



Original Article

Role of C-Reactive Protein and Lactate Dehydrogenase in Predicting the Severity of Acute Pancreatitis

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ABSTRACT

Background: Acute pancreatitis exhibits a wide clinical spectrum ranging from mild self-limiting disease to severe, life-threatening illness. Early assessment of disease severity is essential for optimal management. Inflammatory biomarkers such as C-reactive protein (CRP) and lactate dehydrogenase (LDH) are widely available and may help predict disease severity and clinical outcomes.

Objectives: To evaluate the association of serum CRP and LDH levels with computed tomography (CT) findings and clinical outcomes, including intensive care unit (ICU) admission, in patients with acute pancreatitis.

Materials and Methods: This prospective observational study included 60 patients diagnosed with acute pancreatitis. Serum CRP and LDH levels were measured and correlated with alcohol intake, CT findings (focal inflammation, necrosis, peripancreatic fluid collection, and pseudocyst formation), and requirement for ICU admission. Statistical analysis was performed using appropriate parametric tests, and a p-value <0.05 was considered statistically significant.

Results: The mean serum CRP and LDH levels were 141.33 ± 25.44 mg/ml and 494.67 ± 266.19 IU/L, respectively. CRP and LDH levels did not show a statistically significant association with alcohol consumption. However, both biomarkers demonstrated a significant correlation with CT findings, with higher levels observed in patients with pancreatic necrosis and pseudocyst formation ($p < 0.0001$). While CRP levels were not significantly associated with ICU admission, LDH levels were significantly higher in patients requiring ICU care ($p = 0.0079$).

Conclusion: Serum CRP and LDH levels correlate well with radiological severity in acute pancreatitis. LDH, in particular, is a strong predictor of severe disease and ICU requirement. These biomarkers can serve as valuable, cost-effective tools for early severity assessment and risk stratification in acute pancreatitis.

Keywords: Acute pancreatitis, C-reactive protein (CRP), Lactate dehydrogenase (LDH), Computed tomography (CT), Disease severity, Intensive care unit (ICU) admission.

INTRODUCTION

Acute pancreatitis is a common and potentially life-threatening gastrointestinal emergency characterised by acute inflammation of the pancreas, with variable involvement of regional tissues and distant organ systems. The global incidence of acute pancreatitis ranges from 13 to 45 cases per 100,000 population per year, with a rising trend attributed to increased alcohol consumption and gallstone disease [1,2]. While most cases follow a mild, self-limiting course, approximately 15–20% of patients develop severe acute pancreatitis associated with pancreatic necrosis, systemic inflammatory response syndrome (SIRS), organ failure, and significant mortality [3].

Early identification of patients likely to develop severe disease remains a major clinical challenge. Severe acute pancreatitis is associated with prolonged hospital stay, increased need for intensive care unit (ICU) admission, and higher morbidity and mortality rates [4]. Hence, prompt risk stratification at admission is crucial for timely intervention, appropriate triage, and improved outcomes.

Several clinical scoring systems, such as Ranson's criteria, APACHE II, BISAP, and the Revised Atlanta Classification, have been developed to predict disease severity. However, these scoring systems are often complex, time-consuming, require multiple variables, and may not be readily applicable in all clinical settings, particularly in resource-limited hospitals [5,6]. Consequently, there has been increasing interest in simple, easily available biochemical markers that can reliably predict disease severity early in the course of acute pancreatitis.

C-reactive protein (CRP) is an acute-phase reactant synthesised by the liver in response to inflammatory cytokines, particularly interleukin-6. CRP levels correlate with the intensity of inflammation and tissue necrosis and have been extensively studied as a prognostic marker in acute pancreatitis. Elevated CRP levels, especially values exceeding 150 mg/L within the first 48 hours, have been shown to be associated with pancreatic necrosis and severe disease [7,8]. Due to its wide availability, low cost, and reproducibility, CRP remains one of the most commonly used biomarkers in clinical practice.

Lactate dehydrogenase (LDH) is a cytoplasmic enzyme released during tissue injury and cellular necrosis. Elevated LDH levels reflect the extent of pancreatic and extrapancreatic tissue damage and have been incorporated into several severity scoring systems for acute pancreatitis, including Ranson's criteria [9]. Studies have demonstrated that increased LDH levels are associated with pancreatic necrosis, organ failure, and poor prognosis [10,11]. Despite its potential utility, LDH remains underutilised as a standalone prognostic marker.

Contrast-enhanced computed tomography (CECT) plays a pivotal role in assessing the morphological severity of acute pancreatitis, including the detection of pancreatic necrosis, peripancreatic fluid collections, and local complications [12]. Correlating biochemical markers such as CRP and LDH with CT findings and clinical outcomes, including ICU admission, may provide valuable insights into their predictive value.

Given the burden of acute pancreatitis and the need for early, reliable, and cost-effective predictors of disease severity, this study was undertaken to evaluate the role of C-reactive protein and lactate dehydrogenase in predicting the severity of acute pancreatitis and to assess their association with radiological findings and ICU admission.

MATERIALS AND METHODS

Study Area

The present study was conducted in the Department of General Surgery, Bokaro General Hospital, Bokaro Steel City, Jharkhand, a tertiary care referral centre.

Study Design

This was a prospective observational study.

Study Duration

The study was carried out over a period of 18 months.

Study Population

The study population comprised patients admitted to Bokaro General Hospital with a diagnosis of acute pancreatitis, fulfilling the predefined inclusion and exclusion criteria.

Sample Size

Based on a reference study titled "*Quantification Analysis of Lactate Dehydrogenase and C-Reactive Protein in Evaluation of the Severity and Prognosis of Acute Pancreatitis*", the accordance rate for predicting the development of acute pancreatitis was reported as 84.3% for C-reactive protein (CRP) at a cut-off value of 176 mg/dL, and 90.4% for lactate dehydrogenase (LDH) at a cut-off value of 235.5 U/L.

For calculation of the minimum sample size, the maximum proportion ($P = 0.843$) was considered. Using Cochran's formula for observational studies:

$$N = \frac{Z^2 \times P(1 - P)}{e^2}$$

Where:

- $Z = 1.96$ (95% confidence interval)
- $P = 0.843$
- $e = 0.10$ (allowable error)

$$N = \frac{(1.96)^2 \times 0.843 \times (1 - 0.843)}{(0.10)^2}$$

$$N = 51$$

Thus, the minimum required sample size was 51 patients. To compensate for possible data loss and dropouts, a total of 60 patients were included in the study.

Inclusion Criteria

1. Patients diagnosed with acute pancreatitis based on clinical presentation, laboratory investigations, and imaging findings.
2. Patients presenting to the hospital within a defined time frame from the onset of symptoms to ensure biomarker estimation during the acute phase.
3. Patients aged ≥ 18 years.
4. Patients who had not received prior treatment for acute pancreatitis before hospital admission.
5. Patients who provided written informed consent to participate in the study.

Exclusion Criteria

1. Patients with a history of chronic pancreatitis.
2. Patients with acute on chronic pancreatitis.
3. Patients with chronic inflammatory or autoimmune diseases that could alter CRP levels.
4. Patients with known malignancies, which may influence LDH levels.
5. Pregnant patients.
6. Patients with known chronic liver disease.
7. Patients aged < 18 years.
8. Patients who had received treatment for acute pancreatitis prior to admission.

Method of Data Collection

After obtaining informed consent, detailed demographic and clinical data were collected using a structured proforma. This included:

- Detailed medical history
- Thorough clinical examination
- Laboratory investigations
- Radiological evaluation
- Clinical follow-up during hospital stay

Investigations

All patients underwent the following investigations:

Routine Laboratory Investigations:

- Complete blood count (CBC)
- Renal function tests (RFT)
- Serum electrolytes
- Liver function tests (LFT)
- Random blood sugar (RBS)
- Serum calcium

Specific Biochemical Investigations:

- Serum amylase
- Serum lipase
- Serum C-reactive protein (CRP)
- Serum lactate dehydrogenase (LDH)

Radiological Investigations:

- Plain X-ray abdomen
- Chest X-ray
- Ultrasonography (USG) of abdomen
- Contrast-enhanced computed tomography (CT) of abdomen, where indicated

Data Management and Statistical Analysis

All collected data were entered into a Microsoft Excel spreadsheet and analysed using Statistical Package for Social Sciences (SPSS) software, version 20.0.

Continuous variables were expressed as mean \pm standard deviation (SD), while categorical variables were expressed as frequencies and percentages. Data were tested for normality before analysis. Normally distributed continuous variables

were compared using the Student's t-test, while non-normally distributed variables were analysed using the Mann–Whitney U test. Categorical variables were analysed using the Chi-square test or Fisher's exact test, as appropriate. A p-value <0.05 was considered statistically significant, and a p-value <0.01 was considered highly significant.

RESULTS AND OBSERVATIONS

Table 1: Demographic Distribution of Study Participants (Age and Sex)

Variable	Category	Frequency (n=60)	Percentage (%)
Age Group (years)	≤20	1	1.7
	21–30	16	26.7
	31–40	14	23.3
	41–50	7	11.7
	51–60	9	15.0
	61–70	10	16.7
	≥71	3	5.0
Sex	Male	56	93.3
	Female	4	6.7

Table 2: Clinical, Biochemical, Radiological and Descriptive Characteristics of Study Participants (n = 60)

Variable	Category / Parameter	Frequency / Value	Percentage / Statistics
Alcohol Consumption	Yes	51	85.0%
	No	9	15.0%
Serum Amylase	<140 U/L	16	26.7%
	≥140 U/L	44	73.3%
CT Findings	Focal inflammation	13	21.7%
	Necrosis	24	40.0%
	Peripancreatic fluid collection	20	33.3%
	Pseudocyst	3	5.0%
Age (years)	Mean ± SD	44.98 ± 16.02	—
	Minimum – Maximum	19 – 79	—
	Median	40	—
Duration of Abdominal Pain (days)	Mean ± SD	2.20 ± 0.86	—
	Minimum – Maximum	1 – 4	—
	Median	2	—

Table 3: Distribution of Inflammatory and Enzymatic Biomarkers (CRP and LDH) in Study Participants (n = 60)

Parameter	Number	Mean ± SD	Minimum	Maximum	Median
C-Reactive Protein (mg/mL)	60	141.33 ± 25.44	86.0	190.0	148.0
Lactate Dehydrogenase (IU/L)	60	494.67 ± 266.19	120.0	1220.0	405.0

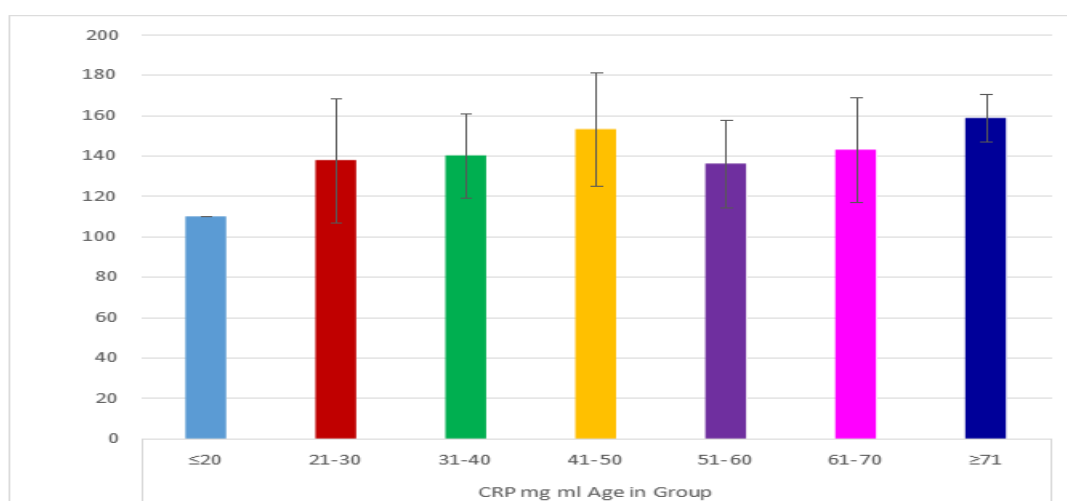


Figure 1: Distribution of mean CRP mg/ ml: Age in Group

Table 4: Distribution of Serum LDH Levels (IU/L) According to Age Group (n = 60)

Age Group (years)	Number (n)	Mean ± SD (IU/L)	Minimum	Maximum	Median
≤20	1	260.00 ± 0.00	260	260	260

21–30	16	488.13 ± 331.37	120	1110	380
31–40	14	567.86 ± 308.80	280	1220	435
41–50	7	482.86 ± 179.97	300	760	410
51–60	9	388.89 ± 187.51	200	750	330
61–70	10	484.00 ± 225.40	200	820	515
≥71	3	646.67 ± 170.39	470	810	660

Statistical test: One-way ANOVA
p-value: 0.6586 (Not significant)

Table 5: Comparison of Mean CRP and LDH Levels According to Alcohol Consumption (n = 60)

Parameter	Alcohol Intake	Number (n)	Mean ± SD	Minimum	Maximum	Median	t-value	p-value
CRP (mg/mL)	No	9	136.11 ± 20.40	108	172	125	0.665	0.509
	Yes	51	142.25 ± 26.29	86	190	148		
LDH (IU/L)	No	9	505.56 ± 340.52	200	1220	410	0.132	0.895
	Yes	51	492.75 ± 255.01	120	1110	400		

Table 6: Association of Mean CRP and LDH Levels with CT Findings and ICU Admission (n = 60)

A. Biomarkers According to CT Findings

CT Finding	N	CRP (mg/mL) Mean ± SD	Median	LDH (IU/L) Mean ± SD	Median
Focal inflammation	13	128.00 ± 31.12	96	393.08 ± 205.97	220
Necrosis	24	160.88 ± 13.54	160	678.75 ± 261.54	605
Peripancreatic fluid collection	20	121.25 ± 8.32	122	286.00 ± 54.81	295
Pseudocyst	3	160.00 ± 32.19	154	623.33 ± 187.17	660

Statistical test: Kruskal–Wallis test

- CRP: Z = 4.0567, p < 0.0001 (Significant)
- LDH: Z = 6.0143, p < 0.0001 (Highly significant)

Table B. Biomarkers According to ICU Admission Status

Parameter	Admission Status	n	Mean ± SD	Minimum	Maximum	Median	t-value	p-value
CRP (mg/mL)	ICU	25	147.16 ± 29.07	86	186	158	1.516	0.135
	Ward	35	137.17 ± 21.99	106	190	128		
LDH (IU/L)	ICU	25	600.80 ± 322.94	120	1220	540	2.752	0.0079
	Ward	35	418.86 ± 187.46	200	820	350		

DISCUSSION

Acute pancreatitis presents with a wide spectrum of clinical severity, ranging from mild self-limiting inflammation to severe disease complicated by pancreatic necrosis, organ failure, and death. Early prediction of disease severity remains a cornerstone in the management of acute pancreatitis, as it allows timely intensive monitoring, early intervention, and appropriate utilisation of healthcare resources. The present study was undertaken to evaluate the role of C-reactive protein (CRP) and lactate dehydrogenase (LDH) as simple, readily available biochemical markers in predicting the severity of acute pancreatitis.

Demographic and Clinical Profile

In the present study, the majority of patients were middle-aged adults, with a mean age of 44.98 ± 16.02 years, and a marked male predominance (93.3%). This demographic profile is consistent with several Indian and international studies, which report a higher incidence of acute pancreatitis among males, largely attributed to alcohol consumption [13,14]. Alcohol was identified as the most common etiological factor in the present study (85%), reinforcing its significant role in the pathogenesis of acute pancreatitis in the Indian population.

CRP as a Marker of Disease Severity

CRP is a well-established acute-phase reactant that reflects the intensity of systemic inflammation. In the present study, the mean CRP level was 141.33 ± 25.44 mg/mL, indicating a substantial inflammatory response among the study population. Although CRP levels did not show a statistically significant association with alcohol consumption or ICU admission, a strong and statistically significant correlation was observed between CRP levels and CT findings (p < 0.0001).

Patients with pancreatic necrosis and pseudocyst formation demonstrated significantly higher CRP levels compared to those with focal inflammation or peripancreatic fluid collection. These findings are in agreement with earlier studies that reported elevated CRP levels in severe acute pancreatitis and pancreatic necrosis [7,15]. Puolakkainen et al. demonstrated that CRP levels exceeding 150 mg/L within 48 hours were strongly associated with pancreatic necrosis and severe disease [8]. Similarly, Cardoso et al. emphasized the prognostic value of CRP when measured after 48 hours of symptom onset [7].

The lack of a statistically significant association between CRP levels and ICU admission in the present study may be attributed to early supportive management and timely intervention, which could have mitigated disease progression in some patients.

LDH as a Marker of Tissue Injury and Severity

LDH is a marker of cellular injury and tissue necrosis and has been included in traditional prognostic scoring systems such as Ranson's criteria. In the present study, the mean LDH level was 494.67 ± 266.19 IU/L, with significantly higher values observed in patients with pancreatic necrosis, pseudocyst, and those requiring ICU admission.

A statistically significant association was found between LDH levels and CT findings ($p < 0.0001$), as well as ICU admission ($p = 0.0079$). Patients admitted to the ICU had markedly higher mean LDH levels compared to those managed in the general ward, indicating that LDH is a sensitive marker of severe disease. These findings are consistent with previous studies that identified elevated LDH levels as a predictor of pancreatic necrosis, organ failure, and mortality [9,16].

De Campos et al. reported that LDH was independently associated with mortality in severe acute pancreatitis [11]. Similarly, Ranson et al. highlighted the prognostic importance of LDH in the early phase of the disease [9]. The present study supports the utility of LDH as a simple and effective biochemical marker for early severity assessment.

Correlation with Radiological Severity

Contrast-enhanced CT remains the gold standard for assessing morphological severity in acute pancreatitis. In the present study, pancreatic necrosis was the most common CT finding (40%), followed by peripancreatic fluid collection. Both CRP and LDH levels showed a strong correlation with CT severity, particularly in patients with necrosis and pseudocyst formation.

These findings are consistent with the observations of Balthazar et al., who demonstrated that biochemical markers correlate well with CT severity index and clinical outcomes [12]. The combined use of biochemical markers and imaging findings enhances the accuracy of early severity prediction.

Clinical Implications

The findings of the present study suggest that CRP and LDH are valuable, cost-effective, and easily available markers that can aid in the early prediction of disease severity in acute pancreatitis. While CRP correlates well with radiological severity, LDH appears to be a stronger predictor of clinical severity and ICU requirement. Their combined use may serve as a practical alternative to complex scoring systems, particularly in resource-limited settings.

Limitations

The present study has certain limitations, including a relatively small sample size and its single-centre design, which may limit the generalizability of the findings. Serial measurement of CRP and LDH was not performed, which could have provided additional prognostic information. Further multicentric studies with larger sample sizes are warranted to validate these findings.

CONCLUSION

CRP and LDH are useful, readily available biomarkers for assessing disease severity in acute pancreatitis. Elevated CRP correlates significantly with CT-detected complications, while raised LDH is strongly associated with severe radiological findings and ICU admission. Their combined use enables early risk stratification and supports timely clinical decision-making, particularly in resource-limited settings.

REFERENCES

1. Banks PA, Freeman ML. Practice guidelines in acute pancreatitis. *Am J Gastroenterol.* 2006;101(10):2379–2400.
2. Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology.* 2013;144(6):1252–1261.
3. Fagenholz PJ, Castillo CF, Harris NS, et al. Increasing United States hospital admissions for acute pancreatitis, 1988–2003. *Ann Surg.* 2007;245(3):474–482.

4. Tenner S, Baillie J, DeWitt J, Vege SS. American College of Gastroenterology guideline: management of acute pancreatitis. *Am J Gastroenterol*. 2013;108(9):1400–1415.
5. Ranson JHC, Rifkind KM, Roses DF, et al. Prognostic signs and the role of operative management in acute pancreatitis. *SurgGynecol Obstet*. 1974;139:69–81.
6. Papachristou GI, Muddana V, Yadav D, et al. Comparison of BISAP, Ranson's, APACHE-II, and CTSI scores in predicting organ failure and mortality in acute pancreatitis. *Am J Gastroenterol*. 2010;105(2):435–441.
7. Cardoso FS, Ricardo LB, Oliveira AM, et al. C-reactive protein prognostic accuracy in acute pancreatitis: timing of measurement and cutoff points. *Eur J Gastroenterol Hepatol*. 2013;25(7):784–789.
8. Puolakkainen P, Valtonen V, Paananen A, Schröder T. C-reactive protein and serum phospholipase A2 in the assessment of severity of acute pancreatitis. *Gut*. 1987;28(7):764–771.
9. Ranson JHC. Etiological and prognostic factors in human acute pancreatitis: a review. *Am J Gastroenterol*. 1982;77(9):633–638.
10. Muddana V, Whitcomb DC, Khalid A, et al. Elevated serum creatinine as a marker of pancreatic necrosis in acute pancreatitis. *Am J Gastroenterol*. 2009;104(1):164–170.
11. De Campos T, Cerqueira C, Kuryura L, et al. Mortality predictors in severe acute pancreatitis. *JOP*. 2008;9(6):690–695.
12. Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. *Radiology*. 2002;223(3):603–613.
13. Negi N, Mokta J, Sharma B, et al. Clinical profile and outcome of acute pancreatitis: A hospital-based study. *J Assoc Physicians India*. 2015;63(3):30–34.
14. Garg PK, Tandon RK. Survey on chronic pancreatitis in the Asia-Pacific region. *J Gastroenterol Hepatol*. 2004;19(9):998–1004.
15. Wilson C, Heads A, Shenkin A, Imrie CW. C-reactive protein, antiproteases, and complement factors as objective markers of severity in acute pancreatitis. *Br J Surg*. 1989;76(2):177–181.
16. Larvin M, McMahon MJ. APACHE-II score for assessment and monitoring of acute pancreatitis. *Lancet*. 1989;334(8656):201–205.