

Case Report**Fatal Paraquat Poisoning in a 15-Year-Old Girl**Rukhsana Parvin¹, Md Kamrul Hasan², Priyanka Sarkar³, Nazmun Nahar Mouri³

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doi: <http://dx.doi.org/10.3329/jemc.v7i2.32657>**Abstract**

Paraquat (dipyridylum) is a highly toxic compound with a pungent smell. It is commonly used for weed killing. Intoxication occurs mainly by oral ingestion. After ingestion severe impairment of multiple organs develops. There is no specific antidote. Treatment includes repeated activated charcoal hemoperfusion with pulse methylprednisolone, cyclophosphamide, and antioxidants. Here we report a case of 15-year-old girl who presented with history of attempted suicide with paraquat. Patient died due to respiratory failure despite aggressive treatment and hemodialysis.

Key words: Paraquat; Hemodialysis; Activated charcoal; Adult respiratory distress syndrome

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Introduction

Paraquat (1,1'-dimethyl-4,4'-dipyridylum) is a broad spectrum quaternary nitrogen liquid herbicide which leads to severe and fatal toxicity after accidental and intentional ingestion.¹ It is sprayed on unwanted weeds and other vegetations before planting crops. It is a fast-acting and nonselective compound. It destroys tissues of green plants on contact and by translocation within the plant. It inhibits reduction of NADP to NADPH during photosynthesis. This disruption leads to the formation of superoxide anion, singlet oxygen, and hydroxyl and peroxy radicals. These reactive oxygen species (ROS) interact with the unsaturated lipids of membranes causing destruction of plant organelles leading to cell death.²

Paraquat was first synthesized in 1882.³ It was first manufactured and sold by Imperial Chemical Industries (ICI) in early 1962³, and is today among the most commonly used herbicides. Commercially available paraquat is produced and sold as the dichloride salt. China is now the world's largest manufacturer of paraquat, producing more than 100,000 tons per year.⁴ Paraquat is also used on non-crop areas such

as public airports, electric transformer stations and around commercial buildings to control weeds. The most common trade name for paraquat is Gramoxone. Under normal storage conditions, paraquat's shelf-life is indefinite.⁵

Acute paraquat poisoning is reported in some parts of Asia, the Pacific and the Caribbean.⁶ It was also a significant cause of self-poisoning in Europe and America in eighties and nineties. It is now banned in most European countries and Sri Lanka.⁷ In Bangladesh, paraquat was not used as a common suicidal agent previously. So far only one case report was published in 2010.⁸ But recently cases of this poisoning have increased. In 2015–2016, there were forty cases in Rangamati Medical College Hospital (G Dewan, personal communication, 10 October 2016), 17 in Rajshahi Medical College Hospital⁹ and seven cases in Dhaka medical college hospital (Md R Amin, personal communication, 11 September 2016) reported. Paraquat poisoning has no specific antidote. Its management includes supportive and intensive therapy taken in intensive care unit. In spite of advances in

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treatment and supportive care, mortality rate remains more than >50%.⁷ Very high case fatality of paraquat is due to its inherent toxicity and the lack of any definitive treatment.⁷ Here we report a case of a 15-year-old female with paraquat poisoning, who was managed conservatively but finally died of the complications.

Case report

A 15-year-old girl hailing from Savar presented in the emergency department of Enam Medical College Hospital with the history of ingestion of about 20 mL paraquat (Fig 1) followed by difficulty in swallowing and pain in throat for 26 hours. Two hours after poisoning she was taken to a nearby hospital where stomach wash was given with normal saline. Then she was managed with intravenous fluid, antibiotics and anti-ulcerants and was referred for further management. The patient had several episodes of vomiting which contained clotted blood. On examination, her tongue was found coated and ulcerated, characteristically known as paraquat tongue (Fig 2), pulse 112 beats/minute, temperature 99°C, respiratory rate 14 breaths/minute and BP was 110/70 mm Hg. Examination of abdomen revealed epigastric tenderness. Blood investigations showed hemoglobin 10.7 gm/dL, WBC count 12,350/cumm and normal serum electrolytes. Endoscopy of upper gastrointestinal tract showed corrosive-induced burn (Zargar classification grade 3a) in the esophagus and stomach (Fig 3). On next day, she developed breathlessness. Examination of both lungs revealed widespread bilateral crepitations. Chest radiography showed bilateral pneumonitis (Fig 4). She was referred to intensive care unit for further respiratory support but the family of the patient denied. Her stool turned black on the same day. After four days of poisoning, she became icteric and her urine became high-colored. Her serum bilirubin was 3.87 mg/dL, SGPT 99 U/L, prothrombin time 17.7 seconds and APTT was 30.4 seconds. Her urine output was reducing gradually from second day of admission. Her serum creatinine was 308 μ mol/L on first day, then 401 μ mol/L on next day and increased up to 524 μ mol/L. She was managed conservatively with intravenous fluid, antiemetic, anti-ulcerant and pulse methylprednisolone including two sessions of hemodialysis. But her breathlessness increased markedly and she became cyanosed. She developed respiratory arrest and finally succumbed to death.



Fig 1. Sample of paraquat



Fig 2. Coated and ulcerated tongue



Fig 3. Endoscopy of upper gastrointestinal tract showing corrosive-induced burn (3rd degree) in the esophagus and stomach



Fig 4. Chest X-ray showing bilateral pneumonitis

Discussion

In developing countries self-poisoning with pesticides is a major public health problem with an estimated 300 000 deaths occurring in the Asia-Pacific region each year.¹⁰ Paraquat poisoning has high case fatality rate (>50%).⁷ Routes of poisoning are ingestion and direct contact with skin. An ingestion of 15 mL of 20% paraquat solution is considered lethal.¹¹ Clinical features depend upon the amount of ingested poison. After ingestion, paraquat causes burning sensation in mouth or throat, abdominal pain, nausea, vomiting and diarrhea. Severe oral ulcers may develop within few days. Tongue may be coated and may appear inflamed. Paraquat is rapidly distributed in most tissues including lungs and kidneys. In lungs it causes pulmonary congestion, edema, hemorrhage, diffuse alveolitis and extensive pulmonary fibrosis. Acute respiratory distress syndrome may occur after 24–48 hours after ingestion. Paraquat causes renal failure by causing hypovolemia, circulatory failure, septicemia and direct toxicity.¹² It also can cause hepatic failure, cardiac failure, shock and convulsion. Direct contact of paraquat with skin causes burns and dermatitis. Contact with eye may cause irritation, burn, corneal damage and scarring.

In a series of 17 patients in India, the most common symptoms were vomiting (100%), followed by

altered sensorium (59%), oral ulceration or dysphagia (53%), dyspnea (41%), or loose stools (24%).¹² In our patient, her tongue became coated one day after taking paraquat. She had melena suggestive of upper gastrointestinal bleeding. She developed renal and hepatic impairment and finally respiratory failure.

Measurement of plasma paraquat concentration is useful both to confirm poisoning and predict prognosis.⁷ A semi-quantitative test using bicarbonate and sodium dithionite can be used as a bedside test to confirm systemic paraquat toxicity. In an alkaline medium, sodium dithionite reduces paraquat to a blue radical. If the urine paraquat concentration is more than 1 mg l⁻¹, the urine will appear blue and this finding alone indicates a very poor prognosis.¹³ There are five nomograms and formulae of plasma paraquat concentrations to predict outcome after self-poisoning.^{14,15} In this case, none of the investigations could be done due to lack of facilities.

Paraquat is rapidly absorbed after ingestion and distributed in lungs, liver, kidney and muscle. Toxicity and its effects increase with the time lapse after ingestion. Our patient was taken to a nearby hospital two hours after ingestion but was not managed properly. She was given stomach wash which is not recommended as paraquat causes caustic injuries. It might also have contributed to the development of mucosal injury followed by development of hematemesis and melena. Clay (Fuller's earth) and activated charcoal are effective absorbents and these should be used for gastric lavage.¹

There is no specific antidote. Pulse therapy with steroids (methylprednisolone or dexamethasone) and cyclophosphamide are given to avoid pulmonary fibrosis.¹⁶ But these immunosuppressive agents do not increase survival.¹⁷ Intravenous salicylates may have some promising role.^{17,18} In our patient, pulsed methylprednisolone was administered, but patient did not show any signs of improvement. High flow oxygen is contraindicated as it can enhance toxicity of paraquat by providing more electron receptors.¹⁹ Hemodialysis is used for acute kidney injury though it cannot increase clearance of the substance. Early insertion of a nasogastric feeding tube will ensure adequate nutrition. Several antioxidants have been tested as potential antidotes in animal studies.²⁰

In human, role of these antioxidants are not well-established as there are no controlled studies.

Paraquat is a highly toxic poison. Clinicians should be aware of the fatal nature of this pesticide along with other pesticide compounds. Government may consider taking necessary steps to ban use of paraquat. Toxicological laboratories should be established to provide investigation facilities for proper diagnosis and management of the poisoning cases. Early establishment of the diagnosis and aggressive management to reduce contamination and prevention of further absorption may help the patient to recover.

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