

Cerebellar Coenurosis with Hind Limb Paralysis in Sheep and Goats in Mongolia

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Abstract: Cerebellar coenurosis is caused by *Coenurus cerebralis*, the larval stage of *Taenia multiceps*, particularly in goats and sheep. In this case report, we describe gross and histopathological characteristics of cerebellar coenurosis in two goats and one sheep in Mongolia. In March 2017, animals showing ataxia and hind limb paralysis were referred to the Laboratory of Veterinary Pathology, Institute of Veterinary Medicine, Ulaanbaatar Mongolia. At necropsy, fluid-filled coenurus cysts were detected exclusively in the cerebellum of all the animals. Histological examination of the affected cerebellar hemispheres revealed necrosis, granulomatous inflammation and diffuse gliosis at the periphery of the cysts. Based on the results of this study, cerebellar coenurosis should be considered a differential diagnosis in cases with neurologic signs such as ataxia and hind limb paralysis, in Mongolia.

Key words: cerebellar ataxia, coenurosis, goat, Mongolia, sheep

Introduction

Coenurosis, which is generally observed in small ruminants such as sheep and goats, is a zoonotic parasitic disease caused by *Coenurus cerebralis*, the larval stage of *Taenia multiceps*, which live in the small intestines of dogs and other canids⁹⁻¹¹. The larvae (*Coenurus cerebralis*) usually affect the cerebrum of the animals and develop into transparent cysts, and the animals present neurological signs such as circling, head pressing, convulsions, paresis, teeth grinding, blindness, and coma. The majority of the

affected cases die from starvation after some weeks from the initial neurological signs¹⁶. Clinical symptoms may be suggestive of the disease, yet definitive diagnosis is made histopathologically, based on the detection of parasitic cysts in the brain¹³. While 80–90% of the cysts are localized in the cerebral hemispheres, 5–10% may be in the cerebellum¹¹.

Animals affected with cerebellar coenurosis have been reported to display incapacitating ataxia, hind limb paresis, inability to stand, swaying, and falling. These signs are typical of cerebellar dysfunction caused by direct compression of the cerebellum by parasitic cysts^{4, 7, 14}.

In past years, cerebellar ataxia induced by plant poisoning have been reported in sheep and goats in Mongolia. The dominant histological feature of the affected animals was

marked Purkinje cell loss¹⁵). There has been no report describing cerebral pathology of sheep and goats affected with cerebellar coenurosis in Mongolia. The purpose of this present study was to demonstrate the pathological findings of sheep and goats affected with cerebellar coenurosis compared with cerebellar diseases, for the differential diagnosis of sick animals showing cerebellar ataxia, in Mongolia.

Materials and Methods

In March 2017, a 12-month-old, Romanov breed sheep (Fig. 1) and two local breed of Cashmere goats aged 3 and 12 months (Fig. 2) with chronic neurologic signs including ataxia, hind limb paralysis, convulsion and culminating in recumbency were referred to the Laboratory of Veterinary Pathology, Institute of Veterinary Medicine, Ulaanbaatar Mongolia. These animals were herded in steppe grasslands, which were near to human settlements where local domestic dogs were observed. At necropsy, fluid-filled coenurus cysts were detected in the cerebellum of all the animals.

Cerebrum and cerebellum tissues were fixed in 10% neutral phosphate buffered formalin and embedded in paraffin. Three-micrometer-thick paraffin sections were cut and stained with hematoxylin and eosin (HE). Immunohistochemistry of selected sections from the cerebellum was performed by using previously reported techniques⁶). Briefly, rabbit polyclonal antibody to glial fibrillary acidic protein (GFAP) (1:100 dilution in phosphate buffered saline (PBS)) (DAKO, Glostrup, Denmark) was used as a primary antibody. After incubation with primary antibody over night at 4°C, the sections were placed in a solution containing a peroxidase-labeled polymer conjugated to a secondary anti-rabbit antibody [EnVision + kit / HRP (DAB), (DAKO)] for 30 min at room temperature. Positive reactions resulted in brown staining with diaminobenzidine, which were then counter-stained with hematoxylin.

Results and Discussion

At necropsy, a 1.5 to 2.0-cm-diameter, fluid-filled cyst was found at the bottom of the cerebellum of the 12-month-old sheep and in the cerebellum of the goats. At the cut sec-

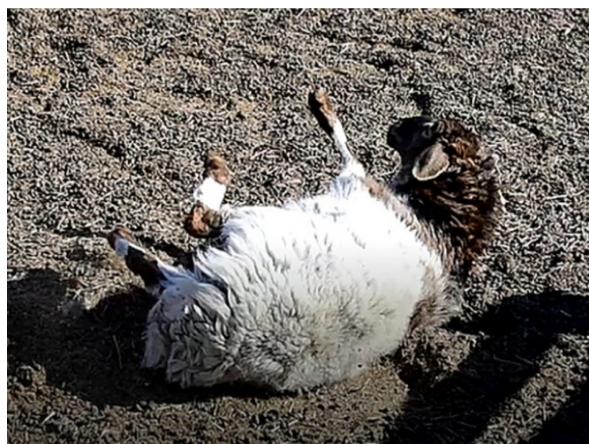


Fig. 1. A 12-month-old sheep showing hind limb paralysis and convulsions.



Fig. 2. A 12-month-old goat showing hind limb paralysis.

tion, in addition to the superficial cyst, a deep compartment with white clusters of scolices was seen in the cerebellar hemisphere, which caused severe pressure atrophy of the cerebellum and brain stem (Fig. 3). The two goats also showed similar coenurus cysts containing white clusters of scolices in the cerebellar hemisphere (Fig. 4). Histological examination of the affected cerebellar hemispheres and brain stem revealed necrosis of cerebellar and brain stem parenchyma, granulomatous inflammation characterized by the formation of the multinucleated giant cells, gliosis, mononuclear cell infiltration and perivascular cuffing at the periphery of the cysts (Fig. 5a); GFAP-positive astrocytic fibers, which surrounded the necrotic and granulomatous lesions, were demonstrated (Fig. 5b). These histologic

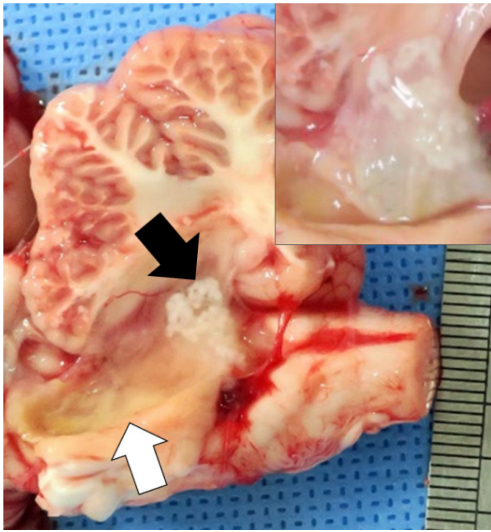


Fig. 3. Coenurus cyst (white arrow) at the bottom of the cerebellum and a large number of scolices as white clusters (black arrow, inset) attached to the cyst wall in the 12-month-old sheep.

changes were common in all the cystic changes observed in the three animals examined in this study.

Parasitological examination of 256 goats from all provinces of Mongolia, during 1994 to 1999, demonstrated high prevalence and intensity of helminth infections including infection from cestode larvae of *Taenia multiceps*¹²⁾. A short communication describing clinical and macroscopic findings of a Mongolian goat affected with spinal coenurosis was reported¹⁷⁾. There are, however, no report describing the cerebral pathology of sheep and goats affected with coenurosis in Mongolia. In this study, typical clinical, macroscopic, and microscopic findings of cerebellar coenurosis were observed. Cerebellar ataxia and coenurus cysts surrounded by necrotic lesions of the brain parenchyma in the cerebellum shown in this study concurred with the findings in previous reports on coenurosis in small ruminants⁹⁻¹¹⁾.

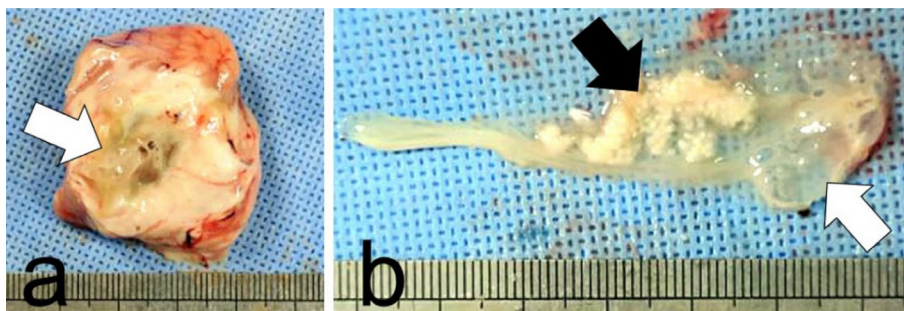


Fig. 4. Coenurus cyst (a: white arrow) (b: white arrow) in the cerebellum and a large number of scolices as white clusters (b: black arrow) attached to the cyst wall in the 12-month-old goat.

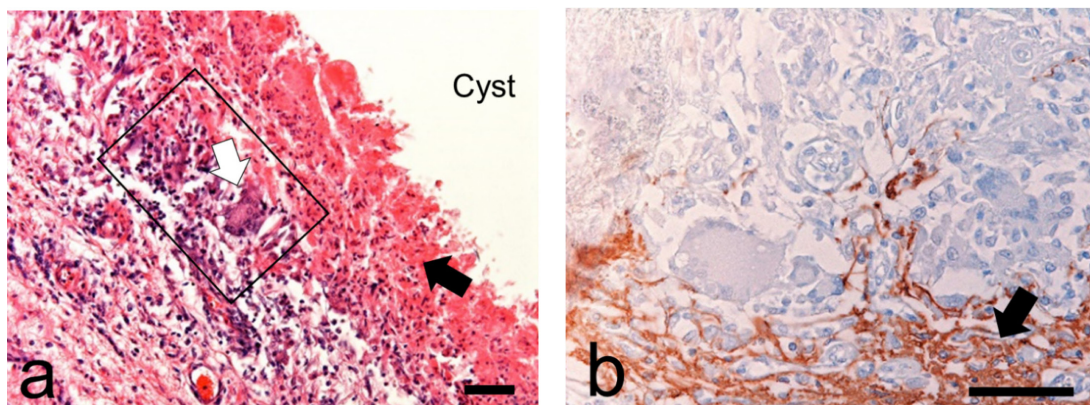


Fig. 5a. Histology of the wall of the coenurus cyst in the cerebellum of the 12-month-old sheep, showing necrosis (black arrow) of cerebellar parenchyma, granulomatous inflammation with giant cells (white arrow), mononuclear cell infiltration and perivascular cuffing. HE stain. Bar = 100 µm.

Fig. 5b. Higher magnification of 5a (rectangle) stained with GFAP immunohistochemistry, showing marked astrocytic gliosis (black arrow) surrounding the granulomatous lesions. Bar = 50 µm.

Cerebral coenurosis is a very common parasitic encephalopathy of the sheep in many countries and the cerebellar form of the disease is not uncommon. *Coenurus cerebralis* cysts in sheep showed a predilection for the cerebral hemispheres according to most of the reported cases^{1,7,14}. While 80–90% of the cysts are localized in the cerebral hemispheres, 5–10% may be located in the cerebellum¹¹. In a study on *Coenurus cerebralis* infection of the sheep, cysts were seen in the cerebral hemisphere in 96% of the cases (43% and 57% for left and right, respectively) and only 4% in the cerebellum. As to the mechanisms of predilection of the coenurus cyst location in the affected animals, association of different genotypes of the parasites⁸ and host-parasite relationship¹⁷ has been suggested. The prediction of cyst locations based on the direction of circling and head deviation had a 62% success rate¹. In this study, all the animals examined had cystic lesions exclusively in the cerebellum. The neurologic signs were ataxia, incoordination, and hind limb paralysis as described in the cerebellar coenurosis literature^{2,4,5,7,14}.

In the last few years, cerebellar ataxia induced by ingestion of *Oxytropis glabra*, a toxic plant containing swainsonine, an indolizidine alkaloid of endophyte origin, has been reported in sheep and goats in Mongolia¹⁵. Severe drought associated with global warming resulted in vegetation degradation, limiting forage choices for animals and increasing animal poisoning incidents from poisonous plants³. Although no gross pathological changes were observed in a variety of organs including the central nervous system of the poisoned animals, microscopic examination of the cerebellum demonstrated degenerative changes, such as vacuolar changes and loss of Purkinje cells, torpedo formation in the granular layer, increased number of spheroids in the cerebellar medulla, and loss of axons and myelin sheaths of Purkinje cells¹⁵. On the other hand, necrosis of the brain parenchyma induced by the parasitic cysts, granulomatous inflammatory changes and surrounding astrocytic gliosis are the characteristic findings of the brain of animals affected with coenurosis, as demonstrated in this study¹³. Based on the results of this study and other related studies in Mongolia, cerebellar coenurosis should be considered as a differential diagnosis in cases showing cerebellar dysfunction.

Environmental contamination with cestode eggs from dogs is expected in steppe grasslands in Mongolia¹². Affected animals in this study were herded in the steppe grasslands, which were near to human settlements where local domestic dogs were observed. In order to control coenurosis, it is important to treat the tapeworm-infected dogs with anthelmintic and to dispose infected animal tissues effectively to prevent access of dogs to the larval stages, and thereby perpetuating the cycle of the parasite infection.

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