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Marijuana Toxicosis

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As the changing legal landscape has made marijuana increasingly accessible in the United States, accidental exposures have become increasingly common in our pets. THC toxicosis is a common presentation in both veterinary ERs and primary care clinics. A 2021 survey of veterinarians throughout the US and Canada found a significant increase in reported case numbers compared to just several years prior.

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Most cases of marijuana toxicosis follow ingestion of either edible products, plant material, or medical THC preparations. Nearly two-thirds of exposures reported to the Pet Poison Helpline involved ingesting commercial or homemade edible products. These products are often intended to be split into many smaller doses and, when consumed in bolus, can contain very high concentrations of THC. Dogs are reported to have more cannabinoid receptors than humans, maybe even more sensitive than their human counterparts to THC's psychoactive effects.

Marijuana ingestion is not always witnessed, and owners are not always certain whether or where their pets may have gained access to THC-containing products. Unfortunately, no reliable bedside tests are available to quickly and definitively diagnose THC toxicity in veterinary patients. Some clinics rely on commercially available urine drug screening tests intended for human use. While a positive on these dipstick tests may prove diagnostically helpful — particularly when exposure status is unknown — false negatives are common. This may be due to differences between human and canine THC metabolism, error associated with sample handling (THC can bind to glass or rubber collection vials), or testing performed too soon after exposure.

The major toxin present in marijuana is THC. THC is rapidly absorbed and distributed into the tissues, crossing the blood-brain barrier. The onset of clinical signs usually occurs within 60 minutes of ingestion. Signs of toxicosis may include CNS depression, ataxia, hyperesthesia, vomiting or hypersalivation, hypothermia, dribbling urine, bradycardia or tachycardia, mydriasis or miosis, hypotension, tremors, or in severe cases, seizure or coma. THC's minimum lethal dose is extremely high — greater than 3g/kg. For reference, the average 1g joint contains approximately 150mg of THC. However, with the rise of easily accessible edible preparations, exposure loads can be relatively high, increasing the likelihood that pets may present with severe CNS effects. Other considerations for pets who present with marijuana toxicosis — particularly pets who have ingested edibles — include potential exposure to other toxins (such as chocolate, xylitol, or raisins) and ingestion of foil or other packaging materials.

Often, patients with only mild symptoms can be discharged for care at home, where they should be monitored in a safe environment and kept away from stairs, pools, or other risks of falls or injury. Severely affected animals — those obtunded, respiratory depressed, experiencing seizures, or comatose — should be hospitalized for monitoring and supportive care. Though marijuana has anti-emetic properties, induction of emesis can be attempted in patients presenting within 60 minutes of exposure or when there is clinical suspicion that a significant amount of ingested material remains within the stomach. Care should be taken not to induce emesis in particularly sedated patients or present with CNS depression to reduce the risk of aspiration. Vomiting can be treated with anti-emetics. For patients with a low anticipated risk of aspiration, activated charcoal can be administered. As THC undergoes hepatic recirculation, one to two additional doses can be repeated every six to eight hours. Clinicians can provide fluid support (note that IV fluid therapy is not expected to enhance THC excretion significantly) and heat support as needed based on clinical status. Phenothiazines or benzodiazepines can be given as needed to manage agitation (avoid acepromazine in significantly hypotensive patients). Because THC is highly lipid soluble, clinicians may consider intravenous lipid emulsion (ILE) therapy for patients presenting with severe CNS signs. Intralipid therapy has been used with variable success in veterinary patients — ILE's mechanism of action is still not fully understood, and further research is needed to elucidate optimal dosing protocols in dogs fully. A typical protocol is a 1.5ml/kg IV bolus given over one to two minutes, followed by a 0.25ml/kg/min CRI for an additional 30 to 60 minutes. If needed, this dose can be repeated once the patient's serum is no longer lipemic (typically four to six hours following the initial dose). When administering lipids, clinicians should collect blood samples before administration, as the hyperlipemia caused by treatment can interfere with lab assays, and consider that ILE may also impact other lipid-soluble drugs (such as sedatives or anticonvulsants).

Overall, the prognosis for THC toxicosis is generally very good for patients receiving appropriate supportive care. Most dogs will recover over 24 to 36 hours. For patients with high exposure loads (particularly those ingesting high concentrations of edible preparations), full resolution of clinical signs may take several days.

Reference

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