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

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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 Acute Kidney Injury during the hospital stay in the Department of Medicine.

Study Period: One year.

Study Design: Prospective observational study.

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.

Results

This study consisted of 112 males and 88 females. Males contributing 56% compared to 44% of females.

Table 1: Gender distribution

Gender	Frequency	Percent
Male	112	56
Female	88	44
Total	200	100

Table 2: USG Abdomen

USG Abdomen	Frequency	Percent
Normal	171	85.5
Bladder Stone	1	0.5
BPH	3	1.5
Cystitis	5	2.5
HM+	18	9
Pyelonephritis	1	0.5
Ure. Stone	1	0.5
Total	200	100

Table 3: Diagnosis and conservative treatment

Diagnosis	Treatment: Conservative		Total
	No	Yes	
ADD AKI	3	31	34
	6.30%	20.40%	17.00%
AGN AKI	-	19	19
	-	12.50%	9.50%
CIN	2	10	12
	4.20%	6.60%	6.00%
CVA AKI	-	9	9
	-	5.90%	4.50%
DIAKI	2	10	12
	4.20%	6.60%	6.00%
HUS/TTP	-	9	9
	-	5.90%	4.50%
Lepto/AKI	12	22	34
	25.00%	14.50%	17.00%
LVF AKI	-	10	10
	-	6.60%	5.00%
MM AKI	-	5	5
	-	3.30%	2.50%
NSAID AKI	6	19	25
	12.50%	12.50%	12.50%
Obst. AKI	2	3	5
	4.20%	2.00%	2.50%
Sepsis AKI	17	5	22
	35.40%	3.30%	11.00%
Viper Bite	4		4
	8.30%		2.00%
Total	48	152	200

Chi Square: 71.389; $P < 0.001$

Table 4: Diagnosis and hemodialysis

Diagnosis	Treatment: HD		Total
	No	Yes	
ADD AKI	31	3	34
	19.70%	7.00%	17.00%
AGN AKI	19		19
	12.10%		9.50%
CIN	10	2	12
	6.40%	4.70%	6.00%
CVA AKI	9		9
	5.70%		4.50%
DIAKI	10	2	12
	6.40%	4.70%	6.00%
HUS/TTP	9		9
	5.70%		4.50%
Lepto/AKI	22	12	34
	14.00%	27.90%	17.00%
LVF AKI	10		10
	6.40%		5.00%
MM AKI	5		5
	3.20%		2.50%
NSAID AKI	19	6	25
	12.10%	14.00%	12.50%
Obst. AKI	3	2	5
	1.90%	4.70%	2.50%
Sepsis AKI	9	13	22
	5.70%	30.20%	11.00%
Viper Bite	1	3	4
	0.60%	7.00%	2.00%
Total	157	43	200

Chi Square: 47.954; $P < 0.001$

Table 5: Outcome and Diagnosis

Diagnosis	Cured	Outcome Relieved	Dead	Total
ADD AKI	34	-	-	34
	22.50%	-	-	17.00%
AGN AKI	9	10	-	19
	6.00%	28.60%	-	9.50%
CIN	10	2	-	12
	6.60%	5.70%	-	6.00%
CVA AKI	9	-	-	9
	6.00%	-	-	4.50%
DIAKI	11	1	-	12
	7.30%	2.90%	-	6.00%
HUS/TTP	-	9	-	9
	-	25.70%	-	4.50%
Lepto/AKI	27	4	3	34
	17.90%	11.40%	21.40%	17.00%
LVF AKI	10	-	-	10
	6.60%	-	-	5.00%
MM AKI	-	5	-	5
	-	14.30%	-	2.50%
NSAID AKI	23	2	-	25
	15.20%	5.70%	-	12.50%
Obst. AKI	3	2	-	5
	2.00%	5.70%	-	2.50%
Sepsis AKI	12	-	10	22
	7.90%	-	71.40%	11.00%
Viper Bite	3	-	1	4
	2.00%	-	7.10%	2.00%
Total	151	35	14	200

Chi Square: 163.616; P < 0.001

Discussion

Out of the 200 patients studied 152 were treated conservatively by fluid management and antibiotics. Of the 152 patients managed conservatively, 20.40% were in the acute diarrheal group, 14.5% were leptospirosis induced AKI, 12.5% were NSAID induced, 12.5% were due to acute glomerulonephritis (P=<0.001). The higher incidence of conservative management in ADD/AKI was that the patients were identified early and appropriate treatment was instituted and that resulted in the reversal of AKI. Similarly patients with history and clinical features suggestive of leptospirosis were aggressively managed resulting in the regression of the disease. Only patients presenting late were at an increased risk of progression of the disease.

NSAID intake was more common in the elderly and those that were managed conservatively were largely devoid of confounding factors that lead to a rapid progression of symptoms. NSAID intake led to dialysis in patients whose renal function was already compromised. Almost all cases of acute post streptococcal glomerulonephritis were managed conservatively and improved. Out of the 200 patients 48 were subjected to Renal replacement therapy (RRT). Most patients subjected for hemodialysis were having septic AKI (30.20%), lepto/AKI (27.90%) and viper bite (7%). (P=<0.001).

Outcome profile was studied according to age group <50yrs and >50yrs, gender and to the mode of treatment given to the patient. Of the 200 patients 151 were cured (75.50%). 35 (17.50%) were relieved of there symptoms but had to undergo more than one hemodialysis sessions. 14 (7%) had died during the study period due to complications of AKI.

The major cause of death in this study was sepsis induced AKI. 10 patients (71.40%) out of the the 14 died (P=<0.001). Of the 22 patients studied with sepsis induced AKI, 13 patients underwent hemodialysis (63.6%)(P=<0.05). This finding was in accordance with the study done by the BEST investigators where they showed a mortality rate of 70.2% in hospitals. The cause of such a high rate of mortality was due to ischaemia-reperfusion injury, direct inflammation injury, coagulation, endothelial dysfunction and apoptosis [10]. Sepsis induced AKI did not respect gender nor age. Mortality is certainly higher among

people more than 50 yrs of age due to preexisting conditions like diabetes, low serum albumin, atherosclerosis which are more common in the elderly.

Another cause of increased mortality in this study is leptospirosis induced AKI. 34 patients presented with leptospirosis and 3 (21.40%) died (P=<0.001). 12 patients required dialysis(27.90%)(P=<0.05). Leptospirosis is endemic to Alappuzha and is a major cause of mortality in young as well as older persons. Early recognition and timely action usually saves the person.

Out of the 43 persons requiring dialysis, 3(7%) were bitten by Russels viper, which is a common poisonous snake found in close proximity to human dwelling and in farm lands. One person died of complications (9.10%) (P=<0.05). There were 5 patients of multiple myeloma(20%) who developed AKI and none of them were subjected to dialysis. There were a total of 9 patients of Hemolytic Uremic syndrome who developed AKI and none of them required hemodialysis.

Conclusion

Septic AKI was the most common cause for hemodialysis in patients with AKI, followed by leptospirosis.

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