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IMAGES IN TOXICOLOGY

Paraquat poisoning with fatal outcome in a 56-year-old agricultural worker

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Abstract. Paraquat is a highly toxic herbicide that can cause caustic lesions, acute kidney failure and delayed pulmonary fibrosis. We report a case of a 56-year-old male agricultural worker who intentionally ingested 20 ml of paraquat, leading to a fatal outcome. He presented with vomiting, diarrhea, dysphagia and sialorrhea. Despite prompt medical intervention with cyclophosphamide, methylprednisolone, and supportive care, the patient developed acute renal failure and progressive pulmonary fibrosis. His condition deteriorated rapidly, and he succumbed to refractory hypoxemia 31 days after hospital admission. This case highlights the lethal nature of paraquat poisoning and the importance of preventive measures to minimize exposure.

Key words: *Paraquat; Herbicides; Pyridinium compounds; Caustics; Tongue; Pulmonary fibrosis.*

Paraquat is a non-selective herbicide that is widely used in agriculture. It is a highly toxic substance that can cause caustic lesions, acute renal failure (usually reversible) and delayed oxygen-dependent pulmonary fibrosis (after 7 to 14 days). Paraquat poisoning is a medical emergency that requires immediate treatment due to its high mortality. We present the case of a 56-year-old male agricultural worker who presented to a low-complexity health center with vomiting and diarrhea, immediately after intentional ingestion of 20 ml of paraquat of unknown concentration.

After 48 hours, due to persistence of symptoms and appearance of dysphagia and sialorrhea, he went to a more complex center. On hospital admission, a whitish depapillated tongue was observed, a sign known as "paraquat tongue" (Fig. 1). Complementary studies were carried out, including chest X-ray and laboratory tests. Analyses showed an elevated white blood cell count (16500/mm3), elevated urea (96 mg/dl) and creatinine (4.3 mg/dl), and alterations in liver enzymes (alanine aminotransferase/ALAT 34 IU/ml and aspartate aminotransferase/ASAT 94 IU/ml). Arterial blood gas was as follow: pH: 7.44, pCO2: 32 mmHg, pO2: 79 mmHg, bicarbonate: 20.8 mEq/l, base excess: -2.1 and Sat O2: 96% (FiO2 at 0.21). The sodium dithionite test in urine was positive, confirming the presence of paraquat in the body (Fig. 2).

Figure 1. 48 hours after ingestion. We can appreciate the "paraquat tongue": depapilated and whitish tongue.

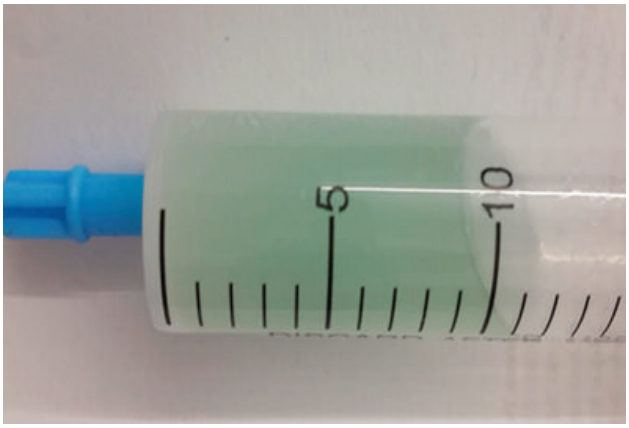

Figure 2. Dithionite test 48 hours after ingestion. Positive result for paraquat: bluish staining of urine.

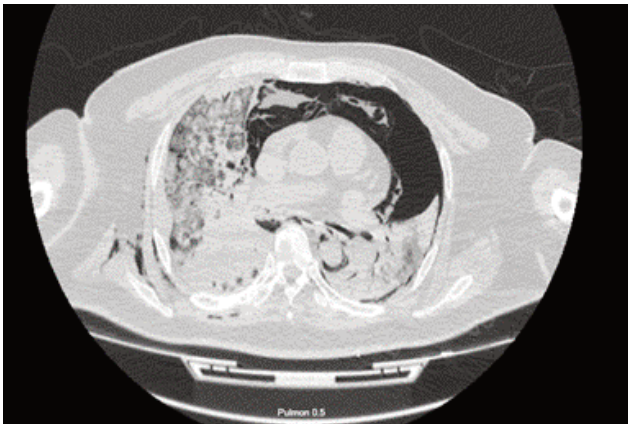

Figure 3. 30 days after ingestion: pneumomediastinum, pneumothorax, pulmonary fibrosis, images of consolidation and subcutaneous emphysema.

Based on the anamnesis, physical examination and complementary studies, it was decided to start immunomodulatory treatment with cyclophosphamide (1 g/day) for 48 hours and methylprednisolone (1 g/day) for 72 hours. The Nephrology Unit opted for a wait-and-see approach to renal replacement therapy. Upper gastrointestinal endoscopy was not performed due to the time elapsed since ingestion.

During hospitalization, the patient presented a deepening of renal injury, with uremia of 287 mg/dl and creatinemia of 8.2 mg/dl, but maintained an adequate diuretic rhythm. From the seventh day after ingestion, he progressed to respiratory failure secondary to progressive pulmonary fibrosis, documented by computed axial tomography (Fig. 3). He required different modalities of ventilatory support, from high-flow nasal cannula to non-invasive ventilation and finally orotracheal intubation. He died from refractory hypoxemia 31 days after hospital admission.

Conflicts of interest

The authors declare no conflicts of interest.

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