Rodenticides

A wide variety of materials are used as rodenticides. They pose particular risks for accidental poisonings for several reasons. First, as agents specifically designed to kill mammals, often their toxicity is very similar for the target rodents and for humans. (Warfarin and other anticoagulant rodenticides were initially developed to overcome this problem by creating compounds that were highly toxic to rodents, particularly after repeated exposures, but much less toxic to humans.) Second, since rodents usually share environments with humans and other mammals, the risk of accidental exposure is an integral part of the placement of baits for the rodents. Finally, as rodents have developed resistance to existing rodenticides, there is a continuous need to develop new and potentially more toxic rodenticides. As rodents have become resistant to warfarin baits, for example, the development of "superwarfarins" has increased the risk to humans.[1,2] It is important to be familiar with use patterns and development of more toxic compounds and to make every effort to identify the actual agent used in order to institute the most appropriate management for these poisonings.

COUMARINS AND INDANDIONES

Toxicology

Warfarin and related compounds (coumarins and indandiones) are the most commonly ingested rodenticides in the United States, with 13,345 exposures reported in 1996.[3] Gastrointestinal absorption of these toxicants is efficient. Warfarin can be absorbed across the skin, but this has occurred only under extraordinary circumstances.

Coumarins and indandiones depress the hepatic synthesis of vitamin K dependent blood-clotting factors (II (prothrombin), VII, IX, and X). The anti-prothrombin effect is best known, and is the basis for detection and assessment of clinical poisoning. The agents also increase permeability of capillaries throughout the body, predisposing the animal to widespread internal hemorrhage. This generally occurs in the rodent after several days of warfarin ingestion due to the long half-lives of the vitamin K dependent clotting factors,[1,2] although lethal hemorrhage may follow smaller doses of the modern, more toxic compounds.[1]

The lengthened prothrombin time (PT) from a toxic dose of coumarins or indandiones may be evident within 24 hours, but usually reaches a maximum

in 36-72 hours.[1,4,5] Lengthened PT occurs in response to doses much lower than that necessary to cause hemorrhage. There is concern that the more toxic modern compounds, such as brodifacoum and difenacoum, may cause serious poisoning of nontarget mammals, including humans, at much lower dosage. Brodifacoum, one of the superwarfarins, is much more toxic, with a dose as low as 1 mg in an adult or 0.014 mg/kg in a child sufficient to produce toxicity.[1]

Symptomatic poisoning, with prolonged symptoms due to the long half-lives of superwarfarins, has been reported even with single exposures; however, these are usually intentional and are large single dosages.[2] Because of their toxicity in relation to warfarin, patients may require higher dosages of vitamin K and will require longer monitoring of their PT. One patient required vitamin K for several months following discharge.[6] Another patient was released from the hospital with significant clinical improvement and only slightly elevated coagulation studies after brodifacoum ingestion. Two and a half weeks later, he presented in a comatose state and was found to have massive intracranial hemorrhage.[7]

Clinical effects of these agents usually begin several days after ingestion, due to the long half-life of the factors. Primary manifestations include nosebleeds, bleeding gums, hematuria, melena, and extensive ecchymoses.[1,2,6,7,8] Patients may also have symptoms of anemia, including fatigue and dyspnea on exertion.[8] If the poisoning is severe, the patient may progress to shock and death.

Unlike the coumarin compounds, some indandiones cause symptoms and signs of neurologic and cardiopulmonary injury in laboratory rats leading to death before hemorrhage occurs. These actions may account for the greater toxicity of indandiones in rodents. Neither neurologic nor cardiopulmonary manifestations have been reported in human poisonings.

Confirmation of Poisoning

Coumarin or indandione poisoning results in an increase in prothrombin time, the result of reduced plasma prothrombin concentration. This is a reliable test for absorption of physiologically significant doses. Detectable reduction in prothrombin occurs within 24-48 hours of ingestion and persists for 1-3 weeks.[1,4,5] The manufacturers can often measure blood levels of the more toxic coumarins.[8]

Treatment

1. Determine quantity ingested. If it is certain that the patient ingested no more than a mouthful or two of warfarin- or indandione-treated bait, or a single swallow or less of bait treated with the more toxic brodifacoum or bromadiolone compounds, medical treatment is probably unnecessary.

2. Vitamin K$_1$. A patient presenting within 24 hours after ingestion will likely have a normal PT. However, in a study of 110 children who were poisoned by superwarfarins, primarily brodifacoum, a child's PT was significantly more likely to be prolonged at 48 hours after having a normal PT at 24 hours.[5] Therefore, for suicidal ingestions with large amounts taken, if there is uncertainty about the amount of bait ingested or the general health of the patient, phytonadione (vitamin K$_1$) given orally protects against the anticoagulant effect of these rodenticides, with essentially no risk to the patient. In accidental ingestions with healthy children involving only a taste or single swallow, no medical treatment is required, but children should be observed for bleeding and bruising. If a larger amount may have been ingested, PT should be monitored at 24 and 48 hours, with phytonadione therapy initiated for elevated PT or clinical signs of bleeding.

Caution: Phytonadione, specifically, is required. Neither vitamin K$_3$ (menadione, Hykinone[R]) nor vitamin K$_4$ (menadiol) is an antidote for these anticoagulants.

Dosage of Phytonadione (oral):

- *Adults and children over 12 years:* 15-25 mg.

- *Children under 12 years:* 5-10 mg.

Alternatively, a colloidal preparation of phytonadione, Aquamephyton[R], may be given intramuscularly. For adults and children over 12 years, give 5-10 mg; for children under 12, give 1-5 mg.

Ensure that patients (especially children) are carefully observed for at least 4-5 days after ingestion. The indandiones and some of the more recently introduced coumarins may have other toxic effects.

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3. Gastrointestinal decontamination. If large amounts of anticoagulant have been ingested within several hours prior to treatment, consider gastric decontamination procedures as outlined Chapter 2.

4. Determine prothrombin time. If anticoagulant has been ingested any time in the preceding 15 days, determination of the PT provides a basis for judging the severity of poisoning. Patients who ingest large amounts, particularly of the superwarfarin compounds, will likely have a very prolonged period of decreased prothrombin activity. Patients may need to be treated for as long as 3 or 4 months.[6,7]

If the prothrombin time is significantly lengthened, give Aquamephyton[R] intramuscularly. See next page for dosage.

> **Dosage of Aquamephyton^R (intramuscular):**
>
> - *Adults and children over 12 years:* 5-10 mg.
>
> - *Children under 12 years:* 1-5 mg.
>
> Decide dose within these ranges according to the degree of prothrombin time lengthening and, in children, the age and weight of the child. Substantially higher doses of phytonadione (50 to 125 mg) have been required in some poisonings with brodifacoum when bleeding and PT elevation persisted despite therapy.[6,7,9]
>
> Repeat prothrombin time in 24 hours. If it has not decreased from the original value, repeat Aquamephyton^R dosage.

5. Bleeding. If victim is bleeding as a result of anticoagulant poisoning, administer Aquamephyton^R intravenously: up to 10 mg in adults and children over 12 years, and up to 5 mg in children under 12 years. Initial dosage should be decided chiefly on the basis of the severity of bleeding. Subsequent dosages may need to be adjusted based on response, especially in the case of the superwarfarins.[6,7,9] Repeat intravenous Aquamephyton^R in 24 hours if bleeding continues. Inject at rates not exceeding 5% of the total dose per minute. Intravenous infusion of the Aquamephyton^R diluted in saline or glucose solution is recommended. Bleeding is usually controlled in 3-6 hours.

Caution: Adverse reactions, some fatal, have occurred from intravenous phytonadione injections, even when recommended dosage limits and injection rates were observed. For this reason, the intravenous route should be used *only* in cases of severe poisoning. Flushing, dizziness, hypotension, dyspnea, and cyanosis have characterized adverse reactions.

Antidotal therapy in cases of severe bleeding should be supplemented with transfusion of fresh blood or plasma. Use of fresh blood or plasma represents the most rapidly effective method of stopping hemorrhage due to these anticoagulants, but the effect may not endure. Therefore, the transfusions should be given along with phytonadione therapy.

Determine PT and hemoglobin concentrations every 6-12 hours to assess effectiveness of antihemorrhagic measures. When normal blood coagulation is restored, it may be advisable to drain large hematomata.

Ferrous sulfate therapy may be appropriate in the recuperative period to rebuild lost erythrocyte mass.

INORGANIC RODENTICIDES

Toxicology

Thallium sulfate is well absorbed from the gut and across the skin. It exhibits a very large volume of distribution (tissue uptake) and is distributed chiefly to the kidney and liver, both of which participate in thallium excretion. Most blood-borne thallium is in the red cells. Elimination half-life from blood in the adult human is about 1.9 days. Most authors report the LD_{50} in humans to be between 10 and 15 mg/kg.[10]

Unlike other inorganic rodenticides like yellow phosphorus and zinc phosphide, thallium poisoning tends to have a more insidious onset with a wide variety of toxic manifestations. Alopecia is a fairly consistent feature of thallium poisoning that is often helpful diagnostically; however, it occurs two weeks or more after poisoning and is not helpful early in the presentation.[10,11] In addition to hair loss, the gastrointestinal system, central nervous system, cardiovascular system, renal system, and skin are prominently affected by toxic intakes.

Early symptoms include abdominal pain, nausea, vomiting, bloody diarrhea, stomatitis, and salivation. Ileus may appear later on. Elevated liver enzymes may occur, indicating tissue damage. Other patients experience signs of central nervous system toxicity including headache, lethargy, muscle weakness, paresthesias, tremor, ptosis, and ataxia. These usually occur several days to more than a week after exposure.[10,12] Extremely painful paraesthesias, either in the presence or absence of gastrointestinal signs, may be the primary presenting complaint.[11,13] Myoclonic movements, convulsions, delirium, and coma reflect more severe neurologic involvement. Fever is a bad prognostic indication of brain damage.

Cardiovascular effects include early hypotension, due at least in part to a toxic myocardiopathy. Ventricular arrythmias may occur. Hypertension occurs later and is probably a result of vasoconstriction. The urine may show protein and red cells. Patients may also develop alveolar edema and hyaline membrane formation in the lungs, consistent with a diagnosis of Acute Respiratory Distress Syndrome.[14] Death from thallium poisoning may be caused by respiratory paralysis or cardiovascular collapse. Absorption of nonlethal doses of thallium has caused protracted painful neuropathies and paresis, optic nerve atrophy, persistent ataxia, dementia, seizures, and coma.[11]

Yellow phosphorus (also known as white phosphorus) is a corrosive agent and damages all tissues it comes in contact with, including skin and the gut lining. Initial symptoms usually reflect mucosal injury and occur a few minutes to 24 hours following ingestion. The first symptoms include severe vomiting and burning pain in the throat, chest, and abdomen. The emesis may be bloody (either red, brown, or black)[15] and on occasion may have a garlic smell.[16,17] In some cases, central nervous system signs such as lethargy, restlessness, and irrita-

bility are the earliest symptoms, followed by symptoms of gastrointestinal injury. Shock and cardiopulmonary arrest leading to death may occur early in severe ingestions.[17]

If the patient survives, a relatively symptom-free period of a few hours or days may occur, although this is not always the case.[15] The third stage of toxicity then ensues with systemic signs indicating severe injury to the liver, myocardium, and brain. This is due to phosphine gas (PH_3) formed in and absorbed from the gut. Nausea and vomiting recur. Hemorrhage occurs at various sites reflecting a depression of clotting factor synthesis in the damaged liver. Also, thrombocytopenia may contribute. Hepatomegaly and jaundice appear. Hypovolemic shock and toxic myocarditis may develop. Brain injury is manifested by convulsions, delirium, and coma. Anuric renal failure commonly develops due to shock and to the toxic effects of phosphorus products and accumulating bilirubin on renal tubules. The mortality rate of phosphorus poisonings may be as high as 50 percent.[15]

Zinc phosphide is much less corrosive to skin and mucous membranes than yellow phosphorus, but inhalation of dust may induce pulmonary edema. The emetic effect of zinc released in the gut may provide a measure of protection; however, phosphine will be produced in the gut and absorbed along with the zinc. Nausea and vomiting, excitement, chills, chest tightness, dyspnea, and cough may progress to pulmonary edema. Patients face many of the same systemic toxicities as encountered with yellow phosphorus, including hepatic failure with jaundice and hemorrhage, delirium, convulsions, and coma (from toxic encephalopathy), tetany from hypocalcemia, and anuria from renal tubular damage. Ventricular arrythmias from cardiomyopathy and shock also occur and are another common cause of death.[16,18] Inhalation of phosphine gas from improper use of phosphide rodenticides has resulted in pulmonary edema, myocardial injury, and multisystem involvement.[19] For more information about the effects of phosphine gas poisoning, see the section on phosphine in Chapter 16, Fumigants.

Confirmation of Poisoning

Phosphorus and phosphides sometimes impart a foul rotten fish odor to vomitus, feces, and sometimes the breath. Luminescence of vomitus or feces is an occasional feature of phosphorus ingestion. Hyperphosphatemia and hypocalcemia occur in some cases, but are not consistent findings.

Thallium can be measured in the serum, urine, and hair. Hair analysis is likely to be useful only in establishing protracted prior absorption. Serum concentration does not exceed 30 mcg per liter in non-exposed persons. The most reliable method for diagnosis is considered a 24-hour urine excretion. The normal value is less than 10 mcg/liter per 24 hours.[10,13]

Treatment: Thallium Sulfate

1. Gastrointestinal decontamination. If thallium sulfate was swallowed less than an hour prior to treatment, consider gastrointestinal decontamination as outlined in Chapter 2. Multiple doses of activated charcoal may be helpful in increasing thallium elimination.[13]

2. Electrolyte and glucose solutions should be given by intravenous infusion to support urinary excretion of thallium by diuresis. Monitor fluid balance carefully to insure that fluid overload does not occur. If shock develops, give whole blood, plasma, or plasma expanders. Pressor amines must be used very carefully in light of myocardial injury. Monitor ECG for arrhythmias.

3. Convulsions. Control seizures and myoclonic jerking as outlined in Chapter 2.

4. Combined hemodialysis and hemoperfusion has proven moderately effective in reducing the body burden of thallium in victims of severe poisoning. In one case, peritoneal dialysis was not effective.

5. Chelation therapy. Several methods for chelating and/or accelerating disposition of thallium have been tested and found either relatively ineffective or hazardous. Chelating agents are not recommended in thallium poisoning. Potassium chloride has been recommended. However it has been reported to increase toxicity to the brain,[11,14] and has not shown to increase elimination in some cases.[20]

6. Potassium ferric ferrocyanide (Prussian Blue) orally enhances fecal excretion of thallium by exchange of potassium for thallium in the gut. It is not available or approved for use in humans in the United States. Reports of its use in humans are anecdotal and do not strongly support its use.

Treatment: Yellow Phosphorus and Zinc Phosphide

1. Skin decontamination. Brush or scrape non-adherent phosphorus from the skin. Wash skin burns with copious amounts of water. Make sure all particles of phosphorus have been removed. If burned area is infected, cover with an antimicrobial creme. See Chapter 2.

2. Supportive management. Poisonings by ingested yellow phosphorus or zinc phosphide are extremely difficult to manage. Treatment is basically supportive and symptomatic. Control of airway and convulsions must be established prior to considering gastrointestinal decontamination as described in Chapter 2.

Caution: Highly toxic phosphine gas may evolve from emesis, lavage fluid, and feces of victims of these poisons. The patient's room should be well ventilated. Persons attending the patient must wear gloves to avoid contact with the phosphorus.

3. Lavage with 1:5000 potassium permanganate solution has been used in the management of ingested phosphorus compounds in the past; however, there is not sufficient evidence for its efficacy and we do not recommend it.

4. Catharsis is probably not indicated, but there may be some benefit in administering mineral oil. Dosage is 100 mL for adults and children over 12 years, and 1.5 mL/kg body weight in children under 12 years. Do not give vegetable oils or fats.

5. Transfusions. Combat shock and acidosis with transfusions of whole blood and appropriate intravenous fluids. Monitor fluid balance and central venous pressure to avoid fluid overload. Monitor blood electrolytes, glucose, and pH to guide choice of intravenous solutions. Administer 100% oxygen by mask or nasal tube.

6. Oxygen. Combat pulmonary edema with intermittent or continuous positive pressure oxygen.

7. Renal protection. Monitor urine albumin, glucose, and sediment to detect early renal injury. Extracorporeal hemodialysis will be required if acute renal failure occurs, but it does not enhance excretion of phosphorus. Monitor ECG to detect myocardial impairment.

8. Liver damage. Monitor serum alkaline phosphatase, LDH, ALT, AST, prothrombin time, and bilirubin to evaluate liver damage. Administer Aquamephyton[R] (vitamin K_1) if prothrombin level declines.

9. Pain management. Morphine sulphate may be necessary to control pain. Adult dose: 2-15 mg IM/IV/SC Q 2-6 hours prn. Child's dose: 0.1-0.2 mg/kg/dose Q 2-4 hours.

10. Phosphine gas. For specific therapy due to phosphine gas, refer to the treatment of phosphine poisoning in Chapter 16, Fumigants.

CONVULSANTS

Toxicology

Crimidine is a synthetic chlorinated pyrimidine compound that, in adequate dosage, causes violent convulsions similar to those produced by strychnine.

Sodium fluoroacetate and fluoroacetamide are readily absorbed by the gut, but only to a limited extent across skin. The toxic mechanism is distinct from that of fluoride salts. Three molecules of fluoroacetate or fluoroacetamide are combined in the liver to form a molecule of fluorocitrate, which poisons critical enzymes of the tricarboxylic acid (Krebs) cycle, blocking cellular respiration. The heart, brain, and kidneys are the organs most prominently affected. The effect on the heart is to cause arrhythmias, progressing to ventricular fibrillation, which is a common cause of death. Metabolic acidosis, shock, electrolyte imbalance, and respiratory distress are all poor prognostic signs. Neurotoxicity is expressed as violent tonic-clonic convulsions, spasms, and rigor, sometimes not occurring for hours after ingestion.[21]

Strychnine is a natural toxin (nux vomica) which causes violent convulsions by direct excitatory action on the cells of the central nervous system, chiefly the spinal cord. Death is caused by convulsive interference with pulmonary function, by depression of respiratory center activity, or both. Strychnine is detoxified in the liver. Residence half-life is about 10 hours in humans. Onset of symptoms is usually within 15-20 minutes of ingestion. Lethal dose in adults is reported to be between 50 and 100 mg, although as little as 15 mg can kill a child.[22]

Confirmation of Poisoning

There are no generally available tests to confirm poisoning by the convulsant rodenticides.

Treatment: Sodium Fluoroacetate and Fluoroacetamide

Poisonings by these compounds have occurred almost entirely as a result of accidental and suicidal ingestions. If the poison was ingested shortly before treatment and convulsions have not yet occurred, the first step in treatment is to remove the toxicant from the gut. If the victim is already convulsing, however, it is necessary first to control the seizures before gastric lavage and catharsis are undertaken.

1. Control seizures as outlined in Chapter 2. Seizure activity from these compounds may be so severe that doses necessary for seizure control may paralyze respiration. For this reason, it is best to intubate the trachea as early as

possible in the course of seizure control, and support pulmonary ventilation mechanically. This has the added advantage of protecting the airway from aspiration of regurgitated gastric contents.

2. Gastrointestinal decontamination. If the patient is seen within an hour of exposure and is not convulsing, consider gastrointestinal decontamination as outlined in Chapter 2.

3. Administer intravenous fluids cautiously to support excretion of absorbed toxicant. It is especially important to avoid fluid overload in the presence of a weak and irritable myocardium.

4. Monitor electocardiogram for arrhythmias and, if detected, treat with an appropriate antiarrhythmic drug. Facilities for electroshock cardioversion should be at hand. Some victims of fluoroacetate poisoning have been rescued after repeated cardioversions.

5. Calcium gluconate (10% solution) given slowly intravenously should be given to relieve hypocalcemia. Care must be taken to avoid extravasation.

Dosage of Calcium Gluconate:
Supplied as 100 mg/mL (10% solution)

- *Adults and children over 12 years:* 10 mL of 10% solution, given slowly, intravenously. Repeat as necessary.

- *Children under 12 years:* 200-500 mg/kg/24 hr divided Q6 hr. For cardiac arrest, 100 mg/kg/dose. Repeat dosage as needed.

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6. Other therapies. Antidotal efficacy of glycerol monacetate and ethanol, observed in animals, has not been substantiated in humans. These therapies are not recommended in humans.

Treatment: Strychnine or Crimidine

Strychnine and crimidine cause violent convulsions shortly following ingestion of toxic doses. Both poisons are probably well adsorbed onto charcoal. If the patient is seen fully conscious and not convulsing a few moments after the ingestion, great benefit may derive from the immediate ingestion of activated charcoal. If the patient is already obtunded or convulsing, the involuntary motor activity must be controlled before steps are taken to empty the gut and limit toxicant absorption.

1. Control seizures as outlined in Chapter 2.

2. Gastrointestinal decontamination. Consider gastrointestinal decontamination if patient is seen within an hour of ingestion.

3. Administer intravenous fluids to support excretion of absorbed toxicants. Inclusion of sodium bicarbonate in the infusion fluid counteracts metabolic acidosis generated by convulsions. Effectiveness of hemodialysis and hemoperfusion has not been tested.

MISCELLANEOUS RODENTICIDES: RED SQUILL AND CHOLECALCIFEROL

Toxicology

Red squill is a little-used rodenticide, consisting of the inner portions of a small cabbage plant grown in eastern Mediterranean countries. Its toxic properties have been known since ancient times and are probably due to cardiac glycosides. For several reasons, mammals other than rodents are unlikely to be poisoned: (1) red squill is intensely nauseant, so that animals which vomit (rodents do not) are unlikely to retain the poison; (2) the glycoside is not efficiently absorbed from the gut; and (3) absorbed glycoside is rapidly excreted. Injection of the glycosides leads to effects typical of digitalis: alterations in cardiac impulse conduction and arrhythmias.

Cholecalciferol is the activated form of vitamin D (vitamin D_3). Its toxic effect is probably a combination of actions on liver, kidney, and possibly the myocardium, the last two toxicities being the result of hypercalcemia. Early symptoms and signs of vitamin D-induced hypercalcemia in humans are fatigue, weakness, headache, and nausea. Polyuria, polydipsia, proteinuria, and azotemia result from acute renal tubular injury by hypercalcemia. This is commonly the cause of death. Prolonged hypercalcemia results ultimately in nephrolithiasis and nephrocalcinosis. Azotemia occurs as renal tubular damage progresses.

Confirmation of Poisoning

Cholecalciferol intoxication is indicated by an elevated concentration of calcium (chiefly the unbound fraction) in the serum. There are no generally available tests for the other rodenticides or their biotransformation products.

Treatment: Red Squill

Red squill is unlikely to cause poisoning unless ingested at substantial dosage. The problem is usually self-correcting due to its intense emetic effect. If, for some reason, the squill is retained, syrup of ipecac, followed by 1-2 glasses of water, should be administered to initiate vomiting. Monitor cardiac status electrocardiographically.

Treatment: Cholecalciferol

Cholecalciferol at high dosage may cause severe poisoning and death. Human poisonings from its use as a rodenticide have not been reported, but vitamin D overdosage has occurred under clinical circumstances. Treatment is directed at limiting gastrointestinal absorption, accelerating excretion, and counteracting the hypercalcemic effect.

1. Gastrointestinal decontamination. If cholecalciferol has been ingested within an hour prior to treatment, consider gastric decontamination, as outlined in Chapter 2. Repeated administration of charcoal at half or more the initial dosage every 2-4 hours may be beneficial.

2. Administer intravenous fluids (normal saline or 5% glucose) at moderate rates to support excretory mechanisms and excretion. Monitor fluid balance to avoid overload, and measure serum electrolytes periodically. Measure total and ionized calcium levels in the blood 24 hours after cholecalciferol ingestion to determine severity of toxic effect. Monitor urine for protein, and red and white cells to assess renal injury.

3. Furosemide (Lasix), 20-40 mg intravenously, or 40-120 mg daily by mouth may be given to promote diuresis. Dosage for children under 12 is approximately 0.5-1.0 mg/kg body weight intravenously, 1.0-2.0 mg/kg body weight orally. Monitor serum potassium after dosage; give potassium chloride if hypokalemia occurs. Consult package insert for additional directions and warnings.

4. Predinisone and similar glucocorticoids reduce elevated blood calcium levels in certain diseases. Although they have not been tested in cholecalciferol overdosage, it is possible that they would be beneficial. Dosage is approximately 1 mg per kilogram per day, to a maximum of 20 mg per day.

5. Calcitonin (salmon calcitonin, Calcimar[R]) is a logical antidote for cholecalciferol actions, but has only very limited use in human poisoning.[23] In other conditions, the usual dosage is 4 International Units per kg body weight every 12 hours, by intramuscular or subcutaneous injection, continued for 2-5 days.

The dose may be doubled if calcium-lowering effect is not sufficient. Calcium gluconate for intravenous injection should be immediately available if indications of hypocalcemia (carpopedal spasm, cardiac arrhythmias) appear. Consult package insert for additional directions and warnings.

6. Cholestryamine appears effective in the treatment of vitamin D toxicity in animals.[24] It has seen very limited use in humans.[25,26]

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