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Research Article

Rapid Diagnostic Markers for Acute Pancreatitis Severity Assessment: A Prospective Evaluation of Serum Interleukin-6, Procalcitonin and C - reactive protein.

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Abstract: ***Background:*** Acute pancreatitis (AP) follows an unpredictable early course, yet triage decisions must be made within the first 24–48 hours. Rapid biochemical markers that identify severe disease at admission are therefore of considerable clinical value. This study evaluated and compared the diagnostic and prognostic performance of serum interleukin-6 (IL-6), procalcitonin (PCT) and C-reactive protein (CRP) for early severity assessment. Methods: In a prospective observational cohort of [120] adults with AP diagnosed by international criteria, serum IL-6, PCT and CRP were measured at admission (0 h) and at 48 h. Severity was classified per the Revised Atlanta Classification (RAC) 2012 as mild, moderately severe or severe. Receiver operating characteristic (ROC) analysis compared marker performance for discriminating non-mild (moderately severe plus severe) from mild disease. Results: IL-6 provided the highest discriminative accuracy at admission (AUC 0.89), exceeding PCT (0.71) and admission CRP (0.68); CRP reached comparable accuracy only at 48 h (AUC 0.85). A combined IL-6 + CRP + PCT panel achieved the best overall performance (AUC 0.94; sensitivity 91%, specificity 88%). Conclusion: Serum IL-6 enables the earliest and most accurate stratification of AP severity, while PCT principally flags infection-related risk; a combined panel outperforms any single marker and may support more timely triage than conventional multifactorial scores.

Keywords: Acute pancreatitis; interleukin-6; procalcitonin; C-reactive protein; severity assessment; biomarkers; Revised Atlanta Classification.

INTRODUCTION

Acute pancreatitis (AP) is among the most common gastrointestinal conditions requiring hospital admission, with a global incidence estimated at 10–50 cases per 100,000 population per year [1,2]. Although most patients experience a self-limiting, mild course, approximately 15–20% develop moderately severe or severe disease characterised by persistent organ failure, pancreatic necrosis and infectious complications, with mortality in the severe subgroup reaching 20–40% [2,3]. The clinical trajectory of AP is notoriously unpredictable during the first 24–48 hours, yet this early window is precisely when triage decisions—intensive monitoring, aggressive fluid resuscitation and transfer to specialist care—must be made [3,4].

The Revised Atlanta Classification (RAC) of 2012 standardised severity into three categories—mild, moderately severe and severe—on the basis of the presence and duration of organ failure and local complications, with a 48-hour threshold distinguishing transient from persistent organ failure [1]. While the RAC provides a robust retrospective framework, it defines severity by outcomes that have already occurred and therefore cannot guide the earliest clinical decisions. Multifactorial scoring systems such as Ranson, APACHE II and the Bedside Index for Severity in Acute Pancreatitis (BISAP) were developed to address this gap, but each is limited by delayed calculation, cumbersome variable collection or modest discriminative accuracy [5,6].

These limitations have driven interest in rapid, single-analyte biochemical markers measurable on admission [11]. C-reactive protein (CRP) remains the most widely used laboratory marker and is often regarded as the reference standard, with a threshold above 150 mg/L at 48 hours indicating a complicated course; however, its value is limited by a delayed peak that typically occurs only 72 hours after symptom onset [4,7]. Procalcitonin (PCT), the propeptide of calcitonin, rises rapidly in bacterial and fungal infection and has been proposed as an early indicator of infected necrosis and severe disease [7,8]. Interleukin-6 (IL-6), a pro-inflammatory cytokine released early in the systemic inflammatory cascade, increases before the acute-phase proteins it induces and has consistently emerged as one of the most promising markers for early severity stratification, with reported

sensitivities of 69–100% and area-under-the-curve (AUC) values ranging from 0.69 to 0.99 [4,9,10].

Despite a growing body of evidence, uncertainty persists regarding which marker—or combination of markers—offers the optimal balance of early availability, diagnostic accuracy and clinical practicality. Head-to-head comparisons remain heterogeneous, and few studies have evaluated these analytes against a common RAC-defined outcome within a single cohort [9,10]. The present study was therefore designed to evaluate and compare the diagnostic and prognostic performance of serum IL-6, PCT and CRP, measured at admission and at 48 hours, for the early identification of severe AP, and to determine whether a combined biomarker panel improves predictive accuracy over any single marker.

MATERIALS AND METHODS

2.1 Study design and setting: This prospective observational cohort study was conducted in the Department of Biochemistry, at SSIMS&RC over a 12-month period. The protocol was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants in accordance with the Declaration of Helsinki.

2.2 Study population: Consecutive adults (≥18 years) admitted with a first episode of AP were screened. Diagnosis required at least two of three criteria: (i) abdominal pain characteristic of AP; (ii) serum amylase or lipase greater than three times the upper limit of normal; and (iii) characteristic findings on contrast-enhanced computed tomography (CECT), magnetic resonance imaging or transabdominal ultrasonography [1]. Patients presenting more than 48 hours after symptom onset, or with chronic pancreatitis, pancreatic malignancy, pre-existing chronic inflammatory or autoimmune disease, chronic kidney or liver failure, or non-pancreatic sepsis were excluded.

2.3 Sample size: The required sample was estimated on the basis of an expected AUC of 0.85 for IL-6, with a type I error of 0.05 and power of 80%, yielding a minimum of [N] patients; [120] were enrolled to allow for attrition.

2.4 Blood sampling and biochemical analysis: Venous blood was collected at admission (0 h) and at 48 h. Serum was separated by centrifugation at 3000 rpm for 10 minutes and, where not analysed immediately, stored at −80 °C. Serum IL-6 was measured by [electrochemiluminescence immunoassay], PCT by [immunofluorescence assay] and CRP by [immunoturbidimetry] on [analyser platform] per manufacturer instructions. Amylase and lipase were determined by standard enzymatic methods. Internal quality controls were run with each batch.

2.5 Outcome definition: The primary outcome was severity classified per RAC 2012 as mild (no organ failure, no local/systemic complications), moderately severe (transient organ failure <48 h and/or local complications) or severe (persistent organ failure >48 h) [1]. Organ failure was defined using the modified Marshall scoring system. For diagnostic-accuracy analyses, moderately severe and severe cases were combined and compared against mild disease. BISAP and APACHE II scores were recorded within 24 h for comparison.

2.6 Statistical analysis: Analyses were performed in [SPSS v.XX / R v.X.X]. Continuous variables were summarised as mean ± SD or median (interquartile range) and compared using the Student t-test or Mann–Whitney U test; categorical variables using the chi-square or Fisher exact test. Discriminative performance was assessed by ROC analysis with AUC and 95% confidence intervals. Optimal cut-offs were derived by the Youden index, with corresponding sensitivity, specificity and predictive values. AUCs were compared using the DeLong test, and multivariable logistic regression identified independent predictors and defined the combined model. A two-tailed $p < 0.05$ was considered significant.

RESULTS

Of [120] enrolled patients, [78] (65.0%) had mild, [26] (21.7%) moderately severe and [16] (13.3%) severe AP by RAC 2012. Baseline demographic and clinical characteristics are summarised in Table 1. Age, sex distribution and aetiology were comparable across severity groups, whereas admission APACHE II score, the proportion with BISAP ≥ 3, and radiological necrosis increased significantly with severity (all $p < 0.001$), confirming appropriate case stratification.

Table 1. Baseline demographic and clinical characteristics by severity group.

Variable	Mild (n=78)	Mod. severe (n=26)	Severe (n=16)	p-value
Age, years (mean ± SD)	44.6 ± 13.2	48.1 ± 12.7	52.3 ± 14.1	0.09
Male, n (%)	50 (64.1)	17 (65.4)	11 (68.8)	0.92
Biliary aetiology, n (%)	34 (43.6)	12 (46.2)	7 (43.8)	0.98
Alcohol aetiology, n (%)	30 (38.5)	10 (38.5)	6 (37.5)	0.99
BISAP ≥ 3, n (%)	4 (5.1)	9 (34.6)	12 (75.0)	<0.001
APACHE II (mean ± SD)	5.1 ± 2.3	8.7 ± 3.1	12.4 ± 3.8	<0.001
Necrosis on CECT, n (%)	2 (2.6)	8 (30.8)	13 (81.3)	<0.001

SD, standard deviation; BISAP, Bedside Index for Severity in Acute Pancreatitis; APACHE II, Acute Physiology and Chronic Health Evaluation II; CECT, contrast-enhanced computed tomography. Values illustrative — replace with study data.

Serum concentrations of all three markers rose progressively with severity at both time points (Table 2). IL-6 was already markedly elevated at admission in more severe disease, whereas the separation of CRP between groups became most pronounced at 48 h, consistent with its delayed acute-phase kinetics. PCT concentrations were highest in the severe group, in keeping with a greater burden of infection and necrosis.

Table 2. Serum biomarker concentrations at admission (0 h) and 48 h, by severity group [median (IQR)].

Marker (time)	Mild (n=78)	Mod. severe (n=26)	Severe (n=16)	p-value
IL-6, pg/mL (0 h)	22 (14–38)	68 (45–102)	156 (98–240)	<0.001
IL-6, pg/mL (48 h)	18 (11–31)	82 (54–120)	210 (140–305)	<0.001
CRP, mg/L (0 h)	28 (12–55)	74 (41–110)	112 (70–165)	<0.001
CRP, mg/L (48 h)	61 (40–98)	168 (120–232)	264 (198–330)	<0.001
PCT, ng/mL (0 h)	0.18 (0.09–0.34)	0.42 (0.22–0.78)	1.30 (0.65–2.40)	<0.001
PCT, ng/mL (48 h)	0.22 (0.11–0.40)	0.85 (0.44–1.55)	2.60 (1.40–4.20)	<0.001

IQR, interquartile range; IL-6, interleukin-6; CRP, C-reactive protein; PCT, procalcitonin. Values illustrative — replace with study data.

The discriminative performance of each marker for identifying non-mild (moderately severe plus severe) disease is shown in Table 3. At admission, IL-6 achieved the highest accuracy (AUC 0.89), substantially exceeding admission CRP (0.68) and PCT (0.71). CRP reached comparable accuracy only at 48 h (AUC 0.85). A combined logistic-regression panel incorporating IL-6, CRP and PCT yielded the best overall discrimination (AUC 0.94), with sensitivity of 91% and specificity of 88%, significantly outperforming any single marker on the DeLong test ($p < 0.05$).

Table 3. Diagnostic performance of individual markers and the combined panel for predicting non-mild acute pancreatitis.

Marker (time)	AUC (95% CI)	Cut-off	Sens. (%)	Spec. (%)
IL-6 (0 h)	0.89 (0.83–0.95)	40 pg/mL	86	82
IL-6 (48 h)	0.91 (0.86–0.96)	45 pg/mL	88	85
CRP (0 h)	0.68 (0.59–0.77)	60 mg/L	64	66
CRP (48 h)	0.85 (0.78–0.92)	150 mg/L	82	80
PCT (0 h)	0.71 (0.62–0.80)	0.35 ng/mL	70	72
PCT (48 h)	0.80 (0.72–0.88)	0.50 ng/mL	74	78
Combined panel*	0.94 (0.90–0.98)	—	91	88

*Combined panel = IL-6 + CRP + PCT (multivariable logistic model). AUC, area under the ROC curve; CI, confidence interval; Sens., sensitivity; Spec., specificity. Values illustrative — replace with study data.

DISCUSSION

In this prospective cohort, serum IL-6 provided the earliest and most accurate discrimination between mild and more severe forms of AP, outperforming both CRP and PCT at admission. This is consistent with the biology of IL-6, which is released rapidly by many cell types in response to tissue injury and drives hepatic synthesis of acute-phase proteins, including CRP [4,9]. Because IL-6 rises before the reactants it induces, it is informative at the stage when clinical decisions are most consequential, whereas CRP does not reach its 150 mg/L threshold until roughly 48–72 hours after symptom onset [4,7]. The finding that CRP achieved comparable accuracy only at 48 h echoes reports identifying it as a robust but temporally delayed marker [7,9].

PCT showed the lowest discriminative accuracy for overall severity, in keeping with its principal role as an indicator of bacterial and fungal infection rather than sterile inflammation [7,8]. PCT nonetheless retains value for the early recognition of infected necrosis and sepsis-related complications, and its inclusion in a multi-marker strategy may capture a dimension of risk not reflected by IL-6 or CRP [8]. Consistent with this, the combined panel achieved higher accuracy than any single analyte, supporting the view that complementary markers reflecting distinct processes—early cytokine activation, the acute-phase response and infection—together provide the most complete assessment of severity [10].

These results align with comparative studies reporting that IL-6 and 48-hour CRP have significantly greater discriminative performance than other inflammatory markers for early severity prediction [9], and with pooled data describing IL-6 AUC values of 0.69–0.99 [10]. The practical appeal of these markers lies in their rapid turnaround and objectivity relative to multifactorial scores such as APACHE II and BISAP, which require collation of numerous variables and, for Ranson criteria, a 48-hour delay before completion [5,6].

Several limitations warrant consideration. The single-centre design, modest sample size and—critically—the illustrative nature of the present dataset limit generalisability and must be addressed with real, prospectively collected data. The absence of serial sampling beyond 48 h precludes full characterisation of marker kinetics, and IL-6 assays are not yet universally available or standardised across laboratories, which may constrain routine implementation. Cost and turnaround time also merit formal economic evaluation. Future work should validate these findings in larger multicentre cohorts, incorporate emerging endothelial markers such as angiopoietin-2, and test prospectively whether biomarker-guided triage improves patient-centred outcomes [10].

CONCLUSION

Early and accurate stratification of AP severity remains a central clinical challenge. In this study, serum IL-6 emerged as the most reliable single marker for early identification of patients at risk of moderately severe or severe disease, offering discriminative accuracy at admission that CRP achieved only after 48 hours, while PCT contributed principally to the recognition of infection-related severity. A combined panel integrating IL-6, CRP and PCT provided the best overall predictive performance and may support more timely triage than conventional scoring systems. Incorporation of rapid biomarker measurement into early management pathways, validated in larger multicentre studies, holds promise for improving resource allocation and outcomes in acute pancreatitis.

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