International Medical Society
http://imedicalsociety.org

International Archives of Medicine
Section: Toxicology & Therapeutics
ISSN: 1755-7682

2016
Vol. 9 No. 160
doi: 10.3823/2031

Paraquat Tongue: Rebirth of Paraquat Intoxication

CASE REPORT

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Abstract

Acute pesticide poisoning accounts for significant morbidity and mortality in the world, especially in developing countries. We present an interesting case of paraquat intoxication here.

A 39-year-old man was brought to our emergency department (ED) by his family soon after attempting suicide by ingesting unknown substances. Initial vital signs, laboratory data, and chest radiography showed normal. Only green-blue color of finger and redness of tongue were noted. Urine unknown drugs screening tests were negative. Initial urine paraquat test was also negative at our ED. Then he confessed to taste 1-2 drips of paraquat (24%) through his right finger. We arranged hemoperfusion therapy due to the strong contact history and some physical examination findings. Finally, the serum paraquat level at ED was 0.1 μg/ml. This case highlights that even in cases with negative urine paraquat tests, detailed history taking and physical examination can help the diagnosis of paraquat poisoning. Early recognition and the appropriate treatment are imperative.

Keywords
Paraquat Tongue; Paraquat Intoxication.

Introduction

The main causes of pesticide poisoning in Taiwan are paraquat, organic phosphates, glyphosate and carbamates [1, 2]. Paraquat, 1, '-dimethyl-4,4'-bipyridinium dichloride, is the one of the most widely used herbicides in the world. Paraquat is a rapidly-acting, non-selective herbicide and relatively inexpensive. It is toxic to human beings and animals by its redox activity, which products superoxide anion, potentially leading to acute respiratory distress syndrome and acute kidney injury [3, 4].

Paraquat is rapidly absorbed from ingestion, dermal exposure and inhalation and can be detected within 1 hour from serum and uri-

ne. According to data published by Proudfoot et al. (1979), Proudfoot (1995) and Smith (1988b), peak concentrations (Cmax) in humans are attained within 2 hours to 4 hours after ingestion [6, 7]. Irrespective of the route of administration, lung and kidney are the organs showing the highest concentrations of paraquat [7-9]. Preferential accumulation in the lung is increasing toxicity due to superoxide anion. Over a period of hours to days, these processes cause multi-organs failure. The most affected organs are those with oxygen tension, high blood flow, and energy requirements, in particular the heart, lung, kidney, and liver. In general, urine paraquat test is useful for screening paraquat poisoning [10]. Management of paraquat intoxication usually depends on the amount ingested and the time elapsed since the exposure.

Case Report

A 39-year-old male was brought to our emergency department (ED) 2 hours after attempting suicide by ingesting unknown herbicides. He has history of depressive disorders without regular follow-up. His Glasgow Coma Scale (GCS) is E4V5M6. His blood pressure was 130/96 mmHg, pulse rate was 82 beats/min, and respiratory rate was 18 breaths/min. Initial laboratory examinations revealed normal kidney function tests and normal serum electrolytes. Unknown drugs screening test of urine, including benzodiazepine, tricyclic antidepressants, barbiturates, cocaine, acetaminophen, tetrahydrocannabinol and phencyclidine, showed negative results. Physical examinations revealed mild green-blue color over distal phalanx of right index finger **(Figure 1)**. In addition, mild reddish without obvious ulceration was noted over the apex of tongue. Chest radiography was unremarkable. Urine paraquat test was also performed but revealed negative findings at initial 1 hour and 2 hours after arrival of ED. He received gastric lavage with NG-tube, and only some yellowish gastric juice was found from NG tube.

The patient finally told the physician that he took a small amount of paraquat (24%) through his right finger. The diagnosis of paraquat intoxication was made based on history taking and physical examinations. Then serum paraquat test was checked at ED. After admission, he received charcoal hemoperfusion therapy for 2 times. The apex region of tongue revealed mild reddish at day 3 **(Figure 2)** and obvious ulcerations at day 6 **(Figure 3)** after admission. Initial

Figure 1: Physical examinations showed mild green-blue color over distal phalanx of right index finger.

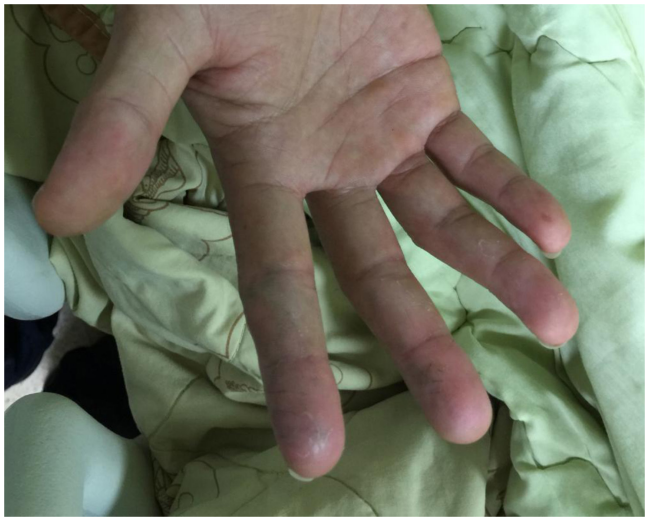
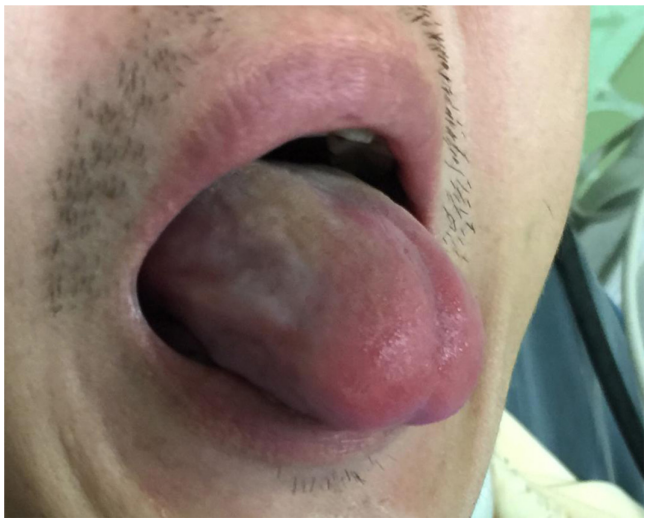
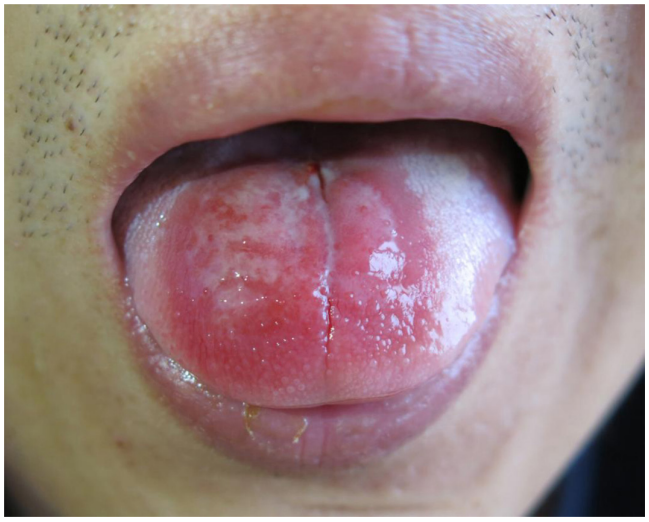


Figure 2: The apex region of tongue showed mild reddish at day 3.



serum paraquat test at ED was positive and the concentration was 0.1 μg/ml. Following kidney function tests and chest radiographies showed normal. The results of serials of serum paraquat concentrations were undetectable after hemoperfusion. Lastly, he was discharged after 10 days in hospital. No obvious respiratory sequelae and kidney injury were found with 6-month follow-up.

Figure 3: The apex region of tongue showed obvious ulcerations at day 6.



Discussion

Paraquat intoxication is life-threatening and can cause major effects on gastrointestinal tract, lung, liver, and kidney. The Median Lethal Dose (LD50) in human beings is approximately 35 mg/kg, which translates into as little as 8-12 ml of a 24% solution [11]. The gastrointestinal tracts such as tongue, mouth, pharynx, esophagus, stomach and intestine are the most initial sites of toxicity, which is manifested by edema, swelling, and painful ulceration. In some severe cases, centrizonal hepatocellular injury can occur, resulting in elevated bilirubin and enzymes like aspartate transaminase (AST), alanine aminotransaminase (ALT) and lactate dehydrogenase (LDH) [12]. The hepatic injury from paraquat can be severe enough to cause jaundice. However, hepatotoxicity is rarely a major determinant to clinical outcome. On the contrary, acute kidney injury plays a critical role in determining the clinical outcome [12].

Pulmonary complication also represents the most lethal and least treatable manifestation. Both type I and II pneumatocytes appear to selectively accumulate paraquat. Biotransformation of paraquat in these cells causes free radical production with promotion lipid peroxidation and cell injury [13, 14]. There is a progressive decline in arterial oxygen tension and CO2 diffusion capacity. Cyanosis and dyspnea reflect deteriorating gas exchange in the damaged lung. In addition, frothy sputum is the early and principal manifestation of pulmonary edema. Such a serious dysfunction of gas exchange results eventual death due to hypoxemia. Acute pulmonary edema and early lung damage can occur within few hours to days after ingestion [13, 14]. In clinical practice, routine investigations such as liver function tests, kidney function tests, and electrolytes are checked only to monitor baseline status of the patient. Several studies predicted survival rates in patients with paraquat intoxication by using plasma paraquat concentrations and/or clinical criteria [16-19].

Paraquat is not metabolised and nearly excreted unchanged in the urine, it can be easily detected by qualitative urine analysis. A semi-quantitative test using bicarbonate and sodium dithionite can be used as a bedside test to confirm systemic paraquat toxicity [19]. In an alkaline medium, sodium dithionite reduces paraquat to a blue radical. If the urine paraquat concentration is more than 1 μg/ml, the urine will appear blue and this finding alone indicates a very poor prognosis [20]. However, negative urine paraquat test could also be fatal [21]. To date, hemoperfusion is proven to be better than homodialysis in treating patients with paraquat poisoning. Hemoperfusion can decrease mortality rates within 6-12 hours after paraquat exposure. Cyclophosphamide, methylprednisolone,

dexamethasone and vitamine C have some therapeutic effects [20, 22, 23].

Conclusion

The present case highlights the importance that physicians should be alert to the possibility of acute paraquat poisoning in high risk patients presenting with unknown skin lesions and mucosal ulcerations. The absence of paraquat in urine could also be fatal. Early recognition and the rapid treatment of paraquat intoxication are imperative, even in cases with negative urine paraquat tests.

Reference

1. Yang CC, Wu JF, Ong HC, Hung SC, Kuo YP, Sa CH, et al. Taiwan National Poison Center: epidemiologic data 1985-1993. J Toxicol Clin Toxicol. 1996; 34(6): 651-63.
2. Hung DZ, Yang CC, Ong HC, Wu JF, Sa CH, Chen SS, et al. The present and feature of poison control center in Taiwan. J Toxicol Sci. 1998; 23 Suppl 2: 280-3.
3. Bonneh-Barkay D, Reaney SH, Langston WJ, Di Monte DA. Redox cycling of the herbicide paraquat in microglial cultures. Brain Res Mol Brain Res. 2005; 134(1): 52-6.
4. Adam A, Smith LL, Cohen GM. Langston WJ, Di Monte DA. An assessment of the role of redox cycling in mediating the toxicity of paraquat and nitrofurantoin. Environ Health Perspect. 1990; 85: 113-7.
5. Price LA, Newman KJ, Clague AE, Wilson PR, Wenck DJ. Paraquat and diquat interference in the analysis of creatinine by the Jaffé reaction. Pathology. 1995; 27(2): 154-6.
6. Proudfoot AT, Stewart MS, Levitt T, Widdop B. Paraquat poisoning: significance of plasma-paraquat concentrations. Lancet. 1979; 2(8138): 330-2.
7. Murray RE, Gibson JE. A comparative study of paraquat intoxication in rats, guinea pigs and monkeys. Exp Mol Pathol. 1972; 17(3): 317-25.
8. Sharp CW, Ottolenghi A, Posner HS. Correlation of paraquat toxicity with tissue concentrations and weight loss of the rat. Toxicol Appl Pharmacol. 1972; 22(2): 241-51
9. Ilett KF, Stripp B, Menard RH, Reid WD, Gillette JR. Studies on the mechanism of the lung toxicity of paraquat: comparison of tissue distribution and some biochemical parameters in rats and rabbits. Toxicol Appl Pharmacol. 1974; 28(2): 216-26.
10. Gil HW, Hong JR, Jang SH, Hong SY. Diagnostic and therapeutic approach for acute paraquat intoxication. J Korean Med Sci. 2014; 29(11):1441-9
11. U.S. National Library of Medicine. Hazardous Substances Databank. Bethesda, MD, 1995. 10-9.
12. Mullick FG, Ishak KG, Mahabir R, Stromeyer FW. Hepatic injury associated with paraquat toxicity in humans. Liver. 1981; 1(3): 209-21.
13. Smith P, Heath D, Kay JM. The pathogenesis and structure of paraquat-indu ced pulmonary fibrosis in rats. J Pathol. 1974; 114(2): 57-67.
14. Honoré P, Hantson P, Fauville JP, Peeters A, Manieu P. Paraquat poisoning. "State of the art". Acta Clin Belg. 1994; 49(5): 220-8.
15. Nordquist RE1, Nguyen H, Poyer JL, Carubelli R. The role of free radicals in paraquat-induced corneal lesions. Free Radic Res. 1995 ; 23(1): 61-71.
16. Ikebuchi J. Evaluation of paraquat concentrations in paraquat poisoning. Arch Toxicol. 1987; 60(4): 304-10.
17. Yoon SC. Clinical outcome of paraquat poisoning. Korean J Intern Med. 2009; 24(2): 93-4.
18. Senarathna L, Eddleston M, Wilks MF, Woollen BH, Tomenson JA, Roberts DM, el al. Prediction of outcome after paraquat poisoning by measurement of the plasma paraquat concentration. QJM. 2009; 102(4): 251-9.
19. Liu Z, Liu D, Zhang X, Wang H, Hao F. Prognosis value of urine paraquat semi-quantitative in the patients with acute paraquat poisoning. Zhonghua Lao Dong Wei Sheng Zhi Ye Bing Za Zhi. 2014; 32(5): 378-80. [Article in Chinese]
20. Gawarammana IB1, Buckley NA. Medical management of paraquat ingestion.Br J Clin Pharmacol. 2011; 72(5):745-57.
21. Monteiro R, Correia L, Simão A, Carvalho A, Costa JN. Negative paraquaturia does not exclude paraquat fatal poisoning. Acta Med Port. 2011 ;24 Suppl 3: 645-8.
22. Peng ZY, Chang P, Wang H, Cen ZR, Zhou J, Liu ZG. Intensive hemoperfusion and long-term hemofitration for treatment of paraquat poisoning: a case report, Nan Fang Yi Ke Da Xue Xue Bao. 2015; 35(10): 1515-8. [Article in Chinese]
23. Chang JB, Lin CC, Chiou JF, Mau SY, Liu TZ, Chen CH. Treatment of acute paraquat intoxication using recommended megadose of vitamin C: a reappraisal. Free Radic Res. 2013; 47(12): 991-1001.