

A PROSPECTIVE STUDY OF AETIOLOGY AND OUTCOME OF ACUTE KIDNEY INJURY IN TYPE 2 DIABETES PATIENTS

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Abstract:

Introduction: An important development in the recent years is the observation that relative small elevation of serum creatinine has been associated with worsening of outcome in various diseases. Acute kidney injury is recognized as a contributor to unfavorable outcome in different clinical settings and should be considered as a risk factor for mortality in a host of clinical entities. AKI is observed in 7% of patients requiring hospitalization and in 36% to 67% of critically ill patients depending on the definition used.

Materials and Methods: 200 patients admitted with AKI are defined according to AKI Network criteria (AKIN) among 1500 acute care hospital admissions in Sri Siddartha Medical College and Research Centre, B.H Road, Agalkote, Tumkur, Karnataka, India. Their clinical and biochemical data were studied. The patients were followed up until discharge/death. Written informed consent was obtained from the patients selected for the study. History collected from the patient and subjected to clinical examination, routine blood investigations including blood urea, serum creatinine, urine routine, ultrasound abdomen, serology for leptospirosis, enteric fever, peripheral smear for malarial parasite and other relevant investigations were performed and values interpreted. Patients were started on appropriate therapy once a diagnosis was made. Wherever possible etiological factors were corrected. Renal replacement therapy was given according to their clinical and biochemical indications.

Results: Among 200 patients with acute kidney injury, 126 male patients and 74 female patients are included in the study. Mean age group in this study is 44.22 Mean blood urea is 116.39 Mean baseline creatinine is 1.02 Mean creatinine is 2.06 Creatinine at diagnosis is 3.22 Mean sodium is 137.98. Mean potassium is 4.5 Mean urine sodium is 31.06. 63% male patients and 37% female patients were studied. Maximum number of patients were in the age group of 51 to 60 (26%), followed by age group of 41 to 50 (21%), followed by age >60 years (16%), followed by 14% in age group 21 to 30 and 31 to 40, followed by 9% under <20 years of age.

Conclusion: The presentation of AKI is predominately oliguric but non-oliguric AKI can also occur. Fever, loose stools, pedal edema and vomiting were other presentations observed. Sepsis is the most common cause of AKI followed by hypotension and acute gastroenteritis. It is essential to prevent and treat multi organ failure to reduce the mortality. Our patients are treated conservatively and dialysis done whenever required. Hemodialysis was the preferred mode of dialysis than peritoneal dialysis. 154 patients recovered fully and 30 patients recovered partially and the mortality rate was 8%. Presence of multi organ failure and complications is the chief

reason for mortality .Organ failure had a greater impact on severity and prognosis of AKI. We observed that early diagnosis and early intervention are responsible for good survival rate in AKI.

Key Words: Acute kidney injury, mortality, pedal edema, peritoneal dialysis.

INTRODUCTION

An important development in the recent years is the observation that relative small elevation of serum creatinine has been associated with worsening of outcome in various diseases.¹ Acute kidney injury is recognized as a contributor to unfavorable outcome in different clinical settings and should be considered as a risk factor for mortality in a host of clinical entities. AKI is observed in 7% of patients requiring hospitalization and in 36% to 67% of critically ill patients depending on the definition used.²

Diabetic population are at risk of developing AKI either resulting from fluid loss associated with diabetic ketoacidosis and non ketotic hyperosmoar coma or due to other adversaries like hypotension, infection or exposure to nephrotoxic substances.³

AKI can result from decreased renal or intrarenal perfusion, a toxic or obstructive insult to the renal tubule, tubulointerstitial inflammation and edema or primary reduction in the filtering capacity of the glomerulus.⁴ Ischemia and toxins, often in the setting of sepsis, account for the largest number of cases of AKI. It is estimated that 19-33% of in hospital AKI causes are attributed to drug nephrotoxicity.⁷⁻⁹ In developed countries trauma and surgery constitute the main cause of acute renal failure whereas in developing countries more than 60% are related to medical cause.⁵

Outcome of AKI depends on three factors: early recognition, establishment of cause and appropriate clinical management. A recent large international study on the epidemiology and outcome of AKI in critically ill adult patients reported an overall in hospital mortality rate of 60%, of those who survived to hospital discharge and 13% remained dialysis dependent.⁶

MATERIALS AND METHODS

Sample size: 200

Type of study: Prospective study

Duration of study: May 2022 to May 2023.

Area of Study: Department of Emergency Medicine, Sri Siddartha Medical College and Research Centre, B.H Road, Agalkote, Tumkur, Karnataka, India. Pin-527107.

Study design and sampling

200 patients admitted with AKI are defined according to AKI Network criteria (AKIN) among 1500 acute care hospital admissions in Sri Siddartha Medical College and Research Centre, B.H Road, Agalkote, Tumkur, Karnataka, India. Their clinical and biochemical data were studied. The patients were followed up until discharge/death.

Inclusion Criteria:

1. Patients of both sex more than 12 years of age

2. Patients with increase in serum creatinine of 0.3 mg/dl from the baseline or elevation of >50% from the baseline (based on AKIN criteria), reduced glomerular filtration rate (GFR) and urine output < 0.3ml/kg/hr for 24 hours or anuria for 12 hours (based on failure category of RIFLE criteria of acute kidney injury).

Exclusion Criteria:

1. Pregnancy
2. Age less than 12 years
3. Chronic contracted kidneys

Methodology:

Patients fulfilling the inclusion and exclusion criteria were included in this study conducted from May 2022 to May 2023. Written informed consent was obtained from the patients selected for the study. History collected from the patient and subjected to clinical examination, routine blood investigations including blood urea, serum creatinine, urine routine, ultrasound abdomen, serology for leptospirosis, enteric fever, peripheral smear for malarial parasite and other relevant investigations were performed and values interpreted. Patients were started on appropriate therapy once a diagnosis was made. Wherever possible etiological factors were corrected. Renal replacement therapy was given according to their clinical and biochemical indications.

Patients with raise in serum creatinine 0.3 mg/dl from the baseline or > 50% elevation from the baseline were followed up and studied.

Statistical analysis: Statistical analysis was performed using SPSS VERSION 21 software. All categorical data are expressed as percentage. The continuous variables are expressed as Mean \pm Standard deviation. Statistical analysis are performed using anova test, unpaired test and kruskal wallis test. Variables with p values <0.05 is considered significant.

RESULTS

AGE IN YEARS	NO OF PATIENTS	PERCENTAGE
< 20	18	9%
21-30	28	14%
31-40	28	14%
41-50	42	21%
51-60	52	26%
> 60	32	16%

Table 1: Age distribution

SEX	NO OF PATIENT	PERCENTAGE
MALE	126	63%
FEMALE	74	37%

Table 2: Gender Distribution

SYMPTOMS	PRESENT	ABSENT
OLIGURIA	132	34
FEVER	72	64
VOMITTING	10	95
LOOSE STOOLS	44	78
PEDAL EDEMA	22	89
JAUNDICE	6	97

Table 3: Symptoms

CAUSES	PRESENT	ABSENT
SEPSIS	44	78
HYPOTENSION	32	84
AGE	38	81
SNAKE BITE	30	85
POISONING	12	94
GLOMERULONEPHRITIS	14	93
FEVER	12	94
DRUGS	2	99
BOO	6	97
RENAL CALCULI	10	95
INJURY	4	98
BURNS	4	98
PANCREATITIS	2	99

Table 4: Causes

LEUCOCYTOSIS	NO OF PATIENTS	PERCENTAGE
PRESENT	76	19%
ABSENT	162	81%

Table 5: Leucocytosis

THROMBOCYTOPENIA	NO OF PATIENTS	PERCENTAGE
PRESENT	28	14%
ABSENT	172	86%

Table 6: Thrombocytopenia

HYPERKALEMIA	NO OF PATIENTS	PERCENTAGE
PRESENT	12	6%
ABSENT	188	94%

Table 7: Hyperkalemia

URINE SODIUM	NO OF PATIENTS	PERCENTAGE
< 20	62	31%
> 20	138	69%

Table 8: Urine Sodium

KINETIC GFR	NO OF PATIENTS	PERCENTAGE
LESS THAN 10	124	62%
10 TO 20	38	19%
20 TO 30	38	19%

Table 9: Kinetic GFR

PROTEINURIA	NO OF PATIENTS	PERCENTAGE
PRESENT	14	7%
ABSENT	186	93%

Table 10: Proteinuria

TYPE OF AKI	NO OF PATIENTS	PERCENTAGE
PRE RENAL	58	29%
RENAL	126	63%
POST RENAL	16	8%

Table 11: Type of AKI

CONSERVATIVE TRT	NO OF PATIENT	PERCENTAGE
DONE	124	62%
NOT DONE	76	38%

Table 12: Conservative Treatment

HEMODIALYSIS	NO OF PATIENTS	PERCENTAGE
DONE	44	24%
NOT DONE	152	76%

Table 13: Hemodialysis

PERITONEAL DIALYSIS	NO OF PATIENTS	PERCENTAGE
DONE	28	14%
NOT DONE	172	86%

Table 14: Peritoneal Dialysis

TREATMENT	NO OF PATIENTS	PERCENTAGE
CONSERVATIVE	124	62%
HEMODIALYSIS	48	24%
PERITONEAL DIALYSIS	28	14%

Table 15: Treatment

IN ALL PATIENTS		
PARAMETERS	MEAN	SD
AGE	44.22	15.91
BLOOD UREA	116.39	46.24
BASELINE CREATININ	1.02	0.19
CREATININE 2	2.06	0.67
CREATININE AT DIAGNOSIS	3.22	1.39
SODIUM	137.98	7.46
POTASSIUM	4.5	0.86
URINE SODIUM	31.06	12.96

Table 16: General Parameters

OUTCOME	NO OF PATIENTS	PERCENTAGE
FULL RECOVERY	154	77%
PARTIAL RECOVERY	30	15%
DEATH	16	8%

Table 17: OUTCOME

Among 200 patients with acute kidney injury, 126 male patients and 74 female patients are included in the study. Mean age group in this study is 44.22 Mean blood urea is 116.39 Mean baseline creatinine is 1.02 Mean creatinine is 2.06 Creatinine at diagnosis is 3.22 Mean sodium is 137.98. Mean potassium is 4.5 Mean urine sodium is 31.06. 63% male patients and 37% female patients were studied. Maximum number of patients were in the age group of 51 to 60 (26%), followed by age group of 41 to 50 (21%), followed by age >60 years (16%), followed by 14% in age group 21 to 30 and 31 to 40, followed by 9% under <20 years of age.

Most common symptoms observed were, Oliguria- 66%, Fever- 36%, Loose stools- 22%, Pedal edema- 11%, Vomiting- 5%, Jaundice- 3% Common causes observed are, Sepsis- 22%, Acute gastroenteritis- 19%, Hypotension- 16%, Snake bite- 15%, Glomerulonephritis- 7% , Fever-6%, Renal calculi- 5%, Bladder outlet obstruction- 3%, Injury- 2%, Burns- 2%, Pancreatitis- 1%, Drugs- 1%.

Sepsis is defined as life threatening organ dysfunction caused by a dysregulated host response to infection. Sepsis is defined according to **CRITERIA IN 1991/2003(SEPSIS 1/SEPSIS 2)**: Suspected (or documented) infection plus ≥ 2 SIRS criteria.

CRITERIA IN 2016 (SEPSIS 3): Suspected (or documented) infection plus acute increase in ≥ 2 sepsis related organ failure assessment (SOFA) points.

Leukocytosis is observed in 19% and thrombocytopenia was observed in 14% and hyperkalemia in 6% of patients. Urine sodium was <20 in 31% of patients.

Kinetic GFR is used for calculating GFR in this study. Serial changing creatinine values (3 creatine values- baseline creatinine, creatinine 1 and creatinine 2) is used.

Kinetic GFR was <10 in 62%, 10-20 in 19%, 20-30 in 19%. Proteinuria was present in 7%. 29% patients had pre renal failure, 63% had intrinsic renal failure and post renal 8%.

Conservative management was done for 62% of patients, hemodialysis for 24% and peritoneal dialysis for 14%. 77% patients had full recovery, 15% had partial recovery, death observed in 8% of patients. Most of the patients with intrinsic renal failure had full recovery. Most of the patients with pre renal failure had partial recovery. Most number of deaths were observed in pre renal AKI. Patients treated with conservative management recovered well. 50% of patients with Kinetic GFR <10 have fully recovered and death was observed in 6% of patients with Kinetic GFR <10.

DISCUSSION

AKI is a potentially fatal but reversible renal disease. The etiology, clinical course and outcome differs in various part of the world and also within India because of geographic and climatic diversity and varying standard of medical care in India.⁷

In our study 200 patients were analysed with 126 males and 74 females. Mean age of occurrence was 44.22. Maximum number of cases occurred in 5th to 6th decade. Oliguric AKI (66%) was predominant in our study. which is in concordance with previous study by M A. Muthusethupathy et al.

Sepsis was the leading cause for AKI in our study, which is in concordance with most of the multicentre trials. A number of studies indicate that onset of AKI is associated with a higher resource utilization and risk of death than in patients without AKI.⁸

Moreover, this increased risk of death rises incrementally with the severity of AKI. In 2005, Uchino et al. found that mortality for patients with severe AKI requiring renal replacement therapy was 60.3%. In a study by Thakar et al, the risk of death increased with the increasing severity of AKI: AKIN stage 1, odds ratio (OR) 2.2; stage 2, OR 6.1 and stage 3, OR 8.6. Data from 2010 indicates that even transient perturbations in kidney function in hospitalized patients increases the risk of death. These findings suggest that even small changes in kidney function carry a notable mortality.⁹

Sepsis accounts for 22% of cases. 58 patients had pre renal AKI. 126 patients had intrinsic renal failure and 16 patients had post renal failure. Conservative management was done for 124 patients. 48 patients were treated with hemodialysis and 28 patients were treated with peritoneal dialysis. Out of the 200 patients studied 154 patients recovered fully, 30 patients recovered partially and death occurred in 16 patients.¹⁰

15% of cases were due to hemotoxic snakebite. Malaria and leptospirosis contributed 5% of patients. There were no death due to malarial AKI as against 42.5% mortality in the study by Zinna et al study. A study from eastern India conducted by Prakash et al reported 4.2% cases of malarial AKI. Mortality is decreased due to early diagnosis and effective treatment of malaria. There was no death due to leptospirosis in contrast to M.A.Muthusethupathy study, which observed 28% mortality. Low mortality in our study is due to early diagnosis and awareness of clinical features of leptospirosis with standard diagnostic test and effective therapy. Ostermann. M Chang R W 2007 detected that RIFLE class Failure has mortality rate of 57% and RIFLE class Injury had 45% mortality.

Prevalance of AKI was found to 6.6%. Associated organ failure had greater impact on outcome than the severity of AKI.

CONCLUSION

The presentation of AKI is predominately oliguric but non-oliguric AKI can also occur. Fever, loose stools, pedal edema and vomiting were other presentations observed. Sepsis is the most common cause of AKI followed by hypotension and acute gastroenteritis. It is essential to prevent and treat multi organ failure to reduce the mortality. Our patients are treated conservatively and dialysis done whenever required. Hemodialysis was the preferred mode of dialysis than peritoneal dialysis. 154 patients recovered fully and 30 patients recovered partially and the mortality rate was 8%. Presence of multi organ failure and complications is the chief reason for mortality. Organ failure had a greater impact on severity and prognosis of AKI. We observed that early diagnosis and early intervention are responsible for good survival rate in AKI.

REFERENCES

1. Mahajan S, Tiwari S, Bharani R, et al. Spectrum of acute renal failure and factors predicting its outcome in an intensive care unit in India. *Ren Fail* 2006;28(2):119-24.
2. Jha V, Malhotra HS, Sakhuja V, et al. Spectrum of hospital-acquired acute renal failure in the developing countries - Chandigarh study. *Q J Med* 1992;83(303):497-505.
3. Prakash J, Singh SP, Kumar OM, et al. Hospital acquired acute renal failure. *Indian J Nephrol* 1996;6:9-13.
4. Ghani AR, Zainudin S, Kamaruddin NA, et al. Acute renal failure following the use of rosiglitazone in a chronic kidney disease patient. *Singapore Med J* 2009;50(1):e32-4.
5. Clemente PA. Feminisation and nephrology. *Nefrologia* 2010;30(1):110-3.
6. Cartin-Ceba R, Kashiouris M, Plataki M, et al. Risk factors for development of acute kidney injury in critically ill patients: a systematic review and meta-analysis of observational studies. Article ID 691013, *Crit Care Res Pract* 2012;2012: p. 15.
7. Schiff H. Renal recovery from acute tubular necrosis requiring renal replacement therapy: a prospective study in critically ill patients. *Nephrol Dial Transplant* 2006;21(5):1248-52.
8. Waikar SS, Liu KD, Chertow GM, et al. The incidence and prognostic significance of acute kidney injury. *Curr Opin Nephrol Hypertens* 2007;16(3):227-36.
9. Bagshaw SM, Uchino S, Bellomo R, et al. Septic acute kidney injury in critically ill patients: clinical characteristics and outcomes. *Clin J Am Soc Nephrol* 2007;2(3):431-9.
10. Singbartl K, Kellum JA. AKI in the ICU: definition, epidemiology, risk stratification and outcomes. *Kidney Int* 2012;81(9):819-25.