Asian Journal of Pharmaceutical and Health Sciences
www.ajphs.com

Acute paraquat poisoning presenting with severe hepatorenal failure

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ARTICLE HISTORY

Received: 16.08.2013

Accepted: 15.09.2013

Available online: 10.11.2013

Keywords:

Paraquat, renal failure, hemofiltration, pulmonary fibrosis

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ABSTRACT

In India, agrochemicals are often used as poisons. However, organophosphorus compounds are more commonly used for this purpose than other chemicals. Paraquat is an herbicide with potential oxidative cytotoxic effects. We here report a fatal case of paraquat poisoning from Eastern India, presenting with acute hepatorenal failure. The patient also had severe burns of her mouth. This is probably the second report of such fatal effect of paraquat poisoning from this part of the country.

INTRODUCTION

In a predominantly agro-based economy like India, agricultural chemicals are often used as poisons, suicidal or homicidal. Different chemicals and pesticides, including Methyl parathion, methomyl, carbofuran and paraquat may be used as poisons [1]. Paraquat or Dipyridylium is an herbicide, once in common use [2]. However its use was slowly discontinued or restricted in many countries due to reports of its toxicity [2].

In India, paraquat is freely available and often used as herbicide or weedicide. However, it is less often used for poisoning, compared to other organophosphates. We here report a fatal case of paraquat poisoning presenting with acute hepatorenal failure. As far as we searched, this is probably the second report of such serious effect of paraquat poisoning from West Bengal/Eastern India.

THE CASE REPORT

An 18 year old rural unmarried female presented to a tertiary care centre of Kolkata with history of suicidal ingestion of 50 ml of weed killer liquid five days ago. The liquid contained 1% paraquat dichloride, 4% cocoamine ethoxylate, dye acid blue 0.05% and emetic dye 0.05%. There was also nonyl phenol ethylene oxide and silicon defoamer. No other poison was taken along with it. She was at first taken to local primary health centre after 12 hours. No decontamination procedure like gastric lavage was done.

The patient suffered from severe burn in mouth after ingestion of the poison. After 3 days, she developed fever and severe nausea. She was at first treated conservatively before referral. When she presented to us, she had icterus and her urine output was markedly decreased (around 500 ml/24 hours). The urine was high coloured. On examination, we found right upper quadrant tenderness and coated tongue. Her sensorium was intact and she had no bleeding manifestation. Laboratory tests revealed hemoglobin of 10 gm/dl with total leukocyte count of 11000/cmm (neutrophil 88%). The platelet count was 1.82 lakhs/cmm. Blood sugar was 75 mg/dl with urea/creatinine of 80 and 4.4 mg/dl respectively. By 2nd day of admission, the urine output had decreased to 100 ml/24 hours. Liver function test revealed bilirubin 9.4 mg/dl, SGOT/SGPT 248/285 IU/L (N :< 40 for both), alkaline phosphatase of 225 IU/L (N: <306) and serum albumin of 2.9 g/dl. Prothrombin time was 18 seconds (control: 11 sec). Electrolyte assay revealed Na/K: 134/3.4 mEq/L. The patient was becoming drowsy. Blood gas analysis revealed pH of 7.31 with serum bicarbonate of 17 mEq/L. Blood and urine cultures were negative for any organism. Chest X ray and ultrasonography of abdomen were normal. Toxicology screen for common poisons like paracetamol and opiates were negative.

The patient was immediately referred for hemofiltration, in

view of the deteriorating hemodynamic status. She underwent one session only. However, by 3rd day, her serum bilirubin had increased to 15 mg/dl and she was in deep coma. The urine output over 24 hours became 30 ml. In spite of best efforts of all concerned, she passed away on the 3rd day.

DISCUSSION

Paraquat is a quaternary nitrogen compound, which acts as a contact poison for plant tissues [3]. This compound blocks conversion of NADP to NADPH, and thus causes formation of oxygen free radicals [3]. However, the exact site of action of this chemical in human body is still debated [4]. While some authors favour the microsomal origin of free radicals, some other authors have found the mitochondria to be the main site of damage [4]. Also, activation of NMDA receptors and ADP-ribosylation are some other probable mechanisms of toxicity of this compound in different organs like liver [4].

Paraquat toxicity is known to cause skin and mucosa burns [3]. Our patient also had severe burns of the tongue and pharynx after ingestion of the poison. Contact with skin or eyes may also cause severe injuries [3]. There is no specific treatment for the burns. Only symptomatic washing with solution containing diphenhydramine, aluminium hydroxide, magnesium hydroxide and lignocaine may bring some relief [5]. However, when a patient presents with mouth burns, further investigations like straight X ray abdomen should be done to look for potential G.I. complications like perforation [5]. Systemic effects of paraquat include hepatic and renal failure, pulmonary toxicity and convulsions [6]. While hepatotoxicity is mainly related to direct pharmacological effect of the chemical on hepatocytes, renal failure is caused by many factors like hypovolemia, acute tubular necrosis or septicaemia [3]. Anoxic brain damage is also reported rarely after paraquat ingestion [7]. However, the main cause of death remains acute hepatic or renal failure or gastrointestinal perforation [5].

Liver injury due to paraquat is biphasic. While the injury is mainly hepatocellular to begin with, in later stages, there is also cholangiocellular damage and cholestasis [8]. Our patient had mainly a rise in SGOT and SGPT. Abdominal imaging also did not show cholestasis. The renal failure in this poisoning may be oliguric or non oliguric [9]. Rarely, a Fanconi syndrome like picture may occur a few days after the poisoning. Usually, renal failure is associated with concomitant pulmonary toxicity [3]. However, our patient did not manifest any such changes, probably due to the rapid clinical deterioration.

Treatment for paraquat poisoning is mainly supportive. There is no specific antidote [3, 10]. A variety of treatments like antioxidants, N acetyl cysteine and immunosuppressives like cyclophosphamide have been used in different studies. But the results are not encouraging [10]. Hemoperfusion may help in certain situations. Progressive pulmonary toxicity may be a rare yet fatal late complication [10].

The prognosis following Paraquat poisoning may be assessed by a clinical scale called SIPP [11]. This requires a serum paraquat level, which is often not available in our setting. In these cases, other critical care scales like MSAPS II may be used [11]. However, serum levels, if available, are still the best prognostic marker. Paraquat poisoning is rarely reported from India [3]. The main reported cases depict kidney injury and respiratory failure to be the principal manifestations [12]. Hepatic failure is rarely found in India. A case similar to ours, with hepatorenal failure, was reported from Rajasthan [3]. That patient also died rapidly of multiorgan failure. In another case series from north India, high serum bilirubin was found in 5 cases of paraquat poisoning [13]. However, alterations in other components of liver function test have not been documented there. Usually, if the patient survives, the liver function normalizes by itself [13]. A case of Paraquat poisoning was reported from rural West Bengal recently. The cause of fatality there was acute lung haemorrhage and respiratory failure [14].

CONCLUSION

Our case highlights the need for early aggressive management of this comparatively rare poisoning. Since treatment is mostly supportive, there should be a multidisciplinary approach in treatment of this condition.

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