A Rare Case of “Paraquat Tongue”

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Sir,

Paraquat is a toxic herbicide with chemical formula N, N’-dimethyl-4,4’-bipyridinium dichloride.[1] It is widely used by Indian farmers for killing weeds and hence easily available in rural areas for deliberate self-poisoning. The actual ingestion of paraquat or spitting it without swallowing results in a typical tongue changes called “Paraquat tongue”[2]

A 20-year-old male was admitted to emergency department after about six hours of intentional consumption of approximately 10 ml of paraquat for suicidal purpose. Patient’s relatives brought the container named ‘Milquat’ containing 24% paraquat dichloride. Patient complained of nausea, vomiting, abdominal pain, burning of mouth and was unable to take fluid or food at the time of admission. The routine investigations revealed elevated levels of serum urea, creatinine, bilirubin and aminotransferases. On follow up, his urea and creatinine levels increased gradually till the fifth day, and then gradually decreased after hemodialysis over next seven days. Serum bilirubin level was increasing gradually till ninth day and aminotransferases level till fifth day and then started decreasing. There was no pulmonary involvement. Clinical management was done by Department of General Medicine with fluid and electrolytes, gastric lavage, hemodialysis, N-acetyl cysteine, methylprednisolone. A bedside dermatology consultation was requested on day 8 to evaluate the tongue lesion. The tongue lesions started with redness and gradually progressed to painful erosions and ulcers. Examination of tongue revealed multiple erosions and superficial ulcers, few of which were coalesced. Floor of the ulcers were covered with yellowish necrotic debris. Center of the tongue was relatively spared [Figure 1]. An upper gastrointestinal endoscopy revealed hyperemia, erosion and edema of esophagus. The tongue lesions were managed by topical application of 20% benzocaine, povidone iodine mouth gargle and topical metronidazole 1% and chlorhexidine 0.25% combination oral gel.

Paraquat causes peroxidation of lipid cell membrane and mitochondria complex I (NADH –ubiquinone oxidoreductase) by free oxygen radical leading to apoptosis.[1] The clinical manifestation of paraquat poisoning depends upon the quantity ingested. Ingestion of more than 50 ml of 20% paraquat is often fatal causing multi organ failure and death.[2] Ingestion of smaller quantity may lead to acute renal failure, hepatic failure and acute alveolitis followed by secondary pulmonary fibrosis.[2,3] Our patient did not develop any pulmonary complication but still he was advised regular follow up for any pulmonary fibrosis.

The mucosal involvement is universal in paraquat poisoning. Initially, tongue becomes erythematous and swollen but later develops erosion and ulcerations which are often covered with yellowish necrotic debris (Paraquat tongue).[4] Associated symptoms include severe pain, burning sensation, dysphagia and excessive salivation. The mucosal lesions of pharynx, esophagus, and stomach are also common and may result in perforation, mediastinitis and pneumomediastinum.[2]

A semi-quantitative test using bicarbonate and sodium dithionite can be used as a bedside test for detecting paraquat in urine and plasma.[2] The medical management includes resuscitation, gastric decontamination, fluid and electrolyte balance, hemodialysis, corticosteroids, cyclophosphamide, antioxidants like Vitamin C, Vitamin E, and N-acetyl cysteine.

The management of paraquat tongue includes daily debridement of slough, topical application of benzocaine, choline salicylate, benzalkonium chloride and contents of multivitamin capsules. Early insertion of a nasogastric feeding tube ensures adequate nutrition.[5] The serial monitoring of renal, hepatic and pulmonary functions and long term follow up are mandatory in cases of paraquat poisoning.[4] Awareness regarding paraquat tongue and high index of suspicion may provide clue to the diagnosis of paraquat poisoning.

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Nil.
Conclusion

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