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Investigating Clinical Factors Influencing Pulmonary Fibrosis in Acute Diquat Poisoning

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Objective: This study aimed to explore the factors influencing pulmonary fibrosis in patients with acute diquat poisoning through logistic regression analysis.

Methods: A retrospective analysis was conducted on 68 cases of acute diquat poisoning due to suicidal intent admitted to our hospital from February 2020 to March 2023. Patients were divided into a combined group (28 cases with pulmonary fibrosis) and an acute diquat poisoning group (40 cases without). A healthy control group consisted of 40 individuals. General data were compared among the three groups, and laboratory indicators were analyzed. Single and multiple logistic regression analyses were performed to identify risk factors for pulmonary fibrosis.

Results: There were no significant differences in gender, age, BMI, poisoning status, or treatment timing among the groups ($P > 0.05$). The combined group had significantly higher diquat ingestion dose, SIRS score, SOFA score, and APACHE II score compared to the poisoning group ($P < 0.05$). In the acute poisoning group, these scores were also higher than in the healthy controls ($P < 0.05$). Laboratory indicators, including Hb, PLT, ALP, DBil, ALB, BUN, Glu, BNP, and pH, showed no significant differences ($P > 0.05$). However, WBC, ALT, TBil, DBil, Cr, K+, Tn I, and Lac levels were significantly higher in the combined group compared to the acute poisoning group ($P < 0.05$). Logistic regression analysis identified factors influencing pulmonary fibrosis as diquat ingestion dose, K+, ALT, PaO2, Lac, and HCO3-.

Conclusion: The factors influencing pulmonary fibrosis in acute diquat poisoning include diquat ingestion dose, K+, ALT, PaO2, Lac, and HCO3-. These findings enhance understanding of pulmonary fibrosis pathogenesis and may inform clinical management for affected patients.

Keywords: acute diquat poisoning, pulmonary fibrosis, K+, ALT, PaO_2

Introduction

As an effective alternative to paraquat, diquat has gradually been widely used in agricultural production in recent years.[1] However, cases of diquat poisoning are becoming increasingly common in clinical practice.[2,3] Diquat, an oxidoreductive pyridine herbicide, is classified by the World Health Organization (WHO) as a moderately toxic pesticide, along with Paraquat, both belonging to the bipyridyl group.[4–7] Acute diquat poisoning is associated with a high mortality rate, as it rapidly distributes throughout various organs and tissues upon entering the human body.[8] Diquat and its metabolites primarily exit the body via urine within 48 hours of ingestion, with a smaller portion excreted through bile metabolism.[9] It exhibits significant toxicity to multiple organs, including the lungs, kidneys, and liver, with pulmonary damage being particularly severe and challenging to treat clinically.[2,10] Mortality is often attributed to acute lung injury and refractory hypoxemia induced by chronic pulmonary fibrosis, accounting for approximately 45% of cases, similar to Paraquat poisoning.[11] Pulmonary fibrosis is a major contributor to mortality and represents a significant clinical challenge.[12,13]

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Currently, there is no established effective treatment for diquat-induced pulmonary fibrosis. Treatment typically involves a combination of various medications and symptomatic therapy based on clinical observations, but overall efficacy remains suboptimal, with a high mortality rate among patients.[14,15] Consequently, investigating the factors contributing to the development of pulmonary fibrosis in patients with acute diquat poisoning has become a key focus of clinical research. However, there is a paucity of studies investigating the factors associated with pulmonary fibrosis in acute diquat poisoning patients. Therefore, this study aims to explore the influencing factors for the development of pulmonary fibrosis in patients with acute diquat poisoning using logistic regression analysis.

Materials and Methods

Clinical Data

A retrospective analysis was conducted on 68 patients with acute diquat poisoning admitted to the first affiliated hospital of Guangxi Medical University from February 2020 to March 2023. Patients were categorized into the Combined Group (28 cases) and the Acute Diquat Poisoning Group (40 cases) based on the presence or absence of pulmonary fibrosis. Combined Group includes patients diagnosed with acute diquat poisoning who subsequently developed pulmonary fibrosis. This group was defined based on specific clinical criteria for pulmonary fibrosis, including symptoms such as dyspnea, reduced blood oxygen saturation (<90%), increased heart rate (>120 beats per minute), and the presence of fine Velcro crackles at the lung bases upon inspiration. Pulmonary function tests and CT imaging confirmed the fibrotic changes, characterized by restrictive ventilatory impairment and scattered fibrous streak-like alterations in the lungs. Additionally, there were 40 healthy individuals recruited as the control group.

Inclusion Criteria Included

(1) diquat poisoning (including oral ingestion, skin contact, and other exposure routes); (2) average time from poisoning to hospital admission ranged from 0.5 to 3.5 hours; (3) no gastric lavage performed before admission; (4) the detection of diquat poisoning is conclusively confirmed through urine testing using sodium bicarbonate/sodium metabisulfite assay techniques; (5) completion of at least one follow-up visit after discharge; (6) In the combined group of patients, clinical symptoms of pulmonary fibrosis manifested as sudden onset of dyspnea, decreased blood oxygen saturation to below 90%, heart rate increased to over 120 beats per minute, and fine Velcro crackles were auscultated at the bases of both lungs during inspiration. Pulmonary function tests revealed restrictive ventilatory impairment, while CT scans showed pulmonary fibrosis characterized by scattered fibrous streak-like changes in unilateral or bilateral lungs.[16]

Exclusion Criteria Included

(1) history of pulmonary, hepatic, or renal diseases, or conditions affecting liver and kidney function or any history of chronic exposure to toxins known to cause pulmonary fibrosis (2) abnormal coagulation function; (3) presence of chronic heart failure; (4) patient refusal of treatment and voluntary discharge against medical advice; (5) concomitant infectious diseases before admission; (6) suspected or confirmed poisoning with other toxins or drugs.

This study protocol complied with the principles outlined in the Declaration of Helsinki.

Methods

Clinical Data

Patient demographics, including age, gender, oral diquat dose, presence of hypotension upon admission, time from ingestion to admission, Systemic Inflammatory Response Syndrome (SIRS) score, Sequential Organ Failure Assessment (SOFA) score, and Acute Physiology and Chronic Health Evaluation II (APACHE II) score, were recorded.

Laboratory Parameters

White Blood Cell count (WBC), Hemoglobin (Hb), Platelet count (PLT), Alanine Aminotransferase (ALT), Alkaline Phosphatase (ALP), Total Bilirubin (TBil), Direct Bilirubin (DBil), Albumin (ALB), Potassium (K+), Blood Urea Nitrogen (BUN), Creatinine (Cr), Glucose (Glu), Troponin I (Tn I), Brain Natriuretic Peptide (BNP), pH, Partial Pressure of Oxygen (PaO2), Partial Pressure of Carbon Dioxide (PaCO2), and Lactate (Lac) levels were collected and analyzed.

Pulmonary Fibrosis Assessment

Pulmonary fibrosis was evaluated using high-resolution computed tomography (HRCT). Fibrotic signs included ground-glass opacities, reticular patterns, or honeycombing. Fibrosis extent was scored based on CT images. Each lung was divided into upper, middle, and lower lobes, and two slices were selected for scoring. Fibrosis severity was scored as follows: 0 points for no fibrosis; 1 point for fibrosis involvement <5%; 2 points for fibrosis involvement <1/4; 3 points for fibrosis involvement between 1/4 and 1/2; 4 points for fibrosis involvement between 1/2 and 3/4; and 5 points for fibrosis involvement >3/4. The total fibrosis score was calculated using a weighted formula. Patients with non-zero scores were classified as having pulmonary fibrosis.

Treatment Regimen

(1) Emesis induction and gastric lavage were performed upon admission, regardless of pre-hospital interventions. Gastric lavage was conducted with water until the lavage fluid became colorless and tasteless. (2) Fluid resuscitation and diuresis therapy included intravenous fluid therapy with balanced electrolyte solutions (such as Normal Saline or Glucose-Normal Saline) combined with furosemide to maintain a urine output of 1–2 mL/kg/h and a total intravenous fluid volume of 3500–4000 mL. (3) Blood purification therapy with hemoperfusion was initiated within 4 hours of admission. Heparinization was performed, and patients were connected to a blood purification device for 2–4 hours per session, once a day, for a total of 3–5 sessions. (4) Pharmacological treatment included administration of glutathione, N-acetylcysteine, vitamin C, and vitamin B for antioxidant therapy. Broad-spectrum antibiotics were administered if evidence of infection was present, followed by antibiotic adjustment based on bacterial culture and sensitivity results. Corticosteroids (methylprednisolone 15 mg/(kg·d)) and/or immunosuppressants (cyclophosphamide 10 mg/(kg·d)) were used in patients with moderate to severe diquat poisoning. (5) Respiratory support included oxygen supplementation via face mask for patients with acute respiratory distress syndrome and/or PaO2 <40 mmHg (5.3 kPa), with endotracheal intubation or tracheostomy performed if necessary.

Observational Parameters

(1) Comparison of general characteristics between the two groups. (2) Statistical analysis of laboratory parameters in both groups. (3) Single-factor logistic regression analysis of risk factors for pulmonary fibrosis in patients with acute diquat poisoning. (4) Multifactorial logistic regression analysis of risk factors for pulmonary fibrosis in patients with acute diquat poisoning.

Statistical Analysis

Data were analyzed using SPSS version 22.0. Categorical variables were expressed as percentages, while continuous variables were expressed as means ± standard deviations or medians. The chi-square test or analysis of variance was used for categorical variables, and the *t*-test was used for continuous variables in single-factor analysis. Single- and multiple-factor logistic regression analyses were conducted to identify risk factors for pulmonary fibrosis in patients with acute diquat poisoning, with $P<0.05$ considered statistically significant.

Results

The general information of the Combined Group (28 individuals), the Acute Diquat Poisoning Group (40 individuals) and healthy control group (40 individuals) was analyzed. The gender distribution in the three groups (male/female) was 18/10, 23/17 and 22/18, respectively, with no significant difference observed ($P>0.05$). The mean ages were 35.65±4.98 years, 36.21±5.47 years and 37.03±4.56 years in three groups, respectively, with no significant difference ($P>0.05$). Similarly, there was no significant difference in BMI among the three groups ($P>0.05$). Regarding the status at the time of poisoning, the difference in poisoning status distribution among the three groups has not been statistically analyzed. Upon admission, the time from ingestion to medical treatment was significantly shorter in the Combined Group (2.23 ±0.27 hours) compared to the Acute Diquat Poisoning Group (2.47±0.32 hours) ($P=0.001$). There was also a significant difference in the oral diquat dose, with the Combined Group receiving a dose of 46.19±4.91 mL, which was significantly

higher than the dose received by the Acute Diquat Poisoning Group (41.93±4.08 mL) (P<0.001). In terms of scoring, the SIRS score (2.37±0.46), SOFA score (2.29±0.39), and APACHE II score (2.32±0.41) in the Combined Group were all significantly higher than those in the Acute Diquat Poisoning Group (2.01±0.37, 2.03±0.32, and 2.04±0.39, respectively), with all P values <0.01. As shown in Table 1.

The laboratory test results revealed significant differences among the three groups in several laboratory parameters. Significant differences were found in several laboratory indicators among the groups. The Combined group showed elevated WBC (29.08±3.29 $\times 10^9$/L), ALT (29.09±4.39 U/L), TBil (13.28±2.32 μmol/L), DBil (5.49 ±0.76 μmol/L), BUN (5.23±0.46 mmol/L), Cr (112.81±18.26 mmol/L), and Lac (3.87±0.54 mmol/L) levels compared to the Acute diquat poisoning and Healthy control groups ($P < 0.05$). K^+ was lower (3.94±0.37 mmol/L), while $PaCO_2$ (24.38±12.76 mmol/L), PaO_2 (95.81±13.07 mmol/L), and HCO_3^- (21.07±2.02 mmol/L) also differed significantly ($P < 0.001$). No significant differences were observed for Hb, PLT, ALP, ALB, Glu, Tn I, BNP, and pH ($P > 0.05$). As shown in Table 2.

Univariate Logistic Regression Analysis of Factors Influencing the Occurrence of Pulmonary Fibrosis in Patients with Acute Diquat Poisoning

The independent variables, including the oral intake dosage of diquat, SIRS score, SOFA score, APACHE II score, WBC count, K+ concentration, ALT level, TBil level, DBil level, Cr level, Tn I level, PaO2 level, PaCO2 level, Lac level and $HCO3^-$ level were compared to assess their association with the occurrence of pulmonary fibrosis in patients with acute diquat poisoning. Single-factor logistic regression analysis was conducted. The results revealed that the influencing factors for pulmonary fibrosis in patients with acute diquat poisoning were the oral intake dosage of diquat, ALT level, K+ concentration, PaO2 level, PaCO2 level, Lac level and $HCO3^-$ level, as shown in Table 3.

Table 1 Comparison of General Information

Item	Combined Group (n=28)	Acute Diquat Poisoning Group (n=40)	Healthy Control Group (40 Individuals)	χ2/t	P
Gender (Male/Female) (n)	18/10	23/17	22/18	0.602	0.740
Age (Years)	35.65±4.98	36.21±5.47	37.03±4.56	1.182	0.241
BMI (kg/m2)	22.38±3.87	23.01±3.47	23.65±3.81	1.344	0.184
State at time of poisoning (n)				0.016	0.899
Satiated	8	12			
Fasting	20	28			
After Drinking	9	11	13	0.281	0.869
Time from medication to visit (h)	2.23±0.27	2.47±0.32		3.354	0.001
Oral dose of diquat (g)	46.19±4.91	41.93±4.08		−3.822	<0.001
SIRS Score (Points)	2.37±0.46	2.01±0.37		−3.486	<0.001
SOFA Score (Points)	2.29±0.39	2.03±0.32		−2.950	0.004
APACHE II Score (Points)	2.32±0.41	2.04±0.39		−2.857	0.006
Occupation (n)				4.958	0.762
Office clerk	2	3	5		
housewife	11	16	17		
Programmer	3	5	6		
accountant	4	4	7		
freelancer	8	12	5		

Abbreviations: BMI, Body Mass Index; SIRS, Systemic Inflammatory Response Syndrome Score; SOFA, Sequential Organ Failure Assessment Score; APACHE II, Acute Physiology and Chronic Health Evaluation II Score.

Table 2 Comparison of Laboratory Indicators Between Two Groups

Item	Combined Group (n=28)	Acute Diquat Poisoning Group (n=40)	Healthy Control Group (40 Individuals)	t	P
WBC ($\times10^9$/L)	29.08±3.29	23.23±3.32	6.83±1.23	−7.178	<0.001
Hb (g/L)	143.19±14.39	140.91±15.03	141.38±14.97	−0.499	0.620
PLT ($\times10^9$/L)	263.92±19.03	260.76±20.07	265.18±22.18	0.244	0.808
ALT (U/L)	29.09±4.39	21.27±4.52	17.98±4.21	−10.542	<0.001
ALP (U/L)	80.91±9.76	82.38±9.98	78.98±10.08	0.603	0.549
TBil (μmol/L)	13.28±2.32	10.09±2.43	7.03±2.09	4.857	<0.001
DBil (μmol/L)	5.49±0.76	4.92±0.69	4.03±0.71	−3.197	0.002
ALB (g/L)	47.81±3.29	48.14±3.36	50.91±3.47	0.407	0.685
K^+ (mmol/L)	3.94±0.37	3.64±0.33	4.12±0.32	−3.480	<0.001
BUN (mmol/L)	5.23±0.46	4.98±0.51	4.01±0.34	−2.121	0.038
Cr (mmol/L)	112.81±18.26	68.93±17.23	60.93±15.29	−10.085	<0.001
Glu (mmol/L)	6.93±1.29	6.76±1.21	6.09±1.07	−0.554	0.581
Tn I (ng/mL)	0.003±0.0003	0.002±0.0004	0.022±0.009	−11.783	<0.001
BNP (pg/mL)	10.65±2.65	10.74±2.73	11.23±2.87	−0.656	0.514
pH	7.45±0.54	7.41±0.58	7.39±0.52	−0.294	0.770
PaO2 (mmol/L)	95.81±13.07	97.98±12.28	97.98±12.28	−6.429	<0.001
PaCO2 (mmol/L)	24.38±12.76	42.94±13.28	44.87±11.17	−6.318	<0.001
Lac (mmol/L)	3.87±0.54	1.32±0.34	1.02±0.29	−22.556	<0.001
$HCO3^-$ (mmol/L)	21.07±2.02	22.23±2.08	25.03±2.21	7.530	<0.001

Abbreviations: WBC, White Blood Cells; Hb, Hemoglobin; PLT, Platelets; ALT, Alanine Aminotransferase; ALP, Alkaline Phosphatase; TBil, Total Bilirubin; DBil, Direct Bilirubin; ALB, Albumin; K+, Potassium; BUN, Blood Urea Nitrogen; Cr, Creatinine; Glu, Glucose; Tn I, Troponin I; BNP, B-type Natriuretic Peptide; pH, Potential of Hydrogen; PaO_2, Partial Pressure of Oxygen; $PaCO_2$, Partial Pressure of Carbon Dioxide; Lac, Lactate; HCO_3-, *Bicarbonate.*

Table 3 Univariate Logistic Regression Analysis of Factors Influencing Pulmonary Fibrosis in Acute Diquat Poisoning Patients

Variable	β	SE	Wald$\chi2$ Value	OR (95% CI)	P value
Oral Diquat Dosage	0.571	0.196	6.524	1.552 (1.078–2.187)	0.001
SIRS Score	1.319	0.703	3.494	0.984 (0.058–1.083)	0.061
SOFA Score	0.864	0.711	1.513	2.276 (0.578–6.758)	0.211
APACHEIIScore	0.105	0.283	0.134	1.124 (0.892–1.326)	0.576
WBC	0.262	0.691	1.457	2.186 (1.001–3.496)	0.226
ALT	0.285	0.206	6.356	1.305 (1.005–1.826)	0.006
K^+	1.872	0.181	17.856	6.087 (1.473–8.987)	<0.001
TBil	3.802	2.006	3.318	3.287 (1.202–7.021)	0.067
DBil	0.256	0.245	1.067	0.987 (0.453–1.345)	0.308
Cr	0.097	0.165	0.336	0.916 (0.073–1.324)	0.566
Tn I	0.082	0.238	0.316	1.082 (0.676–1.716)	0.736
PaO_2	0.526	0.175	2.836	1.653 (1.163–2.358)	0.004
$PaCO_2$	0.387	0.173	2.451	1.465 (1.034–2.006)	0.026
Lac	0.573	0.187	6.532	1.553 (1.071–2.195)	0.001
$HCO3^-$	1.003	0.159	6.382	2.387 (1.021–3.987)	<0.001

Abbreviations: SIRS, Systemic Inflammatory Response Syndrome Score; SOFA, Sequential Organ Failure Assessment Score; APACHE II, Acute Physiology and Chronic Health Evaluation II Score. WBC, White Blood Cells; ALT, Alanine Aminotransferase; TBil, Total Bilirubin; DBil, Direct Bilirubin; K+, Potassium; Cr, Creatinine; Glu, Glucose; Tn I, Troponin I; PaO_2, Partial Pressure of Oxygen; $PaCO_2$, Partial Pressure of Carbon Dioxide; Lac, Lactate; HCO_3-, *Bicarbonate.*

Table 4 Multifactorial Logistic Regression Analysis of Factors Influencing Pulmonary Fibrosis in Acute Diquat Poisoning Patients

Variable	β	SE	Wald χ2 Value	OR (95% CI)	P value
Oral Diquat Dosage	1.042	0.242	17.565	2.863 (1.692–3.883)	<0.001
ALT	2.163	0.494	16.295	7.985 (3.007–18.762)	<0.001
K^+	0.279	0.104	7.646	1.273 (1.046–1.767)	0.004
PaO_2	1.365	0.369	12.178	3.868 (1.761–6.324)	<0.001
$PaCO_2$	0.455	0.275	2.735	1.342 (0.903–2.317)	0.087
Lac	0.226	0.603	7.258	1.649 (1.172–2.187)	0.014
$HCO3^-$	2.239	0.621	12.398	6.398 (2.013–9.871)	<0.001

Abbreviations: ALT, Alanine Aminotransferase; K^+, Potassium; PaO_2, Partial Pressure of Oxygen; $PaCO_2$, Partial Pressure of Carbon Dioxide; Lac, Lactate. HCO_3^-, *Bicarbonate*.

The Multifactorial Logistic Regression Analysis of Factors Influencing the Occurrence of Pulmonary Fibrosis in Patients with Acute Diquat Poisoning

Factors identified as statistically significant in the single-factor analysis were included in the multifactorial analysis, including the oral intake dosage of diquat, ALT level, K+ concentration, PaO2 level, PaCO2 level, Lac level and $HCO3^-$ level. The dependent variable was the occurrence of pulmonary fibrosis in patients with acute diquat poisoning. The results revealed that the influencing factors for pulmonary fibrosis in patients with acute diquat poisoning were the oral intake dosage of diquat, K+ concentration, ALT level, PaO2 level, Lac level and $HCO3^-$ level, as shown in Table 4.

Discussion

In this study, we investigated the clinical factors influencing the development of pulmonary fibrosis in patients with acute diquat poisoning. The results indicated that the dose of diquat ingestion, potassium ions (K+), alanine aminotransferase (ALT), arterial partial pressure of oxygen (PaO2), lactate (Lac), and bicarbonate (HCO3-) were significantly associated with the occurrence of pulmonary fibrosis. These findings provide important insights into the pathogenesis of pulmonary fibrosis following acute diquat poisoning and may guide the development of clinical management strategies. Currently, there is limited research on the epidemiology, toxicokinetics, clinical diagnosis, and treatment of diquat poisoning, and there is a lack of studies with relatively large sample sizes. Most cases of diquat poisoning involve intentional ingestion of concentrated diquat liquid formulations, leading to complications in the liver, kidneys, heart, and gastrointestinal tract, which can be life-threatening.[17,18] Additionally, diquat can also cause toxic reactions through pulmonary, ocular, or dermal exposure, leading to severe complications such as multiple organ failure, acute respiratory distress syndrome, and acute lung injury, ultimately resulting in pulmonary interstitial or alveolar fibrosis, respiratory failure, and posing a serious threat to the patient's life and health.[18,19]

Currently, the mechanism of diquat poisoning is not fully understood. It is believed that diquat entering the body may lead to oxidative stress, inflammatory reactions, alveolar damage, and alveolar epithelial cell reconstruction, thereby inducing pulmonary fibrosis, resulting in hypoxemia and multiorgan failure leading to death.[14,20] Currently, clinical diagnosis is mainly based on detailed collection of medical history, confirmation of toxic exposure history, qualitative and quantitative toxicology testing, and assessment of organ damage. There is no clear grading method available, and clinical judgment is primarily based on the dosage of orally ingested diquat and organ damage to determine the severity of diquat poisoning. However, pulmonary fibrosis occurring after acute diquat poisoning can lead to impaired pulmonary gas diffusion and exchange function, resulting in difficult-to-treat hypoxemia.[21] Early occurrence of pulmonary fibrosis in patients with acute diquat poisoning is associated with poor prognosis and higher mortality rates.[8] Therefore, it is essential to promptly assess whether patients with diquat poisoning have concurrent pulmonary fibrosis.

Studies by Yan[11] et al indicated that patients with better prognosis of acute diquat poisoning were more prone to develop respiratory failure. Consistent with these findings, our results showed that the oral intake dosage of diquat, SIRS score, SOFA score, and APACHE II score were higher in the combined group than in the acute diquat poisoning group ($P<0.05$). This suggests that patients with acute diquat poisoning who develop pulmonary fibrosis have higher oral intake dosage of diquat, SIRS score, SOFA score, and APACHE II score. This may be because a higher dosage of orally ingested diquat leads to higher

drug levels in the urine, which may trigger pulmonary fibrosis, thereby affecting the prognosis of patients. Additionally, SIRS score, SOFA score, and APACHE II score can evaluate the prognosis of critically ill patients and are important for assessing disease severity and mortality rates.[22–24] WBC is an essential component of blood, and its levels increase during inflammatory reactions, enhancing the body's ability to resist pathogen invasion. ALT, TBil, and DBil can assess the severity of liver damage. Elevated ALT is often associated with systemic inflammation and may reflect an inflammatory response that exacerbates fibrosis. ROS can impact both lung and liver tissues, leading to ALT release; thus, oxidative stress—present in both fibrosis and hepatic inflammation—may further contribute to tissue damage. Previous studies have shown that higher serum Cr levels in the early stage of diquat poisoning are associated with a higher risk of respiratory failure and poorer prognosis.[25,26] Tn I is exclusively present in myocardial cells, and its concentration increases in the blood during myocardial injury, indicating a close relationship with heart damage. High concentrations of diquat affect the oxidative-reductive process of renal cells, leading to renal tubular necrosis and affecting K+ absorption. The concentration gradient of K^+ across the cell membrane is essential for maintaining cellular equilibrium. Disruptions in K^+ channel function can alter membrane potential, cellular excitability, and signal transduction, potentially promoting the fibrotic process. Furthermore, K^+ channels interact with calcium channels, impacting intracellular calcium signaling, which may lead to fibroblast proliferation and collagen deposition. PaCO2 is related to the carbon dioxide content in the blood and reflects respiratory factors in acid-base balance. PaO2 indicates the pressure of dissolved oxygen molecules in the plasma and reflects the cell's ability to utilize oxygen, determined by the oxygen partial pressure of inhaled gas and the functional status of external respiration. Lac is derived from erythrocytes, brain tissue, and skeletal muscle, and its serum level reflects tissue metabolic status and oxygen supply. The results of this study indicate that there were no significant differences in Hb, PLT, ALP, DBil, ALB, BUN, Glu, BNP, and pH among the three groups ($P > 0.05$). However, the Combined group exhibited higher levels of WBC, ALT, TBil, DBil, Cr, K^+, Tn I, Lac and lower level of PaO_2, $PaCO_2$, and HCO_3^- compared to the Acute diquat poisoning group. Additionally, the Acute diquat poisoning group had higher levels of WBC, ALT, TBil, DBil, Cr, K^+, Tn I, Lac and lower level of PaO_2, $PaCO_2$, and HCO_3^- than the Healthy control group ($P < 0.05$), indicating heart damage, renal dysfunction, and abnormal blood gas analysis parameters. However, the specific mechanisms require further research for confirmation.

Research by Meng[27] et al suggested that the prognosis of acute diquat poisoning is influenced by the dosage of orally ingested diquat. Zhu[25] et al suggested that Lac can effectively evaluate the severity of diquat poisoning and assess the prognosis level. However, there is limited research on the factors influencing the occurrence of pulmonary fibrosis in patients with acute diquat poisoning. Our study results showed that in the single-factor logistic regression analysis, the influencing factors for the occurrence of pulmonary fibrosis in patients with acute diquat poisoning were the oral intake dosage of diquat, ALT, K+, PaO2, PaCO2, Lac and $HCO3^-$. Further analysis using multifactor logistic regression revealed that the influencing factors for the occurrence of pulmonary fibrosis in patients with acute diquat poisoning were the oral intake dosage of diquat, K+, ALT, PaO2, Lac and $HCO3^-$, suggesting that the oral intake dosage of diquat, K+, ALT, PaO2, Lac and $HCO3^-$ are influencing factors for the occurrence of pulmonary fibrosis in patients with acute diquat poisoning.

The limitations of our study primarily include a relatively small sample size and its nature as a single-center retrospective analysis, which may be subject to geographical and demographic biases, potentially limiting the generalizability of the results. Additionally, due to the observational design of our study, causal relationships cannot be established, and only associations between relevant factors and pulmonary fibrosis can be inferred. Therefore, it is essential to interpret and extrapolate the study findings with caution considering these limitations.

In summary, our study identifies key factors associated with pulmonary fibrosis in acute diquat poisoning patients. Factors such as diquat ingestion dosage, potassium levels, ALT levels, PaO2, lactate and $HCO3^-$ levels are significantly correlated with pulmonary fibrosis development. Our study contributes to improving early detection and management strategies for better patient outcomes.

Data Sharing Statement

The datasets generated and analyzed during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

The study protocol was approved by the Ethics Committee of the First Affiliated Hospital of Guangxi Medical University (2021-09), and the study was performed in accordance with the Helsinki II declaration. Informed consent was obtained from all the study subjects before enrollment.

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Disclosure

The authors declare that they have no competing interests.

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