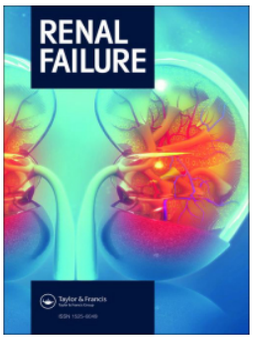

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CASE REPORT

A Fatal Case of Paraquat Poisoning Following Minimal Dermal Exposure

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Paraquat is a pesticide widely used in agriculture. Numerous cases of paraquat intoxication have been reported either accidentally or intentionally as a suicidal attempt. The most severe cases of paraquat poisoning refer to oral ingestion. Complications include respiratory, hepatic, and renal failure, and are usually fatal. Dermal exposure is less frequent and rarely fatal.

This article reports a case of an 81-year-old man with minimal skin burn after accidental paraquat exposure. The patient developed acute renal and respiratory failure and, despite aggressive treatment with hemodialysis, hemoperfusion, and mechanical ventilation, died two days later.

Keywords paraquat, pesticide, poisoning, renal failure, ARDS

INTRODUCTION

Paraquat (1,1'-dimethyl-4,4'-dipyridylium), the nonselective pesticide used worldwide in agriculture,[1] is highly toxic in humans and has caused numerous fatalities. It remains the most common cause of fatal pesticide poisoning in western countries.[2] Paraquat intoxication is characterized by circulatory failure with multiple organ failure in the early phase, as well as by respiratory failure caused by progressive pulmonary fibrosis in a later phase.[3] Although severe cases of poisoning are usually the result of ingestion, accidental or in a suicide attempt, there are also sporadic reports of systemic involvement after skin exposure to paraquat.[4,5]

This study reports a case of fatal paraquat poisoning after minimal dermal exposure.

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CASE REPORT

An 81-year-old male presented to his family doctor because of a skin lesion of the right thigh after accidental contact with paraquat the previous evening. His relatives reported that the pesticide was spread on the trousers, and the old man slept overnight without removing the clothes. The lesion was limited, producing only skin erosion, which was treated empirically with steroid ointments. Four days later, the patient complained of severe breathlessness and was admitted in our department.

On admission, the patient complained of difficulty in breathing. He also reported decreased urine output over the last 48 hours. He did not report fever or other systemic symptoms. His past medical history was unremarkable except for mild hypertension during the last four years, which was treated with perindopril.

On initial physical examination, the patient was alert but highly distressed. Vital signs were (on admission): rectal temperature, 37.2ºC; heart rate, 85/min; respiratory rate, 16/min; and blood pressure, 120/70 mmHg. Superficial chemical burn on the right thigh was calculated as covering 4% of the total body surface area. There were no mouth ulcers. The rest of the examination was negative.

Laboratory tests revealed the following: hematocrit, 42.5%; white blood cells count, $14.6\times10^3/\mu L$ (89.9% neutrophils); PLT, $160\times10^3/\mu L$; urea, 246 mg/dL; creatinine, 10.1 mg/dL; Na, 140 mEq/L; K, 4.2 mEq/L; alkaline phosphatase, 102 IU/L; SGOT, 87 IU/L; SGPT, 100 IU/L; LDH, 431 IU/L; Ca, 9.4 mg/dL; P, 6.0 mg/dL; ESR, 107 mm; PT, 26%; and INR, 2.17. Blood gas analysis on two liters of oxygen showed pH, 7.46; PaO_2, 49.1 mmHg; $PaCO_2$, 27.7 mmHg; and O_2, saturation 83.5%. Chest radiograph showed findings compatible with acute respiratory distress syndrome (ARDS; see Figure 1). Abdomen ultrasound was negative. Urine sodium dithionite test was negative for paraquat, and no pesticide was detected in blood sample. These findings indicated acute lung injury, acute renal injury, leucocytosis, and impaired hepatic function.

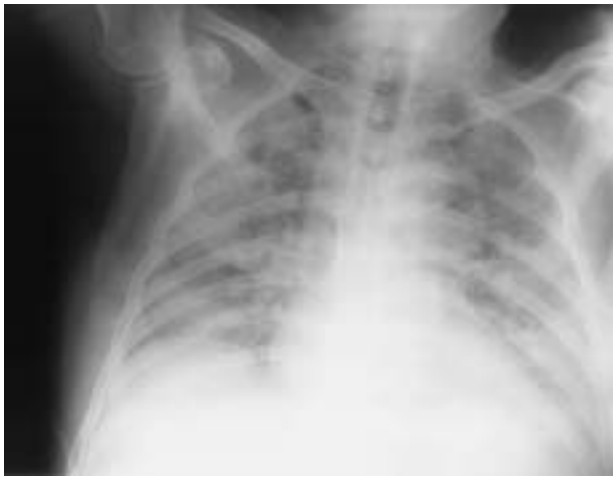

Figure 1. Chest x-ray showing infiltrates of the lower lobes, suggestive of Acute Respiratory Distress Syndrome (ARDS).

The patient was treated with hydration, oxygen supplementation, and intravenous antibiotics. Renal failure was managed by hemodialysis and hemoperfusion. The lung function continued to deteriorate. He was transferred to the Intensive Care Unit, intubated, and ventilated, but died two days later.

DISCUSSION

Paraquat poisoning remains a significant worldwide cause of morbidity and mortality,[1] following pesticide exposure. The mechanism of toxicity in humans has not been clarified, but paraquat has been shown in animals to induce complete oxidation of both NADPH and NADH and increase lipid peroxidation.[6] Additionally, paraquat is a potent generator of acute reactive oxygen species.[6]

The most severe cases of paraquat poisoning occur following ingestion: oral exposure has been demonstrated to correlate with longer mean hospitalization and higher fatality rate.[7] There are reports of dermal exposure to paraquat producing only local lesions on skin and nails without any signs of systemic involvement.[8,9] Nevertheless, some cases of severe systemic involvement and deaths after skin exposure have been reported.[4,5] In the present case, dermal exposure was extremely limited, covering only 4% of the total body surface area.

In vitro and in vivo experiments have indicated low percutaneous permeability of the substance through intact skin.[10,11] Similar animal studies demonstrated that in an aqueous solution, paraquat ions do not penetrate the intact skin of the rabbit, which is more permeable than the human skin.[12] Therefore, damaged skin is essential for significant paraquat absorption.[10,13] Previous skin lesions can be aggravated by paraquat itself. This was the case in the patient featured here, who remained in contact to paraquat through the humid trousers despite the initial chemical burn. Most fatalities via the dermal route are reported in developing countries.[10,13] All cases are also associated with pre-existing skin lesions or prolonged contact of healthy skin with concentrated solutions.[10,13]

There are many analyses in the literature that attempt to determine potent predictors of the outcome in paraquat poisoning. A multiple logistic regression analysis by Jones et al.[14] demonstrated that patients with a given concentration of paraquat have a better chance of survival if they have only recently taken it; moreover, at any time after ingestion, the prognosis is better the lower the plasma concentration. As kidneys are the major organ to excrete paraquat, paraquat-poisoned patients with severe acute renal failure have a prolonged half-life of paraquat and a markedly increased mortality rate.[15]

Retrospective studies showed that good predictors of outcome may be the low plasma and urine concentrations within the first 24 hours of intoxication.[16,17] Nevertheless, the qualitative urine test is not reliable for patients presenting >24 hours after the exposure.[17]

Treatment choices for paraquat poisoning include general treatment of poisoning (aiming to decontaminate the gut and minimize absorption), hypo-oxygenation,[3] lung radiotherapy, hemodialysis, and hemoperfusion,[3] but results are controversial.[18,19] Nevertheless, we decided to treat our patient with hemoperfusion, as there are anecdotal reports of successful treatment even in cases of late referral.[20] There are also reports with promising results regarding the use of combined methylprednisolone and dexamethazone therapy.[21,22] There is one report in the literature for the successful treatment of severe paraquat poisoning after dermal exposure by the combined use of cyclophosphamide and methylprednisolone pulse therapy.[23]

Steroid treatment may decrease the inflammation of paraquat poisoning.[24] Moreover, steroids can suppress superoxide production by neutrophils.[25] Therefore, steroid therapy may be an effective treatment of paraquat poisoning (after the failure of standard therapies), where the inflammation of the lungs may play a major role in the lethal hypoxemia of patients.[22]

In conclusion, it is clear that even minimal dermal exposure to paraquat can be fatal, especially when associated with high concentrations of the substance and/or significant delay in treatment initiation. The exact efficacy of the current medical management remains controversial. Therefore, it is crucial for emergency physicians to suspect paraquat poisoning when facing chemical burns and skin lesions and to investigate for the possibility of intoxication, particularly when skin lesions are associated with systemic symptoms.

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