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Australian tea tree (*Melaleuca alternifolia*) oil poisoning in three purebred cats

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Tea tree oil, a botanical product commonly sold in natural food stores, is an extract of the leaves of the Australian tea tree (*Melaleuca alternifolia*). The tea tree is in the family Myrtaceae, which also contains eucalyptus, cloves, and myrtles. An essential oil, tea tree oil is similar in composition and toxicity to eucalyptus oil.[2] It contains terpenes, sesquiterpenes, hydrocarbons, and related oils. Because of its lipophilic nature, tea tree oil is readily absorbed through the skin. Similar to eucalyptus oil, tea tree oil has a pungent odor.

Herbal medicines, including tea tree oil, are becoming more common in companion animal care as people are seeking natural health care products for themselves and their pets. Tea tree oil is advertised in health food stores as being safe and nontoxic. Tea tree oil toxicosis, but not death, has been reported in cats.[7,9] In this case report, we document tea tree oil poisoning in 3 adult intact female purebred Angora cats, one of which died.

Three female Angora cats were presented to a veterinary clinic in north central Oklahoma. The cats had been recently acquired by a breeder, who had vaccinated them and treated them for fleas. The cats were severely infested with fleas, so they were shaved. The shaving produced no nicks on the skin; however, numerous flea bites were visible. The product used to eliminate fleas was labeled for use as a spot treatment for skin lesions, but a catalog advertised that it would repel fleas when diluted and used as a dip. The product[a] contained 100% oil of *Melaleuca alternifolia.* The oil was applied directly to the cats' skin, and 2 1-oz (approximately 60 ml) bottles were used on the 3 cats.

Within 5 hours of treatment, cat 1 was brought to the veterinarian. It was hypothermic and uncoordinated. It was unable to stand but was alert. Cats 2 and 3 were admitted later that day. Cat 2 was comatose with severe hypothermia and dehydration. Cat 3 was alert and nervous, slightly ataxic, and trembling. All cats had a strong minty odor similar to that of the tea tree oil product.

The cats were bathed in warm water and a mild detergent to remove any remaining oil from the skin. Activated charcoal was administered orally to adsorb any ingested tea tree oil. All cats were given dexamethasone. Cats 1 and 2 were given isotonic saline solution intravenously for rehydration, and their body temperatures were increased using heat lamps and warm water bottles.

Serum chemistry values for all 3 cats were evaluated. Alanine aminotransferase levels for cats 1, 2, and 3 were 233, 135, and 82 IU/liter, respectively (reference range, 10–88 IU/liter). Aspartate aminotransferase levels for cats 1, 2, and 3 were 88, 413, and 107 IU/liter, respectively (reference range, 10–80 IU/liter). Cat 2 had mildly elevated blood urea nitrogen (BUN), although the creatinine level was within normal limits. A differential blood cell count on cat 2 showed leukocytosis (28,000 white blood cells; reference range, 5,500–19,500 cells) with neutrophilia (26,880; reference range, 1,925–10,725). No band cells were present.

Cat 3 recovered within 24 hours of being admitted to the veterinary hospital, and cat 1 recovered after 48 hours. Both cats were sent home to the cattery, and no problems have been reported since. Cat 2 improved over days 2 and 3 but remained ataxic and obtunded. Although cat 2 was being treated with aggressive fluid therapy, it remained dehydrated. On day 3, it began regulating its own body temperature, but it was found dead late that evening. The cause of death is unknown, the carcass was not available for postmortem evaluation.

A pooled sample of urine from all 3 cats was submitted to the Oklahoma Animal Disease Diagnostic Laboratory (OADDL) for analysis. The material remaining in one of the tea tree oil containers was also submitted to be used as a positive control. Urine and tea tree oil samples were extract-

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ed using chloroform. Extracts of tea tree oil and urine were analyzed by gas chromatography–mass spectroscopy (GC-MS)[b] at a flow rate of 1.1 ml/minute, with an injector temperature of 225 C and column[c] temperature of 40–300 C. The tea tree oil product contained 42% terpinen-4-ol, which is consistent with *M. alternifolia* oil. The cat urine contained terpinen-4-ol and unidentified metabolites.

The essential oil of *M. alternifolia* varies in chemical composition[2,6] but usually contains 50–60% terpenes. The main terpenes, which are regulated by the Australian government, are terpinen-4-ol[2–4,7,9] (minimum 30%)[3,4,9] and 1,8-cineole[1–4,9] (maximum 15%).[2,3,9] Terpinen-4-ol is known to have antibacterial and antifungal properties.[2,3,6,7,9] The allowable level of 1,8-cineole is low because this terpene causes irritation of skin and mucous membranes.[2,3,6,9] 1,8-Cineole is also the major component of eucalyptus oil.[1] The concentration of 1,8-cineole in tea tree oil tends to be inversely proportional to the concentration of terpinen-4-ol.[2] Tea tree oil can also contain p-cymene if it is improperly stored or stored for a long period of time.[9]

Tea tree oil was first isolated in 1925.[8] Because it has antibacterial and antifungal properties[4,7,9] and is believed by some to be useful as an antipruritic and an insect repellant,[9] it is currently used in many different products, including pure tea tree oil, gels and body lotions, shampoos, conditioners, balms, liniments, toothpaste, insect repellents, and air conditioner germicides.[2,6] Products that are sold for use on companion animals include shampoos for cats, dogs, ferrets, and horses[2,6,7] and pure tea tree oil products similar to the one used on these cats.

Knowledge of the toxicity of *Melaleuca* oil is variable among health food store personnel and natural health care practitioners. Many health food store personnel believe tea tree oil to be nontoxic. Veterinary and human natural health care practitioners tend to be aware that *Melaleuca* oil can cause serious side effects, and some have had clinical experience with tea tree oil toxicosis or hypersensitivity. Cats may be more susceptible to tea tree oil toxicosis than dogs.

The toxicity of *Melaleuca* oil is actually similar to that of other essential oils, such as eucalyptus oil,[2,8] that contain terpenes.[9] Toxicosis in a human has resulted from ingestion of 0.5–1 ml tea tree oil/kg body weight.[4,8] Approximately 0.67 oz (20 ml) was applied to each cat in this case study.

Terpene metabolism is mostly hepatic and involves phase I and phase II biotransformation.[1,9] Terpenes are ultimately conjugated to glycine or glucuronide.[9] Glycine conjugation is probably more important than glucuronide conjugation in cats, who are poor glucuronide conjugators. Terpene metabolites are excreted primarily in the urine over 2–3 days after exposure.[9] Fecal excretion is a minor route of elimination.[9]

Tea tree oil toxicosis has been reported in humans, rats, dogs, and cats.[3–8] Most patients have clinical signs of central nervous system depression. In 1 case, a child who ingested 10 ml of tea tree oil had signs of confusion, disorientation, incoordination, and unsteady gait.[5] The child recovered within 7 hours.[5] In another report, an adult ingested half of a teacup of tea tree oil and was comatose for 12 hours, was semiconscious and hallucinating for the following 36 hours, and had diarrhea for 6 weeks.[3,8] In a third report, a man who would occasionally ingest half a teaspoon of tea tree oil developed a rash soon after dosing;[3,8] this may have been a hypersensitivity reaction.[8]

Tea tree oil ingestion in rats induced mucosal irritation, respiratory distress, and coma.[8] Dermal toxicity in the rabbit has been tested, and no toxicity was seen with topical doses up to 2 g of oil/kg body weight.[2]

Tea tree oil toxicosis reported in dogs and cats has been associated with misuse of the product, as was the case in this report.[7,8] Dogs and cats will appear weak, obtunded, uncoordinated, and ataxic and may have muscular tremors.[3,7,9] Clinical signs usually begin soon after treatment with the product, and in one case, 2 kittens developed central nervous system depression within minutes of being dipped in a cleaning product containing tea tree oil.[7] The elevated liver enzymes in the cats in this case study suggest that tea tree oil is also hepatotoxic to cats. Death from tea tree oil poisoning has not previously been reported in domestic animals. The cat that died in this case, cat 2, had central nervous system depression typical of tea tree oil poisoning. However, the elevated BUN and persistant dehydration suggests that this cat may have had renal damage unrelated to tea tree oil intoxication.

The terpenes in tea tree oil are rapidly absorbed through the skin and digestive tract because of their highly lipophilic nature.[2,4,9] Toxicity from dermal exposure has not been produced in rabbits[2] but may be possible.[2,4] Cats may be more susceptible to tea tree oil toxicosis after topical application than are other species because of their grooming behavior. In this case, the cats had been shaved, so that tea tree oil was applied directly to the skin, enhancing dermal absorption. The flea bite lesions may have also increased dermal absorption somewhat. Further, grooming efficiency was probably augmented due to shaving, allowing cats to ingest a large amount of the oil applied.

Diagnosis of tea tree oil or other essential oil toxicosis usually relies upon history of exposure. Animals recently exposed to tea tree oil are likely to have a pungent odor from the oil. Exposure can be confirmed by the presence of terpinen-4-ol in the urine of the animal by GC-MS. Quantitation of terpinen-4-ol in the urine was not possible in this case but was not necessary for proper treatment of the cats. The sample was pooled from 3 cats, 2 of which had been treated with intravenous fluids, so the level in the urine was expected to be low.

There is no antidote for tea tree oil toxicosis.[9] General detoxification and supportive care should be given. A mild detergent bath will prevent further dermal absorption of the oil and further oral ingestion due to grooming behavior.[7,9] Activated charcoal and saline cathartics should be administered to adsorb the oil and move it through the gastrointestinal tract, respectively.[7,9] Heart rate, respiratory rate, body temperature, hydration, and serum electrolytes should be monitored and corrective action taken as needed.[9] The prognosis for animals poisoned with *Melaleuca* oil is good with treatment, unless the animal has other health problems.

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Sources and manufacturers

a. Miracle Coat™ Pure Tea Tree Oil, Miracle Corp of Australia, Dayton, OH.
b. Autosystem GC Q-Mass 910, Perkin-Elmer Corp., Norwalk, CT.
c. RTX®-5MS Column, Restek Corp., Bellafonte, PA.

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Lasalocid toxicosis in neonatal calves

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Lasalocid is an ionophore used as a growth promotant feed additive in cattle and as a coccidiostat in cattle, sheep, and poultry.[1,10] Lasalocid toxicosis has been reported in feeder calves following feed mixing errors[4] and in neonatal calves after administration of lasalocid.[9]

In the absence of an approved treatment for cryptosporidiosis in neonatal calves, the off-label use of lasalocid for this purpose has been attempted in the field using several dosage forms. In this report, we describe 2 cases of lasalocid toxicosis resulting from use of a commercially available liquid coccidiostat/antidiarrheal compound containing lasalocid. In an effort to more accurately characterize postmortem lesions and clinical signs and to rule out the effects of the polypharmacy reported in the field cases, 2 newborn calves were dosed experimentally with the commercial coccidiostat used in the field cases.

Two cases of bovine neonatal death loss were submitted to the Iowa State University Veterinary Diagnostic Laboratory. In the first case, 3 calves were submitted for postmortem examination. A liquid oral coccidiostat had been used at 4 times the label dose as a preventative for cryptosporidiosis, providing 200 mg of lasalocid per dose given once daily by depositing the liquid with a syringe in the posterior oral cavity beginning within 12 hours of birth. In addition, the calves were given injections of flunixin meglumine, gentamicin, and *Clostridium perfringens* types C and D antitoxin and oral doses of *E. coli* K99 monoclonal antibody and enrofloxacin. Calves were found dead or in lateral recumbency with opisthotonos within 12–24 hours after administration of the drug. Necropsies were performed, and tissues were collected in formalin and routinely processed for light microscopic examination. Bacteriologic cultures were made from lung, intestinal sections, liver, spleen, and brain. Spleen, lymph node, and colon were used for direct fluorescent antibody examination, feces were used for enzyme-linked immunosorbent assay and lung, spleen, and lymph nodes were used for virus isolation.

In the second case, a 36-hour-old calf developed clinical signs of recumbency, dyspnea, and ataxia and died after receiving since birth 100 mg lasalocid in each twice-daily feeding of milk replacer (total of 3 doses). Fresh and formalin-fixed specimens of brain, cardiac muscle, lung, spleen, kidney, and rumen were submitted for histopathologic examination and bacteriologic and virologic tests.

In the first case, postmortem examination revealed severe pulmonary congestion and moderate interlobular edema. Two of the calves had milk curd in the abomasum. No other gross changes were observed. On histopathologic examination the lungs were diffusely congested and had abundant proteinaceous fluid and fibrin in the alveoli. In addition, 1 calf that had survived for 24 hours had focal areas of purulent bronchopneumonia. No significant histopathologic changes were observed in the brain, thyroid, thymus, cardiac muscle, skeletal muscle, small intestine, large intestine, liver, kidney, or adrenal gland. No evidence of cryptosporidial infection was observed. Bacterial cultures of the tissues did not result in the isolation of any pathogens. Direct fluorescent antibody examination and virus isolation procedures did not demonstrate the presence of rotavirus, coronavirus, or bovine viral diarrhea virus.

Postmortem lesions seen in the second case were pulmo-

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