

# Sago Palm Toxicosis in Dogs

Justine A. Lee, DVM, DACVECC, DABT  
CEO, VETgirl



## YOU HAVE ASKED...

How should we treat sago palm toxicosis in dogs?

## THE EXPERT SAYS...

According to the medical director, during the past 10 years, the ASPCA Animal Poison Control Center has had 1398 sago palm poisonings reported, with 90% of calls related to dogs (Tina Wismer, DVM, DABVT, DABT, personal communication, 2015).

Sago palm, members of the order Cycadaceae and genera *Cycads*, *Macrozamia*, and *Zamia*,<sup>1</sup> are a group of plants naturally found in tropical/subtropical environments such as the southern United States and Hawaii.<sup>1</sup> Plants from this family include Cycad (*Cycas cirinalis*), coontie (*Zamia pumila*), cardboard palm (*Zamia furfuracea*), and

Japanese cycad (*Cycas revoluta*).<sup>1</sup> Sago palm is sold as an indoor ornamental bonsai-type houseplant or landscaping plant, resulting in increased chance of exposure by dogs regardless of location.

The 3 primary toxins of sago palm include: azoglucosides (eg, cycasin), which results in hepatotoxicity; β-methylamino-L-alanine, which results in neurotoxicity; and an unknown high molecular weight agent.<sup>2</sup> All parts of the plant are considered poisonous, with the female plant considered more dangerous.<sup>1</sup> The seeds and nut contain large amounts of cycasin, and ingestion of as few as 2 seeds has been reported to result in clinical signs in dogs.<sup>2</sup>

Clinical signs seen with sago palm toxicosis can occur within 15 minutes to several hours after ingestion. Three main organ systems are affected by sago palm:

**All parts of the plant are considered poisonous, with the female plant considered more dangerous.**

## CLINICAL SIGNS OF SAGO PALM TOXICOSIS\*

- ▶ Inappetance
- ▶ Anorexia
- ▶ Vomiting
- ▶ Hematemesis
- ▶ Generalized malaise
- ▶ Diarrhea
- ▶ Melena
- ▶ Hematochezia
- ▶ Dehydration
- ▶ Ascites
- ▶ Abdominal pain
- ▶ Icterus
- ▶ Ataxia
- ▶ Tremors
- ▶ Seizures
- ▶ Coma
- ▶ Death

\*Ordered by prevalence in the author's clinical experience

the GI tract, liver, and central nerve system (CNS). Typically, GI signs (eg, anorexia, hypersalivation, vomiting, diarrhea) are seen initially. Within 2 to 3 days, signs of acute hepatic necrosis (AHN; eg, increased liver enzymes, icterus, melena, vomiting, hepatic encephalopathy) and CNS signs (eg, weakness, ataxia, seizures, tremors) can develop. (See **Clinical Signs of Sago Palm Toxicosis**.)

### Diagnostics

When presented with a patient affected by sago palm toxicosis, routine diagnostics (eg, CBC, chemistry panel, urinalysis) should be performed. While the patient is hospitalized, daily blood work should include packed cell volume/total solids, blood glucose, and hepatic panel (including liver enzymes and albumin). For patients that develop evidence of hepatic injury, additional clinicopathologic and diagnostic testing should include coagulation parameters (eg, prothrombin time [PT], activated partial thromboplastin time [aPTT], platelet count,) serum bile acids (or ammonia), and abdominal ultrasound.

In dogs with AHN secondary to sago palm toxicosis, clinicopathologic findings may include elevations in liver enzymes (eg, aspartate transaminase, alanine aminotransferase, alkaline

phosphatase) and/or abnormal bile acids; however, elevations may not occur for 24 to 48 hours and may last from 2 to 9 days.<sup>1</sup> Other findings may include anemia (secondary to melena) or hemoconcentration (secondary to dehydration), evidence of liver failure (eg, hyperbilirubinemia, hypoalbuminemia, hypocholesterolemia, hyper- or hypoglycemia, hyperlactatemia), leukocytosis, thrombocytopenia, and coagulopathy (eg, prolonged PT, aPTT). Increased blood urea nitrogen (BUN) caused by prerenal azotemia or GI bleeding<sup>2</sup> or decreased BUN secondary to liver failure may also be seen.

Ideally, abdominal ultrasound should be performed to rule out other underlying disease (see **Differential Diagnoses for Acute Hepatic Injury or Hepatopathy**, page 54). However, ultrasound may not reveal any obvious gross ultrasonographic findings with AHN. To better assess this, liver biopsies should ideally be performed, provided the patient is not coagulopathic. Histopathologic lesions seen with sago palm toxicosis include marked focal centrilobular and midzonal coagulation necrosis, along with cirrhosis.<sup>2</sup>

### Treatment

As ingestion of this plant can result in severe clinical signs and AHN, aggressive decontamination and treatment is warranted. See **Table** for dosages. In patients without signs of toxicosis, such as recent ingestion, decontamination should be performed. Appropriate emetic agents (eg, for dogs, hydrogen peroxide or apomorphine) should be used if the patient is not already vomiting. Multiple doses of activated charcoal (eg, 1-5 g/kg PO) should be administered, with the first dose containing a cathartic (eg, sorbitol) if possible. Antiemetics (eg, dolasetron,

**As ingestion of this plant can result in severe clinical signs and AHN, aggressive decontamination and treatment is warranted.**

**TABLE**

**DRUG DOSES FOR COMMON MEDICATIONS USED TO TREAT SAGO PALM TOXICOSIS<sup>1,6</sup>**

<b>Drug</b>	<b>Use</b>	<b>Dose</b>
Activated charcoal	To minimize absorption from the GI tract	1-5 g/kg PO once. If multiple doses are used, dose at 1-2 g/kg PO 4-6 times a day for 24 hours
Diazepam	Anticonvulsant	0.25-0.5 mg/kg IV prn (no max dose)
Dolasetron	Antiemetic	0.6-1 mg/kg IV, SC twice a day
Levetiracetam	Anticonvulsant	20-60 mg/kg IV 3 times a day or prn
<i>N</i> -acetylcysteine	Hepatoprotectant	140- 280 mg/kg IV or PO once, followed by 70 mg/kg IV or PO every 6 hours for 2-3 days as needed. <i>IV route preferred to prevent interaction with activated charcoal.</i>
Ondansetron	Antiemetic	0.6-1 mg/kg IV, SC twice a day
Plasma (as fresh frozen or frozen)	Used to treat coagulopathy secondary to hepatic injury	10-20 mL/kg IV if coagulopathic
<i>S</i> -adenosylmethionine	Hepatoprotectant	18-20 mg/kg PO once a day
Vitamin K <sub>1</sub>	Used to treat coagulopathy secondary to Vitamin K-dependent factors inactivated during hepatic injury	1 mg/kg PO, SC twice a day if coagulopathic

ondansetron) should be promptly initiated to aid in patient comfort and to prevent emesis of charcoal or secondary aspiration pneumonia.

Additional therapy should include IV fluids, hepatoprotectants (eg, *S*-adenosylmethionine, *N*-acetylcysteine), GI support, appropriate monitoring, and supportive care. Ideally, the use of a balanced crystalloid (eg, Normosol-R) should be used to aid in hepatic perfusion and treat ongoing fluid losses (eg, vomiting, diarrhea). Lactated Ringer solution should be avoided if hepatic injury is evident; with hepatic injury, hyperlactatemia may

occur as a result of inappropriate metabolism of the buffer, lactate, to bicarbonate by the liver. In hypoproteinemic patients (total solids <5 gm/dL), the use of colloids (eg, hetastarch, VetStarch) can be considered to help maintain colloid osmotic pressure.

Hepatoprotectants (eg, *S*-adenosylmethionine, *N*-acetylcysteine) can be used to increase glutathione levels, act as anti-inflammatories, improve microcirculation, act as a reactive oxygen species scavenger, improve membrane stabilization, and serve as antiapoptotic agents.

AHN = acute hepatic necrosis  
 aPTT = activated partial thromboplastin time  
 BUN = blood urea nitrogen  
 PT = prothrombin time

## DIFFERENTIAL DIAGNOSES FOR ACUTE HEPATIC INJURY OR HEPATOPATHY

### Toxicologic

- ▶ Acetaminophen/paracetamol
- ▶ *Amanita* spp mushrooms
- ▶ Blue-green algae
- ▶ NSAIDs (typically chronic or subacute exposure)
- ▶ Xylitol
- ▶ Iron (rare)

### Underlying Disease

- ▶ Anatomic abnormalities (eg, portosystemic shunts, microvascular dysplasia)
- ▶ Cirrhosis
- ▶ Cholangiohepatitis
- ▶ Chronic hepatopathy
- ▶ Extrahepatic biliary duct obstruction (eg, pancreatitis)
- ▶ Neoplasia

AHN = acute hepatic necrosis  
 PT = prothrombin time  
 PTT = partial thromboplastin time

GI support (eg, H<sub>2</sub>-blockers, proton-pump inhibitors, sucralfate) should be initiated for presumptive increased gastrin levels. In patients with neurologic signs, hepatic encephalopathy should be ruled out or treated. Additional therapy for AHN patients may require dextrose supplementation, blood pressure monitoring, anticonvulsants, thermoregulation, coagulation support (eg, Vitamin K<sub>1</sub>, plasma transfusions), and supportive care.

### Prognosis

With sago palm toxicosis, prognosis varies depending on how soon the toxicant is identified, speed of decontamination, and severity of clinical signs. Once AHN develops, prognosis is poor to grave. The published mortality for sago palm toxicosis ranges from 32% to 50%.<sup>3,4</sup> However, unpublished data from the past 10 years at the ASPCA Animal Poison Control Center reveals that of 1398 cases, only 33 dogs died or were euthanized (ie, 2.4% mortality). This is likely due to multiple factors, such as onset of decon-

tamination by pet owners at home, improvements in quality of medicine, and retrospective bias from tertiary academic settings (where the worst cases may have been referred). Ferguson, et al, found that nonsurvivors were more likely to have evidence of increased ALT and total bilirubin, evidence of coagulopathy (prolonged PT/PTT), and hypoalbuminemia.<sup>5</sup> Survivors were more likely to have received activated charcoal,<sup>5</sup> which reiterates the importance of aggressive decontamination with sago palm toxicosis.

### Conclusion

Veterinarians should be aware of the danger of sago palm resulting in severe, potentially fatal acute hepatic necrosis or injury. When in doubt, an animal poison control center should be consulted for this potentially life-threatening plant ingestion. Pet owners should be cautioned about the dangers of sago palm plants. The aggressive use of decontamination and supportive care is necessary to ensure good outcome. ■

### References

1. Klatt CA. Sago Palm. In: Osweiler GD, Hovda LR, Brutlag AG, Lee JA, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*. Iowa City, IA: Wiley-Blackwell; 2010:743-741.
2. Albretsen JC. Cycasin. In: Plumlee KH, ed. *Clinical Veterinary Toxicology*. St. Louis, MO: Elsevier Mosby; 2004:392-394.
3. Albretsen JC, Khan SA, Richardson JA. Cycad palm toxicosis in dogs: 60 cases (1987-1997). *JAVMA*. 1998;213(1):99-101.
4. Fatourechil L, DelGiudice LA, Sookhoo N. Sago palm toxicosis in dogs. *Compend Contin Educ Vet*. 2013;35(4):E1-8.
5. Ferguson D, Crowe M, McLaughlin L, Gaschen F. Survival and prognostic indicators for cycad intoxication in dogs. *JVIM*. 2011;25(4):831-837.
6. Plumb DC. *Plumb's Veterinary Drug Handbook*, 7th ed. St. Louis, MO: Wiley-Blackwell; 2011.