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Original Article

Agreement Between Laboratory and Point-of-Care Creatinine-Based Risk Assessment for Post-Contrast Acute Kidney Injury in Patients Undergoing Urgent Angiography

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ABSTRACT

Background: Rapid identification of patients at risk of contrast-associated acute kidney injury (AKI) is essential in acute settings such as acute myocardial infarction and ischemic stroke. Point-of-care (POC) creatinine testing provides immediate assessment of kidney function; however, its reliability for clinical risk stratification relative to standard laboratory measurements remains uncertain. This study evaluated the agreement between laboratory- and POC creatinine-based risk stratification and their association with subsequent AKI after contrast angiography.

Methods: In this prospective observational study, 295 adults undergoing contrast-enhanced angiography for acute myocardial infarction or acute ischemic stroke were enrolled. Serum creatinine was measured using both standard laboratory methods and a POC device before contrast administration. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI 2021 equation, and predicted risk of post-contrast AKI was assessed using the Mehran risk score. AKI was defined according to KDIGO criteria (≥1.5-fold increase from baseline or ≥26.5 μmol/L increase within 7 days). Agreement between laboratory- and POC-derived risk categories was evaluated using weighted Cohen's kappa.

Results: The median age was 64 years (interquartile range 57–70), and 66.8% of participants were male. Based on laboratory measurements obtained in the hospital central laboratory, categories were low in 8.8%, moderate in 37.3%, high in 25.4%, and very high in 28.5% of patients. Among patients with available follow-up creatinine measurements (n = 127), CA-AKI occurred in 11.0% (14/127). Agreement between laboratory- and POC-based risk classifications was near-perfect ($\kappa = 0.97$, 95% CI 0.95–0.98). The correlation between laboratory and POC creatinine values was moderate ($r = 0.63$, $p < 0.001$).

Conclusion: POC creatinine–based Mehran risk stratification shows excellent diagnostic agreement with laboratory-based assessment for identifying patients at risk of post-contrast AKI. POC testing may facilitate rapid bedside risk assessment in patients undergoing angiography for acute myocardial infarction or ischemic stroke without compromising risk classification reliability.

Keywords: Contrast-Associated Acute Kidney Injury; Point-of-Care Testing; Creatinine; Mehran Risk Score; Percutaneous Coronary Intervention; Angiography; Chronic Kidney Disease

Introduction

Contrast-associated acute kidney injury (CA-AKI) is a common and clinically important complication of contrast-enhanced diagnostic and therapeutic procedures. Even modest increases in serum creatinine following contrast exposure have been associated with prolonged hospitalization, increased healthcare costs, and higher mortality in both the short- and long-term [1–3]. Historically referred to as contrast-induced nephropathy, this complication has been described as the third leading cause of hospital-acquired AKI [4]. More recently, the term CA-AKI has been adopted to reflect uncertainty about direct causality and to align with standardized AKI definitions proposed by the Kidney Disease: Improving Global Outcomes (KDIGO) initiative [5].

Patients presenting with acute coronary syndromes represent a high-risk subgroup due to urgent treatment requirements, hemodynamic instability, and a high burden of comorbidities [2,6]. Similarly, acute stroke pathways prioritize rapid imaging and life-saving intervention, leaving limited opportunity to await standard laboratory results, as treatment delays are directly associated with worse neurological outcomes [7,8].

Risk prediction models such as the Mehran score are widely used to stratify the risk of post-contrast AKI following coronary angiography and percutaneous coronary intervention [2]. However, their clinical use traditionally relies on laboratory-measured serum creatinine and eGFR, which may not be immediately available in emergency or catheterization laboratory settings. Point-of-care creatinine testing provides rapid bedside assessment of kidney function; however, concerns persist regarding its accuracy and whether measurement variability may influence clinical risk classification [9,10].

The importance of reliable renal risk stratification is further underscored by the growing global burden of chronic kidney disease (CKD), affecting approximately 14% of adults worldwide and up to 44% of individuals older than 65 years [11,12]. This rising burden has been observed across diverse healthcare systems, including Central Asia, where nationwide analyses and population-based screening initiatives have reported increasing CKD prevalence and associated cardiovascular risk factors [13,14]. CKD is independently associated with increased risks of cardiovascular events and all-cause mortality across diverse populations, even at moderately reduced levels of kidney function, further underscoring the importance of accurate risk stratification in contrast-exposed patients [15]. Importantly, CKD is a well-established risk factor for AKI, particularly in the setting of contrast exposure, where reduced renal reserve increases susceptibility to hemodynamic stress and nephrotoxic injury [5]. Despite prior studies evaluating agreement between laboratory and POC creatinine measurements, evidence linking creatinine-based risk stratification to observed AKI outcomes in acute angiographic settings remains limited [16]. Therefore, this study aimed to evaluate agreement between laboratory- and POC–derived Mehran risk categories and to assess their association with subsequent AKI.

Methods

Study Design and Population

This prospective observational cohort study was conducted in Astana, Kazakhstan. Adult patients undergoing contrast-enhanced angiography for acute cardiovascular and/or cerebrovascular indications were enrolled between September 2023 and June 2025. The study was conducted at a high-volume center providing emergency percutaneous coronary intervention for acute coronary syndromes and angiography for acute cerebrovascular events. Consecutive eligible patients were approached for enrollment during the index hospitalization, and written informed consent was obtained. The study was approved by the institutional ethics committee (Protocol No. 1/2023/ПЭ, approved on 15 June 2023) and conducted in accordance with the Declaration of Helsinki.

Adults aged 18 years or older undergoing emergency or urgent angiography for suspected or confirmed acute myocardial infarction (including ST-elevation myocardial infarction (STEMI), non–ST-elevation myocardial infarction (NSTEMI), or unstable angina requiring coronary angiography or percutaneous coronary intervention (PCI)) and/or acute cerebrovascular events requiring contrast angiography and/or endovascular intervention were eligible. Inclusion required both a pre-procedural laboratory serum creatinine and a pre-procedural point-of-care (POC) creatinine measurement obtained prior to contrast administration. Patients on chronic dialysis at baseline were excluded. AKI outcome analyses were restricted to participants with sufficient follow-up creatinine measurements to apply KDIGO criteria. Of the 295 enrolled patients, 127 (43%) had post-procedural creatinine measurements available within 7 days and were included in the AKI outcome analysis; the remaining patients were discharged before repeat testing or lacked documented follow-up laboratory

measurements within the predefined assessment window.

Procedures and Creatinine Assessment

Procedures included coronary angiography with or without percutaneous coronary intervention for acute coronary syndromes, as well as contrast-enhanced diagnostic and interventional procedures for acute cerebrovascular events. Procedures were not formally stratified according to type (diagnostic angiography vs. PCI) for analysis, as the primary aim of the study was to evaluate agreement in creatinine-based risk stratification across an acute care population. Contrast volume (mL) administered during the procedure was recorded. Information on the vascular access site (radial vs. femoral) was not consistently available and was therefore excluded from the analysis.

Before contrast administration, serum creatinine was assessed using two methods. Laboratory and point-of-care creatinine measurements were obtained within a short pre-procedural time window, with no clinically significant delay between sampling. Central laboratory creatinine was measured from venous blood samples using the hospital's standard enzymatic method on an automated analyzer in the hospital laboratory, calibrated according to manufacturer standards, and is traceable to isotope dilution mass spectrometry (IDMS) reference standards. Point-of-care creatinine was measured using the StatSensor® Creatinine Meter (Nova Biomedical, Waltham, MA, USA) according to the manufacturer's instructions. Estimated glomerular filtration rate (eGFR) was calculated using the CKD-EPI 2021 creatinine equation, separately for laboratory and POC creatinine values [16]. Neither laboratory nor point-of-care creatinine testing resulted in delays to urgent angiographic procedures. No additional contrast-induced nephropathy–specific preventive measures were implemented beyond standard institutional practice. Standard institutional practice included intravenous isotonic fluid administration at the discretion of the treating physician, avoidance of nephrotoxic medications when feasible, and minimization of contrast volume as required by the procedure. However, detailed data on the use and intensity of preventive measures, including intravenous hydration, were not systematically collected for analysis.

Predicted risk of post-contrast kidney injury was assessed using the original Mehran risk score. The score was calculated twice for each participant, once using laboratory-derived creatinine and eGFR values and a second time using point-of-care–derived values [2]. Mehran risk scores were calculated retrospectively for study purposes using clinical and laboratory data available prior to contrast administration. Clinical variables required for score calculation, including comorbidities, anemia, use of an intra-aortic balloon pump, and contrast volume, were obtained from review of electronic medical records and procedural documentation. Congestive heart failure was defined as NYHA class III–IV or documented pulmonary edema, consistent with the original Mehran score definition. Patients were categorized into four predicted risk strata based on total score: low (<6), moderate (6–10), high (11–15), and very high (≥16).

Outcome Definition

The primary outcome was CA-AKI, defined according to KDIGO creatinine criteria as an increase in serum creatinine of at least 26.5 μmol/L (0.3 mg/dL) within 48 hours or an increase to at least 1.5 times baseline within 7 days following contrast exposure [5]. Urine output criteria were not used because standardized documentation was incomplete. Post-procedural serum creatinine measurements were obtained during hospitalization and, when available, at multiple time points within 7 days after contrast exposure; the highest value was used to determine AKI according to KDIGO criteria. AKI outcome analyses were restricted to participants with sufficient follow-up creatinine measurements to apply KDIGO criteria [5].

Statistical Analysis

Continuous variables were summarized as median with interquartile range, and categorical variables as counts and percentages. Agreement between laboratory- and POC-derived Mehran risk categories was evaluated using percent agreement and weighted Cohen's kappa with 95% confidence intervals. Correlation between laboratory and POC creatinine values was assessed using Pearson correlation. The distribution of observed AKI events across predicted risk categories was examined descriptively and visualized using flow or Sankey diagrams. Statistical analyses were performed using Stata version 16 MP2 (StataCorp LLC, College Station, TX, USA), and a two-sided p-value <0.05 was considered statistically significant.

Results

Baseline Characteristics

A total of 295 patients were included. The median age was 64 years (IQR 57–70), and 197 patients (66.8%) were male (Table 1). Ischemic heart disease was present in 70.0% of patients, cerebrovascular disease in 17.4%, diabetes mellitus in 13.9%, and congestive heart

failure, New York Heart Association (NYHA) class III-IV in 13.9%. Anemia was present in 8.1% of cases, and intra-aortic balloon pump support was used in 8.1% of cases. For calculating the Mehran risk score, anemia was defined as a baseline hematocrit <39% in men and <36% in women, in accordance with the original score definition [2]. Median baseline laboratory eGFR was 88.8 mL/min/1.73 m² (IQR 71.0–99.0), while median POC eGFR was 103.9 mL/min/1.73 m² (IQR 91.4–114.3), suggesting a tendency toward higher eGFR estimates with point-of-care measurements.

Table 1. Baseline sociodemographic and clinical characteristics of patients undergoing contrast angiography (n = 295). Data are presented as n (%) and median (IQR).

Variable	Values
Age, years (median, IQR)	64, 57-70
Male sex, n (%)	197 (66.8)
Comorbidities, n (%) *	
• Ischemic heart disease	149 (70.0)
• Cerebrovascular disease	37 (17.4)
• Cardiac arrhythmias	10 (4.7)
• Cardiomyopathy	10 (4.7)
• Other	7 (3.3)
Diabetes mellitus, n (%) (yes)	41 (13.9)
Congestive heart failure (NYHA class III–IV), n (%)	41 (13.9)
Anemia, n (%) (yes)	24 (8.1)
Use of IABP, n (%) (yes)	24 (8.1)
Prior STEMI, n (%) (yes)	34 (11.5)
Prior NSTEMI or unstable angina, n (%) (yes)	170 (57.6)
Prior ischemic stroke, n (%) (yes)	39 (13.2)
Baseline CKD (eGFR <60 mL/min/1.73 m²), n (%)	40 (13.6)
Baseline lab eGFR, mL/min/1.73 m² (median, IQR)	88.8, 71.0-99.0
Baseline POC eGFR, mL/min/1.73 m² (median, IQR)	103.9, 91.4-114.3
Contrast volume, mL (median, IQR)	100, 60-130

eGFR, estimated glomerular filtration rate; IABP, intra-aortic balloon pump; NSTEMI, non–ST-segment elevation myocardial infarction; POC, point-of-care; STEMI, ST-segment elevation myocardial infarction; NYHA, New York Heart Association. Congestive heart failure is defined as NYHA class III–IV, consistent with the original Mehran risk score definition.

** Medical history variables were unavailable for 82 patients due to incomplete documentation at admission; percentages are calculated using available cases (n=213).*

Agreement Between Laboratory and Point-Of-Care Risk Categories

Agreement between laboratory-based and POC-based Mehran risk categories was excellent (Table 2). Overall percent agreement across all four risk strata was 99.3%, with a weighted kappa of 0.97 (95% CI 0.95–0.98). Discrepancies were rare and occurred almost exclusively between adjacent risk categories. No patients categorized as low risk by laboratory assessment were classified as high or very high risk by POC assessment, and no patients classified as very high risk by laboratory assessment were downgraded to low or moderate risk by POC testing.

Table 2. Agreement between laboratory and point-of-care risk categories

Mehran risk category (points; predicted CA-AKI risk*)	POC Low	POC Moderate	POC High	POC Very High	Total (n, %)
Low (<6 points, 7.5%)	25	1	0	0	26 (8.8)
Moderate (6–10, 14%)	2	106	2	0	110 (37.3)
High (11–15, 26%)	0	5	69	1	75 (25.4)
Very High (≥16, 57%)	0	0	7	77	84 (28.5)
Total (n, %)	27 (9.2)	112 (38)	78 (26.4)	78 (26.4)	295 (100)

Percent agreement = 99.3%; weighted κ = 0.97 (95% CI 0.95–0.98).
**Predicted risk of contrast-associated acute kidney injury (CA-AKI) according to the original Mehran score publication.*

Association Between Predicted Risk and Observed AKI

Follow-up creatinine measurements sufficient to assess AKI according to KDIGO criteria were available for 127 of 295 patients (43%). AKI occurred in 11.0% (14/127). Figure 1 illustrates the relationship between predicted CA-AKI risk categories and observed AKI outcomes based on risk classification derived from (a) POC creatinine and (b) laboratory creatinine. Across both methods, AKI events were disproportionately concentrated among patients categorized as high or very high risk, while patients in low or moderate categories predominantly experienced no AKI. The overall pattern of risk-to-outcome transitions was consistent between POC- and laboratory-based stratification.

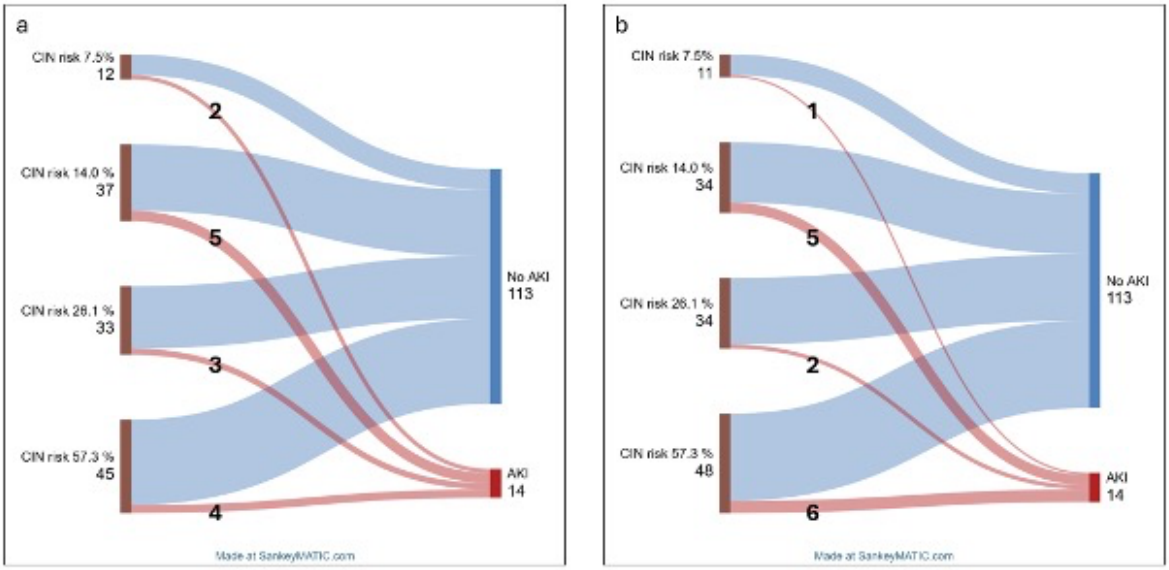

Figure 1. Relationship between predicted contrast-associated acute kidney injury (CA-AKI) risk categories and observed AKI outcomes based on (a) point-of-care and (b) laboratory creatinine.

Association Between Laboratory and Point-Of-Care Creatinine Measurements

Laboratory and POC creatinine values demonstrated a statistically significant positive correlation ($r = 0.63$, $p < 0.001$) (Figure 2). Although dispersion was observed, particularly at higher creatinine values, the results demonstrate a moderate linear association between laboratory and point-of-care creatinine measurements.

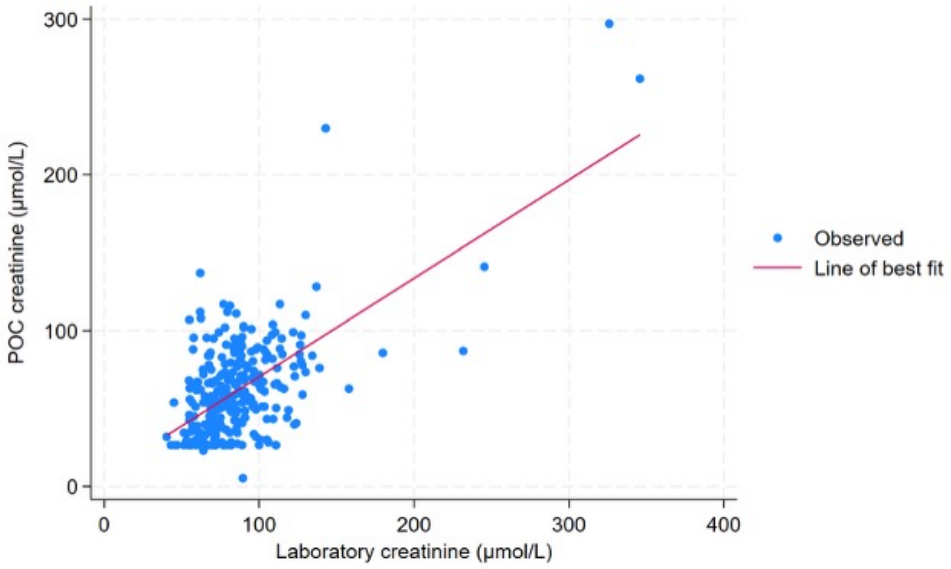

Figure 2. Association between point-of-care and laboratory serum creatinine measurements. Laboratory and POC creatinine showed moderate correlation ($r = 0.63$, $p < 0.001$).

Discussion

In this prospective observational cohort of patients undergoing contrast angiography for acute cardiovascular and cerebrovascular indications, we found near-perfect agreement between laboratory and point-of-care creatinine-based Mehran risk stratification. The excellent agreement in categorical risk stratification (weighted $\kappa = 0.97$) should be interpreted separately from the moderate correlation observed between continuous creatinine values ($r = 0.63$), as these metrics capture fundamentally different aspects of method comparison. Importantly, both approaches demonstrated similar and clinically meaningful relationships between predicted risk strata and observed AKI outcomes. These findings support the clinical validity of POC creatinine for rapid bedside risk stratification in time-sensitive angiography settings.

Our results show that accurate risk stratification does not require exact numerical agreement between laboratory and point-of-care creatinine measurements. Although the correlation between laboratory and POC creatinine values was only moderate, it did not yield meaningful differences in risk classification. Notably, point-of-care–derived eGFR values were higher than laboratory-based estimates, suggesting a tendency toward overestimation. This discrepancy likely reflects measurement variability and calibration differences between point-of-care devices and central laboratory assays. While such differences may be clinically relevant in borderline cases near risk category thresholds, they did not result in meaningful differences in categorical risk stratification in our cohort. Clinical decisions are typically guided by risk categories rather than precise creatinine values, and discordant classifications were uncommon and typically limited to neighboring risk categories. As a result, such discrepancies are unlikely to influence clinical management in most settings.

Our findings are consistent with prior studies evaluating point-of-care creatinine testing in emergency and catheterization laboratory settings, which have shown shorter turnaround times and clinically acceptable agreement with central laboratory measurements, particularly when results are used for risk stratification rather than exact value substitution [9,10,17]. However, published studies have reported variable concordance between point-of-care and laboratory creatinine measurements, and uncertainty regarding their impact on clinical workflow and decision-making has limited widespread adoption [9,17]. Beyond demonstrating agreement, our findings show that higher Mehran risk categories were consistently associated with higher rates of AKI, supporting the clinical relevance of point-of-care–based risk stratification in high-acuity angiography settings.

The importance of efficient and reliable renal risk assessment is reinforced by the high prevalence of CKD and its growth over recent decades [11]. CA-AKI has traditionally been attributed to renal vasoconstriction, medullary hypoxia, and tubular injury [19]. Although renal dysfunction after contrast exposure is often transient, AKI has been consistently associated with worse clinical outcomes, particularly in acute coronary syndrome populations [1,3]. However, contemporary analyses have questioned the extent to

which contrast exposure alone is causally responsible for kidney injury, emphasizing the contribution of underlying hemodynamic instability and comorbid conditions [18]. Moreover, large randomized trials evaluating preventive strategies such as intravenous sodium bicarbonate or N-acetylcysteine have demonstrated limited incremental benefit beyond optimized supportive care [19]. Together, these findings emphasize the importance of identifying patients at highest risk rather than applying universal preventive strategies to all individuals undergoing contrast exposure. In acute care settings where contrast-based angiography is frequently used, rapid and practical approaches to renal risk assessment are essential. Clinical practice guidelines support risk-based evaluation of kidney function prior to contrast exposure [5], reinforcing the need for timely and reliable stratification tools.

The clinical implications of our findings are most relevant to the catheterization laboratory and emergency settings. In acute myocardial infarction or stroke, waiting for central laboratory creatinine results may delay intervention or renal risk assessment. Prior studies have shown that point-of-care creatinine testing can reduce delays to contrast-enhanced imaging while maintaining acceptable agreement with laboratory measurements [9,10,17]. In this setting, point-of-care testing may enable rapid bedside application of risk prediction tools, such as the Mehran score [2], thereby facilitating earlier identification of patients at very high risk and supporting timely preventive measures in accordance with current clinical practice guidelines [5].

Limitations

This study has several limitations. Although 295 patients were enrolled, post-procedural creatinine measurements sufficient to assess AKI were available in only 127 patients (43%), which may introduce selection bias, as patients discharged early or without repeat testing may have differed from those with complete follow-up. Second, the small number of AKI events (14 cases) limited statistical power and precluded stable multivariable modeling. As a result, we were unable to adjust for additional potential confounders such as diabetes mellitus, baseline CKD, or contrast volume, which may influence the risk of post-contrast AKI. Third, urine output criteria were not used for AKI diagnosis due to incomplete standardized documentation, potentially leading to underestimation of AKI incidence. Fourth, this was a single-center study conducted in a high-volume tertiary-care setting, which may limit generalizability to other healthcare systems or resource-limited environments. Additionally, the relatively low prevalence of advanced CKD in this cohort limited our ability to perform stratified analyses according to baseline CKD status. We did not capture the time interval between clinical presentation and angiography, which may reflect patient acuity and could influence the risk of AKI. Finally, although agreement in risk classification was excellent, we did not evaluate long-term renal outcomes or mortality, and further studies are needed to determine whether point-of-care–guided risk stratification translates into improved clinical outcomes. Nevertheless, the prospective design, real-world acute care setting, and near-perfect agreement between risk classification methods support the internal validity and clinical relevance of the findings.

Conclusion

Point-of-care creatinine-based Mehran risk scoring demonstrates excellent agreement with laboratory-based assessment and shows a comparable association with post-contrast AKI outcomes in an acute angiography cohort. Despite only moderate correlation of absolute creatinine values, POC testing provides clinically reliable risk classification and may serve as a practical tool for rapid bedside risk stratification in patients undergoing angiography for acute stroke or myocardial infarction.

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Statement of Ethics: The study was approved by a local institutional ethics committee (Protocol No. 1/2023/ПЭ, approved on 15 June 2023). The study was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants.

Conflict of Interest: The authors have no conflicts of interest to declare.

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Data availability: The data that support the findings of this study are not publicly available due to patient privacy restrictions, but are available from the corresponding author upon reasonable request.

AI use statement: Generative AI tools were used only for language editing and manuscript formatting. All scientific content, analysis, and interpretation were performed by the authors.

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