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CASE REPORT

Death Due to Homicidal Paraquat Poisoning: A Case Report

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Abstract

Background: Paraquat is a widely used herbicide that raises a significant public health concern due to its extreme toxicity even at low doses and its potential for criminal poisoning. However, most of the research is concentrated on pulmonary toxicity of the compound so far, ignoring its other systemic effects. Moreover, a perfect antidote for this poisoning remains still at large. **Case Presentation:** In this case report, we present probably the first case of criminal paraquat poisoning in India, involving a 40-year-old man who was poisoned by family members with paraquat-laced alcohol. Although the patient initially appeared to be recovering, his condition worsened, and he died from multi-organ dysfunction syndrome (MODS). **Discussion:** Paraquat poisoning presents with a spectrum of clinical features, mainly affecting the lungs and kidneys, often leading to respiratory and renal failure. However, its toxicity extends to other organs like liver causing toxic hepatitis that prompts us for a proper diagnosis and management. Current research and management protocols tend to neglect the hepatic and renal toxicities, focusing primarily on pulmonary fibrosis. The manufacture and sale of paraquat in India needs to be reviewed in the light of increasing global bans of this compound. There is also a pressing need for antidote research in dealing with agrochemical substance abuse, particularly paraquat. **Conclusion:** The emergence of paraquat as a lethal homicidal poison highlights the need for stringent regulations and proactive measures to safeguard public health. The whole saga of chemical/pesticide regulation in our country is very naïve when compared to the global standard. It is time to foster collaboration between regulatory bodies, healthcare professionals, and researchers to address the menace of *'paraquat deaths'* in India.

Keywords: Paraquat, Criminal poisoning, Homicide, Herbicide, Dithionate test, Forensic Toxicology

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Introduction

Paraquat dichloride, scientifically labelled as N,N′-dimethyl-4,4′-bipyridinium dichloride, is an organic compound with the chemical formula [(C6H7N)2]Cl2 [1]. It is extensively utilized across the globe as an herbicide in agriculture. The mechanism of action of paraquat is due to its uptake by cells where it produces toxic reactive oxygen species, leading to oxidative stress and cellular damage/death.

Paraquat (PQ) is emerging as a one of the largely abused agrochemical for self-harm in India. It has always been a matter of concern for clinicians because this compound is extremely lethal in even small doses (5-10 ml/one sip) and the research community is equally worried about developing a reliable antidote for this. In addition, there were instances of accidental exposure to PQ during agricultural activities or while handling it at manufacturing sites. Although PQ was sporadically misused for criminal poisoning in other parts of the world, there were no documented cases of its intentional use for homicide in India, except for an unsuccessful attempt involving a child [2]. Here, we present the first documented case of fatal homicidal PQ poisoning in an adult in India.

Case Report

A 40-year-old man was poisoned with paraquat while consuming alcohol by his family members, who colluded to permanently eliminate him. According to the deceased's statement, his wife had engaged in an extramarital affair, leading to heated arguments and marital discord between them. Subsequently, his wife, father-in-law, and brother-in-law conspired together and mixed paraquat into the alcohol consumed by the deceased. The incident took place at the deceased's residence. The investigation revealed that the motive for the murder was to get rid of him due to his constant questioning of his wife's fidelity, his lack of involvement in any gainful employment to contribute to the family expenses, and his excessive spending on alcohol. However, the deceased remained unaware of this until his condition deteriorated, leading to his hospitalization four days later. Additionally, the deceased had been attacked by his father-in-law and brother-in-law six years earlier, resulting in a scuffle and the deceased sustained an upper limb fracture (specific details regarding the site and side of the fracture are unavailable).

The deceased presented at the healthcare facility with complaints of abdominal pain, decreased urine output, yellowish discoloration of conjunctivae and urine, and vomiting for the past 4-5 days. There is no significant past medical history available on record. He is a known alcoholic for the past 10 years. On physical examination, icterus was present, and the blood pressure was 99/60 mmHg. Cardiovascular, central nervous system, and respiratory system examinations were normal. Laboratory data at the time of admission showed TC: 16,700/µL, RFT: serum creatinine at 9.2 mg/dL, blood urea at 264 mg/dL, LFT: AST at 381 IU/L, ALT at 481 IU/L, ALP at 202 IU/L, total bilirubin at 12.8 mg/dL, and RVAB 1 and 2 non-reactive. Serum electrolytes were within normal limits and coagulation profile data is unavailable. The ultrasound of the abdomen and pelvis detected grade 1 renal parenchymal disease and a 6mm calculus in the right kidney (the exact location is not mentioned by the radiologist), thickened gall bladder, and

minimal free fluid in the pelvis. The initial working diagnosis was acute febrile illness with acute kidney injury, and attempts were made to rule out leptospirosis, scrub typhus and other related diseases with similar presentation. However, the associated upper GI bleed and oral mucosal erosions made the attending physician become sceptical about the infectious aetiology. Eventually, the diagnosis of poisoning of unknown origin was made through a thorough history collection process by the physician. The case was subsequently registered as a Medico-Legal Case (MLC), involving police authorities. The deceased himself went to the police station and lodged a complaint about the issue after the attending physician raised suspicions of poisoning in this case during his hospital stay before his condition worsened. Further investigation revealed that the deceased was criminally poisoned with paraquat laced with alcohol. The deceased succumbed to Multi-Organ Dysfunction Syndrome resulting from Acute Kidney Injury and Toxic hepatitis one week after admission to the hospital despite several attempts of haemodialysis and transfusion of fresh frozen plasma. An autopsy was conducted accordingly to ascertain the exact cause of death.

At autopsy, the deceased appeared moderately built and moderately nourished. The body exhibited a yellowish tinge overall, with yellowish conjunctivae. Brownish-red fluid was observed emanating from the mouth and nostrils. Ulcerations were noted throughout the oral mucosa and lips. Upon dissection, both lungs were deeply congested and emitted brownish-red fluid upon sectioning. The liver appeared yellow, enlarged, and gritty upon sectioning. Both the kidneys appeared grossly congested on cutsection. The left ventricular wall of the heart measured 2.4 cm. The lumens of both coronary arteries were patent, with thickened walls, and the aorta exhibited atherosclerotic changes at some sites. Approximately 50 ml of brownish-red fluid was found in the stomach. Viscera were preserved for chemical analysis, including the liver, kidney, stomach and its contents, small intestine and its contents, and blood. Histopathological examination of the lung revealed features of congestion and dilated alveolar spaces. The liver showed congestion and fatty changes histologically whereas the kidneys also showed congestion and oedematous parenchyma. Chemical analysis of the viscera tested positive for paraquat (qualitative analysis) even at about 2 weeks post poisoning. Similarly, a glass of paraquat-laced liquor was seized at the crime scene after autopsy, which also revealed paraquat and ethyl alcohol upon chemical analysis. Quantitative analysis of paraquat was not possible due to resource constraints, both for clinical and forensic purposes. Additionally, the urine dithionate test was not performed during the hospital stay. The cause of death in this case was opined as *"due to paraquat poisoning as per available evidence on record"*. All the accused in this case later admitted to poisoning the deceased while he was consuming alcohol, intending to kill him due to his disruptive and irresponsible behaviour towards family members. Consequently, they were charged with homicide, and the case is currently under legal proceedings.

Discussion

In this case, the patient initially appeared normal for four days after being criminally poisoned with paraquat which is

quite commonplace in practice. The reason why poisoned victims may appear normal after consumption can be attributed to the variations in both the concentration and quantity of PQ consumed. This variability scientifically explains the differing onset times for symptoms, ranging from immediate effects to delayed manifestations. It's common for paraquat poisoning patients to remain asymptomatic for a considerable period (a few hours to days) before the prodromal acute gastroenteric illness-like symptoms begin to manifest. These symptoms then slowly transform into renal failure and respiratory failure/ Multi Organ Dysfunction Syndrome (MODS) as the condition worsens. This is a critical consideration to be borne in mind when discharging patients with paraquat poisoning from hospitals. Point-of-care physicians may occasionally discharge seemingly stable patients who later return in a deteriorated condition. Furthermore, there is an increasing trend of concealed paraquat poisonings in India. Urban, well-educated individuals are obtaining paraquat from online markets, consuming it in small doses for committing suicide, and keeping it undisclosed to others. They only inform their treating doctors about it when the situation deteriorates further and are on the verge of death. This has become a method of clandestine suicide poisoning for some individuals who wish to make their deaths appear as though it was caused by illness rather than intentional poisoning [3].

Paraquat poisoning can mimic various pathological conditions, and incidents of using this lethal herbicide for criminal purposes are increasing globally, with several reported cases [4-9]. The signs and symptoms of paraquat ingestion can resemble other conditions and need differentiation. Paraquat poisoning manifests with a spectrum of clinical features, with pulmonary and renal toxicities being predominant and often contributing to mortality. Gastrointestinal symptoms, though common, are sometimes overlooked as a cause of mortality due to erosion and perforation. Early signs may include the characteristic "Paraquat tongue," along with oesophageal and gastric erosion, accompanied by nausea and vomiting. Pulmonary complications arise due to paraquat distribution to pneumocytes, leading to pneumonitis, Acute Respiratory Distress Syndrome and, in delayed cases, pulmonary fibrosis. Finally, it may result in a multiorgan failure which is usually associated with acute renal injury, hepatic shut down, myocardial injury and internal bleeding. Timely identification and management are crucial in reducing the fatal outcomes of paraquat poisoning.

Oropharyngeal burns, a major sign, can also occur in other herbicidal ingestions, burns, infections like aphthous ulcers, herpes simplex, tonsillitis, and chemotherapy induced toxicity. Acute dyspnoea must be differentiated from emergent pulmonary conditions such as airway obstruction, aspiration, cor-pulmonale, pneumonia, pulmonary embolism, pulmonary hypertension, tension pneumothorax, cystic fibrosis, and cardiac conditions like cardiac tamponade, cardiogenic pulmonary oedema, myocarditis [1]. The diagnostic dilemma in recognising paraquat poisoning lies in the initial clinical presentation of nonspecific symptoms like buccopharyngeal ulcers, sore throat, and vomiting that mimic a natural illness. The treating physician might misdiagnose it as influenza, diphtheria, or dermatitis, leading to delays in PQ

poisoning management. The point of care medical officers should have a high index of suspicion to think on the lines of poisoning especially in cases where proper history isn't available.

If we look at the medical literature, ongoing research, and poisoning management protocols, much is discussed about paraquat lung, while the toxic effects on the kidneys and liver have often not been given proper consideration. Instead, disproportionate attention has been given to Acute Respiratory Distress Syndrome and lung fibrosis. The initial impact of paraquat poisoning is typically observed in the kidneys, resulting in acute kidney injury, often followed by toxic hepatitis in many patients. However, in cases of minor dosage consumption, renal toxicity tends to be self-limiting, with patients exhibiting favourable responses to haemodialysis and avoiding further progression to Multi-Organ Dysfunction Syndrome (MODS). This has led some practitioners to rigorously pursue haemodialysis, although its benefits in high dose/concentration paraquat poisoning are equivocal [11]. There is a pressing need for research on prognostic and mortality indicators in the management of paraquat poisoning. It has been observed by several physicians that aspiration of paraquat during ingestion and extensive GI burns are a few indicators of worse prognosis [1].

As paraquat increasingly becomes a tool for homicide, it is imperative to reconsider regulations regarding its sale in India. Given its significant toxicity to humans, it warrants classification as a highly toxic compound, necessitating a national emphasis on research aimed at minimizing its usage, addressing packaging and labelling concerns, and ultimately phasing it out with sustainable alternatives in agriculture practices. The packaging should prominently display hazard indicators like it is usually displayed on tobacco products. Furthermore, the implementation of technology-based consumer tracking and the employment of digital suicide prevention surveillance are essential in the current context. The ongoing debate about the connection between paraquat exposure and Parkinson's disease remains a controversial issue and adds weight to paraquat ban argument [12].

Various potential antidotes have been identified for management of paraquat poisoning, but antidote research faces limitations due to the substance's unique mechanism of action involving redox cycling and the generation of reactive oxygen species, which are highly toxic at the cellular level. Effective antidotes should aim to either bind to the substance and render it inert or disrupt the cascade of events leading to cellular and molecular toxicity. Further research in mechanistic toxicology of PQ is necessary.

In a medico-legal setting, relying solely on qualitative toxicology data may not suffice. Quantitative data regarding paraquat levels is preferable, despite literature indicating that even a small amount can be lethal. The challenges in quantifying paraquat or its residues in bodily fluids, attributed to factors such as time elapsed and metabolism, are significant considerations within the domain of autopsy toxicology. However, a clinically positive dithionate test, coupled with paraquat detection in body fluids before death, can significantly aid forensic pathologists in determining the cause of death, apart from utilizing the chemical analysis of viscera following autopsy. It's worth noting that while a pathologist can confidently identify the cause of death,

establishing the method/manner of poisoning rests with the law enforcement agencies and always requires additional evidence.

This case report represents the first documented instance of homicidal poisoning involving paraquat in India. It is noteworthy in the context of forensic toxicology and public health awareness. The only other comparable reported incident involved an unsuccessful attempt by a grandmother to poison her 4-year-old grandchild with paraquat [2]. This case underscores the importance of vigilance in detecting and managing paraquat poisoning, particularly in cases involving deliberate harm.

Conclusion

With paraquat emerging as a lethal homicidal weapon, urgent measures are warranted to regulate its sale and use in India. Considering its potential for self-harm and homicide, a ban on this substance should be seriously considered, with efforts directed towards identifying economical alternatives in agriculture. Emergency physicians must remain vigilant, as paraquat poisoning can masquerade as a medical illness, necessitating a high index of suspicion for accurate diagnosis. In cases of uncertainty, the urine dithionate test stands as a crucial point-of-care diagnostic tool, guiding appropriate treatment protocols. However, the persistent lack of an effective antidote for paraquat underscores the pressing need for further research in this area. As we navigate these challenges, it's imperative that regulatory bodies, healthcare professionals, and researchers collaborate closely to mitigate the grave consequences of paraquat poisoning and safeguard public health.

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Conflicts of Interest

The authors declares that they do not have conflict of interest.

Ethical Considerations

All ethical concerns should be addressed to the authors.

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