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Management and outcome of Acute Kidney Injury at a Tertiary Care Hospital

Cijo John[1], Selin Abraham[1]

Author's Affiliation:
[1]Assistant Professor, Department of Medicine, Mount Zion Medical College, Chayalode, Adoor Pathanamthitta, Enadimangalam, Kerala 691556, India.

Corresponding Author:
Selin Abraham
Assistant Professor, Department of Medicine, Mount Zion Medical College, Chayalode, Adoor Pathanamthitta, Enadimangalam, Kerala 691556, India.
E-mail: sowmyatshassan@gmail.com

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Abstract

Introduction: The most frequent causes of postrenal AKI in the elderly include benign prostatic hypertrophy (BPH) or prostate cancer, retroperitoneal adenopathy or malignancies, pelvic neoplasms, and neurogenic bladder. Although BPH and prostate cancer are common in older men, they cause obstruction in only a minority of cases. In elderly women, pelvic and retroperitoneal malignancies are the most frequent causes of postrenal AKI. *Methodology:* This study was conducted on 200 admitted patients who presented with Acute Kidney Injury or developed Acute Kidney Injury during the hospital stay in the Department of Medicine. The symptoms, signs and basic lab data like Routine Blood Examination for Hb, TC, DC, ESR & Platelet count: Renal function tests, Liver function tests, Serum Electrolytes & Routine Urine examination, was noted at the time of admission, during the course of hospital stay and at the date of discharge. Specific investigations like USG Abdomen, Renal Biopsy, Arterial Blood Gas analysis was done accordingly to analyze the etiology. *Results:* Pre renal conditions predominate as the cause for AKI. Post renal causes account for only 2.5% of the total. People above the age group of 50yrs was at an increased risk for the development of AKI. *Conclusion:* Septic AKI was the commonest cause of increased mortality followed by leptospirosis.

Keywords: AKI; Outcome; Leptospirosis.

Introduction

AKI can also develop from acute or rapidly progressive glomerulonephritis. Timely diagnosis and treatment of these conditions is critical to preserve renal function and avoid life-threatening complications. Diffuse proliferative forms of glomerulonephritis can be associated with infections and generally carry a good prognosis in the elderly and in the young [1,2]. Rapidly progressive (crescentic) glomerulonephritis is a fulminant presentation of glomerular disease that will lead to renal failure over days to weeks if left untreated. Evidence suggests that rapidly progressive glomerulonephritis may be more common among the elderly and carries a poorer prognosis [3]. Clinically, patients often present with AKI, hypertension, hematuria, and proteinuria. Characteristically, the urinary sediment demonstrates dysmorphic red blood cells and red blood cell casts. Serologic studies including complement levels, antinuclear antibodies (ANA), antineutrophil cytoplasmic antibodies (ANCA), antiglomerular basement membrane antibodies, cryoglobulin levels, and hepatitis B and C antibodies can be useful in suggesting the cause, although kidney biopsy is nearly universally required for specific diagnosis. Treatment, including high-dose glucocorticoids, immuno-suppressive therapy and plasmapheresis, will be dependent on the specific cause. Despite the potential for treatment associated toxicities, case series have demonstrated that elderly patients with limited comorbidities may tolerate and respond well to therapy [4].

Postrenal or obstructive AKI is more common in the aged than in the young, accounting for 9% to 30%

of cases [5]. Postrenal AKI can be categorized asaffecting either the upper urinary tract (proximal to the bladder) or lower urinary tract (obstruction occurring at the bladder outlet or urethra). Obstruction of the lowertract will affect both kidneys and diminish renal function. In contrast, unilateral uppertract obstructing processes may cause renal colic and unilateral hydronephrosis, butwill not cause deterioration in renal function if the contralateral kidney can compensate. However, if the obstruction is bilateral, is of a unilateral functioning kidney, or if there is significant underlying chronic kidney disease, upper tract obstruction canalso cause AKI.

The most frequent causes of postrenal AKI in the elderly include benign prostatic hypertrophy (BPH) or prostate cancer, retroperitoneal adenopathy or malignancies, pelvic neoplasms, and neurogenic bladder. Although BPH and prostate cancer are common in older men, they cause obstruction in only a minority of cases. In elderly women, pelvic and retroperitoneal malignancies are the most frequent causes of postrenal AKI.

Postrenal AKI may present with either complete or partial obstruction. Complete obstruction is characterized by anuria. The patient may also report flank and abdominal pain or suprapubic fullness. In contrast, the patient with partial obstruction may remain completely asymptomatic or may report similar pain symptoms, as well as voiding complaints including frequency, urgency, hesitancy hematuria, and nocturia. Urine output can be variable, ranging from oliguria to polyuria, or fluctuating between the two [6].

Due to its increased incidence in the elderly and varying presentation, the clinician must maintain a high index of suspicion for postrenal AKI. The diagnosis should especially be considered in patients with BPH or lower urinary tract symptoms, diabetes, kidney stones, abdominal or pelvic malignancies, surgeries or radiation, retroperitoneal adenopathy or neoplasms, and medication use associated with urinary retention. Lower tract obstruction is diagnosed by confirmation of urinary retention using ultrasonographic bladder scans or placement of a bladder catheter. An elevated residual bladder volume (>100–150 mL) after voiding is highly suggestive of postrenal AKI, although, some elderly patients may suffer from chronic urinary retention with elevation in the postvoid residual bladder volume in the absence of kidney dysfunction [7]. Radiographic workup for upper tract obstruction usually begins with ultrasound imaging, which is sensitive and specific in detecting obstruction [8,9]. However, ultrasonography may appear normal in patients presenting with early obstruction or with retroperitoneal processes encasing the kidneys and ureters, preventing ureteral dilation.CT can be valuable in determining the cause and level of obstruction if ultrasound fails to identify the lesion. Together, ultrasound, abdominal plain films, and CT scanning are diagnostic in most cases

Intravenous pyelography has been supplanted by CT imaging and is now only rarely required. Antegrade or retrograde pyelography, however, can be valuable in identifying the site and cause of obstruction, and provides an opportunity for therapeutic intervention. Laboratory findings are nonspecific in postrenal AKI often mimicking prerenal AKI in the early phase and intrinsic AKI later

Treatment of postrenal AKI consists of the rapid detection and relief of obstruction.This can be accomplished by placement of a bladder catheter in lower tract disease or ureteral stents or percutaneous nephrostomy tubes for upper tract disease. A brisk postobstructive diuresis frequently ensues due to water and sodium reabsorptive deficits as well as an osmotic diuresis attributable to previously retained solutes including urea. Careful monitoring of the patient's volume status and electrolytes is essential to avoid the development of volume depletion or serious electrolyte disturbances. Although use of intravenous fluids may be required, it is important to avoid overly aggressive fluid replacement that can drive further diuresis. If the obstruction has been quickly diagnosed and reversed, renal function will improve. However, in patients with a longer duration and higher grade of obstruction, renal functional recovery may be delayed, incomplete, or absent. Brisk urine output following correction of the obstruction does not always correlate with renal recovery and hence close laboratory monitoring remains necessary.

Methodology

Definition of the Study

This study has utilized the classifications called the RIFLE and AKIN. The following definitions have been utilized for the study.

Oliguria: Refers to a 24hr urine output <400ml.

Anuria: Complete absence of urine formation (<100ml/d).

Nonoliguria: Refers to urine output >400ml/d in patients with acute or chronic azotemia.

Calculation of **GFR** by Cockcroft-Gault formula

CrCl (ml/min)=(140-age (years) × weight (kg)× (0.85 if female)/72 × S.Cr (mg/dL).

Add: Acute diorrheal disease including Acute gastroenteritis.

Diaki: Drug induced Acute Kidney Injury including Aminoglycosides, Cisplatin, amphotericin B, vancomycin and others excluding NSAID's.

CIN: Contrast induced nephropathy following iodinated contrast agents.

MM/AKI: Multiple myeloma associated acute kidney injury.

CVA/AKI: Cerebro vascular accidents leading to poor intake and pre-renal failure.

NSAID/AKI: Non-steroidal anti inflammatory drug induced acute kidney injury.

HUS/TTP: Hemolytic uremic syndrome/ thrombotic thrombocytic purpura-characterized by history of recent GI infection or use of calcineurin inhibitors with the presence of schistocytes on peripheral bloodsmear, elevated LDH, anemia and thrombocytopenia.

Study Population

This study was conducted on 200 admitted patients who presented with Acute Kidney Injury or developed

Data Collection Tool: Structured interview schedule.

Study Details

Each case was individually seen and data was collected according to the prepared performa,after obtaining informed consent for participation in the study.

The symptoms, signs and basic lab data like Routine Blood Examination for Hb, TC, DC, ESR & Platelet count: Renal function tests, Liver function tests, Serum Electrolytes & Routine Urine examination, was noted at the time of admission, during the course of hospital stay and at the date of discharge. Specific investigations like USG Abdomen, Renal Biopsy, Arterial Blood Gas analysis was done accordingly to analyse the etiology

Conservative management in the form of removal of precipitating factors for prerenal failure, fluid restriction and use of renoprotective drugs like ACE inhibitor sand interventional treatment in the form of Haemo-dialysis or Peritoneal-dialysis was instituted as needed.

Complications if any like sepsis and worsening of renal reserve was studied according to clinical, radiological and biochemical evidences. Patients was followed up at 3 weeks, 3 months and 6 months after discharge with S. Creatinine, B.Urea, Urine examination results.

Original Research Paper

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Clinical Presentation of Renal Injury at a Tertiary Care Hospital

Cijo John[1], Selin Abraham[1]

Author's Affiliation:
[1]Assistant Professor, Department of Medicine, Mount Zion Medical College, Chayalode, door Pathanamthitta, Enadimangalam, Kerala 691556, India.

Corresponding Author:
Selin Abraham
Assistant Professor, Department of Medicine, Mount Zion Medical College, Chayalode, Adoor Pathanamthitta, Enadimangalam, Kerala 691556, India.
E-mail: sowmyatshassan@gmail.com

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Abstract

Introduction: AKI occurs predominantly in urban intensive care units and is associated with multiorgan failure and sepsis, high mortality, and occurrence in older populations. While cases of AKI in urban areas of the developing world have similar characteristics to those in the developed world, AKI in rural regions commonly develops in response to a single disease and specific conditions (e.g. gastroenteritis) or infections (e.g. severe malaria, leptospirosis, or hemolytic–uremic syndrome) and in younger otherwise healthy individuals. *Methodology*: Acute Kidney Injury,the major inclusion and exclusion criteria were identified. Data regarding etiology,clinical features, outcome to treatment were collected over a period of one year from Jan 2011 to Jan 2012 in total of 200 admitted patients. The outcome of the study was analyzed and documented. *Results*: The youngest person enrolled was 20 yrs and oldest was 86 yrs of age.Amongst the pre renal conditions Acute diarrheal diseases are the commonest. Oliguria dominate as the most common presenting symptom in patients with AKI. *Conclusion*: Acute kidney Injury is commonly seen in men than in women below the age group of 50 yrs.

Keywords: Acute Renal Failure; Glomerulonephritis; AKI.

Introduction

The evolution of the term 'acute renal failure' dates back to 1802, when William Heberden first described it as *IschuriaRenalis*. Since then there are over 35 official definitions of the term; these include: *Acute Bright's disease, war nephritis andcrush syndrome*.It wasn't until 1951 that Homer W. Smith introduced the term 'Acute Renal Failure' [1].

Today, Acute Kidney Injury (AKI) is considered the correct nomenclature for the clinical disorder formerly termed 'Acute Renal Failure'(ARF). AKI, is a protean syndrome of varied severity. It is characterized by a rapid (hours to days) decline in the glomerular filtration rate (GFR) and retention of nitrogenous waste products such as blood urea nitrogen (BUN) and creatinine. Acute kidney injury (AKI) has become increasingly prevalent in both developed and developing countries, and is associated with severe morbidity and mortality [2].

In developed countries, AKI occurs predominantly in urban intensive care units and is associated with multiorgan failure and sepsis, high mortality, and occurrence in older populations. While cases of AKI in urban areas of the developing world have similar characteristics to those in the developed world, AKI in rural regions commonly develops in response to a single disease and specific conditions (e.g. gastroenteritis) or infections (e.g. severe malaria, leptospirosis, or hemolytic–uremic syndrome) and in younger otherwise healthy individuals. Many causes of AKI in rural settings, such as diarrhea, poisoning, malaria, or septic abortion, can be prevented by interventions at the individual, community, and regional levels. Treatment with dialysis is often unavailable or too costly in developing regions, so there must be community-wide efforts to eradicate causes of AKI, expedite diagnosis, and aggressively manage prerenal conditions and specific infections [3].

Despite several advances in our treatment and

understanding of the pathogenesis of acute kidney injury (AKI), many aspects in this field remain subject to controversy, confusion, and lack of consensus. One of these important aspects is the definition of AKI. To make consensus-based recommendations and delineate key questions for future studies,the Acute Dialysis Quality Initiative (ADQI) workgroup identified a definition/classification system for AKI [4].

Accordingly, a multilevel classification system was proposed, in which the complete spectrum of acute renal dysfunction could be included, such as Risk of

the Department of Medicine, and was aimed at identifying the more common causes, clin[illegible] and outcome of treatment of these pati[illegible] with Acute Kidney Injury above 1[illegible] Approval from ethical committee and w[illegible] from patients or his/her relatives were[illegible]

Acute Kidney Injury, the major i[illegible] exclusion criteria were identified. Dat[illegible] etiology, clinical features, outcome to treatment were collected over a period of one year from Jan 2011 to Jan 2012 in total of 200 admitted patients. The outcome of the study was analyzed and documented.

Table 2: Gender distribution between Age groups

Age	Gender		Total
	Male	Female	
< 50 yrs	62	44	106
	55.40%	50.00%	53.00%
>= 50 yrs	50	44	94
	44.60%	50.00%	47.00%
Total	112	88	200

Chi Square: 0.568; P > 0.05

Most patients presented with oliguria as the main symptom.

Table 3: Presenting complaints

Presenting Complaints	Frequency	Percent
None	68	34.0
Oliguria	99	49.5
Anuria	33	16.5
Total	200	100

Table 4: Age wise distribution of presenting complaint

Presenting Complaints	Age		Total
	< 50 yrs	>= 50 yrs	
None	45	23	68
	42.50%	24.50%	34.00%
Oliguria	46	53	99
	43.40%	56.40%	49.50%
Anuria	15	18	33
	14.20%	19.10%	16.50%
Total	106	94	200

Chi Square: 7.191; P < 0.05

Table 5: Etiology and Frequency

Diagnosis	Frequency	Percent
ADD AKI	34	17.0
AGN AKI	19	9.5
CIN	12	6.0
CVA AKI	9	4.5
DIAKI	12	6.0
HUS/TTP	9	4.5
Lepto/AKI	34	17.0
LVF AKI	10	5.0
MM AKI	5	2.5
NSAID AKI	25	12.5
Obst. AKI	5	2.5
Sepsis AKI	22	11.0
Viper Bite	4	2.0
Total	200	100

Discussion

The predominant symptom with which the patients presented was Oliguria (49.5%). 16.5% patients had Anuria as their presenting symptom and 34% of patients did not have either of these symptoms. Oliguria is defined as a urine output that is less than 1 mL/kg/h in infants, less than 0.5 mL/kg/h for six consecutive hours in children and adults, or<400ml/d. The beginning and ending supportive therapy (BEST) kidney investigators highlighted the fact that oliguria was more common in septic AKI and viper bite induced AKI.

It is important to acknowledge, however, that at

least half of all cases of AKI are nonoliguric. This was highlighted by Liano. F, Pascual. M et al in their study on the epidemiology of acute renal failure,in a community based study in Spain. Thus, healthy urine output does not ensure normal renal function. Rarely, ARF comes to the attention of the clinician because of symptoms of uremia (*eg*, anorexia, nausea, vomiting, confusion, pruritus) or laboratory findings compatible with renal failure (metabolic acidosis, hyperkalemia, hyperphosphatemia, hypocalcemia, hyperuricemia, hypermagnesemia, anemia). This finding is also in accordance with the above studies.

It was also observed that oliguria was the predominant symptom in age group above 50 yrs. 56.40% of patients above the age of 50 counted oliguria as their predominant symptom. This finding was statistically significant $P<0.05$. This finding might be due to the fact that kidneys give up early as a fall in GFR as age advances.

associated AKI (112 patients out of 200) (56%). Septic AKI had an equal distribution among age and gender. Post renal failure was more in the age group more than 50 yrs (100%).CIN was also seen in increased incidence in age group more than 50 yrs (11 cases out of 12) (91%). Elderly patients may be at increased risk for true volume depletion due to changes in body composition with aging, leading to decreased total body water as a fraction of body weight, and from an increased burden of comorbid disease [7]. Non steroidalanti inflammatory drugs (NSAIDs), which are used by approximately 10% to 25% of the elderly [8], inhibit production of vasodilatory prostaglandins. NSAID use has been associated with a threefold higher risk of AKI in the general population, (Huerta et al, 2005), and an absolute risk of prerenal AKI of 13% in a nursing home cohort (mean age 87 years)(French study group on acute renal failure).

Postrenal or obstructive AKI is more common in