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PREVALENCE AND ETIOLOGY OF ACUTE KIDNEY INJURY IN ICU PATIENTS

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ABSTRACT

Throughout the world in ICUs, acute kidney injury (AKI) is becoming increasingly found. Admitted kidney patient develop significant complications and require ongoing adjustments in care, as seen in these reader questions. On the basis of Risk, Injury, Failure, Loss of kidney function, End-stage renal disease (RIFLE) criteria, the subsequent pediatric RIFLE (pRIFLE) score, and the Acute Kidney Injury Network (AKIN) criteria. Anatomically AKI is classified into three categories: pre-renal causes, renal causes and post renal causes. Acute kidney injury (AKI) is an abrupt and usually reversible decline in glomerular filtration rate (GFR). The detection, incidence rate in ICU, causes, diagnosis, and prevention of AKI are presented separately. Among hospitalized patients, AKI is most commonly due to either pre-renal etiologies or acute tubular necrosis (ATN) from ischemia, nephrotoxin exposure, or sepsis. Longer hospital stay and economic burden are inevitable. In contrast to western literature, few reliable statistics are available regarding AKI in India. Causes of AKI are frequently categorized as prerenal, intrinsic renal, and post-renal. This classification system oversimplifies the overlapping pathologic mechanisms underlying AKI. **Material and Methods**: AKI was classified according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria in three stages in this study we studied 270 patients in an ICU during a follow-up of period**.** The use of central venous and arterial blood pressure monitoring, urine analysis and microscopy allows assessment of fluid volume status and etiology of AKI. Data elements included: demographics, comorbid conditions, hospital and ICU admission and discharge data, blood and urine laboratory studies including microbiology, medication use, and the use of parenteral and enteral nutrition. The serum creatinine level increases by ≥ 0.35 mg/dl within 48 h or increase in serum creatinine to more than 1.5 times baseline within the previous 7 days. The urine volume contains < 0.5 ml/kg/h for 6 hours. **Results**: Mean age of the patients included in the study was 65.4 years. There were 168 (62.22%) male and 102 (37.78%) females. In 146 (54.07%) hypertension was the associated co-morbid condition. Diabetes was present in 164(60.74%) of the cases. 24 (8.89%) were having associated liver disease. 6 (2.22%0 were HIV (Human Immunodeficiency virus) positive. 49 (18.15) were having COPD (Chronic Obstructive Pulmonary Disease) as a comorbid condition. The overall in-hospital mortality rate was 19.63 % (53/270). The 28-day mortality rate was 7.78 % (21/270) and the ICU mortality rate was 9.63% (26/270). About 124 (45.93%) patients with AKI required dialysis in the ICU. **Conclusion**: Sepsis was the most common cause of AKI in the critically ill patients of our study. Age >60, male gender were prevalent in the majority of AKI patients. More than 60% of the patients had associated comorbidities, with type 2 diabetes, hypertension and coronary artery disease being the three most common. About, 60% of the total patients recovered normal renal function, with 2.4% of the total patients developing CKD. Crude mortality rate among patients with AKI in our study group was 37.04%.

KEYWORDS: ICUs, AKI, GFR, ARF, AKIN, RIFLE, AKI.

INTRODUCTION

Throughout the world in ICUs, acute kidney injury (AKI) is becoming increasingly found. Admitted kidney patient develop significant complications and require ongoing adjustments in care, as seen in these reader questions. Care of the critically ill patient is never static, and hospital clinicians are advised to keep abreast of the current literature and seek advice from other health professionals.

On the basis of Risk, Injury, Failure, Loss of kidney function, End-stage renal disease (RIFLE) criteria, the subsequent pediatric RIFLE (pRIFLE) score, and the Acute Kidney Injury Network (AKIN) criteria. Anatomically AKI is classified into three categories: pre-renal causes, renal causes and post renal causes Acute kidney injury (AKI) is an abrupt and usually reversible decline in glomerular filtration rate (GFR). This results in an increased level of serum blood urea, Blood urea nitrogen(BUN), creatinine and some other metabolic waste products that are normally excreted by the kidney. The term acute kidney injury, rather than acute renal failure (ARF), is increasingly used by the nephrology community to use to the acute kidney function loss. This

term also highlights that injury of the kidney that not means failure of kidney and is also of great clinical significance.

The initial diagnosis of AKI and management of the major complications of AKI are discussed in this research topic. The detection, incidence rate in ICU, causes, diagnosis, and prevention of AKI are presented separately. Among hospitalized patients, AKI is most commonly due to either pre-renal etiologies or acute tubular necrosis (ATN) from ischemia, nephrotoxin exposure, or sepsis.[2]

On the basis of these criteria, prevalence of AKI in ICU was>40%; if the sepsis is present and the mortality rates varied from 15-60%.[3,4] Longer hospital stay and economic burden are inevitable. In contrast to western literature, few reliable statistics are available regarding AKI in India.[5] We retrospectively evaluated patients with AKI, using the RIFLE criteria, to answer questions regarding most susceptible population, etiology, role of dialysis, outcomes and relation of mortality rate with RIFLE class.

Causes of AKI are frequently categorized as prerenal, intrinsic renal, and post-renal. This classification system oversimplifies the overlapping pathologic mechanisms underlying AKI. Renal parenchymal tissue hypo perfusion due to hypovolaemia or hypotension may initially cause a reversible increase in Cr. As cellular dysfunction continues, renal tubular cells may sustain ischemic injury which may persist after correction of the initial hypo perfusion state thereby changing from a pre-renal azotemia to acute tubular necrosis. Rachoin et al. demonstrated that ICU patient with AKI and blood urea nitrogen: Creatinine ratio greater than 20:1 had increased mortality suggesting that pre-renal azotemia is not benign in ICU patients.[6] Acute tubular necrosis (ATN) is the most common form of intrinsic renal failure in the ICU comprising as much as 88% of all cases of AKI. Only one study describes post-renal AKI.[7] Two centers with a combined capacity of over 850 beds only identified 54 patients over a 13-year period suggesting a low overall incidence. Many causes of AKI in ICU patients likely represent multifactorial etiologies.[8]

MATERIALS AND METHODS

The present study was conducted in the Dept. of Medicine at Govt. Doon Medical College Deharadhun in collaboration with CCL and ICU. AKI was classified according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria in three stages in this study we studied 270 patients in an ICU during a follow-up of period**.** Patients with less than 24-hour stay in the ICU and chronic kidney disease were excluded from study. Other exclusion criteria included age <18 years, previous dialysis, kidney transplantation, ARF from urinary tract obstruction and hypovolemia responsive to fluids. Pregnant patients were also excluded. Comparisons were performed by the Student-t tests. Comparisons of frequencies were measured by the Fisher test. Multivariate logistic regression was used to test variables as predictors for AKI and death. Informed consent obtained, the ICU chart was reviewed. For patients with extended ICU stays or prolonged dialysis requirements, data were collected for up to 10 weeks after the day of consultation.

OBSERVATIONS AND DISCUSSION

AKI is depends upon rate of increase of serum creatinine and decrease in urine output. The most of the patients of AKIs are due to prerenal causes like fluid volume deficit, sepsis and renal causes such as in acute tubular injury. The use of central venous and arterial blood pressure monitoring, urine analysis and microscopy allows assessment of fluid volume status and etiology of AKI. Data elements included: demographics, comorbid conditions, hospital and ICU admission and discharge data, blood and urine laboratory studies including microbiology, medication use, and the use of parenteral and enteral nutrition.

Diagnosis of AKI

The serum creatinine level increases by ≥ 0.35 mg/dl within 48 h or increase in serum creatinine to more than 1.5 times baseline within the previous 7 days. The urine volume contains < 0.5 ml/kg/h for 6 hours.

There were 270 patients enrolled in the study. Demographic data were obtained and comorbid conditions and the presumed etiology of AKI was noted.

Table. 1: Demographic characteristics.

		%
Mean age	65.4	
Male	168	62.22
Female	102	37.78

Mean age of the patients included in the study was 65.4 years. There were 168 (62.22%) male and 102 (37.78%) females.

Table 2: Associated Co morbid conditions.

Comorbid conditions	Number	% (n=270)
Hypertension	146	54.07
Diabetes	164	60.74
Liver disease	24	8.89
Coronary heart disease	41	15.19
HIV positive	6	2.22
COPD	49	18.15

In 146 (54.07%) hypertension was the associated co-morbid condition. Diabetes was present in 164(60.74%) of the cases. 24 (8.89%) were having associated liver disease. 6 (2.22%0 were HIV (Human Immunodeficiency virus) positive. 49 (18.15) were having COPD (Chronic Obstructive Pulmonary Disease) as a comorbid condition.

Table 3: Etiological causes of AKI.

Cause	Number	5 (n=270)
Sepsis	84	31.11
Acute tubular necrosis	56	20.74
Hypotension	54	20.00
Renal artery thrombosis	48	17.78
Cardiogenic shock	16	5.93
AKI with no observable cause	12	4.44
	270	100.00

The most common cause of AKI in our study was sepsis (84, 31.11%) followed by Acute tubular necrosis (56, 20.74%). AKI due to hypotension was 54(20%). AKI due to renal artery thrombosis and cardiogenic shock was 48(17.78%) and 16 (5.93%) respectively. No etiological cause was identified in 12 (4.44%) cases in our study.

Table No. 4. Showing Mortality rate in AKI.

N=270	Number	%
Mortality rate	53	19.63
28 days mortality	21	7.78
ICU mortality	26	9.63

The overall in-hospital mortality rate was 19.63% (53/270). The 28-day mortality rate was 7.78% (21/270) and the ICU mortality rate was 9.63% (26/270). About 124 (45.93%) patients with AKI required dialysis in the ICU.

Table No. 5. etiological causes of AKI.

Cause	Number	%
Sepsis	84	31.11
Acute tubular necrosis	56	20.74
Hypotension	54	20.00
Renal artery thrombosis	48	17.78
Cardiogenic shock	16	5.93
AKI with no observable cause	12	4.44
Total	270	100.00

AKI has been considered as a self-limiting disease, with a good prognosis when recovery is noted in hospital.[9] AKI is a major contributor influencing outcomes in critically ill patients. Some studies have shown the epidemiology of AKI in the ICU. These have ranged from descriptions of administrative data sets, retrospective analysis of single- and multicenter cohorts and prospective cohort studies.[10]

In our study major etiological cause was sepsis (84, 31.11%). Sepsis remains the leading cause of death in ICUs. There are adjunctive treatments having the same objective to remove cytokines and other inflammatory or anti-inflammatory mediators to control the evolution of sepsis. "Peak concentration hypothesis" given by Ronco was removal of deleterious peak of mediators secreted during sepsis by blood purification may control host response and avoid organ dysfunction and Honoré thought that hemo filtration is also able to remove mediators from tissue in the he called as "threshold modulation theory.".[11] in our study the overall mortality rate was 53 (19.63%) which was lower than the previous studies in developed and emerging countries.[12,13] The reason may be because some patients from India and China may not have been able to afford dialysis, which could have affected their mortality rates.[14]

In a study percentage of AKI due to sepsis was 52.1 in developing countries and 44.3 in developed countries etiological cause and in our study it was 31.11%. In the same study AKI due to cardiogenic cause was 18.7 in developing countries and 20.4 in developed countries cardiogenic cause and in our study it was 5.93% which was quite less.[14]

Causes of AKI differed, with more glomerulonephritis and acute interstitial nephritis (AIN) in emerging countries and more prerenal AKI, sepsis, and acute tubular necrosis (ATN) in developed countries. In our series sepsis was the main cause followed by acute tubular necrosis and hypotension.[14]

Metcalfe et al. found that 44% required dialysis as a result of chronic renal failure, 36% as a result of AKI, and 20% as a result of acute and chronic renal failure in our study 124 (45.93%) patients required dialysis which was quite high.

It is estimated by the World Health Organization (WHO) in 2014 that 387 million people suffer from Diabetes mellitus (DM), where 90% of the cases are of Type II diabetes.[15] The risk of AKI has been considered to be increase in patients with DM, with an adjusted odds ratio of 1.99, compared to non-DM controls with the same GFR.[16] The higher risk of AKI in patients with DM is the frequent occurrence of complications associated with DM. Some of these complications include cardiovascular disease; heart failure, exposure to medications such as diuretics and others that serve as nephrotoxic agents.[17] In our study 60.74% of the cases with AKI were associated with DM as a comorbitiy. Other co-morbidities associated with the development of AKI including are vascular disease, hypertension and pre-morbid chronic renal disease. In our study hypertension was a comorbid condition was 54.07%.

In this study 2.2% cases were HIV positive with AKI. HIV-associated AKI is mostly due to HIV-mediated viral and immunological disease and or nephrotoxicity from treatments of the same.[18] It also includes low $CD4^+$ levels, AIDS, hepatitis C and liver disease.[19]

CONCLUSION

Sepsis was the most common cause of AKI in the critically ill patients of our study. Age >60, male gender were prevalent in the majority of AKI patients. Avoidance of fluid volume deficit and blood pressure support reduces the incidence of AKI in serious ill

patients. More than 60% of the patients had associated comorbidities, with type 2 diabetes, hypertension and coronary artery disease being the three most common. About, 60% of the total patients recovered normal renal function, with 2.4% of the total patients developing CKD. Crude mortality rate among patients with AKI in our study group was 37.04%.

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