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# Retrospective Analysis of Synthetic Cow Dung Powder Poisoning.

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**Abstract:**
**Introduction:** Synthetic cow-dung powder poisoning is a very common cause of suicidal poisoning among rural population of south India. Yet only a few cases have been reported so far. Auramine-O and Malachite green is highly toxic dyes which are the main constituents of synthetic cow-dung powder leadingfrom vomiting to multiple organ failure. As there is no specific antidote timely management following systematic protocol becomes necessary to reduce mortality and morbidity due to this deadly poison. There is a scarcity of literatures not only documentation of these cases but also in assessing the severity and outcome following treatment.**Aims and Objectives**: Our aim was to study the clinical features, laboratory results, treatment and outcome in Synthetic cow-dung powder poisoning and to assess the severity based on these parameters.**Materials and methods:**This study was conducted retrospectively at a rural based 450 bedded District hospital, Chamarajanagara, India. All the confirmed cases of Synthetic cow-dung powder poisoning were studied. Patient's clinical presentations and routine investigations like Complete Blood Counts, Liver Function Tests, Renal Function Tests, Arterial Blood Gas Analysis, Electrocardiogram, Ultrasound Abdomen and CT Brain were analyzed. Treatment charts and discharge summaries were studied in detail to assess the severity of outcome.**Results:**Out of a total of 310 suicidal poisoning cases, 35 were due to synthetic cow-dung powder poisoning (11.29%). The majority were aged between 20 to 40 years old with a median age of 30. Females were commonly affected with female to male ratio of 5:1.Toxicity analysis revealed Auramine-O powder in 78.0% (27/35) of the cases while Malachite green in 22.0% (08/35) of the cases. 77.14% (27/35) patients presented to hospital within fours of toxin ingestion and all of them recovered well. Jaundice was present in most of the cases 80.0% while raised urea: creatinine ratio in 20.0% and low Glass-glow Coma scale in 14.2% of the cases. Apart from symptomatic treatment, Hepatic and renal impairment was managed with N-acetyl Cystenine and Prednisolone therapies appropriately.**Conclusions:**Early recognition of clinical signs with symptoms and assessing the severity of Synthetic cow-dung powder poisoning is of

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paramount importance in predicting the outcome. Illegal sales and promotion should be completely banned and warrant strict legal action.

**KEYWORDS: POISONING, COW DUNG, SYNTHETIC , MORBIDITY**

**Introduction:**

In India, cattle are worshipped as the "mother of all Gods "and Cow-dung is considered auspicious especially among the rural people. Traditionally it was used as a cleansing agent not only to clean household courtyard and kitchen but also to cleanse temple premises. The cow-dung powder is also used as a mosquito repellent and as bio-gas for cooking purposes. Recently this organic form of cow-dung powder is being replaced by synthetic form due to unavailability and modernization. The synthetic cow-dung powder is commonly known as "Saani powder" in southern parts of India. Chamarajanagar, being the border district of Karnataka and Tamil Nadu shares close geographical and socio-cultural practices. It is a highly toxic compound and known to cause multi-organ failure eventually death. [1,2]

Due to its low cost and easy availability, synthetic cow-dung powder poisoning has become a common cause of suicidal poisoning among rural people. It mainly comes in two varieties: Auramine-O and Malachite green which is chemical dyes and associated with many side effects. [2 ]. Auramine-O is a potent neuro-toxic and hepato-toxic agent. It also causes ocular and gastro-intestinal mucosal injuries. Exposure to Malachite green is associated with multi-organ dysfunction and cardio-toxic. Chronic exposures are associated with pneumoconiosis and carcinogenesis [1,3].

Krishnamoorthy et al had observed various systems involvement [4] . Although "Saani powder" is legally banned, it is still widely available at many grocery shops across the rural belts. Most of the cases present to the hospital very late or go unnoticed. There is a specific antidote for this poisoning [5 ]. Early recognition of cases and prompt symptomatic treatment becomes crucial. However, literature is scarce on documentation of these cases and assessing their severity. In this study an attempt has been made to study the cases of synthetic cow-dung powder poisoning retrospectively, categorize clinical signs and symptoms and analyze the laboratory findings thereby, assessing the severity of this poisoning and predicting the outcome of treatment. Systematic approach and time management helps to reduce mortality and morbidity to a greater extent and preparedness for the future.

**Materials and methods:**

Three years of retrospective data from July 2017 to June 2020,were collected from the medical record department of rural-based District hospital, Chamarajanagar, Karnataka, India.

All cases of synthetic cow-dung powder poisoning were included in this study. Poisoning due to other toxins, combined toxins were excluded. Patients discharged against medical advice and brought dead were also excluded from our study. All the relevant data like clinical presentation, signs, symptoms, clinical examination findings were noted down. Laboratory investigations like Complete blood counts, Liver function tests, renal function tests, Arterial blood gas analysis (ABG), Electrocardiogram (ECG) were analyzed carefully. Ultrasound abdomen & CT brain was done in a few cases. Treatment monitor charts of each patient, discharge summary, and follow advice were studied. Clinical history was also collected from patients who had already recovered from this poisoning.

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Based on the clinical presentations, laboratory reports, and response to treatment, the outcomes of cases were analyzed systematically. Using SPSS software results were correlated and meaningful conclusions were drawn.

**Results:**

Out of a total of 310 suicidal poisoning cases, 35 (11.29%) were due to synthetic cow-dung powder poisoning. There were 29 (82.8%) females and 06 (17.14%) males with female to male ratio of 5:1. Among 35 patients, Mean patient age was 30.9 years (Standard Error of Mean is 1.022 and Inter Quartile Range is 9)

Toxicity analysis revealed Auramine-O powder in 78% (n=27) of the cases while Malachite green in 22% (n=08) of the cases.

Time of presentation to the hospital remains crucial in assessing the severity and deciding the outcome of treatment. Fifteen patients presented within 2 hours of poison ingestion while twelve of them presented between 2 to 4 hours of duration. Eight patients were referred late for more than 4 hours. Two patients were brought dead therefore excluded from this study.

Among the cases Clinical features (Table 1) like Jaundice was noted in 60% (n=21) patients. Persistent vomiting in 17.4% (n=06),severe abdominal pain in 20% (n=07), cardiovascular involvement like palpitations and tachycardia were noted in 8.5% (n=03) each with 1 (2.8%) patient presenting with syncope. low GCS (Glass-Gow Coma Scale) of less than 9 was noted in 14.2% (n=05).

Laboratory analysis revealed (Table 2)liver dysfunction in majority cases in the form of raised bilirubin in 28(80%) cases with elevated liver enzymes in 30(85.7%) cases, renal dysfunction was noted in 14(40%) cases. 9 cases had ECG changes, sinus tachycardia being commonest 8(22.8%) and fatal arrhythmia was seen in 1 (2.8%). Acidosis was seen in 7(20%) on analyzing ABG.

| Sl.no | Clinical features | No. of cases | Death (yes/no) |
|---|---|---|---|
| 1 | Jaundice | 21 (60.0%) | No |
| 2 | Vomiting | 6 (17.4%) | No |
| 3 | Abdominal pain | 7 (20.0%) | No |
| 4 | Palpitation | 3 (8.5%) | Yes (1/3cases) |
| 5 | Syncope attack | 1 (2.8%) | Yes (1/1cases) |
| 6 | Tachycardia | 3 (8.5%) | No |
| 7 | Low GCS score | 5 (14.2%) | Yes (1/5cases) |

Table 1: Clinical features in Synthetic cow-dung powder poisoning

| Sl. no | Parameter | Total number of cases |
|---|---|---|
| 1 | Liver function tests: | |
| | i. Raised Serum Bilirubin | 28 (80.0%) |
| | ii. Raised liver enzymes( SGOT & SGPT) | 30 (85.7%) |
| 2 | Renal function tests: | |

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| | | |
|---|---|---|
| | i. Raised Creatinine levels | 6 (17.14%) |
| | ii. Raised urea-creatinine ratio | 8 (22.8%) |
| 3 | ABG analysis: | |
| | i. Reduced PH | 7 (20.0%) |
| | ii. Reduced bicarbonate levels | 7 (20.0%) |
| 4 | ECG analysis: | |
| | i. Sinus Tachycardia | 8 (22.8%) |
| | ii. Cardiac arrhythmia | 1 (2.8%) |

Table: 2Laboratory parametersin Synthetic cow-dung powder poisoning

**Discussion:**

Suicidal consumption of Synthetic cow-dung powder is very common among the rural population of South India [ 5-6 ]. In our study, 11.29% of total suicidal poisoning cases constitute "Saani powder" poisoning. It mainly comes in two varieties- Auramine-O (yellow powder) and Malachite green (green powder). Both the dyes are highly toxic dyes causing minor gastrointestinal upset to multi-organ failure and eventually death. Although legally banned, it is still available at many local shops. Strict legal action should be taken to stop all its illegal sales and production.

The majority of our patients were young females which is a common finding in most of the studies [2, 3]. In rural parts of India, most of the females are housewives and engage with routine cleaning work using germicidal agents. Synthetic cow-dung powder being easily available at a low cost (25 rupees per packet), females are more vulnerable and susceptible to this poison. Therefore proper education creating public awareness becomes important to prevent exposure only to this poison but also other toxic substances.

Auramine-O is a potent neuro-toxic and hepato-toxic agent [1, 2, 4]. Patients present with pain abdomen, vomiting, yellowish discoloration skin, and mucous membranes. Skin discoloration and features of jaundice were noted in 60% of cases, which could be due to the distribution of toxins into tissue fluids and excretion through skin or deposition of powder on the skin surface as described by Krishnamurthy et al and others [3, 4, 7]. There is no specific antidote for a synthetic cow-dung powder to date, only symptomatic and supportive care was given as per the organs affected.

Liver function tests were severely compromised in most of our cases i.e., 85.7% (n=30) as evidenced by the three-fold rise in liver enzymes i.e., SGOT & SGPT. Serum bilirubin was raised in (n=28)80.0% of the cases. All the patients presenting with hepatic dysfunction were started with N-acetyl Cysteine (NAC)infusion therapy. Many authors suggest and recommend the usage of NAC therapy for acetaminophen liver injuries. [3, 8, 9]. Dramatic improvement was documented in the discharge summary.

Acute renal injury was noted in 14/35 (40.0%) of cases. The increased urea-creatinine ratio was seen in 22.8% cases and metabolic acidosis in 8/35 (22.8%) of cases. Most of the cases recovered with symptomatic treatment and electrolyte balance therapy, while few of them were put on low-dose prednisolone treatment (4%) and tapered gradually. This could be explained by mild renal involvement in our study. Many authors have mentioned renal tubular necrosis; however detailed analysis of pathological changes was not possible which requires advanced diagnostic techniques like renal biopsies. In a rural setup like ours, only baseline investigations are possible therefore early recognition of clinical signs and knowledge about local poisons

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becomes necessary. Few authors have also mentioned about alkalization of urine [7], for enhancing toxin elimination, which could be implemented in future case management. Five of our patients showed a low GCS scale and responded well to conservative management which contradicts studies done by others [1, 5]. This could be explained by a limited number of patients presenting with central nervous system involvement in our study group.

Other important parameters like time of hospitalization and initiation of prompt treatment and ingested dose of toxin also determine the severity of the outcome. Patients with mild symptoms are mostly ones who presented to causality within 2 hours of poison ingestion. They were managed symptomatically like gastric lavage & fluid replacement with continuous monitoring. Those who presented late by >4 hours required intensive care treatment as the toxin was already absorbed into the systemic circulation. Hepatic and renal impairment were managed accordingly which included NAC infusion, prednisolone, and restoration of acid-base balance, while none of them were intubated. Discharge summary and follow-up records showed subsequent normalization of liver and renal function tests. One of the male patients aged about 42 years who presented after 24 hours had severe dyspnea, delirium, and palpitations, and also the amount of poison consumed was unknown. ECG revealed tachycardia and cardiac arrhythmia. He succumbed to death during treatment.

As we studied the cases retrospectively, different treatment modalities and effects of chronic exposure to synthetic cow-dung powder could not be justified. However, this study emphasizes severity assessment based on simple yet reliable clinical & laboratory parameters especially in remote places like ours where timely management with limited medical resources becomes inevitable. It also stresses upon early initiation of NAC infusion and low-dose prednisolone therapy. It also guides less experienced staff & health care workers to manage acute poisoning cases systematically.

**Conclusion:**

Synthetic cow-dung powder being a lethal compound should be completely banned from illegal production and sales.

Seeking early medical attention and timely management in a systematic method and assessing the clinical severity of the cases becomes crucial for a successful outcome. A large number of patients need to be studied prospectively to know more about chronic complications,different treatment modalities and management.

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