# Clinical evaluation of acute kidney injury in Al-Zahraa University Hospital, Cairo, Egypt

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**Background**
Acute kidney injury (AKI) is a very common problem. Early detection of injury with initiation of appropriate supportive care remains the mainstay of therapy.
**Aim**
The aim of the present study was to evaluate the incidence, etiology, prognosis, treatment, and outcome of AKI.
**Patients and methods**
This was a prospective, observational study of 212 patients (137 men and 75 women) who were admitted in all departments of Al-Zahraa University Hospital with AKI during the period from October 2014 to October 2015. Their ages ranged from 22 to 85 years. We included adults aged more than 18 years who presented with AKI, and we excluded patients on regular dialysis. Serum creatinine, sodium, potassium, urea, calcium, phosphorus and uric acid, complete blood count, pelviabdominal ultrasound, and daily measurement of urine output (UOP) were done. AKI patients were classified according to the RIFLE and Acute Kidney Injury Network staging.
**Result**
According to the RIFLE criteria, the number of risk, injury, failure, loss, and end-stage renal disease patients were 55 (25.9%), 62 (29.24%), 33 (15.56%), 38 (17.92%), and 24 (11.32%), respectively. According to the Acute Kidney Injury Network staging system, the number of stages I, II, and III patients were 61 (28.7%), 50 (23.5%), and 101 (47.6%), respectively. The length of hospital stay was significantly associated with severity of AKI. The main cause of AKI was dehydration in 82 (38.7%) patients and sepsis in 71 (33.5%). Oliguric patients were 147 (69.34%) and nonoliguric were 65 (30.66%). Moreover, prognosis of patients was complete recovery in 95 (44.81%), partial recovery in 81 (38.21%), and no recovery in 36 (16.98%).
**Conclusion**
AKI was more common among patients in ICU than those in any other department. Dehydration and sepsis were its leading causes.

**Keywords:**
acute kidney injury, dehydration, sepsis

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## Introduction

Acute kidney injury (AKI) is recognized as a very common problem in critically ill patients, and is strongly associated with increased resource utilization, and higher short-term and long-term mortality [1,2].

The classic definition of AKI included rapid rise of serum creatinine accompanied by a dramatic decrease in urine output (UOP). Due to lagging in the rise of serum creatinine in parallel with the rapid decline in the glomerular filtration rate, other criteria had been included [3,4].

The RIFLE (Risk, Injury, Failure, Loss, and End-stage renal disease) criteria were the first widely accepted definition [5], and later, the Acute Kidney Injury Network (AKIN) criteria was modified RIFLE [6].

## Aim

The presented study aimed to evaluate the incidence, etiology, prognosis, treatment, and outcome of AKI at AL-Zahraa University Hospital, Cairo, Egypt, over a period of 12 months.

## Patients and methods

### Patients

This prospective, observational study was conducted on 212 patients who were admitted in all departments of Al-Zahraa University Hospital with AKI during



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the period from October 2014 to October 2015. There were 137 (64.62%) male patients and 75 (35.38%) female patients, their ages ranging from 22 to 85 years. An informed consent was signed by the patients or their relatives before participation in the study. Approval for this study was obtained from the hospital's Ethical Committee.

Adult patients over 18 years of age who were admitted to the hospital during the study period with AKI were enrolled to the study. Patients with end-stage renal disease (ESRD) and on regular dialysis were excluded from the study.

### Methods

All patients of this study were submitted to the following:

(1) Full medical history, including drug history and surgical operations, full medical examination, and diagnosis at admission.
(2) Laboratory investigations, including serum creatinine (on admission, peak, and on discharge), urea, serum sodium, potassium, calcium, phosphorus, and uric acid. Daily measurement of UOP during admission period. Complete blood count, prothrombin time, prothrombin concentration, and international normalized ratio and pelviabdominal ultrasound were done for each patient.

Patients with AKI were classified according to the RIFLE classification and AKIN staging system (Tables 1 and 2).

### Statistical analysis

Statistical analysis: Data were analyzed using Statistical Program for Social Science (SPSS) (SPSS, version 20.0). Quantitative data were expressed as mean± SD. Qualitative data were expressed as frequency and percentage. A one-way analysis of variance was carried out to compare between more than two means. The $\chi^2$ test of significance was used to compare proportions between two qualitative parameters.

## Results

A total of 212 patients with AKI were studied (AKI was defined according to the RIFLE and AKIN staging of AKI and the patients were followed up for 6 months) – there were 137 (64.62%) male patients, of whom 74 (54.01%) were in stage III AKI according to the AKIN staging, and there were 75 (35.38%) female patients, on whom 27 (36%) were in stage III AKI according to the AKIN staging.

The ages of patients ranged from 22 to 85 years, with a mean±SD of 57.72±13.62 years (Table 3).

According to the RIFLE criteria, the number of risk, injury, failure, loss, and ESRD in the studied patients

**Table 1 RIFLE criteria of acute kidney injury [5]**

| Classes | Urine output criteria | eGFR criteria |
|---|---|---|
| Risk | $<$0.5 ml/kg/h×6 h | Serum creatinine×1.5 or decreased eGFR$>$25% |
| Injury | $<$0.5 ml/kg/h×12 h | Serum creatinine×2 or decreased eGFR$>$50% |
| Failure | $<$0.3 ml/kg/h×24 h, or anuria×12 h | Serum creatinine×3 or serum creatinine≥4 or decreased eGFR$>$75% |
| Loss | – | Persistent acute renal failure=complete loss of kidney function$>$4 weeks |
| End stage | – | End-stage kidney disease$>$3 months |

eGFR, estimated glomerular filtration rate.

**Table 2 Acute Kidney Injury Network staging for acute kidney injury [6]**

| Systems | Serum creatinine criteria | Urine output criteria |
|---|---|---|
| 1 | Serum creatinine increase $>$26.5 μmol/l (≥0.3 mg/dl) or increase to 1.5–2.0-fold from baseline | $<$0.5 ml/kg/h for 6 h |
| 2 | Serum creatinine increase$>$2.0–3.0-fold from baseline | $<$0.5 ml/kg/h for 12 h |
| 3 | Serum creatinine increase $>$3.0-fold from baseline or serum creatinine ≥354 μmol/l (≥4.0 mg/dl) with an acute increase of at least 44 μmol/l (0.5 mg/dl) or need for RRT | $<$0.3 ml/kg/h for 24 h or anuria for 12 h or need for RRT |

RRT, renal replacement therapy.

**Table 3 Demographic data of the studied patients and their relation to different acute kidney injury stages according to the Acute Kidney Injury Network staging**

| Demographic data | Total number of patients in studied group [*N* (%)] | Number of patients in stage I [*N* (%)] | Number of patients in stage II [*N* (%)] | Number of patients in stage III [*N* (%)] | *P*-value |
|---|---|---|---|---|---|
| Male | 137 (64.62) | 33 (24.09) | 30 (21.89) | 74 (54.01) | 0.085 |
| Female | 75 (35.38) | 28 (37.33) | 20 (26.7) | 27 (36) | |
| Age (mean±SD) | 57.72±13.62 | 57.38±15.48 | 62.12±9.49 | 56.95±13.78 | |

were 55 (25.9%), 62 (29.24%), 33 (15.56%), 38 (17.92%), and 24 (11.32%), respectively (Table 4). According to the AKIN staging system, the number of stages I, II, and III in the studied patients were 61 (28.7%), 50 (23.5%), and 101 (47.6%), respectively (Table 5).

Oliguria was noted in 147 (69.34%) patients and 65 (30.66%) patients were nonoliguric (Fig. 1).

Patients allocated from different hospital departments including surgical ICU, medical ICU, internal medicine department, surgery department, urology department, chest department, ophthalmology department, orthopedics department, tropical department, and vascular department were 73 (34.43%), 58 (27.36%), 47 (22.17%), 14 (6.60%), 10 (4.72%), 2 (0.94%), 2 (0.94%), 2 (0.94%), 2(0.94%), and 2 (0.94%), respectively (Fig. 2).

**Table 4 Acute kidney injury staging of 212 studied patients according to the RIFLE classification**

| RIFLE stage | *N* (%) |
|---|---|
| R | 55 (25.9) |
| I | 62 (29.24) |
| F | 33 (15.56) |
| L | 38 (17.92) |
| E | 24 (11.32) |
| Total | 212 (100) |

RIFLE=R, risk; I, injury; F, failure; L, loss; E, end-stage renal disease.

**Table 5 Acute kidney injury staging of 212 studied patients according to the Acute Kidney Injury Network classification**

| AKI stages | *N* (%) |
|---|---|
| I | 61 (28.77) |
| II | 50 (23.58) |
| III | 101 (47.64) |
| Total | 212 (100) |

AKI, acute kidney injury.

**Table 6 Prevalence of acute kidney injury in intensive care unit patients during the study period**

| ICU patients | *n* (%) |
|---|---|
| Total number of patients admitted in surgical and medical ICU | 812 (100) |
| The number of patients developed AKI | 131 (16.13) |

AKI, acute kidney injury.

**Table 7 Mean±SD of length of hospital stay of the studied patients**

| | Min. | Max. | Mean±SD |
|---|---|---|---|
| LOS (days) | 1 | 35 | 11.21±6.38 |

LOS, length of hospital stay; max., maximum; min., minimum.

Out of 812 patients admitted in the surgical and medical ICU during the period of study, 131 (16.13%) patients developed AKI (Table 6).

There were significant associations between the length of hospital stay (LOS) and the AKIN staging. The mean±SD of LOS in stages I, II, and III was 10.47

**Table 8 Mean±SD of length of hospital stay of the studied patients in acute kidney injury according to the Acute Kidney Injury Network staging**

| | Stage I | Stage II | Stage III | *P*-value |
|---|---|---|---|---|
| LOS (days) | 10.47±5.71 | 11.08±5.44) | 12.54±54) | 0.004 |

LOS, length of hospital stay.

**Table 9 Mean±SD of complete blood count and coagulation profile of the studied patients**

| | Min. | Max. | Mean±SD |
|---|---|---|---|
| WBCs | 2.9 | 36.4 | 15.64±7.22 |
| Hb | 3.6 | 15 | 10.92±2.86 |
| HCT | 12.1 | 48 | 36.16±35.53 |
| PLT | 70 | 699 | 277.30±116.20 |
| PT | 11.5 | 55.6 | 16.22±7.47 |
| PC | 30 | 100 | 75.43±25.50 |
| INR | 1 | 6.5 | 1.36±0.75 |

Hb, hemoglobin; HCT, hematocrit; INR, international normalized ratio; max., maximum; min., minimum; PC, prothrombin concentration; PLT, platelet; PT, prothrombin time; WBC, white blood cell.

**Table 10 Mean±SD of laboratory data of the studied patients**

| | Min. | Max. | Mean±SD |
|---|---|---|---|
| Blood urea (mg/dl) | 45 | 407 | 198.16±84.77 |
| Creatinine on admission (mg/dl) | 1.5 | 31 | 5.55±4.41 |
| Peak creatinine (mg/dl) | 2.01 | 31 | 6.31±4.28 |
| Creatinine on discharge (mg/dl) | 0.4 | 11.5 | 2.95±2.36 |
| Calcium (mg/dl) | 4.2 | 10.1 | 8.32±0.96 |
| Phosphorus (mg/dl) | 2.3 | 8 | 5.19±1.62 |
| UA (mg/dl) | 3 | 16 | 7.20±3.06 |
| Albumin (mg/dl) | 1.4 | 5.2 | 3.13±0.66 |
| K | 2.1 | 8 | 4.98±1.19 |
| Na | 140 | 160 | 134.81±19.10 |

max., maximum; min., minimum; UA, uric acid.

**Table 11 The risk factors for develop acute kidney injury in 212 studied patients**

| Risk factors | *N* (%) |
|---|---|
| Sepsis | 71 (33.49) |
| CKD | 38 (17.92) |
| DM+HTN | 35 (16.51) |
| Age >65 | 26 (12.26) |
| Liver cirrhosis | 11 (5.18) |
| HTN | 11 (5.18) |
| DM | 10 (4.71) |
| Heart failure | 8 (3.77) |
| Use of intravenous contrast | 2 (0.94) |

CKD, chronic kidney disease; DM, diabetes mellitus; HTN, hypertension.

**Table 12 Etiology of acute kidney injury in 212 studied patients**

| Etiologies | Total *n* [*N* (%)] | Number of patients stage I [*N* (%)] | Number of patients stage II [*N* (%)] | Number of patients stage III [*N* (%)] | *P*-value |
|---|---|---|---|---|---|
| Prerenal | 139 (65.6) | 42 (30.21) | 38 (27.34) | 59 (42.45) | <0.05 |
| Renal | 51 (24.1) | 15 (29.41) | 8 (15.69) | 28 (54.90) | |
| Postrenal | 22 (10.4) | 4 (18.18) | 4 (18.18) | 14 (63.64) | |

**Table 13 The relationship between the etiology of acute kidney injury and prognosis in 212 studied patients**

| Etiologies | Complete recovery [*N* (%)] | Partial recovery [*N* (%)] | No recovery [*N* (%)] |
|---|---|---|---|
| Prerenal (*n*=139) | 67 (48.2) | 52 (37.41) | 20 (14.39) |
| Postrenal (*n*=22) | 18 (81.82) | 3 (13.64) | 1 (4.55) |
| Renal (*n*=51) | 10 (19.61) | 26 (50.98) | 15 (29.41) |

**Table 14 Causes of acute kidney injury in 212 studied patients**

| Causes | *N* (%) | Death (*n*=100) | Alive (*n*=112) |
|---|---|---|---|
| Dehydration | 82 (38.7) | 21 | 61 |
| Sepsis | 71 (33.5) | 50 | 21 |
| Acute on top of chronic kidney disease | 38 (17.9) | 11 | 27 |
| HRS | 11 (5.2) | 8 | 3 |
| MODS | 10 (4.7) | 10 | 0 |

HRS, hepatorenal syndrome; MODS, multiorgan dysfunction syndrome.

**Table 15 Treatment, prognosis, and outcome distribution of 212 studied patients with acute kidney injury**

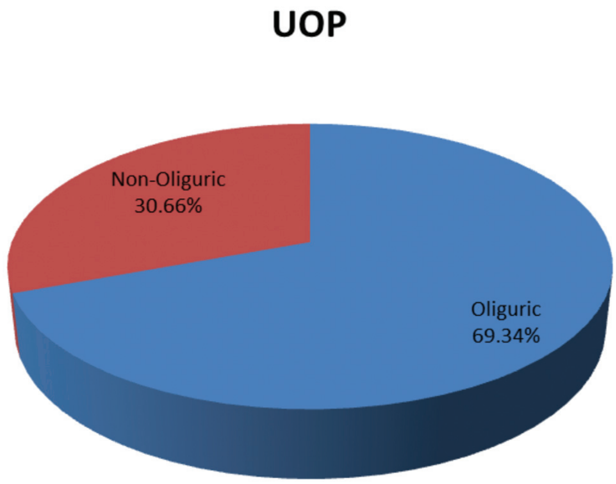
| | *N* (%) |
|---|---|
| Treatment | |
| Conservative | 150 (70.75) |
| Hemodialysis | 62 (29.25) |
| Prognosis | |
| Complete recovery | 95 (44.81) |
| Partial recovery | 81 (38.21) |
| No recovery | 36 (16.98) |
| Outcome | |
| Alive | 112 (52.8) |
| Death | 100 (47.2) |

±5.71 11.08±5.44, and 12.54±54 days, respectively (Tables 7 and 8).

As regards the complete blood count and coagulation profile results of all patients, the mean±SD of white blood cell, hemoglobin, hematocrit, platelet, prothrombin time, prothrombin concentration, and international normalized ratio was 15.64±7.22, 10.92±2.86, 36.16±35.53, 277.30±116.20, 16.22 ±7.47, 75.43±25.50, and 1.36±0.75, respectively (Table 9).

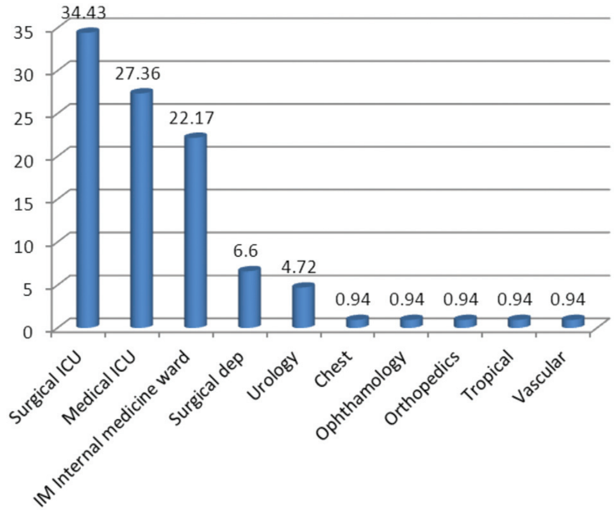
As per the laboratory data results of all patients, the mean±SD of blood urea was 198.16±84.77 mg/dl (range: 45–407 mg/dl); the mean±SD of serum creatinine on admission was 5.55±4.41 mg/dl (range: 1.5–31 mg/dl); peak of serum creatinine was 6.31±4.28 mg/dl (range: 2.01–31 mg/dl); and serum creatinine on discharge was 2.95±

**Figure 1**




The urine output (UOP) distribution of the studied group

**Figure 2**




Department distribution of the studied group

2.36 mg/dl (range: 0.4–11.5 mg/dl). In addition, the mean±SD of serum calcium, phosphorus, uric acid, albumin, sodium, and potassium was 8.32±0.96, 5.19±1.62, 7.20±3.06, 3.13±0.66, 134.81±19.10, and 4.98±1.19, respectively (Table 10).

The number of patients with risk factors for developing AKI, including sepsis, chronic kidney disease (CKD) patients, diabetes and hypertension (HTN), age above 65 years, liver cirrhosis, HTN alone, diabetes alone, heart failure, and patients who developed intravenous contrast nephropathy was 71 (33.49%), 38 (17.92%), 35 (16.5%), 26 (12.26%), 11 (5.18%), 11 (5.18%), 10 (4.71%), 8 (3.77%), and 2 (0.94%), respectively (Table 11).

The patients included in this study were divided into three groups according to the etiology of AKI (prerenal, renal, and postrenal causes). The number of patients in each group was as follows: 139 (65.6), 51 (24.1%), and 22 (10.4%) patients, respectively. There were significant associations with the severity of AKI in prerenal, renal, and postrenal causes ($P>0.035$) (Table 12).

The number of patients with prerenal, renal, and postrenal causes in stage III was 59 (42.45%), 28 (54.90%), and 14 (63.64%), respectively, which was significantly higher than the number of patients with prerenal, renal, and postrenal causes in stage I was 42 (30.21%), 15 (29.41%), 4 (18.18%), and in stage II was 38 (27.34%), 8 (15.69%), 4 (18.18%), respectively ($P<0.05$) (Table 12).

The prognosis of 212 studied patients with AKI was worse in patients with renal causes in whom complete recovery occurred in 10 (19.61%) patients and in patients with postrenal causes in whom complete recovery occurred in 18 (81.82%) patients, and it was better in patients with prerenal causes in whom complete recovery occurred in 67 (48.2%) patients (Table 13).

The causes of AKI of the studied patients, which included dehydration, sepsis, acute-on-CKD, hepatorenal syndrome, and multiorgan dysfunction syndrome were found in 82 (38.7%), 71 (33.5%), 38 (17.9%), 11 (5.2%), and 10 (4.7%) patients, respectively (Table 14).

Dehydration was diagnosed in 82 patients, of whom 21 died; sepsis was diagnosed in 71 patients, of whom 50 died; acute-on-CKD was diagnosed in 38 patients, of whom 11 died; hepatorenal syndrome was diagnosed in 11 patients, of whom eight died; and multiorgan dysfunction syndrome was diagnosed in 10 patients, of whom 10 died (Table 14).

Regarding the treatment of the studied patients, 150 (70.75%) patients were on conservative treatment, whereas 62 (29.25%) patients underwent hemodialysis (HD). As regards prognosis of the studied patients, complete recovery occurred in 95 (44.81%) patients, partial recovery occurred in 81 (38.21%) patients, and there was no recovery in 36 (16.98%) patients. As regards the outcome of the studied patients, 112 (52.8%) patients lived and 100 (47.2%) died (Table 15).

In our study there was a highly significant association between modality of treatment and severity of AKI as all patients who underwent HD were in stage III.

## Discussion

The mean±SD of age of 212 patients with AKI was 57.72±13.62 years, ranging between 22 and 85 years, of whom 137 (64.62%) were male patients and 75 (35.38%) were female patients. This increased incidence in male patients with AKI was also reported by Wijewickrama *et al.* [7] who found that 61.5% of AKI patients were male patient, with a mean age of 47.8 years; and also by Fan *et al.* [8] who found that 63.5% of AKI patients were male patients and 36.5% female patients, with their ages range from 39 to 68 years. However, other authors reported slightly increased female incidence up to 56.7% [9].

We did not find any association between patients' age or sex and severity of AKI, which was noted previously by Fan *et al.* [8] and Wijewickrama *et al.* [7].

When the RIFLE criteria was applied to our patients, 25.9% were classified as risk, 29.24% were classified as injury, 15.56% were classified as failure, 17.92% were classified as loss, and 11.32% were classified as ESRD. Furthermore, in a study by Talaat *et al.* [10], 24% patients were classified as risk, 28.2% as injury, and 47.8% as failure under the RIFLE criteria.

When we categorized AKI patients according to the AKIN criteria, 61 (28.77%) patients were in stage I, 50 (23.58%) patients were in stage II, and 101 (47.64%) patients were in stage III. Similar results were previously reported by Fan *et al.* [8], Talaat *et al.* [10], and Mehta *et al.* [11].

The incidence of AKI was more common in ICU patients compared with patients in other hospital departments. The distribution of patients in surgical ICU was 73 (34.43%), in medical ICU it was 58 (27.36%), and in internal medicine department it was 47 (22.17%).

This was previously noted by Hoste and Schurgers [12] who reported AKI occurring in approximately 50% patients in ICU, and also by Wijewickrama *et al.* [7] who found that during ICU stay, 60.2% developed AKI.

Other authors reported that, the incidence of AKI was higher in the department of internal medicine (52.4%) then it was in the department of surgery (33.2%), ICU (14.4%), and the department of nephrology (14.8%) [8].

The incidence of ICU-acquired AKI reached 16.13% in our studied patients, which was almost similar to that reported previously by other authors, who found that the prevalence of AKI in ICU patients was 21.2 [10], 17 [13], 18 [14], and 22.6% [15].

The mean±SD of the LOS of the studied patients was 11.21±6.38 days . Using Kaplan–Meier curve it was revealed that the LOS was significantly associated with severity of AKI.

This was also reported before by Challiner *et al.* [16] who found that the severity of the injury correlates with the LOS, and by Xiaoxi *et al.* [17] who reported that LOS was significantly higher in patients with AKI, and increased with increasing severity of AKI. Moreover, Chijioke *et al.* [18] reported that a short duration of hospital stay is associated with better outcomes in patients with AKI, and Jinyoung *et al.* [19] reported that duration of AKI influenced mortality rates in hospitalized patients.

The incidence of AKI among the studied patients with sepsis, CKD, diabetes and HTN, elderly above 65 years, liver cirrhosis, HTN alone, diabetes alone, heart failure, and patients who developed intravenous contrast nephropathy was 71 (33.49%), 38 (17.92%), 35 (16.5%), 26 (12.26%), 11 (5.18%), 11 (5.18%), 10 (4.71%), eight (3.77%), and two (0.94%), respectively. Challiner *et al.* [16] reported that the presence of diabetes mellitus (DM), HTN, known CKD, sepsis, diuretics, and age above 65 years were all associated with an increased incidence of AKI. Roberts *et al.* [9] reported that the most common AKI risk factors were HTN, patients treated with angiotensin-converting enzyme inhibitor and diuretics, and also patients with diabetes, CKD, and ischemic heart disease. Moreover, Rifkin *et al.* [20] found that the patients with AKI share several common risk factors including obesity, metabolic syndrome, diabetes, HTN, cardiovascular disease, and microalbuminuria.

The highest incidence of AKI in the presented patients occurred due to prerenal causes [139 (65.6%)] compared with renal [51 (24.1%)] and postrenal [22 (10.4%) causes]. This was in contrast to Fan *et al.* [8] who found that prerenal, renal, and postrenal AKI occurred in 36.5, 46.5, and 17.0% patients, respectively; the highest percent of AKI was due to renal causes.

The high incidence of AKI due to prerenal causes as dehydration and sepsis in our study was possibly due to the old age of the patients, which carries a higher incidence of sepsis-associated AKI, and also the presence of other comorbidities like DM, CKD, and liver disease. Similar results were previously reported by [11,17,21].

In our study, 71 (33.5%) patients out of 212 presented with AKI occurring due to sepsis, which was in agreement with Lopes *et al.* [22], Cruz *et al.* [23], and Bagshaw *et al.* [24], who found that the incidence of sepsis-associated AKI was 31.4, 25.6, and 32.4%, respectively.

In our study 147 (69.34%) patients were oliguric and 65 (30.66%) were nonoliguric, and there was a significant correlation between UOP and severity of AKI. In addition, Bouchard *et al.* [25] and Hoste *et al.* [26] reported the incremental risk for oliguric AKI concurrent with other organ failures, sepsis, and need for dialysis as the major determinants of mortality associated with in-hospital AKI.

Measurement of UOP is advised for AKI patients. Several AKI guidelines have introduced it as a diagnostic criterion along with creatinine. Mehta *et al.* [6] and Jung *et al.* [27] reported that age and anuria duration were independent predictors of renal recovery.

The treatment schedule studied was as follows: 150 (70.75%) patients were on conservative treatment, whereas 62 (29.25%) patients underwent HD. AKI was due to prerenal causes in a large number of our patients; its treatment included correction of the primary cause of AKI, fluid replacement therapy, and nutritional support. Moreover, supportive therapies such as antibiotics and adequate nutrition were given according to the standard management practice. All medications that could potentially affect renal function were discontinued. This was in contrast to Mehta *et al.* [11] who reported that dialysis was more frequently used in low and lower middle-income countries.

In our study we found highly significant association between modality of treatment and severity of AKI as all patients who underwent HD were in stage III. This was also previously noted by other authors [8].

As regards prognosis in our study, complete recovery occurred in 95 (44.81%) patient, partial recovery occurred in 81 (38.21%) patients, and there was no recovery in 36 (16.98%) patients. Mehta *et al.* [11] reported that 28% of their patients had complete recovery and 36% had partial recovery.

In our study we found that there was a highly significant difference in the relationship between the etiology and prognosis of AKI. Prerenal and postrenal causes were associated with higher recovery rates than did renal causes.

In patients with postrenal causes (22 patients) complete recovery occurred in 18 patients, which was significantly higher than the number of partial and no recovery patients, three and one patients, respectively. Most likely, because kidney damage caused by obstruction is transient and mild and is reversible after removal of obstruction, similar results were reported by Fan *et al.* [8], who noticed that postrenal AKI was associated with a higher recovery rate.

In patients with prerenal causes (139 patients) complete recovery occurred in 67 patients, which was significantly higher than the number of partial and no recovery patients, 52 and 20 patients, respectively.

In our studied patients 112 (52.8%) patients lived and 100 (47.2%) died. This was in agreement with Talaat *et al.* [10] who reported an overall mortality rate of 51.7%.

## Conclusion

The incidence of AKI was higher in ICU patients compared with the incidence in those in other hospital departments. The presence of risk factors like sepsis, CKD, heart failure, age more than 65 years, DM, HTN, and liver cirrhosis was associated with an increased development of AKI. Prerenal causes as dehydration and sepsis were the leading causes of AKI.

### Recommendation

(1) Increasing the knowledge in primary care units about AKI and renal diseases regarding causes, symptoms, management, and when to refer for nephrologist or tertiary care hospital.
(2) Long-term follow-up of patients with AKI.

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Nil.

**Conflicts of interest**
There are no conflicts of interest.

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