International Journal of Contemporary Pediatrics
Thomas TV et al. Int J Contemp Pediatr. 2026 Oct;13(10):2157-2161
https://www.ijpediatrics.com
pISSN 2349-3283 | eISSN 2349-3291

DOI: https://dx.doi.org/10.18203/2349-3291.ijcp20263328

Case Report

Paediatric paraquat poisoning – two case reports from a quaternary care centre

Thomas Vechukunnel Thomas, Ranjit B. Joseph*, Aarthi Ramesh

Department of Child and Adolescent Medicine, Aster, Medcity, Kochi, Kerala, India

Received: 13 August 2026
Accepted: 10 September 2026

***Correspondence:**
Dr. Ranjit B. Joseph,
E-mail: ranjitbaby@gmail.com

ABSTRACT

Paraquat (PQ), or N, N′-dimethyl-4,4′-bipyridinium dichloride, is a widely used herbicide. It is highly toxic and enters the human body mainly by swallowing or through damaged skin but may also be inhaled. Due to its properties being corrosive, it can easily damage the skin, thereby making it easy to absorb. Paraquat poisoning has no known cure and is still a serious toxicological emergency with significant death rates. When paraquat enters into the body, it causes redox cycling and generation of reactive oxygen species (ROS). Once inside the cells, paraquat accepts an electron from nicotinamide adenine dinucleotide phosphate (NADPH) forming the paraquat radical which later becomes a superoxide radical by donating its electron to molecular oxygen. During this process, paraquat is regenerated, thereby continuing the vicious cycle. The mechanism of death is usually respiratory failure and may occur within a few days after poisoning or as long as a month later. The mainstay in the management of paraquat poisoning is supportive care with primary importance given to preventing absorption and promoting the excretion of the toxic compounds all whilst mitigating organ damage. This case series of a 14-year-old and a 17-year-old with paraquat ingestion examines the classic clinical presentation, management challenges and high mortality of paraquat toxicity.

Keywords: Paraquat, Poisoning, Adolescent, Multi-organ failure, Pulmonary fibrosis, Extracorporeal membrane oxygenation support

INTRODUCTION

Paraquat (N, N′-dimethyl-4, 4′-bipyridinium dichloride) is a highly effective, non-selective herbicide that has become one of the most widely used agricultural chemicals globally, particularly in developing countries where it remains readily available and affordable.

The clinical significance of paraquat poisoning lies in its exceptionally high mortality rate, often exceeding 70-90% in cases involving significant ingestion, and the absence of any specific antidote.[1] The pathophysiology of paraquat toxicity involves complex molecular mechanisms including the disruption of cellular energy metabolism, induction of apoptosis through dysregulation of pro-apoptotic (Bax) and anti-apoptotic (Bcl-2) genes and finally ability to generate reactive oxygen species (ROS) through redox cycling.[2] These processes culminate in severe damage to the respiratory system, kidneys, liver and other vital organs with the severity of toxicity directly correlating with the amount ingested and the time elapsed before medical intervention.[3] The lungs serve as the primary target organ due to their high oxygen tension and active uptake of paraquat through polyamine transport systems ultimately resulting in progressive pulmonary fibrosis that is often the cause of death.

Clinical manifestations of paraquat poisoning typically progress through distinct phases beginning with gastrointestinal symptoms including nausea, vomiting and oral ulceration followed by the development of acute kidney injury and hepatotoxicity. The most devastating complication is the development of acute respiratory distress syndrome (ARDS) and progressive pulmonary fibrosis which can occur days to weeks after initial exposure and often proves fatal despite intensive

supportive care.[4] The management of paraquat poisoning remains largely supportive focusing on limiting absorption through gastric decontamination, enhancing elimination via hemoperfusion or haemodialysis and providing aggressive supportive care for multi-organ dysfunction.

Pediatric cases of paraquat poisoning are particularly tragic as children may be exposed through accidental ingestion or in some cases, intentional self-harm. The smaller body mass in pediatric patients can result in higher mg/kg doses even with smaller absolute amounts ingested potentially leading to more severe toxicity.[5] Furthermore, the developing organ systems in children may be more susceptible to the oxidative damage caused by paraquat making early recognition and aggressive management crucial.

CASE REPORT

Case 1

A 16-year-old girl presented to our hospital with complaints of multiple episodes of vomiting, loose stools and abdominal pain following food intake from outside. She was treated as acute gastroenteritis with intravenous (IV) fluids and discharged. The following day, in view of persistence of symptoms, she was admitted. On day 2 of admission, she developed ulceration, swallowing difficulty and drooling of saliva. In view of these symptoms, she was started on iv antibiotics. On day 3, she had two episodes of haematemesis and developed jaundice. Liver function tests (LFT) and renal function tests (RFT) done showed derangement. Results were suggestive of acute kidney injury and haemodialysis was initiated. She developed breathing difficulty on day 4 and hence was initiated on 02 support. Chest X-ray taken showed bilateral homogenous opacity with right sided pneumothorax and pneumomediastinum. Ultrasonography (USG) thorax done showed consolidation and pneumothorax. In view of these findings, intercostal drainage (ICD) tube was inserted over the right side. She developed severe respiratory distress following which she was started on BiPap support. Chest X-rays and renal tests taken on the following days showed worsening of lung fields (white out) and kidney functions. Urine toxicology was found to be weakly positive for chlorpyriphos along with low choline esterase levels (680). Correlation of symptoms was along the lines of paraquat poisoning. Upon probing, child confessed to ingestion of paraquat. She had persistent hypoxia and worsening of distress. Hence, she was intubated.

Despite adequate ventilator settings, child had persistent hypoxia and worsening hypercarbia and she was initiated on extracorporeal membrane oxygenation (ECMO) on day 6 of illness. She was on ECMO support for 10 days. However, she succumbed on day 11 to the effects of the toxin.

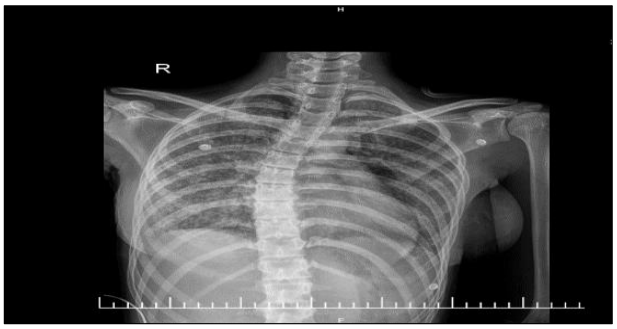

Figure 1: Day 4 of illness.

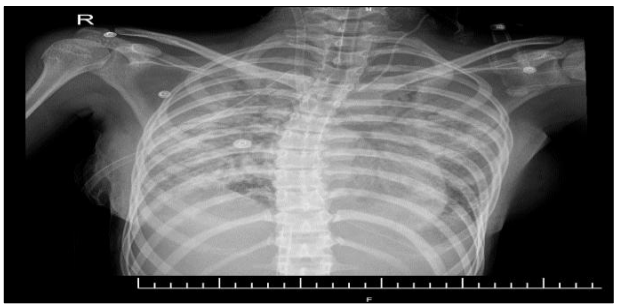

Figure 2: Day 5 of illness.

Case 2

14-year-old female child presented to our emergency department with alleged history of ingestion of paraquat. She presented with multiple episodes of vomiting and was admitted to pediatric intensive care unit (PICU). Oral cavity examination done showed erythematous lesions with mildly edematous and cracked lips. Gastric lavage was done within 1 hour and hemoperfusion with charcoal filters was initiated.

She was started on iv N-acetylcysteine dexamethasone, vitamin C and other supportive measures. On day 2 admission, renal function test showed elevated creatinine (1.26). Hence, hemodialysis was started. Gastric aspirate, blood and urine samples send for toxicology was found to be positive for paraquat. LFT done on day 3 of admission was found to be elevated. Nearing day 6 of admission, she was found to have respiratory distress with saturation drop and hence was commenced on (bilevel positive airway pressure) BiPaP ventilation. Pigtail catheters were inserted in view of spontaneous pneumothorax that developed. Despite insertion of pigtails, child had worsening pneumothorax and catheter repositioning was done. However, she had deteriorating lung functions and was intubated and ventilated. She was started on total parenteral nutrition (TPN) in view of dysphagia and oral ulcers. Proning was initiated in view of refractory hypoxia.

Despite all these measures, she continued to be hypoxic and succumbed on day 10 of ingestion.

Table 1: Laboratory values for hepatic function, renal function, and serum ammonia concentrations during the course of hospitalization.

Lab parameters	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Urea (mg/dl)	110	188	71	160	188	187
Creatinine (mg/dl)	4.74	6.55	1.82	2.93	3.14	2.96
ALT (alanine aminotransferase) (U/l)	170	228	274	197	152	118
AST (aspartate aminotransferase) (U/l)	126	261	221	126	93	79
Ammonia (µg/dl)						

*mg/dl-milligrams per decilitre; *U/l-units per litre; *µg/dl-micrograms per decilitre

Table 2: Laboratory values of hepatic function, renal function, and serum ammonia concentrations during the course of hospitalization.

Lab parameters	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6
Urea (mg/dl)	23.1	32.1	68.9	139.2	171.3	193.7
Creatinine (mg/dl)	0.93	1.26	3.21	6.50	7.22	6.67
ALT (alanine aminotransferase) (U/l)	8.8	10.4	132.7	792	777	414
AST (aspartate aminotransferase) (U/l)	19.1	24.5	256.9	241.9	161	174.7
Ammonia (µg/dl)	51.8					113.8

*mg/dl-milligrams per decilitre; *U/l-units per litre; *µg/dl-micrograms per decilitre

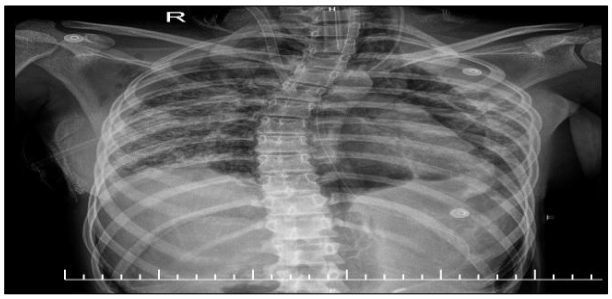

Figure 3: Day 9 of illness.

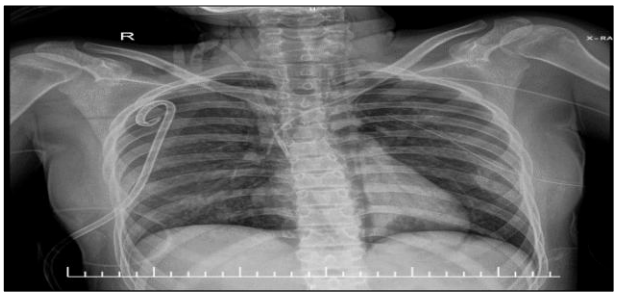

Figure 6: Day 4 of illness-s/p pigtail insertion.

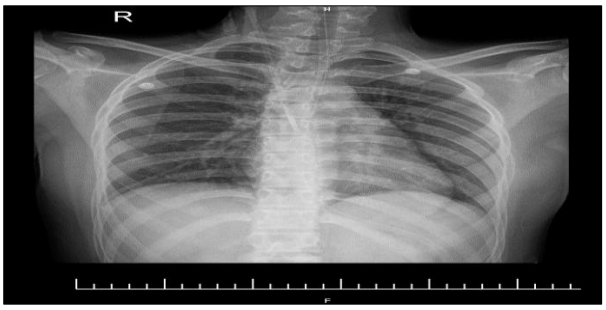

Figure 4: On the day of admission.

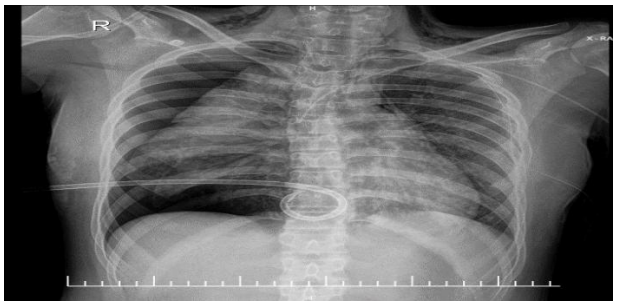

Figure 7: Day 6 of illness.

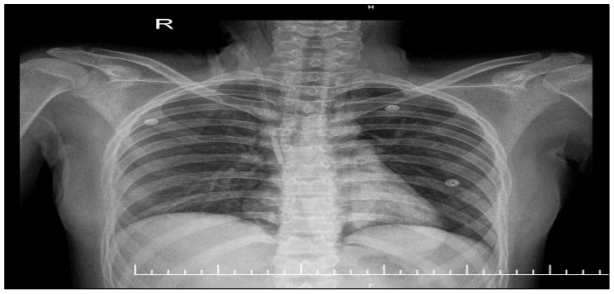

Figure 5: Day 3 of illness.

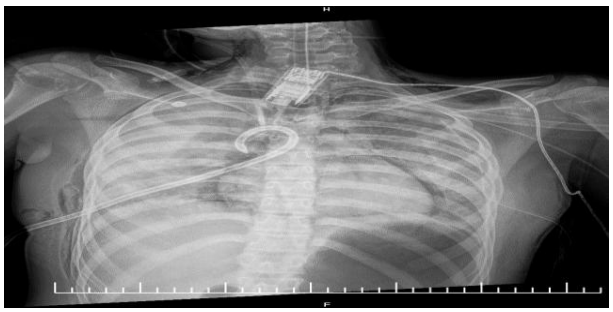

Figure 8: Day 10 of illness.

DISCUSSION

Paraquat is one of the most commonly used herbicides worldwide especially in India. It is usually sold in the form of a concentrated liquid of dichloride salt. Due to its affordability, availability as well as unavailability of antidote, it is deemed a very notorious substance and one of the most common agents responsible for herbicide poisoning. The toxic mechanism of paraquat poisoning includes cell apoptosis by the up-regulation of the proapoptotic gene (Bax) and down regulation of the antiapoptotic gene (Bcl-2). It also leads to the formation of ROS which combine with unsaturated lipids causing cell lysis. Paraquat is most commonly available as a liquid form usually 24% paraquat dichloride.[5] Ingestion of high doses of paraquat (≥60 ml) is associated with a survival rate below 1%, often leading to pulmonary complications, organ failure, renal and hepatic toxicity finally leading to death due to multi organ failure. Lethal dose of paraquat in humans is considered to be around 20-40 mg/kg of total body weight.[6] Studies showed that paraquat ingestion is predominantly seen in males.

Route of toxicity is mainly through oral with other modes of entry like inhalation and cutaneous absorption as well.

Paraquat toxicity usually produces local as well as systemic effects. Clinical manifestations of paraquat poisoning are usually classified into 3 groups depending on the amount of paraquat ingested which is as follows: mild poisoning (20 mg/kg) in which the patients generally have minor gastrointestinal symptoms along with some amount of renal damage; severe poisoning (20-40 mg/kg) in which the patients develop acute kidney injury, acute lung injury and progress into pulmonary fibrosis, symptoms usually seen after nearly 2 weeks of ingestion; and fulminant poisoning (40 mg/kg) in which the patients develop multiple organ failure, considered as a lethal dose usually resulting in deaths within 2 hours to a maximum of a week, death usually caused due to multi organ failure and sometimes cardiogenic shock.[7]

In the above case series, both patients had predominant gastrointestinal symptoms at presentation. However, along the course of illness, respiratory symptoms were markedly seen. Other common symptoms reported include pulmonary edema and fibrosis which can develop rapidly leading to respiratory failure and death. Moreover, hepatitis, abdominal pain, renal failure and also gastrointestinal tract bleeding were also seen in majority of paediatric patients who had ingested paraquat. PQ poisoning induces lung fibrosis through the activation of several signalling pathways including NF-κB, NLRP3 inflammasome, TLRs, MAPKs, AMPK, Rho/ROCK, PI3K/Akt/mTOR, TGF-β/Smad, and Wnt/β-catenin. Additionally, the Keap1/Nrf2 pathway and the Hippo-YAP/TAZ pathway are also implicated in PQ-induced lung fibrosis.[8] Chest X-rays may reveal diffuse consolidation, pneumomediastinum and pneumothorax as can be seen in the following X-rays of our patients.

Liver injury is usually caused due to activation of PI3K-AKT pathway and Bcl-2/Bax pathway which affects cell proliferation and regulates apoptosis respectively. Additionally, NF-κB, MAPK and Nrf2/Keap1 pathways are also implicated in paraquat-induced liver damage. Around 46.5% of patients who ingested PQ suffered from liver injury. Moreover, it is seen that majority of victims of PQ ingestion show gradual or sudden worsening in renal function test.[9] PQ builds up in the kidneys' proximal tubular epithelial cells where it produces ROS and goes through redox cycling. Cellular damage brought on by this oxidative stress includes endoplasmic reticulum degranulation and mitochondrial damage. Nucleotide binding domain, death associated protein kinase and other proteins have been said to be associated with acute kidney injury (AKI) as well. Monitoring creatinine, blood urea nitrogen and keeping track of urine output can be useful indicators of ongoing AKI.[10,11]

CONCLUSION

From various studies, it has been demonstrated that the dose, time lapse from the time of ingestion to that of gastric lavage and worsening of RFT were the various risk factors that would indicate the prognosis of PQ poisoning. Till date, no specific antidote has been found and patients at risk of PQ poisoning are usually given supportive treatment such as steroid pulse therapy and cyclophosphamide to prevent pulmonary fibrosis. Haemodialysis is also indicated to lower down concentration of PQ in the circulation. In general, poor prognosis is seen in patients who have ingested PQ.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Zhang X, Xu X, Li S, Li L, Zhang J, Wang R. A Synthetic Receptor as a Specific Antidote for Paraquat Poisoning. Theranostics. 2019;9(3):633-45.
2. Gawarammana IB, Buckley NA. Medical management of paraquat ingestion. Br J Clin Pharmacol. 2011;72(5):745-57.
3. Saravu K, Sekhar S, Pai A, Barkur AS, Rajesh V, Earla JR. Paraquat - A deadly poison: Report of a case and review. Indian J Crit Care Med. 2013;17(3):182-4.
4. Lazulfa I, Kusumastuti NP, Azis AL, Dharmawati I, Setyaningtyas A, Lestari DP. Deadly paraquat poisoning in a 7-years-old child: A case report. J Med Pharm Chem Res. 2024;6(7):953-8.
5. Jang YJ, Won JH, Back MJ, Fu Z, Jang JM, Ha HC, et al. Paraquat Induces Apoptosis through a Mitochondria-Dependent Pathway in RAW264.7 Cells. Biomol Ther (Seoul). 2015;23(5):407-13.
6. Sukumar CA, Shanbhag V, Shastry AB. Paraquat: The Poison Potion. Indian J Crit Care Med. 2019;23(4):S263-6.

7. Liu X, Yang H, Liu Z. Signaling pathways involved in paraquat-induced pulmonary toxicity: Molecular mechanisms and potential therapeutic drugs. Int Immunopharmacol. 2022;113(A):109301.
8. Wang XL, Li L, Meng X. Interplay between the Redox System and Renal Tubular Transport. Antioxidants (Basel). 2024;13(10):1156.
9. Barma A, Poudel A, Rawal G, Karn M, Unika KC, Ojha L, et al. Fatal paraquat poisoning: a case report and literature review on rapid deterioration and therapeutic challenges. Ann Med Surg. 2025;87(4):2421-5.
10. Narendra S, Vinaykumar S. Paraquat Poisoning: A Case Series in South India. Int J Sci Res. 2015;4(1):561-4.
11. 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(1):123.

Cite this article as: Thomas TV, Joseph RB, Ramesh A. Paediatric paraquat poisoning – two case reports from a quaternary care centre. Int J Contemp Pediatr 2026;13:2157-61.