

Original Research Paper

General Medicine

AKI IN POST MI: A KILLER IN DISGUISE

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ABSTRACT INTRODUCTION: There has been a constant association of adverse clinical outcome in myocardial infarction (MI) patients with acute kidney injury (AKI). However, whether AKI contributes to an excess risk of cardiovascular events is still controversial, as very few studies are conducted in this context and less amount of knowledge regarding AKI's short-term effects after its occurrence as a complication of AMI, so this study was performed.

 $\textbf{AIM:} \ To find the incidence of AKI in post MI patients, study their clinical profile and outcome of AKI in post MI patients.$

MATERIAL AND METHOD: Prospective observational study was undertaken in Saraswathi Medical College and Hospital, Bareilly including 100 patients admitted in the ICU, emergency and ward from 1stMay 2019 to 31stNov 2019. They were subjected to careful history taking and detailed clinical examination; routine investigations were done. The data was computed and was statistically analyzed.

RESULTS: Out of 100, 36 patients had AKI. Mean age of the patients with AKI was 50 ± 6.1 years and those without AKI was 53 ± 5.9 years. Out of 100, 44 had inferior wall MI, 36 had anterior wall MI and 20 had NSTEMI. Mean fasting blood glucose (mg/dL) in patients without AKI was 94.7 ± 32 and in those with AKI was 145 ± 40 . Mean troponin I(ng/ml) was 2.4 ± 3.4 in patients without AKI and 5.29 ± 7.1 in those with AKI which was found statistically significant (p value=0.015). Mean urea (mg/dL) and creatinine (mg/dL) was 33.9 ± 11.9 and 0.74 ± 0.11 respectively in patients without AKI and with AKI was 70 ± 48.9 and 2 ± 1.45 respectively. Patients with AKI after MI had a more number of complications like arrhythmia, heart failure, hypotension, LVEF (<30%), rupture (free wall or septum or papillary muscle) as compared to patients without AKI. Mortality in patients without AKI was 4 and in patients with AKI was 6.

CONCLUSION: High troponin I and older age are the most important risk factors for AKI in patients with acute MI. Post-MI complications and mortality in patients with AKI was more than that in patients without AKI. Careful monitoring of susceptible patients in ICU is recommended for early detection and management of AKI in patients with MI.

KEYWORDS:

INTRODUCTION

There has been a constant association of adverse clinical outcome in myocardial infarction (MI) patients with acute kidney injury (AKI). AKI often complicates the hospital stay and is prevalent in about 30% of cases admitted to ICU. $^{\rm I}$ The incidence of AKI has surpassed the incidence of stroke and has increased by fourfolds in the U.S. since 1988 and is in a rising trend and will probably continue to do so in future as well if proper measures are not taken. $^{\rm I}$

Due to advancement in the field of medical sciences in both, pharmacological as well as revascularization therapy, the overall survival of ST-elevation myocardial infarction (STEMI) patients has considerably improved in the last two decades. The presently the physician's focus is shifting on the subsets whose outcome after MI is still disappointing and mortality rate remains high. Poor prognosis is associated in patients who develop AKI after MI. However, management guideline of STEMI currently does not focus much attention on AKI. Therefore, this study was planned in view of providing a framework of reference to raise the awareness of AKI post MI to physicians, so as to improve the outcome of STEMI patients with AKI.

Even though AKI is a frequent complication seen in the hospitals and is known to have a marked impact on morbidity and mortality, many important questions remain unanswered like whether AKI contributes to a high risk of cardiovascular events. As very few studies are conducted in this context and a lesser amount of knowledge regarding AKI's short-term effects after its occurrences as a complication of AMI. Thus, this study was performed to find out the incidence of AKI in post MI patients and to study their clinical profile and the outcome of AKI in post MI.

MATERIALS AND METHOD:

Prospective observational study was undertaken in

Saraswathi Medical College and Hospital, Bareilly including 100 patients admitted in the medical ICU, emergency and ward from 1 **May 2019 to 31 **Nov 2019.

Detailed history taking and clinical examination was performed and laboratoryinvestigations were done after explaining the nature of study and informed written consent taken from the patients. The following investigations were done:

- Blood glucose: FBS, PPBS
- Renal function tests
- Electrolytes
- Urine routine and microscopy
- Electrocardiography(ECG)
- · Chest x-ray
- Echocardiography
- Cardiac enzymes like troponin, CPK-MB

The data was collected, computed and statistically analyzed.

RESULTS:

Out of 100, 36 patients had AKI. Mean age of these patients was 53 ± 5.9 years in patients with AKI and those without AKI were 50 ± 6.1 years. Out of 100, 44 had inferior wall MI, 36 had anterior wall MI and 20 had NSTEMI.

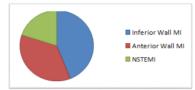


Fig. 1. Showing incidence of inferior wall , anterior wall MI and NSTEMI

Incidence of AKI in acute MI is 36. Mean fasting blood glucose (mg/dL) was 94.7 ± 32.1 in patients without AKI and with that of AKI was 145 ± 40 . Mean troponin I(ng/ml) was 2.4 ± 3.4 in patients without AKI and with that of AKI was 5.29 ± 7.1 which was found statistically significant (p value=0.015). Mean urea (mg/dL) and creatinine (mg/dL) was 33.9 ± 11.9 and 0.74 ± 0.11 respectively in patients without AKI and with that of AKI was 70 ± 48.9 and 2 ± 1.45 respectively.

Table 1. Clinical and Laboratory profile of patients with AKI and without AKI

	No AKI	AKI	P Value
Age	50± 6.1	53±5.9	0.004*
Troponin I(ng/ml)	2.4±3.4	5.29 ± 7.1	0.015*
CPK-MB (ng/ml)	65.3±84.9	44.1±76.5	0.19
Urea (mg/dl)	33.9±11.9	70±48.9	0.00002*
Creatinine (mg/dl)	0.74±0.11	2±1.45	0.000003*
Albumin (g/dl)	3.7±0.6	3.5±0.4	0.086
A/C ratio (mg/g)	84±58	99±86	0.16
Fasting Blood Glucose (mg/dl)	94.7±32.1	145±40	0.013*

^{*}is statistically significant using Chi Square test

Patients with AKI after MI had a more number of complications like arrhythmia, heart failure, hypotension, LVEF (<30%), rupture (free wall or septum or papillary muscle) as compared to patients without AKI as shown in the table below.

Table 2. Complication in post-MI patients with AKI and without AKI

Complications	Post-MI with AKI	Post MI without AKI	P- Value
Arrhythmia	5 (13.9%)	3 (4.7%)	0.048*
Heart Failure	6 (16.7%)	4 (6.3%)	0.046*
Hypotension	21 (58.3%)	19 (29.7%)	<0.001*
LVEF (<30%)	11 (30.6%)	8 (12.5%)	<0.001*
Rupture (free wall or septum or papillary muscle)	1 (2.8%)	0 (0%)	0.477
Death	6 (16.7%)	4(6.3%)	0.0238*

^{*}is statistically significant using Chi Square test

Mortality in patients with AKI was 6 and in patients without AKI was 4, which was found to be statistically significant.

DISCUSSION:

The incidence of AKI increased by fourfolds in the United States since 1988 and is projected to have incidence of 500 per 100,000 population annually, which is more than incidence of stroke annually. AKI is related to markedly increased risk of mortality in hospitalized patients, predominantly in those who were admitted to the ICU wherein mortality rates could be more than 50%.[1]

Acute kidney injury (AKI) is defined by the impairment of kidney filtration and excretory function over days to weeks, resulting in retention of nitrogenous and other waste products normally cleared by the kidneys. By this definition the presence of AKI is defined by an elevation in the serum creatinine concentration or reduction in urine output. AKI is currently defined by a rise in serum creatinine from baseline of at least 0.3 mg/dL within 48 h or at least 50% higher than baseline within 1 week or a reduction in urine output to <0.5 mL/kg per h for longer than 6 h. 7

AKI leads to an increase in cardiac biomarkers of ischemia 2 ; also there is an increase in cytokines (inflammatory) 3 , endothelial dysfunction 4 and dysregulation in mineral metabolism $^{5.6}$. In post MI patients, the complication of AKI, for example, dyselectrolytemia , hypervolumia, acidosis and others leads to increase in mortality and adverse outcome after MI.

In a study by Kirsten E. Fleischmann, Fox CS et al found that clinical factors, for example heart failure, hypotension, and the majority cardiac risk factors; and the procedures such as coronary artery catheterization and coronary artery bypass grafting, were found associated with increasedrisk for AKI. They found that mortality was 2.1% in patients without AKI, as compared to patients with mild, moderate and severe AKI was 6.6%, 14.2%, and 31.8% respectively. AKI was found to be associated with an increased risk of bleeding, i.e. 8.4% in patients without AKI and 32.7% in patients with severe AKI. [8] Parikh RP, Steven G, Coca DO; Yongfei MS, et al found that AKI has a graded and independent association with long-term mortality. They concluded that mild, moderate and severe AKI was associated with a 15%, 23% and 33% increased risk of death respectively at 10 years. [9]

Thus, we conclude that AKI is a frequent complication in AMI that is associated with notable short- and long-term mortality. Post-MI complication and mortality in patients with AKI was more than that in patients without AKI. Careful monitoring of susceptible patients in ICU is recommended for early detection and management of AKI in those patients.

CONCLUSION:

High troponin I and older age are the most important risk factors for AKI in patients with acute MI. Post-MI complication and mortality in patients with AKI was more than that in patients without AKI. Careful monitoring of susceptible patients in ICU is recommended for early detection and management of AKI in those patients.

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