An 8-year-old, 21.8-kilogram (48-pound), spayed female dalmatian presented 24 hours after ingesting 113.4 g (4 ounces) of raisins.

**History.** The owner found the remains of a box of raisins on the floor. The ingestion had occurred sometime during the day, while the owner was at work. The owner had found vomitus containing raisins on the floor, but the dog appeared normal. Intact raisins were seen in the stool during the dog’s evening walk. The following evening, the dog was lethargic and anorectic. The owner found additional evidence of vomiting and diarrhea. The dog had a history of colitis.

**Examination.** On physical examination, the dog had a mildly painful abdomen. Diarrhea was present, the dog was markedly depressed, and 8% dehydration was noted. Vital signs were normal except for mild hypothermia (temperature, 99.5º F).

**ASK YOURSELF…**

What is your initial treatment plan?

A. Hospitalize the dog, obtain baseline chemistry panel and urinalysis, and begin fluid therapy.

B. Inject metoclopramide; prescribe sucralfate, famotidine, and metronidazole. Instruct the owner not to feed the dog for 12 hours and to contact you if no improvement is noted in 24 hours.

C. Treat for colitis, a preexisting medical condition in this dog.

D. Lecture owner about dog’s “counter-surfing”; then prescribe a bland diet.

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**Raisin Toxicosis in a Dalmatian**

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Correct Answer: A
Hospitalize the dog, obtain baseline chemistry panel and urinalysis, and begin fluid therapy.

In 1999, a trend of dogs developing acute renal failure after ingesting raisins or grapes was tracked. The lowest documented raisin dose leading to renal failure is 3.11 g/kg (0.11 oz/kg); the lowest grape dose, 19.85 g/kg (0.7 oz/kg) body weight. Raisins are about 4.5 times more concentrated than grapes on a per-ounce basis.

Pathophysiologic Mechanism. Unknown. Tests of raisins and grapes for pesticides, heavy metals, and mycotoxins have been negative. Some dogs that eat raisins or grapes do not develop any clinical signs; others develop mild gastrointestinal signs and recover; still others develop acute renal failure despite being asymptomatic after exposure.

Clinical Signs. Clinical signs typically begin within 24 hours of ingestion. Vomiting may be immediate or delayed. Common clinical signs include lethargy and anorexia. Diarrhea and abdominal pain are frequently present. Oliguria or anuria, ataxia, and weakness have been associated with increased mortality. Dogs may present with hypertension, hypothermia, dehydra
tion, tremors, and polydipsia. Rarely, arrhythmias, hypersalivation, and seizures may occur; pancreatitis is also rare. Disseminated intravascular coagulopathy has occurred in some dogs during treatment.

Chemistry & Histopathology. Creatinine, phosphorus, and the calcium × phosphorus product may become elevated within 24 hours; BUN and calcium elevate within 3 days of ingestion. Hyperglycemia, elevated alanine transaminase levels, and hyperlipasemia are common. Hypercalcemia, elevated calcium × phosphorus product, hyperkalemia or hypokalemia, and acidosis are associated with higher mortality.

On urinalysis, glycosuria, proteinuria, or cylindruria may be present.

Proximal renal tubular degeneration or necrosis is a consistent finding on histopathologic examination. Tubule basement membranes are generally intact. Evidence of tubular regeneration may be seen. Mineralization of kidneys and other tissues, including gastric mucosa, myocardium, lung, and blood vessel walls, may occur.

Treatment. The first step of treatment is decontamination. Emesis can be effective up to 12 hours and active charcoal up to 24 hours after ingestion. The rapidity and degree of success with decontamination can influence subsequent treatment recommendations.

Obtain baseline chemistry, urinalysis, and CBC. Monitor BUN, creatinine, calcium, phosphorus, calcium × phosphorus product, electrolytes, total protein, and hematocrit on a daily basis and then at less frequent intervals as signs resolve.
Correct dehydration and start fluid therapy at two times the maintenance rate; be sure to check for overhydration during fluid therapy. Monitor urine output; if oliguria develops, mannitol, furosemide, and dopamine may be used to try to increase urine flow.

Control vomiting with metoclopramide. Phenothiazines are contraindicated if the dog is dehydrated. Use histamine antagonists (cimetidine, famotidine) and sucralfate to treat uremic gastritis. If the dog has hyperphosphatemia, aluminum hydroxide is recommended to bind intestinal phosphorus. At this time, it is unknown whether hemodialysis or peritoneal dialysis is effective in binding and removing the toxic component. However, it may give the animal time to regenerate renal tubules.

**Differential Diagnosis.** Any other cause of acute renal failure. Specific differentials include ethylene glycol, leptospirosis, aminoglycosides, bacterial pyelonephritis, and chronic renal failure.

**Treatment Endpoint & Prognosis.** Continue treatment until clinical signs and azotemia resolve. Treatment may be required for several days to several weeks, although most cases resolve within 7 days. If acute renal failure develops, prognosis is guarded.

**Follow-up.** In this case, the dog was azotemic and had elevated creatinine levels. Calcium and calcium × phosphorus product were within normal levels. She was treated with fluid diuresis, metoclopramide, sucralfate, and famotidine. After 5 days, she regained her appetite, and azotemia began to improve. After 8 days, clinical signs had resolved completely.

See Aids & Resources, back page, for references, contacts, and appendices.

**TAKE-HOME MESSAGE**
- Consider grape or raisin toxicosis as a differential in dogs presenting with acute renal failure.

**COMING SOON**

to these pages…

- **Complications:** Treatment of Congestive Heart Failure
- **Devices:** Holter vs Event Cardiac Monitors
- **What’s the Take-Home?** Feline Esophageal Ulcers
- **Applied Behavior:** Leash-Training the Adult Dog
- **Procedures Pro & Applied Cytology:** Cerebrospinal Fluid Taps
- **How to Refer:** Cataracts

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