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Pathological Changes of Abomasum in Naturally Infected Makoyee Sheep with *Teladorsagia circumcincta*

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Abstract: The present study was carried out in order to investigate the lesions in the abomasum of Makoyee breed sheep caused by *Teladorsagia* spp. Infected sheep which were examined during spring and summer 2000, subjected to detail necropsy with special reference to abomasal lesions. Six out of 20 sheep were infected with *T. circumcincta*. Abomasal changes were recorded and tissue samples were collected from abomasum for histopathology. The results were indicated different pathological changes such as congestion of blood vessels, oedema, foci of *T. circumcincta* in the superficial part of mucosa, sometimes deep in the glandular region and increased mucous secretion. Also, cystic spaces were found in some glands and infiltration of leukocytes in the mucosa, submucosa and muscular layers were seen. The other findings were hyperplasia of glands, muscular layer of the abomasum and thickening of blood vessel walls. This study brought to light the significant pathological changes even in the light infection caused by *T. circumcincta* in the abomasum of Makoyee breed sheep.

Key words: *Teladorsagia circumcincta*, Makoyee breed sheep, abomasum, pathological changes

INTRODUCTION

Teladorsagia spp. has been reported from sheep in Iran with high prevalence (Eslami and Nabavi, 1976; Eslami *et al*., 1979, 1980). These nematode causes serious losses by producing weight loss and decreased wool production, either are economically disadvantageous (Kaufmann, 1996; Bowman and Lynn, 1999). It is recalled that larvae of *Teladorsagia* spp. may develop in one of three ways in sheep. First, larvae may remain for some considerable time in the mucosa without development beyond the early fourth stage. Secondly, they stay in the mucosa for a brief period, long enough to pass the third ecdysis, and then pass to the lumen of the abomasum and become adults. Thirdly, the larvae may grow while in the mucosa and migrate to the lumen either as mature or at some other stage of development. *Teladorsagia* spp. overwintering infective third stage larvae resemble those of *Trichostrongylus* on pastures and infect ruminants during the early grazing season. However, arrested development of parasitic larvae is also very well established in *Teladorsagia* spp. and this is both of epidemiological and pathological importance. Type I or summer teladorsagiosis, usually occurs in pastured young ruminants, the worms maturing without first passing through a developmental arrest. On contrary, Type II or winter teladorsagiosis, occurs in late winter when larvae that have remained in arrested development since fall once again become metabolically active and proceed to develop into adults. There are some information dealing with different breeds of sheep infected with *T. circumcincta* (Gruner *et al*., 1986; Stear *et al*., 1997, 2002; Bouix *et al*., 1998; Coltman *et al*., 2001; Gruner *et al*., 2004a,b; Martinez-Valladares *et al*., 2005a,b). Despite the importance of *T. circumcincta* in causing serious economic losses in sheep in Iran, there is no report dealing with pathological changes of abomasum in Makoee breed sheep with natural infection with this nematode in this region. Therefore, the results of this study was compared with investigations of Mckellar *et al.* (1990), O' Callaghan *et al.* (1992), Abd-Rabo *et al.* (1993), Baker *et al.* (1993), Shwakat *et al.* (1994), Hill *et al.* (1994), Lawton *et al.* (1996), Scott and Mckellar (1998), Scott *et al.* (1998) and Przemeck *et al.* (2005).

MATERIALS AND METHODS

Twenty Makoyee sheep, which were slaughtered at Urmia slaughter-house were subjected to detail post-mortem examination with special reference to the abomasal changes and *Teladorsagia* spp. infection.

The abomasum was opened and inspected for the parasite. In cases which were positive for the presence of

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the parasite, the degree of infestation was determined by both counting eggs per gram of feces (EPG) and number of adult worms found in the abomasums (Dunn, 1978).The species of the *Teladorsagia* spp. was identified based on the morphological characteristics (Soulsby, 1986). Also, single infection with *Teladorsagia* spp. was considered by fecal examination and abomasal changes were recorded in animals which were only infected with *Teladorsagia* spp. Sheep with other worms beside *Teladorsagia* spp. in their abomasums were not included in this study.

Tissue samples were collected in 10% buffer formal saline for histopathology and processed. Paraffin blocks were made, 4-5 micron sections were cut and stained with hematoxylin and eosin. They were examined under light microscope and observations were recorded.

RESULTS

Six cases out of 20 sheep (2-4 years old) were found to have light infection (EPG = 50-200 and Number of Worms = 1-1000) with only *Teladorsagia circumcincta*. Animals which had other worms in their abomasums, were not taken into consideration for this study. The identification of the parasite was based on the morphology.

Gross pathology: The abomasal wall was thickened with superficial ulceration. The mucosa was congested and covered with mucous secretions. Adult parasites were macroscopically visible on the mucosa.

Histopathology: The pathological changes observed by light microscopy were thickening of the mucosa, hyperplasia of glands, infiltration of inflammatory cells mainly lymphocytes and macrophages (Fig. 1). Severe inflammatory reactions and in some areas, ulcer formation and some cystic spaces were also noticed. In some foci the penetration of the mucosa by *T. circumcincta* was evident (Fig. 2). The submucosal layer was also thickened, oedematous and infiltrated with inflammatory cells. There was also fibrous tissue proliferation (Fig. 3). The muscular layer of abomasum was hyperplastic which caused the thickening of this layer. Inflammatory cells were also seen in the muscular layer. The serosal layer of abomasum showed no significant pathological changes. Severe changes were observed in the abomasal glands, they showed different degrees of hyperplasia from mild to very severe (Fig. 4). There was increased mucosal secretion and some glands showed cystic changes which were due to the obstruction of the duct by the parasite. Some parasites were found in the glandular region, which were surrounded by macrophages (Fig. 5). Inflammatory changes were also evident. Other general changes were congestion of the blood vessels. Thickening of the wall of blood vessels was due to the hyperplastic changes.

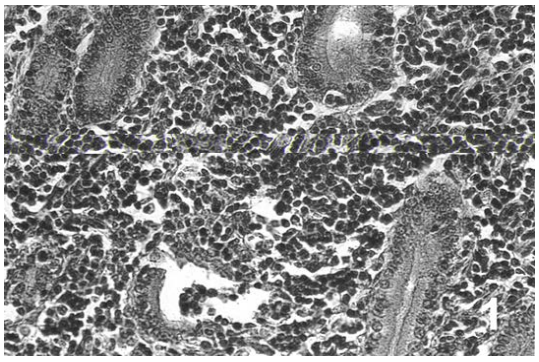

Fig. 1: Infected abomasum- Diffuse leukocytic infiltration in between the hyperplastic glands (H and E X200)

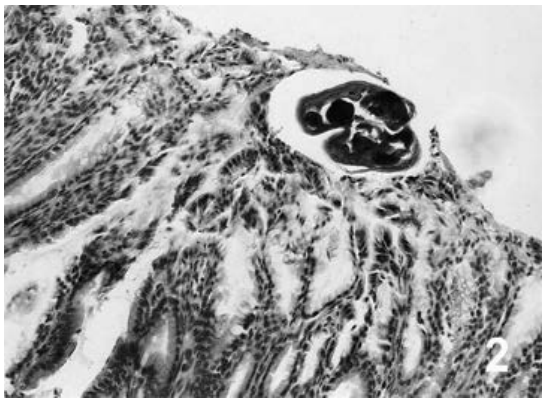

Fig. 2: Presence of *Teladorsagia circumcincta* in the superficial parts of the mucosa (H and E X 100)

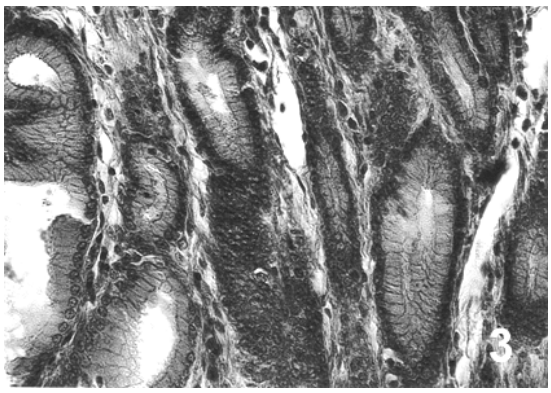

Fig. 3: Severe hyperplasia in the glands (H and E X 200)

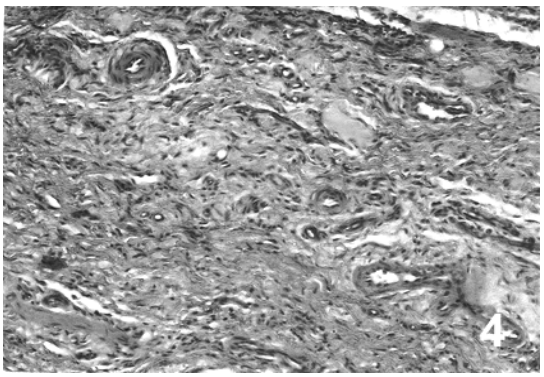

Fig. 4: Fibroblastic proliferation in the submucosal layer of infected abomasums (H and E X 100)

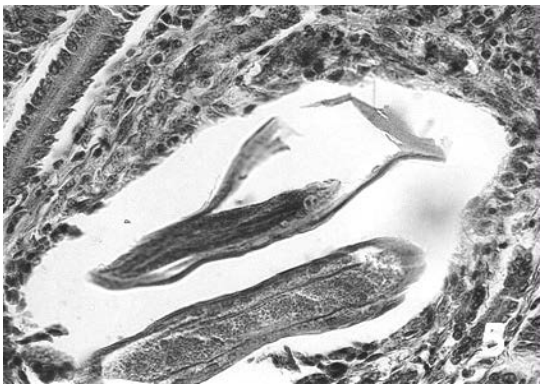

Fig. 5: Presence of *Teladorsagia circumcincta* in the glandular part of the abomasums (H and E X 100)

DISCUSSION

At present study, the histopathological observations clearly showed pathological changes in the infected tissues with *T. circumcincta*. These changes which were observed in the mucosal layer, indicated the extent of the injuries due to different developing stages of the parasite (Abd-Rabo *et al*., 1993; O' Callaghan *et al*., 1992). The inflammatory response in different layers of the abomasum could be due to the release of mediators, as was stated by Baker and Gershwin (1993). The hyperplastic changes directly could be due to chronic irritation by the parasite during the course of its development. Scott *et al.* (1998) explain the hyperplastic changes could be mediated by host growth factor-alpha. Alternatively, the changes in response to the presence of adult worms could be mediated by chemicals that are cytotoxic/inhibitory for cells and released by parasites themselves. On the other hand, Scott and Mckellar (1998), reported that *Teladorsagia* spp. could excrete/secrete products which stimulated the *in vitro* release of pepsinogen from intact abomasal sheets and caused contraction of strips of abomasal smooth muscle, this fact could be the reason for hyperplasia of smooth muscle cells during teladosagiosis. Other investigators also reported that pepsinogen level is increased during the *Teladorsagia* spp. infection (McKellar *et al*., 1990; Baker *et al*., 1993; Shwakat *et al*., 1994; Hill *et al*., 1994; Gruner *et al*., 1986; Lawton *et al*., 1996). The ulcer formation could be attributed to the larvae migration and also should take into consideration the alteration of abomasal pH as a factor (Shwakat *et al*., 1994). Przemeck *et al.* (2005) revealed that excretory/secretory (ES) products of *T. circumcincta* and *Haemonchus contortus* have been implicated in the inhibition of gastric acid secretion and vacuolation, and the loss of parietal cells associated with abomasal parasitism.

This study well documented the different pathological changes of abomasum of Makoyee sheep with *T. circumcincta.*

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