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Blood urea nitrogen to left ventricular ejection fraction ratio and TIMI risk index as predictors for contrast- induced nephropathy in patients with acute coronary syndrome

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ABSTRACT

Background: Acute coronary syndrome (ACS) refers to a spectrum of clinical presentations ranging from those for ST-segment elevation myocardial infarction (STEMI) to presentations found in non–ST-segment elevation myocardial infarction (NSTEMI) or in unstable angina. Contrast-induced nephropathy (CIN) is defined as the impairment of renal function-measured as either a 25% increase in serum creatinine (SCr) from baseline or a 0.5 mg/dL (44 µmol/L) increase in absolute SCr value—within 48-72 hours after intravenous contrast administration.

Objective: To evaluate blood urea nitrogen to left ventricular ejection ratio and TIMI risk index as predictors for occurrence of CIN in patients with acute coronary syndrome who underwent PCI.

Patients and Methods: it included 300 patients (68% male and 32% female), mean of age is 57.4 ± 11.5 years old, with a range from 27 to 80 years old and 68% of them are male at Cardiology Department, Faculty of Medicine, Zagazig University; and Cardiology Department, National Heart Institute starting from July 2019 till July 2020.

Results: Patients were classified into two groups: CIN group vs non CIN group and we found that, hypotension positive inotrope, history of HF and history of TIA or stroke, were statistically high significant predictors for CIN, showed a statistical significant positive correlation between BUN\EFr level and BUN, creatinin level before and after PCI, TIMI risk index, contrast volume and mehran score and showed that sensitivity of BUN\EFr level as a predictor of CIN was 88.9% with ability to exclude 94.5% of truly negative cases, while sensitivity of mehran score in prediction of positive cases was 100% and 90% exclusion of negative cases, both tools had high accuracy above 90% and a high statistically significant tool used in prediction of CIN.

Conclusion: CIN is a frequent complication following PCI and is associated with complicated hospital stay and high mortality rate. Patients who are older and have associated comorbidities such as anemia, acute heart failure, high contrast volume and ratio, high TIMI risk index, high Killip score, high Mehran score and renal insufficiency at the baseline are at High risk of developing CIN. Those patients can be identified, and more vigilant preventive measures can then be applied for the prophylaxis of CIN. BUN/EFr and TIMI risk index score can be used to predict acute kidney injury in patients with ACS undergoing PCI.

Introduction

Acute coronary syndrome refers to a spectrum of clinical presentations ranging from those for ST-segment elevation myocardial infarction to presentations found in non–ST-segment elevation myocardial infarction or in unstable angina. It is almost always associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery**(1)**.

Contrast-induced nephropathy is defined as the impairment of renal function measured as either a 25% increase in serum creatinine from baseline or a 0.5 mg/dL (44 µmol/L) increase in absolute SCr value within 48-72 hours after intravenous contrast administration. For renal insufficiency to be attributable to contrast administration, it should be acute, usually occurring within 2-3 days (although it has been suggested that renal insufficiency developing up to 7 days post–contrast administration be considered CIN); it should also not be attributable to any other identifiable cause of renal failure Contrast-induced nephropathy is a common and potentially severe complication in patients with acute coronary syndrome who were undergoing percutaneous coronary intervention. The presence of CIN is also associated with increased morbidity and mortality **(2)**. CIN is one of the leading causes of hospital-acquired acute kidney injury. It is associated with a significantly higher risk of in hospital and 1-year mortality, even in patients who do not need dialysis. There is a complicated relationship between CIN, comorbidity, and mortality. Most patients who develop CIN do not die from renal failure. Death, if it does occur, is more commonly from either a preexisting non renal complication or a procedural complication **(3)**. The incidence of CIN ranges from 2 to 30% due to variations in study populations, clinical settings, and CIN definitions **(4)**.

Aim of the work

To assess the role of BUN to LVEF ratio and TIMI risk index as predictors for occurrence of CIN in patients with ACS who underwent PCI and to improve risk stratification of CIN to decrease morbidity and mortality in patients with ACS who underwent PCI.

Patients and methods

•**Type of Study**: Cohort study

•**Study Setting**: at Cardiology Department, Faculty of Medicine, Zagazig University; and Cardiology Department, National Heart Institute.

•**Study Period:** starting from July 2019 till July 2020.

•**Inclusion Criteria:** Adults (>18 years). All Patients with ACS who were treated by PCI.

•**Exclusion Criteria**: Patients who have chronic renal failure and on dialysis, Exposure to contrast within the preceding 5 days, Coincidental other emergent situations e.g: gastrointestinal bleeding, Patients who had not been had serum creatinine or Echocardiography at admission or lacked follow up, Patients who had had hematologic diseases, Patients who died during angiography or PCI.

•**Sampling Method**: Simple random sampling.

•**Sample Size:** 300 patients.

•**Ethical Considerations:** Clear explanation of the study was made for all cases and a written informed consent was taken for each. The study protocol was approved by Ethical Committee and Institutional Review Board (IRB) of Zagazig University.

Methods

Initial Assessment: All patients were initially seen.

Thorough examination and assessment were carried out. This included:

1.History: Demographic data including name, age, sex, residence, occupation, marital status, special habits of medical and surgical importance and current diabetes mellitus, dyslipidemia and hypertension. Previous PCI procedures and previous CABG, Other co-morbid conditions, such as previous cerebrovascular stroke, renal impairment, and the presence of peripheral vascular diseases. Smoking index is calculated.

2.Examination: General examination for vital signs (blood pressure, heart rate, respiratory rate and temperature) and body weight. Other systems examination.

Chest and heart auscultation were done.

3.Investigation: Laboratory investigations: Blood urea nitrogen, serum creatinine and other routine labs (CBC, Cardiac enzymes, Blood glucose level, Lipid profile) were measured for all patients. Serum creatinine was measured at admission and follow up serum creatinine was measured every day for the following 3 days post PCI. Blood urea nitrogen may also serve as a comprehensive marker reflecting impaired cardiology function and neurohormonal activation. Blood urea nitrogen: It has been shown that BUN was associated with mortality in patients with acute myocardial infarction. Because BUN reflects both glomerular filtration rate and neurohormonal activations, it may serve as a marker of CIN compared with creatinine. The left ventricular ejection fraction was found to be a predictor for CIN in other risk models.

Electrocardiography: A 12-lead surface ECG was done for each patient on admission and daily during CCU stay. These ECGs were used to assess the degree of ST-segment elevation before initiating mechanical reperfusion therapy. Complete electrocardiography shows types of acute coronary syndrome. The electrocardiograms were recorded at a paper speed of 25 mm/s and an amplification of 10 mm/mV

Echocardiography: We used GE VIVID IQ portable and Siemens ACUSON X700 Echocardiography machines to assess LVEF. The biplane method of disks (modified Simpson's rule) is the currently recommended two-dimensional method to assess LVEF. Modified Simpson method (biplane method of disks) is a modality requiring area tracings of LV cavity. The American Society of Echocardiography recommends this method for measuring LVEF. This method requires the measurement of LVEF by tracing the endocardial border in both the apical four-chamber and two-chamber views in end-systole and end-diastole. These tracings eventually divide the LV cavity into a predetermined number of disks.

Coronary Angiography and PCI procedure: All patients were given 600 mg clopidogrel, 300 mg aspirin, UFH/LMWH (low-dose unfractionated heparin, 50 U/kg, while the standard-dose of unfractionated heparin is 85 U/kg during the procedure and continued during hospital stay unless contraindicated, in addition to the conventional anti-ischemic and antianginal treatment as nitrates.

Results

In our study, the CIN group (59.7 ± 16.01) was older than the non CIN group (56.7 ± 11.1). There was significant decrease in hemoglobin level in CIN group compared with non CIN group. There was no significant relation between DM, Dyslipidemia and CIN. There was significant correlation between creatinine level pre- and post-PCI and development of CIN. There was high significant relation between TIMI risk index and Killip score among CIN group. The Mehran score was significant in predicting the occurrence of CIN.

Sensitivity of BUN\EFr level as a predictor of CIN was 88.9% with ability to exclude 94.5% of truly negative cases, while sensitivity of mehran score in prediction of positive cases was 100% and 90% exclusion of negative cases, both tools had high accuracy above 90% and a high statistically significant tool used in prediction of CIN.

Table (1): Relation between CIN development and basic characteristics of the studied groups.

		CIN N=27		Non-CIN N=273		t-test	P
Age\ years Mean ±SD		**59.7 ± 16.01**		**56.7 ± 11.1**		**0.46**	**0.66 NS**
		N	**%**	**N**	**%**	**X^2**	**P value**
Gender	**Male**	**21**	**77.8**	**183**	**67**	**0.44**	**0.51 NS**
	Female	**6**	**22.2**	**90**	**33**		
Smoking		**9**	**33.3**	**93**	**34.1**	**Fisher**	**0.97 NS**
DM		**15**	**55.6**	**147**	**53.8**	**Fisher**	**0.91 NS**
Hypertension		**15**	**55.6**	**129**	**47.3**	**Fisher**	**0.63 NS**
Hypotension\ positive inotrope		**12**	**44.4**	**9**	**3.3**	**Fisher**	**<0.001 HS**
Dyslipidemia		**12**	**44.4**	**54**	**19.8**	**Fisher**	**0.09 NS**
History of HF		**21**	**77.8**	**12**	**4.4**	**Fisher**	**<0.001 HS**
CAD		**9**	**33.3**	**66**	**24.2**	**Fisher**	**0.34 NS**
Previous PCI		**6**	**22.2**	**60**	**22**	**Fisher**	**0.99 NS**
Prior to TIA\ stroke or DVT		**21**	**77.8**	**18**	**6.7**	**Fisher**	**<0.001 HS**
HR	**Mean ±SD**	**80.6 ± 24.3**		**86.2 ± 9.9**		**1.22***	**0.22 NS**
SBP	**Mean ±SD**	**120 ± 27.7**		**130.9± 16.2**		**1.03***	**0.34 NS**
DBP	**Mean ±SD**	**77.9 ± 15.5**		**84.1 ± 12.1**		**1.05***	**0.33 NS**

Table (2): Relation between CIN development and assessment data among studied groups.

	CIN N=27 Mean ±SD (range)	Non-CIN N=273 Mean ±SD (range)	t-test	P value
LVEF	43.6 ± 6.97 32 – 54	51.98 ± 5.5 40 – 65	3.12	0.01 S
HGB	9.8 ± 1.4 7.5 – 13	11.4 ± 1.17 8.7 – 14	2.87	0.03 S
BUN	38.9 ± 5.24 35 – 50	27.6 ± 4.14 18 – 38	5.53	0.001 S
Creatinin pre-PCI	1.5 ± 0.48 0.8 – 2.2	0.87 ± 0.22 0.3 - 1.3	7.14	<0.001 HS
Creatinin post-PCI	2.93 ± 0.41 2.4 – 3.5	0.84 ± 0.24 0.4 – 1.4	10.64	<0.001 HS
BUN\EFR	0.96 ± 0.16 0.75– 1.14	0.54 ± 0.12 0.29– 0.8	7.01	<0.001 HS

Table (3): Relation between CIN development and clinical data among both studied groups.

	CIN N=27	Non-CIN N=273	X^2 t-test*	P value
Contrast volume (cc)				
Mean ±SD	227.8 ± 50.7	158.5 ± 50.2	9.61*	0.003
(Range)	(150 - 300)	(50 – 300)		S
Affected vessel n (%)				
Single vessel	9 (33.3%)	183 (67%)	Fisher	0.04 S
Two vessels	14 (44.4%)	54 (19.8%)	Fisher	0.08 NS
MVD	6 (22.2%)	36 (13.2%)	Fisher	0.46 NS
Balloon or stenting n (%)				
Yes	24 (88.9%)	228 (83.5%)	Fisher	0.68
No	3 (11.1%)	135 (16.5%)		NS

Table (4): Distribution of ACS types among both studied groups.

ACS	CIN N=27	Non-CIN N=273	X^2	P value
STEMI	27 (100%)	186 (68.1%)		
Non-STEMI	0 (0.0%)	54 (18.7%)	4.04	0.13
Unstable angina	0 (0.0%)	36 (13.2%)		NS

Table (5): Diagnostic scores among both studied groups.

	CIN N=27	Non-CIN N=273	X^2 MW*	P value
TIMI risk index				
Mean ±SD	**45 ± 18.1**	**20.9 ± 8.51**	**4.71***	**<0.001**
Median (Range)	**38 (26 – 84)**	**20 (6 – 63)**		**HS**
Mehran score				
Mean ±SD	**8.78 ± 5.1**	**5 ± 3.39**	**2.36***	**0.02**
Median (Range)	**7 (3 – 19)**	**3 (0 – 16)**		**S**
Killip class n (%)				
1	**12 (44.4%)**	**222 (81.3%)**		
2	**6 (22.2%)**	**42 (15.4%)**	**16.04**	**0.001**
3	**6 (22.2%)**	**3 (1.1%)**		**S**
4	**3 (11.1%)**	**6 (2.2%)**		

Table (6): Validity data of BUN\EFr level and mehran score as predictors of CIN among studied cases.

	BUN\EFr	Mehran score
Cut off	**>0.6**	**>6.5**
AUC (95% CI)	**0.924 (0.807-1.04)**	**0.998 (0.993-1.003)**
Sensitivity	**88.9%**	**100%**
Specificity	**94.5%**	**98.9%**
PVP	**61.5%**	**90%**
PVN	**98.9%**	**100%**
Accuracy	**93%**	**99%**
P- value	**<0.001 HS**	**<0.001 HS**

Table (7): correlation between BUN\EFr and other clinical data of the studied patients.

	BUN\EFr	
	R	**P value**
Age	**0.180**	**0.201 NS**
HR	**0.282**	**0.121 NS**
SBP	**-0.331**	**0.231 NS**
DBP	**-0.183**	**0.504 NS**
LVEF	**-0.723**	**<0.001 HS**
HGB	**-0.324**	**<0.001 HS**
BUN	**0.339**	**<0.001 HS**
Creatinin before PCI	**0.598**	**<0.001 HS**
Creatinin after PCI	**0.276**	**<0.001 HS**
Contrast volume	**0.371**	**<0.001 HS**
TIMI risk index	**0.580**	**<0.001 HS**
TIMI flow after PCI	**0.287**	**<0.001 HS**
Mehran score	**0.397**	**<0.001 HS**

Table (8): multivariate analysis of significant predictors of CIN among studied patients.

Variables	Multivariate analysis		
	B	95% CI	P value
History of HF	0.08	0.398 – 1.05	0.81 NS
Prior to TIA or stroke	-0.032	0.213-1.83	0.04 S
Hypotension\ positive inotrope	0.120	0.435-2.14	0.02 S
LVEF	0.002	0.02-0.321	0.564
HGB	0.435	0.753-1.34	0.212
BUN	1.824	0.27- 2.982	0.243
BUN\EFr	-0.001	1.24-3.34	0.001 S
TIMI risk index	-0.921	0.432-4.912	0.004 S
Mehran score	0.002	0.523-2.45	0.007 S
Creatinine pre-PCI	-0.923	0.532-1.76	0.123
Creatinine post-PCI	-0.005	0.032-0.832	0.09
Contrast volume	0.004	1.12 -5.23	0.02 S
MVD	0.005	0.213-675	0.06
Killip class >2	-0.007	0.342-0.934	0.432
Furosemide usage	0.001	0.13-0.97	0.114 NS
LM vs affection	0.002	0.234-1.03	0.32 NS

Table (9): correlation between mehran score and other clinical data of the studied patients.

	Mehran score	
	r	P value
Age	0.09	0.901 NS
HR	0.201	0.721 NS
SBP	-0.101	0.631 NS
DBP	-0.233	0.714 NS
LVEF	-0.423	<0.001 HS
HGB	-0.524	<0.001 HS
BUN	0.439	<0.001 HS
Creatinine before PCI	0.498	<0.001 HS
Creatinine after PCI	0.366	<0.001 HS
Contrast volume	0.344	<0.001 HS
TIMI risk index	0.580	<0.001 HS
TIMI flow after PCI	0.287	<0.001 HS

Discussion

Contrast-induced nephropathy is a possible complication of coronary diagnostic and interventional procedures. Its development has been associated with increased in-hospital and long-term morbidity and mortality, prolonged hospitalization, and long-term renal impairment **(1).** CIN is an acute kidney injury that follows intravascular administration of

radio-opaque contrast media in susceptible individuals. It remains responsible for a third of all hospital-acquired acute kidney injury and affects between 1% and 2% of the general population. Acute kidney injury is defined as any of the following: increase in Serum Creatinine (SCr) by ≥0.3 mg/dl (≥ 26.5 μmol/l) within 48 hours; OR increase in SCr to ≥1.5 times baseline, which is known or presumed to have occurred within prior 7 days; OR Urine volume <0.5 ml/kg/h for 6 hours. TIMI risk index for ST-Elevation Myocardial Infarction (STEMI) is a simple risk score designed to be used at initial presentation to predict 30-day mortality in STEMI patients treated with fibrinolytics. The TRI is a continuous index derived from three readily available clinical variables and is calculated using the equation: (heart rate × [age/10]2/systolic blood pressure). But in our study used it was used for ACS patients with significant correlation.

The current study was done to evaluate blood urea nitrogen to left ventricular ejection ratio and TIMI risk index as predictors for occurrence of CIN in patients with acute coronary syndrome, who underwent PCI. It included 300 patients (204 male and 96 female), mean of age is 57.4 ± 11.5 years old and with a range from 27 to 80 years old and 68% of them are male, 34% smokers, 54% diabetics, 48% hypertensive, 11% had past history of HF and 22% had history of previous PCI, 25% with CAD. It also shows that 71% of studied group had STEMI type of ACS, 17% were non- STEMI and only 12% had unstable angina, shows that 64% of the studied patients presented with single vessel affection and 14% only presented with MVD, 84% of them with balloon or stenting, and shows that 71% of studied group had STEMI type of ACS, 17% were non- STEMI and only 12% had unstable angina. It also shows that all patients with CIN had STEMI type of ACS versus 68.1% of non-CIN, there was non-STEMI and unstable angina cases among non-CIN group (18.7% and 13.2% respectively) with no significant difference among both groups.

In our study, the CIN group (59.7 ± 16.01) was older than the non CIN group (56.7 ± 11.1). This may be explained by the gradual decline of renal function which is a recognized feature of the aging process despite presence of wide variability in the rate of decline among individuals **(2).** Our study is concordant with **Kirris T. et al., (3)** which evaluated 1140 consecutive patients with ACS who were treated with PCI between January 2008 and July 2015 who presented with the diagnosis of ACS and underwent PCI shows that advanced age has high significant correlation with development of CIN. These findings also agree with **Marenzi et al. (4)** who stated that the age of CIN group was (67y ± 12) while the age in non CIN group was (61y ± 11). Also, **Liu et al., (5),** stated that patients with CI-AKI were older and **Victor et al., (6)** showed significant increase in CI-AKI among patients with age >70 years.

Our study showed significant decrease in haemoglobin level in CIN group compared with non CIN group, this may be explained by that the decrease in the hemoglobin level was associated with decrease in renal tissue oxygenation with associated decrease in the kidney ability to function normally **(7).** This coincides with **Victor et al. (6)** who stated that the presence of anemia was associated with CIN and **Liu et al., (8)** who stated that CI-AKI group experienced a higher incidence of anemia.

It is also concordant with **Kaya A. et al., (9)** which included 963 patients who presented with the diagnosis of STEMI between September 2015 and January 2018 and underwent PCI and showed that there is significant correlation between low hemoglobin level and development of CIN. This is concordant with **Kirris T. et al., (3)** which evaluated 1140 consecutive patients with ACS who were treated with PCI between January 2008 and July 2015 who presented with the diagnosis of ACS and underwent PCI shows that anemia has high significant correlation with development of CIN. Our study showed that there was no

significant relation between smoking, HTN and CIN which was disconcordant with **Wykrzykowska et al., (10)**, who studied 1208 who represented to CCU with Acute Coronary Syndrome, that reported smoking and hypertension had significant relation with CIN with p value (<.001) for both and this may be due to the large number of patients included in the study. This is disconcordant with **Kirris T. et al., (3)** which shows that Hypertension and smoking has no significant correlation with development of CIN.

Our study showed that there was no significant relation between DM, Dyslipidemia and CIN which was concordant with **Wykrzykowska et al., (10)**, who studied 1208 who represented to CCU with Acute Coronary Syndrome that showed that there was no significant relation between ACEF score and the other risk factor DM, Dyslipidemia.

However, our study was disconcordant with **Kaya A. et al., (9)** in which there was high significant correlation between glucose level and development of CIN, Otherwise, there was no correlation between dyslipidemia and development of CIN. This is concordant with **Kirris T. et al., (3)** which showed that has no significant correlation between DM, Dyslipidemia and development of CIN. In our study, there is highly significant increase in incidence of HF, Prior TIA, Stroke or DVT in CIN group compared with non CIN group. This is concordant with **Kirris T. et al., (3)** which showed that there was significant correlation between history of HF, prior TIA, stroke or DVT and development of CIN. In our study, we found that there was significant decrease in EF in CIN group compared with non CIN group; this can be explained by the decrease in the tissue perfusion occurring in heart failure which is represented by ejection fraction with subsequent decrease in the renal GFR **(7).** This coincides with **Andò et al. (11)** that stated that patient with CIN had a more severe impairment of basal EF. Also, **Akkoyun et al. (12)** stated that CIN-positive patients had lower left ventricular ejection fraction. Similar results were obtained by **Kim et al. (13)** who stated that in multivariate logistic regression analyses, the development of CI-AKI was associated with left ventricular systolic dysfunction. **Gaskina et al. (14)** also stated that Patients with versus without CI-AKI were with lower LV EF (37±10% versus 41±14%, p<0.05) and **Marenzi et al., (4)** stated that Patients developing CIN had lower left ventricular ejection fraction. Our study showed that there was highly significant increase in creatinine level before PCI and after PCI in CIN group compare with non CIN group , the higher creatinine level before, the higher creatinine level after PCI and this was concordant with **Ando et al., (11)** , who studied 481 patients that referred to the Coronary Care Unit(CCU) of the University Hospital of Messina from January 2008 to June 2011 for primary PCI in the course of STEMI ,in being serum creatinine is independent predictors of AKI development after primary PCI for STEMI with p value (<0.001).

Also, this was concordant with **Dziewierz et al., (15)**, 1425 patient with ACS admitted to 29 community hospitals of Poland. Data were collected during two separate enrollment periods: from February 2005 to March 2005 and from December 2005 to January 2006, in being serum creatinine is independent predictors of AKI development after elective with p value (<0.001). It is also concordant with **Kaya A. et al., (9)** which included 963 patients who presented with the diagnosis of STEMI between September 2015 and January 2018 and underwent PCI and showed that there is significant correlation between creatinine level pre- and post-PCI and development of CIN. In our study, there was significant increase in amount of used contrast volume in CIN group compared with non CIN group. This may be explained by vasoconstriction resulting in renal medullary hypoxia and direct toxicity caused by the contrast media to renal tubular cells. This result coincides with the results of **Marenzi et al., (4)** who stated that CIN patients received a higher volume of the contrast agent during PCI than patients without CIN. **Gaskina et al., (14)** who stated that CIN patients received a higher volume (282±94 versus 236±85 ml, p<0.05), **Narula et al., (16)** who stated that

contrast volume is an independent predictors of CI-AKI and **Marenzi et al., (17)** who stated that Patients who received more than the maximum contrast dose (contrast ratio >1) had a more complicated in-hospital clinical course and higher mortality rate (13% versus 2.8%; P <0.001) than did patients with a contrast ratio less than 1.

It is also concordant with **Kaya A. et al., (9)** which included 963 patients who presented with the diagnosis of STEMI between September 2015 and January 2018 and underwent PCI and showed that there is significant correlation between amount of contrast and development of CIN. On the other hand, **Liu et al. (8)** stated that there was no significant difference between two groups in Contrast volume.

This was concordant with **Singhal et al., (18)**, who studied 300 consecutive coronary artery disease patient streated with coronary angioplasty in North India, that showed that the dose of the contrast was highly significant with p value (0.00) as the contrast induced Nephropathy was associated with contrast volume more than 250 ml.

In our study, the Mehran score was significant in predicting the occurrence of CIN. This coincides with **Kul et al. (19)** that stated that Patients with CI-AKI had higher Mehran score. It also correlates with **R. Mehran et al., (20)** which concluded that Mehran's score is one of the most common risk scores in prediction of the risk for CIN.

Our study showed that sensitivity of BUN\EFr level as a predictor of CIN was 88.9% with ability to exclude 94.5% of truly negative cases, while sensitivity of mehran score in prediction of positive cases was 100% and 90% exclusion of negative cases, both tools had high accuracy above 90% and a high statistically significant tool used in prediction of CIN.

This is concordant with **Kirris T. et al., (3)** study which evaluated 1140 consecutive patients with ACS who were treated with PCI between January 2008 and July 2015 who presented with the diagnosis of ACS and underwent PCI shows that BUN\EFr has high significant correlation with development of CIN. Our study showed that there was high significant relation between TIMI risk index and Killip score among CIN group. TIMI risk index is an easily applicable simple and useful score which is obtained by the formulation of age, systolic blood pressure, and heart rate without laboratory parameters.

Our study showed that there was high significant relation between TIMI risk index and Killip score among CIN group. TIMI risk index has asensitivity 100% and 74.7% exclusion of negative while his accuracy was 77%. This is concordant with **Kaya A. et al., (9)** which included 963 patients who presented with the diagnosis of STEMI between September 2015 and January 2018 and underwent PCI shows that Killip score and TIMI risk index high significant correlation with development of CIN. The is concordant with **Kirris T. et al., (3)** which showed that there was significant correlation between that Killip score and TIMI risk index and development of CIN.

Conclusion

CIN is a frequent complication following PCI and is associated with complicated hospital stay and high mortality rate. Patients who are older and have associated comorbidities such as anemia, acute heart failure, high contrast volume and ratio, high TIMI risk index, high Killip score, high Mehran score and renal insufficiency at the baseline are at High risk of developing CIN. Those patients can be identified, and more vigilant preventive measures can then be applied for the prophylaxis of CIN. BUN/EFr and TIMI risk index score can be used to predict acute kidney injury in patients with ACS undergoing PCI.

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