# *Cassia occidentalis* Poisoning Causes Fatal Coma in Children in Western Uttar Pradesh

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*We investigated cases of the annual seasonal outbreaks of acute hepato-myo-encephalopathy in young children in western Uttar Pradesh for causal association with Cassia occidentalis poisoning, by a prospective survey in 2006. During September-October homes of 10 consecutive cases were visited and history of eating Cassia beans was obtained in all. Nine children died within 4-5 days. There appears to be an etiological association between consumption of Cassia occidentalis beans and acute hepato-myo-encephalopathy.*

**Key words:** *Cassia occidentalis, Encephalopathy, Hepato-myo-encephalopathy.*

Many outbreaks of acute childhood illnesses with severe brain dysfunction (other than Japanese encephalitis, JE) occur in different parts of India. They may be different diseases at different times and places(1-4). Western Uttar Pradesh (UP) is noted for annual seasonal outbreaks of what is called "encephalitis"(2,5). Despite several investigations by Public Health agencies, no viral agent has been detected; yet they are presumed to be viral encephalitis(2,5,6).

We found the disease neither infectious nor encephalitis, but a hepatomyoencephalopthy syndrome. Pathology excluded infectious nature but suggested toxic cell necrosis with little inflammation. Circumstantial evidence pointed to plant toxin as the probable cause(2). We conducted a case-control study of risk factors and found significant association with exposure to *Cassia occidentalis* (Coffee senna, common name; Kasondi, Pamaad, Hindi) (*Fig. 1*)(7). The plant grows luxuriantly in western UP, with flowering season in August-September and seed-pods season in September-December, correlating with the seasonality of 'outbreaks'(2,5,7). It is known to cause severe poisoning in different animal species(7,8). Information on human toxicity is scarce and confined to occasional case report on side effects in adults(9).

From toxicology experts in the country we learned that the seeds contain too many potentially toxic chemicals to allow the easy detection of a specific toxin or set of toxins, through blood tests. Another way of confirming cause-and-effect association is to directly correlate effect with the putative cause. So during the outbreak season of 2006 we investigated cases as they were brought to the hospital by home visits for checking out history on consumption of *C. occidentalis* pods or beans.

## Subjects and Methods

The study hypothesis was: "cases of acute hepatomyoencephalopathy would not have the antecedent history of eating the Kasondi plant beans" (null hypothesis). As the plant is inedible by common knowledge–not even cattle and sheep/goats eat them–eating the beans would be a rare event if at all it occurs. We sought for the frequency of negating the null hypothesis. First we identified a 'case' according to clinical criteria as described previously(2). The home of each case-child was visited by trained field workers within 2 days of hospitalization. Parents and

other family members were asked direct questions regarding their knowledge of the affected child eating or not eating the plant pods or beans prior to illness.

During last week of September till October 2006, we identified and investigated 10 consecutive case-children in 9 families. Eight were 2-4 years old; 8 were girls; mean age was 47.7 months and median age 48.0 months. Nine children died within 3 days of hospitalization; one child was discharged well 3 days after admission. Two siblings were affected in one family. All case-children were well prior to the onset of illness that developed suddenly (duration of illness before presentation was <48 hours in 8 cases) and progressed rapidly to altered sensorium and coma within 24 hours of onset. In most children vomiting started first, usually early in the morning followed by fever, altered sensorium, abnormal body movements and signs of extreme irritability and agitation (biting, scratching clothes and body and grimacing with protruding and moving tongue/lips). Generalised convulsions occurred in 3 children. The main laboratory findings are summarized in *Table I*.

Homes were visited within 2 days of hospitalizing each case. All families were very poor and rural residents. Most parents were daily wage-laborers. We searched for *C. occidentalis* in the environs and discussed the antecedent history of the child regarding eating/not eating cassia beans during the 2 days immediately prior to the onset of illness.

## Results

*Cassia occidentalis* was found in abundance within 100-200 meters of all but one of the houses; in one case (no. 3) the weed was found on the way to her school. Although adults in all households recognized the plant and knew it to be inedible, none knew that it was poisonous.

A definite history of the case-child consuming the beans of the plant the day prior to onset of illness was obtained in all cases. Parents had seen the case-child eating the beans in cases no. 4, 5, 7, 8, 9, and 10 (*Table I*). In other either friends or close relatives verified that the child had eaten the beans. However, the exact number of pods or amount of beans consumed by them could not be ascertained. In most cases the beans were consumed while playing with friends in the field or in their backyard playing 'kitchen games'. Apparently, other children who were playing with the case-children had also consumed the beans but in very small quantities. In all instances the symptoms began after a few to many hours. All except one (no. 8) died within 4-5 days of consuming the beans, most within 48 hours. One interesting association was strong history of pica in almost all children who had consumed cassia beans.

## Discussion

The history of consumption of *Cassia occidentalis* beans by all case-children before falling

**TABLE I**–*Laboratory Iinvestigations in Case-Children (n = 10).*

| Case.No. | TLC (per cmm) | Serum glucose(mg %) | Serum ALT (U/L) | Serum AST (U/L) | Serum CPK (IU/L) | Serum LDH (U/L) | PT (sec) |
|---|---|---|---|---|---|---|---|
| 1. | 5400 | 12.2 | 9160 | 8880 | 986 | 3288 | 46 |
| 2. | 7860 | 97.8 | 1340 | 1480 | 720 | 2235 | 16 |
| 3. | 10100 | 9.7 | 948 | 905 | 1072 | 1776 | 17 |
| 4. | 4200 | 5.3 | 3870 | 4060 | 1050 | 2335 | 32 |
| 5. | 2890 | 10.2 | 1590 | 1674 | 140 | 445 | 24 |
| 6. | 8900 | 192.8 | 1625 | 1710 | 4890 | 6990 | 22 |
| 7. | 4500 | 116.7 | 2030 | 2160 | 246 | 245 | 18 |
| 8. | 7600 | 9.5 | 2180 | 2320 | 910 | 2450 | 22 |
| 9. | 3900 | 15.5 | 3690 | 3990 | 42 | 646 | 24 |
| 10. | 3800 | 96.9 | 3560 | 3910 | 217 | 1788 | 17 |

TLC - Total leucocyte count; ALT - Alanine Amino Transferase; AST - Aspartate Amino Transferase; CPK - Creatine Phosphokinase; LDH - Lactic Dehydrogenase; PT- Prothrombin time

**What this Study Adds**

- Consumption of *Cassia occidentalis* beans is associated with onset of acute hepato-myo-encephalopathy syndrome in young children in western Uttar Pradesh.

sick disproves our null hypothesis. Hence, the results confirm that *C. occidentalis* poisoning in children results in acute hepatomyoencephalopathy.

Poverty, hunger, lack of parental supervision, ignorance, children playing by themselves, non-availability of many toys / play objects, easy access to the plant, its plentiful availability and similarity to other green legumes seem to be the reasons why children in this part of the world would eat the plant. Pica, an acknowledged risk factor in acute poisoning in children is another possible reason why some children do consume large quantity of the beans while others do not(10). Surprisingly, most parents and other local natives were not aware about toxic nature of the weed.

Toxicity of Cassia beans is dose dependent, but not directly related to cumulated total dose over time (7). According to published reports on animal toxicity, the toxic dose of beans varies from as small as 0.05% to 0.5% of body weight(11). 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.

The *Cassia occidentalis* poisoning in children seems to affect mainly three systems–hepatic, skeletal muscles and brain. The exact toxic principles are yet to be defined but various anthraquinones and their derivatives are usually blamed for *Cassia occidentalis* toxicity(11,12).

Since finding positive history of environmental poisoning is difficult especially in young children, this report should provide a new perspective while investigating cases of acute encephalopathy in other places and seasons. The possibility of phytotoxin poisoning should be specifically investigated while searching for any viral etiology. In western UP there is urgent need to launch massive educational campaigns to make people aware of the risk so that future outbreaks may be prevented.

*Contributors:* VMV designed the study, collected and analyzed the data, and wrote the first draft. He will act as guarantor of the study. AK helped in acquisition of data, analyzed the data and performed statistical analysis. TJJ conceived and coordinated the study and substantially contributed to design, interpretation, and data analysis. NCN had substantial intellectual input into manuscript writing.

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*Fig. 1. Cassia occidentalis plant along with flowers and pods.*

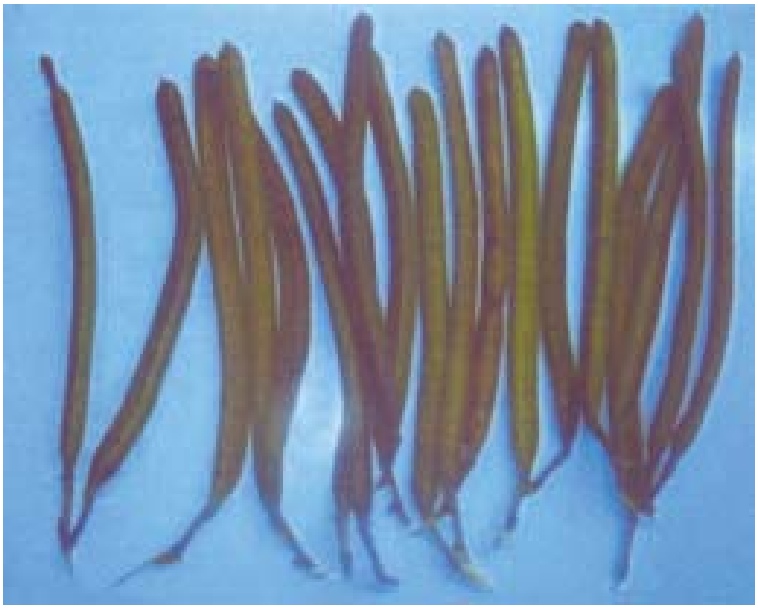

*Fig. 2. Pods (fruits) of Cassia occidentalis plant.*

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