ISSN: 3048-8397

Bio Vet Innovator Magazine
(Fueling The Future of Science…)
Volume 3 (Issue 7) JULY 2026

OPEN ACCESS | World Zoonosis Day – 06th July 2026 | Popular Article

Pharmaceutical (Drug) Toxicities in Animals

Saloni Singla and Rashmi Sagar Bhullar
Associate Professor
Department of Veterinary Pharmacology and Toxicology,
College of Veterinary Science,
Guru Angad Dev Veterinary and Animal Science University, Ludhiana. India
***Corresponding Author:** salonivpt@gmail.com
DOI: https://doi.org/10.5281/zenodo.21453753

Received: July 10, 2026
Published: July 14, 2026

Abstract:

Pharmaceutical toxicities in animals are common and often stem from off-label use, dosing errors, accidental ingestion, species differences in metabolism, and idiosyncratic reactions. Companion animals especially dogs and cats are the most frequently affected. Human over-the-counter and prescription drugs are a major source, with nonsteroidal anti-inflammatory drugs (NSAID, cardiovascular drugs (e.g., calcium channel blockers) repeatedly implicated. Clinical signs range from gastrointestinal upset and ulceration to renal, hepatic, cardiovascular, or neurologic injury, depending on the agent and dose. Species sensitivities vary markedly. Management focuses on rapid decontamination when appropriate, organ support, and targeted antidotes where available. Prevention relies on client education, secure storage of medications, and veterinary guidance before administering human drugs to animals.

Introduction:

Pharmaceutical (drug) toxicity is one of the most common causes of poisoning encountered in veterinary medicine. Drug toxicity occurs when an animal is exposed to a medication at a dose that exceeds its therapeutic index, receives an inappropriate drug for its species, or develops an adverse reaction due to altered metabolism or drug interactions. Companion animals, particularly dogs and cats, account for the majority of reported cases because of accidental ingestion of human medications. Livestock and horses are more commonly affected by dosing errors, extra-label drug use, contaminated feed, or accidental overdosing.

Drug toxicity may result in damage to multiple organ systems, including the gastrointestinal, nervous, cardiovascular, hepatic, renal, and hematopoietic systems. The severity of poisoning depends on several factors, including the drug involved, dose, route of administration, species, breed, age, body weight, and pre-existing disease.

Causes of Pharmaceutical Toxicity:

Common causes include: Accidental ingestion of human medications, overdose of veterinary medicines medication errors (incorrect dose, frequency, or route), off-label drug administration, drug-drug interactions, accidental repeated dosing by owners, breed-specific drug sensitivity, impaired hepatic or renal metabolism and long-term administration leading to cumulative toxicity

Classification of Pharmaceutical Toxicities:

- **Analgesics and Anti-inflammatory Drugs:**

  Acetaminophen poisoning is one of the most serious drug toxicities in companion animals, particularly cats. Normally, acetaminophen is metabolized in the liver through glucuronidation and sulfation. Cats possess deficient glucuronyl transferase activity, leading to accumulation of the toxic metabolite N-acetyl-p-benzoquinone imine (NAPQI). This metabolite causes oxidative damage to hepatocytes and red blood cells. Clinical signs develop within a few hours and include facial swelling, cyanosis, dyspnea, depression, vomiting, weakness, brown-colored mucous membranes due to methemoglobinemia, hypothermia, and jaundice. Severe poisoning results in hepatic necrosis, intravascular hemolysis, and death. Dogs primarily develop liver injury at high doses, whereas cats suffer both hepatic and hematological damage at much lower doses. Diagnosis is based on exposure history, elevated liver enzymes, methemoglobin levels, Heinz body formation, and biochemical abnormalities. Treatment involves administration of **N-acetylcysteine (NAC)** as the specific antidote, oxygen supplementation, intravenous fluids, vitamin C to reduce methemoglobin, S-adenosylmethionine, blood transfusion if required, and intensive supportive care.

- **Non-steroidal anti-inflammatory drugs (NSAIDs):**

  These are among the most frequently encountered causes of drug poisoning in veterinary medicine. Common veterinary NSAIDs include carprofen, meloxicam, flunixin meglumine, ketoprofen, and phenylbutazone, while human NSAIDs such as ibuprofen, naproxen, diclofenac, and aspirin are frequent causes of accidental poisoning in pets. NSAIDs inhibit cyclooxygenase (COX-1 and COX-2) enzymes responsible for prostaglandin synthesis. Although this reduces inflammation and pain, prostaglandins also protect the gastric mucosa, maintain renal blood flow, and support platelet function. Excessive inhibition leads to gastric ulceration, gastrointestinal hemorrhage, renal ischemia, and acute kidney injury. Dogs commonly develop vomiting, diarrhea, abdominal pain, melena, anorexia, dehydration, weakness, and renal failure following NSAID overdose. Cats are particularly sensitive because they metabolize these drugs more slowly, increasing the risk of prolonged toxicity. Birds, especially vultures, are extremely susceptible to diclofenac toxicity, which causes visceral gout and acute renal failure. Diagnosis relies on exposure history, hematology, serum biochemistry, urinalysis, and endoscopy in severe gastrointestinal cases. Treatment includes

immediate decontamination with emetics and activated charcoal when appropriate, intravenous fluid therapy, gastroprotectants such as omeprazole and sucralfate, antiemetics, renal monitoring, and blood transfusion in severe gastrointestinal hemorrhage.

- **Antiparasitic Drug Toxicity:**

  Macrocyclic lactones, including ivermectin, moxidectin, doramectin, selamectin, and milbemycin, are widely used antiparasitic drugs. Toxicity occurs primarily after overdose or administration to genetically susceptible breeds carrying mutations in the **MDR1 (ABCB1)** gene, such as Collies, Australian Shepherds, Border Collies, and Shetland Sheepdogs. The defective P-glycoprotein transporter allows ivermectin to cross the blood-brain barrier and accumulate within the central nervous system. Affected animals exhibit depression, hypersalivation, mydriasis, blindness, ataxia, muscle tremors, recumbency, seizures, coma, respiratory depression, and death. Diagnosis is based on exposure history and neurological examination. Treatment consists of activated charcoal for recent ingestion, intravenous fluids, ventilatory support, when necessary, seizure control, nutritional support, and lipid emulsion therapy in selected cases. Recovery may require several weeks because of the prolonged elimination half-life.

- **Digoxin Toxicity:**

  Digoxin is used to treat congestive heart failure and atrial fibrillation in dogs. It possesses a narrow therapeutic index, making overdose relatively common. Digoxin inhibits the sodium-potassium ATPase pump, increasing intracellular calcium concentrations but predisposing animals to cardiac arrhythmias.

  Clinical signs include vomiting, anorexia, diarrhea, weakness, depression, bradyarrhythmias, ventricular arrhythmias, syncope, hyperkalemia, and sudden death. Diagnosis involves serum digoxin concentration measurement, electrocardiography, and electrolyte evaluation. Treatment includes discontinuation of the drug, correction of electrolyte abnormalities, antiarrhythmic therapy, atropine for severe bradycardia, and digoxin-specific antibody fragments where available.

- **Chemotherapeutic Drug Toxicity:**

  Anticancer drugs possess narrow safety margins because they target rapidly dividing cells. Agents such as doxorubicin, cyclophosphamide, vincristine, cisplatin, carboplatin, and methotrexate may cause severe adverse effects. Bone marrow suppression is the most common toxicity, resulting in neutropenia, anemia, thrombocytopenia, and increased susceptibility to infections. Gastrointestinal toxicity includes vomiting, diarrhea, mucositis, anorexia, and dehydration. Doxorubicin causes cumulative cardiomyopathy in dogs, whereas cisplatin induces fatal pulmonary edema in cats and is therefore contraindicated in that species. Treatment includes

hospitalization, broad-spectrum antibiotics for neutropenia, antiemetics, intravenous fluids, nutritional support, colony-stimulating factors where indicated, and dose adjustment.

- **Antibiotic Toxicity:**

Although antibiotics are generally safe when used appropriately, overdoses or prolonged use can result in significant toxicity. Aminoglycosides such as gentamicin, amikacin, and neomycin are nephrotoxic because they accumulate within renal proximal tubular epithelial cells, causing oxidative damage and tubular necrosis. They may also damage the vestibular and auditory systems, leading to ototoxicity.

- Tetracyclines may produce hepatotoxicity, gastrointestinal disturbances, and permanent tooth discoloration in young animals. Chloramphenicol can suppress bone marrow, resulting in aplastic anemia and pancytopenia. Fluoroquinolones, including enrofloxacin, may cause irreversible retinal degeneration and blindness in cats when administered at excessive doses. Ionophore antibiotics such as monensin, lasalocid, and salinomycin commonly cause skeletal muscle necrosis and myocardial degeneration in horses and dogs due to disruption of intracellular calcium homeostasis. Clinical signs include anorexia, vomiting, weakness, ataxia, deafness, muscle tremors, cardiac arrhythmias, renal dysfunction, and collapse. Treatment requires immediate discontinuation of the offending drug, aggressive fluid therapy, renal support, electrolyte correction, antioxidants, and symptomatic management.

- **Corticosteroid Toxicity:**

Glucocorticoids such as prednisolone, dexamethasone, and methylprednisolone are widely prescribed anti-inflammatory and immunosuppressive drugs. Acute overdose is usually less severe than chronic excessive administration. Long-term exposure produces iatrogenic hyperadrenocorticism (Cushing's syndrome).

- Clinical signs include polyuria, polydipsia, polyphagia, muscle wasting, abdominal enlargement, hepatomegaly, skin thinning, alopecia, delayed wound healing, immunosuppression, secondary infections, hyperglycemia, hypertension, osteoporosis, and adrenal suppression. Diagnosis includes endocrine testing, serum biochemistry, and imaging. Management involves gradual tapering of corticosteroids, treatment of secondary complications, and monitoring adrenal recovery.

Diagnosis of Drug Toxicity:

Diagnosis involves obtaining a thorough history of drug exposure, identifying clinical signs, performing physical examination, complete blood count, serum biochemistry, urinalysis, toxicological screening when available, electrocardiography, radiography, ultrasonography, and organ-specific functional tests. Early diagnosis significantly improves survival.

General Treatment Principles:

Management of drug toxicity focuses on preventing further absorption, enhancing elimination, administering specific antidotes when available, and providing supportive care. Gastrointestinal decontamination includes emesis (when appropriate), gastric lavage, and activated charcoal. Intravenous fluid therapy helps maintain hydration and renal perfusion. Oxygen therapy, seizure control, antiemetics, gastroprotectants, blood transfusion, lipid emulsion therapy, hemodialysis (for selected toxins), and intensive monitoring are essential in severe cases.

Prevention:

We should store all medications securely and out of reach of animals and should never administer human medicines without veterinary advice. Always calculate drug dosages accurately based on species and body weight, use species-appropriate formulations and avoid off-label use unless justified. We should educate owners about breed-specific drug sensitivities and proper medication handling and monitor animals receiving long-term therapy with periodic liver, kidney, and hematological evaluations.

Conclusion:

Pharmaceutical toxicities are a major cause of veterinary emergencies and can affect multiple organ systems. Companion animals are particularly vulnerable to accidental ingestion of human medications, whereas livestock and horses are more often affected by dosing errors or inappropriate drug use. Prompt recognition of exposure, early decontamination when indicated, appropriate antidote administration, and intensive supportive care are critical for improving outcomes. Preventive measures, including owner education, accurate dosing, and awareness of species- and breed-specific sensitivities, remain the most effective strategies for reducing the incidence of drug-induced poisoning in animals.

References:

Ettinger, S. J., Feldman, E. C., & Côté, E. (2024). *Textbook of Veterinary Internal Medicine* (9th ed.). Elsevier.

Gupta, R. C. (Ed.). (2022). *Veterinary Toxicology: Basic and Clinical Principles* (3rd ed.). Academic Press.

Merck Veterinary Manual. Drug Poisoning and Toxicology sections.

Papich, M. G. (2020). *Saunders Handbook of Veterinary Drugs* (5th ed.). Elsevier.

Peterson, M. E., & Talcott, P. A. (Eds.). (2021). *Small Animal Toxicology* (4th ed.). Elsevier.

Plumlee, K. H. (2019). *Plum's Veterinary Drug Handbook* (9th ed.). Wiley-Blackwell.

Citation: Saloni Singla and Rashmi Sagar Bhullar. (2026). Pharmaceutical (Drug) Toxicities in Animals. In Bio Vet Innovator Magazine (Vol. 3, Issue 7 pp. 28–32). Bio Vet Innovator Magazine. https://doi.org/10.5281/zenodo.21453753