

POT IS NOT FOR PETS!

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With the legalization of marijuana in several states, there has been an increased prevalence of accidental exposure to dogs, cats, and children within the past few years.¹⁻³ As a result, veterinarians need to be aware of this growing toxicant. Judicious history taking, along with rapid recognition of clinical signs, is imperative, as pet owners are often unwilling to admit to this illicit drug toxicosis in their pets. Thankfully, with appropriate decontamination and treatment, the prognosis is excellent with symptomatic and supportive care.

Marijuana, found in the *Cannabis sativa* plant, contains the toxic ingredient tetrahydrocannabinol (THC). Marijuana is also common known under the nicknames Mary Jane, pot, hemp, hashish, pot, grass, weed, devil weed/week, etc.⁴ Synthetic marijuana, commonly found in stores, causes similar clinical signs (see treatment). THC directly affects cannabinoid (CB1) receptors in the brain, affecting neurotransmitters [e.g. dopamine, serotonin, gamma-aminobutyric acid (GABA)].^{4,5} This can result in either stimulatory or inhibitory signs.

Pharmacokinetics

Marijuana is rapidly absorbed either orally or when smoked (e.g., via inhalation), and is eliminated via the liver, bile (55%), feces (45%), and urine (17%).⁴ Some enterohepatic recycling occurs.⁵ Duration of signs typically occur within 30 minutes, and can last up to 3 days (with an average duration of clinical signs of 18-24 hours).⁴ Important to note is that clinical signs can be seen at very low doses of marijuana exposure; that said, the LD₅₀ in dogs is extremely high (considered to be > 3 g/kg).⁵

Clinical signs

Animals may exhibit mild to moderate signs after inhalational exposure (e.g., smoke), but are more likely to become symptomatic after ingestion (which is the more common route of accidental animal exposure). Clinical signs include: central nervous system signs (e.g., ataxia, disorientation, hyperesthesia, agitation, hyperactivity, dysphoria, mydriasis, behavioral changes, tremors, seizures, coma), gastrointestinal signs (e.g., hypersalivation, vomiting), cardiopulmonary signs (e.g., bradycardia, tachycardia, hypoventilation), and miscellaneous other signs [e.g., urinary incontinence, temperature changes (e.g., hypo- or hyperthermia), death]. Clinical signs of marijuana toxicosis can quickly progress to obtundation, coma, bradycardia, hypotension, tremors, seizures, and rarely, death. Signs can develop quickly (within 5 minutes) or be delayed up to 12 hours; most often, occur with 1-3 hours of exposure.⁴

Clinicopathologic testing

With marijuana poisoning, no significant clinicopathologic findings are noted. In patients suspected of having underlying disease (e.g., metabolic, geriatric, etc.), a baseline complete blood count and general chemistry panel can be considered. In patients suspected of hypoventilating secondary to the sedative effects of marijuana, a venous blood gas should be performed to evaluate the partial pressure of carbon dioxide (pCO₂; normal reference range 30-35 mm Hg). Patients with a severe respiratory acidosis (e.g., pCO₂ > 50 mm Hg) are candidates for intubation and mechanical ventilation. If hypoxemia (e.g., defined as a partial pressure of oxygen of < 80 mm Hg; normal reference range 80-100 mm Hg) is a concern, the use of an arterial blood gas as a gold standard should be considered.

While some emergency veterinary clinics often screen for marijuana poisoning with human urinary drug screen tests, the author cautions that careful interpretation of the results is imperative. False negative results can be seen with THC due to the unique metabolites in dogs as compared to humans.⁵ That said, a positive result (e.g., even a weak positive) is consistent with marijuana poisoning.

Treatment

Treatment for marijuana poisoning includes appropriate decontamination; however, this should be performed judiciously. Keep in mind that emesis induction is often not rewarding, due to the antiemetic effect of THC. If the patient is already symptomatic, emesis should *not* be induced. For example, if the patient has a decreased gag reflex or is excessively sedate, emesis should not be induced due to the risk of aspiration pneumonia. In this situation, if the marijuana is thought to still be present in the stomach (e.g., wads of buds ingested), it is safer to perform gastric lavage (with an inflated endotracheal tube to protect the airway) under sedation for gavage, followed by activated charcoal administration directly through the orogastric tube. Typically, multiple doses of activated charcoal can be administered due to enterohepatic recirculation.

Additional treatment includes gastrointestinal support. Anti-emetics (e.g., maropitant, dolasetron, ondansetron) should be considered to prevent vomiting and secondary aspiration. Fluid therapy with a balanced, maintenance crystalloid can be implemented to maintain hydration and perfusion. Symptomatic supportive care, including thermoregulation, nursing care, anxiolytics (for agitated, tachycardia, and/or hypertensive patients including acepromazine or benzodiazepines), and monitoring (e.g., electrocardiogram, blood pressure, pulse oximetry, end-tidal CO₂, etc.) should be performed.

Life-threatening clinical signs are less commonly seen with marijuana toxicosis, but can potentially result in demise of the patient. In patients that are hypoventilating (e.g., respiratory rate < 6 bpm, increased end-tidal CO₂, respiratory acidosis, hypercapnea,

etc.), intubation and mechanical ventilation are recommended. Bradycardiac patients (canine patients with a heart rate < 40-50 bpm) may require the use of atropine (0.01 mg/kg IM, IV PRN). If tachycardiac is seen (canine patients with a HR > 180 bpm), a blood pressure should be checked. If the patient is both tachycardiac and *hypertensive*, the use of anxiolytics (e.g., acepromazine at 0.05 mg/kg, IV, IM PRN) or beta-blockers (e.g., propranolol 0.02 mg/kg IV, up to max dose of 0.1 mg/kg) are warranted. If the patient is both tachycardiac and *hypotensive*, a crystalloid fluid bolus should be considered (e.g., 20-30 ml/kg IV).

While there is no “cure” for marijuana poisoning in veterinary medicine, the prognosis is generally excellent with supportive care. One potential “antidote” that can be in severe, potentially life-threatening cases is the use of intravenous lipid emulsion (ILE). Please see “IV Lipid Therapy: Are we really giving IV fat?” proceedings more for information. Dosing for ILE in veterinary medicine is extrapolated from human medicine at:⁶

- 20% solution: 1.5 – 4 ml/kg IV over 1 minute, followed by 0.25 mg/kg/min, over 30-60 minutes.⁶
- Re-dosing of aliquots of 1.5 ml/kg q 4-6 hours can also be continued for 24 hours if needed OR follow-up CRI doses of 0.5 ml/kg/hr can be continued until clinical signs improve (not to exceed 24 hours).⁶

Conclusion

Thankfully, with rapid recognition of clinical signs and prompt treatment, the prognosis for marijuana toxicosis in veterinary medicine is excellent with supportive care. Veterinarians should be aware of the growing prevalence of exposure in dogs and cats due to the legalization of medical marijuana in certain states, and should be able to rapidly recognize and treat this ever-growing toxicity.

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