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### Case Report

## Regurgitations in a Lamb with Acute Coenurosis- A case Report

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Received 21 Feb 2015  
Accepted 11 Apr 2015

#### **Keywords:**

Sheep,  
Acute coenurosis,  
Regurgitation,  
*Coenurus cerebralis*,  
Neurological disease

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#### **Abstract**

Coenurosis is a disease of the central nervous system in sheep, caused by *Coenurus cerebralis*, the larval stage of *Multiceps multiceps*, which inhabits the small intestine of Canidae. A case of regurgitations in a 2.5 month old lamb with acute coenurosis is being reported. The lamb was presented with a sudden onset of ataxia and regurgitations for 10 days. The post-mortem examination revealed 4 immature *C. cerebralis* cysts between 0.5 and 1.5 cm in diameter located in the brainstem and cerebellum, and histopathological examination revealed multifocal pyogranulomatous meningoencephalitis, so a diagnosis of acute coenurosis was established. Thus, acute coenurosis should be included in the differential diagnosis of regurgitations in lambs.

### Introduction

Coenurosis is a fatal disease of the central nervous system mainly affecting sheep, but it has also been reported in goats, cattle, horses, pigs, and hu-

mans. Coenurosis is caused by *Coenurus cerebralis*, the larval stage of *Multiceps multiceps* (*Taenia multiceps*) which inhabits the small intestine of dogs and wild Canidae, who serve as defini-

tive host. The intermediate hosts become infected after consuming contaminated feedstuffs with *M. multiceps* eggs (1).

The disease can be presented in two clinical forms, as chronic or acute coenurosis. Chronic coenurosis is the most common form, and is usually observed in young, 6–18 months old sheep (2). The clinical manifestations are those of a space-occupying lesion and depend on the localization of the developed *Coenurus* cyst. Acute coenurosis is produced by the penetration and migration of the oncospheres in the brain. The clinical signs and the gravity of acute form are correlated with the number of ingested eggs, the immune status of the host, the migrating route localization of parasites in the brain, and the intensity of inflammatory response (3). Symptoms may include depression or excitability, ataxia, scoliosis, dysmetria, muscle tremors, blindness, nystagmus, and coma (4, 5). Pyrexia is not a consistent finding. Death without prior symptoms has also been reported (6- 8). Symptoms are caused by an acute inflammatory response due to toxic and allergic reaction rather than by mechanical action of the migrating larvae (3). We report a case of regurgitations as the main clinical finding in a lamb with acute coenurosis.

### **Case History**

A 2.5 month-old male Lacaune lamb was presented to the Farm Animal Clinic of Aristotle University in Thessaloniki with ataxia and regurgitations. According to the owner the symptoms appeared suddenly 10 days earlier. The flock from which the lamb originated had a history of acute coenurosis, as this had been previously diagnosed.

### **Clinico-pathological findings**

On clinical examination, the lamb was depressed, with slightly decreased appetite and ataxic gait, while its rectal temperature was normal. Both nostrils were soiled by dark greenish dried discharge. On closer examination, the discharge was identified as dried ingested food, due to the frequent regurgitations

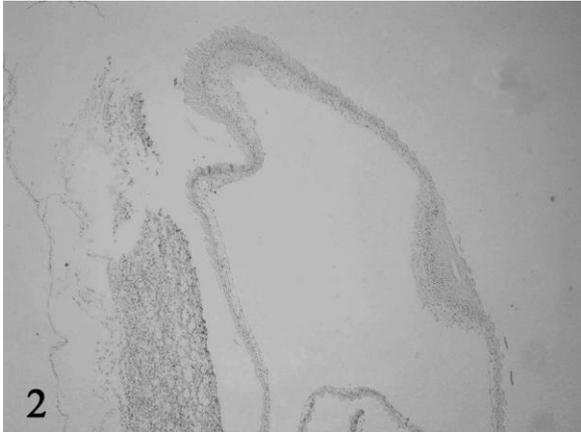
(Fig. 1). Hematological and serum biochemical examination revealed no abnormalities compared to normal values (9, 10). The lamb was kept in the clinic for further observation. In the following days its clinical condition worsened and as the prognosis was poor, the lamb was euthanized for welfare reasons.



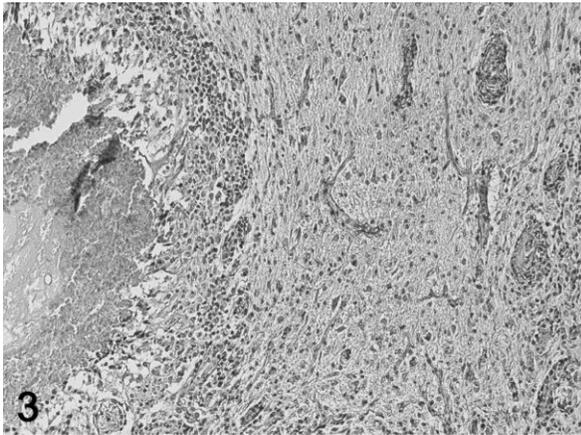
**Fig. 1:** Lamb with depression and dark greenish discharge as the result of regurgitations

A detailed post-mortem examination was performed. Gross examination revealed the presence of 4 fluid filled cystic formations between 0.5-1.5 cm in diameter with translucent walls, one located on the surface of the cerebellum and the others within the brainstem parenchyma, surrounded by a thin layer of purulent yellowish exudates, which was also present as a layer covering part of the brainstem surface. Hyperemia of the leptomeninges was also evident. The findings were limited to the brain during gross examination, as no other lesions were evident. The four stomachs and esophagus appeared normal, and no aspiration pneumonia was observed. Brain tissue samples were obtained and fixed in 10% buffered neutral formalin for histopathological examination. Evaluation of the brain sections revealed foci of cavitations accompanied by compression of adjacent parenchyma (interpreted as malacic lesions by the migratory larvae). Each cavity was surrounded by cellular debris and numerous macrophages, fewer

lymphocytes and plasma cells and a peripheral rim of congested blood vessels. In one cavity the thick eosinophilic tegument of a cestode larva (*Coenurus*) was found (Fig. 2).



**Fig. 2:** Detail of the eosinophilic tegument of a larva, (haematoxylin-eosin staining, magnification x 200)



**Fig. 3:** Part of a granuloma (left) with central necrosis surrounded by numerous macrophages, neutrophils, lymphocytes and plasma cells, and multifocal perivascular cuffs in the adjacent parenchyma (haematoxylin-eosin staining, magnification x 200)

There were also multiple granulomas consisting of central dark eosinophilic amorphous material that was surrounded by multinucleated giant cells, degenerated and non-degenerated neutrophils, lymphocytes and

plasma cells (Fig. 3). In the white matter, perivascular cuffs of lymphocytes and plasma cells, gliosis and formation of glial nodules were detected. Based on the above mentioned findings the morphological diagnosis was pyogranulomatous meningoencephalitis, multi-focal, severe with necrosis, compression and coenuri and lymphocytic perivascular cuffing. The cysts were examined in the Laboratory of Parasitology and Parasitic Diseases, Faculty of Veterinary Medicine, Aristotle University and the findings were as reported by Giadinis et al. (7). Due to the gross and microscopic lesions documented in this case, acute coenurosis diagnosis was established.

## Discussion

Due to the lack of lesions concerning the esophagus, regurgitation of the lamb was attributed to the meningoencephalitis caused by acute coenurosis. This could be explained by the severe lesions observed in the brainstem, where nucleus ambiguus and parasympathetic nucleus of the vagus nerve are located, controlling esophageal motility. From the former the glossopharyngeal nerve (IX), the general somatic efferent axons of the vagus nerve (X) and the accessory nerve (XI) originate and from the latter the general visceral efferent preganglionic neurons of the vagus nerve (X) originate (11). Branches of the glossopharyngeal and the vagus nerve innervate the striated muscles of the esophagus. Another hypothesis is that these nerves were affected by the inflammation of the meninges caused in this case by the larvae migration (12), as both of the cranial nerves (IX, X) travel through the subarachnoid space and exit the cranial cavity through the jugular foramen (11). Vomiting in a lamb with acute coenurosis was reported in 1982, at the 20<sup>th</sup> day post experimental infection with 2000 *T. multiceps* eggs, but this report did not focus on this clinical finding, either excluded other possible causes of vomiting (4). Nevertheless, vomiting and regurgitation is

differentiated as a clinical manifestation (13, 14).

Regurgitation in small ruminants is a rare phenomenon. It is defined as the expulsion through the mouth or nostrils of feed, saliva and other substances after they have been swallowed, but before reaching the forestomachs (13, 14). Braun et al. (13) reported regurgitation in a ram with megaesophagus that was attributed to myo-neural disturbance due to severe inflammation caused by infestation with sarcosporidia (*Sarcocystis* spp.). Some serotypes of Bluetongue virus were found to cause regurgitation in sheep (15- 17) and cattle (17), due to damage of the esophageal musculature resulting to esophageal paresis. Regurgitation has also been reported as a clinical sign of Scrapie in sheep and goats (18). Various plant toxicoses are manifested with regurgitation or vomiting when consumed by ruminants, such as *Geigeria* spp. causing vermeersiekte (19). Physical stenosis of the esophagus may cause regurgitation, resulting from obstruction of the lumen associated with feed particles or foreign bodies, tumors, esophagitis, even masses of healing tissue after esophageal rupture (20). Compression of the esophagus leading to stenosis may result from mediastinal masses which include abscesses, enlarged mediastinal lymph nodes, caseous lymphadenitis (21), mediastinal tumors such as thymoma and mediastinal haematomas (22). Diverticulum of the thoracic region of the esophagus (23), hiatal hernia, congenital and acquired idiopathic megaesophagus, are disorders associated with regurgitation in cattle. Milk fever (14, 24) and botulism type B may cause regurgitation in cattle as well (25). Van Biervliet et al. (12) reported otitis media/interna and spontaneous regurgitation in three calves, with evidence of meningitis in two of them. In a retrospective study of megaesophagus in 15 llamas, organophosphate poisoning was identified as the causative agent in one case (26).

## Conclusion

Acute coenurosis should be included in the differential diagnosis of regurgitation in sheep.

## Acknowledgements

The authors declare that there is no conflict of interests.

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