TISSUE REACTION OF THE LIVER OF CATTLE TO AN ARTIFICIAL OR NATURAL INFECTION WITH CYSTICERCUS BOVIS

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Abstract. An evaluation of the tissue reaction to Cysticercus bovis in the liver of calves with an artificial infection and that of oxen with a natural infection provided evidence for the fact that the liver of these animals responded to an infection with C. bovis with an extensive granulomatous reaction. A comparison of tissue reaction to cysticerci at an identical stage of development showed that the reaction of the liver parenchyma was much more intensive than that of the heart and the skeletal muscles. In the described cases, this intensive reaction caused the death of the cysticerci before the completion of their development. Evidence was obtained for the first time that also in the liver C. bovis was located in the lymphatic vessels.

One of the first reports on the location of Cysticercus bovis in the liver was published by Ostertag (1923 — ex van Logtestijn and Westendorp 1971). Le Coultre (1928), von Ostertag and Schönberg (1955), Ginsberg (1960), Chabasse and Genthon (1962), Koudela (1965, 1969), Mitchell (1967), Šlabais (1970) and Koudela and Trefny (1971) found solitary cysticerci in the liver in cases only of a massive infestation of the masseter, tongue, skeletal muscles and the heart. This exceptional incidence of C. bovis in the liver was supported by the results of an artificial infection of animals reported by Mango and Mango (1972) and Kozakiewicz (1977).

Two unusual cases of a massive liver cysticercosis were found by Ginsberg and Griewe (1959) at the Kenya abattoir. These authors regarded an occasional incidence of C. bovis in the liver only as quite common.

There is no description of liver reaction available in any of these studies. A brief account of the initial inflammatory reaction to C. bovis located in the liver of a calf was given by Silverman and Hulland (1961) in their paper on the development of cysticerci and the tissue reaction in the muscles.

MATERIALS AND METHODS

The material in which we evaluated the tissue reaction of the liver to C. bovis consisted of 6 tissue samples with cysts obtained from artificially infected calves. The infection experiments were performed by Dr. B. Machnická, Parasitological Institute, Polish Academy of Sciences, Warsaw. Two cysts were removed on day 83 p.i., 4 on day 102 p.i. A description of the experiment has been given by the first author (Šteřba 1974) in an earlier paper. Two additional tissue samples were obtained from killing cattle (oxen) with a massive spontaneous infection at the abattoir.

The tissue samples were fixed with 10 % neutral formol and treated with standard paraffin techniques. Either partial or complete series of histological sections were made of the material from experimental infections. Calcaneous formations were treated with a method suggested by Šlabais (1960, 1970) and a modification of these procedures (Šteřba and Šlabais 1972, 1974). Of the staining methods we used haematoxylin eosin, van Gieson and van Gieson's elastic method, Masson's method and Goldner's method complemented with Gomori's impregnation method, Kossa's method for the detection of calcium, PAS for mast cells.
RESULTS

Cysts recovered from the parenchyma on day 83 p.i. contained necrotic and collapsed cysticerci; their parenchymal part was not yet enclosed in the bladder. Although otherwise conform to young developmental stages, their scolex was evaginated.

Cysticerci at the same stage of development were found three times in cysts recovered from the liver on day 102 p.i. One cyst of the same age contained a more advanced stage, i.e., an incompletely enclosed cysticercus. From this experimental infection we obtained either live cysticerci (3 times) or those in which a necrobiotic had started. Two of these cysticerci were located intraparenchymally, two subcapsularly (Plate I, Fig. 1).

The developmental stage of cysticerci obtained from the liver of spontaneously infected animals could not be identified, the reason being an advanced necrobiotic and resorption.

We found that all cysticerci in our samples were located in the lymphatic vessels of the liver. The wall of their cysts originated from the walls of these vessels. (Plate I, Fig. 2A, B). Typical of the tissue reaction were inflammatory changes originating in a topical relationship to the parenchymal part of the parasite (Plate II, Fig. 1).

Endothelial cells disappeared from the dilated lymphatic vessel at the site of the tissue reaction, and there originated an inflammatory rim. The character of its inner zone was that of a pseudoepithelial border with a dominance of histiocyctic cells, fibroblasts and containing an occasional plasmocyte and both neutrophilic and eosinophilic leukocytes. Towards the periphery, the border passed into a differently developed zone of granulation tissue entering irregularly the pseudoepithelial border and thus producing folds which bulged into the cavity at various sites.

The quality and extent of the inflammatory reaction to the four cysts aged 102 days were not completely uniform. Sometimes, the structure of the lymphatic vessels was untouched, sometimes, vessels invaded by the parasites were thickened by connective tissue and succumbed to a segmentary or circular granulomatous histiocytic destruction (Plate III, Figs. 1, 2). In some parts, the focal granulomatous destruction was so much advanced that the lymphatic vessel could no longer be identified.

A granulomatous inflammatory destruction of the lymphatic vessel occurred always at the site facing the parenchymal part of the cysticercus. It was composed of epitheliodal histiocytes with newly formed argyrophilic fibres and an occasional multinucleate giant cell among them. The inner part of this section was covered with a fibrinous membrane. The inner layer of the histiocytic reaction became necrotic and the same occurred in the deeper layer. The width of the segmentary or circular histiocytic reaction was unequal. The pseudoepithelial border was folded. The outside of the histiocytic reaction was bordered by a lymphocytic infiltration, and, over a small distance, the latter was overlaid by a strip of cellular collagenous connective tissue.

The cellular composition of the granulomatous reaction differed in some details in the various sites. Sometimes, the nuclei of a larger number of multinucleate giant cells were organized in rings or densely packed (Plate III, Fig. 2). Elsewhere, the lymphocytic border adjoining the histiocytes was remarkably thick, and there was a sparse distribution of plasmocytes among the lymphocytes and lymphoblasts. The necrotic exudate inside the disintegrated lymphatic vessel sometimes contained foci of dystrophic calcification. Similarly, calcification occurred in the inflammatory infiltrate abutting the damaged lymphatic vessel.

In one of the four samples, we recovered a C. bovis from the site of bifurcation of the lymphatic vessel. Its parenchymal part was extended into one branch of the
vessel, the other branch contained the terminal portion of its bladder only. The segmentary granulomatos reaction was more intensive in the area surrounding the scolex portion of the incompletely enclosed cysticercus. The bladder portion was surrounded by a small quantity of cellular exudate affected partly by necrosis.

An occasional focal necrosis of hepatocytes which were to some part surrounded by histiocytes and lymphocytes was seen in the vicinity of the liver parenchyma. At the site of a necrosis of the hepatocytes, we observed an occasional granuloma. Cysts obtained from the liver parenchyma on day 38 p.i. displayed a marked affinity to the portal area. In both instances, the dilated lymphatic vessels contained necrotic and collapsed cysticerci surrounded by a large quantity of purulent exudate. At some sites, the pus was disintegrating necrotically or succumbing to a crumbling dystrophic calcification. The heavily infiltrated necrotic tissue formed a wide border containing sites of calcification and dystrophically changed multinucleate cells. On the periphery, the necrotic tissue passed into a zone of granulation tissue heavily infiltrated with lymphocytes which contained also multinucleate histiocytes and multinucleate giant cells (Plate IV, Fig. 1). The granulation tissue entered the necrotic tissue by means of palisade-like organized histiocytes (Plate IV, Fig. 2).

A similarly extensive granulomatos reaction was seen in the liver of animals with a spontaneous infection. However, in these cases the degree of necrosis and resorption was too advanced to identify the developmental stage of the cysticerci.

**DISCUSSION**

It was found that liver reacted more intensively than the heart and the skeletal muscles to the presence of *C. bovis* (Štěrba 1974), and that in the first, the reaction was granulomatus in nature. The differences in the intensity of the reaction emerged from the results of a comparison of tissue reaction in calves of the same experiment (the same infective dose and duration of infection).

In all cases this intensive tissue reaction was responsible for the death of most of the cysticerci before the completion of their development. However, according to our findings in the same material and to data in the literature (Ginsberg and Griewe 1959, Price 1961, Mango and Mango 1972, Kozakiewicz 1977), a *C. bovis* is capable of attaining its infective stage in the liver. Since we had to be certain that these data could be relied upon, we considered those only given by authors who had either tested the viability of the parasite or made a histological examination of the cyst based on a detailed knowledge of the morphology and morphogenesis of *C. bovis*. A similar influence of the heart on the development of *C. bovis* to that of the liver was observed by Štěrba (1974) and Machnicka et al. (in press). By contrast, the character of the reaction of the skeletal muscles was that of a sectorial inflammation and the majority of cysticerci located in these muscles attained their infective stage within the same span of time (Štěrba 1974, Machnicka et al., in press).

The location of *C. bovis* in the liver was identical to that in the skeletal muscles and in the heart, i.e., it was found in the lymphatic vessels. The growing parasite dilated the lymphatic vessel over a short distance and formed the wall of its cyst from the wall of the vessel.

**ТКАНЕВАЯ РЕАКЦИЯ В ПЕЧЕНИ СКОТА ПРИ ЭКСПЕРИМЕНТАЛЬНОМ И ЕСТЕСТВЕННОМ ЗАРАЖЕНИИ ЦИСТИЦЕРКАМИ CYSTICERCUS BOVIS**

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**Резюме.** При изучении тканевой реакции на *Cysticercus bovis* в печени экспериментально зараженных теленков и естественно зараженных быков было показано, что локализация
C. bovis в печени вызывает большую гранулематозную реакцию. Сравнение тканевых реакций на цистицерки одинакового возраста показало, что тканевая реакция в паренхиме печени значительно более интенсивна, чем реакция в сердце и в скелетной мускулатуре. В описанных случаях интенсивная тканевая реакция была причиной отмирания цистицерков перед окончанием их развития. В первый раз было показано, что даже в печени C. bovis локализован в лимфатических сосудах.

REFERENCES


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Fig. 1. *C. bovic* in a subcapsular location in the liver of a calf. Haematoxylin-eosin (× 27).

Fig. 2A. Part of the cyst wall with the preserved endothelium of the dilated lymphatic vessel (L): bladder of *C. bovic* (B) (× 200).

Fig. 2B. Detailed view on the endothelium of the lymphatic vessel. Haematoxylin-eosin (× 500).
Fig. 1. Inflammatory reaction originating in a topic relationship to the parenchymal part of *C. bovis* (P). Haematoxylin-eosin (× 150).

Fig. 2. Developed inflammatory reaction around the entire cysticercus. Haematoxylin-eosin (× 30).
Fig. 1. Granulomatous reaction in an inflamatorily thickened lymphatic vessel. Haematoxylin-eosin (× 150).

Fig. 2. Concentration of multinucleate giant cells in the granulomatous reaction. Haematoxylin-eosin (× 300).
Fig. 1. Necrosis of the cyst contents. Part of a necrotic *C. bosis* (C). Haematoxylin-eosin (× 20).

Fig. 2. Granulation tissue penetrating the purulent exudate by means of palisade-like organized histiocytes (H). Haematoxylin-eosin (× 135).