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Case Report

Report of a Case of Paraquat Poisoning and Mediastinal Involvement

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Abstract

Paraquat has been recognized as a highly toxic agent for pest removal and is used worldwide.In adults, paraquat poisoning for suicidal attempts is much more common than accidental exposure poisoning. Approximately 20% of patients with paraquat poisoning develop pneumomediastonium as a complication with a mortality rate of approximately 100%. A 19-year-old man patient was admitted to the poisoning emergency department of Khorshid hospital, who had ingested paraquat. He had nausea and vomiting and had normal vital signs and examination in admission. Initial treatment for the patient was done. The patient signs got worsened on the 21st day of hospitalization and had severe emphysema of the superficial and deep spaces of the neck, followed by bilateral pneumothorax, and severe pneumomediastinum. Unfortunately, the patient died on the 27th day of hospitalization. Purpose of the current study is to raise awareness of rare paraquat toxicity complications, treatment, and especially its lethal complications, including pneumomomediastonium.

Keywords: *Herbicides, intoxication, paraquat, pneumomediastinum*

Introduction

Poisoning with pesticides and other agricultural chemicals is a major public health problem worldwide, especially in developing countries. Paraquat has been recognized as a highly toxic agent for pest removal and is used worldwide.[1] Paraquat is a nonselective, contact herbicide of the bipyridynium group. It is a toxic corrosive liquid green, with a strong odor that is formulated with the formulation "N,N′-d imethyl-4,4′-bipyridinium dichloride."[2,3] Paraquat poisoning in adults for suicide attempts is much more common than accidental exposure poisoning.[3]

After ingestion, paraquat enters organs that receive a large amount of bloodstreams, such as the lungs, kidneys, liver, and muscles. The concentration of paraquat in the lung parenchyma due to its active uptake by pneumocytes type 1 and 2 is about 10–20 times higher than that in plasma; therefore, the lung is a major target of paraquat poisoning and respiratory failure due to common lung injury. The most common cause of death is from the use of this substance.[1] Lipid peroxidation-induced pulmonary fibrosis is also one of the main symptoms of paraquat poisoning.[4] Previous studies have shown that approximately 20% of patients with paraquat poisoning develop pneumomediastinum as a complication with a mortality rate of approximately 100%.[5]

Our goal in writing this article is to raise awareness of the pulmonary manifestations of paraquat use, treatment, and its toxicities, including pneumomediastinum. In the following article, we will describe a case of paraquat toxicity for the suicidal attempt. In the present case subcutaneous emphysema in the neck, pneumothorax, and pneumomediastinum as complications of paraquat poisoning, eventually led to the surgery and death.

Case Report

A 19 year old male patient weighing 61 kg who had been admitted with 50 cc of paraquat ingestion in an attempt to suicide, was brought to the poisoning emergency department of the Khorshid Hospital in Isfahan on September 16, 2018, about 2 hours after repeated nausea and vomiting. Glasgow Coma scale, and vital signs were normal, blood pressure was 120/80 mmHg, pulse rate was 90/min, respiratory rate was 18/min, and the temperature was 37°C, and O2 saturation was 95%. Cardiac and pulmonary examinations were normal at the time of admission.

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The patient had a history of hypothyroidism that was controlled by levothyroxine tablets. Treatment began with a nasogastric tube and gastrointestinal (GI) washing at the time of admission. GI washing continued until the GI secretions were clear and then 60 grams of activated charcoal was gavaged to the patient and subsequently maintained for the nasogastric tube. Dithionite test was performed and the result was positive (dark blue). A catheter was immediately implanted for hemodialysis through the right femoral vein, and the patient underwent hemodialysis three times and every time for 8 h. 2 h after each hemodialysis, a dithionate test was performed to evaluate the toxicity reduction.

Other treatments for the patient include antioxidant administration of *N*-acetylcysteine 300 mg/kg/day, infusion of Vitamin C 150 mg/h, Vitamin E, 300 units every 8 hours in the form of intramuscular injection, and methylprednisolone 10 mg/kg IV. Pantoprazole ampule 40 mg was administered twice a day to protect GI tract, and a full dose of silymarin, 6000 mg/day. Every other day chest X ray was taken and the patient was in a respiratory recovery condition, which suddenly worsened after 21 days of hospitalization and the patient said he had cervical pain and his breathing had gotten very difficult. After physical examination, the right cervical emphysema was detected.

Immediately chest X-ray and lateral neck graphy performed and surgical and lung service consultation were requested. In graphies, the air was seen in the soft tissue of the neck and the frontal area of the trachea as well as in the mediastinum. Bilateral pneumothorax without mediastinal shift was observed. The diagnosis of severe emphysema of superfcial and deep spaces of the neck, followed by bilateral pneumothorax and severe pneumomediastinum was established [Figures 1-3]. The bilateral chest tube was fixed and the patient was transferred to the pulmonary intensive care unit (ICU). During hospitalization, broad-spectrum antibiotic was administered to the patient due to fever and symptoms of pulmonary infection as well as sepsis. In addition to continued treatment of paraquat poisoning, supportive ICU care and treatment by lung and surgical services were also performed.

During hospitalization, level of alanine aminotransferase (ALT) got abnormal from day 12 of hospitalization, then it decreased to the normal range from day 23, but at day 27 it increased remarkably and reached 691 U/L. The level of Aspartate transaminase (AST) was in normal range during hospitalization but increased to 1372 U/L on day 27. Since the forth day of hospitalization, the level of Blood urea nitrogen (BUN) ascended and got out of normal range. During hospitalization, the level of BUN had fluctuation with the maximum level of 54.6 mg/dL recorded on the 21st day. Creatinine level got abnormal since day 3 of hospitalization and increased since day 11 of hospitalization and remained abnormal. Hb level was less than normal and the patient was anemic during hospitalization. Since day 21, Platelet level got out of the normal range and got into a downtrend. The international normalized ratio (INR)

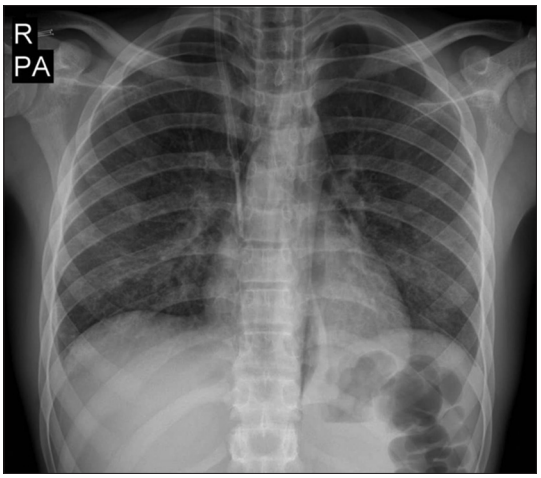

Figure 1: First chest X-ray

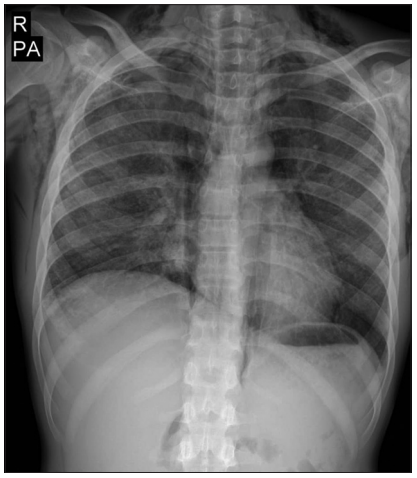

Figure 2: Chest X-ray after neck emphysema

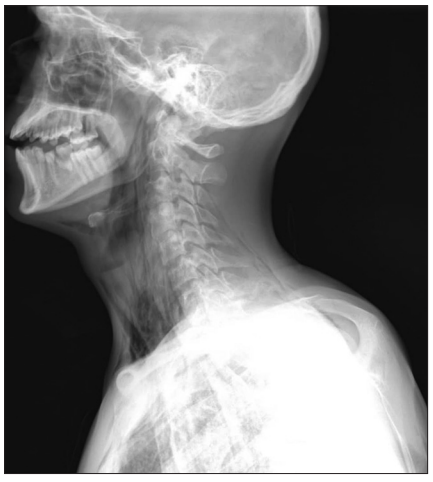

Figure 3: Lateral soft-tissue neck graphy

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level was in the range of 1.27–1.52 and on the 27th day reached 2.29. The patient was died on the 27th day due to severe pneumomediastinum and despite treatment with experimental.

Discussion

Paraquat is an herbicide that has been on the international market since 1958.[6] This compound causes direct cell damage by producing superoxide radicals or other reactive oxygen species and nitrate radicals.[1] In humans, paraquat is highly toxic, with an estimated lethal dose in adults of about 3–6 g. The most common route of poisoning is swallowing (whether intentional or accidental) of concentrated solution.[1]

If taken orally, paraquat in the lungs, unlike gradient, is secreted by active transport and releases hydrogen and superoxide anions, which cause fat damage to the cell membrane. It also generates oxygen-free radicals, leading to hepatotoxicity, nephrotoxicity, and pulmonary fibrosis. Paraquat mainly affects the lungs, where it accumulates at most 6–10 times the plasma concentration.[1]

In lethal cases, histopathologic findings range from pulmonary congestion, edema, and bleeding to extensive pulmonary fibrosis.[1] Since the 1970s, the deaths of patients with paraquat poisoning have been known mainly due to severe pulmonary injury.[6]

Subcutaneous pneumomediastinum and emphysema after paraquat poisoning may be due to corrosion and esophageal perforation due to the corrosive effect of paraquat. Another suggested cause is air leakage from the torn alveoli along the peribronchovascular area, which is caused by alveolar exudation, fibrosis, and increased alveolar tension and shear forces.[6]

Other mechanisms proposed include esophageal injury due to severe vomiting after intoxication or repeated gastric lavage, airway injury due to the ventilator used, or other factors.[5]

Im *et al*. examined radiographic findings and chest CT scan after paraquat poisoning and described abnormal changes observed on radiographs and their clinically visible outcomes. In this study, all patients who developed pneumomediastinum died within 7 days.[7]

Symptoms of paraquat use are usually dose-dependent, and the severity of poisoning can be classified as mild, moderate, and severe. Mild poisoning at doses of 20 mg/kg may occur which causes minor GI problems such as premature vomiting, diarrhea, and burns to the mouth, but it is usually possible to recover completely. Moderate poisoning with doses ranging from 20 mg/kg to 50 mg/kg may be fatal. The patient may suffer lung damage, pulmonary fbrosis, acute renal failure, and in most cases, death occurs within 2–3 weeks. Because of multiple organ failure. In patients who survive longer, fibrotic changes in the alveoli result in gas exchange in the lungs and may progress to ARDS.[3]

Urinary dithionate test for paraquat identification is used to confirm the diagnosis. If urinary paraquat concentration is <1 mg/l within 24 h after paraquat poisoning, the probability of survival is high. Plasma concentrations above 1.6 pg/ml have been found to be lethal 12 h after admission.[1]

Common treatments include nasogastric tube fixation, gastric lavage with normal saline, gavage with charcoal-sorbitol powder, forced alkalinized diuresis, and hemodialysis. Up to 4 h after paraquat poisoning, hemoperfusion with active charcoal is effective.[3]

Antioxidants such as N-acetylcysteine, Vitamin E, Vitamin C, and salicylates can be useful in inhibiting free radicals, inflammatory effects, and nuclear factor kappa B.[1]

Our study limitation were that we could not determine the serum level of paraquat and no esophageal or bronchial damage were observed in bronchoscopy to show the pathology of pneumomediastinum.

Conclusion

Pulmonary fibrosis is a common and lethal complication of paraquat toxicity and almost all of severe cases have this lethal complication. In our case, the patient had this complication but not in severe form, and our planning was discharge depend on his stable situation; but suddenly, he was deteriorate during few hours because of a rare event, means pneumomediastinum.

Declaration of patient consent

The authors certify that they have obtained appropriate patient consent forms. In the form, the patient's father has given his consent for patient's clinical information to be reported in the journal. He understood that his name will not be published and due efforts will be made to conceal his identity.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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