Section: Community Medicine

Original Research Article

A STUDY ON ETIOLOGY AND CLINICAL OUTCOME OF TROPICAL ACUTE KIDNEY INJURY (AKI) IN A TERTIARY CARE HOSPITAL

Vemula Rushendra Kumari[1], C.V. Subbarayudu[2], K. Naga Jyothi[3], C. Sangeetha[4], C.M. Prabhas Naidu[5]

[1]Associate Professor, Department of General Medicine, Tirupati, S.V. Medical College, Tirupati, Andhra Pradesh. India.
[2]Assistant Professor, Department of Community Medicine, S.V. Medical College, Tirupati, Andhra Pradesh. Corresponding author
[3]Assistant Professor, Department of Nephrology, Andhra Medical College, Visakapatnam Andhra Pradesh
[4]Senior Resident, Department of General Medicine, S.V. Medical College, Tirupati, Andhra Pradesh
[5]Student, Narayana Medical College, Nellore, Andhra Pradesh

Received : 18/05/2023
Received in revised form : 22/06/2023
Accepted : 04/07/2023

Keywords:
RRT, Tropical Diseases, AKI

Corresponding Author:
Dr. C. Venkata Subbarayudu
Email: rushendravemula@gmail.com

DOI: 10.47009/jamp.2023.5.4.87

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5(4); 436-441

Abstract

Background: Acute kidney injury(AKI) is linked with considerable mortality and morbidity in India. It is a spectrum ranging from a mild form to severe form requiring Renal Replacement Therapy (RRT). Detection of AKI in the early stage of disease and aggressive management of underlying cause will reduce mortality. If treated partially or left untreated, it was observed to increase risk of developing CKD and progression to End-Stage Renal Disease (ESRD)[4]. Patients in tropical countries continue to face diseases that are due to public health problems such as unsafe water, poor sanitation, lack of infection control, and out-of-hospital childbirth[6]. Potential use of indigenous Indian remedies also seemed to increase the risk of AKI in tropical and developing countries. The aim is to study the etiology and clinical outcome of tropical Acute Kidney Injury (AKI) in a tertiary care hospital. **Materials and Methods:** This prospective observational study was conducted in general medical wards and acute medical care unit (AMC) in SVRRGGH, Tirupati. The study was conducted for a period of 1 year (From February 2020 to January 2021) on 160 subjects. Urine output was measured every 6 hours on day 1 of hospital stay and later for every 24 hours. Urine output for patients in the ICU was measured every 6 hours. Oliguria, hypotension, encephalopathy, acute respiratory distress syndrome (ARDS), sepsis, the requirement for mechanical ventilation and ionotropic support were noted. Serial monitoring of serum creatinine and urine output was individualized for each patient. The patients of AKI were managed with fluid resuscitation, inotropic support, antibiotic therapy and treating the underlying cause. Renal replacement therapy, if required was initiated after consultation with a nephrologist. Each patient was followed up for 3months with serum creatinine. **Result:** Of the total 160 patients of AKI, 104 (65%) were males and 56 (35%) were females. Causes of AKI included in the study were acute diarrheal disease, toxins, glomerulonephritis, sepsis, leptospirosis, malaria, scrub typhus, and dengue, CCF, HRS and BPH. According to AKIN staging, 42.5% of patients were stage 1, 16.3% were stage 2 and 41.3% were AKIN stage 3. Among the causes, Renal AKI (76.3%) was the commonest cause of AKI followed by prerenal (18.7%) and post-renal (5%) AKI. In the study, 121(75.6%) patients were treated conservatively and 39(24.4%) patients underwent renal replacement therapy. 105 (65.6%) patients of AKI completely recovered, 43 (26.9%) patients died and 12 (7.5%) patients progressed to chronic kidney disease. **Conclusion:** Sepsis was the commonest cause of AKI in this study. Risk stratification and early diagnosis & management were the best time-tested approach in treating AKI. Most of the infectious and environmental causes were largely preventable which were associated with significant morbidity and mortality.

INTRODUCTION

Acute kidney injury (AKI) refers to an abrupt decline in renal function over a period and results in decreased urine output and clearance of metabolic waste products, electrolyte imbalance, and dysregulation of acid-base homeostasis.[1] AKI is linked with considerable mortality and morbidity. AKI is a spectrum extending from a mild form to severe form which requires Renal Replacement Therapy (RRT).[2] Detection of AKI in the early stage of disease and aggressive management of underlying cause will reduce mortality.[3] Recently it was observed that the patients who had a complete or near-complete recovery, were at risk of developing CKD and increased risk of progression to End-Stage Renal Disease (ESRD).[4]The incidence of Acute kidney injury differs between the developing and developed countries.[5] Patients in tropical countries continue to face diseases that are due to public health problems such as unsafe water, poor sanitation, lack of infection control, and out-of-hospital childbirth.[6] High temperature, the fragilityof soil, and the leaching of minerals & organic compounds result in the high prevalence of water-borne diseases. Heterogeneity can be seen in the population of the tropics than in temperate climates in terms of socioeconomic status, lack of education, and access to services and medical care.[7] Poverty acts as a risk factor in the population who encounter the industrial toxins that can cause AKI. Community-acquired AKI (CA-AKI) is more common than hospital-acquired AKI (HA-AKI) among developing nations.[8] However, within the Indian subcontinent, different etiologies are reported from various centres which are geographically distinct and also demonstrated changes in the etiological spectrum over a period. The potential use of indigenous tropical remedies results in the development of AKI. From 1983 – 2008, the incidence of surgical, diarrheal, and obstetrical AKI decreased significantly whereas AKI associated with sepsis, malaria, liver disease, and nephrotoxic drugs increased.[9] In some studies, comorbidities like Diabetes Mellitus (DM), hypertension, cardiac disease were associated with the risk of developing AKI.[10] Recent studies in the Indian population revealed nephrotoxic drugs as the most cause of AKI among medical cases and sepsis among surgical cases and in ICU. To determine AKI, volume status, urine output, altered serum creatinine values, and proteinuria are crucial parameters.[11] The main key for treating AKI is assuring adequate renal perfusion and the therapy related to the severity, individualized to a patient. AKI is usually treated with adequate hydration and RRT,[12] along with treatment of complications (such as metabolic acidosis, hyperkalemia) associated with AKI.[13] AKI as a risk factor for CKD and ESRD is reported in long-term studies. According to the frequency and mortality rates of AKI patients, prevention by 20% had shown to reduce the number of complications, deaths, and associated costs.[14] The purpose of this study was to evaluate various etiologies and their outcomes regarding age, sex, and their clinical & laboratory profile.

MATERIALSANDMETHODS

Study Design and Setting

This prospective observational study was conducted from February 2020 to January 2021, in the department of General Medicine of S V R R G General Hospital, Tirupati, Andhra Pradesh, India.Total 160 patients with AKI were included in the study. The study was approved by the institutional Scientific and Ethics Committee at Sri Venkateswara Medical College, Tirupati dated 01/02/2020, Lr.no.19/2020. Written informed consent was taken from all the participants at the time of enrollment.

Inclusion Criteria

Patients with age more than 18 years with features of AKI such as Increase in serum creatinine of 0.3 mg/dl or more within 48 hours of observation, increase in serum creatinine of 1.5 times from baseline or greater, which was known or presumed to occur within 7 days or a reduction in urine volume below 0.5 ml/kg/h for 6 hours.

Exclusion Criteria

Patients with Chronic Kidney disease according to KIDGO criteria and pregnant females were excluded from the study.

Study Procedure

The study included patients admitted in medical wards, AMC, RICU through OPD and emergency. Detailed history and clinical examination was performed and records were checked to confirm the presence of comorbidities such as Diabetes, Hypertension, Coronary Artery disease and other illnesses. Complete hemogram, Serum creatinine, serum electrolytes, liver function tests were done. During the hospital stay, patients were followed up with daily clinical examinations. Urine output was measured every 6 hours on day 1 of hospital stay and later for every 24 hours. Urine output for patients in the ICU was measured every 6 hours. Oliguria, hypotension, encephalopathy, acute respiratory distress syndrome (ARDS), sepsis, the requirement for mechanical ventilation and ionotropic support were noted. Workup was individualized for each patient to achieve a definitive diagnosis. Serial monitoring of serum creatinine and urine output was individualized for each patient. The maximum serum creatinine and maximum stage of AKI for each patient were noted accordingly. The patients of AKI were managed with fluid resuscitation, inotropic support, antibiotic therapy and treating the underlying cause. Renal replacement therapy if required was initiated after consultation with a nephrologist. Each patient was followed up for 3months with serum creatinine.

ISSN(O): 2687-5365; ISSN(P): 2753-6556

Complete renal recovery was defined as the return of serum creatinine to within 0.5 mg/dL baseline creatinine (ATN trail).

Hypotension was defined as the systolic BP<90 mm Hg or a requirement of vasopressors to maintain a mean arterial pressure of 65 mm Hg.Oliguria was defined as urine output <0,5 ml / Kg/hr for more than 6 hrs and anuria as urine output <100 ml for 12 hrs.Hypertension was defined as the systolic BP >150 mmHg and/or diastolic BP >90 mmHg .

Hypernatremia and Hyponatremia were defined as serum sodium level> 145 meq/L and <135 meq/L respectively. Hyperkalemia and Hypokalemia were defined as serum potassium levels> 5.5 meq/L and <3.5 meq/L, respectively. Metabolic acidosis was defined as pH < 7.3 and bicarbonate<20 meq/dl

Serum creatinine at most recent admission (7days to 3months) was considered as baseline.

Stage 1- Increase in serum creatinine by 0.3 mg/dL (within 24hrs) or increase of 150–200% from baseline (known or presumed to have occurred within 7days).Decreased urine output by <0.5 mL/kg/h for6 hrs

Stage 2 - Increase in serum creatinine by >200-300% from baseline, Decreased urine output by<0.5 mL/kg/h for 12 hrs

Stage 3 - Increase in serum creatinine by >300% from baseline or ≥4 mg/dL with an acute increase of ≥0.5 mg/dL or requirement of RRT, Decreased urine output by< 0.3 mL/kg/h for 24 h or anuria for >24hr.

The duration of AKI is defined as the number of days from the first day when the patient met the AKI criteria and until they no longer did.

Analysis

In the present study, SPSS version 21 was used for statistical analysis. Descriptive statistics, such as percentage, frequency, mean, median, and standard deviation were used to summarize patient's baseline clinical characteristics. The Mean and standard deviations were used for continuous variables and Frequencies and proportions were used for categorical data, while the Crosstabs were used for the comparison of proportions of categorical variables. Statistical significance was considered at the p-value of less than 0.05 on the chi-square test.

RESULTS

Among 160 participants, 104 (65%) patients were males and 56 (35%) patients were females. Gender The Mean ± SD of the age of the study population was 47± 16. In the present study, the majority of the patients were between 51 and 60 years of age (23.1%) followed by the age group 41-50 years (21.8%). Only 5.6% of the study population belonged to older age more than 70 years. The minimum and maximum age of the study population was 18 and 78 respectively.

Acute Gastroenteritis, Cardiac illness, Sepsis, Dengue, Malaria, Leptospirosis, Scrub typhus, Hepatorenal syndrome (HRS), Toxins, Glomerulonephritis and Bladder outlet obstruction were the causes of AKI observed in this study. Sepsis (34.6%) was the leading cause of AKI in the present study. Among sepsis, urinary tract infection (17.5%) was the most common cause followed by pneumonia (6.9%). Toxins (23.1%) were the second cause of AKI. Toxins such as plant, chemical, drug and biological toxins were observed to be causative in the study. Hypovolemia due to diarrheal disease was the cause of AKI in 9.4% of patients. Dengue, Malaria, scrub typhus and leptospirosis were seen in 3.8%, 3.8%, 3.1% and 5.6% respectively. Among all patients, 2.5% had AKI secondary to glomerulonephritis. Obstructive uropathy secondary to carcinoma cervix, renal or ureteric calculi and benign prostatic hypertrophy were observed in this study. It accounted for 4.4% of patients.

Among the study population, comorbidities seen were Diabetes mellitus (27.5%), hypertension (15.6%), coronary artery disease (CAD) (8.8%) and chronic obstructive pulmonary disease (COPD) (3.75%).Renal AKI (76.3%) was the common cause of AKI followed by prerenal (18.7%) and post-renal (5%) AKI. Out of 160 patients, 121(75.6%) patients were treated conservatively and 39 (24.4%) patients underwent renal replacement therapy.

In the present study, complete recovery was seen in 105 (65.6%) patients and 43 (26.9%) patients died and 12 (7.5%) patients progressed to chronic kidney disease.Most of the patients (83.75%) required hospitalization for around 2 weeks during which the majority of patients recovered. About 16.25% of patients required hospitalization for >3 weeks.

Table 1: Comparison of AKIN STAGING results between survival and non-survival groups

AKIN	RECOVERY (n)	DIED (n)	CKD (n)
STAGE 1	59	2	7
STAGE 2	24	2	0
STAGE 3	22	39	5
Pearson Chi-square test- 64.139			
Significance -<0.001			

The study population with AKIN stage 3 was associated with high mortality compared to stages 2 and 3. AKIN staging showed a significant association with outcome.

ISSN(O): 2687-5365; ISSN(P): 2753-6556

Table 2: Comparison of ETIOLOGY AND OUTCOME results between survival and non-survival groups

ETIOLOGY		RECOVERY(n)	DIED(n)	CKD(n)
ACUTE GE		12	2	1
CCF		10	1	0
SEPSIS	PNEUMONIA	3	8	0
	UTI	20	5	3
	LIVER ABSCESS	0	2	0
	MENINGITIS	3	0	0
	LOCAL INFECTION	2	1	0
	CELLULITIS	0	3	3
	DIABETIC FOOT	0	1	1
DENGUE		6	0	0
MALARIA		5	1	0
LEPTOSPIROSIS		5	0	0
SCRUB TYPHUS		9	0	0
HRS		2	3	0
TOXINS		20	14	3
AGE		4	0	0
BPH/BOO		4	2	1
Pearson Chi-square test- 76. 860				
SIGNIFICANCE - <0.001				

DISCUSSION

AKI is a potentially fatal but reversible renal disease. The etiology, clinical course and outcome differ in various parts of the world and also within India because of geographic and climatic diversity and varying standards of medical care in India. The mean age of the present study was 47 years which correlates with the studies done by Vikrant S et al study,[15] (49 years) and Khan et al study,[16] (48.1). In other studies, by Bernieh B et al,[17] and Ravindra L et al,[18] the mean age was a little higher compared to the present study i.e., 56.2 years and 59.5 years respectively.

In this study, 67.5% were males and 32.5 were females. The male to female ratio is 1.8:1. Bernieh B et al,[17] Ravindra et al,[18] and Vikrant S et al,[15] studies also reported more or less equal distribution of both males and females with slightly more males, whereas a study by Rekha et al,[19]was comparable to the present study. The higher percentage of males may be due to occupational hazards among males that predisposes them to vectorborne diseases and envenomation.

Among various etiologies of AKI, sepsis has topped the list with 34.6% of patients followed by toxins (21.1%). Sepsis pertained to be the common cause of AKI and it correlates with other studies. Bagshaw SM et al., in their prospective observational study, conducted on critically ill patients at 54 hospitals in 23 countries reported that sepsis was the leading aetiology for AKI in 47.5% of patients.[20] UTI was another major cause of sepsis-associated AKI in this study accounted for 17.5% of cases similar to Vikrant S et al study (8%).[15]

In a study from Yemen, only 0.5% of patients with AKI accounts for pyelonephritis.[21] Pyelonephritis was the predominant cause of UTI in this study. Urine cultures were positive in 16 cases, in which E. coli was isolated predominantly. It showed comparable results with Kaaviya R et al study.[22]Urosepsis may cause sudden deterioration in renal function.

Next common cause of sepsis in the present study was pneumonia (6.9%). Around 34% of patients with community-acquired pneumonia (CAP) had AKI and also reported that AKI was common in non-severe categories of CAP in the Murugan et al study.[23] A significant proportion of patients who were in sepsis showed higher mortality than patients without sepsis. It was observed that 2(1.3%) patients had liver abscesses, 3(1.9%) patients had meningitis and 2(1.3%) patients had a diabetic foot. In India, diarrheal disease-related AKI ranged from 20.6% to 30.5%. Only 9.4% of patients had diarrheal diseases in this study which correlates with Vikrant S et al study (6.5%) but was lower than Prakash S et al (18%),[24] and Bhattacharya et al studies (17.3%).[25] A significant decline in acute diarrheal disease-related AKI has been also reported in India by Prakash S et al study.[24] Due to better hygienic habits, good facilities and effective management at the level of primary and secondary care hospitals, acute diarrheal diseases have been decreasing. In this study, 6(3.8%) patients were presented with Dengue that was slightly higher than Mathew et al study (2%).[26] A recent study from India reported AKI in 10.8% of patients with dengue.[27] Leptospirosis, a zoonotic disease was responsible for AKI in 18.5% of patients in the Vikrant et al,[15] study and it was higher than the present study (5.6%). The majority of patients with leptospirosis were non-oliguric. Scrub typhus is an important cause of acute undifferentiated febrile illness in the Indian subcontinent and is considered a differential diagnosis for acute febrile illness with AKI. AKI related to scrub typhus ranges from 20% to up to 60%.

AKI is considered as resultant of multi-organ dysfunction. Toxins were observed as a cause of AKI in 23.1%of patients. It included snake bite (10.6%), nephrotoxic drugs (1.78%), herbal medications (0.62%) and poisonous chemicals (10.6%). Snakebite is considered an important

ISSN(O): 2687-5365; ISSN(P): 2753-6556

cause of AKI in tropical countries. The mortality related to snake bite-induced AKI ranges from 1% to 20%.[28] The previous studies from India reported that the incidence of snake bite-related AKI was comparatively less. For example, in a study by Muthusethupathi et al. in Chennai,[29] only 6 out of 187 patients had AKI due to snakebite. Patients with snake bite-related AKI were slightly more (8.7%) in the present study and was consistent with Vikrant S et al (6.1%) study. Early administration of anti-snake venom (ASV) was a vital measure. Treatment for established AKI is largely supportive and RRT is considered as the cornerstone. Among toxins, 6.1% of patients developed AKI secondary to nephrotoxic drugs. Drug-induced AKI accounts for 20% of all AKI in an Indian study (148). Patients with drug-induced AKI were comparatively more in Prakash S et al,[24] (4%)and Vikrant S et al,[15] (5.3%) studies than in the present study. Among toxins, chemicals involved in AKI were organophosphorus poisoning (8.1%), rodenticide (8.1%), carbamate (2.7%), supervasmol (8.1%) and paraquat poisonings (16.2%). Herbal medicine induced AKI accounts for 0.6% in the present study. The results of glomerulonephritis (2.5%) were consistent with the Vikrant S et al,[15] study (1.9%) but lower than Prakash S et al (6%),[24] and Bhattacharya et al studies (5.3%).[25] It was observed that Obstructive uropathy was secondary to renal or uretericcalculi, carcinoma cervix, and prostatic enlargement. It was responsible for AKI in 4.4% of patients compared to Prakash S et al study (4%),[24] whereas it accounted for 6.6% in the Vikrant S et al study.[15]

Clinical and laboratory characteristics: Oliguria was the most common clinical feature in 78 (48.8%) patients with AKI and correlates with Prakash S et al (45.3%),[24] and Vikrant S et al studies (45.3).[15] Patients with oliguria were less in number in Bhattacharya et al (25.8),[25] and Shafeeq Usman V et al (36.6%) studies[30] when compared to other features. It was found out oliguric AKI had a far worse prognosis than non- oliguric AKI.[31] Out of all, 20.6% of patients were presented with encephalopathy and it was similar to other studies except Vikrant S et al (9%),[15]and Bhattacharya et al (5.33%),[25]studies.

It was observed that out of 31 patients with hypotension, 15 succumbed to illness in the present study. However, the results of hypotension were inconsistent with other studies. Hyperkalemia was the major electrolyte abnormality in 50% of the patients in the Prakash S et al study,[24] and three times more when compared to the present study (13.1%).

Multi-organ dysfunction occurred in 16.3% of patients in the present study which did not correlate with Vikrant S et al study (38.2%).[15] The mortality was 65.3% in patients with MODS in this study.Metabolic acidosis accounted for 14.3% mortality which was less than in other studies.

The mean ± SD of Urea was 71.5 ± 14.2 in this study which was lower than in Vikrant S et al study (143 ± 68).[15] The mean ± SD of serum creatinine was 3.54 ± 2.13 which was similar to other studies. In a study by Bhattacharya et al study (140), mean creatinine was 2.37 ± 0.90 mg/dl and mean ± SD of Serum urea was 92.44 ± 39.67 mg/dl which was inconsistent with the present study.Daily serum urea and serum creatinine were found to be excellent tools to monitor and predict the outcome of AKI in this study. Similarly worsening serum urea and serum creatinine value despite optimum conservative management showed poor survival outcomes.

In the present study, 7(4.4%) patients were kept on mechanical ventilation that was lower than in other studies. Mathew et al,[26]conducted a study in MICU, where 56% of patients were kept on mechanical ventilation. In this study, mortality of patients on ventilatory support was 100% and it was higher when compared to Mathew et al (70.2%).[26]

In the present study, 18.8% of patients were treated with Ionotropic support which correlates with other studies. In a study by Mathew et al,[26] 83.3% of patients were treated with ionotropic support. Studies including the present one had shown a significant association between Inotropic support and outcome (p=0.001). The common underlying cause in patients who required ionotropic support was sepsis (53.3%).

In the present study, comorbidities observed were DM, HTN and CAD. Diabetes mellitus was the common risk factor associated with sepsis. It accounted for 27.5% of patients, higher than the other studies. About 44 (27.5%) patients had diabetes, 25(15.6%) had hypertension and 14(8.8%) had CAD. Out of all, 10% of patients had both DM and HTN.

The average duration of hospital stay of 7.34 days, was shorter than the duration in the Bhattacharya et al study (8.16 days).[25] In this study, patients with a shorter duration of hospital stay (≤7 days) were associated with mortality similar to a study done in Nigeria (155). The reason may be the limited resources to detect AKI early in primary hospitals and health centres, which leads to late referral and admission of patients associated with severe stage of AKI, usually with complications, and who died early on hospital admission. In the present study, out of 160 patients, 121(75.6%) patients were treated conservatively similar to Prakash S et al (76%),[24] and Rekha et al (74%),[19]studies. Among all the patients, 24.4% underwent dialysis similar to Prakash S et al (24%),[24] and Rekha et al(26%).[19]

In Ali et al study, the mortality in patients who underwent renal replacement therapy was 57% similar to the present study (58.97%). In SHARF (Stuivenberg Hospital Acute Renal Failure) study mortality was 58% among those AKI patients who had undergone hemodialysis and 43% among those treated conservatively. The mortality was 16.53% among patients managed conservatively which was

ISSN(O): 2687-5365; ISSN(P): 2753-6556

lower than the SHARF study. So, a critical approach is warranted in initiating RRT in AKI. The overall mortality of AKI in the present study (26.9%) is much lower compared to findings from other studies; 36.9% in Cameroon and 44.4% in Malawi. This may be due to the difference in patient's site of admission (i.e., ICU vs general medical ward), design, and underlying disease of patients. Sepsis had the highest hospital mortality of 46%, which is similar to data from other Indian studies. Worldwide, the incidence of sepsis-induced AKI is increasingand it represents an independent risk factor for in-hospital mortality in these patients.

AKI, irrespective of residual kidney function, increases long-term mortality risk. Complete recovery was achieved in the majority (65.6%) of the present patients and about 7.5% progressed to CKD, at follow up (90 days). Out of 6 patients with paraquat poisoning, 5 succumbed to death (4 patients underwent hemodialysis). A study done in India with six cases of paraquat poisoning had reported AKI in all the cases with 66% mortality which was lower than the present study (83.3%). Patients with non-oliguric AKI had a better prognosis than oliguric renal failure, probably due in large measure to the decreased severity of the insult.

CONCLUSION

Sepsis was the most common cause of AKI in this study. Renal AKI was the common cause of AKI. Risk stratification and early diagnosis & management were the best time-tested approach in treating AKI. Most of the infectious and environmental causes were largely preventable which were associated with significant morbidity and mortality.

REFERENCES

1. Alan F Almeida, Jatin P Kothari, Acute kidney injury; API Text book of medicine; Y P Munjal: 9th edition; 2012:Jaypee; PP 1291.
2. UK Renal Association. Clinical Practice Guidelines. Module 5: Acute Kidney Injury. 4th edition. Final version. 2008.
3. Anandh U et al. Acute renal failure in the tropics: emerging trends from a tertiary care hospital in South India. Clin Nephrol. 2003;59:341-4.
4. Aggarwal HK et al. Spectrum of renal disorders in a tertiary care hospital in Haryana. J Assoc Physicians India. 2007; 55:198-202.
5. Abraham G, Gupta RK, Senthilselvan A, van der Meulen J, Johny KV: Cause and prognosis of acute renal failure in Kuwait: A 2-year prospective study. J Trop Med Hyg 92: 325–329, 1989
6. Schoolwerth, A. C. et al. Chronic kidney disease: a public health problem that needs a public health action plan. Prev. Chronic Dis. 3, A57 (2006).
7. Tiessen H, Cuevas E, Salcedo IH. Organic matter stability and nutrient availability under temperate and tropical conditions. Adv Geoecol. 1997;31:415–227.
8. Cerda J, Bagga A, Kher V, Chakravarthi RM. The contrasting characteristics of acute kidney injury in developed and developing countries. Nat Clin Pract Nephrol.2008;4:138–53.
9. Sakhuja V, Sud K. Acute renal failure in the tropics. Saudi J Kidney Dis Transplant.1999;9(3):247-60
10. 10. Hsiao CY, Yang HY, Hsiao MC, Hung PH, Wang MC. Risk factors for development of acute kidney injury in patients with urinary tract infection. PLoS ONE. 2015;10(7):e0133835.
11. Rahman M, Shad F, Smith MC. Acute kidney injury: a guide to diagnosis and management. Am Fam Physician. 2012;86(7):631-39.
12. William D, Jenana H. Acute Kidney Injury. In: Joseph TD, ed. by. Pharmacotherapy: A Pathophysiologic Approach. 7th ed. New york: McGraw-Hill Medical; 208. Pp. 723-45.
13. Levy EM, Viscoli CM, Horwitz RI. The effect of acute renal failure on mortality. A cohort analysis. JAMA. 1996;275(19):1489-94.
14. Acute kidney injury: prevention, detection and management | Guidance and guidelines | NICE [Internet]. Nice.org.uk. 2013 [cited 2 April 2016].
15. Vikrant S, Gupta D, Singh M. Epidemiology and outcome of acute kidney injury from a tertiary care hospital in India. Saudi J Kidney Dis Transpl. 2018 Jul-Aug;29(4):956-966. doi: 10.4103/1319-2442.239633. PMID: 30152435.
16. Md. Yousuf Khan, P. Deepak, A. Praveen Kumar, Krishna Kumar T V. Study of etiology, clinical profile and outcome of acute kidney injury (AKI) in Medical Intensive Care Unit. International Journal of Contemporary Medical Research 2017;4(11):2225-2228.
17. Bernieh B, Levy DW. Chaudhuri MD. Pattern of acute renal failure. Transplantations proceeding 2003 ; 36 : 1780 - 1783.
18. Mehta RL, Kellum JA, Shah SV, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Crit Care. 2007;11(2):R31. 81
19. Rekha NH, Venkatesha BM, CLINICAL PROFILE OF ACUTE RENAL FAILURE, Sch.J.App.Med.Sci., Dec 2016;4(12A):4197-4199.
20. Bagshaw SM, Uchino S, Bellomo R, Morimatsu H, Morgera S, Schetz M, Tan I, Bouman C, Macedo E, Gibney N, Tolwani A, Oudemans-van Straaten HM, Ronco C, Kellum JA; Beginning and Ending Supportive Therapy for the Kidney (BEST Kidney) Investigators. Septic acute kidney injury in critically ill patients: clinical characteristics and outcomes. Clin J Am Soc Nephrol. 2007 May;2(3):431-9.
21. Al Rohani M, Aljawshaei H, Aduolimi E. Acute renal failure in Yemeni patients. Saudi J Kidney Dis Transpl. 2011;22:829–33.
22. Kaaviya R, Vadivelan M, Balamurugan N, Parameswaran S, Thabah MM. Community Acquired AKI: A Prospective Observational Study from a Tertiary Level Hospital in Southern India. Indian J Nephrol. 2019;29(4):254-260.
23. Murugan R, Karajala-Subramanyam V, Lee M, et al. Acute kidney injury in non-severe pneumonia is associated with an increased immune response and lower survival. Kidney Int 2010;77:527-35.
24. Prakash Shende, S. K. Sharma, V. B. Vikhe, Yashodeep Gaikwad, Adarshvatge, Rahul Patne. Clinical profile of acute kidney injury in intensive care unit in tertiary care centre. Indian Journal of Basic and Applied Medical Research; March 2014: Vol.-3, Issue- 2, P.511-516.
25. Bhattacharya PK, Roy A, Jamil M, Barman B, Murti SV, Marak PR. Clinical profile and determinants of short-term outcome of acute kidney injury: A hospital-based prospective study from Northeastern India. J Lab Physicians. 2019;11(1):5-10.
26. Mathew, D. M. K., &Radha , D. T. R. (2019). Etiological Factors and Clinical Profile of Acute Kidney Injury in Medical Intensive Care Unit. Journal of Current Medical Research and Opinion, 2(11), 350-366. https://doi.org/10.15520/jcmro.v2i11.235.
27. Prakash J, Singh TB, Ghosh B, Malhotra V, Rathore SS, Vohra R, et al. Changing epidemiology of community-acquired acute kidney injury in developing countries: Analysis of 2405 cases in 26 years from eastern India. Clin Kidney J. 2013;6:150–5.
28. Kanjanabuch T, Sitprija V. Snakebite nephrotoxicity in Asia. Semin Nephrol 2008;28:363-72.
29. Muthusethupathi MA, Shivakumar S. Acute renal failure in south India. Our experience with 187 patients. J Assoc Physicians India. 1987;35:504–7.
30. Usman VS, Narayan R. Clinical profile, outcome and prognostic factors of acute kidney injury - a tertiary hospital-based study. Galore International Journal of Health Sciences & Research. 2019; 4(4): 91-98.
31. Singh TB, Rathore SS, Choudhury TA, Shukla VK, Singh DK, Prakash J. Hospital acquired acute kidney injury in medical, surgical, and intensive care unit: A comparative study. Indian J Nephrol 2013 Jan;23(1):24–9.

ISSN(O): 2687-5365; ISSN(P): 2753-6556