Paraquat lung – a rare cause of diffuse parenchymal lung disease

Mehul Agarwal¹, Manohar Lal Gupta², Kunal Deokar¹, Neha Bharti³, Priyank Jain¹

¹Department of Pulmonary medicine, All India Institute of Medical Sciences, Jodhpur, India; ²Department of Pulmonary Medicine, Santokba Durlabhji Memorial hospital, Jaipur, India; ³Department of Anaesthesiology and Critical care, All India Institute of Medical Sciences, Jodhpur, India

To the Editor,

A 25 years old female, farmer, no co-morbidities, presented with progressively increasing shortness of breath and decreased urine output for 4 days. She had nausea, vomiting and oral ulcers 10-12 days back for which she took symptomatic treatment. She had tachycardia, tachypnea, and peripheral oxygen saturation on room air was 48%. Bilateral basal fine inspiratory crepitations were present on chest auscultation. She was put on non-rebreather face mask at 15 liter/minute oxygen flow. Arterial blood gas revealed respiratory alkalosis with hypoxemia. She was put on pressure controlled bilevel positive airway pressure support. Chest radiograph showed bilateral mid and basal haziness. Subsequently after 30 minutes she was intubated and put on mechanical ventilator in view of deteriorating respiratory distress. Laboratory results revealed haemoglobin of 7.4 g/dl, TLC – 24700/cmm. Kidney and liver function tests were normal.

Intravenous antibiotics, methylprednisolone and other supportive treatment was started. High resolution computed tomography of chest showed bilateral mid and basal ground glass opacities, traction bronchiectasis and subpleural basal honeycomb cysts.

Post intubation she developed right sided pneumothorax and subcutaneous emphysema for which intercostal tube drainage was done. Her anti-nuclear antibody test was negative and two-dimension echocardiography was normal. Clinical history was reviewed from attendants. There was no previous history of joint pain/swelling, Raynaud’s phenomenon, skin rashes, oral ulcers, dysphagia, facial swelling, loss of appetite, medication use, industrial or radiation exposure. She had an accidental ingestion of approximately 15ml of commercial grade herbicide 10-12 days back that was dissolved in a water bottle. That herbicide contained PARAQUAT. A diagnosis of Paraquat lung was made. Patient succumbed to her illness on day 3 of admission.

Use of herbicides is common in agricultural practices and their use/role is now indispensable in developing and under-developed countries. Paraquat (1,1’- dimethyl- 4,4’- bipyridinium) is the most commonly used herbicide in developing and under-developed countries due to its low cost and easy availability. It is safe as dermal, or spray exposure and causes only limited local injury/irritation without any systemic involvement1. Systemic toxic effects are seen with oral ingestion at a dose as low as 10 ml. Following ingestion, paraquat is rapidly and incompletely absorbed and is distributed in tissues, most commonly in lungs, kidneys, liver and muscle tissue. Maximum tissue levels are reached within six hours and its elimination is primarily by kidneys². Paraquat toxicity involves generation of superoxide anions leading to formation of hydrogen peroxide and hydroxyl radical, which ultimately consumes NADPH – cell’s key antioxidant defense. This oxidative stress directly causes cell damage and triggers a pronounced inflammatory response³. Symptoms of paraquat toxicity are usually dose dependent with low doses (<20mg/kg) mostly causing vomiting, diarrhea, mouth ulcers while higher doses (>20mg/kg) usually result in pulmonary fibrosis, acute renal failure and death⁴. Common
radiologic findings include dense consolidation, focal fibrotic lesions with areas of focal honeycombing. Definitive diagnosis can be made by urinary sodium dithionite test and measuring paraquat concentrations in the plasma. Our patient reported after 10-12 days of poison ingestion and since we had definite history of ingestion along with clinico-radiological findings, we did not perform these tests. There is no definitive antidote for paraquat, and management aims at avoiding systemic absorption of the toxin and supportive care. Haemoperfusion using activated charcoal or fuller’s earth when given within 4 hours of ingestion has been proven to be effective.

Paraquat poisoning should be suspected in a farmer presenting with unexplained combination of gastrointestinal, pulmonary and renal symptoms. Early suspicion and diagnosis should be followed by aggressive decontamination and other supportive measures which may help in successful management and reduce the mortality rate.

Conflicts of interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article.

References

1. Fitzgerald GR, Braniville G, Black J, Silke B, Carmody M, O’Dwyer WF. Paraquat poisoning in agricultural workers. Ir Med J. 1978;71:336-42.
2. Wunnapuk K, Mohammed F, Gawarammana I, Liu X, Verbeeck RK, Buckley NA, et al. Prediction of paraquat exposure and toxicity in clinically ill poisoned patients: a model based approach. Br J Clin Pharmacol. 2014;78:855-66.
3. Suntres ZE. Role of antioxidants in paraquat toxicity. Toxicology. 2002;180:65-77.
4. Saravu K, Sekhar S, Pai A, Barkur AS, Rajesh V, Earla JR. Paraquat – a deadly poison: report of a case and review. Indian J Crit Care Med. 2013;17:182-4.
5. Im JG, Lee KS, Han MC, Kim SJ, Kim IO. Paraquat poisoning: Findings on chest radiography and CT in 42 patients. Am J Roentgen. 1991;157:697-701.
6. Gawarammana I, Buckley NA. Medical management of paraquat ingestion. Br J Clin Pharmol. 2011;72:745-57.

Correspondence:
Received: 14 July 2020
Accepted: 20 July 2020
Kunal Deokar; MD, DNB
DM fellow
Department of Pulmonary medicine
All India Institute of Medical Sciences, Jodhpur
Email id: dkunal@live.in