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Effects of oral 3% hydrogen peroxide used as an emetic on the gastroduodenal mucosa of healthy dogs

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Abstract

Objective – To characterize the extent of mucosal injury on the upper gastrointestinal tract following oral administration of 3% hydrogen peroxide (H_2O_2) to induce emesis in normal dogs.
Design – Prospective clinical study.
Setting – Specialty referral hospital.
Animals – Seven staff-owned, healthy, adult dogs.
Interventions – Six dogs were assigned to the H_2O_2 group and 1 dog was assigned as the apomorphine control. Dogs were anesthetized for gastroduodenoscopy with gross inspection and gastroduodenal biopsies at time 0 and 4 hours, 24 hours, 1 week, and 2 weeks following administration of oral 3% H_2O_2 or subconjunctival apomorphine. Gross esophageal, gastric, and duodenal mucosal lesion scoring was performed by 2 blinded, experienced scorers. Biopsy samples were evaluated histologically by a veterinary pathologist.
Measurements and Main Results – Grade I esophagitis was noted in 2 dogs at 4 hours and in 1 dog at 2 weeks, while grade III esophagitis was observed in 1 dog 1 week following H_2O_2 administration. At 4 hours, gastric mucosal lesions were visualized in all dogs, and lesions worsened by 24 hours. Mild to moderate duodenal mucosal lesions were visualized up to 24 hours after administration. Histopathology identified the most severe gastric lesions at 4 hours as hemorrhage; at 24 hours as degeneration, necrosis, and mucosal edema; and at 1 week as inflammation. By 2 weeks, most visual and histopathologic lesions were resolved. No histopathologic lesions were identified at any time point in the dog administered apomorphine.
Conclusions – Significant visual and histopathologic gastric lesions occurred following administration of 3% H_2O_2 in all dogs. Less severe visual duodenal lesions were identified. As compared to H_2O_2 dogs, minimal gross gastroduodenal lesions and normal histopathology were identified in the apomorphine control.

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Keywords: canine, decontamination, emesis, esophagitis, gastritis, toxicosis

Abbreviations

H_2O_2	hydrogen peroxide
ICC	intraclass correlation coefficient

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The authors disclose no conflicts of interest.

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Introduction

Hydrogen peroxide is a clear, odorless liquid commonly found in households, hospitals, and industries. It ranges in concentration from 3% to 90%.[1] Lower concentrations of H_2O_2 (3–9%) are used for disinfecting, tooth whitening, removing cerumen, and bleaching.[1] At higher concentrations (> 10%), H_2O_2 is used as a strong oxidizing agent for bleaching and deodorizing textiles, wool, and paper. It has even found use in the production of rocket fuel and foam rubber.[1,2]

In veterinary medicine, H_2O_2 is frequently used to induce emesis in toxicologic emergencies. Pet owners are often advised by veterinarians to administer H_2O_2, a common household product, for immediate emesis at home. The published dose recommended is 1–2 mL/kg of 3% H_2O_2 administered orally, with 1 subsequent dose

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if emesis is not achieved.[3] While oral administration of small volumes of 3% H_2O_2 is reported to be benign, veterinary studies to support this assumption are lacking.[1] A recent study reported that minor, self-limiting adverse effects were observed in 14% of animals after induction of emesis with 3% H_2O_2.[4] This study evaluated only clinical signs reported by owners, without endoscopic or histopathologic evaluation of the gastrointestinal mucosa. There are case reports and retrospective series on accidental and deliberate ingestions of 3% H_2O_2 in people. Although most human ingestions of the substance are assumed to be nontoxic, there are reports of severe complications.[5] Gastric ulcers, hemorrhagic gastritis, portal venous emboli, and death have been described.[5,6]

To the authors' knowledge, the effects of 3% H_2O_2 administration on gastrointestinal mucosal integrity in dogs have not been reported. The purpose of this investigation was to determine the extent of upper gastrointestinal mucosal injury following oral administration of 3% H_2O_2 in healthy dogs. The authors hypothesized that H_2O_2 would cause significant damage to the gastrointestinal mucosa at the published dose and concentration recommended for veterinary use, and that significant evidence of mucosal injury would be apparent histologically as a direct result of H_2O_2 administration.

Materials and Methods

Nine staff-owned dogs were recruited from June 2013 to March 2014 for this prospective clinical study. Eight of the 9 dogs were recruited for the H_2O_2 study group, while 1 was recruited as the apomorphine study control. Dogs were included in the study based on a normal physical examination; gastroduodenoscopy without evidence of gross lesions and normal histologic findings; negative heartworm antigen test; and normal CBC, serum biochemistry, T4, and urinalysis results at baseline. Two dogs were excluded due to preexisting gastritis identified at the baseline gastroduodenoscopy. Each of the remaining 6 dogs in the H_2O_2 group were administered 1 dose of 3% hydrogen peroxide[a] from a new, previously unopened bottle at 2 mL/kg, not to exceed a total dose of 45 mL. This dose was repeated once after 10 minutes if emesis was not achieved. This dosing was chosen based on the recommended published dose of 3% H_2O_2.[3,7] The control dog received 0.25 mg/kg apomorphine[b] crushed and applied to the conjunctival sac.[8] The conjunctival sac was rinsed with sterile saline after induction of emesis. No anti-emetic medication was administered after emetic administration in any dog. All staff provided informed consent prior to patient inclusion, and measures were taken to minimize discomfort. In case of intractable or persistent vomiting (> 20 minutes of continuous vomiting), a rescue protocol was developed, and consisted of placement of an IV catheter followed by administration of IV fluids, anti-emetic medications, and gastrointestinal protectants for ≥ 24 hours. None of the study dogs required the rescue protocol.

Table 1: Savary–Miller classification of esophageal injury

Grade of esophagitis	Description
I	Single erosion
II	Confluent erosions
III	Circular, confluent erosions
IV	Esophageal ulceration, stenosis, or perforation

Criteria previously published in Rousseau A, Prittie J, Broussard JD, et al.[9]

Dogs were anesthetized for gastroduodenoscopy with gross inspection, photography, and biopsy sampling at 4 hours, 24 hours, 1 week, and 2 weeks following oral administration of 3% H_2O_2 or apomorphine. All endoscopic procedures were performed with a flexible video-gastroscope.[c] Propofol[d] (4–6 mg/kg IV) was administered to effect as an induction agent. Following endotracheal intubation, general anesthesia was maintained with isoflurane[e] in oxygen (100 mL/kg/min) with 0.9% NaCl solution[f] delivered at 10 mL/kg/h IV. Dogs were placed in left lateral recumbency and gastroduodenoscopy was performed by 1 of 2 experienced clinicians. Each procedure was performed in such a way to avoid iatrogenic lesions prior to photography.

The endoscope was inserted into the esophagus. With insufflation, the upper and lower esophageal sphincters as well as the proximal, mid, and distal esophagus were evaluated visually. Photographs were taken at each section. The endoscope was gently advanced through the lower esophageal sphincter into the stomach and insufflated to distend the rugal folds. Photographs were taken of the gastric body, pyloric antrum, pyloric sphincter, angularis incisura, and cardia. The endoscope was then advanced carefully through the pyloric sphincter into the proximal descending duodenum. Photographs of the proximal descending duodenum were obtained. Following photography, multiple biopsies (ranging from 6–8 each) were taken of the proximal duodenum and stomach. All dogs recovered well from anesthesia and were observed for several hours after the procedure. No medications were administered to the dogs between endoscopic examinations.

Endoscopic photographs of the esophagus, lower esophageal sphincter, gastric cardia, gastric body, angularis incisura, and duodenum were scored by 2 experienced observers blinded to each of the studies. Esophageal lesions were scored based upon the Savary–Miller classification (Table 1).[9] Gastric and duodenal

Table 2: Gastroduodenal lesion scores

Score	Description
1	Normal
2	1 mucosal hemorrhage
3	2–5 mucosal hemorrhages
4	> 5 mucosal hemorrhages
5	1 erosion
6	2–5 erosions
7	> 5 erosions
8	1 ulcer
9	2 ulcers
10	> 2 ulcers
11	Perforating ulcer

Criteria and table previously published by Graham AH, Leib MS.[10]

lesions were assigned a score from 1 to 11 for each region based upon a previously described scale (Table 2).[10] A mucosal hemorrhage was defined as a small, erythematous lesion with intact mucosal epithelium. An erosion was defined as a defect in mucosal epithelium. An ulcer was described as a large mucosal defect with a deep center and raised margins. Summation of scores from all regions was then performed, and a total score was given to each dog for each endoscopic exam. The intraclass correlation coefficient (ICC) was computed to assess agreement between the 2 observers' scores. The ICC (2,1), based on a 2-way random effects analysis of variance (ANOVA) model, was used because the same pair of observers scored each dog.[11] The ICC was calculated using the "irr" package in R version 3.1.3.[g]

Gastric and duodenal endoscopic biopsies were fixed in 10% neutral buffered formalin[h] for ≥ 24 hours then processed into paraffin,[i] sliced at 5 micron sections and stained with standard hematoxylin and eosin staining.[j,k] All endoscopic biopsies were evaluated by a board-certified veterinary pathologist based on the World Small Animal Veterinary Association guidelines for evaluation of intestinal biopsies developed by the Association's International Gastrointestinal Standardization Group.[12] Sections were evaluated for epithelial degeneration, necrosis, and regeneration; mucosal inflammation; mucosal edema; and mucosal hemorrhage. Each of these parameters was evaluated as normal, minimal, mild, moderate, or severe.

In the first dog, the final gastroduodenoscopy was performed 1 week following 3% H_2O_2 administration. Since histopathologic changes were still present at 1 week in this dog's biopsy samples, the remaining dogs also underwent gastroduodenoscopy 2 weeks following H_2O_2 administration. The histopathology results for the initial dog were not available until the third week, and therefore, no 2-week data could be collected from this animal.

Results

Seven staff-owned dogs (2 females, 5 males), with a median age of 5 years (range, 2–8 years) and median body weight of 14.2 kg (range, 4.6–35.5 kg) were included in the study. Six of the 7 dogs were placed in the 3% H_2O_2 group, while 1 dog was used as the apomorphine control. Mixed breed dogs were most common (57%), followed by the Pembroke Welsh Corgi (28.6%) and the Golden Retriever (14%). Small, medium, and large-sized dogs were included into the study (28.6% small, 42.9% medium, and 28.6% large).

Emesis was successfully induced with H_2O_2 and apomorphine in all dogs. Five of the 6 dogs vomited after receiving 1 dose of H_2O_2, with the remaining dog requiring a second dose. The mean time to onset of emesis with 3% H_2O_2 was 4.5 minutes. The time to onset of emesis with conjunctival administration of apomorphine was 2 minutes. The mean number of times emesis occurred following 3% H_2O_2 was 1.8 times. The apomorphine study control dog vomited 7 times following its administration. The ICC was 0.95 (95% confidence interval, 0.65–0.99) that indicates "excellent" agreement of the observers' endoscopic examination scores.[13]

Visual scoring in dogs receiving H_2O_2

Esophageal – The majority of the dogs had minimal esophageal lesions. Four hours following H_2O_2 administration, 2 of the 6 dogs had a Grade I esophagitis, which resolved by 24 hours. One week following H_2O_2 administration, 1 dog had Grade III esophagitis, which improved to Grade I esophagitis by the 2 week mark. Another dog had Grade I esophagitis at 2 weeks post hydrogen peroxide treatment.

Gastric – By 4 hours after H_2O_2 exposure, all dogs had developed gastric mucosal lesions, ranging from a single mucosal hemorrhage to 2–5 mucosal erosions and an ulcer. All 6 dogs showed worsening of gastric lesion scores in ≥ 1 area of the stomach from 4 hours to 24 hours post 3% H_2O_2. Three dogs had 3 regions of the stomach in which gastric mucosal lesions grossly deteriorated in appearance from 4 hours to 24 hours. Additionally, 1 dog was subjectively noted to have significantly increased hemorrhage and friability of gastric tissues at the time of biopsy up to 24 hours.

Median total gastric mucosal lesion scores showed evidence of moderate gross mucosal injury, which generally improved by 2 weeks following ingestion of 3% H_2O_2 (Figure 1). Median gastrointestinal lesion scores of the gastric body, pylorus, and cardia increased from 4 hours to 24 hours, while the angularis incisura improved from 4 hours to 24 hours. Median gastrointestinal lesion scores improved consistently from 24 hours to 2 weeks in all areas of the stomach. For the first 24 hours following H_2O_2

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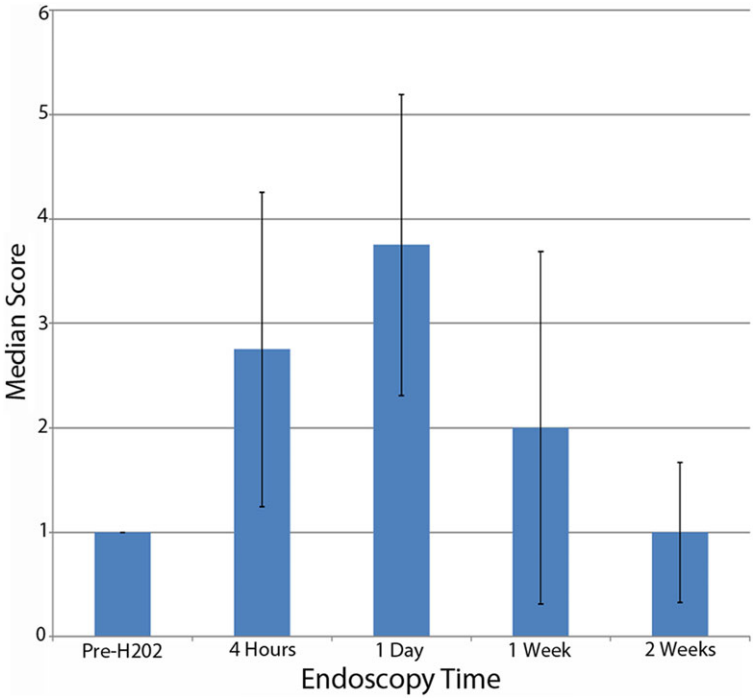

Figure 1: Median total gastric mucosal lesion scores before and after 3% hydrogen peroxide administration. Error bars represent the range of total endoscopic scores. The most severe lesions were noted at 24 hours ("1 Day"), with gradual improvement or resolution by 2 weeks.

administration, the highest average gastroduodenal mucosal lesion scores were noted at the gastric body, while at 1–2 weeks following H_2O_2 exposure, the pylorus had the highest average gastroduodenal mucosal lesion scores.

An ulcer was identified at the gastric body at 4 hours and 24 hours in 2 different dogs. Two ulcerations were identified at the cardia in 1 dog at 24 hours. By 2 weeks following H_2O_2 administration, all ulcers were resolving, but not completely healed. Perforating ulcers or > 2 ulcers were not identified at any time point in this study.

Duodenal – By 4 hours, 83% of dogs had duodenal mucosal lesions visually identified, ranging from 1 mucosal hemorrhage to > 5 erosions. Significant median total duodenal mucosal lesion scores were identified 4 hours following 3% H_2O_2 administration, and most lesions improved visually by 2 weeks (Figure 2). On average, the duodenum had the lowest median mucosal lesion scores at all study points. Further, appreciable hemorrhage and fragility of the duodenal mucosa was observed at the time of biopsy in 1 dog at 4 hours and 24 hours.

Visual scoring apomorphine

No esophageal lesions were identified visually at any time following emesis induced by conjunctival administration of apomorphine. Gastric mucosal hemorrhages (2–5 mucosal hemorrhages) were identified at the pylorus at 4 hours and 24 hours, and at the angularis incisura 1 week following emesis induced by apomorphine administration. The pyloric hemorrhages resolved by 1 week, and the angularis incisura lesions resolved by 2 weeks following emesis induced by apomorphine administration. Duodenal mucosal lesion scores were normal at all-time points studied following emesis induced by conjunctival apomorphine administration.

Histopathology

All biopsy samples from all animals were histologically normal before treatment. Significant histopathologic injury to the gastric mucosa was noted in all dogs administered 3% H_2O_2 (Table 3). No significant histologic duodenal changes were noted in any dog after H_2O_2 treatment. No changes were noted in any biopsy sample from the apomorphine-treated dog at any time point.

Follow-up

Upon follow-up examination at 12 months following H_2O_2 exposure, 5 of 6 dogs showed no reported clinical signs of gastrointestinal distress. The other dog was reportedly reluctant to eat for 1 month following H_2O_2 administration, with a gradual return of appetite to normal. The dog having received apomorphine remained clinically normal at 6 months follow-up.

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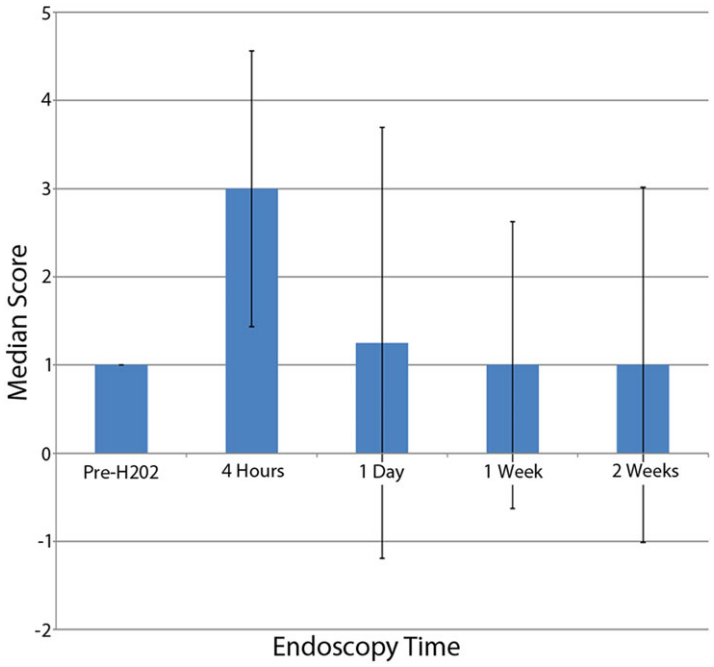

Figure 2: Median total duodenal mucosal lesion scores before and after 3% hydrogen peroxide administration. Error bars represent the range of total endoscopic scores. The most severe lesions were noted at 4 hours with gradual improvement or resolution by 2 weeks.

Table 3: Histopathology results (hydrogen peroxide group)

	Epithelial degeneration	Epithelial necrosis	Inflammatory infiltrates	Mucosal edema	Mucosal hemorrhage
Pre-H_2O_2	0/6	0/6	0/6	0/6	0/6
4 Hours	6/6 (minimal to moderate)	5/6 (minimal to moderate)	6/6 (minimal to early mildly neutrophilic)	6/6 (minimal to moderate)	5/6 (minimal to mild)
24 Hours	6/6 (minimal to moderate)	6/6 (minimal to moderate)	6/6 (minimal to mildly neutrophilic)	6/6 (minimal to mild)	5/6 (minimal to mild)
1 Week	2/6 (minimal or mild)	1/6 (minimal necrosis)	2/6 (minimal or mildly neutrophilic) 3/6 (mild lymphoplasmacytic inflammation)	4/6 (minimal to mild)	2/6 (minimal or mild hemorrhage)
2 Weeks	0/6	0/6	1/6 (mild lymphoplasmacytic gastritis)	0/6	0/6

Discussion

This study found that gross and histopathologic gastric lesions occurred following administration of 3% H_2O_2 for induction of emesis in normal dogs, which challenges the notion that H_2O_2 administration in dogs is benign. The most severe lesions identified were gastric ulcers and gastric degeneration and necrosis, evident at 4 and 24 hours following H_2O_2 ingestion. Most gastroduodenal lesions were present for up to 1 week, with resolution by 2 weeks. The duodenum was less affected grossly than the esophagus or the stomach, and was less affected than the stomach histologically. The esophagus was not evaluated histologically.

In people, extent of mucosal injury determined by esophagogastroduodenoscopy strongly correlates with the risk of death and systemic complications following ingestion of 35% H_2O_2.[1] Endoscopic assessment within 24 hours of H_2O_2 ingestion is therefore used to estimate prognosis in people.[14]

Somewhat surprisingly, minimal gross esophageal lesions were noted following H_2O_2 administration in this study. In people, hemorrhagic blebs, diffuse

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superficial ulcerations, and erythema of the lower esophageal sphincter have been identified within 24 hours following H_2O_2 ingestion on esophagoscopy.[14] Esophageal stricture formation is reported to occur 6–12 weeks thereafter.[1] To prevent serious esophageal injury, physicians often advise immediate ingestion of large quantities of water following H_2O_2 ingestion.[1] Gross evidence of minimal, spontaneously resolving esophagitis (Grade I esophagitis) was observed in 2 of 6 dogs 4 hours following H_2O_2 administration. Two dogs without previously identified esophagitis developed mild esophagitis at the 1 week mark and minimal esophagitis 2 weeks following H_2O_2 exposure. The significance of spontaneously arising esophagitis identified at these time points is unknown. Gastric reflux subsequent to gastroduodenoscopy was considered a possibility. Minor esophageal effects seen in these study dogs could be explained by a rapid esophageal transit time or ingestion of a low concentration of H_2O_2, as most esophageal injuries reported in people follow ingestion of $\geq$ 10% H_2O_2.

Friability of the gastric mucosa has been reported in people upon gastroduodenoscopy following H_2O_2 ingestion.[14] Subjectively, this observation was also noted when obtaining biopsies in all study dogs up to 24 hours following H_2O_2 exposure. Notable fragility of the gastric and duodenal mucosa was appreciated in 1 dog 4 and 24 hours following H_2O_2 administration. These time points corresponded with the most severe histopathologic and gross lesions. In this dog, the gastroduodenal mucosa was friable, and tore easily with immediate, moderate hemorrhage upon performing biopsies. Based on this information, oral H_2O_2 administration may be dangerous in animals with disease affecting the integrity of the gastrointestinal mucosa, such as acute gastroenteritis, shock, or inflammatory bowel disease, or in those animals that have ingested a potentially caustic substance. These dogs may be at a higher risk of substantial gastrointestinal injury, such as sloughing of the mucosa or perforation.

Apomorphine administration appeared safe and effective in the single control dog in this study. No histopathologic lesions were identified at any time point evaluated, and minimal gross gastric hemorrhagic lesions were noted at 4 hours, 24 hours, and 1 week after apomorphine administration. This control dog vomited 4 times more than the mean number of times of emesis occurred in the dogs that received H_2O_2. This situation supports the theory that gastroduodenal mucosal injury is secondary to direct caustic injury of 3% H_2O_2, rather than the act of vomiting. Hydrogen peroxide is reported to cause morbidity by 3 mechanisms: direct caustic injury, oxygen gas formation, and lipid peroxidation.[1] As an unstable oxidizing agent, H_2O_2 releases water and oxygen upon its decomposition. In the presence of catalase, H_2O_2 dissociates into these components. Catalase is found in mucous membranes, liver, kidney, RBCs, and bone marrow.[1] Formation of oxygen gas could theoretically lead to perforation of a hollow organ or a gas embolism. Common locations of gas emboli in humans include the portal venous system, gastric wall, and brain.[14] Also, ingestion of H_2O_2 causes lipid peroxidation and subsequent direct cytotoxicity.[1]

This study used a measured dose (2 mL/kg) of 3% H_2O_2 in each dog, which was repeated once if emesis did not occur after administration of the first dose. In the home environment, the volume of H_2O_2 administered, number of doses, and concentration of H_2O_2 given likely vary. Therefore, the potential exists for more severe adverse effects with administration at home. Potential lethal adverse effects reported in people include gas embolism, perforation of a hollow organ, or death. These complications have been documented in human case reports even with small volume ingestion of 3% H_2O_2. Ingestion of 2–4 ounces (60–120 mL) and "one handful" of 3% H_2O_2 have caused multiple gastric ulcers and diffuse hemorrhagic gastritis with portal venous gas embolism in people, respectively.[5,6]

Despite the gross mucosal injury identified with gastroduodenoscopy, most of the dogs in this study, including the dog that received 2 doses of H_2O_2, appeared clinically normal following the acute emesis event. Only 1 of the dogs showed long-term effects following H_2O_2 ingestion, specifically a decreased appetite of 1 month duration. Interestingly, this dog suffered the most duodenal mucosal injury following H_2O_2 at all time points, ranging from $>$ 5 mucosal hemorrhages to $>$ 5 erosions. At 2 weeks, visual mucosal lesions remained present, although improving, in all scored areas of the stomach, and duodenal injury was still present, with an average gastroduodenal lesion score of 5. Moderate, multifocal lymphoplasmacytic gastritis with normal duodenum was noted in this dog histopathologically at 2 weeks. The observation of persistent gross and histopathologic mucosal injury secondary to H_2O_2 in this dog may have been reflected in its decreased appetite.

A primary limitation of this investigation was the number of study dogs, as this limited the ability for statistical analysis. A formal statistical hypothesis test to compare histological outcomes for dogs treated with 3% H_2O_2 versus apomorphine was not possible because only 1 dog was treated with apomorphine. Estimates of both the mean and variability in groups' histological outcomes are needed to compare groups, but no estimate of variability can be obtained from a single study subject. Future studies with larger sample sizes are warranted to provide more data for statistical analysis and provide stronger evidence regarding the risk of H_2O_2

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administration for emesis induction. Another limitation to this study was the inability to definitively distinguish previous biopsy sites from new, active mucosal lesions occurring due to the H_2O_2. This limitation may have biased our visual scoring system to more severe lesions identified at the earlier and more frequent time points between studies. However, this limitation did not appear to be problematic with the apomorphine control, further highlighting their differences in affecting gastric mucosa.

Results of this investigation suggest that apomorphine should be considered in lieu of H_2O_2 when considering upper gastrointestinal tract injury. If apomorphine is not readily available for use within 1–2 hours of toxin ingestion, a veterinarian may consider advising at-home use of H_2O_2, if the benefits of decontamination outweigh the risks of its use. Emetic use in people, both at home and in the emergency department, has been challenged greatly in the past several years and generally is not advised due to severe adverse effects and apparent indifferent influence on prognosis.[13] Overall, results of this study suggests that the use of 3% H_2O_2 to induce emesis in dogs should not be considered entirely innocuous. Results of this study serve as a reminder that H_2O_2 is a caustic substance, directly causing substantial gastric mucosal degeneration, necrosis, inflammation, hemorrhage, and edema. Effects were sustained for 1 week following H_2O_2 administration, and generally resolved by 2 weeks. Further studies with larger sample sizes are needed to support or refute the results of this investigation.

Footnotes

[a] 3% Hydrogen Peroxide, Henry Schein Animal Health, Dublin, OH.
[b] Apomorphine HCl, Diamondback Drugs, Scottsdale, AZ.
[c] Flexible Video Gastroscope, Order No. 60814NKS, Karl Storz GmbH & Co. KG, Tuttlingen, Germany.
[d] Propofol, 1% Injectable Emulsion, Hospira, Inc., Lake Forest, IL.
[e] Isoflurane USP, Abbott Laboratories, North Chicago, IL.
[f] 0.9% Sodium Chloride Injection USP, Hospira, Inc.
[g] R Development Core Team. R: A language and environment for statistical computing. R Foundation for Statistical Computing, 2015; Vienna, Austria.
[h] 10% Neutral Buffered Formalin, SARL Scientific, Kalamazoo, MI.
[i] Paraffin, Leica Biosystems Richmond, Inc., Richmond, IL.
[j] Hematoxylin, Mercedes Medical, Orlando, FL.
[k] Eosin Y, Harleco-EM Science, Gibbstown, NJ.

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