Vet Education Pty Ltd
In Conjunction with
The ASPCA Poison Control Centre
Presents...

The 2nd Annual Online Veterinary Toxicology Symposium

Cycad (Sago) Palm Toxicity in Dogs and Cats
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Sago Palm Toxicity

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Plant overview, origins, images

Origins in Mesozoic era (age of the Cycads, if you’re not a zoologist; age of the dinosaurs if you are))

Cycas is most widely distributed cycad genus; Madagascar to east coast of Africa, northern Australia to southern Japan. In North America, landscape plant in Florida, along Gulf Coast, southern Texas and southern California. Sold as house plants, as well, so climate no longer a limiting factor.

History

First documented toxicity reported are related to Captain James Cook’s explorations in Australia. The men ate roasted husks found in an aboriginal campfire, which they had assumed to be edible; the seeds turned out to be noxious, causing severe diarrhea and vomiting. The same seeds, when fed to pigs, caused severe signs – including deaths over several weeks. They were unaware, at the time, that native peoples prepared the seeds of Cycas species by repeated washing/soaking and drying.

Toxins

The plant contains glycosides of methylazoxymethanol (MAM) and a neurotoxic amino acid (B-N-methylamino-L-alanine (L-BMAA)), as well as yet unidentified toxins. Principle glycosides are cycasin and macrozamin, which are subject to B-glucosidases in a plant or by bacterial enzymes in the gut. This chemical process releases the aglycone MAM and sugars.

Mechanisms

The aglycone MAM alkylates DNA and RNA, with the primary effect of acute hepatic changes.

The neurotoxin, L-BMAA, is primarily linked to ataxia and hind limb paresis in cattle. There is overlap with the clinical signs, and often those with neurologic signs also experience hepatopathy.

Toxic doses

All parts of the plant contain the toxins, with the cycasin concentrations highest in the leaves and, the roots, lower in the seeds, and lowest in the pith; the L-BMAA is most concentrated in the seeds. There is no “safe” level for ingestion of the plant.
Clinical signs by species

Dogs: Vomiting, diarrhea, lethargy, tremors, liver damage with progressive evidence biochemical changes characteristic of that damage (elevated ALT, bilirubinaemia, hypoalbuminaemia, prolonged PT / aPTT, and death (reported mortality rates up to 50% even in treated patients, vary between surveys and may be due to classifications by history vs evidence)

Sheep: Similar to the hepatic signs noted in dogs, as well as neurologic signs. More likely to be sub-acute to chronic, with emaciation, pallor, icterus, lethargy; diarrhea is rare

Cattle: Most often neurologic signs, resulting from days to weeks of ingestion of the leaves. Proprioception deficits and progressive weakness of the pelvic limbs – stumbling, staggering, ataxia, abnormal gait. This syndrome has been observed in tropical regions such as the Caribbean, not in North America.

Pathology, histopathology

In dogs, sheep and cattle, there are changes both in brain and in liver. Even when no gross changes – as when only neurologic signs in cattle – there are generally significant changes to neuro tissues.

Management of ingestion/intoxication in dogs

Asymptomatic patients – focus is on decontamination and close monitoring: activated charcoal, baseline and follow up biochemical profiles. If decontaminated within hours and no clinical signs or biochemical changes by 24-36 hours, good prognosis for remaining healthy

Symptomatic patients – need to control presenting signs while proceeding with decontamination and supportive care: anti-emetics, activated charcoal if can be done safely, gastrointestinal protectants, aggressive fluid support, serial monitoring of biochemical profiles, liver protectants, n-acetylcystiene, s-adenosylmethionine (s-AME)

Case studies – a tale of two dogs

Review signalment, history, initial presentations

Clinical treatment, responses