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PARAQUAT POISONING: A CASE REPORT

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ABSTRACT

Paraquat (1, 1' dimethyl 4, 4' dipyridylium) is a highly toxic chemical herbicide or week killer. It is one of the most common herbicides used today, but it can cause fatal poisoning when ingested or inhaled. It is often difficult to diagnose in the face of nonspecific clinical features. Although it is freely available Herbicide, few cases have been reported from India. Ingestion of toxic doses of Paraquat has serious complications on the lungs, gastrointestinal tract, kidney, liver, and other organs. Due to its inherent toxicity and the lack of a specific antidote, it has a high case fatality rate. Despite being restricted to commercially licensed users in India, it is a common herbicide causing both intentional and accidental poisoning. However, the prognosis is poor for acute poisoning or high doses of Paraquat. Since, there is currently no effective treatment for Paraquat poisoning, preventative efforts must be prioritized. We have reported a case of fatal ParaquatPoisoning at C.U.Shah Medical College and Hospital, Surendranagar. This case report details the clinical presentation, management strategies, and outcomes of a patient with Paraquat poisoning. Paraquat poisoning remains a significant clinical challenge with high mortality rates. This case report emphasizes the importance of early recognition, aggressive decontamination, and advanced supportive care in managing Paraquat toxicity. Further research is imperative to explore novel treatment strategies and improve outcomes in cases of severe Paraquat poisoning.

KEYWORDS: Paraquat poisoning, chemical herbicide, acute lung injury, steroids, intentional ingestion, Surendranagar.

INTRODUCTION

Paraquat (1, 1′-dimethyl-4, 4′-bipyridinium) is a fast-acting Contact herbicide that is widely used in agriculture and horticulture industries throughout the world.[1,4] Its herbicidal Properties were discovered in 1858 and it became available for Commercial use in 1962.[5] Initially, it was used to kill marijuanaWeeds in the United States and Mexico, later it became popular world wide as it is a cheap and an effective herbicide.[6] It is currently the world's second most popular herbicide, with Sales in over 100 countries.[7]

Paraquat is extremely toxic for humans as well as animals. Ingestion (intentional or accidental) is the most common route of poisoning.[8] Additionally, poisoning via other routes such as dermal or mucus contact.[9,10] injection.[11] and inhalation.[12] have also been reported. Dermal exposure, particularly in Individuals with pre-existing skin lesions, has been documented to lead to severe Paraquat poisoning. Inhalation of sprayed Paraquat solution typically causes local irritation with minimal systemic absorption.[15] Upon ingestion, approximately 20% Of Paraquat is absorbed by the gastrointestinal tract, with higher absorption rates noted in the presence of ulcerated mucosa or an empty stomach.[16] Paraquat undergoes minimal metabolic processing in the body, with over 90% excreted unchanged by the kidneys. The estimated lethal dose in

an adult person is around 30 mg/kg.[13] or 3–6 g of Paraquat ion.[8] which is similarto 10 to 20 ml of a 20% solution.

At the tissue level, Paraquat undergoes reduction to Paraquat radicals in the presence of reduced nicotinamide adenine dinucleotide phosphate (NADPH).[17] Subsequently, these Paraquat radicals interact with oxygen molecules, leading to the production of superoxide anion (O2 –).[18]

Excessive levels of superoxide anion facilitate the formation ofhydroxyl free radicals (OH–), which have the potentialto induce cellular damage through processes such as lipid per oxidation and inhibition of crucial cellular enzymes.[18] This mechanism elucidates why the lungs are particularly vulnerable to Paraquat poisoning, as they exhibit high tissue concentrations resulting From active uptake mechanisms and abundant oxygen availability, facilitating the formation of reactive oxygen radicals.[18,19]

Clinical symptoms due to Paraquat poisoning are mostly due to the formation of intracellular reactive oxygen species, whichCause cellular damage through lipid per oxidation, nuclear factor kappa B activation, mitochondrial damage, and apoptosis.[14] Paraquat is readily taken up into lung tissue against a concentration gradient, promoting pneumonitis and lung fibrosis.[14] Additionally, it affects multiple organs including heart, kidneys, liver, adrenal glands, central nervous system, muscles and spleen causing multiple organ failure.[14] The case fatality rate is extremelyhigh, ranging from 50% to 90%, but in situations of purposeful self-poisoning using concentrated formulations, the mortality rate reaches 100%.[14] Paraquat's high case fatality rate is owing to its inherent toxicity as well as due to the lack of an effective antidote. There are no universally-acknowledged standards for treating Paraquat poisoning. Treatment varies from supportive care alone to various combinations of immune-modulation, antioxidant therapy, hemoperfusion and hemodialysis.[14]

Between 1985 and 1990, approximately 340,000 cases of agricultural and horticultural poisoning were reported in the UnitedStates, resulting in 97 deaths. Although Paraquat poisoning comprised only 0.34% of these cases, it had the highest mortality rate, responsible for 13%of all fatal cases.[20,21] In Indian scenario, at a tertiary care institute, Household and agricultural poisons together make up the majority (approximately52%) of the substances found in suspected poisoning cases.[22]

CASE REPORT

A **24**-year-old **female** patient was brought to the medical emergency department of C U Shah Medical College and Hospital, Surendranagar on **18/04/2025** at 11:56 a.m. There was alleged history of suicidal attempt in which an unknown quantity of a liquid form of poison suspected to be paraquat was consumed by her at her residence before one day. The patient's family reported finding an empty container of a commonly available herbicide nearby.

Upon arrival, the patient was conscious and oriented. She complained of throat pain and burning in oral cavity since last 24 hours. There was no history of pain in abdomen, vomiting, palpitation, perspiration, and chest pain, shortness of breath, seizure, hematemesis, malena, decrease urine output or cough.

On examination her vitals were found to be normal with heart rate of 70 per minute, BP was 110/70 mm of hg and respiratory rate was 22. Her temperature was raised to 101.4°F. Cardiovascular system was normal. There was no difficulty in breathing. No added sounds were present on auscultation of respiratory system. Oxygen saturation (SpO_2) was observed as 99% and was continuously monitored. Pupils were bilaterally equal and reactive to light. She was admitted in ICU as a suspect case of paraquat poisoning.

On investigation at the time of admission, her in the complete blood count tests, her White blood cell count was abnormal (13,200 cells/cu.mm), her renal and liver function tests were normal, her electrolytes show decrease level of Na and Chloride (Na 135.4 mmol/L and Chloride 97.1 mmol/L). Her urine examination shows Albumin + + + and Ketone + + +**,** her urine pregnancy test was negative. Bed side X ray chest and ECG was normal on day 1.

Patient was initially treated with inj. MPS 40mg in NS 100cc IV 8 hourly, Inj Lactaguard (Cefoperazone + Sulbactum) 3 gm IV stat followed by 1.5 gm 8 hourly, Inj Pantaprazole 40 mg And Inj ondansetron 4 mg IV 12 hourly, Inj Metrogyl 100 ml IV 8 hourly and Inj DNS 500ml + Inj ipvit iv 80 ml/hour.

On the day of **admission at 7 p.m.** patient suddenly become unconscious. She was unable give response to deep painful stimuli and unable to follow verbal command. She develops difficulty in breathing. Her vitals were pulse 126/minute, BP 96/56 mm of hg, Spo2 74%. patient's relatives were explained regarding patient condition and urgent requirement of intubation. After taking positive consent of patient's relatives, she was intubated by ET Tube no.7 and mouth gaze placed. post intubation vitals were pulse 116/minute, BP 96/66 mm of hg and Spo2 98 % with PRVC (Pressure regulated volume control).

On the **Day 2 (19/4/25)** patent's vitals were, Temp. Normal, Pulse: 88/minute, BP: 102/70 mm of hg, RS: BLAE (+), CVS: S1S2 (+), Pupils: BERL, Ptosis (-nt), CNS: conscious, oriented, Spo2: 99% with PRVC, Urine output: 2200 ml. Same line of treatment of day 1 was continue on day 2 .Ryle's tube feeding was started (150 ml/2hourly).S.creatinine was normal (1.18).

On the **day 3 (20/4/25)**, the patient's vital signs, including O2 saturation with PRVC remained stable. Same line of treatment was continuing.

On the **day 4 (21/4/25),** patient had good respiratory effort and vitally stable, so she shifted from PRVC to PSPRO.At night Again patient shift to PRVC. All blood investigation except Total count (22600 cells/cu.mm) were normal. Albumin and Ketone were absent in urine examination.

On the **day 5 (22/4/25),** patient had good respiratory effort and vitally stable, so again she shifted to PSPRO. Rest of all treatment was continued.

On the **day 6 (23/4/25),** because of good respiratory support patient was shifted from PSPRO to T Piece with 4 L oxygen. Same day night patient Spo2 became 95% with T piece (4 L o2), so again patient shift to PSPRO. All blood investigation were normal. Same line of treatment was continuing.

On the **day 7 (24/4/25)**, patient maintain Spo2 100% on PSPRO, so again patient shift to T piece with 4 L o2. Same day night because of good respiratory effort, good muscle tone and normal level of consciousness ET tube and Mouth piece was removed. Post extubation patient's Spo2 was 97% on RA (room air).

On the **day 8 (25/4/25),** Spo2 of patient was 92 % with 2 L o2 with nasal pronge. Patient was shifted in ward. All blood investigation except Total count (11200 cells /cu.mm) was normal.

On the **day 9 (26/4/25),** Spo2 of patient was 99%with 1L o2. Rest of vitals were normal. X-Ray chest shows haziness in left lower zone. Same line of treatment was continuing. Same day evening o2 was stop. Patient maintain Spo2 97% on RA.

On the **day 10 (27/4/25),** patient's Spo2 gradually decreased from 97% to 92 % on RA.

On the **day 11 (28/4/25),** patient's Spo2 suddenly dropped to 74% on RA.Patient's Spo2 reach up to 90%with 2L o2 support. Rest of vitals was normal. ABG shows ph 7.51, pco2 20, po2 43, hco3 16, tco2 16.6, be -7.0 O2 saturation was 84%. Patient was put on Tab.c-furo-cv 1 bd, Tab Doxo 400 ½ bd, Cap cypra 1 od, Tab Mucinac 1bd with ½ glass of water, syp solvin exp 15ml tds, and inj MPS 40 mg od. Neb ipravent 8 hourly and Budecort 12 hourly started. Tab pirfenidone 200 tds, syp sparcid ds 15 ml bd was started.

On the **day 12 (29/4/25),** patient had difficulty in breathing, tachycardia and her spo2was 90%with 2L o2 and 78% with room air. HRCT Thorax shows diffuse ground glass opacities involving both lung fields, Consolidation involving lateral and posterior segment of bilateral lower lobe, Extensive pneumomediastinum s/o possibility of paraquat induced pneumonitis.S.cretinine was 0.91 (normal). same line of treatment was continuing.

On the **day 13(30/4/25),** patient had poor respiratory efforts and her Spo2 was 76% on RA and 92% with 4L o2, pulse was 126/minute and BP was 96/62 mm of Hg.HRCT of Thorax shows bilateral lung changes s/o paraquat induced lung disease. USG of abdomen shows bilateral increased cortical echogenisity and CM differentiation was maintained. Her blood investigation shows Total counts 21,800 cells/cu mm, CRP was 45.91 mg/dL.

On the same day 2:30 pm patient was shifted to higher center (Rajkot Gov. hospital) for further treatment. Next day morning (1/5/25) despite all efforts, the patient collapsed and could not be revived.

Table 1: Laboratory investigation.

INVESTIGATION	18/4/25	21/4	23/4	25/4	29/4	30/4
Urea	35	22.20	42.60			
Creatinine	0.9	0.96	0.99	0.64	0.91	0.71
Na+	**135.4**	136.3	136.9			
K+	3.78	3.97	4.72	4.57	3.33	
Chloride	**97.1**	98.1	99.0			
Hb	12	12.5	13.20	12.30		12.30
Tc	**13200**	**22600**	**10000**	**11700**		**21800**
Rbs	67					
Sgpt	10	23	18			14
Choline esterase	3882.49					
UPT	-ve					
U-Alb	+++	Absent				
U-Ketone	+++	Absent				
CRP						**45.91**

Table 2: Vital Data.

	Ad.	18/4/25	19/4	23/4	24/4	25/4

Pulse	70/min.	126	88	76	62	62
BP	110/70 mm of Hg	96/56	102/70	116/70	116/72	116/66
Spo2	99% RA	74% RA	99% PRVC	95% Tpiece 4 L o2	97% RA	92% Nasal prong 2L o2

Table 2: Vital Data (Continued)

	26/4/25	**27/4**	**28/4**	**29/4**	**30/4**
Pulse	82/min.	90	102	122	126
BP	122/72 mm of Hg	110/70	112/74	112/70	96/52
Spo2	97% RA	92% RA	74% RA 90% 2Lo2	78%RA 90% 2Lo2	76% RA 92% 4 L o2

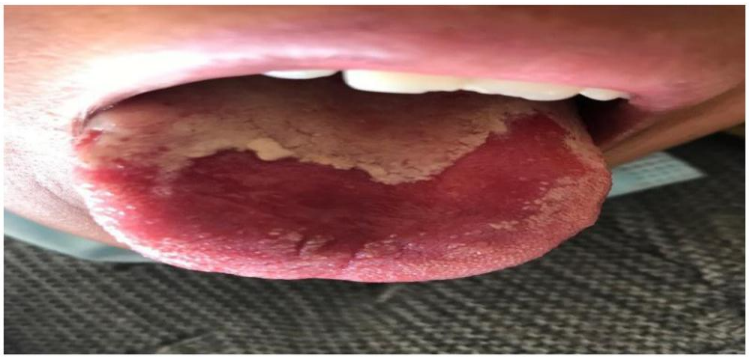


Figure 1: Patient's tongue at the time of admission.

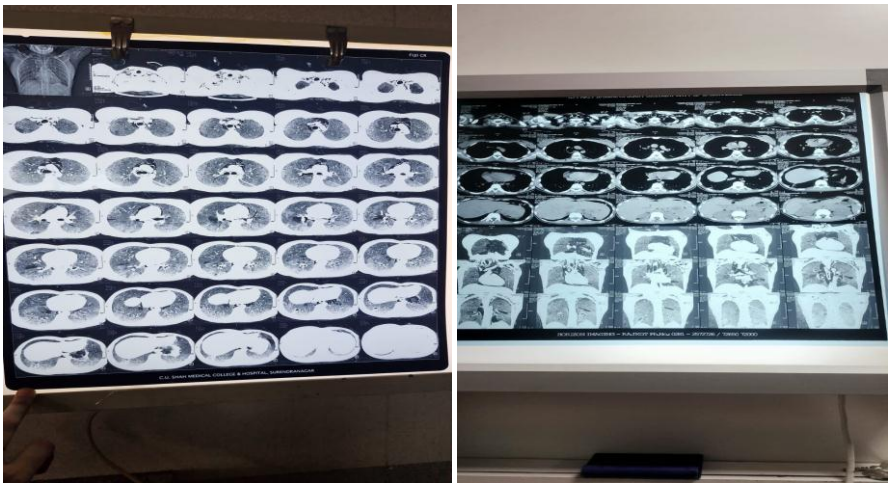
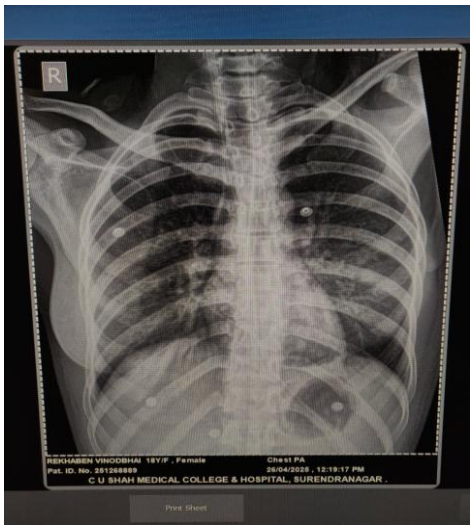
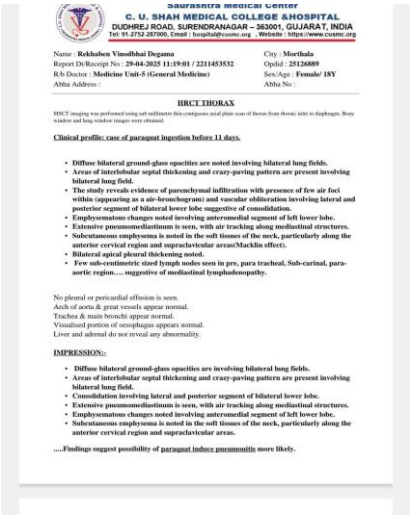


Figure 2: HRCT of Thorax (29/4/25).





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Opdid : 25126889
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Abha No :

HRCT THORAX

MSCT imaging was performed using sub millimetre thin contiguous axial plain scan of thorax from thoracic inlet to diaphragm. Bony window and lung window images were obtained.

Clinical profile: case of paraquat ingestion before 11 days.

- Diffuse bilateral ground-glass opacities are noted involving bilateral lung fields.
- Areas of interlobular septal thickening and crazy-paving pattern are present involving bilateral lung field.
- The study reveals evidence of parenchymal infiltration with presence of few air foci within (appearing as a air-bronchogram) and vascular obliteration involving lateral and posterior segment of bilateral lower lobe suggestive of consolidation.
- Emphysematous changes noted involving anteromedial segment of left lower lobe.
- Extensive pneumomediastinum is seen, with air tracking along mediastinal structures.
- Subcutaneous emphysema is noted in the soft tissues of the neck, particularly along the anterior cervical region and supraclavicular areas(Macklin effect).
- Bilateral apical pleural thickening noted.
- Few sub-centimetric sized lymph nodes seen in pre, para tracheal, Sub-carinal, para-aortic region.... suggestive of mediastinal lymphadenopathy.

No pleural or pericardial effusion is seen.
Arch of aorta & great vessels appear normal.
Trachea & main bronchi appear normal.
Visualised portion of oesophagus appears normal.
Liver and adrenal do not reveal any abnormality.

IMPRESSION:-

- Diffuse bilateral ground-glass opacities are involving bilateral lung fields.
- Areas of interlobular septal thickening and crazy-paving pattern are present involving bilateral lung field.
- Consolidation involving lateral and posterior segment of bilateral lower lobe.
- Extensive pneumomediastinum is seen, with air tracking along mediastinal structures.
- Emphysematous changes noted involving anteromedial segment of left lower lobe.
- Subcutaneous emphysema is noted in the soft tissues of the neck, particularly along the anterior cervical region and supraclavicular areas.

....Findings suggest possibility of paraquat induce pneumonitis more likely.

Figure 3: HRCT THORAX X-Ray chest

CONCLUSION

Paraquat poisoining remains a critical medical emergency with a high mortality rate, especially when treatment is delayed.In this case, a 24 year old female patient presented 24 hour after intentional ingestion and , despite supportive measures, succumbed after 13th day due to progressive pulmonary fibrosis. The outcome reflect the ireversible pulmonary damage caused by paraquat and the lack of effective antidote. This case reinforce the urgent need for early diagnosis, immediate decontamination, a and stringent control over the availabiliy of highly toxic herbicide like paraquat. Enhaanced awareness and public health policies are essential to reduce the burden of such preventable poisoining.

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