ORIGINAL ARTICLE

Etiology, risk factors and outcome of acute kidney injury in a medical intensive care unit

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Abstract

Background: Acute Kidney Injury (AKI) is a common yet serious illness with significant complications having high incidence and prevalence rates. Various studies have been conducted to understand the causes and risk factors of AKI and its outcomes. *Aim and Objectives:* The present research aimed to assess the etiology, risk factors and outcome of AKI in Medical Intensive Care Unit (MICU). *Material and Methods:* In this longitudinal study, 100 participants were recruited between the age group 20-80 years and diagnosed with AKI either at the time of admission or during their stay in MICU. *Results:* Mean age was 61.3 ± 15.43 years. The study participants included 68 male (68%) and 32 female participants (32%). Majority of the participants had diabetes mellitus as a co-morbidity (49%) followed by systemic hypertension (36%). The commonest etiology of AKI in our study, was infections (61.22%). Thirteen individuals progressed to Chronic Kidney Disease (CKD) and 27 succumbed to AKI. The negative outcome with respect to progression to CKD and death was significantly associated with invasive ventilation status ($p = 0.0005$), requirement of dialysis ($p = 0.0365$) and exposure to nephrotoxic drugs ($p = 0.0135$). *Conclusion:* In the present study, infection was the commonest etiology for AKI. Diabetes as well as hypertension were significant risk factors for AKI. A significant number of individuals progressed to CKD and 27 out of 100 individuals succumbed to AKI.

Keywords: Acute Kidney Injury, Etiology, Intensive Care Unit, Outcome, Risk Factors

Introduction

Acute Kidney Injury (AKI) is defined as an acute reduction in kidney function as well as structural damage resulting in loss of function [1]. Worldwide, AKI is considered a serious illness with significant complications resulting in increased overall economic and health care burden [2]. Most nephrology consultations in hospitals are for AKI. Some investigators also believe AKI to be a hospital- acquired disease. The terms "acute renal failure" and "acute renal insufficiency" have been now replaced with the term AKI, which were used for the description of this condition [2].

A meta-analysis used pooled incidence rate of AKI according to the Kidney Disease Improving Global Outcomes (KDIGO) equivalent definition and found the overall, worldwide AKI incidence to be 23.2%; while it was 21.6% in adults, it was 33.7% in pediatric population [3]. The incidence of AKI in children, according to a study from South India, was 5.2% in the pediatric wards and 25.1% in the pediatric Intensive Care Unit (ICU) [4]. A prospective study from North India observing 64,288 patients being hospitalized found that 364 of them developed AKI, indicating 0.56% incidence [5].

However, this condition can be reversed if treated at an early stage [6].

AKI is commonly witnessed in ICUs which is related to numerous factors leading to Chronic Kidney Disease (CKD), all along contributing to enormous financial burden to the patient [7]. Drugs administered to patients while in the hospital have been implicated in up to 35% of all AKIs and the pathophysiology being acute necrosis of the tubules or allergic nephritis [7]. Hospitalized individuals regularly receive antimicrobials, which often result in significant adverse reactions including renal damage. Few commonly used drugs in ICU which cause nephrotoxicity are Non-steroidal Anti-inflammatory Drugs (NSAIDs) resulting in interstitial nephritis and glomerulonephritis; amphotericin causing tubular toxicity; beta lactams and acyclovir causing interstitial nephritis; Angiotensin-Converting Enzyme (ACE) inhibitors leading to glomerular damage; clopidogrel and contrast agents causing tubular cell toxicity; and proton pump inhibitors causing acute interstitial nephritis [8]. Incidence of antimicrobial nephrotoxicity is estimated to be about 35% [7].

The diagnosis of AKI, historically, was based on serum markers such as creatinine and urea along with the measurement of urine output. Researchers later brought the Risk, Injury, Failure, Loss and End-stage, the (RIFLE) definition, which now remains as one of the accepted definitions for AKI [2]. RIFLE criteria for AKI recognizes two key indicators, serum creatinine level and urine output [7]. It is said that individuals who have had AKI and recovered, have a shortened life span in comparison to normal individuals, indicating that recovery from an acute event can still have pathophysiological consequences [9]. Various studies have identified advancing age as a risk factor for development of AKI along with obesity, atherosclerosis, dyslipidemia, smoking and polypharmacy [1]. With this background, the current research was undertaken to assess the etiologies, risk factors and outcome of AKI in Medical Intensive Care Unit (MICU).

Material and Methods

This longitudinal study was undertaken in the Department of General Medicine of a tertiary care hospital, comprising of 100 participants in the age group of 20-80 years of both sexes. All the participants who were diagnosed with AKI at the time of admission to the MICU or during their stay in MICU were included. Subjects with pre-existing kidney disease or renal transplant recipients were excluded from the research.

Institutional Ethics Committee approved the research (IHEC/17/399) on 27.12.2017. National trial registration number was not required as the present study did not involve any intervention. All procedures were followed in accordance with the ethical standards of the responsible committee on human experimentation and with the Helsinki Declaration of 1975.

The inclusion of participants was based on the RIFLE criteria and outcome was measured up to 3 months of follow-up and was considered as recovered when estimated Glomerular Filtration Rate (eGFR) $\geqslant$ 60 ml/min/1.73m^2, CKD-eGFR < 60 ml/min/1.73m^2 or Death [10].

Sample size calculation was done using the following formula:

$$n = \frac{Z^2 P(1 - P)}{d^2}$$

where, n is the sample size required, P is the proportion, Z is the standard normal variate corresponding to level of confidence, d is error

term. The mortality rate of ICU admitted AKI cases, according to one study, was found to be 41% [11] and with 95% confidence level and relative error of 10. Hence the sample size was calculated as,

$$n = \frac{(1.96)^2 (0.41)(1 - 0.41)}{(0.1)^2}$$

$n = 92.92$

By assuming non-response rate as 7.5%, the final sample size was determined as 100 cases.

Informed consent was obtained from either the participants or the next of kin. The participants' complete history consisting of the comorbidities namely diabetes, hypertension, hypothyroidism, dyslipidemia, coronary artery disease and history of smoking and alcoholism along with drug history and native medicine history were recorded or collected from the case files. Clinical parameters such as blood pressure, pulse rate, respiratory rate, oxygen saturation and Glasgow Coma Scale scores were recorded along with laboratory parameters such as serum urea, creatinine, albumin, potassium, HCO_3, hemoglobin and platelet counts.

Data analysis was done using R software (Version i3863.6.3). Continuous variables were represented by Mean ± Standard Deviation. Categorical variables were represented by frequency tables. Continuous data were compared using ANOVA followed by Tukey HSD as post hoc test or Kruskal Wallis followed by pair wise Mann-Whitney U-test with Bonferroni adjustment as post hoc, depending on the normality distribution. Categorical data were compared using chi-square test or chi-square test with simulation. p-value < 0.05 was considered as statistically significant.

Results

Demographic details

One hundred participants were included in this prospective research. Participants were in the age range between 20 to 80 years with mean age of 61.3 ± 15.43 years. There were 68 male (68%) and 32 female participants (32%). Majority of them had diabetes mellitus as a co-morbidity (49%) and as per the statistical analysis, the negative or positive outcome was significantly associated with gender (p = 0.0195) and none of the co-morbidities were significantly associated with the outcome except for coronary artery disease (p = 0.0290). (Table 1)

Laboratory and clinical findings

Table 2 presented the comparison between groups based on laboratory and clinical data. Mean pulse rate (p = 0.0349) and mean HCO_3 levels (p = 0.0113) were shown to be significantly associated with the outcome. When the etiologic profile for AKI was assessed, infection was found to be a major factor (49%), followed by pulmonary causes (17%) such as bronchial asthma and Chronic Obstructive Pulmonary Disease (COPD) and cardiac causes (16% and 17% respectively). Heart failure was present in 21 participants (7%) and 45 of them had to be on invasive ventilation. The association with different etiological factors was represented in Table 3 and showed negative outcome which was significantly associated with invasive ventilation status (p = 0.0005), requirement of dialysis (p = 0.0365) and exposure to nephrotoxic drugs (p = 0.0135). When the outcome was assessed, 61% had complete recovery, 13 progressed to CKD and 27 succumbed to AKI.

Participant status

Among the participants whose condition improved, 40% needed invasive support, 40.43% participants required inotropes, 78.57% were exposed to nephrotoxic drugs and all participants were exposed to contrast agents (Figure 1).

Table 1: Comparison between groups based on the demographic data and comorbidities

Parameter	Sub-categories	Recovery (n=61)	Chronic Kidney Disease (n=13)	Death (n=27)	*p*
Age (year)		60.31 ± 16.93	69.67 ± 9.59	59.81 ± 12.99	0.1332[A]
Gender	**Male**	37 (54.41%)	7 (10.29%)	24 (35.29%)	0.0195[CS]
	Female	24 (75%)	5 (15.63%)	3 (9.38%)	
Diabetes	**Yes**	30 (61.22%)	8 (16.33%)	11 (22.45%)	0.3268[C]
	No	31 (60.78%)	4 (7.84%)	16 (31.37%)	

A indicates ANOVA test; C indicates Chi-square test; CS indicates Chi-square test with simulation; KW indicates Kruskal Wallis test

Table 2: Comparison between groups based on the laboratory and clinical data

Parameter	Recovery (n=61)	Chronic Kidney Disease (n=13)	Death (n=27)	*p*
GCS (mean ± SD)	12.77 ± 4.03	13.92 ± 2.64	11.74 ± 4.46	0.0675[KW]
Pulse rate (%)	101.95 ± 25.39	92.17 ± 22.84	112.48 ± 19.13	0.0349[A]
SpO_2 (mean ± SD)	86.7 ± 14.23	86.42 ± 11.70	77.78 ± 12.78	0.0040[KW]
HCO_3	19.14 ± 5.40	21.48 ± 8.11	15.73 ± 6.28	0.0113 [A]

A indicates ANOVA test; C indicates Chi-square test; CS indicates Chi-square test with simulation; KW indicates Kruskal Wallis test

Table 3: Comparison between groups based on the aetiologies, use of mechanical ventilation, heart failure, albuminuria, dialysis

Parameter	Sub-categories	Recovery (n=61)	Chronic KidneyDisease (n=13)	Death (n=27)	*p*
Aetiology	**Infections**	30 (61.22%)	5 (10.2%)	14 (28.57%)	0.4418 [CS]
	Cardiac	11 (68.75%)	2 (12.5%)	3 (18.75%)	
	Gastrointestinal	5 (50%)	1 (10%)	4 (40%)	
	Pulmonary	11 (64.71%)	4 (23.53%)	2 (11.76%)	
	Neurological	3 (75%)	0 (0%)	1 (25%)	
	Others	1 (25%)	0 (0%)	3 (75%)	
Use of Mechanical Ventilator (%)	**Yes**	40%	4.44%	55.56%	0.0005
	No	78.18%	18.18%	3.64%	
Dialysis (%)	**Yes**	38.89%	27.78%	33.33%	0.0365
	No	65.85%	8.54%	25.61%	
Heart Failure	**Normal**	46 (58.23%)	9 (11.39%)	24 (30.38%)	0.2709 [CS]
	mild	3 (75%)	1 (25%)	0 (0%)	
	Moderate	9 (90%)	1 (10%)	0 (0%)	
	Severe	3 (42.86%)	1 (14.29%)	3 (42.86%)	
Exposure to Nephrotoxic Drugs	**Exposed**	78.57%	14.29%	7.14%	0.0135
	Not exposed	54.17%	11.11%	34.72%	

A indicates ANOVA test; C indicates Chi-square test; CS indicates Chi-square test with simulation; KW indicates Kruskal Wallis test

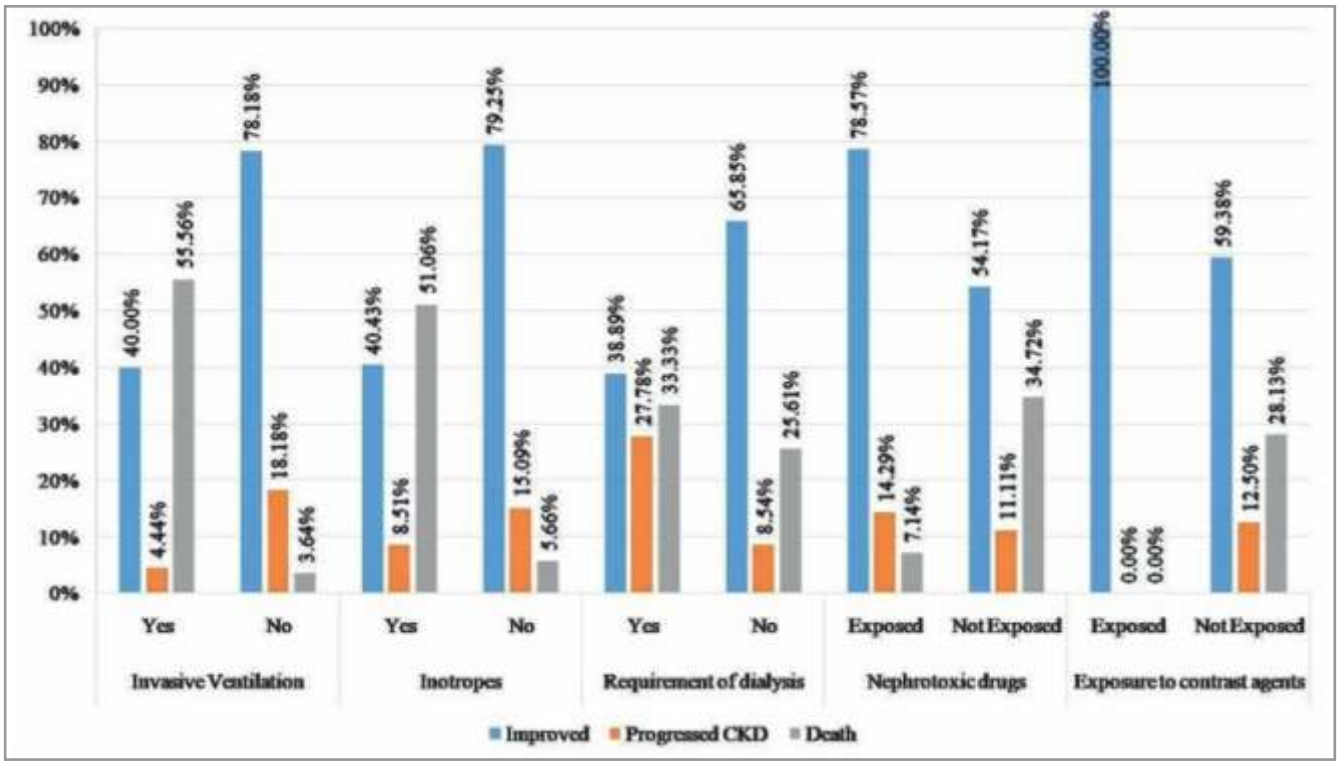

Figure 1: Clinical factors and outcome

Discussion

AKI is known to affect about 1.3 crore individuals per year worldwide and maybe a cause of death in about 2 million individuals. There has been research pointing out that economic, cultural and even geographic causes are risk factors for the development of AKI and this is more pronounced in the developing countries. Differences between the individual's area of residence such as urban or rural have been acknowledged as a risk factor. Furthermore, variations between cultures, seasons and pathogens involved have also been implicated in the development of AKI [12]. Incidence and prevalence of AKI had been determined to vary based on the classification criteria used, whether the RIFLE or Acute Kidney Injury Network (AKIN), and is recorded between 10 to 67% [13-14].

Medve *et al*., (2011) found the mean age of the participants to be 59.6 years [14]. A study by Pinheiro *et al*., (2019) reported the mean age of the participants to be 71 years and 54% of them were males [15]. Another study by Srisawat *et al*., (2020) on 5377 individuals found the mean age to be 65.1 years and 45.8% were females [16]. In the present research, the mean age was found to be 61.3 ± 15.43 years and majority of them were between 60-79 years of age. Akin to previous studies, males were affected by AKI more than females. One study by Hoste *et al*., (2006) found that gender or race do not alter the vulnerability for AKI, but age is a widely accepted risk factor [17]. Another study contrastingly found female gender to be a risk factor for AKI [16]. Neugarten *et al*., (2018) studied sex differences in hospital associated AKI

and concluded that female gender had a protective role for AKI. The authors suggested that AKI is not a disease per se but a congregation of disorders having different sexual dimorphism; there might be a male preponderance for AKI [18].

In the present research, diabetes was the most common comorbidity, followed by hypertension and cardiovascular disease, and this may be due to the variation in geography as well as food habits such as predominantly carbohydrate rich diet further predisposing an individual to the development of diabetes. A meta-analysis found that low e-GFR and high albuminuria were associated with higher AKI, irrespective of diabetes or hypertension [19]. Srisawat *et al*., (2020) found hypertension in 43.4% of their samples, followed by diabetes in 27.6%, coronary artery disease in 10.5% and cardiovascular disease in 6.4% of their study sample [16]. Piccinni *et al*., (2011) also found a higher percentage of individuals who had hypertension (52%) followed by cardiovascular diseases in 48% and diabetes mellitus in 21% [20]. Wonnacott *et al*., (2014) found hypertension to be the most common comorbidity (31.4%) followed by diabetes (24%) [21]. It has been mentioned in literature that comorbidities may augment kidney damage in AKI [16]. One study found that about 10.3% of the AKI subjects were smokers whereas in the present study, a higher percentage of the subjects were smokers (33%) and alcoholics (27%) [12]. It was stated that smoking and COPD may not be the risk factors for AKI, as just 5 out of 32 patients who developed post operative AKI, in their study were smokers and 7 out of 32 patients who developed post-operative AKI had COPD. However, when the data were analyzed using univariate regression analysis, at 95% confidence interval, the odds ratio for smoking was found to be 1.61 (p = 0.38) and for COPD, the odds ratio was 3.92 (p = 0.009). This shows that COPD may a contributing risk factor for the development of AKI [22].

An Australian study analyzing 120,123 patients for a period of five years, between the years 2000 to 2005 found AKI in 36% of them. The most common etiology in their study was sepsis (32.4%) [23]. Another study by Medve *et al*., (2011) found the incidence to be 24.4% in Hungary where they analyzed patients admitted across seven ICUs between 1st October 2009 to 30th November 2009. Most common etiology for ICU admission in their study was gastrointestinal surgery [14].

In the present research, infection was a major cause (49%) for AKI, followed by pulmonary and cardiac causes, Abd ElHafeez *et al*., (2017) found a higher percentage of cardiovascular aetiology (52%) in their study participants, followed by sepsis (36%), diabetes (27.5%), hypovolemia (22%), liver disorders (13%) and COPD (4.8%) [12]. Srisawat *et al*., (2020) found sepsis to be a major cause in their study too with 47% of AKI being due to it, followed by pulmonary causes (6%) and hypoperfusion (3.2%) [21]. Pinheiro *et al*., (2019) found a much higher percentage of sepsis etiology in their samples (77%) in the study [15].

The current study had a higher serum creatinine at the time of admission while hemoglobin and albumin levels were similar to previous studies. Piccinni *et al*., (2011) had the serological parameters such as serum creatinine, urea, sodium and potassium levels assessed at baseline and accordingly, the serum creatinine was 1 mg/dL (0.8-1.1) [20]. Zeng *et al*., (2014) found serum

creatinine at 1.0 (0.8-1.5) mg/dL and hemoglobin at 11.1 (9.7-12.7) g/dL [24].

A study by Wang *et al.,* (2012) observed that their AKI patients had ICU stay for an average of 7 days (4-13 days) [25], while the current research showed the mean ICU stay to be 4.14±2.92 days. A study by Abd ElHafeez *et al*., (2017) established that about 10% of their patients had been exposed to nephrotoxic drugs such as the NSAIDs [12], whereas a study by Piccinni *et al*., (2011) found a much higher (28.1%) exposure to nephrotoxic drugs, primarily NSAIDs [20]. Similarly, about 28% of the present study population had a history of exposure to nephrotoxic drugs. The common drug was vancomycin and one participant had exposure to tenofovir and another to acyclovir. Vancomycin has been found to be the commonest nephrotoxic drug causing as much as 7.2-14.4% nephrotoxicity, and the toxicity was found to be higher when concomitant piperacillin and tazobactam are administered. Aminoglycosides and cancer chemotherapeutic agents are the other drugs that are implicated in the development of AKI [12]. Contrastingly, only 2% of the samples were drug related in the study by Medve *et al*., (2014)[14]. Incidence of Contrast Induced AKI (CIAKI) in ICU patients has been found to range from 11-19% [13]. In this research, only 4% of the participants were exposed to contrast agents. CIAKI is majorly caused due to the use of iodine-based contrast agents. It is estimated that about 8.5% of clinically stable patients when subjected to non-coronary angiography suffered AKI. Certain risk factors such as diabetes, congestive heart failure, pre-existing liver disease, renal insufficiency, use of hyper-osmol contrast agents, etc., have been implicated in development of CIAKI. Prevention of CIAKI is suggested to include low osmol contrast agents, administration of intravenous bicarbonate or saline during the contrast radiographic procedure and also discontinuation of nephrotoxic drugs [26]. Oeyen *et al*., (2015) found that hypertension, nephrotoxic drug exposure and albuminuria were associated with the worst outcome in AKI but did not find any association between other variables. The case fatality was close to 26% and 13% of AKI had progressed to CKD, also 19% of the samples were dialysis dependent [27]. Srisawat *et al*., (2020) found that cardiovascular disease, diabetes, hypertension and coronary artery diseases, all were significant risk factors for AKI [16].

It is noteworthy that AKI patients who progress to CKD have an overall increased risk of development of End Stage Renal Disease (ESRD) and the estimated risk is 13%, and if they had a pre-existing CKD, the risk goes up to 40% [28]. Mortality following AKI has always been high, and about 40-80% AKI patients in the ICU succumb to it. The combination of sepsis and AKI results in increased mortality [16]. Challiner *et al*., (2014) found a lower mortality rate (11.4%) in AKI. They further detected that the presence of diabetes, hypertension, sepsis, age >65 years and diuretics were all statistically significant risk factors, with increased odds ratio for the development of AKI. They did not find any increased odds for contrast agents or nephrotoxic drugs [28]. A higher mortality was also found in the study by Wijewickrama *et al.,* (2014) from Sri Lanka, where 34 out of 65 patients died due to AKI. However, the author states that, whether this increased risk of death is due to complications resulting from AKI per se cannot be determined from this study [29].

Another research by Medve *et al*., (2011)in Hungarian ICUs showed that age and mechanical ventilators were independent risk factors for mortality [14]. The current results depict a similar picture where 27% succumbed to AKI and 12% progressed to CKD but found significant association of AKI with invasive ventilation. The availability of novel biomarkers for AKI has eased the diagnosis and classification of AKI. Some of them include the NGAL-Neutrophil Gelatinase Associated Lipoclain (NGAL), CyC-Cystatin C, Interleukin 18, KIM-1-Kidney Injury Molecule-1, Hepcidin etc [12, 28]. NGAL is one of the most investigated novel biomarkers and the elevation of this marker is found to be much earlier than serum creatinine, thereby facilitating early diagnosis. The usage of these markers may pick up early AKI, termed as subclinical AKI, which might go undiagnosed if traditional markers of GFR are used. They are also more sensitive and have a role to play in prognosis [8, 28]. The treatment of a patient with AKI varies depending on the clinical situation and resource availability. Although the efficacy of a number of frequently used therapies is still debatable, evidence supporting a number of interventions, particularly when administered simultaneously, has grown over the last decade [30]. The limitation of the present study was the small sample size utilized and single centre nature of the study. A longer follow up period would also help in assessing the long-term effects of AKI.

Conclusion

The current research has delineated the etiological profile and the risk factors of AKI as well as their outcome in MICU individuals from Southern India. Infection was noted as the major cause for AKI while diabetes and hypertension were the most common comorbidities. The current research also found a significant association of AKI with invasive ventilation status, requirement of dialysis and exposure to nephrotoxic drugs. Mortality rate, analogous to various previous research was significant and a moderate number of individuals progressed to CKD. The clinical implications of the present study are that the results highlight the importance of identifying AKI in ICU set up, addressing the risk factors of AKI and preventing them to avoid significant morbidity and mortality. A larger sample size would have been helpful in throwing more insights to understanding AKI.

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