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Serious Accidental Poisoning Due to Consumption of Cassia Occidentalis Seeds in A 3-Year-Old Girl from Greece

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1. Abstract

The authors describe a 3-year-old previously healthy girl who presented with new-onset hepatic encephalopathy due to accidental consumption of Cassia occidentalis seeds. The child found the seeds four days before her presentation in the dehiscent fruits of a plant that was kept in a flowerpot in a neighbor's house. Both parents and the neighbor witnessed the consumption but were unaware of its toxic potential. The plant was identified as Cassia occidentalis by the physicians on call based on digital photographs provided by the parents. She developed increasing somnolence, hypoglycemia, high lactic acid, severe transaminasemia, high serum lactate dehydrogenase, direct hyperbilirubinemia and coagulation disorders, requiring her urgent transport to a pediatric intensive care unit (PICU). In the PICU, hyperammonemia was also documented. She required mechanical ventilation and aggressive medical support but recovered without neurological or other sequelae. Unintentional exposure of young children to Cassia occidentalis causes a condition known as hepatomyoencephalopathy due to the presence of toxic anthraquinones, a class of secondary plant metabolites known to provoke severe liver damage. Public and clinician awareness is extremely important regarding the hepatotoxicity of Cassia occidentalis. A thorough history of ingestion of toxic beans is of paramount importance for the immediate institution of appropriate supportive care in case of toxicity.

2. Introduction

Cassia (Senna) occidentalis is a common agricultural plant in Europe, North and South America, Mexico, Africa, Asia, and Australia [1]. All parts of the plant are highly toxic to herbivore animals, while the dried plant is used for fuel in India and elsewhere [2]. Toxicity in humans has mainly been reported from India, where several reports of acute liver failure have been associated with its consumption, mainly in young children [3-7] but also in an elderly woman [8]. We present the case of a 3-year-old Greek girl who developed near-fatal acute liver failure after accidentally consuming seeds of this plant.

3. Case Report

A 3-year-old girl was brought to the pediatric emergency room of the University General Hospital of Alexandroupolis by her parents in late October due to increased somnolence over the last several hours. Two days ago, she started having decreased oral intake. At the same time, she vomited twice and looked tired. The parents reported that four days ago, she had consumed the seeds of an ornamental plant during a visit to a neighbor's house. The plant was identified by the physicians on call (authors ST and MT) as Cassia occidentalis, based on digital photographs of the plant taken by the parents. Her personal medical history was unremarkable, and she was in good health before the events described. In the emergency room, a point-of-care dipstick glucose measurement was low (35 mg/dL), for which she was immediately given 2 ml/kg 10% dextrose and started on continuous intravenous hydration with 5% dextrose in normal saline. The initial complete blood count showed leukocytes 3,010/μL (neutrophils 54%, lymphocytes 20%, monocytes 26%), hemoglobin 12.2 g/ dL, hematocrit 36%, and platelets 140.000/μL. Serum biochemical tests disclosed sodium 134 mmol/L, potassium 4.6 mmol/l, calcium 8.4 mg/dL, aspartate aminotransferase (AST) 5,675 U/L, alanine aminotransferase (ALT) 7,910 U/L, lactate dehydrogenase (LDH) 2,743 U/L, total bilirubin 4.03 mg/dL, direct bilirubin 2.75 mg/dL, indirect bilirubin 1.28 mg/dL, γGT 44 U/L, albumin 3.7 g/dL, and C-reactive protein 0.48 mg/dL. Coagulation studies revealed severely prolonged prothrombin time (PT 83.9 sec, reference range 10.1- 12.9 sec), International Normalized Ratio (INR) 7.31, a prolonged activated partial thromboplastin type (aPTT) 45.1 sec (reference range 25-37 sec), fibrinogen 147 mg/dL (reference range 220-490 mg/dL), and D-dimers 1,407 ng/mL. Arterial blood gas determination while breathing supplemental oxygen showed pH 7.37, pCO2 29mmHg, pO2 133 mmHg, HCO3 16.8 mmol/L, base deficit 8.5 mmol/L, and lactate 3.4 mmol/L. Work-up for viral hepatitides was positive only for IgG antibodies against hepatitis A and IgG antibodies against hepatitis B surface antigen due to prior immunization. Because of laboratory evidence of incipient liver failure, she was given twice vitamin K 1mg intravenously along with 120 ml of fresh frozen plasma. The Greek National Poisoning Center was conducted by phone, and she was started according to their advice on N-acetylcysteine (NAC) as a continuous infusion intravenously en route to the nearest pediatric intensive care unit (PICU). On arrival in the PICU, the patient was hemodynamically stable, she was breathing spontaneously and had a Glasgow Coma Scale (GCS) of 13/15, mild drowsiness, adequate ventilation and oxygenation. Laboratory investigations for liver function tests, bilirubin, ammonia and INR were initially performed every 8 hours. Based on her laboratory findings in the PICU, that were indicative of hepatic encephalopathy (hypoglycemia, hyperammonemia, high lactic acid, severe transaminasemia, high LDH, direct hyperbilirubinemia, and coagulation disorders), she was prescribed rifaximin for intestinal sterilization, prophylactic systemic antibiotics (tazobactam, metronidazole), antifungals (fluconazole), and antivirals (cidofovir), and mannitol as a neuroprotective agent. In addition, ursodeoxycholic acid, gastroprotection with proton pump

inhibitors, lactulose and enemas were prescribed. Laboratory studies to exclude autoimmune hepatitis (ANA, SMA, ANCA, LKM, GP1, LC1) were negative, while the serum immunoglobulin concentrations were within the normal range for age. Serological testing for adenovirus, CMV, EBV, HSV, VZV, HHV-6, HHV-7, parvovirus B19, toxoplasma, rubella, and enterovirus was negative for acute infection, as it was a film array of respiratory secretions and stool. Urine organic acids and serum and urine acetylcarnitines were also examined and were normal. A pediatric hepatologist (author IR) and the Greek National Transplant Organization were notified from the start, and contact was made with a transplant center in Italy for possible transfer and emergency liver transplantation in case of deteriorating hepatic failure. The criteria set for the patient's transfer were the presence of worsening hepatic encephalopathy, the need for continuous venovenous hemodialysis for uncontrolled hyperammonemia, the need for high respiratory support, INR value > 6.5, or bilirubin > 15mg/dL. Plasma administration was reserved in case of bleeding or surgery and platelet administration was reserved for platelet counts < 50,000/μL. Approximately 24 hours after the PICU admission, the patient developed a gradual decline in the level of consciousness, with a GSC of 8/15. She was intubated, sedated and placed on mechanical ventilation while a brain CT scan was performed, which did not show signs of increased intracranial pressure. On the first hospital day, an ultrasound of the liver, biliary tract and spleen was performed and on day 2, a CT scan of the upper and lower abdomen was completed and disclosed a hypodense liver without focal lesions or other abnormalities. Four days later, the sedation was gradually discontinued, and she was successfully weaned off the ventilator on day 7, i.e., after 6 days of mechanical ventilation. At that time, the child was oriented, responsive to stimuli and tetrakinetic but still showed mild somnolence. The serial changes in liver enzymes, coagulation studies, lactic acid and plasma ammonia levels are shown in Table 1. As shown, the plasma lactate and ammonia peaked on hospital days 1, 5 and 8, creating a characteristic triphasic course that was followed by a progressive decline. Figure 1 shows the serial changes over time of total and direct bilirubin, while Figure 2 shows the serial changes over time for AST, ALT, and LDH. On hospital day 10, she was transferred to the 3rd Department of Pediatrics of Aristotle University of Thessaloniki, where hospitalization continued for a few more days with improving clinical and laboratory findings. She is currently healthy, approximately 5 months after the events described.

4. Discussion

We report a 3-year-old girl who developed nearly fatal hepatic encephalopathy due to accidental consumption of Cassia (Senna) occidentalis seeds during a visit to a neighbor's house who kept the ornamental plant in a flowerpot. Both parents and their neighbor witnessed the consumption but were completely unaware of the severe hepatotoxic potential of the plant. Severe herbal hepatotoxicity is exceptionally uncommon in children, although its incidence is likely underestimated due to underreporting by parents and/or caregivers who frequently refuse to disclose the consumption of herbal products to physicians [9,10]. Exposure to plants accounts for approximately 5% of human poisonings reported by poison control centers in North America and Europe [10]. Unintentional plant exposure, mainly in children <5 years of age, accounts for more than 90% of cases [10]. Gastrointestinal symptoms are prevalent, and treatment is predominantly supportive. Severe and fatal cases are rare, emphasizing the usually favorable outcome of poisoning in children. Our patient was prescribed NAC en route to the PICU. An updated meta-analysis and systematic review have shown that in patients with non-acetaminophen-related acute liver failure, NAC significantly improves overall, post-transplant, and transplant-free survival while reducing the overall length of hospital stay [11]. Cassia (Senna) occidentalis, commonly known as styptic weed or coffee senna, is a flowering plant that belongs to the family Fabaceae and is native to many continents [1]. Because of its nice, yellow flowers that are arranged in groups of two to four, with fertile stamens in each flower, it is commonly used as an ornamental flower in Greece and elsewhere. Flowering occurs all year and its dry, elongated, dehiscent fruit contains many seeds. Although all parts of the plant have been used as food and medicine by Indian tribal populations, several reports exist describing its consumption as a cause of death in children due to hepatic encephalopathy [3-8]. In recent years, due to the publicized association of the plant with acute liver failure, the number of deaths has declined dramatically, although apparently, many people keep the plant at home for decorative reasons, completely unaware of its toxicity [12]. Commercially available Senna is a common laxative derived from Cassia acutifolia and Cassia angustifolia, which are also hepatotoxic. A high index of suspicion for Senna toxicity must be kept in mind in patients who consumed herbal medications and presented with vomiting, diarrhea, and rapidly evolving encephalopathy. Vashishtha et al. in 2007 first described 55 children aged 2 to 10 years of age who were hospitalized from 2003 to 2005 in Bijnor, Uttar Pradesh, India, with features of acute encephalopathy likely induced by a phytotoxin [3]. The disease peaked in early winter in previously healthy, rural children of very low socio-economic status. Almost all had vomiting followed by unconsciousness and a majority had low-grade fever and abnormal behavior with agitation. Abnormal posture of the trunk and limbs were distinctive features. Serum transaminases, creatinine phosphokinase, and LDH levels were found markedly elevated in all cases in whom the tests were performed. Serum glucose was <50 mg/dL in 47.3% of the affected children at presentation, while the cerebrospinal fluid was under normal or low pressure and without pleocytosis in all.

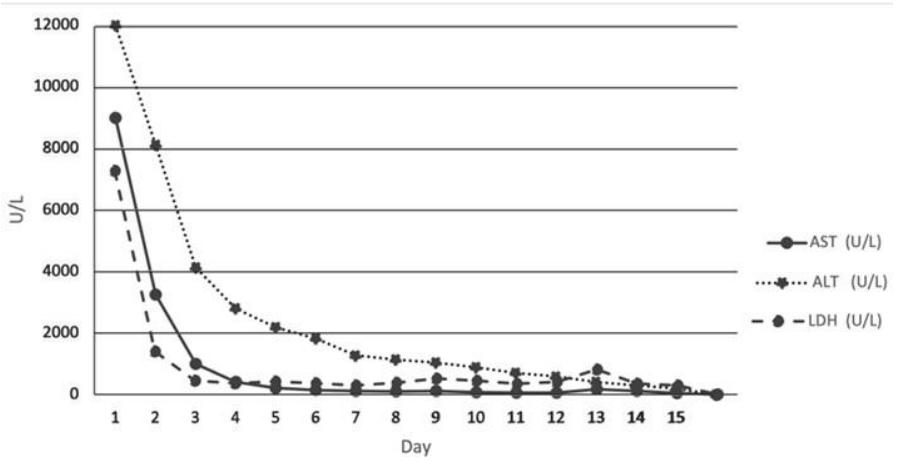

Figure1: Serial changes of AST, ALT, and LDH during the patient's hospitalization.

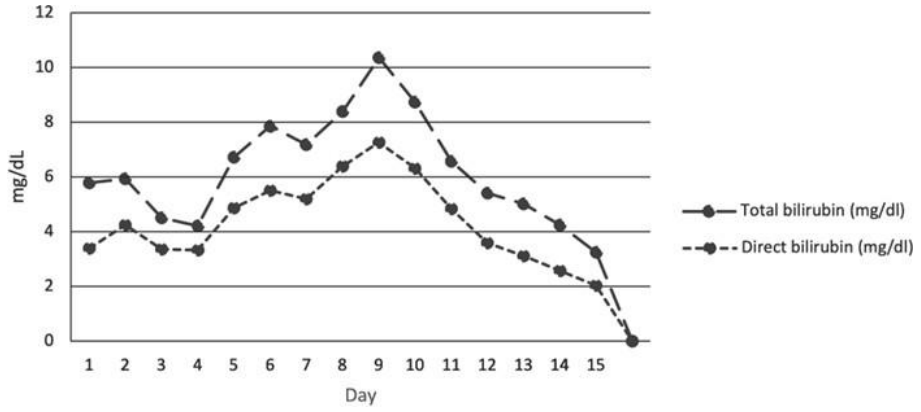

Figure2: Serial changes of total and direct bilirubin during the patient's hospitalization.

Table 1: Serial changes in liver enzymes, bilirubin, creatinine phosphokinase, amylase, INR, ammonia, and lactic acid during the patient's hospitalization.

	Hospital day															
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**	**13**	**14**	**15**	**25**
AST (U/L)	8993	3243	996	419	226	155	119	113	118	84	66	70	180	118	58	46
ALT (U/L)	12000	8117	4138	2801	2192	1830	1276	1138	1049	891	706	591	418	309	200	44
γGT (U/L)	47	46	33	37	51	49	49	61	83	101	133	169	248	238	161	31
Alkaline phosphatase (U/L)	339	361	281	258	320	286	246	263	261	223	191	189	177	178	165	244
LDH (U/L)	7289	1394	448	356	418	360	283	366	507	440	343	402	803	350	286	284
Total bilirubin (mg/dL)	5,78	5,93	4,51	4,21	6,72	7,86	7,18	8,39	10,4	8,7	6,6	5,4	5	4,3	3,2	0,8
Direct bilirubin (mg/dL)	3,39	4,25	3,36	3,33	4,87	5,51	5,19	6,4	7,27	6,3	4,8	3,6	3,1	2,6	2	0,5
CPK (U/L)	550	173	253	205	103	66	49	58	83	66	94	46	58	20	27	63
Amylase (U/L)	112	40	57	149	75	115	95	824	335	79	34	88	259	165	122	54
INR	5,47	3,85	3,42	3,20	2,23	2,22	1,91	1,84	1,47	1,43	1,24	1,13	1,00	1,05	0,99	1,12
Ammonia (μmol/L)	121	98	63	72	137	83		113	76	67						
Lactic acid (mol/L)	5,4	2,5	1,9	3,5	4,8	5,3	4,1	3,6	4	3						

Overall, 42 children succumbed to their illness, most within 72 hours of presentation, for a case fatality rate of 76.4%, while survivors did not show neurological deficits. Panwar and Kumar in 2008 provided convincing evidence that the cause of this hepatomyoencephalopathy was not a virus but rather Cassia occidentalis toxicity of which the local Indian population was unaware. Two years later, i.e., in 2010 the deadly disease had stopped in Saharanpur due to a successful education campaign in the affected districts of India [12]. Cases of patients who consumed seeds of C. occidentalis along with affected patients but in smaller quantities have been described. They also developed transaminitis but without full-blown encephalopathy [5]. Nirupam et al in 2013 described a near-fatal case of Cassia occidentalis poisoning in a 3-year-old girl from western Uttar Pradesh, who was saved with intensive support [6]. On admission, she had vomiting, low- grade fever, excessive crying, and irritability with violent and self- mutilating behavior [6]. Exposure to Cassia occidentalis causes liver failure and the well-described hepatomyoencephalopathy syndrome due to the presence of toxic anthraquinones, a class of secondary plant metabolites known to provoke damage to vital organs such as the liver, brain, kidney, spleen, and muscles [13]. Animal studies in rodents [14], as well as clinical studies in humans, have shown that Cassia occidentalis' toxicity is associated with elevated transaminases and LDH, necrotic hepatic lesions, and muscle vacuolization associated with increased serum creatinine phosphokinase. Cassia occidentalis' consumption also causes neuronal damage by disturbing the levels of various neuronal proteins, such as glial fibrillary acidic protein and beta-tubulin III. Anthraquinones disturb cellular homeostasis via binding to DNA, increasing the production of reactive oxygen species and inhibiting essential enzymes. In conclusion, public and clinician awareness is crucial to prevent a syndrome mimicking virally induced hepatic encephalopathy caused by ingestion of parts of Cassia occidentalis. A meticulous history of any potential ingestion of toxic beans is essential for the immediate institution of appropriate supportive care in case of hepatic encephalopathy.

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