Acute Kidney Injury in Patients of Emergency and Surgical Intensive Care Units: Incidence and Risk Factors

Samia Abdelrahman El Wakeel, Khaled Mohamed El Sayed,
Olfat Abdelmoniem Ibrahem, Sherif Omar Hasan Ali
Department of Anesthesia and Surgical Intensive Care, Faculty of Medicine – Zagazig University
*Corresponding Author: Sherif Omar Hasan Ali, **Mobile:** +20 01113103492, **Email:** sherif.omar9291@gmail.com

ABSTRACT
Background: Acute kidney injury is an abrupt or rapid decline in renal filtration function.
Objective: The study aimed to identify the incidence and analyze the risk factors (age and sex) for acute kidney injury development in patients of emergency and surgical intensive care units.
Patients and Methods: This study was carried out at emergency and surgical intensive care units of Zagazig University Hospitals over a period of 6 months. Data collection were1: Peak of serum creatinine level. 2: Peak of decreased urine output. 3: At the peak of AKI: AKIN (Acute Kidney Injury Network) score.
Results: Incidence of AKI in patients with normal kidney functions at admission and without history of any previous renal problems equal 7.015% according to AKIN score.
Conclusion: our results reported that incidence rate of developing AKI in patients of emergency and postoperative critical care units along the period of 6 months according to the inclusion criteria and AKIN score is 7.015 %. Age and sex weren't risk factors for AKI.
Keywords: Acute kidney injury, Incidence, Risk factors.

INTRODUCTION

Acute kidney injury (AKI)—or acute renal failure (ARF), as it was previously termed—is defined as an abrupt or rapid decline in renal filtration function. This condition is usually marked by a rise in serum creatinine concentration or by azotemia (a rise in blood urea nitrogen [BUN] concentration) [1].

However, immediately after a kidney injury, BUN or creatinine levels may be normal, and the only sign of a kidney injury may be decreased urine production [2].

The disorder is generally characterized by an abrupt deterioration in kidney function that disrupts metabolic, electrolyte and fluid homeostasis over a period of hours to days. The spectrum of AKI is broad, ranging from small changes in the levels of biochemical markers of kidney function to overt kidney failure requiring initiation of renal replacement therapy (RRT) [3].

AKI is a common and devastating condition associated with significant morbidity and mortality. Efforts to identify biomarkers to assist with the early diagnosis of AKI have yielded many promising candidates, such as kidney injury molecule-1 (KIM-1), neutrophil gelatinase associated lipocalin (NGAL), interleukin-18 (IL-18), cystatin C, clusterin, fatty acid binding protein (FABP), and osteopontin [4].

In 2013, the World Kidney Day Steering Committee focused on AKI, directing awareness to its impact and calling for campaigns to promote the prevention and prompt identification of AKI in patients at risk as well as the implementation of evidence-informed protocols and policies to mitigate its impact [5].

Incidence of AKI in intensive care unit (ICU) patients ranges between 20-70% according to settings, and, among these, patients who undergo RRT portend even worse outcome [6].

Risk factors for AKI in patients with severe illnesses are often multiple rather than single. These features can be grouped into several categories: certain underlying background predisposes patients to the development of AKI. Aged patients tend to acquire AKI more frequently than their younger counterparts, owing to the physiologic ageing of kidneys, multiple morbidities, and impaired renal recoverability [7].

Comorbidities include those with underlying diabetes mellitus (DM), hypertension, chronic kidney disease (CKD), and heart failure all reportedly set the backstage of subsequent renal injury, through the interplay of disrupted renal auto-regulation, preexisting renal damage, and concomitant use of nephrotoxic medications [8, 9]. Sepsis or systemic inflammatory response syndrome (SIRS) contributes to AKI development [10].

Hypotension, shock at presentation, and use of vasopressors/inotropes, also account for part of the clinical settings that subsequently spawn AKI [8, 9]. Several high-risk procedures or operations, such as cardiac surgeries (with cardio-pulmonary bypass), emergent surgeries, or lengthy surgery period, serve as a predisposing factor for AKI after operations [10].

Transfusion with packed red blood cells or use of furosemide perioperatively could also be associated with AKI [11]. Medications are often the one neglected component of the preludes for AKI. Angiotensin-converting enzyme inhibitors or angiotensin receptor blockers constitute one important example. Their use during coronary angiography is reported to increase risk of subsequent contrast induced nephropathy by nearly 50% [12].

The study aimed to identify the incidence and analyze the risk factors (age and sex) for acute kidney injury development in patients of emergency and surgical intensive care units.

PATIENTS AND METHODS

Study design: Retrospective cohort study.
Study setting: This study was carried out at emergency and surgical intensive care units of Zagazig University Hospitals over a period of 6 months.

Ethical approval:
   An approval of the study was obtained from Zagazig University academic and ethical committee. Every patient signed an informed written consent for acceptance of the operation.

Inclusion criteria:
  The study included the patients meeting the following inclusion criteria:
1: Patients with no history of chronic kidney disease and normal serum creatinine at time of admission in ICU but developed increase in serum creatinine level > 1.2 mg/dl later.
2: Patients with no history of chronic kidney disease and normal urine output at time of admission in ICU but developed decrease in urine output < 0.5 ml / kg /hour for > 6 hours later.
3: Patients with history of other medical diseases as hypertension, diabetes mellitus, chronic liver disease, hypo or hyperthyroidism and cerebrovascular stroke either controlled on treatment or not.

Exclusion criteria
 1: Patients with history of chronic kidney disease.
 2: End stage renal disease patients on regular dialysis.
 3: Patients with history of renal congenital anomalies as polycystic kidney or absent kidney.

All patients that had been admitted in emergency and surgical intensive care units of Zagazig University Hospitals over a period of 6 months that started at October 2018 and ended at March 2019 are included in this study.

Method of sample collection:
   Nonprobability sampling which involves the selection of elements based on inclusion and exclusion criteria.

Data collection methods:
Quantitative data collection:
1: Peak of serum creatinine level.
2: Peak of decreased urine output.
3: At the peak of AKI: AKIN (Acute Kidney Injury Network) score.

Table (1): Acute Kidney Injury Network Classification/Staging System for AKI [13].

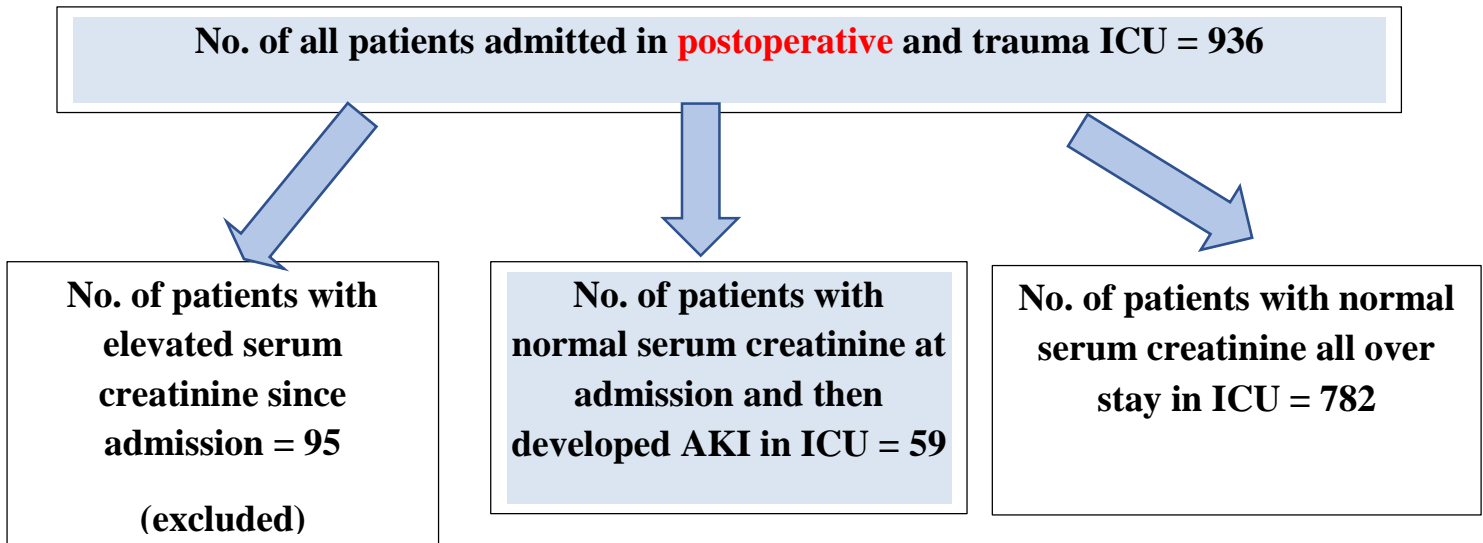| Stage	Serum creatinine criteria	Urine output criteria
1	• Increase in serum creatinine of more than or equal to 0.3 mg/dl or • increase to more than or equal to 150% to 200% from base line	• Less than 0.5 ml/kg per hour for more than 6 hours.
2	• Increase in serum creatinine to more than 200% to 300% from baseline	• Less than 0.5 ml/kg per hour for more than 12 hours.
3	• Increase in serum creatinine to more than 300% from baseline or • serum creatinine of more than or equal to 4.0 mg/dl with acute increase of at least 0.5 mg/dl	• Less than 0.3 ml/kg per hour for 24 hours or • Anuria for 12 hours.

Qualitative data collection:
1: Age.
2: Gender.
3: Admission cause.

Statistical analysis
   Data collected throughout history, basic clinical examination, laboratory investigations and outcome measures coded, entered and analyzed using Microsoft Excel software. Data were then imported into Statistical Package for the Social Sciences (SPSS version 20.0) software for analysis. According to the type of data; qualitative are represented as number and percentage and quantitative continues group are represented by mean ± SD, median, and range. Chi square test was used for difference and association of qualitative variable. Independent t-test was used for differences between quantitative variables. P value was set at <0.05 for significant results.

RESULTS

No. of all patients admitted in **postoperative** and trauma ICU = 936

No. of patients with elevated serum creatinine since admission = 95 (excluded)	No. of patients with normal serum creatinine at admission and then developed AKI in ICU = 59	No. of patients with normal serum creatinine all over stay in ICU = 782

There was no significant difference between groups as regard age or sex as shown in table 2.

Table (2): Comparison of age and sex between AKI and Non-AKI cases

Group			Non-AKI	AKI	t-test	P-value
Age (year)			50.24±17.64	50.62±15.89	-0.16	0.87
Gender	Female	N	411	34		
		%	52.6%	57.6%		
	Male	N	371	25		
		%	47.4%	42.4%		
Total		N	782	59	0.56	0.45
		%	100.0%	100.0%		

(AKI= acute kidney injury).
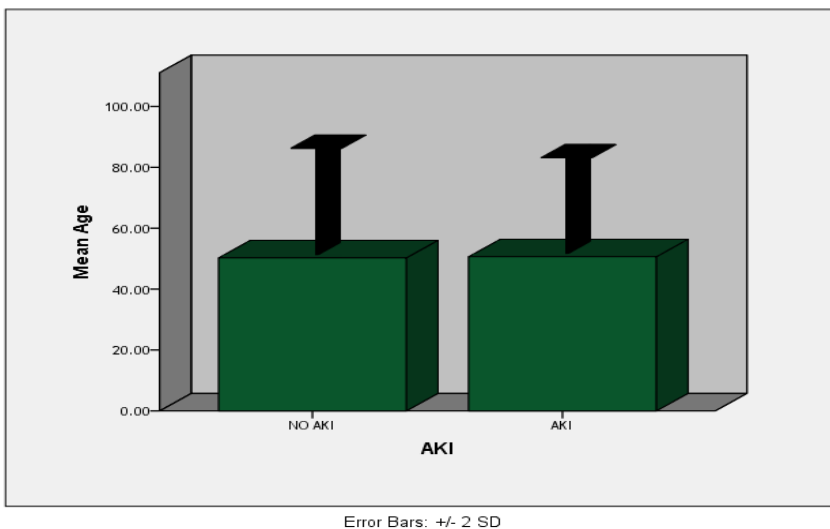

Error Bars: +/- 2 SD

Figure (1): Bar chart showing comparison of age of AKI and Non-AKI cases.

Table 3 shows that peak of decreased urine output was 0.55 ml/kg/hour ±0.15

Table (3): Peak of decreased urine output (UOP) among AKI cases

	Peak of decreased Urine Output
Mean± SD	0.55±0.15 ml/kg/hour
Median (Range)	0.54 (0.26-1.1) ml/kg/hour

Table (5): Peak of serum creatinine among AKI cases

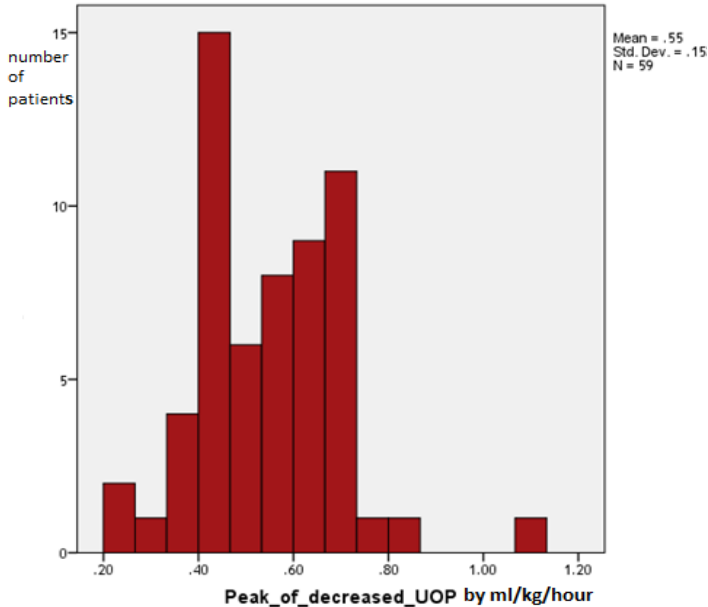|	Peak of serum creatinine
Mean+SD	2.199±0.49 mg/dl
Median (range)	1.84 (1.40 – 4.30) mg/dl

Figure (2): Bar chart showing distribution of UOP.

Regarding AKIN staging; the majority was in stage 1 (Table 4).

Table (4): AKIN staging distribution among AKI cases

Stage	Frequency	Percent
Stage 1	36	61.0 %
Stage 2	17	28.8 %
Stage 3	6	10.2 %
Total	59	100.0 %

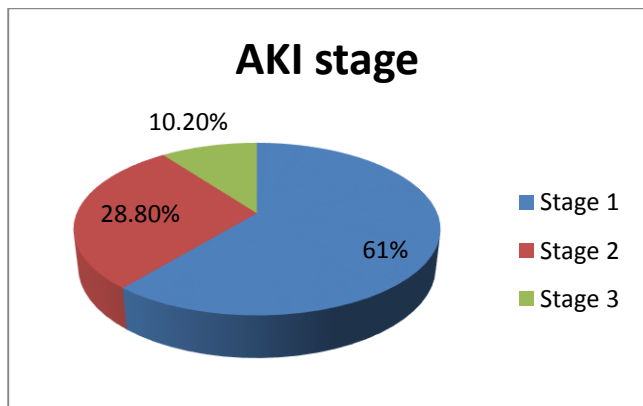**Figure (3): Pie chart showing AKI staging according to AKIN score.**

Mean of increased serum creatinine was 2.2 mg/dl (Table 5).

DISCUSSION

In our study the incidence rate of developing AKI in patients met the inclusion criteria and according to AKIN score was 7.015%.

This is near to the incidence rate of AKI, which was reported in **the study of Ali et al. [14]**, which was held at hospitals of Grampian region of Scotland over a period of 6 months and used the rise of serum creatinine or fall in GFR to assign a category in RIFLE classification and did not use urine output as a criterion for classification and the incidence rate of AKI according to RIFLE score and was 8.9% by 474 patients of total 5321 patients.

Our study differs from **that of Koeze et al. [15]**, which included all patients admitted in ICU from January 1st 2014 till June 11th 2014 and used serum creatinine and urine output according to RIFLE , AKIN and KDIGO classification.

The incidence of AKI in **Koeze et al. [15]** was (7.8%) using the RIFLE based on serum creatinine and (28%) based on urine output during the first week of ICU admission. The incidence according to AKIN was (12%) based on serum creatinine and (28%) based on urine output during the first week of ICU admission. The incidence according to KDIGO was (11%) based on serum creatinine and (28%) based on urine output during the first week of admission. Most probably the cause of difference between our study and that study of **Koeze et al. [15]** is that study held on medical, postoperative and emergency critical units and didn't exclude CKD patients and using different classification to define and stage AKI.

In our study distribution of AKI was more among female patients by (57.6%) where the mean of age was (50.62±15.89) with no agreement with **Hoste et al.s [16]** study as the mean of age was 65.0 and it was more among male patients by percentage was 63.0 % and the cause is that study excluded all patients under 18 years old.

In our study and according to AKIN SCORE, the majority of patients were stage 1 by (61%) followed by stage 2 by (28.8%) then stage 3 by (10.2%). The mean of peak of decreased UOP was (0.55±0.15) and the mean of creatinine (2.19±0.78), while in the study **of Koeze et al. [15]** and also according to AKIN score with UOP criteria : stage 1 incidence was 20 % and stage 2 incidence was 9.2 % and stage 3 was 7.9 %.

There are several limitations of this study: First, we specifically focused on patients who were admitted to emergency and postoperative ICUs only. Second, this study represents a snapshot in time of only 6 months. Especially in certain critical care units where a limited number of patients were included, this may have led to sampling bias. Third, inclusion and exclusion criteria which made us choose only group of patients to assess the results.

CONCLUSION

Our results reported that incidence rate of developing AKI in patients of emergency and postoperative critical care units along the period of 6 months started from October 2018 till the end of March 2019 according to the inclusion criteria and AKIN score is 7.015 %. Age and sex weren't risk factors for AKI.

REFERENCES:

1. **Schrier R, Wang W, Poole B et al. (2004):** Acute renal failure: definitions, diagnosis, pathogenesis, and therapy. J Clin Invest., 114(1):5–14.
2. **Waikar S, Bonventre J (2009):** Creatinine kinetics and the definition of acute kidney injury. J Am Soc Nephrol., 20(3):672–9.
3. **Lameire N, Bagga A, Cruz et al. (2013):** Acute kidney injury: an increasing global concern. Lancet, 382(9887):170–9.
4. **Vaidya V, Ferguson M, Bonventre J (2008):** Biomarkers of acute kidney injury. Annu Rev Pharmacol Toxicol., 48:463–93.
5. **Li P, Burdmann E, Mehta R (2013):** Acute kidney injury: global health alert. Kidney Int., 83(3):372–6.
6. **Clec'h C, Darmon M, Lautrette et al. (2012):** Efficacy of renal replacement therapy in critically ill patients: a propensity analysis. Crit Care, 16(6): 236-42.
7. **Chao C, Wu V, Lai C et al. (2012):** Advanced age affects the outcome-predictive power of RIFLE classification in geriatric patients with acute kidney injury. Kidney Int., 82(8):920–7.
8. **Bagshaw S, George C, Bellomo R (2008):** Early acute kidney injury and sepsis: a multicentre evaluation. Crit Care, 12(2): 47-52.
9. **Chawla L, Abell L, Mazhari R et al. (2005):** Identifying critically ill patients at high risk for developing acute renal failure: a pilot study. Kidney Int., 68(5):2274–80.
10. **Bellomo R, Wan L, Langenberg C et al. (2011):** Septic acute kidney injury: the glomerular arterioles. Contrib Nephrol., 174:98–107.
11. **Loef B, Epema A, Smilde T et al. (2005):** Immediate postoperative renal function deterioration in cardiac surgical patients predicts in-hospital mortality and long-term survival. J Am Soc Nephrol., 16(1):195–200.
12. **Barkhordari K, Fakhre Yasseri A, Yousefshahi F et al. (2018):** Risk factors for acute kidney injury in coronary artery bypass graft surgery patients based on the Acute Kidney Injury Network Criteria. J Tehran Heart Cent [Internet], 13(2):52–7.
13. **Mehta R, Kellum J, Shah S et al. (2007):** Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. Critical Care (London, England), 11: 31-36.
14. **Ali T, Khan I, Simpson W et al. (2007):** Incidence and outcomes in acute kidney injury: A comprehensive population-based study. J Am Soc Nephrol., 18(4):1292–8.
15. **Koeze J, Keus F, Dieperink W et al. (2017):** Incidence, timing and outcome of AKI in critically ill patients varies with the definition used and the addition of urine output criteria. BMC Nephrol., 18(1):1–9.
16. **Hoste E, Bagshaw S, Bellomo R et al. (2015):** Epidemiology of acute kidney injury in critically ill patients: the multinational AKI-EPI study. Intensive Care Med., 41(8):1411–23.