Accidental poisoning with *Cassia occidentalis*: A rare cause of fatal acute encephalopathy in children

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**Abstract**
In pediatric population, accidental poisonings are not an uncommon cause for presentation as acute encephalopathy syndrome (AES). Certain plants found in India such as *Ricinus Communis* (Dhatura) and *Cannabis* are known to produce a clinical picture of AES. Hereby, we share our experience of accidental poisoning with *Cassia occidentalis* in three young children who presented with acute encephalopathy to the emergency department of a tertiary care center in Haryana, North India.

**Keywords:** Acute encephalopathy syndrome, *Cassia occidentalis*, poisoning

**INTRODUCTION**

*Cassia occidentalis* (local names – Kasondi in Haryana and Pamaad in Uttar Pradesh) is a wild shrub which grows abundantly in rural and roadside areas of some states in North India. It has been proposed to cause accidental poisoning and sporadic outbreaks of acute hepatomyoencephalopathy syndrome in young children with high case fatality in Western Uttar Pradesh.[1-3] The flowering season of the *C. occidentalis* is August–September and seed pod season is September–December, correlating with the seasonality of outbreaks in Western Uttar Pradesh.[1,2,4] It is known to cause severe poisoning in different animal species.[2,5] Most poisoning occurs when animals eat the pods and beans.[5] The toxic effects are seen in the skeletal muscle, liver, kidney, and heart.[7] Acute liver and muscle degeneration can be rapidly fatal in most animals.[3,8,9]

**CASE REPORTS**

A 3-year-old male child presented to the pediatric emergency with a history of multiple episodes of vomiting, low-grade fever for the past 2 days, three episodes of seizures, and altered sensorium for the past 12 h. The child had one episode of seizure on the way to the hospital and he received in cardiorespiratory arrest. The child was appropriately resuscitated, blood samples were withdrawn for baseline tests, and he was shifted to the pediatric intensive care unit (PICU). Examination revealed Glasgow Coma Scale (GCS)-3/15, generalized hypotonia, deep tendon reflexes (DTRs) were absent, plantar were mute, and bilateral (B/L) pupils were dilated and nonreactive to light. Blood sugar level was low. The child was treated keeping a working diagnosis of acute encephalitis or cerebral malaria. In spite of best efforts, the child expired after 8 h of intensive care unit (ICU) stay.

Sibling of the child, 6-year-old female, was also brought on the same day (after 12 h) with complaints of pain in...
the abdomen, multiple episodes of vomiting for the past 3 days, low-grade fever for the past 2 days, and altered sensorium for the past few hours. At presentation, the child was hypoglycemic. On examination, the child had low-grade fever with temperature of 100°F, heart rates (HRs) – 158/min, respiratory rates (RRs) – 40/min, and peripheral pulses were weakly palpable; chest examination revealed bilateral crepitation and wheezing; per abdomen examination indicated hepatomegaly (liver span 8 cm); and cardiovascular examination was normal. Central nervous system examination revealed GCS-8/15 (E2 V2 M4), both pupils were mid dilated with sluggish reaction to light, hypertonia in all four limbs, DTRs were brisk, and bilateral plantar reflexes were extensor. No bite marks were present on the body. The child was shifted to the PICU and was managed as per the protocol.

Third child, an 8-year-old male, who was living in the neighborhood of previous children, brought to the emergency department along with the second child. He also had a similar presentation with multiple episodes of vomiting for 3 days, low-grade fever for the past 1 day, one episode of generalized tonic-clonic seizure 12 h back, and altered sensorium for the past 12 h. This child was also hypoglycemic at the time of presentation to casualty. On examination, the child had HR – 194/min, RR – 20/min, poor respiratory efforts, and peripheral pulses were not palpable; chest examination revealed B/L crepitation and wheezing; per abdomen examination revealed liver span of 6.5 cm; cardiovascular examination revealed marked tachycardia; and central nervous system examination revealed GCS-4/15 (E1 V1 M2), both pupils were mid dilated with sluggish reaction to light, hypertonia in all four limbs, brisk knee jerks, and bilateral plantar reflexes were extensor. The child was immediately shifted to the PICU and treated. All children were neurologically and developmentally normal prior to this event. Clinical and biochemical findings shown in Table 1.

On taking a detailed history in the PICU and repeated probing, the parents of the children told that the children were seen playing with the beans of some unknown plant 3 days back and there is a possibility of ingestion of those beans. The exact amount of beans or pods consumed by the children was not known. The picture of that unknown plant was received through phone [Figure 1], and the plant was identified as *C. occidentalis* by an expert botanist. All the children were from a low socioeconomic status, whose parents were daily wage laborer (brick furnace workers), and the children used to play outside without adult supervision. There was no history of any insect bite or dog bite. No household kept pigs. All the three children expired during the treatment.

**DISCUSSION**

These cases highlight the very high toxicity of *C. occidentalis* seeds in children.

Low socioeconomic conditions, lack of knowledge regarding the toxic nature of the plant, easy access to the plant, lack of parental supervision for a longer duration, and playing with the beans could be the reasons that children eat such inedible plant’s bean. The parents of all the three kids were unaware about the toxic nature of the plant and they knew that their children used to play with the pods of the plant. Pica is considered as a reason, why some of the children do consume a large quantity of beans, while others do not.[10] Toxicity of Cassia beans is dose dependent but not directly related to cumulated dose over time.[2] According to the published reports on animal toxicity, the toxic dose of beans varies from as small as 0.05%–0.5% of body weight.[7] Hence, while consumption of 1–2 pods by a young child may not have any deleterious impact, a large “binge” can lead to serious disease and death.

**CONCLUSION**

These case reports highlight the need for public awareness regarding the toxicity of this plant to prevent further such disasters. Clinician knowledge about the toxicity features of *C. occidentalis* is also very crucial so that adequate early supportive care can be initiated to reduce the mortality. There is no specific antidote for this poisoning, so the preventive measures should be addressed seriously.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate
Table 1: Clinical and biochemical findings

|                      | Case 1                        | Case 2                        | Case 3                        |
|----------------------|-------------------------------|-------------------------------|-------------------------------|
| Ingestion prior to admission (days) | 2 days                        | 3 days                        | 3 days                        |
| Age and sex          | 3 year male                   | 6 year female                 | 8 years male                  |
| Clinical presentation | Vomiting, fever, seizure and altered sensorium | Pain abdomen, vomiting, fever and altered sensorium | Vomiting, fever, seizure and altered sensorium |
| Pupils at presentation | Dilated and nonreactive       | Mid dilated and sluggish reaction | Mid dilated and sluggish reaction |
| Hypoglycemia at presentation | Yes (23 mg/dl)               | Yes (28 mg/dl)                | Yes (36 mg/dl)                |
| Complete hemogram    |                               |                               |                               |
| Hb                   | 8.4                           | 8.6                           | 10.2                          |
| TLC/mm³              | 2300                          | <1000                         | <1000                         |
| DLC                  | 65/30/3/2                     | 3/4/2/1                       | 4/2/3/1                       |
| Platelets            | 36000                         | 50,000                        | <20000                        |
| PBF                  | M/H                           | 395                           | 387                           |
| Liver function test  |                               |                               |                               |
| SGOT                 | 5856                          | 6082                          | 9810                          |
| PT                   | 9415                          | 9820                          | 9480                          |
| Alk.P04              | 466                           | 395                           | 387                           |
| Serum bilirubin: Total (direct/indirect) | 2.8 (1.8/1.0)                | 2.9 (1.9/0.97)                | 3.1 (1.8/1.3)                 |
| PT/INR               | 41.2/3.7                      | 93.6/7.35                     | 68.3/5.75                     |
| aPTT                 | 36.0                          | 63.4                          | 45.7                          |
| Serum creatinine     | 1.8                           | 2.4                           | 1.7                           |
| Serum electrolyte (Na/K) | 134/5                        | 138/5.1                       | 132/5.6                       |
| Urine C/E (albumin and blood) | 1+/2+                       | 2+/3+                         | 2+/3+                         |
| CPK                  | 1165                          | 950                           | 1040                          |
| Blood C/S            | Sterile                       | Sterile                       | Sterile                       |
| Duration of stay (h) | 12                            | 36                            | 22                            |

Hb: Hemoglobin, TLC: Total leukocyte count, DLC: Differential leukocyte count, SGOT: Serum Glutamic Oxaloacetic Transaminase, PT/INR: Prothrombin time and international normalized ratio, CPK: Creatine phospho kinase, aPTT: Activated partial thromboplastin time, C/S: Culture sensitivity, C/E: Complete Examination, Na: Sodium, K: Potassium, h: hours

patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest
There are no conflicts of interest.

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