



ORIGINAL RESEARCH PAPER

Cardiology

ACUTE KIDNEY INJURY IN ST ELEVATION MYOCARDIAL INFARCTION – INCIDENCE, COMORBID CONDITIONS AND ITS SIGNIFICANCE

KEY WORDS: AKI, STEMI, Inhospital Mortality, Thrombolysis

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ABSTRACT

BACKGROUND – The identification and prevention of acute kidney injury (AKI) is very important in a setting of STEMI.

OBJECTIVE – To find out the incidence, inhospital mortality and associated comorbid conditions in AKI complicating STEMI.

RESULTS – Out of the total 150 patients, 107 patients did not develop AKI and 43 patients developed AKI. Of the 43 patients (28.6%) who developed AKI, 29 patients (19.3%) had Stage I, 10 patients (6.7%) had stage II, and 4 patients (2.7%) had stage III AKI. The comparison between mortality and AKI shows highly significant p value =0.001 which means that the presence of AKI has higher mortality rate (30.2%) than the non AKI patients (9.3%). The percentage of patients who underwent thrombolysis with Non-AKI group was 59% and that of AKI group was 86.0% showing a high statistical significance with p value=0.002.

CONCLUSION – Development of AKI in STEMI significantly increased inhospital mortality.

INTRODUCTION

AKI is an important and common complication occurring after acute myocardial infarction (AMI), the development of which is associated with unfavourable outcomes and higher mortality after an AMI. The latest definition of Acute Kidney Injury (AKI), as per Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Work Group, 2012^[1] is : AKI is defined as any of the following (Not Graded): Increase in Serum creatinine (SCr) by 0.3 mg/dl ($\times 26.5 \mu\text{mol/l}$) within 48 hours; or increase in SCr to $\times 1.5$ times 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. The mechanisms causing AKI in the first few days after an AMI are multifactorial, including systemic and renal hemodynamic changes secondary to an impaired cardiac output and an imbalance of vasodilators and vasoconstrictors, the use of contrast media, and immunological and inflammatory kidney damage resulting from crosstalk between the heart and the kidney^[2].

Therefore, AKI, if not identified & intervened at the earliest, may become partly irreversible or even progress to chronic kidney disease, which has a profound impact on cardiovascular outcomes in patients with acute coronary syndromes as well as in the general population.^[3] Worsening of renal function during admission for myocardial infarction is a powerful and independent predictor of in-hospital and 1-year mortality^[4]. The purpose of this study is to evaluate the incidence of AKI, as defined by the KDIGO, in the acute phase of a myocardial infarction, thereby making vigorous efforts to preserve renal function as we attempt to salvage and protect cardiac muscle. The study also included evaluating the comorbidities associated with development of AKI along with 5-day in-hospital mortality in MI patients.

MATERIALS & METHODS

This cohort study was done on 150 patients at Institute of Non-Communicable Diseases, Government Royapettah Hospital, Kilpauk Medical College, Chennai. Individuals who got admitted with Acute Myocardial Infarction in the Intensive Coronary Care Unit of Government Royapettah Hospital were included in the study. The period of study is 6 months. Patients admitted with Acute Myocardial Infarction in Intensive Coronary Care Unit at Govt. Royapettah hospital were included in this study. Patients less than 18 years and those with pre-existing Chronic Kidney disease were excluded.

According to the classic World Health Organization criteria, diagnosis of acute myocardial infarction requires two of the following three criteria: A history suggestive of coronary ischemia for a prolonged period of time (>30 minutes), Positive cardiac biomarkers (Troponin I, Creatine Kinase –MB, Myoglobin) or Electrocardiographic evidence of acute myocardial infarction.

TABLE 1 -Staging of Acute Kidney Injury (AKI) (according to KDIGO guidelines)

Stage	Serum Creatinine	Urine Output
1	1.5-1.9 times baseline (or) $\geq 0.3 \text{ mg/dl}$ ($\geq 26.5 \mu\text{mol/l}$) increase	$<0.5 \text{ ml/kg/h}$ for 6–12 hours
2	2.0-2.9 times baseline	$<0.5 \text{ ml/kg/h}$ for ≥ 12 hours
3	3.0 times baseline (or) Increase in serum creatinine to $\geq 4.0 \text{ mg/dl}$ ($\geq 353.6 \mu\text{mol/l}$) (or) Initiation of renal replacement therapy	$<0.3 \text{ ml/kg/h}$ for ≥ 24 hours (or) Anuria for ≥ 12 hours

Blood glucose levels, serum triglycerides and hemoglobin levels were estimated as well. These patients were followed up during a period of next 48 hours and serum creatinine estimation was done to detect AKI (urine output is not included as a criterion in our study) & the factors associated with the development of AKI were studied, while the treatment of Acute myocardial infarction were not compromised during the study.

Subsequently, the percentage of in-hospital 5-day mortality of these patients was estimated. The collected data were analysed with IBM.SPSS statistics software 23.0 Version

RESULTS

TABLE 2: Baseline Characteristics of the study population (n=150)

Parameters	Mean \pm S.D.	Minimum	Maximum
Age (years)	60 \pm 9	36	85
Systolic Blood Pressure (mm Hg)	134 \pm 32	60	220
Diastolic Blood Pressure (mm Hg)	84 \pm 10	50	120
Blood Sugar on admission (mg/dL)	164 \pm 59	78	347
Serum Creatinine-on admission (mg/dL)	0.9 \pm 0.3	0.6	2.4
Serum Creatinine –after 48 hours (mg/dL)	1.1 \pm 0.5	0.6	4.1
Serum Cholesterol (mg/dL)	184 \pm 42	81	320
Serum Triglycerides	131 \pm 61	54	500

(mg/dL)			
Chest pain duration (in hours)	6±8	0	72
Hemoglobin (gm/dL)	12±1.5	8	15

S.D., Standard Deviation

Table 2 represents all the baseline characteristics of the entire study population. Table 3 represents, out of the total 150 patients, 107 patients did not develop Acute Kidney Injury (Non-AKI group) and 43 patients developed Acute Kidney Injury (AKI group). Of the 43 patients (28.6%) who developed AKI, 29 patient (19.3%) had Stage I AKI, 10 patients (6.7%) had stage II AKI, and 4 patients (2.7%) had stage III AKI. However in Fox C S et al study, the percentage of AKI in patients with acute myocardial infarction was only 16% [5]. The total number of deaths during the hospital stay among 150 patients were 23 (15.3%).

TABLE 3: Distribution of Non-AKI and various stages of AKI among the total study population (n=150)

Parameters	Non-AKI	AKI		
		Stage I	Stage II	Stage III
N	107	29	10	4
Percentage(%)	71.3	19.3	6.7	2.7

AKI, Acute Kidney Injury; Non-AKI, Non-Acute Kidney Injury

From Table 3, the mean±S.D. age of 43 patients who presented with AKI was 63±10 years, which was higher compared to the mean age of patients in Non-AKI group and has p value= 0.027, significant at 0.01 < P ≤ .05. The mean±S.D systolic and diastolic BP of these AKI patients were 130±42 mm Hg and 84±12 mm Hg. The mean random blood sugar of the AKI patients was 154±52 mg/dL, which was however less than that of non-AKI patients.

Also, the mean±S.D of serum creatinine value at the time of admission of AKI patients was 0.9±0.3 mg/dL, which was same as that of non-AKI patients. And the mean±S.D. of serum creatinine value after 48 hours in AKI patients was 1.6±0.5 mg/dL, which was

higher than that of non-AKI group, which was 0.9±0.2 mg/dL (Table 4).

Among AKI patients, the mean±S.D values of blood sugar at the time of admission was 154±52 mg/dL serum cholesterol was 192±51 gm/dL, and that of serum triglycerides was 122±28 gm/dL and the mean hemoglobin value of patients who developed AKI was 11.8±1.7 gm/dL. The mean duration of chest pain in patients who developed AKI was 10 hours, which was higher than that of non-AKI patients, and with a statistically significant p=0.009, which meant longer the duration of chest pain, more is the tendency to develop AKI in MI patients.

The percentage of female with Non-AKI was 48.6% and with AKI was 41.9%. But the comparison between sex and AKI shows no statistical significance (p=0.454) which reveals there is no gender predilection for the development of AKI in MI patients. The percentage of patients with history of hypertension with Non-AKI was 54.2% and percentage of patients with history of hypertension with AKI was 51.2%. By Chi-square test, the comparison between history of hypertension and AKI shows no statistical significance (p=0.736), which reveals the presence of history of hypertension is not associated with development of AKI in MI patients (Table 4).

The percentage of patients with history of diabetes with Non-AKI was 36.4% and the percentage of history of diabetes with AKI was 41.9%, which appears to be higher. But by Chi-square test, the comparison between history of diabetes and AKI shows no statistical significance (p=0.537) which again, reveals the presence of history of diabetes is not associated with development of AKI in MI patients (Table 4).

The percentage of patients with history of Coronary Artery Disease (CAD) with Non-AKI was 23.4% and the percentage of patients with history of CAD with AKI was 34.9%, which appears higher compared to that of non-AKI patients. However, the comparison between history of CAD and AKI shows no statistical significance with p=0.149, which reveals the presence of past history of CAD is not associated with development of AKI in MI patients, during the

TABLE 4: Group statistics of study population in AKI (n=43) and

Non-AKI-(n=107) groups (T-test)

Parameters	Non-AKI n(107)			AKI n(43)			p value
	Mean	Std. Deviation	Std. Error	Mean	Std. Deviation	Std. Error	
Age(years)	59	8	0.8	63	10	1.6	0.027
Systolic BP(mm Hg)	136	26	2.5	130	42	6.4	0.492*
Diastolic BP(mm Hg)	84	11	1.0	84	12	1.8	0.677*
Blood sugar on admission (mg/dL)	168	62	5.9	154	52	7.8	0.217*
Serum Creatinine – On admission (mg/dL)	0.9	0.3	.03	0.9	0.3	.04	0.846*
Serum Creatinine – after 48 hours (mg/dL)	0.9	0.2	.02	1.6	0.5	.08	<0.001
Serum Cholesterol (mg/dL)	180	38	3.7	192	51	7.8	0.206*
Serum Triglycerides (mg/dL)	134	70	6.8	122	28	4.3	0.123*
Chest pain duration (in hours)	4	4	0.3	10	13	1.9	0.009
Hemoglobin (gm/dL)	12.0	1.5	0.1	11.8	1.7	0.3	0.354*

AKI, Acute Kidney Injury; Non-AKI, Non-Acute Kidney Injury; * p value- Not significant

present episodeThe percentage of patients with history of smoking with Non-AKI group was 27.1% and those with AKI was 32.6%. Yet again, the comparison between history of smoking and AKI shows the no statistical significance with p=0.504 reveals the presence of history of smoking is not a risk factor for the development of AKI in MI patients.

The percentage of patients with history of alcohol intake with Non-AKI patients was 23.4% and the percentage of history of alcohol intake with AKI patients was 18.6%. However the comparison between history of alcohol intake and AKI shows no statistical significance with p=0.525 reveals the presence of history of alcohol consumption is not a risk factor for the development of AKI in MI patients.

The percentage of patients who underwent thrombolysis with Non-AKI group was 59.% and that of AKI group was 86.0%. The comparison between thrombolysis and AKI shows the highly statistical significance with p value=0.002, which reveals that patients with acute myocardial infarction who are treated with thrombolysis are more prone for development of AKI, which however needs further studies which would compare different modalities of treatment of acute MI- such as thrombolysis, Percutaneous Coronary Intervention. The comparison between mortality and AKI shows highly significant p value =0.001 which means the presence of AKI have higher mortality rate (30.2%) than the non AKI patients (9.3%), thus making Acute Kidney Injury a definite risk factor for mortality in MI patients, which is the primary aim of our study.

DISCUSSION

Acute Kidney injury is one of the complications, if developed in patients with Acute Myocardial Infarction, affects adversely the outcome of patients, as evidenced by the results from previous studies[6-9]. Hence there is a dire need to study more about the renal parameters as a routine in patients who present with acute myocardial infarction, as attempts to salvage the heart are made because it will influence both short- and long-term mortality of these patients.

In our study, 43 patients (28.6%) of the total study population developed AKI, which is greater than the C.S Fox et al study which documented 16% of AKI in hospitalised acute MI patients [10] and Bruetto RG et al study that documented 14.6% of AKI in acute MI patients [11].

Most number of MI patients who developed AKI fall within the elderly age group of 61-70 yrs. Previous studies have revealed that development of renal insufficiency in elderly MI patients adversely affects outcome in terms of mortality which continues until 6 months after myocardial infarction[12,13]. Among 43 patients who developed AKI, 25 were male (58.2%) and 18 female (41.8%). Here, though the percentage of male who developed AKI is higher, by statistical analysis, gender does not prove to be an individual risk factor for developing AKI in hospitalized acute MI patients. Of the 18 female who developed AKI, 6 female (33.3%) had co-existing hypertension, 6 female (33.3%) were diabetics and history of coronary artery disease was present in 8 (44.4%) of them.

However, in our study, there was no significance in terms of comorbidities such as history of hypertension, diabetes mellitus, prior history of CAD and personal habits such as smoking, alcohol consumption, to be a risk factor for developing AKI in hospitalized patients with acute myocardial infarction, which proved to be conflicting results compared to CS Fox et al study. Hence more studies are required in analysing the role of these parameters in development of AKI following MI. Both male and female population who developed AKI, fall into stage I, accounting for 19.3%, Stage II with 6.7% and stage III with 2.7%, thus adding upto a cumulative percentage of 28.6%, the distribution of which was similar to the results from CS Fox et al study across various stages [10].

Of the total 150 study population, 23 patients had died during their 5-day in-hospital stay. Of the 23 patients who died, 13 patients (5 female) had developed AKI, thus proving that patients with AMI who had developed AKI had poor outcome in terms of 5-day in-hospital mortality, which is very much similar to the results obtained from the previous studies [14-17]

CONCLUSION

1. The incidence of Acute Kidney Injury in hospitalized patients presenting with Acute myocardial Infarction is 28.6%
2. History of hypertension, diabetes, prior CAD, and personal habits such as smoking and alcohol consumption were not significantly associated with development of AKI in acute MI patients in this study.
3. Development of AKI is associated with poor outcome in terms of 5-day in-hospital mortality of acute MI patients, with p= 0.001.

Acute Kidney injury is an important and common complication occurring in acute MI patients. All the more, there is an increase need for more prospective studies to document the incidence of acute renal insufficiency occurring during critical illness such as MI, and the associated comorbidities so that the treatment modalities can be altered accordingly so as to prevent further damage to the kidneys during the acute phase of illness, and by educating the patient to be on long term follow up to identify early before these acutely injured pair of kidneys fail irreversibly so that necessary measures can be initiated to ensure better quality of life in such patients.

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