International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Gross and Histopathological Changes in Lungs in Cases of Death Due to Acute Transoral Paraquat Poisoning

Sayak Sovan Dutta[1], Sandip Mukhopadhyay[2], Palas Bhattacharyya[3], Deepmalya Sengupta[4], Mukesh M[5], Suklal Mandi[6]

[1,2] Associate Professor, Department of FMT, NRS Medical College, Kolkata.
[3] Associate Professor, Department of Pathology, NRS Medical College, Kolkata.
[4] Assistant Professor, Department of FMT, NRS Medical College, Kolkata.
[5,6] Junior Resident, Department of FMT, NRS Medical College, Kolkata.

Corresponding Author:

Dr. Deepmalya Sengupta
Assistant Professor, Department of FMT, NRS Medical College, Kolkata.

Received: 26-10-2025
Accepted: 20-11-2025
Available online: 30-11-2025

ABSTRACT

Background: Paraquat, a widely used herbicide, is associated with significant human toxicity, primarily affecting the lungs. Its ingestion leads to oxidative stress, inflammation, and progressive pulmonary damage. This study aims to correlate the gross and histopathological changes in the lungs of paraquat poisoning victims with the survival period.

Methods: This descriptive, cross-sectional study was conducted over six months, involving 24 cases of transoral paraquat poisoning. Lung specimens were examined macroscopically and microscopically for congestion, edema, hemorrhage, and fibrotic changes. Histopathological analysis included diffuse alveolar damage, inflammatory infiltrates, cellular vacuolization, and fibrosis. Data were analyzed descriptively.

Results: Macroscopic findings showed pulmonary hemorrhage (66.67%), congestion (62.5%), and edema (58.33%). Fibrotic changes were observed in 45.83%, primarily in cases surviving beyond 97 hours. Microscopically, diffuse alveolar damage (91.67%) and inflammatory infiltrates (100%) were prevalent. Fibrosis increased with survival duration, absent in deaths within 24 hours but pronounced in those surviving beyond one week. The findings align with existing literature, indicating paraquat-induced oxidative stress and a temporally progressive pathology.

Conclusion: Paraquat poisoning results in significant pulmonary pathology, characterized by congestion, hemorrhage, and progressive fibrosis. The severity correlates with survival duration, underscoring the need for timely intervention. Further research is essential to explore therapeutic strategies and improve clinical outcomes.

Keywords: *Paraquat; herbicide; histopathology; pulmonary pathology; pulmonary hemorrhage, fibrosis.*

INTRODUCTION

Suicide emerged as a serious health issue in India as per Report of World Health Organization with a reported national suicide rate of 10.4 (calculated per lakh of population) [1]. 14% to 20% global suicide rate was estimated due to consuming pesticide causing death of 110,000-168,000 per year [2]. Easy availability of highly hazardous pesticides and herbicides at Home and local shops and no proper State guidelines to their sell in Asian Countries including India, contribute to a large of deaths by consuming those poisons [3] forming only the tip of the iceberg whereas unreported suicidal attempts is mostly ignored which could be a real health concern in the society [4].

In the year 2022 among the total number of 3400 Medicolegal autopsies performed at NRSMCH Morgue, Kolkata, 382 deaths were said to be due to consummation of poison, out of which 218 cases (57%) were due to consummation of

Paraquat poison as revealed from the in supplied inquest/ requisition papers for Medicolegal autopsy from Police and Toxicological Examination report from State Forensic Science Laboratory.

Paraquat, a widely used herbicide, has been a cornerstone of agricultural practices worldwide. Its effectiveness in controlling weed growth has made it indispensable to farming communities for decades. However, the growing incidence of paraquat poisoning in humans has raised significant concerns about its safety and associated health risks.

Chemically known as 1,1'-dimethyl-4,4'-bipyridinium dichloride, paraquat was first introduced in the 1960s and quickly gained popularity due to its broad-spectrum herbicidal properties. The herbicide disrupts the photosynthetic process in plants, causing their rapid death. Despite its utility in agriculture, paraquat's toxic nature has been a concern since its inception. Human exposure primarily occurs through ingestion, skin contact, or inhalation during spraying, posing health risks to farmers, agricultural workers, and residents near treated areas.

The mechanisms of paraquat toxicity are multifaceted, primarily involving the generation of reactive oxygen species (ROS) within the body. Paraquat undergoes redox cycling, producing superoxide radicals and other highly reactive molecules. These ROS cause significant damage to cellular structures, particularly in the lungs, kidneys, and liver, leading to oxidative stress and inflammation. The lungs, as the primary site of paraquat accumulation, are most severely affected. Upon inhalation or ingestion, paraquat accumulates in pulmonary tissues, initiating a cascade of events that culminates in acute respiratory distress syndrome (ARDS). The oxidative stress induced by paraquat leads to lipid peroxidation, DNA damage, and protein oxidation in lung cells. This oxidative damage compromises the alveolar epithelium and capillary endothelium, increasing vascular permeability and causing pulmonary edema. Additionally, paraquat toxicity elicits a pro-inflammatory response in the lungs, exacerbating tissue injury. Prolonged inflammation may lead to fibrosis, impairing lung function and contributing to respiratory failure [5].

Clinical manifestations of paraquat poisoning vary depending on concentration and dosage, ranging from mild gastrointestinal symptoms to severe multi-organ failure. Ingestion of even small amounts can cause nausea, vomiting, abdominal pain, and diarrhea within hours. As toxicity progresses, symptoms such as respiratory distress, pulmonary edema, hemoptysis, and renal failure may develop [6]. The rapid onset and progression of symptoms make early detection and intervention crucial for improving prognosis.

Diagnosing paraquat poisoning poses challenges due to the absence of a specific antidote, making timely identification essential for effective management [7]. The lack of a rapid and widely available diagnostic test further complicates the issue. Diagnosis often relies on clinical presentation, exposure history, and laboratory tests, such as measuring paraquat levels in blood or urine. Unfortunately, by the time symptoms appear, irreversible damage may have occurred, emphasizing the need for improved diagnostic tools.

Treatment of paraquat poisoning is complex and often unsuccessful. While supportive care, including respiratory and hemodynamic support, is critical, specific therapeutic options are limited. Activated charcoal may be used to limit further absorption, and certain medications, such as cyclophosphamide and immunosuppressive agents, have been explored to mitigate inflammatory responses. Hemodialysis and hemoperfusion may be beneficial if initiated within 12 hours of ingestion [7]. However, the prognosis for severe paraquat poisoning remains grim, with high mortality rates.

Histopathological examination reveals key oxidative stress-induced changes, including cellular swelling, vacuolization, and inflammatory infiltrates. These findings provide critical diagnostic information and insights into potential therapeutic targets for mitigating oxidative damage. Paraquat exposure significantly alters the pulmonary parenchymal architecture, with histopathological findings such as alveolar damage, interstitial fibrosis, and consolidation. The severity of these changes correlates with the dose and duration of exposure. Histopathology aids in confirming diagnoses and assessing the progression of pulmonary damage, helping clinicians determine appropriate treatments.

Recognizing specific histopathological patterns, such as diffuse alveolar damage, hyaline membrane formation, and interstitial fibrosis, is crucial for accurately diagnosing paraquat poisoning, especially when clinical presentations mimic other respiratory conditions. Microscopic examination of lung tissues also provides valuable information on the temporal progression of pulmonary toxicity. Early stages are characterized by edema, inflammation, and hemorrhage, while later stages exhibit fibrosis and irreversible damage [8]. Understanding these patterns is essential for prognosis and guiding therapeutic interventions.

Gross and microscopic examinations allow for the identification of oxidative stress-induced alterations, parenchymal damage, and inflammatory responses. This comprehensive understanding of paraquat-induced pulmonary toxicity is invaluable for clinicians, pathologists, and researchers, aiding in the development of effective therapeutic strategies and advancing the management of paraquat poisoning.

OBJECTIVES

Aim: Gross and Histopathological changes in lungs in cases of death due to transoral paraquat poisoning.
Specific Objective: To correlate the gross and histopathological changes in lungs in relation to Period of survival (duration between ingestion and death)

MATERIALS AND METHODS

a. Study Design: Descriptive, cross sectional study

b. Study Period and Sample size: period of 6 months after obtaining approval from Institutional Ethics Committee involving all cases (24) of transoral paraquat poisoning except those excluded by the exclusion criteria

c. Study area: Department of Forensic Medicine and Toxicology and Department of Pathology of a tertiary care Medical College and Hospital

d. Study Population:

i) Inclusion Criteria:
All cases involved medicolegal autopsies, either of individuals brought in dead or those who died in the hospital with a confirmed history of paraquat ingestion. This information was obtained from the inquest or requisition papers provided by the police prior to the autopsy, or from relatives of the deceased who either identified the substance by name or presented the poison container.

ii) Exclusion criteria:
a) Death with clinical diagnosis of Poisoning, but mentioned as "Unknown poison" in Medical Certificate of Cause of Death (MCCD)/police inquest/requisition and definite identification of the poison could not be made out from information furnished by the relatives of deceased and Autopsy findings.
b) Deceased with pre existing diseased condition of lungs.

e. Data and Sample collection procedure:
Cases were selected based on inclusion and exclusion criteria, following which informed consent was obtained from the next of kin or the legal claimant of the deceased. The lungs were then removed using standard autopsy techniques, washed, and weighed. Any macroscopic evidence of edema or focal hemorrhage was noted prior to dissection. After dissection, any evidence of hemorrhage or fibrotic changes was examined in different regions of the lungs. The cut lung specimens were preserved in 10% formol saline and sent to the Department of Pathology for paraffin block preparation and Hematoxylin and Eosin staining. The sections were visualized under light microscopy for cytomorphological studies.

f. Data Analysis:
Data was collected and compiled in a spreadsheet. Descriptive analysis was performed using numbers and percentages, and appropriate tables were utilized for presentation.

g. Human Subject Protection:
Approval from the Institutional Ethics Committee was obtained via Memo No. NRSMC/IEC/23/2024, dated 03.03.2024. Informed consent was secured from the next of kin or legal claimant of the deceased prior to conducting the study, and confidentiality regarding the identity of the deceased was upheld.

RESULTS

Table1: Distribution of study population based on age and sex

Age	Male	Percentage	Female	Percentage	Total	Percentage
Below 10 yrs	0	0%	1	7.15%	1	4.17%
11-20 yrs	0	0%	2	14.28%	2	8.33%
21-30 yrs	2	20%	3	21.43%	5	20.83%
31-40 yrs	4	40%	2	14.28%	6	25%
41-50 yrs	1	10%	3	21.43%	4	16.67%
51-60 yrs	3	60%	2	14.28%	5	20.83%
61-70 yrs	0	0%	1	7.15%	1	4.17%
Above 70 yrs	0	0%	0	0%	0	0%
Total	10	100%	14	100%	24	100%

The majority of cases were between 21-60 years, with females comprising a slightly higher proportion (58.33%) than males (41.67%).

Table 2: Distribution of study population based on Religion

Religion	Number of cases	Percentage
Hindu	18	75%
Muslim	6	25%
Christian	0	0%

Others	0	0%
Total	24	100%

Most cases (75%) were Hindu, followed by 25% Muslim, with no cases from other religions.

Table 3: Distribution of study population based on Marital status

Marital status	Number of cases	Percentage
Married	16	66.67%
Unmarried	8	33.33%
Others	0	0%
Total	24	100%

Two-thirds of the cases (66.67%) were married, while the remaining were unmarried.

Table 4: Distribution of study population based on duration between ingestion of poison and first hospital admission

Duration b/w ingestion & admission	Number of cases	Percentage
Below 1 hr	3	12.5%
1-6 hrs	15	62.5%
7-12 hrs	2	8.33%
12-24 hrs	3	12.5%
Above 24 hrs	1	4.17%
Total	24	100&

A majority (62.5%) of the cases reached the hospital within 1-6 hours of paraquat ingestion.

Table 5: Distribution of study population based on time interval between ingestion of poison and death

Duration b/w ingestion & death	Number of cases	Percentage
Below 24 hr	3	12.5%
24-48 hr	5	20.83%
48-72 hr	1	4.17%
73-96 hr	2	8.33%
97hr - 1 week	7	29.17%
Above 1 week	6	25%
Total	24	100%

The largest proportion of deaths (29.17%) occurred 97 hours to 1 week after ingestion, with 25% occurring beyond 1 week.

Table 6: Distribution of cases based on macroscopic appearance of oedema of lungs

Oedema	Number of cases	Percentage
Present	14	58.33%
Absent	10	41.67%
Total	24	100%

Pulmonary edema was present in 58.33% of cases in gross examination of lungs.

Table 7: Distribution of cases based on macroscopic detection of congestion of lungs

Congestion	Number of cases	Percentage
Present	15	62.5%
Absent	9	37.5%
Total	24	100%

Congestion of the lungs was detected in 62.5% of cases macroscopically.

Table 8: Distribution of cases based on macroscopic evidence of pulmonary hemorrhage

Haemorrhage	Number of cases	Percentage
Present	16	66.67%
Absent	8	33.33%
Total	24	100%

Petechial or patchy areas of Pulmonary hemorrhage was observed in 66.67% of cases in naked eye examination of lungs.

Table 9: Distribution of cases based on macroscopic evidence of fibrotic changes in lungs

Fibrotic change	Number of cases	Percentage
Present	11	45.83%
Absent	13	54.17%

Total	24	100%

Macroscopic fibrotic changes in the lungs were seen in 45.83% of cases following dissection of lungs.

Table 10: Distribution of cases showing relation between period of survival and macroscopic evidence of pulmonary fibrotic change

Duration b/w ingestion & death	Fibrosis present	Percentage	Fibrosis absent	Percentage	Total	Percentage
Below 24 hr	0	0%	3	23.08%	3	12.5%
24-48 hr	1	9.09%	4	30.77%	5	20.83%
48-72 hr	0	0%	1	7.69%	1	4.17%
73-96 hr	1	9.09%	1	7.69%	2	8.33%
97 - 1 week	4	36.36%	3	23.08%	7	29.17%
Above 1 week	5	45.46%	1	7.69%	6	25%
Total	11	100&	13	100%	24	100%

Fibrotic changes were more frequently observed in cases surviving beyond 97 hours, with 45.46% showing fibrosis above 1 week.

Table 11: Distribution of cases based on microscopic evidence of lung parenchymal congestion

Parenchymal congestion	Number of cases	Percentage
NIL	5	20.83%
(1+)	8	33.33%
(2+)	6	25%
(3+)	5	20.83%
Total	24	100%

Microscopic evidence of parenchymal congestion was seen in 79.17% of cases, with 33.33% showing mild congestion.

Table 12: Distribution of cases based on microscopic evidence of cellular swelling in lungs

Cellular swelling	Number of cases	Percentage
NIL	3	12.5%
(1+)	14	58.33%
(2+)	2	8.33%
(3+)	5	20.83%
Total	24	100%

Cellular swelling in the lungs was noted in 87.5% of cases, with mild swelling being most common (58.33%) as revealed under microscope.

Table 13: Distribution of cases based on microscopic evidence of cellular vacuolization in lung parenchyma

Vacuolization	Number of cases	Percentage
NIL	6	25%
(1+)	14	58.33%
(2+)	4	16.67%
(3+)	0	0%
Total	24	100%

Cellular vacuolization was evident in 75% of cases, primarily mild in severity (58.33%).

Table 14: Distribution of cases based on microscopic evidence of pulmonary fibrosis

Fibrosis	Number of cases	Percentage
NIL	6	25%
(1+)	11	45.83%
(2+)	5	20.83%
(3+)	2	8.33%
Total	24	100%

Pulmonary fibrosis was observed microscopically in 75% of cases, with mild fibrosis in 45.83%.

Table 15: Distribution of cases showing relation between period of survival and microscopic evidence of pulmonary fibrotic change

Duration b/w ingestion & death	Fibrosis nil	Fibrosis 1+	Fibrosis 2+	Fibrosis 3+	Total number of cases
Below 24 hr	3 (50%)	0 (0%)	0 (0%)	0 (0%)	3 (12.5%)
24-48 hr	2 (33.33%)	3 (27.27%)	0 (0%)	0 (0%)	5 (20.83%)

48-72 hr	0 (0%)	0 (0%)	1 (20%)	0 (0%)	1 (4.17%)
73-96 hr	1 (16.67%)	0 (0%)	1 (20%)	0 (0%)	2 (8.33%)
97 - 1 week	0 (0%)	5 (45.45%)	2 (40%)	0 (0%)	7 (29.17%)
Above 1 week	0 (0%)	3 (27.27%)	1 (20%)	2 (100%)	6 (25%)
Total	6 (100%)	11 (100%)	5 (100%)	2 (100%)	24 (100%)

Fibrotic changes were absent in cases dying within 24 hours but became more prominent with increasing survival time, particularly beyond 1 week.

Table 16: Distribution of cases based on microscopic evidence of cellular inflammatory infiltrates in lungs

Inflammatory infiltrates	Number of cases	Percentage
NIL	0	0%
(1+)	12	50%
(2+)	7	29.17%
(3+)	5	20.83%
Total	24	100%

Inflammatory infiltrates were present in all cases, with mild infiltration in 50%.

Table 17: Distribution of cases based on microscopic evidence of alveolar damage

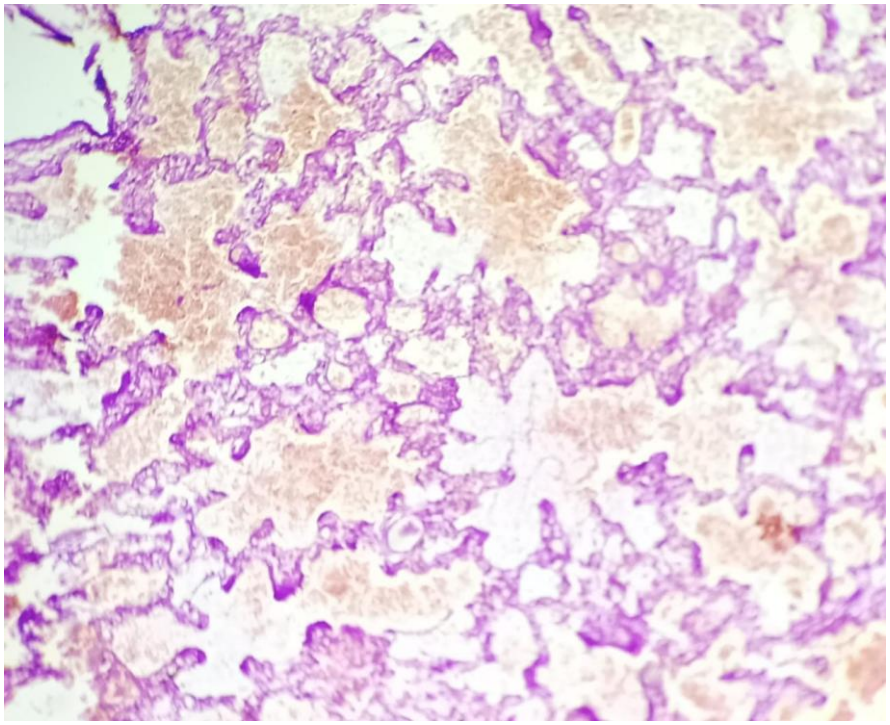
Diffuse alveolar damage	Number of cases	Percentage
NIL	2	8.33%
(1+)	8	33.33%
(2+)	9	37.5%
(3+)	5	20.83%
Total	24	100%

Diffuse alveolar damage was observed in 91.67% of cases, with moderate damage being most common (37.5%).

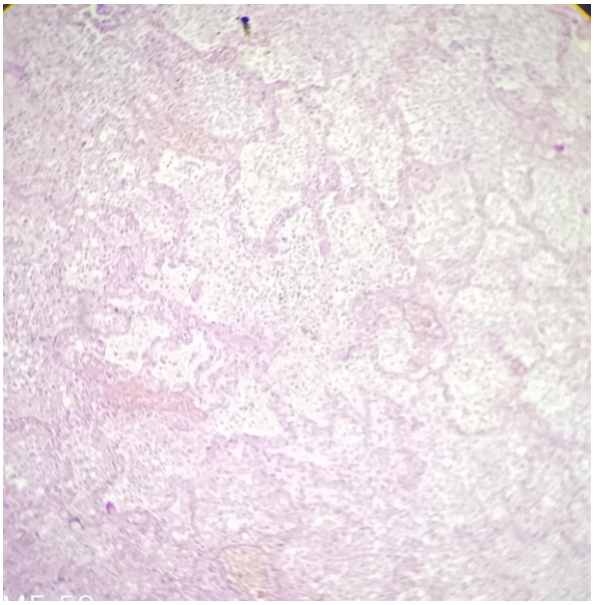
Table 18: Distribution of cases based on microscopic evidence of necrotic changes in lung parenchyma

Necrosis	Number of cases	Percentage
NIL	13	54.17%
(1+)	6	25%
(2+)	3	12.5%
(3+)	2	8.33%
Total	24	100%

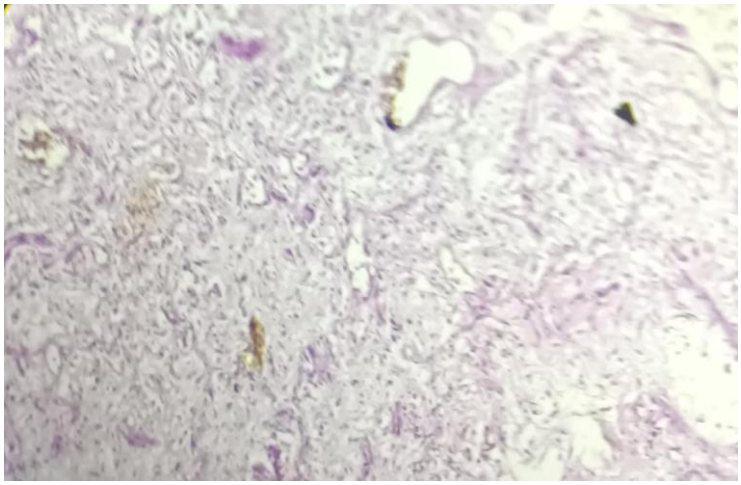
Necrotic changes were observed in 45.83% of cases, with mild necrosis in 25%.



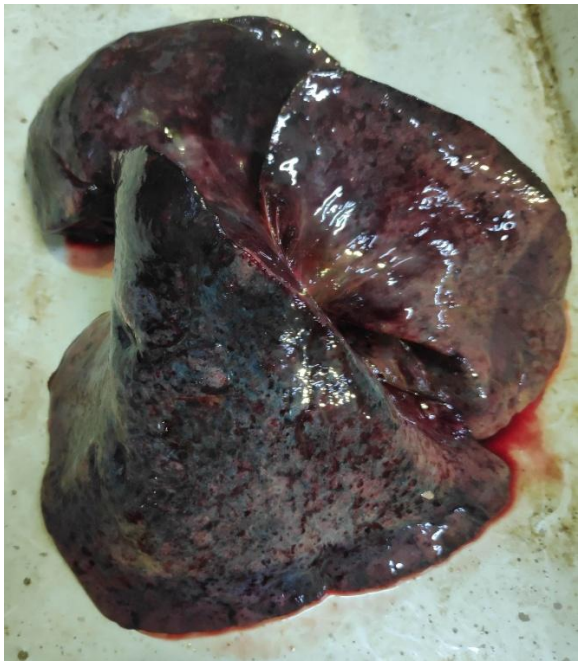
Pic 1: Congestion of lungs with presence of RBC in alveolar spaces



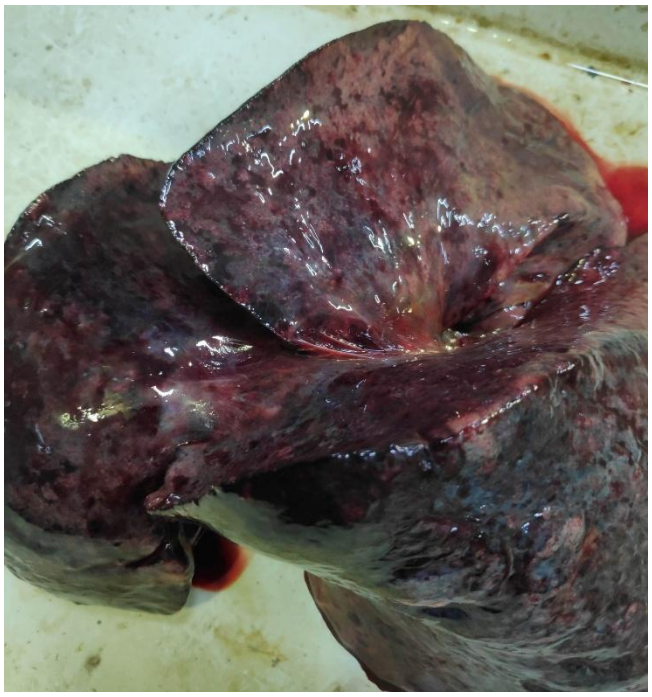
Pic 2: Necrosis and inflammation of lung



Pic 3: Fibrosis in lung parenchyma



Pic 4: Gross examination of lung shows patchy areas of hemorrhage



Pic 5: Diffuse pulmonary hemorrhage in gross examination

DISCUSSION

The study investigates the gross and histopathological changes in the lungs of individuals who succumbed to transoral paraquat poisoning, correlating these findings with the survival period. The results provide a comprehensive understanding of the progressive pulmonary pathology caused by paraquat toxicity.

The most frequent macroscopic findings in the lungs included pulmonary hemorrhage (66.67%), congestion (62.5%), and edema (58.33%). Fibrotic changes were observed in 45.83% of cases, with a higher prevalence in those surviving beyond 97 hours. Similar findings were noted in studies by Senthil Kumar, Shikha Gupta, Yogender Singh Bansal et al. entitled 'Pulmonary Histopathology in Fatal Paraquat Poisoning' who reported pulmonary hemorrhage and edema as prominent findings regardless of survival period[8]. The gradual progression to fibrosis corresponds with observations by G. Rabello and J.K. Mason, who described fibrosis and honeycombing as hallmark features of paraquat-induced pulmonary damage, linked to sustained tissue concentrations of the toxin in their research entitled 'Pulmonary histological appearances in fatal paraquat poisoning' [9].

Histopathological examination revealed diffuse alveolar damage in 91.67% of cases, inflammatory infiltrates in 100%, and varying degrees of fibrosis in 75%. Cellular swelling, vacuolization, and parenchymal congestion were also significant features, with parenchymal congestion present in 79.17% of cases. These findings align with Senthil Kumar et al., who observed hyaline membrane formation and septal congestion, particularly in cases surviving beyond four days [8].

Thrombotic microangiopathy, though not explicitly examined in this study, has been highlighted in case report published in Journal of Lung, Pulmonary and Respiratory Research entitled 'Paraquat poisoning and the lung pathology vascular injury leading to thrombotic microangiopathy is the primary pathological event' [10]. This aspect might warrant further exploration to deepen the understanding of vascular injury in this context. Similarly, emphysematous changes reported in other studies were not prominently identified, possibly due to differences in survival durations [10].

This study demonstrates that the extent and severity of pulmonary changes correlate with the duration between paraquat ingestion and death. Fibrotic changes were absent in deaths within 24 hours but became pronounced in cases surviving beyond one week (45.46% showing fibrosis). This progression supports findings by Tohru Takahashi, Yoshinobu Takahashi, Masaki Nio who described paraquat lung as a self-accelerating lesion characterized by alveolar remodeling and septal thickening over time in their study entitled 'Remodeling of the alveolar structure in the paraquat lung of humans: A morphometric study' [11].

J.G. Im et al. provided imaging evidence that corroborates these histopathological findings, reporting diffuse consolidations and parenchymal changes in the initial week, evolving into focal honeycombing after four weeks [12].

Although imaging data were not part of this study, the histological evidence complements these radiological observations.

This study highlights a pattern of acute to chronic transition in lung pathology in paraquat poisoning, with findings comparable to those reported in literature. The progressive fibrotic changes and inflammatory response illustrate the toxic dynamics of paraquat, paralleling chronic interstitial lung diseases. The histopathological similarities to paraquat-induced damage described by Rabello and Mason emphasize the relevance of temporal changes in understanding paraquat toxicity [9].

While the study provides valuable insights, certain limitations include a relatively small sample size (n=24) and the exclusion of imaging correlations. Future studies might integrate advanced imaging and molecular analyses to better characterize the disease's progression.

CONCLUSION

The study underscores the pathological progression in paraquat poisoning, with pulmonary congestion, hemorrhage, and diffuse alveolar damage being prominent initial findings. Fibrotic changes and inflammatory infiltrates intensify with prolonged survival. These results align with existing literature, reinforcing the understanding of paraquat-induced pulmonary damage as a temporally progressive and self-accelerating pathology.

Further research focusing on the role of vascular changes and potential therapeutic interventions may improve clinical management and outcomes in paraquat poisoning cases.

Conflict of interest: none declared
Source of funding: nil

REFERENCES

1. World Health Organization. Suicide - India. [Internet]. Available from: https://www.who.int/news-room/fact-sheets/detail/suicide
2. Mew EJ, Padmanathan P, Konradsen F, Eddleston M, Sen CS, Phillips MR, et al. The global burden of fatal self-poisoning with pesticides 2006–15: Systematic review. J Affect Disord. 2017;219:93–104.
3. Bose A, Sandal Sejbaek C, Suganthy P, Raghava V, Alex R, Muliyil J, et al. Self-harm and self-poisoning in southern India: choice of poisoning agents and treatment. Trop Med Int Health. 2009;14:761–5.
4. Carroll R, Metcalfe C, Gunnell D. Hospital presenting self-harm and risk of fatal and non-fatal repetition: systematic review and meta-analysis. PLoS One. 2014;9:e89944.
5. Aggrawal A. Textbook of Forensic Medicine and Toxicology. 2nd ed. Sirmour: Avichal Publishing Company; 2021. p. 610.
6. Mukherjee JB. Forensic Medicine and Toxicology. 5th ed. Kolkata: Academic Publishers; 2018. p. 866.
7. Reddy KSN, Murty OP. The Essentials of Forensic Medicine and Toxicology. 35th ed. New Delhi: Jaypee Brothers Medical Publishers; 2022. p. 399.
8. Kumar S, Gupta S, Bansal YS, Bal A, Rastogi P, Muthu V, et al. Pulmonary histopathology in fatal paraquat poisoning. Autops Case Rep. 2021;11:e2021342.
9. Rebello G, Mason JK. Pulmonary histological appearances in fatal paraquat poisoning. Histopathology. 1978;2(1):53–66.
10. Paraquat poisoning and the lung pathology vascular injury leading to thrombotic microangiopathy is the primary pathological event [Internet]. MedCrave. Available from: https://medcraveonline.com/JLPRR/paraquat-poisoning-and-the-lung-pathology-vascular-injury-leading-to-thrombotic-microangiopathy-is-the-primary-pathological-event.html
11. Takahashi T, Takahashi Y, Nio M. Remodeling of the alveolar structure in the paraquat lung of humans: A morphometric study. Hum Pathol. 1994;25(7):702–8.
12. Im JG, Lee KS, Han MC, Kim SJ, Kim IO. Paraquat poisoning: findings on chest radiography and CT in 42 patients. AJR Am J Roentgenol. 1991;157(4):697–701.