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SERUM CORTISOL LEVELS & INSULIN RESISTANCE IN DETECTION AND DIFFERENTIATION OF SUBTYPES OF ACUTE CORONARY SYNDROME: AN OBSERVATIONAL CROSS-SECTIONAL STUDY

Biochemistry

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ABSTRACT

Background: Activation of the hypothalamic-pituitary-adrenal (HPA) axis during acute myocardial ischaemia results in hypercortisolaemia, which may drive insulin resistance and worsen cardiovascular prognosis. Systematic characterisation of serum cortisol across Acute Coronary Syndrome (ACS) subtypes and its metabolic correlates is lacking in Indian populations.**Methods:** An observational cross-sectional study enrolled 150 newly diagnosed ACS patients at tertiary care centre of central india. Serum cortisol was estimated by Chemiluminescent Immunoassay (CLIA); insulin resistance was calculated by HOMA-IR from fasting glucose and insulin. Statistical analysis used ANOVA, Student's t-test, Chi-square, Pearson correlation, and multiple linear regression. **Results:** Elevated cortisol (>23 µg/dL) was found in 46.7% of patients (n=70), exclusively among males and exclusively in STEMI.Mean cortisol showed a highly significant severity gradient: STEMI (24.37 ± 1.44 µg/dL) > NSTEMI (18.54 ± 0.60) > Unstable Angina (15.01 ± 0.52; F=678.56, p<0.001). Males had significantly higher cortisol than females (23.28 ± 3.02 vs. 17.51 ± 1.65 µg/dL; p<0.001). Cortisol rose progressively with age (F=14.533, p<0.001). Pearson correlation with HOMA-IR was r=+0.612 (p<0.001); cortisol was an independent predictor on multivariate analysis (β=+0.124, p<0.001). **Conclusion:** Serum cortisol in ACS is severity-graded, gender-specific, and an independent predictor of insulin resistance. Routine cortisol assessment should be incorporated into ACS metabolic evaluation.

KEYWORDS

Acute Coronary Syndrome, Serum Cortisol, HPA Axis, Hypercortisolaemia, HOMA-IR, Insulin Resistance, STEMI, Cardiovascular Biomarkers

INTRODUCTION

Acute Coronary Syndrome (ACS) encompasses three clinically and pathophysiologically distinct but interrelated conditions: ST-elevation myocardial infarction (STEMI), non-ST-elevation myocardial infarction (NSTEMI), and Unstable Angina (UA). Together, they represent the most lethal manifestation of coronary artery disease and constitute the leading cause of cardiovascular mortality globally. India's burden is compounded by premature onset— approximately 50% of myocardial infarctions in India occur before the age of 50 — and by the high prevalence of STEMI as the predominant ACS subtype, which the CREATE registry documents at nearly 70% of Indian presentations, compared to a lower proportion in Western cohorts. Within Central India and Madhya Pradesh, in-hospital STEMI mortality ranges from 5–10%, reflecting both disease severity and systemic healthcare access challenges.

Cortisol, the principal glucocorticoid, is secreted in direct proportion to the severity of physiological stress. The acute cardiac event activates the hypothalamic-pituitary-adrenal (HPA) axis through afferent pain signals, haemodynamiccompromise, and inflammatory cytokine release, triggering a surge of adrenocorticotropic hormone (ACTH) and consequent elevation in circulating cortisol. While this neuroendocrine response serves an acute adaptive purpose — mobilising energy substrates, maintaining vascular tone, and modulating immune activity — its sustained or excessive activation carries well-characterised adverse metabolic consequences. These include impaired insulin-mediated glucose uptake in skeletal muscle, enhanced hepatic gluconeogenesis, increased lipolysis with free fatty acid release, and suppression of pancreatic insulin secretion, collectively promoting insulin resistance.

Insulin resistance in ACS, even in the absence of established diabetes, is clinically significant. Lazzeri et al. demonstrated that HOMA-IR positivity in ACS independently predicted greater myocardial damage, reduced left ventricular function, elevated troponin levels, and in-hospital mortality. The mechanistic link between HPA axis activation, cortisol-mediated insulin resistance, and ACS severity is therefore of substantial clinical relevance. Yet, systematic data from Indian tertiary care populations quantifying the distribution of serum cortisol across ACS subtypes and its independent correlation with HOMA-IR remain limited. The present study was designed to address this gap.

The study objectives were: (1) to characterise the distribution of serum cortisol in ACS patients by severity, gender, and age group; (2) to determine the correlation between serum cortisol and insulin resistance as measured by HOMA-IR; and (3) to assess cortisol's independent contribution to insulin resistance in a multivariate model.

MATERIALAND METHODS

Study Design and Setting: An observational, cross-sectional, hospital-based study was conducted in the Department of Medical Biochemistry in collaboration with the Department of Cardiology, at tertiary care centre of central india. All biochemical investigations were performed at the Central Clinical Laboratory (CCL), tertiary care centre of central india. Ethical clearance was obtained from the Institutional Ethics Committee. Written informed consent was obtained from all participants or, in the case of unconscious patients, from their relatives.

Sample Size and Participants: The minimum sample was calculated at 142 cases (ACS prevalence 10.3% in India; z=1.96; d=0.05); 150 were enrolled to account for potential dropouts. Inclusion criteria: newly diagnosed ACS patients, age ≥18 years, either sex, presenting to Cardiology Emergency/IPD before initiation of any therapy, willing to provide informed consent. Exclusion criteria: known Diabetes Mellitus, BMI >30 kg/m² (obesity), chronic diseases (HIV infection, CHF, COPD), malignancy, chronic opioid abuse, immunosuppressant or anti-androgenic medications, severe hepatic or renal dysfunction.

Sample Collection: Fasting venous blood (5 mL) was collected after 8–12 hours of overnight fasting, in the morning, under aseptic technique. Morning collection was standardised to account for the circadian peak of cortisol secretion. Serum was separated by centrifugation at 3000 rpm for 10 minutes, aliquoted immediately, and stored at −20°C until analysis within one freeze-thaw cycle.

Serum Cortisol Estimation (Method): Cortisol was measured by Chemiluminescent Immunoassay (CLIA) on the Beckman Coulter UniCel DxI 800 Access Immunoassay System. The assay employs a competitive binding principle: cortisol in the patient sample competes with alkaline-phosphatase-labelled cortisol for a limited number of anti-cortisol antibody sites on paramagnetic particles. After incubation and magnetic separation, the chemiluminescent substrate (Lumi-Phos 530) is cleaved by bound enzyme, producing light emission inversely proportional to cortisol concentration. The luminometer signal is converted to cortisol concentration via a calibration curve. Normal morning reference range: 5–23 µg/dL. Elevated cortisol was defined as >23 µg/dL. All tests followed validated Standard Operating Procedures; internal and external quality control was maintained.

HOMA-IR Calculation: Fasting plasma glucose was estimated by the Hexokinase method (Beckman Coulter AU5800). Fasting serum insulin was measured by Chemiluminescent Microparticle Immunoassay (CMIA). HOMA-IR = [Fasting Insulin (µIU/mL) × Fasting Glucose (mg/dL)] ÷ 405. Values <1.0 indicated insulin sensitivity; 1.0–2.49 indicated early resistance; ≥2.5 indicated significant insulin resistance.

Statistical Analysis: Data were analysed using EpiInfo software. Continuous variables are expressed as mean ± standard deviation (SD). Categorical variables are presented as frequencies and percentages. Group comparisons used one-way ANOVA (for continuous variables across ≥3 groups) and Student's t-test (for two groups). Categorical associations used Chi-square test. Pearson correlation coefficient (r) quantified linear associations between cortisol and HOMA-IR. Multiple linear regression identified independent predictors of HOMA-IR, with the model including age, gender, ACS type, smoking, serum cortisol, and serum testosterone. Statistical significance was set at $p<0.05$.

RESULTS AND DISCUSSION

Of 150 enrolled patients, 107 (71.3%) were male and 43 (28.7%) female, yielding a male-to-female ratio of approximately 2.5:1. The highest ACS incidence was in the 51–60 years age group (40.0%), followed by 41–50 years (39.3%), and >60 years (20.7%). STEMI was the most common ACS type (60.7%, n=91), followed by NSTEMI (26.7%, n=40) and Unstable Angina (12.7%, n=19). Five patients (3.3%) expired during hospitalisation; 145 (96.7%) were discharged improved. The demographic profile is consistent with the well-documented epidemiology of premature and male-predominant ACS in India and Central Indian registries.

Overall Distribution of Serum Cortisol

Of 150 ACS patients, 80 (53.3%) had normal serum cortisol (5–23 µg/dL) and 70 (46.7%) had elevated cortisol (>23 µg/dL). The overall mean was 21.63 ± 3.76 µg/dL (Table 1, Figure 1). Nearly half the cohort therefore demonstrated hypercortisolaemia at presentation — a direct reflection of robust HPA axis activation during the acute coronary event.

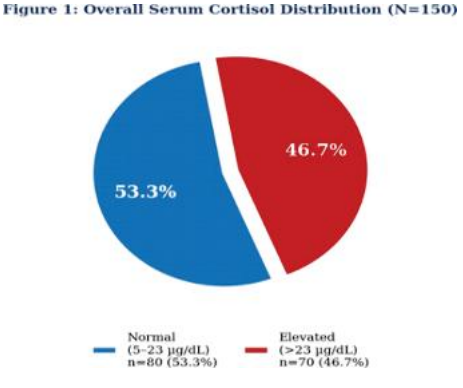


Figure 1: Pie Chart — Overall Serum Cortisol Distribution in ACS Patients (N=150)

Table 1: Overall Serum Cortisol Distribution in ACS Patients (N=150)

Normal (5–23 µg/dL)	80	53.3
Elevated (>23 µg/dL)	**70**	**46.7**
Total (N=150)	**150**	**100.0**

[Reference range: 5–23 µg/dL (morning fasting sample, CLIA method)]

This prevalence is consistent with published evidence. Mujawar et al. (2010) reported cortisol levels of 1237 nmol/L in acute MI patients versus 253 nmol/L in controls — more than a fourfold elevation. Helaly et al. (2013) documented significantly higher cortisol in ACS patients compared to stable coronary disease patients and healthy controls. Pilz et al. (2021) confirmed that higher cortisol was associated with adverse cardiovascular risk profiles including elevated blood pressure, glucose, and triglycerides. The present study's 46.7% prevalence of hypercortisolaemia in an ACS cohort is clinically striking, particularly given the deliberate exclusion of known diabetics, which limits the confounding effect of pre-existing glucocorticoid-mediated metabolic disease.

Serum Cortisol Across Age Groups

Serum cortisol increased progressively and significantly with age. The 41–50 years group had a mean of 19.88 ± 3.64 µg/dL; the 51–60 years group, 22.23 ± 3.28 µg/dL; and the >60 years group, 23.79 ± 3.42 µg/dL ($F=14.533$, $p<0.001$). The proportion with elevated cortisol also increased with age — 20.3% in the youngest group rising to 71.0% in those over 60 years — with a highly significant association ($\chi^2=28.072$, $p<0.001$; Table 2, Figures 2 and 3).

Table 2: Serum Cortisol Distribution and Mean Values Across Age Groups (N=150)

Age Group (Years)	n	Normal n (%)	Elevated n (%)	Mean ± SD (µg/dL)
41–50	59	47 (79.7%)	**12 (20.3%)**	19.88 ± 3.64
51–60	60	24 (40.0%)	**36 (60.0%)**	22.23 ± 3.28
>60	31	9 (29.0%)	**22 (71.0%)**	23.79 ± 3.42
Total (N=150)	**150**	80 (53.3%)	**70 (46.7%)**	**21.63 ± 3.76**
χ^2 Statistic / p-value		*$\chi^2 = 28.072$*	**p < 0.001***	**F = 14.533 p < 0.001***

[One-way ANOVA for continuous comparison; Chi-square for categorical; * p<0.001 statistically significant]

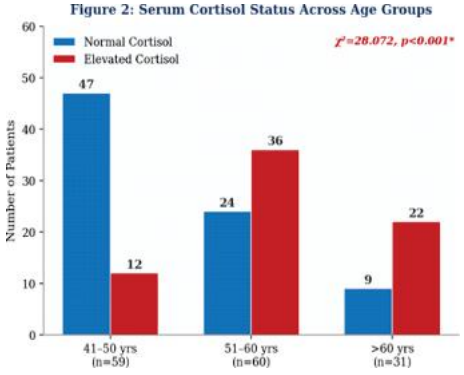


Figure 2: Grouped Bar Chart — Normal vs Elevated Serum Cortisol by Age Group

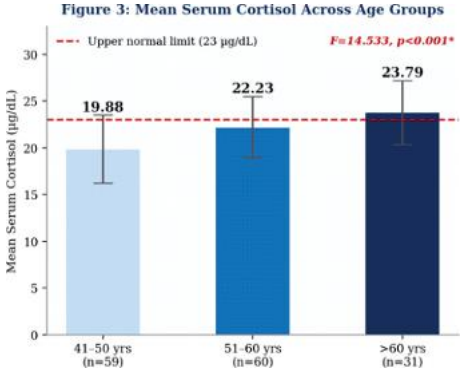


Figure 3: Mean Serum Cortisol (±SD) Across Age Groups with Reference Limit

The age-related rise in cortisol reflects cumulative HPA axis dysregulation in older individuals, characterised by declining glucocorticoid receptor sensitivity, reduced negative feedback efficacy, and blunted suppression of cortisol secretion. This is superimposed upon the age-related increase in ACS severity and the greater haemodynamiccompromise associated with more advanced coronary artery disease. The concentration of elevated cortisol in older age groups carries prognostic implications: older ACS patients face a compounded metabolic risk from both hypercortisolaemia and age-related deterioration in insulin sensitivity, amplifying the severity of HOMA-IR derangement documented in this cohort.

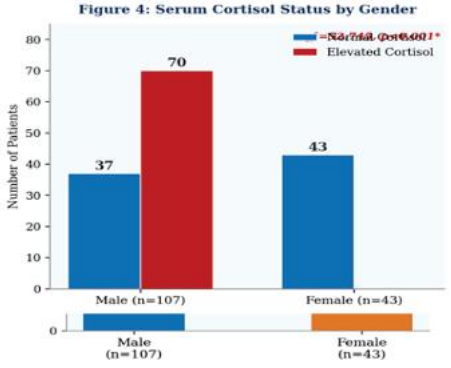
Gender-Specific Serum Cortisol Distribution

Male patients had significantly higher mean cortisol (23.28 ± 3.02 µg/dL) compared to females (17.51 ± 1.65 µg/dL; t=11.809, p<0.001). Most strikingly, all 70 patients with elevated cortisol were male, while all 43 female patients had normal cortisol values (χ^2=52.745, p<0.001; Table 3, Figures 4 and 5).

Table 3: Serum Cortisol Distribution and Mean Values by Gender (N=150)

Gender	n	Normal n (%)	Elevated n (%)	Mean ± SD (µg/dL)
Male	107	37 (34.6%)	**70 (65.4%)**	23.28 ± 3.02
Female	43	43 (100%)	0 (0.0%)	17.51 ± 1.65
Total (N=150)	**150**	80 (53.3%)	**70 (46.7%)**	**21.63 ± 3.76**
t-statistic / p-value			**χ^2=52.745 p<0.001***	**t=11.809 p<0.001***

[Student's t-test for continuous comparison; Chi-square for categorical; * p<0.001]



The gender difference in cortisol is primarily explained by the differential ACS subtype distribution between sexes: males bore the overwhelming STEMI burden in this cohort, and elevated cortisol was found exclusively in STEMI. Female patients, who were predominantly represented in NSTEMI and Unstable Angina, had cortisol levels within normal limits. Additionally, biological sex differences in HPA axis reactivity play a role: oestrogen in premenopausal women modulates CRH secretion and reduces peak cortisol responses to psychophysiological stress stimuli. This aligns with findings from Helaly et al. (2013) and Mujawar et al. (2010), both of whom studied exclusively male ACS patients and documented marked hypercortisolaemia. The present study extends these observations by demonstrating the gender dimension in a mixed-sex cohort.

Serum Cortisol Across ACS Types — The Severity Gradient

The most clinically compelling result of this study is the extraordinarily significant gradient of serum cortisol across ACS severity subtypes. Mean cortisol was highest in STEMI (24.37 ± 1.44 µg/dL), intermediate in NSTEMI (18.54 ± 0.60 µg/dL), and lowest in Unstable Angina (15.01 ± 0.52 µg/dL). The one-way ANOVA yielded F=678.56, p<0.001 — the highest F-statistic of any variable in the entire dataset, underscoring the exceptional discriminative power of cortisol across ACS subtypes (Table 4, Figures 6 and 7). Elevated cortisol was present in 76.9% of STEMI cases, in 0% of NSTEMI cases, and in 0% of Unstable Angina cases (χ^2=85.096, p<0.001).

Table 4: Serum Cortisol Distribution and Mean Values Across ACS Types (N=150)

ACS Type	n	Normal n (%)	Elevated n (%)	Mean ± SD
STEMI	91	21 (23.1%)	**70 (76.9%)**	24.37 ± 1.44
NSTEMI	40	40 (100%)	0 (0.0%)	18.54 ± 0.60
Unstable Angina	19	19 (100%)	0 (0.0%)	15.01 ± 0.52
Total (N=150)	**150**	80 (53.3%)	**70 (46.7%)**	**21.63 ± 3.76**
χ^2 / F-statistic p-value		**χ^2=85.096 p<0.001***		**F=678.56 p<0.001***

[One-way ANOVA for continuous; Chi-square for categorical; * p<0.001 | STEMI=ST-elevation MI; NSTEMI=Non-ST-elevation MI]

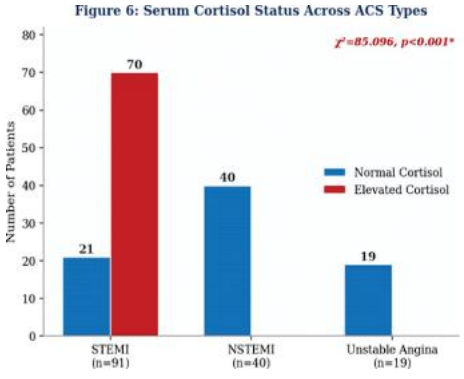


Figure 6: Grouped Bar Chart — Normal vs Elevated Serum Cortisol Across ACS Types

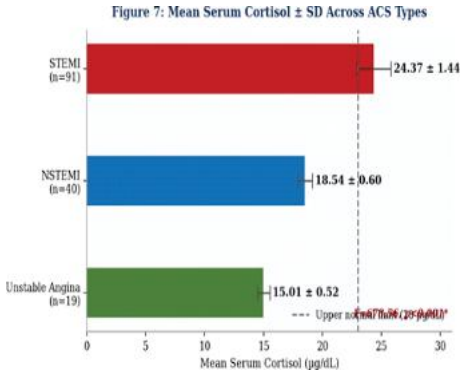


Figure 7: Mean Serum Cortisol (±SD) Across ACS Types with Upper Reference Limit

This severity-cortisol gradient has a robust physiological explanation. Complete coronary occlusion in STEMI produces transmural myocardial necrosis, maximal haemodynamic compromise, intense pain, and systemic inflammatory activation — all potent stimuli for HPA axis engagement. The resulting cortisol surge is proportionate to the magnitude of this physiological threat. Partial occlusion in NSTEMI produces non-transmural ischaemia with a lesser stress stimulus, while the demand-supply mismatch of Unstable Angina generates the least HPA activation.The finding that cortisol differentiates not merely between ACS and non-ACS states but grades finely across ACS subtypes establishes it as a clinically informative severity biomarker. These observations corroborate Smith et al. (2005), who in the Caerphilly prospective study documented that elevated cortisol-to-testosterone ratios predicted ischaemic heart disease risk with an age-adjusted OR of 1.22 per SD increase, and Mujawar et al. (2010), who documented a strong positive correlation between cortisol and mean blood pressure in AMI (r=0.457, p<0.001). Pilz et al. (2021) noted that cortisol was associated with adverse cardiovascular risk factor profiles, though it was not an independent predictor of mortality after full covariate adjustment in their elective angiography cohort — a population with lower acute stress stimulus than the present ACS cohort.

Correlation of Serum Cortisol with Insulin Resistance (HOMA-IR)

Pearson correlation analysis demonstrated a significant moderate positive correlation between serum cortisol and HOMA-IR (r=+0.612, p<0.001), indicating that patients with higher cortisol had greater insulin resistance (Table 5, Figure 8). On multiple linear regression

analysis adjusting for age, gender, ACS type, smoking, and serum testosterone, cortisol remained a significant independent positive predictor of HOMA-IR (β=+0.124, p<0.001). The full model explained 48.7% of the variance in HOMA-IR (R^2=0.487, adjusted R^2=0.461, p<0.001).

Table 5: Pearson Correlation and Multiple Regression — Cortisol with HOMA-IR (N=150)

Variable Pair	r (Pearson)	p-value	β (MLR)
Serum Cortisol (µg/dL) vs HOMA-IR	**+0.612**	**< 0.001***	**+0.124**
Serum Testosterone (ng/mL) vs HOMA-IR	**−0.647**	**< 0.001***	**−0.387**
Cortisol / Testosterone Ratio vs HOMA-IR	**+0.683**	**< 0.001***	—

[r = Pearson correlation coefficient; β = standardised regression coefficient from MLR; * p<0.001]

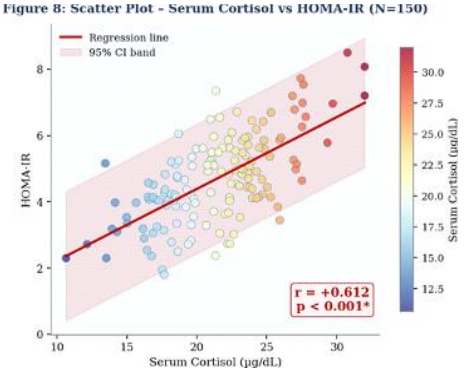


Figure 8: Scatter Plot — Serum Cortisol vs HOMA-IR Showing Positive Correlation (r=+0.612, p<0.001) with 95% CI Band

A total of 98% of patients (n=147) had significant insulin resistance (HOMA-IR >2.5), despite the deliberate exclusion of known diabetics and obese patients. This striking prevalence confirms that insulin resistance in the ACS setting is an acute, severity-dependent metabolic derangement driven by the neuroendocrine milieu of the coronary event — not merely a reflection of pre-existing metabolic disease. The mechanisms by which cortisol generates insulin resistance are well characterised: (i) cortisol inhibits GLUT4 translocation and expression in skeletal muscle, impairing insulin-stimulated glucose uptake; (ii) it stimulates hepatic glucose production via gluconeogenesis and glycogenolysis; (iii) it activates hormone-sensitive lipase in adipose tissue, releasing free fatty acids that impair insulin signalling; and (iv) it suppresses insulin secretion from pancreatic β-cells at high concentrations. These pathways collectively explain the strong and independent positive correlation between cortisol and HOMA-IR observed in the present cohort. The finding aligns with Anagnostis et al. (2009), who comprehensively reviewed the pathogenetic role of cortisol in metabolic syndrome, and with Lazzeri et al. (2014), who demonstrated that acute HOMA-IR positivity in ACS independently predicted in-hospital mortality.

CONCLUSION

The present study demonstrates, with robust statistical power and comprehensive subgroup characterisation, that serum cortisol in Acute Coronary Syndrome is a pervasive, severity-graded, gender-specific, and age-dependent biomarker of the neuroendocrine response to myocardial ischaemia. The stepwise increase in mean cortisol from Unstable Angina (15.01 µg/dL) through NSTEMI (18.54 µg/dL) to STEMI (24.37 µg/dL) — with an F-statistic of678.56 — represents the most statistically discriminant finding in the study and establishes cortisol as a graded biological reporter of ACS severity, not merely a non-specific stress marker.

Hypercortisolaemia in this cohort was gender-specific (exclusive to males) and age-progressive, reflecting the compounded effect of advanced coronary disease severity in older patients and sex-based differences in HPA axis regulation. Cortisol independently predicted insulin resistance in the multivariate model, confirming its direct mechanistic contribution to the near-universal HOMA-IR derangement observed in ACS patients — even after excluding known diabetics and obese individuals.

These findings collectively support the clinical utility of serum cortisol as a routinely measured biomarker in ACS evaluation. As an affordable, rapidly available analyte measurable on standard immunoassay platforms present in all tertiary care biochemistry laboratories, cortisol can be seamlessly incorporated into the admission biochemical workup of ACS patients. It provides clinically meaningful, non-redundant information regarding metabolic severity and hormonal derangement that complements traditional cardiovascular risk stratification tools. We recommend its routine inclusion in ACS biochemical panels, particularly in Indian tertiary care settings. Larger multicentre prospective studies across diverse Indian geographies are warranted to validate cortisol's prognostic value for long-term outcomes and to evaluate whether cortisol-directed metabolic interventions improve prognosis in this high-risk population.

LIMITATIONS OF THE STUDY

- Single time-point measurement: Cortisol was measured at a single morning fasting time point. Given its well-characterised circadian rhythm and stress-induced pulsatility, serial measurements during hospitalisation would better capture the dynamic arc of HPA axis activation and recovery across the acute coronary event.
- Single-centre tertiary design: Enrolment at one institution introduces referral bias, with disproportionate representation of severe and complex ACS. Generalisability to primary care and community ACS populations, and to patients in rural Central India, is limited.
- ACTH not measured: Concurrent ACTH measurement was not performed, preventing classification of hypercortisolaemia as centrally driven (HPG axis) versus adrenal autonomous, limiting mechanistic conclusions about the cortisol pathway.
- Inflammatory biomarkers absent: High-sensitivity CRP, IL-6, and TNF-α were not assessed. These mediators link cortisol-mediated immune activation to insulin resistance and atherosclerosis progression; their inclusion would have strengthened pathophysiological interpretation.
- No longitudinal follow-up: The cross-sectional design precludes assessment of whether admission cortisol elevation predicts major adverse cardiovascular events, rehospitalisation, or mortality beyond the index hospitalisation period. A minimum 1–2 year prospective follow-up design is warranted in future studies.

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