IJCRT.ORG ISSN : 2320-2882

INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

Paraquat - A Deadly Poison Report Of A Case And Review

P. Lakshmi Prasanna
Department of Forensic science
Parul university
Vadodara, Gujarat, India

Abstract:
Paraquat is a Bipyridilium herbicide used widely in our country and is a highly toxic compound. A 16-year-old female patient was admitted to the emergency department of our tertiary care hospital in South India with the history of alleged consumption of paraquat poison. Since there is dearth of High Quality evidence- based treatment for this poisoning, different treatment modalities have been tried to manage patient's condition. In this case, none of the strategies could work well. Most of the patients reported with paraquat intoxication are from agricultural background; usually such patients cannot afford the treatment expenses. This paper presents a fatal case of acute poisoning with paraquat who succumbed to acute respiratory distress syndrome (ARDS).

Keywords: Acute respiratory distress syndrome, case study, clinical toxicology, herbicide, paraquat, pesticide poisoning

Introduction:

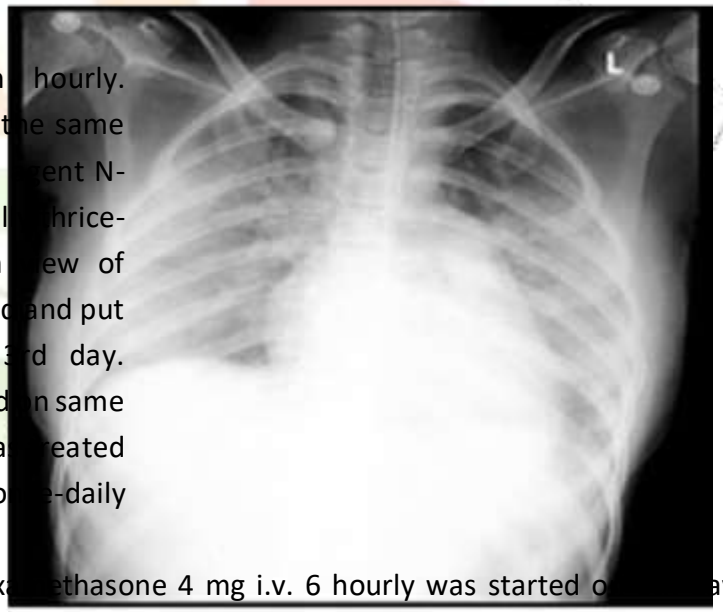
Paraquat, a toxic bipyridyl herbicide, is a bright green corrosive liquid with pungent smell. Its herbicidal properties were discovered in 1950s and first marketed in 1962. Once, it was encouraged by US for usage in Mexico to abolish marijuana plants. Presently, it is the second highest-selling weed killer globally and is available in a 20% solution form and that needs to be diluted before agricultural use.[1] Usually, adult cases of intoxication are due to suicidal attempts instead of homicidal or accidental exposure. The main acute systemic effects are pulmonary Edema, convulsions, cardiac, renal, and hepatic failure.[2] The LD50 in humans is approximately 35 mg/kg, which translates into as little as 10-15 ml of a 20% solution. This paper represents a case of acute poisoning with paraquat and detailed review of the intoxication

Case Report:

Access this article online Website: www.ijccm.org DOI: 10.4103/0972-5229.117074 Quick Response Code: A 16-year-old female patient was admitted to emergency department of our tertiary care hospital with history of alleged consumption of paraquat poison, 13 days before at 19.00 hours at her residence due to failure in matriculation examination. History revealed that after consumption of the poison, she was taken to the local hospital after ½ an hour. Meanwhile, she was reported to have 6 episodes of vomiting. She was treated conservatively; gastric lavage followed by activated charcoal

1 gm/kg was given as an adsorbent and discharged after 6 days from a local hospital. Since then, she was suffering from fever and cough without shortness of breath, orthopnea, chest, and abdominal pain. After 1 week at 21.00 hours, she was brought to our hospital. Her O2 saturation was 40% on room air, pulse rate was 78 beats/minute, respiratory rate28 beats/minute, and blood pressure was 100/60 mmHg. Respiratory examination revealed bilateral crepitation. Patient was administered O2 by 60% venturi, and her saturation picked up to 95%. Laboratory tests showed leukocytosis, neutrophilia, elevated ESR, and metabolic acidosis with normal renal and liver function. On the second day, she was shifted to ICU due to increasing breathlessness.

Empiricalantibiotic therapy was started with intravenous piperacillin-tazobactam 4.5 gm stat and continued 8th hourly. Antioxidant therapy was initiated on the same day with vitamin E and C. Detoxifying agent N-Acetyl cysteine 600 mg was given orally thrice-daily from second day onwards. In view of persistence hypoxia, she was intubated and put on mechanical ventilator on the 3rd day. Midazolam and Morphine were started on same day and continued for 1 week. She was treated with oral cyclophosphamide 50 mg once-daily on

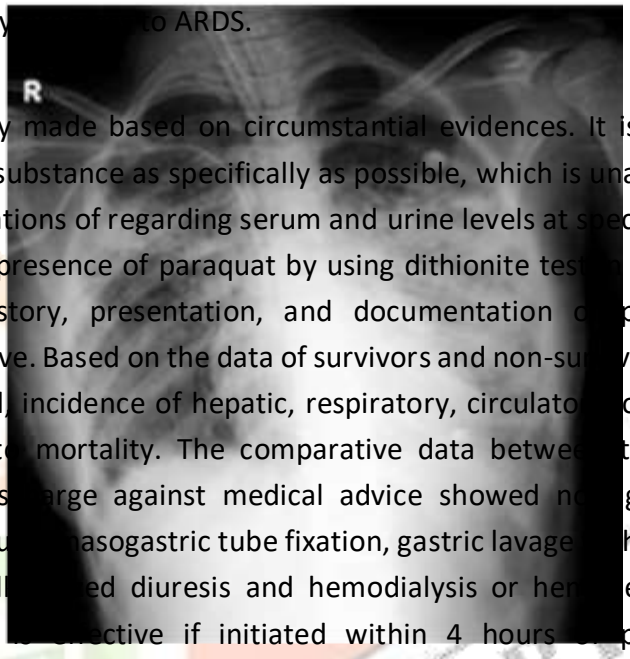


3rd day till 12th day, and steroid dexamethasone 4 mg i.v. 6 hourly was started on 6th day and stopped after one week. Subsequently, she was treated with linezolid and piperacillin-tazobactam when she developed ventilator-associated pneumonia (VAP). Piperacillin-tazobactam was changed to cefoperazone-sulbactum based on culture and sensitivity of endotracheal aspirate, which grew Klebsiella pneumoniae and Acinetobacters pecies. The serial chest X-rays done are shown in the Figures 1-3. Patient could not afford to continue the treatment; hence, she was discharged against medical advice in a critical condition.

Discussion:

Since bipyridyl salts are caustic, the gastrointestinal tract can be severely injured after ingestion of a concentrated solution.[3] Once large concentration of this poison accumulates in lungs or renal cells, it leads to generation of toxic reactive oxygen species through redox cycling, which devastate cellular

defensive system. Renal failure can result due to the direct toxicity and hemodynamic changes. Conservation of renal function is vital to reduce plasma paraquat levels and thereby reduce accumulation in lung cells.[3] Symptoms of parquat ingestion are usually dose-dependent, and intoxication can be categorized to mild, moderate, and fulminant. Mild intoxication can happen with doses ≤20 mg/kg, which usually produce minor gastrointestinal problems like transient vomiting, diarrhea, and oropharyngeal burns, but usually complete recovery is possible. Moderate intoxication can occur with doses between >20 mg/kg and <50 mg/kg of the poison. Patient may suffer lung injury, pulmonary fibrosis, acute renal failure, and in majority of cases, death occurs within 2-3 weeks. Fulminant intoxication of ≥50 mg/kg of the poison, may lead to death within 3 days, because of multiple organ failure. In patients who survive longer, f ibrotic changes in the alveoli result in gas exchange interference in the lungs and may [illegible] to ARDS.



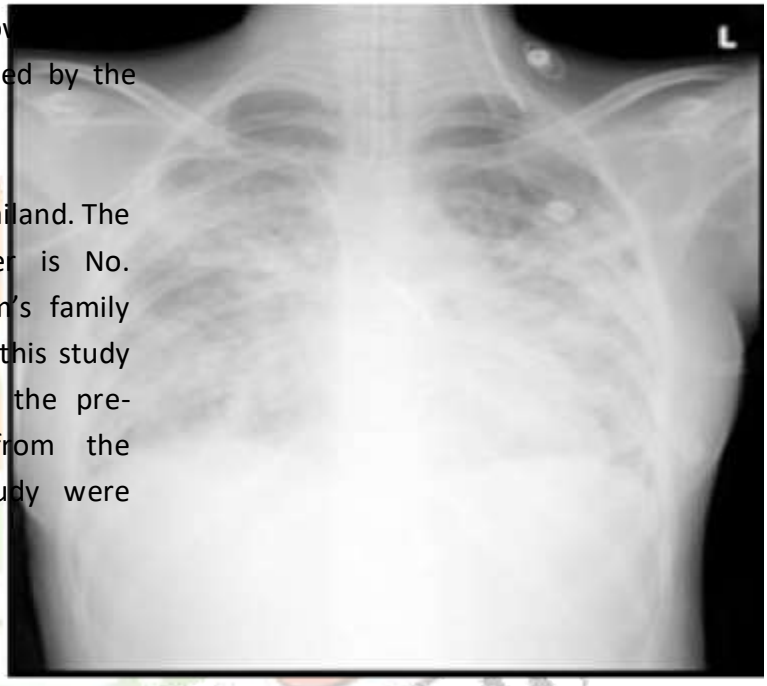
Diagnosis of paraquat poisoning is usually made based on circumstantial evidences. It is always important to identify ingested amount of substance as specifically as possible, which is unavailable in this patient. Urinary paraquat concentrations of regarding serum and urine levels at specific time points is lacking. We did not confirm the presence of paraquat by using dithionite test [illegible] urine or plasma. In our patient, the clinical history, presentation, and documentation o[illegible] paraquat consumption endorses the diagnosis positive. Based on the data of survivors and non-su[illegible]vors from the Indian study, delayed hospital referral, incidence of hepatic, respiratory, circulato[illegible]or multi-organ failure were significantly related t[illegible] mortality. The comparative data betwe[illegible]the non-survivors and the patients who took dis[illegible]arge against medical advice showed n[illegible]gnificant difference.[6] Conventional treatment inclu[illegible]nasogastric tube fixation, gastric lavage [illegible]h normal saline, charcoal-sorbitol lavage, forced al[illegible]ed diuresis and hemodialysis or her[illegible]erfusion. Hemoperfusion with activated charcoal [illegible]ective if initiated within 4 hours [illegible]paraquat intoxication. Since the patient reported after few days, benefit was not expected from this in our patient.[6] Paraquat accumulation in lung tissues exerts a destructive effect, leading to hypoxemia, requiring mechanical ventilation. Ironically, oxygen supplementation may have a deleterious effect because it increases the number of toxic radicals. Oxygen should, therefore, be given only to correct hypoxemia.[1] Subsequent management includes antibiotics for supervening infection, supporting renal function with hemodialysis or filtration. Potent analgesics such as opiates may be required to alleviate intense pain from gastrointestinal tract injury, ulceration, and inflammation. Some antioxidants like vitamins C and E have been clinically used to protect against free-radical toxicity. N-acetyl cysteine is also used as an antioxidant because of its free radial scavenging property, and it will increase intracellular glutathione levels. Thereby, it will provide protective effect on lung parenchymal cells.[3-4] In a study by Lin et al., therapeutic effect has been reported with high dose cyclophosphamide and glucocorticoid where survival is about 75%.[8] An intensive care unit study and a meta-analysis conducted by Agarwal et al., concluded that immunosuppressive therapy with cyclophosphamide and glucocorticoids have potential role in management of paraquat poisoning in moderate to severe poisoning cases.[9,10] Since there is lack of clear evidence-based therapy for paraquat intoxication, different approaches have been tried for supportive management. In the present case, our therapeutic stratagems did not improve patient's clinical condition.

Materials and Methods:

This study was a cross-sectional retrospective review carried out at the Department of Forensic Medicine in a tertiary care teaching hospital in Thailand from 1 January 2016 to 31 December 2019. Autopsies were performed on all deaths due to PQ poisoning, and the relevant viscera, serum, and urine were sent to the toxicology laboratory for chemical analysis. Toxicological analysis was performed using urine sodium dithionite test and LC MS/MS.Nospecific sample size was calculated, and all confirmed cases of PQ poisoning were included in the study. Data for this study was collected from the post-mortem records of all confirmed cases.

2.1. Ethical Approval

The study was conducted following the Declaration of Helsinki and approved by the Institutional Review Board (Human Ethics Committee), the Faculty of Medicine, Chiang Mai University, Thailand. The ethics approval reference number is No. EXEMPTION 9375/2023. The victim's family consent was not required because this study was retrospec tive and reviewed the pre-existing confidential database from the hospital. The results of this study were reported anonymously.



2.2. Case Reports and Autopsies

This study included seven fatal cases of PQ poisoning, comprising five men and two women. PQintoxication was screened using the urine sodium dithionite test and subse quently confirmed by measuring serum PQ concentration through LC-MS/MS. Autopsies were performed, and samples of cardiac blood, femoral blood, urine, brain, heart, lung, liver, spleen, kidney, adrenal gland, esophagus, stomach, and intestine were collected. Serum samples were directly subjected to LC-MS/MS procedures, while the samples of brain, heart, lung, liver, spleen, kidney, adrenal gland, esophagus, stomach, and intestine were subjected to qualitative histopathological analysis using hematoxylin and eosin (H&E) staining (Sigma-Aldrich, St. Louis, MO, USA). The histopathological examination was conducted at the Department of Pathology, Chiang Mai University. Tissue samples were initially fixed in a 10% buffered formaldehyde solution. Following fixation, the samples underwent routine histopathological processing, which involved paraffin embedding, sectioning, and staining with H&E. This standard procedure enabled the clear visualization of cellular and tissue architecture under light microscopy. The histological slides were reviewed by both a histopathologist

and a forensic doctor to confirm the cause of death. Consequently, the evaluation criteria focused on

identifying specific pathological changes in cellular or tissue structures that could assist in determining the cause of death

Table 1: Reported fatal cases and analytical methods of paraquat poisoning

Paraquat Concentration (μg/mL)				Number of Cases	Method of Detection	Reference
Blood	Urine	Gastric Content	Other Samples			
0.5–372.0	N/A	N/A	N/A	7	LC-MS/MS	Present study
0.3–2.2 (Heart) 1.0–1.6 (Peripheral)	0.1–5.3	0.6–1.7	0.2–6.1 (Bile) 0.2 (Vitreous humor)	4	HPLC-MS/MS	[27]
150.0 (Heart) 80.0 (Peripheral)	910.0	N/A	320.0 (Bile) 60.0 (Vitreous humor)	1	HPLC-MS/MS	[28]
291.5 * (2.9–1108.8)	N/A	6028.3 * (3.0–21,617.2)	200.0 (Vitreous humor)	16	LC-MS/MS	[23]
3.3 * (0.1–9.5)	3.0 * (0.1–13.5)	N/A	4.1 * (0.5–11.9) (mg/g of lung)	5	GC-ion trap MS	[19]
68.7 * (1.5–335.9)	N/A	N/A	N/A	7	HPLC (Liquid-Liquid-Extraction)	[32]
5.1	6.0	17.2	80.6 (mg/kg of kidney)	1	HPLC-DAD	[33]
30.1–636.6	N/A	N/A	N/A	24	Spectrophotometry	[34]
2.0	5.1	N/A	52.1 (μg/g of kidney)	1	HPLC	[25]
7.9 * (0.2–25.0)	192.0	N/A	41.5 * (0.3–146.0) (μg/g of liver)	5	HPLC ion-pair extraction	[35]
7.2 * (1.1–13.3)	N/A	N/A	N/A	3	Spectrophotometry	[36]

- Mean, GC-ion trap MS, Gas chromatography – ion trap mass spectrometry; HPLC-DAD, high-performance liquid chromatography with diode-array detector, N/Are presents “not available”.

2.3. Toxicological Evaluation

2.3.1. Reagents

Paraquat dichloride hydrate (PQ) was purchased from Dr . Ehren Storfer GmbH (Augsburg, Germany). Ethyl viologen dibromide (EV)was purchased from Sigma-Aldrich (St. Louis, MO,USA).Acetonitrile was purchased from J.T. Baker (Phillipsburg, NJ,USA). Ammonium for mate was purchased from LOBACHEMIEPYT. LTD. (Mumbai, Maharashtra, India). Formic acid was purchased from Fisher Scientific (Hampton, NH,USA). All chemicals were mass spectroscopy grade. 2.3.2.Preparation of Standards and Quality Control and Samples

The stock solutions of PQ and EV were prepared at a concentration of 1mg/m Lin deionized water (DW). To prepare a 100μg/mL PQ working solution, 10μL of the stock solution was diluted with 100μL of water. Six PQ calibration standards were then created by adding the working solutions to blank human serum, covering arrange from 50 to 2000ng/mL (50,100,200,500,1000,and2000). QC samples at three concentration levels (75,600,and1500ng/mL) were also prepared using the same procedure. The stock and working solutions were store dat4◦C.

2.3.3. Forensic Sample Preparation

For each case, a serum sample of 250µL was mixed with 500µL of Cold acetonitrile (−20to−10◦C) and 20µL of the internal standard (EV,5µg/mL). Next, the mixture underwent Vortexing (Vortex-Genie2 mixer, Scientific Industries, Inc., New York, NY, USA) at a speed of 2700rpm for10s, followed by centrifugation (Centrifuge5810R, Eppendorf, Hamburg, Germany) at 14,500rpm for 15min at 4◦C. Finally, 10µL of the super natant was introduced into the LC-MS/MS system[24].

2.3.4.Method Validation

Linearity and Lower Limit of Quantification (LLOQ) Linearity was determined by evaluating PQ calibration standards covering a range from 50to2000ng/ml. Six PQ calibration standards were generated by introducing working solutions into a blank human serum. To establish the calibration curve, a weighted linear least-squares regression (with a weight of 1/x) was employed, correlating the peak area ratios of PQ to EV with their corresponding concentrations. Regarding the Lack of Fit statistic, if the p-value is greater than the significance level (0.05), it suggests that the Lack of Fit is not statistically significant and the model fits well. The LLOQ was established as the lowest concentration on the calibration curve, determined through visual evaluation, at which the analyte could be reliably detected with acceptable precision and accuracy. The observed value was anticipated to fall within ±20% of the expected value [24,37]. Determining the LLOQ involved utilizing a blank serum sample and signal-to-noise ratios of 3 [24,37].

Accuracy and Precision:

The QC samples were Analyzed at three concentration levels (75, 600, and 1500 ng/mL) within the same day to assess intra-day accuracy and precision or on consecutive days to study inter-day accuracy and precision. Intra-day accuracy and precision were assessed by analyzing five replicates of QC samples at three concentration levels within a single run. To evaluate inter-day accuracy and precision, three replicates of QC samples were analyzed on four different validation days. Precision was indicated by the coefficient of variation (% CV), with the target result within ±15% for accuracy and reliability [24,38]. Accuracy was expressed as (mean concentration)/(spiked concentration) × 100%.

2.3.5. Procedure of Analysis and LC-MS/MS Instrumentation

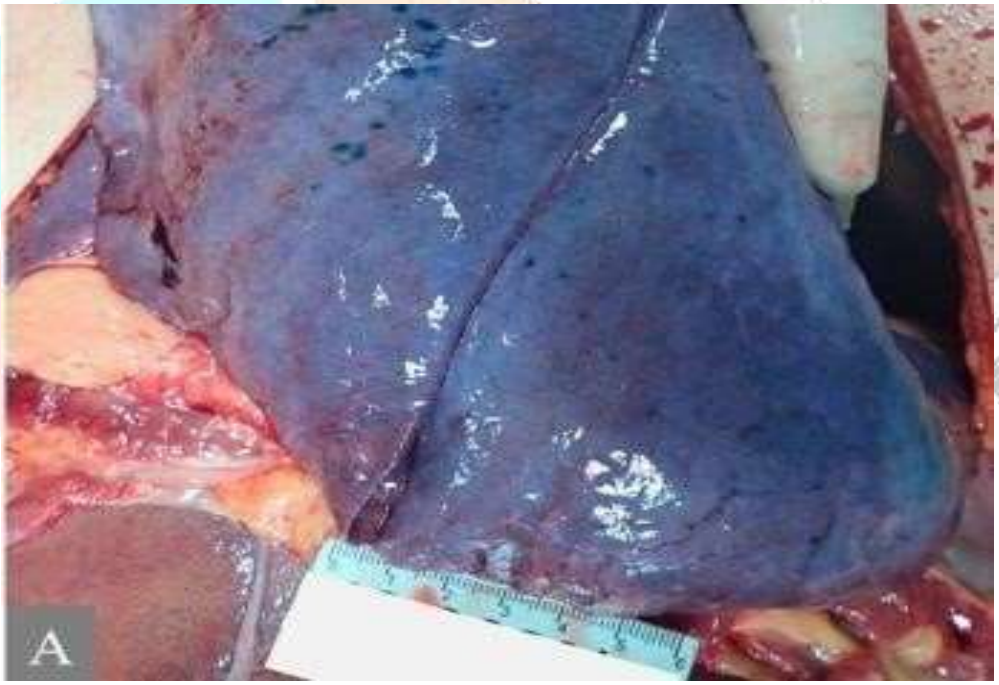
PQ in post-mortem human serum samples was assayed by modifying a previously described procedure using LC-MS/MS[24]. The LC-MS/MS system consisted of an Agilent1290 Infinity HPLC system coupled with a 6460 triple quadrupole mass spectrometer (Agi lent Technologies, Inc., Santa Clara, CA, USA). Quantitative data analysis was performed and processed using the Mass Hunter software (version B.04.01, Agilent Technologies). Chromatographic separation of PQ was performed on a Poro shell 120 HILIC-Z column (100 × 2.1mml. D., 2.7 µm particle size, Agilent Technologies, Inc., Santa Clara, CA, USA). The mobile phase was a mixture of solvent A (20 mM ammonium for mate containing 0.8% formic acid in DW at pH = 2–3) and solvent B (20 mM ammonium for mate containing 0.8% formic acid in 10% acetonitrile at pH = 3–4), with a solvent ratio of 20:80 v/v. This mixture was delivered using isocratic elution. The column temperature

was set at 30 ◦C, and the f low rate was 0.3 mL/min for a total runtime of six min. Tandem mass spectrometry was used to detect PQ and EV(used as an internal standard) using ESI in positive ion mode. The precursor ions were fragmented by varying the fragment voltage to obtain product ions. The collision energy for PQ and EV is presented in Table 2. Multiple reaction monitoring (MRM)technique was used for the selective quantification of PQ. Additionally, the second m/z to first m/z ratio must be consistent with the calibrators and within a toleranceof ±20%toconfirm the presence of PQ in the samples [24].

Table 2. Precursor ions, fragment voltage, product ions, and collision energy for paraquat and ethyl viologen.

Chemicals	Precursor Ions (*m/z*)	Fragment Voltage (V)	Product Ions (*m/z*)	Collision Energy (V)
Ethyl viologen	215.1	92	186.1	14
			158.1	34
Paraquat	187.1	92	172.1	18
			156.1	46

Result:



In this study, seven fatal cases of PQ poisoning were included, comprising five men and two women aged between 39 and 70. The most prominent autoptic and macroscopic findings were observed in the lungs, which exhibited an increased weight due to edema, congestion, hemorrh age, and fibrosis (Figure1). The liver and kidneys were also significantly altered, with liver steatosis, liver jaundice, and acute kidney injury being present. Additionally, erosion nuclears, corrosive burns, mucosal damage and gastritis were observed due to the potential irritation caused by PQ. This study also found alterations in the heart, which included petechial hemorrh age, myocardial hemorrh age, and coronary occlusion. The exact amount of PQ ingested was unknown, and four out of the seven cases resulted in immediate death. The remaining three cases were admitted to the hospital in a serious condition but unfortunately died. In Case6, the amount of ingestion was based on the patient's past medical history. A summary of the autoptic and macroscopic findings is presented in Tables 3 and 4.

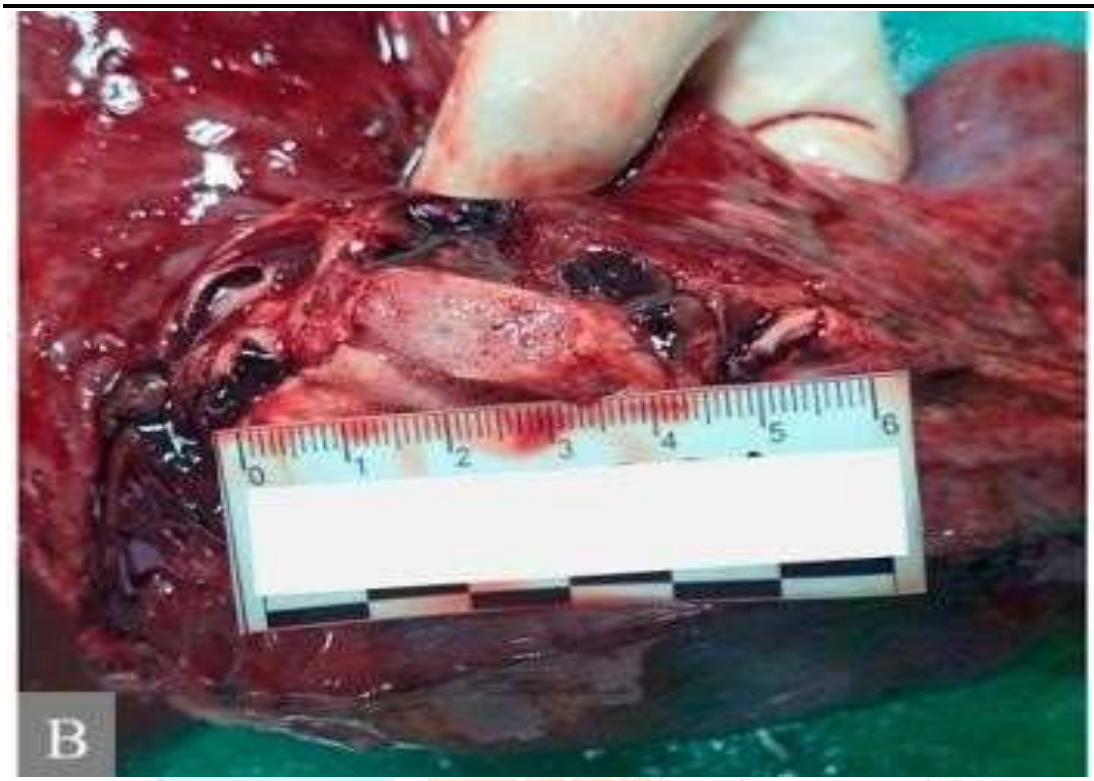


Figure1. Autoptic macroscopic pathomorphological findings of the lung.

(A)shows pulmonary congestion from Case 7.

(B) shows pulmonary edema: frothy fluid in the main bronchi from case 7.

Table 3. Demographic characteristics of fatal cases and autoptic findings.

Autopsy Case No.	Age	Gender	Weight (g)						Manner/Amount of Ingestion (mL)	Survival Time (Day)
			Brain	Heart	R/L Lung	Liver	Spleen	R/L Kidney		
1	44	M	N/A	310 (~)	740 (↑)/625 (↑)	1515 (~)	50 (~)	100 (~)/110 (~)	Suicide/N/A	N/A
2	45	M	1450 (↑)	344 (~)	710 (↑)/670 (↑)	1560 (↑)	105 (~)	145 (↑)/135 (↑)	N/A	N/A
3	39	M	1410 (~)	535 (↑)	1044 (↑)/1000 (↑)	N/A	N/A	N/A	Suicide/N/A	1
4	44	F	1381 (↑)	246 (~)	1009 (↑)/728 (↑)	1520 (↑)	88 (~)	141 (↑)/146 (↑)	N/A	2
5	70	F	1120 (↓)	295 (~)	779 (↑)/553 (↑)	1027 (↓)	120 (~)	140 (~)/115 (~)	N/A	N/A
6	54	M	978 (~)	253 (~)	270 (~)/322 (~)	1140 (~)	87 (~)	81 (~)/73 (~)	N/A/500	1
7	42	M	1356 (~)	380 (↑)	880 (↑)/773 (↑)	1683 (↑)	177 (↑)	210 (↑)/225 (↑)	N/A	N/A

Table4. Macroscopic pathomorphological findings .

Autopsy Case No.	Heart	Lung	Liver	Stomach/Intestine	Kidney
1	Subendocardial, myocardial hemorrhage	Pulmonary edema, hemorrhage	Steatosis/necrosis, jaundice	Greenish blue color (lips, nose, larynx, trachea, stomach, intestine)	Acute kidney injury
2	N/A	Pulmonary edema, greenish blue color (trachea)	N/A	Greenish blue color (stomach, intestine)	N/A

Autopsy Case No.	Heart	Lung	Liver	Stomach/Intestine	Kidney
3	Petechial hemorrhage, myocardial hemorrhage	Pulmonary congestion, hemorrhage	Steatosis	Gastritis	N/A
4	N/A	Severe pulmonary edema, intrapulmonary hemorrhage	Steatosis	Gastritis, mucosal hemorrhage, corrosive burn of esophagus	N/A
5	Congestion	Pulmonary fibrosis, edema, congestion	Jaundice	Mucosal damage	N/A
6	Petechial hemorrhage, coronary occlusion	Adhesion of all lobes, pulmonary hemorrhage	N/A	Erosion ulcer, petechial hemorrhage (esophagus, stomach), greenish-blue color (gastric content)	Acute kidney injury
7	N/A	Pulmonary edema, congestion	N/A	Gastritis	N/A

3.2. Microscopic Histopathological Findings All fatal cases exhibited pulmonary histological alterations, except for Case 2, for which lung histological data were not available due to post-mortem changes. The most notable histological findings were edema and congestion, accompanied by diffuse alve olar damage, fibrosis, petechial hemorrhage, and the presence of fibrin-platelet thrombi (Figure 2). Additionally, hepatic histological changes were observed in six out of the seven fatal cases. The most prominent histological finding was steatosis, and hepatocyte degeneration and canaliculi cholestasis were also observed (Figure 3). Cardiac histopathology was present in five out of the seven fatal cases, which included hemorrhage at the subendocardial and myocardial levels, as well as thrombosis, atherosclerosis, coronary vessel occlusion, myocardial necrosis, and infarction (Figure 4). Lesions in the esophagus and stomach were also observed, likely due to irritation. The histological findings showed mucosal hemorrhage, infarction, and inflammation (Figure 5). Partial autolysis was observed in the kidneys, along with the adrenal gland. Renal histological alterations included acute tubular necrosis and the presence of fibrin platelet thrombi in glomeruli (Figure 6). The adrenal gland exhibited focal cortical necrosis and lipid depletion of cortical cells, indicating cellular stress. Spleen congestion was found in three out of the seven fatal cases. Lesions in the brain were characterized by subarachnoid congestion, the presence of red blood cells within the subarachnoid space due to hemorrhage, and eosinophilic neurons, indicating acute neuronal injury (Figure 7). A summary of the microscopic histopathological findings is presented.

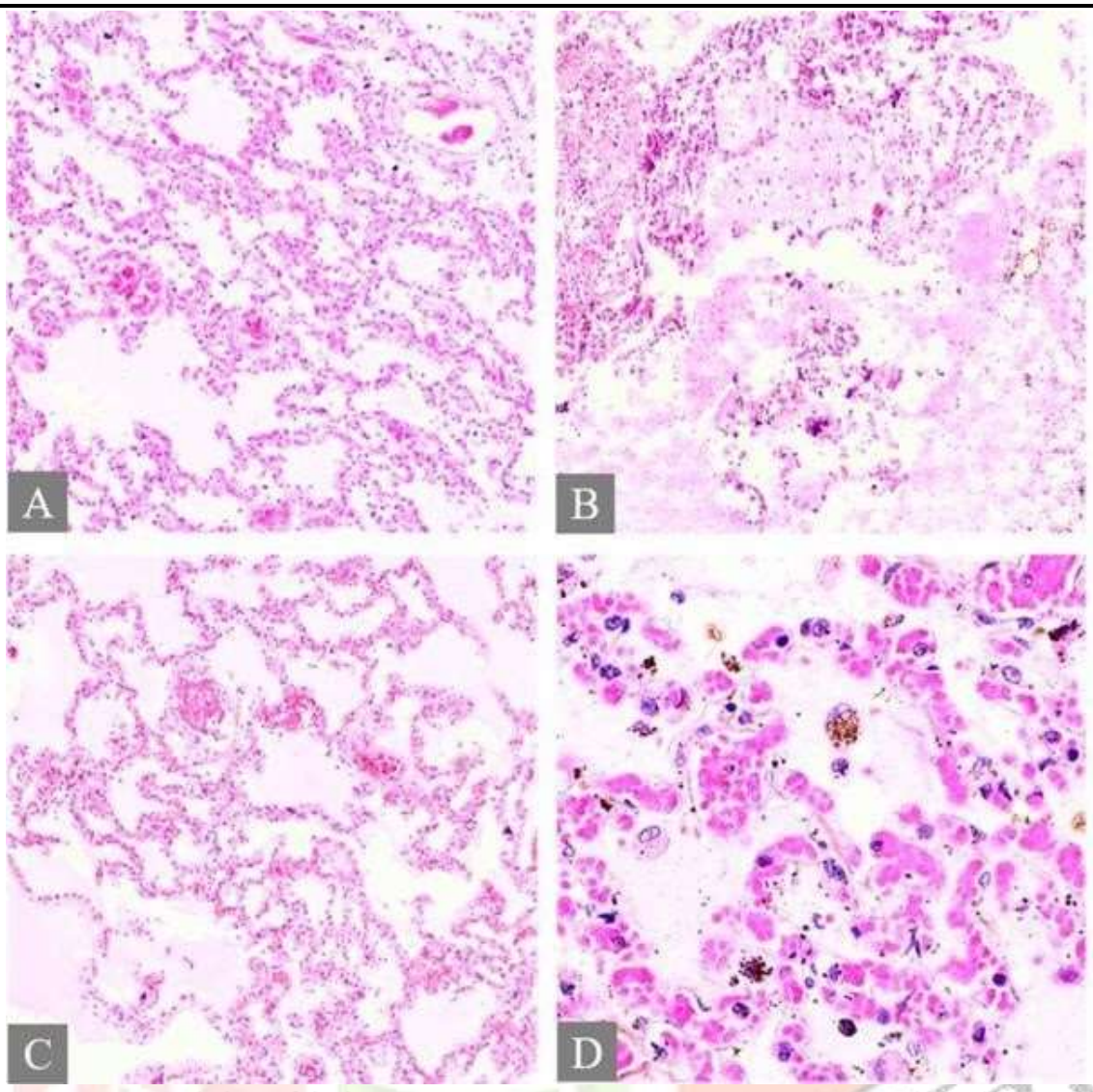


Figure 2. Histopathology of the lung.

(A) shows pulmonary congestion from Case 7 (H&E, 10×).

(B) shows diffuse alveolar damage from Case 3 (H&E, 10×).

(C) shows fibrin platelet thrombi from Case 3 (H&E, 10×).

(D) shows pulmonary hemosiderin indicating hemorrhage from Case 3 (H&E,,40×).

Conclusion:

The data on paraquat poisoning from our country is scanty. We report our experience of acute paraquat poisoning with ARDS. The unexplained combination of gastrointestinal symptoms, acute renal injury, and respiratory failure must be suspected of paraquat toxicity, even in the absence of ingestion history. Both urine and serum concentrations of samples at known time intervals post-ingestion are to be determined for assessing severity of the intoxication and to predict survival chance. If patient presents early, the therapeutic interventions with hemoperfusion and dialysis is

recommended to prevent pulmonary and multi-organ failure. We did not find any significant benefits for the cyclophosphamide-dexamethasone regimen.

References:

1. Arts J, Schuit G, Schipper A, Kleij van der B. A case report of paraquat poisoning. Eur J Hosp Pharm 2006;12:22-4.
2. Chen HW, Tseng TK, Ding LW. Intravenous paraquat poisoning. J Chin Med Assoc 2009;72:547-50. Dinis-Oliveira RJ, Duarte JA, Sánchez-Navarro A, Remião F, Bastos ML, Carvalho F. Paraquat poisonings: Mechanisms of lung toxicity, clinical features, and treatment. Crit Rev Toxicol 2008;38:13-71.
3. Dinis-Oliveira RJ, Sarmento A, Reis P, Amaro A, Remiao F, Bastos ML, et al. Acute paraquat poisoning: Report of a survival case following intake of a potential lethal dose. Pediatr Emerg Care 2006;22:537-40
4. Soloukides A, Moutzouris DA, Kassimatis T, Metaxatos G, Hadjiconstantinou V. A fatal case of paraquat poisoning following minimal dermal exposure. Ren Fail 2007;29:375-7. Sandhu JS, Dhiman A, Mahajan R,
5. Sandhu P. Outcome of paraquat poisoning - a five year study. Indian J Nephrol 2003;13:64-8.
6. Botella de Maglia J, BelenguerTarín JE. Paraquat poisoning. A study of 29 cases and evaluation of the effectiveness of the Caribbean scheme. Med Clin (Barc) 2000;115:530-3.
7. Afzali S, Gholyaf M. The Effectiveness of Combined Treatment with Methylprednisolone and Cyclophosphamide in Oral Paraquat Poisoning. Arch Iranian Med 2008;11:387-91.
8. Agarwal R, Srinivas R, Aggarwal AN, Gupta D. Experience with paraquat poisoning in a respiratory intensive care unit in North India. Singapore Med J 2006;47:1033-7. 10.
9. Agarwal R, Srinivas R, Aggarwal AN, Gupta D. Immunosuppressive therapy in lung injury due to Paraquat poisoning: A meta-analysis. Singapore Med J 2007;48:1000-5.
10. Soontornniyomkij, V.; Bunyaratvej, S. Fatal paraquat poisoning: A light microscopic study in eight autopsy cases. J. Med. Assoc. Thai 1992, 75 (Suppl. 1), 98–105.
11. Kaojarern, S.; Ongphiphadhanakul, B. Predicting outcomes in paraquat poisonings. Vet. Hum. Toxicol. 1991, 33, 115–118. [PubMed]
12. Tajai, P.; Fedeles, B.I.; Suriyo, T.; Navasumrit, P.; Kanitwithayanun, J.; Essigmann, J.M.; Satayavivad, J. An engineered cell line lacking OGG1 and MUTYHglycosylases implicates the accumulation of genomic 8-oxoguanine as the basis for paraquat mutagenicity. Free Radic. Biol. Med. 2018, 116, 64–72. [CrossRef] [PubMed]
13. Kanchan, T.; Bakkannavar, S.M.; Acharya, P.R. Paraquat Poisoning: Analysis of an Uncommon Cause of Fatal Poisoning from Manipal, South India. Toxicol. Int. 2015, 22, 30–34. [CrossRef] [PubMed]
14. Liu, H.-L.; Chen, W.-L.; Yang, M.-C.; Lin, H.-M.; Chou, C.-C.; Chang, C.-F.; Lin, T.-J.; Liu, K.-T.; Lin, Y.-R. Prediction of early mortality in patients with paraquat intoxication. J. Acute Med. 2013, 3, 6–10. [CrossRef]

15. Tajai, P.; Kornjirakasemsan, A. Predicting mortality in paraquat poisoning through clinical findings, with a focus on pulmonary and cardiovascular system disorders. J. Pharm. Policy Pract. 2023, 16, 123. [CrossRef]