Original Article

Causes and Outcome of Acute Kidney Injury: Gezira Experience

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Abstract

Introduction: A precise operational definition of acute kidney injury remains elusive. Conceptually, acute kidney injury is defined as the loss of renal function, measured by decline in glomerular filtration rate, developing over a period of hours to days. Clinical manifestations of acute kidney injury (AKI) are highly variable; in some patients, the only manifestation may be biochemical abnormalities on routine blood sampling, while other patients will present with overt uremic signs or symptoms.

Objectives: We evaluated the aetiology and the clinical outcome of all patients admitted to Gezira Hospital for Renal diseases and Surgery with AKI.

Methods: Clinical data of patients admitted from January 2008 through December 2009 were reviewed and analysed.

Results: Total number of patients included in this study was 122. Out of them 39.3% (48) were females. The mean age was 51.34 (SD 22.18) years. The most common causes of acute kidney injury are ischemic acute tubular necrosis, followed by sepsis and obstruction respectively. Mortality rate was 18.9%.

Conclusion: Mortality rate of AKI in our set up was 18.9%.

Key words: glomerular, tubular necrosis, creatinine.

Descriptions of acute kidney injury (AKI) are dated back to the ancient Greek period[1], when the diagnosis was possible only by observing a reduction in urine volume. The modern day conception of AKI has evolved alongside developments in pathology and clinical biochemistry, which have permitted clinicopathologic correlations and early diagnosis[2]. Descriptions of AKI from the early 20th century centered around specific conditions, such as crush injuries[3], war nephritis[4], and falciparum malaria[5]. Sir William Osler in 1912 described several recognizable causes of AKI under the heading of "acute Bright's disease," including sepsis, pregnancy, burns, and toxins[6].

Conceptually, AKI is defined as the loss of renal function, measured by a decline in glomerular filtration rate (GFR), developing over a period of hours to days.

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Clinically, AKI is manifested by the retention of creatinine, urea, and other metabolic waste products that are normally excreted by the kidney. Clinical manifestations of AKI are also highly variable. In some patients, the only manifestation may be biochemical abnormalities on routine blood sampling, while other patients will present with overt uremic signs or symptoms. Recent epidemiological studies have demonstrated wide variations in the aetiology and risk factors associated with AKI[7-10].

Limited data are available regarding the causes and outcome of acute kidney injury in Sudan in general and Gezira State in particular[11].

Materials and Methods: Data of all patients admitted to Gezira Hospital for Renal diseases and Surgery with AKI from January 2008 to the December 2009 were collected.

AKI was defined in this study as an increase in serum creatinine of 0.5 mg/dl or more with a baseline serum creatinine < 1.5 mg/dl or a percentage increase in the serum creatinine concentration of ≥ 50%.

Relevant data were collected from history,

and physical examination, and laboratory investigations that included urinalysis, blood urea, serum creatinine and serum electrolytes. Ultrasonography of the kidneys and bladder were performed on all patients, while CT-scan of the kidneys and renal biopsy were performed when indicated.

Exclusion criteria: Patient with hepato-renal syndrome, patients younger than 18 years, and renal transplanted patients were excluded. The data were analysed using Statistical Package of Social Sciences (SPSS)

Results: *A total of 122 patients were included in this study 60.7% (74) were males. The mean (±SD) age was 51.34 (±22.18) years. The mean (±SD) of hospitalization period was 9.54 (±6.76) days. The means (±SD) serum creatinine and blood urea on admission were 6.89 (±5.93) and 146.25mg/dl (±81.17) respectively while the means of serum creatinine and blood urea at discharge were 2.67 (±2.10) and 71.12 (±40.71) respectively. The mean (±SD) of serum sodium level at presentation was 130.67 (±7.76). 40 (32.78%) patients presented with hyponatremia. The mean (±SD) serum potassium level at presentation was 4.01 (±1.07). 34 (27.87%) patients had hypokalemia and 19 (15.57%) had hyperekalemia.*

Table 1 summarizes the major causes of AKI. 62(50.82%) were on regular haemodialysis, and only one patient had peritoneal dialysis**.** Three patients refused renal replacement therapy and 56(45.9%) were treated conservatively.

Table 1- Causes of AKI

		Frequency
Renal	Acute interstitial nephritis	4 (3.3%)
	GN	4 (3.3%)
	Snake bite	11 (9%)
	Ischemic ATN	26 (21.3%)
	Hair dye	11 (9%)
Pre renal	Volume depletion	19 15.6%)
	Sepsis	11 (9%)
Post renal	Obstruction	34(27.9%)
	Total	122(100%)

GN= Glomerulonephritis

Table 2 shows the outcome of AKI. Fig1 summarizes the causes of AKI in deceased patients, the mean age in this group was 63.91 (SD 16.04), 63.64% (14) were males; the mean of hospitalization in this group was 4.1 (SD 4.92) days.

Table 2- The Outcome of AKI

	Frequency
Recovery	74(60.7%)
CKD	18(14.8%)
DAMA	7(5.7%)
Death	23(18.8%)
Total	122(100%)

CKD=Chronic kidney disease
DAMA= Discharge against medical advice

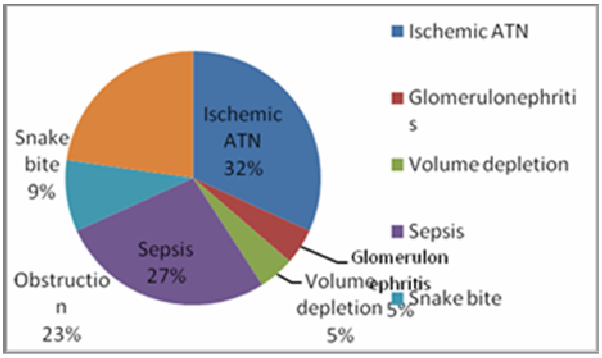

Fig 1 - Causes of AKI in Deceased Patients

Table 3 summarizes the mortality rate according to cause of AKI.

Discussion:-

Lacking clear agreement regarding the definition of acute kidney injure make studying this topic difficult[12].

In our study we found that the most common cause of AKI was ischemic acute tubular necrosis (ATN), followed by sepsis and obstruction respectively. This is comparable to the multi-centric report by Cruz et al[13] In this study the mortality rate was 18.9%, which is again consistent with that reported by Cruz [13]. However, we found that mortality rate was 54.55% in patients who suffered from sepsis as a cause of AKI. This is also consistent with other reports[14]. Lopes et al conducted retrospective study from 2003 to

2007 to evaluate the capacity of this system in predicting in-hospital mortality of septic patients and found that 31.4% of those patients had AKI with a mortality rate of 25.3% in patients with sepsis and AKI[15]. The prevalence of AKI secondary to sepsis in Sudan is not known.

Renal failure is an important complication of snake bite and a major cause of mortality. In this study snake bites had precipitated AKI with mortality rate of 18%**.** This is in keeping with reports from Turkey[16].

Table -3- Mortality Rate According to Cause of AKI

		Frequency	No Deceased patients
Renal	Acute interstitial nephritis	4	0
	Glomerulonephritis	4	1
	Snake bite	11	2
	Ischemic ATN	26	7
	Hair dye	11	0
Pre- renal	Volume depletion	19	1
	Sepsis	11	6
Post renal	Obstruction	34	5
	Total	122	22

In this study the survival rate of patients with AKI secondary to hair dye [Paraphenylenediamine (PPA)] ingestion was 100% in contrast to Kaballo's report mortality of 40%[11] these could be explained by early presentation to our nephrology unit and early renal replacement therapy (RRT).

The optimal timing of the initiation, the modality, and the dose of renal replacement therapy in AKI are still controversial[17,18].

In this study 14.8% of the patients ended with chronic kidney disease(CKD); Coca conducted meta-analysis that included 48 studies that contained a total of 47,017 participants and concluded that the incidence rate of CKD after an episode of AKI was 7.8 events/100 patient-years, and the rate of end-stage kidney disease was 4.9 events/100 patient-years[19] while Cartin-Ceba et al found that 4.9% of AKI progressed to end stage renal disease (ESRD)[20] and Schiffl et al stated that 19-31% may develop chronic kidney disease[21]. Wald reported that the incidence rate of chronic dialysis was 2.63 per 100 person-years among individuals with acute kidney injury[22].

Conclusion: Causes of AKI in our study were more or less similar to other places while the mortality rate of AKI in our set up was 18.9%.

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