



**Emergency treatment  
of cholecalciferol  
rodenticide poisoning –  
advice for veterinarians**

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## Key points

- Cholecalciferol is a vitamin D compound. It is not an anticoagulant rodenticide.
- The main risks from cholecalciferol poisoning are hypercalcaemia and hyperphosphataemia which can cause gastrointestinal, renal, cardiac and neurological signs.
- Tissue calcification is irreversible.
- Treatment is aimed at controlling the calcium concentration with IV fluids, furosemide, steroids, antiemetics, gastroprotectants, a phosphate binder and a bisphosphonate.

## Product description

BASF produces a cholecalciferol rodenticide, under the global brand Selontra. Selontra® is a rodenticide bait containing cholecalciferol (vitamin D<sub>3</sub>) at 0.075% (0.75 mg/g, 750 ppm). It is available as a grey to green, semi-solid soft paste block with a faint sweetish odour. Each 20 g bait block is square and shrink-wrapped in a non-absorbent, transparent, odour-permeable polyolefin film. Each bait block contains 15 mg of cholecalciferol.

### Amount of cholecalciferol in one 20 g block of Selontra®:

Grams	Milligrams	Micrograms	International Units
0.015 g	15 mg	15000 µg	600,000 IU

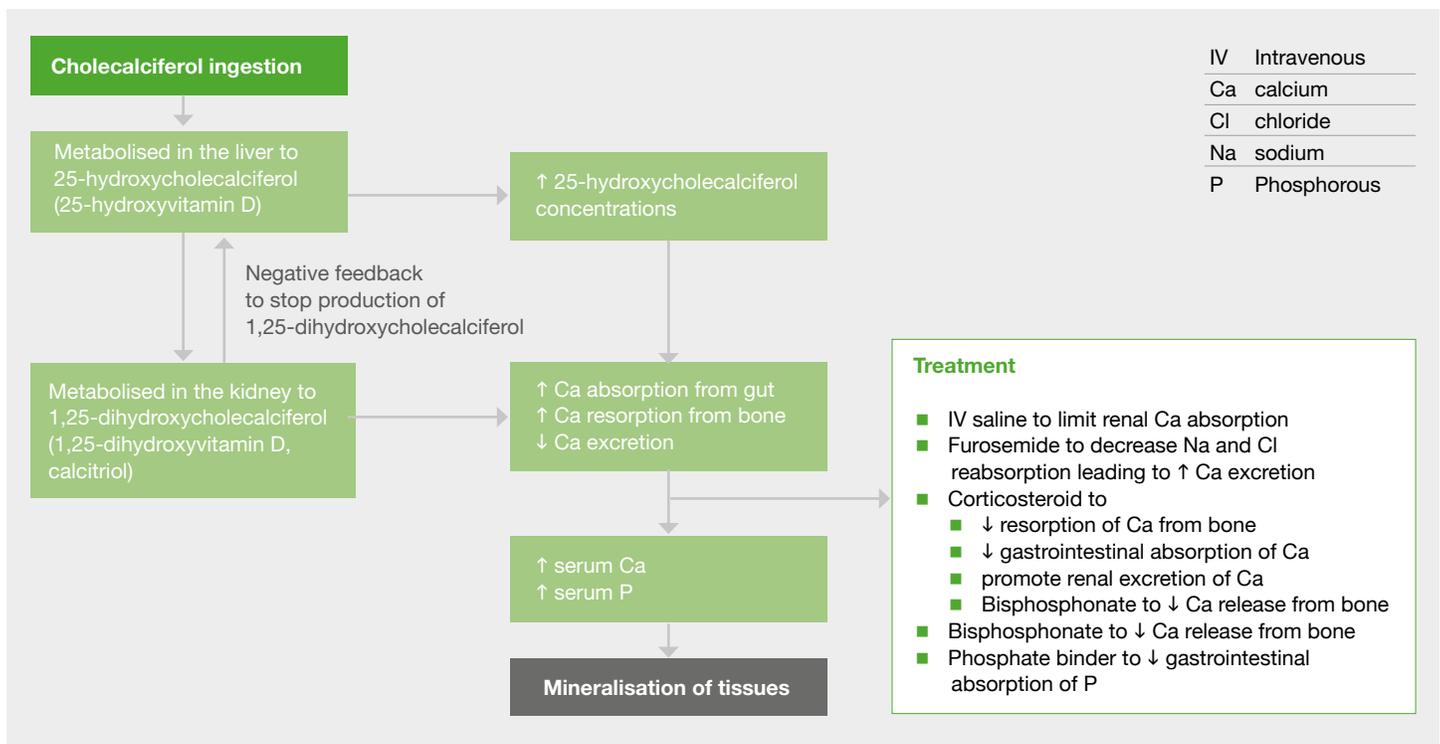
## Pharmacokinetics and mechanism of toxicity

Absorption of cholecalciferol is rapid and complete in the small intestine. It is metabolised in the liver to 25-hydroxycholecalciferol (25-hydroxyvitamin D) which is the principal circulating metabolite. It has limited activity, but at high concentrations can exert metabolic effects. This metabolite is converted by renal 1 $\alpha$ -hydroxylase to the physiologically active 1,25-dihydroxycholecalciferol (1,25-dihydroxyvitamin D, calcitriol). This activation is normally dependent on and regulated by plasma concentrations of parathyroid hormone, calcium, phosphorous and calcitriol. When 1,25-dihydroxycholecalciferol concentrations reach a certain level, further activation of 25-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol is suppressed by negative feedback. However, there is no similar control over production of 25-hydroxycholecalciferol; therefore, concentrations of this compound rise to a level where it will begin to exert similar effects to the active intermediate. 1,25-dihydroxycholecalciferol binds to vitamin D receptors with an affinity 500 times greater than that of 25-hydroxycholecalciferol and 1000 times greater than that of cholecalciferol (Gupta, 2007). The major metabolites of these pathways are excreted primarily via bile and in the faeces. Less than one third is excreted in the urine. In dogs given a single dose of cholecalciferol (8 mg/kg) the half-life of 25-hydroxyvitamin D was 29 days (Rumbeiha *et al.*, 1999).

In overdose, 25-hydroxycholecalciferol and to a lesser extent 1,25-dihydroxycholecalciferol, result in increased resorption of calcium from bone, increased absorption of calcium from the gut, intestinal calcium transport and proximal renal tubule reabsorption of calcium in the kidney (Figure 1). This gives rise to hypercalcaemia, hyperphosphataemia and toxic effects.



**Figure 1. An outline of the toxic mechanism of cholecalciferol in overdose.**



## Toxic dose

There is interindividual variability in susceptibility to cholecalciferol toxicosis.

- The oral LD<sub>50</sub> of cholecalciferol in dogs is reported as 10-80 mg/kg (EU, 2018), 80 mg/kg (Gunther *et al.*, 1988) and 88 mg/kg (Marshall, 1984). This is equivalent to 13.3-117.3 g of Selontra® bait per kilogram body weight.
- Signs of poisoning can occur after ingestion of 0.5 mg (20,000 IU) of cholecalciferol per kilogram body weight (Peterson and Fluegeman, 2013; Rumbelha, 2013). This is equivalent to 0.67 g of Selontra® bait per kilogram body weight.
- Fatalities in dogs are reported after 2-13 mg/kg (Gunther *et al.*, 1988; Dorman, 1990; Talcott *et al.*, 1991; Rumbelha *et al.*, 1999; Hare *et al.*, 2000). This is equivalent to 2.7-17.3 g of Selontra® bait per kilogram body weight.
- In an experimental study, 8 of 10 cats given cholecalciferol 0.375 mg (15,000 IU)/kg/day died on days 3-31. Death occurred 1-2 days after the onset of clinical signs. The other two cats remained well (Morita *et al.*, 1995). This dose is equivalent to 0.5 g of Selontra® bait per kilogram body weight.
- Severe cholecalciferol poisoning has been reported in cats but in most cases the dose ingested was unknown (Moore *et al.*, 1988; Peterson *et al.*, 1991). Acute ingestion of 50 g of another 0.075% bait in a 6 month old domestic shorthaired cat was fatal, although he presented 4 days after ingestion (Thomas *et al.*, 1990).



## Secondary poisoning

The risk of secondary exposure to cholecalciferol from ingestion of animals that have died from poisoning is low.

- Twelve cats, fed whole carcasses of cholecalciferol-poisoned possums (from 0.8% bait) as their only food for 5 consecutive days, remained well with serum calcium concentration within or close to the normal range (Eason *et al.*, 1996; Eason *et al.*, 2000).
- Dogs fed rats that had died after ingestion of cholecalciferol bait (0.075%) for 14 days developed no adverse effects (Marshall, 1984).
- Dogs fed cholecalciferol-poisoned possums (from 0.8% bait) daily for 5 days had increased serum calcium concentrations within a week of starting to feed, and the concentrations remained above the normal range for a further 1-2 weeks. Serum urea concentrations also exceeded normal values by day 4, indicating toxin-induced renal damage or dehydration secondary to anorexia. Serum urea returned to normal by day 28 after dosing. No toxin-related changes were observed in the haematology in any treatment group (Eason *et al.*, 2000).
- Dogs fed possums euthanised 48 hours after ingestion of cholecalciferol bait (0.8%), thus containing high concentrations of 25-hydroxycholecalciferol and residual bait in the gut, exhibited anorexia and varying degrees of lethargy from day 4 to day 14 after dosing. By day 7, mean body weights had decreased by 5% compared to pre-treatment values. All affected dogs began to recover about 14 days after the onset of exposure. Histopathological examination revealed mineralisation of the kidneys, consistent with cholecalciferol toxicosis, in all dogs euthanised on day 14 that had been hypercalcaemic (Eason *et al.*, 2000).

## Clinical effects

Calcium concentrations given are total calcium. Refer to Table 1 for reference values and Table 2 for conversions.

**Table 1 Reference values for calcium and hypercalcaemia in cats and dogs**

	Dog	Cat
Normal total calcium	2.2-2.8 mmol/L (9-11.5 mg/dL)	2.0-2.6 mmol/L (8-10.5 mg/dL)
Normal ionized calcium	1.2-1.5 mmol/L (4.8-6.0 mg/dL)	1.1-1.4 mmol/L (4.4-5.6 mg/dL)
Hypercalcaemia (total calcium)	>3 mmol/L (>12 mg/dL)	>2.7 mmol/L (>11 mg/dL)
Hypercalcaemia (ionised calcium)	>1.45 mmol/L (>5.7 mg/dL)	>1.4 mmol/L (>5.6 mg/dL)

**Table 2 Conversions for vitamin D, calcium and phosphorous**

<b>Vitamin D</b>	1 mg = 41000 IU approximately 1 µg = 41 IU approximately 1 IU = 0.025 µg approximately
<b>Calcium</b>	1 mmol is equivalent to 40.46 mg 1 mg is equivalent to 0.025 mmol or 25 µmol To convert mmol/L to mg/dL divide by 0.2495 To convert mg/dL to mmol/L multiply by 0.2495
<b>Phosphorous</b>	1 mmol is equivalent to 30.95 mg 1 mg is equivalent to 0.032 mmol or 32 µmol To convert mmol/L to mg/dL divide by 0.323 To convert mg/dL to mmol/L multiply by 0.323

The onset of effects after ingestion of cholecalciferol is variable and signs usually occur within 12-36 hours.

Initial signs can be non-specific with polydipsia (sometimes in the absence of any other signs), anorexia, depression, weakness, lethargy, recumbency, polyuria, polydipsia, profuse vomiting and diarrhoea. Signs become more pronounced as the calcium concentration rises above 3 mmol/L (12 mg/dL) which usually occurs from 24-36 hours. There may be severe vomiting, anorexia, constipation, ataxia, dyspnoea, tachypnoea, muscle spasms, twitching and convulsions.

In severe cases hypercalcaemia, hyperphosphataemia, and deposition of calcium in tissues may cause initial tachycardia, then bradycardia and renal insufficiency or renal failure. Pain may occur from calcification of tissues. Haematemesis and haemorrhagic diarrhoea may occur as a result of calcification in the gastrointestinal tract; pulmonary haemorrhage has also been reported. Metabolic acidosis, leucocytosis and thrombocytopenia are occasionally reported. Cardiac abnormalities may be seen on the ECG (commonly shortened QT segment, prolonged PR interval and ventricular arrhythmias). Hypercalcaemic cardiac failure may occur if the serum calcium concentration exceeds 3.5 mmol/L (14 mg/dL). Death can occur from acute renal failure or calcification of tissues of the gastrointestinal tract, kidneys or heart. Pulmonary calcification has also been reported (Wehner *et al.*, 2013).

Animals that do not die in the acute phase of cholecalciferol poisoning may develop chronic renal failure due to calcification of the kidneys.



## Diagnosis

- A serum total calcium concentration of more than 3 mmol/L (12 mg/dL) is characteristic of cholecalciferol toxicosis.
- Anorexia is an early sign of a rising calcium blood concentration.
- Calciuria is also a common early sign but is often missed as it is not routinely measured.
- From 12-72 hours the phosphorous concentration (>1.7 mmol/L; >5.2 mg/dL) increases and can precede hypercalcaemia.
- Urea and creatinine concentrations are elevated and urine specific gravity may range from 1.002-1.006.
- As the condition advances X-rays may show mineralisation of the stomach, upper gastrointestinal tract and other tissues.).

Differential diagnoses for hypercalcaemia are given in Box 1.

## Post-mortem findings

In animals that have died or been euthanised after cholecalciferol toxicosis there will usually be evidence of severe dehydration, calcification of soft tissues and blood vessels, petechial haemorrhages, pale streaks in the kidney, pitted mottled kidneys, diffuse haemorrhage of the gastrointestinal tract, roughened raised plaques on the intima of the great vessels and on the surface of the lung and abdominal viscera. The organs commonly affected by calcification are the kidneys (Morita *et al.*, 1995; Rumbeiha *et al.*, 1999), stomach (Rumbeiha *et al.*, 1999), heart (Gunter *et al.*, 1988; Thomas *et al.*, 1990; Morita *et al.*, 1995) and lungs (Thomas *et al.*, 1990; Peterson *et al.*, 1991; Talcott and Kowitz, 1991; Morita *et al.*, 1995; Rumbeiha *et al.*, 1999).

Microscopic lesions include mineralisation of kidney tubules, coronary arteries, gastric mucosa, parietal pleura, pulmonary bronchioles, pancreas and urinary bladder. The renal tubules may be degenerative or necrotic and the myocardium may also show evidence of necrosis.

## Treatment

- The aim of therapy is to ensure adequate hydration and urine output, controlling serum calcium concentrations and reducing phosphorous concentrations.
- The aim is to keep the total calcium concentration < 3 mmol/L (12.5 mg/dL), ionised calcium <1.3 mmol/L (5.4 mg/dL) and the phosphorous <2.3 mmol/L (7 mg/dL) (Dee and Hovda, 2012).
- Drug therapy includes IV saline with furosemide, steroids, an antiemetic, gastroprotectants, a phosphate binder and a bisphosphonate.

### Gut decontamination

- If the animal presents within 2 hours of ingestion vomiting can be induced followed by activated charcoal (1 g/kg every 4 hours) (Peterson and Fluegeman, 2013).
- Oral colestyramine (0.3-1 g/kg) 3 times daily for 4 days may be given instead (DeClementi and Sobczak, 2012; Peterson and Fluegeman, 2013).

### Box 1. Differential diagnoses for hypercalcaemia

- Primary hyperparathyroidism
- Pseudohyperparathyroidism resulting from lymphosarcoma, multiple myeloma and various other cancers
- Hypoadrenocorticism
- Haemoconcentration
- Osteomyelitis
- Renal disease
- Poisoning from grapes, raisins, sultanas or currants
- Idiopathic hypercalcaemia of cats
- Juvenile hypercalcaemia



## Monitoring

- The serum total calcium concentration should be monitored every 12 hours initially and then once daily for 4 days or more (depending on clinical signs). The calcium concentration should be monitored for 4 days even in asymptomatic cases. If the calcium concentration remains normal and the animal has no signs after 4 days then nothing further is required (DeClementi and Sobczak, 2012).
- Other electrolytes (particularly phosphorous, potassium and magnesium) should also be monitored as long-term fluid administration can cause hypokalaemia and hypomagnesaemia.
- It is essential to monitor renal function, urine specific gravity, fluid balance and urine output. The body weight should be measured several times daily (Peterson and Fluegeman, 2013).
- Blood gases should be monitored and in symptomatic animals the ECG should be checked.

## General care

- It is important to ensure adequate hydration and good urine output in animals with cholecalciferol toxicosis.
- Antiemetics should be given for severe or persistent vomiting.
- Gut protectants should be given to help prevent damage to the gastric mucosa (Table 3).
- Phosphate binders such as aluminium hydroxide can be used in animals with hyperphosphataemia.
- Diazepam may be given for seizures, if required.
- Analgesia may be required.

## Management of hypercalcaemia

If the total calcium concentration is more than 3 mmol/L (>12 mg/dL) or a toxic dose has been ingested then calciuresis should be attempted.

- IV fluids prevent volume depletion and limit renal absorption of calcium.
- Saline is used rather than other fluids, as it contains no calcium, and sodium ions enhance calcium excretion by reducing tubular reabsorption of calcium and enhancing calciuresis (DeClementi and Sobczak, 2012). Calcium-containing fluids (e.g., lactated Ringer's) should be avoided. Saline (0.9%) should be started at 2-3 times the maintenance rate and once hydration status is corrected furosemide should be added (Table 3).
- Furosemide decreases sodium and chloride reabsorption in the ascending loop of Henle leading to increased calcium excretion. Thiazide diuretics should not be used as they lower renal calcium excretion. Furosemide may be needed over a period of many days and it may be preferable to switch to oral dosing later. Care must be taken to ensure hypokalaemia does not result from prolonged use of furosemide. This is a potential result of co-administration with corticosteroids (see below), and potassium supplementation may be required.
- Sodium bicarbonate may be needed to correct metabolic acidosis or to induce a mild alkalosis which would enhance conversion of active ionised calcium to its inactive non-ionised form.
- Aggressive management of hypercalcaemia can sometimes result in hypocalcaemia and this should be corrected with oral calcium salts, if mild, or parenteral calcium gluconate, if severe.

## Corticosteroids

Corticosteroids are recommended in cholecalciferol toxicosis as they decrease resorption of calcium from bone, decrease gastrointestinal absorption and promote renal excretion of calcium. The dosage should be tapered off if used for more than 2 weeks to prevent adrenocortical insufficiency.



**Table 3. Dosages of drugs used in the management of cholecalciferol toxicosis in dogs and cats**

Drug	Dosage	Notes
<b>Calciuresis</b>		
Saline 0.9%	IV, start at 2-3 times maintenance dose.	<ul style="list-style-type: none"> <li>Once hydration status is corrected add furosemide</li> </ul>
Furosemide	Initial bolus dose of 0.66 mg/kg IV bolus, followed by 0.66 mg/kg/hour CRI.	<ul style="list-style-type: none"> <li>Furosemide may be needed over a period of many days and later on it may be preferable to use oral dosing (1-5 mg/kg orally, 1-3 times daily).</li> <li>Furosemide may be needed for 1-2 weeks after saline diuresis has been discontinued and then dose tapered off.</li> </ul>
<b>Corticosteroid</b>		
Prednisolone	1-3 mg/kg orally every 12 hours for 2-4 weeks	<ul style="list-style-type: none"> <li>Monitor for hypokalaemia</li> </ul>
<b>Antiemetics</b>		
Maropitant	<i>Dogs:</i> 1 mg/kg SC every 24 hours or 2 mg/kg orally every 24 hours. <i>Cats:</i> 1 mg/kg SC or orally every 24 hours	
Metoclopramide	0.25-0.5 mg/kg IV, IM, SC or orally every 12 hours, or 0.17-0.33 mg/kg IV, IM, SC or orally every 8 hours.	
<b>Gastroprotectants</b>		
Famotidine	0.5-1.0 mg/kg orally every 12-24 hours	<ul style="list-style-type: none"> <li>Where adsorbents have been administered a period of 2 hours should elapse before famotidine is administered to minimise charcoal binding to drug.</li> <li>If sucralfate is to be used then a period of 2 hours should elapse before famotidine is administered.</li> </ul>
Cimetidine	<i>Dogs:</i> 5-10 mg/kg orally, IV, IM every 8 hours <i>Cats:</i> 2.5-5 orally, IV, IM every 12 hours	<ul style="list-style-type: none"> <li>If adsorbents have been administered then parenteral routes should be employed in the initial stages.</li> <li>If IV route is to be used then administer by slow IV injection over 30 minutes to reduce risk of hypotension and arrhythmias.</li> <li>If metoclopramide has been used or sucralfate is to be used then a period of two hours should elapse before cimetidine is administered.</li> </ul>
Ranitidine	<i>Dogs:</i> 2 mg/kg slow IV, SC, orally every 8-12 hours <i>Cats:</i> 2 mg/kg CRI, 2.5 mg/kg slow IV every 12 hours, 3.5 mg/kg orally every 12 hours	<ul style="list-style-type: none"> <li>If adsorbents have been administered then parenteral routes should be employed in the initial stages.</li> <li>If metoclopramide has been used or sucralfate is to be used then a period of two hours should elapse before ranitidine is administered.</li> </ul>
Omeprazole	<i>Dogs:</i> 0.5-1.5 mg/kg IV or orally every 24 hours <i>Cats:</i> 0.75-1 mg/kg orally every 24 hours	
<b>Management of hypercalcaemia</b>		
Give <b>one</b> of the following. A bisphosphonate is preferred (DeClementi and Sobczak, 2012), usually pamidronate.		
<b>Pamidronate</b> (most commonly used)	<i>Dogs:</i> 1.3-2 mg/kg, diluted in 0.9% saline and given over 2-4 hours IV. <i>Cats:</i> 1-2 mg/kg IV over 4 hours	<ul style="list-style-type: none"> <li><b>Not to be given with calcitonin.</b></li> <li>Should reduce the calcium concentration within 24-48 hours.</li> <li>Dogs with refractory hypercalcaemia may need a single re-treatment 5-7 days later.</li> <li>High doses of pamidronate (&gt;10 mg/kg) can cause nephrotoxicity.</li> </ul>
<b>Clodronate</b>	<i>Dogs:</i> 5-14 mg/kg by slow IV over at least 2 hours. It can be diluted in 0.9% saline (Ulutas <i>et al.</i> , 2006) <b>OR</b> 10-30 mg/kg orally every 8-12 hours. <i>Cats:</i> No information available.	<ul style="list-style-type: none"> <li><b>Not to be given with calcitonin.</b></li> <li>Clodronate should be given at least one hour after food and at least 2 hours either before or after cimetidine administration.</li> </ul>
<b>Calcitonin</b>	4-7 IU/kg SC every 6-8 hours.	<ul style="list-style-type: none"> <li><b>Not to be given with bisphosphonate.</b></li> <li>Should be continued until serum calcium stabilises at &lt;3 mmol/L (&lt;12 mg/dL) (could be 3-4 weeks, because calcitonin has a short half-life in dogs).</li> <li>Animals may become refractory to treatment.</li> <li>Anorexia and vomiting are common side effects.</li> <li>Dogs tend to regain their appetite about 18 hours after calcitonin administration is stopped (Hare <i>et al.</i>, 2000).</li> </ul>
<b>Other drugs</b>		
<b>Diazepam</b>	0.5-1.0 mg/kg IV repeated every 10 minutes up to 3 times if needed.	

CRI continuous rate infusion, IM intramuscular, IV intravenous, SC subcutaneous



### **Bisphosphonates or calcitonin**

Bisphosphonates (particularly pamidronate but sometimes clodronate) or calcitonin can be used in the management of cholecalciferol-induced hypercalcaemia. Bisphosphonates inhibit osteoclast action and suppress calcium release from bone and they have been used effectively in experimental (Rumbeiha *et al.*, 1999; Ulutas *et al.*, 2006) and clinical cases (Pesillo *et al.*, 2002; Hostutler *et al.*, 2005) of cholecalciferol toxicosis in dogs. These drugs are relatively expensive but rapidly lower the calcium concentration and may allow daily outpatient monitoring (Morrow and Volmer, 2002).

If a bisphosphonate is not available, calcitonin, a synthetic salmon calcitonin compound that inhibits bone and intestinal resorption of calcium, may be given. A bisphosphonate is preferred (DeClementi and Sobczak, 2012), as calcitonin is less effective and has a very short-life necessitating repeated dosing. Note that a bisphosphonate or calcitonin should be used, not both. Use of bisphosphonates and calcitonin together, either together or sequentially, is controversial as experimental studies in animals receiving both did not do as well as animals receiving one or the other. In humans, however, short-term use of calcitonin concomitantly with a bisphosphonate therapy is the preferred treatment for emergency hypercalcaemia of malignancy (Morrow, 2001).

Saline diuresis and other therapies (furosemide, phosphate binders, steroids) should be continued while on bisphosphonate or calcitonin therapy.

### **Ongoing care**

- IV fluids should be continued until the calcium concentration normalises (Peterson and Fluegeman, 2013).
- Furosemide and prednisolone (or other corticosteroids) should then be continued for a further 2-4 weeks with a gradual tapering off of the dose, however these drugs should be stopped if there is risk of hypocalcaemia.
- Once the calcium concentration has stabilised the animal can be weaned off fluids but it is still necessary to monitor calcium and phosphorous concentrations and renal function. The calcium concentration and renal function should be assessed at 24, 48, 72 hours and then 2-3 times weekly for 2 weeks and finally once a week for 2 weeks after cessation of treatment. If urea and creatinine concentrations are elevated, then conventional treatment for renal failure is recommended. If the calcium starts to rise saline diuresis should be re-started and another dose of pamidronate (if used) given.
- A low phosphorous, low calcium diet should be given for 4 weeks initially in animals with cholecalciferol toxicosis. Calcification of soft tissues is irreversible, but it is advisable to restrict dietary calcium and multivitamin/mineral supplement intake.

## **Prognosis**

The outcome in animals with cholecalciferol poisoning depends on the severity and length of hypercalcaemia. The prognosis of animals with acute cholecalciferol toxicosis is generally good if treatment is started early, calcium concentrations are controlled and tissue calcification is prevented. Prognosis is guarded if signs are severe or advanced, particularly in animals with calcification of the gastrointestinal tract or cardiac tissue. Rapid reduction in calcium concentrations may not necessarily lead to a favourable outcome if tissue mineralisation has already occurred (Fan *et al.*, 1998). Haematemesis is considered a poor sign since it indicates severe gastrointestinal ulceration (Rumbeiha, 2013). Severely poisoned animals usually die within 2-5 days of onset of clinical signs.

In adult animals where the product of calcium [Ca] and phosphorous [P] concentrations (as measured in mg/dL) exceeds 60, tissue mineralisation is likely to occur (i.e. if [Ca] x [P] is >60 where units of both are in mg/dL). Note a [Ca] x [P] of more than 70 may be normal in puppies and kittens. Puppies are more sensitive to cholecalciferol toxicosis and this will be exacerbated as cholecalciferol is concentrated in their mother's milk (it is very lipophilic) (Peterson and Fluegeman, 2013). Dogs with pre-existing kidney disease are more sensitive to cholecalciferol toxicosis (Rumbeiha, 2013).





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This paper was written for BASF by VPIS

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