

## Patterns of Acute Kidney Injury in Banha Teaching Hospital

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### ABSTRACT

**Background:** Acute kidney injury (AKI) is a common and preventable condition that affects 21% of adults and 33% of children globally, with higher mortality rates among the elderly and those with multiple comorbidities in developed countries. The lack of data on AKI in Africa may be due to limited access to renal replacement medication, and identifying high-risk individuals is crucial to avoiding the development of chronic kidney disease. **Aim:** The study aimed to evaluate patterns of AKI in Banha Teaching Hospital. **Patients and Methods:** The study collected data on patients aged 18 and older with AKI, including epidemiological, anamnestic, clinical, biological, imaging, treatment, and outcome data. AKI was defined according to the KDIGO classification and classified according to the mechanism of occurrence. Total and partial recovery was defined based on the evolution of renal function. **Results:** The study found that diabetes, coma, high creatinine levels, and sepsis were positively correlated with death occurrence. Gender, blood pressure, heart disease, and certain types of AKI did not show significant correlation with death occurrence.

**Conclusion:** AKI is frequent among the population and often fatal, but can potentially be prevented by expanding extracellular volume to improve renal blood flow and decrease exposure to nephrotoxic agents.

**Keywords:** Acute Kidney Injury, Patterns, Epidemiology, Mortality

### INTRODUCTION

A medical condition known as acute kidney injury (AKI) might have been brought on by one of a number of different factors. Acute kidney injury (AKI) is characterized by a sudden and reversible decline in renal function. One of the telltale signs of AKI is an elevation in plasma creatinine levels. This condition, which may be avoided simply and is quickly treated, is quite common in hospitals and other in-patient institutions. According to **Hoste et al.**<sup>(1)</sup>, AKI affects 21% of adults and 33% of children all over the globe. The mortality rate for adults is 23%, while the mortality rate for children is just 13%. AKI may occur in one of two ways: either before hospitalization in the community or as a complication when the patient is already in the hospital. The majority of those who suffer from community-acquired acute kidney damage are younger individuals, and the condition is more prevalent in nations that have less medical resources<sup>(2)</sup>. According to **Yang**,<sup>(3)</sup> however, hospital-acquired AKI is more common in developed countries, especially among the elderly and those who have a number of comorbidities.

Epidemiologically speaking, it is difficult to determine the prevalence of AKI in Africa. Nigeria, which is located in Sub-Saharan Africa, is the only nation having data on the prevalence of AKI, according to a 2015 global meta-analysis of 154 nations<sup>(4)</sup>. The lack of knowledge regarding AKI is likely due to the lack of drugs available to treat renal replacement therapy. AKI is more common in hospitals than in the general population, according to several studies<sup>(5)</sup>.

Identifying individuals at risk of developing AKI, whether acquired in the community or in a hospital, is crucial. Following an episode of AKI, inflammatory lesions may progress to renal fibrosis, leading to the development of chronic kidney disease. Therefore, it is essential to identify high-risk individuals<sup>(2)</sup>.

The present study aimed to evaluate patterns of acute kidney injury in Banha Teaching Hospital

### PATIENTS AND METHODS

This was a study done in in Benha University Hospital between the years August 2022 – February 2023 The study was conducted on 320 subjects. All patients were monitored in the Internal Medicine and Dialysis units of the Benha Medical Schools.

Any adult patient who was diagnosed with AKI was qualified for participation into the study. Those who had chronic renal illness, regardless of whether or not they were receiving hemodialysis, as well as those who did not have acute kidney injury, were excluded from the study. For each patient who participated in the study, the following information was collected: Information on the patient's age, gender, and residency that was used for epidemiological purposes. Data on the patient's anamnestic history, which includes information on the patient's medical history such as hypertension, diabetes, and heart disease, as well as the patient's medications and the cause for hospitalization. Data from clinical examinations, including the patient's blood pressure and temperature at the time of admission, as well as their state of awareness, hydration status, and rate of diuresis. Information pertaining to the patient's biology, such as the blood count, serum creatinine level, and plasma urea level. Imaging data, such as renal ultrasounds, which may offer further information about the kidneys are examples of this kind of data. The treatment that was received, which may have an effect on how the patient ultimately fared. Data about the patient's outcomes, such as whether or not they were released, died, were discharged against the recommendation of their medical providers, or were moved to another hospital.

The operational definitions adopted were as follows: The KDIGO categorization method was used throughout the study investigation to appropriately characterize cases of acute kidney injury (AKI). The severity of AKI was determined using baseline creatinine levels, which served as the foundation for

categorization. Stage I was defined as a rise in creatinine levels of 1.5 times the baseline level, Stage II as an increase of 2 times the baseline level, and Stage III as an increase of 3 times the baseline level <sup>(6)</sup>. **Levey et al.** <sup>(7)</sup> first published the baseline creatinine levels norms. A number of evaluations, including interviews, physical exams, blood tests, and ultrasounds, were used to determine the pre-renal, renal, and post-renal causes of AKI. AKI was then classified using the renal, pre-renal, and post-renal categories based on its underlying mechanism of occurrence, which encompassed pre-renal, renal, and post-renal causes.

Diarrhea, vomiting, hemorrhage, heart failure, cirrhosis, sepsis, nonsteroidal anti-inflammatory drug (NSAID) use, diuretic use, and angiotensin-converting enzyme (ACE) inhibitor use have all been linked to the development of functional AKI in individuals. If the patient had been given nephrotoxic medications like aminoglycosides or traditional toxicants and their urea to creatinine ratio was less than 10, the doctor would diagnose them with an organic form of AKI. Ultrasound evidence of pyelocaliceal cavity dilatation on the left or right side of the body was used to diagnose obstructive AKI. Following and based on study of **Tia et al.** <sup>(2)</sup> According to the evolution of renal function, total recovery was defined by the return to normal renal function i.e. serum creatinine  $\leq$  13 mg/l, partial recovery was defined by a 50% decrease of the maximum serum creatinine value.

A blood creatinine level of 13 mg/l or below was used as the threshold for recovery in both models.

**Ethical Approval: Prior to participating in the study, all subjects provided written consent, acknowledging the potential risks associated with their involvement. The Ethics Board of Banha University approved the research project, ensuring adherence to guidelines set forth by the World Medical Association's Declaration of Helsinki concerning studies involving human subjects.**

**Statistical Analysis:** Data were analyzed using IBM SPSS 24 (May 2016). Data were presented as frequency and percentage. Spearman's correlation was calculated. Results with a p-value of less than 0.05 were considered significant. Quantitative data were presented as mean and standard deviation (SD) and distribution of data was detected by Shapiro-Wilk Test, if data is normally distributed, data were compared by t-test if not we used Mann Whitney U test. Qualitative data were presented as frequency and percentage and were compared by Chi2 test. Hazard Ratio was used for survival analysis to compare the risk of an event occurring between two groups. If the P-values were less than 0.05, we deemed the results statistically significant.

**RESULTS**

The study included 320 subjects, with a majority being over the age of 55 and male. Most of the subjects lived in urban areas (Table 1).

**Table (1):** Demographic data of included subjects (n=320)

	Parameters	Number	Percentage
Age	• 15 - 24	20	6.25
	• 25 - 34	45	14.06
	• 35 - 44	29	9.06
	• 45 - 54	49	15.31
	• 55 - 64	75	23.44
	• $\geq$ 65	102	31.88
Gender	• Male	187	58.44
	• Female	133	41.56
Residence	• Urban	219	68.44
	• Rural	101	31.56

The most common cause of admission was fever, and the most prevalent comorbidity was hypertension. The majority of patients complained of fever, and physical signs included high blood pressure, oliguria, and normal urine output (Table 2).

**Table (2):** Clinical data of AKI patients (n=320)

Parameters	Number	Percentage
Cause of admission		
• Fever	75	23.44
• Coma	65	20.31
• Adynamia	25	7.81
• Oedema	23	7.19
• Dyspnea	22	6.88
• Cardiac failure	20	6.25
Comorbidities		
• Hypertension	138	43.13
• Heart disease	56	17.5
• Diabetes	55	17.19
• Gastritis	26	8.13
• Sickle cell disease	17	5.31
• Herbal remedies exposure	36	11.25
• NSAID intake	7	2.19
Complaints		
• Fever	222	69.38
• Vomiting	88	27.5
• Diarrhea	45	14.06
• Edema	39	12.19
• Digestive hemorrhage	10	3.13
Physical signs		
• Fever	217	67.81
• High blood pressure	125	39.06
• Tachycardia	52	16.25
• Anuria	3	0.94
• Oliguria	149	46.56
• Normal urine output	168	52.5
• Hypovolemia	71	22.19
• Cutaneous pallor	70	21.88
• Ascites	52	16.25

NSAID: Non-Steroidal Anti Inflammatory Drug

Based on the KDIGO classification, the majority of patients had AKI stage 1. In terms of type, most of the cases were functional (Table 3).

Table (3): Severity and type of acute renal failure (n=320)

Diagnosis of acute kidney injury	Number	Percentage
Severity of AKI (KDIGO)		
• AKI stage 1	204	63.75
• AKI stage 2	71	22.19
• AKI stage 3	45	14.06
Type of AKI		
• Functional	298	93.12
• Organic	22	6.88

AKI: Acute Kidney Injury

The majority of patients died, while 36.56% were discharged on medical treatment (Table 4).

Table (4): Outcomes of AKI patients (n=320)

Outcome	Number	Percentage
Death	145	45.31
Discharge on medical treatment	117	36.56
Discharge	30	9.38
Transferred	28	8.75

The most common cause of death was cardiorespiratory arrest (Table 5).

Table (5): Cause of death among included subjects (n=320)

Cause of death	Number	Percentage
Cardiorespiratory arrest	37	25.52
Stroke	19	13.1
Cardiovascular shock	17	11.72
Hemodynamic shock	19	13.1
Hypovolemic shock	9	6.21
Sepsis	16	11.03
Coma	13	8.97
Respiratory distress	14	9.66
Acute pulmonary edema	1	0.69

The results showed that diabetes, coma, creatinine levels within the range of 30-60 mg/l, creatinine levels > 60 mg/l, and sepsis were positively correlated with death occurrence. Gender, blood pressure, heart disease, organic and functional AKI, and also creatinine levels in the range of 14-30 mg/l did not show a significant correlation with death occurrence (Table 6).

Table (6): Correlation between different parameters and death occurrence (n=320)

	Death	
	r	P. Value
Male	-0.089	0.443
Female	0.089	0.443
High blood pressure	-0.131	0.192
Diabetes	0.229	0.012*
Heart disease	-0.029	0.779
Coma	0.447	<0.0001*
Non coma	-0.447	<0.0001*
Creatinine [14- 30] mg/l	0.067	0.308
Creatinine [30 - 60] mg/l	0.253	0.003*
Creatinine > 60 mg/l	0.442	<0.0001*
Sepsis	0.338	<0.0001*
No sepsis	-0.338	<0.0001*
Functional	-0.185	0.06
Organic	0.071	0.434

r: Pearson Correlation, \*: Significant

## DISCUSSION

The epidemiological profile of AKI in our study is explained by the cross-sectional study that was carried out. Patients whose socioeconomic conditions were precarious and who would have had difficulty obtaining medical care were given priority in the investigation's major focus area. Variables about the patient, especially their advanced age, may be used as reliable indicators of their likelihood of developing AKI. The similar result was reached by **Kellum et al.** <sup>(5)</sup>, who found that older individuals constitute a significant portion of those who are diagnosed with AKI.

The development of much comorbidity in addition to a decreased renal reserve both contribute to an increased sensitivity to nephrotoxic causes in elderly persons, which is defined as those who are older than 70 years old. People over the age of 60 have a continuous loss of renal reserve (about 0.75 ml/1.73m<sup>2</sup> per year starting at age 30), which is partially compensated for by a drop in muscle mass. This loss of renal reserve begins at age 30 and continues until people reach the age of 60. After the age of 30, this loss begins to take place. This rate typically decreases to 125 ml/min when individuals are young adults, 80 ml/min when individuals are aged 60, and 60 ml/min when individuals are aged 80. The average glomerular filtration rate for young persons is 125 milliliters per minute (ml/min). The study that was conducted by **Selmi et al.** <sup>(8)</sup> found that elderly persons are also more prone to suffer volume depletion as a consequence of a lessened sensation of thirst as well as a lower ability to maintain ideal salt levels and maximum urine concentration.

**Fuhrman et al.** <sup>(9)</sup> observed that males had a greater prevalence of acute kidney injury (AKI), which

is consistent with the results of the overwhelming majority of studies. There was no indication of obstructive nephropathy that we could uncover; functional AKI came in first, followed by organic AKI in second place. Changes in renal function that are not accompanied with structural damage are one of the hallmarks of functional acute kidney injury (AKI). If this hypoperfusion is not addressed as rapidly as it should be, it may result in ischemic acute tubular necrosis (ATN), which is a kind of kidney damage known as organic acute kidney injury (AKI). The study that was conducted by **Tia *et al.***<sup>(2)</sup> found that ischemic ATN and functional AKI both represent distinct stages of the symptoms that are caused by renal hypoperfusion.

By addressing the extra-renal factors that are responsible for renal hypoperfusion, it is feasible to correct pre-renal azotemia. This occurs when there is decreased blood flow to the kidneys. It is feasible to restore renal blood flow during the functional period by increasing extracellular volume or by making use of vasodilators; any of these approaches may still correct renal function. According to **Kanbay *et al.***<sup>(10)</sup>, even after AKI has been treated, organ damage continues to be present, and restoration of perfusion does not improve renal function.

The factors that enhanced the chance of mortality among our patients were coma, levels of creatinine ranging from 30 to 60 mg/l, levels of creatinine that were more than 60 mg/l, and infection. Infection and/or sepsis were the primary contributors to the development of acute kidney injury (AKI), which is the medical term for kidney damage that occurs suddenly. The mortality rate that is related with septic acute kidney injury (AKI), as stated by **Srisawat *et al.***<sup>(11)</sup>, might be as high as 74.5 percent.

After suffering from septic acute kidney injury (AKI), a patient may have renal hypoperfusion due to a variety of different causes. Alterations in systemic hemodynamics, such as hypotension, renal hemodynamic abnormalities that lead to renal ischemia, and disruptions in the distribution of blood flow to the kidneys are all factors that might contribute to this condition. Antibiotic toxicity and inflammatory cell infiltration into the renal parenchyma are two additional potential causes of acute kidney damage. Inflammatory cells in the renal parenchyma have been hypothesized to have a role in the onset of acute kidney injury<sup>(12)</sup>.

**Silver and Chertow**,<sup>(13)</sup> found that only 20% of fatalities in their sample were caused by severe AKI.

## CONCLUSION

Acute kidney injury (AKI) is a common occurrence and can have severe consequences, leading to renal failure and potentially even death. One potential strategy to prevent AKI is to increase the extracellular space, which can result in increased urine output, decreased activation of vasoconstrictor receptors, and improved

blood flow to the kidneys. These changes can reduce the body's exposure to nephrotoxic substances, thereby potentially reducing the risk of AKI.

## DECLARATIONS

- **Consent for publication:** I attest that all authors have agreed to submit the work.
- **Availability of data and material:** Available
- **Competing interests:** None
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- **Conflicts of interest:** no conflicts of interest.

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