1. Introduction

Trichinellosis is a parasitic zoonosis transmitted to humans through a variety of mammals. Although traditionally herbivores have been considered non-specific hosts, it is now known that they can participate in the transmission of this disease to human beings. Thus, outbreaks of trichinellosis in which the origin was horsemeat have been reported [3, 12, 16, 36]. Furthermore, it has been experimentally proven that not only horses [15, 18, 27, 30, 31, 36], but other herbivores such as bovine [28] or sheep [1, 27, 34] can also be suitable hosts for Trichinella spiralis. In spite of the interest in this type of research, there are not available data about the behaviour of other herbivores, such as the goat, with regard to Trichinella spiralis infection. The goat is, probably, the most important livestock species in the Canary Islands, Spain, where this experiment was carried out.
As indicator of the establishment of Trichinella sp. in this atypical host, the present paper shows the description of the pathological effects of this parasite in the goat.

2. Materials and methods

A) ANIMALS

A total of 10 goats (six males and four females), two months old and about 10 kg body weight were used in this experiment, according with a previous paper [22].

B) PARASITE

The experiment was carried out using a synanthropic isolate of Trichinella spiralis as infecting material [22], obtained from domestic pigs and typified according to the International Commission on Trichinellosis (ICT) as "T1" [19].

C) EXPERIMENTAL DESIGN AND SAMPLE COLLECTION

The animals were divided into the following groups:

Group 1: Eight experimental animals, infected with 1,000 larvae kg⁻¹ body weight (approx. 10,000 larvae per animal), "per os" administration of infected muscle tissue from Swiss NBC mice.

Group 2: Two non-infected animals, used as control and housed in the same experimental conditions.

Animals of group 1 were painlessly killed at 5, 7, 20, 30, 45, 60 and 90 days post infection (dpi). One animal, programmed to be sacrificed on day 75 pi died on day 23 pi, possibly due to a special and individual susceptibility. This chronological period was established according to the duration of the biological cycle phases of T. spiralis and for studying the lesions produced. The animals of group 2 (control group) were killed on days 20 and 90 pi.

After sacrifice, the animals were necropsied and samples from abomasum, small and large intestine, skeletal muscles, myocardium, liver, kidney, spleen, lymph nodes, lung and central nervous system were taken in order to analyse the anatopathological and histopathological changes. These samples were fixed in 10 % neutral buffered formalin and absolute ethanol during 24 hours, embedded in paraffin, sectioned at 2-3 µm and stained with hematoxylin and eosin for light microscopy findings.

3. Results

No clinical or pathological abnormalities were observed in the animals of group two.

The animals of group one, showed an extensive lesional picture. The gross examination of the intestine of animals sacrificed on days 5 and 8 pi revealed a moderate congestive appearance with mucous fluid and a pronounced hypertrrophy of Peyer's patches. These lesions were clearly evident from day 20 pi and became so strong that caused relieves on the ileo-cecal mucosal at the end of the experiment (45-90 dpi).

A desquamative catarrhal enteritis (Fig. 1) was microscopically observed in the animals necropsied on days 5 and 8 pi, which evolved with decreasing intensity until day 30 pi. Hyperplasic and chronic enteritis (Fig. 2), moderate on days 30 and 45 pi and clearly manifest on days 60 and 90 pi, was observed. These lesions are characterised by a lymphoid hyperplasia and a very strong reticulohistiocytosis.

Macroscopically, the intestinal smooth muscle showed a congestive appearance. Microscopically, hyperemia and oedema were detected. These lesions were very strong during the first days of the experiment, diminishing slowly until day 45 pi and, finally, disappeared in the animals sacrificed on days 60 and 90 pi. Likewise, myofibrolisis and simple myositis appeared after day 45 pi, maintaining their intensity until the end of the experiment (Fig. 3).

In the samples taken from the diaphragm, masseters, tongue, intercostal and limb flexor and extensor muscles, a considerable congestion was observed in the initial phases of the disease (20 dpi), similar to the aforementioned in the smooth muscle. After day 40 pi and more clearly on day 90 pi, degenerative areas with a whitish appearance could be found in the diaphragm. On a microscopic level, a vascular reaction was observed after day 5 pi, characterised by hyperemia and interfascicular oedema, which remain strong at day 8 pi, moderate at days 20 and 23 pi, slight at days 30 and 45 pi, and then disappeared. In this tissue a simple myositis was observed, which followed a similar evolution pattern after the day 20 pi. From this date, Zenker degeneration was found, which progressively increased until the end of the experiment (Fig 4). From day 30 pi on, this degeneration coincided with the appearance of myofibrilar lysis, whose intensity runs parallel to the degeneration. The same evolution could be observed in the chronic inflammatory reactions, firstly detected as a granulomatous process, which became very strong at the end of the experiment (Fig. 5). The presence of intrafibrilar parasitic cysts after day 20 pi should be underlined. They were few in number and not wholly formed on this date, but increased in number and development after days 23 and 45 pi.

In the heart, congestive areas on the surface of the epicardium were detected after the first days of the experiment until day 30 pi. After day 45 pi, they became degenerative, with a whitish appearance. The most obvious microscopic lesions were only observed in the vascular processes (hyperemia and oedema) and in the myositic ones. Initially, this myositis appeared as simple myocarditis progressing between days 5 and 30 pi. After day 45 pi, it turned into granulomatous myositis which reached its maximum expression on day 90 pi (Fig. 6).

Macroscopically, the liver appeared swollen, globate, with slightly discoloured edges which gives it a degenerate aspect. This is confirmed by the microscopic observation of a moderate microvascular glycogen degeneration on the first sacrificed animal (Fig. 7). This lesion was clear throughout the experiment with little variation in intensity. Furthermore, it is worth to mention the existence of a light granulomatous hepatitis formed by mononuclear cells on day 45 pi, with stronger symptoms in the goat sacrificed on day 60 pi, and even stronger in the one of day 90 pi (Fig. 8).
PATHOLOGICAL CHANGES IN GOATS EXPERIMENTALLY INFECTED WITH *TRICHINELLA SPIRALIS*

**Figure 1.** — Desquamative enteritis; (8 DPI), x 250.

**Figure 2.** — Hyperplasic and chronic enteritis (44 DPI), x 180.

**Figure 3.** — Intestinal smooth muscle. Simple myositis (90 DPI), x 180.

**Figure 4.** — Zenker’s degeneration (90 DPI), x 250.

**Figure 5.** — Parasitic cysts. Granulomatous reaction (44 DPI), x 250.

**Figure 6.** — Granulomatous myocarditis (90 DPI), 180.
The kidneys showed alterations which affected not only the glomerulus but also the interstice and tubular system. Macroscopically, until day 30 pi the kidneys looked spherically, with cortical hemorrhages, and the medulla strong in colour. Its decapsulation occurred easily. After day 40 pi the capsule was strongly attached to the parenchyma and there was no evidence of hemorrhages. Microscopically, at 5-8 dpi a minimal glomerulonephritis was detected. At 20-30 dpi, the glomerulonephritis was membranous, becoming proliferative after this date (Fig. 9). In the tubular system, and after 8 dpi, there was a tubular degeneration, which steadily increases in intensity until the final days of the experiment. As a result of this degeneration, which includes desquamation of epithelial cells, from day 30 pi on a progressive and evident tubular necrosis appeared (Fig. 10).

The presence of oedema was evident at the interstitial tissue, being moderate on day 5 pi, stronger from day 8 pi to day 30 pi. After day 45 pi, the oedema was replaced by a lymphoplasmonic cellular infiltration, showing an interstitial nephritis, which increased until the end of the experiment (Fig. 11).

Macroscopically, the spleen showed a splenomegaly with considerable congestion, from the beginning of the experiment until day 30 pi. Likewise, it appeared whitish due to the spreading of the white spleen pulp, which rose in intensity parallel to the experimental process. Microscopically, a hyperplasic reactive splenitis was detected, being acute until day 30 pi., with a strong vascular stasis due to the spreading of the red spleen pulp. In the final days it became chronic (Fig. 12) with predominance of white pulp. Furthermore, after day 40 pi, there was lymphocytolysis and reticulohistiocytosis.

The lymph nodes showed lesions similar to the ones observed in the spleen. A lymphoadenomegaly with oedema was evident until day 30 pi. The animals sacrificed after this date didn’t show this oedematous appearance. Microscopically, hyperplasic reactive lymphadenitis was detected, which was acute at the beginning of the experiment (Fig. 13) and chronic at the end (Fig. 14). A follicular hypertrophy with lymphocytolysis and reticulohistiocytosis corroborated the macroscopic observations, especially at the end of the experiment. The medullar area showed a strong sinusal catarrh (Fig. 15).

During the first phases of the experiment the lungs showed a congestive aspect. However, at the end of the experiment this organ appeared greyish with signs of hepatization. Also, a marked enlargement of the intra and interlobular septa appeared. Catarrhal bronchitis with mucous secretion and leukocyte infiltration (Fig. 16), as well as a fibrinous bronchopneumonia, mainly in the cranial lobes, were microscopically observed. Both phenomena were very strong during the first days of the cycle (5 and 8 dpi) and progressively diminished to an interseptal cellular infiltrate, constituted by lymphocytes and histiocytes, after day 20 pi. This interstitial pneumonia, was more acute at the end of the experiment (Fig. 17).

Finally, the central nervous system showed a congestive aspect with wide vessels and presence of fibrin in the animals sacrificed during the first days. At the end of the experiment, it appeared "cooked" and degenerate. Microscopically, hyperemia and moderate perivascular oedema were detected with dilation of the Virchow-Robbins areas. After day 20 pi an encephalitic reaction was evident, with glia cells proliferation which placed around the neurone. Also neurophagy phenomena were detected (Fig. 18). This lesional picture became more acute at the end of the experiment (days 60 and 90 pi).

4. Discussion

Concerning to the outbreaks of human trichinellosis caused by horsemeat, a large number of researchers have carried out experimental infections in other herbivores like bovine or sheep (see Introduction). However, none of them have studied the alterations that *T. spiralis* may produce in the organs and tissues of these hosts. Perhaps the only one exception could be the histopathological research in the muscles of experimentally infected horses [31, 32]. The present paper is, probably, the first study related to the pathological picture produced by *Trichinella* in herbivores.

Due to the establishment of the new released larvae 1 into the intestine, the first lesions are evident both at macro and microscopic level. Macroscopic observations, such as congestion and abundant mucous secretion, were previously described [4, 38]. These organic reactions included the microscopic evidence of a catarrhal enteritis with cellular desquamation during at least the first 23 dpi, which were also described in mice [21, 25], and in dogs [23]. From day 30 to day 90 pi, a progressive chronic inflammation occurred, as well as RACE et al. [21] reported. The inflammation was always accompanied by hypertrophy of Peyer's patches, findings previously reported in mice [21], and in dogs [23]. These histopathological observations lead us to believe that the correspondence between the expulsion of the adults and the evolution of the enteritis. After expulsion, the acute desquamation became hyperplasic and chronic, while the hypertrophias of Peyer's patches remained until the end of the experiment. These findings could be related with an intestinal response to a toxic process produced by the larvae [6].

Before the arrival and establishment of the larvae at the muscles, the first alterations in this tissue became noticeable. So, hyperemia and interfascicular oedema were detected. These alterations were possibly produced by the aforementioned toxic process. Nevertheless, the lesions became obvious and much more accentuated after the larvae establishment. The main lesions observed were Zenker degeneration with intrafibrilar cysts from day 20 pi and granulomatus myositis with myofibrosis from day 30 pi. These lesions are also described by REINA et al. [23], PROKOPOWICZ et al. [20] and GUTOWSKA et al. [9], which found that the myositis appear very early (8, 10 and 14-15 dpi, respectively). However, it is universally accepted that there is a radical increase of the muscle lesions after the arrival of the larvae [5]. Coinciding with the findings of HULÍNSKÁ. et al. [10], at the end of the experiment (90 dpi) we observed a predominance of degenerative lesions in the skeletal muscles of the animals infected with *Trichinella* sp.
PATHOLOGICAL CHANGES IN GOATS EXPERIMENTALLY INFECTED WITH *TRICHINELLA SPIRALIS*

FIGURE 7. — Glycogen degeneration (90 DPI), x 350.

FIGURE 8. — Granulomatous hepatitis (90 DPI), x 250.

FIGURE 9. — Endothelomesangial-proliferative glomerulonephritis (45 DPI), x 350.

FIGURE 10. — Tubular necrosis (45-60 DPI), x 250.

FIGURE 11. — Interstitial nephritis (90 DPI), x 350.

FIGURE 12. — Chronic hyperplasic reactive splenitis. Follicular hypertrophy (45 DPI), x 250.
In our study, larvae of first stage were also found in the myocardium after day 23 pi, although in a few number. The lesions observed in this tissue were basically limited to the vascular processes, producing a granulomatous myocarditis, as previously described other researchers in other hosts [17, 35].

The presence of glycogen degeneration and small granulomas is similar to the ones found by REINA et al. [23] in dogs. With the exception of this paper we have not found other reference to this particular lesion. However, GABRYEL et al. [7] detected granulomatous infiltrate in mice infected with T. pseudospiralis.

Conversely to the findings of PAMBUCIAN and CIRO-NEAU [14] in rats, macroscopically we did not observe hepatomegalia in the experimental goats, merely a slight diffuse fading, as external expression of the glycogen degeneration.

It seems to be that the membranoproliferative and/or endotheliomesiangial-proliferative glomerulonephritis, as the main kidney lesions caused by Trichinella, are generally accepted [7, 33, 38]. The latter authors affirm that, after day 22 the first glomerular hypertrophy could be detected and from this date the intensity of this process increases, being very strong at day 56 pi. These observations coincide mostly with the evolution of glomerulonephritis observed in this experiment.

In the lymph nodes and spleen, hypertrophy has been reported [2, 7, 38]. This macroscopic observation, due to the reactive and hyperplastic lymphoidenitis and splenitis, is microscopically evident at the beginning of the experiment, but acquires strength at the end (after day 45 pi). Following the same evolution, we detected follicular hypertrophy, with a loss of lymphocytes and the presence of reticulohistiocytosis.

Vascular and inflammatory processes were evident in the lungs, where catarrhal bronchitis was frequently found. Nevertheless, and in spite of previous reports [8, 17, 35], evidence of hemorrhagic foci or oedema was not found. On the other hand, coinciding with the findings of these authors, the goats showed signs of bronchopneumonia which evolved to interstitial pneumonia with interseptal accumulation of histiocytes and lymphocytes.

Like URSELL et al. [35], we found oedema and hyperemia in the central nervous system of our goats. Furthermore, there was evidence of an encephalitic reaction [35]. These findings are in contradiction with the lack of morphological alterations described by GABRYEL et al. [7] in the nervous system of mice infected with T. pseudospiralis.

Considering all aforementioned alterations, it is possible to conclude that trichinellosis in goats provokes a widespread inflammatory reaction. The most important lesions are located in the intestine, liver, kidneys and striated muscles and their intensity bear a strict resemblance with the endogenous cycle of the parasite.

The histopathological findings observed in the animals of this experiment demonstrate that the goat is a perfect host to T. spiralis. The parasite may complete its biological cycle, producing some morphological alterations due to its biological and physiological needs, which are similar to those detected in other more common hosts.

Acknowledgments

The authors are very grateful to Mr M. GÓMEZ-BLÁZQUEZ and Mr G. FERNÁNDEZ for their technical assistance. Dr M. GARCÍA-ALONSO assisted in the English revision of the manuscript.

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PATHOLOGICAL CHANGES IN GOATS EXPERIMENTALLY INFECTED WITH *TRICHINIELLA SPIRALIS*


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