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Original Research Article

The Association between Serum Procalcitonin Levels and Severity of Coronary Artery Disease Assessed by Syntax Score in Patients with Acute Coronary Syndrome

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Abstract:

Background and Rationale: Inflammatory pathways occupy a central position in the pathogenesis of atherosclerotic plaque instability that underpins acute coronary events. Among various circulating mediators, serum Procalcitonin (PCT)—conventionally exploited as a surrogate for bacterial sepsis—has recently attracted attention in the context of ischaemic heart disease, where cytokine-driven transcription of its precursor peptide is also activated. Concurrently, the SYNTAX score has been validated as a reproducible angiographic instrument capable of expressing the cumulative anatomical load of coronary artery disease (CAD). Whether circulating PCT mirrors the angiographic complexity captured by this scoring system across distinct presentations of acute coronary syndrome (ACS) warrants dedicated investigation.

Objectives: To determine whether admission serum PCT concentrations are independently associated with the anatomical burden of CAD quantified by the SYNTAX score, and to compare this relationship across three ACS categories: ST-elevation myocardial infarction (STEMI), Non-ST-elevation ACS (NSTE-ACS), and Unstable Angina (UA).

Methods: Three hundred consecutive ACS patients (100 per subtype) admitted to SCBMCH between December 2023 and August 2025 were enrolled in this single-centre, cross-sectional, observational investigation. Venous PCT was quantified at admission using a validated immunoassay. Coronary angiography employed the Judkins technique; the resulting SYNTAX score was computed by a blinded interventional cardiologist. Participants were dichotomised into low (≤32) and high (>33) SYNTAX strata. Group comparisons used independent t-tests, chi-square, and one-way ANOVA, with $p<0.05$ denoting significance.

Key Findings: A statistically meaningful elevation of PCT was documented in the high-SYNTAX cohort relative to the low-SYNTAX cohort (0.07 ± 0.03 vs. 0.06 ± 0.03 ng/mL; $p = 0.0298$). Supra-threshold PCT (>0.0704 ng/mL) was present in 48% of STEMI and 51% of NSTE-ACS patients versus merely 1% of UA patients. NSTE-ACS carried the heaviest angiographic burden, recording the highest mean SYNTAX score (30.01 ± 8.71). Conventional cardiovascular risk determinants failed to discriminate between SYNTAX categories (all $p >0.05$).

Conclusion: Admission PCT levels reflect the angiographic complexity of coronary anatomy in ACS patients. This readily accessible biomarker may augment conventional risk scoring, particularly in resource-limited environments where expedited triage decisions are required.

Keywords: Procalcitonin; SYNTAX score; Acute Coronary Syndrome; Coronary Artery Disease; Inflammation; STEMI; NSTE-ACS.

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Introduction

Cardiovascular disease continues to exact a profound global toll, with acute coronary syndrome occupying a pre-eminent position among causes of sudden death and premature disability worldwide.[1] The overarching term ACS encompasses a pathophysiological spectrum anchored by unstable angina at one pole and transmural ST-elevation myocardial infarction at the other, with non-ST-elevation myocardial infarction bridging these extremes.[2] Each presentation carries a distinct prognosis shaped not merely by the acuteness of the thrombotic event but also by the extent and intricacy of the underlying coronary architecture—a dimension that warrants precise characterisation for optimal therapeutic planning.

An internationally validated instrument addressing

this need is the SYNTAX (Synergy between Percutaneous Coronary Intervention with Taxus and Cardiac Surgery) score, which encodes the anatomical intricacy of coronary lesions based on their number, location, morphological characteristics, and haemodynamic significance.[3] Scores exceeding 33 have consistently signalled a domain of coronary complexity where outcomes following percutaneous coronary intervention (PCI) are inferior, thereby favouring surgical revascularisation.[4] Despite its prognostic potency, the SYNTAX score demands formal coronary angiography, a resource-intensive procedure not universally available at the point of first medical contact.

Parallel scientific effort has sought circulating molecular signals capable of approximating coronary disease severity non-invasively. The inflammatory model of atherogenesis, consolidated over several decades of basic and clinical research, posits that innate immune activation, cytokine elaboration, and myeloid cell infiltration of the vascular wall are not epiphenomena of atherosclerosis but rather its fundamental drivers.[5] High-sensitivity C-reactive protein (hs-CRP) and interleukin-6 have been the most extensively studied in this paradigm.[6,7] Yet their diagnostic granularity remains constrained by the non-specificity of these signals across a broad range of inflammatory conditions.

Procalcitonin (PCT), a 116-amino-acid precursor of the calcium-regulating hormone calcitonin, undergoes systemic upregulation under the influence of bacterial endotoxins and pro-inflammatory mediators—principally tumour necrosis factor-alpha, interleukin-1 beta, and interleukin-6.[8] Critically, these same cytokine networks are activated during myocardial ischaemia-reperfusion injury, endothelial disruption, and plaque fissuring.[9] Consequently, PCT elevation in the absence of overt infection has been documented in the setting of acute myocardial necrosis, raising the possibility that this peptide tracks the intensity of the vascular inflammatory cascade rather than infection per se.[10,11]

Notwithstanding these mechanistic plausibility arguments, a direct empirical linkage between quantitative PCT levels and the formal angiographic complexity score has not been robustly characterised across all three major ACS subtypes simultaneously.[12] Existing publications have predominantly examined PCT in isolated STEMI cohorts or mixed ACS populations without subtype-stratified angiographic correlation. This knowledge deficit is clinically relevant: a validated association between PCT and SYNTAX score would permit early inflammatory profiling to augment or even temporise the need for formal coronary evaluation in resource-constrained or logistically challenging scenarios.

The present investigation was accordingly designed to rigorously evaluate the relationship between admission PCT and the SYNTAX-derived anatomical burden of coronary artery disease in 300 ACS patients recruited from a quaternary cardiac care unit in eastern India. A secondary aim was to appraise whether circulating inflammatory biomarkers convey risk information beyond the classical cardiovascular risk factor compendium when classifying coronary anatomical complexity.

Materials and Methods

Study Architecture and Ethical Framework: This investigation adhered to a prospective, single-centre, observational, cross-sectional design executed within the Department of Cardiology at SCBMCH, Cuttack—a quaternary-referral government institution serving eastern Odisha—between December 2023 and August 2025. Ethical clearance was obtained from the Institutional Ethics Committee, and each participant provided voluntary written informed consent in accordance with the ethical tenets of the Declaration of Helsinki.

Participant Selection: Consecutive patients aged 18–75 years who fulfilled diagnostic criteria for any ACS subtype and who presented in normal sinus rhythm were considered for enrolment. Three hundred subjects meeting the eligibility threshold were recruited, distributed equally across three groups of 100 each: STEMI, NSTE-ACS, and Unstable Angina.

Eligibility Boundaries: Participants were ineligible if any of the following applied: haemodynamic collapse meeting criteria for cardiogenic shock at presentation; documented prior surgical coronary revascularisation via CABG; any concurrent or recently resolved infectious illness; established connective tissue disorder or other chronic inflammatory condition; clinically significant hepatic impairment (Child-Pugh B/C) or reduced kidney function (estimated glomerular filtration rate <30 mL/min/1.73 m^2); or an active or suspected malignant neoplasm. These restrictions were designed to neutralise confounders capable of independently modulating PCT concentrations.

Baseline Characterisation: A structured proforma documented sociodemographic data (age, biological sex), anthropometric measurements (height, weight, body mass index), and a comprehensive cardiovascular risk inventory encompassing systemic hypertension, type 2 diabetes mellitus, dyslipidaemia, active and remote tobacco exposure, and first-degree family history of premature CAD. A twelve-lead electrocardiogram was acquired at presentation and repeated daily or whenever clinically prompted.

Biochemical Evaluation: Venous blood was withdrawn from the antecubital fossa at the time of initial presentation, prior to any reperfusion intervention. The analyte panel encompassed serum PCT (validated electrochemiluminescence immunoassay), high-sensitivity C-reactive protein, complete haematological profile, renal function (serum creatinine), hepatic enzymes, fasting plasma glucose, and a full lipid panel (total cholesterol, LDL-C, HDL-C, triglycerides). All biochemical assays were processed in the hospital's National Accreditation Board for Testing and Calibration Laboratories (NABL)-accredited clinical biochemistry laboratory under standardised pre-analytical and analytical conditions.

Coronary Angiography Protocol: Invasive coronary assessment was performed using the Judkins technique with vascular access obtained via either the radial or femoral artery, determined by operator discretion and individual anatomical suitability. Imaging was conducted on whichever of two cath-lab systems was operationally available in a random-allocation sequence: (i) PHILIPS AZURION 7 M20 digital flat-detector angiography system (Philips Healthcare, Amsterdam, Netherlands); or (ii) SIEMENS ARTIS ZEE FLOOR system (Serial No. 135885, Siemens Healthineers, Erlangen, Germany). Angiographic interpretation was carried out by a senior interventional cardiologist who remained unaware of each patient's biomarker results.

SYNTAX Score Derivation: The SYNTAX score was calculated in conformity with the methodology described by Sianos et al. Every coronary segment with a reference vessel diameter ≥1.5 mm exhibiting ≥50% diameter stenosis was incorporated into the analysis. Scores were derived using the validated online SYNTAX score calculator (www.syntaxscore.com). For group-comparative purposes, patients were categorised as low-complexity CAD (SYNTAX ≤32) or high-complexity CAD (SYNTAX >33).

Statistical Framework: Data were processed using SPSS version 26.0 (IBM Corp., Armonk, NY). Normally distributed continuous variables were summarised as mean ± standard deviation and compared via independent-samples t-test or one-way ANOVA; skewed variables were evaluated using the Mann-Whitney U test. Categorical data were expressed as absolute counts and percentages, with inter-group comparisons performed by Pearson's chi-square or Fisher's exact test as dictated by expected cell frequencies. Pearson's r coefficient was employed for correlation analyses between PCT and SYNTAX score. Statistical significance was defined at a two-tailed alpha of 0.05.

Results

Cohort Profile: The aggregate study cohort (n=300) had a mean age of 48.6 ± 13.9 years. Female participants constituted a marginal majority (51% versus 49% male), and the cohort's mean BMI of 26.1 ± 2.6 kg/m² placed it within the overweight band—a finding consistent with adiposity-associated cardiometabolic risk. Systemic hypertension affected 55.3% of participants, while type 2 diabetes mellitus was documented in 48.6%. Dyslipidaemia was identified in three of every five enrolled patients (60%). Regarding tobacco exposure, 38.3% were active smokers and 36.7% were ex-smokers. A first-degree family history of CAD was reported by 36.7%. Critically, none of the baseline demographic or risk factor variables demonstrated statistically significant inter-group differences when the SYNTAX ≤32 and SYNTAX >33 cohorts were compared (all p >0.05), confirming baseline equivalence between the two anatomical severity strata.

Coronary Anatomical Complexity Across ACS Presentations

Table 1: Frequency Distribution of SYNTAX Score Categories Across ACS Subtypes

ACS Subtype	SYNTAX ≤32 (n)	SYNTAX >33 (n)	Total (n)
STEMI	71	29	100
NSTE-ACS	70	30	100
Unstable Angina	85	15	100
Total	226	74	300

STEMI: ST-Elevation Myocardial Infarction; NSTE-ACS: Non-ST-Elevation Acute Coronary Syndrome; SYNTAX score cut-off: ≤32 (low) vs >33 (high)

Table 1 reveals that complex angiographic anatomy (SYNTAX >33) was documented in 29% of STEMI and 30% of NSTE-ACS patients—figures that contrast sharply with the 15% observed among UA participants. This gradient corroborates the expectation that STEMI and NSTE-ACS, characterised by rupture-prone plaques and extensive thrombus burden, are more frequently accompanied by diffuse multivessel coronary involvement than the comparatively quiescent pathobiology of UA.

Procalcitonin Elevation Stratified by ACS Subtype

Table 2: Proportion of Patients with Supra-Threshold Procalcitonin (>0.0704 ng/mL) by ACS Category

ACS Subtype	PCT ≤0.0704 ng/mL	PCT >0.0704 ng/mL	Total
STEMI	52 (52%)	48 (48%)	100
NSTE-ACS	49 (49%)	51 (51%)	100
Unstable Angina	99 (99%)	1 (1%)	100
Total	200	100	300

PCT: Procalcitonin; threshold derived from ROC analysis of the full cohort

A compelling divergence in PCT positivity rates was evident across ACS subtypes (Table 2). Elevated PCT was recorded in 48% of STEMI and 51% of NSTE-ACS patients, whereas the rate in UA was negligible at 1%. This differential highlights that PCT elevation in ACS is intimately tied to the magnitude of myocardial necrosis and the attendant systemic inflammatory amplification rather than being a feature uniformly shared across the syndrome spectrum.

Inflammatory Biomarker Profiles Stratified by Angiographic Complexity

Table 3: Inflammatory Biomarkers Compared Across SYNTAX Score Strata (Mean ± SD)

Biomarker	SYNTAX ≤32	SYNTAX >33	p-value
hs-CRP (mg/L)	4.86 ± 1.90	5.38 ± 2.19	0.0762
Procalcitonin (ng/mL)	0.06 ± 0.03	0.07 ± 0.03	0.0298 *

* $p<0.05$ statistically significant; hs-CRP: high-sensitivity C-reactive protein; PCT: Procalcitonin

Among the inflammatory mediators examined, PCT achieved statistical significance as a discriminator between SYNTAX strata ($p = 0.0298$; Table 3). Patients carrying a SYNTAX score >33 harboured higher circulating PCT relative to those with less complex anatomy (0.07 ± 0.03 vs. 0.06 ± 0.03 ng/mL). The hs-CRP differential (5.38 ± 2.19 vs. 4.86 ± 1.90 mg/L), while numerically consistent in direction, did not cross the conventional significance boundary ($p = 0.0762$), suggesting PCT may possess greater discriminatory granularity for angiographic severity than this non-specific acute-phase reactant.

Parallel Behaviour of SYNTAX Score and Procalcitonin Across ACS Categories

Table 4: Mean SYNTAX Score and Procalcitonin Concentrations Disaggregated by ACS Subtype

ACS Subtype	SYNTAX Score (Mean±SD)	PCT ng/mL (Mean±SD)	n
Unstable Angina	25.98 ± 8.30	0.050 ± 0.010	100
STEMI	28.62 ± 10.31	0.071 ± 0.030	100
NSTE-ACS	30.01 ± 8.71	0.073 ± 0.029	100

SD: Standard Deviation; values ordered by ascending angiographic complexity

A coherent parallel ascent was observed in both SYNTAX score and PCT across ACS subtypes (Table 4). UA patients occupied the lowest tier on both dimensions (SYNTAX 25.98 ± 8.30; PCT 0.050 ± 0.010 ng/mL). STEMI patients occupied an intermediate position (SYNTAX 28.62 ± 10.31; PCT 0.071 ± 0.030 ng/mL). Paradoxically, NSTE-ACS participants recorded the highest values on both parameters (SYNTAX 30.01 ± 8.71; PCT 0.073 ± 0.029 ng/mL), notwithstanding their less dramatic acute clinical presentation. This pattern implies that the angiographic and inflammatory burden in NSTE-ACS may surpass that of STEMI when examined at the population level—a nuance with significant implications for revascularisation strategy.

In-Hospital Clinical Outcomes: In-hospital mortality followed an inverse gradient of disease severity: STEMI recorded the highest case fatality (10 of 100 patients; 10%), followed by NSTE-ACS (3%) and UA (1%). These outcome data contextualise the clinical stakes associated with imprecise risk stratification and lend weight to the pursuit of early biomarker-based severity surrogates.

Discussion

The centrepiece finding of this investigation is a statistically significant inter-strata difference in serum PCT concentrations when patients are segregated by SYNTAX score, with higher PCT accompanying anatomically more elaborate coronary disease ($p = 0.0298$). This empirical observation furnishes a novel evidential plank for the proposition that a circulating inflammatory peptide—originally characterised as a bacteraemia sentinel—encodes information about the extent and complexity of atherothrombotic coronary pathology.

The mechanistic framework underpinning this finding draws on well-established immunological principles. Rupture of a coronary atheromatous plaque triggers a local and systemic inflammatory cascade in which cytokines—particularly TNF-α, IL-1β, and IL-6—surge substantially in

circulation.[13,14] These molecules are precisely the signals that drive hepatic and parenchymal synthesis of PCT independently of any infectious stimulus.[15,16] It follows that, in patients whose coronary anatomy harbours more extensive and unstable disease, the magnitude of cytokine elaboration—and consequently PCT production—would be proportionally amplified. Our findings are consistent with this model, as the highest PCT values were recorded in the subgroup bearing the most complex coronary anatomy.

The near-total absence of PCT elevation in UA patients (1% supra-threshold) contrasted with the near-equal prevalence of elevation in STEMI and NSTE-ACS (48% and 51%, respectively). This differential aligns with established knowledge regarding the pathobiology of each entity.[17] In UA, vulnerable plaque erosion or fissuring provokes transient ischaemia without frank myocardial cell death; consequently, the inflammatory signal remains subdued. In STEMI and NSTE-ACS, however, transmural or subendocardial necrosis propagates a robust systemic inflammatory response whose intensity appears sufficient to upregulate PCT synthesis detectably.[18]

An intriguing observation was that NSTE-ACS participants exhibited the highest mean SYNTAX score despite presenting with a clinically less dramatic syndrome than STEMI. This has a plausible anatomical explanation: STEMI characteristically results from a single culprit vessel occluded by acute thrombus superimposed on a focal plaque rupture, whereas NSTE-ACS more frequently reflects multivessel atherosclerotic involvement, with multiple unstable but subtotally obstructing lesions.[19,20] The wider angiographic disease canvas in NSTE-ACS thus produces a higher aggregate SYNTAX score, and the correspondingly broader inflammatory activation translates to marginally higher PCT levels—precisely the pattern we observed.

Comparing PCT with hs-CRP, we found that only PCT reached statistical significance as a discriminator of SYNTAX category, with hs-CRP showing a concordant directional trend that fell short of significance. This may not be coincidental.[21] C-reactive protein is a downstream acute-phase reactant induced by IL-6 from multiple tissue sources, rendering it comparatively non-specific. PCT, by contrast, may be more tightly coupled to the magnitude of acute cytokine release events associated with focal vascular necrosis.[22] If replicated in larger cohorts, this could position PCT as a more diagnostically precise inflammatory marker of anatomical CAD complexity than hs-CRP.

Traditional cardiovascular risk factors—hypertension, diabetes, dyslipidaemia, smoking, and familial predisposition—were uniformly distributed between the low and high SYNTAX groups in our cohort, reinforcing the view that these chronically operating determinants establish the substrate for atherosclerosis but do not reliably predict the resultant angiographic complexity at any given clinical juncture.[23,24] This finding supports a growing consensus that prognostic models relying solely on classic risk factors are insufficient for classifying coronary anatomical burden, and that inflammatory profiling adds a complementary dimension.[25]

From an applied clinical perspective, PCT measurement is inexpensive, widely available even in peripheral hospitals, and delivers a quantitative result within two hours of venepuncture. If an elevated PCT at ACS presentation signals likely high-SYNTAX coronary anatomy, this information could accelerate triage decisions—specifically, guiding urgent transfer to catheterisation-capable centres or prioritising early invasive strategy.[26] A composite biomarker index combining PCT with troponin and hs-CRP might yield superior discriminatory accuracy than any single analyte, a hypothesis meriting prospective validation. [27,28]

In-hospital mortality data from this cohort—STEMI 10%, NSTE-ACS 3%, UA 1%—follows the expected prognostic hierarchy and underscores the clinical stakes associated with late or inadequate risk stratification.[29] Patients with high SYNTAX scores carry substantially elevated revascularisation risk and adverse outcomes post-PCI; their early identification before angiography could influence pre-procedure planning, including heart team discussions regarding PCI versus CABG.[30]

Study Limitations: Several constraints warrant transparent acknowledgement. The cross-sectional architecture precludes causal inference or assessment of temporal PCT kinetics in relation to evolving angiographic change. The single-centre setting may limit external generalisability to populations with differing ethnic and dietary profiles. PCT was sampled at a single time point; serial measurements capturing peak inflammatory response were not performed. The sample of 100 per ACS subtype, while adequate for primary comparisons, may have limited power to detect subtler inter-strata differences for secondary outcomes. Additionally, subclinical dental or pulmonary infections—not formally excluded by culture—could theoretically contribute to PCT elevation. Future large-scale, multicentre, longitudinal studies with serial biosampling and clinical follow-up are warranted to validate and extend these observations.

Conclusion

This cross-sectional investigation involving 300

ACS patients demonstrates that serum Procalcitonin levels, measured at the time of hospital admission, carry a statistically meaningful association with the angiographic complexity of coronary artery disease as quantified by the SYNTAX score. The inflammatory signal indexed by PCT was most pronounced in STEMI and NSTE-ACS, where myocardial necrosis and vascular injury are most severe, and was virtually absent in Unstable Angina. Conventional cardiovascular risk determinants did not discriminate between anatomical severity strata, accentuating the incremental informational yield provided by inflammatory biomarker assessment.

These data collectively support a role for PCT as a low-cost, rapidly accessible, and clinically actionable biomarker for initial coronary risk stratification in the ACS setting. Its incorporation alongside established biomarkers within structured triage protocols could facilitate earlier identification of patients bearing anatomically complex coronary disease who may derive the greatest benefit from prompt invasive evaluation and appropriately tailored revascularisation. Prospective, multicentre validation of these findings remains the necessary next step towards integrating PCT into evidence-based ACS management pathways.

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