Lung in Paraquat Poisoning

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ABSTRACT

Insecticide poisoning is a common problem in the developing world. Organophosphorus is the most commonly misused pesticide, yet, quite a large number of suicidal victims tend to use paraquat as a means of self-harm. We present a case of a 70-year-old male with alleged history of consumption of herbicide poison (Paraquat 24%). He was treated with intravenous (IV) fluids, antacids, antibiotics and supportive measures, as well as activated charcoal as adsorbent. The patient later developed oral ulcers, respiratory and metabolic acidosis, and pulmonary fibrosis. His relatives insisted on discharge against medical advice. This case highlights the rapidity of onset of fibrosis, within 52 hours of ingestion of the lethal compound with serial chest radiographs indicating development of widespread fibrosis within 72 hours. It also stresses on avoiding oxygen therapy in cases of paraquat poisoning.

Keywords: Paraquat, insecticide poisoning, fibrosis, acute respiratory distress

Insecticide poisoning is a major burden in the developing world.1 While organophosphorus is the most commonly misused pesticide, quite a large number of suicidal victims tend to use paraquat as a means of self-harm. Moreover, it has been abused in multiple routes with dismal prognosis in all.2,3 Despite being classified as a Class II-moderately hazardous pesticide by the World Health Organization (WHO), its easy availability favors wide usage.4 The major factor affecting the prognosis of patients is the ingested amount of paraquat as the compound demonstrates concentration-dependent toxicity.5

Very few successful cases have been reported in the literature.6,7 Since, there is no identified antidote for paraquat, intoxication is predominantly fatal. Lung is the most common organ involved in paraquat intoxication. The mechanisms of lung injury are numerous.8 The patterns of lung injury could be pneumothorax, acute respiratory distress syndrome (ARDS) and finally fibrosis.9 Pulmonary fibrosis has been reported as early as 5 days in paraquat ingestion.10-13 Here, we report a case of paraquat poisoning with eventual lung fibrosis.

CASE REPORT

A 70-year-old male patient, without any comorbidities and addictions, was brought to emergency room after 3 hours of alleged history of consumption of herbicide poison (Paraquat 24%) about 50 mL mixed with water. Patient was referred for further management from local hospital where stomach wash was done, managed with intravenous (IV) fluids and antiemetic. The patient had one episode of vomiting. There was no history of diarrhea, oliguria, cough, abdominal pain, chest pain and fever.

On examination, vital signs were stable (afebrile, blood pressure [BP] - 160/90 mmHg, pulse rate - 78 bpm, respiratory rate - 16/min, oxygen saturation - 98% in room air, capillary blood glucose - 110 mg/dL). The patient was conscious and oriented; abdomen was soft and nontender; heart sounds were normal without murmurs; bilateral chest movements were symmetrical with good air entry. Patient was admitted in intensive care unit (ICU), treated with IV fluids, antacids, antibiotics and supportive measures accordingly. Ryle’s tube was inserted and activated charcoal was used as adsorbent.

On Day 1, his blood parameters were normal (random blood sugar [RBS] - 118 mg/dL; urea - 25 mg/dL; creatinine - 0.8 mg/dL; serum glutamic oxaloacetic
transaminase [SGOT] - 34 U/L; serum glutamic pyruvic transaminase [SGPT] - 29 U/L; alkaline phosphatase [ALP] - 152 IU/mL; Na - 144.6 mEq/L; K - 4.29 mEq/L; Cl - 106.6 mEq/L; complete blood cell [CBC]: Hemoglobin - 11.8 g%, white blood cell [WBC] count - 15,900/μL, platelet - 2.66 lakhs/μL, erythrocyte sedimentation rate [ESR] 6 mm and 13 mm at 30 minutes and 1 hour, respectively). Electrocardiogram (ECG) revealed sinus tachycardia while chest radiograph was interpreted normal (Fig. 1).

Over the next 2 days, the patient developed difficulty in opening mouth owing to the development of multiple oral ulcers. The oral ulcers, initially hemorrhagic, eventually became necrotic (Fig. 2), hence resulted in dysphagia. Oromaxillary facial surgery opinion was taken and the patient was started on vitamin C therapy. Serial arterial blood gas (ABG) analysis revealed metabolic acidosis at admission, progressing to respiratory alkalosis in the next 2 days.

At 54 hours of ingestion of the poison, patient developed tachypnea and fall in saturation (SpO₂ = 50% with room air). Chest radiograph on the same day (Day 3) revealed fibrotic strands in the right lung mid zone and lower zone (Fig. 3).

Simultaneously, he developed hypokalemia (2.53 mEq/L). Potassium correction was given with IV potassium chloride infusion in normal saline.

Few hours later, the patient began to gasp for breath and was intubated. Chest radiograph on Day 4 revealed fibrotic stands in bilateral lung fields (Fig. 4).

The patient soon developed both respiratory and metabolic acidosis, and rapidly progressed to shock. His blood reports on Day 4 were urea - 73 mg/dL; creatinine - 3.7 mg/dL; SGOT - 410 U/L; SGPT - 344 U/L; ALP - 237 IU/L; total protein: 4.10 g%; albumin - 3.44 g%; globulin - 2.37 g%; INR - 1.09; Na - 143.1 mEq/L; K - 3.70 mEq/L; Cl - 114.8 mEq/L; serum methemoglobin - 2.1%. He was on mechanical ventilation. However, patient’s relatives insisted on discharge against medical advice.
DISCUSSION

Paraquat is a commonly used agricultural pesticide. Concentration-dependent toxicity is well-established for this deadly compound. Intoxications are predominantly lethal via ingestion as well as by IV route.2,3 Intentional ingestion often results in accumulation of the toxin in lungs due to its high affinity to alveolar cells. Subsequently, free radical mediated tissue injury occurs resulting in alveolitis, pneumothorax, acute respiratory distress and finally fibrosis.5,9

Interstitial alveolitis is the precursor lesion for development of fibrotic strands later during the course of the disease. The progression is usually slow, with pulmonary fibrosis developing weeks after the intoxication, which was evident even in rare survivors. Limited earlier studies have reported acute lung fibrosis developing within few days.10-13

Our case highlights the rapidity of onset of fibrosis, within 52 hours of ingestion of the lethal compound. Serial chest radiographs of our patient indicate development of widespread fibrosis within 72 hours (Figs. 1, 3 and 4).

Our patient was an elderly male with history of one episode of spontaneous vomiting. We propose that minimal aspiration of the toxin into the lungs could have exaggerated the immune response resulting in rapid fibrosis within 4 days of intoxication. Vomiting and age have been found to be important variables in the prognosis of the patient.14 Use of oxygen therapy in maintaining oxygen saturation has mixed effect, as it aggravates tissue injury by forming more and more reactive oxygen species. Therefore, nasal oxygen was deferred.

Next to lungs, kidneys are involved in paraquat poisoning, as the compound finds its way out of the body through urine. Following kidneys, organs with rich blood supply, like liver and brain are damaged. Eventually, the patient succumbs to multiple organ damage. In our patient too, renal parameters were elevated thrice the baseline, whereas liver enzymes were raised to more than 10 times the baseline by the 4th day.

Being caustic in nature, the paraquat compound corrodes the epithelium of the gastrointestinal tract. It is well-known to cause severe mucosal ulcerations, hemorrhagic erosions of the gut wall, esophageal perforation leading to mediastinitis, clinically presenting as chest pain, cough, dysphagia and extensive oral ulcers.15,16 Our patient developed extensive ulcerations of the oral mucosa by Day 3, that restricted mouth opening due to stricture formation, as shown in Figure 2. The use of steroids was restricted to avoid esophageal perforation.

Few reliable techniques are available for predicting survival in intentional paraquat poisoning.17,18 However, majority of victims yield to multiorgan failure within few days-weeks. Prior studies advocate the use of fuller’s earth/activated charcoal as adsorbents to decrease systemic absorption, immunosuppressant therapy to tackle hyperimmune response that foreruns pulmonary fibrosis and antioxidants to bring down the free radical injury.11,19 Our patient had received activated charcoal as well as antioxidants. Early hemoperfusion therapy has been found to be indispensable in reducing mortality in severely poisoned patients. Hence, it should be initiated as soon as possible, particularly when the patient arrives early with history of paraquat poisoning.20 However, for our patient, this could not be done.

In our patient, the clinical history, presentation and documentation of paraquat consumption endorses the positive diagnosis, further supported by elevated serum methemoglobin.21 In circumstances, where reliable history is unavailable and there is clinical suspicion of paraquat poisoning, its presence needs to be confirmed by using dithionite test in urine or plasma.22

Paraquat ban decreased intentional herbicide ingestion and contributed to lowering herbicide poisoning-associated mortality.23 It has already been banned in Kerala, India. Alternative human friendly pesticides could be promoted to avoid the morbidity and mortality associated with paraquat exposure. The need of the hour is to educate the susceptible population regarding the harmful effects of paraquat exposure. In victims of paraquat poisoning, the use of gastric lavage and oxygen may be avoided while the use of activated charcoal and antioxidants should be encouraged. Further studies are warranted to identify an antidote and suitable therapeutic strategies in overcoming this highly fatal toxin.

CONCLUSION

Lungs are most commonly affected in paraquat intoxication. The compound demonstrates concentration-dependent toxicity. Lung injury is characterized by alveolitis, pneumothorax, acute respiratory distress and finally fibrosis. Kidneys and liver also get affected eventually, and the patient succumbs to multiple organ damage.
CASE REPORT

This case highlights the appropriate management approach for a patient with suspected paraquat poisoning.

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Access to medical products and creating an enabling legal and trade environment for the public were critical to achieving the Sustainable Development Goals 2030 Agenda, Union Health Minister Dr Harsh Vardhan said.

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