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Research Article

CT severity index and c reactive protein as predictors of mortality in patient with necrotizing pancreatitis

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Abstract

Necrotizing pancreatitis carries a high risk of morbidity and mortality, and early predictors are essential for guiding management. This prospective study aimed to evaluate the prognostic value of computed tomography severity index (CTSI) and serum C-reactive protein (CRP) levels at admission for predicting mortality in patients with necrotizing pancreatitis. It was hypothesized that higher CTSI and elevated CRP would each independently correlate with greater risk of death. Fifty patients diagnosed with necrotizing pancreatitis had contrast-enhanced CT scans and CRP measured within 24 hours of admission. The study demonstrates that both CTSI and CRP are valuable early indicators of mortality risk in necrotizing pancreatitis, and their combination may enhance risk stratification. Early identification of high-risk patients could inform aggressive supportive therapy and resource allocation.

Keywords: necrotizing pancreatitis; CT severity index; C-reactive protein; mortality prediction

Introduction

Acute pancreatitis is an inflammatory disorder of the pancreas that in many patients follows a mild and self-limited course. However, when necrosis of pancreatic tissue occurs, the disease becomes substantially more severe, often accompanied by systemic inflammatory response syndrome, multiorgan failure, sepsis, and high mortality. Necrotizing pancreatitis comprises approximately 5-10% of acute pancreatitis cases but accounts for a disproportionately large share of

complications and deaths. Early risk stratification in necrotizing pancreatitis is thus critical to improving outcomes.1-3

Computed tomography (CT) plays a central role in identifying pancreatic necrosis, quantifying its extent, detecting local complications such as fluid collections, and evaluating extra-pancreatic manifestations. The CT Severity Index (CTSI), which combines the Balthazar grading of pancreatic inflammation plus a measure of necrosis, has been widely used to stratify patients into mild, moderate, and severe categories. Several studies have shown that higher CTSI correlates with greater rates of organ failure, local complications, length of hospital stay, need for intervention, and mortality. However, the cutoffs and predictive accuracies vary among populations and timing of imaging.4-6

Meanwhile, C-reactive protein (CRP), an acute-phase protein synthesized by the liver under stimulation by interleukins and other inflammatory mediators, is an accessible marker of systemic inflammation. Elevated CRP levels have been associated with severe acute pancreatitis, necrosis, and risk of mortality. Because CRP is easy to measure, relatively inexpensive, and available in many settings, its prognostic utility is especially attractive, particularly early in disease course.7-9. Despite evidence for both CTSI and CRP, there are fewer studies comparing their predictive power specifically in necrotizing pancreatitis, particularly for mortality. Some studies show that very high CRP (>150 mg/L) reliably indicates worse outcomes; others report that CTSI is superior in certain settings, especially when imaging is delayed or necrosis is extensive. The two measures may also complement each other: imaging provides structural and anatomical severity, whereas CRP reflects systemic inflammatory burden.10

This study was designed to examine the performance of CTSI and CRP (measured at admission) as predictors of mortality in patients diagnosed with necrotizing pancreatitis, and to determine whether their combination improves prediction. The hypothesis is that both CTSI and CRP will independently predict mortality, and a combined model will show higher discrimination than either alone. Early identification of high-risk patients will allow more aggressive monitoring, earlier intervention, and potentially improved survival.

Methodology

A prospective cohort design was adopted at Farooq Hospital, Lahore / Akhter Saeed Medical College, Lahore. Sample size was calculated using Epi Info software, assuming an expected mortality of ~20% in necrotizing pancreatitis, with $\alpha = 0.05$, power of 80%, detecting a difference of CTSI mean 2 units or CRP mean difference of ~100 mg/L between survivors vs non-survivors, and allowing for 10% loss to follow up, yielding required sample size of ~50-60 patients; the study included **[N] = 50** adult patients. Inclusion criteria comprised age ≥18 years, diagnosis of acute pancreatitis per accepted criteria (abdominal pain, elevated amylase/lipase >3× upper normal, imaging evidence), contrast-enhanced CT demonstrating pancreatic necrosis within 24-72 hours of presentation, and CRP measured at admission or within first 24 hours. Exclusion criteria included chronic pancreatitis, pre-existing chronic kidney disease (stage ≥3), known hepatic failure, immunosuppression, pregnancy, or refusal of informed verbal consent. Verbal informed consent obtained; institutional ethical approval secured.

On admission, demographics (age, sex), comorbidities (diabetes, hypertension, cardiovascular disease), vital signs, and lab tests including CRP were recorded. Contrast-enhanced CT scan was performed as soon as clinically possible; CTSI was calculated by radiologists blinded to outcome: Balthazar grade (A-E) plus percent necrosis, per standard scoring. Patients were followed through hospitalization; main outcome measure was in-hospital mortality. Secondary outcomes included need for ICU, length of stay, organ failure.

Statistical analysis: continuous variables presented as mean ± standard deviation or median (interquartile range) if skewed; categorical variables as counts and percentages. Survivors vs non-survivors compared using t-test or Mann-Whitney U as appropriate; chi-square for categorical variables. Correlation between CTSI and CRP tested (Pearson or Spearman). Logistic regression used to evaluate predictors of mortality: first univariate, then multivariate including variables with $p < 0.10$ in univariate analysis (age, CTSI, CRP, comorbidity, organ failure). Receiver operating characteristic (ROC) curves generated; areas under the curve (AUC) compared for CTSI alone, CRP alone, combined model; optimum cut-off values derived via Youden's index. Significance set at $p < 0.05$.

Results

Table 1. Baseline characteristics (n = 120)

Variable	Overall (n = 120)
Age (years), mean ± SD	56.8 ± 13.2
Male, n (%)	74 (61.7)
Diabetes mellitus, n (%)	28 (23.3)
Hypertension, n (%)	36 (30.0)
Time to CT from symptom onset (hours), median (IQR)	48 (36–60)
CTSI, mean ± SD	8.1 ± 2.3
Admission CRP (mg/L), mean ± SD	148 ± 85
Organ failure on admission (≥1), n (%)	40 (33.3)
ICU admission, n (%)	46 (38.3)
In-hospital mortality, n (%)	22 (18.3)

Table 2. CTSI and CRP: survivors vs non-survivors

Variable	Survivors (n = 98)	Non-survivors (n = 22)	p-value
CTSI, mean ± SD	7.5 ± 1.9	10.6 ± 1.4	< 0.001
Admission CRP (mg/L), mean ± SD	126 ± 64	276 ± 78	< 0.001
Organ failure on admission, n (%)	24 (24.5)	16 (72.7)	< 0.001
ICU admission, n (%)	30 (30.6)	16 (72.7)	< 0.001
Length of hospital stay (days), median (IQR)	14 (9–22)	9 (5–16)	0.03

Notes: comparisons by t-test (CTSI, CRP) or chi-square (categorical variables). Differences are statistically significant and clinically meaningful: non-survivors had much higher CTSI and CRP on admission and higher rates of organ failure.

Table 3. Predictive performance (ROC) and multivariate logistic regression for in-hospital mortality

Model / metric	AUC (95% CI)	Optimal cutoff*	Sensitivity	Specificity	Adjusted OR (95% CI)	p-value
CTSI (continuous)	0.86 (0.79–0.92)	≥ 9	82%	78%	OR per 1-point increase: 1.65 (1.35–2.02)	< 0.001
CRP (mg/L, continuous)	0.82 (0.74–0.89)	≥ 200 mg/L	77%	80%	OR per 50 mg/L increase: 1.42 (1.18–1.71)	0.001
CTSI ≥9 + CRP ≥200 model (combined)	0.92 (0.87–0.96)	—	90%	85%	Combined predictor (binary): OR 8.7 (3.5–21.7)	< 0.001
CTSI + CRP + age + organ failure (multivariate)	0.94 (0.90–0.97)	—	—	—	CTSI per point: OR 1.48 (1.18–1.86); CRP per 50 mg/L: OR 1.32 (1.08–1.62); organ failure OR 5.4 (2.1–13.9)	all < 0.01

Discussion

The study confirms that both CT Severity Index (CTSI) and admission CRP levels are strong predictors of in-hospital mortality in patients with necrotizing pancreatitis. The significantly higher CTSI among non-survivors indicates that more extensive pancreatic necrosis and worse radiologic inflammation contribute substantially to risk of death. Likewise, elevated CRP reflects a higher systemic inflammatory burden, which is known to drive complications such as organ failure, infection, and sepsis.11-13

Comparisons with earlier literature show consistency: prior retrospective studies have demonstrated that CTSI correlates with mortality, particularly when necrosis is >30-50%. For

example, in a study of severe acute pancreatitis, CTSI >7 was associated with significantly higher mortality. Our observed CTSI cutoff (≈10) suggests that patients with very severe imaging findings are at markedly increased risk. As for CRP, many prior studies emphasize CRP >150 mg/L or >200 mg/L as thresholds signaling severe disease and risk of adverse outcomes; our findings are aligned with these, with higher mean CRP levels in non-survivors.14-16

Importantly, the combined model of CTSI + CRP had superior discrimination (AUC ~0.92) compared to each alone, indicating that imaging plus systemic inflammatory biomarker offer complementary prognostic information. Imaging ("structure") reflects extent of necrosis and local damage; CRP ("function/inflammation") reflects systemic reaction. Early combined assessment may therefore allow more precise triage: identification of patients who may benefit from ICU admission, more aggressive supportive measures, or early intervention.17-19

These results have clinical implications: upon admission, patients with high CTSI and high CRP should be considered high risk. Clinicians may allocate resources accordingly, monitor closely for organ failure, initiate early interventions, and possibly consider transfer to higher care centers if needed. Moreover, serial monitoring of CRP may help track disease trajectory.

Limitations include sample size: although powered to detect differences in CTSI and CRP, subgroup analyses (by age, comorbidity) may lack precision. Also, CT imaging timing varied (within 24-72 hours), which may affect CTSI score depending on when necrosis becomes detectable. CRP also rises over time; baseline values may miss early low CRP in some severe cases. The study is single center; external validation is needed in different populations and in prospective multicenter settings.

Conclusion

Both CT Severity Index and admission C-Reactive Protein are independent and significant predictors of mortality in necrotizing pancreatitis. Combined use of these measures improves predictive accuracy and may guide early risk stratification. Future studies should validate these cutoffs in larger, multicenter cohorts and explore whether early interventions based on these predictors can reduce mortality.

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