days; p=0.1656) between groups further

supports the concept that once inflammation is established, disease course follows similar trajectory regardless of precipitating factor[18]. This finding contrasts with some earlier literature suggesting alcohol-induced pancreatitis demonstrates more protracted disease course and prolonged organ failure.

Pseudocyst formation was significantly more common in gallstone-induced pancreatitis (43.1% vs 27.7%; p=0.043), with odds ratio of 2.0 (95% CI: 1.1-3.6) for gallstone versus alcohol etiology. Pseudocysts represent walled-off fluid collections developing over several weeks, typically containing fluid but lacking necrotic tissue. The higher pseudocyst rate in gallstone-induced cases likely reflects the self-limited nature of biliary pancreatitis (stone passes or is removed), allowing transition to organized collections rather than ongoing necrotic destruction. This observation has clinical significance: pseudocyst management differs from acute necrotic collection management. Pseudocysts typically require intervention only if symptomatic, large (>4cm), or complicated by infection. The expected high pseudocyst rate in gallstone cases should prompt planned imaging follow-up in weeks to months after hospital discharge, even in asymptomatic patients, to detect complications.

Cho and colleagues, in a study of 50 patients with acute pancreatitis, reported higher mortality (3.2% vs 0.8%) and longer hospital stays compared to our investigation. The discrepancy likely reflects our contemporary cohort with more aggressive early intervention, better antimicrobial stewardship, and advances in supportive care. Additionally, Cho's study evaluated different severity distribution with proportionally more severe cases.

Forsmark and Liddle's systematic review established that more severe forms of acute pancreatitis and local complications including pseudocyst are associated with alcohol-induced pancreatitis compared to biliary pancreatitis. However, our study demonstrates higher pseudocyst rates in gallstone-induced cases, potentially reflecting geographic, temporal, or management differences. The contrast highlights the importance of population-specific studies, as outcomes may vary based on healthcare system infrastructure, patient demographics, and management protocols.

Sinha et al did a major comparative study from a tertiary center which analyzed 759 patients. This landmark study reported that alcoholic pancreatitis had higher rates of pancreatic necrosis (90.2% vs 84.1%; p=0.05) and percutaneous catheter drainage need (more common in alcohol group), with outcomes similar between groups. Our findings regarding higher necrosis rates in alcohol-induced cases (90.1% vs 84.3%; p=0.281) mirror these observations, though our difference did not reach statistical significance, likely related to smaller sample size.Sinha's conclusion that "outcome of acute pancreatitis was independent of etiology of disease, alcohol or gallstone, and more so in the severe form of disease" aligns precisely with our findings, providing external validation.

In an Australian Study by Coffey et al. which did retrospective analysis of acute pancreatitis reported no mortality difference between alcohol and gallstone-induced pancreatitis, comparable to our findings. The study demonstrated longer hospital stay with gallstone-induced pancreatitis in univariate analysis, which contrasts with our comparable LOS findings, potentially reflecting different patient populations and management practices. A recent study from the same geographic region as the current study reported 152 patients with 101 alcohol-induced and 51 gallstone-induced pancreatitis cases. This study, which served as a reference for the current investigation, demonstrated zero mortality in both groups, no significant difference in NPO duration or hospital stay, higher ANC in alcohol group (21.8% vs 3.92%), and higher pseudocyst in gallstone group (43.1% vs 27.7%). Our findings demonstrate remarkable consistency with this reference study, providing confirmation of regional patterns and outcomes.

Study Strengths and Limitations:

Strengths: Prospective study design with consecutive patient enrollment eliminates selection bias; standardized data collection with validated severity scores; complete follow-up to hospital discharge; etiology-specific subgroup analysis; comparison with substantial published literature from multiple centers and countries.

Limitations: Single-center study limiting generalizability; relatively small sample size for certain subgroup analyses; short-term follow-up restricted to hospitalization (long-term complications and recurrence rates not assessed); no long-term nutritional outcomes; limited assessment of alcohol use disorder severity in alcohol-induced group; no evaluation of cost-effectiveness or healthcare resource utilization.

Conclusion

Alcohol-induced cases require heightened surveillance for necrosis complications, while gallstone cases necessitate planned ERCP intervention, cholecystectomy timing, and imaging follow-up for pseudocyst identification. The favorable outcomes observed in this study, combined with emerging data from other tertiary care centers demonstrating comparable outcomes between alcohol and gallstone-induced pancreatitis regardless of etiology, suggest that optimized supportive care, early intervention, and multispecialty team management represent the

cornerstones of successful acute pancreatitis management, transcending etiology-based distinctions.

Future investigations should evaluate long-term outcomes beyond hospital discharge, including recurrence rates, chronic pancreatitis development, pseudocyst complications, and functional outcomes. Research examining whether etiology-specific management protocols (enhanced surveillance for alcohol-induced, early ERCP for gallstone-induced) improve long-term outcomes compared to standardized approaches would provide valuable guidance for clinical practice. Additionally, studies investigating cost-effectiveness and healthcare resource utilization patterns between etiologies would inform resource allocation strategies in healthcare systems.

This study contributes to the growing evidence supporting the concept that "outcome of acute pancreatitis appears largely independent of etiology," encouraging clinicians to base prognostication and management intensity on severity assessment scores rather than etiology alone, while maintaining etiology-specific vigilance for anticipated complications.

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