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Research Article

Early Diagnostic Value of KIM-1, NGAL, and NLR in Acute Kidney Injury Caused by Diquat Poisoning

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Background. The kidney is the main excretory organ after diquat absorption. Acute kidney injury (AKI) is a common complication in diquat poisoning patients. *Objectives*. To identify the value of combined detection of neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), and neutrophil-lymphocyte ratio (NLR) in the early diagnosis of diquat-induced AKI. *Materials and Methods*. The retrospective cohort study included 42 diquat poisoning patients. *Results*. Forty-two patients with diquat poisoning were included, of which 20 had fulminant poisoning (47.6%). At admission (0 h), levels of KIM-1, NGAL, NLR, and acute physiology and chronic health evaluation (APACHE) II scores in the fulminant poisoning group were higher than that of the moderate to severe poisoning group ($P < 0.05$), and they were all higher than in the control group ($P < 0.05$), while blood urea nitrogen (BUN) and uric acid (UA) levels did not significantly differ across the three groups ($P > 0.05$). At 12 h and 24 h, the levels of KIM-1, NGAL, NLR, UA, BUN, and APACHE II scores of patients in the fulminant poisoning group were higher than those in the moderate to severe poisoning group ($P < 0.05$), 12 h were higher than 0 h, and 24 h were higher than 12 h ($P < 0.05$). Among 42 patients with diquat poisoning, 28 had AKI (66.7%). At 0 h, the AKI group had higher levels of KIM-1, NGAL, NLR, and APACHE II scores than in the non-AKI (NAKI) group ($P < 0.05$), while there was no significant difference in BUN and UA levels between the two groups ($P > 0.05$). At 12 h and 24 h, the levels of KIM-1, NGAL, NLR, UA, BUN, and APACHE II scores in the AKI group were higher than those in the NAKI group ($P < 0.05$), 12 h were higher than 0 h, and 24 h were higher than 12 h ($P < 0.05$). KIM-1, NGAL, and NLR are independent risk markers for AKI in diquat poisoning patients. At admission (0 h), the combined application of KIM-1, NGAL, and NLR's sensitivity, specificity, and area under the curve (AUC) for predicting AKI in diquat poisoning patients was 0.893, 0.859, and 0.903, respectively. *Conclusions*. KIM-1, NGAL, and NLR can be employed as early diagnostic indicators for the clinical prediction of AKI in diquat poisoning patients. Our findings may help clinicians reduce the occurrence of AKI.

1. Introduction

Diquat is a bipyridine compound with a slow absorption rate but a rapid and widespread dispersion. Within 48 h of oral poisoning, 45% of the intake is excreted through the kidneys and intestines. The primary excretory organ is the kidney. When less than 1 g of diquation was consumed, the prognosis was better; more patients died within 48 h when the intake was greater than 12 g; and multiorgan dysfunction, particularly acute renal failure, is the most common manifestation when the intake was between the two [1]. As a recent study reported, acute kidney injury (AKI) with obvious tubular necrosis will develop in 81% of diquat poisoning patients [2]. AKI is clinically diagnosed by serum creatinine (Scr), but Scr elevation frequently lags behind AKI diagnosis [3]. For the past few years, neutrophil-lymphocyte ratio (NLR), neutrophil gelatinase-associated lipocalin (NGAL), and kidney injury molecule-1 (KIM-1) have all been identified as biomarkers for kidney injury [4, 5]. The purpose of this study was to perform dynamic monitoring of KIM-1, NGAL, and NLR as well as to investigate the value of combining KIM-1, NGAL, and NLR detection in the early prediction of AKI caused by diquat poisoning.

2. Materials and Methods

2.1. Subjects. Forty-two diquat poisoning patients who visited Harrison International Peace Hospital's emergency room between August 2019 and September 2021 served as the study's subjects.

The diagnostic criteria of diquat poisoning refer to the expert consensus on diagnosis and treatment of acute diquat poisoning formulated by the Expert Consensus Group on Diagnosis and Treatment of Acute diquat Poisoning [1]; AKI was diagnosed using the diagnostic criteria established by Kidney Disease: Improving Global Outcomes (KDIGO) in 2012 [6].

Patients, who met the inclusion criteria had a clear oral history of taking diquat, were admitted within 2 h of taking diquat orally and survived for more than 24 h after admission. Exclusion criteria include previous heart, lungs, and renal insufficiency, pregnancy, tumors, trauma, immunological illnesses, metabolic problems, and other poisoned patients.

This study protocol was reviewed and approved by the Ethics Committee of the Harrison International Peace Hospital affiliated with Hebei Medical University, and all examinations and treatments were carried out with the patients' or their families' informed consent.

2.2. Treatment and Groups. All patients admitted to the hospital received gastric lavage, adsorption, purgation, and whole bowel irrigation; contaminated clothing was removed, and the residual pesticide in the contact area was thoroughly washed with water; rehydration, diuresis, hemoperfusion, and hemodialysis; drug-assisted antioxidation and scavenging of oxygen-free radicals, scavenging of inflammatory mediators, and symptomatic supportive treatment. According to the Expert Consensus on Diagnosis and Treatment of acute diquat poisoning, the enrolled patients were divided into two groups as follows: those with moderate to severe poisoning (22 cases, ingestion of 1~12 g diquation, namely, 20% commodity of 9.35–112.20 mL) and those with fulminant poisoning (20 cases, ingestion >12 g, namely, 20% commodity >112.20 mL). In the moderate to severe poisoning group, multiple organ dysfunction syndrome (MODS) with acute renal failure as the main manifestation and two-thirds of patients can recover. In the fulminant poisoning group, the disease progressed rapidly to most organ failures, and the majority of the patients died within 24~48 h. Based on the KDIGO-AKI criteria, the patients were divided into two groups, namely, the AKI group (28 cases) and the nonacute kidney injury (NAKI) group (14 cases). During the same period, 25 healthy subjects served as the control group.

2.3. Indicators. At the time of admission (0 h), as well as 12 and 24 h after treatment, 10 mL of elbow venous blood was drawn for anticoagulation treatment, the blood samples were centrifuged, and the supernatant was taken for the detection of various indexes. NGAL concentrations were measured by immunofluorescence assay (reference range: blood NGAL <150 ng/mL). KIM-1 was tested using an enzyme-linked immunosorbent assay (Wuhan Huamei Biological Engineering Co., Ltd.), which was carried out exactly as directed. The coefficient of variation CV (%) of both NGAL and KIM-1 should be ≤12% within the lot, and the coefficient of variation CV(%) between the lot should be ≤15%. Neutrophil count and lymphocyte count were measured by blood routine (Sysmex-XN2800 automatic blood cell analyzer, Japan), and the NLR value was calculated (NLR = neutrophil/lymphocyte). The levels of uric acid (UA) and blood urea nitrogen (BUN) were detected by a 7,600 automatic biochemical analyzer (Hitachi, Japan). At the same time, the acute physiology and chronic health status (APACHE) II scores were observed.

2.4. Statistical Analysis. For the statistical analysis, SPSS23.0 software was used, and the measurement data were represented as the mean ± standard deviation ($\overline{x} \pm s$). The differences between the data of the two groups were evaluated by T-test, and the comparison of sample rates was performed by Chi-square test (χ^2 test). The kappa test was used to evaluate the consistency of general clinical data. The logistic regression analysis was used to identify the independent risk markers for AKI. The predictive value of KIM-1, NGAL, and NLR for AKI in patients with diquat poisoning was assessed by receiver operating characteristic (ROC) curves. $P = 0.05$ served as the test level and $P < 0.05$ was statistically significant.

3. Results

3.1. Comparison of Observation Index Levels in Patients with Acute Diquat Poisoning. Table 1 displayed the clinical data for the three groups and demonstrated that there were no significant differences in the three groups' age, gender, body mass index, systolic blood pressure, diastolic blood pressure, serum creatinine, or time from poisoning to first gastric lavage ($P > 0.05$). As seen in Table 2, at 0 h, the levels of serum KIM-1, NGAL, NLR, and APACHE II scores in the fulminant group were higher than those in the moderate to severe group ($P < 0.05$), and all levels in the moderate to severe and fulminant groups were higher than those in the control group ($P < 0.05$), while there was no statistically significant difference among the three groups in terms of BUN and UA levels ($P > 0.05$). The levels of serum KIM-1, NGAL, NLR, UA, BUN, and APACHE II scores were higher in the fulminant group at 12 h and 24 h than they were in the moderate to severe group ($P < 0.05$), and their levels in the moderate to severe group and the fulminant group were higher at 12 h than 0 h and higher at 24 h than 12 h ($P < 0.05$).

3.2. Comparison of the Observation Index Values in the AKI Group and NAKI Group. According to Table 3, there was no discernible difference in BUN and UA levels between the AKI group and the NAKI group ($P > 0.05$), whereas the levels of serum KIM-1, NGAL, NLR, and APACHE II scores in the AKI group were greater than those in the NAKI group

Table 1: Comparison of clinical data among three groups ($\overline{x} \pm s$).

	Control group ($n = 25$)	Moderate to severe group ($n = 22$)	Fulminant group ($n = 20$)	F (χ^2) value	P value
Age (years, mean ± SD)	40.18 ± 9.34	39.96 ± 9.52	40.55 ± 9.52	2.356	0.154
Gender (M/F)	12/13	10/12	8/12	0.294[a]	0.863
Body mass index (kg/m^2, mean ± SD)	23.15 ± 4.23	23.45 ± 3.62	23.27 ± 3.98	0.884	0.562
Systolic pressure (mmHg, mean ± SD)	121.75 ± 10.56	128.38 ± 10.12	132.02 ± 11.73	0.738	0.427
Diastolic pressure (mmHg, Mean ± SD)	74.38 ± 9.37	73.97 ± 9.68	74.04 ± 8.13	0.941	0.315
Serum creatinine (μmol/L, mean ± SD)	65.92 ± 10.41	66.3 ± 38.36	68.13 ± 43.55	0.578	0.448
Time from poisoning to first gastric lavage (h, mean ± SD)	—	1.99 ± 0.63	1.92 ± 0.58	2.023	0.356
Length of hospital (d)	—	13.6 ± 4.29	4.22 ± 2.15	9.172	0.000
Renal replacement therapy (n, %)	—	3 (13.64)	9 (45.00)	5.505	0.025

a: χ^2 value.

Table 2: Comparison of observation index levels at 0 h, 12 h, and 24 h after admission in patients with acute diquat poisoning ($\overline{x} \pm s$).

	Control group ($n = 25$)	Moderate to severe group ($n = 22$)	Fulminant group ($n = 20$)	F/t	P value
KIM-1/(ng·mL^{-1}) at 0 h	1.98 ± 0.36	2.36 ± 0.41	2.91 ± 0.49[a]	30.778	0.000
KIM-1/(ng·mL^{-1}) at 12 h	—	2.83 ± 0.57[b]	3.34 ± 0.68[ab]	2.583	0.014
KIM-1/(ng·mL^{-1}) at 24 h	—	3.61 ± 0.60[bc]	4.40 ± 0.79[abc]	3.089	0.004
NGAL/(ng·mL^{-1}) at 0 h	82.39 ± 13.61	170.15 ± 30.45	211.32 ± 44.55[a]	29.604	0.000
NGAL/(ng·mL^{-1}) at 12 h	—	264.84 ± 43.09[b]	349.63 ± 61.02[ab]	2.395	0.021
NGAL/(ng·mL^{-1}) at 24 h	—	349.41 ± 58.62[bc]	487.43 ± 96.43[abc]	4.360	0.000
NLR at 0 h	1.56 ± 0.32	2.35 ± 0.67	6.54 ± 1.05[a]	272.607	0.000
NLR at 12 h	—	5.61 ± 0.98[b]	9.21 ± 2.14[ab]	8.480	0.000
NLR at 24 h	—	9.05 ± 1.52[bc]	11.16 ± 3.41[abc]	2.641	0.012
UA/(g/g cr) at 0 h	5.12 ± 0.97	5.14 ± 0.99	5.13 ± 0.95	1.199	0.308
UA/(g/g cr) at 12 h	—	6.02 ± 1.55[b]	7.51 ± 1.87[ab]	6.019	0.000
UA/(g/g cr) at 24 h	—	7.23 ± 1.52[bc]	8.67 ± 1.91[abc]	4.984	0.000
BUN/(mg·dl^{-1}) at 0 h	19.87 ± 2.23	20.04 ± 2.07	19.73 ± 1.86	0.120	0.887
BUN/(mg·dl^{-1}) at 12 h	—	29.99 ± 4.77[b]	38.71 ± 7.95[ab]	4.358	0.000
BUN/(mg·dl^{-1}) at 24 h	—	41.88 ± 8.03[bc]	49.89 ± 12.77[abc]	2.114	0.041
APACHE II score at 0 h	5.57 ± 1.21	10.29 ± 2.10	14.35 ± 2.71[a]	77.763	0.000
APACHE II score at 12 h	—	14.74 ± 3.98[b]	19.62 ± 5.04[ab]	4.798	0.000
APACHE II score at 24 h	—	18.71 ± 4.22[bc]	23.86 ± 7.29[abc]	3.598	0.001

KIM-1: kidney injury molecule-1; NGAL: neutrophil gelatinase-associated lipocalin; NLR: neutrophil-lymphocyte ratio; UA: uric acid; BUN: blood urea nitrogen; APACHE II score: acute physiology and chronic health status II score. [a]$P < 0.05$ vs moderate to severe group; [b]$P < 0.05$ vs 0 h; [c]$P < 0.05$ vs 12 h.

($P < 0.05$). At 12 h and 24 h, the levels of serum KIM-1, NGAL, NLR, UA, BUN, and APACHE II scores were higher in the AKI group than in the NAKI group ($P < 0.05$), and their levels in both groups were greater at 12 h than 0 h and higher at 24 h than 12 h ($P < 0.05$).

3.3. Clinical Predictors of AKI Injury in Diquat Poisoning Patients. The variables with $P < 0.2$ in the one-way ANOVA were included in the binary logistic regression analysis (Table 4). KIM-1, NGAL, and NLR were found to be three independent risk markers for AKI in patients with diquat poisoning, with OR values of 8.126, 1.033, and 2.637 and P values of <0.01, <0.01, and 0.012, respectively.

Analysis of KIM-1, NGAL, and APACHE II scores in the hospitalized serum of patients with acute diquat poisoning for the ability to predict AKI: Figure 1 depicts the ROC curve of KIM-1, NGAL, NLR, and APACHE II score levels at admission on AKI in patients with acute diquat poisoning. The curve analysis shows that the AUC (area under the curve) of KIM-1, NGAL, and NLR was greater than that of the APACHE II score, and their predictive abilities for AKI in diquat patients were also higher than that of the APACHE II score. Besides, the KIM-1, NGAL, and NLR cutoff values were 2.23 ng/ml, 188.9 ng/mL, and 3.96, respectively. In addition, the sensitivity, specificity, and AUC of the combined application of KIM-1, NGAL, and NLR to predict AKI in diquat patients were 0.893, 0.859, and 0.903, respectively (Table 5).

4. Discussion

This study investigated the relationship between serum KIM-1, NGAL, and NLR levels at admission and AKI in diquat poisoning patients. KIM-1, NGAL, and NLR levels in the blood were found to be higher in the fulminant group and the AKI group than in the moderate to severe group and the NAKI group (both $P < 0.05$). Indicators with P value less

Table 3: Comparison of observation index levels in two groups at 0 h, 12 h, and 24 h after admission ($\overline{x} \pm s$).

	NAKI group ($n = 14$)	AKI group ($n = 28$)	t	P value
KIM-1/(ng·mL^{-1}) at 0 h	2.07 ± 0.50	2.73 ± 0.75[a]	8.840	0.000
KIM-1/(ng·mL^{-1}) at 12 h	2.11 ± 0.49	3.19 ± 0.77[ab]	7.283	0.000
KIM-1/(ng·mL^{-1}) at 24 h	1.96 ± 0.63	4.38 ± 1.35[abc]	12.488	0.000
NGAL/(ng·mL^{-1}) at 0 h	81.89 ± 17.98	202.19 ± 37.74[a]	13.527	0.000
NGAL/(ng·mL^{-1}) at 12 h	81.64 ± 19.08	311.68 ± 57.13[ab]	14.536	0.000
NGAL/(ng·mL^{-1}) at 24 h	82.67 ± 18.21	425.73 ± 71.66[abc]	23.598	0.000
NLR at 0 h	2.12 ± 0.76	4.63 ± 1.13[a]	5.756	0.000
NLR at 12 h	4.45 ± 1.05[b]	8.27 ± 2.13[ab]	8.201	0.000
NLR at 24 h	6.60 ± 1.73[bc]	11.32 ± 3.21[abc]	6.733	0.000
UA/(g/g cr) at 0 h	5.12 ± 0.92	5.14 ± 0.86	0.018	0.986
UA/(g/g cr) at 12 h	5.13 ± 1.03	6.42 ± 1.56[ab]	5.927	0.000
UA/(g/g cr) at 24 h	5.11 ± 0.97	7.79 ± 1.88[abc]	7.611	0.000
BUN/(mg·dl^{-1}) at 0 h	21.32 ± 4.13	21.17 ± 3.82	0.018	0.986
BUN/(mg·dl^{-1}) at 12 h	21.22 ± 3.88	35.29 ± 6.58[ab]	7.359	0.000
BUN/(mg·dl^{-1}) at 24 h	21.14 ± 5.49	43.13 ± 10.55[abc]	7.289	0.000
APACHE II score at 0 h	9.19 ± 2.60	13.05 ± 3.70[a]	3.550	0.000
APACHE II score at 12 h	13.64 ± 4.28[b]	17.83 ± 5.65[ab]	4.134	0.000
APACHE II score at 24 h	16.86 ± 5.95[bc]	21.53 ± 6.40[abc]	3.070	0.004

KIM-1: kidney injury molecule-1; NGAL: neutrophil gelatinase-associated lipocalin; NLR: neutrophil-lymphocyte ratio; UA: uric acid; BUN: blood urea nitrogen; APACHE II score: acute physiology and chronic health status II score; AKI: acute kidney injury; NAKI: nonacute kidney injury. [a]$P < 0.05$ vs NAKI group; [b]$P < 0.05$ vs 0 h; [c]$P < 0.05$ vs 12 h.

Table 4: Binary logistic regression analysis of independent risk markers for AKI in patients with acute diquat poisoning.

Variable	β	S.E	Wald	P	OR	95% CI
KIM-1	2.095	0.739	8.029	<0.01	8.126	1.908~4.613
NGAL	0.033	0.010	9.793	<0.01	1.033	1.012~1.055
NLR	0.969	0.384	6.363	0.012	2.637	1.214~5.600
APACHE II score	0.020	0.096	5.287	0.016	1.246	1.033~1.503
Constant	−8.113	2.047	15.713	<0.01	0.000	

KIM-1: kidney injury molecule-1; NGAL: neutrophil gelatinase-associated lipocalin; NLR: neutrophil-lymphocyte ratio; APACHE II score: acute physiology and chronic health status II score; β: regression coefficient; S.E: standard error; OR: odds ratio; CI: confidence interval.

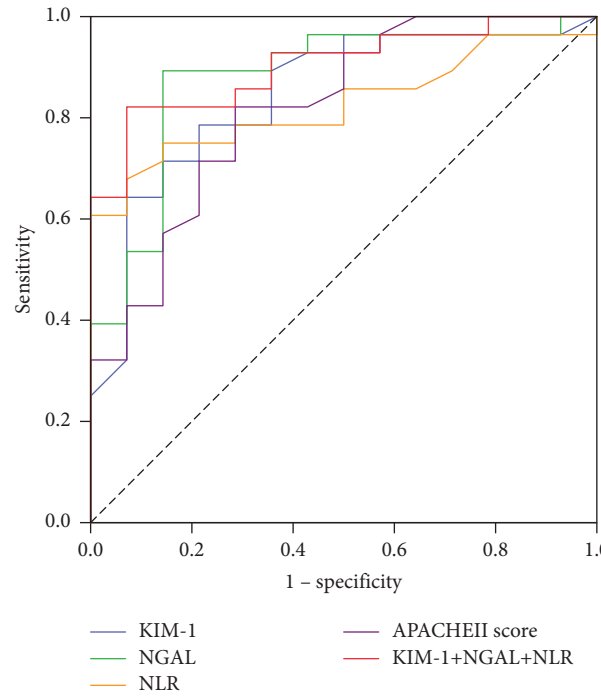

Figure 1: The receiver operating characteristic (ROC) curves of KIM-1, NGAL, NLR, and APACHE II score levels at admission on AKI in patients with acute diquat poisoning.

than 0.2 in univariate analysis were included in the binary logistic regression analysis, and the results showed that KIM-1, NGAL, and NLR were independent risk markers for AKI in patients with diquat poisoning as well as clinical indicators associated with early diagnosis and intervention in diquat poisoning patients. Our research may provide clinical guidance for reducing the occurrence of AKI.

Pesticide poisoning is a serious public health concern in China, which is an agricultural country. Diquat poisoning has increased following the paraquat prohibition in 2016 [7]. Because of its rapid onset, high fatality rate, and absence of a viable antidote, acute diquat poisoning has gained extensive attention. The kidney is the most commonly damaged organ as a result of diquat poisoning, and kidney injury contributes significantly to diquat death. Following kidney damage, diquat excretion rates decline, exacerbating kidney damage and eventually leading to renal failure. However, it is still unknown how kidney damage occurs. Kidney damage biomarkers have been conventional, including Scr, cystatin C, and BUN. Cystatin C has no sensitivity or specificity for the diagnosis of AKI [8]. The changes in Scr, BUN, and other markers are lagged behind changes in the renal function, which may lead the patients to miss the therapeutic opportunity [8, 9]. As a result, finding early kidney injury markers aids in early illness identification and treatment, reducing the occurrence of major adverse kidney events (MAKE) [10], and improving the prognosis of diquat poisoning patients. KIM-1, a kidney injury biomarker, has been

Table 5: The analysis of the predictive ability of KIM-1, NGAL, NLR, and APACHE II score levels at admission on AKI in patients with acute diquat poisoning.

Index	AUC	95% CI	Cut-off value	Sensitivity	Specificity
KIM-1	0.844	0.717~0.972	2.23 ng/ml	0.821	0.786
NGAL	0.878	0.761~0.994	188.9 ng/mL	0.893	0.857
NLR	0.827	0.704~0.949	3.96	0.750	0.814
APACHE II score	0.818	0.681~0.954	10.94	0.714	0.786
KIM-1 + NGAL + NLR	0.903	0.813~0.993		0.893	0.859

KIM-1: kidney injury molecule-1; NGAL: neutrophil gelatinase-associated lipocalin; NLR: neutrophil-lymphocyte ratio; APACHE II score: acute physiology and chronic health status II score; AUC: area under the curve.

shown to be an immunomodulatory protein produced by subepithelial cells in the proximal convoluted tubules of the kidney that is essential for both the process of renal interstitial fibrosis and the early healing of damage [4]. KIM-1 expression is higher when AKI first develops, but it remains high when renal function recovers and Scr returns to normal [4]. Therefore, KIM-1 could be employed as an early biological indicator of AKI in diquat poisoning patients. The study's findings revealed that the serum KIM-1 level of the patients was higher than that in the control group at admission, higher in the fulminant group than in the moderate to severe group ($P < 0.05$), and higher in the AKI group than in the NAKI group ($P < 0.05$). However, there was no noticeable difference ($P > 0.05$) in serum UA and BUN levels upon admission between the moderate to severe group and the fulminant group, as well as between the AKI group and the NAKI group.

Acute diquat poisoning is a common emergency room poisoning condition. Despite recent advances in emergency care, the fatality rate of diquat poisoning patients remains high. Following the combination of AKI, the patient's condition deteriorates and the risk of death increases. According to research, there are three categories of early biological indicators for detecting AKI: inflammatory markers, cell damage markers, and cell cycle markers. NLR is defined as the ratio of peripheral neutrophil count to lymphocyte count, and it is one of the most effective markers, reflecting the body's systemic inflammatory response, as well as a cheap, simple, and repeatable inflammatory marker [11, 12]. NLR is of great significance for the assessment of the condition and prognosis of inflammation-related diseases and can be used as a major predictor for AKI [5, 13]. The advantage of NLR is that it is simple to evaluate upon admission. This study found that diquat patients had greater NLR levels than the control group, AKI patients had higher NLR levels than NAKI patients ($P < 0.05$), 12 h was higher than admission ($P < 0.05$), 24 h was higher than 12 h ($P < 0.05$), and dynamic monitoring of NLR could predict the inflammatory response. NLR was identified as an independent risk factor for AKI in diquat poisoning patients in the binary logistic regression research. NGAL, another early biological marker of renal injury, is a lipid carrier protein that is expressed on the surface of neutrophils and secreted in small amounts in the liver cells and epithelial cells under physiological conditions. NGAL is released by neutrophils during sepsis, and a huge number of inflammatory factors are released to damage the renal tubular epithelial cells, inducing high NGAL expression [14, 15], and NGAL levels significantly increase within 3 h of AKI. According to the study's findings, the serum NGAL levels in diquat patients had risen upon admission and increased with disease severity, with the fulminant group greater than the moderate to severe group ($P < 0.05$) and the AKI group higher than the NAKI group ($P < 0.05$). The serum UA and BUN levels of patients admitted to the hospital were not statistically different from those in the control group ($P > 0.05$). According to our research, NGAL is expressed earlier than UA and BUN, and it has an early predictive value for the occurrence of AKI in diquat patients, allowing for early treatment and preventing the occurrence of MAKE, thereby improving patient prognosis and survival rate.

Glomerular filtration rate, urine volume, and blood creatinine are currently the most important indications of AKI diagnosis in patients and the recovery of renal function following AKI [6, 16], but there is a lag that cannot reflect the occurrence of AKI at an early stage. The simultaneous detection of several new biomarkers may aid in the clinical diagnosis of AKI. According to the results of the regression analysis, KIM-1, NGAL, and NLR are the independent risk markers for AKI in diquat poisoning patients. The ability of KIM-1 and NGAL to predict the occurrence of AKI in patients with diquat poisoning was examined and results showed that the sensitivity, specificity, positive predictive value, and negative predictive value of KIM-1 were 0.821, 0.786, 0.793, and 0.688, respectively; the sensitivity, specificity, positive predictive value, and negative predictive value of NGAL were 0.893, 0.857, 0.926, and 0.800, respectively, indicating that NGAL was higher than KIM-1. The combined detection of KIM-1, NGAL, and NLR has higher sensitivity and specificity, as well as a larger AUC, according to the ROC curve results. Our findings indicated that KIM-1, NGAL, and NLR can be used as early predictive indicators for AKI caused by diquat.

5. Conclusions

In summary, KIM-1, NGAL, and NLR can be used as indicators for the early diagnosis of AKI in diquat poisoning patients, and they may be involved in the pathogenesis of AKI associated with diquat poisoning, and the indicator changes in patients can be dynamically monitored to indicate the disease changes and adjust the treatment strategy. The increase in KIM-1, NGAL, and NLR levels was earlier

than the changes in UA and BUN, the traditional markers of kidney injury, and the combined detection was better than the single index, making early clinical diagnosis and treatment more conducive. Despite this, since this study is a single-center study with a limited number of cases, it still needs to be further confirmed by a larger sample size, multicenter, and prospective clinical studies.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

Acknowledgments

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