Toxicology Brief managing common poisonings in companion animals

❖PEER-REVIEWED

Cycad toxicosis in dogs

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Often referred to as *sago palms*, cycads are hardy evergreen yard plants that grow in warm states, such as Florida, North Carolina, and Georgia, and are also used as ornamental houseplants.[1] Lately, their increased use as houseplants has made them available all over the world.[2] Cycads, such as *Zamia floridana*, *Cycas revoluta*, and *Cycas circinalis*, arose from vegetation that dominated in the Mesozoic era.

Cycad leaves are one-pinnately divided and glossy green (*Figure 1*). The leaflets are narrow, with a sunken midrib, and the margins roll downward. These palmlike plants are dioecious (separate male and female plants in the same species). The female plant has a cone-shaped middle that produces seeds.[3-5] The pollen cone is large and erect; the seeds are loosely arranged with leaves around the stem. Each seed is large, plumlike, and pale-yellowish-tan.[1] Male cones are elongated and do not produce seeds.

Cycad toxicosis has been described in people and many animals, including dogs, sheep, and cattle. In this article, I focus on the clinical signs, diagnosis, and treatment of cycad toxicosis in dogs.

1. The glossy green leaves of *Cycas revoluta*. (*Photo courtesy of Dr. Safdar Khan, APCC, ASPCA.*)

TOXICITY AND MECHANISM OF ACTION

Cycad palms produce three toxins: cycasin, beta-methylamino-L-alanine, and an unidentified toxin.[3-6]

Cycasin, which is converted to its aglycone, methylazoxymethanol, may cause centrilobular and midzonal coagulative hepatic necrosis and gastrointestinal irritation. In addition, cycasin is carcinogenic, mutagenic, and teratogenic.[3,5,6]

Beta-methylamino-L-alanine, a neurotoxic amino acid, causes ataxia in rats and is implicated in Guam disease in people. Guam disease is characterized by symptoms similar to those of Alzheimer, Parkinson, and Lou Gehrig (amyotrophic lateral sclerosis) diseases.[3,5,6]

The unidentified toxin, a high-molecular-weight compound, may cause hindlimb paralysis in cattle because of axonal degeneration in the central nervous system.[3,5,6]

All parts of the plant are toxic, but the seeds contain higher amounts of cycasin than do other parts of the plant. Dogs usually ingest the seeds. Although toxic, the young leaves are palatable.[7-9]

CLINICAL SIGNS

In one study, the most common signs in dogs ingesting cycad plants were gastrointestinal, hepatic, and neurologic.[10] In cases reported to the American Society for the Prevention of Cruelty to Animals (ASPCA) Animal Poison Control Center and recorded in the AnTox database between 1988 and 1998, the most common signs of cycad toxicosis in dogs were vomiting (with or without blood), depression, diarrhea (with or without blood), and anorexia.[11] AnTox data from 2000 through 2007 show the same

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common signs of cycad toxicosis in dogs as mentioned above.

Gastrointestinal signs may develop within 24 hours, but bilirubin concentrations and serum alanine aminotransferase and alkaline phosphatase activities may not become elevated for 24 to 48 hours.[10] Signs may last from 24 hours to nine days.[11]

PATHOLOGY

Gastrointestinal abnormalities in dogs with cycad poisoning include hemorrhage and mucosal necrosis. Histologic abnormalities in the livers of these dogs include cirrhosis with marked focal centrilobular and midzonal coagulation necrosis.[2]

DIAGNOSIS

Cycad toxicosis is diagnosed based on a history of known exposure (*e.g.* observed ingestion, identification of chewed plants, identification of plant material in vomitus) and compatible clinical signs. Although cycasin and beta-methylamino-L-alanine can be found in the livers of animals that ingest cycad palms, no diagnostic laboratories routinely test for these compounds.[2]

TREATMENT AND MONITORING

Treatment of cycad toxicosis is symptomatic and supportive. No antidote for any cycad toxin is available.[2] Measure hepatic enzyme activities and bilirubin concentrations on presentation, and monitor these values daily for 72 hours. If elevations are found, monitor the parameters until they return to normal. Administer supportive therapy as needed.

Asymptomatic patients

Perform gastric lavage or induce emesis by using hydrogen peroxide (1 ml/lb; maximum = 45 ml) in asymptomatic dogs as soon as possible after suspected ingestion. Repeated doses of activated charcoal at 1 to 2 g/kg may be given after emesis.

Monitor dogs for secondary effects of liver failure, and treat these conditions as needed.

Symptomatic patients

If clinical signs are present, supportive therapy is indicated. For gastrointestinal signs, administer sucralfate (1 g in large dogs or 0.5 g in small dogs orally every eight hours) and cimetidine (5 to 10 mg/kg intravenously or orally every eight hours if the dog is not vomiting) or other gastric acid inhibitors.[12] Supportive fluid therapy with 5% dextrose in 0.9% saline solution is recommended, and blood glucose concentrations should be closely monitored.[13] Seizures and tremors may be controlled with diazepam (2 to 5 mg/kg intravenously in dogs, as needed).[12] If gastrointestinal tract hemorrhage is severe, blood transfusions may be necessary.

Monitor dogs for secondary effects of liver failure, such as coagulopathy, hepatic encephalopathy, hypoproteinemia, or renal failure, and treat these conditions as needed.[10] A low-protein bland diet is recommended. S-adenosyl-methionine (17 to 20 mg/kg daily in dogs) has been recommended in the long-term management of hepatic injury.[12]

PROGNOSIS

A patient's prognosis is good if treatment is instituted soon after cycad ingestion. However, if the patient is showing clinical signs, its prognosis is guarded.[2] The reported mortality rate in dogs with clinical signs is 32.1%.[11] ❖

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