Evaluation of Acute Kidney Injuries Among Acutely Intoxicated Patients by RIFLE Classification

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KEYWORDS
Acute kidney injury,
AKI,
Intoxication,
Toxin,
Poison.

ABSTRACT

Acute kidney injury (AKI) develops in many of the patients admitted with a history of envenomation and poisoning. Tubular injury initiated by toxins often results from a combination of acute renal vasoconstriction and direct cellular toxicity due to intracellular accumulation of the toxin. The present study aimed to evaluate (percentage, causes, and outcome) acute kidney injuries among acutely poisoned patients. This cross-sectional study was carried out on all patients with acute kidney injury admitted to intermediate or intensive care unit (ICU) of Poison Control Center in Ain-Shams University Hospitals due to acute toxicity with drugs and toxins causing acute kidney injury. Participants were allocated retrospectively in the period from January 2018 to December 2018 and prospectively from April 2019 to the end of September 2019. The RIFLE staging in acutely intoxicated patients is significantly impacted by serum creatinine, serum urea, and eGFR. The outcome in acutely intoxicated patients is significantly impacted by serum creatinine and eGFR. Patients should be pre-hydrated and GFR should be frequently monitored during the administration of a potentially nephrotoxic drug with careful assessment of hemodynamic and volume status using vital signs and physical examination.

Introduction

Acute renal failure (ARF) is a common problem in intensive care medicine. Mild degrees of ARF that don't require dialysis can increase the risk of death about five folds. Today, patients with ARF have high co-morbidity and complication rates. In addition to effects on patient mortality, ARF prolongs hospitalization by an average of 10 days (Evenepoel, 2004).

Acute kidney injury (AKI) develops in many of the patients admitted with a history of envenomation and poisoning (Mohapatra et al., 2011).

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Nephrotoxic snake bites, scorpion stings, and wasp stings have been known to produce AKI in the group of envenomation. In snake envenomation, delayed presentation to health care, delayed administration of Antiserum venom (ASV), and incomplete dosages are the main factors in predicting the outcome of AKI. Acute kidney injury associated with bites and stings has a better prognosis than AKI associated with chemical poisoning. Among the poisons, paraquat, rat killer paste, copper sulfate, hair dye, toxic chemicals, and organophosphorus compounds can cause renal failure (Naqvi, 2017).

In addition, many poisonous plant substances can cause multiple organ dysfunction syndromes in which renal failure is also one of the components. Acute kidney injury can also develop in patients admitted with a history of envenomation and poisoning because of sepsis and pre-renal etiology. It is

an important cause of morbidity and mortality in these groups of patients. It increases the incidence of infections in these groups of patients by catheter-related sepsis and its attendant risks (DK, 2018).

Tubular injury initiated by toxins often results from a combination of acute renal vasoconstriction and direct cellular toxicity due to intracellular accumulation of the toxin. Also, it may be mediated immunologically in the case of interstitial nephritis (Evenepoel, 2004).

Awareness of the range of toxins on one hand and simple measures such as adequate pre-hydration of the patient and drug monitoring, on the other hand, may be sufficient to avoid drug-induced ARF or minimize its clinical severity in susceptible patients (Evenepoel, 2004).

The present study aimed to evaluate (percentage, causes, and outcome) acute kidney injuries among acutely poisoned patients.

Material and Methods:

This cross-sectional study was carried out on all patients with acute kidney injury admitted to Poison Control Center in Ain-Shams University Hospitals retrospectively in the period from January 2018 to December 2018 and prospectively during April 2019 to the end of September 2019. Acute toxicity was confirmed by complete screening and drug level. Approval from Ethical Committee in our department and informed written consent were obtained.

Inclusion criteria:

Patients admitted to the intermediate or intensive care unit (ICU) at Ain-Shams University Hospital due to acute toxicity with drugs and toxins causing acute kidney injury were enrolled in the study.

Exclusion criteria:

Patients with a history of debilitating diseases as diabetes, hypertension, known chronic kidney disease, or those on any drugs that can increase serum creatinine were excluded from the study

All patients included in this study were subjected to complete demographic, medical history, and clinical examination including vital signs and any presenting symptoms. Patients were scored according to Reed`s classification of coma (Amiri et al., 2016) and RIFLE staging. The RIFLE classification is based on serum creatinine and urine output determinants and considers three severity classes of AKI (Risk, Injury and Failure), according to the variations in SCr (serum creatinine) and/or UO (urine output), and two outcome classes (loss of kidney function and end-stage kidney disease) (Bellomo et al., 2004). Data on the current situation including the type of the toxic agent that causes intoxication, time of intoxication, delay time between intoxication and admission, route of exposure (oral, inhalation, injection, dermal or bite), and mode of intoxication were recorded. Routine lab investigations such as CBC, kidney function profile, and serum electrolytes were performed.

Statistical analysis:

Statistical presentation and analysis of the present study were conducted using SPSS. Quantitative variables were expressed as mean and standard deviation (SD) and were compared using the F test among the three groups. Categorical variables were expressed as frequency and percentage and were statistically analyzed by the Chi-square test.

The overall diagnostic performance was assessed by ROC curve analysis. P-value ≤ 0.05 was considered statistically significant.

Results:

At the end of the period of the study, it was found that 70 patients had ARF from drug or poison intake. Acute toxicity was confirmed with complete screening and drug level. During the period of the study, the percentage of studied patients from the total number (10893) of patients admitted to Ain Shams Poison Control Center (ASPCC) was 0.64%. Studied patients were poisoned by 20 drugs and toxins.

Patients' percentages with AKI due to acute drug or toxin intake admitted to ASPCC are shown in table (1).

Table (1): Patients' percentages with AKI due to acute drug or toxin intake admitted to ASPCC (n:70)

Toxic agent	Number	Percentage%
Opiate	12	17.14
Tramadol	4	5.71
Hashish	5	7.14
Organophosphorus poisoning	9	12.86
Carbamate	3	4.29
Dormer	3	4.29
Methanol	7	10
Corrosive	3	4.29
PPD	6	8.57
Aluminum phosphide	3	4.29
Digoxin	1	1.43
Theophylline	2	2.86
Snakebite	2	2.86
Metformin	2	2.86
Warfarin	1	1.43
Ibuprofen	1	1.43
Lithium	1	1.43
Clozapine	2	2.86
Carbamazepine	1	1.43
CO toxicity	2	2.86
Total	70	100

Table (2) showed acute kidney injury patients' percentages of each drug or toxin (N1) about total cases (N2) admitted to ASPCC.

Table (2): Patients' numbers and percentages with AKI due to acute toxic agent intake (n1) about total cases of this agent admitted to ASPCC (n2).

Toxic agent	n1	n2	Percentage (%)
Opiate	12	573	2.09
Tramadol	4	368	1.09
Hashish	5	1431	0.35
Organophosphorus poisoning	9	1725	0.52
Carbamate	3	1212	0.25
Dormer	3	7	42.86
Methanol	7	63	11.11
Corrosive	3	2090	0.14
PPD	6	43	13.95
Aluminum phosphide	3	66	4.55
Digoxin	1	27	3.7
Theophylline	2	816	0.25
Snakebite	2	210	0.95
Metformin	2	560	0.36
Warfarin	1	34	2.94
Ibuprofen	1	120	0.83
Lithium	1	8	12.5
Clozapine	2	400	0.5
Carbamazepine	1	420	0.24
CO toxicity	2	720	0.28
Total	70	10893	

n1 represents the number of patients affected by each toxic agent that caused acute kidney injury. **n2** represents the total number of patients admitted by each toxic agent during the period of the study. **AKI:** acute kidney injury.

Results showed that males intoxicated patients were more than females. Also, the route of exposure in the studied patients was mostly oral then intravenous then inhalation then sting, and bite. The mode of poisoning was mostly accidental addict then suicidal then accidental nonaddict then iatrogenic and finally criminal. While no patients were in coma grade III, most patients were in coma status grade 0 than grade IV then grade I then grade II. Most patients had no urinary manifestations (non oliguric phase) and therefore did not need dialysis. About (4.29%) of studied patients were in the risk stage while (40%) were in the injury stage and about (51.43%) were in the failure stage and only (2.86%) were in the loss of function stage and (1.43%) of them were in the end-stage kidney disease. It was found that almost half of the patients had complete recovery while patients who had chronic kidney disease were (5.71%) and about (42.86%) of studied patients died (Table 3)

Table (3): Percentage of patients with AKI about variant parameters (n:70)

		Number	Percentage (%)
Age group (year)	0-14	2	2.86
	15-20	9	12.86
	21-30	14	20
	31-40	24	34.29
	41-50	21	30
Route of exposure	Oral	53	75.71
	IV	12	17.14
	Inhalation	3	4.29
	Sting and Bite	2	2.86
Mode of poisoning	Accidental nonaddict	19	27.14
	Accidental addict	26	37.14
	Suicidal	23	32.86
	Iatrogenic	1	1.43
	Criminal	1	1.43
Conscious state	Coma grade 0	25	35.71
	Coma grade I	13	18.57
	Coma grade II	12	17.14
	Coma grade III	0	0
	Coma grade IV	20	28.57
Urinary manifestations	None	53	75.71
	Anuria	6	8.57
	Oliguria	6	8.57
	Hematuria	5	7.14
Dialysis	No	55	78.57
	Yes	15	21.43
RIFLE staging	Risk	3	4.29
	Injury	28	40
	Failure	36	51.43
	Loss of function	2	2.86
	End-stage kidney disease	1	1.43
Outcome	Complete recovery	36	51.43
	Chronic kidney disease	4	5.71
	Death	30	42.86

n: number, **AKI:** acute kidney injury.

Table (4) showed that most patients were admitted after a delay time of 1-6 hours followed by a delay time of 6-24 hours then a delay time of 1-3 days. Table (5) describes the results of some laboratory investigations.

Table (4): Percentage of patients with AKI about delay time between intoxication and admission (n:70)

Delay time	Number	Percentage%
1-6 hours	43	61.4
6-24 hours	18	25.7
1-3 days	9	12.85

n: number, **AKI:** acute kidney injury.

Table (5): Percentage of patients with AKI about CBC parameters and electrolytes (n:70)

CBC parameters		Number	Percentage (%)
HB level	Low	33	47.2
	Normal	37	52.8
WBCs	High	29	41.4
	Normal	41	58.6
Platelets	Low	23	32.9
	Normal	47	67.1
Electrolytes			
Potassium level	Normal	39	55.71
	Low	12	17.14
	High	19	27.14
Sodium level	Normal	58	82.86
	Low	8	11.43
	High	4	5.71

Normal values for: Hemoglobin level: 13-15 g/dl, WBCs: 4,500 to 11,000/ microliter. Platelets: 150,000 to 450,000 /microliter, Potassium level: 3.5 - 5.0 mmol/L, Sodium level: 135 - 145 mEq/L.

There was a significant statistical difference of serum urea associated with different urinary system manifestations. There was a significant statistical difference in patients with different urinary manifestations about both injury and failure stages. There was a significant statistical difference in patients with different urinary manifestations about both complete recovery and death (Table 6).

Table (6): Urinary system manifestations about RIFLE staging, outcome, and renal parameters (n:70)

	Urinary system manifestations								Total number	p-value
	None (n:53)		Anuria (n:6)		Oliguria (n:6)		Hematuria (n:5)			
	n	%	n	%	n	%	n	%		
RIFLE staging										
Risk	3	100	0	0	0	0	0	0	3	-
Injury	25	89.29	1	3.57	1	3.57	1	3.57	28	<0.001*
Failure	24	66.67	3	8.33	5	13.9	4	11.11	36	<0.001*
Loss of function	1	50	1	50	0	0	0	0	2	1
End-stage kidney disease	0	0	1	100	0	0	0	0	1	-
Outcome										
Complete recovery	31	86.11	1	2.78	1	2.78	3	8.33	36	<0.001*
Chronic kidney disease	2	50	2	50	0	0	0	0	4	1
Death	20	66.67	3	10	5	16.6	2	6.67	30	<0.001*
Renal parameters										
Serum urea (mg/dL)	92.94 ±8.67		79.83 ±6.63		152.67 ±9.36		91.6 ±8.96			0.024*
Serum creatinine (mg/dL)	3.11 ±66		3.76 ±15		4.08 ±59		3.88 ±56			0.465
eGFR (ml /min /1.73 m^2)	29.25 ±3.91		20.2 5 ±07		21.05 ±2.25		20.6 ±1.09			0.154

eGFR: estimated glomerular filtration rate. n: number. RIFLE: risk, injury, failure, loss of function, end-stage renal disease. mg: milligram. dl: deciliter. ml: milliliter. min: minute. m: meter.

There was a significant statistical difference in serum creatinine in acutely intoxicated patients about different RIFLE stages. There was a significant statistical difference of serum urea in acutely kidney injured patients about different RIFLE stages. Also, there was a significant statistical difference of eGFR about different RIFLE stages (Table 7).

Table (7): RIFLE (risk, injury, failure, loss of function, end-stage kidney disease) staging about serum creatinine, serum urea, and eGFR.

	RIFLE staging					
	Risk	**Injury**	**Failure**	**Loss of Function**	**End-stage kidney disease**	**p-value**
	mean ± SD	**mean ± SD**	**mean ± SD**	**mean ± SD**	**mean ± SD**	
Serum creatinine (mg/dL)	1.6 ± 0.05	2.2 ± 0.42	4 ± 1.7	4.95 ± 2.3	10 ± 0	<0.001*
Serum urea (mg/dL)	49 ± 21.65	74.5 ± 39.3	118 ± 48.2	96 ±16.97	106 ± 0	0.002*
eGFR (ml/min/1.73m)	54.26 ± 4.1	37.58± 9.4	18.15 ± 5.7	12.95 ± 3.9	6.5 ± 0	<0.001*

eGFR: estimated glomerular filtration rate, SD: standard deviation.

While there was a significant statistical difference of serum creatinine about the outcome, there was a nonsignificant statistical difference of serum urea about different outcomes. There was a significant statistical difference of eGFR about different outcomes (Table 8).

Table (8): Outcome stages about serum creatinine, serum urea, and eGFR

	Outcome			
	Complete recovery	**Chronic kidney disease**	**Death**	**p-value**
	mean ± SD	**mean ± SD**	**mean ± SD**	
Serum creatinine (mg/dL)	2.747 ± 1.052	7.425 ± 3.164	3.43 ± 1.615	<0.001*
Serum urea (mg/dL)	84.056 ± 35.031	121.75 ± 46.133	108.867 ± 58.56	0.064
eGFR (ml /min /1.73 m^2)	30.76 ± 13.7	9.8 ± 4.27	25.147 ± 11.9	0.006*

eGFR: estimated glomerular filtration rate, SD: standard deviation.

The ROC curve analysis to assess urine output as a predictor of survival with a cut off value. 1200 revealed a corresponding sensitivity of (72.5%), specificity (56.67%), PPV (69%), and NPV (60.7%) with accuracy rate (61.9%) (Figure 1).

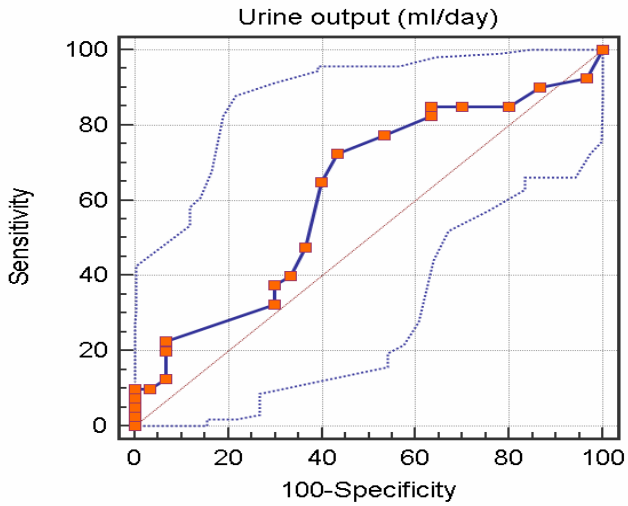

Fig. (1): ROC curve of urine output as a predictor of survival

The ROC curve analysis to assess eGFR as a predictor of survival with a cut off value >24.5, revealed a corresponding sensitivity of (55%), specificity (63.33%), PPV (66.7%), and NPV (51.4%) with accuracy rate (56.9%) (Figure 2).

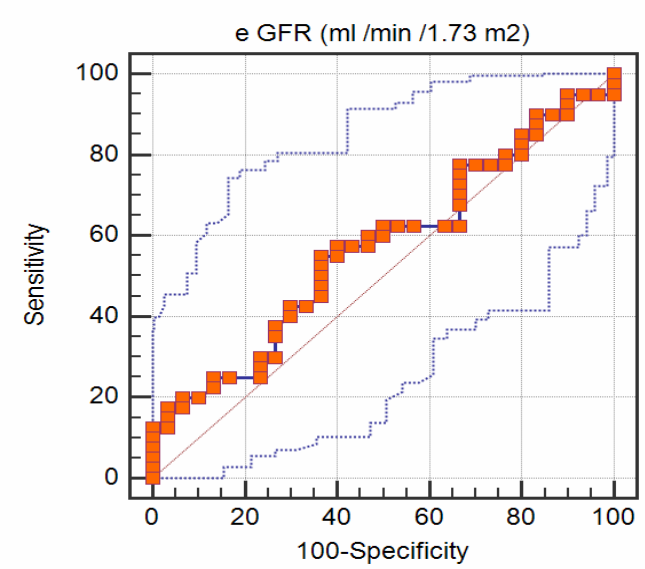

Fig. (2): ROC curve of eGFR as a predictor of survival

Discussion

Acute kidney injury (AKI), formerly known as acute renal failure (ARF) is common, especially in hospitalized patients, and is independently and strongly associated with morbidity and mortality. Acute kidney injury is not a single disease, but a complex clinical syndrome that may arise in response to many etiologies, such as circulatory shock, sepsis, and nephrotoxins (Bellomo et al., 2012).

The present work aimed to study cases of acutely poisoned patients by drugs and toxins with acute kidney injury in Poison Control Center Ain-Shams University Hospitals (ASPCC) in the period from January 2018 to the end of December 2018 and the period of April 2019 to the end of September 2019.

At the end of the study, the total patients admitted to Ain Shams Poisoning Center during the period of the study were 10893 patients. 70 patients had acute kidney injury with a percentage of (0.64%). In agreement with the current study, Sweni et al. (2012) found in a tertiary care center of South India in the period of 12 months from August 2006 to July 2007 that 1,250 cases were admitted to their toxicology center and only 32 cases (2.5%) developed AKI. Also, Naqvi (2017) found that in Pakistan from January 1990 to May 2016, 184 cases (0.74%) developed AKI during the study period.

In a similar study done in India in the period of 6 months from February 2017 to July 2017, SivaKumar and Karthikeyan (2018) found that a total number of 1210 patients were admitted with a history of poisoning and envenomation. The authors found that 68 of them developed AKI during the study period with a percentage of 5.6%.

The present study showed that the most common related toxins of AKI were opiate, organophosphorus, methanol, PPD, hashish, and tramadol. Contrary to the present results, a study made by Sweni et al. (2012)in a

tertiary care center of South India, the snake bite was the most common cause of AKI. Dichromate, indigenous medicines, and PPD were the least causes for ARF. None of the patients with organophosphate or overdose of medicines developed AKI. In another study by Naqvi (2017) in Pakistan, the commonest causes of AKI were PPD, methanol, organophosphorus compounds, paraquat, copper sulfate, tartaric acid, phenobarbitone, and benzodiazepine toxicity. SivaKumar and Karthikeyan (2018) in India also found that the commonest causes of AKI were snake bites, paraquat, rat killer paste, copper sulfate, ethylene glycol, and organophosphorus poisoning respectively. Overall, this difference might be due to the factors associated with AKI across studies such as differences in the study population, potency and composition of snake venom, which differs across geographic regions, and management facilities (Paul and Dasgupta, 2012).

In the present study, opiate caused AKI by the percentage of 2.09% of all opiate intoxicated patients and that represents 17.14% of all patients who developed AKI. Similar to the current results, Rice et al. (2000) in Australia identified a group of 27 patients who developed renal failure after recent intravenous heroin use and stated that there was an increase in patients admitted with rhabdomyolysis-induced renal failure associated with heroin use in their hospitals. In agreement with the present study, Mohammad et al. (2017) in the United States presented two case reports of opiate overdose developed acute kidney injury and stated that acute kidney injury (AKI) following heroin overdose is emerging as a major problem. The usual mechanism for AKI with opioid use is in the setting of multi-organ failure from respiratory depression, hypoxia, and volume depletion with or without rhabdomyolysis (Feng et al., 2015).

In the present study, organophosphorus caused AKI in 0.52% of all organophosphorus intoxicated patients representing12.86% of all patients who developed AKI. Contrary to the present study, a prospective study conducted by Banday et al. (2015) on 133 patients with OP poisoning in Karnataka in India over 1 year, 15.03% of patients had acute renal failure following exposure to OP poisoning. Also contrary to the present results, Lee et al. (2015) published a large cohort study that highlighted AKI development after exposure to OP poisoning during the period from 2000 to 2011. Patients with OP poisoning were associated with a 6.17 fold higher risk of AKI. Different mechanisms have been suggested that can cause AKI. Organophosphorous poisoning might cause oxidative stress, direct damage to the renal tubules, rhabdomyolysis, and hypovolemia due to dehydration (Zafar et al., 2017).

In the present study, methanol caused AKI in 11.11% of all methanol intoxicated patients(10% of all patients who developed AKI).In agreement with the present study, Salek et al. (2014) found that 15.4% of studied methanol patients developed AKI, while a study made by Lee et al. (2014) indicated that AKI developed in 59.4% of patients after methanol exposure. The etiologies of methanol nephrotoxicity may be due to high blood methanol, formate concentration, hemolysis, and myoglobinuria (Chang et al., 2019).

In the present study, Paraphenyldiamine dye caused AKI in 13.95% of all PPD intoxicated patients (8.57% of all patients who developed AKI) to Ain Shams Hospitals. In agreement to the present study, a study conducted by Ramulu et al. (2016) in India, AKI was seen in 19.3% of patients who ingested more than 50 ml of hair dye and was not seen in patients who ingested less than 50 ml of the dye. On the contrary to the current study, a study done by Magdy et al. (2015) in

Upper Egypt, AKI developed in 76% of studied PPD intoxicated patients. The difference may be attributed to the high prevalence of the use of dyes in Upper Egypt. Acute kidney injury in PPD-toxicity has several mechanisms. It has a direct toxic action of PPD or its metabolites on the renal parenchyma. It has a high filtration rate and after oxidation is converted to nephrotoxic agents specially quinone-diamine. Its indirect action is through hemolysis, met-hemoglobinemia, and myoglobin casts leading to tubular damage (Ahmed and Alturki, 2018).

The current study showed that about 75.71% of studied patients had no urinary manifestations, only 8.57% had anuria, where 8.57% had oliguria and about 7.14% had hematuria.

On the contrary, a study conducted by Sweni et al. (2012) showed that oliguria was the most common symptom of renal dysfunction while the time of onset of oligo-anuria was similar.

Also, Naqvi (2017) found that 93% of patients had oligoanuria and 86% of them had haematuria.

In the present study, the range of creatinine level was 1.4 to 10.1 mg/dl and the mean value of creatinine level was 3.3 mg/dl.

In concordance with the present study, Sivakumar and Karthikeyan (2018) found that the mean peak creatinine value in 50 patients was 5.13 mg/dl.

Also, on the contrary, Sweni et al. (2012) found that the mean blood creatinine value was 6.1 and 6.8 mg/dl in envenomation and chemical poisoning respectively.

In the present study, the range of urea level was 17 to 221 mg/dl and the mean urea level was 96.8 mg/dl.

On the contrary, Sweni et al. (2012) found that the mean value of blood urea was 276 and 318 mg/dl in envenomation and chemical poisoning respectively.

The present study showed that about 51.43% of studied patients had complete recovery, where 42.86% died and only 5.71% had chronic kidney disease.

In agreement with the current study, Naqvi (2017)showed that complete recovery occurred in 72.28% of patients and death occurred in 20%.

The present study showed that dialysis was done only in 21.43% of studied patients.

On the contrary, Sivakumar and Karthikeyan's (2018) study showed that dialysis requirement was in about 86% of patients.

The present study revealed that the percentage of intoxicated male patients was higher than females (75.71% and 24.29% respectively).

These results approximately go hand in hand with the results recorded by Sivakumar and Karthikeyan (2018)who found that 78% of intoxicated patients were males and 22% were females.

The present study showed that 35.71% of studied cases were in coma grade 0, about 28.57% of them were in coma grade IV, where 18.57% of them were in coma grade II and about 17.14% of them were in coma grade I. No patients were in coma grade III.

On the contrary, Naqvi (2017) found that only 16% of patients had an altered level of consciousness.

In the current study, accidental addict poisoning was about (37.14%) than suicidal (32.86%) then accidental non-addict (27.14 %), and finally iatrogenic (1.43%), and criminal (1.43%).

On the contrary, Mostafa et al. (2014) found that self-poisoning was the most common manner of poisoning followed by accidental poisoning than an overdose of drugs or abuse.

The likely causes of intentional poisoning are unemployment, low socioeconomic status, and marital discord (Mostafa et al., 2014).

The current study showed that the route of exposure in studied patients was mostly oral. Generally, the oral route was the most common route of poisoning as mentioned by Mostafa et al. (2014) in a study among adolescent patients admitted to the poison control center, Ain Shams University Hospitals over six months. This could be explained by the convenience and easy availability of orally consumable poisons as mentioned by Tarvadi et al. (2013).

Future research should evaluate whether the use of early markers of tubular damage like KIM-1, NGAL, IL-18, Clusterin, and Cystatin C can reduce the incidence of nephrotoxicity.

Conclusions:

During the period of study, the percentage of studied patients from a total number (10893) of patients admitted to Ain Shams poisoning control center (ASPCC) was 0.0064%. The most common drugs and toxins causing acute kidney injury were opiates, organophosphorus, methanol, and PPD. The majority of studied patients were in the failure stage followed by the injury stage, the risk stage, the loss of function stage, and the end-stage kidney disease respectively.

Recommendations:

Patients should be pre-hydrated and GFR & urine output should be frequently monitored during the treatment with a potentially nephrotoxic drug with careful assessment of hemodynamic and volume status using vital signs and physical examination.

Acknowledgments and sponsorship: Nil

Conflict of Interest: Nil

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تقييم إصابات الكلى الحادة بين مرضى التسمم الحاد بمعايير ريفل

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تعد الاصابات الحادة للكلي من المشاكل الشائعة في العناية المركزة. وبالرغم من أن الدرجات البسيطة للإصابة لا تتطلب غسيل الكلى الا انها تزيد من خطر الموت حوالي خمسة أضعاف. كما انها تزيد من معدل المضاعفات والامراض المصاحبة لها. بالإضافة إلى تأثيرها علي زيادة معدل الوفيات، فإنها تطيل مدة الاقامة بالمستشفى بمعدل ١٠ أيام. تحدث إصابة الكلى الحادة في العديد من حالات التسمم مثل اللدغات والتسمم بالعقاقير. تهدف هذه الدراسة إلى تحديد النسبة المئوية لإصابات الكلى الحادة بين مرضى التسمم الحاد وتحليل النتيجة والتشخيص وفقا لمعايير ريفل لإصابة الكلى الحادة.تعد هذه الدراسة دراسة مستعرضة بأثر رجعي ومستقبلية. تم تنفيذها على ٧٠ مريضا يعانون من إصابة الكلى الحادة من تناول أدوية أو سموم وتم إدخالهم مركز علاج التسمم بعين شمس خلال الفترة من يناير ٢٠١٨ إلى نهاية ديسمبر ٢٠١٨ والفترة من أبريل ٢٠١٩ إلى نهاية سبتمبر ٢٠١٩. وقد تبين وجود علاقة ذات دلالة إحصائية بين التشخيص وفقاً لمعايير ريفل في مرضى التسمم الحاد ومستويات الكرياتينين واليوريا في الدم بالإضافة إلى معدل الترشيح. كما تبين وجود علاقة بين كلاً من متوسط معدل الترشيح الكلوي ونسبة الكرياتينين بالدم ونسبة الشفاء. ومن هذه الدراسة نخلص إلى أهمية التقييم الدقيق لحالة الدورة الدموية باستخدام المؤشرات الحيوية والفحص البدني للمريض.يجب ان يتم اعطاء محاليل للمرضي مسبقا ومتابعة معدل الترشيح الكلوي بصفة دورية عند علاج تسمم دواء يحتمل ان يكون ساما للكلى.