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A RARE CASE OF SURVIVAL FROM ACUTE PARAQUAT POISONING

Pediatrics

Dr.Unnati Shah 2nd year resident in department of paediatrics, Sumandeep University - Corresponder
Dr.Sunil Pathak Assistant Professor in department of paediatrics, Sumandeep University
Dr.Dulari Gandhi Professor and head of department of paediatrics, Sumandeep University

ABSTRACT

Introduction: Paraquat (1, r-dimethyl-4,4'-bipyridium dichloride), a brown syrupy liquid is an effective herbicide that has low chronic toxicity with poor prognosis.at present there is no specific antidote to paraquat poisoning.
Case report: A 15 years male child was admitted to the emergency room Dhiraj hospital with an alleged history of accidental ingestion of unknown quantity of paraquat at his home. He had complain of throat pain and burning sensation of throat and stomach. There were two episode of vomiting after 2 hours of ingestion. On admission he was conscious and oriented. On physical examination patient was afebrile with HR 122/min, RR 60/MIN, SPO2 85% on room air with 2 lit o2 SPO2 95% with hypertension (140/98) >95th percentile. Pupils were normal sized and B/L reactive to light. On systemic examination auscultation of lungs revealed B/L crepitation with mild respiratory distress, per abdomen hepatomegaly, and CVS examination revealed loud P2 with tachycardia. After 25 days of ingestion he developed focal seizure involving left Upper limb and left lower limb.
Conclusion: Paraquat a herbicide which mainly involves lungs and kidney leading to pulmonary fibrosis and acute kidney injury. There is not specific antidote of this poison. Treatment remains supportive. Antioxidant vitamin E vitamin C and N acetyl cysteine are helpful in preventing the damage cause by free radicals. Oxigen is contraindicated in its treatment.

KEYWORDS:

Paraquat (1, r-dimethyl-4,4'-bipyridium dichloride) Poisoning

INTRODUCTION

Paraquat (1, r-dimethyl-4,4'-bipyridium dichloride), is a brown liquid, is an effective herbicide .It has low chronic toxicity because when It comes in contact with soil, its rapid deactivation occurs.[1-2] Although its uncommon, paraquat ingestion can cause severe and often fatal toxicity. Although paraquat is widely available, reports of this poisoning are very uncommon in India. Fatal dose of paraquat is very small (>10 ml) poison can cause damage lungs permanently. No any diagnostic test is available, therefore diagnosis is difficult. Most common route of its exposure is inhalation[3].

Case Report

A 15 years male child was admitted to the PICU with an alleged history of accidental ingestion of unknown quantity of paraquat at his home. He had complain of throat pain and burning sensation of throat and stomach. There were two episode of vomiting after 2 hours of ingestion. On admission he was conscious and oriented. On physical examination patient was afebrile with pulse 122/min, RR 60/MIN, SPO2 85% on room air, with 2 litre O2 SPO2 increased to 95% and hypertension (140/98mmHg). Pupils were normal sized and bilaterally reactive to light. On examination of respiratory system there was tachypnea, intercostal and subcostal retraction, on auscultation bilateral crepitation were present. Abdomen was soft, nontender with hepatomegaly, and CVS examination was normal except loud P2. Laboratory finding s/o high urea (129)and high creatinine(2.3) which was reach to maximum on day 8th and then gradually become normal(23,0.6 respectively) on day 29. He was given a symptomatic treatment. General condition was improved. Tachypnea subsided in 1 week. On 25th days of ingestion he developed one episode of focal seizure involving left upper limb and left lower limb that lasted for 3-5 minutes. Chest x ray- showed linear fibrotic opacity seen in left upper zone and bilateral middle zone. USG chest-bilateral consolidation. 2D-echo was suggestive of mild tricuspid regurgitation, mild pulmonary artery hypertension (PAH). MRI and EEG was not done because of unaffordability.

On follow up clinical condition of patient was better, his blood pressure was 110/68 mmHg, no convulsion after discharge. Chest x-ray became normal after 6 month. Renal function test normal. Antihypertensive and Antiepileptic medications were tapered and stopped after 6 months.

TREATMENT CHALLENGES

When patient come to us we didn't know what kind of poison patient ingested, so to prevent respiratory distress we had start oxygen but within few hours, after going through literature, we came to know about nature of poison and as Oxygen is contraindicated so despite of tachypnea (RR-60) we have to stop oxygen . When patient's general condition was improved and patient was fully stable he was discharged on phenytoin, Nifedipine and Losartan, N-acetyl cysteine, Vitamin C, Vitamin E and Pantoprazole.

DISCUSSION

When consumed orally, Paraquat accumulates in the lungs and it leads to a release of hydrogen and superoxide anions, it causes lipid peroxidation and damage to the cell membranes. Paraquat toxicity produces both local and systemic effects. As seen in this case, paraquat ingestion caused throat pain and burning sensation of throat and stomach. There is free radical release by paraquat, that causes hepatotoxicity, nephrotoxicity and pulmonary fibrosis.[4] In this case on systemic examination there was respiratory distress, hepatomegaly, and loud P2. Oliguria suggestive of involvement of renal system. On 25th days he developed focal seizure involving left Upper limb and left lower limb s/o central nervous system involvement.

The pulmonary manifestations begins with diffuse consolidation, after several days later into cystic lesions and focal fibrotic lesions with high mortality.[4] In fatal cases of paraquat poisoning, findings ranges from pulmonary congestion, pulmonary oedema, haemorrhage and to extensive pulmonary fibrosis. Large amounts ingestion leads to multiorgan failure and cardiogenic shock and is uniformly fatal. Its plasma concentration of >1.6 pg. /ml 12 h after ingestion is universally fatal. There is no specific antidote for paraquat poisoning, therefore general management for poisoning was initiated. Supportive treatment was started. N acetyl cysteine, Vitamin C and E were given to avoid free radical injury to lungs along with pulse methyl prednisolone for 5 days followed by low dose oral prednisolone to prevent pulmonary fibrosis.[3] As the present case had renal failure, we could not use cyclophosphamide. Although the use of oxygen can increase the Paraquat toxicity, it should be given in the hypoxic patients in low concentration.[3] In spite of advances in medical care, treatment and supportive care, mortality is very high (due to multiorgan system failure) in patients with paraquat poisoning. Although there have been isolated case reports of survivors (due to the small dose and early treatment)[5], an ingestion of a high dose of poisoning has a poor prognosis. When it has been ingested, decontamination in the form of gastric lavage should be given to prevent further absorption.

Conclusion

Small amount of paraquat poisoning can have fatal outcome, so early diagnosis and aggressive management can reduce the risk of mortality. It mainly involves lungs and kidney leading to pulmonary fibrosis and acute kidney injury. There is no any specific antidote of this poison so treatment remains supportive in nature, and mortality rate is very high.

Antioxidant N acetyl cysteine, vitamin E and vitamin C are helpful in preventing the damage cause by free radicals. Oxygen is contraindicated in its treatment. So we shall always think about this poison in any patient with unexplained respiratory and renal damage after consuming unknown poison.

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