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Research Article

Hemoglobin Level and Left Ventricular Function as Predictors of Acute Kidney Injury in Patients with ST Elevation Myocardial Infarction Undergoing Primary Percutaneous Intervention

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Abstract

Background: Among patients with ST-segment elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (PCI), worsening of renal function resulting in acute kidney injury (AKI) is a frequent complication known to be associated with adverse outcomes. Also, anemia on admission is associated with an increased risk for (AKI). The relation between echocardiographic parameters of left ventricular (LV) function and the risk of AKI among patients with STEMI undergoing PCI is still controversial.

Objective: Detection of the relationship between hemoglobin level and LV systolic and diastolic function as a predictors for developing AKI after primary PCI in patients with STEMI.

Methods: A prospective observational cohort study conducted from June 2016 to June 2017, at cardiovascular medicine department, Tanta University Hospitals in Gharbia Governorate, Egypt. The study enrolled 38 consecutive adult patients of both genders who were diagnosed with definite STEMI within 12 hours from the time of symptoms onset and were treated by PCI who developed AKI after the procedure. The study population (38 patients) was then subdivided according to severity of renal impairment according to creatinine clearance (Cr cl) into two groups:

1. **Group I:** Those who developed severe renal impairment with Cr.cl ≤ 30 ml/min (17 patients).
2. **Group II:** Those who developed mild to moderate renal impairment with Cr.cl ≥ 30 ml/min (21 patients).

They were subjected to full clinical examination, laboratory investigation including serum creatinine (sCr) level that was determined on hospital admission, before primary PCI, and at least once a day during the cardiac intensive care unit stay,complete blood count with special attention to hemoglobin (Hb) level at hospital admission before primary PCI. All patients underwent a screening echocardiography within three days of admission, measurement of LV systolic function was performed using the commercially available machine (Vivid 7, GE Medical System, Horten, Norway) with a 3.5-MHz transducer. LV systolic function was assessed using M-mode in parasternal long axis view.

Results: Group I patients were older (70.65 ± 10.36 years vs 57.43 ± 12.09 years, p = 0.001), but there was no statistacillay significant difference between both groups for risk factors, smoking, dyslipidemia, diabetes, time to reperfusion, addiction, site of infarction or Killip's classification; but there was more prevelance of prior MI in group I than group II. There was a statistically significant difference between both groups as regard Hb level (10.65 ± 0.996 gm/dl vs 11.62 ± 1.396 gm/dl, p = 0.017), EF (40.18 ± 7.40 % vs 48.67 ± 8.05%, p = 0.002), Cr cl (26.35 ± 2.18 ml/min vs 39.10 ± 5.61 ml/min, p = 0.001), sCr on admission (1.36 ± 0.21 ml/dl vs 1.06 ± 0.20 ml/dl, p = 0.001), peak sCr (2.08 ± 0.32 ml/dl vs 1.79 ± 0.32 ml/dl, p = 0.010) and s.Cr level at discharge (1.55 ± 0.21 ml/dl vs 1.36 ± 0.18 ml/dl, p = 0.004).

Conclusion: Older ages, previous history of myocardial infarction, TIMI flow after PCI, anemia on admission, level of serum creatinine on admission, creatinine clearance and impaired systolic function of left ventricle were strongly statistically different with developing AKI and related to its severity, while no significant statistical difference could be found as regard gender, smoking, addiction, diabetes, dyslipidemia, Killip classification, time to reperfusion, or site of infarction with incidence of developing AKI.

Keywords: *Acute Kidney Injury; Percutaneous Coronary Intervention; Ejection Fraction Percentage; Hb Level*

Introduction

Worldwide, acute ST-segment myocardial infarction (STEMI) is one of the most important cardiovascular diseases that increase risk of morbidity and mortality [1].

The primary goal in management of acute STEMI is reperfusion therapy with intravenous fibrinolysis or primary percutaneous intervention (PCI) [2], which is the preferred strategy, if it is performed on time by experienced personnel. However; it still has some side effects [2].

Acute kidney injury (AKI) is a frequent complication among patients who undergo PCI shown to be associated with adverse outcomes [3-6]. Other important factors among this specific patient population includes an adverse hemodynamic state resulting in reduced renal perfusion, as well as other metabolic factors [7-9].

Some studies specifically demonstrated that low ejection fraction (EF) < 30% to 40% is an independent predictor of AKI in patients with CHF [10-12].

Anemia is a common finding known to have a decrimental effect on the outcome of STEMI [13,14]. Previous studies have shown that anemia increases the risk of contrast-induced nephropathy in patient undergoing primary percutaneous intervention. Admission hemoglobin (Hb) level was shown to be a predictor of in-hospital mortality among STEMI patients who underwent primary PCI [15].

It is well known that patients with an eGFR < 60 mL/min/1.73 m^2 are more likely to have anemia and that prevalence and severity of anemia increase with declining renal function [16].

Patients and Methods

The study was conducted at the Department of Cardiovascular Medicine, Tanta University hospital in Gharbia Governorate, Egypt, during the period between June 2016 to June 2017. It was carried out on 38 patients diagnosed definitively with STEMI and treated with primary PCI who developed contrast induced nephropathy after the procedure. An informed consent was taken from all participants.

Patients included in this study fulfilled the following criteria: Patients admitted to the cardiac intensive care unit with STEMI who underwent primary PCI within 12h from symptom onset or between 12 and 24h with evidence of continuing ischemia who developed contrast induced nephropathy post procedure.

Exclusion criteria were:

- Patients who were treated either conservatively or by thrombolysis.
- Patients whose final diagnosis on discharge was other than STEMI (e.g. myocarditis).
- Patients who died within 24 hours of admission because we presumed there was insufficient time for AKI to occur.
- Patients requiring chronic peritoneal dialysis or hemodialysis treatment.
- Patients without a pre procedure serum creatinine level.
- Patient with cardiogenic shock at time of presentation.

All included patients were subjected to detailed:

1. History taking: (Personal history - Risk factors: hypertension (HTN), smoking, addiction, diabetes mellitus (DM), chronic kidney disease - Past history of premature coronary artery disease - sPast drug history of nephrotoxic drugs - Family history for coronary artery disease).
2. Clinical examination: Vital signs: e.g. heart rate, blood pressure, Signs of heart failure/hemodynamic instability.
3. Local cardiac examination.
4. Twelve leads surface ECG: Routine 12-lead ECG was done for the patients to detect changes suggestive of STEMI.
5. Venous sampling for laboratory data including:

a. Biomarkers of myocardial injury (troponin and CKMB)

b. Complete blood count with special attention to Hb level at hospital admission before primary PCI. Anemia was defined as hemoglobin level < 12 g/dL in females and < 13 g/dL in males according to World Health Organization criteria [17].

c. Serum creatinine (sCr) level was determined on hospital admission, before primary PCI and at least once a daily during the cardiac intensive care unit stay and was available for all analyzed patients.

d. Creatinine clearance is calculated by the following formula: CreatClear = Sex * ((140 - Age)/(SerumCreat)) * (Weight/72) [18].

6. Primary percutaneous intervention for Infarct related artery (IRA).

All included patients were subjected to primary PCI for the infarct related artery (IRA) according to the ESC guidelines. Coronary angiography was performed under local anesthesia from the femoral approach according to the standard technique. Reperfusion success was assessed according to TIMI blood flow grade, and (IRA) was identified according to the culprit lesion on the basis of the infarct location on the admission ECG, and the angiographic findings (target vessel, lesion characteristics).The Choice of stents (bare-metal stent or drug-eluting stent) was left to the operator's discretion. The success of primary PCI was defined as achievement of the TIMI flow of the infarct related artery to grade III.

Echocardiography

Within three days of admission, measurement of left ventricular (LV) systolic function was performed using the commercially available machine (Vivid 7, GE Medical System, Horten, Norway) with a 3.5-MHz transducer. LV systolic function is assessed using Simpson's method in the apical 4 and apical 2 views and M-mode in parasternal long axis view by directing the M-mode cursor across the mid- LV.

Statistical presentation and analysis of the present study was conducted, using the mean, standard deviation (SD) and Chi-square test by SPSS Version 20. Numerical data was presented as mean and standard deviation (SD), and categorical data was presented as number and percentage. For comparison Chi-square test was used for variables, and independent student 't' test for numerical variables. The level of significance was adopted at $p < 0.05$.

Subjects were informed about the purpose and procedure of the study and benefits of sharing in it. Ethical considerations of the study were carried out according to that of Declaration of Helsinki.

Results

The present study was conducted in Cardiology Department, Faculty of Medicine Tanta University. It was carried out on 38 adult patients of both genders diagnosed definitively with acute kidney injury after having primary PCI as revascularization strategy for STEMI, within 12 hours from the time of symptoms onset. The study was conducted from June 2016 to June 2017.

The study population was subdivided into two groups:

- **Group I:** Those who developed severe renal impairment with Cr.cl ≤ 30 ml/min (17 patients).
- **Group II:** Those who developed mild to moderate renal impairment with Cr.cl ≥ 30 ml/min (21 patients).

In the study we examined the relationship between patient characteristics, factors in initial clinical evaluation of the patients with prediction of occurrence of acute kidney injury and its severity.

Age

In group I, the age ranged from 50 to 85 years with mean ± SD age 70.65 ± 10.36 years and in group II, it ranged from 38 to 82 years with mean ± SD age 57.43 ± 12.09 years. There was significant difference among studied groups regarding age ($P < 0.001$) (Table 1).

Gender

Among the study population 68.40 % of the patients were males and 31.60 % were females. There was no statistically significant difference among studied groups regarding gender (Table 1).

Characteristics	Group I (n = 17) n (%)	Group II (n = 21) n (%)	Total (n = 38)	Sig. Test	P
Age (in years)					
Mean ± SD	70.65 ± 10.36	57.43 ± 12.09	63.34 ± 13.031	T	
Range	50 - 85	38 - 82	38 - 85	3.568	0.001*
Gender					
Male	10 (58.8%)	16 (76.2%)	26 (68.4%)	χ^2	
Female	7 (41.2%)	5 (23.8%)	12 (31.6%)	1.311	0.252

***Table 1**: Socio-demographic characteristics of the studied patient groups.*

*t: t for independent t test; *: Statistically significant;χ^2: Chi square test.*

Clinical charactaristics and risk factors among both study groups

There was no statistically significant difference between both groups as regard diabetes, hypertension, dyslipidemia, obesity, smoking or addiction, but there was a statistically significant difference between both study groups as regard history of prior MI ($p < 0.05$) (Tables 2 and 3).

Characteristics	Group I (n = 17) n (%)	Group II (n = 21) n (%)	χ^2	P
Diabetes Mellitus	10 (58.8%)	10 (47.6%)	0.473	0.492
Hypertension	8 (47.1%)	10 (47.6%)	0.131	0.973
Dyslipidemia	10 (58.8%)	15 (71.4%)	0.663	0.415
Obesity	7 (41.2%)	12 (57.1%)	0.958	0.328

***Table 2:** Clinical characteristics of both study groups.*

**: Statistically significant;χ^2: Chi square test.*

Characteristics	Group I (n = 17) n (%)	Group II (n = 21) n (%)	χ^2	P
Smoking	9 (52.9%)	12 (57.1%)	0.67	0.796
Addiction	1 (5.9%)	5 (23.8%)	2.271	0.132
Prior MI	6 (35.3%)	2 (9.5%)	3.754	0.05*

***Table 3:** Smoking, addiction, and prior MI in both study groups.*

**: Statistically significant;χ^2: Chi square test.*

Site of infarction

In group I, 3 patients (17.7%) had anterior MI,10 patients (58.8%) had inferior MI and 4 (23.5%) patients had multiple infarction site. In group II, 5 patient (23.8%) had anterior MI, 13 patients (61.9%) had inferior MI and 3 patients (14.3%) had multiple infarction site. There was no statistically significant difference between the two groups as regard infarction site (P = 0.557) (Table 4).

Site of infarction	Group I (n = 17) n (%)	Group II (n = 21) n (%)	χ^2	p
Anterior	3 (17.7%)	5 (23.8%)	2.075	0.557
Inferior	10 (58.8%)	13 (61.9%)		
Multiple	4 (23.5%)	3 (14.3%)		
Anterior, posterior	2 (11.8%)	1 (4.7%)		
Anterior, Right	2 (11.8%)	1 (4.7%)		
Anterior, posterior, Right, lateral	0 (0%)	1 (4.7%)		

Table 4: *Site of infarction among both study group patients.*

**: Statistically significant;χ^2: Chi square test.*

Time to reperfusion and contrast volume used in both study groups

There was no significant difference as regard wether time to reperfusion (67.94 ± 26.04 min vs 57.14 ± 24.11 min) or contrast volume (288.24 ± 33.21 ml vs 285.71 ± 82.37 ml) in group I and II respectively (Table 5).

	Group I (n = 17)	Group II (n = 21)	T	P
Time to reperfusion (in minutes)	67.94 ± 26.04	57.14 ± 24.11	1.313	0.198
Contrast volume	288.24 ± 33.21	285.71 ± 82.37	0.128	0.899

Table 5: *Time to reperfusion and contrast volume in both study group patients.*

t: t for independent t test.

Number of vessels affected

There was no statistically significant difference between the two groups as regard the number of affected vessels (P = 0.073) (Table 6).

Characteristics	Group I (n = 17) n (%)	Group II (n = 21) n (%)	χ^2	P
One vessel	6 (35.2%)	12 (57.2%)	5.233	0.073
Two vessel	4 (23.5%)	7 (33.3%)		
Three vessel	7 (41.3%)	2 (9.5%)		

Table 6: *Number of affected vessels in both study group patients.*

**: Statistically significant;χ^2: Chi square test.*

The results of PCI, assessed by TIMI flow

There was statistically significant difference between the two groups as regard TIMI flow after PCI (P = 0.022) (Table 7).

Characteristics	Group I (n = 17) n (%)	Group II (n = 21) n (%)	χ^2	P
II	7 (41.2%)	2 (9.5%)	5.208	0.022*
III	10 (58.8%)	19 (90.5%)		

Table 7: *TIMI flow after PCI in both study group patients.*

**: Statistically significant; χ^2: Chi square test.*

Killip classification of the studied population

There was no statistically significant difference between the two groups as regard Killip Classification (P = 0.318) (Table 8).

Characteristics	Group I (n = 17) n (%)	Group II (n = 21) n (%)	χ^2	P
I	11 (64.7%)	18 (85.7%)	2.294	0.318
II	4 (23.5%)	2 (9.5%)		
III	2 (11.8%)	19 (90.5%)		

Table 8: *Killip classification for the studied population.*

**: Statistically significant;χ^2: Chi square test.*

Renal function

In group I, the mean Cr cl was 26.35 ± 2.18 ml/min. The mean creatinine levels were 1.36 ± 0.21 mg/dl on admission, 2.08 ± 0.32 mg/dl at peak and 1.55 ± 0.21 mg/dl at discharge. In group II, the mean Cr cl was 39.10 ± 5.61 ml/min. The mean creatinine levels were 1.06 ± 0.20 mg/dl on admission, 1.79 ± 0.32 mg/dl at peak, and 1.36 ± 0.18 mg/dl at discharge.There was statistically significant difference between the two groups as regard Cr cl level (P < 0.001), s.Cr level on admission (P < 0.001), peak s.Cr level (P.value = 0.010), and s.Cr level at discharge with (P < 0.004) (Table 9).

Characteristics	Group I (n = 17) Mean ± SD	Group II (n = 21) Mean ± SD	t	P
Creatinine clearance (ml/min)	26.35 ± 2.18	39.10 ± 5.61	-8.821	0.001*
Creatinine level (mg/dl)				
On admission	1.36 ± 0.21	1.06 ± 0.20	4.603	0.001*
Peak	2.08 ± 0.32	1.79 ± 0.32	2.726	0.010*
At discharge	1.55 ± 0.21	1.36 ± 0.18	3.142	0.004*

Table 9: *Renal function in both study group patients.*

*t: t for independent t test; *: Statistically significant.*

Creatinine level

The creatinine level among group I patients changed with a statistically significant difference between on admission, at peak, and at discharge (P = 0.001). Among group II patients, the creatinine level also changed in a statistically significant way (P = 0.001) (Table 10).

	Creatinine level (mg/dl)				
	On admission	Peak	At discharge	F	P
	Group I				
Mean ± SD	1.36 ± 0.21	2.08 ± 0.32	1.55 ± 0.21	37.337	0.001*
	Group II				
Mean ± SD	1.06 ± 0.20	1.79 ± 0.32	1.36 ± 0.18	45.643	0.001*

Table 10: *Follow up of the creatinine level among both study groups.*

*F: ANOVA (Analysis of Variance); *: Statistically significant.*

Hb level

In group I, the mean Hb level 10.65 ± 0.996 gm/dl while in group II, mean Hb level 11.62 ± 1.396 gm/dl. There was a statistically significant difference between the two groups (P = 0.017) (Table 11).

Admission Hb	Group I (n = 17)	Group II (n = 21)	t	P
Mean ± SD	10.65 ± 0.996	11.62 ± 1.396	-2.50	0.017*

Table 11: *Admission HB level in both study groups.*

*t: t for independent t test; *: Statistically significant.*

Ejection fraction percentage

In group I, the mean Ejection fraction percentage (EF %)was 40.18 ± 7.40% while in group II, mean Ejection fraction percentage was 48.67 ± 8.05%. There was a statistically significant difference between the two groups regarding the ejection fraction percentage (P 0.002) (Table 12).

Ejection fraction %	Group I (n = 17)	Group II (n = 21)	t	P
Mean ± SD	40.18 ± 7.40	48.67 ± 8.05	-3.381	0.002*

Table 12: *Ejection fraction % in both study groups.*

*t: t for independent t test; *: Statistically significant.*

Creatinine clearance in relation to HB level

In 32 patients with anemia the mean Cr cl level was 32.09 ± 5.279 ml/min while in 6 patients with normal HB level mean Cr cl level was 40.33 ± 7.511 ml/min. There was a statistically significant difference between the two groups (P = 0.009) (Table 13).

Creatinine clearance (ml/min)	Patients with Anemia (n = 32)	Patients with normal HB (n = 6)	t	P
Mean ± SD	32.09 ± 5.279	40.33 ± 7.511	-3.255	0.009*

Table 13: *Creatinine clearance in relation to HB level.*

*t: t for independent t test; *: Statistically significant.*

Creatinine clearance in relation to systolic function

In 11 patients with normal systolic function the mean Cr cl level was 38.00 ± 8.56 ml/min while in 27 patients with impaired systolic function mean Cr cl level was 31.52 ± 6.71 ml/min. There was a statistically significant difference between the two groups (P = 0.040) (Table 14).

Creatinine clearance (ml/min)	Normal Systolic Function Ej% ≥ 50% (n = 11)	Abnormal Systolic Function Ej < 50% (n = 27)	t	P
Mean ± SD	38.00 ± 8.56	31.52 ± 6.71	2.247	0.040*

Table 14: *Creatinine clearance in relation to systolic function.*

*t: t for independent t test; *: Statistically significant.*

Correlation between AKI parameters with admission Hb and Ejection fraction

There was a statistically significant correlation between Cr cl, Hb level and Ejection fraction. Also, there was a statistically significant correlation between creatinine level and Ejection fraction (Table 15) and (Figure 1-3).

Correlations	r.	P
	Creatinine clearance (ml/min)	
Hb level	0.412	0.01*
Ejection fraction %	0.513	0.001*
	Creatinine level (mg/ml)	
Hb level	-0.063	0.708
Ejection fraction %	-0.463	0.003*

Table 15: *Correlation between AKI parameters with admission Hb and Ejection fraction.*

r: Pearson correlation coefficient

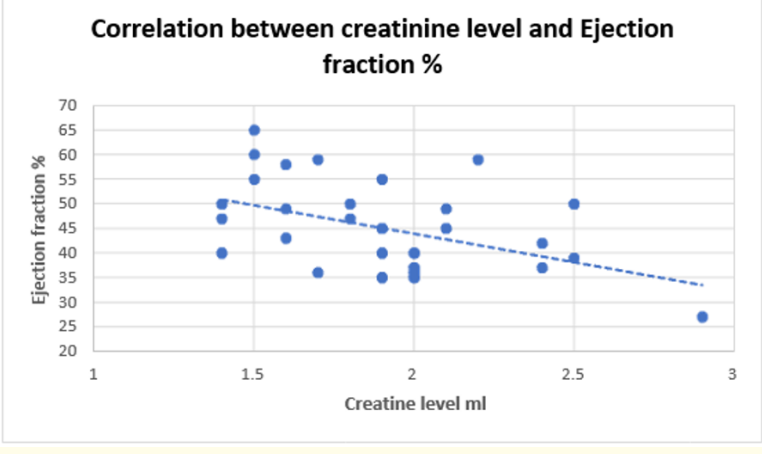

Figure 1: *Correlation between creatinine level and ejection fraction.*

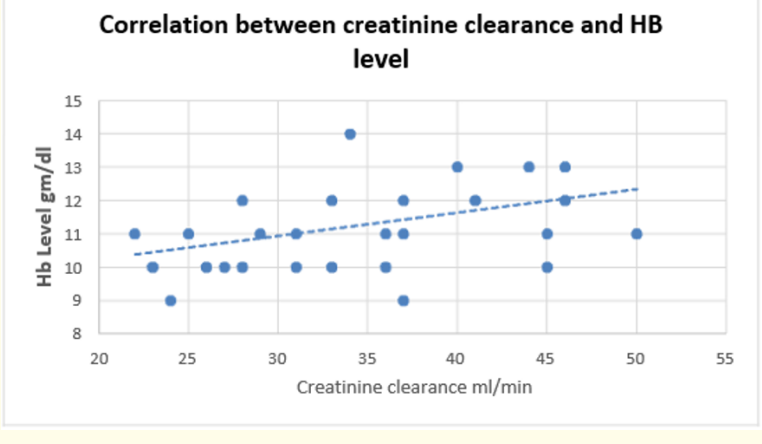

Figure 2: *Correlation between Cr cl anf Hb level.*

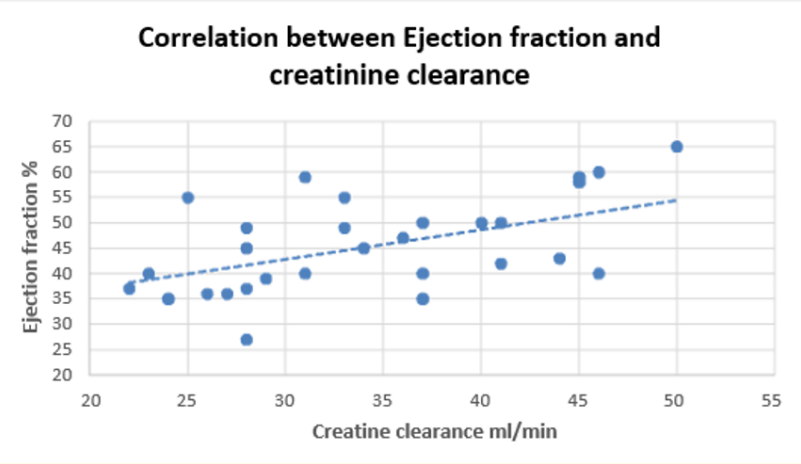

Figure 3: *Correlation between Ejection fraction and Cr cl.*

Table 16 reveals that significant Relation between EF %, Time to reperfusion and prediction of outcome (AKI) (Table 16).

	B	SE	Sig.	OR	95% CI	
					LL	UL
EF %	-0.265	0.132	0.045*	0.767	0.592	0.994
Hb level	-1.259	0.686	0.066	0.284	0.074	1.089
Age	0.102	0.058	0.080	1.107	0.988	1.241
Time to reperfusion	-0.054	0.038	0.150	0.947	0.880	1.020
TIMI flow after procedure	3.310	1.647	0.044*	27.374	1.085	90.426
Prior MI	-0.503	1.503	0.738	0.605	0.032	11.508

Table 16: *Multivariate analysis logistic regression for AKI.*

*B: Un standardized Coefficients SE: Standard Error; OR: Odds ratio; CI: Confidence interval; LL: Lower limit; L: Upper Limit; *: Statistically significant at p ≤ 0.05.*

The Receiver Operating Characteristics (ROC) analysis was performed to compare the performance and predictive accuracy of EF%, and Hb level for predicting AKI. The areas under the curve (AUC) for EF % and Hb level were 0.804, and 0.702 (P< 0.001, P = 0.035, respectively).

The EF % carried the highest sensitivity 81% in predicting AKI with cutoff value > 41, compared with admission Hb level that showed sensitivity of 76% with cutoff value > 10. The positive predictive value of EF % and admission Hb level with respect to AKI was 25% and 38 respectively and The negative predictive value of EF % and admission Hb level with respect to AKI was 22% and 36 respectively (Table 17) and (Figure 4).

	Cut off	AUC	P	Sensitivity	Specificity	PPV	NPV
EF%	41.0	0.804	0.001*	81.0	70.6	0.52	22.7
Admission HB	10.5	0.702	0.035*	67.2	47.1	38.5	36.0

Table 17: *Agreement (sensitivity, specificity and accuracy) for EF%, and HB level with severity of AKI.*

AUC: Area under the curve; PPV: Positive predictive value; NPV: Negative predictive value.

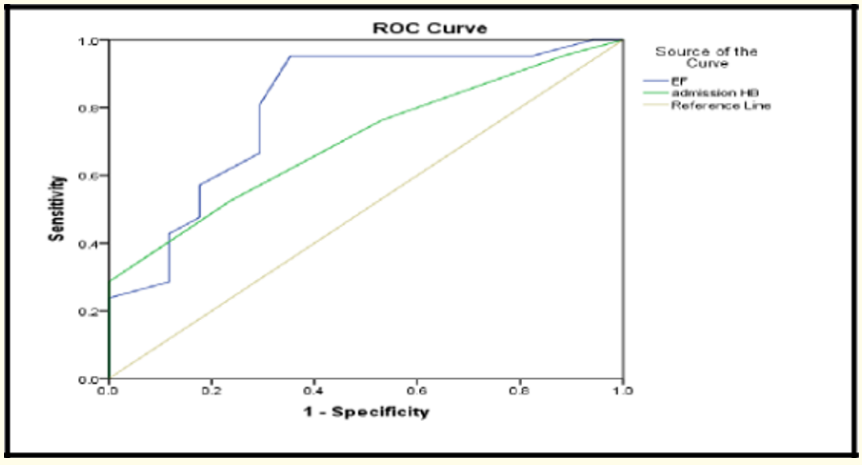

Figure 4: *ROC curve for sensitivity and specificity.*

Discussion

Acute kidney injury is a common and a serious complication of primary PCI after STEMI that affects both mortality and morbidity and affects outcome of the procedure and can be the cause of death and MACE rather than STEMI complications itself. Patients who develop CIN have a 5.5 fold increased risk of death when compared to the patients without CIN [19].

In 2014, the European Society of Cardiology published updated guidelines on CIN prevention which provides a framework for the use of evidence-based strategies for prevention [20].

In the aim of prevention of occurrence, contrast induced nephropathy after primary PCI risk factors and predictors should be properly identified and well understood so that preventive measures and precautions could be applied.

Many predictive risk scores were developed to estimate risk of CIN as Mehran risk score developed by Mehran R., *et al.* [21] in 2004. Based on the attained score, patients were further divided into low, moderate, high, very high risk groups, and the incidence of CIN, risk of dialysis and mortality are calculated for each group, also the ACEF score (Age, Creatnine clearance and Ejection Fraction) is proved as useful score for CIN prediction by Giuseppe Andò., *et al* [22].

Novel risk prediction algorithm using computational tool proposed by Gurm., *et al.* [23] may prove useful for both bedside clinical decision making and risk assessment.

In the present study we aim to correlate baseline patient characteristics and initial clinical evaluation at presentation with prevelance and severity of AKI.

As regard age: In group I a higher mean age (70.65 ± 10.36) was observed while in group II younger ages were more prevalent with significant correlation of age with prevalence and severity of AKI.

Similarly in 2015 Taku Inohara., *et al.* [24] in their study that aimed at the development and validation of a pre-PCI risk model for CIN prediction and included 358 patients who developed CIN in a COHORT study showed that those patients tend to be older with mean age 72.1 ± 12.1 [24].

With concordance with the present study also a prospective observational study carried out by Eleni Palli., *et al.* [25] suggested that critically ill patients aged 65 or more years old are more prone to present with renal injury after the intravenous infusion of radiocontrast media compared to patients aged less than 65 years old even with normal baseline renal function [25].

As regard sex, the present study couldn't find any significant difference in gender either in prevalence or severity of AKI.

In controversy with our study, the study by Taku Inohara., *et al.* [24] also demonstrated CIN is commoner among female gender in their study [24].

There is a lack of data regarding CIN and gender association. Despite discordant and inconsistent data, a large study suggested female sex as an independent risk factor for CIN. Various characteristics related to female sex such as advanced age at the time of presenting with STEMI, comorbidities and reduced body surface might increase the risk of CIN [26].

In the present study smoking and addiction were not found to have any statistical significant relation with the risk of developing AKI.

Similarly Mehran R., *et al.* [21] in their study conducted on 891 patients to validate Mehran risk score for prediction of CIN demonstrated that smoking and ex-smoking was not correlated to the risk of developing renal impairment after primary PCI.

As regard Diabetes mellitus, the present study showed that there is no correlation between DM and AKI with no difference among the group with severe renal impairment compared to the group who developed mild to moderate impairment.

On the contrary, in a study carried on Italian patients by Salvatore Evola., *et al.* [27] to assess risk factors of CIN 42% of 105 patients were found diabetics with P.value = 0.03 in comparison with those who did not develop AKI.

As regard hypertension, the present study showed that there is no correlation between hypertension and AKI with no difference among the group with severe renal impairment compared to the group who developed mild to moderate impairment. There was no statistical significant relation with the risk of developing AKI with (P. value = 0.973).

On the contrary, Salvatore Evola., *et al.* [27] in their study also demonstrated arterial hypertension as strong independent risk predictor for CIN with 80% prevalence among their CIN group and $P < 0.05$. This difference may be due to few number of patients in the present study.

As regard dyslipidemia, the present study cannot find any significant correlation between dyslipidemia and incidence or severity of AKI.

Similarly in the previously mentioned study conducted by Alberto Bouzas-Mosquera., *et al.* [28] hypercholesterolemia was found to have no significant correlation with contrast induced nephropathy in their affected population 33% had hypercholesterolemia.

As regard previous history of prior MI, the present study correlated presence of prior MI as a predictor for AKI occurrence with more prevalence among the group with severe renal impairment with ($P < 0.05$) compared to the group who developed mild to moderate impairment.

In the present study, contrast volume was not found to have any statistical significant relation with the risk of developing AKI, in group I the volume was 288.24 ± 33.21 ml and in group II it was 285.71 ± 82.37 ml with (P = 0.899).

Similarly, data on contrast volume used were available in only 418 (38 had AKI) patients, however, its amount during PCI did not differ between patients with or without AKI (134 - 49 ml vs 147 - 47 ml, respectively; P ¼ 0.136), or in multivariate models [29].

As regard TIMI flow after PCI, the present study showed that less TIMI flow is more prevalent among the group with severe renal impairment with (P = 0.022) compared to the group who developed mild to moderate impairment.

As regard change in creatinine level, the study showed that higher creatinine level in the group with severe renal impairment with mean ± SD 1.36 ± 0.21 on admission, 2.08 ± 0.32 peak and 1.55 ± 0.21 SD at discharge with (P = 0.001) compared to the other group who developed mild to moderate impairement mean ± SD level was 1.06 ± 0.20 on admission, 1.79 ± 0.32 peak and 1.36 ± 0.18 gm/dl at discharge with (P = 0.001).

As regard hemoglobin level, in this present study, it was significantly correlated to incidence of AKI where anemic patients had more risk of developing renal impairment and it tends to be more severe (P = 0.017).

In concordance with the present study, another retrospective, single-centre observational study at the Tel-Aviv Sourasky Medical Center, a tertiary referral hospital with a 24/7 PPCI service by Shacham., *et al.* [29] found that patients with AKI had significantly lower admission hemoglobin levels (13.6 - 1.7 g/dL vs 14.4 - 1.5 g/dL; P < 0.001) and were more likely to be anemic (27% vs 12%; P < 0.001).

The prevalence of AKI in anemic patients was two fold higher than in non anemic patients (37% vs 19%; P ¼ 0.001), yet there was a substantially lower AKI prevalence compared with patients with reduced baseline kidney function.

In a multivariate logistic regression model, a lower admission hemoglobin level (odds ratio [OR], 0.86; 95% confidence interval [CI], 0.74 - 0.98; P ¼ 0.04) and anemia at admission (OR, 1.76; 95% CI, 1.02 - 3.02; P ¼ 0.04) emerged as independent predictors of AKI [29].

It is reported that contrast media could increase oxygen affinity of hemoglobin, so oxygen delivery to the peripheral tissues might be impaired. Local renal hypoxia can be more aggravated in patients with low hemoglobin after exposure to contrast media; hence, the combination of contrast-induced vasoconstriction and anemia may decrease oxygen delivery sufficiently to cause renal medullary hypoxia. Thus, it is intuitive that anemia may play a role in CIN risk [30].

Regarding left ventricular function, the present study showed that 48% of the study population had LV ejection fraction less than 50% with more prevalence of these myopathic patients among the group I members who developed severe renal impairment LV EF < 50%, with a mean ± SD EF of 40.18 ± 7.40.

In a similar study performed by Yacov Shacham., *et al.* [31] to assess the association of left ventricular function and acute kidney injury among STIMI Patients treated by primary percutaneous intervention, a retrospective, single center observational study at the Tel-Aviv Sourasky Medical Center, a tertiary referral hospital with a 24/7 primary PCI service, 34 patients admitted from June 2011 to December 2013 to the cardiac intensive care unit with the diagnosis of acute STEMI, underwent primary PCI and developed CIN were enrolled [31]. The patients were found to be older, to have more co-morbidities, longer time to reperfusion. In this cohort of patients with STEMI who underwent PCI, the occurrence of AKI after primary PCI was associated with worse LV systolic and diastolic function; however, only LVEF emerged as an independent predictor of AKI. For every 1% reduction in EF, the risk of AKI increased (odds ratio 1.1, 95% confidence interval 0.86 to 0.96, p. value 0.001 [31].

The sudden myocardial insult in STEMI results in an acute reduction of the LV pumping function, and this leads to reduced effective renal blood flow, consequently causing hypoxic change and the synthesis of reactive oxygen species. In addition the increased sympathetic tone, renin angiotensin-aldosterone system activation, the overproduction of many humoral factors such as vasopressin, catecholamines, endothelin, proinflammatory cytokines, and decreased nitric oxide levels can cause vascular endothelial cell damage, further aggravating blood flow disturbances and making the kidney more susceptible to CIN [31].

Conclusion

AKI could be a serious outcome complicating primary PCI for STEMI patients affecting both morbidity and mortality and the incidence of AKI is greatly affected by many risk factors. Pre-procedural risk factors and clinical status are the most important and common predictors of developing renal impairment post contrast medium exposure.

In this study our patients are evaluated pre-procedurally and risk factor are well assessed then s.Cr and Cr cl are observed post-procedure for identifying patients with AKI as defined before. Findings in clinical evaluations and patient's characteristics and baseline risk factor are then correlated to the risk of developing AKI and its severity.

Older ages, previous history of myocardial infarction, TIMI flow after PCI, anemia on admission, level of serum creatinine on admission, creatinine clearance and impaired systolic function of left ventricle were strongly statistically different with developing AKI and to its severity. While no significant statistical difference could be found as regard gender, smoking, addiction, diabetes, dyslipidemia, Killip classification, time to reperfusion, or site of infarction with incidence of developing AKI.

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