Paraquat Tongue as an Infrequent Complication in Paraquat Intoxication

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ABSTRACT

Paraquat tongue is a rare manifestation of paraquat poisoning. This study reports a case of multiple ulcerations in the tongue of a 31-year-old depressed man who attempted suicide by ingesting a paraquat solution. Chest radiography showed diffuse ground glass opacities of both lungs with a thickening of the intralobular interstitium, compatible with interstitial pneumonitis. Urine paraquat was 10mg/mL. The patient was treated with an immediate detoxification by gastric lavage, active charcoal, charcoal hemoperfusion, pulse therapies of cyclophosphamide (15mg/kg/day for 2 days) and methylprednisolone pulse therapies (1g/day for 3 days), followed by prolonged dexamethasone therapy (20mg/day for 14 days), resulting a favorable prognosis. Furthermore, early intensive detoxification, the mainstay treatment, proved to effective in our case.

Key words: Paraquat, Paraquate tongue, Cyclophosphamide

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**Introduction**

Paraquat was synthesized in 1882 and has been used as an herbicide since 1955.\(^1\) There are three degrees of ingested paraquat poisoning severity. Mild poisoning causes oral irritation and gastrointestinal discomfort but eventually leads to complete resolution. Moderate-to-severe poisoning causes acute kidney injury and, in severe conditions, pneumonitis or pulmonary fibrosis following acute hepatitis, often resulting in high mortality rate between two and three weeks.\(^2\) Those patients often die within several hours to days because of multiple organ dysfunction syndromes after ingesting about 40mL of a 24% paraquat solution. After ingesting approximately 16 mL, moderate-to-severe poisoning and death within 1 to 2 weeks may follow resulting from pulmonary fibrosis and severe hypoxemia.\(^3\) Herein we report a case of paraquat tongue in case involving a man who intentionally drank paraquat to poison himself.

**Case Presentation**

A 31-year-old depressed man attempted suicide by ingesting unknown amount of 24% paraquat solution. He presented three hours after ingestion complaining of intolerable oral irritation and gastrointestinal discomfort. The patient had drunk alcohol wine (200mL, 16%v/v alcohol) daily for the previous three years. He had no history of hypertension, diabetes mellitus, or other chronic illnesses. On arrival, his body temperature was 36.8°C, his pulse was 78 beats/min, his respiratory rate was 18 breaths/min, and his blood pressure was 126/73mmHg. On physical examination, the patient was alert and moderately anxious. Some ulcerative wounds were noted in the oral cavity. Other systems revealed no remarkable abnormal findings. The sodium dithionite test of urine showed a dark blue color and a urine paraquat concentration of 10μg/mL three hours after ingestion. Other laboratory tests reported white blood cells to be 18000/mL, hemoglobin 13.6g/dL, platelets 181,000/mL, blood urea nitrogen 35.7mg/dL, serum creatinine 1.99mg/dL, aspartate aminotransferase 64 units/L, and alanine aminotransferase 24 units/L. Chest radiograph taken in the emergency department showed no active lung lesion. Twelve hours after ingestion of the toxin, a series of standard treatments including gastric lavage, active charcoal and magnesium citrate catharsis, and emergent hemoperfusion with 300g of active charcoal cartridge was initiated. Two 8-hour courses of hemoperfusion were completed, resulting in a negative sodium dithionite urine test. The patient was started on 1g of methylprednisolone in 100mL of dextrose 5% water (D5W) administered over one hour repeated daily for three days. Cyclophosphamide, 15mg/kg, in 100mL of D5W was administered over one hour daily for two days. The patient was then admitted to our toxicology unit for continued monitoring. Unfortunately, on day 4, he suffered aggravated oral mucosal ulcerations [Figure 1].

Upper gastrointestinal panendoscopy showed that esophageal necrotizing ulcers as well as his gastric and duodenal ulcers were compatible with corrosive injury and esophagocardiac junction bleeding due to Mallory Weiss tear following the management with hemoclips. To evaluate whether there were potentially late paraquat poisoning complications, we followed the patient using chest radiographs and tests for renal function, liver function, and arterial blood gas with the patient breathing room air. On day 5, chest radiography showed diffuse...
ground glass opacities of both lungs, with thickening of the intralobular interstitium, compatible with interstitial pneumonitis. 2000mg of intravenous ceftriaxone was initiated daily due to fever accompanied with aggravated dyspnea, and 5 mg of intravenous dexamethasone was administered every eight hours. His clinical condition gradually recovered. The patient was discharged on day 23, with outpatient clinic follow-up. At three months follow-up, the patient remained free of clinical manifestations.

**Discussion**

In the present study, patient with paraquat poisoning had multiple oral ulcers and presented with later complications, including pulmonary fibrosis and interstitial pneumonitis. Ingestion of paraquat may cause oral damage and can lead to mucosal lesions of the mouth and the tongue called “paraquat tongue”. Paraquat tongue starts to develop within several days of poisoning and ulcerations may develop with or without bleeding. Mucosal lesions in the pharynx, esophagus and stomach are also very common and much more insidious. Those complications may result in perforation, mediastinitis and pneumomediastinum. It is fortunate that our patient responded well to an intensive detoxification protocol involving gastric lavage, active charcoal, charcoal hemoperfusion, as well as cyclophosphamide and repeated methylprednisolone pulse therapies, followed by prolonged dexamethasone therapy. In our case, the patient’s initial oral mucosal lesions gradually improved to near-normal after 3 months of follow-up, even in the presence of corrosive injuries.

**Figure 1. Anterior view (top) and lateral view (bottom): Ingestion of paraquat may cause aggravated oral mucosal ulcerations with or without bleeding and can lead to mucosal lesions of the mouth and the tongue called “paraquat tongue”**.
There has been great progress in strategies to treat paraquat intoxication introduced by Lin JL et al.[4-7] In 1996 and 1999, Lin et al conducted a randomized control studies proving that better survival can be achieved using cyclophosphamide and methylprednisolone (treatment group, 25% and 18.2% mortality, respectively; conservatively treated group 70.6% and 57.1%, respectively). In 2006 and 2011, Lin et al further conducted two studies of pulse cyclophosphamide and pulse methylprednisolone and found they could achieve even better survival following these treatment protocols (treatment groups, 31.3% and 66% mortality, respectively; conservatively treated groups 85.7% and 92%, respectively). The severity of inflammation plays a major challenge in the treatment of patients with paraquat tongue during the subacute period of intoxication. Repeated methylprednisolone pulse therapy after dexamethasone treatment can attenuate the severity of inflammation following paraquat poisoning.[5] A definite diagnosis of paraquat poisoning and an immediate institution of a detoxification protocol are a prerequisite for a favorable outcome.

Conflicts of Interest Statement

The authors declare no conflicts of interest.

References

巴拉刈中毒患者的罕見表現：
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摘    要

巴拉刈中毒患者的預後通常都不好，巴拉刈舌頭更是巴拉刈中毒患者的罕見表現之一。一名31歲的抑鬱症男子因服用巴拉刈除草劑自殺而導致的多處舌頭潰瘍，胸部X光顯現大量的毛玻璃樣斑塊合併肺葉間質性增厚與間質性肺炎，尿液巴拉刈濃度為10mg/dL。因此立即根據巴拉刈解毒指引給予活性炭洗胃並且執行血液灌流術合併環磷醯胺與類固醇脈衝療法。巴拉刈中毒早期依據巴拉刈解毒指引去治療可以有效提高巴拉刈中毒患者的預後。

關鍵詞：巴拉刈、巴拉刈舌頭、環磷醯胺

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