Case Report

Fatal fulminant myocarditis and multiorgan failure following accidental *Cassia occidentalis* ingestion in a child: A case report and review of literature

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ABSTRACT

Cassia occidentalis (Coffee Senna) is a toxic leguminous weed widely distributed across tropical and subtropical regions, including rural India, where accidental ingestion by children continues to pose a serious but underrecognized public health threat. Its seeds contain potent anthraquinone derivatives and toxalbumins that disrupt mitochondrial oxidative phosphorylation, leading to a fulminant multiorgan syndrome encompassing hepatic failure, rhabdomyolysis, toxic encephalopathy, and myocarditis. We report the case of a 4-year-old previously healthy child who presented to the pediatric intensive care unit 2 days after the accidental ingestion of *C. occidentalis* seeds, with progressive vomiting, lethargy, and hemodynamic instability. Clinical examination revealed hypotension, tachycardia, tachypnea, drowsiness, and hepatomegaly. Electrocardiography revealed ST-segment elevations in multiple leads, and echocardiography confirmed severe left ventricular systolic dysfunction with an ejection fraction of 25%, consistent with fulminant myocarditis. Despite aggressive intensive care management, including mechanical ventilation, inotropic support with dopamine and adrenaline, fresh frozen plasma transfusions and vitamin K. The child deteriorated rapidly and succumbed within 12 h of admission due to refractory cardiogenic shock and multiorgan failure. This case highlights the fulminant and near-universally fatal course of *C. occidentalis* toxicity when cardiac involvement occurs and underscores the critical need for early suspicion, prompt referral to tertiary pediatric critical care facilities, and robust community education campaigns for prevention in endemic rural areas.

Key words: *Cassia occidentalis*, Coffee Senna, Fulminant myocarditis, Hepatic failure, Multiorgan dysfunction, Pediatric intensive care unit, Pediatric myocarditis, Poisoning

Cassia occidentalis (Coffee Senna), also known as *Senna occidentalis*, is a wild leguminous weed belonging to the family Fabaceae. It is widely distributed across tropical and subtropical regions and is particularly prevalent in rural parts of India, Africa, and South America, where it grows abundantly in agricultural fields, roadsides, and wastelands [1,2]. The plant bears seeds that closely resemble coffee beans in color and shape, making them especially attractive to young children who may inadvertently ingest them during play near harvested fields (Fig. 1).

The toxic constituents of *C. occidentalis* include anthraquinone derivatives (including chrysarobin and emodin), naphthopyrones, and toxalbumins. These compounds impair mitochondrial oxidative phosphorylation, generate reactive oxygen species (ROS), and cause energy depletion in metabolically active tissues, including hepatocytes, skeletal muscle fibers, and cardiac myocytes [3-7]. The resulting clinicopathologic syndrome, termed hepatomyoencephalopathy, is characterized by progressive hepatic failure, rhabdomyolysis, toxic encephalopathy, and, in severe cases, fulminant myocarditis with cardiogenic shock. *C. occidentalis* poisoning has been implicated in several pediatric outbreak deaths in northern India, particularly in western Uttar Pradesh and Bihar, where case-fatality rates exceeding 70% have been documented [1,2]. Despite this, the condition remains underrecognized by primary care providers in rural and peri-urban settings, and most children present to tertiary facilities at an advanced stage of organ dysfunction.

The present case is reported for several reasons. First, fulminant myocarditis as a manifestation of *C. occidentalis* toxicity in a child is infrequently documented in the indexed literature. Second, the combination of severe left ventricular (LV) systolic dysfunction, acute hepatic failure, renal impairment,

Access this article online	
Received - 17 March 2026 Initial Review - 02 April 2026 Accepted - 27 April 2026	Quick Response code
DOI: 10.32677/ijcr.v12i5.8175	

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Figure 1: Cassia occidentalis

and coagulopathy, constituting true multiorgan failure in a young child following seed ingestion, warrants detailed documentation. Third, the case illustrates the characteristic 2-day delay in presentation that is typical of this poisoning, a feature with important implications for clinical recognition and management. This report aims to heighten awareness among pediatricians, pediatric intensivists, and emergency physicians regarding the need for early suspicion and aggressive intervention in cases of suspected *C. occidentalis* ingestion.

CASE REPORT

A 4-year-old previously healthy male child presented to the pediatric emergency department with a 2-day history of progressive illness following accidental ingestion of *C. occidentalis* seeds. According to the accompanying family members, the child had been playing near agricultural fields in a rural area approximately 48 h before presentation, during which time, he reportedly ingested an unknown quantity of seeds identified by the family as belonging to the *C. occidentalis* plant, locally known as "Chakunda," that grew abundantly in surrounding fields.

On the day of ingestion (day 1), the child developed nausea and multiple episodes of non-bilious, non-projectile vomiting, along with mild abdominal discomfort. There was no immediate loss of consciousness, seizure activity, or obvious hemodynamic compromise at that time. The family, residing in a semi-urban area with limited healthcare awareness and access, attributed the symptoms to a minor gastrointestinal upset and initially managed the child at home with oral rehydration. There was no

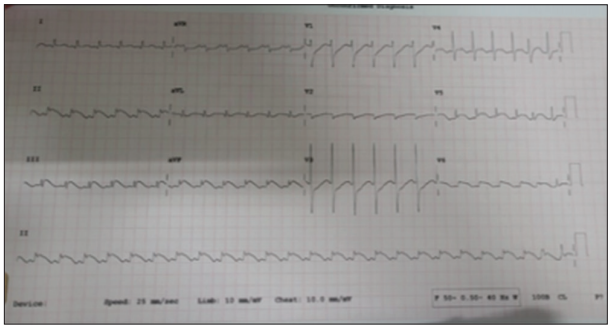

Figure 2: ECG – ST Changes

prior visit to a healthcare facility during this interval. The delay in seeking medical attention was attributable to a combination of factors: Initial perceived mildness of symptoms, lack of awareness regarding the toxicity of *C. occidentalis* seeds, socioeconomic constraints limiting access to tertiary care, and the gradual rather than abrupt onset of serious manifestations.

Over the following 24–36 h (Day 2), the child's condition progressively worsened. Vomiting became more frequent and was accompanied by increasing lethargy, refusal of feeds, and progressive pallor. In the hours immediately preceding presentation, the family noted that the child had become increasingly unresponsive and had markedly reduced urine output. The child was then brought to a local health facility, from where he was referred to the tertiary pediatric center and subsequently admitted to the pediatric intensive care unit (PICU).

On admission (approximately 48 h post-ingestion), the child was critically ill. Vital signs revealed heart rate 160 beats/min, blood pressure 70/40 mmHg, respiratory rate 52 breaths/min, temperature 37.2°C, and oxygen saturation (SpO_2) 88% on room air. The child was drowsy with a Glasgow Coma Scale score of 10/15 (E3V3M4). There was no icterus noted on initial examination. On systemic examination, cardiovascular assessment revealed tachycardia with feeble peripheral pulses and a prolonged capillary refill time of 5 s. Hepatomegaly of 3 cm below the right costal margin was noted on abdominal examination; the spleen was not palpable. Respiratory examination revealed bilateral coarse crepitations consistent with early pulmonary edema. Neurological examination showed generalized hypotonia with no focal deficits. There was no rash, lymphadenopathy, or petechiae. Urinary output was estimated at <0.5 mL/kg/h, consistent with oliguria.

Laboratory investigations on admission revealed hemoglobin 10.2 g/dL, total leukocyte count 14,200/mm^3 with neutrophilic predominance, and platelet count 98,000/mm^3. Liver function tests showed severe transaminitis with aspartate aminotransferase 980 IU/L and alanine aminotransferase 750 IU/L; serum bilirubin was mildly elevated at 2.8 mg/dL (direct 1.9 mg/dL). Coagulation studies revealed a markedly prolonged international normalized ratio (INR) of 7.1 with a prothrombin time of 78 s. Serum ammonia was elevated at 110 μmol/L (reference <80 μmol/L). Arterial blood

gas (ABG) analysis demonstrated severe metabolic acidosis: pH 7.18, pCO_2 28 mmHg, HCO_3 10 mEq/L, and base excess -16. Serum lactate was markedly elevated at 6.8 mmol/L. Renal parameters showed blood urea nitrogen 48 mg/dL and serum creatinine 1.8 mg/dL, consistent with acute kidney injury (AKI). Serum electrolytes revealed hyponatremia (Na^+ 128 mEq/L) and hypoglycemia with random blood glucose 42 mg/dL. Cardiac biomarkers were strikingly abnormal: Creatine kinase-myocardial band fraction was 430 IU/L; NT-proBNP was 18,000 pg/mL. Serum troponin-I was not available at the time of admission. Creatine phosphokinase total was markedly elevated at 3,400 IU/L, consistent with significant rhabdomyolysis.

Electrocardiography (ECG) demonstrated sinus tachycardia with diffuse ST-segment elevation in leads II, III, aVF, and V3–V6, consistent with myopericarditis or fulminant myocarditis (Fig. 2). Chest X-ray revealed cardiomegaly with pulmonary venous congestion. A bedside 2D echocardiogram performed within 1 h of admission confirmed severely impaired LV systolic function with an ejection fraction (EF) of 25%, global LV hypokinesia, and mild pericardial effusion without tamponade physiology. The right ventricular function was moderately impaired. No structural defects were identified.

Given the clinical and biochemical picture of fulminant myocarditis, acute hepatic failure, AKI, coagulopathy, and metabolic derangements in the context of *C. occidentalis* ingestion, a diagnosis of *C. occidentalis*-induced multiorgan failure was established.

The child was immediately intubated and mechanically ventilated for airway protection and respiratory failure. Hemodynamic resuscitation was initiated with isotonic crystalloids (10 mL/kg boluses, cautiously titrated given cardiomyopathy), followed by inotropic support with adrenaline (0.1 mg/kg/min) in view of hypotension. Dextrose infusions were initiated to correct hypoglycemia and maintain euglycemia. Intravenous vitamin K and fresh frozen plasma (FFP) were administered for coagulopathy correction. Empiric broad-spectrum antibiotics were initiated pending culture results. In view of AKI and persistent metabolic acidosis despite initial management., the child was planned to start on continous renal replacement therapy.

Despite maximal intensive care support, the child's condition continued to deteriorate. Inotropic requirements escalated progressively. Repeat ABG showed worsening acidosis (pH 7.10). The child developed bradycardia followed by pulseless electrical activity cardiac arrest approximately 10 h post-admission. Cardiopulmonary resuscitation was performed for 20 min with no return of spontaneous circulation. Death was declared 12 h after PICU admission, approximately 60 h following the initial ingestion of *C. occidentalis* seeds.

DISCUSSION

C. occidentalis poisoning represents a severe but underrecognized cause of toxic myocarditis and hepatic failure among children in rural and agrarian regions of India [1,2]. The toxin exposure usually results from accidental ingestion of its coffee-like seeds, often by children playing near harvested fields. The illness has been linked to several outbreaks of hepatomyoencephalopathy and toxic myocarditis, particularly in western Uttar Pradesh and Bihar [1-3]. The present case underscores the fulminant nature of *C. occidentalis* poisoning, characterized by a combination of cardiogenic shock, acute hepatic failure, and rapid progression to multiorgan dysfunction.

Pathophysiologically, *C. occidentalis* contains several anthraquinone derivatives, naphthopyrones, and toxalbumins that induce mitochondrial uncoupling, impair oxidative phosphorylation, and generate ROS [4-7]. The resulting energy failure and oxidative stress lead to necrosis of hepatocytes, skeletal muscle fibers, and cardiac myocytes. Experimental studies by O'Hara, Osweiler, Pugh, Brewer, and Helman demonstrated uncoupling of oxidative phosphorylation in myocardial mitochondria, establishing a direct mechanistic link between the toxin and cardiac dysfunction [6]. More recent biochemical analyses by Shukla *et al*. have further confirmed that anthraquinones are responsible for mitochondrial swelling, adenosine triphosphate depletion, and cell death [7].

Clinically, the presentation often begins with non-specific gastrointestinal symptoms such as vomiting, abdominal pain, and malaise, followed by encephalopathy, jaundice, and shock within 48-72 h [1,3,8]. Cardiac involvement ranges from mild enzyme elevation to overt myocarditis with ST-segment elevation, arrhythmias, and severe LV systolic dysfunction. In the current case, the marked elevation in NT-proBNP (18,000 pg/mL) and ECG changes with ST elevations were consistent with fulminant myocarditis. The coexistent hepatic failure and elevated INR (>7.0) reflected profound synthetic dysfunction, a hallmark of severe intoxication.

A critical and clinically relevant feature of the present case and one that merits focused discussion is the 2-day delay between ingestion and PICU presentation. This delayed presentation is characteristic of *C. occidentalis* toxicity and has been previously noted in outbreak investigations [1,3]. Several factors contribute to this pattern. First, the initial symptoms of nausea, vomiting, and abdominal discomfort are non-specific and may be indistinguishable from common gastrointestinal illnesses, leading families to manage the child at home without seeking medical attention. Second, the toxic syndrome evolves over 24–72 h as mitochondrial injury to multiple organs accumulates, meaning that life-threatening manifestations such as hepatic failure, cardiogenic shock, and encephalopathy typically do not become apparent until 48 h or more post-ingestion. Third, socioeconomic barriers, including limited health literacy, geographic remoteness, financial constraints, and the absence of local tertiary care facilities, delay referral even when symptoms worsen. Fourth, in endemic regions, the plant is not universally recognized as toxic

by rural communities, and primary care providers may not consider plant poisoning in the differential diagnosis of a febrile child with vomiting. This delay has profound prognostic implications, as children presenting beyond 48 h with established multiorgan dysfunction have near-universally fatal outcomes [1,4,9].

Outbreak investigations have established a strong epidemiologic correlation between *C. occidentalis* ingestion and pediatric mortality [1,2]. In the landmark study by Vashishtha *et al*., case–control data from western Uttar Pradesh confirmed *C. occidentalis* bean ingestion as the most probable cause of hepatomyoencephalopathy in children, with a mortality rate exceeding 70% [1]. Other reports from tertiary centers across India describe similar patterns, including elevated transaminases, hyperammonemia, and rapid onset of refractory shock [3-5,9].

Mechanistic studies demonstrate that anthraquinones and other Cassia toxins exert multiorgan mitochondrial toxicity leading to hepatic, muscular, and myocardial necrosis [5-7,10]. The cardiac pathology observed in fatal cases is often characterized by interstitial edema, myocardial fiber necrosis, and inflammatory infiltrates consistent with toxin-induced myocarditis [4,6]. Autopsy-based studies have confirmed diffuse mitochondrial injury within both hepatocytes and cardiomyocytes, correlating with the clinical presentation of hepatic and cardiac failure [6,7].

Mortality predictors include delayed presentation (>48 h post-ingestion), severe metabolic acidosis, high transaminase and ferritin levels, prolonged INR (>5), and depressed LV EF (<30%) [1-4,8,9]. Children presenting in shock with elevated cardiac biomarkers or encephalopathy typically have a rapidly fatal course despite optimal management. In our case, death occurred within 12 h of presentation, consistent with the hyperacute progression reported in previous Indian pediatric case series.

Management of *C. occidentalis* poisoning remains largely supportive, as no specific antidote exists [1,2]. Therapy focuses on maintaining hemodynamic stability through inotropes, correcting metabolic derangements, and addressing hepatic coagulopathy with FFP and vitamin K. Careful attention should be paid to fluid balance to prevent hepatic congestion and pulmonary edema. Advanced modalities such as molecular adsorbent recirculating systems or liver transplantation may be considered in centers equipped for acute liver failure management, though pediatric data remain limited [3,4,9]. Continuous renal replacement therapy may be useful for the management of refractory AKI, fluid overload, and severe metabolic acidosis, as employed in the present case.

Globally, reports of *C. occidentalis* poisoning are rare but not limited to India. Cases have been reported from Africa and South America, with similar toxic profiles involving hepatic and cardiac systems [11,12]. A recent European case documented by Sdougka *et al*. described a toddler presenting with acute liver failure following seed ingestion, highlighting the worldwide potential of this underrecognized toxin [13]. Thus, *C. occidentalis* represents a global toxicological concern, and awareness should extend beyond endemic regions.

Public health awareness campaigns and community education are essential preventive measures, especially in rural settings where *C. occidentalis* grows wild. Avoiding the use of its seeds as play objects or unintentional food substitutes can prevent accidental ingestion. The inclusion of Cassia toxicity in pediatric toxicology curricula and awareness programs among rural healthcare providers is crucial for early recognition and prompt intervention [10-12].

Taken together, *C. occidentalis* toxicity in children presents as a unique clinicopathologic syndrome of hepatomyoencephalopathy and fulminant myocarditis. Early recognition, aggressive supportive care in a PICU setting, and prevention through community education remain the cornerstones of management. The present case adds to the growing body of literature emphasizing the fulminant and near-universally fatal nature of this poisoning when cardiac involvement occurs, and underscores the need for increased vigilance among pediatricians and pediatric intensivists.

CONCLUSION

C. occidentalis toxicity should be considered in any child from an endemic rural area presenting with unexplained vomiting, hepatic dysfunction, and hemodynamic instability, even when symptom onset predates presentation by 24–72 h. The fulminant multiorgan syndrome encompassing toxic myocarditis, acute hepatic failure, AKI, coagulopathy, and metabolic acidosis carries an exceptionally high mortality risk, particularly once cardiac involvement is established. Early recognition, timely referral to a tertiary PICU, and aggressive multimodal supportive care are essential components of management, though outcomes remain poor in advanced cases. Pediatric clinicians and intensivists, especially those practicing in India and other endemic regions, must maintain a high index of suspicion for this preventable but potentially fatal cause of multiorgan failure.

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Funding: Nil; Conflicts of interest: Nil.

How to cite this article: Deora C, Gupta N. Fatal fulminant myocarditis and multiorgan failure following accidental *Cassia occidentalis* ingestion in a child: A case report and review of literature. Indian J Case Reports. 2026;12(5):318-322.