

Absence of diarrhea in purge nut ingestion: A case series of eight children

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ABSTRACT

There are very few case reports in literature of *J. curcas* poisoning. Previously grown as an ornamental plant; it is presently being cultivated on a large scale for its seed oil, which is used as biodiesel. This has brought this plant in close vicinity to the human population, exposing them to the chance ingestion. We are presenting clinical and biochemical profile of eight children with *J. curcas* poisoning. The plant is commonly known to be a purgative and gastrointestinal irritant but the most conspicuous feature in our patients was absence of diarrhea. Lethargy, severe abdominal pain, inability to ingest anything, and intense thirst were the most prominent complaints in all children. The symptoms in all the patients were significant enough to merit admission and intravenous fluid therapy. Hematological and biochemical workup revealed neutrophilia and raised serum alkaline phosphatase in all patients while leukocytosis was observed in 5 of them. Electrocardiography was normal in all the patients.

Key words: *Ayurveda, curcas, jatropha, multifida, plant poisoning*

INTRODUCTION

Jatropha curcas; known to be a gastrointestinal irritant and purgative has been used, both in Ayurveda and the African system of medicine, for varied illnesses. Presently it is being cultivated on a large scale for use as biodiesel in India. Although an uncommonly reported source of poisoning; its cultivation for extracting biodiesel and as an ornamental plant has brought this plant in close vicinity to humans, thereby increasing its incidence of chance ingestion. We are presenting a series of eight children who were admitted with this hitherto uncommon poisoning, with a conspicuous absence of diarrhea as a manifestation.

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CASE REPORT

Eight children were brought to the pediatric emergency at 8 PM, with alleged history of ingestion of some wild seed 6-7 hours ago. All the children were between 3 and 12 years of age and were from the same household. The children ate the seeds, on the way back from school and half-an-hour later, they all started vomiting. The vomitus contained only the ingested food particles and no blood. Vomiting was associated with severe abdominal pain in all the children. The pain and vomiting progressively increased and they became lethargic before reaching the hospital. There was no history of diarrhea, fever, skin rash, bleeding from any site, altered sensorium, or convulsions.

All the children on arrival were conscious and oriented, but lethargic. Vital signs (heart rate, respiratory rate, blood pressure, and temperature) in all the children were normal for the age. All of them had markedly increased thirst, without any clinical signs of dehydration. Central nervous system examination was otherwise normal. Rest of the systemic examination was unremarkable.

The investigations of all the patients are shown in [Table 1]. Five of the eight children showed hemoglobin of less than 9 gm%. Blood counts revealed leukocytosis in five of the eight children, and neutrophilia and normal platelet count in all the children. Serum alkaline phosphatase was raised in all the patients. Blood urea levels were raised in four children, but serum creatinine was normal in all of

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them. All the patients had normal liver function test and total serum calcium levels. Electrocardiography was normal in all the children.

Keeping in mind the fact that the history was essentially provided by the children and could be unreliable, on arrival in the Pediatric Emergency, empirically gastric lavage was done and activated charcoal was administered through the Ryle's tube. Gastric aspirate contained fresh unaltered blood in all but two children. All the patients required and were administered intravenous fluids. In addition, symptomatic treatment was started with antiemetic, oral antacid, and injection ranitidine. The vomiting stopped in all the patients over 6 hours. None of the patients had loose stools (0%) during the illness. There was no complaint regarding increased or decreased frequency of urination by the children during their stay in the hospital. Initially, the seed could not be identified because the adult relative accompanying the children could bring only the outer pericarp of the seed. The relatives brought the stem of the plant with fruit and the seeds next day. The plant was then identified with the help of experts from forensic medicine and Indian system of medicine. The children were kept under observation for 36 hours before being discharged. Vomiting had subsided and all the patients were accepting feeds well, at the time of discharge.

On reviewing the history, it was found that the 7-year-old girl first ate the seed and then offered it to the rest of the siblings. It was not the black seed, but the shiny white kernel after removing the outer pericarp, which attracted them. All the children ate it believing it to be cashew nut.

DISCUSSION

J. curcas belongs to the family, Euphorbiaceae and is commonly known as purge nut, barbados nut, black vomit nut, curcas bean, and physic nut. It is being cultivated in India for its oil which is being used as biodiesel. In other parts of world it is also grown as an ornamental plant.^[1]

The seed has a black coat with a white shiny kernel inside [Figure 1]. Its agreeable taste makes children ingest more than one seed. All parts of the plant are toxic particularly the seeds. The oil yielded from the seeds is known as hell oil, oleum infernale or oleum ricini majoris.

Almost all parts of the plant have been used in traditional system of medicine in India (Ayurveda) and Africa for a wide range of ailments. The seeds of *J. curcas* have been used in the Indian system of medicine: As purgative, antihelminthic, and abortifacient; as well as for treating ascites, gout, paralysis,



Figure 1: Image of the *Jatropha curcas* stem, fruit (dried), and seed with and without pericarp

and skin diseases.^[2] The seed oil of the plant has been used in treatment of rheumatic conditions, itch, parasitic skin diseases, gonorrhea, and as mouthwash.^[2] In Africa it has been specifically utilized for its purgative effect.^[3] The toxicity of the plant is due to curcin, hydrocyanic acid, tetramethylpyrazine, and an atropine-like substance.^[4]

Onset of symptoms in *J. curcas* poisoning is very rapid. Although uncommon in adults, toxicity is same as in children.^[5] All our patients developed symptoms within half-an-hour of ingestion; vomiting initially, followed by pain abdomen. The severity gradually increased and after 3-4 hours they were unable to ingest anything orally and became lethargic.

Although the toxic symptoms described are primarily gastrointestinal; with diarrhea as the main symptom, interestingly none of our patients had diarrhea. Occasional reports have previously reported absence of diarrhea.^[1,6-8] As has been suggested in literature, one reason for this could be the difference of species of the *Jatropha* seeds.^[4] Curd is also known to neutralize toxic effects of *Jatropha*.^[9] It was not possible to precisely identify the species of *Jatropha*, as only the stem and the seed of the plant were available to us. Since the most common species of *Jatropha* in our region is *curcas*, the plant was assumed to be *curcas*. None of our patients had taken curd, so this was ruled out as reason for absence of diarrhea.

All our patients had mild dehydration due to decreased oral intake and losses in vomitus. There was no history of increased urinary loss, salivation, sweating.

Lethargy was seen in all our patients. Except Abdu-Aguye *et al.*, who reported two patients with restlessness, all

other reports of *J. curcas* poisoning have been associated with lethargy [Table 2].^[10] Since none of the children were severely dehydrated, the lethargy was more likely due to direct effect of the *Jatropha* seed, as also reported by Koltin *et al.*^[11]

Isolated case reports have described both meiosis and mydriasis as manifestations of *Jatropha* poisoning.^[1,4,7] But none of our patients had meiosis or mydriasis. Electrocardiographic abnormalities and mild transient elevated alanine aminotransferase/aspartate aminotransferase (ALT/AST) have been reported in poisoning with *Jatropha*.^[11] None of our patients had deranged liver function test and electrocardiograph (ECG).

Hemoconcentration is expected in dehydrated patients; but in our patients it is not possible to comment on hemoconcentration accurately, as the premorbid hemoglobin is not known. Since anemia is very prevalent in our community; especially so in lower socioeconomic strata, the higher hemoglobin in case number 2 and 5 may indicate hemoconcentration [Table 1]. This is further corroborated by elevated urea level in these children. We found leukocytosis in five children which has been reported only in two earlier reports.^[1,12] Differential leukocyte count revealed neutrophilia in all the patients

including those with normal leukocyte count. This may be due to an acute phase reaction to the stress of the poisoning, or a direct effect of the toxins.

In our patients, severity of manifestations did not correlate with the number of seeds eaten. Although the children were asked the history of number of seeds eaten within 1 day, the possibility of recall bias among the children cannot be ruled out. Since literature search did not present any study correlating the number of seeds ingested with the toxicity, we tried to correlate the severity of toxic effects with the number of seeds eaten.

Maximum number of seeds was eaten by case number 2, 3, and 4. But in two of these (case number 3 and 4) gastric bleeding was absent. Elevated urea with normal creatinine in cases 2,3,4,5, and 8 indicate prerenal azotemia due to dehydration. This also did not correlate with the number of seeds ingested. We believe that the severity and time of onset of vomiting is a major confounder because rapid onset of severe vomiting can result in reduced toxic effects due to the agent being vomited out. Another confounding factor could be the age of the patient, as younger patients can have more severe manifestations with the same dose.

Table 1: Summary of the cases

Names	Child-1	Child-2	Child-3	Child-4	Child-5	Child-6	Child-7	Child-8
Age (years)	12	8	7	7	6	6	5	3
Sex	M	M	F	F	M	M	F	F
Weight (kg)	21	18	15	15	20	16	16	8
Blood in gastric aspirate	+	++	None	None	+	+	+	+
No. of seeds eaten	3	10	10	10	5	3	4	5
No. of vomitings	7-8	7-8	10-12	>10	8-10	10-12	7-8	8-10
Hb (g%)	10.3	12.6	8.9	7.9	13.6	8	8.4	8.9
TLC (' $10^3/\mu$ l)	18.6	8.0	14.9	9.2	20.0	13.6	14.0	4.6
DLC (P/L/M/E)	85/12/1/2	70/25/2/3	85/12/1/2	87/10/1/2	71/26/2/1	70/26/2/2	78/20/1/1	74/20/2/4
Platelets (' $10^5/\mu$ l)	2.3	2.5	2.6	1.8	1.7	2.4	1.9	1.7
RBS (mg/dl)	WNL	WNL	WNL	WNL	WNL	WNL	WNL	WNL
Na (mEq/L)	140	141	139	137	140	144	132	141
K (mEq/L)	3.7	4.1	4	4.3	4	3.8	4.6	4.4
Urea (mg/dl)	26	42	39	40	43	36	30	42
Creat (mg/dl)	0.8	0.9	0.8	0.8	0.8	0.8	0.8	0.8
Ca (mg/dl)	8.3	9.5	9.5	9.5	9.9	10.1	9.2	9.7
TSB (mg/dl)	0.7	0.2	0.5	0.5	0.3	0.2	0.6	0.8
ALP (IU/l)	618	661	630	824	678	670	564	564
SGOT (IU/l)	41	44	41	47	35	55	37	38
SGPT (IU/l)	30	31	37	48	31	49	18	31
Total proteins (g/dl)	7.2	6.5	6.8	7	6.7	6.7	6.4	6.6
Albumin (g/dl)	4.6	4.3	4.1	4.5	4.2	4.4	0	4.4
ECG	WNL	WNL	WNL	WNL	WNL	WNL	WNL	WNL

M = Male sex, F = female, + = Mildly blood stained, ++ = Grossly blood stained, Hb = Hemoglobin, TLC = Total leukocyte count, DLC = Differential leukocyte count, P = Polymorphs, L = Lymphocytes, M = Monocytes, E = Eosinophils, RBS = Random blood sugar, WNL = Within normal limits, Na = Serum sodium, K = Serum potassium, Creat = Serum creatinine, Ca = Calcium, TSB = Total serum bilirubin, ALP = Serum alkaline phosphatase, SGOT = Serum glutamic oxaloacetic transaminase, SGPT = Serum glutamic-pyruvic transaminase, ECG = Electrocardiograph

Table 2: Summary of cases of *Jatropha* poisoning in literature

Name of the author	Seed type	No. of patients	Age of patients (years)	Time from ingestion to onset of symptoms	No. of seeds eaten	Clinical features (% of the total patients)
Esperanza 2012 ^[8]	Not specified	34	7-12	Not specified	Not specified	Nausea, vomiting, dehydration (100%); abdominal pain (53%); diarrhea (12%)
Chomchai <i>et al.</i> , 2011 ^[11]	C	75	2-14	Not specified	1-20	Nausea (100%), vomiting (100%), diarrhea reported as one of the most common presentation but exact number not specified, abdominal pain (100%). Three patients had mild, transient AST/ALT derangement at 48-72 h
Shah 2010 ^[5]	C	5	13, 35, 18, 38, 17	10-15 min	1-3	Vomiting (100%), diarrhea (100%), abdominal pain (100%), dehydration (60%)
Singh and Singh 2010 ^[7]	C	4	5-8	1-2 h	Not specified	Vomiting (100%), abdominal pain (100%), diarrhea (25%), dehydration (0%), miosis (25%)
Lurie <i>et al.</i> , 2008 ^[12]	M	1	12	Not specified	2	Diarrhea, vomiting, abdominal pain, leukocytosis
Koltin 2006 ^[13]	M	4	3,4,5,7	3 h	Not specified	Vomiting (100%), diarrhea (100%), meiosis (100%), dehydration (100%), 2/4 (50%) mildly obtunded, miosis and leukocytosis (100%)
Kulkarni <i>et al.</i> , 2005 ^[6]	C	20	8-13	0.5-2 h	1-4	Vomiting (95%), diarrhea (50%), headache (40%), fever (40%), abdominal pain (25%), asymptomatic (5%)
Levin <i>et al.</i> , 2000 ^[13]	M	2	9.5, 8.5	1.5 h	10 each	Vomiting (100%), abdominal pain (100%), diarrhea (100%), obtunded (100%), dehydration (100%). One patient had prerenal azotemia and respiratory acidosis
Abdu-Aguye <i>et al.</i> , 1986 ^[10]	C	2	3, 5	Not specified	Not specified	Vomiting (100%), DH (100%), restlessness (100%)
Joubert <i>et al.</i> , 1984 ^[14]	C	8	2-9	Not specified	Not specified	Nausea (100%), vomiting (100%), diarrhea (100%), abdominal cramps (100%), dehydration (5/8, 62.5%)

C = *Jatropha curcas*, M = *Jatropha multifida*, AST = Aspartate aminotransferase, ALT = Alanine aminotransferase

All our patients responded to initial intravenous rehydration fluid therapy along with oral antacids and gastric lavage with activated charcoal. Although treatment is essentially symptomatic and there is no antidote, rehydration is critical. Rehydration can be given either orally or intravenously if the patient cannot accept oral intake. Gastric lavage should be performed promptly, when the patients present early. Activated charcoal may be given orally or via Ryle's tube to decrease absorption. For severe gastritis patients may be started on antacids along with a histamine-2 receptor blocker or proton pump inhibitor. Although fatalities with *Jatropha* poisoning are rare, Abdu-Aguye *et al.*, demonstrated that oral seeds administered to mice can cause macroscopic anal hemorrhage and death. They also found that extract of the seed administered intraperitoneally in mice can cause death in doses as low as 1 mg/kg.^[10] Thus, patients need to be monitored clinically and biochemically for fluid and electrolyte disturbances, acid-base status, full blood count, and renal and hepatic function. Specific therapy may be indicated for hemorrhagic gastrointestinal damage, skeletal muscle and gastrointestinal spasm, and hemoglobinuria. As fatality is rare and most of the patients can become asymptomatic within 24 hours, patients need to be observed till they are able to take oral feeds well.

CONCLUSION

Acute accidental poisoning with a variety of organic plant irritants is common, especially in children. Minimal or no history is available due to the inability of the child victim to convey the features of the exact plant, the part of which may have been consumed. In cases of chemical ingestion, information regarding the nature of the chemical, its clinical features, antidote, etc. are readily available on the container of such chemical, but identification of a poisonous plant is very difficult especially for the clinician in the emergency units. It would be in the interest of the hospitals to maintain a list of the locally growing wild/garden plants so as to correctly diagnose and initiate appropriate management measures in case of poisoning with such plants. Moreover, it would be desirable for the upcoming Poison Control Centers of the individual hospitals to include a physician trained in the Indian system of medicine or a botanist so that such plants can be correctly and quickly identified and appropriate treatment instituted. In this era of information technology, identification of the plant can also be easily done by creating a webpage where the treating physician can upload the image of the plant from a mobile phone camera, which can then be reviewed by experts from other systems of medicine. Timely identification of the plant can help a treating physician give accurate treatment

and prognosis and curtail unnecessary hospitalization and reduce treatment cost. Information regarding toxic effects of plants grown locally for commercial or ornamental purposes should be widely disseminated in the community as a preventive measure.

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