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Exploring the Correlation Between Fibrinogen-to-Albumin Ratio and Contrast-Induced Nephropathy in the Study Population

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Abstract

Background: Contrast-Induced Nephropathy (CIN) presents a significant risk in non-ST elevation acute coronary syndrome (NSTE-ACS) patients undergoing percutaneous coronary intervention (PCI). This study investigates the association between the Fibrinogen-to-Albumin Ratio (FAR) and CIN in such patients. **Material & Methods:** This cross-sectional study was conducted at the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, from June 2019 to May 2020. 200 NSTE-ACS patients undergoing PCI were categorized into two groups based on FAR: Group I (FAR ≥ 0.106, n=100) and Group II (FAR < 0.106, n=100). Clinical parameters, including pulse rate, blood pressure, and biochemical markers, were analyzed. The incidence of CIN and the role of FAR as a predictive marker were statistically evaluated. **Results:** Baseline clinical parameters showed no significant differences between the groups, with pulse rates averaging 88.8±16.4 bpm in Group I and 87.7±11.4 bpm in Group II (p=0.62). Troponin I levels were higher in Group I (42.1±24.6 ng/dl) compared to Group II (35.5±25.6 ng/dl, p=0.07). Group I also exhibited higher hemoglobin levels (12.5±1.5 gm/dl vs. 12.0±1.5 gm/dl in Group II, p=0.02). The incidence of CIN was significantly higher in Group I at 12%, compared to 2% in Group II. FAR was identified as a significant predictor of CIN, with an odds ratio of 11.45 (p=0.006). **Conclusions:** The study establishes FAR as a significant independent predictor of CIN in NSTE-ACS patients undergoing PCI. These findings suggest that FAR can be an effective biomarker for assessing CIN risk, potentially guiding more tailored patient management strategies in this high-risk group.

Keywords:- Fibrinogen-to-Albumin Ratio (FAR), Contrast-Induced Nephropathy (CIN), Biomarkers, Cardiovascular

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INTRODUCTION

Acute coronary syndrome (ACS), particularly non-ST segment elevation ACS (NSTEMI), represents a significant portion of cardiovascular emergencies, often necessitating percutaneous coronary intervention (PCI) as a primary treatment modality.[1] Globally, and particularly in Bangladesh, the prevalence of NSTEMI and the subsequent use of PCI have been steadily increasing, reflecting a growing burden on healthcare systems.[2] This rise underscores the need for a deeper understanding of the complications associated with PCI, notably contrast-induced nephropathy (CIN). CIN, defined as acute renal impairment following intravascular administration of contrast media, is a notable complication post-PCI, accounting for approximately 10% of all hospital-acquired acute kidney injuries.[3] The incidence of CIN in NSTEMI patients undergoing PCI is particularly concerning due to its association with increased morbidity and mortality. The clinical implications of CIN extend beyond the immediate post-procedural period, encompassing both short- and long-term adverse outcomes, making its prediction and prevention a clinical priority. In this context, the Fibrinogen-to-Albumin Ratio (FAR) emerges as a potential biomarker of interest. FAR, indicative of the balance between coagulation and albumin levels, has been increasingly recognized for its role in cardiovascular diseases, especially in ACS.[4] While existing literature has explored various biomarkers like the red blood cell distribution width-to-albumin ratio and the C-reactive protein/albumin ratio (CAR) in predicting CIN, the specific role of FAR in this context remains underexplored.[3,5]

Percutaneous coronary intervention (PCI) is the well-established recommended treatment of the reperfusion strategy and it contributes to the salvage of the myocardium and improvement of the prognosis.[6] However, even after successful PCI, both the short- and long-term morbidity and mortality of NSTEMI patients undergoing PCI remain high.[7,8] One such complication is contrast-induced nephropathy (CIN). CIN can be defined as the elevation of serum creatinine ≥ 0.5mg/dL or 44 µmol/L or 25% increase in baseline serum creatinine levels within 48 hours after PCI. Contrast-induced nephropathy (CIN) also described as contrast induced acute kidney injury (AKI) is an acute renal failure occurring within the days after exposure to intravascular radiographic contrast material that is not attributable to other causes. It is the third most common cause of hospital-acquired renal failure, after decreased renal perfusion and use of nephrotoxic medications.[9] Patients having PCI are at higher risk for CIN, although most of them do not have pre-procedural renal dysfunction.[10] CIN is low (<3%) in patients without known renal dysfunction or renal risk factors, whereas it is ≤ 50% in patients with known renal risk factors and dysfunction. In case of PCI, the risk of CIN is 10% to 20%.[11] The risk of CIN is significantly higher among patients with acute MI undergoing Emergency PCI than among the general population undergoing elective PCI (Wi et al., 2011). Usually, CIN develops within the first 2 to 3 days with a peak in serum creatinine level 3 to 5 days after contrast exposure. In general, serum creatinine normalizes within 7 to 10 days after contrast exposure. However, in patients with STEMI, after primary PCI the impaired renal function might persist in almost half of the patients with CIN.[11] Several risk

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factors for CIN have been identified; some are associated with pre-existing kidney vulnerability (chronic kidney disease, age, diabetes), some are due to the clinical situation (hypoxia, anaemia, heart failure, haemodynamic instability), and some are due directly to the toxicity of contrast media (type of contrast used and the volume of contrast injected.[9] The rationale for this study stems from the observed gap in current research regarding the correlation between FAR and CIN in NSTEMI patients post-PCI. Understanding this relationship is crucial, as it could lead to improved risk stratification and prophylactic strategies, potentially mitigating the incidence and severity of CIN. The significance of this study is further amplified by the potential clinical implications of identifying a reliable and easily measurable biomarker like FAR, which could aid in the early identification of patients at higher risk of developing CIN.

MATERIAL AND METHODS

This cross-sectional study was conducted at the Department of Cardiology, National Institute of Cardiovascular Diseases (NICVD), Dhaka, from June 2019 to May 2020. It focused on Non-ST Elevation Acute Coronary Syndrome (Non-ST ACS) patients undergoing Percutaneous Coronary Intervention (PCI). Using purposive sampling, 200 patients were divided into two groups based on the Fibrinogen-to-Albumin Ratio (FAR): Group 1 (FAR ≥ 0.106) and Group 2 (FAR < 0.106), with 100 patients in each group. Ethical approval was obtained from the NICVD Ethical Review Committee, and informed consent was secured from each participant or their legal guardians, ensuring the right to confidentiality and withdrawal at any stage. Inclusion criteria encompassed patients with Non-ST Elevation acute coronary syndromes undergoing PCI during their hospital stay. Exclusion criteria were established to omit patients with known kidney disease, recent contrast medium administration, previous PCI or CABG, use of nephrotoxic drugs, active infectious or inflammatory diseases, hepatic cirrhosis, malignancy, significant cardiac conditions, and severe hypotension or shock. Participants underwent detailed history taking and clinical examination, with data recorded in a structured questionnaire. This included demographic information, risk factor profiling, and a 12-lead resting ECG at admission. Treatment followed current guidelines, with nephrotoxic medications withheld upon admission. Laboratory investigations included serum creatinine, measured by Beckman Coulter Inc AU 480 Auto analyzer, and serum albumin, using the bromocresol green reagent. Serum fibrinogen levels were determined using an Automated Coagulation Analyzer STA Compact Max with STA –liquid Fib kit reagent. FAR was calculated from these measurements taken the day before PCI, forming the basis for group allocation. PCI was performed in line with standard clinical practices and institutional protocols. The contrast dose and type were at the discretion of the interventional cardiologist. Key procedural details, such as the number of vessels treated, duration, and complications, were documented. Post-procedural serum creatinine levels were measured within 24 to 48 hours to assess CIN development. Data analysis involved statistical evaluation using SPSS 17.0. Continuous variables were expressed as mean ± standard deviation and analyzed using the unpaired Student's t-test, while categorical variables were assessed using the Chi-squared or Fisher's exact

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test as appropriate. Logistic regression analysis was also employed, with statistical significance set at $p \leq 0.05$.

RESULTS

In the study comprising 200 participants, divided into Group I (n=100) and Group II (n=100), the baseline characteristics were analyzed. Age distribution across the groups showed a similar pattern, with the majority of participants in the 50-59 age range (38% in both groups). The mean age was slightly higher in Group I (56.3±8.9 years) compared to Group II (54.1±9.1 years), but this difference was not statistically significant (p=0.09). Gender distribution revealed a higher proportion of males in both groups, with 78% in Group I and 86% in Group II, though the difference was not significant (p=0.14). Body Mass Index (BMI) categories showed that overweight individuals (BMI 23 – 24.9) were the most common in both groups, accounting for 48% in Group I and 40% in Group II. The proportion of obese participants (BMI ≥ 25) was higher in Group I (38%) compared to Group II (28%), but these differences did not reach statistical significance. Regarding risk factors, smoking was reported by 42% of Group I and 34% of Group II, hypertension by 48% of Group I and 42% of Group II, and diabetes mellitus by 48% in Group I and 44% in Group II. The prevalence of dyslipidemia was identical in both groups (36%). Family history of Coronary Artery Disease (CAD) was slightly higher in Group I (14%) compared to Group II (10%), but this difference was not statistically significant. Symptom-wise, chest pain was nearly universal in both groups (100% in Group I and 99% in Group II). Shortness of breath was reported by a higher percentage in Group I (94%) compared to Group II (86%), and palpitations were reported by 12% of Group I and 10% of Group II participants. However, none of these symptom differences between the groups reached statistical significance. [Table 1]

The findings revealed a uniform distribution of Non-ST Elevation Myocardial Infarction (NSTEMI) and Unstable Angina (UA) across both groups. Specifically, NSTEMI was present in 90% of the patients in both Group I and Group II. Similarly, UA was reported in 10% of the patients in each group. The identical distribution of NSTEMI and UA in both groups resulted in a p-value of 1, indicating no statistically significant difference between Group I and Group II in terms of the types of non-ST ACS. [Table 2]

The average pulse rate was similar between the groups, with Group I at 88.8±16.4 beats per minute and Group II at 87.7±11.4 beats per minute (p=0.62). Systolic blood pressure was slightly higher in Group I (140.8±29.4 mmHg) compared to Group II (133.0±25.9 mmHg), but this difference was not statistically significant (p=0.11). Diastolic blood pressure also showed a higher mean in Group I (89.6±15.0 mmHg) than in Group II (82.1±13.2 mmHg), yet without reaching statistical significance (p=0.22). Random Blood Sugar (RBS) levels were comparable between the groups, with Group I having 12.1±5.1 mmol/L and Group II having 11.6±4.6 mmol/L (p=0.47). Troponin I levels were higher in Group I (42.1±24.6 ng/dl) compared to Group II (35.5±25.6 ng/dl), approaching statistical significance (p=0.07). Hemoglobin (Hb) levels were significantly different, with Group I showing a higher mean (12.5±1.5 gm/dl) than Group II (12.0±1.5 gm/dl) (p=0.02). White Blood Cell (WBC)

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Page no- 50-61 | Section- Research Article (Cardiology)

count, Hematocrit, Neutrophil, Lymphocyte, and Platelet counts all showed statistically significant differences between the groups. Group I had higher counts in all these parameters, with WBC at 14.7±3.2 (109/L), Hematocrit at 56.8±6.8%, Neutrophil at 11.5±2.9 (109/L), Lymphocyte at 2.6±0.9 (109/L), and Platelet at 376000±66815, compared to Group II, which had lower counts in these parameters ($p<0.001$ for all). Lipid profiles, including Total Cholesterol, Triglyceride, HDL, and LDL cholesterol, showed no significant differences between the groups, with p-values ranging from 0.05 to 0.34. However, Fibrinogen levels were significantly higher in Group I (4.7±0.6 g/L) compared to Group II (3.3±0.6 g/L) ($p<0.001$). Serum Albumin levels were significantly lower in Group I (31.4±1.8 g/L) compared to Group II (38.2±3.1 g/L) ($p<0.001$). Consequently, the Fibrinogen to Albumin Ratio (FAR) was significantly higher in Group I (0.150±0.023) compared to Group II (0.086±0.013) ($p<0.001$). [Table 3]

In assessing the volume of contrast agent used during Percutaneous Coronary Intervention (PCI) in the study of 200 non-ST Elevation Acute Coronary Syndrome (non-ST ACS) patients, a comparison was made between Group I (n=100) and Group II (n=100). The data revealed that in Group I, 64% of the patients (64 out of 100) received more than 150 ml of contrast agent, while in Group II, this proportion was slightly lower at 60% (60 out of 100). Conversely, 36% of patients in Group I and 40% of patients in Group II received 150 ml or less of the contrast agent. However, the difference in the volume of contrast agent used between the two groups was not statistically significant, with a p-value of 0.56. [Table 4]

In the study involving 200 non-ST Elevation Acute Coronary Syndrome (non-ST ACS) patients, the Left Ventricular Ejection Fraction (LVEF) was compared between Group I (n=100) and Group II (n=100). The distribution of LVEF percentages indicated that 38% of patients in Group I had moderate LV dysfunction (LVEF 36-45%), compared to 32% in Group II. Meanwhile, mild LV dysfunction (LVEF 45-54%) was observed in 62% of patients in Group I and 68% in Group II. The mean LVEF was slightly higher in Group II (46.5±4.4%) compared to Group I (45.8±3.0%). However, these differences in the distribution of LVEF categories and the mean LVEF values between the two groups were not statistically significant, with a p-value of 0.26. [Table 5]

In evaluating the incidence of contrast-induced nephropathy (CIN) in the study of 200 non-ST Elevation Acute Coronary Syndrome (non-ST ACS) patients, a significant difference was observed between Group I (n=100) and Group II (n=100). The data revealed that CIN developed in 12% of patients in Group I (12 out of 100), whereas in Group II, only 2% of the patients (2 out of 100) developed CIN. Conversely, 88% of patients in Group I and 98% in Group II did not develop CIN. This difference in the incidence of CIN between the two groups was statistically significant, with a p-value of less than 0.001. [Table 6]

In the study comprising 200 non-ST Elevation Acute Coronary Syndrome (non-ST ACS) patients, significant differences were observed in serum fibrinogen, albumin levels, and the Fibrinogen to Albumin Ratio (FAR) between Group I (n=100) and Group II (n=100). Group I exhibited a higher mean serum fibrinogen level (4.8±1.1 g/l) compared to Group II (3.9±0.9 g/l),

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Page no- 50-61 | Section- Research Article (Cardiology)

with the difference being statistically significant (p<0.001). Similarly, serum albumin levels were lower in Group I (31.4±1.7 g/l) than in Group II (35.1±4.3 g/l), and this difference was also statistically significant (p=0.002). Furthermore, the mean FAR was significantly higher in Group I (0.153±0.036) compared to Group II (0.116±0.036), with a p-value of less than 0.001. These findings indicate a notable difference in the biochemical profile related to fibrinogen and albumin levels between the two groups, which is directly relevant to the study's focus on the association between FAR and the development of CIN in non-ST ACS patients post-PCI. [Table 7]

In identifying independent predictors for contrast-induced nephropathy (CIN) in the study of 200 non-ST Elevation Acute Coronary Syndrome (non-ST ACS) patients, a multivariate logistic regression analysis revealed several key variables. Age greater than 60 years was a significant predictor of CIN, with a regression coefficient of 2.351, an odds ratio (OR) of 10.49, and a 95% confidence interval (CI) ranging from 2.091 to 52.646 (p=0.004). Hypertension showed an OR of 3.82 (95% CI: 0.866-16.868) and a regression coefficient of 1.341, approaching statistical significance (p=0.07). Diabetes mellitus, with a regression coefficient of 1.025 and an OR of 2.79 (95% CI: 0.485-16.018), and contrast volume greater than 150ml, with a regression coefficient of 1.84 and an OR of 6.29 (95% CI: 0.688-57.586), were also identified as potential predictors, but these did not reach statistical significance (p=0.25 and p=0.1, respectively). Notably, a raised Fibrinogen to Albumin Ratio (FAR) of ≥0.106 emerged as a significant independent predictor of CIN. The regression coefficient for raised FAR was 2.438, with an OR of 11.45 and a 95% CI of 2.014-65.097, indicating a strong association with the development of CIN (p=0.006). [Table 8]

Table 1: Baseline characteristics distribution of the participants (N=200)

Variable	**Group I (n=100)**		**Group II (n=100)**		**p-value**
	Frequency	**Percentage**	**Frequency**	**Percentage**	
Age					
30 – 39	2	2%	4	4%	0.09
40 – 49	16	16%	22	22%	
50 – 59	38	38%	38	38%	
60 - 69	35	35%	34	34%	
70 - 79	9	9%	2	2%	
Mean ± SD	56.3±8.9		54.1±9.1		
(Range)	(38-73)		(36-72)		
Gender					
Male	78	78%	86	86%	0.14
Female	22	22%	14	14%	
		BMI			
< 18.5 (Underweight)	0	0%	4	4%	0.13
18.5 – 22.9 (Normal weight)	24	24%	28	28%	0.52

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Page no- 50-61 | Section- Research Article (Cardiology)

23 – 24.9 (Overweight)	48	48%	40	40%	0.25
≥ 25 (Obese)	38	38%	28	28%	0.13
Risk factors					
Smoking	42	42%	34	34%	0.24
Hypertension	48	48%	42	42%	0.39
Diabetes mellitus	48	48%	44	44%	0.57
Dyslipidemia	36	36%	36	36%	1
Family H/O CAD	14	14%	10	10%	0.38
Symptoms					
Chest pain	100	100%	99	99%	0.17
Shortness of breath	94	94%	86	86%	0.06
Palpitation	12	12%	10	10%	0.65

Table 2: Distribution of non ST-ACS patients clinically (N=200)

Types of non ST-ACS	**Group I (n = 100)**		**Group II (n = 100)**		**p-value**
	Frequency	**Percentage**	**Frequency**	**Percentage**	
NSTEMI	90	90%	90	90%	1
UA	10	10%	10	10%	

Table 3: Distribution of Baseline clinical parameters and biochemical findings of the study patients (N=200)

Clinical and Biochemical variables	**Group I (n=100)**	**Group II (n=100)**	**p-value**
	Mean ± SD	**Mean ± SD**	
Pulse/min	88.8±16.4	87.7±11.4	0.62
Systolic Blood pressure (mmHg)	140.8±29.4	133.0±25.9	0.11
Diastolic Blood pressure (mmHg)	89.6±15.0	82.1±13.2	0.22
RBS (mmol/L)	12.1±5.1	11.6±4.6	0.47
Troponin I (ng/dl)	42.1±24.6	35.5±25.6	0.07
Hb (gm/dl)	12.5±1.5	12.0±1.5	0.02
WBC (109/L)	14.7±3.2	12.2±2.2	<0.001
Hematocrit	56.8±6.8	37.3±6.1	<0.001
Neutrophil (109/L)	11.5±2.9	9.4±2.4	<0.001
Lymphocyte (109/L)	2.6±0.9	2.2±0.6	<0.001
Platelet	376000±66815	314000±94432	<0.001
Total Cholesterol (mg/dl)	189.6±37.6	180.7±27.5	0.06
Triglyceride (mg/dl)	301.2±63.6	292.6±64.4	0.34
HDL cholesterol (mg/dl)	41.9±6.2	43.5±6.4	0.08
LDL cholesterol (mg/dl)	143.6±25.1	136.6±24.2	0.05
Fibrinogen (g/L)	4.7±0.6	3.3±0.6	<0.001
Serum Albumin (g/L)	31.4±1.8	38.2±3.1	<0.001
Fibrinogen to Albumin Ratio (FAR)	0.150±0.023	0.086±0.013	<0.001

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Page no- 50-61 | Section- Research Article (Cardiology)

Table 4: Comparison of amount of contrast agent used between two groups (N=200)

Volume of Contrast	Group I (n = 100)		Group II (n = 100)		p-value
	Frequency	Percentage	Frequency	Percentage	
>150 ml	64	64%	60	60%	0.56
≤ 150 ml	36	36%	40	40%	

Table 5: Comparison of LVEF between two groups (N=200)

LVEF %	Group I (n=100)		Group II (n=100)		p-value
	Frequency	Percentage	Frequency	Percentage	
Moderate LV dysfunction (36-45)	38	38%	32	32%	0.26
Mild LV dysfunction (45-54)	62	62%	68	68%	
Mean ± SD	45.8±3.0		46.5±4.4		

Table 6: Incidence of contrast induced nephropathy among studied patients (N=200)

CIN	Group I (n = 100)		Group II (n = 100)		p-value
	Frequency	Percentage	Frequency	Percentage	
Developed	12	12%	2	2%	<0.001
Not developed	88	88%	98	98%	

Table 7: Comparison of serum fibrinogen, albumin and FAR with contrast induced nephropathy (CIN) (N=200)

Variables	Group I (n=100)	Group II (n=100)	p-value
	Mean ± SD	Mean ± SD	
Serum fibrinogen (g/l)	4.8±1.1	3.9±0.9	<001
Serum albumin (g/l)	31.4±1.7	35.1±4.3	0.002
Fibrinogen to Albumin Ratio (FAR)	0.153±0.036	0.116±0.036	<0.001

Table 8: Independent predictors for contrast induced nephropathy (N=200)

Variables of interest	Regression coefficient	OR	95% CI of OR	p value
Age>60 yrs	2.351	10.49	2.091-52.646	0.004
Hypertension	1.341	3.82	0.866-16.868	0.07
Diabetes mellitus	1.025	2.79	0.485-16.018	0.25
Contrast volume >150ml	1.84	6.29	0.688-57.586	0.1
Raised FAR≥0.106	2.438	11.45	2.014-65.097	0.006

DISCUSSION

The present study's exploration into the association between Fibrinogen-to-Albumin Ratio (FAR) and Contrast-Induced Nephropathy (CIN) in non-ST elevation acute coronary syndrome (NSTE-ACS) patients undergoing percutaneous coronary intervention (PCI) has yielded several significant insights. The baseline characteristics of our study population indicated a balanced age distribution across both groups, with the

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https://doi.org/10.53339/aimdr.2024.10.1.7
Page no- 50-61 | Section- Research Article (Cardiology)

majority in the 50-59 age range (38% in both groups). This demographic parity is crucial as it ensures that age-related factors do not confound the results, aligning with the findings of Kurtul et al., who also focused on a similar patient demographic in their study on Neutrophil-to-Lymphocyte Ratio (NLR) and CIN.[12] The study's baseline clinical parameters, including pulse rate, blood pressure, and Random Blood Sugar (RBS), showed no significant differences between Group I and Group II. The pulse rates were almost identical, with Group I averaging 88.8±16.4 beats per minute and Group II at 87.7±11.4 beats per minute (p=0.62). Similarly, systolic and diastolic blood pressures were slightly higher in Group I (140.8±29.4 mmHg and 89.6±15.0 mmHg, respectively) compared to Group II (133.0±25.9 mmHg and 82.1±13.2 mmHg, respectively), but these differences were not statistically significant (p=0.11 and p=0.22, respectively). These findings suggest that the two groups were well-matched in terms of these basic cardiovascular parameters, which is crucial for ensuring the validity of the study's comparisons. The biochemical parameters, however, revealed more pronounced differences. Troponin I levels were higher in Group I (42.1±24.6 ng/dl) compared to Group II (35.5±25.6 ng/dl), nearing statistical significance (p=0.07). This could indicate a higher degree of myocardial injury in Group I, which is a critical factor in the context of NSTE-ACS. Hemoglobin levels were significantly different, with Group I showing a higher mean (12.5±1.5 gm/dl) than Group II (12.0±1.5 gm/dl) (p=0.02). The higher hemoglobin in Group I might suggest a better initial oxygen-carrying capacity, which could have implications for patient outcomes. Notably, the white blood cell (WBC) count, hematocrit, neutrophil, lymphocyte, and platelet counts all showed statistically significant differences between the groups. Group I had higher counts in all these parameters (p<0.001 for all), suggesting a more pronounced inflammatory response or a higher baseline level of physiological stress. This finding aligns with the study by Kurtul et al., which emphasized the role of inflammatory markers in patients with NSTE-ACS.[12] The lipid profiles, including total cholesterol, triglycerides, HDL, and LDL cholesterol, showed no significant differences between the groups. However, fibrinogen levels were significantly higher in Group I (4.7±0.6 g/L) compared to Group II (3.3±0.6 g/L) (p<0.001), and serum albumin levels were significantly lower in Group I (31.4±1.8 g/L) compared to Group II (38.2±3.1 g/L) (p<0.001). Consequently, the FAR was significantly higher in Group I (0.150±0.023) compared to Group II (0.086±0.013) (p<0.001). These findings are crucial as they highlight the potential role of FAR as a biomarker in NSTE-ACS patients, a concept supported by the study of He et al.[13] Regarding the volume of contrast agent used, our study found no significant difference between the two groups, with 64% of Group I and 60% of Group II receiving more than 150 ml of contrast agent (p=0.56). This suggests that the contrast volume alone may not be a determining factor in the development of CIN, a finding that adds to the ongoing debate on the role of contrast volume in CIN as discussed in the study by Şaylık et al.[14] The comparison of Left Ventricular Ejection Fraction (LVEF) between the two groups also did not reveal any significant differences. The distribution of LVEF percentages indicated that moderate LV dysfunction (LVEF 36-45%) was present in 38%

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Vol-10, Issue-1 | Jan-Feb 2024
https://doi.org/10.53339/aimdr.2024.10.1.7
Page no- 50-61 | Section- Research Article (Cardiology)

of patients in Group I and 32% in Group II, while mild LV dysfunction (LVEF 45-54%) was observed in 62% of Group I and 68% of Group II. The mean LVEF was slightly higher in Group II (46.5±4.4%) compared to Group I (45.8±3.0%), but these differences were not statistically significant (p=0.26). This finding is consistent with the study by Satılmış and Karabulut, which also did not find a significant association between CAR and LVEF in NSTEMI patients.[3] The incidence of CIN in our study was notably higher in Group I (12%) compared to Group II (2%), with a significant p-value of less than 0.001. This finding is particularly relevant when juxtaposed with the study by Şaylık et al., which investigated Serum Uric Acid to Albumin Ratio (UAR) as a predictor for CIN in STEMI patients undergoing PCI.[14] While Şaylık et al. focused on a different biomarker, the emphasis on predictive ratios in CIN development is a common thread. Our study's identification of FAR as a significant independent predictor for CIN, with an odds ratio of 11.45 (p=0.006), resonates with the findings of He et al. (13). They found FAR to be a significant prognostic indicator in NSTE-ACS patients post-PCI, further validating our results. Additionally, the study by Ma et al., which developed a nomogram model incorporating FAR, supports our approach in using comprehensive models for risk stratification.[4] Interestingly, our study's focus on FAR as a predictive biomarker for CIN finds parallels in the work of Satılmış and Karabulut, who assessed the C-reactive protein/albumin ratio (CAR) in NSTEMI patients.[3] Their findings, which highlight the significance of inflammatory biomarkers in predicting CIN, align with our study's emphasis on FAR. Furthermore, the study by Sun et al. introduces the red blood cell distribution width-to-albumin ratio as a novel biomarker for predicting CIN after emergency PCI.[5] This study, along with the research by Winardi and Sitepu, which explores the Platelet-Lymphocyte Ratio (PLR) in STEMI patients, underscores the diverse yet potentially complementary approaches in identifying risk factors for CIN.[15] Our study also aligns with the recent research by Miyagawa et al., focusing on non-gated computed tomography in NSTE-ACS patients.[16] While their study does not directly relate to FAR, it provides context for diagnostic approaches in NSTE-ACS, which could influence the development of CIN. In conclusion, our study contributes significantly to the existing literature by highlighting the predictive value of FAR in CIN development among NSTE-ACS patients undergoing PCI. The comparative analysis with existing studies emphasizes the need for incorporating such biomarkers in clinical risk assessment protocols and suggests a potential avenue for future research in optimizing patient management strategies.

Limitations of The Study

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

CONCLUSIONS

In conclusion, this study has provided valuable insights into the association between the Fibrinogen-to-Albumin Ratio (FAR) and Contrast-Induced Nephropathy (CIN) in patients with non-ST elevation acute coronary syndrome (NSTE-ACS) undergoing percutaneous coronary intervention (PCI). Our findings highlight that FAR is a significant

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Page no- 50-61 | Section- Research Article (Cardiology)

independent predictor of CIN, emphasizing its potential as a crucial biomarker in this patient group. The study's detailed analysis of baseline characteristics and clinical parameters revealed well-matched cohorts, ensuring the validity of our comparisons. The higher incidence of CIN in one group compared to the other underscores the clinical relevance of FAR in patient management and risk stratification. This study contributes to the understanding of the complex interplay between various clinical and biochemical factors in NSTE-ACS patients and the development of CIN post-PCI. The emphasis on FAR, alongside the exploration of other biomarkers, illustrates the diverse approaches in identifying risk factors for CIN. Overall, this study underscores the need for incorporating biomarkers like FAR in clinical protocols for assessing the risk of CIN in NSTE-ACS patients undergoing PCI. The findings suggest potential avenues for future research in optimizing patient management strategies and improving clinical outcomes in this patient population.

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