



LA SOCIÉTÉ TUNISIENNE
DE MÉDECINE D'URGENCE



THE OFFICIAL JOURNAL
OF THE TUNISIAN SOCIETY
OF EMERGENCY MEDICINE

**MARCH
2026**

**Volume 4
ISSUE 1**



ISSN : 3061-9165

TUNISIAN JOURNAL OF EMERGENCY MEDICINE

Editorial Board

Honorable Editor in Chief

Semir NOUIRA

STMU chair

Imen REJEB

Editors-in-chief

Olfa CHAKROUN-WALHA

Rim KARRAY

Past Editor-in-chief

Riadh BOUKEF

Sami SOUISSI

Editorial Board

Riadh BOUKEF

Sami SOUISSI

Asma ZORGATI

Hamdi BOUBAKER

Hanen GHAZALI

Mounir HAGUI

Olfa DJEBBI

Rym HAMED

Zied MEZGAR

Founding Editorial Board

Anouar Yahmadi, Mohamed Habib Grissa, Abdelwaheb Morjane, Hamdi Boubaker, Asma Zorgati, Lotfi Boukadida, Rym Hamed, Samir Abdelmoumen, Naoufel Somrani, Zohra Dridi, Hanene Ghazali, Kaouthar Beltaief, Nebiha Borsali, Zied Mezgar, Riadh Boukef, Mounir Naija, Wahid Bouida, Sami Hafi, Majdi Omri and Sami Souissi

Website for submitting your publication: www.tjem.tn

Acute kidney injury in critically ill patients: a comparison between the RIFLE, AKIN, CK and KDIGO classifications

Rania Ammar¹, Emna Ennouri¹, Mabrouk Bahloul¹, Chokri Ben Hamida¹

1: Medical resuscitation department of CHU Habib Bourguiba Teaching Hospital, Sfax, Tunisia, University of Sfax, Tunisia

Corresponding author: Rania Ammar; Address: Road El Ain km 1, Postal code 3029 Sfax, Tunisia; FAX: +216 74 243 427; Phone: +216 21469841; Email: rania.ammarzayani@gmail.com

Abstract

Objective: Compare the incidence of acute kidney injury (AKI) and the ability to predict in-hospital mortality by using RIFLE, AKIN, CK, and KDIGO classifications in critically ill patients.

Methods: A prospective, observational, single-center study conducted over 6 months. We included patients aged ≥ 18 years, and with an in-hospital stay ≥ 48 hours.

Results: We included 171 patients. The incidence of AKI using the RIFLE, AKIN, CK, and KDIGO criteria were 32.16%, 46.8%, 45%, and 46.8%, respectively. AKIN and KDIGO were similar, and they were more sensitive than RIFLE (46.8% vs 32.16%, $p < 0.001$) and CK (46.8% vs 45%, $p < 0.001$). In-hospital mortality was significantly higher for AKI patients than for non-AKI patients, regardless of the definition used: RIFLE (17.54% vs 12.86%, $p < 0.0001$), CK (23.39% vs 7.01%, $p < 0.0001$), and both KDIGO and AKIN (24.56% vs 5.84%, $p < 0.0001$). Mortality rate of patients identified as AKI by KDIGO and AKIN was higher than RIFLE (24.56% vs 17.54%, $p = 10^{-3}$), and CK (24.56% vs 23.39, $p = 10^{-3}$). Compared with RIFLE and CK criteria, both KDIGO and AKIN had greater predictive ability to predict the in-hospital mortality. The area-under-ROC curves for in-hospital mortality for RIFLE, AKIN, CK and KDIGO criteria were: 0.694, 0.761, 0.75, and 0.761 respectively.

Conclusion: KDIGO and AKIN identified more AKI patients and were more predictive for in-hospital mortality than RIFLE and CK.

Keywords: Acute kidney injury; Acute kidney failure; Diagnosis; Incidence, In-hospital mortality

INTRODUCTION

Acute kidney injury (AKI) is a frequent complication in hospitalized patients in the intensive care unit (ICU). It affects one in five hospitalized patients (1) and occurs in up to 25% (2,3). Renal dysfunction is an independent risk factor for mortality, especially in patients treated with renal replacement therapy (RRT) (4). There were about 13.3 million cases of AKI reported worldwide in 2013, with about 85% of cases occurring in low and middle-income countries (5). The effective treatment of AKI is strongly dependent on a timely diagnosis. However, the mean missed diagnosis rate of AKI is reportedly as high as 74.2% (6), which may increase mortality (7). Since 2004, at least four criteria have been used to define and stage AKI. The RIFLE (Risk, Injury, Failure, Loss, and End-stage kidney disease) criteria was the first consensus definition (8). The Acute Kidney Injury Network (AKIN) criteria (9) modified RIFLE by incorporating an absolute increase in creatinine after the finding that small increases in serum creatinine (SCr) were of prognostic significance (10). In 2009, Waikar and Bonventre proposed a creatinine kinetics (CK)-based definition of AKI using absolute changes in SCr over 24 hours or 48 hours (11). Finally, in 2012, the Kidney Disease Improving Global Outcomes (KDIGO) Work Group proposed another definition that builds upon the AKIN definition (12). Few studies have compared these four criteria to determine the incidence of AKI in critically ill patients.

Our study aimed to compare the incidence of AKI and the ability to predict in-hospital mortality by using RIFLE, AKIN, CK, and KDIGO classifications in critically ill patients.

METHODS

Setting: A prospective, observational, single-center study conducted over 6 months (January 2018 to June 2018) in a 22-bed intensive care unit (ICU) of the Habib Bourguiba University Hospital, Sfax, Tunisia. We included all patients admitted to the ICU during the study period, aged ≥ 18 years, and with an in-hospital stay of at least 48 hours. AKI was defined according to SCr-based criteria per RIFLE, AKIN, CK, and KDIGO; urine output data were not available. Patients aged < 18 years, with end-stage chronic kidney disease (CKD) already undergoing hemodialysis, with CKD without baseline serum creatinine information, and/or with an in-hospital stay of less than 48 hours were not included.

Data collection: SCr was recorded for 10 days or until discharge from the ICU. Complete recovery or loss of renal function, the need for renal replacement therapy (RRT) was also recorded. Age, gender, co-morbidities, primary diagnosis, the need for mechanical ventilation, PaO₂/FiO₂ ratio, the use of catecholamine drugs, in-hospital mortality, length of mechanical ventilation, length of stay in ICU, and pneumonia acquired under mechanical ventilation (PAMV) were noted. Severity was assessed by APACHE II score (13) (Acute Physiology and Chronic Health Evaluation)

and SOFA (14) (Sequential Organ Failure Assessment). Patients were divided into two groups: those who had AKI according to the most sensitive classification (AKI) group, and those who did not have AKI (No-AKI) group.

Definition of acute kidney injury: AKI was defined by using RIFLE, AKIN, CK, and KDIGO criteria. Patients were categorized according to serum creatinine (SCr) and not urine output. We used the lowest known SCr value during the previous 3 months as the baseline creatinine. For patients without a known baseline SCr, we used an estimated baseline. The baseline creatinine was estimated using the simplified modification of diet in renal disease (MDRD) formula, assuming a GFR of 75 mL/min per 1.73 m (2 15,16).

In 2004, the ADQI (Acute Dialysis Quality Initiative) collective (8) developed a system to define and classify AKI by RIFLE (acronym indicating Risk of renal dysfunction; Injury to the kidney; Failure of kidney function, Loss of kidney function, and End-stage kidney disease) criteria. AKI was defined as a 1.5 threshold increase in baseline SCr ($\geq 50\%$) within 7 days. In 2007, AKIN (Acute Kidney Injury Network) (9) adopted the severity criteria of the RIFLE classification with a modification of stage 1 and therefore of the definition which then defined AKI by the abrupt onset (in 48 hours) of a reduction in renal function defined by an increase in blood creatinine ≥ 0.3 mg/dl ($\geq 26.5\mu\text{mol/L}$) or $\geq 50\%$ of its base value within 48 h or to 1.5 to 1.9 times baseline. Waikar and

Bonventre published in 2009 (11) a new classification based on creatinine kinetics (CK). It defines AKI as an increase in creatinine ≥ 0.3 mg/dl ($\geq 26.5\mu\text{mol/L}$) over 24 hours or 0.5 mg/dl ($44\mu\text{mol/L}$) within 48 hours. The KDIGO group classification (Kidney Disease Improving Global Outcomes) was established in 2012 and represents an optimized synthesis of the three previous classifications to define an AKI by the presence of at least 1 of the following 3 diagnostic criteria: increased plasma creatinine ≥ 0.3 mg/dl ($\geq 26.5\mu\text{mol/L}$) within 48 hours or increase in serum creatinine ≥ 1.5 -1.9 times the baseline value within 7 days ($\geq 50\%$)¹². In AKIN and KDIGO stage-3 criteria were not only defined by an increase of baseline SCr ≥ 3 times, but also by initiation of RRT (17).

Renal prognosis was classified as complete recovery or loss of renal function based on the SCr level at discharge compared to that at baseline. Complete recovery of kidney function was defined as a SCr level of no more than 0.5 mg/dL ($44\mu\text{mol/L}$) greater than the baseline value. Loss of renal function was defined as a continuously increasing SCr value or the need for RRT (18).

Statistical analysis: The categorical data were reported as proportions and compared by using Fisher's exact test or the Pearson Chi-square test. Continuous variables were presented as the mean \pm standard deviation, median (IQL), and compared by Student's t-test or by the non-parametric Mann-Whitney U test in case of non-normal distribution. Receiver operating

curves ROC by calculating the area under the curve (AUC) were used to compare the predictive ability for mortality. Logistic regression analysis was used in calculating odds ratios and 95% confidence intervals. A p-value ≤ 0.05 was significant.

RESULTS

Demographic Characteristics, Clinical and Biological Findings

During the study period, 428 patients were admitted. After checking the exclusion criteria, 171 patients were included (Figure 1).

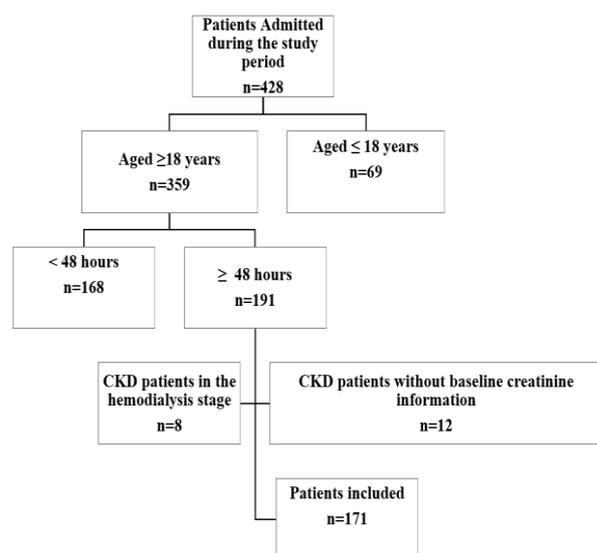


Figure 1 : Flow Chart of study
CKD: end-stage chronic kidney disease

The mean age of patients was 49.26 ± 19.86 years, ranging from 18 to 88 years. 113 (66.08%) were men with a sex ratio of 1.94. Mean APACHE II (SD) 15.1 ± 7.5 . Mean SOFA (SD) 6.7 ± 2.9 . Mean SCr (SD) at admission was 148.65 ± 19.95 $\mu\text{mol/L}$. The previous SCr value was found in only 15.2%. The mean baseline SCr (SD) estimated by MDRD was 92.34 ± 13.52 $\mu\text{mol/L}$. The characteristics of

the whole patient population are detailed in Table 1.

Table 1 : Clinical characteristics of patients on admission

	All patients	No-AKI (n=91)	AKI (n=80)	p
Age (mean \pm SD)	49.26 \pm 19.86	42.66 \pm 19	56.69 \pm 18.2	0.000
Male (gender), n (%)	113 (66.08)	62 (36.25)	51 (29.82)	0.6
APACHE II (mean \pm SD)	15.1 \pm 7.5	12.9 \pm 6.6	17.49 \pm 7.7	0.000
SOFA (mean \pm SD)	6.7 \pm 2.9	5.99 \pm 2.6	7.48 \pm 3.01	0.001
HBP, n (%)	44(25.7)	14 (67.4)	29 (32.6)	0.002
Diabetes, n (%)	32(18.7)	9 (28.1)	23 (71.9)	0.002
Chronic heart failure, n(%)	25(14.6)	6 (24)	19 (76)	0.002
Chronic kidney diseases*, n (%)	2(1.2)			
Chronic lung disease, n(%)	27(15.8)	12 (44.4)	15 (55.6)	0.335
Shock at admission, n (%)	18(10)	3 (16.7%)	15(18.51)	0.001
Polytrauma, n(%)	48(28)	35(72.9)	12 (48%)	0.001
Surgical admission, n (%)	25(15)	13 (52%)	12 (48%)	0.91
Vasopressors, n(%)	96(56.14)	46	50	0.09
Oedema of the lower limbs, n (%)	17(9.9)	5 (29.4%)	12 (70%)	0.04
Mechanical Ventilation, n (%)	151(88.3)	82(47.95)	69(40.35)	0.5
PaO ₂ /FiO ₂ ratio	251.47	275.95 \pm 10	224.67 \pm 85	0.001
Length of stay, median (IQR) (days)	10.5(5-19)	9(5-17)	13(6-20)	0.14
Length of mechanical ventilation, median (IQR) (days)	8(4-13)	6(3-11)	10(4.5-16)	0.036
PAMV, n (%)	82(48%)	32 (39.5%)	49 (60.5%)	0.001
SCr on admission (mean \pm SD)	148.65 \pm 19.95	64.93 \pm 20.76	161.18 \pm 14.28	0.000
Serum creatinine (SCr) baseline using MDRD (mean \pm SD)	92.34 \pm 13.52	95.03 \pm 14.14	89.24 \pm 12.14	0.052
Mortality, n (%)	52(30.4)	10(5.84)	42(24.46)	0.00

AKI: acute kidney injury; HBP: high blood pressure; MDRD: the modification formula of diet in renal disease; SCr: Serum creatinine; APACHE II, Acute Physiology and Chronic Health Evaluation; SOFA: Sequential Organ Failure Assessment; PAMV: Pneumonia Acquired under Mechanical Ventilation

*Chronic renal insufficiency without stage of hemodialysis,

Comparison of the incidence of acute kidney injury by the different classifications

AKI was diagnosed in 55 patients (32.16%) by using the RIFLE classification: 22 with Risk, 18 with Injury, and 15 with Failure (Table 2).

Table 2: Correlation between RIFLE and KDIGO classifications

Definition	RIFLE				Total
	No-AKI	Stage 1	Stage 2	Stage 3	
KDIGO					
No-AKI	91(53.2)	0	0	0	91(53.2)
Stage 1	25(14.6)	22(12.9)	0	0	47(27.5)
Stage 2	0	0	18(10.5)	0	18(10.5)
Stage 3	0	0	0	15(8.8)	15(8.8)
Total	116(67.8)	22(12.9)	18(10.5)	15(8.8)	71(100)

AKI, acute kidney injury; **KDIGO**, Kidney Disease: Improving Global Outcomes; **RIFLE**, Risk, Injury, Failure, Loss of Kidney Function, and End-stage Kidney Disease

The incidence of AKI according to AKIN and KDIGO was identical (Table 3).

Table 3: Correlation between AKIN and KDIGO classifications

Definition	No-AKI	AKIN			Total
		Stage 1	Stage 2	Stage 3	
KDIGO					
No-AKI	91(53.2)	0	0	0	91(53.2)
Stage 1	0	47(27.5)	0	0	47(27.5)
Stage 2	0	0	18(10.5)	0	18(10.5)
Stage 3	0	0	0	15(8.8)	15(8.8)
Total	91(53.2)	47(27.5)	18(10.5)	15(8.8)	171(100)

AKI, acute kidney injury; **AKIN**, Acute Kidney Injury Network; **KDIGO**, Kidney Disease: Improving Global Outcomes.

AKI occurred in 80 patients (46.8%): 47 with stage 1, 18 with stage 2, and 15 with stage 3. When CK criteria were used, AKI occurred in 77 patients (45%): 37 with stage 1, 21 with stage 2, and 19 with stage 3 (Table 4).

Table 4 : Correlation between CK and KDIGO classifications

Definition	No-AKI	CK			Total
		Stage 1	Stage 2	Stage 3	
KDIGO					
No-AKI	91(53.2)	0	0	0	91(53.2)
Stage 1	3 (1.7)	35(20.5)	9(5.3)	0	47(27.5)
Stage 2	0	2(1.16)	12(7)	4(2.34)	18(10.5)
Stage 3	0	0	0	15(8.8)	15(8.8)
Total	94(55)	37(21.6)	21(12.3)	19(11.1)	171(100)

AKI, acute kidney injury; **KDIGO**, Kidney Disease: Improving Global Outcomes; **CK**, creatinine kinetics

The KDIGO criteria identified 25 additional patients with AKI than the RIFLE criteria did. They were identified as stage 1 (Table 2).

Compared with the CK criteria, KDIGO diagnosed 3 more patients as having AKI. Two patients were identified as stage 2 by KDIGO and stage 1 by CK. Nine patients were identified as stage 1 by KDIGO and stage 2 by CK. Four patients were identified as stage 2 by KDIGO and stage 3 by CK (Table 4). The KDIGO and AKIN criteria were more sensitive than RIFLE (46.8% versus 32.16 %, $p < 0.001$) and CK (46.8% versus 45%, $p < 0.001$).

A total of 18 patients (10.52%) received renal replacement therapy (RRT) within 10 days after ICU admission. According to the KDIGO and AKIN criteria, these 18 patients were identified as AKI: 4 with stage 1, 2 with stage 2, and 12 with stage 3. Based on the RIFLE criteria, 17 of the 18 patients were identified as AKI: 3 with Risk, 2 with injury, and 12 with failure; the other patient without AKI received RRT for hyperkalemia. Based on the CK criteria, these 18 patients were identified as AKI: 3 with stage 1, 3 with stage 2, and 12 with stage 3.

Thirty-four patients (42.5%) had complete recovery of renal function, 4 patients needed long-short RRT, and 11 patients (13.5%) lost recovery of renal function and developed chronic kidney failure.

Comparison of outcomes

In-hospital mortality

In-hospital mortality was significantly higher for AKI patients than for non-AKI patients,

regardless of the definition used (Table 1): RIFLE (17.54% versus 12.86%, $p < 0.0001$), CK (23.39% versus 7.01%, $p < 0.0001$) and both KDIGO and AKIN (24.56% versus 5.84%, $p < 0.0001$) criteria (table 5).

Table 5: In-hospital mortality according to AKI stratified by the RIFLE, AKIN, and KDIGO classification schemes

Category	RIFLE	AKIN	CK	KDIGO
None (%)	22(12.86)	10(5.84)	12(7.01)	10(5.84)
Risk/Stage 1 (%)	11(6.43)	23(13.45)	17(9.94)	23(13.45)
Injury/Stage 2 (%)	8(4.68)	8(4.68)	10(5.84)	8(4.68)
Failure/Stage 3 (%)	11(6.43)	11(6.43)	13(7.6)	11(6.43)
Any category (%)	30(17.54)	42(24.56)	40(23.39)	42(24.56)

AKI: acute kidney injury; KDIGO: Kidney Disease: Improving Global Outcomes; RIFLE: Risk: Injury: Failure: Loss of Kidney Function: and End-stage kidney disease; CK: creatinine kinetics

It is worth mentioning that the AKIN and KDIGO classifications have similar results. Mortality rate of patients identified as AKI by KDIGO and AKIN was higher than RIFLE (24.56% versus 17.54%, $p = 0.000$) or CK (24.56% versus 23.39, $p = 0.000$).

Length of stays in the intensive care unit

In our study, the ICU-length of stay was greater in patients with AKI than in those without AKI; however, it was not a significant difference ($p = 0.14$) (Table 1).

Predictive ability for mortality

AKI was associated with in-hospital mortality regardless of the definition used.

We found high Odds ratios for stage 3 of the AKIN-KDIGO and CK classifications of 22.28 and 14.806, respectively. The odds ratio for stage 3 was only 3.4 in the RIFLE classification. The odds ratio increases from

stage 1 to stage 3 for AKIN-KDIGO and CK classifications. But the odds ratio for stage 1 was higher than for stage 3 and stage 2 according to the RIFLE classification (Table 6).

Table 6: Association of different acute kidney injury categories with mortality by multivariable logistic regression models

Criteria	Odds ratio	p	95% CI
RIFLE			
Stage 1	4.7	0.002	1.774 - 12.449
Stage 2	1.2	0.020	1.209 - 9.662
Stage 3	3.4	0.000	3.418 - 40.398
AKIN			
Stage 1	8.1	0.000	3.377 - 19.431
Stage 2	6.48	0.001	2.076 - 20.229
Stage 3	22.28	0.000	5.953 - 83.346
KDIGO			
Stage 1	8.1	0.000	3.377 - 19.431
Stage 2	6.48	0.001	2.076 - 20.229
Stage 3	22.28	0.000	5.953 - 83.346
CK			
Stage 1	5.808	0.000	2.395 - 14.088
Stage 2	6.833	0.000	2.354 - 19.832
Stage 3	14.806	0.000	4.729 - 46.357

CI: confidence interval, AKI: acute kidney injury; AKIN: Acute Kidney Injury Network; KDIGO: Kidney Disease: Improving Global Outcomes; CK: creatinine kinetics; RIFLE: Risk, Injury, Failure, Loss of Kidney Function, and End-stage Kidney Disease

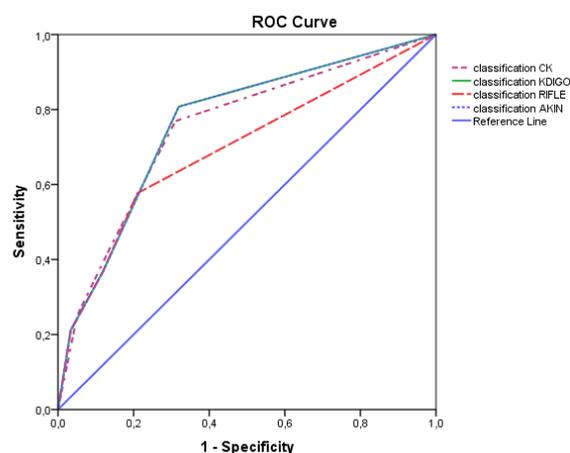


Figure 2: Area under the curves for RIFLE, AKIN, CK and KDIGO classification schemes comparing the predictive ability of RIFLE, AKIN, CK and KDIGO classification schemes for in-hospital mortality.

AKIN, Acute Kidney Injury Network; KDIGO, Kidney Disease: Improving Global Outcomes; RIFLE, Risk, Injury, Failure, Loss of Kidney Function, and End-stage Kidney Disease; ROC, receiver operating characteristic. RIFLE: AUC 0.694 (95% CI 0.603-0.785, $p < 0.001$). AKIN: AUC 0.761 (95% CI 0.682-0.84, $p < 0.001$). CK: AUC 0.75 (95% CI 0.668-0.831, $p < 0.001$). KDIGO: AUC 0.761 (95% CI 0.682-0.84, $p < 0.001$).

The area-under-ROC curves for in-hospital mortality for RIFLE, AKIN, CK, and KDIGO criteria were: 0.694 ($p < 0.001$, $CI_{95\%}$ [0.603-0.785]), 0.761 ($p < 0.001$, $CI_{95\%}$ [0.682-0.84]), 0.75 ($p < 0.001$, $CI_{95\%}$ [0.668-0.831]) and 0.761 ($p < 0.001$, $CI_{95\%}$ [0.682-0.84]) respectively. AKIN and KDIGO were identical. Compared with RIFLE and CK criteria, KDIGO and AKIN had greater predictive ability for in-hospital mortality ($p < 0.001$) (Figure 2).

DISCUSSION

Incidence of acute kidney injury

Our study compared the four criteria and showed that KDIGO and AKIN identified more patients as AKI than RIFLE and CK. Both KDIGO and AKIN criteria identified 25 more patients with AKI than the RIFLE criteria did categorize as stage 1. Compared with the CK criteria, both KDIGO and AKIN diagnosed 3 more patients with AKI. In the literature, a few studies have compared these four criteria in critically ill patients (19–21). Consistent with our results, most of these studies confirmed that KDIGO is the most sensitive criterion and identifies more AKI (17, 18). Zhou et al. compared the four criteria and found the incidences of AKI of 26.4% by RIFLE, 34.1% by AKIN, 37.8% by KDIGO, and 36.1% by Cys-C criteria (22). This finding was comparable to our results. Xiaoxi et al. compared the four definition criteria and found that AKI incidence was the highest with the KDIGO definition (18.3%), followed by the AKIN (16.6%), RIFLE (16.1%), and CK (7%) definitions. AKI incidence appeared markedly

higher in those with low baseline serum creatinine according to the KDIGO, AKIN, and RIFLE definitions, in which AKI may be defined by a 50% increase over baseline (17). Emilio et al. compared the four criteria in patients with sepsis and found that the AKI rate was 74.3% for RIFLE, 81.7% for AKIN, 81.7% for KDIGO, and 77.5% for CK (21). In our sample, higher rates may be explained by the severity of patients on admission, compared to those reported in previous studies. In a large multicenter study conducted in China, Luo et al. compared only 3 criteria (RIFLE, AKIN, and KDIGO), and found that the highest incidence of AKI was diagnosed with the KDIGO criteria (51%), followed by RIFLE (46.9%) and AKIN (38.4%) (23). Another research compared only the RIFLE and the AKIN classifications (24). The reported incidence of AKI differed across the various diagnostic criteria. The incidence of AKI according to the KDIGO definition is the highest due to the addition of an absolute increase criterion (≥ 0.3 mg/dl over 48 hours) to the RIFLE definition and expansion of the time limit for percentage increase ($\geq 50\%$) in the AKIN and CK definition from 48 hours to 7 days (17). Furthermore, without a baseline SCr to serve as a reference point, it is difficult to accurately identify acute SCr elevations and evaluate patient recovery (20). Koeze et al. compared RIFLE, AKIN or KDIGO. They found that AKI incidence rates were respectively 15%, 21%, and 20%, using SCr of RIFLE, AKIN, and KDIGO criteria. Adding

urine output criteria increased AKI incidence rates to 35, 38, and 38% using RIFLE, AKIN, and KDIGO definitions (20). Compared with our study, we used only SCr; we would have found a higher incidence of AKI if we had added urine output. KDIGO and AKIN have similar results, allegedly because most of our patients developed AKI within admission and 48 hours. Moreover, KDIGO and AKIN identify more AKI at stage 3 because they include the need for RRT in the definition of stage 3.

In-hospital mortality

Our results showed that patients diagnosed as AKI had significantly higher in-hospital mortality than non-AKI patients, regardless of the diagnostic criteria used. Actually, AKI is associated with significantly increased mortality, which was related directly to the severity of AKI (10,17,19,20). Furthermore, patients in our sample had very high SOFA and SAPSII scores. Luo et al. (23) demonstrated that in-hospital mortality was significantly higher for patients diagnosed with AKI compared to those without, across all evaluated criteria: RIFLE (27.8% vs. 7%, $p < 0.001$), AKIN (32.2% vs. 7.1%, $p < 0.001$), and KDIGO (27.4% vs. 5.6%, $p < 0.001$). This was attributed to the KDIGO and AKIN criteria identifying more patients with AKI than the alternative definitions. Our result was comparable (26). Conversely, a study by Ülger et al. (27) evaluating in-hospital mortality among critically ill trauma patients showed that first-day ICU mortality did not significantly differ

between AKI and non-AKI groups when applying the RIFLE ($p = 0.565$), AKIN ($p = 0.362$), and KDIGO ($p = 0.362$) definitions. A significant increase in first-day mortality was only observed when AKI was defined by the CK criteria ($p = 0.045$).

Length of stays in the intensive care unit

In our study, ICU length of stay was longer for patients with AKI than for those without AKI, this difference was not significant. Similar findings are reported in the literature regardless of the criteria used (19,22,27).

Mortality prediction

AKI was a heavy global burden that was associated with both short and long-term mortality (7). In our study, KDIGO and AKIN were more predictive of in-hospital mortality than RIFLE and CK. As shown in our results, most studies found that predicted in-hospital mortality increases from stage 1 to stage 3 (19,21,23,26,27). Luo et al. found that compared with the RIFLE criteria, KDIGO was more predictive of in-hospital mortality (AUC 0.757; $p < 0.001$), but there was no significant difference between AKIN and KDIGO (23). In a retrospective analysis of 457 critically ill patients with severe sepsis or septic shock, Pereira et al. (29) found that AKI defined by the AKIN and KDIGO criteria was associated with in-hospital mortality, whereas AKI defined by RIFLE was not—a finding consistent with our own results. Nevertheless, the AUC for in-hospital mortality was comparable across all three classifications: RIFLE (0.652), AKIN (0.686), and KDIGO (0.658), with $p < 0.001$ for

all. Conversely, a separate prospective cohort study (26) demonstrated that the RIFLE, AKIN, and KDIGO criteria were all effective predictors of mortality in critically ill patients, with no significant differences among them. In that study, the AUC was 0.735 for RIFLE, 0.740 for AKIN, and 0.733 for KDIGO ($p < 0.001$ for all).

Limits: There are some limitations to our study. First, we used the simplified MDRD formula as a baseline for patients without known baseline creatinine. Second, we used only serum creatinine to define AKI without considering urine output; this may underestimate the incidence of AKI. Third, the sample size is small, and we have not calculated the power. Even so, this study is one of the few studies conducted in Africa that compares the four criteria of AKI.

CONCLUSION

In conclusion, the KDIGO and AKIN criteria identified a greater number of AKI cases than the RIFLE and CK classifications. Regardless of the definition applied, patients diagnosed with AKI experienced significantly higher in-hospital mortality than those without the condition. Furthermore, KDIGO and AKIN demonstrated superior predictive value for in-hospital mortality compared to RIFLE and CK. Given the high acuity of the emergency setting, utilizing these sensitive and highly predictive criteria is critical for early risk stratification, enabling clinicians to implement timely interventions and potentially improve patient outcomes.

Acknowledgments: None

Contributing authors: All contributed to the writing of the manuscript

Funding: This work was done as part of our daily activities. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of conflict of interest: The authors declare that they have no conflict of interest.

REFERENCES

1. Susantitaphong P, Cruz DN, Cerda J, Abulfaraj M, Alqahtani F, Koulouridis I, et al. World Incidence of AKI: A Meta-Analysis. *Clin J Am Soc Nephrol*. 6 sept 2013;8(9):1482-93.
2. Bouchard J, Acharya A, Cerda J, Maccariello ER, Madarasu RC, Tolwani AJ, et al. A Prospective International Multicenter Study of AKI in the Intensive Care Unit. *Clin J Am Soc Nephrol CJASN*. 7 août 2015;10(8):1324-31.
3. Vincent J-L, Marshall JC, Namendys-Silva SA, François B, Martin-Loeches I, Lipman J, et al. Assessment of the worldwide burden of critical illness: the intensive care over nations (ICON) audit. *Lancet Respir Med*. mai 2014;2(5):380-6.
4. Cruz DN, Ronco C. Acute kidney injury in the intensive care unit: current trends in incidence and outcome. *Crit Care*. 2007;11(4):149.
5. Lewington AJ, Cerdá J, Mehta RL. Raising Awareness of Acute Kidney Injury: A Global Perspective of a Silent Killer. *Kidney Int*. sept 2013;84(3):457-67.
6. Yang L, Xing G, Wang L, Wu Y, Li S, Xu G, et al. Acute kidney injury in China: a cross-sectional survey. *Lancet Lond Engl*. 10 oct 2015;386(10002):1465-71.
7. Cheng X, Wu B, Liu Y, Mao H, Xing C. Incidence and diagnosis of Acute kidney injury in hospitalized adult patients: a retrospective observational study in a tertiary teaching Hospital in Southeast China. *BMC Nephrol*. déc 2017;18(1):203.
8. Bellomo R, Ronco C, Kellum JA, Mehta RL, Palevsky P. Acute renal failure – definition, outcome measures, animal models, fluid therapy and information technology needs: the Second International Consensus Conference of the Acute Dialysis Quality Initiative (ADQI) Group. *Crit Care*. 2004;8(4):R204.
9. Mehta RL, Kellum JA, Shah SV, Molitoris BA, Ronco C, Warnock DG, et al. Acute Kidney Injury Network: report of an initiative to improve outcomes in acute kidney injury. *Crit Care*. 2007;11(2):R31.

10. Chertow GM, Burdick E, Honour M, Bonventre JV, Bates DW. Acute kidney injury, mortality, length of stay, and costs in hospitalized patients. *J Am Soc Nephrol JASN.* nov 2005;16(11):3365-70.
11. Waikar SS, Bonventre JV. Creatinine Kinetics and the Definition of Acute Kidney Injury. *J Am Soc Nephrol.* mars 2009;20(3):672-9.
12. Khwaja A. KDIGO Clinical Practice Guidelines for Acute Kidney Injury. *Nephron.* 7 août 2012;120(4):c179-84.
13. Akavipat P, Thinkhamrop J, Thinkhamrop B, Sriraj W. ACUTE PHYSIOLOGY AND CHRONIC HEALTH EVALUATION (APACHE) II SCORE – THE CLINICAL PREDICTOR IN NEUROSURGICAL INTENSIVE CARE UNIT. *Acta Clin Croat.* mars 2019;58(1):50-6.
14. Vincent JL, Moreno R, Takala J, Willatts S, De Mendonça A, Bruining H, et al. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. *Intensive Care Med.* juill 1996;707-10.
15. Ma Y-C, Zuo L, Chen J-H, Luo Q, Yu X-Q, Li Y, et al. Improved GFR estimation by combined creatinine and cystatin C measurements. *Kidney Int.* déc 2007;72(12):1535-42.
16. Bagshaw SM, Uchino S, Cruz D, Bellomo R, Morimatsu H, Morgera S, et al. A comparison of observed versus estimated baseline creatinine for determination of RIFLE class in patients with acute kidney injury. *Nephrol Dial Transplant.* 1 sept 2009;24(9):2739-44.
17. Zeng X, McMahon GM, Brunelli SM, Bates DW, Waikar SS. Incidence, Outcomes, and Comparisons across Definitions of AKI in Hospitalized Individuals. *Clin J Am Soc Nephrol.* 7 janv 2014;9(1):12-20.
18. The VA/NIH Acute Renal Failure Trial Network*. Intensity of Renal Support in Critically Ill Patients with Acute Kidney Injury. *N Engl J Med.* 3 juill 2008;359(1):7-20.
19. Tsai T-Y, Chien H, Tsai F-C, Pan H-C, Yang H-Y, Lee S-Y, et al. Comparison of RIFLE, AKIN, and KDIGO classifications for assessing prognosis of patients on extracorporeal membrane oxygenation. *J Formos Med Assoc.* nov 2017;116(11):844-51.
20. Koeze J, Keus F, Dieperink W, van der Horst ICC, Zijlstra JG, van Meurs M. Incidence, timing and outcome of AKI in critically ill patients varies with the definition used and the addition of urine output criteria. *BMC Nephrol.* déc 2017;18(1):70.
21. Rodrigo E, Suberviola B, Albines Z, Castellanos Á, Heras M, Rodriguez-Borregán JC, et al. A comparison of acute kidney injury classification systems in sepsis. *Nefrol Publicacion Of Soc Espanola Nefrol.* oct 2016;36(5):530-4.
22. Zhou J, Liu Y, Tang Y, Liu F, Zhang L, Zeng X, et al. A comparison of RIFLE, AKIN, KDIGO, and Cys-C criteria for the definition of acute kidney injury in critically ill patients. *Int Urol Nephrol.* janv 2016;48(1):125-32.
23. Luo X, Jiang L, Du B, Wen Y, Wang M, Xi X. A comparison of different diagnostic criteria of acute kidney injury in critically ill patients. *Crit Care.* 2014;18(4):R144.
24. Lopes JA, Fernandes P, Jorge S, Gonçalves S, Alvarez A, Costa e Silva Z, et al. Acute kidney injury in intensive care unit patients: a comparison between the RIFLE and the Acute Kidney Injury Network classifications. *Crit Care.* 2008;12(4):R110.
25. Bellomo R, Ronco C, Mehta RL, Asfar P, Boissramé-Helms J, Darmon M, et al. Acute kidney injury in the ICU: from injury to recovery: reports from the 5th Paris International Conference. *Ann Intensive Care.* déc 2017;7(1):49.
26. Levi TM, de Souza SP, de Magalhães JG, de Carvalho MS, Cunha ALB, Dantas JGA de O, et al. Comparison of the RIFLE, AKIN and KDIGO criteria to predict mortality in critically ill patients. *Rev Bras Ter Intensiva.* 2013;25(4):290-6.
27. Ülger F, Pehlivanlar Küçük M, Küçük AO, İlkaya NK, Murat N, Bilgiç B, et al. Evaluation of acute kidney injury (AKI) with RIFLE, AKIN, CK, and KDIGO in critically ill trauma patients. *Eur J Trauma Emerg Surg.* août 2018;44(4):597-605.
28. Libório AB, Leite TT, Neves FM de O, Teles F, Bezerra CT de M. AKI Complications in Critically Ill Patients: Association with Mortality Rates and RRT. *Clin J Am Soc Nephrol.* 7 janv 2015;10(1):21-8.
29. Pereira M, Rodrigues N, Godinho I, Gameiro J, Neves M, Gouveia J, et al. Acute kidney injury in patients with severe sepsis or septic shock: a comparison between the 'Risk, Injury, Failure, Loss of kidney function, End-stage kidney disease' (RIFLE), Acute Kidney Injury Network (AKIN) and Kidney Disease: Improving Global Outcomes (KDIGO) classifications. *Clin Kidney J.* 13 déc 2016;sfw107.

Knowledge and Practices of Mothers Regarding First Aid in Domestic Accidents: A Cross-Sectional Study in Sfax, Tunisia

Mahmoud Ladhar ¹, Zouhour Hadrich ¹, Farah Bouattour ², Chahd Ben Alaya ², Yosri Smeoui ¹, Badria Moalla ², Faiza Safti ¹.

Department[s] and Institutional Affiliation[s]:

(1) Pediatric Intensive Care Unit, CHU Hedi Chaker, University of Sfax; Faculty of Medicine of Sfax, Sfax, Tunisia.

(2) Private Faculty of Paramedical Sciences of Sfax

Corresponding Author: Ladhar Mahmoud; Pediatric Intensive Care Unit, CHU Hedi Chaker, University of Sfax, Tunisia: Email :

Mahmoud.ladhar@gmail.com; Phone : +216 52 278 276

Abstract

Background: Domestic accidents are among the leading causes of morbidity and mortality in children worldwide, particularly in low- and middle-income countries. Mothers, as primary caregivers, play a critical role in preventing and managing these injuries through appropriate first-aid practices. However, their level of knowledge and preparedness remains poorly documented in Tunisia.

Aim: This study aimed to assess the knowledge and practices of mothers regarding first aid in domestic accidents among children in Sfax, Tunisia.

Methods: A descriptive cross-sectional study was conducted among 50 mothers attending the pediatric and pediatric intensive care units at Hadi Chaker University Hospital in Sfax during 2025. Data were collected using a structured questionnaire composed of 35 items evaluating knowledge and practical skills in six domains: fractures, burns, choking, wounds, poisoning, and electrocution. Scores were categorized as poor, moderate, or good.

Results: Most participants were aged between 31 and 40 years old (42%) and lived in urban areas (58%). Overall, 56% of mothers demonstrated an average level of first-aid knowledge, while only 22% achieved a good level. Knowledge gaps were most evident in the management of burns (65% poor knowledge) and poisoning (70% poor knowledge). Mothers with higher educational levels and previous first-aid training scored significantly better ($p < 0.05$).

Conclusion: The study highlights insufficient first-aid knowledge and practices among Tunisian mothers, especially in handling burns, poisoning, and choking. Integrating community-based education and structured first-aid training programs for mothers is crucial to reducing childhood morbidity and mortality related to domestic accidents.

Keywords: Domestic accidents; First aid; Mothers; Knowledge; Practices; Child safety.

INTRODUCTION

Domestic accidents represent a major public health problem and are a leading cause of preventable injuries and deaths among children. According to the World Health Organization, nearly 950,000 children die annually due to unintentional injuries, mostly occurring at home (1, 2). In developing countries, these accidents often result from limited parental awareness and inadequate first-aid responses (3).

Mothers, as primary caregivers, are often the first to respond when accidents occur. Their actions can determine whether a child recovers safely or faces long-term complications. However, studies across the Middle East and North Africa have revealed substantial gaps in maternal first-aid knowledge, with many relying on inappropriate traditional practices such as applying toothpaste to burns or inducing vomiting after poisoning (4). This study aims to assess the knowledge level and practices of mothers regarding first aid in domestic accidents in Sfax, Tunisia.

METHODS

A descriptive cross-sectional study was conducted between January and April 2025 in the pediatric and pediatric intensive care units of Hadi Chaker University Hospital, Sfax, Tunisia.

Participants: Fifty mothers aged 18 years or older who resided in Sfax and consented to participate were included.

Sampling: A non-probabilistic purposive sampling technique was used, selecting participants who met the inclusion criteria. The investigator explained to the patient and/or his accompanying relatives the aim of the study, obtained the consent, and then began the interview.

Data collection: A validated 35-item questionnaire (Appendix 1) assessed knowledge and practices regarding six types of domestic accidents: fractures, burns, wounds, choking, poisoning, and electrical injuries. A data collection instrument for calculating scores (Appendix 2). We used a questionnaire inspired by three studies focusing on home-related children's accidents (5-7). The questionnaire used consisted of 35 items. For each item, answers were proposed, and there was one correct option. Each correct answer was coded as 1 point. The overall Knowledge and Practice Score was 44 points. Knowledge and Practice were considered as low (score 0-13), moderate (score 14-25), or good (score ≥ 26). The overall Knowledge Score was 25 points. Knowledge was considered low (score 0-8), moderate (score 9-16), or good (score ≥ 17). Practice Score was 19 points. Knowledge was considered low (score 0-6), moderate (score 7-11), or good (score ≥ 12).

Data analysis: Data were analyzed with descriptive statistics and chi-square tests ($p < 0.05$).

Ethics: Oral informed consent was obtained before participation, and confidentiality was maintained.

RESULTS

Sociodemographic Characteristics

A total of 50 mothers participated in this study. Most participants were aged between 31 and 40 years (42%), followed by 18–30 years (36%) and 41–50 years (22%). The majority lived in urban areas (58%), while 42% resided in rural zones. Regarding education, 46% had completed secondary education, 22% university education, and 32% had primary or no formal education.

Most participants were married (86%), and 68% were housewives. Approximately 48% had two or more children, and 60% reported that at least one of their children had previously experienced a domestic accident (Table 1).

Table 1. Demographic characteristics of participating mothers (n = 50)

Variable	Category	n (%)
Age (years)	18–30	18 (36%)
	31–40	21 (42%)
	41–50	11 (22%)
Residence	Urban	29 (58%)
	Rural	21 (42%)
Education level	Primary or less	16 (32%)
	Secondary	23 (46%)
	University	11 (22%)
Marital status	Married	43 (86%)
	Divorced/Widowed	7 (14%)
Employment status	Housewife	34 (68%)
	Employed	16 (32%)

Overall Knowledge and Practice Scores

The overall knowledge mean score was 14.8 ± 4.3 out of 25, indicating a moderate level. Twenty-two percent of mothers demonstrated good knowledge, 56% had moderate knowledge, and 22% showed poor knowledge. Similarly, the mean practice score was 10.1 ± 3.9 out of 19, with 18% achieving good performance, 60% moderate, and 22% poor. Mothers who had previously received first-aid training scored significantly higher in both knowledge (mean 17.6 vs 13.4, $p < 0.01$) and practice ($p < 0.05$).

Sixty percent of mothers correctly identified fracture symptoms, but only 36% knew that immobilization is the first step. Good knowledge in 24%, moderate in 60%, and poor in 16%.

Sixty-five percent had poor knowledge; 72% incorrectly believed toothpaste or oil should be applied. Only 22% mentioned cooling the burn under running water. Knowledge correlated with education level ($p < 0.001$).

Forty-six percent had a poor understanding of choking management. Only 38% identified the Heimlich maneuver, while 30% proposed an unsafe method to insert fingers.

Fifty-eight percent showed moderate knowledge. Forty percent would wash with water and antiseptic, while 34% would apply inappropriate substances. Twenty-two percent mentioned tetanus prophylaxis.

Seventy percent had poor knowledge. Eighty percent reported they would induce vomiting, while only 12% would call emergency services.

Sixty-two percent failed to mention cutting electricity before touching the victim. Twenty-two percent knew resuscitation should begin after ensuring safety. Table 2 details mothers' knowledge regarding domestic accidents.

Table 2: Knowledge of Mothers Regarding Domestic Accidents

Domains		Poor (%)	Moderate rate (%)	Good (%)
Fractures	Knowledge	64	34	2
	Practical skills	78	22	-
Burns	Knowledge	70	30	-
	Practical skills	78	20	2
Choking	Knowledge	46	24	30
	Practical skills	58	38	4
Wounds	Knowledge	48	-	52
	Practical skills	38	46	16
Poisoning	Knowledge	90	-	10
	Practical skills	88	-	12
Electrical accidents	Knowledge	62	38	-
	Practical skills	70	-	30
Total	Knowledge	28	52	20
	Practical skills	28	64	8

A significant correlation was found between education level and both knowledge ($r = 0.62$, $p < 0.01$) and practice ($r = 0.54$, $p < 0.05$). Prior first-aid training was also associated with better performance ($p < 0.05$). No significant association was found with age, marital status, or employment.

DISCUSSION

This study offers a comprehensive assessment of mothers' knowledge and practices concerning pediatric emergencies—such as fractures, burns, choking, chemical intoxications, and electrical accidents—and highlights significant gaps that may compromise the safety and outcomes of affected children. The results demonstrate substantial discrepancies between theoretical knowledge and the practical ability to perform appropriate first-aid measures.

Overall, 52% of mothers had a moderate knowledge level, while 28% had insufficient knowledge and only 20% reached an adequate level. Practical performance was even more concerning, with 64% demonstrating moderate practices, 28% poor practices, and just 8% achieving a good level. This discordance between awareness and behavior likely reflects limited access to structured first-aid training, persistent cultural misconceptions, and reliance on informal and unreliable information sources. Although 30% of participants had received some form of first-aid education, only 16% held official certification, and fewer than half (44%) had a first-aid kit at home.

Specific emergencies revealed several critical shortcomings. Regarding fractures, although immobilization was generally known, recognition of clinical signs was limited, and some reported inappropriate actions, findings consistent with previous research (8). In burn

management, cultural beliefs persisted, with 20% applying toothpaste and 8% using honey-unsafe practices also reported elsewhere (3, 9). Choking management emerged as the most alarming domain; only 14% of mothers knew the Heimlich maneuver, while many suggested potentially harmful responses such as giving water, echoing trends noted in other studies (10). Chemical intoxication responses were largely inaccurate, with dangerous behaviors such as inducing vomiting or giving milk remaining common, consistent with earlier findings (11). Electrocutation management also reflected gaps: although 64% recognized the need to remove the child from the electrical source, only 30% mentioned turning off the power beforehand, a critical step emphasized in prior work (12).

Despite these shortcomings, partial knowledge in certain areas suggests that targeted educational strategies could be effective. Future programs should combine structured theoretical instruction with practical, hands-on training, adapted to the sociocultural context of the population to ensure optimal and sustainable improvements.

Limitations include the scarcity of local literature, time constraints, potential response bias, a relatively small sample size, and participant reluctance. Nonetheless, the study provides important insights: it is one of the first Tunisian investigations on maternal

preparedness for pediatric emergencies, employs a validated questionnaire, identifies clinically relevant deficiencies, and offers evidence-based recommendations for future community interventions.

CONCLUSION

Mothers in Sfax demonstrate moderate but insufficient knowledge and practices regarding first aid during domestic accidents. Implementing structured education programs and regular awareness campaigns is essential to improve emergency preparedness and reduce childhood morbidity and mortality.

Acknowledgments

The authors thank all participating mothers and the staff of Hedi Chaker University Hospital for their cooperation.

Conflict of interest: None declared.

Funding: No external funding was received.

REFERENCES

- 1- Peden M. *World report on child injury prevention appeals to Keep Kids Safe. Inj Prev* 2008;14:413–4
- 2- Li C, Jiao J., Hua G. *Global burden of all cause-specific injuries among children and adolescents from 1990 to 2019: a prospective cohort study. International Journal of Surgery (2024)* 110:2092–2103.
- 3- Nguyen T, Hoang M, Pham L. *Assessing caregiver responses to pediatric burns in low-resource settings. J Burn Care Res.* 2019;40(1):67-73.
- 4- Hazazi, Y. O., Mahmoud, M. A., & Al Ali, M. O. (2021). *Knowledge and awareness of parents in the Kingdom of Saudi Arabia regarding unintentional home injuries in children 2019 a descriptive cross-sectional study. Journal of Family Medicine and Primary Care*, 10, 243–248.
- 5- Al-Bshri et Jahan, « *Prevalence of home-related injuries among children under 5 years old and practice of mothers toward first aid in Buraidah, Qassim* ». *J Family Med Prim Care.* 2021;10:1234-1240
- 6- Anazi et al., « *Impact of Health Education on Maternal Knowledge Regarding Choking Prevention and First Aid in Children, Riyadh, Saudi Arabia. Front Public Health.* 2024;12:1376033.
- 7- Wani et al., « *Pediatric First Aid, Trauma Knowledge, and Attitude among Parents and General Population in Aseer Region, Southern Saudi Arabia* ». *SAGE Open Med.* 2022;10:20503121221126762

8- Garcia P, Torres L, Mendes R. *Parental knowledge and management of pediatric fractures: A cross-sectional analysis. J Pediatr Emerg Care.* 2018;34(2):115-21.

9- Ghosh S. *Cultural beliefs and traditional remedies in burn management: A global review. Burns.* 2020;46(5):1121-8.

10- White R, Johnson P, Clarke S. *Caregivers' knowledge of choking management and factors influencing first-aid competence. Child Health J.* 2020;24(3):301-9.

11- Hernandez J, Ruiz M, Lopez A. *Misconceptions in the management of household chemical intoxications among caregivers. Clin Toxicol.* 2019;57(7):566-73.

12- Liu X, Wang Y. *Safety practices and parental awareness in pediatric electrical injuries. Pediatr Emerg Med.* 2020;37(4):245-52.

Appendix 1

Questionnaire on Mothers' Knowledge and Practices in Pediatric Emergency

Situations

This questionnaire aims to assess mothers' awareness and behaviors regarding pediatric first aid. You are kindly requested to complete this questionnaire and share it with other participants.

I. Sociodemographic Data

Age:

City:

Region:

Marital Status: Married () Divorced ()

Educational Level:

Illiterate () Primary School () Middle School ()

High School () University ()

Employment Status:

Housewife () Employed ()

Number of Children:

1 () 2 () ≥ 3 ()

Children's Age Groups (multiple answers possible):

1 day–1 month () 1 month–1 year () 2–3 years

() 4–6 years ()

7–12 years () 13–18 years ()

Self-assessed level of knowledge in pediatric first aid:

Good () Very Good () Excellent ()

Insufficient ()

Have you attended a first aid training course?

Yes () No ()

Do you hold a first aid certification?

Yes () No ()

Sources of information (multiple answers possible):

Physician () Books/Articles ()

Internet/Television ()

Family/Relatives/Friends () Other:

Do you have a first aid kit (bag, home, or car)?

Yes () No ()

In which of the following situations would you be able to provide first aid? (multiple answers possible)

Burns () Fractures () Minor wounds ()

Choking () Poisoning () Other:

II. Knowledge and Practices in Emergency Situations

Fractures

Knowledge: (1)

Do you know how to manage a suspected fracture in a child?

Yes () No ()

Common symptoms of fractures (multiple answers possible):

Severe pain () Swelling ()

Bruising/discoloration ()

Bleeding at fracture site () Nausea/vomiting ()

Fainting () Deformity () Inability to move ()

Other:

Practices:

If you suspect a limb fracture, you should:

Immobilize with a splint immediately ()

Prevent movement and seek medical care ()

Place in an ice bath ()

I do not know ()

Burns

Knowledge:

Do you know the degrees of burns?

Yes () No ()

Do you know how to provide first aid for each degree?

Yes () No () First-degree only () First and second degree only () Other:

Most common causes of burns at home (multiple answers possible):

Fire () Hot liquids/steam () Sun exposure ()

Household appliances () Electricity ()

Chemicals ()

Have you ever managed a third- or fourth-degree burn?

Yes () No ()

Practices:

If yes, what did you do?

Treated as superficial burn ()

Took child directly to hospital ()

Applied ointment and waited ()

Other:

Management of superficial burns (multiple answers possible):

Cold water () Ice () Toothpaste () Honey ()

Oils () Other:

Choking

Knowledge: (2)

Is choking a major cause of death in preschool children?

Yes () No () I do not know ()

Do you know first aid for choking?

Yes () No () I do not know ()

At what age can children safely chew solid foods such as nuts?

1 year () 2 years () 3 years () 4 years ()

Practices:

Prevention measures (multiple answers possible):

Avoid small toys ()

Supervise during eating and play ()

Encourage laughing while eating ()

Place large amounts of food in the mouth ()

An 8-month-old infant is choking but conscious:

Back blows and chest thrusts ()

Hang upside down ()

Start CPR ()
Unconscious 10-year-old choking in restaurant:
Abdominal thrusts ()
Finger sweep ()
Start CPR ()
Back slap ()
Conscious 7-year-old unable to speak:
Give water ()
Abdominal thrusts ()
Encourage coughing ()
Ask to take a deep breath ()

Wounds

Knowledge:
Most common superficial injuries (multiple answers possible):
Head injuries () Dental trauma ()
Limb injuries () Eye injuries ()
Practices:
Wash wound with water ()
Apply pressure with a cloth ()
Apply ice ()
Disinfect and bandage ()
Leave to heal ()
Other:

Chemical Poisoning

Knowledge: (3)
Has your child experienced chemical or medication poisoning?
Yes () No ()
Practices:
If yes, what did you do?
Induce vomiting ()
Give cold water ()
Give milk and egg white ()
Take to the hospital ()
Other:

Electrical injuries:

Knowledge:
Electrocution is:
Passage of electric current causing injury ()
Passage of electric current causing death ()
Passage of electric current causes mild tingling ()
Practices:
First action to take in case of an electrical accident:
Cut off the electricity source ()
Move the victim away from the source ()
Place in recovery position ()

1- Al-Bshri et Jahan, « Prevalence of home-related injuries among children under 5 years old and practice of mothers toward first aid in Buraidah, Qassim ».

2- Anazi et al., « Impact of Health Education on Maternal Knowledge Regarding Choking Prevention and First Aid in Children, Riyadh, Saudi Arabia.

3- Wani et al., « Pediatric First Aid, Trauma Knowledge, and Attitude among Parents and General Population in Aseer Region, Southern Saudi Arabia ».

Appendix 2

Data Collection Instrument and Scoring System

This study combined four validated questionnaires derived from a cross-sectional study, comprising 35 items.

Overall Knowledge and Practice Score (Total = 44):

0–13: Low
14–25: Moderate
≥26: Good

Overall Knowledge Score (Total = 25):

0–8: Low
9–16: Moderate
≥17: Good

Overall Practice Score (Total = 19):

0–6: Low
7–11: Moderate
≥12: Good

Subscores:

Fractures:

Knowledge (6): 0–2 Low | 3–4 Moderate | ≥5 Good

Practice (2): 0–1 Low | ≥2 Good

Wounds:

Knowledge (1): 0 Low | ≥1 Good

Practice (2): 0–1 Low | ≥2 Good

Burns:

Knowledge (6): 0–3 Low | 4 Moderate | ≥5 Good

Practice (4): 0–2 Low | 3 Moderate | ≥4 Good

Choking:

Knowledge (3): 0–1 Low | 2 Moderate | ≥3 Good

Practice (5): 0–2 Low | 3–4 Moderate | ≥5 Good

Chemical Poisoning:

Knowledge (1): 0 Low | ≥1 Good

Practice (1): 0 Low | ≥1 Good

Electrical accidents:

Knowledge (2): 0 Low | 1 Moderate | ≥2 Good

Practice (1): 0 Low | ≥1 Good

Assessment of occupational stress among emergency department staff: a descriptive study at Ibn Jazzar Hospital in Kairouan

Imen Mabrouk, Imen Ketata, Raja Kalai, Chafiaa Bouhamed

Emergency Department, Ibn Jazzar Hospital, Kairouan, Tunisia.

Corresponding author: Imen Mabrouk; Emergency Department, Ibn Jazzar Hospital, Kairouan, Tunisia

Abstract

Introduction: Work in emergency departments is recognized as highly exposed to occupational stress due to heavy workloads, time pressure, and the severity of clinical situations. This stress can have negative consequences on healthcare workers' health, work quality, and patient safety.

Objective: To assess the level of occupational stress among emergency department staff and to identify associated organizational factors.

Methods: This is a descriptive study conducted in the emergency department of Ibn Jazzar Hospital in Kairouan between November 2024 and January 2025. Occupational stress was assessed using the Karasek questionnaire among 44 healthcare professionals.

Results: The prevalence of high psychological demand was high (65.1%). Most participants (90.5%) showed moderate to high decision latitude. Social support was considered insufficient among a large proportion of participants. Sixty percent of professionals were classified as being in a job strain situation, mainly general practitioners, predominantly female, aged between 30 and 49 years.

Conclusion: Occupational stress is very common among emergency department staff. These findings highlight the need to improve work organization and strengthen institutional support to preserve healthcare workers' health and the quality of care.

Keywords: Occupational stress, Emergency department, Karasek questionnaire, Job strain, General practitioners.

INTRODUCTION

Professional practice in emergency departments is recognized as one of the most stressful work environments in healthcare. Professionals are repeatedly confronted with life-threatening emergencies, heavy workloads,

long working hours, and strong emotional pressure.

This chronic stress can lead to emotional exhaustion, reduced quality of care, and negative impacts on the physical and psychological well-being of healthcare workers. Assessing occupational stress,

therefore, represents a major challenge in improving working conditions and preventing psychosocial risks.

This study aimed to assess the level of stress among emergency department staff and to identify the main contributing factors.

METHODS

Type and Setting of the Study: This was a descriptive cross-sectional study conducted in the emergency department of Ibn Jazzar Hospital in Kairouan.

Study Population: The study included 44 healthcare professionals working in the emergency department during the period from November 2024 to January 31, 2025. The questionnaire was self-administered. The investigator explained the aims, obtained the consent, delivered the questionnaire to the participants, and then collected the completed survey instrument.

Data Collection Tool: Occupational stress was assessed using the Karasek questionnaire, which explores three main dimensions: Decision latitude (questions 1 to 9), Psychological demand (questions 10 to 18), Social support, including supervisory support (questions 19 to 23), and coworker support (questions 24 to 29) [1, 2]. Responses were scored using a four-point Likert scale. Job strain is defined as high psychological demand (>20) combined with low decision latitude (<71). Isostrain corresponds to a job strain situation associated with low social support (<24).

Statistical Analysis: Data were analyzed using descriptive statistics. Results are expressed as means, medians, standard deviations, and percentages.

RESULTS

The sample consisted mainly of general practitioners (86%), with a predominance of females (sex ratio = 4.5). The most represented age group was 45–49 years (34.9%), followed by 35–39 years (23.3%). Regarding working hours, 72.7% of participants reported 24-hour shifts, compared with 22.7% working 8-hour shifts.

Assessment of Occupational Stress: High psychological demand was observed in 65.1% of participants, with a median score of 29.

Decision latitude was moderate to high in 90.5% of professionals.

Social support was considered insufficient, with a median score of 28, reflecting low perceived support.

Overall, 60% of participants were classified as being in a job strain situation, mainly female general practitioners aged between 30 and 49 years.

Table 1: Karasek questionnaire scores among emergency department staff.

Dimension	N	Mean	Standard Deviation	Median
Psychological demand	44	28.2	4.6	29
Decision latitude	44	29.5	3.9	29.5
Social support	44	27.5	3.2	28
Global Karasek score	44	58.5	5.2	60

DISCUSSION

The present study highlights a high prevalence of occupational stress among emergency department staff, with 60% of participants classified as being in a job strain situation according to the Karasek model. This finding confirms that emergency departments represent a high psychosocial risk work environment, as widely reported in the literature [1,2].

High Psychological Demand: The high psychological demand observed in 65.1% of participants reflects a substantial mental workload. Emergency professionals are exposed to constant time pressure, unpredictable activity levels, and often difficult clinical situations, which are recognized as major determinants of occupational stress [3]. Previous studies have shown that emergency physicians experience higher levels of psychological demand than those observed in other medical specialties [4].

According to the Karasek model, excessive psychological demand that exceeds individuals' coping capacities is associated with an increased risk of psychological and somatic disorders [1,2].

Decision Latitude: An Insufficient Protective Factor: In our study, 90.5% of participants exhibited moderate to high decision latitude, reflecting a certain level of professional autonomy. Decision latitude is traditionally considered a protective factor against occupational stress [1]. However, despite this autonomy, a significant proportion of

participants remained in a job strain situation. This suggests that decision-making autonomy alone is insufficient to counterbalance the effects of excessive psychological demand, particularly when workload is chronic [5].

Social Support and Risk of Isolation: Perceived social support was generally low in our population. Social support, whether from supervisors or colleagues, plays a fundamental role in preventing stress and burnout [6]. In the Karasek model, the combination of high psychological demand, low decision latitude, and low social support defines the isostrain situation, considered the most harmful to health [7]. Nevertheless, the high level of agreement regarding help from colleagues suggests a certain level of team cohesion, which could serve as a lever for improving the psychosocial work climate.

Most professionals classified as being in a job strain situation were general practitioners, predominantly female, aged between 30 and 49 years. Several studies have shown that female physicians are more exposed to occupational stress and burnout, partly due to the dual burden of professional and family responsibilities [8]. This age range also corresponds to a period of high professional involvement, increasing vulnerability to chronic stress.

In our study, 72.7% of participants worked 24-hour shifts, a factor widely recognized as a source of stress and fatigue. Prolonged working hours and shift work disrupt circadian rhythms,

impair sleep quality, and increase the risk of medical errors [9]. Several studies have established a direct link between excessive working hours, emotional exhaustion, and deterioration in the quality of care [10,11].

The results of this study emphasize the need to implement organizational interventions to reduce psychological workload. Strategies targeting work organization, schedule optimization, and strengthening institutional support are recognized as more effective than isolated individual approaches in preventing occupational stress [12].

LIMITATIONS: This study has several limitations, including its single-center design and the small sample size, which may limit the generalizability of the results. The use of a self-administered questionnaire may introduce reporting bias. Nevertheless, the use of the internationally validated Karasek questionnaire provides strong methodological robustness to the assessment of occupational stress.

CONCLUSION

Occupational stress is frequent and concerning among emergency department staff. Improving

working conditions, better organization of work schedules, and strengthening social support appear essential to reduce the risk of job strain and to preserve the quality of care.

REFERENCES

1. Karasek RA. Job demands, job decision latitude, and mental strain: implications for job redesign. *Adm Sci Q.* 1979;24:285–308.
2. Karasek R, Theorell T. *Healthy work: stress, productivity, and the reconstruction of working life.* New York: Basic Books; 1990.
3. McVicar A. Scoping the common antecedents of job stress and job satisfaction for nurses. *Int J Nurs Stud.* 2016;52:421–432.
4. Shanafelt TD, et al. Burnout and satisfaction with work-life balance among US physicians. *Arch Intern Med.* 2015;172:1377–1385.
5. Johnson JV, et al. Job strain, work place social support, and cardiovascular disease. *Am J Public Health.* 2008;98:427–433.
6. Cohen S, Wills TA. Stress, social support, and the buffering hypothesis. *Psychol Bull.* 1985;98:310–357.
7. Theorell T, et al. A systematic review of studies in the demand–control–support model of work stress. *Scand J Work Environ Health.* 2015;41:1–14.
8. Guille C, et al. Work-family conflict and burnout among physicians. *J Womens Health.* 2017;26:1–8.
9. Lockley SW, et al. Effects of health care provider work hours and sleep deprivation on safety and performance. *Sleep.* 2007;30:1–8.
10. West CP, et al. Interventions to prevent and reduce physician burnout. *Lancet.* 2018;388:2272–2281.
11. Chakroun W O, Rejeb I , Kammoun L et al. Evaluation of stress among emergency staff: survey in a Tunisian emergency department. *Ann Fr Anesth Reanim.* 2013;32:565-71.
12. Ruotsalainen JH, et al. Preventing occupational stress in healthcare workers. *Cochrane Database Syst Rev.* 2015.

Acute Poisoning with Organophosphate Insecticides: A Report of 26 Cases and Literature Review

Nabila Choubane

University of Health Sciences, Faculty of Medicine Youcef El Khatib, Department of Emergency Medicine, University Hospital of Algiers, Algeria

Corresponding author: Nabila Choubane; University of Health Sciences, Faculty of Medicine Youcef El Khatib, Department of Emergency Medicine, University Hospital of Algiers, Algeria; email: nabila.choubane@yahoo.com

Abstract

Introduction: Acute organophosphate (OP) poisoning remains a major public health concern in regions with intensive agricultural activity. This study aimed to describe the clinical presentation, biological findings, therapeutic management, and outcomes of patients admitted with acute OP poisoning to a tertiary emergency department.

Methods: We conducted a retrospective descriptive study over 12 months (January–December 2023) at the University Hospital of Algiers emergency department. Patients with a confirmed diagnosis of acute OP poisoning were included. Data collected encompassed demographics, clinical presentation, laboratory findings, therapeutic interventions, and patient outcomes.

Results: Twenty-six patients were included, with a mean age of 29.1 ± 10 years. Females accounted for 69% of cases, and 65% of poisonings were intentional. Muscarinic manifestations were present in all patients, while central nervous system signs and nicotinic manifestations were observed in 42% and 19%, respectively. The mean plasma cholinesterase level was 800 IU/L. All patients received atropine; pralidoxime was administered in 19% due to limited availability. Mechanical ventilation was required in 35%, vasopressors in 23%, and anticonvulsants in 8% of cases. The overall mortality rate was 23%, and the mean hospital stay was 11 ± 2.3 days.

Conclusion: Acute OP poisoning is common, severe, and potentially fatal. Early recognition, prompt supportive care, and improved access to specific antidotes such as oximes are crucial. Preventive strategies, including pesticide regulation and psychosocial support, are essential to reduce morbidity and mortality.

Keywords: Organophosphate; Poisoning; Clinical characteristics; Therapeutic management; Outcomes

INTRODUCTION

Organophosphate (OP) insecticides are widely used in agriculture due to their efficacy, low cost, and availability (1,2). However, their high toxicity makes them a common cause of acute poisoning, particularly in low- and middle-income countries and rural areas (1,2). OPs exert their

toxic effects through irreversible inhibition of acetylcholinesterase, leading to accumulation of acetylcholine at synaptic junctions and overstimulation of muscarinic, nicotinic, and central cholinergic receptors (3).

Pesticide poisoning represents a major public health problem worldwide. According to WHO estimates reported by Mew et al.,

approximately 3 million cases occur each year, resulting in over 250,000 deaths, the majority of which are attributable to organophosphates (3). Suicide due to pesticide ingestion constitutes a significant proportion of these fatalities, particularly in South Asia, where nationally representative surveys have documented high suicide mortality rates (4).

Clinically, acute OP poisoning presents with muscarinic signs (hypersalivation, bradycardia, bronchospasm), nicotinic manifestations (fasciculations, muscle weakness), and central nervous system disturbances (confusion, seizures, coma) (5,6). Prognosis depends on the ingested dose, compound type, route of exposure, and timeliness of medical intervention (3,7,8).

In Algeria, epidemiological data on OP poisoning remain limited, despite frequent exposure in rural areas. This study aimed to describe the clinical, biological, therapeutic, and prognostic characteristics of patients admitted for acute OP poisoning to a university emergency department in Algiers, to inform management strategies and preventive measures.

METHODS

This was a retrospective descriptive study conducted in the emergency department of the University Hospital of Algiers over a 12-month period, from January 1st to December 31st, 2023. All patients admitted for acute organophosphate poisoning were included if

the diagnosis was confirmed both clinically (presence of cholinergic syndrome) and biologically (marked decrease in plasma cholinesterase activity). Patients with mixed or undocumented poisonings were excluded.

Data were extracted from medical records and included: Demographics (age, sex); Circumstances of poisoning: intentional vs. accidental; Clinical presentation (muscarinic, nicotinic, and central nervous system signs); Biological investigations (plasma acetylcholinesterase levels (Ellman's method)); Therapeutic management (atropine, pralidoxime, mechanical ventilation, vasopressors, anticonvulsants, gastric lavage); and Outcomes (length of hospital stay, mortality).

Data analysis was descriptive, using Microsoft Excel 2023. Results are reported as means, frequencies, and percentages.

The study was conducted in accordance with the Declaration of Helsinki. Due to the retrospective and anonymous nature of the data, formal informed consent was not required. Institutional approval was obtained.

RESULTS

A total of 26 patients were included. The mean age was 29.1 ± 10 years, with females accounting for 69% (n=18) of cases. Most poisonings (65%) were intentional. All patients presented with a muscarinic syndrome. Central nervous system manifestations occurred in 42% of cases, and nicotinic signs in 19%. The mean plasma

cholinesterase activity was 800 IU/L, indicating significant enzymatic inhibition (Table 1).

Table 1. Demographic and clinical characteristics of patients with acute organophosphate poisoning (n = 26)

Parameters	Value
Mean age (\pm SD); years	29.1 \pm 10
Female sex; n(%)	18 (69)
Male; n(%)	8 (31)
Intentional poisoning; n(%)	17 (65)
Accidental poisoning; n(%)	9 (35)
Muscarinic syndrome; n(%)	26 (100)
Central nervous system signs; n(%)	11 (42)
Nicotinic signs; n(%)	5 (19)
Mean cholinesterase level; IU/L	800

All patients received atropine, while pralidoxime was administered in only five cases (19%) due to limited availability. Nine patients (35%) required mechanical ventilation, six (23%) needed vasopressor support, and two (8%) were treated with anticonvulsants. The overall mortality rate was 23%, and the mean hospital stay was 11 \pm 2.3 days (Table 2).

Table 2. Therapeutic management and outcomes of patients with acute organophosphate poisoning

Therapeutic Intervention/Outcomes	Value
Atropine; n(%)	26 (100)
Pralidoxime; n(%)	05 (19)
Mechanical ventilation; n(%)	09 (35)
Vasopressor support; n(%)	06 (23)
Anticonvulsants (benzodiazepines); n(%)	02 (8)
Mean length of hospital stay; days	11 \pm 2.3
Deaths; n(%)	06 (23)

DISCUSSION

This retrospective study describes the clinical and epidemiological characteristics of acute OP poisoning in a hospital setting in Algeria. The predominance of intentional exposure in

our cohort aligns with previous hospital-based and epidemiological data, confirming that pesticide ingestion is a major method of self-harm in many low- and middle-income countries [1,2,4]. Systematic reviews and national surveys have highlighted the substantial contribution of pesticide self-poisoning to global suicide mortality [2–4].

In our series, women accounted for a considerable proportion of self-poisoning cases. Similar patterns have been reported in hospital-based cohorts from resource-limited settings, although gender distributions vary across regions and study populations [1,4]. This variability likely reflects sociocultural and demographic differences rather than a uniform epidemiological pattern.

Clinically, the predominance of muscarinic manifestations observed in our patients is consistent with the established mechanism of acetylcholinesterase inhibition, leading to acetylcholine accumulation at synaptic junctions [5]. The frequent occurrence of nicotinic and central nervous system signs further illustrates the broad clinical spectrum described in prospective studies [5–7]. Previous research has shown that clinical severity depends not only on the ingested dose and time to treatment but also on the specific OP compound involved, as toxicity varies markedly among agents [5,12].

Measurement of plasma cholinesterase activity remains a useful supportive diagnostic and severity marker. The markedly

reduced levels observed in our cohort reflect significant enzyme inhibition, consistent with previous clinical and toxicological studies [5,8]. Nevertheless, laboratory results should always be interpreted in conjunction with clinical findings, particularly in settings where timely assays may not be available [5]. The systematic administration of atropine in our cohort is in line with established management principles, where early and adequate atropinization remains the cornerstone of treatment [9,13]. The limited use of pralidoxime reflects issues of availability. While oximes are recommended as specific antidotes, their impact on mortality and clinical outcomes remains debated. Randomized trials and systematic reviews have reported heterogeneous results, with uncertainty regarding their benefit, particularly in severe cases or when treatment is delayed [9–11]. These data support cautious interpretation of oxime efficacy while acknowledging their continued indication when accessible.

The high rates of mechanical ventilation and vasopressor support indicate a severe cholinergic crisis with respiratory and cardiovascular compromise. Comparable needs for advanced supportive care have been reported in intensive care cohorts from regions with high OP exposure [6,12]. Respiratory failure remains a key determinant of prognosis in acute OP poisoning [5,6].

The observed mortality rate in our series was 23.1%, highlighting the severity of acute OP poisoning. This rate is consistent with other hospital-based studies from regions with a high incidence of OP exposure, where reported mortality generally ranges between 15% and 25% [6,12,13]. Differences between studies may reflect variations in clinical severity, access to intensive care, and overall management resources.

From a public health perspective, these findings reinforce the importance of preventive strategies targeting access to highly hazardous pesticides. Restricting availability has been proposed as a key intervention to reduce fatal self-poisoning in developing countries [14].

This study has several limitations. The sample size was relatively small, which limits statistical power, and the retrospective design without long-term follow-up prevented the assessment of functional or psychological sequelae after poisoning. In addition, precise identification of the ingested organophosphate compounds was sometimes not possible, restricting analysis according to compound type. Despite these limitations, this series provides essential data for Algeria and serves as a foundation for prospective multicenter studies aimed at refining therapeutic and preventive strategies.

CONCLUSION

Acute organophosphate poisoning remains a frequent and life-threatening medical

emergency. Our findings confirm the predominance of intentional exposure, the characteristic cholinergic clinical pattern, and the substantial need for intensive supportive care. While atropine remains the cornerstone of therapy, limited access to oximes and variability in compound toxicity may influence outcomes. Strengthening early management and implementing preventive strategies, including pesticide regulation and psychosocial interventions, are essential to reduce mortality. Prospective multicenter studies are warranted to clarify optimal therapeutic approaches.

Conflict of interest: The authors declare no conflict of interest.

REFERENCES

1. Peter JV, Jerobin J, Nair A, et al. Clinical profile and outcome of patients with organophosphate poisoning needing intensive care. *Indian J Crit Care Med.* 2014;18(9):576–80.
2. Gunnell D, Eddleston M, Phillips MR, Konradsen F. The global distribution of fatal pesticide self-poisoning: systematic review. *BMC Public Health.* 2007;7:357.
3. Mew EJ, Padmanathan P, Konradsen F, Eddleston M, Chang SS, Phillips MR, Gunnell D. The global burden of fatal self-poisoning with pesticides 2006–15: Systematic review. *J Affect Disord.* 2017;219:93–105.
4. Patel V, Ramasundarahettige C, Vijayakumar L, et al. Suicide mortality in India: a nationally representative survey. *Lancet.* 2012;379(9834):2343–51.
5. Eddleston M, Eyer P, Worek F, et al. Differences between organophosphorus insecticides in human self-poisoning: a prospective cohort study. *Lancet.* 2005;366(9495):1452–9.
6. Karunarathne A, Jayarathne S, Kularatne SA, et al. Outcome of organophosphate poisoning in rural Sri Lanka: A tertiary care hospital-based study. *Indian J Crit Care Med.* 2021;25(Suppl 2):S139–47.
7. Banerjee I, Tripathi SK, Roy AS. Organophosphorus poisoning: A clinicopathological study in rural Bengal. *N Am J Med Sci.* 2012;4(3):147–50.
8. Shadnia S, Azizi E, Hosseini A, et al. Evaluation of oxidative stress and genotoxicity in organophosphate pesticide manufacturing workers. *Hum Exp Toxicol.* 2005;24(6):297–300.
9. Vale JA, Meredith TJ, Buckley NA. Oximes in organophosphate poisoning: a systematic review of clinical trials. *Clin Toxicol (Phila).* 2016;54(8):627–34.
10. Buckley NA, Eddleston M, Li Y, et al. Oximes for acute organophosphate pesticide poisoning. *Cochrane Database Syst Rev.* 2011;(2):CD005085.
11. Roberts DM, Eddleston M, Wilks MF, et al. Clinical outcomes and kinetics of pralidoxime in acute organophosphate poisoning: a multicenter randomized controlled trial. *Lancet.* 2007;369(9564):1035–41.
12. Dawson AH, Eddleston M, Senarathna L, et al. Acute human lethal toxicity of pesticides: a prospective cohort study. *Crit Care Med.* 2010;38(3):832–8.
13. Pawar KS, Bhoite RR, Pillay CP, et al. Management of organophosphorus poisoning with a single bolus of atropine. *Lancet.* 2006;368(9553):2136–41.
14. Konradsen F, van der Hoek W, Cole DC, et al. Reducing acute poisoning in developing countries, options for restricting the availability of pesticides. *Toxicol Lett.* 2003;137(3):167–74.

Neuroimaging Features Of Central Nervous System Diseases In HIV-Infected Patients: A Comprehensive Review

Wiem Feki¹, Fatma Hammami², Makram Koubaa², Emna Daoud¹, Mounir Ben Jemaa², Zaineb Mnif¹

1. Radiology Department, Hedi Chaker University Hospital, University of Sfax, Tunisia

2. Infectious Diseases Department, Hedi Chaker University Hospital, University of Sfax, Tunisia

Abstract

Neurological complications remain a major cause of morbidity and mortality in HIV-infected individuals, especially those with advanced immunosuppression. Opportunistic infections such as cerebral toxoplasmosis, central nervous system tuberculosis, cryptococcal infection, and progressive multifocal leukoencephalopathy present with diverse and sometimes overlapping neuroimaging findings. Magnetic resonance imaging is the modality of choice for early detection and characterization of these conditions. In the emergency setting, neuroimaging plays a pivotal role in rapidly identifying life-threatening central nervous system complications in HIV-infected patients presenting with acute neurological symptoms. Our work aims to provide a comprehensive overview of the most common brain diseases encountered in HIV-infected patients and to highlight the crucial role of neuroimaging in facilitating accurate diagnosis and timely treatment, thereby improving clinical outcomes.

Keywords

HIV infection ; Brain diseases ; Magnetic resonance imaging ; Cerebral toxoplasmosis ; Central nervous system tuberculosis

INTRODUCTION

Central nervous system (CNS) involvement remains one of the most serious and life-threatening complications in patients infected with the human immunodeficiency virus (HIV). Despite advances in antiretroviral therapy (ART), opportunistic infections and HIV-associated neurocognitive disorders continue to affect a significant proportion of

immunocompromised individuals, particularly those with low CD4 cell counts [1,2]. These CNS manifestations are varied, ranging from infections such as toxoplasmosis and cryptococcosis to demyelinating processes like progressive multifocal leukoencephalopathy (PML) and direct viral effects such as HIV-associated encephalitis [3].

Accurate and timely diagnosis is essential, as clinical presentations are often nonspecific and overlapping. Headache, confusion, seizures, and focal neurological deficits may be seen in several of these conditions, making clinical differentiation challenging.

In the emergency department, where time-sensitive decisions are crucial, the identification of these neurological complications can be particularly challenging. Delay in diagnosis may lead to rapid deterioration or irreversible damage. Therefore, neuroimaging, especially magnetic resonance imaging (MRI), plays a pivotal role in guiding diagnosis, differentiating pathologies, and assessing response to therapy [4–6]. Awareness of these imaging patterns is essential for emergency physicians confronted with HIV-infected patients presenting with acute neurological symptoms.

This review aims to provide a comprehensive overview of the most common brain diseases encountered in HIV-infected patients. We focus on five major entities: cerebral toxoplasmosis, CNS tuberculosis, cryptococcal infection, PML, and HIV-associated encephalopathy, highlighting the characteristic imaging findings and key differentiating features.

1/ Cerebral toxoplasmosis (Figures 1,2):

Toxoplasmosis is the most frequent opportunistic brain infection in HIV-infected patients. It is caused by the parasite *Toxoplasma gondii* and typically occurs in

individuals with CD₄ counts below 100 cells/ μ L [7]. Pathologically, it results in necrotizing encephalitis.



Figure 1 : Cerebral toxoplasmosis : Axial T2 (A), coronal (B) and sagittal (C) T1 sections after injection of gadolinium: showing multiple rounded lesions including the left frontal lesion (red arrow) taking contrast in the periphery and surrounded by significant lesion oedema (yellow arrow

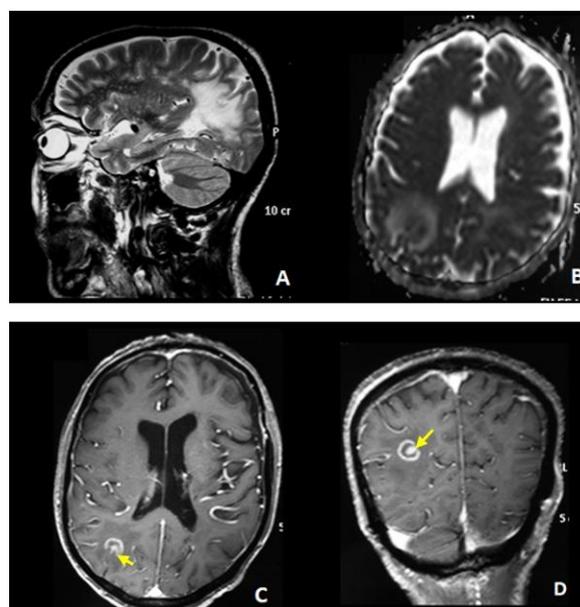


Figure 2 : Cerebral toxoplasmosis : Saggiital T2 section (A), ADC (B), T1 after injection axial (C) and coronal (D) : The “eccentric target sign” (yellow arrow)

MRI typically reveals multiple lesions, 1–4 cm in size, with a predilection for the basal ganglia, thalamus, and corticomedullary junction [7]. On T1-weighted images, the lesions appear hypointense, while T2-weighted images show iso- to hypointense signal, often with surrounding vasogenic edema and mass effect.

The enhancement pattern is usually ring-shaped or nodular. A distinctive but infrequent sign is the eccentric target sign, where a small enhancing nodule lies eccentrically within a ring—seen in about 30% of cases and relatively specific for toxoplasmosis [8].

Magnetic resonance spectroscopy (MRS) may show elevated lipid and lactate peaks, indicating necrosis and anaerobic metabolism [9]. Diagnosis is often clinical and radiological, confirmed by therapeutic response to anti-toxoplasmosis treatment, thereby avoiding invasive biopsy [10].

Differential diagnoses include primary CNS lymphoma and HIV encephalopathy, and may require advanced imaging techniques or brain biopsy in equivocal cases.

SUMMARY

Multifocal peripherally enhancing lesions involving the basal ganglia and corticomedullary junction are the hallmark of cerebral toxoplasmosis. Look for the eccentric target sign. Differentiation from lymphoma may require advanced imaging, a therapeutic trial, or biopsy.

2. CNS Tuberculosis (Figure 3)

The HIV epidemic has significantly increased the incidence of tuberculosis worldwide. HIV infection is the major risk factor for reactivation of latent tuberculosis, increasing the risk approximately threefold compared to HIV-negative individuals and raising the proportion of extrapulmonary tuberculosis cases fivefold [11]. CNS tuberculosis usually

occurs in patients with CD4 counts less than 500 cells/ μ L [12].

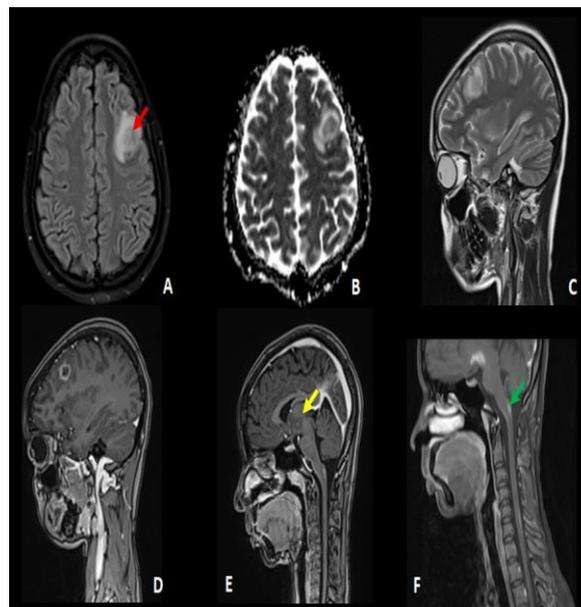


Figure 3 :Tuberculoma with tuberculous meningitis :Axial FLAIR (A), ADC (B) and sagittal T2 (C), T1 after gadolinium injection (D, E, F): left frontal lesion (red arrow) rounded in central T2 hyposignal surrounded by a peripheral T2 hypersignal taking contrast in the periphery in an annular fashion. There is a second lesion of the same type at the base of the cerebral peduncle (yellow arrow) and meningeal contrast opposite the gracile fasciculus (green arrow).

CNS tuberculosis presents in various forms:

- Intracranial intra-axial tuberculous granuloma (tuberculoma): These are ring-enhancing, round lesions 3–15 mm in diameter, often surrounded by extensive vasogenic edema. They are typically hypointense on T1-weighted images and hyperintense on T2-weighted images, sometimes with a hypointense rim. Tuberculomas can occur in both supra- and infratentorial regions but are more common in white matter and subcortical areas. Caseating tuberculomas with liquid centers may

resemble tuberculous abscesses, though abscesses tend to be larger and solitary [13].

-Meningeal (extra-axial) tuberculous granulomas: Appear as small (<6 mm) round foci on the brain surface, sometimes with cortical involvement, showing ring enhancement on contrast-enhanced T1-weighted images [14].

-Tuberculous leptomeningitis: Characterized by predominant basal meningeal enhancement, similar to patterns seen in immunocompetent patients [12].

Summary

Tuberculosis can affect multiple CNS regions with varied imaging features. CNS involvement generally occurs at CD4 counts below 500 cells/ μ L but can present at any level of immunosuppression.

3. Cryptococcal infection (Figures 4,5)

Infection caused by *Cryptococcus neoformans* typically occurs with CD4 counts below 100 cells/ μ L [15]. Pathologically, there are three main forms of cryptococcal infection: meningitis, gelatinous pseudocysts, and cryptococcomas.

MRI findings are generally nonspecific. Cryptococcal meningitis presents as focal (chronic basilar meningitis) or diffuse meningeal enhancement. Mild dilatation of the ventricular system can also be detected [16].

Cryptococcus neoformans pseudocysts occupy and dilate perivascular Virchow-Robin spaces, resulting in rapidly growing,

non-enhancing “cysts” seen symmetrically in the basal ganglia and thalamus.

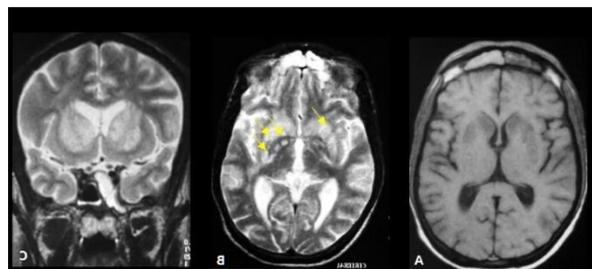


Figure 4: Cryptococcal infection: *Cryptococcus neoformans* pseudocysts and dilate perivascular Virchow-Robin spaces, non enhancing “cysts” seen symmetrically in basal ganglia (yellow arrows). These lesions are hypointense on T1W images (A) and hyperintense on T2W MR images (B,C).

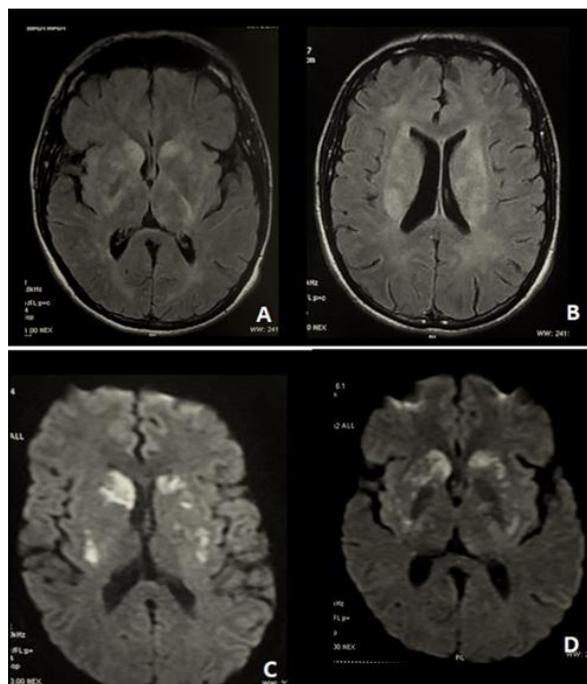


Figure 5: Cryptococcosis with acute ischemic lesions :FLAIR (A,B) and diffusion (C,D) axial sections: multiple bilateral and asymmetric FLAIR hypersignal patchy signal anomalies with diffusion restriction in the basal ganglia and external capsule in relation to ischaemic lesions

These lesions are hypointense on T1-weighted images and hyperintense on T2-weighted MR images [17]. Cryptococcomas are rare mass-like parenchymal lesions with

nodular enhancement and have a predilection for the basal ganglia, thalamus, and cerebellum [18].

SUMMARY

Variable patterns of cerebral involvement occur, with the most characteristic being perivascular gelatinous pseudocyst formation.

4. Progressive multifocal leukoencephalopathy (Figure 6)

PML is a demyelinating disorder caused by latent reactivation of the Papovavirus (JC virus). It typically occurs in patients with CD4 counts between 50 and 100 cells/ μ L [19].

MRI demonstrates multifocal, bilateral but asymmetric areas of T2 hyperintensity predominantly involving the periventricular and subcortical white matter, usually without mass effect or contrast enhancement. Subcortical U-fibers are commonly involved, and occasional cortical involvement is noted.

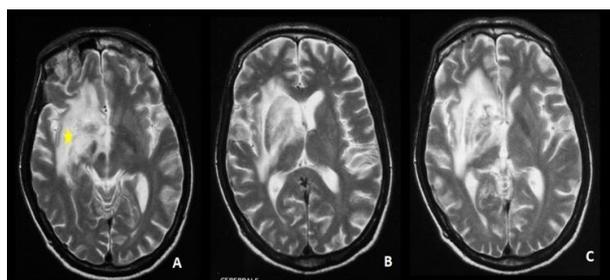


Figure 6: Progressive multifocal leukoencephalopathy: axial T2 sections: T2 hyperintensity area (yellow star) involving predominantly periventricular and subcortical white matter, without mass effect or enhancement

Diffusion restriction may be present at the advancing edge of acute lesions, and cystic changes may develop in late stages. Lesions

have a predilection for the parieto-occipital regions, basal ganglia, and thalami [20,21].

SUMMARY

PML is a multifocal demyelinating disorder occurring in advanced immunosuppression. Its asymmetric distribution, absence of mass effect, and lack of enhancement help distinguish it from HIV encephalopathy, lymphoma, and toxoplasmosis.

5. HIV Encephalitis

HIV encephalitis results from direct neuronal injury caused by the HIV virus. The spectrum of neurocognitive symptoms is broad, with imaging correlates most evident in advanced disease, such as HIV-associated dementia [22].

Imaging findings are nonspecific and include symmetrical cerebral atrophy disproportionate to age, with confluent, bilateral, almost symmetrical and diffuse T2 hyperintensity involving deep and periventricular white matter. There is often a frontal lobe predominance. No mass effect or enhancement is seen in HIV encephalopathy [23,24].

SUMMARY

HIV encephalitis manifests as nonspecific, diffuse deep white matter disease due to direct viral effects. Its prevalence may paradoxically increase with longer survival of HIV-positive patients.

CONCLUSION

Brain infections and neurological complications remain a significant cause of morbidity and mortality in HIV-infected

patients, particularly in those with advanced immunosuppression. Neuroimaging, primarily MRI, plays a critical role in the early detection, characterization, and differentiation of opportunistic infections. Although imaging features can be similar, identifying specific patterns is key to enabling timely diagnosis and guiding appropriate treatment. In the emergency setting, prompt neuroimaging is often essential for the rapid evaluation of HIV-infected patients presenting with acute neurological symptoms, where early intervention can be lifesaving. As survival improves with ART, awareness of these varied presentations and advances in imaging techniques remain essential for optimal management and improved outcomes in this vulnerable population.

Acknowledgments : None

Author contributions : (I) Conception and design: WF, FH, MK, (II) Administrative support: WF, FH, ED; (III) Provision of study materials or patients: WF, FH, MBJ; (IV) Collection and assembly of data: FH, ED, ZM; (V) Data analysis and interpretation: WF, FH, MK, MBJ, ED; ZM (VI) Manuscript writing: WF, FH; (VII) Final approval and revision of manuscript: All authors.

Conflict of interest statement :The authors have no conflict of interest to declare.

REFERENCES

1. Vittinghoff E, Scheer S, O'Malley P, Colfax G, Holmberg SD, Katz MH. CD4+ T-cell count and the risk of opportunistic infections in HIV-infected patients. *J Acquir Immune Defic Syndr*. 2003;33(4):521–6.

2. Saylor D, Dickens AM, Sacktor N, Haughey N, Slusher B, Pletnikov M, et al. HIV-associated neurocognitive disorder—pathogenesis and prospects for treatment. *Nat Rev Neurol*. 2016;12(4):234–48.
3. Patel K, Clifford DB. HIV-associated neurocognitive disorder. *Curr Opin HIV AIDS*. 2020;15(3):146–53.
4. Post MJ, Sheldon JJ, Hensley GT, Diaz PS, Morgello S, Bussiere J, et al. Central nervous system disease in acquired immunodeficiency syndrome: prospective correlation using CT, MR imaging, and pathologic studies. *Radiology*. 1986;158(1):141–8.
5. Zimmerman RD, Heier LA, Deck MDF. Neuroimaging of AIDS. *Radiol Clin North Am*. 1994;32(4):889–908.
6. Grant PM, Hammoud DA. Neuroimaging in HIV infection. *Curr HIV/AIDS Rep*. 2014;11(4):349–57.
7. Porter SB, Sande MA. Toxoplasmosis of the central nervous system in the acquired immunodeficiency syndrome. *N Engl J Med*. 1992;327(23):1643–8.
8. Vidal JE, Hernández AV, de Oliveira ACP, et al. Sensitivity and specificity of the eccentric target sign on MRI for the diagnosis of cerebral toxoplasmosis in AIDS patients. *Clin Radiol*. 2007;62(10):1002–7.
9. Chang L, Ernst T, Tornatore C, et al. Metabolite abnormalities in the basal ganglia of HIV-infected patients. *AJNR Am J Neuroradiol*. 1999;20(2):195–200.
10. Torre D, Casari S, Speranza F, et al. Randomized trial of empirical therapy with pyrimethamine-sulfadiazine vs. trimethoprim-sulfamethoxazole for AIDS patients with presumptive cerebral toxoplasmosis. *Clin Infect Dis*. 2004;39(10):1381–4.
11. Getahun H, Gunneberg C, Granich R, Nunn P. HIV infection-associated tuberculosis: the epidemiology and the response. *Clin Infect Dis*. 2010;50 Suppl 3:S201–7.
12. Rock RB, Olin M, Baker CA, Molitor TW, Peterson PK. Central nervous system tuberculosis: pathogenesis and clinical aspects. *Clin Microbiol Rev*. 2008;21(2):243–61.
13. Bernaerts A, Vanhoenacker FM, Parizel PM, et al. Tuberculosis of the central nervous system: overview of neuroradiological findings. *Eur Radiol*. 2003;13(8):1876–90.
14. Kumar R, Prasad KN, Husain N, et al. MRI of intracranial tuberculosis. *AJR Am J Roentgenol*. 1998;171(5):1497–504.
15. Kiertiburanakul S, Wirojtananugoon S, Mahaisavariya P, Sungkanuparph S. Cryptococcosis in human immunodeficiency virus-negative patients. *Int J Infect Dis*. 2006;10(1):72–8.
16. Brat DJ, Meyers SP, Henson JW, et al. Imaging of cryptococcal CNS infections: correlation with pathologic findings. *AJNR Am J Neuroradiol*. 1999;20(4):655–64.
17. Chang RC-C, Yu J-C, Chang WN, et al. Cryptococcal infection of the central nervous system in AIDS: CT and MR imaging findings. *AJNR Am J Neuroradiol*. 1993;14(4):781–7.

18. Tseng H-F, Hsiao Y-H, Lin W-C, et al. *Cryptococcomas in HIV-negative patients: clinical features and imaging findings. Clin Radiol.* 2014;69(9):e391–7.
19. Tan IL, McArthur JC, Clifford DB, et al. *Progressive multifocal leukoencephalopathy in HIV infection: clinical and radiographic correlates. Ann Neurol.* 2010;68(6):853–61.
20. Koralnik IJ. *Progressive multifocal leukoencephalopathy revisited: Has the disease outgrown its name? Ann Neurol.* 2006;60(2):162–73.
21. Calderón C, Martín M, de Felipe S, et al. *MR imaging features of progressive multifocal leukoencephalopathy: initial and follow-up study. AJNR Am J Neuroradiol.* 2003;24(7):1410–9.
22. Wiley CA, Achim CL, Westmoreland SV. *HIV dementia and the central nervous system. J Neurovirol.* 1998;4(1):37–43.
23. Ances BM, Ellis RJ. *Dementia and neurocognitive disorders due to HIV-1 infection. Semin Neurol.* 2007;27(1):86–92.
24. Navia BA, Cho ES, Petito CK, Price RW. *The AIDS dementia complex: I. Clinical features. Ann Neurol.* 1986;19(6):517–24.

Managing Acute Hemorrhage in Glanzmann Thrombasthenia in the Emergency Department : A Two-Case Report

Rim Karray¹, Fedi Ben Ali¹, Hela Ben Jmeaa², Racem Mnif¹, Hana Kessentini¹, Olfa Chakroun¹, Imed Frikha², Nouredine Rekik¹.

1. *Emergency Department, Habib Bourguiba University Hospital, Sfax, Tunisia.*

2. *Cardiovascular and Thoracic Surgery Department, Habib Bourguiba University Hospital, Sfax, Tunisia.*

Abstract

Background: Glanzmann Thrombasthenia (GT) is a rare inherited platelet function disorder caused by a quantitative or qualitative deficiency of the glycoprotein IIb/IIIa complex, leading to defective platelet aggregation. Although typically associated with mucocutaneous bleeding, life-threatening hemorrhagic complications may occur and pose significant challenges in emergency settings.

Cases presentation: We report two cases of acute hemorrhage in patients with GT managed in the emergency department. The first case involved a 32-year-old man presenting with acute dyspnea due to a massive spontaneous left hemothorax. Despite prompt resuscitation, blood and platelet transfusions, antifibrinolytic therapy, and emergency chest drainage, the patient developed respiratory failure and subsequently died from septic shock. The second case concerned a 19-year-old man admitted after facial trauma complicated by persistent epistaxis refractory to local measures and tranexamic acid. Hemostasis was successfully achieved after platelet transfusion.

Conclusion: These cases highlight the wide clinical spectrum of hemorrhagic emergencies in GT and the limitations of standard coagulation tests, which are often normal. Management relies on rapid assessment, early antifibrinolytic therapy, and platelet transfusion, with recombinant activated factor VII as a valuable option in refractory or allo-immunized patients. Emergency physicians should be aware of this rare condition, as timely multidisciplinary management is crucial to reduce morbidity and mortality.

Keywords: Glanzmann Thrombasthenia, emergency, acute hemorrhage, management.

INTRODUCTION

The management of rare or complex hemorrhagic emergencies represents a real challenge for emergency department teams. Glanzmann's

thrombopathy (GT), a rare congenital platelet disorder caused by a qualitative or quantitative deficiency of the glycoprotein IIb/IIIa complex, is one such condition. Although it is classically

associated with mucosal bleeding (epistaxis, gingival bleeding, menorrhagia, etc.), serious hemorrhagic complications such as spontaneous hemothorax have been reported, even in the absence of trauma. The risk is potentially higher in the presence of stress factors.

We report the cases of two patients who were admitted to the emergency department for bleeding associated with Glanzmann's disease, massive hemothorax, and epistaxis.

Case 1

A 32-year-old man was admitted to the emergency department with dyspnea. Initial examination using the ABCDE approach showed a clear upper airway, a respiratory rate of 22 breaths per minute, and a pulse oxygen saturation of 94% on room air. Pulmonary auscultation revealed decreased vesicular breath sounds over the left hemithorax, with no rales or pleural friction rub. The patient's blood pressure was 120/70 mmHg, and heart rate was 97 beats per minute. He was conscious, cooperative, and afebrile. The medical history revealed that the patient had been treated since childhood for GT and had stopped treatment several years ago. There was no cough or expectoration, nor any recent falls or trauma.

A point-of-care ultrasound showed a large left pleural effusion. An urgent chest X-ray revealed homogeneous opacity of the left hemithorax, associated with contralateral tracheal deviation, suggestive of a large pleural effusion. **(Figure 1)**

An urgent chest scan confirmed the presence of a large left pleural effusion, with blood density, causing total lung collapse and mediastinal deviation compressing the right heart chambers.



Figure 1: A chest X-ray revealing homogeneous opacity of the left hemithorax, associated with contralateral tracheal deviation, suggestive of a large pleural effusion.

Biological tests, including hemostasis, showed anemia, with hemoglobin at 8.2 g/dl, platelets at 229,000/mm³, PT 67%, APTT 28/30, and fibrinogen 2.7g/L.

The collegial decision with the cardiovascular and thoracic surgery team was to insert a chest drain on the left side. The patient was prepared before the interventional procedure. A transfusion of red blood cell concentrates was administered, supplemented by apheresis platelet concentrate to rapidly correct the hemostasis disorder. Treatment with tranexamic acid was also started to limit the extent of blood loss. After this preparatory phase, emergency chest drainage was performed laterally at the fourth intercostal space. It allowed for the immediate evacuation of 400 ml of old-looking blood, after which the drain was clamped and then unclamped in order to limit further blood loss while allowing for careful monitoring of subsequent flow. It brought back a total of 1600 cc/24 hours. The patient's respiratory condition progressively worsened the following day, requiring emergency orotracheal intubation and mechanical ventilation.

He was then transferred to intensive care. The prognosis was unfavorable, and the patient died after one month in septic shock following pneumonia acquired while on mechanical ventilation.

Case 2

A 19-year-old man, known to have GT since childhood, was admitted to the emergency room for craniofacial trauma following an assault. The initial examination following the ABCDE approach was normal. There was epistaxis, blood pressure of 110/70 mmHg, heart rate of 82 bpm, and oxygen saturation of 96% in ambient air. The patient had a periorbital ecchymosis and a bleeding wound on his upper lip, which was sutured. No other signs of physical assault were noted. The patient presented with moderate epistaxis that did not resolve after digital compression, anterior packing, and administration of tranexamic acid.

A brain scan was performed and revealed no abnormalities. The biological assessment, in this case hemostasis, showed no abnormalities (hemoglobin at 12.8 g/dl, platelets at 258,000/mm³, PT 68%, APTT 29/30, and fibrinogen 3 g/L).

The joint decision with the otolaryngology and hematology teams was to administer platelet concentrates to optimize the patient's hemostasis.

The outcome was favorable. The epistaxis subsided after the platelet concentrate transfusion.

He was discharged after 24 hours of monitoring.

DISCUSSION

Glanzmann's thrombasthenia is a rare disease characterized by a quantitative or qualitative

deficiency of the GPIIb/IIIa complex (platelet integrin α IIb β 3), which is essential for platelet aggregation and clot formation. The clinical presentation of Glanzmann's thrombasthenia is dominated by severe mucocutaneous bleeding and the risk of major hemorrhagic complications (1,2). The diagnosis of this disease may be delayed because platelet counts and standard hemostasis tests (PT, APTT, fibrinogen) are generally normal: only aggregometry and immunophenotyping can confirm the diagnosis. It is crucial not to eliminate Glanzmann's thrombasthenia in cases of severe bleeding despite normal standard test results (1).

In an emergency, it is imperative to rapidly evaluate the severity of bleeding and administer appropriate treatment to prevent life-threatening risks (3).

Globally, the management of hemorrhage relies on rapid recognition of its severity, immediate control of the source of bleeding, and hemodynamic stabilization of the patient according to the ABCDE approach. It includes early vascular access, judicious fluid resuscitation with transfusion if necessary, and early administration of tranexamic acid. The correction of coagulation disorders and the rapid referral to specialized care are essential to improve prognosis (4).

In Glanzmann's thrombasthenia, initial control of bleeding relies on local measures such as compression and cryotherapy, combined with systemic administration of antifibrinolytics, particularly tranexamic acid, which is well documented in the literature as helping to control menometrorrhagia and other superficial blood loss. However, these measures are rarely

sufficient in cases of major hemorrhage, where transfusion of HLA-compatible platelet concentrates remains the gold standard (5-8).

Platelet concentrate transfusion is the cornerstone of treatment for moderate to severe bleeding. However, alloimmunization against platelet glycoproteins can lead to transfusion resistance, making management more complex (1,6).

For refractory patients, the use of recombinant factor VIIa (rFVIIa, Novo Seven) often achieves effective hemostasis, with a precise dosing regimen and strict monitoring to prevent thromboembolic complications (8, 9). This factor has proven to be a promising therapeutic option for the treatment of bleeding episodes in patients with Glanzmann's thrombasthenia, particularly in cases of alloimmunization or refractoriness to traditional platelet transfusions (10).

Optimal management requires multidisciplinary coordination, integrating emergency services, hematology, anesthesia, and blood banks, ensuring rapid access to appropriate treatments and personalized follow-up. As part of a comprehensive approach, it is essential to plan for long-term coordination with reference centers to anticipate bleeding episodes, particularly during trauma, surgery, or obstetric procedures (9,11).

These recommendations are supported by international consensus and data from prospective registries, which emphasize the importance of individualized protocols and rigorous follow-up to improve patient prognosis (1,9,11).

CONCLUSION

The management of acute bleeding in a patient with Glanzmann's thrombasthenia must be rapid and coordinated to avoid severe blood loss or

hemorrhagic shock. Immediate measures include local treatment, antifibrinolytics, and appropriate platelet transfusions. Alloimmunization can cause these measures to be ineffective; in this situation, recombinant factor VIIa proves to be a crucial treatment. Multidisciplinary collaboration between emergency services, hematology, blood banks, surgeons, and the implementation of personalized protocols are essential to optimize hemorrhage control and limit mortality.

REFERENCES

1. Nurden AT, Pillois X, Nurden P. Thrombasthenia of Glanzmann and related disorders of platelet function. *Haematologica*. 2015 Jul;100(7):434-8. doi:10.3324/haematol.2014.117784.
2. Pillois X, et al. Emergency management of patients with Glanzmann thrombasthenia: consensus recommendations from the French reference center for inherited platelet disorders. *Haematologica*. 2023 Jun 28. PubMed PMID: 37386449.
3. Solh T, Botsford A, Solh M. Glanzmann's thrombasthenia: pathogenesis, diagnosis, and current and emerging treatment options. *J Blood Med*. 2015;6:219-27.
4. Jacques Duranteau, et al. Recommandations sur la réanimation du choc hémorragique. *Anesth Reanim*. 2015; 1: 62-74
5. White GH, St John AE. Management of Epistaxis in the Emergency Department. *Clinician*. 2022 Jul 14;167:22-9.
6. Hasanpour M., et al. (2024). Patient with a history of Glanzmann thrombasthenia presented with chronic subdural hematoma: a case report. *Oxford Medical Case Reports*, 2024
7. Kim J, et al. Emergency Surgery for Spontaneous Hemopneumothorax. *JCPSP*. 2014 Jun;24(6):428-31.
8. Almesedin G.S., Alshmaily H.O., Alshammari K.A., Albalawi R.S. (2023). Two case reports of Glanzmann thrombasthenia with intracranial hemorrhage and a review of the literature. *Surgical Neurology International*, 14, 448.
9. Haute Autorité de Santé (HAS), CRPP. (2020). *Protocole National de Diagnostic et de Soins (PNDS) Thrombasthénie de Glanzmann et pathologies plaquettaires apparentées*.
10. Almatar E., Alsharidah S., Hashem O.A. Outcomes of recombinant activated factor VIIa (NovoSeven) therapy in Glanzmann thrombasthenia: two case reports. *Blood Coagul Fibrinolysis*. 2025 Sep 1;36(6):293-295.
11. *Thrombasthénie de Glanzmann : quelle prise en charge de nos jours ?* *Revue Francophone d'Hémostase et de Thrombose*, 26(1), 34-43.

Spinal cord injury caused by stab wounds after a penetrating trauma: About 2 cases

Hana Kessentini¹, Rim Karray¹, Fedi Ben Ali¹, Haifa Snoussi², Marwa Bellakhal¹,
Olfa Chakroun¹, Nouredine Rekik¹

1. Emergency Department Habib Bourguiba University Hospital Sfax, Tunisia

2. Emergency Department, Regional Hospital Mahres, Sfax, Tunisia

Abstract

Background: Penetrating spinal cord injury caused by stab wounds is rare but represents a distinct clinical entity within traumatic spinal cord injuries. Non-missile penetrating injuries account for a small proportion of spinal trauma, most often occurring in the context of interpersonal violence. Their management remains challenging due to limited clinician experience and the absence of clear treatment guidelines. In addition to immediate neural damage, these injuries may result in delayed complications such as infection, cerebrospinal fluid leakage, and chronic neuropathic pain.

Case presentation: We report two cases of cervical spinal cord injury caused by stab wounds without associated vertebral fracture or dislocation.

The first patient, a 50-year-old woman, presented with complete tetraplegia following a cervical stab wound. Imaging showed a transverse spinal cord contusion with hemorrhagic edema at C4. Despite conservative management including immobilization, prophylaxis, and corticosteroid therapy, neurological status remained unchanged at three months.

The second patient, her daughter, sustained a lateral cervical stab wound and presented with right hemiparesis. MRI demonstrated an incomplete spinal cord transection at C5–C6. She was treated conservatively with corticosteroids and rehabilitation, resulting in slight neurological improvement at three-month follow-up.

Conclusion: Spinal cord injury caused by stab wounds is uncommon but potentially devastating. MRI plays a central role in diagnosis and prognostic evaluation. In the absence of compressive lesions or foreign bodies, conservative management with close neurological monitoring remains an appropriate strategy.

Key words : Penetrating trauma - spinal cord injury – MRI – Conservative treatment

INTRODUCTION

Spinal Cord Injury caused by stab Wounds (SCIW) is rare. It represent an uncommon cause of traumatic spinal cord injury. Nevertheless, they constitute a distinct clinical entity and rank as the third leading cause of open spinal cord trauma, after gunshot wounds and injuries related to road traffic accidents (1).

These rare injuries present a significant clinical challenge due to the limited experience of clinicians and the absence of specific management guidelines. In addition to direct tissue damage, spinal cord injuries caused by stab wounds can lead to delayed complications, including infection, cerebrospinal fluid leakage, and chronic pain(2).

In this context, we report the cases of two patients admitted for spinal cord trauma secondary to vertebro-spinal penetrating injuries that were not associated with vertebral fractures or dislocation.

Case 1

A 50-year-old woman with no significant past medical history was admitted to the emergency department following a stab wound assault inflicted by her son.

The initial assessment following the ABCDE approach revealed a patent airway. Respiratory rate was 16 breaths per minute, with symmetrical chest expansion, normal lung auscultation, and an oxygen saturation of

98% on room air. Hemodynamically, the patient was stable, with a blood pressure of 120/70 mmHg and a heart rate of 80 bpm, and no peripheral signs of shock.

Neurologically, the patient was conscious, cooperative, and had a Glasgow Coma Scale score of 15/15. Examination revealed complete tetraplegia associated with absent anal tone on digital rectal examination. Deep tendon reflexes were absent in both the upper and lower limbs (patellar and Achilles reflexes) with a sensory level at C4. The remainder of the physical examination identified two linear wounds with minimal oozing: one measuring 3 cm located on the posterolateral aspect of the neck, and another measuring 2 cm on the scalp. No other impact sites were found.

Initial management consisted of strict cervical spine immobilization with a rigid cervical collar, meticulous wound closure, and appropriate prophylactic measures, including antibiotics, tetanus immunization, and passive tetanus antitoxin administration. A whole-body CT scan performed 3 hours after admission did not reveal any additional traumatic lesions, except for soft-tissue infiltration along the wound trajectory.

Repeated neurological examinations performed over time by different clinicians confirmed the persistence of a complete spinal cord syndrome, unchanged from the initial findings. A cervical MRI demonstrated

a linear transverse spinal cord contusion with hemorrhagic edema at the level of the C4 vertebral body, without associated vertebral fracture or dislocation. (Figure1).

At three months of follow-up, the patient's clinical condition remained unchanged, with continued tetraplegia.



Figure 1: (a) Sagittal section of spinal cord MRI (b) Transversal section of spinal cord MRI

Orange arrow: linear transverse spinal cord contusion with hemorrhagic edema at the level of the C4 vertebral body

Case 2

The daughter of the same patient was also a victim in the same assault, sustaining multiple scalp lacerations and a small punctiform wound to the left lateral cervical region.

On admission, the ABCDE assessment confirmed a patent airway. She was breathing at 18 breaths per minute with symmetrical chest expansion, normal lung sounds, and an SpO₂ of 97% on room air. Hemodynamics were stable. Neurological assessment yielded a GCS of 15/15, with weakness of the right hemibody. Anal sphincter tone was normal on rectal exam. Wound management and suturing were completed. CT scans of the brain and cervical spine were unremarkable

Subsequent cervical spine MRI demonstrated an incomplete transection of the spinal cord at the C5–C6 level, predominantly on the left side. The only plausible explanation for this lesion was direct penetration of the spinal cord by the knife without associated vascular or osseous injury.

A multidisciplinary team decision, in collaboration with neurosurgeons, opted for conservative treatment. High-dose corticosteroid therapy with methylprednisolone was indicated following the same protocol as the first patient. The patient was then transferred to the neurosurgery department.

At three months, follow-up revealed a slight improvement in the right hemiparesis with physiotherapy.

DISCUSSION

Spinal cord injury caused by stab wounds (SCISW) represents one of the rarest causes of traumatic spinal cord injury. (3) (4). It results from a partial or complete transection of the cord(5).

Stab wounds are generally inflicted in the setting of assault and produce a sharp, pointed pathway of damage(6). Injury can be both immediate from the direct trauma of the penetrating object and delayed, as can occur with infection, spinal fluid leak, and chronic pain.

In the emergency department, the initial evaluation of patients is performed according to a systematic, well-codified approach developed by Advanced Trauma Life Support (ATLS) (7).

In the absence of neurological deficits, CT imaging is used to evaluate bony structures, delineate the trajectory of the stab wound, and detect the presence of pneumocephalus (8). Spinal MRI remains the only imaging modality capable of accurately identifying spinal cord lesions and evaluating disc-ligamentous integrity(9). This exam played a crucial role in ensuring diagnostic accuracy in our two patients.

Considerable controversy persists regarding the neurosurgical management of patients with penetrating spinal injuries(9). There is no clear consensus on the indications for urgent surgical intervention within the first

few hours. In general, surgery is recommended only in cases of neural compression caused by a bone fragment, retained foreign body (such as a blade fragment), or hematoma, as well as in the presence of persistent cerebrospinal fluid leakage. It consists of a laminectomy, followed by the removal of devitalized tissue and compressive elements, and concludes with an airtight closure of the dura mater (10) (11).

The role of high-dose corticosteroid therapy is highly debatable, although the NASCIS study shows that the use of methylprednisolone in humans is beneficial in combating the various mechanisms leading to spinal cord ischemia. However, the impact of management on long-term neurological outcomes remains uncertain (9) (12).

Both patients were managed conservatively with high-dose intravenous corticosteroid therapy. At three months of follow-up, the clinical course remained largely unchanged, with persistent paraplegia in the first patient, while the second patient showed slight improvement, marked by partial regression of right-sided hemiparesis.

CONCLUSION

Direct stabbing injuries rarely lead to complete transection of the spinal cord, as in our first case. Computed tomography (CT) scans, or plain radiographs, are necessary to exclude retained foreign bodies. MRI, as a further imaging tool,

can confirm the Spinal cord injury and may be useful as a predictor of outcomes. Regarding optimal management, conservative treatment should be preferred over surgical intervention in the absence of a foreign body at the injury site.

REFERENCES

1. Naja A, Tahir Y, Laidi A, Jamal O, Edderaz L, Saïdy J. Les plaies vertébro-médullaires par arme blanche : à propos de 13 cas. *Neurochirurgie [Internet]*. 1 avr 2019 [cité 3 févr 2026];65(2):129. Disponible sur: <https://www.sciencedirect.com/science/article/pii/S002837701930102X>
2. Szymoniuik M, Kochański M, Dryla A, Kamieniak P. Stabbing injury of the spinal cord: A case report and systematic literature review. *Clinical Neurology and Neurosurgery [Internet]*. 1 déc 2024 [cité 13 févr 2026];247:108629. Disponible sur: <https://www.sciencedirect.com/science/article/pii/S030384672400516X>
3. Szymoniuik M, Kochański M, Dryla A, Kamieniak P. Stabbing injury of the spinal cord: A case report and systematic literature review. *Clin Neurol Neurosurg*. déc 2024;247:108629.
4. Kamaoui I, Maaroufi M, Benzagmout M, Sqalli Houssaini N, Boujraf S, Tizniti S. MRI findings in spinal cord penetrating injury: three case reports. *J Neuroradiol*. 2007 Oct;34(4):276-9. Disponible sur: <https://www.sciencedirect.com/science/article/abs/pii/S0150986107002520>
5. McCaughey EJ, Purcell M, Barnett SC, Allan DB. Spinal Cord Injury Caused by Stab Wounds: Incidence, Natural History, and Relevance for Future Research. *J Neurotrauma*. 1 août 2016;33(15):1416-21.
6. de Cayenne S. Plaies par arme blanche. 2010;
7. Kool DR, Blickman JG. Advanced Trauma Life Support®. ABCDE from a radiological point of view. *Emerg Radiol [Internet]*. juill 2007 [cité 18 févr 2026];14(3):135-41. Disponible sur: <https://pmc.ncbi.nlm.nih.gov/articles/PMC1914302/>
8. Sqalli Houssaini N, Maaroufi M, Kamaoui I, Hamdi D, Lamhadri M, Tizniti S. NR63 Apport de l'imagerie dans les plaies vertebro-médullaires. A propos de 4 cas. *Journal de Radiologie [Internet]*. 1 oct 2005 [cité 13 févr 2026];86(10):1518. Disponible sur: <https://www.sciencedirect.com/science/article/pii/S021036305761187>
9. Assoumane II, Dossou MW, Amadou Moussa AW, Lawson LD, Agada KN, Hamma OI, et al. Dorsal vertebro-medullary stab wound: Two cases from Niamey National Hospital and review of literature. *Interdisciplinary Neurosurgery [Internet]*. 1 déc 2023 [cité 10 févr 2026];34:101828. Disponible sur: <https://www.sciencedirect.com/science/article/pii/S2214751923001111>
10. Smith C, White JB. Penetrating knife injuries to the spine: Management considerations and literature review. *Interdisciplinary Neurosurgery [Internet]*. 1

- mars 2014 [cité 10 févr 2026];1(1):3-4. Disponible sur: <https://www.sciencedirect.com/science/article/pii/S2214751914000024>
11. Dran G, Fontaine D, Litrico S, Grellier P, Paquis P. Plaie médullaire cervicale par arme blanche: Présentation de deux cas. *Neurochirurgie [Internet]*. 1 nov 2005 [cité 10 févr 2026];51(5):476-80. Disponible sur: <https://www.sciencedirect.com/science/article/pii/S0028377005835064>
 12. Neurosurgical management of penetrating spinal injury. - Abstract - Europe PMC [Internet]. [cité 12 févr 2026]. Disponible sur: <https://europepmc.org/article/med/9122834>