



Case Report

Accidental *Ricinus communis* Oil Cake Intoxication in a Dairy Cow

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Abstract

The aim of the study to report a case of spontaneous toxicity in a 5 year old crossbred Jersey cow with a history of accidental ingestion of *Ricinus communis* oil cake during the month of November 2017. The animal had anorexia and profuse watery yellowish mucoid diarrhoea. On clinical examination the animal showed dullness and depression, congested conjunctival mucous membrane, tachycardia and doughy rumen with absence of rumen motility. The whole blood and sera samples were collected for haemato-biochemical analysis. The haematological values showed mild haemoconcentration with normocytic normochromic anaemia. There was no abnormality in biochemical values. Electrocardiography revealed sinus tachycardia. On ultrasound examination the liver and kidney showed the normal contour and intestinal loops showed increased wall thickness without distension. The animal was treated with activated charcoal, egg white, fluid therapy, purgatives, antioxidants (ascorbic acid) and low dose of sodium bicarbonate. The animal recovered uneventfully after three days of treatment. Recovery is generally less common in ricin poisonings.

Key words: Castor Oil Cake, Cattle, Ricin, *Ricinus communis*

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Introduction

Castor bean (*Ricinus communis*) is one of the industrial oil seed crop belonging to Euphorbiaceae family. It is widespread in the tropical region and mostly cultivated in India, China and Brazil (Miller *et al.*, 2009; Rosiane *et al.*, 2011). These plants are mainly cultivated for oil production. The oil is very useful in industries and medical field as lubricant. The seed after oil extraction is a rich source of protein; it has



limited use as animal feed due to the presence of most potent phytotoxin ricin, less toxic alkaloid ricinine and thermostable allergens (Akande *et al.*, 2011). All plant parts are toxic, but the highest level of ricin can be found in hard seed coat, which needs to be crushed or broken to release toxic compound (Sebastian, 2007). The ricin molecule is composed of two chains, A and B. The chain A is responsible for the enzymatic activity of the toxin. It removes the adenine from 28S RNA of ribosome and inhibits protein synthesis, leading to cell death. The B chain is essential for delivering the A Chain into the cell to produce lethal effect (Puri and Kumar, 2011). The ricin is toxic to all species; the lethal oral dose of castor seed in horse, cow, goat and hen is varied from 7 to >300 mg/kg, 1-2 g/kg, 5.5 g/kg and 14 g/kg, respectively (Bornemann, 1992; Anonymous, 2008). Intoxication in the animal can occur after accidental ingestion of feed contaminated with crushed or broken seed (Worbs *et al.*, 2011). The farm animals showed symptoms of weakness, profuse watery diarrhoea, dehydration, sunken eyeball, dilation of pupils, depression, tachycardia, dyspnoea and colic within few hours to three days later (Aslani *et al.*, 2007). The present article describes about the accidental intoxication of *Ricinus communis* oil cake in a dairy cow and its clinical management.

Materials and Methods

A five year old Crossbred Jersey cow was presented to the Large Animal Medicine, Referral clinics of Veterinary College and Research Institute, Orathanadu, Thanjavur district of Tamil Nadu, India with a history of accidental ingestion of 1.0 kg castor oil cake, which was kept in a small bag and hanged in a tree of the farm. This oil cake was meant for use as an organic fertilizer for gardens. The cow had anorexia and profuse yellowish mucoid watery diarrhoea. On clinical examination the animal showed dullness and depression, congested conjunctival mucous membrane and doughy rumen on palpation. The rectal temperature was 39°C, respiratory rate, heart rate and the pulse rate 40/min, 157/min and 136/min, respectively. The lymph node was of normal size; there was a suspension of rumen motility. The whole blood and sera samples were collected for haemato-biochemical analysis. Electrocardiography (ECG) was recorded with base apex lead system with 50 mm paper speed. Ultrasonographic examination of liver, kidney and intestines were carried out by using 2.5-5.0 MHz convex probe (Esaote, Europe).

Results and Discussion

The haematological values showed mild haemoconcentration with normocytic normochromic anaemia (Table 1). This was coinciding with the report of Aslani *et al.* (2007) and Worbs *et al.* (2011) which stated that profuse watery diarrhoea in ricin intoxicated animals leads to severe dehydration and haemoconcentration. In this study, there were no alterations in biochemical values (Table 1). This was contradictory to the report of Worbs *et al.* (2011), who reported that elevated level of liver enzymes (AST), creatine kinase, BUN and creatinine occur in animals exposed to *Ricinus communis* seeds experimentally.

Table 1: Haemato-biochemical analysis before and after treatment

Haemogram			Serum biochemistry		
Parameters	Before Treatment	After Treatment	Parameters	Before Treatment	After Treatment
Haemoglobin(g/dl)	12.9	10.4	Total protein (g/dl)	6.8	6.93
PCV (%)	42	38	Albumin (g/dl)	2.97	3.03
RBC ($10^6/\mu\text{l}$)	10.3	9.25	Globulin (g/dl)	3.83	3.9
WBC ($10^3/\mu\text{l}$)	6	11.19	Glucose (mg/dl)	80	68
MCV (fl)	40.7	41	BUN (mg/dl)	34	24
MCH (pg)	12.5	11.2	Creatinine (mg/dl)	1.5	1.3
MCHC (g/dl)	30.7	27.3	AST (U/L)	110	84

ECG showed sinus tachycardia. No major changes in ECG were observed. On ultrasound (USG) examination, the liver and kidney showed the normal contour. The intestinal loops showed increased wall thickness without distension (Fig.1).

**Fig.1:** Transabdominal Ultrasonography-Thickened intestinal wall with homogenous echogenic contents in multiple loops of jejunum**Fig. 2:** Haemorrhagic watery diarrhoea in a Crossbred Jersey cow affected with ricin toxicity**Fig. 3:** Haemorrhagic watery diarrhoea with sloughing off intestinal mucosa

The thickened intestinal wall observed here could possibly may due to inflammation. Currently, no specific antidotes are available against ricin intoxications. However, the treatments focused on supportive therapy

like intravenous fluids to maintain blood volume, injection Vitamin B₁, B₆ and B₁₂ (Tribivet) IV, oral drenching of liquid paraffin 1.0 Litre and purgatives (Bovilax 500g dissolved in one litre of water) and feeding of activated charcoal (250g) along with egg white on first day to prevent the toxin absorption and expedite the elimination of the toxin from the body. On second day the animal showed severe bloody diarrhoea with sloughing off mucosal layer of intestines possibly due to gastrointestinal irritation by ingested toxin (Fig. 2 & 3).

On day two the animal was treated with multiple electrolyte solutions (2.0 Litre), normal saline (2.0 Litre) and Inj. Sodium bicarbonate 50 ml IV, inj. Ascorbic acid 10 ml IM, Inj. Streptopenicillin 2.5 g IM and Chlorpheniramine maleate 15ml IM along with supportive herbal treatment like feeding of aloe vera and grounded fenugreek seed 250g. Maintenance of alkaline urine flow with sodium bicarbonate 5 to 15g/day promotes the renal excretion of the toxin (Nellis, 1997). Administration of ascorbic acid prevents the binding of ricin B-chain on the cell. If the B-chain does not bind, the A-chain cannot enter the cytoplasm to exert its lethal effect of inhibiting protein synthesis (Holtsclaw and Clark, 1977). On third day the animal voided greenish semisolid dung and resumed the adequate feed intake and was ruminating normally. Not much reports available on the clinical management of ricin poisoning in dairy cattle. In the available literatures in public domain, fluid therapy and activated charcoal were only used as supportive care. However, majority of cases were reportedly died in such cases. In the current study the dairy cow recovered uneventfully.

Conclusion

Ricin intoxication in the animal was managed with activated charcoal, egg white, fluid therapy, purgatives, sodium bicarbonate and ascorbic acid and it made uneventful recovery.

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