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Research Article

CLINICAL PROFILE AND OUTCOME OF HOSPITAL ACQUIRED ACUTE KIDNEY INJURY VERSUS COMMUNITY ACQUIRED ACUTE KIDNEY INJURY- A PROSPECTIVE STUDY FROM CENTRAL INDIA

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ABSTRACT
Although the epidemiology and the impact of acute kidney injury on outcomes are well known in the western literature, good data is lacking from central India. Little is known about patients sustaining acute kidney injury (AKI) in the community acquired acute kidney injury (CAAKI) and how this differs from AKI in hospital acquired acute kidney injury (HAAKI). The objective of this study is to compare epidemiology, clinical characteristics, aetiologies, severity and outcomes of patients of these two categories. A prospective study was conducted during 24 months from October 2013 to October 2015 in Sri Aurobindo Medical College and Post Graduate Institute, Indore. All patients admitted to different departments of the hospital and having AKI were included in the study. AKI was verified by applying the RIFLE criteria, and patients were categorized as CAAKI if RIFLE criteria were met at admission. HAAKI was defined as if RIFLE criteria were met twenty four hours or longer after hospitalization. Among the 500 patients with AKI, 286 were classified as CAAKI (57.2%). There was no significant difference in age average and co morbidities between CAAKI and HAAKI. Dehydration and volume depletion were significantly more prevalent in patients with CAAKI (47.9% *vs.* 28.97% for HAAKI $p<0.0001$). While HAAKI was associated with a significantly higher prevalence of drug induced AKI (23.36% *vs.* 7% in CAAKI $p<0.0001$). Patients with HAAKI were sicker than compared to patients with CAAKI. Maximum RIFLE class was more severe, and mortality rate was higher with class F in both groups. The mortality in hospital was significantly higher in the HAAKI group compared to CAAKI group (51% *versus* 20% $p<0.0001$). This study highlights that risk factors for CAAKI and HAAKI are similar, with pre-existing CKD, diabetes, heart disease, hypertension, cancer and tuberculosis being common risk factors for both. As our study had shown, early recognition of AKI that is in RIFLE class I has better outcome in both the groups. So, mortality and morbidity can be safely reduced with team approach and involving nephrologists early in the course of illness.

Keywords: *Acute Kidney Injury, Community Acquired Acute Kidney Injury, Hospital Acquired Acute Kidney Injury, Aetiology, Chronic Kidney Disease*

INTRODUCTION
Acute kidney injury is an increasingly common and potentially catastrophic complication in hospitalized patients. Early observational studies from the 1980s (Hou *et al.,* 1983) and 1990s (Liano *et al.,* 1996) established the general epidemiologic features of acute kidney injury: the incidence, prognostic significance, and predisposing medical and surgical conditions. Recent multicenter observational cohorts and administrative databases have enhanced our understanding of the overall disease burden of acute kidney injury and trends in its epidemiology. An increasing number of clinical studies focusing on specific types of acute kidney injury [*e.g.*, in the setting of intravenous contrast (Mitchell and Kline, 2007) sepsis (Yegenaga *et al.,* 2004) and major surgery (Thakar *et al.,* 2005)] have provided further details into this heterogeneous syndrome. Despite our sophisticated understanding of the epidemiology and pathobiology of acute kidney injury, current prevention strategies are inadequate and current treatment options outside of renal replacement therapy are nonexistent. There has been increasing interest in the identification and validation of novel biomarkers (Wagener *et al.,* 2006) of acute kidney injury that may permit earlier and more accurate diagnosis. Acute Kidney Injury is a syndrome characterized by

rapid decline in glomerular filtration rate and retention of nitrogenous waste products such as blood urea nitrogen and creatinine. Acute kidney injury in the hospitalized patients is associated with high rates of morbidity, mortality and consumption of healthcare resources. Occurrence of renal failure in patients hospitalized for non-renal problems complicates the hospital course with profound impact on patient outcomes.

International Society of nephrology has recognised acute kidney injury as a major health concern and it aims to curb down mortality associated with acute kidney injury to zero by 2025 (0 by 25 initiative) (Mehta *et al.*, 2015). Knowledge of incidence and risk factors is crucial because it drives local and international efforts on detection and treatment. Also, notable differences exist between developing and developed countries: Incidence seems higher in the former, but underreporting compounded by age and gender disparities makes available data unreliable. In developing countries, incidence varies seasonally; incidence peaks cause critical shortages in medical and nursing personnel. Finally, in developing countries higher incidence of sepsis, lack of trained persons to deal with intensive care, lack of systematic evaluation of the role of falciparum malaria, obstetric mechanisms, and hemolytic uremic syndrome on AKI hampers efforts to prevent acute kidney injury.

Limited data are available on the aetiology and outcome acute kidney injury in Indian population especially from central India. In this observational study, we compare clinical characteristics, aetiologies, and outcomes of patients admitted to the hospital with community acquired acute kidney injury in contrast to those who acquired AKI during their inpatient stay.

MATERIALS AND METHODS

We conducted a prospective study of all patients who were admitted in Sri Aurobindo Medical College and Postgraduate Institute, Indore from October 2013 to October 2015. The facility is a tertiary care referral center in central India catering to the states of Madhya Pradesh, Uttar Pradesh, Rajasthan and Gujarat. All the patients of age more than 18, admitted during the study period with acute kidney injury (AKI) or who developed acute kidney injury during the hospital stay were included. The patients were subjected to detailed history and thorough clinical examination. Particulars such as name, age, sex, address and contact information was noted in a pre-structured proforma. Subjects were also enquired regarding and past medical illness which can be a risk factor for acute kidney injury. Detailed history regarding drugs and other ayurvedic or naturopathy preparation has been asked. Investigations were done to confirm the diagnosis. Complete blood count, renal function test, liver function test, electrolytes, serum proteins, Sugar levels electrolytes, LDH, peripheral smear, urine routine, chest X ray, ultrasonography of abdomen, electrocardiogram were done in all the patients. Further set of Investigation were selectively done to confirm the diagnosis and aetiology. Immunological Profile, HBA1c, blood Urine throat or fluid cultures, Renal colour Doppler, 2 D Echo was done in patients as per the primary suspicion, Cardiac and pulmonary evaluation was done as and when required. Kidney biopsy was done if there was no renal recovery in 3 weeks.

After Confirming the diagnosis the patients were classified into hospital acquired acute kidney injury and community acquired kidney injury. Hospital acquired acute kidney injury was defined as increase in serum creatinine by 0.5 mg/dl for patients with baseline creatinine <1.2 mg /dl and a rise of 1 mg/dl for serum creatinine ≥ 1.2 mg /dl or decline in urine output 0.5 ml/kg/hr as per the RIFLE criteria (Bellomo *et al.*, 2004). Community acquired acute kidney injury was defined as patients who had creatinine > 1.2 mg/dl at the time of admission with rise of 0.5 mg /dl for patients with baseline creatinine < 1.2 mg/dl and a rise of 1 mg/dl for baseline creatinine >1.2 mg/dl. The lowest creatinine level during admission was taken as baseline. The most probable cause of AKI was assigned as given below:

1) Decreased renal perfusion was identified by one or more of (a) Decrease in blood pressure to less than 90/60 mm Hg (b) Evidence of congestive heart failure (c) Signs of volume depletion (d) Improvement with restoration of blood flow.

2) Drugs were identified as cause of AKI when there was a temporal relationship to administration of the drug, in the absence of other pathological mechanisms.

3) Sepsis was defined as two or more of the following as a result of proven or suspected infection (a) temperature >38°C or <36°C (b) heart rate>90/min (c) respiratory rate >24 /min (d) White blood count >12000/micro litre, <4000/micro litre or >10% band forms.
4) Radiographic contrast nephropathy was defined as either a 25% increase in serum creatinine or an absolute increase in serum creatinine of 0.5 mg/dl within 24 hours of the procedure.
5) Obstruction was attributed to cause AKI if there was evidence on imaging.
6) Post operative, in patients with post operative AKI the operative and anaesthetics notes were reviewed to identify the most probable cause of AKI.
Patients were followed daily until the last day of hospital stay and 3 months after discharge. Serial records of urine output and serum creatinine were maintained. Renal replacement therapy was instituted according to standard clinical indications. We have classified patients according to the maximum RIFLE class (class R, class I or class F) reached during their hospital stay and Outcome in each class of RIFLE (Table 1).

Table 1: RIFLE Classification (Bellomo *et al.*, 2004)

Class	GFR	Urine Output
Risk	↑ S Cr × 1.5 or ↓ GFR >25%	<0.5 mL/kg/h × 6 h
Injury	↑ S Cr × 2 or ↓ GFR >50%	<0.5 mL/kg/h × 12 h
Failure	↑ S Cr × 3 or ↓ GFR >75% or if baseline S Cr ≥353.6 μmol/L (≥4 mg/dL) ↑ S Cr >44.2 μmol/L (>0.5 mg/dL)	<0.3 mL/kg/h × 24 h or anuria × 12 h
Loss of kidney function	Complete loss of kidney function >4 weeks	
End-stage kidney Disease	Complete loss of kidneyfunction >3 months	

Outcome was compared between Hospital acquired acute kidney injury and community acquired acute kidney injury. Outcomes of acute kidney injury in this study were measured in terms of: Length of hospital stays (LOS) - defined as the number of days from admission to discharge, death, or leave against medical advice from hospital. Treatment outcome: dies or discharged with improved renal function. Complete recovery was defined as decrease in the serum creatinine to <1.2 mg/dl along with improvement in urine output during the hospital stay.
Partial recovery of renal function will be defined as the improvement in renal function (as determined by an increase in urine output and a decrease in serum creatinine) but serum creatinine levels still >1.2 mg/dl at the time of discharge from the hospital. Dialysis dependent were those who required dialysis for life long.

Statistical Analysis

Statistical analysis was carried out using SPSS software, version 20. A descriptive analysis was performed; Continuous data was presented as mean and standard deviation (m±Sd) and categorical data as a percent and 95% Confident Interval (CI). At the univariate analysis, proportions were compared between groups using a Pearson chi-squared test. Continuous data were compared using t-test when comparisons were between two groups.

Ethical Considerations

An informed consent for participating in the study was obtained for all patients. No invasive investigation means was used. The authors declare no conflict of interest.

RESULTS AND DISCUSSION

Results

We included 500 patients having AKI; out of which 214 patients had hospital acquired acute kidney injury and 286 had community acquired acute kidney injury. The demographic and clinical profiles of

both groups of patients are shown in Table 2. Mean age of HAAKI and CAAKI patients was 44 ± 17.3years (range16-92) and 48 ± 17 years (range 20-85) respectively. The patients with CAAKI are older compared to HAAKI. The male patients were dominant in both HAAKI and CAAKI groups (59.6% and 64.2% of cases, respectively). Both the groups had association with various co morbid conditions as described in Table 2.

Table 2: Demographic Features of Patients with HAAKI and CAAKI

Parameter	HAAKI(214)	CAAKI(286)	p
Mean age (y)	44 ± 17.3	48 ± 17	0.04
Male	128(59.8)	181 (64.5)	0.18
Female	86 (40.4)	102 (35.5)	
Diabetes mellitus	40 (18.6%)	55 (19.2%)	0.43
Hypertension	38 (17.75%)	52 (18.18%)	0.45
Pre-existing CKD	28(13.08%)	40(13.98%)	0.38
Coronary artery disease	15(7.0%)	20 (6.99%)	0.49
Malignancy	18(8.4%)	28(9.7%)	0.29
COPD	6(2.8%)	9(3.1%)	0.41
Tuberculosis	10(4.67%)	15(5.2%)	0.38

The etiological factors of hospital acquired acute kidney injury and community acquired acute kidney injury differ, as shown in Table 3, the leading cause was sepsis (35%) in HAAKI and it was more than CAAKI (26.92%), the difference was statistically significant. Volume depletion was more in CAAKI than HAAKI (47.9% versus 28.97 % respectively) and the difference was statistically significant (p <0.0001). In volume depletion, acute gastroenteritis was more prevalent in CAAKI (32% versus 9.8% in HAAKI). Acute glomerulonephritis was more in CAAKI than HAAKI (8.39 % Vs 0.46% respectively with p <0.001).

The drugs being the third common cause of AKI in HAAKI, it is more in HAAKI than CAAKI (23.36% versus 7 % respectively with p being significant.

Table 3: Aetiology of AKI in Patients with HAAKI versus CAAKI

Aetiology OF AKI	HAAKI n = 214	CAAKI n = 286	*p*
Volume Depletion & Hypoperfusion	62(28.97)	137(47.9%)	<0.0001
Acute Gastroenteritis	21(9.81%)	94(32%)	<0.0001
Other Causes of Volume depletion	41(19%)	43(15.35)	<0.1112
SEPSIS	75(35%)	77(26.92%)	<0.025
UTI	23(10.74)	34(11.88%)	
Respiratory Tract infection	32(14.95)	24(8.3%)	
Abdominal infection/Wound Infection	15(7%)	7(2.44%)	
Malaria	0	10(3.4%)	
Dengue	0	1(0.34%)	
Snake Bite	0	1(0.34%)	
DRUGS	50(23.36%)	20(7%)	<0.0001
CIN	9(4.2)	0	
Acute Glomerulonephritis	1(0.46%)	24(8.39%)	<0.0001
TMA	0	2(0.69%)	
Urinary Tract Obstruction	0	26(9%)	

Contrast induced nephropathy was seen only in HAAKI in 4.2% cases. Thrombotic miocroangiopathy and urinary tract obstruction was seen only in CAAKI with incidence of 2 % and 26% respectively.
The patients with HAAKI are sicker than CAAKI as described in table 4.

Table 4: Comparison of Severity in HAAKI and CAAKI

Particulars	HA-AKI(n =214)	CA-AKI (n = 286)	P value
ICU care	56(26.16%)	40(13.98%)	0.0003
Ventilator	45(21%)	20(07%)	0.0001
Dialysis	40(19%)	85(30%)	<0.006
Duration of Hospital stay	14.74 days	13.23 days	0.19

The need of ICU care was required in 26.16 % of HAAKI patients while only 13.98 % of CAAKI required ICU care. The need of ventilator was also significantly more in HAAKI than CAAKI (21% versus 7 % respectively). Though the need of dialysis was more with CAAKI than HAAKI (30% versus 19%) but the length of hospital stay was almost same.
RIFLE class I and II had very low mortality in community acquired group (4%) collectively as compared to 24 % In HAAKI group depicted in table 5. RIFLE Class III groups also had statistically higher mortality in HAAKI versus CAAKI (27.1 % versus 16%). This contributes to more severe form of AKI in HAAKI even if they are picked early during RIFLE class I and II the outcome remains poor.

Table 5: The RIFLE Classification and its Association with Mortality

RIFLE Class	HA-AKI	CA-AKI	P Value
Class I (R)	10 (4.6%)	3(1%)	<0.0001
Class II (I)	42(19.3%)	9(3%)	<0.005
Class III (F)	58(27.1%)	45(16%)	<0.0001
Total	110(51%)	57 (20%)	<0.0001

Outcome was better of community acquired acute kidney injury group, there was full recovery in 59% of CAAKI but only 30% of HAAKI patients.

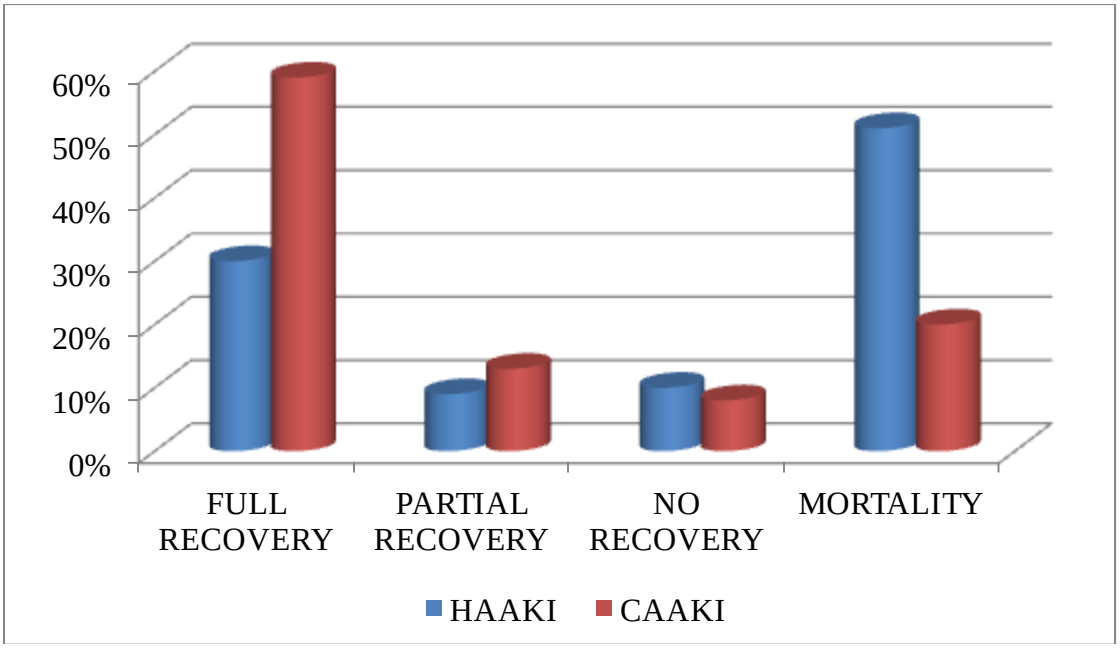

Figure 1: Comparison of Outcome between HAAKI and CAAKI

Mortality was significantly higher in HAAKI (51% versus 20% in CAAKI) owing to higher percentage of sepsis induced AKI in this group.

Discussion

Incidence and associated mortality risks of AKI in critically ill patients are well documented (Mehta *et al.*, 2004; Uchino *et al.*, 2005; Bagshaw *et al.*, 2008). Increases in creatinine in non critically ill hospitalized patients are also common and carry heightened mortality (Liangos *et al.*, 2006). Studies describing and comparing incidence, risk factors, and outcomes of patients who sustain AKI in the hospital versus who sustain AKI in community are limited, and the few studies performed have large difference in the patient numbers. In this study, we identified 286 patients who sustained AKI in the community and were subsequently admitted to the hospital. We compared this cohort with 214 patients who sustained AKI during a hospital stay. The incidence of CAAKI was low in present study than in previous studies (Wonnacott *et al.*, 2014; Bardai *et al.*, 2015). However, this probably remains an underestimation of the true amount of CAAKI that exists, because numerous patients with CAAKI neither have blood tests performed nor get admitted to the hospital.

This study highlights that risk factors for CAAKI and HAAKI are similar, with CAAKI also being more common in male patients with pre existing CKD, diabetes, heart disease, hypertension, COPD, cancer and tuberculosis. This highlights the demographic characteristics of people in the community who may benefit from more frequent blood tests in the event of an acute illness or medication change.

Even mild AKI is no longer considered to be benign, but rather an independent predictor of mortality (Lafrance and Miller, 2010; Liangos *et al.*, 2006; Meier *et al.*, 2011). Patients with CAAKI had more severe AKI, shorter hospitalizations, yet better long-term survival than patients with HAAKI. Superior survival in CAAKI was surprising because these patients had co morbid conditions similar to those of patients with HAAKI. The reasons for these differences remain unclear. Interestingly, a study performed in Scotland demonstrated that inadequacies in recognition and management of hospital based AKI were particularly high in patients with mild AKI (Aitken *et al.*, 2013).

Thus, more severe CAAKI may have been more appropriately managed, leading to better outcomes. Perhaps further contributing to differences in management of CAAKI and HAAKI is that on admission to the hospital patients are generally assessed thoroughly and all laboratory investigation are done within 6 to 24 hours. However, after this, further management is dependent on symptoms of patients with investigations only done when needed, so oliguric AKI is missed in hospitalized patients in absence of symptoms as it is well known that uremic symptoms develop very late in course, so only those patients are picked early who develop reduced urine output. This may also underlie differences in early appropriate recognition and management of CAAKI and HAAKI, which may ultimately influence differences in outcomes.

Although, we could identify patients with unrecognized AKI at the point of discharge from hospital, we were unable to assess whether AKI was missed or mismanaged during inpatient stay. It is, however, evident that patients with CAAKI were more likely to be referred to nephrology, thus, further suggesting that they were more likely to be recognized and appropriately managed. Important differences in the aetiology of AKI may also be a factor in dictating short and long-term outcomes. With previous work that suggested vasculitis, GN, and obstructive uropathy may be more prevalent in CAAKI, while prerenal failure and acute tubular necrosis were more common in patients with HAAKI (Liano *et al.*, 1996). It is clear that the incidence and mortality of CAAKI are significant and clinically underappreciated. The incidence of CAAKI was 2.91 % and HAAKI was 2.18 % in our study.

Three percent of AKI episodes were mild (RIFLE I) in CAAKI group and 4.6 % from HAAKI presented in class I. whereas most patients (70%) had sever renal insufficiency (RIFLE III), and 24% class II. Length of hospital stay was a mean of 12.5±13.5 days. The global mortality rate among all patients in the study was 35%. The patients of our study group were younger compared to other study (Wonnacott *et al.*, 2014) because the overall survival is less in Indian population. Pre-existing CKD was observed in 13.53% of patients with AKI, with similar proportions across the CAAKI and HAAKI groups (13.98% versus 13.08%; p=NS).

Comparison of prevalence of various co morbid conditions in patients with CAAKI and HAAKI revealed approximately equal proportions of such diagnoses as diabetes, hypertension, heart disease and cancer as seen in previous study (Bardai *et al.,* 2015).

In our group we found that history of tuberculosis also had significant association with AKI in both groups CAAKI and HAAKI, this need further attention. The physiologic characteristics of AKI were divided into three categories; prerenal, intrarenal, and post renal. Intrarenal causes of AKI accounted for a greater proportion of HAAKI (49% vs. 36%; p< 0.05), while prerenal causes were more common among patients with CAAKI (48% vs. 43%; p=NS). Dehydration and volume depletion was significantly more prevalent in patients with CAAKI (47.9% vs. 28.97; p<0.0001) and attribute to better recovery rates of this group. The frequency of glomerulonephritis was higher in CAAKI (8.39%% versus 0.46%: p <0.001). CAAKI was associated with a significantly lower prevalence of acute tubular necrosis (ATN) than HAAKI (12.1 vs. 50%; p<0.0001). The Contrast induced nephropathy was exclusively seen in HAAKI (4.2% Vs 0). Sepsis was more common in HAAKI (35 % Vs 26.92) and could be the reason of high mortality in this group.

We have also investigated the data for acute mortality and short-term outcomes. Outcomes in patients with CAAKI and HAAKI are shown in Figure 1. Severity of AKI in term of ICU care; Ventilator requirement was much more in HAAKI than CAAKI. There were significant differences between the numbers of patients requiring renal replacement therapy; it was high in CAAKI (19% in the HAAKI group and 30% in the CAAKI group). The length of hospital stay; the median stay in both patients with CAAKI and HAAKI was similar (13.23 and 14.5 days respectively). However, mortality in hospital was significantly higher in the HAAKI group compared to CAAKI group (51% HAAKI versus 20% CAAKI; p<0.0001).

The current study found that incidence of CAAKI was comparable to HAAKI, accounting for nearly half of the patients with a diagnosis of AKI, probably because our center is tertiary center with all super specialties, all extensive surgeries, cardiothoracic, neurology, bariatric, burn unit with all Intensive care units. This finding is not in consistent with previous reports (Wonnacott *et al.,* 2014) identified 686 patients who sustained AKI in the community. They compared this cohort with 334 patients who sustained AKI during a hospital stay.

The incidence of CAAKI was found at 86.2% in this study. In two earlier studies, Obialo *et al.,* (2000) performed a retrospective study of 100 African Americans with AKI in which 80% of patients had CAAKI and Wang *et al.,* (2007) reported that 60% of 211 Chinese patients with AKI had CAAKI. The absence of a reliable baseline serum creatinine was a significant limitation in both studies. The availability for a baseline creatinine in the current study allowed us to accurately identify patients with CAAKI, to define the prevalence of CKD in our cohort, and to accurately classify the severity of AKI.

It has been previously noted that mortality in CAAKI may be up to 20% lower than that of HA-AKI (Brivet *et al.,* 1996). According to some recent reports, the mortality rate in CAAKI ranged from 15% to 26% (Liano *et al.,* 1996), whereas the mortality rate in HAAKI ranged from 25% to 70 % (Brivet *et al.,* 1996).

Also, the mortality rates observed in our study were consistent with these published reports. In this study, although AKI severity and comorbidity had a similar distribution between CAAKI and HAAKI groups, the mortality rate was significantly higher in the HAAKI group compared to documented predictors of mortality such as oliguria, sepsis, multiorgan failure, and ICU stay or mechanical ventilation occurred more frequently in patients HAAKI (Chertow *et al.,* 2005).

In our study, we actually found the same finding, in fact, HAAKI group had higher prevalence of mechanical ventilation (21% versus 7%; p value <0.0001), higher rate of multiorgan failure (17% vs. 14%. p=NS), higher prevalence of anuria (15.1% vs. 8.3% p= NS) and a higher rate of ICU stay (26.16% versus 13.98%; p<0.003). The predictors of severity in our study are same as seen in study by Biradar *et al.,* (2004). The Long term outcome of our study group as depicted in figure 1, full recovery was significantly more in CAAKI (59% Versus 30%: p value 0.0001) was mainly because of difference in aetiological factors.

Conclusion

This current report is one of few prospective study comparing CAAKI and HAAKI. Our data suggest that CAAKI is a common cause of AKI that is as severe as that seen in HAAKI. AKI has a significant impact on length of stay, mortality, and the development and/or progression of CKD. Development of strategies to limit the risk of CAAKI such as high risk factor subject screening may have a significant impact on healthcare costs and patient's prognosis.

Despite this, patients with CAAKI have better short and long-term outcomes, the reasons for which are unknown.

The reason may be the difference in aetiology as the most common aetiology of CAAKI is volume depletion thus, it gets full recovery.

AKI is a catastrophic disease and to deal with such a cataclysmic disease leading to high mortality and morbidity, frequent epidemiological studies from all parts of country are needed to devise the preventive and therapeutic strategies for this condition. Early recognition and nephrology intervention can lead to better outcome.

Abbreviations

AKI: Acute Kidney Injury,
CKD: Chronic Kidney Disease;
CAAKI: Community Acquired AKI;
HAAKI: Hospital-acquired acute kidney injury;
RIFLE: Risk, Injury, Failure, Loss, ESRD
sCr: serum Creatinine
ICU: Intensive Care Unit
ATN: Acute Tubular Necrosis;
GN: Glomerulonephritis
KDIGO: Kidney Disease Improving Global Outcomes

Conflicts of Interest

The authors declare that they have no conflicts of interest.

REFERENCES

Aitken E, Carruthers C, Gall L *et al.*, (2013). Acute kidney injury: outcomes and quality of care. *Quarterly Journal of Medicine* **106**(4) hcs237.

Bagshaw SM, George C, Gibney RN & Bellomo R (2008). A multi-center evaluation of early acute kidney injury in critically ill trauma patients. *Renal Failure* **30**(6) 581-589.

Bellomo R, Ronco C, Kellum JA, Mehta *et al.*, (2004). Acute renal failure–definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Critical Care* **8**(4) 1.

Biradar V, Urmila A, Renuka S & Pais P (2004). Clinical spectrum of hospital acquired renal failure: a study from tertiary care hospital. *Indian Journal of Nephrology* **14** 93-6.

Brivet FG, Kleinknecht DJ, Loirat P *et al.*, (1996). Acute renal failure in intensive care units--causes, outcome, and prognostic factors of hospital mortality: a prospective, multicenter study. *Critical Care Medicine* **24**(2) 192-198.

Chertow GM, Burdick E, Honour M *et al.*, (2005). Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *Journal of the American Society of Nephrology* **16**(11) 3365-3370.

El Bardai G, Kabbali N, Najdi A, Arrayhani M & Sqalli T (2015). Comparison of Hospital-Acquired and Community-Acquired Acute Kidney Injury in Hospitalized Patients. *Open Journal of Nephrology* **1**(2)

Hou SH, Bushinsky DA, Wish JB *et al.*, (1983). Hospital-acquired renal insufficiency: a prospective study. *The American Journal of Medicine* **74**(2) 243-248.

Lafrance JP & Miller DR (2010). Acute kidney injury associates with increased long-term mortality. *Journal of the American Society of Nephrology* **21**(2) 345-352.

Liangos O, Wald R, O'Bell JW *et al.*, (2006). Epidemiology and outcomes of acute renal failure in hospitalized patients: a national survey. *Clinical Journal of the American Society of Nephrology* **1**(1) 43-51.

Liano F, Pascual J & Madrid Acute Renal Failure Study Group (1996). Epidemiology of acute renal failure: a prospective, multicenter, community-based study. *Kidney International* **50**(3) 811-818.

Mehta RL, Cerdá J, Burdmann EA *et al.*, (2015). International Society of Nephrology's 0by25 initiative for acute kidney injury (zero preventable deaths by 2025): a human rights case for nephrology. *The Lancet* **385**(9987) 2616-2643.

Mehta RL, Pascual MT, Soroko S *et al.*, & Program to Improve Care in Acute Renal Disease (PICARD) (2004). Spectrum of acute renal failure in the intensive care unit: the PICARD experience. *Kidney International* **66**(4) 1613-1621.

Meier P, Bonfils RM, Vogt B, Burnand B & Burnier M (2011). Referral patterns and outcomes in non critically ill patients with hospital-acquired acute kidney injury. *Clinical Journal of the American Society of Nephrology* **6**(9) 2215-2225.

Mitchell AM & Kline JA (2007). Contrast nephropathy following computed tomography angiography of the chest for pulmonary embolism in the emergency department. *Journal of Thrombosis and Haemostasis* **5**(1) 50-54.

Obialo CI, Okonofua EC, Tayade AS & Riley LJ (2000). Epidemiology of de novo acute renal failure in hospitalized African Americans: comparing community-acquired vs hospital-acquired disease. *Archives of Internal Medicine* **160**(9) 1309-1313.

Thakar CV, Worley S, Arrigain S *et al.*, (2005). Influence of renal dysfunction on mortality after cardiac surgery: modifying effect of preoperative renal function. *Kidney International* **67**(3) 1112-1119.

Uchino S, Kellum JA, Bellomo R *et al.*, (2005). Acute renal failure in critically ill patients: a multinational, multicenter study. *The Journal of the American Medical Association* **294**(7) 813-818.

Wagener G, Jan M, Kim M *et al.*, (2006). Association between increases in urinary neutrophil gelatinase–associated lipocalin and acute renal dysfunction after adult cardiac surgery. *The Journal of the American Society of Anesthesiologists* **105**(3) 485-491.6.

Wang Y, Cui Z & Fan M (2007). Hospital-acquired and community-acquired acute renal failure in hospitalized Chinese: a ten-year review. *Renal Failure* **29**(2) 163-168.

Wonnacott A, Meran S, Amphlett B *et al.*, (2014). Epidemiology and outcomes in community-acquired versus hospital-acquired AKI. *Clinical Journal of the American Society of Nephrology* **9**(6) 1007-1014.

Yegenaga I, Hoste E, Van Biesen W *et al.*, (2004). Clinical characteristics of patients developing ARF due to sepsis/systemic inflammatory response syndrome: results of a prospective study. *American Journal of Kidney Diseases* **43**(5) 817-824.