Pathogenic Studies of Bovine Progressive Degenerative Myeloencephalopathy (Weaver) of Brown Swiss Cattle

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Introduction

Bovine progressive myeloencephalopathy (weaver) of purebred Brown Swiss cattle has been described fairly recently in the United States.[4-6, 8-10] This disease has also been reported recently from Canada, Denmark, and Switzerland.[1-3,7] This paper presents further findings in the ongoing study of this disease.

Materials and Methods

Cattle affected with bovine progressive degenerative myeloencephalopathy are part of our studies into the nature, cause, and effect of congenital defects in cattle, the methods of which have been outlined previously.[5,6] The condition occurred in purebred Brown Swiss cattle in various parts of the United States. Affected cattle were transported to the Congenital Defects Laboratory in Kansas State University for further clinical and pathological studies. All cattle were euthanitized with T-61 intravenously via jugular vein. Brain, spinal cord, and other tissue samples were removed within 30 minutes of euthanasia and placed into 10% buffered neutral formalin for further processing. Slides were routinely stained with hematoxylin and eosin and various special stains were used.

Results

Clinical Signs

Bovine progressive degenerative myeloencephalopathy of purebred Brown Swiss cattle is a progressive disease with clinical signs implicating the central nervous system. Four basic criteria were used to establish a clinical diagnosis:

1. Onset of bilateral hind leg weakness and ataxia between 5 and 8 months of age;
2. Deficient proprioceptive reflexes, normal motor and sensory reflexes, with no other clinically detectable neurological abnormality;
3. Absence of clinically significant skeletal or muscular abnormality; and
4. Adherence to familial relationship.

Bovine progressive degenerative myeloencephalopathy involved both sexes. Clinical signs were hind leg weakness, ataxia, and dysmetria with an onset of 5 to 8 months of age, progressing to eventual recumbency and frequently death from rumen tympany. These clinical signs were present without other neurological or musculoskeletal abnormalities. Finally, the animals became recumbent and died or had to be destroyed. A deficit of unconscious proprioception was postulated as the cause of the hind leg signs.

Pathologic Lesions

There were no gross lesions typical for weavers. Primary microscopic lesions were confined to the central nervous system. Spinal cord lesions in H&E stained sections consisted of axonal degeneration, including spheroid formation, loss of axons and myelin, and vacuolation of white matter. Vacuoles were usually devoid of stainable material; however, a few contained foamy glitter cells. Luxol fast blue-cresyl echt violet-stained section confirmed loss of myelin. Lesions were qualitatively similar at all spinal cord levels, but quantitatively dissimilar at different levels in the same funiculi. Both ascending and descending fibers were involved. In all cases, lesions were most severe in thoracic spinal cord segments. Little or no glial response, no inflammatory response, and no involvement of gray matter were observed in the spinal cord sections. Cerebellar lesions were limited to selective degeneration and loss of Purkinje cells and occasional swelling of Purkinje cell axons (torpedoes) in the granular layer of the cerebellar cortex. Lesions were inconsistent or absent in other parts of the brain.

There were lesions in the reproductive system of females and males. Ovaries were smaller than normal and frequently had multiple or single, small and large fluid filled cysts. Testicles were small, soft and flabby. Histologic examination disclosed testicular atrophy and cessation of sperm production.

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Epidemiological studies failed to indicate any specific geographical or environmental factors. The incidence of recorded cases has increased greatly during the past 8 years. Breeding records indicated that there were was a familial pattern to the occurrence of this disease.

Discussion

Congenital defects are defined as abnormalities of structure, formation, and function present at birth. Bovine progressive degenerative myeloencephalopathy falls into the category of formation. In its clinical signs and pathologic lesions it is quite different from other known genetic CNS problems in cattle.[6] Weaver in purebred Brown Swiss cattle may be a congenital metabolic defect of axon transport that appears to be inherited as a simple autosomal recessive trait.

Bovine progressive degenerative myeloencephalopathy shares many features with heredofamilial central nervous diseases in man involving primarily spinal cord, cerebellum and/or brainstem include motorneuron disease, neuroaxonal dystrophy and spinocerebellar degeneration. Thus, bovine progressive degenerative myeloencephalopathy may also serve as a biomedical model for the study of similar disease processes in man.

Summary

Clinical signs of bovine progressive degenerative myeloencephalopathy ("weaver") in Brown Swiss cattle, appear when calves are 6 to 8 months old and progress during the next 12 to 18 months to recumbency. Four basic criteria for diagnosis are: 1. onset of bilateral hind leg weakness and ataxia between 5 and 8 months of age; 2. deficient proprioceptive reflexes, normal motor and sensory reflexes, and no other clinically detectable neurologic abnormality; 3. absence of clinically significant skeletal or muscular abnormality; and 4. adherence to familial relationship.

There are no gross lesions typical for weavers, and primary microscopic lesions are confined to the central nervous system. Spinal cord lesions consisted of axonal degeneration including spheroid formation, loss of axons and myelin, and vacuolation of white matter due to large, empty intercellular spaces. Lesions were qualitatively similar at all spinal cord levels but quantitatively dissimilar at different levels in the same funiculi. Cerebellar lesions were limited to selective degeneration and loss of Purkinje cells and occasional swelling of Purkinje cell axons in the granular layer of the cerebellar cortex. Progressive degenerative myeloencephalopathy is the single most important genetic disease of purebred Brown Swiss cattle and may be an inherited congenital metabolic defect of axon transport.

References

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