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Auramine-o and Malachite Green Poisoning: Rare and Fatal

Hisham Md[1], Vijayakumar A[1]*, Rajesh N[1], Sivakumar M.N[2]

[1] Drug and Poison Information Center, Department of Pharmacy Practice, KMCH College of Pharmacy, Coimbatore, Tamil Nadu, India

[2]Intensive Care Medicine, Kovai Medical Center and Hospital, Coimbatore, Tamil Nadu, India

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ABSTRACT

Traditionally it was believed that the cow dung has germicide property. In modern era, due to unavailability of actual cow dung people started using commercially available synthetic one. The synthetic cow dung powder called 'Sani Powder' in local parlance used to clean courtyards, house and temple premises. It is a dye which can be a lethal poison with no available antidote. Cow dung powder is available in two different colors: yellow powder (Auramine-O) and green powder (Malachite Green), commonly used in rural Tamil Nadu (South India) in the districts of Coimbatore, Tirupur and Erode. Even though the sale is legally banned, the powder is easily available in grocery shops. High toxicity profile of the dye is evident from limited toxicological data demonstrated from clinical and experimental studies. Auramine is life threatening as it is neurotoxic and also causes severe hepatic damage. Animal and observational research confirms that Malachite Green is multi-organ toxin with delayed toxicity. Very few cases have been reported with Auramine poisoning while there are no references so far about Malachite Green poisoning.

Keywords: Auramine poisoning, Dye, Hepatotoxicity, Malachite Green, Neurotoxicity.

INTRODUCTION

Ancient history of south-Indian culture regarding their personal hygiene and community welfare etc is mentioned in many places in the literature. During that period, people cleaned the living premises using cow dung but now people use commercially available powder which is synthetic and in local parlance known as 'Sani Powder' and is commonly used in South-India (Tamil Nadu) to clean courtyards, house and temple premises. It is used as a germicide but the chemical being used is a dye. It is basically composed of Auramine-O (diaryl methane dye) as yellow powder and Malachite Green (triphenyl methane dye) as green powder. Though the sale is legally banned, the powder can be cheaply purchased from grocery shops at Rs.5 per packet. In rural Tamil Nadu (South India), especially in the districts of Coimbatore, Tirupur and Erode cow dung powder is commonly used as a suicidal poison. There is no specific antidote for these dyes. It has a very high toxicity profile due to which death occurs within hours of ingestion. Auramine is a neurotoxic poison which causes CNS depression which is clearly manifested from the low Glasgow Coma Scale (GCS). Severe hepatic damage as a result of centrilobular necrosis due to toxic metabolite related toxicity may be illustrated by jaundice, upper abdominal pain, and vomiting. Auramine being a cationic dye causes severe ocular injury on eye contact and damages the gastrointestinal mucosa on ingestion. Chronic effects of Auramine dye include carcinogenicity, mutagenicity and its long term inhalation leads to pneumoconiosis. Malachite Green is multi-organ toxin which shows delayed toxicity. Rarely do these cases get referred to tertiary or teaching hospitals which add to the reason why synthetic cow dung poisoning is not reported in literatures.[1]

Address for Correspondence:

Vijayakumar. A, Drug and Poison Information Center, Department of Pharmacy Practice, KMCH College of Pharmacy, Coimbatore, Tamil Nadu, India – 641014.

E-mail: vijayspharm@gmail.com

CASE REPORT 1

A 19 year old male ingested yellow sani powder with the intention of committing suicide. He was taken to a nearby government hospital with complaints of yellow discoloration of oral cavity, epigastric pain and abdominal discomfort. Gastric lavage was given and he was referred to a higher center. He was brought to the hospital after an hour. On examination he was conscious, tachycardic with a pulse rate of 140beats/min, blood pressure110/80mmHg, respiratory rate was 40 breaths/min, SpO_2 89%, GCS was 10/15 (E4 V2 M4) and he was icteric. Ultrasound (USG) abdomen revealed mild hepatomegaly, had no transaminitis and the patient was hyperglycemic. ABG analysis showed metabolic acidosis with hypoxia. He was closely monitored and supportively managed in ICU for two days. On the fourth day he had acute hepatitis with SGPT of 693U/L (normal upto 37U/L) and total billirubin of 4.36mg/dL (normal upto 1mg/dL). His coagulation parameters were also prolonged with Prothrombin time 21 seconds (control 14 seconds) and INR 1.72. He was treated with intravenous injection of Vitamin K. Progressively on the sixth day patient had vomiting and severe transaminitis with SGPT of 2050U/L. On further treatment, the SGPT decreased to 895U/L and total bilirubin decreased to 1.08mg/dL as on the ninth day. Patient was stabilized and discharged on the 10th day.

CASE REPORT 2

A 31 year old woman consumed unknown quantity of yellow cow dung powder as a suicide attempt. She was rushed to a nearby hospital and gastric lavage was given. She was referred to a tertiary hospital for higher care. On the way, she had developed recurrent episodes of seizure (status epilepticus). She was brought to the hospital unconscious and in gasping state. She also had frothy secretions from her mouth. On examination, her pulse rate was 139beats/min, blood pressure 140/60mmHg, SpO_2 was 67% and GCS was 4/15 (E1 V1 M2). She was intubated, revived and shifted to ICU. She was hypoxic with aspiration pneumonitis and ABG analysis showed metabolic acidosis. Her coagulation parameters were prolonged with Prothrombin time 20 seconds (control 14 seconds) and INR 1.65. She was hyperglycemic and had no transaminitis. She responded well to treatment and weaned from ventilator after 16 hours on the second day and shifted to room. On the fourth day, she had severe transaminitis with SGPT of 644U/L and total billirubin of 6.72mg/dL which gradually reduced later. She was stabilized, slowly recovered and was discharged on the sixth day.

CASE REPORT 3

A 28 year old female was brought to our hospital an hour after consumption of green cow dung powder in an attempt to suicide. She had complaints of vomiting within 5 minutes of consumption and burning sensation over the abdomen. On examination, she was conscious, pulse rate 116beats/min, blood pressure 90/60mmHg, respiratory rate 24breaths/min, SpO_2 was 98% and GCS was 15/15. Next day she had cough with expectoration, bilateral wheeze on auscultation and bronchitis. Her cough persisted for four days. She also had episodic chest pain and discomfort. She had no transaminitis. Patient was closely monitored, stabilized with intravenous fluids, antibiotics and bronchodilators, counseled and discharged on eighth day.

DISCUSSION

Auramine-O is a yellow dye with a molecular formula of $C_{17}H_{21}N_3$.HCl. Toxicological data demonstrated in different animal models show acute oral LD_{50} of 150-1500 mg/kg. Other studies also confirm DNA damage induced by Auramine in liver, kidney, and bone marrow of rats and mice, and in human cell line. Eye contact of Auramine (cationic dyes) produces a range of injuries from conjunctival oedema, hyperemia and purulent discharge to total opacification. Chronic health effects after exposure to Auramine are carcinogenic and mutagenic with higher incidence of bladder cancer, lymphatic cancer and also cause reproductive damage in humans.[2] Long term inhalation causes pneumoconiosis which results from sedimentation of particles less than 0.5 micron in the lungs. Surprisingly, there are no cautionary labels on the packet despite being a known poison in the locality. Even though it is legally banned, the poison is widely available in market and no step was taken to prevent it.

Acute exposure initially shows neurological features like convulsions, non-specific muscle cramps, spasms, focal deficit and coma. Except for any primary focal neurological deficit, seizures are one of the deadly events caused by many poisons. The sudden onset of seizure episode in poisoning signifies the involvement of both the cerebral hemispheres. Direct CNS effect of the poison is clearly evident from the low GCS score of the patients. Patients show severe jaundice and late transaminitis due to centrilobular necrosis of liver and biliary stasis. Centrilobular necrosis is often dose-related or toxic metabolite related hepatotoxicity. The damage spreads outward from the middle lobe of the liver. Mild necrosis is due to small amounts of parenchymal damage with asymptomatic transaminitis. Severe necrosis is accompanied either by nausea, vomiting, upper abdominal pain and jaundice. [3] The yellow dye is a GI tract irritant which causes damage to the mucosal membrane hence, causing epigastric pain and discomfort.

The similarity between the two yellow cow dung poisoning were tachycardia over a short duration of time, metabolic acidosis and they both were hyperglycemic. These were not reported in any literatures before.

Malachite Green has a molecular formula of $C_{23}H_{25}ClN_2$ and is traditionally used as dye. It's also used as a parasiticide in fish industry. No human experimental and toxicological studies are available. Chemical safety data demonstrated in animal (mouse) model shows acute oral LD_{50} of 80-120 mg/kg.[4] Interestingly rats fed malachite green illustrated a dose related liver damage along with hepatic tumors and lung adenomas.[5] Clinical and experimental observations reported so far revealed that malachite green is a multi-organ toxin and takes a while longer to affect the organs. It is very difficult to state whether the cough with expectoration, bilateral wheeze, chest discomfort and bronchitis in the case report 3 is due to the direct action of the toxin in the respiratory system. Also there is no case report published on Malachite Green toxicity in humans.

CONCLUSION

The treatment of poisoning caused by an uncommon compound is a challenge and the situation becomes graver when the patient does not respond properly on treatment. Further studies are necessary to elucidate this rare and fatal poison in a broader aspect and this article will serve as a guide for future research. The authors wish to highlight the easy availability of such fatal poisons must be banned completely

from the market and necessary action should be taken against those who sell them.

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