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Cow Dung Powder Poisoining

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Abstract
In India, Cow is considered auspicious and is worshipped as the "Mother of all Gods". Cow-dung has spiritual significance and used as a cleansing agent due to its germicidal properties, mosquito repellent and as a bio-gas. Due to unavailability of natural cow dung due to modernization, synthetic cow dung powder is being prepared which is known as "Saani powder" in southern parts of India. Being a highly toxic compound it is known to cause multi-organ failure and death eventually. It has become a common cause of suicidal poisoning among rural people as it is easily available at a low cost. As there is no specific antidote for this poisoning, early recognition of cases and prompt symptomatic treatment becomes crucial in reducing mortality and morbidity to a greater extent and preparedness for the future.

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I. Introduction

'Saani' powder is available in two varieties: 1. Yellow powder: Auramine-O, chemically known as Diaryl methane dye. 2. Green powder: Malachite Green, chemical constituent being triphenyl methane.[1] Auramine O is a fluorescent stain and appears as yellow needle like crystals and is soluble in water and ethanol. Due to very high toxicity profile and unavailability of a specific antidote death occurs within hours of ingestion. Lethal dose is considered to be 0.5mg/kg body weight.[2] Auramine is a neurotoxic poison leading to CNS depression. The lethal effect of this agent is attributed to hepatotoxicity and fulminant hepatic failure as a result of centrilobular necrosis due to toxic metabolite may be illustrated by jaundice, upper abdominal pain, and vomiting.[3] Auramine is a cationic dye causes ocular injury on eye contact and damages the gastrointestinal mucosa on ingestion. It is considered to be a pro-carcinogenic inducing DNA damage in liver, kidney and bone marrow cells in vivo.[6] Malachite Green is multi-organ toxin which shows delayed toxicity. The stages of hepatotoxicity were similar in saani powder poisoining and acetaminophen toxicity which led to observational studies on the role of NAC to counteract the toxic effect of the chemical and prevention of multiorgan damage.[4]

1ststage: <24hours-aymptomatic/gastrointestinal irritation. 2nd stage: 24-72hours: alteration in LFT 3rd stage: >72hours: until resolution of symptoms/death

Early deaths usually on day 1 are due to cerebral edema leading to convulsions, coma, cardiorespiratory arrest

Deaths on day 2 and 3 were due to hepatotoxicity, fulminant hepatic failure, hepatic encephalopathy, coagulopathy

Ursodeoxycholic acid was useful in many patients due to its choleretic, immunomodulatory and antiapoptotic properties. [5]

Role of NAC was quoted in the literature due to its antioxidative effects, but none of the literature quoted NAC as an antidote which requires large study population to prove the efficacy of the drug.NAC has been tried inpatients presenting within 24hours of consumption of saani powder at doses recommended for acetaminophen toxicity i.e a loading dose of 150 mg/kg over 60 min, followed by 50 mg/kg for next 4 hours, 100 mg/kg for next 16 hours- in 5% Dextrose solution] and studies proved a significant reduction in the morbidity and mortality.[3,5]

II. Case Report

A 17year old male has ingested yellow saani powder about 2 packets of 5gm each mixed with water with the intention of committing suicide. He was taken to a nearby government hospital in about 1hour of ingestion with complaints of yellow discoloration of oral cavity, abdominal discomfort and multiple episodes of vomitings. Gastric lavage was given, ursodeoxycholic acid was started and was referred to our center after 2days in view of deranged LFT. On examination patient is conscious, oriented and yellowish discolouration noted all over the body, ABG analysis was within normal limits. Investigations at the earlier hospital suggested a mild increase in the bilirubin, SGOT and SGPT levels on day 2, these values continued to increase on day 3 which was the day of presentation to our hospital, where we started NAC infusion and continued ursodeoxycholic acid. There was mild elevation in PT and INR but the patient did not have any signs of

coagulopathy. Bilirubin levels, SGOT and SGPT showed a decreasing trend from day 5, However yellowish discolouration of skin persisted. Patient was discharged in stable condition on day 10.

III. Discussion

Auramine causes centrilobular necrosis of liver. It is also a gastrointestinal tract irritant causing mucosal damage, epigastric discomfort and vomitings.[1] Our patient had consumed yellow cow dung powder, he had features of toxic hepatitis from day three of poison intake. This is similar to a study by Md H *et al*. who observed toxic hepatitis from day four of poison intake in their patients.[2] In our patient, SGOT and SGPT and bilirubin levels were elevated but the coagulation parameters were normal. In contrast, Md H *et al*. had observed that apart from SGOT and SGPT, bilirubin levels and coagulation parameters were also elevated.[2]

Our patients had yellowish discoloration of skin from day one of poison intake, but elevation in the bilirubin, SGOT and SGPT levels were observed from day three. Hence, the discoloration of skin could be due to deposition of powder in different parts of the body as observed by Krishnamoorthy *et al*.[4]

Auramine is a neurotoxic poison which causes central nervous system (CNS) depression.[1] Md H *et al*. also had observed poor GCS in one of their patients requiring intubation.[2] Our patient was neurologically and hemodynamically stable since the day of presentation, this can be attributed to the early gastric lavage done at the primary health care centre where the patient initially presented which led to removal of agent from the body.

Tachycardia, metabolic acidosis, and hyperglycemia observed by Md H *et al*. were not seen in our patient. [3]

Krishnamoorthy *et al*. in his retrospective analysis of cow dung powder poisoning had listed number of patients with multisystem involvements.
[4] However, our patient did not have multisystem involvement.
There is no specific antidote for these dyes. [1] Supportive therapy is the mainstay of treatment.

Ursodeoxycholic acid was started at the PHC before referring the patient to our centre inspite of which we observed an elevation in the bilirubin, SGOT and SGPT levels, we started the patient on NAC infusion after which the bilirubin, SGOT and SGPT levels began to decreasing and daily monitoring showed a decreasing trend.

Alkalization of urine keeps the dye in ionized state there by reducing the distribution of toxin in the body-decreasing the nuerotoxicity, promotes the solubility of the dye and enhances its renal excretion.[2] Continuous infusions of sodium bicarbonate are recommended to prevent the toxicity of the poison.[4]

Strong recommendations for Early GASTRIC LAVAGE, NAC Infusion and URINE ALKALIZATION to form the mainstay of treatment protocol along with other supportive care.

DNA damage in the liver, kidney and bone marrow induced by auramine in animal studies and carcinogenesis involving bladder, lymphatics and reproductive system on chronic exposure in humans.[6]

IV. Conclusion

NAC trial regimen gave hope of survival in patients with saani powder poisoining and found to prevent further liver damage.
More studies are required to prove the efficacy of sodium bicarbonate in reducing cerebral toxicity.

Large number of patients needs to be studied to know more about the mechanism of action, clinical presentation, and management of cow dung powder poisoning and a standard treatment protocol should be implemented to reduce morbidity and mortality.

The easy availability and illegal sale of such deadly poisons must be banned from the market and strict legislative actions must be ensured.

Public awareness, psychosocial councelling of the patients and curtailing its availability for public use is an urgent need of the hour.

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