PEER-REVIEWED

The 10 most common toxicoses in cats

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Cats are sensitive to many toxic agents, sometimes in ways unique to their species. In addition, cats are less likely than dogs to expose themselves through curious ingestions, but cats will nibble on potentially deadly agents, such as lilies. Cats also can jump to high places and obtain materials assumed to be out of reach. And because of their grooming behavior, cats with dermal exposure are likely to receive an oral dose as well.

In this article, we describe 10 common toxicoses in cats. The agents discussed were selected based on the 10 most frequent feline exposures reported to the ASPCA Animal Poison Control Center (APCC) in the past four years.

Canine permethrin insecticides

The topical application of a permethrin spot-on or dip product labeled for use only in dogs can lead to tremors and seizures in cats. These products, which generally contain 45% or 65% permethrin in spot-ons and 3% or more permethrin in dips, are applied to cats accidentally or by individuals who ignore the warnings on the label. In some instances, cats have developed signs of permethrin toxicosis after being in close contact with (sleeping near or grooming) a dog recently treated with a permethrin spot-on product. Initial signs may appear within a few hours but can take 24 to 72 hours to manifest. Full-body tremors are the most common finding, although seizures may also occur.[1] Other pyrethroids, including phenothrin and etofenprox, can cause a similar syndrome in cats when used at high concentrations.[2]

Treatment consists of bathing the cat in a liquid hand dishwashing detergent (*e.g.* Dawn Dishwashing Liquid—Procter & Gamble) to remove the sebum in which the product is distributed. If the cat is symptomatic, delay the bath until the tremors have been controlled. The tremors are best treated with slow intravenous boluses of methocarbamol (Robaxin-V—Fort Dodge Animal Health; total initial dose 55 to 220 mg/kg).[1] Repeat the methocarbamol as needed, but do not exceed a dose of 330 mg/kg/day or respiratory depression may occur.[3] If methocarbamol is not effective, then barbiturates, propofol, or both can be used. Diazepam is generally ineffective for the tremors but should be used if seizures are present.

Additional care should include monitoring the patient's body temperature and administering intravenous fluids to protect the kidneys from myoglobinuria due to muscle breakdown. Atropine is not antidotal for permethrin; no true antidote exists. The prognosis is generally good with aggressive supportive care.

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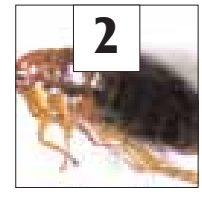


Other topical insecticides

Besides permethrin products, many other flea control products are on the market today. Topical flea control products commonly include insect growth regulators such as s-methoprene and pyriproxyfen, which have low oral and dermal toxic potential in mammals. Insecticide ingredients may include organophosphates or carbamates, pyrethroids, imidacloprid, fipronil, and selamectin, all of which when used appropriately (including low-concentration pyrethroid products) have a low risk of causing serious problems.[4,5]

In general, topical flea control products applied according to label directions will not cause systemic effects in cats.[4,5] Any topically applied product can cause either dermal irritation or a dermal hypersensitivity-like reaction. If dermal signs appear, wash the product off with a mild detergent. If the irritation is localized, the contents of a vitamin E capsule or a corticosteroid cream can be applied. If the irritation is more widespread, corticosteroids or antihistamines may be used systemically.

If a cat licks a topically applied product, a taste reaction—characterized by hypersalivation, agitation, and occasionally vomiting—may develop. These signs are simply a reaction to the bitter taste and can sometimes be quite dramatic. Removing the product from the tongue by giving the cat milk or liquid from a tuna fish can should resolve the signs.

Venlafaxine

Venlafaxine (Effexor, Effexor XR—Wyeth) is a bicyclic antidepressant available in tablets and capsules of 25, 37.5, 50, 75, 100, and 150 mg. Venlafaxine acts as a serotonin and norepinephrine reuptake inhibitor as well as a weak dopamine reuptake inhibitor. Cats seem to readily eat venlafaxine capsules (ASPCA APCC Database: Unpublished data, 2003-2005). Less than one 37.5-mg capsule is enough to cause mydriasis, vomiting,

tachypnea, tachycardia, ataxia, and agitation (ASPCA APCC Database: Unpublished data, 2002-2005). Signs generally begin within one to eight hours after exposure (later if an extended-release formulation was ingested).

Emesis may be initiated in asymptomatic patients. Activated charcoal is effective; repeat the dose in four to six hours if the animal was exposed to an extended-release formulation. Be sure to monitor heart rate and blood pressure. Cyproheptadine (1.1 mg/kg orally or rectally up to three or four times a day) can be used as a serotonin antagonist, and acepromazine or chlorpromazine can be used to treat agitation. Generally, the prognosis is good with close monitoring and treatment of signs.

Glow jewelry and sticks

Glow jewelry and sticks are plastic bracelets, necklaces, and wands that contain a liquid that glows in the dark. The jewelry is popular throughout the summer, especially around the Fourth of July and at Halloween. Cats frequently bite into the jewelry. The main ingredient is dibutyl phthalate, an oily liquid that has a wide margin of safety with an oral LD_{50} in rats of greater than 8 g/kg.[6] So ingesting the contents of a piece of glow jewelry should not cause any serious effects. The chemical has an extremely unpleasant taste, and most cats will not ingest more than a small amount.

Almost immediately after biting into a piece of glow jewelry, cats exhibit signs of a taste reaction, including hypersalivation, agitation, and, occasionally, vomiting. The behavioral changes are likely due to the cat's reacting to the unpleasant taste. A tasty treat such as milk, liquid from a tuna fish can, or other palatable food can ameliorate the taste reaction. Remove any liquid on the fur with a wet washcloth to prevent re-exposure; take the cat into a darkened room to help you identify the product on the coat.[6]

Lilies

Cats ingesting lilies can develop acute renal failure. While many plants are called *lilies*, renal failure has been seen only with *Lilium* species (*e.g.* Easter lilies, Stargazer lilies, tiger lilies, Asiatic lilies, Oriental lilies) and *Hemerocallis* species (day lilies).[7] Ingesting any part of the plant (including the pollen) may cause signs, and even the smallest of exposures should be aggressively treated.

After ingesting lilies, cats generally develop vomiting and depression within two to four hours. Often the cats seem to recover and then begin to deteriorate rapidly about 24 to 72 hours after the exposure with signs of polyuria, polydipsia, and more severe depression.[8] A serum chemistry profile reveals elevated creatinine, blood urea nitrogen (BUN), and phosphorus concentrations; the creatinine concentration is often elevated disproportionately to the BUN concentration.[7] Urinalysis may show cellular casts beginning about 18 hours after exposure.

Treatment consists of immediate decontamination, including emesis and activated charcoal. Start fluid diuresis as soon as possible, and continue it for at least 48 hours. The prognosis is good with prompt, aggressive treatment. Once renal failure develops, some recovery is possible but may take weeks, and the cat may require peritoneal dialysis for support.[7] The development of oliguria or anuria is a poor prognostic sign.[7]

Liquid potpourri

Liquid potpourri is used as household fragrance, often placed in a bowl over a candle or heat source. Cats may lick the product from the container or from their fur if exposed to a spill. Liquid potpourri may contain high concentrations of cationic detergents, essential oils, or a combination of both.[9] Cationic detergents are corrosive to the oral mucosa and can cause severe gastrointestinal upset, drooling, central nervous system (CNS) depression, and hypotension. Cats may exhibit dermal irritation and ulceration as well as severe corneal ulceration if skin or eye exposure occurs. Essential oils may cause gastrointestinal and oral irritation and CNS depression.[9]

If the exposure is detected quickly, dilution with milk or water should be performed; do not induce vomiting or administer activated charcoal. Hospitalize symptomatic cats. Sucralfate slurries can be used to coat and protect oral and esophageal lesions while they heal. Pain management with opioids can make the cats more comfortable. Monitor the white blood cell count and begin antibiotics if signs of infection are evident. Give intravenous fluids for hydration. Cats may be anorectic for several days, so forced feeding or alimentation through a feeding tube may be needed until the cats recover. Endoscopy may be required to evaluate esophageal damage, but be sure to avoid further damage to or perforation of a devitalized esophagus. The prognosis with supportive care is good unless esophageal damage has occurred.

Nonsteroidal anti-inflammatory drugs

Cats may be exposed to nonsteroidal anti-inflammatory drugs (NSAIDs) either by owner administration or, more rarely, by self-ingestion, often with canine chewable formulations. NSAIDs can cause gastrointestinal upset, including vomiting, diarrhea, ulceration, hemorrhage, and ulcer perforation. Acute renal failure can occur at higher dosages. Some NSAIDs

have been associated with CNS signs such as seizures and comas at high doses in cats. The more common drugs that can cause this syndrome include carprofen, ibuprofen, deracoxib, naproxen, etodolac, meloxicam, and indomethacin.[10,11]

In general, cats have a low tolerance for NSAIDs. For example, cats are thought to be at least twice as sensitive to ibuprofen as dogs are.[10] Gastrointestinal ulceration can occur in cats exposed to 4 mg/kg of carprofen; acute renal failure can develop at doses greater than 8 mg/kg (ASPCA APCC Database: Unpublished data, 2001-2005). Because of this sensitivity, most exposures require aggressive treatment.

Initial treatment should consist of gastric decontamination. If spontaneous vomiting has not begun and the ingestion was less than four hours earlier, induce emesis. Then administer activated charcoal and give repeated doses when exposure involves an NSAID that undergoes enterohepatic recirculation. To prevent gastrointestinal ulceration, administer an acid reducer such as an H_2 blocker (*e.g.* ranitidine or famotidine) or proton-pump inhibitor (*e.g.* omeprazole), as well as sucralfate and misoprostol (1 to 3 µg/kg orally b.i.d.)[12] for seven to 10 days. Monitor the cat for signs of gastrointestinal hemorrhage, such as melena or a decreased packed cell volume. Initiate fluid diuresis at twice the maintenance rate for at least 48 hours to prevent renal damage, and monitor the results of renal function tests.[11]

Acetaminophen

As with NSAIDs, acetaminophen is often administered to sick cats by their owners. Acetaminophen has a narrow margin of safety in cats. One adult tablet (325 to 500 mg) could be lethal. Clinical signs such as depression, vomiting, dyspnea, brown discoloration of the mucous membranes and blood due to methemoglobinemia, respiratory distress, swelling of the face and paws, and hepatic necrosis can develop at almost any level of exposure.[11] Signs of methemoglobinemia generally occur within hours of exposure, and liver damage may take a couple of days to manifest.

In asymptomatic cats, emesis may be initiated and activated charcoal administered. If methemoglobinemia is present, start oxygen therapy combined with a blood transfusion or polymerized bovine hemoglobin solution (Oxyglobin—Biopure) administration. Begin *N*-acetylcysteine (*e.g.* Mucomyst—Bristol-Myers Squibb) therapy immediately in any case of acetaminophen exposure in a cat. Dilute the *N*-acetylcysteine solution to a 5% concentration with 5% dextrose or sterile water; this will

yield a 50-mg/ml solution. The loading dose is 140 mg/kg followed by 70 mg/kg every six hours for seven additional doses. Administer *N*-acetylcysteine orally unless either a bacteriostatic filter or a sterile solution of *N*-acetylcysteine (Acetadote—Cumberland Pharmaceuticals) is available. Adjunctive therapy includes intravenous fluids, cimetidine (to inhibit CP450 liver enzymes that activate acetaminophen to the toxic metabolite), and ascorbic acid, which may be used to help reduce methemoglobin to hemoglobin.[11] The prognosis in these cases is fair to guarded.

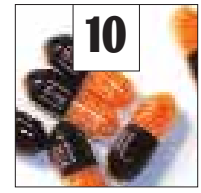
Anticoagulant rodenticides

Although rodent baits are all similar in appearance, do not confuse anticoagulant rodenticides with bromethalin (a neurotoxin) or cholecalciferol (a vitamin D analogue). Small doses of anticoagulants can cause coagulopathy by inhibiting the recycling of vitamin K_1 and blocking the synthesis of clotting factors II, VII, IX, and X. Clinical signs generally occur three to seven days after exposure when circulating clotting factors are depleted. Bleeding may occur in any location, so signs may be nonspecific and include weakness, lethargy, and dyspnea.[13] Hemorrhage is most common in the lungs, so cough or respiratory difficulty is a common finding.[14] Frank hemorrhage or ecchymoses may be seen. Lameness may occur if bleeding occurs in a joint, and various neurologic signs may be noted if bleeding occurs in the brain or spinal cord.[13]

Anticoagulant rodenticide poisoning can be diagnosed by measuring the prothrombin time (PT). PIVKA (proteins induced by vitamin K_1 absence or antagonism) and Thrombotest (Axis-Shield) time are other screening tests for anticoagulant toxicosis. PT and PIVKA tests are most sensitive to depletions of factor VII because it has the shortest half-life.[14]

If performed within two to four hours of exposure, decontamination by inducing emesis and administering activated charcoal is effective at reducing the amount absorbed systemically. Otherwise, treatment with vitamin K_1 (3 to 5 mg/kg orally divided twice daily) is antidotal. Vitamin K_1 should be given for 14 days after warfarin exposure, for 21 days after bromadiolone exposure, and for 30 days after brodifacoum and all other anticoagulant exposure or unknown anticoagulant exposure.[14]

Also test the PT or PIVKA about 48 hours after cessation of vitamin K_1 treatment to determine whether the patient was treated long enough. If an animal presents in hemorrhagic crisis, treatment is generally supportive and should consist of whole blood or plasma transfusions and stabilization as needed as well as vitamin K_1.[13] If treatment is started before coagulopathy, the prognosis is excellent. The prognosis is guarded if the patient is already bleeding.



Amphetamines

In people, amphetamines in prescription medications are used for appetite suppression, attention deficit disorder, and narcolepsy. Another source of amphetamine exposure is illicit preparations of amphetamine, methamphetamine, and 3,4-methylenedioxymethamphetamine (MDMA), also known as *Ecstasy*. Amphetamines act as CNS stimulants by increasing catecholamine release, inhibiting catecholamine reuptake, and increasing release of serotonin.[15] Almost any exposure in a cat can result in clinical signs such as agitation, hyperthermia, tremors, seizures, tachycardia, hypertension, cardiac arrhythmias, and coma (ASPCA APCC Database: Unpublished data, 2002-2005).

Treatment should include gastric decontamination if the animal is asymptomatic, but a rapidity in the onset of clinical signs may limit the possibility for this. Monitor cardiovascular and CNS signs closely. Also monitor body temperature, and maintain it in a normal range. Administer acepromazine or chlorpromazine for agitation, and barbiturates may be used to control seizures.[16] Cyproheptadine may be used as a serotonin antagonist. Treat cardiac arrhythmias as needed (*e.g.* propranolol if tachycardia is present). Intravenous fluids will help promote elimination. Consider administering ammonium chloride or ascorbic acid to acidify the urine and promote elimination if acid-base balance can be monitored. The half-life of the drug and the duration of signs depend on the urinary pH, and signs may be seen for 12 to 48 hours or more.[16] The prognosis with aggressive supportive care is good in most cases.

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