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ORIGINAL ARTICLE

Etiology of acute pancreatitis in a tertiary care teaching hospital.

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ABSTRACT… Objective: To determine the frequency of etiological factors of acute pancreatitis at a tertiary care hospital, and their association, particularly gallstone disease, with demographic/clinical characteristics. **Study Design:** Descriptive, Prospective Observational study. **Setting:** General Surgery, MTI–Lady Reading Hospital, Peshawar. **Period:** Six Months (1st July–31st December 2025). **Methods:** Patients aged 30–70 with abdominal pain plus amylase >300 U/L and lipase >480 U/L were enrolled after consent, differing from the Revised Atlanta Classification; duct dilation, pseudocyst, bleeding disorders, and end-stage renal/liver disease were excluded. Gallstones, idiopathic disease, autoimmune disease, dyslipidaemia, and smoking were assessed (alcohol excluded); gallstone etiology was compared by Chi-square/Fisher's exact test ($p<0.05$). **Results:** Of 140 screened, 101 met the case definition (mean age 45.75±9.03 years; 54, 53.5% female). Gallstone disease was leading (54, 53.5%, 95% CI 43.8–62.9%; 61, 60.4% overall), followed by idiopathic disease (16, 15.8%), smoking alone (10, 9.9%), dyslipidaemia (16, 15.8%), autoimmune disease (2, 2.0%), and multiple factors (12, 11.9%); no significant demographic/clinical association emerged. **Conclusion:** Gallstone disease was commonest, followed by idiopathic disease, smoking, and dyslipidaemia. As the case definition differs from the Revised Atlanta Classification and alcohol history was unrecorded, findings warrant cautious interpretation.

Key words: Acute Pancreatitis, Dyslipidemias, Etiology, Gallstones, Idiopathic Diseases, Smoking, Tertiary Care Centers.

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INTRODUCTION

Acute pancreatitis is a common condition and a leading gastrointestinal cause of hospital admission.[1] The pancreas performs exocrine and endocrine functions: pancreatic juice from acinar cells drains via the pancreatic ducts, while insulin and glucagon from the islets of Langerhans enter the circulation directly.[2,3]

Its clinical course is highly variable, ranging from mild oedematous disease to severe necrotizing pancreatitis. The internationally accepted standard, the Revised Atlanta Classification, requires two of three features (typical pain, amylase/lipase ≥3× normal, or characteristic imaging) and grades severity by organ failure.[4] Reported mortality is ~3% in mild disease but rises to 20% with necrosis.[5] Predicting course and outcome remains difficult, and severity is occasionally under-recognised: roughly one in ten severe patients may die before diagnosis.[6,7] A recent global meta-analysis found incidence rising steadily over six decades, particularly in North America and Europe.[8]

Gallstones and alcohol together account for ~70% of cases, gallstones (including microlithiasis) alone causing 35–40%; reported gallstone-related prevalence ranges from 40–70% across populations, with alcohol the next commonest cause. Other recognised causes include post-ERCP and drug-induced pancreatitis, infections, toxins, hypertriglyceridaemia, hypercalcaemia, genetic/autoimmune disease, and vascular causes, alongside an idiopathic group with no identifiable cause.[9,10] A Pakistani tertiary-care series reported gallstones in 37% and idiopathic disease in 12%[11], while regional South Asian/East African series have similarly described autoimmune disease, dyslipidaemia, and smoking each contributing smaller proportions, with variation between settings.[12]

Given the rising global incidence linked to lifestyle change and metabolic disease, local etiological data can help clinicians identify at-risk populations, yet Pakistani data remain scarce. This study therefore determined the frequency of etiological factors of acute pancreatitis at our tertiary care teaching hospital and, secondarily, explored

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whether gallstone disease, the commonest cause, was associated with demographic or clinical characteristics.

METHODS

This descriptive, prospective observational study was conducted in the Department of General Surgery, MTI–Lady Reading Hospital, Peshawar, over six months (1st July–31st December 2025) after taking approvel from ethical committee (Ref: 533-LRH-MTI, Dated: 16-12-2024), with patients enrolled consecutively and assessed prospectively. A sample size of 136 was calculated using the WHO calculator, based on the previously reported frequency of dyslipidaemia (6%)[12], with 95% confidence and 4% margin of error; dyslipidaemia was chosen as the anchor proportion, being the least frequent reported category, to ensure adequate power for less common factors and not only gallstones. A total of 140 consecutive patients were screened using non-probability consecutive sampling.

Acute pancreatitis was operationally defined as abdominal pain (VAS>4) with tenderness, nausea, and vomiting, plus elevated serum amylase (>300 U/L) and lipase (>480 U/L) — differing from the Revised Atlanta Classification, which requires only two of three criteria (typical pain, a single enzyme ≥3× normal, or characteristic imaging);[4] this distinction is addressed in the Discussion. Patients aged 30–70 of either gender were eligible; those with dilated pancreatic duct, pseudocyst, bleeding disorder, end-stage renal disease, or chronic liver disease were excluded, since these could confound diagnosis or etiological assessment. Alcohol history was not part of the pre-specified protocol and was not recorded; this is discussed as a limitation.

After ethical approval and informed consent, demographic data (age, gender, BMI, education, occupation, residence, socioeconomic status) and histories of hypertension/diabetes were recorded on a standardised proforma. Each patient was examined by a surgeon with ≥5 years' post-fellowship experience for predefined etiological factors: gallstone disease (clinical plus ultrasound evidence of echogenic stones, posterior acoustic shadowing, or gallbladder wall thickening); idiopathic disease (no cause after comprehensive evaluation); autoimmune disease (positive ANA/ASCA serology or histological immune infiltration — more limited than the International Consensus Diagnostic Criteria, which also require IgG4, imaging, and steroid responsiveness[13]); dyslipidaemia (operationally, ≥2 of triglycerides >150 mg/dl, LDL >100 mg/dl, total cholesterol >200 mg/dl, or HDL <40 mg/dl — differing from the higher triglyceride thresholds, >500 or >1000 mg/dl, used for hypertriglyceridaemia-induced pancreatitis[14]); and smoking (any cigarette/cigar/pipe history, recorded as exposure rather than confirmed cause). A single researcher performed all data collection; proforma completeness ranged from 75.0–95.8% (mean 14.4% missing fields/patient), autoimmune serology/histology being the most incomplete domains.

Data were analysed in SPSS v.23. Numerical variables (age, BMI) were tested for normality (Shapiro-Wilk) and summarised as mean±SD or median (IQR); categorical variables were summarised as frequencies/percentages with 95% CI (Wilson score). Missing data were not imputed (available-case analysis). Gallstone etiology, the only adequately powered category, was stratified by age, gender, residence, occupation, education, hypertension, and diabetes using Chi-square/Fisher's exact test, $p<0.05$; very small categories (autoimmune, n=2) were not formally tested owing to inadequate power.

RESULTS

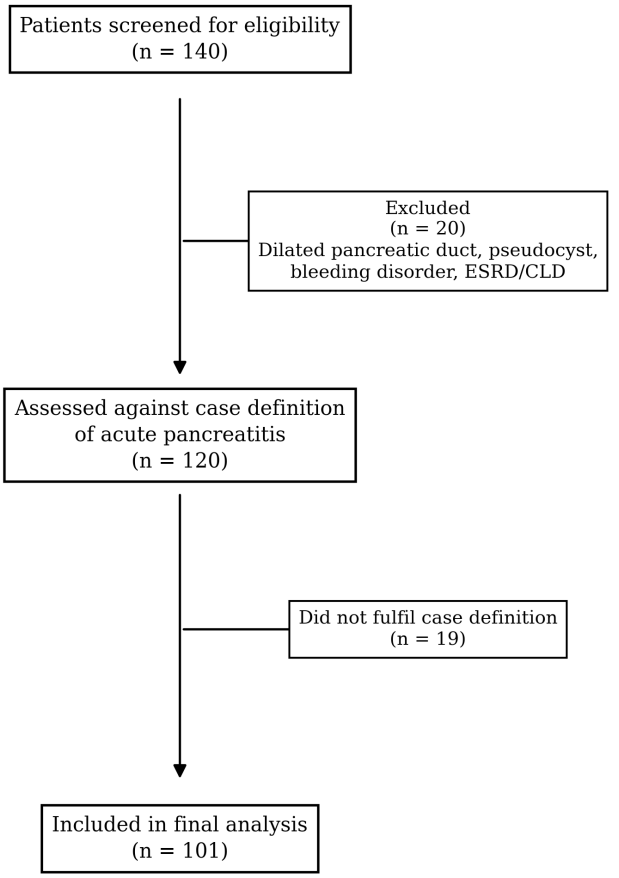
Of 140 patients screened, 20 were excluded and 19 did not meet the operational case definition; the remaining 101 formed the analytic sample (yield 72.1%; Figure-1).

Mean age was 45.75±9.03 years (median 45, IQR 39–53), with a mild female preponderance (54, 53.5% vs 47 male, 46.5%). Mean BMI was 27.65±4.16 kg/m^2, with 26 (25.7%) normal weight, 46 (45.5%) overweight, and 29 (28.7%) obese. Sixty-four (63.4%) were rural, 58 (57.4%) employed, and 51 (50.5%) illiterate; hypertension was present in 23 (22.8%) and diabetes in 34 (33.7%). All 101 patients were classified as lower socioeconomic class, reflecting this public-sector hospital's referral population rather than true absence of variability

(Table-I).

FIGURE-1

Flow of patients through the study, from screening to final analysis.



Etiological Factors of Acute Pancreatitis

Gallstone disease was the leading factor, the primary classification in 54/101 (53.5%, 95% CI 43.8–62.9%) and present, alone or with another factor, in 61 (60.4%, 95% CI 50.6–69.4%). Idiopathic disease accounted for 16 (15.8%). Smoking was the sole exposure in 10 (9.9%) as primary classification and present overall (alone or coexisting) in 13 (12.9%); as a recognized risk factor rather than a confirmed cause, this reflects exposure, not definitive attribution. Study-defined dyslipidaemia was primary in 7 (6.9%) and present overall in 16 (15.8%). A further 7 (6.9%) had multiple, non-dominant factors, and 7 (6.9%) had none identified. Autoimmune disease was found in only 2 (2.0%, 95% CI 0.5–6.9%); however, ANA was untested in 77 (76.2%), ASCA in 74 (73.3%), and histology unavailable in 97 (96.0%), so this is a minimum estimate. Full frequencies with 95% CIs appear in Table-II (primary classification) and Table-III (all identified factors).

Table-I

Demographic and clinical characteristics of the study population (n = 101).

Variable	Category	n (%)
Age (years)	Mean ± SD	45.75 ± 9.03
	Median (IQR)	45 (39–53)
Gender	Male	47 (46.5%)
	Female	54 (53.5%)
BMI (kg/m²)	Mean ± SD	27.65 ± 4.16
BMI category	Normal (18.5–24.9)	26 (25.7%)
	Overweight (25–29.9)	46 (45.5%)
	Obese (≥30)	29 (28.7%)
Residence area	Urban	37 (36.6%)
	Rural	64 (63.4%)
Occupation status	Employed	58 (57.4%)
	Unemployed	43 (42.6%)
Education status	Literate	50 (49.5%)
	Illiterate	51 (50.5%)
Socioeconomic status	Lower class	101 (100.0%)
Hypertension	Yes	23 (22.8%)
	No	78 (77.2%)
Diabetes mellitus	Yes	34 (33.7%)
	No	67 (66.3%)

TABLE-II

Primary etiological classification according to the study protocol.

Primary Etiological Classification (n = 101)	n	%	95% CI
Gallstone disease	54	53.5%	43.8–62.9%
Idiopathic	16	15.8%	10.0–24.2%
Smoking history (only identified exposure)	10	9.9%	5.5–17.3%
Multiple / overlapping etiological factors	7	6.9%	3.4–13.6%
Dyslipidaemia (study-defined)	7	6.9%	3.4–13.6%
No predefined etiological factor identified	7	6.9%	3.4–13.6%

TABLE-III

Identified etiological factors, including coexisting factors (not mutually exclusive).

Identified Etiological Factors (Any Patient Positive)	n	%	95% CI
Gallstone	61	60.4%	50.6–69.4%
Idiopathic	16	15.8%	10.0–24.2%
Dyslipidaemia (study-defined)	16	15.8%	10.0–24.2%
Smoking history (exposure)	13	12.9%	7.7–20.8%
Autoimmune	2	2.0%	0.5–6.9%
Two or more etiological factors co-existing	12	11.9%	6.9–19.6%

Overlap Between Identified Etiological Factors

Twelve patients (11.9%, 95% CI 6.9–19.6%) had more than one factor, most often gallstone with dyslipidaemia; given the sparse, uneven overlap across five categories, Table-IV lists the exact combinations rather than a Venn diagram.

TABLE-IV

Exact combinations of identified etiological factors observed.

Combination of Identified Etiological Factors	n	%
Gallstone only	49	48.5%
Idiopathic (no cause identified)	16	15.8%
Smoking exposure only	10	9.9%
Dyslipidaemia only	7	6.9%
Gallstone + Dyslipidaemia	7	6.9%
No predefined etiological factor identified	7	6.9%
Gallstone + Autoimmune	2	2.0%
Gallstone + Dyslipidaemia + Smoking Exposure	2	2.0%
Gallstone + Smoking Exposure	1	1.0%

Stratification of Gallstone Etiology

Gallstone etiology was stratified by gender, age, residence, occupation, education, hypertension, and diabetes (Table-V); autoimmune disease (n=2) was not stratified for inadequate power. Gallstone disease was numerically more common among females (68.5% vs 51.1% males), unemployed patients (72.1% vs 51.7%), and literate patients (70.0% vs 51.0%), but none of these, nor age, residence, hypertension, or diabetes, reached significance (all $p>0.05$), indicating no strong effect modification.

TABLE-V

Stratification of gallstone etiology by demographic and clinical variables.

Variable	Gallstone Present n (%)	Gallstone Absent n (%)	P-Value*
Gender-Male	24 (51.1%)	23 (48.9%)	0.113
Gender-Female	37 (68.5%)	17 (31.5%)	
Age 30-50 yrs	43 (59.7%)	29 (40.3%)	1.000
Age 51-70 yrs	18 (62.1%)	11 (37.9%)	
Residence-Urban	20 (54.1%)	17 (45.9%)	0.436
Residence-Rural	41 (64.1%)	23 (35.9%)	
Occupation-Employed	30 (51.7%)	28 (48.3%)	0.062
Occupation-Unemployed	31 (72.1%)	12 (27.9%)	
Education-Literate	35 (70.0%)	15 (30.0%)	0.080
Education-Illiterate	26 (51.0%)	25 (49.0%)	
Hypertension-Yes	12 (52.2%)	11 (47.8%)	0.500
Hypertension-No	49 (62.8%)	29 (37.2%)	
Diabetes-Yes	21 (61.8%)	13 (38.2%)	1.000
Diabetes-No	40 (59.7%)	27 (40.3%)	

*Chi-square test / Fisher's exact test, as appropriate; $p<0.05$ considered significant.

DISCUSSION

This study found gallstone disease to be the leading etiological factor of acute pancreatitis, accounting for just over half of cases as the primary classification and three in five when coexisting factors were included. This is broadly consistent with the literature (35–70% depending on population)[9,10] and aligns with a Pakistani series reporting gallstones in 37% and idiopathic disease in 12%.[11] Two other Pakistani tertiary-care series similarly identified biliary/gallstone disease as the largest group, followed by idiopathic and drug-induced disease.[15,16] The somewhat higher gallstone proportion and female preponderance observed

here are consistent with the recognized association between gallstone disease and female gender in South Asian populations.

An important limitation concerns the case definition, which required both amylase and lipase elevated above fixed thresholds rather than the Revised Atlanta Classification's two-of-three criteria (typical pain, either enzyme ≥3× normal, or imaging).[4] Patients meeting Atlanta criteria but not both biochemical thresholds may have been excluded, introducing possible selection bias and limiting comparison with guideline-based studies. Alcohol history, one of the two classical major causes alongside gallstones, was not part of the pre-specified protocol and was not recorded in any patient; some cases classified here as idiopathic (15.8%, toward the upper end of reported ranges) may in fact have been alcohol-related.

Smoking was the only identified exposure in 9.9% (primary) and 12.9% overall; we describe this as exposure rather than a confirmed diagnosis, since smoking is a recognised epidemiological risk factor but not, alone, a definitive per-patient cause. A meta-analysis found current smokers at significantly higher risk than never-smokers, with a dose-response relationship.[17] As a modifiable exposure, routinely documenting smoking history and offering cessation counselling remains a reasonable, low-cost measure regardless of its role in any individual case.

Study-defined dyslipidaemia was identified in 15.8% overall, higher than the 6% anchor used for sample-size calculation[12], likely because our operational definition required only two of four routine criteria rather than the substantially higher triglyceride thresholds (>500, often >1000 mg/dl) used to define hypertriglyceridaemia-induced pancreatitis.[14] Of the 16 patients meeting our criteria, 10 (62.5%) had triglycerides above 500 mg/dl and one above 1000 mg/dl, suggesting a majority are plausibly lipid-associated, while others likely reflect incidental dyslipidaemia; given that nearly three-quarters of the cohort were overweight or obese, this is a clinically relevant co-occurring finding, though our design cannot establish whether routine lipid profiling would alter management.

Autoimmune disease was identified in only 2 patients (2.0%) using ANA/ASCA positivity or histology, a more limited work-up than the International Consensus Diagnostic Criteria (IgG4, imaging, histology, steroid response).[13] Since ANA was untested in 76.2%, ASCA in 73.3%, and histology unavailable in 96.0% of the cohort, our figure is best regarded as a floor estimate.

Twelve patients (11.9%) had more than one factor, most often gallstone disease with dyslipidaemia (Table-IV), reinforcing that etiological work-up should continue even once an apparent cause is identified. On stratified analysis, none of gender, age, residence, occupation, education, hypertension, or diabetes was significantly associated with gallstone etiology, although it was numerically more common among women, unemployed, and literate patients (p=0.113, 0.062, and 0.080, respectively); given modest cell sizes, these findings are exploratory rather than confirmatory, and non-significance should not be over-interpreted as evidence of no association.

This single-centre, prospective observational study used non-probability consecutive sampling, limiting generalisability. Disease severity per the Revised Atlanta Classification was not recorded, so etiological differences by severity could not be evaluated. Data completeness ranged from 75.0–95.8%, autoimmune assessment being most incomplete, and several stratified comparisons (e.g., autoimmune, n=2) were underpowered. Larger, multicentre, prospective studies using guideline-concordant definitions, a validated hypertriglyceridaemia threshold, structured alcohol history, and systematic severity grading are recommended.

CONCLUSION

In this tertiary care teaching hospital population, gallstone disease was the commonest etiological factor of acute pancreatitis, followed by idiopathic disease, smoking exposure, and study-defined dyslipidaemia; autoimmune disease was uncommon but also the least completely investigated category. A meaningful proportion had more than one factor, and none of the demographic or clinical variables

studied was significantly associated with gallstone etiology. Because the case definition differs from the Revised Atlanta Classification, alcohol history was not captured, and autoimmune disease/severity could not be systematically assessed, these findings should be interpreted as local epidemiological data rather than directly generalisable practice recommendations. Future studies using guideline-based criteria, structured alcohol history, standardised autoimmune assessment, accepted hypertriglyceridaemia criteria, and systematic severity grading are required before clinical translation.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Bakir Khan:** Conceptualization, data collection, drafting.
2	**Waqar Alam Jan:** Supervision, proof reading.
3	**Muhammad Noman:** Data curation, data analysis.
4	**Mehmood Ahmad:** Data collection.