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Acute Kidney Injury- Incidence, Etiology and Clinical Outcome in A Multispeciality Hospital

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Abstract: Acute kidney injury (AKI) is a syndrome of varied severity. It is characterized by a rapid (hrs to weeks) decline in the glomerular filtration rate (GFR) and retention of nitrogenous waste products such as blood urea nitrogen (BUN) and creatinine. In recent years, it has been recognized that the time-honored term acute renal failure (ARF) fails to adequately describe what is a dynamic process extending across initiation, maintenance and recovery phases, each of which may be of variable duration and severity. The aim of the study was to know the incidence of AKI, to study the etiology and risk factors and clinical outcome of the AKI. All the patients above 18 yrs age who have a rise in serum creatinine levels of atleast 0.3mg/dl or 50% of base line within 24-48 and reduction in urine output to 0.5ml/kg/hr for longer than 6hrs were considered. Of all screened patient 100 satisfied the criteria of hospital acquired acute renal failure which represents 2.27 percent of patients who suffered from hospital acquired acute kidney injury. All patients had only one episode of acute renal failure. In this study, eighteen (18%) patients who developed acute renal failure died. Septicemia was the commonest cause of death in patients of acute renal failure numbering 6 (33.3%).Four (22.2%) patients died due to peripheral circulatory failure. Uncontrolled renal failure was responsible for 3 (16.7%) deaths. Hyperkalemia was the cause of death in 2 patients (11.1%). 2 patients (11.1%) died of gastrointestinal bleed and 1 patient (5.6%) died of pulmonary edema. The frequent and close pre intervention monitoring of serum creatinine in pertinent clinical settings is essential for early detection of AKI; in patients with risk factors.

Keywords: Acute Kidney Injury, Septicemia, Renal Perfusion, Serum Creatinine, Blood Urea, Oliguria, Hyperkalemia.

INTRODUCTION

Acute kidney injury (AKI), previously called acute renal failure (ARF) is a rapid loss of kidney function. Its causes are numerous and include low blood volume, exposure to toxins, and prostate enlargement. AKI is diagnosed on the basis of clinical history, such as decreased urine production, and characteristic laboratory findings, such as elevated blood urea nitrogen and creatinine. Depending on its severity, AKI may lead to a number of complications, including metabolic acidosis, high potassium levels, changes in body fluid balance, and effects to other organ systems. Management includes supportive care, such as renal replacement therapy, as well as treatment of the underlying condition.

The AKI in hospitalized patients appear as a complication of preexisting renal disease, nephrotoxic agents and it is more frequent in critically ill patients and those who are admitted in intensive care units. The incidence and spectrum of hospital acquired AKI is variable in different studies. From a variety of predominantly single centre studies it is estimated that 3% to 7% of hospitalized patients develop AKI [1-3]

The incidence of hospital acquired acute renal failure in one Indian study was 0.64%. The higher incidence has been reported in ICUs [4]. Hospital-acquired AKI often occurs in an ICU setting and is commonly part of multi organ failure. This dichotomy in the etiology of AKI explains the increased mortality rate, dialysis requirements, and rates of progression to end-stage renal failure seen in hospital-acquired ARF compared with community-acquired ARF [5].

The important cause of hospital acquired acute renal failure includes hypo perfusion to the kidney, nephrotoxic drugs, major surgery, contrast media and complicating medical illnesses like hepatorenal syndrome, cardiac failure and vascular occlusion of renal vessels. The etiological factors causing hospital acquired kidney injury differ markedly from study to study.

Since there is paucity of literature on the subject AKI in hospitalized patients in our country therefore the present hospital based study was undertaken to find out if there is any new etiological factors and risk factors for development of AKI and if any measures can be taken to decrease the risk.

AIM OF THE STUDY

Aim of this study is to know the incidence of acute kidney injury, study the etiology and risk factors for AKI and the clinical outcome of the AKI.

MATERIALS AND METHODS

The present study entitled spectrum of acquired acute kidney injury in hospitalized patients was carried out in the department of medicine, surgery, obstetrics and gynecology, ISPAT GENERAL HOSPITAL ROURKELA over a time period of eighteen months from November 2012 to April 2014. All patients that satisfied our inclusion and exclusion criteria were undertaken for this study. Approval from ethical committee and written consent from the scientific committee were obtained. Written and well informed consent was obtained from the patients and guardian of patients selected for the study. Clinical history, physical findings and lab parameters were recorded in a predetermined proforma. All patients fulfilling the inclusion criteria then underwent a fixed set of investigations.

Inclusion criteria

Patients above 18 yrs age who have:-

- A rise of serum Creatinine of atleast 0.3mg/dl or 50% of base line within 24-48
- Reduction in urine output to 0.5ml/kg/hr for longer than 6hrs.
- Both male and female subjects are considered.

Exlusion criteria

- Patients with chronic kidney injury.
- Other renal impairment cases where there is difficulty in making a clear cut diagnosis of AKI.

A serial record of urine output and serum creatinine was maintained during the course of AKI till death, discharge or return of renal function to normal. Data was calculated in the form of percentages for clinical parameters, final outcome and etiological factors. Ratio was calculated for male and female subjects.

All the admitted patients were evaluated for their serum creatinine values frequently. The first value of the serum creatinine after the admission to hospital was taken as the baseline value. If an increase of the serum creatinine as defined previously, occurred during the hospital stay, it was labeled as hospital acquired acute kidney injury. A detailed history, physical examination and laboratory investigation was performed in such cases in order to establish a cause for acute renal failure. All such patients were followed up to discharge, death or return of the renal function to baseline level. A serial record of urine output and serum creatinine was maintained.

History and examination

To assign a specific cause for acute renal failure following clinical criteria was used. Decreased renal perfusion was identified as a cause of acute renal failure when there was documented fall of blood pressure to below 90/60 and postural hypotension, loss of skin turgor or congestive heart failure. Major surgery was thought to be the cause for acute renal failure whenever rises in serum creatinine occurred as defined in the criteria within 72 hours of surgery with or without documented hypotension. Radiographic contrast media was thought to be the cause of ARF when acute renal failure occurs within 72 hours of a radiological procedure using contrast agent. Nephrotoxic drug was considered to be the cause of acute renal failure when the drug was taken for a minimum of 3 days prior to the defined increase in serum creatinine. Hepato-renal syndrome was considered to be the cause for acute renal failure when ARF developed in patients with liver diseases with urinary sodium less than 10/meg/liter. Septicemia was identified as a cause of acute renal failure when temperature was > 100° F and total leucocyte count was >10 x 109/L in an appropriate clinical setting or positive blood culture.

The complications (Bleeding diathesis, metabolic acidosis, neuropsychiatric manifestations, hyperkalemia and pulmonary edema) associated with ARF were looked especially in each patients and recorded. Hemodialysis or peritoneal dialysis was instituted using standard clinical indication. Death was directly attributed to AKI if death has occurred complicating hyperkalemia, uremia, pulmonary edema or bleeding diathesis secondary to oliguria and azotemia.

RESULTS

The present study entitles “Spectrum of AKI in hospitalized Patients” was carried out in the department of medicine ISPAT general hospital, ROURKELA. Of all screened patient 100 satisfied the criteria of hospital acquired acute renal failure which represents 2.27 percent of patients who suffered from hospital acquired acute kidney injury. All patients had only one episode of acute renal failure. The incidence of acute renal failure in our hospital is shown in Table-1.

Table -1: Incidence of acute kidney injury in our hospital

Study period	No of patients of AKI	Total no of patients admitted in IGH(all deparments included)	Incidence
Sept 2012-April 2014	100	44012	2.27

Maximum number of patients with hospital acquired acute kidney injury was in their fourth and fifth decade. The youngest patient was 18 years old and the oldest was 75 years. The male to female sex ratio was 1.22:1. Out of 100 patients who developed hospital acquired ARF, 55 (55%) were male and 45 (45%) were female.

The Etiological spectrum of hospital acquired AKI is shown in Table-2. In majority (90) of the cases only one factor was responsible for ARF and in remaining patients (10) multiple causative agents were responsible for hospital acquired ARF.

Table-2: Etiological spectrum of Acute Kidney Injury in our hospital (n = 100)

Etiological Factor	No of Patients	Percentage
Decreased renal perfusion	30	30
Volume depletion	22	22
Urinary catheterization	3	3
Heart failure	5	5
Hepato renal syndrome	7	7
Nephrotoxic drug	8	8
Septicemia	20	20
Intravascular hemolysis	35	35

Among the post-operative cases sepsis was the commonest etiological factor followed by decreased renal perfusion and nephrotoxic drugs. Among the drugs NSAIDs were the common cause.

All patients included in our study were screened for the evidence of risk factors which makes the kidney vulnerable to ischemic or toxic injury. 38 (38%) patients had definite factors which increases the risk for AKI (Table-3). The commonest factor was the age>60 years observed in 12 (12%) patients. Hypertension, pre-existing liver disease, were observed in 9 (9%), 7 (7%) patients respectively. Multiple nephrological insults like dehydration, sepsis and acidosis were observed in 10 (10%) patients.

Table-3:- Risk factors in patients of acute kidney injury in our hospital (n = 100)

Risk factor	No of Patients	Percentage
Age>60 years	12	12
Hypertension (> 140>90)	9	9
Pre-existing liver disease	7	7
Multiple nephrological insults	10	10

In this study, 64 (64%) patients had oliguria (Urine output <400ml/d) and 36 (36%) patients were non-oliguric. Majority of the non-oliguric patients were having drug induced renal toxicity. Neuropsychiatric manifestations were present in 11 (11%) patients in the form of drowsiness, confusion or coma. 7 (7%) patients had hyperkalemia (serum potassium>6 meq/L). Pulmonary edema was present in 4 (4%) patients, pericarditis in 4 (4%) patients and bleeding diathesis was present in 1 (1%) patient. 28 (28%) patients required dialysis. 72 (72%) patients were undialysed (Table-4).

Table-4 Clinical features in patients of acute kidney injury in our hospital (n = 100)

Parameter	No of Patients	Percentage
Oliguria	64	64
Non- Oliguric	36	36
Neuropsychiatric features	11	11
Hyperkalemia (>5.5meg/L)	7	7
Pulmonary edema	4	4
Pericarditis	4	4
Bleeding diathesis	1	1

When the creatinine profile of AKI was taken into consideration in this study, it was found that all the patients had creatinine value less than 1.5 and 15 patients had high creatinine value (>8) (Table-5). Most

of the patients had blood urea levels of more than 100 at the time of admission and over the days they recovered and had decreased urea levels at the time of discharge (Table-6). Serum electrolytes levels were altered in many cases at the time of admission which were reverted back to normal or near normal at the time of discharge (Table-7 & 8). In this study, duration of hospital stay was less than 5 days in many cases.

Table-5: Creatinine profile in patients of AKI

Range of Serum Creatinine	No of patients at admission	No of patients at discharge/death
<1.5	0	80
1.5-2.9	22	4
3-4.9	18	5
5-7.9	45	6
>8	15	5

Table-6: Blood Urea profile in patients of AKI

Range of Blood Urea	No of patients at admission	No of patients at death/discharge
<100	27	64
100-199	44	28
200-299	21	6
>300	8	2
Total	100	100

Table-7: Serum sodium profile in patients of AKI

Range of Sr Sodium	No of patients at admission	No of patients at discharge/death
105-115	3	0
116-130	12	16
>130	95	84

Table-8: Serum Potassium profile of AKI

Range of Serum potassium	No of patients at admission	No of patients at discharge/death
3.5-5.5	93	97
>5.5	7	3

In this study, eighteen (18%) patients who developed acute renal failure died. Septicemia was the commonest cause of death in patients of acute renal failure numbering 6 (33.3%) (Table-9).

Four (22.2%) patients died due to peripheral circulatory failure. Uncontrolled renal failure was responsible for 3 (16.7%) deaths. Hyperkalemia was the cause of death in 2 patients (11.1%). 2 patients (11.1%) died of gastrointestinal bleed and 1 patient (5.6%) died of pulmonary edema.

The organisms which caused deaths due to sepsis were Pseudomonas in 3 patients, Acinetobacter in 2 patients. In 1 patient organism could not be identified**.**

Table–9: Causes of mortality in patients of acute kidney injury in our hospital (n = 18)

Cause of death	No of Patients	Percentage
Septicemia	6	33.3
Peripheral circulatory failure	4	22.2
Uremia	3	16.7
Hyperkalemia	2	11.1
Gastrointestinal bleed	2	11.1
Pulmonary edema	1	5.6
Total	18	100.0

DISCUSSION

Acute kidney injury is an important cause of mortality and morbidity. It is more common in patients who are seriously ill or admitted in intensive care units. Various studies have been done in the western countries but little work has been done in the developing countries on this topic [1, 3, 6]. The aim of this prospective study was to analyze the incidence, etiological spectrum and outcome of patients with hospital acquired AKI in our hospital. The data from the

technologically advances countries cannot be applied to the developing countries on this subject (1,6).

In the present study acute renal failure in hospitalized patients complicated 2.27 percent of all patients admitted in the general medical ward and department. Hou et al reported an incidence of 4.9 per cent for hospital acquired acute renal failure amongst patients admitted to the Tuft New England Medical Center in Boston [4]. Shusterman *et al.*, reported the incidence of 2 per cent for hospital acquired AKI amongst the patients who developed this complication at the University of Pennsylvania Hospital in Philadelphia [7]. The Indian study conducted by Jha *et al.*, in north Indian at Chandigarh revealed the incidence of this complication to be 0.64 per cent [3]. This difference in the incidence appears because of the different criteria used in different studies. Hou et al was more liberal in defining criteria for acute renal failure [4].

Shusterman et al included medical, surgical and gynecological patients in his study. Hou *et al.*, looked at this problem only among patients of general medical and general surgical services [4]. Jha *et al*., looked the patients of medicine, surgery, gynecology, pediatrics, dermatology, psychiatry, ophthalmology, ENT and orthopedics [3]. In our study, the patients were included from general medicine wards and their specialties. This difference in the group of patients included in various studies may be another cause for difference in the incidence of hospital acquired ARF in different studies. The incidence in our study is quite close to the Chandigarh study despite the inclusion of patients from various other departments in Chandigarh study probably because ARF rarely develops in those additional departments like dermatology, ophthalmology, ENT and psychiatry [3].

Despite the wide variation in the spectrum of etiological factors observed in different studies, only few etiological factors, such as nephrotoxic drugs, decreased renal perfusion, surgery and septicemia were operating in majority of the cases of hospital acquired renal failure. Decreased renal perfusion was the etiological factor, being responsible in 30 (30%) patients in the present study which is similar to the reports of Hou *et al*., where also the same etiological factors were responsible for hospital acquired acute renal failure; in maximum number of patients (42%). In the Chandigarh study nephrotoxic drugs were the most common etiological factor responsible for hospital acquired acute renal failure being responsible in 29% of the patients [3]. Decreased renal perfusion and intravascular hemolysis are the commonest cause of hospital acquired acute renal failure in our study. The observations highlight the need of maintaining adequate renal perfusion in the hospitalized patients. Thus all the critically ill patients must be watched daily for their hydration status and fluid balance. The use of nephrotoxic must be carefully monitored in serious patients and intensive care units. Administration of nephrotoxic drugs specially the aminoglycosides was the most frequent (29%) cause of hospital acquired acute renal failure in Chandigarh study while nephotoxic ARF was seen in 8 (8%) patients in our study. This highlights the need to monitor renal function carefully in every patient who receives aminoglycoside drugs such as gentamycin and amikacin. All patients who are getting aminoglycosides should have their base line serum creatinine done before the start of therapy and aminogoglycosides should preferably be avoided if the patient is already having renal disease or other risk factors for hospital acquired acute renal failure. Non steroidal anti inflammatory drugs can produce AKI, particularly in patients with certain risk factors such as congestive heart failure, cirrhosis, age greater than 60 year and diabetic nephropathy [8, 9]. Non steroidal anti inflammatory drugs were responsible in 5 (5%) patients in our study. Radiographic contrast media was responsible in 1(1%) cases in the present study while it was operating in 4 per cent patients in the study of Jha *et al*., [3]. The incidence of contrast associated ARF varies from 20-22 per cent.

Contrast induced ARF may be prevented if its use is avoided in patients with pre-existing renal disease, diabetic nephropathy and elderly patients. However such patients should be well hydrated and adequate dieresis should be maintained before and after the procedure in order to prevent contrast associated renal failure.

Septicemia also was one important cause for hospital acquired acute renal failure, being responsible in 20 (20%) patients, which highlights the need of controlling the infection early in the disease with specific antibiotics. All the critically patients must have a barrier nursing care where the nursing staff should take all aseptic precautions while dealing with such patients. The control and treatment of infection using appropriate and meticulous measures are important steps towards the prevention of septicemic acute renal failure. Since most of these patients are on intravenous lines and indwelling catheters, the use of intravenous lines and indwelling catheters which break the normal anatomical barriers should be minimized [3]. This figure is close to the study of Hou *et al.*, who reported 18.6% and the Chandigarh study reporting 18% [4].

Thirty Five (35%) patients developed hospital acquired acute renal failure because of intravascular hemolysis. All patients of intravascular hemolysis were suffering from malarial fever. This hospital lies in an endemic area of the Plasmodium falciparum parasite and malaria is a common occurrence.

Catheterization was responsible in 3 (3%) patients for development of hospital acquired acute

renal failure. This again stresses the need for minimizing the use of urinary catheters in hospitalized patients and if at all they are necessary proper aseptic precaution should be taken.

Eighteen of 100 patients died with a mortality rate of 18%. Mortality of the ARF was 17.4% in an Indian study. However, the higher morality (41.0%) was reported by Hou *et al*., [4]. The difference in mortality in different study may be due to selection of criteria of patients with AKI. Severity of the primary disease, and associated sepsis. Septicemia was the commonest (33.3%) cause of death. Thus, the control of infection remains the comer stone of therapy in hospital acquired AKI and it was found to be helpful in decreasing the morbidity and mortality associated with ARF. Oliguria, severity of renal failure, multi organ failure and development of complication of AKI contribute to mortality adversely [10]. Other causes of mortality were peripheral circulatory failure (22.2%), uncontrolled uremia (16.7%), hyperkalemia (11.1%), gastrointestinal bleed (11.1%) and pulmonary edema (5.5%).

CONCLUSION

Acute renal failure is an important cause of mortality and morbidity. Despite a wide variability in the clinical setting of ATN, the etiological factors have remained relatively constant. The meticulous search for clinical circumstances known to cause AKI should be made appropriately in all hospitalized patients. The patients with pre-existing renal disease, elderly patients, and patients with decreased intravascular volume and dehydrated patients are at more risk for the development of acute renal failure. The frequent and close pre intervention monitoring of serum creatinine in pertinent clinical settings is essential for early detection of AKI; in patients with risk factors.

REFERENCES

1. Lameire N, Van Biesen W. Vanholdre R: Acute Renal Failure. Lancet 2005; 365:417-430.
2. Nash K, Hafeez A, Hou S. Hospital-Acquired Renal Insufficiency. Am J Kidney Dis 2002; 39:930-936.
3. Jha V, Malhotra HS, Sakhuja V, Chugh KS. Spectrum of hospital acquired acute renal failure in the developing countries, Chandigarh study. Quarterly Journal of Medicine, New Series, July 1992; 84, 303 : 497-505.
4. Hou SH, Bushinsky DA, Wish JB, Cohen JJ, Harrington JT. Hospital acquired renal insufficiency. A prospective study Am. J. Med. 1983; 74: 243-248.
5. Wen J, Cheng Q, Zhao J. Hospital acquired acute kidney injury in Chinese very elderly patients. J Nephrol. 2013 May-Jun;26(3) : 572-9.
6. Brady Hr, Singer GG. Acute Renal Failure Lancet 1995; 346: 1533- 1540
7. Shusterman N, Storm BL, Murray TG, Morrison G, West SL, Maislin G. Risk factors and outcome of hospital acquired acute final failure Am. J. Med. 1987, 83 : 65-71.
8. Rasmussen HH, Ibels, LS. Acute renal failure multivariate analysis of causes and risk factors, Am. J. Med. 1982, 73 : 211- 213.
9. Blackshear JL, Davidman M, Stillman MT. Identification of risk for renal insufficiency from non-steroidal anti-inflammatory drugs. Arch. Intern. Med. 1983, 143: 1130-1134.
10. Kleinknecht D, Ganeval D, Gonzalez-Duque LA, Fermanian J. Furosemide in acute oliguric renal failure A controlled trial. Nephron. 1976;17(1):51-8.