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Sucrose Permeability Testing as an Indicator of Gastric Ulceration in Horses (21-Nov-2003)

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Abstracts

There is great need for a simple and economical method for diagnosing equine gastric ulceration. Sucrose permeability testing is an accurate method for detection of moderate to severe gastric ulceration in horses.

1. Introduction

The prevalence of gastric ulceration in horses is high [1]. Currently, gastric endoscopy (gastrosocopy) is the only method for definitively diagnosing and monitoring the healing of gastric ulcers in horses. The large size and high cost of endoscopic equipment for equine gastroscopy limit availability of the technique. Thus, the availability of a simple and accurate test for detecting gastric ulcers in horses is much needed by equine practitioners.

Sucrose permeability testing has been documented to be reliable for detecting gastric ulceration in dogs and humans [2,3]. Sucrose is not synthesized or hydrolyzed in the circulatory system; therefore, once sucrose is absorbed across the gastric mucosa, it is filtered from the blood by the kidneys, where it is then concentrated and excreted in the urine. Thus, urinary sucrose concentration after administration or ingestion of sucrose is a useful indicator of gastric ulceration.

The purpose of this study was to determine if urinary sucrose concentration in a sample collected at a specified time after sucrose administration could be used to accurately predict the presence and severity of gastric ulceration in horses.

2. Materials and Methods

Thirteen healthy horses of varying breeds were used in a cross-over design: sucrose permeability testing was performed after each horse was subjected to a feed deprivation model to induce gastric ulceration; the same horses were then evaluated after 21 days of omeprazole [a] treatment to heal any induced ulcers. For the induction of ulcers, each horse was subjected to feed deprivation: each horse was alternately fed twice daily for 24 h and then fasted for 24 h until a total of 96 h of fasting had been obtained. The beginning of sucrose permeability testing coincided with the end of the fasting period. On the morning of sucrose testing, a soft flexible stallion catheter was aseptically passed in both mares and geldings. The contents of the bladder were emptied, urine volume was recorded, and several aliquots were placed in collection tubes and frozen at -80° until analysis. Horses were then allowed to consume a meal consisting of 1 kg of concentrate. At the conclusion of the meal, 454 g of sucrose (as a 10% solution in water) was administered by nasogastric intubation. Urine was then collected 2 and 4 h after sucrose administration. All samples were frozen at -80° immediately after collection. At the conclusion of evaluation with induced ulceration, each horse was administered omeprazole at 4 mg/kg for 21 days to heal any induced ulcers. At the conclusion of omeprazole treatment, horses were again evaluated using the sucrose permeability testing protocol. After sucrose permeability testing, all horses were endoscopically evaluated to determine the severity of gastric ulceration (scored on 0 - 3 scale) [4]. Urine samples were analyzed by high-performance liquid chromatography and pulsed amperometric detection. Urinary sucrose concentrations from the 2-h collection time with and without induced gastric ulcers were analyzed using the Wilcoxon sign-rank test. A generalized linear model was used to determine the association between gastric ulcer severity score and sucrose concentration.

3. Results

The feed-deprivation model was successful in inducing gastric ulcers in 11 of 13 horses. Omeprazole treatment resulted in a significant ($P = 0.001$) decrease in ulcer severity among the 11 horses in which the feed-deprivation model was successful. Sucrose concentrations were significantly ($P = 0.001$) higher after ulcer induction in these 11 horses. Significant ($P < 0.05$) differences were observed between ulcer severity scores of 2 or 3 and ulcer severity scores of 0 or 1. Using a cut-point of 0.7 mg/ml, the apparent sensitivity and specificity of urinary sucrose testing to detect gastric ulcers with score >1 were 83% and 90%, respectively.

Differences in urinary sucrose concentration were also calculated using the ratio of urine sucrose concentration per urine osmolality for each of the 11 horses in which ulcer induction was successful. Values of this ratio 2 h after sucrose administration were significantly ($P = 0.002$) higher in horses after ulcer induction. Urinary sucrose concentrations were significantly ($P = 0.046$) lower in horses with ulcer severity scores ≤ 1 than for those with ulcer severity scores >1 .

4. Discussion

Our data indicate that sucrose permeability testing is a useful method for detecting equine gastric ulceration. Urinary sucrose concentrations were significantly higher in those horses with induced gastric ulcers compared with the same horses after 21 days of omeprazole treatment. More importantly, urinary sucrose concentrations were significantly correlated with the severity of gastric ulcers. Reliable detection of clinically important ulcers may be one of the most important findings from the study presented here.

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Footnote

[a] Gastrogard, Merial, 3239 Satellite Blvd., Building 500, Duluth, GA 30096, USA.

References

1. Andrews FM, Nadeau JA. Clinical syndromes of gastric ulceration in foals and mature horses. *Equine Vet J* 1999; 29 (Suppl):30-33.
2. Meddings JB, Kirk D, Olson ME. Noninvasive detection of nonsteroidal anti-inflammatory drug-induced gastropathy in dogs. *Am J Vet Res* 1995; 56:977-981.
3. Sutherland LR, Verhoef Marja, Wallace JL, et al. A simple, non-invasive marker of gastric damage: Sucrose permeability. *Lancet* 1994; 343:998-1000.
4. Vattistas NJ, Sifferman RL, Holste J, et al. Induction and maintenance of gastric ulceration in horses in simulated race training. *Equine Vet J* 1999; 29(Suppl):40-44.

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