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From Toxin to Triage: Nursing Strategies in Paraquat Poisoning Management

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ABSTRACT

Background:

Paraquat (N,N′-dimethyl-4,4′-bipyridinium dichloride) is a widely used herbicide that is associated with high fatality rates following ingestion. Despite global bans, paraquat remains available in India which is an important factor contributing to intentional and accidental poisonings. Its toxicity results in severe multiorgan dysfunction, particularly pulmonary fibrosis, renal failure, and hepatic injury, with no specific antidote available. Supportive care and evidence-based nursing interventions are therefore central to improving outcomes.

Case Report:

We report the case of a 19-year-old male who presented to the medical intensive care unit following intentional ingestion of approximately 100 mL of a 24% paraquat solution. The patient developed systemic manifestations including vomiting, respiratory distress, acute kidney injury, hepatic dysfunction and leukocytosis. He required non-invasive ventilation, haemodialysis, and multimodal pharmacotherapy, including N-acetylcysteine, corticosteroids, antioxidants, antibiotics and hepatoprotective agents. Laboratory investigations showed derangements in renal and liver function, elevated bilirubin, and leukocytosis, with gradual biochemical improvement after supportive therapy. Nursing care emphasized airway and oxygenation monitoring, fluid balance, neurological status, infection prevention, and psychological support. Despite aggressive supportive measures, the prognosis in paraquat poisoning remains guarded, underscoring the importance of early recognition, comprehensive medical management, and structured nursing protocols.

Conclusion:

This case highlights the lethal nature of paraquat poisoning and the absence of a curative antidote. Multidisciplinary collaboration, vigilant nursing interventions, and early supportive care are pivotal in mitigating morbidity and mortality. Nursing strategies that combine physiological monitoring with psychosocial support play a crucial role in patient recovery.

Keywords: Acute Kidney Injury, Multidisciplinary Care, Paraquat Poisoning, Supportive Care

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INTRODUCTION

Paraquat (N,N′-dimethyl-4,4′-bipyridinium dichloride) is a highly toxic non-selective chemical that chemical that is used as a herbicide in agriculture usually for controlling the weeds and grasses in India.[1] It's an odourless, yellow to green liquid with a chemical formula of $C_{12}H_{14}C1_2N_2$ Paraquat was discovered in the 1950s and commercially introduced in 1961 under the name "Gramoxone" and remains one of the most widely used herbicides across the globe.[2,3] Paraquat has been widely banned due to its health risks as well as the consumers but still available in markets and used in India. A significant concern among Indian healthcare professionals is the high mortality rate and morbidity linked to both accidental and intentional paraquat (PQ) consumption. PQ poisoning is frequently fatal and leads to C severe complications such as hepatic and renal failure, progressive pulmonary fibrosis, and Parkinsonism-like symptoms. The absence of a specific antidote makes treating PQ poisoning particularly difficult, strengthening the argument for its prohibition.[4]

Acute pesticide poisoning is a widespread global issue, especially in developing countries. While hospital data captures only severe cases, WHO estimates suggest around 1 million serious accidental

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poisonings and 2 million suicide attempts with pesticides occur annually. However, minor cases are vastly underreported, with surveys indicating that up to 25 million agricultural workers in developing regions may experience poisoning each year.[5] In clinical settings, particularly in primary and secondary level hospitals in India, there is no standardized protocol for the treatment and nursing care of patients with paraquat poisoning. This lack of uniformity often leads to inconsistent care and contributes to a high mortality rate. Since there is currently no specific antidote available for paraquat poisoning, immediate supportive measures such as blood purification therapy, gastrointestinal management, nutritional support, and psychological care play a vital role in patient outcomes.[6] Therefore, managing paraquat poisoning continues to be a major challenge in acute and critical care nursing practice.

CASE REPORT

A 19-year-old male was admitted to the Medical Intensive Care Unit (MICU), on 19th March 2025, with a diagnosis of paraquat poisoning complicated by acute kidney injury (AKI) and multiple organ dysfunction syndrome (MODS). The patient had no known pre-existing medical or psychiatric illness and was reportedly in good health prior to the incident. On 11th March 2025 at approximately 7:00 PM, he intentionally ingested around 100 ml of a 24% paraquat solution at his agricultural field following an episode of severe emotional distress due to unresolved interpersonal conflicts with family at home. Immediately following ingestion, he developed episodes of vomiting and abdominal discomfort and was taken to a nearby local hospital by his family. He remained there under supportive care for three days, during which his clinical condition worsened, prompting referral to the tertiary care centre AIIMS, Mangalagiri for advanced management. Initial assessment indicated multi-organ involvement, necessitating admission to the intensive care unit. There is no history of previous hospitalizations, surgeries, or chronic illnesses. The patient's family history is non-contributory, with no known hereditary or psychiatric disorders. This case highlights the aggressive nature of paraquat toxicity, the importance of early diagnosis, and the critical role of multidisciplinary management in cases of suicidal pesticide ingestion.

Upon arrival, the patient was conscious but exhibited symptoms of systemic toxicity, including shortness of breath, palpitations, persistent dry cough, intermittent fever with chills, and severe vomiting. On physical examination, his c BMI: 27.5 kg/m^2; yellowish discolouration of eyes and pupils were bilaterally equal and reacting to light. His oral cavity was erythematous and edematous. Heart sounds were normal. Grade II edema was present on bilateral limbs. Urinary elimination was through a 14 French foley's catheter. The urine was orange in color.

On examination, initial vital signs showed tachycardia, tachypnoea, fever 100-degree Fahrenheit and SPO2 level of 86%. ECG showed sinus tachycardia and 2-D Echocardiography revealed good LV systolic function EF 72% with mild pericardial effusion and Doppler study revealed that mild pulmonary hypertension (PVSP of 38mmHg) done. Laboratory investigations revealed rising creatinine and blood urea levels, suggestive of acute kidney injury and underwent 4 cycles of haemodialysis. Chest X-ray done and pulmonary evaluation raised concerns of evolving pulmonary infiltrates. After 3 days of admission the CBC results showed that Hb:12gm/Dl, Haematocrit value of 33%, TLC is 17700cells with a normal platelet and coagulation profile. KFT reports showed that Serum creatinine level came down to 1.8mg/dL, Blood urea level: 113.7mg/Dl BUN level:53.1mg/dL with normal serum electrolyte levels. On 24th March urinary sodium dithionite test was done and showed a negative result (Table 1).

S.No	Date	Name of Investigation	Patient Value
1	26/03/25	**Complete Blood Count**	
		Hemoglobin	12gm/dL
		Haematocrit	33%
		TLC	17700cells/mm^3
		Platelet Count	274×10^5 cells/mm^3
		Procalcitonin	0.23%
2	26/03/25	**Kidney Function Test**	
		Blood urea	33.1mg/dL
		Blood urea Nitrogen	113.7mg/dL
		Serum Creatinine	1.8mg/dL
		Serum Uric acid	5.6mg/dL
		Serum Sodium	133mEq/L
		Serum Potassium	3.9mEq/L
3	26/03/25	**Liver Function Test**	
		Serum Bilirubin (T)	6.2mg/dL
		Bilirubin (C)	4.5mg/dL
		Bilirubin (UC)	1.7mg/dL
		Total Protein	4.7gm/dL
		Albumin	2.9gm/dL
		Globulin	253 IU/L
		ALT	42 IU/L
		AST	254.6 IU/L
		ALP	
4	24/03/25	Urinary sodium dithionite test	Negative
5		2-D ECHO	Normal EF Mild PAH Mild pericardial effusion
6	22/03/25	aPTT	27.35 sec
		PT	17.45 sec
		INR	1.3

Table No:1 List of investigations

Therapeutic Intervention

As the patient got admitted to medical intensive care unit got the following treatment (Table 2)

1. **N-Acetylcysteine** – 600 mg IV, three times daily (TID)
2. **Vitamin C** – Oral tablet, one tablet TID
3. **Dexamethasone** – 8 mg IV, TID
4. **Ursodeoxycholic acid (UDILIV)** – 300 mg orally, TID
5. **Ondansetron (Emeset)** – 4 mg IV, as needed (SOS) for nausea/vomiting
6. **Paracetamol (Acetaminophen)** – 1 g IV, as needed (SOS) for fever/pain
7. **Lactulose syrup** – 30 mL at bedtime (HS)
8. **Meropenem** – 1 g IV, TID
9. **Pantoprazole** – 40 mg IV, once daily (OD) for gastric prophylaxis
10. **Vitamin K** – 10 mg IV, OD for 3 days

Table No 2: Therapeutic Interventions Administered in the ICU

The patient received a multimodal treatment approach targeting oxidative stress, inflammation, and secondary complications of paraquat toxicity. Similar cases were treated using this same modlaities.[7] As the patient developed severe paraquat toxicity, manifesting as acute kidney injury (AKI), acute liver injury, and leukocytosis, along with respiratory distress (tachypnoea and hypoxia) necessitating non-invasive ventilation (NIV) with pressure support (PS) of 14 cm H_2O and PEEP of 6 cm H_2O to improve oxygenation and reduce work of breathing. N-Acetylcysteine (600 mg IV TID) was prioritized to counteract oxidative-mediated damage to the lungs, kidneys, and liver, while dexamethasone (8 mg IV TID) aimed to attenuate inflammatory pneumonitis and pulmonary fibrosis. Vitamin C (oral TID) provided adjunctive antioxidant support. Given oliguric AKI (evidenced by orange-colored urine via a 14Fr Foley catheter), strict fluid balance was maintained with judicious IV hydration to optimize renal perfusion without volume overload. Ursodeoxycholic acid (UDILIV, 300 mg oral TID) and vitamin K (10 mg IV OD for 3 days) were administered to mitigate hepatocellular injury and correct coagulopathy. Broad-spectrum meropenem (1 g IV TID) covered for sepsis risk secondary to immunosuppression and leukocytosis. Pantoprazole (40 mg IV OD) prevented stress ulcers, and lactulose (30 mL HS) aided ammonia clearance in hepatic dysfunction. Symptom control included ondansetron (4 mg IV SOS) for nausea and cautious paracetamol (1 g IV SOS, adjusted for AKI) for analgesia. Renal function was closely monitored, with readiness for renal replacement therapy if AKI progressed.

Nursing management of patient with paraquat poisoning in an intensive care unit

Nursing care of the patient with paraquat poisoning was quite challenging because there was no existing clinical protocol.[6]

1. Assessment

- ABC assessment (Airway, Breathing, Circulation)
- Gastric lavage (if<1 hour) – with caution
- Activated charcoal
- Monitoring of vital signs including regular body weight checking to check for retention of fluids
- Hourly monitoring of SpO_2, ABGs, and respiratory effort was performed.
- Ensure proper fit of NIV mask to prevent air leaks and skin breakdown.
- Maintain settings (PS 14 cm H_2O, PEEP 6 cm H_2O) and document response.
- Suction secretions PRN while minimizing NIV disconnection.
- Monitoring of neurological status
- Proper positioning of the patient in semi-fowler's position
- Strict intake output monitoring
- Monitor the patient for signs of bleeding
- Regular foley's catheter care to prevent the occurrence of CAUTI.

1.Nursing Diagnosis

As per the patient condition, existing treatment modalities and identified needs and future complications that may occur following nursing diagnoses were considered while treating a patient with paraquat poisoning.

1) Impaired gas exchange related to alveolar damage from paraquat toxicity as evidenced by hypoxia SpO_2 level of 88%

- Assess the respiratory status of the patient
- Monitor oxygen saturation of the patients
- Position the patient to improve the lung compliance
- Ensure proper delivery of oxygen via NIV
- Monitor for leakage through the sides of NIV mask
- Suction secretions whenever needed

2) Acute kidney injury related to paraquat nephrotoxicity as evidenced by increased serum creatinine level

- Monitor the renal function of the patients
- Monitor intake output
- Observe for the signs of fluid retention
- Restrict potassium rich foods

3) Risk for fluid volume excess related to reduced renal function and decreased plasma protein level

- Monitor intake output chart and monitor fluid balance
- Daily weight monitoring
- Auscultate lung sound for crackles at bases
- Restrict sodium intake·Observe for signs of fluid excess

1) Risk for confusion related to hepatic encephalopathy as evidenced by elevated serum bilirubin level
- Monitor mental status
- Observe for changes in the behaviour
- Implement safety measures
- Administer lactulose as per physician's prescription

2) Risk for Infection related to immunosuppression and invasive devices.
- Monitor for signs of infections
- Monitor vital signs HR, Temperature
- Perform hand hygiene before all procedures
- Strict aseptic technique
- Check lab values for signs of infection TLC level

3) Risk for bleeding to liver injury and coagulopathy
- Monitor for signs of bleeding
- Monitor PT, CT, aPTT, INR
- Monitor urine and stool for occult blood
- Monitor vital signs
- Administer prescribed anticoagulants to the patient
- Apply pressure in the site after puncture

4) Anxiety related to disease process and ICU environment as evidenced by facial expression
- Establish a rapport with the patient
- Clarify the doubts of the patient in clear and simple language
- Give simple explanation before every procedure
- Encourage family to involve in patient care
- Tell the patient to hear some music or song

In ICUs, nurses spend more time with patients than their relatives. This patient, being very young, attempted suicide due to familial conflict. It is important to encourage patients and families to face and address such conflicts proactively to prevent such actions.5 Educate the family regarding the herbicides and its consequences when its kept to near reach of family members especially to children.[8]

DISCUSSION

Self-poisoning is fatal. In rural areas most of these self-poisoning commonly occurs due to ingestion of herbicides which easily available their homes.[9-11] Herbicides are the leading type of pesticides which consumed by the Indian farmers to protect their crops.[12,13] When these highly hazardous pesticides are readily available in Indian markets raises a concern in relation to the pesticide suicides which is serious public health issue among agricultural workers and their families.[14-16] Even though the reporting of such case were less due to the medico legal case impacts.[17] Numerous studies have investigated the correlation between pesticides and suicide, revealing a positive relationship that increases the risk of both suicides and suicidal behavior.[18] Paraquat self-poisoning is not only a problem of the Asia–Pacific region and in Europe and America as well. The case fatality of paraquat poisoning is very high due to its molecular properties, high toxicity, and lack of standardized management protocols.[19,20]

Paraquat toxicity primarily impacts the lungs and kidneys due to its distinctive distribution pattern. The molecular structure of the poison is similar to that of natural polyamines, which enables it to selectively accumulate in alveolar type I and II cells via active uptake mechanisms. Likewise, the kidneys actively excrete paraquat, resulting in its concentration within proximal tubule epithelial cells. Once it has accumulated in these targeted tissues, paraquat triggers a damaging cycle of redox reactions that produce harmful reactive oxygen species. This oxidative damage rapidly surpasses the body's inherent antioxidant defences. In the lungs, this mechanism leads to progressive harm, starting with acute alveolitis that can develop into potentially lethal pulmonary fibrosis, especially at moderate levels of exposure. The fibrotic change occurs due to abnormal activation and proliferation of fibroblasts, which gradually destroys the normal structure of the lungs. Renal injury is characterized by vacuolization and necrosis of proximal tubule cells, while liver damage arises from mitochondrial dysfunction and damage to the endoplasmic reticulum.[21,22]

Early decontamination with activated charcoal or Fuller's earth within 2-4 hours is critical, along with prophylactic nasogastric tube insertion if pharyngeal burns are present or paraquat is detected in urine. Prognosis is determined through urine dithionite testing (with repeat testing if initially negative) and plasma paraquat levels. Daily monitoring of electrolytes, liver function, and blood gases helps track organ dysfunction progression. Supportive care includes IV fluids for hypotension or swallowing difficulty, with strict fluid balance monitoring to detect acute kidney injury. Oxygen should be avoided unless absolutely necessary, as it may worsen lung injury. Hemoperfusion or haemodialysis may be considered only for early presentations (within 2 hours) or borderline exposures, but is futile in advanced poisoning. Continuous cardiorespiratory monitoring is essential, as hypotension or pneumonitis indicates poor prognosis. Pain and agitation should be managed with opiates and benzodiazepines as needed. Mechanical ventilation is generally avoided due to high mortality in pneumonitis and fibrosis cases. The approach emphasizes early intervention, cautious oxygen use, and prognosis-guided decision making based on toxicant levels and organ function.[23,24]

CONCLUSION

The management of paraquat poisoning has primarily been supportive, and the outcomes of treatments for paraquat poisoning, which include absorbents, pharmacological methods, radiotherapy, haemodialysis, and hemoperfusions, have been unsatisfactory. At present, there are no genuine pharmacological antagonists available for paraquat, nor are there any chelating agents that can effectively bind the toxin in the bloodstream or other tissues.23 A patient suffering from severe paraquat poisoning faces a grim prognosis due to the absence of a specific antidote. The mortality rate is significant, making it crucial to recognize the clinical symptoms and obtain a history of exposure to this lethal poison. While medical management is crucial in treating paraquat poisoning, nursing care plays an equally vital role in determining patient outcomes. Effective treatment requires a holistic approach that addresses not just the physical manifestations of toxicity, but also the patient's psychological distress, family concerns, and social needs. Comprehensive care must integrate both medical interventions and compassionate nursing support to achieve optimal recovery and quality of life.

Conflict of interest

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