

Diagnostic Methods and Economic Impact Assessment of Coenurosis in Sheep and Goats: Review

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Abstract: Coenurosis (gid or sturdy), a fatal disease of sheep and goats, is caused by the larval stage of *Taenia multiceps*. The cystic larvae (*Coenurus cerebralis*) develop in the brain and spinal cord of sheep, goats and sometimes cattle and have also been reported in human and horses. Coenurosis causes a serious problem in sheep and goats production. The pathology and pathogenesis of coenurosis is followed by loads of cysts in vital organs. The life cycle can be interrupted most satisfactorily by control of tape worm infection in dogs and preventing dogs having access to sheep carcasses. In physical examination, neurological symptoms like rotating around itself, leaning the head to the left side, incoordination and ataxia are detected. The clinical signs are variable and may be confused with other nervous conditions. Therefore, confirmatory diagnostic methods like; haematological, biochemical, postmortem findings and histopathological examinations are used for identification of coenurosis. Since clinical sign is showed after the development of irreversible lesions in organs, especially brain tissues, the disease causes great economical losses. Control and prevention of coenurosis diseases approached mainly by identifying dogs from sheep and goats pens and through farmer education.

Key words: Coenurosis • Diagnosis • Economic • Ethiopia • Goats • Sheep

INTRODUCTION

Ethiopia is a home for many livestock species and for production of livestock. As estimate indicates that the country is the home for about 25.5 million of sheep and 24.06 million of goats [1]. About 99.8% of the sheep and nearly all goat populations of the country are local breeds [2]. *Coenurus cerebralis* is distributed worldwide especially, most common in the developing countries of Africa and Asia where sheep and goat rearing is a common source of income [3-6].

Coenurus cerebralis (*C. cerebralis*) is the metacestode or larval form of the dog tapeworm *Taenia multiceps* (*T. multiceps*), which causes coenurosis also known as gid or sturdy [7]. The larval stage of *T. multiceps* is mainly found in the brain and in some instances in the spinal cord of sheep and goats. The cysts of *T. multiceps* may be present elsewhere in the brain and spinal cord, protruding into the cerebral ventricles, but they often found near the surface of parietal cerebral

cortex. The predilection sites of *C. cerebralis* are in the cerebral hemispheres of the brain, especially in the subarachnoid space, which facilitates the nourishment of the cysts by cerebro-spinal fluid (CSF) of the brain [8]. *C. cerebralis* is a commonly occurring parasitic disease that affects ruminants, horses, pigs, canines and human beings [9, 10].

The adult parasite of *C. cerebralis* is found in small intestines of the definitive host (domestic dogs and wild canids such as coyotes, foxes and jackals). The adult parasite tapeworm of dogs reaches maturity after 40-42 days. The life cycle of *C. cerebralis* starts when the definitive host, mainly dogs, starts to disseminate the gravid proglottids which contain almost 37,000 eggs that are released from proglottids with the feces. Eggs then contaminate the environment and water which resists for 15 days under dry environmental conditions or 30 days with high level of humidity [11]. The intermediate hosts, sheep and goats, are infected with ingesting the eggs deposited in the grass or water. In the small intestines of

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sheep and goats the eggs hatch to the oncospheres (embryos) which then penetrate the wall of small intestine and circulate in blood to lodge in the brain of sheep and goats [12]. The circulations of this parasite between intermediate-host species of animals and environment would result in transition of disease and causes reduction in production and reproductions of sheep and goats.

Therefore; the objectives of this review were to:

- Describe the distributions of Coenurosis parasites of sheep and goats in Ethiopia;
- Thrash-out the different diagnostic methods for identification of coenurosis and
- Highlights the economic significance of coenurosis disease in the country.

Literature Review

Actiology and Host Species of Coenurosis: Coenurosis is a parasitic disease of the central nervous system. It is caused by a tapeworm (cestode) called *Taeniamulticeps* (*T. multiceps*), which lives relatively benignly in the definitive canine host (including dogs, foxes, jackals and coyote) but causes significant disease in the intermediate host, where the larval stage of the tapeworm migrates to the brain and spinal cord and matures into a fluid-filled cyst. Sheep are the main intermediate host but there have been rare cases reported in cattle, pigs, deer, horses and humans. Dogs and other canines such as foxes, coyotes and jackals are the definitive hosts of the tapeworm *T. multiceps*. Canine hosts shed tapeworm eggs in their feces which contaminates the pasture for the intermediate host to ingest. *T. multiceps* infection on a farm is significant as it confirms an unbroken sheep and dog life cycle, which in turn implies the existence of more important tapeworms such as *Echinococcusgranulosus*. This dog/sheep tapeworm usually infects sheep and forms cysts in the lungs and liver, which if consumed by humans will cause a very serious disease that is very difficult to treat [13].

Biomorphology: Morphologically, *C. cerebralis* reaches full development in the brain of sheep and goats 6-8 months and can grow to a size of 5 cm or more with cysts containing a considerable amount of fluid and germinal epithelium with 500-700 scolices distributed in the non-linear groups [14]. The study reported at necropsy, a 4-cm-diameter, fluid-filled and superficial cyst with white clusters of scolices was found in the right cerebrum. At the cut section, in addition to the superficial cyst, a

deep compartment was seen in the right cerebral hemisphere that caused severe pressure atrophy of cerebral grey and white matter. A narrow opening was located between these compartments. There were 300 scolices within the superficial compartment of the cyst and none in the deep compartment [15].

The cyst seems like round or oval, large and bladder like. They measured approximately 3-4cm in diameter and filled with large amount of fluid. The scolex showed four cap-shaped suckers and a rostellum armed with 18-34 typical taeniid hooks arranged in double rows (large and small) [16].

Host-Specification and life-cycle of Coenurosis:

Coenurosis is a disease settled into small intestines of dogs caused by *C. cerebralis* which is larvae of the taenia called *T. multiceps* and seen commonly in goats and sheep worldwide and affects central nervous system [7, 17]. The disease, apart from sheep and goats, is seen in cattle, horses, pigs and humans [18]. Dogs play an important role in that they are final host and spread eggs with feces. At the end of taking the herbs contaminated with intermediate hosts, oncospheres set free in small intestines of intermediate hosts are carried into brain and spinal cord. 6 to 8 months after the intake of eggs, a transparent and delicate coenurus cyst with liquid content forms in these organs [19]. At the end of taking those tissues containing coenurus cyst by dogs and other wild carnivorous, adult taenia occur in dogs' intestines and in this manner life cycle of the parasite becomes complete [7].

The life cycle of *T. multiceps* follows the same intermediate-host and definitive-host life cycle like that of coenurosis cysts. The intermediate host is infected through ingestion of *T. multiceps* eggs. Each egg contains an onchosphere which hatches and is activated in the small intestine. The onchosphere penetrates the mucosa and is carried via the blood stream to the brain or spinal cord. In goats, the cysts can form in subcutaneous and muscular sites as well as the brain and spinal cord. The onchosphere develops into a metacestode larval stage called *Coenurosiscerebralis*. The *Coenurosiscerebralis* matures into a thin-walled fluid-filled cyst about 5cm in diameter. The life cycle is complete when the canine eats the raw infected brain, spinal cord or offal contaminated by the fluid from the ruptured cyst. The scolex (head of the tapeworm) embeds itself into the wall of the small intestine where it begins to grow and shed new eggs [20].

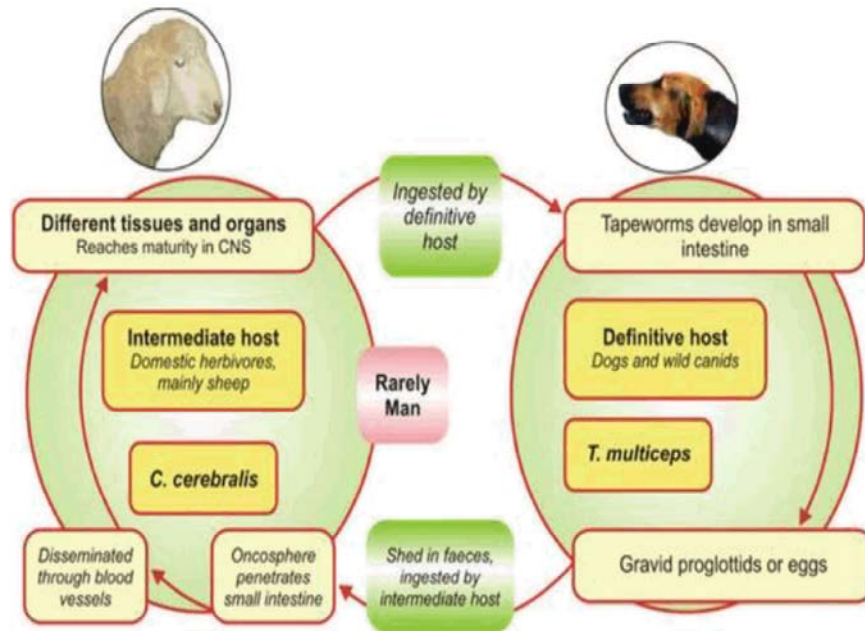


Fig. 1: *Coenurus cerebralis* life cycle involving intermediate and definitive host. Source [21].

Table 1: Localization of *Coenurus cerebralis* cysts in the brain of sheep and goats [2].

Animal examined	No. examined	No. positive	Localization of the cyst in the brain				χ ²	P-value
			RCH	LCH	MF	CRL		
Sheep	384	19	8	9	1	1	3.840	<0.001
Goats	384	45	9	29	6	1		
Total	768	64	17	38	7	2		

RCH= Right side cerebral hemisphere; LCH= Left side cerebral hemisphere
 MF= Median fissure of the cerebrum; CRL= Cerebellum

Pathogenesis and Clinical Sign: Normally *Coenurus* cyst remains limited with brain and spinal cord (Table 1). In addition to this, it's seen in muscles and subcutaneous tissues of sheep [22, 23]. It takes months that the cyst occurred in brain reaches the size that can cause clinical findings. A completely developed *coenurus* cyst may have 5-6 cm width and lead to the increased intracranial pressure resulted in ataxia, hypermetric, blindness, head deviation, headache, stumble and paralysis [13,19, 24]. When cysts occur in spinal cord, hindlimb ataxia, paresis and paralysis develop [25]. The cysts in the muscles lead to muscle pain or functional impairment of organs. However, animals remain normal without showing clinical signs in most cases and this situation is diagnosed after the death of animal [7].

The pathogenic effects of *C. cerebralis* are that of the space occupying lesion and the pressure applied to the brain by the cyst during its development. The clinical signs depend on the size and sites of cyst in the brain.

Affected sheep and goats may become blind in one or both eyes and indifferent to feed and water which results in emaciation and death of the animals [26, 27].

Coenurosis can occur in both an acute and a chronic disease form. Acute *coenurosis* occurs during the migratory phase of the disease, usually about 10 days after the ingestion of large numbers of tapeworm eggs. Young lambs aged 6-8 weeks are most likely to show signs of acute disease. The signs are associated with an inflammatory and allergic reaction. There is transient pyrexia and relatively mild neurological signs such as listlessness and a slight head aversion. Occasionally the signs are more severe and the animal may develop encephalitis, convulse and die within 4–5 days [28]. Acute disease is an important differential diagnosis for Cerebrocortical necrosis (CCN). Chronic *coenurosis* typically occurs in sheep of 16-18 months of age. The time taken for the larvae to hatch, migrate and grow large enough to present nervous dysfunction varies from 2 to

6 months. The earliest signs are often behavioral, with the affected animal tending to stand apart from the flock and react slowly to external stimuli. As the cyst grows the clinical signs progress to depression, unilateral blindness, circling, altered head position, in-coordination, paralysis [29] and recumbency. Unless treated surgically, the animal will die [28].

Epidemiological Status of Coenurosis: It is fairly uncommon, but seen in certain geographical areas. The real prevalence of coenurosis is difficult to assess, because farmers and vets often diagnose the disease and send the animal for slaughter without confirmation or report. A large proportion of infected lambs may also be sold fat before clinical signs have developed [13]. Infection by the larval stage of the tapeworm *T. multiceps* in small ruminants is common in worldwide [26]. It has been documented in scattered foci throughout the world, including the Americas and parts of Europe and is distributed in the worldwide [3, 7]. In Africa, the disease (coenurosis) has been documented in Ethiopia, Ghana, Mozambique, Uganda, Egypt, Democratic Republic of Congo, Senegal, Sudan, Chad, Angola, Kenya and Southern Africa [30]. It has been reported that 2.9% sheep in Jordan [3], 18.65 % in Uramia abattoir, Iran [31], 14.8 % in Tete municipal abattoir, Mozambique [32], 44.4 % Ngorongoro district, Tanzania [30] and 3.1–28.5% in Kars Province of Turkey [4, 33] have been infected with the cerebral form of the *C. cerebralis*. There are many reports regarding the cerebral form of the coenurosis in Europe, including Greece [27]. The disease also has been reported in sheep, almost in all 31 provinces of Iran. Prevalence of 18.65% in West Azarbaijan Province, northwestern Iran [31], 0.007% in Kerman province eastern Iran [3]. It has been reported that this disease is seen commonly in sheep and goats in Turkey [34].

The prevalence of coenurosis in sheep and goats were reported 4.9% and 11.7%, respectively in ELFORA export abattoir [2]. The study done at Ethiopian Health and Nutrition Research Institute (EHNRI), Addis Ababa by Asefa *et al.* [35] was reported 4.7% *C. cerebralis* cysts in sheep. The *C. cerebralis* prevalence 2.3% and 12.44% in sheep and goats, respectively, was also reported by Adem [36] in Ashim export abattoirs in Ethiopia.

Economic Losses Due to Coenurosis: The significant economic loss incurred by *C. cerebralis* in small ruminants slaughtered at abattoirs in Ethiopia is due to the condemnation of edible organ (brain) of small ruminants affected with *C. cerebralis* [37]. The financial loss from

domestic and international markets due to brain condemnation of small ruminants with *C. cerebralis* cysts at ELFORA export abattoir was estimated by adopting the formula [38].

$EL = Srx * Coy * Roz$; Where, EL- Estimated annual economic loss of the brain from domestic or international market due to coenurosis

- Srx- Annual sheep and goats slaughter rate of the abattoir
- Coy- Average cost of the brains in the abattoir
- Roz - Condemnation rates of sheep and goats brains

Accordingly, the financial loss from domestic animals at national and international markets due to brain condemnation of small ruminants with *C. cerebralis* cysts at ELFORA export abattoir in Ethiopia was estimated at 18,127.2 USD or 335,353.2 ETB. Where the annual slaughter rate of sheep and goats were 149,760, the rejection rate of brain was 8.3%, average cost of brain was 8.3% and average cost of brain was 1.75 USD/kg [2].

Farmer or the owner often facilitate the contamination of the environment by opening the skull of infected sheep leaving the *Coenurus* cyst free to be eaten by dogs or, feeding them directly with the definitive host [12]. The higher percentages of ecological variables (rainfall, relative humidity and air temperature) are considered to be the influencing factors for coenurosis. In rainy season, rain causes spread of feces of dog, fox (final host) over the grasses and these contaminants are responsible for the increased occurrence of gid during rainy season [39]. According to Gicik *et al.* [33] selling of sick animals to abattoirs or market by owners as soon as they noticed the coenurosis without informing the local authorities leads to the high prevalence of *C. cerebralis* in the area.

Diagnostic techniques for coenurosis: The diagnosis of coenurosis is benefit from clinical findings, neurological and ultrasonographic examination and post-mortem examination [40, 41]. Histopathologic examination, hematological and biochemical examinations are the assenting diagnostic methods for coenurosis. Computed tomography also has been used successfully for the evaluation of coenurosis including the determination of the location and definite size of cysts [42]. Immunodiagnosis tests such as skin test for immediate hypersensitivity, indirect haemagglutination antibody test, immuno-electrophoresis, gel double

diffusion, ELISA tests have been used experimentally. Despite the availability of these tests which have their own practical challenges, post mortem findings of a thin walled cyst filled with transparent fluid and with numerous scoleces in the wall remain the definitive diagnosis [32].

According to Miran *et al.* [30] post mortem examination for the diagnosis of coenurus is as the following: The heads of slaughtered sheep and goats collected, followed by skin removal and careful opening of the skull using a machete or other instrument without damaging the brain. Meninges incised using a scalpel blade to expose brain tissue. The whole brain of each individual animal collected and examined for visible evidence of cyst (*C. cerebralis*). The number and location of cysts seen (described as right hemisphere, left hemisphere or cerebellum) recorded. Therefore, among all the following diagnostic methods are peculiar for isolation and identifications of coenurosis in sheep and goats.

Clinical Examination: Clinical findings in coenurosis vary from breeds of animal, settlement site and size of the cyst [43]. Main clinical signs seen in sheep and goats are dullness, circling, torticollis, sometimes one-sided blindness, pain response depending on pressure of cyst in the brain and leaning of head towards right or left, feet stamping or walking in straight line [44, 45]. Clinical signs seen in coenurosis in cattle are incoordination and visual impairment [43], more specifically uncontrolled movements, stroke in legs, circling from time to time, fatigue and death have been reported [46]. Circling is a common symptom [7] and Achenef *et al.* [10] have concluded that circling side is towards cyst site. It also detected like rotating around itself, leaning the head to the left side, incoordination, drawing circles while walking and ataxia. One author also reported similar results by physical examination, neurological symptoms like rotating around itself, leaning the head to the left side, incoordination, drawing circles while walking, ataxia, bruxism and weight loss [34].

Haematological Findings: As its shown in the table below no abnormality was found in blood parameters other than stress leukogram (Table 2) [2].

Ghosh *et al.* [48] reported that number of erythrocyte, hemoglobin and packed cell volume (PCV) has decreased but number of eosinophil has increased and blood values get back to normal after removing Coenurus cyst surgically. Toos and Adib [49] reported that total protein level has decreased in the sheep with coenurosis and

Table 2: Hematologic parameters of a Simmental bull with Coenurosis.

Parameter	Results	Normal range [47]
WBC (x103/iL)	11.02	4.0-12.0
LYM (x103/iL)	3.46	2.5-7.5
MON (x103/iL)	0.22	0.03-0.8
NEU (x103/iL)	7.29	0.6-4.0
EOS (x103/iL)	0.04	0-2.4
BAS (x103/iL)	0.00	<0.2
RBC (x106/iL)	7.65	5.0-10.0
HGB (g/dL)	9.8	8-15
HCT (%)	31.78	24-46
MCV (fL)	42	37-51
MCH (ng)	12.8	13-18
MCHC (g/dL)	30.8	33-37
RDWc (%)	23.6	16-24
PLT (x103/iL)	260	200-730

Table 3. Biochemical parameters of a Simmental bull with Coenurosis.

Parameter	Results	Normal range [50]
AST (u/L)	135	60-125
ALT (u/L)	28	6.9-35
GGT (u/L)	21	6-17.4
ALP (u/L)	149	18-153
LDH (u/L)	1476	309-938
CHOL (mg/dL)	61	62-193
TBIL (mg/dL)	0.15	0-1.6
TP (g/dL)	9.4	6.7-7.5
ALB (g/dL)	3.4	2.5-3.8
GLB (g/dL)	6.0	3.0-3.5
Ca (mg/dL)	9.3	8.0-11.4
P (mg/dL)	7.3	5.6-8.0
Mg (mg/dL)	2.5	1.5-2.9

AST and creatinine phosphokinase (CPK) levels have increased. But, Shimalis *et al.* [2] reported in their study during hematological investigation they observed stress leukogram depending on the injection of dexamethasone[2].

Biochemical Findings: It is detected that there is an increase in the levels of aspartate aminotransferase (AST), alkaline phosphatase (ALP), globulin (GLOB) and total protein (TP), on the other hand the levels of albumin (ALB), gamma glutamil transferase (GGT), total bilirubin (TBIL), Cholesterol (CHOL), calcium (Ca), magnesium (Mg) and phosphorus (P) were in the reference range in biochemical examination (Table 3) [34]. They detected that there was an increase in the levels of AST, ALP, GLOB and TP; on the other hand, the levels of ALB, GGT, TBIL, CHOL, Ca, Mg and P were inside the reference range in biochemical examination [34].

Postmortem Examination: A transparent 7x5 cm sized cyst in the ventral region of brain's left hemisphere (Fig. 3B) was observed during necropsy. After the

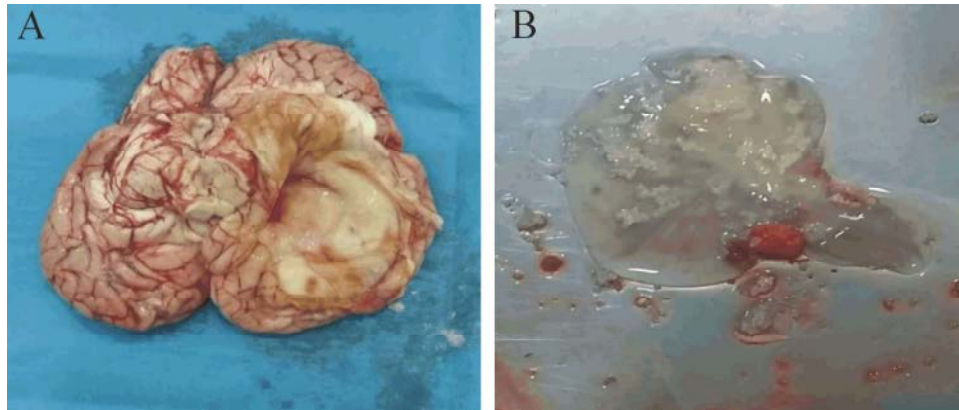


Fig. 2: The localization of cyst and atrophy in brain (A).The cyst containing grey-white fluid including protoscolex (B) [34].

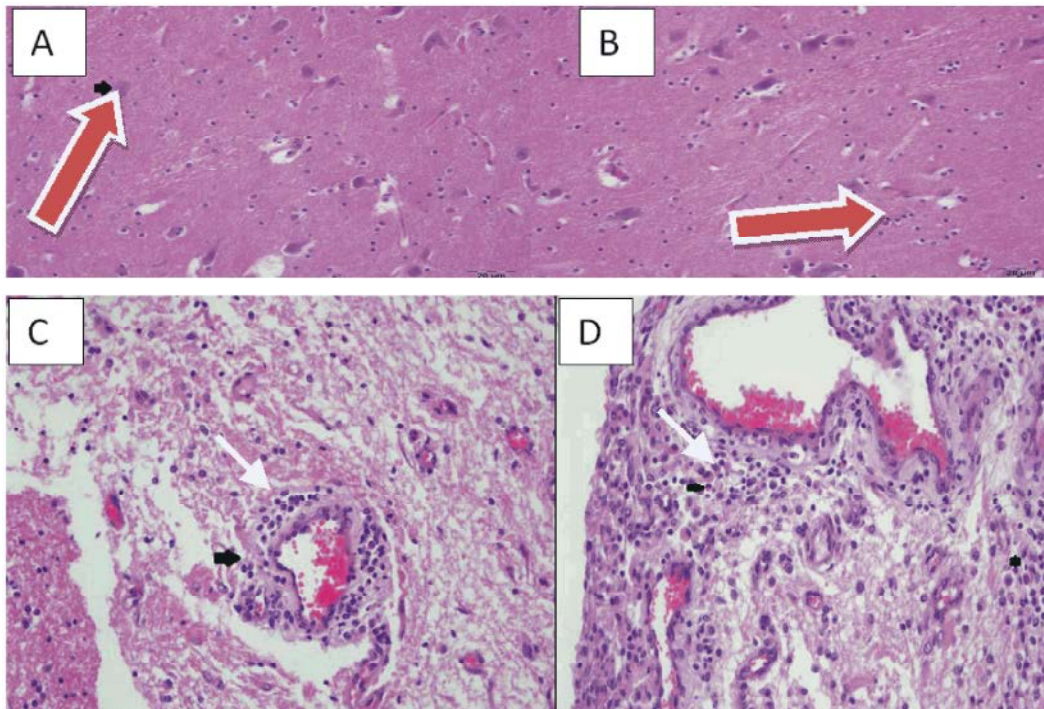


Fig. 3:A: Neuronal necrosis as shown with an arrow in the figure. H&E.X400. B) Gliosis as shown with an arrow in the figure. H&E.X400. C) Hyperemia in vessels and infiltration of perivascular mono-nuclear cell as shown with an arrow in the figure. H&E.X400. D) The infiltration of eosinophil leukocyte shown with an arrow and mononuclear cell shown with a star in the figure. H&E.X400 [34].

dissection of this cyst, there was a grey-white fluid including protoscolex and the formation of atrophy in brain due to this cyst was detected [34] (Fig. 3B).

Histopathological Examination: Histopathological examination of the affected cerebral hemisphere exposed showed that, multiple scolices growing on the internal layer of the cyst, neuronal degeneration and necrosis,

hyperaemia and astrocytosis. Common features of histopathological lesions seen in coenurosis are congestion, focal hemorrhage, demyelination, satellitosis, perivascular cuffing liquefactive necrosis and gliosis and those lead to microglial nodule formation [51-53]. A severe level of neuronal necrosis (Fig. 3A) and gliosis (Fig. 3B), a milder level of hyperemia in vessels and infiltration of perivascular mononuclear cell (Fig. 3C) and the infiltration

of eosinophil leukocyte and mononuclear cell in the close regions of the wall of the cyst (Fig. 3D) [34].

The other histopathological lesions reported by coenurosis are neuronal degeneration and pressure atrophy in the skull [51, 33]. A case reported from Turkey determined a severe level of neuronal necrosis and gliosis, a milder level of hyperemia in vessels and infiltration of perivascular mononuclear cell and the infiltration of eosinophil leukocyte and mononuclear cell in the close regions of the wall of the cyst [34].

Molecular Characterization of Coenurosis: Molecular genetic markers of mitochondrial DNA were applied phylogenetically to resolve the questionable relationship between *C. gaigeri* and *C. cerebralis*. Results based on phylogenetic analysis of the mitochondrial DNA (CO1 and ND1) suggest that the larval stages of *C. gaigeri* and *C. cerebralis*, which showed similar morphological criteria, are monophyletic species. However, *C. gaigeri* interestingly were situated in the biceps femoris, triceps and abdominal muscles without localization in the nervous system [54].

Georgios *et al.* [55] reports the causative agents of cerebral and non-cerebral coenurosis in livestock by determining the mitochondrial genotypes and morphological phenotypes of *T. multiceps* isolates from a wide geographical range in Europe, Africa and western Asia. Three studies were conducted: (1) a morphological comparison of the rostellar hooks of cerebral and non-cerebral cysts of sheep and goats, (2) a morphological comparison of adult worms experimentally produced in dogs and (3) a molecular analysis of three partial mitochondrial genes (*nad1*, *cox1* and 12S rRNA) of the same isolates. No significant morphological or genetic differences were associated with the species of the intermediate host. Adult parasites originating from cerebral and non-cerebral cysts differed morphologically, e.g. the shape of the small hooks and the distribution of the testes in the mature proglottids. The phylogenetic analysis of the mitochondrial haplotypes produced three distinct clusters: one cluster including both cerebral isolates from Greece and non-cerebral isolates from tropical and subtropical countries and two clusters including cerebral isolates from Greece. The majority of the non-cerebral specimens clustered together but did not form a monophyletic group. No monophyletic groups were observed based on geography, although specimens from the same region tended to cluster. The clustering indicates high intraspecific diversity. The phylogenetic analysis suggests that all variants of *T. multiceps* can

cause cerebral coenurosis in sheep (which may be the ancestral phenotype) and some variants, predominantly from one genetic cluster, acquired the additional capacity to produce non-cerebral forms in goats and more rarely in sheep [55].

Molecular characterization by PCR shows positive result for cerebral cysts in the naturally and experimentally infected sheep and goats, by producing the expected fragments for COX-1 and NAD1 genes. Sequence analysis showed that the sheep and goats samples examined in the naturally and experimentally infected samples are 100% identical to each other and 100% similar to adult worms recovered from dogs based on both mitochondrial markers [26].

Differential Diagnosis: Coenuruscerebralis may be found upon necropsy in the brain of sheep and goat but the condition needs to be differentiated from other local space occupying lesions of the cranial cavity and spinal cord including abscess and tumor. Hemorrhage in the early stage of the disease may be confused with encephalitis because of signs of brain irritation [56].

Listeriosis, loupingill, scrapie and brain abscessation and tumor should be considered as the differential diagnosis of the cerebral coenurosis [40,57]. Scrapie would typically affect sheep older than three years; polioencephalomalacia causes diffuse bilateral cerebral signs, listeriosis results in multiple unilateral cranial nerve deficits, while focal symmetrical encephalomalacia results in rapid death. A thorough neurological examination should therefore permit an accurate diagnosis of *coenurosis* [17].

Listeriosis is an infection caused by the bacterium *Listeria monocytogenes*. The disease can affect sheep, goats and cattle. Symptoms include depression, decreased appetite, fever, stumbling or moving in one direction only, head pulled to flank with rigid neck, facial paralysis on one side, slack jaw and abortions. The disease is curable by use of antibiotics such as procaine penicillin [30].

Scrapie is an infectious transmissible fatal degenerative disease affecting the central nervous system of sheep and goats. The disease is caused by a prion (protein particle similar to a virus but lacking nucleic acid) and is usually observed in animals older than 2 years. Early signs include subtle changes in behavior or temperament. These changes may be followed by scratching and rubbing against fixed objects, loss of coordination, weakness, weight loss despite retention of appetite, biting of feet and limbs, lip smacking and gait

abnormalities, (high-stepping of the forelegs, hopping like a rabbit and swaying of the back end) and the disease is often accompanied by pruritus [30, 58].

Louping ill results from infection by louping ill virus, a member of the genus *Flavivirus* in the family *Flaviviridae*. This virus is closely related to tick borne encephalitis virus. The incubation period for louping ill is six to 18 days in sheep. Louping ill is characterized by an initial febrile viremic stage, which may be accompanied by depression and anorexia, followed in some cases by neurological signs. Affected sheep may develop an unusual hopping gait, called a "louping gait," during which they move both hindlegs, then both forelegs, forward in unison. Death is common among animals with neurological signs, often within a few days. Peracute deaths can also be seen. Surviving animals may have residual CNS deficits. Louping ill should be suspected in sheep with fever and neurological signs, particularly when the flock has recently been introduced to tick-infested pastures. It should also be a consideration in grouse with a fatal illness [59].

Control and Prevention Methods of Coenurosis:

Currently the only treatment that can be recommended is the surgical removal of the coenurus cyst from the brain of the affected animal [60, 61]. This treatment can be very successful and most cases will show a dramatic recovery, with return to full neurological function [62], however not all affected animals can undergo surgery as it largely depends on the location of the cyst [63]. If this is not possible, the control and prevention of coenurosis should be based on routine anthelmintic dosing of dogs, preferably every three months. The vet will have to decide whether there is a chance the animal will recover or whether it is better to destroy the affected animal humanely to prevent further suffering. The best control and prevention method of coenurosis is to prevent dogs from having access to sheep and cattle carcasses and not to feed them uncooked meat [64].

Public footpaths running through the sheep fields used by people walking their dogs can be a particular problem. Farmers could display a sign explaining the disease risks and encouraging local people walking their dogs on these fields to have their dogs wormed. If cases of coenurosis are a regular occurrence on the farm, eliminating the disease from the farm should be part of the overall health plan [29]. The treatment of the disease in sheep and goat is not satisfactory, except surgical removal of the cyst that is not economical, so the most effective method is prevention of the disease by

controlling dog contact with pasture, community awareness regarding the transmission way of the disease [65].

Zoonotic Importance of *Coenurosiscerebralis*:

Coenurus cerebralis in human beings diagnosed for the first time in 1913 in Paris, when a man presented symptoms of central nervous system nerve degeneration. He had convulsions and trouble speaking/ understanding speech. During his autopsy, two coenuri were found in his brain. Recently (within the last 25 years), human cases have been recorded in Uganda, Kenya, Ghana, South Africa, Rwanda, Nigeria, Italy, Israel, Mexico, Canada and the United States and animal cases have been found in many other countries as well. In 1983, a 4-year-old girl in the USA was admitted to the hospital with progressive, generalized muscle weakness, inability to walk, rash, abdominal pain and deteriorating neurological ability. When the doctors did a CT scan, they saw fluid filled lumps in her brain and decided to operate. While operating, coenuri were found and the patient was immediately given chemotherapy with praziquantel. Unfortunately, the coenurosis had already done too much damage in the central nervous system and the little girl did not survive. In these cases, the infected individuals had been exposed to wild dogs in regions where canid tapeworm is considered endemic and probably ingested the parasite accidentally through contact with contaminated food or water [66].

Coenuruses is a relatively rare zoonotic disease of humans, caused by the larval stage of a dog tape worm *Taenia (Multiceps) multiceps*. Human infection occurs if eggs are accidentally ingested as result of poor personal hygiene after being shed in the faces of the dog. After ingestion of the eggs, larvae hatch, penetrate the intestinal wall and migrate to various tissues, where they develop in to large, cystic larvae. Symptoms are secondary to the presence of a cyst in a vital structure. Patients with coenuruses present with headache and papille edema. The cysts have been responsible for epilepsy, hemiplegia, monoplegia and cerebral ataxia. When the spinal cord is affected there may be spastic paraplegia, lymphadenopathy, fever and malaise can occur, raising the suspicion of lymphoma [57].

The cerebral form of coenurosis in human is the most serious one. Several years may pass between infection and the appearance of symptoms and the symptoms varies with the neuroanatomical localization of the coenurus: cerebral coenurosis is manifested by signs of intracranial hypertension and the disease is very difficult

to distinguish clinically from neuro cysticercosis or cerebral hydatidosis. Symptoms that may be observed consist of headache, vomiting, paraplegia, hemiplegia, aphasia and epileptic form of seizures. Papilledema is a sign of increased intracranial pressure. The coenurus can also develop in the vitreous humor and may affect the retina and choroid. The degree of damage to vision depends on the size of the coenurus and the extent of the choroido retinal lesion. The prognosis for coenurosis of the nervous tissue is always serious and the only treatment is surgery, although recently, the testing of treatment with praziquantel or albendazole has begun [9]. There are more than 100 reports of human infection with these metacestodes. The cerebral coenurosis create serious problems and even death in patients [26].

Humans are dead-end intermediate hosts and become infected by ingesting eggs passed in the excrement of a definitive host. The ingested eggs release oncospheres in the host intestine that penetrate the intestinal wall and migrate toward target organs through the blood stream, usually lodging in the brain, spinal canal, or eye. In the brain, the coenurus causes inflammation (coenurosis) in the parenchyma and its presence along the cerebrospinal fluid pathways eventually leads to basal arachnoiditis or ependymitis. The most common signs and symptoms of this condition are headache, seizures, vomiting and papille edema [67].

CONCLUSION

This review showed that coenurosis was an important parasitic disease in sheep and goats caused by the larval stages *C. cerebralis* and induced severe economic loss due to brain condemnation. Inappropriate disposal of heads of small ruminants being practiced by some of the abattoirs could enhance the continuation of the life cycle of *C. cerebralis* between the intermediate (sheep and goats) and final hosts (dogs). The main reservoir and facilitates transmission of disease are dogs species. This disease has very similar clinical sign with other diseases like; listeriosis, scrapie and louping-ill. Therefore, different diagnostic methods should have to be available to confirm the diseases in particular areas of suspicions. Therefore, depending on the written literature and importance of diseases the following recommendations are forwarded:

- Awareness creation programs should be launched for the butchers, abattoir workers, meat-sellers and dog owners as to the danger of the *C. cerebralis* to human as well as animal health.

- Efforts should be geared towards preventing dogs and other canines contaminating the pasture with tapeworm eggs, by stopping those eating sheep carcasses.
- Dispose of sheep carcasses quickly and correctly.
- Hygienic measures should also be observed or vegetables should be thoroughly washed and cooked before consumption.
- Worm residential dogs regularly (every 3 months) and prohibition of backyard slaughter.
- If possible local people walking dogs on the land should be encouraged to have their dogs wormed.

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