

A Novel Source of Acrylamide in Migraine Caps Containing Hydrogel

Leah D Swanson, Renee D Schmid* and Lynn R Hovda

Pet Poison Helpline/SafetyCall International, LLC, USA

***Corresponding author:** Renee D Schmid, Pet Poison Helpline/SafetyCall International, LLC, 3600 American Blvd W #725, Bloomington, MN 55431

Received: January 22, 2026

Published: March 16, 2026

Pet Poison Helpline® (PPH), a 24/7 international animal poison control center, has observed a concerning trend in acute oral canine exposures to popular migraine hydrogel masks resulting in gastrointestinal and life-threatening neurologic signs. Affected canines have presented with profound neurologic signs within several hours of ingestion. In one case, ingestion of approximately one tablespoon of migraine wrap hydrogel by a 4.7 kg canine resulted in severe signs including vomiting, tachycardia, ataxia, generalized tremors, and grand-mal seizures. The patient was discharged with neurologic deficits, and the outcome is unknown due to loss of follow-up. Similarly, among dogs that have survived after ingesting migraine wrap hydrogel, a subset has exhibited persistent neurologic deficits upon discharge, the long-term resolutions of which remains undetermined.

Evaluation of the safety data sheets for a variety of these hydrogels lists polyacrylamide, water, and glycerol as the main constituents. Glycerol has been implicated in neurologic signs attributed to osmotic shifts in several species; however, it has a wide margin of safety and unlikely to be responsible for neurologic signs seen in these cases. In canines, gastrointestinal tract irritation and osmotic effects have been reported at doses exceeding 5,600mg/kg following administration of 100% glycerol [1]. Per PPH's database, patients consuming the hydrogel have not developed classic electrolyte disturbances due to an osmotic shift, ruling out glycerol as the primary concern for neurologic signs. Polyacrylamide itself has a wide margin of safety, with a single acute oral dose of 4.0 g/kg in rats being well tolerated [2]. However, polyacrylamide gels may contain residual acrylamide monomer ranging from 0.01% to 0.1% [2].

Residual acrylamide monomers are regarded as impurities in most polyacrylamide preparations and may become a toxicologic concern at higher concentrations if monomer polymerization is incomplete. Acrylamide is a reactive neurologic toxicant, in contrast to the polymerized form, which is generally regarded as having low toxicity. In addition to residual monomer from the production process, acrylamide may also be generated under high-temperature conditions, like microwaving, through the Maillard reaction, which involves reducing starches and amino acids, a well-established pathway in

food and industrial manufacturing processes [3]. Furthermore, a formulation component like glycerol could interfere with polymer cross-linking and promote physical destabilization of the polymer network, potentially facilitating the release of unpolymerized acrylamide monomer [4]. Collectively, elevated acrylamide monomer levels may reflect pre-production issues (e.g., incomplete polymerization), formulation-related instability, or post-production degradation of polyacrylamide gels.

Liquid and gas chromatography-mass spectrometry (LC and GC-MS) was performed on gel samples obtained from the masks purchased new and used in accordance per label instructions. Acrylamide monomer, a known neurologic toxicant, was detected, correlating with the acute clinical syndrome exhibited by these patients. Quantitative acrylamide analysis via LC/GC-MS is pending and will further elucidate exposure levels.

Unlike polyacrylamide polymer, acrylamide monomer possesses significant acute toxicity with a narrow margin of safety. It is readily absorbed via oral, dermal, and inhalational routes, and distributes rapidly and systemically, causing pronounced neurotoxic effects at relatively low doses in mammals [5,6]. Acute exposure in animal studies have demonstrated neurologic signs in cats at doses as low as 65mg/kg, with seizures and mortality reported at 100mg/kg and an estimated LD50 ranging from 100 to 200mg/kg [5]. Consistent with this data, acrylamide induced neurotoxicity has been associated with central and peripheral nervous system impairment including ataxia, tremors, seizures, and other neurologic abnormalities [6]. There have been case reports in canines in veterinary literature of acute neurologic signs and deaths resulting from acrylamide toxicosis suspected to be formed by the Maillard reaction in burned porridge [3].

Although the underlying pathophysiology of acute neurologic signs in the case of acrylamide toxicosis has not been fully elucidated, three primary mechanisms have been proposed in the literature to explain acute neurologic signs: (1) oxidative stress secondary to glutathione depletion; (2) disruption of neurotransmission, resulting in decreased acetylcholine, dopamine, and norepinephrine levels; and (3) longer-term effects associated with axonopathy characterized by terminal axonal degeneration [5].

In cases reported to PPH, canines have developed severe neurologic signs such as ataxia, tremors, hyperesthesia, gait abnormalities, dull mentation, and seizures. Routine diagnostic evaluation, including complete blood count, serum biochemical analysis, electrolyte profile, and ethylene glycol screening, was unremarkable in these cases, except for marked hemoconcentration in some cases. This finding was consistent with dehydration likely secondary to severe acute gastrointestinal signs observed immediately preceding the onset of neurologic signs.

Given the rapid and life-threatening nature of the clinical signs described with this toxicosis, immediate decontamination in the asymptomatic patient followed by aggressive symptomatic and supportive care are indicated upon presentation to a veterinary facility. Consultation with a pet poison center is recommended as these cases are complicated and evolve rapidly. The potential for chronic neurologic sequelae in survivors aligns with acrylamide's established neurotoxic profile and underscores the need for heightened clinical vigilance and further research into both acute and long-term outcomes following exposure to acrylamide.

In conclusion, the qualitative confirmation of acrylamide within the hydrogel in conjunction with the severity of the associated clinical manifestations, is consistent with acrylamide toxicosis profiles described in the literature. These findings identify a previously unrecognized source of acrylamide expo-

sure, expanding the current understanding of a potential toxicologic risk and warranting further consideration by clinicians. Prevention of exposure by pet owners at home, increased clinical vigilance, and prompt implementation of symptomatic and supportive therapeutic measures may reduce adverse outcomes associated with these hydrogel migraine mask exposures.

References

1. EFSA Panel on Food Additives and Nutrient Sources added to Food (ANS), Mortensen A, Aguilar F, Crebelli R, Di Domenico A, Dusemund B, et al. Re-evaluation of glycerol (E 422) as a food additive. *EFSA J Eur Food Saf Auth*, 2017; 15(3): e04720.
2. Amended Final Report on the Safety Assessment of Polyacrylamide and Acrylamide Residues in Cosmetics I. *Int J Toxicol*, 2005; 24(2 suppl): 21–50.
3. Le Roux-Pullen L, Lessing D. Should veterinarians consider acrylamide that potentially occurs in starch-rich foodstuffs as a neurotoxin in dogs? *J S Afr Vet Assoc*, 2011; 82(2): 129–130.
4. Bol'shakov AI, Kuzina SI, Kiryukhin DP. Spontaneous polymerization of acrylamide in glycerol: Active centers and specific features. *Polym Sci Ser A*, 2008; 50(10): 1045–1051.
5. Yamamoto R, Yasuoka T, Matsushima J, Tsubouchi Y, Kanazashi H, Sakurai K, et al. Acute acrylamide poisoning with severe symptoms in a short time: a case report. *Int J Emerg Med*, 2023; 16(1): 41.
6. Faria M, Ziv T, Gómez-Canela C, Ben-Lulu S, Prats E, Novoa-Luna KA, et al. Acrylamide acute neurotoxicity in adult zebrafish. *Sci Rep*, 2018; 8(1): 7918.