Case Report

Cow dung powder poisoning

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Abstract

Cow dung, which has germicidal property, was used in ancient days to clean living premises in South India. Nowadays, people are using commercially available synthetic cow dung powder. It is locally known as "saani powder" in Tamil Nadu. It is freely available in homes and is sometimes accidentally consumed by children. It is available in two colors - yellow and green. Cow dung powder poisoning is common in districts of Tamil Nadu such as Coimbatore, Tirupur, and Erode. We report two cases of yellow cow dung powder poisoning from our hospital.

Keywords: Auramine, cow dung powder, malachite green

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Introduction

Cow dung, which has germicidal property, was used in ancient days to clean living premises in South India. Nowadays, people are using commercially available synthetic cow dung powder.[1] It is locally known as "saani powder" in Tamil Nadu. It is freely available in homes and is sometimes accidentally consumed by children. It is available in two colors -yellow and green.[1,2] Cow dung powder poisoning is common in certain districts of Tamil Nadu such as Coimbatore, Tirupur, and Erode and many deaths have been reported due to this.[1,3] However, there are very few articles in literature regarding this poisoning. Hence, the mechanism of action, clinical presentation, and cause of death is not clearly known.

Case Reports

Case 1

A 13-year-old-female with H/O yellow cow dung powder intake was initially taken to a peripheral hospital. She had multiple episodes of vomiting and yellowish discoloration of skin. Hence, she was referred to our hospital on 3rd day. O/E she was conscious, oriented. Her systemic examination and vitals were normal. She had yellowish discoloration of the skin – more on face and hands. Investigations on admission such as complete hemogram, renal function test (RFT), serum electrolytes, and coagulation profile were normal. Liver function tests (LFT) showed normal bilirubin levels, elevated serum glutamic oxaloacetic transaminase (SGOT), and serum glutamic pyruvic transaminase (SGPT) values (SGOT – 1040 and SGPT – 1125). She was started on antiemetics, proton pump inhibitors, steroids, and Vitamin K. Medical gastroenterologist opinion was obtained, and ursodeoxycholic acid and metadoxine were added. Next day, her SGOT and SGPT values started decreasing (SGOT – 125, SGPT – 744). Other blood investigations were normal. However, yellowish discoloration of the skin persisted. She was totally asymptomatic and discharged 2 days later.

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Case 2

A 36-year-old-male with H/O yellow cow dung powder intake was taken to a peripheral hospital for initial management and was referred to our hospital on the same day. He had multiple episodes of vomiting while shifting. On examination, he was unconscious, not responding to painful stimuli. His Glasgow coma scale (GCS) was $E_1V_1M_3$. Pupils were 2 mm, equal and reacting to light. He had yellowish discoloration of the skin. Cardiovascular and respiratory system examination were normal. His saturation and vitals were normal. Due to poor GCS, the patient was intubated and mechanical ventilation was started. His investigations such as hemogram, RFT, and LFT were all normal. Stomach wash, activated charcoal, antibiotic, antiemetic, PPI, and steroid were started. Next day, he regained consciousness, was slowly weaned and extubated on day three. His blood investigations on day three were normal except for elevated SGOT and SGPT values (SGOT – 87 and SGPT – 259). Medical gastroenterologist opinion was obtained and ursodeoxycholic acid was given. He was discharged on day four.

Discussion

Two types of cow dung powder are available – yellow powder (AURAMINE O - diarylmethane dye) and green powder (malachite green - triphenylmethane dye).[1]

Krishnamoorthy *et al*. in his retrospective analysis of cow dung powder poisoning had just listed the number of patients developing various system involvements. However, the clinical presentation and biochemical abnormalities were not discussed.[4]

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

Our patients had yellowish discoloration of skin from day one of poison intake, but bilirubin levels were normal. SGOT and SGPT elevations were observed only on 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] One of our patients had poor GCS requiring intubation. He was hemodynamically stable, and other system examinations were normal. Next day, he regained consciousness and was extubated on day three. Hisham *et al*. also had observed poor GCS in one of their patients requiring intubation. Direct CNS effect of the poison is clearly evident from the low GCS of the patients.[1]

Tachycardia, metabolic acidosis, and hyperglycemia observed by Hisham *et al*. were not seen in our patients.[1]

There is no specific antidote for these dyes.[1] Supportive therapy is the mainstay of treatment. Ursodeoxycholic acid was tried in both our patients because in addition to its choleretic effect, it also has immunomodulatory and antiapoptotic properties.[5] More studies are needed to determine the management of toxic hepatitis and the role of N-acetyl cysteine in cow dung powder poisoning.

Even though other organs looked unaffected in our cases, more number of patients should be studied before concluding.

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 can produce injuries such as conjunctival edema, hyperemia, purulent discharge, and total opacification. Chronic health effects of auramine exposure are carcinogenic and mutagenic with higher incidence of bladder cancer, lymphatic cancer, and also cause reproductive damage in humans.[6]

Conclusion

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.

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Conflicts of interest

There are no conflicts of interest.

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