Diagnostic Exercise

From The Davis-Thompson Foundation*

Case #: **181** Month: **January** Year: **2022**
Answer Sheet

Tittle: *Bovine coenurosis*

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Clinical History: An 18-month-old mixed breed steer had incoordination, blindness, and opisthotonos for ten days. The clinical condition progressed to lethargy and lateral recumbency, and the steer was euthanized due to poor prognosis.

Necropsy Findings: There is a cyst located mainly on the left side of the brain, moderately distending the third ventricle and protruding through the transverse fissure into the right dorsal thalamic region anterior to the rostral colliculi). The cyst is 3.3 x 3.5 x 3 cm, consists of a thin transparent membrane filled with translucent fluid, and has numerous slightly elongated opaque white structures of approximately 1 mm (protoscolices) adhered to the inner aspect of the capsule (Figure 1). The cyst causes compression and atrophy of the thalamus and hippocampus, more pronounced on the left side (Figure 2). There is dilation of the lateral ventricles, moderate on the left side and mild on the right side. The mesencephalic aqueduct is moderately distended.

Macroscopic Images:

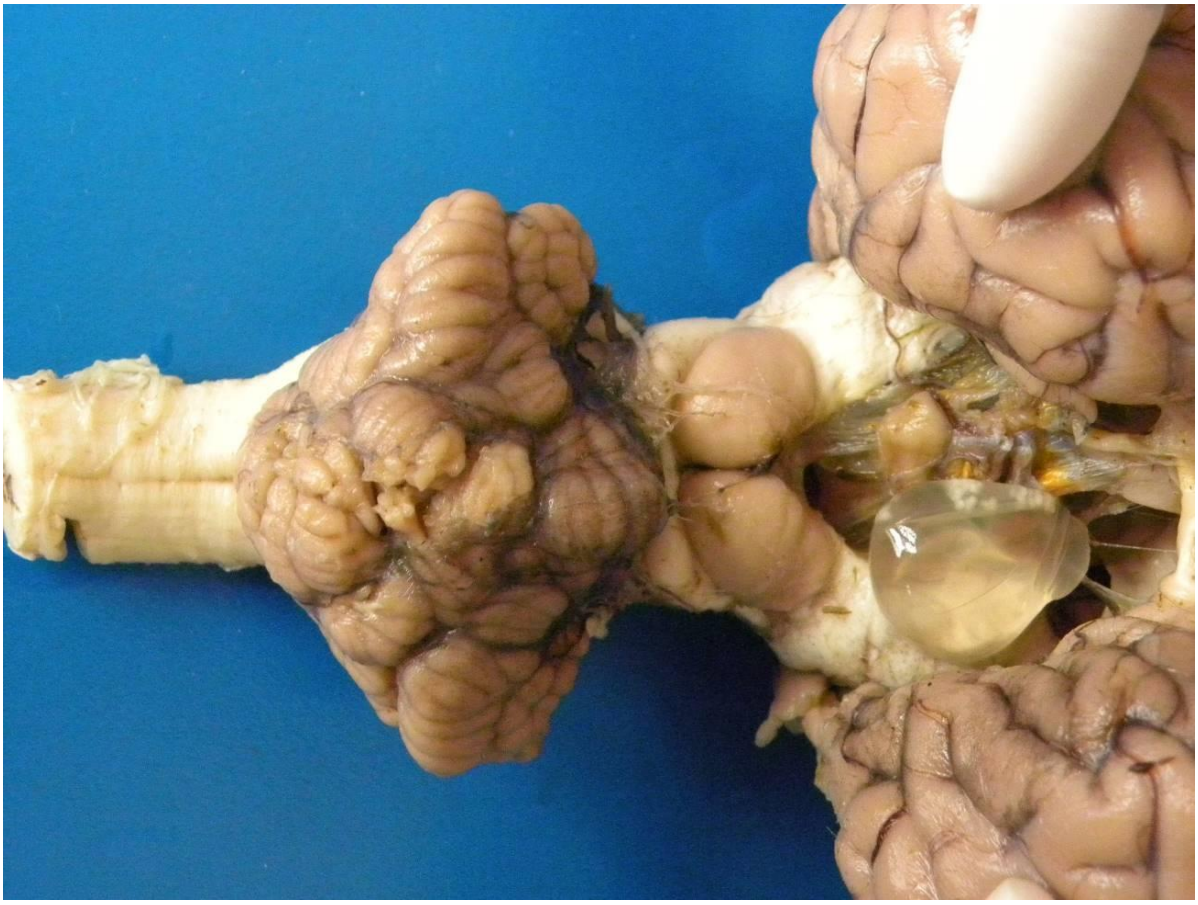

Figure 1. Bovine brain (formalin-fixed specimen). This is a closer view of the brain from the previous illustration. Part of a cyst protrudes into the right side of the thalamus, anteriorly to the rostral colliculi. The cyst has a thin wall and, in its upper portion, has two clusters of opaque white protoscolices of approximately 1 mm in diameter each.

Figure 2. Bovine brain (formalin-fixed specimen). A large cyst distends the third ventricle, compressing the left side of the thalamus and hippocampus and leading to hydrocephalus of the third and lateral ventricles. The cyst contents lost their translucency due to the accumulation of exudate, yellow pigment (hemosiderin), and cellular debris.

Microscopic findings

The cyst was lined by an eosinophilic (bladder) membrane with an external dense layer and an internal loose layer (Figure 3). Numerous scolices ranging from 100 nm to 1000 mm in diameter protruded from the internal aspect of the cyst wall. They lacked a body cavity and intestinal tract, and had a tegument and parenchyma containing abundant deeply basophilic, granular material (calcareous corpuscles) (Figure 4). There were multifocal infoldings of the tegument. The rostellum, suckers, and hooks were evident in multiple sections. Lymphocytes, plasma cells, eosinophils, macrophages, and multinucleated giant cells (foreign body and Langhans types) infiltrated the leptomeninges and neuroparenchyma adjacent to the cyst (Figure 5).

Macrophages often contained brown, granular, cytoplasmic pigment (hemosiderin). There were multiple foci of hemorrhage within the leptomeninges and accumulation of cellular debris, fibrin, and mineral. The neuroparenchyma was multifocally hypercellular due to hyperplasia and hypertrophy of astrocytes and microglial cells and had occasional pale lacy areas (edema).

Microscopic Images:

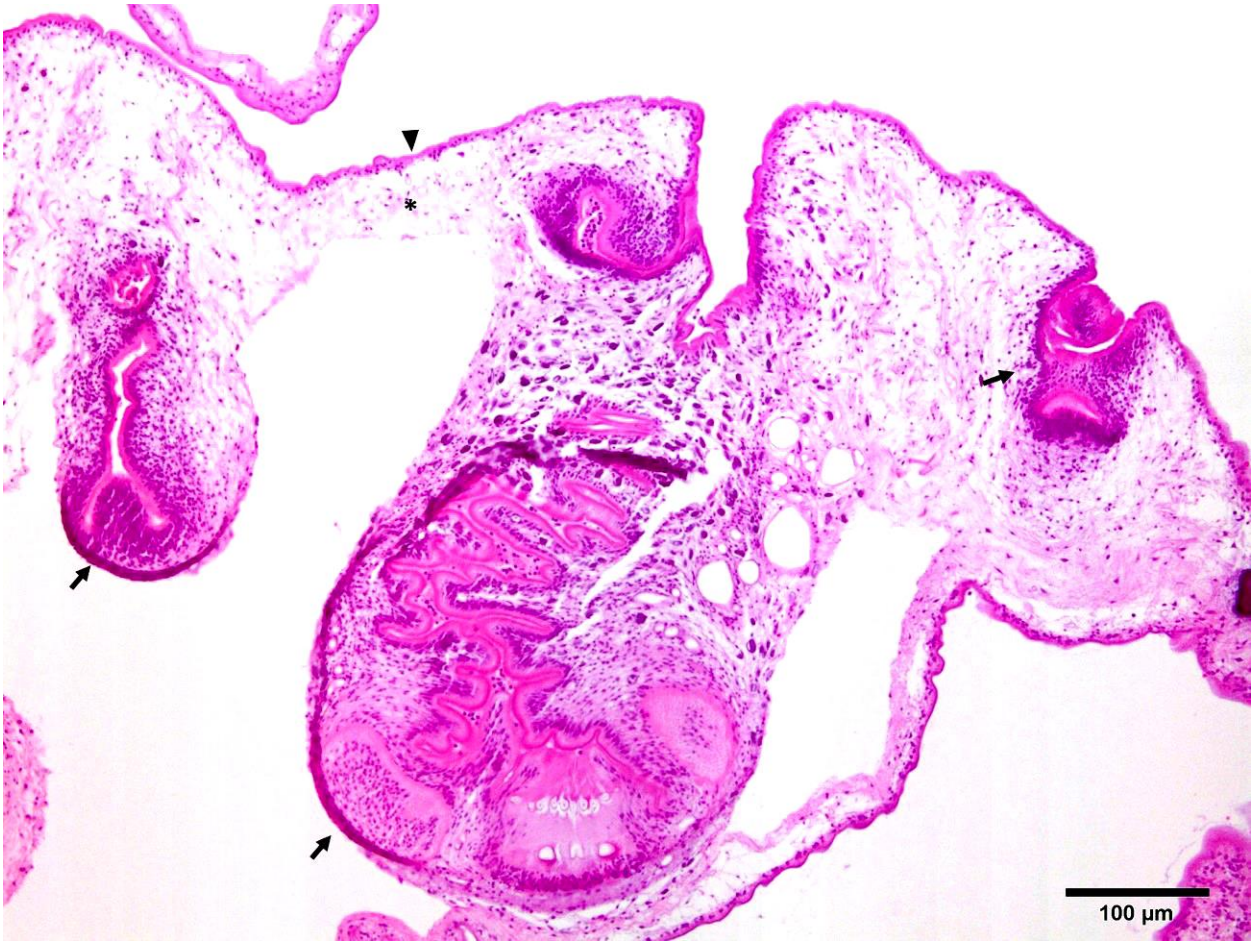

Figure 3. Multiple scolices (arrows) invaginate from the bladder membrane. The membrane is composed of an outer, dense, eosinophilic layer (arrowhead) and an inner loose layer (asterisk).

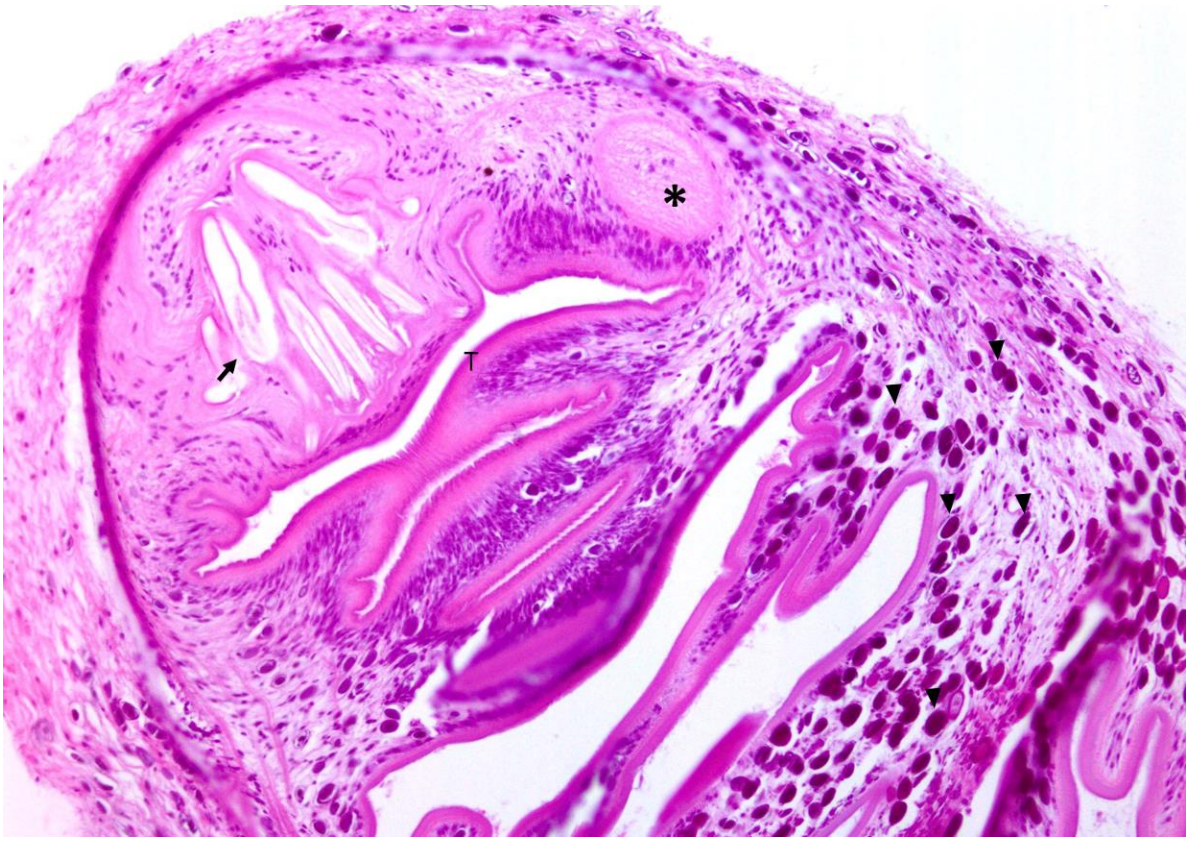

Figure 4. A scolex with infoldings of the tegument (T) and lack of a body cavity and digestive tract. The parenchyma contains numerous, deeply basophilic, 5-15 μm calcareous corpuscles (arrowheads). The rostellum contains suckers (asterisk) and hooks (arrow).

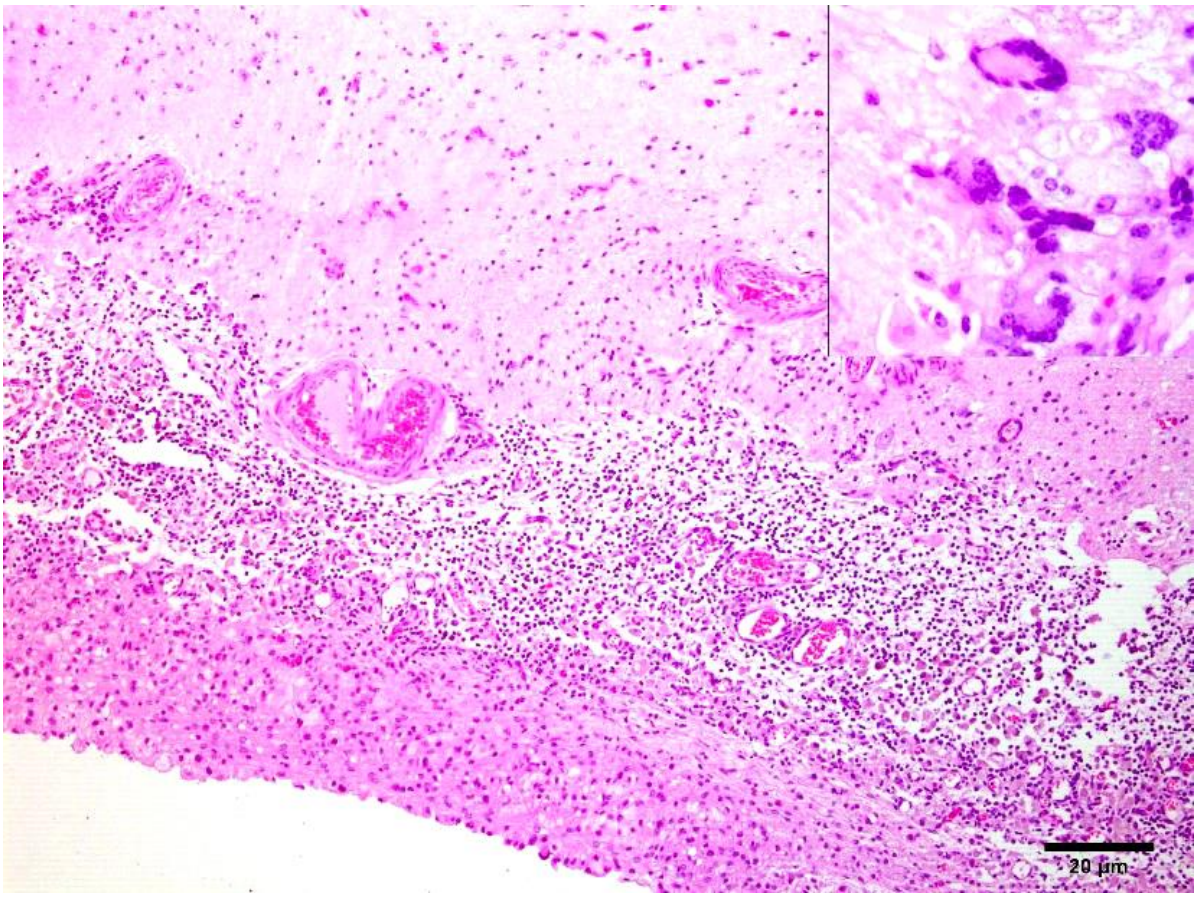

Figure 5. The leptomeninges are expanded by an inflammatory infiltrate consisting of macrophages, lymphocytes, plasma cells, eosinophils, and multinucleated giant cells (inset).

• **Morphologic diagnosis**: Brain, third ventricle, parasitic cyst accompanied by moderate, locally extensive, lymphoplasmacytic and granulomatous meningoencephalitis.

• **Etiology**: *Coenurus cerebralis* (larval stage of *Taenia multiceps*)

• **Name of the condition**: Bovine coenurosis

Comments:

The clinical signs and gross and histopathological findings are consistent with the diagnosis of coenurosis in the current case. The acoelomate parasites are characteristic of the Class Cestoda or Trematoda. The calcareous corpuscles in the parenchyma and the absence of a digestive tract allow the classification of the organism as a cestode (Taylor et al. 2007). The hooks are characteristic of a member of the Taeniidae family. Among the genera of metacestodes in this family, *Coenurus*

sp. is the one that has numerous scolices and does not have a broad capsule, which differentiates it from the hydatid cyst (Taylor et al. 2007). The location within the nervous system and the morphological aspects are characteristic of the metacestode larvae of *T. multiceps*.

Coenurosis is colloquially known by various sobriquets like gid, sturdy, goggle-turn, giddy-turning, and water-brain (Innes and Saunders 1962). *C. cerebralis* is the larval stage (metacestode) of *Taenia multiceps* (Class Cestoda, Family Taeniidae), which affects commonly sheep (Rissi et al. 2008) and less so other species like goats (Nourani and Kheirabadi 2009), cattle (Greig and Holmes 1977, Giadinis et al 2007, Avcioglu et al 2012), deer, antelopes, chamois, gazelle, camels, rabbits, hares, and rarely horses (Innes and Saunders 1962) and man (Schellhas and Norris 1985). Intermediate hosts are the dog and other carnivores, like foxes and jackals (Innes and Saunders 1962). In Southern Brazil, the disease is common in sheep and known as "torneio" (Portuguese for "turning-sickness").

Infection occurs when the intermediate herbivore host ingests the pasture contaminated with embryonated eggs and/or gravid proglottids of *T. multiceps* shed in the feces of the definitive carnivore host. Each egg contains an oncosphere. The hatching of the eggs releases the oncosphere in the small intestine of the intermediate host. The oncosphere is then activated, penetrates the intestinal mucosa, and reaches the central nervous system through the bloodstream. There, each oncosphere evolves into the larval stage (metacestodes) of *T. multiceps* called *Coenurus cerebralis*. A mature *C. cerebralis* is a translucent cyst of 5 cm or more filled with serous fluid containing clusters of dozens of protoscolices attached to the internal aspect of the cyst membrane. The life cycle concludes when the final host, a canid, ingests the brain or spinal cord of an affected intermediate host (Taylor et al. 2007).

Neurologic signs depend on (i) the effects produced by the entry of the larvae (acute phase) in the central nervous system and (ii) on the space-occupying effects of the fully-developed cyst (chronic stage).

Acute disease is uncommon and occurs mainly in sheep grazed on pasture heavily contaminated with feces of infected dogs. The onset of the acute stage is approximately 10-14 days after the cerebral invasion when some (~20%) of affected

animals, which develop the chronic disease later, may show signs of acute meningoencephalitis (Innes and Saunders 1962).

The signs in the chronic stage start 2-4 months after infection and include incoordination, high stepping gait, blindness, head tilt, stumbling, and paralysis depending on the location of the parasitic cyst in the brain or in the spinal cord. If the parasitic cyst occurs in the spinal cord, there will be unilateral or bilateral paralysis caudal to the segment of the cord involved (Innes and Saunders 1962).

The lesions in sheep in the acute phase are suppurative meningoencephalitis, mostly in the telencephalon, at the base of the brain adjacent to the optic chiasm, and in the interpeduncular fossa (Innes and Saunders 1962). At cut surface, tracts of malacia due to migration of oncospheres may be present (Taylor et al. 2007).

In a review of 16 cases of coenurosis in sheep (Rissi et al. 2008), we found *Coenurus* sp. cysts in the telencephalon (12/16), in the cerebellum (3/16), and in the spinal cord (3/16). In all cases, the lesions corresponded to the chronic phase, with the cysts compressing and displacing the adjacent nervous tissue. Histologically, parasitic cysts found in the 16 sheep (Rissi et al. 2008) had a faint eosinophilic double membrane from which protrude multiple spherical protoscolices. Adjacent to the vesicle wall, successive layers existed, composed of a zone of necrosis and mineralization, an intermediate zone of epithelioid macrophages and multinucleated giant cells, and an external capsule of fibrovascular tissue infiltrated by histiocytes, lymphocytes, and plasma cells. The cysts caused compression atrophy of the adjacent white and gray matter (Rissi et al. 2008).

Coenurosis in cattle is uncommon, but where livestock has access to grazing pastures contaminated by dog feces, cattle have the risk of developing the metacestode stage. One factor that may have facilitated the infection in the present case was that cattle stayed in crowded small paddocks to which dogs had access during the night.

Morbidity in cattle is about 1-2% (Ferreira et al. 1992, Giadinis et al. 2007), and the progression of clinical signs extends over six months or more (Ferreira et al. 1992); however, they are generally shorter due to euthanasia early in the disease process (Giadinis et al. 2007), which was the case with the steer of this report. Generally, young cattle between 6 months and two years of age are affected, and clinical signs include circling, dysmetria, and blindness (Ferreira et al. 1992, Giadinis

et al. 2007), although cattle without clinical signs may show the lesions of coenurosis at postmortem examination in slaughterhouses (Avcioglu et al. 2012). Gross and microscopic features are similar to those described for sheep.

The disease is challenging to diagnose clinically, and diseases such as listeriosis, oestrosis, and louping ill should be on the list of differential diagnoses in sheep. Several neurological diseases such as rabies, bovine necrotizing herpesviral meningoencephalitis, and babesiosis may also be included as differential diagnoses when cattle are affected, although these are acute diseases. Bovine spongiform encephalopathy is a chronic neurological debilitating disease and may need to be consider when cattle older than 24 months are affected.

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