MSD MANUAL
Veterinary Manual

PROFESSIONAL VERSION

Cholecalciferol (Vitamin D_3) Poisoning in Animals

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Since the introduction of the EPA regulatory changes, exposures to cholecalciferol (vitamin D_3) have increased markedly in frequency in the US. Available in many formulations, including soft baits, hard blocks, and pellets, cholecalciferol is usually found in a concentration of 0.075% (0.75 mg/g).

As with other active ingredients and products, nontarget species are exposed to cholecalciferol typically after inadvertent ingestion of the bait product itself. Acute relay toxicosis has not been documented in research settings; however, chronic ingestion of prey or carrion that had consumed or died of cholecalciferol exposure has been shown to lead to mild reversible clinical signs consistent with poisoning. Given the narrow margin of safety of this active ingredient, rare relay toxicosis may theoretically be possible in companion animals.

Cholecalciferol mechanistically disrupts calcium and phosphorus homeostasis within the body. Transported systemically from the gut via specific binding proteins, cholecalciferol travels first to the liver for conversion to 25-hydroxycholecalciferol (calcifediol) and then to the renal tubules for conversion to 1,25-dihydroxycholecalciferol (calcitriol). Although calcitriol is the most bioactive form of vitamin D, ingestion of large quantities of cholecalciferol, as are found in rodenticide products, overwhelmingly increases calcifediol concentrations that impart harmful effects on calcium homeostasis as well. Peak concentrations of calcitriol are reached after 48–96 hours, accounting for some amount of delay in both clinical signs and laboratory changes.

After ingestion and conversion in the liver and kidneys, vitamin D_3 ultimately disrupts calcium homeostasis by increasing calcium and phosphorus absorption from the gut, increasing calcium absorption from the distal renal tubules, and inducing mobilization of calcium from the bone with osteoclastic effects—an influence that is not entirely understood.

Toxic doses of cholecalciferol are much lower than reported acute lethal doses (13 mg/kg) and LD_{50} (88 mg/kg), and important clinical changes with notable morbidity, and even death, may be noted with relatively low doses. Therefore, although the acute lethal dose and LD_{50} are recognized, they are rarely used in guiding aggressive care and monitoring in companion animal patients.

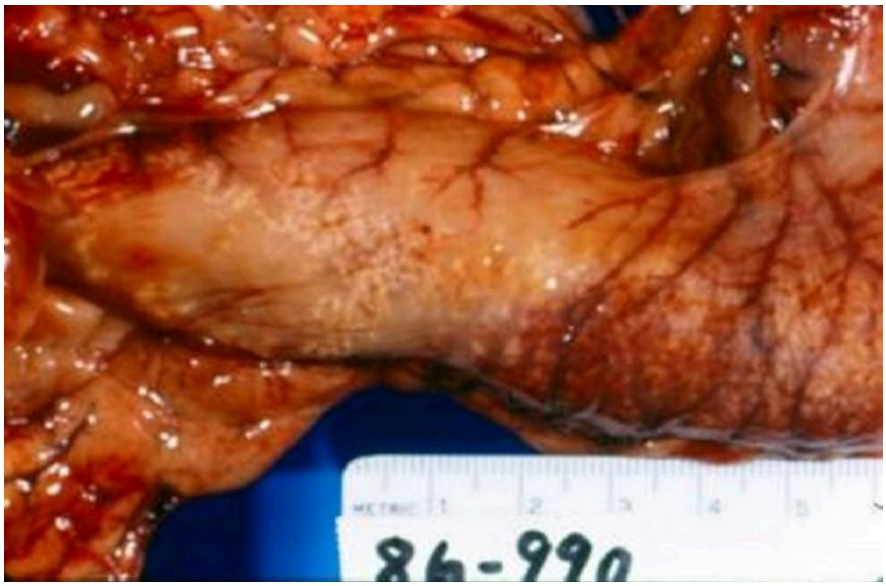
Clinical signs may be noted in doses as low as 0.1 mg/kg, with appreciable elevations in calcium and phosphorus concentrations leading to metastatic soft tissue mineralization in doses

exceeding 0.5 mg/kg. Soft bait or bait block products often weigh between 14 and 28 g, thus containing 10.5–21 mg of cholecalciferol per bait.

Clinical signs and laboratory changes often develop within 12–48 hours after ingestion of cholecalciferol; they may include **weakness, anorexia, vomiting, polyuria and polydipsia, dehydration, metastatic mineralization of the soft tissues, and consequent systemic effects** depending on the organ tissues affected (see serosal calcification image).

Serosal calcification, cholecalciferol poisoning

IMAGE



COURTESY OF DR. FREDERICK W. OEHME.

Persistent elevations in calcium and phosphorus leading to metastatic mineralization commonly affect the kidneys and may lead to renal injury, dysfunction, or failure. However, any soft tissue, including that of the heart, lungs, and GI tract, may be affected, and clinical signs may vary to some extent, depending on the affected organ system.

Patients that remain clinically normal and maintain stable and normal calcium and phosphorus values 72–96 hours after ingestion would not be expected to develop clinical signs or changes associated with toxicosis.

Pearls & Pitfalls

- Patients that remain clinically normal and maintain stable and normal calcium and phosphorus values 72–96 hours after cholecalciferol ingestion would not be expected to develop clinical signs or changes associated with toxicosis.

Differential diagnoses may include exposure to vitamin D_3 supplements or vitamin D_3 analogues (calcitriol and calcipotriene), renal disease, and neoplasia.

Decontamination:

- Induction of emesis, if ingestion occurred within 4 hours, using the following:
    - In dogs: apomorphine, ropinirole, or hydrogen peroxide
    - In cats: dexmedetomidine, hydromorphone, or xylazine
- Dose-dependent administration of activated charcoal:
    - Cholecalciferol dose of 0.1–0.5 mg/kg: single dose of activated charcoal (1–2 g/kg, PO as aqueous slurry) with a cathartic
    - Cholecalciferol dose > 0.5 mg/kg: single dose of activated charcoal (1–2 g/kg, PO as aqueous slurry) with a cathartic, followed by cholestyramine administration:
        - Cholestyramine (0.3–1 g/kg, PO, every 8 hours for 3–4 days). Caution: there is a version of this product for humans that contains xylitol.
        - If cholestyramine is not available, administer activated charcoal (1–2 g/kg, PO as aqueous slurry) with a cathartic, followed by activated charcoal without a cathartic, every 8 hours for up to two additional doses. Ensure that the patient is at low risk of aspiration, receives IV fluid therapy for a minimum of 4–6 hours after the last dose, maintains normal and stable sodium concentrations as checked before each dose, and is passing stool.

Treatment in nonclinically affected patients (dose dependent):

- Cholecalciferol dose of 0.1–0.5 mg/kg: SC fluid therapy and outpatient laboratory testing monitoring the following:
    - Baseline calcium concentration (ionized calcium concentration is ideal), phosphorus concentration, PCV, TP concentration, BUN concentration, creatinine concentration, electrolyte panel, and prefluid urinalysis
    - Recheck of calcium concentration, phosphorus concentration, PCV, TP concentration, BUN concentration, creatinine concentration, and body weight 72 hours after ingestion—sooner if clinical signs are noted
- Cholecalciferol dose of > 0.5 mg/kg:
    - Baseline calcium concentration (ionized calcium concentration is ideal), phosphorus concentration, PCV, TP

concentration, BUN concentration, creatinine concentration, electrolyte panel, and prefluid urinalysis

- Recheck of calcium concentration, phosphorus concentration, PCV, TP concentration, BUN concentration, creatinine concentration, and body weight every 24 hours until 72–96 hours after ingestion
- IV fluid therapy at twice the maintenance dose for 24 hours to safely facilitate the administration of multiple doses of activated charcoal
- Cholestyramine (0.3–1 g/kg, PO, every 8 hours for 3–4 days) to further circumvent enterohepatic circulation, spaced between charcoal doses for the first day

Treatment in clinically affected patients:

- GI support as needed
- Hyperphosphatemic patients: aluminum hydroxide (30–100 mg/kg every 24 hours, PO mixed with food, divided with each meal)
- Hypercalcemic patients: monitoring of calcium concentration from patient baselines in young animals where concentrations may be naturally elevated secondary to growth.
    - Bisphosphonates such as pamidronate or zolendronate are favored for effectiveness at decreasing osteoclastic bone activity. Most patients will respond to a single IV dose with notably lower calcium concentrations within 1–3 days. Some patients may require a second dose.
    - Prednisone in dogs or prednisolone in cats (1 mg/kg, PO, every 12 hours, tapered as calcium concentration improves) to decrease calcium absorption from the gut and bone and to enhance excretion from the kidneys
    - Furosemide may be considered (2 mg/kg, SC, IV, or PO, every 8–12 hours) to aid in calcium excretion through the kidneys; however, it can be dehydrating and lead to electrolyte disturbances. Furosemide administration is generally discontinued when a bisphosphonate is dosed.
    - Salmon calcitonin may be considered; however, it is rarely used because of the refractory response after repeated doses and the improved affordability and availability of the more effective bisphosphonate products.

The prognosis with cholecalciferol poisoning varies depending on the dose ingested and the clinical course. **The extended half-life of cholecalciferol and its metabolites often results in prolonged**

clinical signs that may require weeks of consistent laboratory monitoring and sporadic adjustments to treatment.

If metastatic calcification occurs, chronic systemic disease (eg, renal disease) may occur and require some amount of long-term management and care.

Key Points

- **Cholecalciferol poisoning disrupts calcium homeostasis, causing an elevation in phosphorus and calcium concentrations within 72 hours and resulting in soft tissue calcification, commonly in the kidneys.**
- **No antidote for cholecalciferol poisoning is available.**
- **Due to cholecalciferol's long half-life, resolution of hypercalcemia and resulting effects can take weeks of consistent monitoring and adjustments to treatment.**

For More Information

- Rumbeiha WK. Cholecalciferol. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology*. 3rd ed. Elsevier; 2013:489-497
- Klainbart S. Aroch I. Cholecalciferol. In: Hovda LR, Brutlag AG, Poppenga RH, Epstein SE, eds. *Blackwell's Five-Minute Veterinary Consult Clinical Companion: Small Animal Toxicology*. 3rd ed. Wiley-Blackwell; 2024:801-806.
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