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Original Research Article

Epidemiology, clinical profile and outcome of acute kidney injury in intensive coronary care unit

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ABSTRACT

Background: The incidence of AKI in cardiac ICU is attributed mainly to Heart Failure and Acute Coronary Syndrome. AKI occurs commonly in the setting of AHF, and is termed CRS type 1. Biomarkers and bioelectrical impedance analysis can be helpful in estimating the real volume overload and may be useful to predict and avoid AKI. The role of UF remains controversial, and it is currently recommended only for diuretic-resistant patients. Objective of current study was to study demographic & clinical profile and outcome of patients with AKI in intensive coronary care unit.
Methods: This prospective study was conducted in ICCU of R.N.T. Medical College, Udaipur. All the patients with increase in serum creatinine ≥50% were included in the study. Detailed investigations like urinary analysis, renal function tests (blood urea, serum creatinine, serum electrolytes), USG whole abdomen, 12 lead ECG, Echocardiography and Troponin T.
Results: Among cases 56.67% had ADHF, 25% had MI, 10% had structural heart disease, 3.3% had systemic illness, 1.67% had cardiogenic shock, 1.67% were cardiac surgery associated and 1.67% had other causes of AKI. 30.0% of cases required ionotropic support while 2.5% of controls required ionotropic support. 5.0% of cases required ventilator support & renal replacement therapy while none of the controls required these.
Conclusions: Patients with AKI had worse outcomes when compared to non-AKI. Mortality among cases was significantly higher than controls, 10% among cases versus only 2.5% in controls.

Keywords: AKI, ICCU, CRS, Biomarkers, MI

INTRODUCTION

Acute Kidney Injury (AKI) is a common clinical problem in intensive care unit (ICU) especially in intensive coronary care unit patients and independently predicts poor outcome. Recently, multi-centre cohort studies reported the occurrence of AKI in an estimated 36% of all patients admitted to the ICU.[1-3]

The incidence of AKI in cardiac ICU is attributed mainly to Heart Failure and Acute Coronary Syndrome. The incidence of AKI varies between 25-33% and 9-19% in Heart failure and ACS respectively. This broad range in incidence is also attributable to the different timeframe used to ascertain renal impairment. Moreover, additional observational data indicate that the incidence of AKI is rising.[3]

RIFLE criteria for acute renal dysfunction and a revision of the criteria were proposed by the acute kidney injury Network (AKIN) - a group representing members of Acute Dialysis Quality Initiative, nephrology and critical care societies. The proposed diagnostic criteria for AKI is an abrupt (within 48 hours) reduction in kidney function defined as an absolute increase in serum creatinine level of >26.4 mmol/l (0.3 mg/dl) or a percentage increase in

serum creatinine level of > 50% (1.5 fold from baseline) or a reduction in urine output (documented oliguria of <0.5 ml/kg/hour for > 6 hours). These criteria should be applied in the context of the clinical presentation and following adequate fluid resuscitation when applicable.

The coexistence of renal and cardiac disease can be today defined as cardiorenal syndrome (CRS). The definition of cardiorenal syndrome (CRS) was proposed by the acute dialysis quality initiative (ADQI) to describe 'Disorders of the heart and kidneys whereby acute or chronic dysfunction in one organ may induce acute or chronic dysfunction of the other'. CRS is classified into five subtypes, considering which organ is affected first and whether it is affected acutely or chronically.[4]

AKI occurs commonly in the setting of AHF, and is termed CRS type. It is associated with adverse clinical outcomes, including increased mortality, rehospitalization, and increased healthcare expenditures. Multiple pathophysiologic mechanisms have been implicated, including neurohormonal activation, venous congestion, inflammation, effects of pharmacologic therapy for HF (RAAS antagonists and diuretics), and nephrotoxic exposure. Prevention is of paramount importance, consisting of avoiding acute decompensation of chronic HF, and, among patients already presenting with AHF, prompt recognition of those at increased risk for AKI. Among patients with established AKI, diuretics remain the cornerstone of therapy. IV administration by bolus or continuous infusion appears to be equally efficacious. Biomarkers and bioelectrical impedance analysis can be helpful in estimating the real volume overload and may be useful to predict and avoid AKI. The role of UF remains controversial, and it is currently recommended only for diuretic-resistant patients.

Aims and objectives

Aim and objective of current study was to investigate demographic & clinical profile and outcome of patients with AKI in intensive coronary care unit.

METHODS

This prospective study was conducted in intensive coronary care unit (ICCU) of RNT medical college, Udaipur during the period January 2021 to June 2021. All the patients with increase in serum creatinine ≥50% were included in the study.

Patients with CKD and/ or abnormal kidney size and abnormal cortico medullary differentiation were excluded from the study.

Cases: Patients admitted in intensive coronary care unit with AKI. Controls: Patients admitted in intensive coronary care unit not having AKI. A thorough diagnostic evaluation was done by a detailed history, physical examination, urinary analysis, Renal Function tests (blood urea, serum creatinine, serum electrolytes), USG whole abdomen, 12 lead ECG, Echocardiography and Troponin T. Renal function tests like (S. urea and S. Creatinine) were taken on day of admission (Day 1) and were repeated within 24 hours of admission (Day 2) and between 24-48 hours of admission (Day 3). Wherever possible the etiological factors were treated. Renal Replacement Therapy was given according to the clinical and biochemical indications.

Statistical method

Data was entered in Microsoft excel and it was analysed by SPSS software version (V27). Chi Square test was used to compare cases and controls on qualitative parameters and T test was done for quantitative parameters. P value were calculated and significance of comparison was tested.

RESULTS

Among cases mean age was 61.20 years and among controls mean age was 60.73 years (p=0.86, NS). Among cases 76.67% were males while 70.00% were males in controls.

Table 1: Demographic Data.

Gender	AKI (Cases n=60)	Non-AKI (Controls n=40)
Males (%)	76.67	70.00
Females (%)	23.33	30.00
Mean age (years)	61.20	60.73

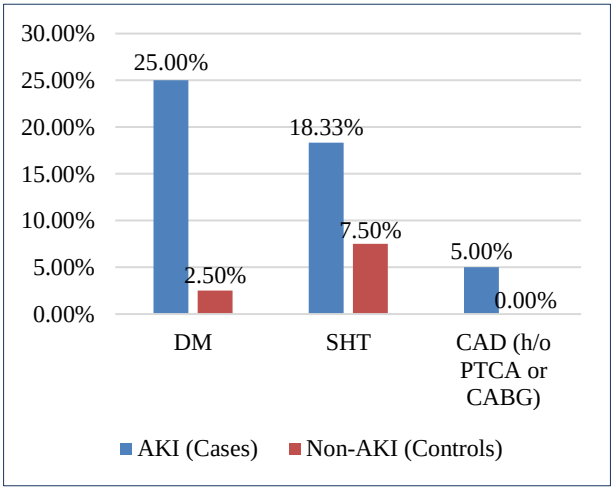

Figure 1: Comorbidity.

Comorbidity

Among cases 25.00% were diabetic while among controls 2.50% were diabetic. Among cases 18.33% had Hypertension while among controls 7.50% had hypertension. Among cases 5.00% had previous history of

CAD (PTCA or CABG) while among controls Nil (0.00%) had such history.

Among cases 56.67% had ADHF, 25% had MI, 10% had structural heart disease, 3.3% had systemic illness, 1.67% had cardiogenic shock, 1.67% were cardiac surgery associated and 1.67% had other causes of AKI. Among Controls 72.50% had Acute Decompensated Heart Failure, 12.50% had MI, 2.50% had structural heart disease, none had systemic illness and cardiogenic shock, 2.50% were cardiac surgery associated and 10.00% had other diseases. 30.0% of cases required ionotropic support while 2.5% of controls required ionotropic support. 5.0% of cases required ventilator support while none of the controls required ventilator support. 5.0% of cases required renal replacement therapy while none of the controls required renal replacement therapy. Mortality rate in cases was 10.0% while in controls it was 2.5%.

Table 2: Aetiology of AKI.

Parameters	Cases		Controls	
Aetiology of AKI (Cases)	**N**	**%**	**N**	**%**
Acute decompensated heart failure (ADHF)	34	56.67	29	72.50
Myocardial infarction (MI)	15	25.00	5	12.50
Structural heart diseases	6	10.00	1	2.50
Cardiogenic shock	1	1.67	0	0.00
Cardiac surgery associated	1	1.67	0	0.00
Systemic illness	2	3.33	1	2.50
Others	1	1.67	4	10.00
Total	60	100.0	40	100.0

DISCUSSION

AKI is a very common complication observed in ICU patients, especially intensive coronary care units and accounts for independent predictor of mortality and morbidity.

Table 3: Outcome

Outcome	Cases (%)	Controls (%)	P value
Ionotropic support	30.00	2.50	<0.001 (HS)
Ventilatory support	5.00	0.00	<0.001 (HS)
Requirement of renal replacement therapy	5.00	0.00	<0.001 (HS)
Mortality	10.00	2.50	<0.001 (HS)

Among cases mean age was 61.2 years and among controls it was 60.73 years and the p value calculated was 0.86 (>0.05) which showed no significant statistical difference between both these groups. Most of the patients with AKI (cases) were in age group of 61-80 years while most patients without AKI (controls) were in age group of 41-60 years (p value 0.56 i.e., >0.05). Similar to ours Jacob C et al had mean age of patients with AKI as 67.5 years. Buargub et al the mean age of those with AKI was 61.6 years and of those without AKI was 54.7 years (p value >0.05) and most of the patients with AKI were between 45-70 years and without AKI between 40-70 years (p value > 0.05) similar to that in our study.[5] Insignificant difference in age of cases and controls signifies that other risk factors like underlying illness, comorbidities, drugs and other factors being implicated in development of AKI.

Among cases 25% were diabetic while among controls only 2.5% were diabetic and this difference is statistically significant (p value <0.05). Several studies evaluated AKI epidemiology in diabetic patients. Mehta and colleagues performed a retrospective analysis, based on the Society of Thoracic Surgeons National Database.[6] All patients included between 2002 and 2004 were analysed, with a total number of 449,524 individuals. The total prevalence of DM was 33%. Dialysis treatment became mandatory in 6,451 patients after surgery. In individuals requiring dialysis, diabetes was diagnosed more frequently than in those without renal replacement therapy (49 versus 33%, p<0.0001). In addition, more detailed analysis using a multivariate logistic regression model revealed diabetes as independent risk factor for developing AKI after cardiac surgery. Another study published by Oliveira and colleagues prospectively evaluated patients undergoing aminoglycoside treatment (n=980).[7] The primary endpoint was a reduction in the glomerular filtration rate (GFR) of 20% or more.[8] The diabetes prevalence was 19.6% in patients that fulfilled the endpoint versus 9.3% without GFR reduction (p=0.007). Comparable to the study by Mehta et al. Oliveira and colleagues performed logistic regression analyses as well.[6] These showed several independent AKI risk factors: baseline. GFR of <60 mL/min/1.73 m^2, the use of iodinated contrast media, hypotension, concomitant use of nephrotoxic drugs, and diabetes (p=0.046).

Girman et al differed in the following categories: obesity, congestive heart failure, hypertension, alcohol and tobacco exposure, past AKI episodes, CKD prevalence, therapy with ACE inhibitors/angiotensin receptor blockers, therapy with other antihypertensive drugs, statin treatment, and NSAID use (p values in every category below 0.001).[8] Hsu and colleagues compared 1,746 hospitalized adults (Kaiser Permanente Northern California) that developed dialysis requiring AKI with over 600,000 individuals without such a complication.[9] The following parameters were identified as independent AKI risk factors: preadmission diabetes mellitus, arterial hypertension, and pre-existing proteinuria similar observations have been made in a retrospective study by Patschan et al where they concluded that hyperglycaemia is a well-known risk factor for endothelial dysfunction.[10] Even quite early after being

exposed to a hyperglycaemic milieu, for instance, induced by the administration of "advanced glycation end-products" (AGEs), cultured endothelial cells show impaired production of nitric oxide which reflects the loss of cellular competence and AKI ensues from transient renal hypo perfusion or ischemia.[11] Our results were in accordance to above studies and showed a positive relation between AKI and diabetes mellitus.

Among cases 5% had prior history of CAD (history of PTCA/CABG) while none of the controls had such history and this difference was statistically significant (p value<0.05). Similar results were obtained by study published in Saudi Journal by Buargub et al where 5 out of 35 patients with AKI had h/o heart disease as compared to 3 out of 49 controls and it was concluded that various mechanisms are postulated for this, like ischemic injury to kidney, low ejection fraction after MI leading to more pronounced renal injury, drugs causing nephrotoxicity and hypo perfusion post-surgical procedure.[5,12]

Among cases the aetiology of AKI was as follows: Acute Decompensated Heart failure accounting for 56.67%, Myocardial infarction accounting for 25%, Structural heart disease accounting for 10%, Systemic illness accounting for 3.33%, 1.67% each to cardiogenic shock, Cardiac surgery associated and other causes. The major bulk of AKI cases had Acute decompensated heart failure and myocardial infarction as the aetiology and similar findings have been observed in a study published in European Journal by Ronco et al where it was observed that about 40% patients with acute decompensated heart failure developed AKI and 27% patients with MI developed AKI and in both Acute Decompensated Heart Failure and Acute Coronary Syndrome, the development of AKI has been associated with greater short- and long-term all-cause and cardiovascular mortality, prolonged duration of hospitalization, increased readmission rates, accelerated progression to CKD stages 4-5, and higher healthcare costs. [13-17] Among cases 30% required ionotropic support while only 2.5% of controls required ionotropic support, 5% of cases required ventilator support while none of the controls required ventilator support, moreover 5% cases required Renal replacement therapy while none of the cases required it. All these differences were statistically significant (p value<0.05). Finally, in our study the mortality among cases was 10% while among controls was 2.5% and this difference was also statistically significant (p value<0.05). A Multicentre study by Li Jiang et al "Epidemiology of acute kidney injury in intensive care units in Beijing" where a total of 3107 patients were included 665 patients required ionotropic support out of which 366 (23.1%) patients had AKI and 299 (19.6%) patients didn't have AKI and difference was statistically significant (p<0.05), 2344 patients required Ventilator support out of which 1260 (79.5%) had AKI and 1084 (71.2%) didn't have AKI and difference was statistically significant (p<0.05) and 281 patients with AKI required Renal replacement therapy (270 patients with AKI and 11 patients without AKI with p value <0.05) and it was also concluded in this study that patients with AKI had worse outcomes in form of prolonged hospital stay, more morbidity, more multi organ dysfunction and mortality.[18] In above mentioned study Mortality among patients with AKI was 28.1% compared to 3.2% in patients not having AKI (p<0.05). Buargub et al also in his study of 84 patients in cardiac ICU reported 25.7% death in patients with AKI in comparison to 6.12% in patients not having AKI. In a study by Wang et al with 19,249 hospitalised patients, AKI occurred in over 1 of 5 hospitalized patients and was associated with an over fourfold increased mortality.[5,12]

Many of the effects of AKI are difficult to identify and quantify independently, but experimental data are increasingly elucidating the subclinical effects of AKI on distant organ function.[19] It is obvious that problems such as refractory hyperkalaemia, pulmonary oedema, or uremic manifestations such as pericarditis are related to AKI when they acutely develop in the appropriate setting. Other uremic manifestations may have several explanations (encephalopathy, acidosis), or may be occult causes of other complications (bleeding diathesis and gastrointestinal bleed, leukocyte dysfunction with immunosuppression and nosocomial infection). In addition to the emerging data that appear to confirm an independent role of AKI in increasing mortality in the ICU, it is also clinically obvious that AKI is a cause of significant morbidity (including ALI and other distant organ injury) and severely complicates ICU management like in our case.

AKI occurs commonly in the setting of AHF, and is termed CRS type 1. It is associated with adverse clinical outcomes, including increased mortality, rehospitalization, and increased healthcare expenditures. Multiple pathophysiologic mechanisms have been implicated, including neurohormonal activation, venous congestion, inflammation, effects of pharmacologic therapy for HF (RAAS antagonists and diuretics), and nephrotoxic exposure. Prevention is of paramount importance, consisting of avoiding acute decompensation of chronic HF, and, among patients already presenting with AHF, prompt recognition of those at increased risk for AKI. Among patients with established AKI, diuretics remain the cornerstone of therapy. IV administration by bolus or continuous infusion appears to be equally efficacious. Biomarkers and bioelectrical impedance analysis can be helpful in estimating the real volume overload and may be useful to predict and avoid AKI. The role of UF remains controversial, and it is currently recommended only for diuretic-resistant patients.

CONCLUSION

Aetiology among non-AKI individuals were as follows: Acute decompensated heart failure 79.50%, Myocardial infarction 5%, Structural Heart disease and systemic illness 2.5% while others were accounting for 10% and none of the patient had cardiogenic shock and cardiac surgery associated aetiology. Patients with AKI (Cases)

had worse outcomes when compared to non-AKI (Controls). 30% cases required ionotropic support while only 2.5% controls required ionotropic support. 5% cases required ventilator support while none of the controls required ventilator support. 5% cases required Renal Replacement therapy while none of the controls required it. Finally, mortality among cases was significantly higher than controls, 10% among cases versus only 2.5% in controls.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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