THE HAZARDS OF ICE MELTS
to Dogs and Cats

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It may not always cause clinical signs, but ice melt exposure can have serious side effects.

A glistening layer of ice has covered the sidewalks and driveway. You take out a bag of ice melt to throw over the ice, not realizing that your curious dog is only too anxious to taste the white granules.

Liquid and solid ice melts designed to de-ice roadways and sidewalks can be potentially harmful to pets. Solid ice melts, which are available in a variety of formulas, contain such ingredients as sodium, potassium, magnesium, and calcium chloride, calcium carbonate, calcium magnesium acetate, and urea.[1] Solid ice melt formulas are most commonly available for purchase by individual consumers. Liquid ice melts, which are generally used commercially for the purpose of de-icing roads and highways, may contain magnesium and calcium chloride, propylene and ethylene glycol, urea, or brine mixtures.[2] Since dogs and cats are more apt to have access to solid ice melt formulas, this column focuses on how to treat ingestions of solid ice melts.

CLINICAL SIGNS AND TREATMENT

Clinical signs associated with solid ice melt exposures include salivation, depression, polydipsia, diarrhea, vomiting, anorexia, vocalizing/crying, disorientation, and apprehension.[2] If exposure is known to have occurred, decontamination procedures may prevent the development of signs. Since most solid ice melts are dermal irritants, it is generally recommended to bathe the exposed animal in a mild, noninsecticidal pet shampoo and rinse well to sufficiently remove any ice melt residue. Inducing emesis[a] should be considered in asymptomatic pets that have ingested large quantities; however, this may be contraindicated with some ice melt formulations. Pets that have recently had surgery or those with underlying health conditions (e.g., epilepsy) in which forced vomition poses health risks should be decontaminated in a clinic rather than at home. Activated charcoal is unlikely to be beneficial because it poorly adsorbs to the salts in ice melts.[1] Once signs develop, management is largely symptomatic and supportive and often includes fluid therapy, emesis management, electrolyte monitoring, and possibly gastrointestinal (GI) protection.

Although many cases of ice melt exposure do not result in serious clinical signs, the potential for development of more serious side effects exists, depending on the formulation ingested.

Sodium Chloride

When large quantities of sodium chloride ice melts are ingested, sodium ion toxicosis may potentially develop as a result of hypernatremia (increased sodium levels in the blood). Hypernatremia is diagnosed when serum sodium concentrations reach more than 155 mEq/L.[3,4] Dogs that ingest 4 g/kg or more of sodium chloride are at high risk of death.[1,5] When large quantities of salt are ingested, the body tries to compensate for the increased burden of sodium in the bloodstream by conserving water (resulting in concentrated urine) and increasing water intake through stimulation of the thirst response mechanism.[3,6] When water intake is denied or insufficient in diluting the increased level of sodium, water is drawn from internal sources. Neurologic signs may develop if electrolyte imbalances continue or if sodium dilution in chronically hypernatremic patients occurs too rapidly for the body to adjust, resulting in brain swelling.

Dogs and cats that have ingested large quantities of sodium chloride ice melts will likely vomit as a result of their body's response to the ingestion. In effect, the vomiting that results serves as a form of decontamination, possibly resulting in a decreased dose of sodium chloride and making it less likely for healthy dogs and cats to develop hypernatremia. Although it is rare for healthy dogs and cats to develop hypernatremia due to increased salt intake, those with underlying cardiac or renal insufficiency are at greater risk since their ability to manage sodium overloads is already compromised.[3,4] Small animals with impaired thirst response mechanisms or those denied access to water are also at increased risk of hypernatremia.[3,4]

The main clinical signs associated with sodium ion toxicosis may include vomiting, polydipsia, polyuria, sinus tachycardia, tremors, seizures, and metabolic acidosis.[1] Additional signs may include diarrhea, muscle weakness, disorientation, behavior changes, coma, and death.[4]

Vomiting must be carefully managed since withholding water or continued vomiting increases the possibility of hypernatremia. Treatment of hypernatremia primarily involves replacing water and electrolytes while assisting with renal excretion of excess sodium.[1,4] It is suggested that serum sodium concentrations be gradually reduced over 48 to 72 hours to prevent brain cells from undergoing osmotic injury.[1,4] Although there is some controversy as to which fluid therapy is best, 5% dextrose is often preferred in cases of acute toxicosis.[4] Loop diuretics (e.g., furosemide) may be beneficial for animals with cardiac disease or oliguria due to kidney disease.[4] In patients with these health conditions, there is a serious threat of development of pulmonary edema during fluid administration.[1,4] Sodium bicarbonate should be used cautiously when treating acidosis to avoid exacerbating hypernatremia and hyperosmolality.[1] Anticonvulsant medications are indicated for patients showing neurologic signs.[1]

Potassium Chloride

Potassium chloride is a severe irritant with the potential to cause GI distress, possibly to the point of hemorrhage.[7,8] Ingestion of potassium chloride ice melts may result in sustained hyperkalemia in patients with decreased renal efficiency.[1,9] Hyperkalemia is most likely to develop in oliguric or anuric small animals.[9]

Clinical signs associated with hyperkalemia may include muscle weakness, vomiting, diarrhea, hypotension, and abnormal cardiac conduction.[1,7] Cardiac problems are more likely to occur in patients with underlying cardiac disease.[8]

Due to the irritating nature of potassium chloride, emesis is controversial in the management of potassium chloride ice melt ingestion.[1] Treatment of hyperkalemia requires intravenous fluid therapy and furosemide or hydrochlorothiazide.[1] Recommended fluids include lactated Ringer's solution or 0.9% sodium chloride solution.[1] Monitoring of serum electrolytes, blood urea nitrogen, blood glucose concentrations, and blood gases is suggested.[1] If continued monitoring is possible, administration of sodium bicarbonate may be warranted if blood gases indicate acidosis.[1]

Magnesium Chloride

Ice melts containing magnesium chloride may contain magnesium dust that is potentially irritating and may cause GI upset.[1] Small animals with impaired health conditions are more susceptible to additional complications associated with magnesium overdosages. More specifically, patients with renal failure are more prone to develop hypermagnesemia because impaired glomerular filtration prevents excretion of magnesium at a rate proportional to the serum concentration.[1,7]

Ingestion of magnesium may result in such signs as vomiting and diarrhea. More severe clinical signs include weakness, hypotension, respiratory depression, impaired neuromuscular transmission, and cardiac abnormalities (e.g., atrioventricular block, prolonged QT intervals, bradycardia).[1,7] Very large magnesium overdoses may result in disruption of neuromuscular activity and cardiac arrest.[7]

Treatment of magnesium chloride ingestion is symptomatic and supportive.[1] Inducing vomiting is recommended with any exposure to reduce absorption of magnesium chloride if ingestion has occurred within 2 hours.[1] Monitoring healthy patients for development of signs may be all that is required. In animals with compromised health, clinical monitoring may be recommended to avoid complications and to provide symptomatic and supportive care as needed.

Calcium Salts

Under certain circumstances, ingestion of large quantities of calcium salts can lead to elevated serum calcium concentrations; however, the requirement of an acidic pH, parathyroid hormone, and vitamin D for absorption of calcium salts makes it unlikely that acute ingestion of ice melts containing calcium salts would cause increased serum calcium concentrations.[1] Calcium carbonate and calcium magnesium acetate are moderate irritants with the potential to cause gastritis.[1] Calcium chloride is the most severe irritant among calcium salts and may result in ocular irritation, skin irritation (which may include burns), and GI irritation and/or hemorrhage.[10]

Acute ingestion of most calcium salts is not likely to cause severe signs unless calcium chloride is present.[10] Due to the potential for severe irritation, dilution with water and dermal decontamination is suggested in calcium chloride exposures.[10] Inducing emesis should be carefully considered due to the possibility of sustaining severe GI and esophageal irritation during the process of vomition. In most cases of calcium salt ingestion, emesis is not warranted unless other potentially problematic substances have been ingested as well.[1] Treatment is symptomatic and supportive if signs develop.

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[a]Feeding a soft meal and administering fresh 3% hydrogen peroxide at a dose of 1 teaspoon per 5 lb of body weight or 1 tablespoon per 20 lb of body weight (not to exceed 3 tablespoons in a dog 60 lb or larger) is likely to be effective. This can be repeated once if no vomiting occurs within 10 to 15 minutes of the first dose.

Toxicology Brief is contributed by veterinary technicians at the American Society for the Prevention of Cruelty to Animals–Animal Poison Control Center, 1717 S. Philo Rd., Suite 36, Urbana, IL 61802; hotline: 888-4ANI-HELP (888-426-4435) or 900-680-0000 (a $45 consultation fee is charged to the caller's telephone bill); email: callen@napcc.aspca.org (for nonemergency information only); web site: www.apcc.aspca.org.

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Urea

Ruminants and large bowel fermenters are most susceptible to urea toxicosis from ingestion of urea ice melts.[1] The microflora in these animals creates an environment in which urea is hydrolyzed and releases carbon dioxide and ammonia.[1] Monogastric animals lack the intestinal flora required to establish an environment conducive to urea toxicosis.

Monogastric animals may develop such clinical signs as hypersalivation, gastroenteritis, abdominal pain, and possibly increased blood ammonia concentrations[1]; less commonly, methemoglobinemia, weakness, and tremors may occur.[1] Treatment includes inducing emesis, electrolyte monitoring, and symptomatic and supportive care as needed.[1]

CONCLUSION

It is important to educate clients on the hazards that ice melt ingestions pose to their pets and inform them about proper storage and use of these products so that exposures may be avoided. In most cases of ice melt exposures, signs are limited to mild dermal irritation and GI upset. However, the possibility exists that some ice melt exposures may be severe enough to stimulate development of compounding syndromes (e.g., sodium ion toxicosis, hyperkalemia, hypermagnesemia, and urea toxicosis), which lead to more severe signs requiring intense management. Obtaining accurate information about the patient's health status as well as the type and amount of ice melt ingested is important to proper case management.

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