Research Paper

Prognostic Factors of Paraquat Toxicity in Urmia: Seven-Year Retrospective Investigation

Mohammad Majidi[1*], Mohammad Delirrad[1], Ayda Mostafazadeh[2]

1. Department of Forensic Medicine and Toxicology, School of Medicine, Urmia University of Medical Sciences, Urmia, Iran.
2. Department of Neurology, Tabriz University of Medical Sciences, Tabriz, Iran.

Citation Majidi M, Delirrad M, Mostafazadeh A. Prognostic Factors of Paraquat Toxicity in Urmia: Seven-Year Retrospective Investigation. International Journal of Medical Toxicology and Forensic Medicine. 2025; 15(4):E50017.

https://doi.org/10.22037/ijmtfm.v15i4.50017

Article info:
Received: 22 Aug 2025
First Revision: 19 Sep 2025
Accepted: 24 Sep 2025
Published: 30 Oct 2025

Keywords:
Pesticides, Herbicides, Paraquat, Acute Poisoning, Iran

ABSTRACT

Background: Paraquat is a very fatal herbicide. It causes such serious complications as acute respiratory distress syndrome, fibrotic lungs, metabolic acidosis, cardiac shock, renal failure, and even death. For this reason, in this study, the prognostic factors of paraquat intoxication were evaluated.

Methods: In this retrospective study, all paraquat-intoxicated patients hospitalized to Taleghani Hospital in Urmia, Iran, from 2014 to 2020 were assessed. The demographics, clinical, and laboratory findings were evaluated using a checklist and investigated in relation to the patients' outcomes. Then, the collected data were analyzed using SPSS version 21.

Results: Our study evaluated 102 cases (71 males & 31 females). The mortality rate was 46.1% (31.4% in males and 14.7% in females) in these patients. The patients' age was (31.4 years ± 13.3 standard deviation) (min=14, max=79). The Median ingestion amount of paraquat was 174 ml (min = 10 ml, max = 1500 ml). Nausea 86 (84.3%), vomiting 78 (76.5%), mucosal lesions of the oral cavity and pharynx 60 (58.8%), epigastric pain 35 (34.3%), and acute respiratory failure 30 (29.4%), loss of consciousness 13 (12.7%), were the important findings in the investigated patients, respectively. Significant relationships (P<0.05) were observed between the patients' outcomes and demographic (the ward & duration of admission), clinical findings (the level of consciousness, vomiting, epigastric pain, acute respiratory failure, hypertension, & sinus bradycardia) and laboratory indices, such as White Blood Cells, Bilirubin, Serum Hco3, Aspartate transaminase (SGOT), Alanine aminotransferase (SGPT), Creatinine, Blood glucose, Platelet, Calcium, Potassium, Leukocytosis, Metabolic acidosis, Hypokalemia.

Conclusion: Some factors, especially laboratory and clinical features, can be applied as a guide to the severity of patients' poisoning, which can be used for effective management by healthcare providers. However, more studies will be needed to clarify absolute prognostic factors in patients with paraquat toxicity.

*** Corresponding Author:**
Mohammad Majidi, MD
Department of Forensic Medicine and Toxicology, School of Medicine, Urmia University of Medical Sciences, Urmia, Iran. Postal Code: 57159-74677. Tel: (+98) 44 3344 2000.
E-mail: *majidi_m@umsu.ac.ir, mohammad.majidi57@yahoo.com*

Introduction

Acute poisoning is one of the important causes of death in the world [1]. Previous studies revealed that the mortality rate from pesticide poisoning in the Asian Pacific countries was 300,000 deaths every year [2]. Pesticide toxicity occurs intentionally (suicidal attempts), accidentally, and criminally, respectively [3]. Herbicide poisoning, such as paraquat, has become very common in developing agricultural countries such as Iran [4]. Paraquat (dimethyl bipyridinium dichloride) is a non-selective and dose-dependent herbicide and is used for suicide attempts due to its availability and cheapness. Paraquat can inhibit the conversion of NADH to NADPH, leading to cellular damage, organ damage, and ultimately, death due to the production of oxygen-free radicals and inflammatory reactions [5-7]. Oral ingestion is the main manner of toxicity due to paraquat [7]. Paraquat affects the heart, liver, and kidneys and causes severe complications, including pulmonary, coagulation, and fibrinolysis disorders [8-10]. Progressive pulmonary damage and acute renal failure are the important causes of death in paraquat toxicity [9, 10]. Recent studies have shown that the minimum lethal dose of paraquat poisoning for adults and children is 7-8 mL and 30 mg/kg, respectively [7, 11]. Also, recent studies have revealed that the lethal doses of paraquat toxicity are 20 cc of a 20% solution and 40 cc of a 24% solution [10, 12]. Because of high mortality due to paraquat, in some countries, such as the United States, the production and selling of paraquat have been stopped since 1994 [5, 7]. Diagnosis of paraquat poisoning involves clinical findings, blood levels, and urine tests [3, 10].

Paraquat poisoning has no specific antidote; however, some treatments can be used for paraquat toxicity, including gastric lavage, ingestion of activated charcoal, charcoal hemoperfusion, pulmonary support, corticosteroids, immunosuppressants, and parenteral nutrition with antioxidants [6, 10]. Recent studies have shown that increased leukocytes, prothrombin, blood and urine creatinine concentrations, paraquat consumption, pancreatic enzymes, and arterial lactate were poor prognosticators in paraquat poisoning [10, 12] since laboratory indices and their usefulness in predicting the outcome of paraquat-poisoned patients have been reported in limited studies [8-10]. Therefore, in this study, laboratory findings and their association with clinical findings and prognosis in patients with paraquat toxicity.

Materials and Methods

Our descriptive-analytical study was performed on 102 paraquat-intoxicated patients hospitalized at Taleghani Hospital in Urmia, Iran, from 2014 to 2020. To perform this study, all admitted paraquat-intoxicated patients aged 14 years or older were included. In each patient, the confirmation of paraquat poisoning had been made based on clinical findings, with evidence of paraquat consumption collected from the patient or other family members. The specialists also visited patients and provided more information to confirm the diagnosis at the time of hospitalization. Table 1 presents some demographic and clinical findings, as well as their impact on outcome. Patients who did not have complete documents and laboratory tests during admission were excluded from this study. All biochemical indices were evaluated upon admission. Biochemical indices and prognosis were retrospectively collected using a checklist, as presented in Table 2.

Since paraquat toxicity has no effective antidote, the patient's treatment was only supportive. For all patients, gastric lavage with 0.9% sodium chloride and activated charcoal was prescribed. Other components of supportive care administered included N-acetylcysteine, Antioxidants (Vit C and E), corticosteroids, dopamine, norepinephrine, and hemodialysis. Also, a Comparison of mortality rates between the two groups (survivors and non-survivors) from 2014 to 2020 is presented in Table 3. No personal identification data was recorded, and all data was kept confidential. This study was conducted after approval by the ethics committee of Urmia University of Medical Sciences, Iran. (Code: IR.UMSU.REC.1398.436). The data included demographic, clinical, and laboratory features for every patient.

Additionally, the variables were classified into survivor and non-survivor groups. In this investigation, the differences in quantitative variables with normal and abnormal distributions were assessed using the T-test and the Mann–Whitney U-test, respectively. The correlations between categorical variables and the patients' outcome were also assessed using the Chi-square test. In all patients, a confidence interval of 95% and a p-value <0.05 were considered to be significant. Finally, the data were analyzed using SPSS version 21.

Results

In this research, 102 patients (71 males and 31 females) were evaluated. The male-to-female ratio was 2:3. The mortality rate was 46.1% (31.4% in males and 14.7% in females). The patients' mean ± SD age (min-max) was 31.4 ± 13.3 (14-79) years. The most paraquat-intoxicated patients, 60 (58.3%), were in the age group of 14-29 years. The volume of consumed paraquat was approximately expressed. For example, a container cap, mouthful, cup, and mug were considered to be 25 ml, 30 ml, 150 ml, and 250 ml, respectively. The Mean±SD amount of paraquat ingestion was 174 ± 242 ml (min=10 ml, max=1500 ml). The mean ± SD of the length of hospitalization was 5.1 ± 4.36 days. Table 1 presents demographic and clinical findings, along with their impact on outcome. Most intoxications occurred in summer and spring (36.3%, 30%, respectively). The majority of patients (92, 90.2%) had no history of drug abuse. Also, 59 (57.8%) and 43 (42.2%) of the patients lived in rural and urban areas, respectively. Most of the intoxicated patients were from Khoy 53, 51.5%) and Urmia (40, 38.8%). About 63 (61.8%) of the poisoning patients were married. The majority of patients (90, 88.2%) had no history of suicide attempts. About 99 (97.1%) of patients ingested the paraquat orally, and 3 (2.9%) of patients had other routes of poisoning, such as inhalational and dermal. The main causes of paraquat ingestion were suicidal attempt (89.2%) and accidental (10.8%), respectively. Nausea 86 (84.3%), vomiting 78 (76.5%), mucosal lesions of oral cavity and pharynx 60 (58.8%), epigastric pain 35 (34.3%), acute respiratory failure 30 (29.4%), loss of consciousness 13 (12.7%), sinus tachycardia 13 (12.7%), hyper- tension 10 (9.8%), vertigo 9 (8.8%), sinus bradycardia 5(4.9%), weakness 5 (4.9%), miosis 4 (3.9%), agitation 3 (2.9%), diarrhea 3 (2.9%), headache 3 (2.9%) and hypotension 2 (1.9%) were the important clinical findings in this research, respectively. Other signs and symptoms included tremors, sweating, chest pain, headache, and mydriasis. Some laboratory tests and their effect on outcome and P-value are presented in Table 2. Furthermore, 49 (48%), 22 (21.6%), 2 (1.9%), and 1 (0.9%) of the studied patients had Leukocytosis, Hypernatremia, Hyperglycemia, and Hyperkalemia, respectively. Additionally, 36 (35.3%), 3 (2.9%), 1 (0.9%), and 1 (0.9%) of these patients had Hypokalemia, Thrombocytopenia, Hyponatremia, and Hypoglycemia, respectively. Approximately 10 patients (9.8%) underwent endoscopy, and various degrees of gastrointestinal lesions were reported. Sixty-nine (67%) of patients underwent hemodialysis. The mean ± SD of hemodialysis was 6 ± 7.2 hours. The average frequency of hemodialysis was 2 ± 2.34 times. Also, there was no significant relationship between treatment outcomes and hemodialysis or its frequency, but all patients who underwent hemodialysis more than 6 times died. Significant relationships (P value <0.05) were found between the patients' outcomes and

Table 1. Some demographic and clinical findings and their effect on outcome.

Items	Survivor group (number=55)	Non-survivor group (number=47)	P value (Chi-square test)
A). Demographic			
Admission places Ward, Intensive care unit (ICU) Ward and ICU, Simultaneously	 30 (54.5%) 4 (7.3%) 21 (38.2%)	 8 (17%) 20 (42.6%) 19 (40.4%)	0.000
Duration of hospital stay (days)	6.1±4.4	4±3.8	0.01 (Independent sample T-Test)
B) Some Clinical disorders (using the square test)			
Level of consciousness GCS, 15/15 GCS (8/15 to 15/15) GCS lower than 8/15	 52 (94.5%) 3 (5.5%) 0	 37 (78.7%) 10 (21.3%) 0	0.03
Vomiting	38 (69%)	40 (85%)	0.04
Epigastric pain	19 (34%)	16 (34%)	0.03
Hypertension	1 (1.8%)	9 (19.1%)	0.003
Sinus bradycardia	0	5 (10.6%)	0.01
Acute respiratory failure	6 (10.9 %)	24 (51%)	0.001
Acute Liver Failure	0	5 (10.6%)	0.01
Acute Renal Failure	1 (1.8%)	13 (27.6%)	0.000

International Journal of
Medical Toxicology & Forensic Medicine

SD: standard deviation, GCS: Glasgow Coma Score

Table 2. Some laboratory tests and their effect on outcome and P-value.

Quantitative variables	Survivor group (number=55)	Non-survivor group (number=47)	P value
A) Blood tests with normal distributions (Mean±SD) and using the T-test.			
Platelet (×10³/L)	522 ± 219	250 ± 79	0.007
Calcium (mg/dl)	9.44 ± 0.6	13 ± 1.6	0.002
Direct Bilirubin (mg/dL)	0.27 ± 0.07	1.8 ± 0.89	0.001
B) Blood tests with abnormal distributions (median (min, max)) and using the Mann-Whitney U-test.			
White Blood Cells (×10³/L)	9.2 (4.9 – 17.1)	12.4 (9.1 – 22.9)	0.000
Serum Hco3	23.2 (17 – 34)	17.9 (12.6 – 27.8)	0.000
Blood Glucose (mg/dl)	99 (23 – 255)	134 (77 – 216)	0.000
Aspartate transaminase (IU/L)	18 (11– 30)	26 (11 – 594)	0.000
Alanine aminotransferase (IU/L)	13 (10 – 53)	20 (9 – 385)	0.001
Creatinine (meq/l)	1 (0.7 – 3.8)	1.5 (0.8 – 11.2)	0.000
Potassium (mg/dl)	3.6 (1.1 – 6.9)	3.2 (2.4 – 4.6)	0.000
C) Other abnormal tests and using chi-square			
Leukocytosis (WBC>11000 /mm³)	19 (34.5%)	30 (63.8%)	0.005
Hypokalemia (K<3.5 meq/l)	6 (10.9%)	30 (63.8%)	0.000
Metabolic Acidosis	1 (1.8%)	8 (17%)	0.007

International Journal of Medical Toxicology & Forensic Medicine

demographic (admission service & the duration of hospital stay), clinical findings (the level of consciousness, vomiting, epigastric pain, acute respiratory failure, hypertension, & sinus bradycardia) and biochemistry tests, such as White Blood Cells, Direct Bilirubin, Serum Hco3, Aspartate transaminase, Alanine aminotransferase, Creatinine, Blood Glucose, Platelet, Calcium and Potassium. Moreover, statistically significant relationships were observed between the patients' outcomes and Leukocytosis, Metabolic Acidosis, and Hypokalemia (Table 2). However, there was no significant relationship between the patients' outcomes and demographic characteristics (History of substance abuse, living area, marital status, a history of suicidal attempts, the amount of ingested paraquat and hemodialysis or its frequency), clinical features (diarrhea, sinus tachycardia, hypotension, mucosal lesions of oral cavity and pharynx, vertigo, agitation and headache), and laboratory profile, such as Sodium, Blood urea nitrogen, Creatine kinas, Hemoglobin, Hematocrit, Partial Thromboplastin Time, Phosphor, Total bilirubin, Alkaline Phosphatase, Magnesium, Amylase and Lipase.

Discussion

Paraquat is a very toxic herbicide with severe complications and mortality. The average age of the patients was 31.4 years. Overall, the results of this investigation are similar to those of other studies. These studies show that paraquat poisoning has increased among young people in the age group of 14-30 years [5, 13, 14] (Table 4). Contrary to this study, the majority of patients were female [9, 13, 14]. The

Table 3. Comparison of mortality rates between two groups (survivors and non-survivors) from 2014 to 2020.

Date of hospitalization	Survivor group (number=55)		Non-survivor group (number=47)		Total (number=102)	Mortality Rate (%)
	Male	Female	Male	Female		
2014	2	0	4	2	8	75 %
2015	2	1	2	4	9	66.6 %
2016	8	0	4	5	17	52.9 %
2017	11	8	6	3	28	32.1%
2018	5	3	5	3	16	50 %
2019	7	1	6	0	14	42.8%
2020	6	1	3	0	10	30%

International Journal of Medical Toxicology & Forensic Medicine

Table 4. Mortality rates and prognostic indices in this study are compared to those in other investigations.

Articles	Non-survived/Total patients, (Mortality %)	P value<0. 05																
		Admission service	Serum Hco3	Loss of consciousness	Hypokalemia	Leukocytosis	Age	SGOT and SGPT	pH and Metabolic Acidosis	Duration of hospital stay	Acute respiratory failure	Acute Liver Failure	Acute Renal Failure	Amount of ingested paraquat (mL)	Increased serum creatinine	Dithionite urine test	Plasma paraquat concentration
Our study (2014-2020)	47/102, (46.1%)	+	+	+	+	+	NSR	+	+	+	+	+	+	NI	+	NI	NI
Delirrad et al [4] (2007-2013)	19/41, (46.3%)	+	NI	+	NI	+	NSR	NI	NI	+	+	+	+	+	+	NI	NI
Flechel et al [13] (2008-2014)	6/26, (23%)	+	+	NI	+	NI	+	NI	+	NI	NI	NI	+	NSR	NSR	+	NI
Jamshidi et al [5] (2004-2015)	63/159, (39.6%)	NI	NI	NI	NI	NI	+	NI	+	NI	+	NI	+	+	NI	NI	NI
Feng et al [9] (2012-2017)	58/ 96, (60.4%)	NI	NI	NI	NI	+	NSR	+	NI	NI	NI	NI	NI	NI	+	NI	+
Zhou et al [14] (2010-2015)	105/202 (51.98%)	NI	NI	NI	+	+	+	NSR	+	NI	+	NI	NI	+	+	NI	+

International Journal of
Medical Toxicology & Forensic Medicine

NSR = not significant relationship, NI= not implemented

suicidal attempts in this study were similar to the investigation of Delirrad et al [4] and Jamshidi et al [5]. In this study, the Median ingestion amount of paraquat was 100 ml, which was 5 times more than the study by Zhou et al. [14]. The majority of poisonings occurred in the summer and spring. The majority of poisonings occurred in summer and spring, as this area is one of the important agricultural centers, and 57.8% of the patients were rural [4]. Similar to this study, the main clinical findings at hospitalization were gastrointestinal disorders such as nausea and vomiting [4, 5]. Contrary to this study, the majority of patients were single [5].

Similar to this study, most patients ingested the paraquat orally and for deliberate self-harm [4, 5]. In our study, the approximate volume of paraquat consumed on admission differs between the survivor and non-survivor groups. Also, these data had valuable information without prognostic value in poisoned patients. In the assessment of Arterial blood gases, low Serum pH and HCO3 were observed in both surviving and non-surviving groups, which was similar to other studies. [5, 13]. Interestingly, the median blood glucose level in this study was very low in the surviving group compared to the non-surviving group.

The mortality rate due to paraquat poisoning in this study was 46.1%. In contrast, the mortality rate reported by Flechel et al. [13] was 23%, which is lower than the rate in this study and other previous studies [4, 5, 9, 14]. In some studies, it has been demonstrated that APACHE II, SOFA2, Hyperuricemia, Hyperamylasemia, increasing prothrombin time, and Arterial lactate have a poor prognosis in paraquat intoxication [10, 15, 16]. Our study demonstrated that Admission service, loss of consciousness, Epigastric

pain, hypertension, Platelet Count, Calcium, Blood Glucose, Aspartate transaminase, and Alanine aminotransferase were significantly varied between the two groups (survivors and non-survivors). Since toxicity with paraquat has no specific treatments or antidote. Therefore, early diagnosis using prognostic factors of mortality may provide appropriate care for these patients. Finally, to achieve better results, follow-up studies are recommended to investigate other prognostic factors.

Study Limitations

The important limitations of this study were the laboratory facilities and staff available to measure the levels of urinary dithionite and blood paraquat.

Conclusion

Some clinical findings, such as oral and pharyngeal mucosal lesions, acute respiratory failure, and a history of pesticide ingestion, can be applied for the diagnosis of paraquat poisoning. Some Laboratory tests, such as urine analysis, Sodium Dithionite, and Plasma paraquat concentration, confirm the paraquat poisoning. Although the amount of ingested paraquat on arrival provides valuable information, these data had no prognostic value in patients with paraquat toxicity. In previous research, various prognostic factors and their impact on prognosis have been investigated. Considering the impact of certain factors on patient prognosis, it is essential to incorporate these factors into patient assessment and management. Some factors, especially laboratory and clinical features, can be applied as a guide to the severity of patients' poisoning, which can be used for effective management by healthcare providers. However, more studies will be needed to clarify absolute prognostic factors in patients with paraquat toxicity.

Ethical Considerations

Compliance with ethical guidelines

The Ethics Committee of Urmia University of Medical Sciences approved this study (Code: IR.UMSU.REC.1398.436).

Acknowledgment

The authors would like to thank the management and staff of Taleghani Hospitals, Urmia, Iran, for approving the protocol prior to its implementation and for supporting this study.

Funding

Urmia University of Medical Science supported this article.

Conflicts of Interest

The authors declared no conflict of interest.

References

[1] Jamshidi F, Ghorbani A, Darvishi S, Davoodzadeh H. Study of laboratory profile in patients with aluminium phosphide poisoning in the southwest of Iran from 2010 to 2015. Arch Med Sadowej Kryminol. 2016;66(3):149-57. [DOI: 10.5114/amsik.2016.66399]

[2] Hassanian Moghaddam H, Zamani N. Therapeutic role of hyperinsulinemia/euglycemia in aluminum phosphide poisoning. Medicine (Baltimore). 2016;95(31):e4349. [DOI: 10.1097/MD.0000000000004349]

[3] Cao ZX, Zhao Y, Gao J, et al. Comparison of severity index and plasma paraquat concentration for predicting survival after paraquat poisoning: A meta-analysis. Medicine (Baltimore). 2020; 99(6): e19063. [DOI: 10.1097/MD.0000000000019063]

[4] Delirrad M, Majidi M, Boushehri B. Clinical features and prognosis of paraquat poisoning: a review of 41 cases. Int J Clin Exp Med. 2015;8(5):8122-8. [Link]

[5] Jamshidi F, Fathi G, Davoodzadeh H. Investigation Paraquat Poisoning in Southwest of Iran - from Sign to Mortality and Morbidity. Arch Med Sadowej Kryminol. 2017;67(1):35-45. [DOI: 10.5114/amsik.2017.70336]

[6] Meng Z, Dong Y, Gao H, et al. The effects of ω-3 fish oil emulsion-based parenteral nutrition plus combination treatment for acute paraquat poisoning. J Int Med Res. 2019;47(2):600-14. [DOI: 10.1177/0300060518806110]

[7] Wang X, Zhang M, Ma J, et al. Metabolic changes in paraquat poisoned patients and support vector machine model of discrimination. Biol Pharm Bull. 2015;38(3):470-5. [DOI: 10.1248/bpb.b14-00781]

[8] Hu X, Guo R, Chen X, Chen Y. Increased plasma prothrombin time is associated with poor prognosis in patients with paraquat poisoning. J Clin Lab Anal. 2018; 32(9):e22597. [DOI: 10.1002/jcla.22597]

[9] Feng S, Gao J, Li Y. A retrospective analysis of leucocyte count as a strong predictor of survival for patients with acute paraquat poisoning. PLOS One. 2018;13(7):e0201200. [DOI: 10.1371/journal.pone.0201200]

[10] Feng MX, Li YN, Ruan WS, Lu YQ. Predictive value of the maximum serum creatinine value and growth rate in acute paraquat poisoning patients. Sci Rep. 2018;8(1):11587. [DOI: 10.1038/s41598-018-29800-0]

[11] Song Y, Li C, Luo F, Tao Y. Clinical features and risk factors of acute kidney injury in children with acute paraquat intoxication. J Int Med Res. 2019;47(9):4194-203. [DOI: 10.1177/0300060519860032]

[12] Wang WJ, Cao ZX, Feng SY, et al. Platelet-lymphocyte ratio is not a prognostic predictor for acute paraquat-intoxicated patients: A retrospective analysis. Medicine (Baltimore). 2019;98(20):e15702. [DOI: 10.1097/MD.0000000000015702]

[13] Flechel A, Jolivet A, Boukhari R, Misslin-Tritsch C, Manca MF, Wiel E, et al. Paraquat poisoning in Western French Guyana: a public health problem persisting ten years after its withdrawal from the French market. Eur Rev Med Pharmacol Sci. 2018;22(20):7034-8. [DOI: 10.26355/eurrev_201810_16175]

[14] Zhou DC, Zhang H, Luo ZM, Zhu QX, Zhou CF. Prognostic value of hematological parameters in patients with paraquat poisoning. Sci Rep. 2016, 8; 6:36235. [DOI: 10.1038/srep36235]

[15] Kavousi-Gharbi S, Jalli R, Rasekhi-Kazerouni A, Habibagahi Z, Marashi SM. Discernment scheme for paraquat poisoning: A five-year experience in Shiraz, Iran. World J Exp Med. 2017;7(1):31-9. [DOI: 10.5493/wjem. v7.i1.31]

[16] Zhang J, Zhao Y, Bai Y, Lv G, Wu J, Chen Y. The significance of serum uric acid level in humans with acute paraquat poisoning. Sci Rep. 2015;5:9168. [DOI: 10.1038/srep09168]