Pacific Tide

An informational monthly newsletter

June 2012
Volume 6, Issue 1

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Author of the month:

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Dr. Good graduated from Cornell University with a Doctorate in Veterinary Medicine in 1999. Her internship in small animal medicine and surgery at Angell Memorial Hospital in Boston followed, and was completed in 2000. She continued on at Angell Memorial for her residency training in emergency medicine and critical care (2000-2003), and joined PVSES upon completion. She became board certified in 2004. Dr. Good finds the challenge of helping animals through critical medical emergencies to be extremely rewarding.

Amanita Mushroom Toxicity

Amanita mushrooms - why should I care about those?

Unfortunately, the Monterey Bay area of California has a high prevalence of Amanita mushrooms, one species of which is also known as the Death Cap mushroom (Amanita phalloides). Dogs, puppies in particular, seem interested in, and prone to ingestion of these mushrooms; typically resulting in severe, often fatal complications. 10+ years ago, many believed that patients who were clinical for this toxicity would universally have a fatal result. However, we now know that with rapid and very intensive intervention, some of these patients can be saved.

Anyone who hikes in this area is likely aware of the multitude of mushroom species that grow in greater Monterey Bay. Not all cause the severe degree of pathology as the Amanita species. Many are non-toxic or may only cause transient GI distress. The focus of this article will solely be on Amanita mushrooms, although other non-Amanita mushrooms may cause serious pathology as well. Distinguishing between toxic and non-toxic mushrooms based on appearance can be very difficult, and is best left to trained individuals. Identification of chewed or vomited mushrooms can be even more difficult to interpret. Intact Amanita phalloides, the Death Cap mushroom, has a typical "toad stool" appearance with a smooth, wide cap that can have a yellow to greenish pigment. There are white gills under the cap. The stalk is white to pallid with a rounded bulb at the base.

Amanita phalloides:

http://www.amanitashop.com/amanita-poisoning/deathcap.htm#description

The North American Mycological Association keeps a list of individuals grouped by location that are willing to identify mushrooms. We have had good success utilizing some of these people for identification of mushrooms that were chewed or ingested by our patients. The web link is: http://namyco.org/toxicology/identifiers.html

Clinical Signs

The clinical signs associated with Amanita toxicity vary dependent on severity of cyclopeptide toxic effect, as well as time course of illness. This can make establishing a suspicion of Amanita toxicity as a differential more difficult, especially early in the course of illness.

Up to 12 hours after ingestion, many patients will exhibit non-specific gastrointestinal signs including vomiting, diarrhea, and abdominal discomfort. Elevation of liver values may not be significant at this time. Some patients may appear to improve for 12-24 hours, although bloodwork can show mild elevations in ALT. Following this phase of apparent improvement, Amanita toxicity patients will exhibit more significant clinical deterioration with lethargy, recurrent GI signs, hypoglycemia, coagulopathy, severe hepatic dysfunction, and sometimes renal failure. Blood tests at this time typically show very severe elevation in ALT. Unfortunately, some in-house blood chemistry analyzers are unable to accurately assess this degree of elevation and may not give a test result. ***If you do not get an accurate reading, please consider that the value may be too high for your analyzer to read.

Expected Test Results

At the time of acute liver failure, chemistry results typically reveal severe elevation in ALT – in the multiple to tens of thousands. Alk Phos is also typically severely elevated. Bilirubin elevation may lag behind the ALT increase. Patients are often hypoglycemic, and this may persist despite dextrose supplementation. PT and PTT are typically prolonged in the severely affected patients. Azotemia may develop if there is a concurrent hepatorenal component of illness.

Confirmation of Amanita toxicity can be made through documentation of the Amanita toxin in urine, blood, liver, or kidney tissue. The Toxicology Laboratory at UC Davis will run this test.

Treatment Options

Induction of emesis is indicated if there is known, recent ingestion of a potentially toxic mushroom. Treatment with activated charcoal is typically recommended, and repeated dosing may help to reduce enterohepatic recirculation.

General medical support including correction of hydration deficits and electrolyte imbalance is essential. Synthetic colloid support can be useful in patients with decreased oncotic pressure. H2 blockers to reduce gastric acidity are frequently used. Anti-emetic therapy – sometimes with multiple medications, can be necessary to try to control vomiting. Coverage with broad spectrum parenteral antibiotics is recommended due to the potential for bacterial translocation.

Hypoglycemia is a frequent complication of Amanita toxicity. Supplementation of crystalloids with dextrose is often necessary. Some patients will develop refractory hypoglycemia despite supplementation of up to 10% dextrose. As phlebitis with higher dextrose concentrations is a concern, placement of a central catheter should be

considered if dextrose needs exceed 5%.

Coagulopathy is also a frequent complication of Amanita toxicity. Intensive and repeated treatment with fresh frozen plasma is often necessary to try to optimize individuals' coagulation statuses. Fresh frozen plasma has many additional benefits including protein and enzyme support. Therapy with Vitamin K is also recommended as liver failure-induced coagulopathy can have relative Vitamin K deficiency as a component of pathology.

There is no antidote available in the United States for Amanita poisoning. Silibinin dihydrogen given in injectable form has been shown to increase survival rates in humans, but is not available in the US. Silymarin – derived from milk thistle, may have similar properties but is less bioavailable. Still, therapy with silymarin is recommended. Acetylcysteine is also typically used for hepatoprotectant and antioxidant properties. After an initial regimen of injectable acetylcysteine over multiple doses, conversion to SAMe when the patient is improved and holding down food is often considered.

Some patients will develop neurologic deterioration from hepatic encephalopathy. Therapy with lactulose and mannitol is sometimes considered. Neurologic deterioration in the absence of hypoglycemia warrants consideration of hepatic encephalopathy.

Prognostic Indicators

Multiple factors including amount ingested, time course of medical intervention, and level of intensive care provided likely explain variation in outcome of patients that have ingested Amanita mushrooms. Patients can appear *severely* affected, but may still survive with intensive support. At PVSES we have found certain parameters to be associated with survival. Patients who have refractory hypoglycemia requiring progressively higher (7.5-10% or more) dextrose supplementation as a CRI seem to be correlated with a worse outcome, whereas patients that stabilize on 5% or less dextrose appear to do better overall. Very high ALTs are seen even in patients who ultimately survive, but in my experience ALTs >20,000 within the first 36 hours of presentation are typically associated with poor outcome. Although I have seen some patients with hepatic encephalopathy eventually recover, neurologic signs in a euglycemic patient warrants a very guarded prognosis. Concurrent azotemia is a marker for concern, and oliguria may develop. Anuria despite medical therapy is typically associated with a dire outcome.

Conclusion

Unfortunately, in this area Amanita toxicity is something that we as veterinarians face in our patients. It should be on our differential list when compatible clinical signs or labwork results are identified. The suspicion of Amanita toxicity warrants rapid and intensive medical intervention to give the best possible chance for patient survival. Hospitalization for 24 hour care with multi-modal medical therapy including repeated plasma transfusions will optimize patient stability and potential for recovery. Although some cases may still have a fatal result, other dogs are able to be saved when a committed family elects to move forward with very intensive medical care.

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About Our Organization

PVSES was founded to provide high quality, specialized medical care to companion animal patients. Our practice is dedicated to serving the veterinary community as a partner in total patient care. We offer comprehensive specialized services including endoscopy, Doppler ultrasound, surgery, 24-hour ICU care, and emergency and critical care. Our staff is committed to providing compassionate and thorough medical care that meets the needs of the patient, client, and referring veterinarian.

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