

Clinical spectrum and prognosis of patients with AKI in acute myocardial infarction prospective study at tertiary care hospital north India

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ABSTRACT

Aims and objectives: To highlight the clinical spectrum of myocardial infarction related AKI and its prognosis.

Materials and methods: It was a prospective observational study.

Results and conclusions: We found out that presence of ACS with increased creatinine on admission is associated with increase in 1 year mortality and morbidity independent of other conventional risk factors.

Keywords: AKI, MI, Prognosis

INTRODUCTION

Acute kidney injury (AKI), formally called acute renal failure (ARF), is commonly defined as an abrupt decline in renal function, clinically manifesting as a reversible acute increase in nitrogen waste products measured by blood urea nitrogen (BUN) and serum creatinine over the course of hours to weeks. The vague nature of this definition has historically made epidemiologic study on and around AKI difficult to generalize to patient populations and compare between scholarly works. Several classification systems were developed to streamline research and clinical practice with respect to AKI. ⁽¹⁻⁴⁾

In the United States, approximately 1% of patients admitted to hospitals have AKI at the time of admission. The estimated incidence rate of AKI during hospitalization is 2-5%. AKI develops within 30 days postoperatively in approximately 1% of general surgery cases and arises in up to 67% of intensive care unit (ICU) patients. ⁽⁵⁾ The definition of AKI was based on an absolute increase in serum creatinine (SCr) depending on the admission SCr:

increase in SCr of ≥ 0.5 mg/dl if admission SCr ≤ 1.9 mg/dl; increase of ≥ 1.0 mg/dl for admission SCr of 2.0 to 4.9; or an increase ≥ 1.5 mg/dl for SCr ≥ 5.0 mg/dl. ⁽⁶⁾ Acute kidney injury (AKI) is characterized by a sudden impairment of kidney function occurring over a period of hours to days. A diagnosis of AKI is currently made on the basis of the presence of increased serum creatinine and/or blood urea nitrogen (BUN) levels and/or a decreased urine output, despite their well-known limitations ⁽⁷⁾. It should be noted that changes in BUN and serum creatinine may represent not only renal injury, but also normal responses of the kidney to extracellular volume depletion or decreased renal blood flow. Recent developments in the diagnosis of AKI include 1) use of the RIFLE (R-renal risk, I Injury, F-failure, L-loss of kidney function, E-end stage kidney disease and AKIN *Manuscript Click here to view linked References (Acute Kidney Injury Network) criteria and 2) use of biomarkers of AKI. The Acute Dialysis Quality Initiative developed a consensus definition

and classification of AKI based on creatinine increase and decrease in glomerular filtration rate (GFR) or urine output: the RIFLE criteria. More recently, the RIFLE criteria were modified by the AKIN. The criteria are identical to the first three stages of RIFLE, with the exception of a shorter time frame of AKI within 48 hours, and a lower creatinine threshold of greater than 0.3 mg/dL from Baseline to peak value^(8,9)

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. It is almost always associated with rupture of an atherosclerotic plaque and partial or complete thrombosis of the infarct-related artery.

Classification is based on ECG changes and presence or absence of cardiac markers in blood. Distinguishing NSTEMI and STEMI is useful because prognosis and treatment are different: Unstable angina: Rest angina that is prolonged (usually > 20 min) New-onset angina of at least class 3 severity NSTEMI AND STEMI Non-ST-segment elevation MI (NSTEMI, subendocardial MI) is myocardial necrosis (evidenced by cardiac markers in blood; troponin I or troponin T and CK will be elevated) without acute ST-segment elevation or Q waves. ECG changes such as ST-segment depression, T-wave inversion, or both may be present⁽¹⁰⁾

Acute kidney injury (AKI), formally called acute renal failure (ARF), is commonly defined as an abrupt decline in renal function, clinically manifesting as a reversible acute increase in nitrogen waste products measured by blood urea nitrogen (BUN) and serum creatinine over the course of hours to weeks. The definition of AKI was based on an absolute increase in serum creatinine (SCr) depending on the admission SCr: increase in SCr of ≥ 0.5 mg/dl if admission SCr ≤ 1.9 mg/dl; increase of ≥ 1.0 mg/dl for admission SCr of 2.0 to 4.9; or an increase ≥ 1.5 mg/dl for SCr ≥ 5.0 mg/dl. Multiple studies assessing various cohorts of patients have shown that subsets of AKI survivors are at high risk for progression to advanced stage CKD^(11,12,13,14). 30-Day mortality for patients with AKI is 50– 80%,

there is an obvious desire to target AKI during the initiation phase.¹⁵

Materials and methods:

It was a prospective study which was conducted under cardiology department of SKIMS Srinagar, where patients admitted with acute coronary syndrome were assessed for AKI. Sample of 200 patients were taken and assessment was done by using AKIN Criteria (Acute kidney injury network,2007 modified version of Rife) where patients Serum creatinine at time of admission,24,48 hours, predischage. Patients were divided into mild, moderate, severe based on differences in serum creatinine levels. Mild 0.3_0.4mg/dl rise Moderate 0.5_1mg/dl rise Severe > 1mg/dl rise Patients were categorized into following categories:

1. Patients with no AKI.
2. Patients with mild AKI that resolve by discharge.
3. Patients with mild AKI that didn't resolve by discharge.
4. Patients with moderate AKI that resolve by discharge.
5. Patients with moderate AKI that didn't resolve by discharge.
6. Patient with severe AKI that resolve by discharge .
7. Patient with severe AKI that didn't resolve by discharge.

At the time of admission, eGFR was calculated using Modification of diet in renal disease equation: Estimated e GFR = $186 (\text{Sr.Cr.level in mg/dl}) (\text{age in years})$. The product of this formula is multiplied by a correction of .742 for women. Those patients who had AKI were followed for a period of 1 year. At 1, 3,6,12 months patients S.Cr , eGFR,was calculated .CKD was diagnosed in patients with e GFR 3 months. UAAP patient's 3ml blood was taken in plain vial and after centrifugation serum creatinine level was done and eGFR was calculated. Follow up of the patient for short term (30 days) and long term period was done .Relation b/w AKI and mortality after ACS was analysed to know the primary outcome of study.

Results:**Patient characteristics****Table 1**

Total patients	Male	Female	Mean age	Smoker	Non smoker	Dyslipidemia	Hypertension
200	160	40	58 years	120	80	140	160

Patient characteristics who got AKI**Table 2**

Total AKI	Male AKI	Female AKI	smoker	Non smoker	dyslipidemias	Mean age	hypertension
35	30	05	33	02	34	58	35

Table 3**Relationship of ACE inhibitor use and b blocker use and AKI in MI**

MI PATIENTS WITH	ACE Inhibitor use	No ACE Use	Beta blocker use	No beta blocker use
WITH AKI	32	03	0	35
WITHOUT AKI	156	09	05	160
TOTAL	188	12	05	195

Table 4

Mortality and AKI Severity Relationship in MI

Category	Dead	Alive	Total
Normal	1	0	1
Mild	0	3	3
Moderate	1	11	12
Severe	4	15	19
Total	6	29	35

Discussion:

In our study Patients were divided into two group: MI Patients with AKI and without AKI. Mean age of patients with AKI was 57 years and without AKI was 59 years .Similar age distribution was seen in study conducted by Maranzie et al ⁽¹⁶⁾.

In AKI patients 87.5% were male, 14.3% were female and without AKI 84.8% were male, and 15.2% were female. Overall in MI patients 85% were male, 15% were female. Zehran et al ¹⁷ and Mehmet Akif caker et al ¹⁸ had also observed similar sex distribution in a study conducted by them.

In patients with AKI 5.2 % were nonsmokers and 94.3% were smokers, without AKI 47.9 % were nonsmoker and 52.1% were smokers. Among comorbidities, 97.1% had dyslipidemia in AKI patients and among patients without AKI 64.8% were having dyslipidemia, Hypertension was present in almost

every MI patient. Similar comorbidities were seen in study conducted by Rosana G bruetto et al (19).

We have observed that those patients who had higher creatinine during hospital admission had significant impact on 1 year mortality and morbidity. Reinecke et al (19) had also observed in a study conducted by them that long-term mortality increases with higher creatinine levels, with a significant difference that starts at a level of 1.3 mg/dL and higher.

In HIJAMI Registry, Yamaguchi et al (20) showed that in-hospital mortality of patients with AMI was greater when the serum creatinine concentration was elevated. This adverse outcome was observed not only in patients with severe renal dysfunction but also in those with mild renal dysfunction.

Carolinin x fox et al (21) also observed in a study conducted by them that, 16.1% had AKI, In-hospital mortality rates for those with mild, moderate and severe AKI were 6.6%, 14.2%, and 31.8% compared

to 2.1% without AKI. Only difference in our study was that in mild grade of severity of AKI we don't have higher mortality which may be because of early use of diuretic and fluid and salt restriction.

Results and conclusions: We found out that presence of ACS with increased creatinine on admission is associated with increase in 1 year mortality and morbidity independent of other conventional risk factors.

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