Essential Fatty Acid Supplementation as a Preventative for Carbohydrate Overload-Induced Laminitis

Kelly A. Neelley, DVM and Douglas J. Herthel, DVM

Essential fatty acid supplementation prevents laminitis in horses challenged with carbohydrate overload. Possible mechanisms that would explain these findings include the known effects that essential fatty acids have on inflammation, vasoconstriction, hypertension, and coagulation in laboratory animals and man. Authors' address: Alamo Pintado Equine Medical Center, P.O. Box 249, Los Olivos, CA 93441.

1. Introduction

The onset of laminitis is a devastating disease seen in horses. Etiologies include carbohydrate overload, endometritis, endotoxemia, grass overload, and surgical complications.[1–6] Despite extensive research concerning the etiology and pathophysiology of laminitis, the mechanism of the disease is still incompletely understood.

There have been conflicting theories concerning the mechanisms of the disease and how the etiologies are possibly interrelated. Questions have been raised concerning vasoconstriction, anastomotic shunting, hypertension, endothelial damage, and mucosal damage. It is now commonly accepted that laminitis, despite the inciting factors, is a vascular disease that causes area of ischemia and necrosis within the hoof.[7–12] At present, there are no known preventatives for laminitis of alimentary origin once the carbohydrate has been ingested and digested.

Essential fatty acid (EFA) supplementation has been studied extensively in human medicine to prevent and treat cardiovascular disease, hypertension, and inflammation.[13–18] Little research to date has been conducted in the horse to examine the possible relationship between EFA supplementation and the prevention of alimentary laminitis.

The purpose of this research was to determine whether or not a diet supplemented with EFA's would affect the outcome of carbohydrate overload-induced laminitis. Because acute laminitis is most commonly diagnosed on clinical signs,[6] the parameters were based on the presence and severity or the absence of clinical signs. The other causes of laminitis were not addressed in this study.

2. Materials and Methods

Twelve mature horses that were healthy and free of clinical and radiographic signs of laminitis were used. Complete blood counts, coagulation, and serum biochemical profiles were analyzed just before carbohydrate challenge. Six horses (the principals) received 1 cup of the EFA supplement[a] orally twice daily for a minimum of 1 month before being challenged with carbohydrate and continued to receive

NOTES

the supplement throughout the experimental period. Six horses (the controls) received no supplementation.

Each horse was sedated with detomidine[b] and given the laminitis ration[c] through a nasogastric tube.[19] The full dose was administered at one time. During the following 48 h (experimental period), each horse was evaluated at 4-h intervals. Parameters measured included vital signs, feces, presence or absence of digital pulses, warmth of the hoof, and the presence, severity, or absence of lameness. Lameness severity was graded according to the guidelines determined by Obel.[1]

3. Results

All six principals showed similar trends in their coagulation profiles. Activated clotting times were increased and thrombin times were decreased. Platelet counts and all other parameters were within normal limits. The six controls showed no abnormal values.

All 12 horses in this study developed signs of gastrointestinal disturbance within 12 h of receiving the ration. Of the six principals, one horse exhibited a transient mild increase in digital pulses without lameness that dissipated without therapy. All six controls exhibited increased digital pulses, warmth over the hoof wall, and lameness (two horses had Obel grade I lameness, three horses had Obel grade II lameness, and one horse exhibited Obel grade III lameness) within 24 h. After 48 h of observation, the experiment was terminated and the controls were treated with bute.[d]

4. Discussion

Based on clinical signs, laminitis was prevented in those horses receiving the supplement. The principal horses showed alimentary signs induced by carbohydrate overload, as expected, but failed to show expected signs of lameness brought on by carbohydrate overload.

The proposed mechanisms by which EFA's prevented signs of lameness in this study include (a) decreased inflammation, (b) decreased vasoconstriction, (c) control of hypertension, and (d) effects on coagulation. Determining how EFA's prevent laminitis following carbohydrate overload is made more difficult by the fact that the mechanisms involved in the onset of carbohydrate-overload induced laminitis are still not completely understood.

5. Conclusions

To summarize briefly, the use of anti-inflammatories in the treatment of laminitis[3,4,5,9,20] and the histologic evidence that inflammation plays a major role in the pathology of laminitis[6] provide evidence that EFA's may decrease signs of laminitis by inhibiting inflammatory mediators.[16,22] EFA's are known to decrease systemic hypertension in man[14,17] and may have the same effect in horses. According to the theory that hoof blood flow increases during acute laminitis as a result of decreased vascular resistance and systemic hypertension,[10] EFA resolution of hypertension may prevent laminitis. Current research involving the use of heparin as a therapeutic modality[21] may point to the effect EFA's have on diminishing platelet aggregation. This is surmised by the fact that EFA's are known to decrease platelet aggregation in man.[13,15,17]

A conclusive determination of the mechanisms involved requires further research, including in-depth vascular studies to determine the effects of EFA's on the blood flow to the foot during a carbohydrate-overload insult.

References and Footnotes

1. Stashak TS. Lameness. In: Staskak TS, ed. *Adams' lameness in horses.* 3rd ed. Philadelphia: Lea and Febiger, 1987;486–499.
2. Colles CM, Jeffcott LB. Laminitis in the horse. *Vet Rec* 1977;100:262–264.
3. Linford RL. Laminitis (founder). In: Smith BP, ed. *Large animal internal medicine.* St. Louis: Mosby, 1990; 1158–1168.
4. Stick JA. Laminitis. In: Robinson NE, ed. *Current therapy in equine medicine.* Vol. 2. Philadelphia: Saunders, 1987;277–281.
5. Green EM, Barner HE, Sprouse RF. Laminitis. In: Colahan PT, Mayhew IG, Merritt AM, et al., eds. *Equine medicine and surgery.* 4th ed. Goleta, CA: American Veterinary, 1991;1354–1366.
6. Hood DM, Grosenbaugh DA, Mostafa MB, et al. The role of vascular mechanisms in the development of acute equine laminitis. *J Vet Intern Med* 1993;7:228–234.
7. Coffman JR, Johnson JH, Guffy MM, et al. Hoof circulation in equine laminitis. *J Am Vet Med Assoc* 1970;156:76–83.
8. Hood DM, Amoss MS, Hightower D, et al. Equine laminitis I: radioisotopic analysis of the hemodynamics of the foot during the acute disease. *J Equine Med Surg* 1978;2: 439–444.
9. Garner HE. Update on laminitis. *Vet Clin North Am [Large Anim Pract]* 1980;2:25–32.
10. Robinson NE, Scott JB, Dabney JM, et al. Digital vascular responses and permeability in equine alimentary laminitis. *Am J Vet Res* 1976;37:1171–1176.
11. Garner HE, Coffman JR, Hahn AW, et al. Equine laminitis and associated hypertension: a review. *J Am Vet Med Assoc* 1975;166:56–57.
12. Hood DM, Stephens KA. Physiopathology of equine laminitis. *Comp Cont Educ* 1981;3:s454–s460.
13. Vanhoutte PM, Shimokawa H, Boulanger C. Fish oil and the platelet-blood vessel wall interaction. *World Rev Nutr Diet* 1991;66:233–244.
14. Knapp HR. Hypostensive effects of w3 fatty acids: mechanistic aspects. *World Rev Nutr Diet* 1991;66:313–328.
15. Harris WS, Connor WE, Goodnight SH. Dietary fish oils, plasma lipids and platelets in man. *World Rev Nutr Diet* 1991;66:343–350.
16. Bonta IL, Parnham MJ. Prostaglandins, essential fatty acids, and cell-tissue interactions in immune-inflammation. *J Plt Leuk Throm* 1994;20:617–623.
17. Lorenz R, et al. Platelet function, thromboxane formation and blood pressure control during supplementation of the western diet with cod liver oil. *Circulation* 1983;67:504–511.
18. DeCaterina R, et al. Omega-3 fatty acids and endothelial

leukocyte adhesion molecules. *PGs Leuk, EFAs* 1995;52: 191–195.
19. Garner HE, Coffman JR, Hahn AW, et al. Equine laminitis of alimentary origin: an experimental model. *Am J Vet Res* 1975;36:441–444.
20. Mostafa MB. Studies on experimental laminitis in the horse. Cairo: Cairo University; 1986. Thesis.
21. Hood DM, Stephens KA, Amoss MS. Heparin as a preventative for equine laminitis: a preliminary report. *Am Assoc Equine Pract Newslett* 1982;2:142–146.
22. Moore JN, Barton MH. Effect of dietary alpha-linolenic acid on responses of horses to endotoxin. *Vet Clin Nut* 1996;3: 16–18.

[a]Platinum Performance, Platinum Nutrition, Lompoc, CA 93436.
[b]Detomidine, Pfizer Animal Health, Pfizer Inc., West Chester, PA 19380; dosage of 0.3 mg/kg IV.
[c]Special laminitis ration, Dr. D. Hood and the Hoof Project, Texas A & M College of Veterinary Medicine, College Station, TX 77843; dosage of 17.6 g/kg of body weight.
[d]Phenylbutazone, Burns Veterinary Supply, Inc., Rockville Centre, NY 11570; dosage of 2 g q 12 h IV for 3 days.