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ORIGINAL RESEARCH

Assessment of cases of acute kidney injury

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Abstract

Background: Acute Kidney Injury (AKI) is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function). The present study was conducted to determine the cases of AKI.

Materials & Methods: 86 patients of AKI of both genders were included and comorbid conditions, laboratory findings and outcome of AKI was recorded.

Results: Out of 86 patients, males were 50 and females were 36. The mean serum urea level in patients was 142.6 mg/dl, serum creatinine was 5.8 mg/dl, serum sodium was 134.4 meq/dl and serum potassium was 4.7 meq/dl. Common comorbidities observed was chronic obstructive pulmonary disease in 21, coronary artery disease in 42, cerebrovascular disease in 18, diabetes in 56 and hypertension in 43. The difference was significant ($P< 0.05$). Out of 86 patients, 65 survived and 21 died. The difference was significant ($P< 0.05$).

Conclusion: Common comorbidities seen among AKI patients were diabetes, hypertension, CAD, cerebrovascular disease and chronic obstructive pulmonary disease.

Key words: AKI, comorbidities, chronic obstructive pulmonary disease

Introduction

Acute Kidney Injury (AKI) is defined as an abrupt (within hours) decrease in kidney function, which encompasses both injury (structural damage) and impairment (loss of function).[1] It is a syndrome that rarely has a sole and distinct pathophysiology.[3] Many patients with AKI have a mixed etiology where the presence of sepsis, ischaemia and nephrotoxicity often co-exist and complicate recognition and treatment.[2] Furthermore the syndrome is quite common among patients without critical illness and it is essential that health care professionals, particularly those without specialization in renal disorders, detect it easily.[3]

According to AKI network, the most current consensus diagnostic criteria for AKI is an abrupt (within 48 h) reduction in kidney function currently defined as an absolute increase in serum creatinine of more than or equal to 0.3 mg/dl, a % increase in serum creatinine of 50% (1.5-fold from baseline), or a reduction in UO (documented oliguria of 0.5 ml/kg/hr for 6 hrs).[4]

The pathophysiology of AKI is multifactorial and complex. The most common cause of AKI is ischaemia, which can occur for a number of reasons. Physiological adaptations, in response to the reduction in blood flow can compensate to a certain degree, but when delivery of oxygen and metabolic substrates becomes inadequate, the resulting cellular injury leads to

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organ dysfunction.[5] The kidney is highly susceptible to injury related to ischaemia, resulting in vasoconstriction, endothelial injury, and activation inflammatory processes.[6] The present study was conducted to determine the cases of AKI.

Materials & Methods

The present study comprised of 86 patients of AKI of both genders. All were informed regarding the study and written consent was obtained.

Data such as name, age, gender etc. was recorded. A thorough clinical examination was done in all patients. Severities of illness and survival prediction were assessed using acute physiology and chronic health evaluation (APACHE)-IV score. Comorbid conditions, laboratory findings and outcome of AKI was recorded. Results thus obtained were subjected to statistical analysis. P value less than 0.05 was considered significant.

Results

Table I Distribution of patients

Total- 86		
Gender	**Males**	**Females**
Number	50	36

Table I shows that out of 86 patients, males were 50 and females were 36.

Table II Assessment of laboratory findings

Parameters	Mean	SD
Serum urea (mg/dl)	142.6	36.2
Serum creatinine (mg/dl)	5.8	1.7
Serum sodium (meq/dl)	134.4	12.6
Serum potassium (meq/dl)	4.7	2.1

Table III shows that mean serum urea level in patients was 142.6 mg/dl, serum creatinine was 5.8 mg/dl, serum sodium was 134.4 meq/dl and serum potassium was 4.7 meq/dl.

Table III Presence of comorbidities

Comorbidities	Number	P value
Chronic obstructive pulmonary disease	21	0.05
Coronary artery disease	42	
Cerebrovascular disease	18	
Diabetes	56	
Hypertension	43	

Table III, graph I shows that common comorbidities observed was chronic obstructive pulmonary disease in 21, coronary artery disease in 42, cerebrovascular disease in 18, diabetes in 56 and hypertension in 43. The difference was significant ($P< 0.05$).

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Graph I Presence of comorbidities

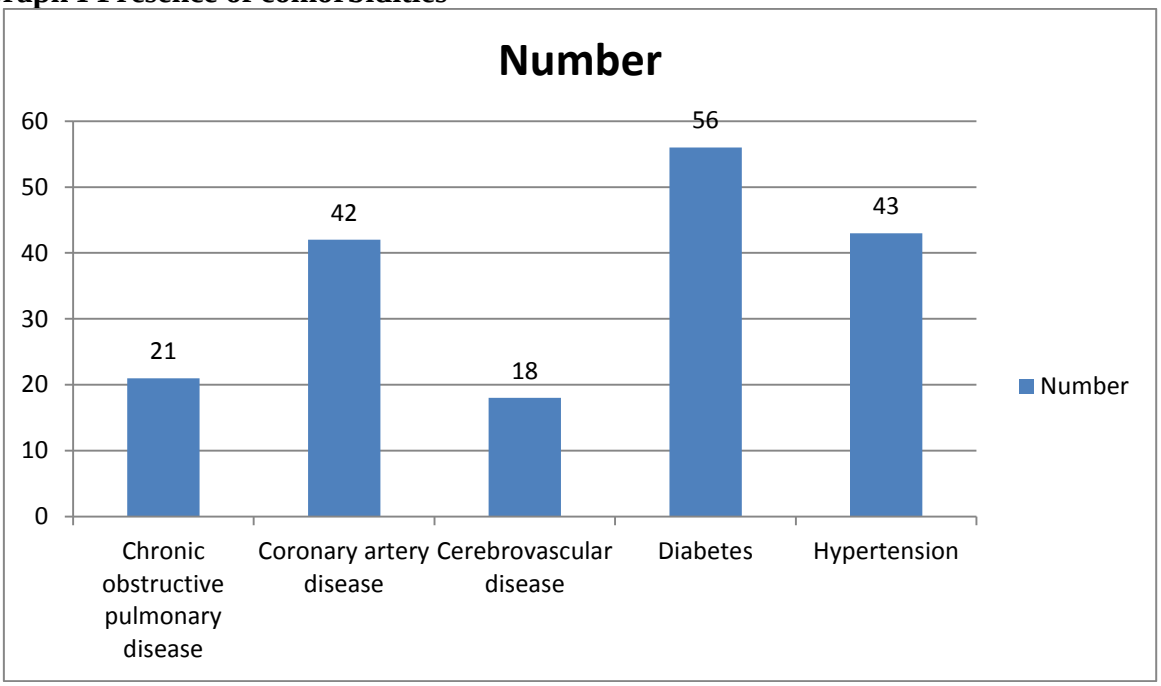

Table IV Outcome of AKI

Outcome	Number	P value
Survival	65	0.01
Death	21	

Table IV shows that out of 86 patients, 65 survived and 21 died. The difference was significant (P< 0.05).

Discussion

Acute kidney injury (AKI) is characterized by a rapid decline glomerular filtration rate (GFR) and retention of nitrogenous waste products such as blood urea nitrogen (BUN) and creatinine.[7] For the purpose of diagnosis and management AKI is divided into three categories: Prerenal, intrarenal and postrenal AKI.[8,9] Recently a new definition of AKI has been widely accepted which is referred to by the acronym RIFLE. AKI in the setting of intensive care unit (ICU) has attracted number of publications for the past two decades.[10,11]

We found that Out of 86 patients, males were 50 and females were 36. Anderson et al[12] included 422 AKI and acute on chronic kidney disease patients. The mean age was 65.8 ± 14.1. Majority of patients were male (58.2%) of Chinese ethnicity (68.8%). One hundred and thirty-two patients (32.6%) were diagnosed in acute care units. Seventy-five percent of patients developed AKI during admission in a non-Renal specialty. Mean baseline eGFR was 50.2 ± 27.7 mL/min. Mean creatinine at AKI diagnosis was 297 ± 161 µmol/L. Renal consultations were initiated at KDIGO Stages 1, 2 and 3 in 58.9, 24.5 and 16.6% of patients, respectively. Three hundred and ten (76.7%) patients had a single etiology of AKI with the 3 most common etiologies of AKI being pre-renal (27.7%), sepsis-associated (25.5%) and ischemic acute tubular necrosis (15.3%). One hundred and nine (27%) patients received acute renal replacement therapy. In-hospital mortality was 20.3%. Six-month mortality post-AKI event was 9.4%. On survival analysis, patients with KDIGO Stage 3 AKI had significantly shorter survival than other stages.

We observed that the mean serum urea level in patients was 142.6 mg/dl, serum creatinine was 5.8 mg/dl, serum sodium was 134.4 meq/dl and serum potassium was 4.7 meq/dl. Uchino S et al[13] determined the period prevalence of ARF in intensive care unit (ICU) patients. Of 29

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269 critically ill patients admitted during the study period, 1738 (5.7%; 95% confidence interval [CI], 5.5%-6.0%) had ARF during their ICU stay, including 1260 who were treated with RRT. The most common contributing factor to ARF was septic shock (47.5%; 95% CI, 45.2%-49.5%). Approximately 30% of patients had preadmission renal dysfunction. Overall hospital mortality was 60.3% (95% CI, 58.0%-62.6%). Dialysis dependence at hospital discharge was 13.8% (95% CI, 11.2%-16.3%) for survivors. Independent risk factors for hospital mortality included use of vasopressors (odds ratio [OR], 1.95; 95% CI, 1.50-2.55; P<.001), mechanical ventilation (OR, 2.11; 95% CI, 1.58-2.82; P<.001), septic shock (OR, 1.36; 95% CI, 1.03-1.79; P = .03), cardiogenic shock (OR, 1.41; 95% CI, 1.05-1.90; P = .02), and hepatorenal syndrome.

We found that common comorbidities observed was chronic obstructive pulmonary disease in 21, coronary artery disease in 42, cerebrovascular disease in 18, diabetes in 56 and hypertension in 43. The difference was significant (P< 0.05). Out of 86 patients, 65 survived and 21 died. Mehta et al[14] found that the mean age was 59.5 years, 41% were women, and 20% were of minority race or ethnicity. There was extensive comorbidity; 30% had chronic kidney disease, 37% had coronary artery disease, 29% had diabetes mellitus, and 21% had chronic liver disease. Acute renal failure was accompanied by extrarenal organ system failure in most patients, even those who did not require dialysis. Three hundred and ninety-eight (64%) patients required dialysis. The in-hospital mortality rate was 37%, and the rate of mortality or non recovery of renal function was 50%. The median hospital length of stay was 25 days (26 days, excluding patients who died).

The shortcoming of the study is small sample size.

Conclusion

Authors found common comorbidities seen among AKI patients were diabetes, hypertension, CAD, cerebrovascular disease and chronic obstructive pulmonary disease.

References

1. Gaião S, Cruz DN. Baseline creatinine to define acute kidney injury: is there any consensus? Nephrol Dial Transplant. 2010;25:3812–4.
2. Clarkson MR, Friedwald JJ, Eustace JA, Rabib H. Acute Kidney Injury. In Brenner and Rector's The Kidney 8th ed. Philadelphia, USA: Saunders Press; 2004. p. 943-86.
3. Nash K, Hafeez A, Hou S. Hospital-acquired renal insufficiency. Am J Kidney Dis 2002;39:930-6.
4. Katagiri D, Doi K, Honda K, Negishi K, Fujita T, Hisagi M, et al. Combination of two urinary biomarkers predicts acute kidney injury after adult cardiac surgery. Ann Thorac Surg. 2012;93:577–83.
5. Eknoyan G, Agodoa L. On improving outcomes and quality of dialysis care, and more. Am J Kidney Dis. 2002;39:889–91.
6. Ostermann M, Chang RW. Acute kidney injury in the intensive care unit according to RIFLE. Crit Care Med 2007;35:1837-43.
7. Cerda J, Lameire N, Eggers P, Pannu N, Uchino S, Wang H, et al. Epidemiology of Acute kidney injury. Clin J Am Soc Nephrol. 2008;3(3):881–6.
8. Musso CG, Reynaldi J, Martinez B, Pierángelo A, Vilas M, Algranati L. Renal reserve in the oldest old. Int Urol Nephrol. 2011;43(1):253–6.
9. Waikar SS, Betensky RA, Emerson SC, Bonventre JV. Imperfect gold standards for kidney injury biomarker evaluation. J Am Soc Nephrol. 2012;23:13–21.
10. Nakamura M, Seki G, Iwadoh K, Nakajima I, Fuchinoue S, Fujita T, et al. Acute kidney injury as defined by the RIFLE criteria is a risk factor for kidney transplant graft failure. Clin Transpl. 2012;26(4):520–8.

ISSN: 0975-3583,0976-2833 VOL13, ISSUE 08, 2022

11. Lameire NH, Bagga A, Cruz D, De Maeseneer J, Endre Z, Kellum JA, et al. Acute kidney injury: an increasing global concern. Lancet. 2013;382:170–9.
12. Anderson S, Eldadah B, Halter JB, Hazzard WR, Himmelfarb J, Horne FM, et al. Acute kidney injury in older adults. J Am Soc Nephrol. 2011;22(1):28–38.
13. Uchino S, Kellum JA, Bellomo R, Doig GS, Morimatsu H, Morgera S, et al. Acute renal failure in critically ill patients: A multinational, multicenter study. JAMA 2005;294:813-8.
14. Mehta RL, Pascual MT, Soroko S, Savage BR, Himmelfarb J, Ikizler TA, et al. Spectrum of acute renal failure in the intensive care unit: The PICARD experience. Kidney Int 2004; 66:1613-21.