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A brief understanding of *Trichuris ovis* in ruminants

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Abstract
Trichuris ovis, commonly known as whipworm of sheep occurs in the caecum and colon of sheep, goats, cattle and other ruminants in all parts of the world but is relatively harmless. However, clinical diseases due to *T. ovis* may also be found in sheep and cattle. Heavy infection may be occurred in very young lambs, kids and calves. The infected hosts may suffer from bloody colitis and diphtheritic caecitis which may cause ulcerative and necrotic lesions on the mucosa. In animals with high worm burden severe anaemia and dehydration and jaundice may lead to the death of the animals. Methyridine, fenbendazole and oxfendazole are the example of anthelmintics which can be used to treat trichuriasis in sheep, goat and cattle.

Keywords: *Trichuris ovis*, pathogenesis, treatment, sheep, goat, cattle

Introduction
Trichuris is a widespread gastrointestinal parasite that can be found in a broad range of hosts and causes trichuriasis, a neglected tropical disease causing significant animal and human health problems as well as considerable socio-economic consequences worldwide [1, 2, 3, 4]. Among the various species of *Trichuris*, *Trichuris ovis* is a whipworm infecting the caecum and colon of sheep, goats, cattle and other ruminants in all parts of the world [1, 2] particularly in South and North America, Australia, Europe and Asia but is relatively harmless [3]. Its life cycle is direct where definitive host acquired infection through ingestion of infective stage of L_1 within the eggs which hatch in the small intestine and the released larvae burrow into the intestinal wall of the caecum and proximal colon where they develop to mature worms [1, 2]. The clinical diseases caused by *T. ovis* have been reported in sheep and cattle [5]. The substitute control methods are liable to be more dependent on sound understanding of the species, life cycle, their pathogenicity and epidemiology which are elaborately reviewed in the present communication.

Morphology
The male measures 50-80 mm in length of which the narrow and filamentous anterior end constitutes three-quarters of the length [1]. The female is 37- 70 mm long of which the narrow and filamentous anterior end forms two-thirds to four-fifths [2]. The fully evaginated spicule is 5-6 mm long and having a sheath which bears an oblong swelling a short distance from its distal extremity and is covered with minute spines which decrease in size towards the distal extremity [1, 6]. The characteristics eggs are brown, barrel shaped or lemon shaped with a transparent, conspicuous plugs at both ends and measures 70-80 by 30-42µm including the plugs; they contain an unsegmented embryo when laid [1,2].

Life cycle
Its life cycle is direct where orally ingested embryonated eggs hatch in the small intestine and the released larvae burrow into the intestinal wall of the caecum and proximal colon where they develop to mature worms. The anterior end of the worm forms syncytial tunnels of epithelial cells around itself as it burrows through the mucosa [7]. The posterior end protrudes into the lumen facilitating copulation and egg release. The infective stage is L_1 within the egg which develops in three weeks under favourable conditions [2]. Infective stage develops in one or two months of being passed in the faeces depending on the temperatures (e.g. 6-20 °C),

since development is related to soil moisture and temperature [1]. Under optimal conditions these may subsequently survive for several years [2].

After ingestion, the plugs are digested and the released L_1 penetrate the glands of the anterior small intestine for 2 to 10 days before they move to caecal mucosa, where they develop to adults [2]. Subsequently all four moults occur within these glands, the adult emerging to lie on the mucosal surface with their anterior ends embedded in the mucosa. The prepatent period is 7-9 weeks [1].

Pathogenesis

The filamentous narrow anterior end of *T. ovis* may be embedded in mucosa, surrounded with fibroblast and leucocytes without sign of fibrosis and necrosis [8]. *T. ovis* inserted in the lining of the large intestine predominantly in caecum of the hosts may be found exclusively beneath the mucosal layer covered by the apical surface of mucosal epithelium, forming a tunnel. The crypts of Leiberkuhn, submucosa, muscularis mucosae and muscularis externa may be obviously distinguished with slight infiltration of mononuclear cells leucocytes, goblet cells along with epithelial changes and overall mucosal architecture in vicinity of worm penetration [9]. Leucocyte infiltration around the worm embedded under the mucosa but there is no sign of rigid encapsulation, fibrosis, and necrosis [8]. This is probably due to the reason that the penetration of anterior end to receive its nutrition is a normal phenomenon and parasite move to new location after a brief period. But since the penetration of parasite inflame the tissues, leucocytes invasion takes place in the vicinity of the parasite in the inflamed areas [8]. Goat infected with *T. ovis* show active hyperaemia, haemorrhages and lymphocytic infiltrations in the mucosa [8].

The animal gets infection through ingestion of infective larval stage within the eggs [1, 2]. After ingestion eggs are hatched out to L_1, the larvae penetrate into small intestine's mucosa and stayed for about two weeks which probably provoke desquamation of the epithelium, hyperaemia, mucoid dystrophy, eosinophilic infiltration around the larvae [8]. The larvae and mature worm also induce mechanical damage to the mucosa of small and large intestines followed by extensive local inflammation and haemorrhages.

As the parasitic infection is a long standing encroachment on the intestinal wall, especially in untreated animals, it causes destruction of the lining epithelium where they predominantly inhabit [1]. Due to this continuous irritation of the adult parasites on the intestinal wall, catarrhal inflammation occurs. That is why goblet cells were increased in numbers and size [8].

Clinical signs

A high incidence of light infections with negligible clinical signs is characteristic feature of *Trichuris* infection in ruminants but isolated outbreaks have been recorded [1, 5]. Most of the infections caused by *T. ovis* are generally light and asymptomatic [1.2]. In some cases a large number of worms cause a diphtheritic inflammation of the caecal mucosa. They cause mild to moderate degree of damage in the intestinal surface, resulting in petechial haemorrhages during the process of penetration of the anterior part of the *T. ovis* [8]. If the host is heavily infected, a large portion of the blood vessels located in the caecal wall will be consumed. This eventually results in the thickening of the wall, thus preventing that region of the large intestine from absorbing fluids causing the host to have diarrhoea [10].

Lambs with heavy infection may suffer from bloody colitis and diphtheritic caecitis due to ulcerative and necrotic lesions on the mucosa. Moreover, in animals with high worm burden severe anaemia and dehydration and jaundice may lead to the death of the animals [1, 4, 11].

Post-mortem examination

Infection of *Trichuris* in large intestine caused thickening of mucosa, lymphoid nodules on the lamina propria is enlarged from which the parasite may produce some chemical mediators that cause lymphoid proliferation locally [12]. Grossly, congestion, haemorrhagic spots, ulcer formation and nodule formation with thickening of the caecal valve may be observed in large intestine [8].

Epidemiology

In sheep, goat and cattle naturally acquired infections are not usually severe enough to cause clinical disease and sheep over eight months of age show an age resistance to infection and resistance to reinfection 2-3 weeks after infection [1,2]. Heavy infection may be found in very young lambs. The pathology is a result of the host immune response, which is assumed to be generated to eliminate invading pathogens. Lesions may also be caused by the direct injure commencing worm attachment or the indirect damage from soluble products released by the worms [13]. Moreover, bacterial incursion takes place at the site of tissue damage which may elicit immune response against the bacteria and aggravate the lesion by enhanced cellular infiltrations [14]. The foremost expression of the host against the parasite invasion is reflected by the changes in the formed elements of blood. The leucocyte response vary from host to host against a particular infection, which may also vary with age and frequency of exposure, being more severe in non-specific host while it is feeble in the specific host as an adjustment is made between the host and parasite [8]. Sometimes, it is specific and characteristic and may be taken in conjunction with other findings in diagnosis of a particular species.

Diagnosis

The clinical signs are not the pathognomonic hence the diagnosis is made by demonstration of the characteristic barrel-shaped eggs with conspicuous plugs at both ends in the faeces. However, since clinical signs may occur during the prepatent period, diagnosis in food animals may depend on necropsy [2].

Treatment

The following anthelmintics can be used in sheep and cattle

1. Methyridine (200 mg/kg) given orally or subcutaneously is highly effective [1].
2. Fenbendazole (5-20mg/kg) is effective.
3. Oxfendazole (2.5mg/kg) is effective against adult parasites (89-99% efficacy) and immature parasites (62-100% efficacy) [1].

Conclusions

After a large number research over the century on epidemiology, biology and development, pathogenesis, clinical signs, and control, this parasite continues to be an essential constraint on ruminant production. Novel developments for the management of this nematode parasite such as vaccines, biological anthelmintics, genetic markers, and selective breeding may, in the future, provide additional or alternative means of parasite control.

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