

Ice melts are health hazards

by

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Pets may be exposed to ice melts that have been spilled, applied to sidewalks, or improperly stored. In 1998, more than 50 cases of ice-melt exposure were reported to the ASPCA Animal Poison Control Center (ASPCA APCC Antox[®] Database: Unpublished data, 1998). Vomiting - the most prevalent sign - occurred in 30% of the cases. Other effects included diarrhea, salivation, depression, anorexia, tremors, disorientation, polydipsia, seizures, and death.

Before initiating treatment for ice-melt ingestion, it's critical to know the ice melt's ingredients and the animal's health status. Inducing emesis is controversial if the product contains a large percentage of potassium chloride and is contraindicated in some species and health conditions. For example, rabbits, rodents, and birds don't vomit, and poorly controlled epileptics may experience seizures with induction of vomiting and shouldn't be induced at home. Activated charcoal doesn't usually adsorb the salts in ice melts. If an animal has walked through the product, rolled in it, or placed its face in a bag of ice melt, bathe the animal and monitor it for skin irritation. Also monitor and correct abnormalities in hydration status, electrolyte concentrations, and heart muscle activity. As mentioned above, vomiting and diarrhea are the most common side effects, but large ingestions can cause more serious problems.

Many brands of sidewalk ice melts are on the market. The most common ingredients in these ice melts are sodium chloride, potassium chloride, magnesium chloride, calcium carbonate, and calcium magnesium acetate. A few ice melts contain urea.

Sodium chloride

Sodium ion toxicosis is possible after large ingestions of ice melts, salt, or rock salt. A dose of 4 g/kg of sodium chloride can be lethal in dogs.¹ Ingesting large amounts of sodium leads to hypernatremia, osmotic diuresis, and increased urine osmolality. Increases in sodium retention let sodium passively diffuse into the cerebrospinal fluid. Active transport is required to remove sodium from the cerebrospinal fluid. Because energy is required for active transport, extracellular sodium concentrations usually decrease faster than sodium concentrations in the cerebrospinal fluid. Sodium trapped in the central nervous system attracts water because of the osmotic gradient, resulting in cerebral edema.^{1,2} The gastroenteritis and diuresis that occur may combine to cause dehydration, further worsening the patient's condition.

The clinical signs of hypernatremia are primarily neurologic and related to osmotic movement of water out of brain cells. The severity of the clinical signs is related more to the rapidity of the onset of hypernatremia than the magnitude.² Signs reported in one dog with fatal hypernatremia from salt ingestion included vomiting, polydipsia, polyuria, fine muscular fasciculations, sinus tachycardia, metabolic acidosis, and seizures.³

Diagnosis of sodium ion toxicosis is generally based on serum, cerebrospinal fluid, or brain sodium concentrations and a history of sodium ingestion or lack of water intake. Normal sodium concentrations in animals are 135 to 155 mEq/L in plasma and 135 to 150 mEq/L in cerebrospinal fluid. Serum and cerebrospinal fluid sodium concentrations above 160 mEq/L or brain sodium concentrations above 1,800 ppm are considered diagnostic of sodium ion toxicosis.⁴

The main goals in treating hypernatremia are to replace water and electrolytes and, if necessary, facilitate renal excretion of excess sodium. Serum sodium concentrations should be lowered gradually over 48 to 72 hours to avoid osmotic injury to cells.² There are arguments about what fluid works best, but many people suggest using either 5% dextrose in water or saline solution.^{2,3} Use sodium bicarbonate cautiously for treating acidosis because of possible worsening of hypernatremia and hyperosmolality. Loop diuretics, such as furosemide (2 to 4 mg/kg t.i.d. or q.i.d. orally, intravenously, or intramuscularly), may help in acute toxicosis and may prevent the development of pulmonary edema during fluid therapy. Anticonvulsant medication may be necessary to control central nervous system signs during therapy.

Potassium chloride

Increased potassium intake is unlikely to cause sustained hyperkalemia unless renal excretion is also impaired. Signs associated with hyperkalemia include muscle weakness, gastrointestinal disturbance, and cardiac conduction disturbances. A serum potassium concentration greater than 8 mEq/L is considered diagnostic of hyperkalemia in dogs and cats.⁵ Ingestion of potassium chloride tablets has caused gastric stenosis, bowel strictures, and esophageal ulcerations in people.⁶⁻⁸

Treatment of hyperkalemia includes administering intravenous fluids (*e.g.* lactated Ringer's solution, 0.9% sodium chloride solution) and furosemide or hydrochlorothiazide (2 to 4 mg/kg orally). Because of the irritating nature of these salts, emesis is controversial. Activated charcoal does not bind to potassium. Monitor serum electrolyte, blood urea nitrogen, and blood glucose concentrations and blood gases. If blood gases indicate acidosis and continued monitoring is possible, 0.5 to 2 mEq/kg of sodium bicarbonate may be administered intravenously over 20 to 30 minutes.⁹

Magnesium chloride

Hypermagnesemia can occur after ice-melt ingestion. Normal serum magnesium concentrations in dogs and cats are 1.8 to 3 mg/dl and 1.9 to 2.28 mg/dl, respectively.⁹ Hypermagnesemia can cause hypotension, hypophosphatemia, cardiac abnormalities (atrioventricular block, prolonged QT intervals, and bradycardia), weakness, and impaired neuromuscular transmission. Patients with renal failure are more susceptible to developing hypermagnesemia. The LD₅₀ of magnesium chloride in rats is about 4,000 mg/kg. Dust from products containing magnesium may be irritating and can cause upset stomach.¹¹ Treatment of magnesium ingestion is symptomatic and supportive. Emesis may reduce the amount absorbed if induced within two hours of ingestion.

Calcium carbonate and calcium magnesium acetate

Acute ingestion of these calcium salts is unlikely to increase serum calcium concentrations because of the requirement of an acidic pH, parathyroid hormone, and vitamin D for absorption. They are, however, moderately severe irritants and may cause gastritis and conjunctivitis. Emesis is generally not warranted unless these salts have been ingested with other problematic substances. Treat gastric upset symptomatically.

Urea

Monogastric animals are not susceptible to urea poisoning but may exhibit increased blood ammonia concentrations.¹ Ruminants and large-bowel fermenters are susceptible because their intestinal microflora provides an ideal environment for the hydrolysis of urea, releasing carbon dioxide and ammonia. Ingestion of urea by dogs usually results in local irritant signs such as hypersalivation, gastroenteritis, and abdominal pain. Less frequent signs include methemoglobinemia, weakness, and tremors.⁹ Managing urea ingestion in monogastric animals includes inducing emesis and monitoring electrolyte concentrations.

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