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Cassia occidentalis poisoning as the probable cause of hepatomyoencephalopathy in children in western Uttar Pradesh

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***Background & objectives*: Recurrent annual outbreaks of acute encephalopathy illness affecting young children have been reported for several years in many districts of western Uttar Pradesh (UP). Our earlier investigations over three consecutive years (2002-2005) proved that these outbreaks were due to a fatal multi-system disease (hepatomyoencephalopathy syndrome) probably caused by some phytotoxin and not due to viral encephalitis as believed so far. We conducted a case-control study to investigate the risk, if any, from various environmental factors and also to identify the putative toxic plant responsible for development of this syndrome.**

***Methods*: Eighteen cases with acute hepatomyoencephalopathy syndrome admitted in 2005 in a secondary care paediatric hospital of Bijnor district of western UP were included in the study. Three age-matched controls were selected for each case. A semi-structured questionnaire was developed and applied to all 18 cases and 54 controls. All interviews were conducted within one week of discharge or death of each case. Quantitative data were analyzed using the relevant established statistical tests.**

***Results*: Parents of 8 (44.4%) cases gave a definite history of their children eating beans of *Cassia occidentalis* weed before falling ill, compared with 3 (5.6% controls), the odds ratio being 12.9 (95% CI 2.6-88.8, *P*<0.001). History of pica was the other associated factor with the disease, odds ratio 5.20 (95% CI 1.4-19.5, *P*<0.01). No other factor was found significantly associated with the disease.**

***Interpretation & conclusions*: Consumption of *C. occidentalis* beans probably caused these outbreaks, described earlier as hepatomyoencephalopathy syndrome. Public education has the potential to prevent future outbreaks.**

Key words : Acute encephalopathy - *Cassia occidentalis* - hepatomyoencephalopathy syndrome

Outbreaks of childhood encephalopathy syndrome with high case-fatality have become a recurrent annual seasonal feature in Saharanpur and eight other districts of western Uttar Pradesh (UP) and one adjoining district of Uttaranchal during the last many years[1-3]. Despite several investigations by public health agencies, no detailed clinical description has been published but it had been assumed to be viral encephalitis[2,3]. No viral agent has been detected in these outbreaks, but Chandipura virus aetiology was suggested, based on studies conducted in another outbreak in central India[4]. The lack of adequate epidemiological studies has been

pointed out in both the western UP disease and the outbreak declared to be Chandipura virus encephalitis[5-10]. The Chandipura virus aetiology was disputed even in the central India outbreak[10].

We investigated children in the outbreaks in Bijnor, the district adjacent to Saharanpur in western UP, during the three consecutive years, 2003-2005[11]. We developed diagnostic clinical criteria and conducted tissue pathology examination. The pathology in liver, skeletal muscles and brain had non inflammatory focal necrosis. Therefore, we named it a syndrome of hepatomyoencephalopathy . Rural children from poor socio-economic background were most vulnerable. A majority (87.3%) of cases occurred in October and November[11]. The mean age of affected children was 3.78 ± 1.23 yr. The onset was sudden in a previously healthy child, with vomiting, agitation and abnormal movements, rapidly worsening to unconsciousness. Fever was not common at onset, but it occurred later and was usually mild. The cerebrospinal fluid (CSF) was not under pressure and it had no pleocytosis[11]. Serum aminotransferases, creatine phophokinase and lactic dehydrogenase were markedly raised in the majority. Serum glucose was found low in nearly half the children. No microorganisms could be isolated from sera, CSF, throat swab and visceral specimens. Thus, all evidences suggested non-infectious pathogenesis[11]. Of 55 children in the study, 42 died, mostly within 72 h of presentation. Others recovered equally rapidly and did not show any neurological deficit on follow up[11].

During home visits of affected children in 2004, we learned from parents about incidents of young, poorly supervised children eating certain weedy plants and their fruits. This information had alerted us to the possibility of plant poisoning as a possible cause. In the second week of October 2005, while planning for an epidemiological study, we came across a local newspaper report on children dying after consuming a weed, in Khekra village, Baghpat district, western UP. During *Ramadan* (month of fasting), 8 young children of Muslim families ate a weed, locally known as '*Pamaad*', fell sick with vomiting and altered sensorium and two died within 24 h. Our staff visited the households of these children, collected samples of the weed and got them identified as *Cassia occidentalis* (Synonyms: Coffee senna, Coffee weed, *Senna occidentalis*). We carried out a community based case-control study to find out the risk, if any from various environmental factors including the coffee weed and to identify the toxic plant responsible for development of this syndrome.

Material & Methods

All children with acute hepatomyoencephalopathy fulfilling the criteria described earlier, admitted during the second week of September through December 2005 were included in this study[11]. The inclusion criteria were acute onset and rapid progression to unconsciousness in a previously well young child; CSF without pleocytosis; no malarial parasite on blood smear; and no plausible clinical diagnosis to explain the disease[11]. For every case, we selected three age-matched (± 1yr) neighbourhood controls, sharing the same house cluster and similar socio-economic conditions. At least one of the three controls was a child living in the same household as the case and was designated as 'contact' for the purpose of the study.

A semi-structured questionnaire with 59 variables was applied to the parents or guardians of the 18 cases and 54 controls, in their homes. The variables included questions pertaining to drug history, physical environment, fauna and flora, consumption of ground nuts, food habits, play habits, availability of cereal stock at home, practices of storing and spraying pesticides/ insecticides, knowledge about '*Pamaad*' (*C. occidentalis*) and any deleterious effects, history of the child seen playing/handling/eating the pods (beans) of the plant, history of the child eating other weedy plants/ fruits/seeds, *etc*. All interviews were conducted in Hindi, mostly within one week of discharge or death of each case. The main respondents were parents/guardians; occasionally neighbours and friends helped with information about the eating and play habits of the deceased children.

Quantitative data were analyzed using Epi-info 6. The estimation of risk was made using odds ratio (OR) with 95 per cent confidence interval (CI). Significance of association between the variables was determined by Chi Squire and Fisher Exact tests.

Results

All children belonged to rural or semi-rural areas and low socio-economic background. The mean ages of the cases and controls were 41.72 ± 17.61 and 47.96 ± 10.89 months, respectively.

Most parents were labourers working in the field or small-scale vegetable growers. The socio-demographic characteristics of cases and controls were similar (Table I), none of the differences being statistically significant.

No sibling or contact of case had any illness affecting brain functions. However, in one instance, a cousin of

one case who visited the family had an identical illness and had died. Parents of that child could not be contacted. None of the cases had illness requiring medical attention during the 2 wk recall period prior to the episode of hepatomyoencephelopathy. However, three controls had short fever during a similar time interval, one of whom had taken some medicine from a local practitioner. Only four control households had some 'allopathic' (modern) or 'ayurvedic' (herbal) medicines kept at home. Neither case nor control had used aspirin during the two-week recall period. Thirteen cases (72.2%) had received some treatment for the acute illness from local practitioners, before bringing to the hospital.

The residences of cases were scattered in an area of over around 1800 square kilometers. All houses had poor sanitary conditions and open drains. The habitats were surrounded by agricultural cultivations. There was no significant difference among the cases and controls in terms of environmental conditions such as house dampness and water logging around the house, proximity to domestic animals and abundance of mosquitoes and rats. No household kept pigs. There was very little use and no storage of pesticides in the study or control households (Table I). A majority of respondents occasionally ate poultry, mutton and fish, but not pork. Most of them ate green leafy vegetables that were grown in the neighbouring fields. None of these factors showed any significant difference in the two groups. There was no significant difference between the cases and the controls in terms of consumption of old groundnuts (that could have acted as source of aflatoxin) or water chestnuts (on which insecticide was likely to have been sprayed) (Table I).

Pamaad (*C. occidentalis*) was found to grow in abundance in the vicinity of houses of both cases and controls. A majority of parents recognized the plant (Table II). There was no significant difference in their ability to recognize the plant or their low risk perception related to it. They considered it to be an insignificant harmless weed that was not even eaten by animals. While none of the parents of cases perceived *C. occidentalis* as a poisonous plant, only 3(5.6%) parents of controls reported that they had some idea about the possible harmful effects of the plant. Some of the families were found using the dried plant along with its pods for fuel. Many parents knew that their children used to play with the pods of the plant. A majority of them had seen children handling or playing with the plant and the pods (Table II).

The parents of 8 (44.4%) cases gave a definite history of their child having eaten *Pamaad* beans before the onset of disease. Some of the parents could verify this on their own while others confirmed it with the help of children of friends/relatives/neighbours. However, the exact number of pods or amount of the beans consumed by them could not be ascertained. Three (5.6%) controls were also reported to have consumed the beans reportedly in a

Table I. Baseline comparison of cases and controls

Parameter	Cases (n=18)		Controls (n=54)	
Demographical & environmental:	N	(%)	N	(%)
Male	9	(50)	29	(54)
Hindu	7	(39)	27	(50)
Thatched hut	9	(50)	23	(43)
Dampness in house	7	(39)	18	(33)
Water logging in close vicinity	10	(56)	23	(43)
Proximity with animals	15	(83)	44	(81)
Death of any animals in recent past	0	(0)	0	(0)
Abundance of rats & mosquitoes	17	(94)	49	(91)
Pesticides available in house	0	(0)	1	(2)
History of insecticide spray	1	(6)	4	(7)
Dietary history:				
Non vegetarian	11	(61)	40	(74)
History of consuming green leafy vegetables	14	(78)	47	(87)
History of consuming 'Bagad'	12	(67)	46	(85)
History of consuming groundnuts stored for long duration	5	(28)	12	(22)
History of consuming water chestnuts	11	(61)	43	(80)

Table II. Distribution of potential risk factors pertaining to *C. occidentalis* plant for hepatomyoencephalopathy in cases and controls

Potential risk factors	Cases (N = 18)		Controls (N = 54)		Statistical analysis
	N	(%)	N	(%)	
Cassia plant found in close vicinity of house	17	(94.4)	45	(83.3)	*P* = 0.24 Odds Ratio = 3.36 95% CI: 0.41-157.65
Family members recognized the plant	15	(83.3)	38	(70.4)	*P* = 0.28 Odds Ratio = 2.09 95% CI: 0.49 - 12.77
The child was seen touching or playing with Cassia pods	12	(66.7)	32	(59.3)	*P* =0.78 Odds Ratio = 1.32 95% CI: 0.45 - 4.22
The child ate *Cassia* pods (beans) very recently	8	(44.4)	3	(5.6)	*P* <0.001 Odds Ratio = 12.89 95% CI: 2.56 - 88.77
The child had history of pica	12	(66.7)	15	(27.8)	*P* <0.01 Odds Ratio = 5.20 95% CI: 1.44 - 19.54

very small quantity. The history of consumption of cassia beans had a strong statistical association with the disease ($P< 0.001$). The children who had consumed cassia beans showed 12.9 times higher risk of developing hepatomyoencephelopathy as compared to those who were reported not to have eaten them.

There was a statistically significant difference in the history of pica among the cases and the controls ($P< 0.01$). Over 66 per cent cases and 27.8 per cent control had pica. Children with pica were at 5.20 fold higher risk of developing hepatomyoencephelopathy in comparison to controls. Of the 11 children who had consumed cassia, 9 had pica (Table II).

Discussion

The findings of this study strongly suggested that consumption of *C. occidentalis* beans was responsible for the disease. Children who had the disease, had significant association with the history of eating the beans of cassia. On the contrary, children who had either not eaten the beans or had eaten only small quantities of it, did not develop the disease. We suspect that children would eat the beans out of sight of parents and a negative history was less reliable than a positive one.

The age group, epidemiological profile and outcome of cases in our series were very similar to those of children in the study reported by Meda *et al*[12] on poisoning due to consumption of unripe ackee fruit leading to acute encephalopathy syndrome in Burkino Faso.

The weed was identified by expert botanists as *C. occidentalis* (Synonyms: Coffee senna, Coffee weed, *Senna occidentalis*) (Fig.). It is a leguminous plant (family: *Fabaceaesae*, Genus:*Cassia* Species: *occidentalis*) found ubiquitously in Asia and Europe as a weed among various crops[13]. It is an opportunist annual herb that grows along roadsides, fence lines and over heaps of waste material[14]. In India, the weed is widely prevalent (known locally as *Pamaad, Kasondi*). *Pamaad* is a misnomer, the name of a similar-looking weed, *Cassia obtusifolia*, which is found much less frequently and is commonly mistaken for *C. occidentalis* (*Kasondi*). However, what the local people called *Pamaad* was actually *C. occidentalis*. Its leaves and roots are used in some herbal remedies, but its pods or beans are usually avoided. Rarely local people boil the leaves and use them as vegetable. The flowering time of *C. occidentalis* in this part of India is after the heavy monsoon rains (after September)[15]. The fruits of the plant are thin pods, flat, 3-4 inches long and pale green when tender, thick and dark green when mature. The beans are green, soft and juicy (Fig.) The poding time is from September to November[14,15]. From December onwards, the pods start drying up, become brown and the beans (seeds) turn dark brown[15].

We found no definitive study on human toxicity of *C. occidentalis*. However, the plant is known to cause poisoning in different animal species[15-18]. All parts of the plant are toxic but different parts have different levels of toxicity. Most poisoning occurs when animals eat the

Fig. *C. occidentalis plant* along with flowers and pods.

pods and beans[16-18]. *C. occidentalis* beans fed to cattle at 0.5 per cent of their body weight are known to induce severe muscle degeneration[19]. However, according to one report, even a dose of ground beans as small as 0.05 per cent of body weight was able to produce toxicity in 26 calves[20]. High doses of the plant induce liver degeneration and death before myodegeneration has time to develop[19, 21]. Appearance of the illness and duration are inversely proportional to the dose, but not directly related to cumulated total dose[20]. The toxic principles have not been clearly established[22]. Various anthraquinones and their derivatives, emodin glycosides, toxalbumins and other alkaloids are the few compounds blamed for the cassia toxicity[13, 23]. The toxic effects are on skeletal muscles, liver, kidney and heart[20]. The acute liver and muscle degeneration can be rapidly fatal in most animals[17, 19].

The clinical signs in animals with *C. occidentalis* toxicity include lethargy, recumbancy, jerky respiration, tremors, diarrhoea, ataxia, hyperpnea, and incoordination[20,24]. Death may occur within 24 h after signs of acute illness and is almost inevitable in animals that become recumbent[20]. The signs, symptoms and the lesions of toxicity also vary in different animal species. For example, horses may not exhibit the signs of muscle degeneration seen in cattle. They succumb to liver degeneration[17]. In most species liver and muscle enzymes are markedly raised and histopathology shows features of microscopic myodegeneration and moderate centrilobular necrosis and fatty degeneration in liver[19, 20].

The natural cycle of *C. occidentalis* explains the strong seasonality of the disease, which occurs during September through December, with most cases occurring in October and November[11]. The tender beans are not unpalatable and tastes like raw edible beans. They do look attractive and form popular play objects among young (2-8 yr old) children. As part of their game, they pretend to cook and serve these beans in the play marriages of their dolls . While most children used to throw them away, some seem to eat the beans. Lack of parental supervision, children left to themselves, easy access to the plant, the plentiful availability, hunger and similarity to other green vegetables seem to be the reasons why children in this part of the country eat the plant. Another possible reason may be pica which had a strong association with the cases. Basavaraj *et al*[25] had also found pica behaviour as a risk factor in acute poisoning in young children.

There are only scanty reports on actual occurrence of toxicity by consumption of *C. occidentalis* fruits (beans) in humans in literature. According to one report prolonged ingestion of a health drink by an adult made from senna extract (*Cassia acutifolia*) resulted in severe hepatotoxicity[26]. Another reference mentions, "In human, ingestion of *C.occidentalis* can cause severe purging. Whereas this may produce great discomfort and pain in adults, the result in a child can be death"[27]. Hence, while consumption of few pods might not have any ill effect in an adult or older child, it can prove fatal for a young child.

Our histopathological findings had pointed toward a phytotoxin as the possible cause of the disease[11]. The characteristic histopathology, especially the liver histology resembled that of toxicity caused by plants like *Senecio* spp, *Crotalaria* spp. and *Heliotropium* groups of weeds producing pyrrolizidine alkaloid or a known hepatotoxic agent[28]. The seeds of *Senecio vulgaris* plant containing pyrrolizidine alkaloids are used to adulterate certain cereals including '*Bagad*'- a cheap staple diet of rural population, which is extracted from inferior quality of rice. We did not find any evidence of accidental or deliberate consumption of these plants or their seeds by the local people or children. Apart from

strong history of consumption of beans of *C. occidentalis* fruits, no other significant association with any other toxic plant could be found.

The histopathological and biochemical changes in liver and muscle in animals that died due to *C. occidentalis* poisoning resemble the features seen in our cases[11,20]. Further, animal studies performed on rats[29], and chickens[30,31] that were fed a ration contaminated with *C. occidentalis* seeds at different doses had shown histopathological changes similar to our histopathological findings on muscle, liver and brain biopsies[11].

Since there are hundreds of different anthraquinone derivatives and other alkaloids in *C. occidentalis*, and no single principal toxin is known, it was not possible for us to perform toxicology studies on cases and controls in this study. We need to carry out further toxicological studies to document chemical evidence of our conclusion that the children died of *Cassia* poisoning.

In conclusion, our study showed a significant association of *C. occidentalis* poisoning with hepatomyoencephalopathy in young children in western UP. In the absence of any other possible causative agent, and in view of the histopathology evidence negating infectious aetiology but agreeing with toxic necrosis, we propose it as the probable cause of the outbreak-like clustering of cases during the *Cassia* poding season. We believe that intensive educational campaigns to make people aware of this risk and environmental measures to reduce the weed density may prevent future outbreaks.

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