Clinical and morphopathological characteristics of an enzootic occurrence of acute coenurosis (*Coenurus cerebralis*) in a sheep herd

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Abstract In this study, 30 sheep from a flock suddenly showed acute neurological symptoms associated with more than 30 % mortality. At necropsy, thickening associated with congestion and turbidity of meningeal membranes particularly on cerebellum, focal to multifocal necrotic areas and whitish spots measuring 1 to 3 cm in diameter were observed in the cortex of cerebrum and cerebellum. Grossly, numerous white tracts were also observed in the myocardium. Histopathologically, the cross sections of coenurus larvae associated with necrotic suppurative meningoencephalitis were observed. Multiple necrotic areas were also observed in the gray matter of cerebellum due to migration of the larvae with an extensive infiltration of eosinophils and neutrophils. In the heart, multifocal granulomatous myocarditis was diagnosed. No growth was seen in bacterial cultures of the brain and heart. Also, no bacteria were seen in these tissues stained with Ziehl–Neelsen and Brown–Brenn Gram stain. On basis of gross and histopathologic lesions, acute coenurosis was diagnosed. Unlike chronic coenurosis, acute form of the disease rarely reported in sheep.

Keywords *Coenurus cerebralis* · Sheep · Histopathology · Acute coenurosis

Introduction

Coenurus cerebralis is the intermediary larval stage of *Taenia multiceps*: a tapeworm which is found in the small intestine of dogs and wild canids (Scott 2000). Sheep are considered to be the animal species most susceptible (Ozmen et al. 2005; Scala et al. 2007). Ovine coenurosis is found in Africa and Asia, with prevalence ranging from 1.3 to 9.8 % (Achenef et al. 1999; Sharma and Chauhan 2006). *C. cerebralis* inhabits the central nervous system and causing a disease known as “coenurosis”, “gid” or “sturdy” (Abo-Shehada et al. 2002). *C. cerebralis* reaches the central nervous system hematogenously and it usually affects the young animals (Ghazaei 2005). Two clinical forms of coenurosis have been described in sheep, the most common is the chronic form (Komnenou et al. 2000; Acha and Szyfres 2003) whilst its acute form is more rare (Scott 2007). The acute form is caused by the migration of the immature parasites in the CNS (Achenef et al. 1999; Komnenou et al. 2000; Scala and Varcasia 2006; Scott 2007).

Symptoms vary depending on the cyst’s location, size, and compression the brain (Sharma and Chauhan 2006; Christodouloupoulos 2007; Gul et al. 2007). This study describes clinical, morphological and pathological characteristics of an enzootic occurrence of acute coenurosis in a sheep herd in Iran.

Materials and Methods

In autumn 2012, three young male sheep about 3–6 months old from the feedlot Iranian crossbred herd were referred to the Department of Clinical Studies, School of Veterinary Medicine, Shiraz University, Iran, because of a syndrome of acute neurological dysfunction with increased mortality.

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The client claimed that he had lost 10 sheep with similar signs during last 2 weeks and other remained sheep are at risk. Physical examinations of the affected lambs showed tachypnea, tachycardia, and absence of menace reflex with presence of direct pupillary light reflex indicated central blindness. Ophthalmoscopic examination revealed moderate congestion of retinal venules. The CSF obtained from lumbosacral region, was examined grossly and microscopically, and also cultured microbiologically. Cases were referred to the Department of Pathology and necropsy was performed. Appropriate tissue samples were taken from the heart and brain, fixed in 10 % neutral buffered formalin, processed and embedded in paraffin. Sections of 5 μ m were cut, stained with Hematoxylin-Eosin (H & E), Ziehl-Neelsen and Brown-Brenn Gram stain, and studied microscopically. Similar tissue samples were submitted for bacteriological examination. A follow-up study in other sheep of the herd showed most of animals were affected so that they referred to the slaughterhouse and on basis of similar morphopathological lesions in the brain, acute coenurosis was confirmed.

Results

The affected sheep showed typical clinical neurological signs such as depression, incoordination, circling behavior, visual defects, teeth grinding and poor appetite. Absence of menace reflex with presence of direct pupillary light reflex indicated central blindness. The CSF examination showed inflammatory cells with predominant eosinophils (eosinophils/lymphocytes ratio was 70:30). The CSF proteins concentration was slightly increased. No bacterial growth occurred on microbiologic culture.

Gross examination of brains revealed the thickening, congestion and turbidity of meningeal membranes. Focal to multifocal necrotic areas were distributed in the cortex of cerebrum and cerebellum. Whitish spots measuring 1–3 cm in diameter were observed on the surface and in the cortex of the brain. Cut surface of formalin-fixed brains showed multiple pores sizes 1–2 cm due to migration of the parasite larva (Fig. 1). Grossly, numerous white tracts were observed in the myocardium. No growth was seen in bacterial cultures of the brain and heart.

Microscopic examination of H&E stained brains showed the cross sections of coenurus larvae associated with hemorrhage and necrosis (Fig. 2). Necrotic suppurative meningoencephalitis with multiple necrotic areas was observed in the gray matter of cerebellum due to migration of the larvae with an extensive infiltration of eosinophils and neutrophils. Congestion, hemorrhage, fibrinous exudates, neuronal necrosis, perivascular cuffing (PVC), and fibrin thrombi in two vessels were also observed.

Fig. 1 Brain: cut surface of formalin-fixed brains showed multiple pores sizes 1–2 cm due to migration of the parasite larva (arrows)

Fig. 2 Brain: cross section of coenurus larvae (arrow) associated with hemorrhage and necrosis of the brain around the cyst. (H&E stain $\times 80$)

Consequences of migration of coenurus larvae in the brain, liquefactive necrosis associated with thin walled larvae and infiltration of inflammatory cells, PVC, hemorrhage and necrosis were observed (Fig. 3). Liquefactive necrosis with mild to moderate infiltration of the inflammatory cells such as eosinophils, lymphocytes, macrophages and neutrophils were seen in the cerebellar cortex. In the heart, multifocal granulomatous myocarditis may be due to calcified coenurus larvae in the center of granuloma were seen. The granuloma is surrounded by inflammatory cells included eosinophils, lymphocytes, macrophages, epithelioid macrophages and giant cells (Fig. 4). Numerous eosinophils were scattered between the myofibers and around the Purkinje fibers. No bacteria were seen in the brain and heart tissues stained with Ziehl-Neelsen and Gram stain.

Discussion

In this study, acute coenurosis as a syndrome of acute neurological dysfunction with increased mortality was observed in a feedlot Iranian crossbred herd sheep in Fars province. The acute form of coenurosis is rare with involvement of young animals usually ages between 1 and 10 months old. The disease occurs due to migration of the immature parasites in the CNS in the first few weeks after ingestion of the parasite eggs (Edwards and Herbert 1982; Achenef et al. 1999; Scala and Varcasia 2006).

Clinical signs in *C. cerebralis* vary depending on the location of the cyst (Sharma and Chauhan 2006; Christodouloupoulos 2007). Reported clinical signs in affected sheep are included: dullness, depression, deviation of the head to one side, often loud bleating and reduced appetite to complete anorexia, circling, incoordination, a high-stepping gait and blindness, torticollis, head tilt towards the side of the cyst location, uncontrolled movements, ataxia, teeth grinding, salivation, paresis and convulsions (Yoshino and Momotani 1988; Nooruddin et al. 1996; Khodakaram-Tafti et al. 1997; Sharma and Chauhan 2006). Impaired vision or blindness has been reported as one of the clinical symptoms of coenurosis (Yoshino and Momotani 1988; Moghaddar et al. 1992). In our study, clinical signs of affected animals were similar to other reports.

In this study, CSF showed eosinophilia with eosinophil/lymphocyte ratio 70:30 and increase of the proteins amount. This large range of cell values reinforces the point that CSF collection findings with clinical examination could be indicative of a parasitic infection. Ghosh et al. (1998) reported lowered erythrocyte count, hemoglobin, packed cell volume but increased eosinophils in coenurosis, which

Fig. 3 Brain: consequences of migration of coenurus larvae in the brain. liquefactive necrosis associated with thin walled larvae (small arrow) and infiltration of inflammatory cells, PVC, hemorrhage and necrosis (large arrow). (H&E stain $\times 200$)

Fig. 4 Heart: granulomatous myocarditis may be due to calcified coenurus larvae in the center of granuloma (arrow). The granuloma is surrounded by inflammatory cells included eosinophils, lymphocytes, macrophages, epithelioid macrophages and giant cells. (H&E stain $\times 80$)

returned to normal values after surgical removal of the coenurus cysts. Altintas et al. (1997) reported significant increase in CSF proteins in lambs infected with *C. cerebralis*. Similar to our study, Scala and Varcasia (2006) reported that the meninges particularly that covering the cerebellum were congested associated with turbidity and focal to multifocal distribution of necrotic areas.

Major features of the microscopic lesions of coenurosis in CNS of sheep and goats are purulent meningoencephalitis, congestion, liquefactive necrosis, neuronophagia, focal haemorrhage, demyelination, perivascular cuffing, necrosis and gliosis leading to formation of microglial nodules (Gogoi et al. 1991; Khodakaram-Tafti et al. 1997; Giadinis et al. 2012). In the present study, in addition to purulent meningoencephalitis, other acute lesions such as liquefactive necrosis associated with migratory thin walled cystic larvae and infiltration of inflammatory cells including eosinophils, lymphocytes and macrophages were observed. Yoshino and Momotani (1988) described eosinophilic infiltration with palisading histiocytes, caseation and calcification around the cyst in the left lateral ventricle of an affected calf. The CNS lesions of chronic coenurosis are reported by a number of researchers (Biyikoglu and Doganay 1998; Oruc and Ulsu 2006; Desouky et al. 2011). Similar to the present study, in some reports the molecular layer of the cerebellar folia showed spongiosis, degeneration and necrosis of purkinje cells, with eosinophilic infiltration (Mouchira 2010; Desouky et al. 2011). Thrombosis of vessels by parasitic elements, inflammation and parasitic toxins has also been implicated in brain necrosis (Mohi El-Din 2010).

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