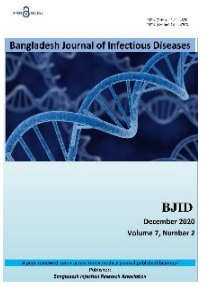
OPEN ACCESS Freely available online
http://www.banglajol.info/index.php/BJID/index
Original Article
Bangladesh Journal of Infectious Diseases
June 2025, Volume 12, Number 1, Page 134-140
ISSN (Online) 2411-670X
ISSN (Print) 2411-4820
NLM ID: 101761093
DOI: https://doi.org/10.3329/bjid.v12i1.84824



Post Percutaneous Coronary Intervention Outcomes Related to the Renal Seromarkers of Contrast-Induced Nephropathy Patients with Acute Coronary Syndrome

AKM Mohiuddin Bhuiyan[1], KAM Mahbub Hasan[2], Adneen Moureen[3], Md. Kamrul Hasan[4], Md. Zobayer[5], Shahnaz Choudhury[6]

[1]Professor and Head, Department of Cardiology, Popular Medical College & Hospital, Dhaka, Bangladesh; [2]Assistant Professor, Department of Cardiac Electrophysiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh; [3]Associate Professor, Department of Microbiology, Armed Forces Medical College, Dhaka, Bangladesh; [4]Assistant Registrar, Department of Cardiology, National Institute of Cardiovascular Diseases, Dhaka, Bangladesh; [5]Assistant Registrar, Department of Cardiology, Popular Medical College & Hospital, Dhaka, Bangladesh; [6]Associate Professor, Department of Radiology and Imaging, Bangladesh Medical University, Dhaka, Bangladesh

Abstract

Background: Urinary albumin excretion is recognized as an early marker of renal dysfunction and has been associated with adverse renal outcomes following percutaneous coronary intervention in patients with acute coronary syndrome. **Objective:** The purpose of this present study was to assess the development of contrast-induced nephropathy among patients with acute coronary syndrome undergoing percutaneous coronary intervention. **Methodology:** This cross-sectional analytical study was conducted in the Department of Cardiology at the National Heart Foundation Hospital and Research Institute (NHFH & RI), Dhaka, from August 2021 to July 2023. A total of 164 consecutive acute coronary syndrome patients undergoing percutaneous coronary intervention were enrolled and categorized into two groups based on urinary albumin excretion: microalbuminuria and normoalbuminuria. Microalbuminuria (MA) was defined as a urinary albumin-to-creatinine ratio (uACR) of 30–300 mg/g in a random spot urine sample. The relationship between pre-procedural microalbuminuria and the occurrence of contrast-induced nephropathy was further assessed using receiver operating characteristic (ROC) curve analysis. **Results:** Among the 164 patients, 67 (41%) had microalbuminuria and 97 (59%) had normoalbuminuria. The mean uACR was significantly higher in the microalbuminuria group (73.75 ± 5.6 mg/g) compared to the normoalbuminuria group (16.24 ± 6.43 mg/g). contrast-induced nephropathy developed in 24 patients (36%) in the microalbuminuria group, whereas only 15 patients (16%) in the normoalbuminuria group experienced contrast-induced nephropathy. ROC curve analysis identified a uACR threshold of 223.17 mg/g as the optimal cut-off for predicting contrast-induced nephropathy, with the best balance of sensitivity and specificity. **Conclusion:** In conclusion, pre-procedural microalbuminuria is significantly associated with an increased risk of contrast-induced nephropathy in acute coronary syndrome patients undergoing percutaneous coronary intervention. [*Bangladesh Journal of Infectious Diseases, June 2025;12(1):134-140*]

Keywords: Microalbuminuria; contrast-induced nephropathy; acute coronary syndrome; primary percutaneous coronary intervention

Correspondence: Prof. Dr. AKM Mohiuddin Bhuiyan, Professor and Head, Department of Cardiology, Popular Medical College & Hospital, Dhanmondhi-2, Dhaka-1205, Bangladesh**; Email:** mbhuiyan24@yahoo.com; **Cell No.:** +8801711-822986; **ORCID:** https://orcid.org/0009-0002-2155-0856

Introduction

Despite substantial progress in interventional cardiology, in-hospital mortality following primary percutaneous coronary intervention remains higher among patients presenting with certain subtypes of ST-elevation myocardial infarction[1]. Several clinical predictors of in-hospital mortality in acute coronary syndrome patients have been identified, including elevated Killip class, low systolic blood pressure, tachycardia, cardiac arrest at presentation, advanced age, prior myocardial infarction (MI) or heart failure, peripheral arterial disease, chronic kidney disease (CKD), and raised baseline serum creatinine levels[2].

Urinary albumin excretion has emerged as one of the earliest markers of renal injury, reflecting underlying endothelial dysfunction and increased glomerular permeability. Importantly, albuminuria is independently associated with both short- and long-term adverse cardiovascular outcomes in the general population, irrespective of the presence of hypertension or diabetes[3-4]. Acute changes in albuminuria have been reported in acute coronary syndrome patients, with peak urinary albumin levels occurring on the first day of the event[5]. Several comorbid conditions, such as obesity, diabetes, and dyslipidemia—which themselves correlate strongly with coronary artery disease—are closely linked to the occurrence of albuminuria[6]. Multiple studies have also demonstrated a significant association between microalbuminuria and increased mortality following acute coronary syndrome, including in patients undergoing primary percutaneous coronary intervention[7-8].

Another major complication in this setting is contrast-induced nephropathy, which remains the third leading cause of hospital-acquired acute kidney injury. contrast-induced nephropathy is associated with higher risks of myocardial infarction, need for dialysis, longer hospital stays, increased healthcare costs, and mortality[9-10]. Even minimal rises in serum creatinine following contrast exposure have been linked to adverse outcomes, underscoring why contrast-induced nephropathy prevention and prediction have become global healthcare priorities.

Contrast-induced nephropathy is particularly relevant among acute coronary syndrome patients undergoing PCI, as these patients frequently present with hemodynamic instability, high thrombotic burden, and reduced renal perfusion. Studies have shown that contrast-induced nephropathy occurs more often in acute coronary syndrome patients undergoing urgent PCI compared to stable outpatients, partly because prophylactic measures like hydration protocols are more challenging in emergency settings[11-12].

The incidence of contrast-induced nephropathy varies according to patient population and clinical context. In low-risk outpatients undergoing routine radiological imaging with iodinated contrast media, incidence rates are generally less than 5.0% cases[13-14]. However, the risk rises substantially to 10.0% to 15.0% in patients undergoing coronary angiography[15-16]. Among high-risk acute coronary syndrome patients undergoing primary percutaneous coronary intervention, reported contrast-induced nephropathy rates range widely from 12.0% to 46.0% cases[17-18]. While the overall incidence of contrast-induced nephropathy has declined in recent years due to improved awareness, better prophylactic strategies, and the use of newer contrast media with lower nephrotoxicity, it remains a significant complication[19].

Several risk factors for contrast-induced nephropathy development have been identified, even in patients without known preexisting kidney disease. These include hemodynamic compromise, thromboembolic events, and adverse drug interactions. The type, volume, and osmolarity of contrast media, as well as the adequacy of preventive measures, also play critical roles[17]. High doses of iodinated contrast media are often necessary during PCI, increasing the risk of renal injury despite the generally good tolerance of modern contrast agents[13].

Traditional predictors of contrast-induced nephropathy include low estimated glomerular filtration rate (eGFR), advanced age, diabetes mellitus, and volume depletion[15]. Baseline renal impairment, indicated by elevated serum creatinine or reduced eGFR, is consistently recognized as one of the strongest independent predictors[19]. Albuminuria has gained recognition as an important biomarker, not only for acute kidney injury (AKI) during the early phase[11], but also in the chronic setting, where it plays a central role in the definition, staging, and prognosis of chronic kidney disease[13]. However, whether albuminuria specifically predicts non-recovery after contrast-induced nephropathy remains less clearly established.

Overall, contrast-induced nephropathy in acute coronary syndrome patients post-PCI is a multifactorial complication that can occur even in individuals with preserved baseline renal function.

While its clinical course may be relatively benign in patients with normal renal reserve, contrast-induced nephropathy is often associated with worse in-hospital outcomes and increased long-term risks, including chronic renal impairment and mortality. Consequently, risk stratification, early identification of susceptible patients, and implementation of preventive measures remain essential in clinical practice to mitigate contrast-induced nephropathy-related morbidity and mortality. The purpose of this present study was to assess the development of contrast-induced nephropathy among patients with acute coronary syndrome undergoing percutaneous coronary intervention.

Methodology

Study Settings and Population: This cross-sectional analytical study was conducted in the Department of Cardiology at the National Heart Foundation Hospital and Research Institute (NHFH&RI), Dhaka, Bangladesh. The study period spanned from August 2021 to July 2023. A total of 164 patients of both sexes, who were admitted with acute coronary syndrome and underwent percutaneous coronary intervention (PCI), were included in the study through purposive sampling.

Selection Criteria: Inclusion criteria encompassed all adult patients presenting with acute coronary syndrome who underwent percutaneous coronary intervention during the study period. exclusion criteria were acute coronary syndrome patients with macroalbuminuria, acute coronary syndrome patients with hemodynamic instability (cardiogenic shock or acute left ventricular failure), Patients with chronic coronary syndrome undergoing PCI, patients with acute kidney injury or pre-existing chronic kidney disease, patients receiving NSAIDs or other nephrotoxic drugs within 48 hours before the procedure, Patients with severe liver dysfunction, malignancy, infectious diseases, thyroid dysfunction, or coagulation disorders, Patients with recent (within two weeks) exposure to contrast media,

Grouping of Study Population: Based on urinary albumin-creatinine ratio (uACR), study subjects were stratified into two groups called group I microalbuminuria (uACR >30 mg/g, *n=67*) and group II: normoalbuminuria (uACR <30 mg/g, *n=97*).

Study Procedure: After obtaining ethical clearance, eligible patients were enrolled following written informed consent. Baseline demographic information, medical history, and clinical data were systematically collected. All patients underwent a 12-lead ECG on admission and echocardiographic evaluation within the first 24 hours. Blood samples were collected for High-sensitivity Troponin I (hs-TnI), NT-proBNP, Fasting lipid profile, Complete blood count (CBC), and serum creatinine, sodium, and potassium. Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) formula. Urinary albumin was measured from a random pre-procedural urine sample using the turbidimetric immunological technique with Dimension EXL with LM Integrated Chemistry System. The urinary albumin-to-creatinine ratio (uACR) was calculated and categorized into normoalbuminuria (<30 mg/g) and microalbuminuria (30–300 mg/g). All patients underwent coronary angiography and PCI was performed by an expert interventional team following international guidelines. The primary endpoint was the occurrence of contrast-induced nephropathy, defined as either a ≥25% relative rise or an absolute rise of ≥0.5 mg/dL (44 µmol/L) in serum creatinine measured within 48–72 hours post-procedure, in the absence of any alternative cause.

Statistical Analysis: Data were compiled and analyzed using SPSS version 23.0 (IBM, USA). Descriptive statistics were expressed as frequency and percentage for categorical variables, and as mean ± standard deviation (SD) for continuous variables. Group comparisons were conducted using the Chi-square test for categorical data and the unpaired Student's t-test for continuous data. The predictive performance of pre-procedural uACR for contrast-induced nephropathy was evaluated using receiver operating characteristic (ROC) curve analysis. Furthermore, multivariate logistic regression was applied to determine the independent association of microalbuminuria with the risk of contrast-induced nephropathy. A p-value <0.05 was considered statistically significant.

Ethical Considerations: The study protocol was reviewed and approved by the Ethics Review Committee of NHFH & RI. Written informed consent was obtained from all patients or their legal guardians after explaining the study objectives, potential risks, and benefits. Participation was entirely voluntary, and patients retained the right to withdraw at any stage without justification. Strict measures were taken to ensure confidentiality, anonymity, and privacy of the study participants, and clinical care was not compromised by study participation.

Results

This cross-sectional analytical study was conducted in the Department of Cardiology, National Heart Foundation Hospital and Research Institute, Dhaka, between August 2021 and July 2023. The primary objective was to evaluate the relationship between microalbuminuria and the development of contrast-induced nephropathy among patients with acute coronary syndrome undergoing percutaneous coronary intervention (PCI). A total of 164 consecutive acute coronary syndrome patients were included, of whom 67 had microalbuminuria and 97 had normoalbuminuria. Patients were stratified into two groups: Group I (Microalbuminuria) and Group II (Normoalbuminuria). Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal urinary albumin-to-creatinine ratio (uACR) cutoff value for contrast-induced nephropathy prediction.

Table 1: Age Distribution According to Albuminuria Status (n = 164)

Age Group	Group I	Group II	P value
30 to 40 Years	7(36.8%)	12(63.2%)	
41 to 50 Years	17(40.5%)	25(59.5%)	0.373
51 to 60 Years	17(33.4%)	34(66.6%)	
>60 Years	26(50.0%)	26(50.0%)	
Total	**67**	**97**	
Mean ± SD	55.5±11.7	54.4±10.7	0.514

Group I: Microalbuminuria; Group II: Normoalbuminuria

The majority of patients in both groups were above 60 years of age. While the 51 to 60 years group was more prevalent in Group II, the mean age was comparable between the two groups ($p > 0.05$) (Table 1).

Table 2: Distribution of Risk Factors by Albuminuria Status (n = 164)

Risk Factors	Group I	Group II	P value
DM	31(46.3%)	24(24.7%)	0.004
Hypertension	36(53.7%)	55(56.7%)	0.707
Dyslipidemia	16(23.9%)	17(17.5%)	0.318
Smoking	42(62.7%)	58(59.8%)	0.709
BMI (kg/m²)	25.8±2.6	26.1±2.72	0.443

Group I: Microalbuminuria; Group II: Normoalbuminuria; DM=Diabetes Mellitus

The prevalence of diabetes mellitus was significantly higher in the microalbuminuria group ($p < 0.05$). Other risk factors such as hypertension, dyslipidemia, smoking, and BMI did not differ significantly between the groups (Table 2).

Table 3: Distribution by Clinical Diagnosis (n = 164)

Diagnosis	Group I	Group II	P value
STEMI	39(58.2%)	41(42.3%)	
NSTEMI	25(37.3%)	52(53.6%)	0.115ns
UA	3(4.5%)	4(4.1%)	

Group I: Microalbuminuria; Group II: Normoalbuminuria; ns = non-significant.

The distribution of clinical presentations (STEMI, NSTEMI, and UA) was comparable between the two groups, with no statistically significant differences (Table 3).

Table 4: Comparison of Biochemical Variables (Mean ± SD)

Biochemical Variable	Group I	Group II	P value
Hemoglobin(%)	11.2±1.13	11.7±1.16	0.003
eGFR(mL/min/ 1.73m²)	81.1±14.82	83.0±17.61	0.479
NT-Pro-BNP (pg/mL)	387.3±92.7	214.1±89.64	0.001
S. Troponin I (ng/mL)	29.9±14.46	30.8±14.81	0.954

Group I: Microalbuminuria; Group II: Normoalbuminuria

Patients in the microalbuminuria group had significantly lower hemoglobin levels and higher NT-proBNP values compared to those in the normoalbuminuria group ($p < 0.05$). Serum creatinine-derived eGFR and troponin I levels did not differ significantly between groups.

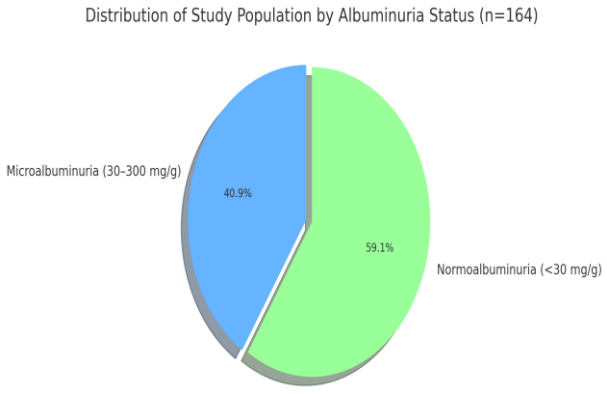


Figure I: Distribution of study population by albuminuria status (n = 164)

The pie chart (Figure I) illustrates that 40.9% of patients had uACR between 30–300 mg/g (Group I: Microalbuminuria), while the remaining 59.1% had uACR <30 mg/g (Group II: Normoalbuminuria).

Discussion

In the present study, the majority of patients were older than 60 years, reflecting the known age-related predisposition to coronary artery disease (CAD). The mean age in group I (microalbuminuria) was 55.53 ± 11.66 years, while in group II (normoalbuminuria) it was 54.38 ± 10.71 years. These findings are consistent with the results reported by Ali et al[13], who also observed a comparable mean age among patients with microalbuminuria. Similarly, Anwar et al[16] demonstrated that patients who developed contrast-induced nephropathy had a significantly higher mean age (58.17 ± 9.20 years) compared to those who did not (52.32 ± 10.88 years), which parallels the trends observed in our study. Adetunji et al[19] also reported a mean age of 62 years in microalbuminuric patients, supporting the notion that contrast-induced nephropathy age is a crucial determinant in the development of microalbuminuria and its sequelae. In contrast, Meng et al[20] found a statistically significant difference in mean age between microalbuminuric and normoalbuminuric groups, suggesting that age itself may be an independent factor. Variations in mean age across studies could be attributed to differences in geographic location, ethnicity, racial background, and genetic predisposition. The increased vulnerability of elderly individuals to microalbuminuria likely stems from multifactorial mechanisms, including age-related renal changes such as declining glomerular filtration rate, impaired tubular function, and reduced concentrating ability.

Diabetes mellitus emerged as one of the strongest predictors of microalbuminuria in this study. Among patients in group I, 31 (46.3%) were diabetic compared to 24 (24.7%) in group II, and this difference was statistically significant. This finding aligns with prior studies, including those by Anwar et al[16], Islam et al[22], Meng et al[20] and Anwar et al[16] all of which demonstrated a strong association between diabetes and microalbuminuria. Ali et al[13] highlighted the role of diabetes in the development of microvascular complications such as retinopathy and neuropathy, which in turn predispose to microalbuminuria and contrast-induced nephropathy. The observed differences across studies may be influenced by variations in diet, lifestyle practices, physical activity, family history, and socio-cultural background.

Hypertension is widely recognized as a major risk factor for chronic kidney disease (CKD). However, its association with microalbuminuria is more complex. In our study, hypertension was present in 36 (53.7%) patients in group I and 55 (56.7%) patients in group II, a difference that was not statistically significant. Similar nonsignificant results were reported by Islam et al[22] and Mridha et al[23]. On the other hand, Liu et al[14] and Adetunji et al[19] observed a significant association, with microalbuminuric patients having higher systolic and diastolic blood pressures compared to normoalbuminuric individuals. These contrasting findings may reflect differences in study populations, diagnostic thresholds, and blood pressure control across different cohorts.

Dyslipidemia is another well-established contributor to the excess burden of CAD in South Asian populations. In our study, dyslipidemia was detected in 16 (23.9%) patients in group I and 17 (17.5%) patients in group II, a difference that was not statistically significant. These results are consistent with those reported by Anwar et al[16], Mridha et al[23] and Siddike et al[24], suggesting that while dyslipidemia is prevalent in CAD patients overall, it may not independently distinguish between microalbuminuric and normoalbuminuric subgroups.

Obesity, often associated with metabolic syndrome, is also linked to microalbuminuria. In our study, obesity was present in 5 (13.4%) patients in group I compared to 13 (7.5%) in group II, but this difference was not statistically significant. These findings mirror those of Mridha et al[23], Meng et al[20], and Ali et al[13], indicating that obesity may contribute to microalbuminuria but does not consistently emerge as an independent predictor in all populations.

Anemia was also evaluated in this study. The mean hemoglobin level was 11.17 g/dl in group I compared to 11.73 g/dl in group II, findings that are in agreement with Adetunji et al[19] who reported a higher prevalence of anemia in microalbuminuric patients. They suggested that this association cannot be solely explained by impaired renal function, but may be related to reduced erythropoietin production. Similarly, Liu et al[14] identified anemia as an independent risk factor for contrast-induced nephropathy, particularly in patients with microalbuminuria.

Renal function, assessed by estimated glomerular filtration rate (eGFR), was also comparable between the two groups in our study. The mean eGFR was 81.11 ± 14.82 ml/min/1.73 m² in group I and 82.98 ± 17.61 ml/min/1.73 m² in group II, with no significant difference. These findings are in line

with those reported by Anwar et al[16]. In contrast, Adetunji et al[19] and Meng et al[20] found significant differences between the groups, suggesting that eGFR decline may not uniformly correlate with microalbuminuria across populations.

Finally, NT-proBNP, a marker of cardiac stress, showed a significant association with micro-albuminuria in our study. The mean NT-proBNP level in group I was 387.31 ± 92.72 pg/ml compared to 214.14 ± 89.64 pg/ml in group II, a statistically significant difference. This finding is supported by Mridha et al[23], who reported higher NT-proBNP levels in diabetic patients with vascular complications and microalbuminuria, and by Siddike et al[24], who also established a positive association. While the exact mechanism remains unclear, NT-proBNP may reflect the combined burden of cardiac dysfunction and renal microvascular damage in these patients.

Conclusion

In conclusion, the study analyzed the distribution of albuminuria status among patients, revealing that microalbuminuria was significantly associated with a higher prevalence of diabetes mellitus and notable differences in biochemical variables such as hemoglobin and NT-proBNP levels. Despite age distributions being comparable and no significant differences in clinical diagnoses between the groups, these findings highlight the importance of monitoring kidney function and associated risk factors in cardiovascular contexts. Overall, this research underscores the relevance of albuminuria as a critical indicator in managing patients at risk for cardiac events.

Acknowledgments
None

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

Financial Disclosure
None

Authors' contributions
Bhuiyan AKMM, Hasan KAMM: Conceptualization, Supervision, Investigation, Data curation, Resources; Moureen A, Hasan MK: Writing - original draft, Formal analysis, Validation, Methodology, Funding acquisition, Visualization, Project administration; Zobayer M, Choudhury S: Writing - review & editing, Validation; Bhuiyan AKMM: Investigation, Data curation, Software. All authors read and approved the final manuscript.

Data Availability
Any inquiries regarding supporting data availability of this study should be directed to the corresponding author and are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate
Ethical approval for this study was obtained from the Ethical Committee of State University of Bangladesh. Written informed consent was obtained from all participants prior to their inclusion in the study.

How to cite this article: Bhuiyan AKMM, Hasan KAMM, Moureen A, Hasan MK, Zobayer M, Choudhury S. Post Percutaneous Coronary Intervention Outcomes Related to the Renal Seromarkers of Contrast-Induced Nephropathy Patients with Acute Coronary Syndrome. Bangladesh J Infect Dis 2025;12(1):134-140

ORCID
AKM Mohiuddin Bhuiyan**:**
https://orcid.org/0009-0002-2155-0856
KAM Mahbub Hasan**:** https://orcid.org/0000-0002-4585-7345
Adneen Moureen: https://orcid.org/0000-0001-8732-6481
Md. Kamrul Hasan**:** https://orcid.org/0009-0004-2149-0886
Md. Zobayer: https://orcid.org/0009-0006-3332-066X
Shahnaz Choudhury**:** https://orcid.org/0009-0006-4425-1524

Article Info
Received on: 1 March 2025
Accepted on: 20 April 2025
Published on: 1 June 2025

References

1. Thrane PG, Olesen KK, Thim T, Gyldenkerne C, Mortensen MB, Kristensen SD, Maeng M. Mortality trends after primary percutaneous coronary intervention for ST-segment elevation myocardial infarction. Journal of the American College of Cardiology. 2023;82(10):999-1010.
2. Del Buono MG, Montone RA, Rinaldi R, Gurgoglione FL, Meucci MC, Camilli M, Iannaccone G, Sanna T, Pedicino D, Trani C, Niccoli G. Clinical predictors and prognostic role of high Killip class in patients with a first episode of anterior ST-segment elevation acute myocardial infarction. Journal of Cardiovascular Medicine. 2021;22(7):530-8.
3. Pafundi PC, Garofalo C, Galiero R, Borrelli S, Caturano A, Rinaldi L, Provenzano M, Salvatore T, De Nicola L, Minutolo R, Sasso FC. Role of albuminuria in detecting cardio-renal risk and outcome in diabetic subjects. Diagnostics. 2021;11(2):290.
4. Min KD, Matsumoto Y, Asakura M, Ishihara M. Rediscovery of the implication of albuminuria in heart failure: emerging classic index for cardiorenal interaction. ESC Heart Failure. 2024;11(6):3470-87
5. Åkerblom A, Clare RM, Lokhnygina Y, Wallentin L, Held C, Van de Werf F, et al. Albuminuria and cardiovascular events in patients with acute coronary syndromes: results from the TRACER trial. American heart journal. 2016;178:1-8
6. Bozkurt B, Aguilar D, Deswal A, Dunbar SB, Francis GS, Horwich T, et al. Contributory risk and management of comorbidities of hypertension, obesity, diabetes mellitus, hyperlipidemia, and metabolic syndrome in chronic heart

failure: a scientific statement from the American Heart Association. Circulation. 2016;134(23):e535-78.
7. Khalil AO, Frere AE, Abd El-Samei MM, Al-Daydamony MM. Microalbuminuria as a Predictor of Outcome in Non-Diabetic Patients Undergo Percutaneous Coronary Intervention for Acute Coronary Syndrome. Egyptian Journal of Hospital Medicine. 2021;85(2):4284-9.
8. Pan D, Chen H. Relationship between serum albumin level and hospitalization duration following percutaneous coronary intervention for acute coronary syndrome. Scientific Reports. 2024;14(1):23883.
9. Chen JW, Wang YL, Li HW. Elevated admission microalbuminuria predicts poor myocardial blood flow and 6-month mortality in ST-segment elevation myocardial infarction patients undergoing primary percutaneous coronary intervention. Clinical cardiology. 2012;35(4):219-24.
10. Mok Y, Ballew SH, Sang Y, Grams ME, Coresh J, Evans M, Barany P, Ärnlöv J, Carrero JJ, Matsushita K. Albuminuria as a predictor of cardiovascular outcomes in patients with acute myocardial infarction. Journal of the American Heart Association. 2019;8(8):e010546.
11. Bugani G, Ponticelli F, Giannini F, Gallo F, Gaudenzi E, Laricchia A, et al. Practical guide to prevention of contrast-induced acute kidney injury after percutaneous coronary intervention. Catheterization and Cardiovascular Interventions. 2021;97(3):443-50.
12. Tomey MI, Chyou JY. Management considerations for acute coronary syndromes in chronic kidney disease. Current Cardiology Reports. 2024;26(5):303-12.
13. Ali A, Bhan C, Malik MB, Ahmad MQ, Sami SA. The prevention and management of contrast-induced acute kidney injury: a mini-review of the literature. Cureus. 2018;10(9).
14. Liu Y, Hong D, Wang AY, Guo R, Smyth B, Liu J, Sun G, Chen S, Tan N, Jardine M, Brieger D. Effects of intravenous hydration on risk of contrast induced nephropathy and in-hospital mortality in STEMI patients undergoing primary percutaneous coronary intervention: a systematic review and meta-analysis of randomized controlled trials. BMC Cardiovascular Disorders. 2019;19(1):87.
15. Rear R. The effect of remote ischaemic preconditioning on contrast-induced nephropathy in the clinical setting of coronary angiography and percutaneous coronary intervention [Doctoral dissertation, UCL (University College London)]
16. Anwar MR, Hai AN, Debnath DK, Faraji MA, Hasan KM. Incidence and Risk Factors of Contrast Induced Nephropathy in Patients Following Coronary Angiography. Journal of Science Foundation. 2017;15(1):20-5.
17. Bhatt S, Rajpal N, Rathi V, Avasthi R. Contrast-induced nephropathy with intravenous iodinated contrast media in routine diagnostic imaging: an initial experience in a tertiary care hospital. Radiology research and practice. 2016;2016(1): 8792984.
18. Chipere TA. Contrast-Induced Nephropathy in Coronary Angiography Patients When Using Ioversol and Iomeprol: A Meta-Analysis (Master's thesis, University of South Africa (South Africa)).
19. Adetunji OR, Mani H, Olujohungbe A, Abraham KA, Gill GV. 'Microalbuminuric anaemia'—the relationship between haemoglobin levels and albuminuria in diabetes. Diabetes research and clinical practice. 2009;85(2):179-82.
20. Meng H, Wu P, Zhao Y, Xu Z, Wang ZM, Li C, Wang L, Yang Z. Microalbuminuria in patients with preserved renal function as a risk factor for contrast-induced acute kidney injury following invasive coronary angiography. European Journal of Radiology. 2016;85(6):1063-7
21. Goldfarb S, McCullough PA, McDermott J, Gay SB. Contrast-induced acute kidney injury: specialty-specific protocols for interventional radiology, diagnostic computed tomography radiology, and interventional cardiology. InMayo Clinic Proceedings 2009;84(2):170-179
22. Islam N, Badiuzzaman M, Asifudduza M, Faysal O, Quadir R, Ali S, et al. Association of Calculated Pre-Procedure Serum Osmolality with Development of Contrast-Induced Nephropathy among Patients Undergoing Percutaneous Coronary Intervention. Journal of Cardiovascular Disease Research. 2023;14(4):1558-1572
23. Mridha S, Rashid M, Kundu S, Momen A, Afroza N, Masum H, Alam I. Exploring the Correlation Between Fibrinogen-to-Albumin Ratio and Contrast-Induced Nephropathy in the Study Population. Annals of International Medical and Dental Research, 2024;10(1):50-61
24. Siddike S, Mamunur Rashid M, Karmakar PK, Hamid MA, Islam AR, Das S, et al. Association between contrast-induced nephropathy and CHA2DS2-VASc score in patient with non-ST elevation myocardial infarction after percutaneous coronary intervention. International Journal of Research in Medical Sciences. 2024;12(2):403