

Cerebellar Disease of Ruminants



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KEYWORDS

- Cerebellar hypoplasia • Cerebellar abiotrophy • Lysosomal storage disease

KEY POINTS

- The cerebellum functions to regulate and coordinate motor activity.
- Clinical signs of cerebellar disease include hypermetric gait in all limbs, normal to increased muscle tone, wide-based stance, swaying (truncal ataxia), and intention tremor. Vestibular signs may be observed.
- Cerebellar hypoplasia is most commonly caused by viral disease, and animals are affected from birth.
- Cerebellar abiotrophy is most often heritable, and offspring are typically born normal and develop neurologic signs later in life.
- Plant toxicities and congenital lysosomal storage disorders are differentials for cerebellar disease.

INTRODUCTION AND ANATOMY OF THE CEREBELLUM

The cerebellum is composed of several distinct areas. Knowledge of the anatomy is important when attempting to localize a lesion in the cerebellum. There is a central median region (also called the vermis) with lateral hemispheres on either side. The flocculonodular lobe (the vestibular cerebellum) is located on the ventral, central aspect of the cerebellum. Three cerebellar peduncles attach the cerebellum to the brainstem. The peduncles attach in a caudal to rostral position: the spinal cord and medulla, the pons, and the mesencephalon, respectively. The cerebellar medulla is composed of white matter and surrounded by the cerebellar cortex. The cerebellum receives afferent input from the cerebral cortex and brain stem, vestibular centers, and spino-cerebellar pathways. Efferent pathways connect to the brain stem and cerebrocortical areas via Purkinje fibers.¹ The primary function of the cerebellum is to regulate and coordinate motor activity.^{1,2}

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CLINICAL PRESENTATION OF CEREBELLAR DISEASE

Clinical signs of cerebellar disease vary depending on the location of the disease process within the cerebellum, although diffuse disease is common. Clinical signs characteristic of cerebellar disease include bilaterally symmetric ataxia without paresis and with normal to increased muscle tone. Where there are unilateral lesions, ataxia will be seen on the ipsilateral side to the lesion. Classically the animal will display a hypermetric gait in all 4 limbs, whereby voluntary movement is exaggerated. The animal may stand with a wide-based stance and sway from side to side while ambulating (truncal ataxia). If the cerebellar cortex is affected, a head intention tremor is observed. The intention tremor may cease when the animal is recumbent and muscles are relaxed.³ In severe disease, the animal may be in lateral recumbency and unable to right itself and display opisthotonus (where the rostral lobe is involved). When performing a neurologic examination, postural reactions may be delayed and then exaggerated. Nystagmus may be present, with variable directions observed when the head is moved.² Abnormal menace reaction may also be present¹ with the head moving away from the threatening gesture, but no blink reflex is observed. Neurologic signs may be enhanced if the animal is suddenly released from restraint, which may be observed as cerebellar convulsions.¹ If the flocculonodular lobe is affected, vestibular signs may be apparent, including head tilt and leaning and falling to the contralateral side of the lesion.¹

DISEASES OF THE CEREBELLUM

Differentiating Cerebellar Abiotrophy and Hypoplasia

Cerebellar abiotrophy occurs because of a variety of familial diseases. Typically, the animal will be born neurologically normal, with the clinical signs developing at weeks to months of age. The cerebellum may be small on necropsy in abiotrophy cases; however, typically it is normal in size. A small cerebellum is found grossly with cerebellar hypoplasia. In abiotrophy cases, the Purkinje cells in the cerebellar cortex are degenerate.⁴ In hypoplasia cases, animals are affected from birth, most commonly as a result of viral disease contracted during gestation. Ratio of the cerebellum to whole brain weight can confirm hypoplasia.⁴

Viral-Induced Cerebellar Disease

Cerebellar hypoplasia has been reported in cases of experimental⁵ and natural exposure of pregnant cattle to bovine viral diarrhea virus (BVDV).⁶ Congenital abnormalities of BVDV (both cerebellar hypoplasia and ocular abnormalities) occur following infection of the cow at 125 to 180 days in gestation. Administration of a modified live BVDV vaccine induced teratogenesis during 90 to 118 days' gestation.⁷ Clinical signs of cerebellar disease may be variable in affected calves, from mild to severe.⁸ Cerebellar hypoplasia has been recognized as part of the congenital defects associated with the sheep *Pestivirus* (border disease virus)⁹ and experimental infection of ewes with BVDV virus.¹⁰ Precolostral serum of calves born with congenital cerebellar infection as a result of BVD had BVD antibody¹¹ and testing calves before consumption of colostrum may be helpful in the diagnosis of congenital BVD infection.

Schmallenberg virus was first recognized in Northern Europe in 2011 when malformed calves, lambs, and kids were born. Gross lesions recognized included porencephaly, hydraencephaly, cerebellar dysplasia, dysplasia of the brainstem and spinal cord, brachygnathia inferior, arthrogryposis, and vertebral column malformations. Some cases also showed encephalomyelitis. Ninety-five percent of lambs and 45% of calves affected in the Netherlands had cerebellar dysplasia of varying degrees.¹² Microscopically, the cerebellar cortex was affected with loss of Purkinje cells and

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