Labrador retriever breeding. Unfortunately, the breeding trials and pedigree analysis required to determine if this is a case of cerebellar abiotrophy were not possible.

Acknowledgment

We thank Shelley Ebbett for photographic assistance.

CVJ

References

1. De Lahunta A. Veterinary Neuroanatomy and Clinical Neurology, 2nd ed. Philadelphia: WB Saunders, 1983: 255–278.
2. De Lahunta A. Abiotrophy in domestic animals: a review. Can J Vet Res 1990; 54: 65–76.
3. Perille AL, Baer K, Joseph RJ, *et al.* Postnatal cerebellar cortical degeneration in Labrador Retriever puppies. Can Vet J 1991; 32: 619–621.
4. Jones TC, Hunt RD. Veterinary Pathology, 5th ed. Philadelphia: Lea & Febiger, 1983: 1657–1658.
5. Percy DH, Carmichael LE, Albert DM, *et al.* Lesions in puppies surviving infection with canine herpesvirus. Vet Pathol 1971; 8: 37–53.
6. Tatalick LM, Marks SL, Baszler TV. Cerebellar abiotrophy characterized by granular cell loss in a Brittany Spaniel. Vet Pathol 1993; 30: 385–388.
7. Steinberg HS, Troncoso JC, Cork LC, *et al.* Clinical features of inherited cerebellar degeneration in Gordon Setters. J Am Vet Med Assoc 1981; 179: 886–890.
8. Jenkins T. Functional Mammalian Neuroanatomy. Philadelphia: Lea & Febiger, 1978: 238–251.
9. Yasuba M, Okimoto K, Iida M, *et al.* Cerebellar cortical degeneration in Beagle dogs. Vet Pathol 1988; 25: 315–317.
10. Cork LC, Troncoso JC, Price DL. Canine inherited ataxia. Ann Neurol 1981; 9: 492–499.

CROSS-CANADA DISEASE REPORT

RAPPORT DES MALADIES DIAGNOSTIQUÉES AU CANADA

Ontario

Mechanobullous disease in a Belgian foal in eastern Ontario

A mechanobullous disease with a suspected inherited etiology has been reported in Belgian foals in the United States (1). Affected foals develop bullae of the skin, and mucosal and mucocutaneous membranes that progress to irregular and asymmetrical erosions and ulcers following minor trauma. Corneal ulcers and dystrophic teeth are also described. Lesions may be present at birth or develop in the neonate. Death results from secondary bacterial infections and reduced feed intake due to oral lesions. Anecdotal reports of this disease have existed in Ontario for several years.

In May 1992, formalin-fixed tissues from a 7-day-old, purebred, female, Belgian foal were submitted to the Kemptville Regional Veterinary Laboratory, Ontario Ministry of Agriculture, Food and Rural Affairs, for histological evaluation. The foal had been normal at birth and had nursed aggressively. By 2 d of age, large irregular erosions and ulcers of the skin over pressure points were observed; similar defects appeared at the oral mucocutaneous junction, and on the tongue, hard palate and mucosa over the erupted incisor teeth. Ulcers of the coronary band were present on both front feet, resulting in sloughing of 1 hoof wall by 6 d of age. A clinical diagnosis of mechanobullous disease of Belgian foals was made and the foal was euthanized at 7 d of age.

Histopathology of the skin was characterized by subepidermal clefts. The basal cell layer was intact and attached to the epidermis, and there was intracellular edema of rare basal cells. Simple cleft formation was unaccompanied by inflammation; where this had progressed to epidermal sloughing and ulceration, the dermal surface consisted of granulation tissue covered with fibrin, neutrophils, and bacteria. At the periphery of ulcers, there was marked, irregular, epidermal hyperplasia; fibrin and neutrophils from the ulcer extended into the adjacent exposed subepidermal clefts, accompanied by an intense superficial dermal perivascular neutrophilic infiltrate. Lesions of the buccal, gingival, and glossal mucosae and the mucocutaneous junction were similar to those in the skin.

In a published study (2), electron microscopic examination has shown that separation of the dermoepidermal junction occurs through the lamina lucida with destruction of basilar hemodesmosomes. This and other ultrastructural morphology was interpreted by the authors of that study as being similar to human epidermolysis bullosa of the junctional type. An autosomal recessive mode of inheritance is suspected.

The histopathology of this case is being reported because anecdotal reports of mechanobullous disease of Belgian foals in eastern Ontario are rarely substantiated by laboratory tests; clinical signs are usually considered diagnostic.

References

1. Kohn CW, Johnson GC, Garry F, Johnson CW, Martin S. Mechanobullous disease in two Belgian foals. Equine Vet J 1989; 21: 297–301.
2. Johnson GC, Kohn CW, Johnson CW, Garry F, Scott D, Martin S. Ultrastructure of junctional epidermolysis bullosa in Belgian foals. J Comp Pathol 1988; 98: 329–336.

Janet Shapiro, *Ontario Ministry of Agriculture, Food and Rural Affairs, Veterinary Laboratory Services, Mail Bag 2005, Kemptville, Ontario K0G 1J0, and* ***Beverly McEwen,*** *Ontario Ministry of Agriculture, Food and Rural Affairs, Veterinary Laboratory Services, Box 3612, Guelph, Ontario N1H 6R8.*