

Chinaberry Tree (*Melia azedarach*) Poisoning in Dog: A Case Report

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This article describes a case of Chinaberry tree poisoning diagnosed in a dog. The initial clinical signs were variable and included tremors (muscular seizures) and a moderate limp in the dog's back leg, which evolved to a more severe condition in the following hours. Abdominal radiographic evaluation was requested, and abundant small, foreign, radio-dense bodies were detected, which were associated with Chinaberry tree fruits after surgical extraction. Adequate treatment was established, and the patient recovered completely. In addition, we compare clinical and gross postmortem findings in other similar cases reported in the literature. There is a general lack of information of such poisoning in pets.

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Keywords: Chinaberry, plant, dog, poisoning, toxicology

Chinaberry tree (*Melia azedarach*), also known as white cedar, pride of India, or Indian lilac, is a fast-growing deciduous tree with worldwide distribution. It is used in some countries for medicinal purposes,¹ including as an antihelmintic, a tonic, an antipyretic; also in the treatment of leprosy, eczema, and the relief of asthma attacks;² and even as an antiparasitic and antifungal agent.³ Nevertheless, it is mainly appreciated as an ornamental tree, and for this reason it has been widely cultivated for its shade around houses and along streets and roadsides, mainly in southern Europe. The fruits (Fig 1) consist of a small, round to ovoid seedpod or drupe that is approximately 1.5 cm in diameter, initially green and smooth but turning a pale yellow.⁴ This constitutes the most toxic part of the tree (the mature trees more than the green trees), whereas the leaves, bark, and flowers are only mildly toxic and usually cause no problems. Toxicity of the fruits is found within the pulp, whereas the shell and kernel are quite harmless. Most poisoning occurs in autumn and winter, when the berries ripen.^{5,6}

Death has been reported in children eating 6 to 8 ripe fruits; they developed malignant ulcers after forcing fruit into the nose, although death may not occur for some days after eating the fruit.⁷ The toxic components of the tree are tetranortriterpene neurotoxins of the cytotoxic limonoid class, known as meliatoxins A1, A2, B1, and B2,^{2,8} which are con-

centrated in the fruit and act as enterotoxins and neurotoxins. Other potentially toxic constituents include azadarin (alkaloid), meliotannic acid, and benzoic acid, or resins such as azaridine, parisine, and margosinine (found in the fruit, leaves, and bark, respectively).⁴ Curiously, some experiments have indicated that toxicity of the plant could be different according to the growing area or stage of growth and may be entirely absent in some trees.² In this sense, in Argentina the fruits are often ingested by children and domestic animals without causing intoxication,⁵ although this is a very old observation and more study is necessary.

The effects on the central nervous system are not unlike those produced by nicotine or anabasine alkaloids, with initial stimulation of sympathetic and parasympathetic ganglia followed by persistent depolarization and complete ganglionic blockage. The gastroenteritis is mainly associated with local irritation and inflammation.⁴

Melia azedarach intoxication has been observed in humans and domestic animals due to the ingestion of leaves or, mainly, fallen fruits, although they have a bitter taste; clinical signs in humans include nausea, vomiting, diarrhea, thirst, sweating, grinding of teeth, sleepiness, and convulsions.⁶ Chinaberry poisoning has been reported in horses, cattle, sheep, goats, pigs, dogs, rabbits, rats, guinea pigs, and poultry.^{1,6,9-15}

Clinical signs in domestic animals appear quickly, usually within 2 to 4 hours after ingestion of the plant. The main clinical symptoms are of 2 types and are quite similar to those observed in humans. The first involves nausea, vomiting, constipation, or diarrhea, frequently with blood, and colic, and the second is associated with the acute nervous system: excitement or depression, weakened heart action, convulsions, ataxia, paresis, dyspnea, coma, and death, with collapse of the circulatory system and insufficient breathing.^{2,6,16} The present study reports an unusual case of accidental

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1527-3369/06/0604-0171/00/0

doi:10.1053/j.tcam.2009.07.001



Figure 1. General aspect of Chinaberry tree fruits. (Color version of figure is available online.)

Chinaberry tree poisoning in a dog. This case is compared with other similar reports affecting not only canines but other domestic animals.

Clinical Report

A 4-year-old female hard-hair Teckel, 8 kg, was received and examined at the veterinary hospital with clinical signs of tremors (muscular seizures) and a moderate limp in its back leg, where sensor reflex was observed. Other notable clinical signs were absent, except for the abdomen, where colic pain and resistance were evident during abdominal palpation. Moreover, abdominal radiographic evaluation was requested, and the presence of abundant, small, foreign radiodense bodies was detected. In absence of a clear diagnosis, an initial treatment was established with paraffin oil (Hodernal, 6 mL/8 h/2 d, orally; Rottapharm, Valencia, Spain) and carprofen (Rimadyl, 4 mg/kg/d, orally; Pfizer, Madrid, Spain), and evolution of the pathology was closely observed.

The following day, the patient showed a slight recovery, becoming more active with a moderate decrease in pain, but abdominal auscultation revealed the presence of a large amount of gas associated with intestinal fermentation. Twenty-four hours later, the general status of the patient became stable, with no improvements, and an increase in pain (mainly around the abdominal area) was observed. The animal was normothermic (37.8°C) but somewhat depressed. Ataxia became more and more evident, especially in the hind legs. New radiological examinations were performed, in which the previously indicated foreign bodies were clearly observed and associated with a significant amount of gas (Fig 2) and intestinal handles strongly contracted. That same evening, both exploratory laparotomy and enterotomy were performed, and a great amount of complete and partially destroyed seeds from the Chinaberry tree (*Melia azedarach*) were identified. The patient remained at the hospital, and a complete treatment was

established, with therapy consisting of intravenous fluids (Ringer's lactate) to treat dehydration, corticosteroids (dexamethasone, 0.1 mg/kg/d, Voren; Boehringer-Ingelheim, Barcelona, Spain) to decrease inflammation and pain, and intramuscular metabolism stimulant with phosphor and vitamin (Catosal, 0.5 mL/24 h; Bayer, Barcelona, Spain) to stimulate the nervous system. With this general treatment, the following day the patient seemed completely recovered and was sent home.

The fifth day after the process started, evaluation of the nervous system determined a slight increase of ataxia, and a moderate recovery of abdominal pain was also observed. The previous treatment was thus modified as follows: a vitamin B1-B6-B12 supplement was administered (Hidroxil, 1 tablet/24 h, Almirall, Barcelona, Spain). We also advised passive rehabilitation to improve muscular tonicity.

Eleven and 14 days after surgical treatment, the patient was received at consultation to evaluate its evolution. Even if the nervous system problems had not become more serious, the dog was unable to stand for more than 4 to 5 seconds, and the animal tried to make slight movements of the hind legs. With those observations, the general treatment was maintained until complete recovery.

In the end, the patient was only able to stand without trouble after an entire month had passed, although a moderate ataxia was still observed when any kind of displacement was realized. The total disappearance of the pathological signs was delayed for more than 2 months.

Discussion

Toxic plants and seeds are found in apartments, houses, and gardens worldwide, and it is relatively common for dogs to accidentally consume house or outdoor plants.^{17,18} Nevertheless, there is a general lack of information concerning pet poisoning associated with plants, and most of the literature is



Figure 2. Lateral radiography of the affected dog, which shows the presence of foreign bodies and gas along the digestive system.

mainly associated with common garden plants, based on the clinical picture and management of those plant poisonings,^{17,19} although there has been a serious advance in this area in recent years.

In many cases, animals that ingest poisonous plants remain asymptomatic or only have moderate and transient gastrointestinal signs. However, sometimes severe clinical processes can be developed, depending on the toxic plant consumed and the amount, part, and developmental stage of the plant. Moreover, death can occur if poisoned pets are not brought immediately to veterinary emergency units and if they are not treated in time,²⁰ because, as with other potential poisoning, the sooner the management is initiated, the better the outcome.¹⁸

In this sense, when fatal poisoning associated with ingestion of Chinaberry tree fruits has been reported, on necropsy, acute cases reveal evidence of gastrointestinal irritation only, but in animals that survive for several days, histological examination shows fatty degeneration and hyperemia of the liver and kidneys.² The necropsy of a dog revealed severe renal congestion, moderate hepatic congestion, and a moderate amount of sero-sanguineous fluid in the abdominal area,¹¹ with fatal signs associated with circulatory collapse and respiratory distress. It must be considered that for severe poisoning, the animal has to chew or bite the fruits to release the toxicity.²¹

Some experimental Chinaberry poisonings have been developed for ruminants. Calves that received 20 g of fruits per kilogram body weight showed apathy and ruminal atony, sternal decubitus, difficulty drinking, hypothermia, dyspnea, and coma followed by death. Macroscopic findings were associated with intestinal congestion, focal or diffuse yellow discoloration of the liver, and brain congestion.¹² Similarly, swine exposed to 5 g of fruits per kg BW had slight diarrhea and spontaneous recovery, whereas those exposed to 10 to 20 g showed neurologic signs, hypothermia, and mortality. In this case, macroscopic signs were associated with congestion of intestinal and gastric mucous.¹³ Nevertheless, alterations in clinical pathology are, in general, inconsistent, with the majority of abnormal values related to the animal's response to stress and the release of endogenous steroids.¹⁹

Moreover, birds, such as ostriches, can be seriously affected by this plant after they engage in coprophagic and pecking activity. Clinical signs are quite similar to those previously described in mammals; macroscopic findings include intestinal congestion, focal or diffuse yellow discoloration of the liver, and brain congestion.¹⁴ Nevertheless, birds, goats, horses, donkeys, and ducks appear to be more resistant to poisoning because toxicosis is more difficult to produce, even experimentally.⁴

No specific, controlled, toxicokinetic studies have been performed in animals for Chinaberry poisoning. However, information from clinical and experimental reports suggests that absorption of the toxic properties is rapid after the ingestion of fruit, with clinical signs usually appearing within 1 to 2 hours after ingestion and most animals dying within 48 hours after the onset of adverse clinical signs.⁴

With respect to the treatment, no antidote exists. Animals

must be aggressively decontaminated before clinical signs become apparent.¹⁶ The rest of the treatment must be realized to eliminate the observed clinical signs. Even with an adequate treatment, prognosis is usually grave to poor after clinical signs progress to the nervous system. The prognosis is good if decontamination efforts (such as those made in the present case) are successful before the development of clinical signs.⁴

The diagnostic approach described here provided information on the diagnosis of Chinaberry poisoning in a dog. The popularity of *Melia azedarach* as garden and street plants in Spain as well as in other European countries provides pets, farming animals, and even children with access. The main interest of our case is to explain some of the nervous system signs associated with this type of poisoning, which, along with the digestive symptoms, could help in establishing a correct diagnosis. The rarity of poisoning incidents suggests that the risk of poisoning is very small compared with that of other toxic plants, but veterinarians must be aware of Chinaberry's toxicity; prevention and treatment of this poisoning would be enhanced by adequate veterinary education.

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