Assessment of Risk Factor for Developing Acute Kidney Injury in the Setting of Acute ST Elevation Myocardial Infarction

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Abstract

Background: An acute kidney injury (AKI) is a recognized complication in patients with ST elevation myocardial infarction (STEMI) by several mechanisms and it adversely affects morbidity and mortality on long-term bases [1, 2, and 3]. Few studies have investigated which patients with STEMI are at risk of developing AKI [6], and this study is aiming to do this assessment.

Patients and Methods: We enrolled patients with STEMI who were admitted to the CCU. KDIGO definitions was utilized to search for development of AKI in these patients throughout their stay in the CCU (their baseline serum creatinine was measured and traced daily thereafter; and their urine output was monitored). Common epidemiological and some relevant medical parameters were recorded for all patients. **Results and conclusion:** low baseline systolic blood pressure, ejection fraction % and estimated glomerular filtration rate (eGFR); in addition to use of diuretics; are the major risk factors for the development of AKI in the setting of STEMI.

Keywords: *acute kidney injury, ST elevation myocardial infarction*

Introduction

Deterioration in renal function is well recognized in patients struck by STEMI; and it is important to know what parameters or risk factors would make patients more vulnerable to such complication. These are investigated by some researchers who found that severity and hemodynamic impairment due to STEMI is the most important predictor [1]. Acute kidney injury- previously called "acute renal failure "– affects many patients with acute myocardial infarction (AMI) especially with cardiogenic shock and is associated with high morbidity and mortality [2,3]. AKI that complicate AMI is supposed to be mediated by hemodynamic changes that activate renin-angiotensin-aldosterone system, sympathetic nervous system and anti-diuretic hormone release [2]. So, patients taking ACE inhibitors/ARBs are most susceptible to hemodynamically mediated AKI as blocking angiotensin action with ACE inhibitors or ARBs decreases efferent arteriolar tone and decreases glomerular capillary perfusion pressure specially when blood volume is reduced for any reason as in case of intravascular volume depletion caused by diuretics use which may result in prerenal azotemia. Prolonged renal hypoperfusion may lead to acute tubular necrosis (ATN) [5]. Use of contrast media and other nephrotoxic agents play a role in development of AKI [5]. It was difficult to study the outcomes of these conditions because the diagnosis of acute kidney injury has not been standardized. Rodrigues et al reported finding more than 30 definitions of "acute renal failure" in the medical literature; this variety causes confusion and made it difficult to compare the results of multiple literatures that were published by different authors [7-11]. New definitions have been set to standardize the diagnosis of acute renal failure. These established the concept of acute kidney **injury** by emphasizing the subtle but important differences between the terms "acute kidney injury" and "acute renal failure". One important distinction between these is that injury precedes failure.

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Several studies have shown that small increases in serum creatinine levels during hospitalization are associated with a worse prognosis for the patients. In 2012, the Kidney Disease Improving Global Outcomes **(KDIGO)** Clinical Practice Guidelines for AKI were designed to compile information systematically on this topic by experts in the field. In short, AKI is defined as increase in serum creatinine by ≥0.3 mg/dl within 48 hours **OR** increase in serum creatinine to ≥1.5-fold the baseline, which is known or presumed to have occurred within the prior 7 days **OR** Urine volume <0.5 ml / kg/h for 6 hours [12-17]. Few studies have investigated the risk factors for developing AKI in early periods after AMI; and these revealed that AKI development (as evidenced by increase in serum creatinine) is associated with high morbidity and mortality [18-20]. The effect of pre-existing impaired estimated glomerular filtration on occurrence of AKI and mortality with AMI in such patients remains controversial [21, 22].

Myocardial infarction: In industrialized countries; acute myocardial infarction (AMI) is one of the most common diagnoses in hospitalized patients. AMI related deaths occur before reaching the hospital exceeds 50% but in-hospital mortality has declined from 10% to about 6% over the past decade with 15% 1-year mortality specially in elderly group [23]. ST-segment elevation myocardial infarction (STEMI) is a clinical syndrome defined by characteristic symptoms of myocardial ischemia in association with persistent electrocardiographic (ECG) ST elevation and subsequent release of biomarkers of myocardial necrosis [24].

Cardiorenal Syndrome (CRS): Although many definitions has been proposed ; the term (cardiorenal syndrome) can simply be thought to reflect the interplay between abnormalities of heart and kidney function, with deteriorating function of one organ while therapy is administered to preserve the other; so this term is being recognized increasingly as a complication of acute decompensation of heart failure (ADHF). About 30% of patients hospitalized with ADHF have abnormal baseline renal function, with associated increase in morbidity and mortality [28-30].

Patients & Methods

This is a cross sectional study, done at al-Sadr teaching hospital between April 1, 2015 and December 31, 2015.

Inclusion criteria: Patients enrolled in the study were those who were admitted to the coronary care unit with the diagnosis of acute ST elevation myocardial infarction, with no exclusion criteria.

Exclusion criteria:

1. Patients who were referred to the cardiac center for doing primary percutaneous intervention (PCI).

2. Presence of obstructive uropathy (depending on baseline clinical presentation and investigations).

3. Patients on maintenance dialysis.

4. Patients who died on arrival before doing the required management.

Data collection: Acute STEMI was diagnosed according to the Third universal definition of myocardial infarction 2012 depending on symptoms, ECG abnormalities and cardiac enzymes. [38].

The following data were collected for each patient:

1. Age ,sex, history of H.T ,history of D.M, history of coronary artery disease, history of dyslipideamia, smoking and drug history.

2. Serum createnine level was measured for every patient on admission and daily thereafter during the stay period in the coronary care unit, with continuous monitoring of urine output. Estimated GFR (eGFR) was calculated using the CKD-EPI CREATININE 2009 EQUATION.

3. Systolic and diastolic blood pressures monitored using mercurial sphygmomanometer.

4. Troponin I was assessed by rapid test cassette (Halogen Company).

5. Ejection fraction (EF) was calculated by 2D echocardiograghy.

6. Abdominal ultrasound was done to assess renal status and to exclude urinary tract obstruction.

AKI was defined according to KDIGO – AKI Work Group guidelines [39] .

Statistical analysis: Statistical package for the social sciences version 21.0 was used for analysis of data (SPSS inc.2013).all variables were examined for normally distribution and descriptive statistics presented as frequency and proportion for categorical variables and as mean ± standard deviation for continuous variables. A student's t-test and analysis of variances tests were used to compare means. Chi square test was used to compare frequencies and proportions. **Level of significant (P. value (was set at ≤ 0.05 to be considered as significant difference or correlation.**

Results

A total of 55 patients were included and forty of them were males (72.7%) as illustrated in **table-1** which also revealed prevalence of important comorbidities in the study group and different therapeutic interventions used. In addition to baseline serum creatinine (and hence eGFR); some other parameters were also recorded initially on admission, and outlined in **table-2**. The patients enrolled were monitored for development of acute kidney injury; where only **seven out of fifty five had developed AKI**. **Table-3** reveals the correlation between the site of AMI and the development of AKI; and there were no significant relation. **Table-4** reveals association between AKI occurrence and all variables recorded for the study group, where patients who developed AKI were more likely to have lower mean systolic blood pressure, ejection fraction and eGFR. Moreover; diuretic use revealed the same significant association.

Discussion: Most of the study group lies between the age of 51-70 yrs. and the majority were males which is well expected as male are known risk group for coronary artery disease [(23)]. The main prevalent risk factors in the history of the studied group (in order of frequency) are hypertension, diabetes mellitus, dyslipidemias and smoking (table-1). All patients in the study were prescribed Aspirin, clopidogrel and an anticoagulant; while 40 of them were candidate to undergo thrombolysis according to guidelines which reflect good access to the facility in the recommended time limit for use of thrombolytics. Because of its favorable role in improving outcome of patients with STEMI; ACEi were prescribed for the majority (72.5%) of studied group, while less than 20% were in need for adding diuretics, which possibly reflects good outcome due to successful reperfusion achieved by thrombolytics in about 80% of patients. Although about 70% of patients had history of hypertension before the heart attack; we noticed that mean blood pressure was only 130.7 mmHg (+/- 23.7) which may be related to myocardial depression secondary to acute MI as evidenced by reduced mean ejection fraction of 48.5%. All patients were well above eGFR of 60 ml/min/1.73m^2 and hence accepted renal function and will provide good media for searching for future development of acute kidney injury. We found that patients with inferior MI comprise the largest portion when compared to other sites of MI, followed by those with anterior MI (19 patients). During their in-hospital stay we monitored the patients for development of acute kidney injury; and found that seven patients developed features of acute kidney injury. All of those patients were treated conservatively except one who required dialysis. Similarly, Ying Liao et al, had found that incidence of AKI in AMI patients were 12.1 %[(15)].

Table 1. Illustration of demographic features, risk factors and therapeutic interventions of the studied group (N=55)

Variable		No.	%
Age (year)	≤ 50	15	27.3
	51 - 60	21	38.2
	61 - 70	14	25.5
	> 70	5	9.1

Cont... **Table 1. Illustration of demographic features, risk factors and therapeutic interventions of the studied group (N=55)**

Sex	Male	40	72.7
	Female	15	27.3
Smoking	Smoker	19	34.5
	Non-smoker	36	65.5
History of Hypertension		29	69.0
History of Diabetes Mellitus		25	59.5
History of Coronary Artery Disease		14	33.3
History of Dyslipidemias		22	52.4
Use of Aspirin		55	100.0
Use of Clopidogrel		55	100.0
Use of Anticoagulant		55	100.0
Use of Thrombolytic		40	78.4
Use of ACEi*		37	72.5
Use of Diuretic		10	19.6
Need for RRT**		1	1.8

*angiotensin converting enzyme inhibitor
**renal replacement therapy

Table 2. Illustration of clinical parameters and investigations recorded for the studied group (N=55)

Parameter	**Mean**	**SD**
Systolic blood pressure	130.7	23.7
Heart rate	83.9	10.1
Ejection Fraction %	48.5	10.2
White Blood Cell count	10.2	2.9
Hemoglobin	12.9	1.4
Baseline serum creatinine	0.78	.24
eGFR*	92.1	25.5

*estimated glomerular filtration rate

Table 3. Correlation between site of myocardial infarction and development of acute kidney injury (N=55)

	Acute kidney injury						
	Yes		**No**		**Total**		
Site of MI	Count	Row N %	Count	Row N %	Count	Row N %	
Inferior	3	13.0	20	87.0	23	41.82	P value = 0.857
Anterior	2	10.5	17	89.5	19	34.55	
Antero-septal	2	22.2	7	77.8	9	16.36	
Antero-lateral	0	0.0	2	100.0	2	3.64	
Lateral	0	0.0	2	100.0	2	3.64	
Total	7	12.7	48	87.3	55	100.00	

Table 4. Correlation matrix for the correlation between incidence of AKI and patients' variables (N = 55)

Variable	**R****	**P. value**
Age	-0.122	0.375
Sex	0.011	0.936
Smoking	0.067	0.628
Hypertension	-0.185	0.177
Diabetes Mellitus	-0.020	0.885
Coronary Artery Disease	0.153	0.266
Dyslipidemias	0.022	0.872
Systolic Blood Pressure	-0.337	0.012*
Heart rate	-0.091	0.507
Ejection Fraction %	-0.316	0.019*
eGFR	-0.456	<0.001*
Baseline Creatinine	0.160	0.244
Hemoglobin	0.048	0.729
White Blood Cell count	-0.025	0.858
Angiotensin Converting Enzyme inhibitors	-0.082	0.550
Diuretic	0.386	0.004*
Thrombolytic	-0.011	0.936
Hospital stay	-0.023	0.870
Death	0.217	0.111
* Significant at P<0.05, ** R: correlation coefficient		

Conclusions

1. Low baseline systolic blood pressure, ejection fraction % and eGFR are the major risk factors for the development of acute kidney injury in the setting of ST elevation myocardial infarction

2. Use of diuretics in the setting of ST elevation myocardial infarction is associated with high risk of development of acute kidney injury.

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Conflict of Interest: None to declare.

Ethical Clearance: In our research, all protocols were approved under the Al-hakeem General Hospital, Najaf, Iraq and all methods were carried out in accordance with approved guidelines.

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