

## IMMUNOMORPHOLOGICAL CHANGES IN EXPERIMENTAL BOVINE CYSTICERCOSIS\*

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### Introduction

Studies on bovine cysticercosis revealed that, the cysticerci (*Cysticercus bovis*) evoke a marked tissue reaction which at one stage or the other, may lead to the dystrophy of the parasite, (Silverman & Hulland, 1961; Sliac, 1971; Kozakiewicz, 1977). On the other hand, different antibody responses were detected in cattle infected with *C. bovis* (Mocina, 1965; Machinicka et al, 1977). However, immunomorphological changes in different organs, at various periods post infection were not thoroughly investigated.

This paper describes the immunomorphological changes and tissue reaction in different organs following the course of experimental bovine cysticercosis.

### Materials and Methods;

Eggs of *Taenia saginata* freshly recovered from gravid proglotids, were suspended in saline. Viability of oncospheres was tested according to the method described by Silverman (1954). Eight calves 3-5 months old free of *C. bovis* infection were each given orally 300,000 viable oncospheres. Two of the infected calves (No. 1 and No. 80) were challenged with a single dose of 600,000 oncospheres, 20 and 40 days after initial infection. Two additional calves were kept, as non-infected controls. Post-mortem examination of animals was done at different periods of 15 to 120 days post infection. Tissue samples were taken from various organs (skeletal muscle, heart, liver, lung intestine, lymph nodes and spleen), for histopathological and histochemical studies.

The specimens were fixed in 10% neutral formalin, or Carnoy's fixative solution, embedded in paraffin wax & sectioned at 6-7 microns thick. Sections were stained with Haematoxylin and Eosin according to standard procedures. Besides, the following histochemical and staining methods were employed: glycogen, periodic acid-Schiff reaction (Mcmanus, 1946); sulfomucins, Steedman Alcian blue method

(Pearse, 1968); heparin of mast cells, toluidine blue method (Chybush, 1961); ribonucleic acid (RNA), methyl green pyronin (Brachit methos, cited in Pearse, 1968).

### Results.

Tissue sections revealed that, the histopathological picture of the organs from infected animals varied according to the stage of development, or the degenerating process in the cysticerci. In the initial phase of infection, host response was that of an early inflammatory reaction, and was fairly intense. The cysticercus bladder was sheathed with a thin capsule of circular connective tissue fibres. The capsule was infiltrated with cells which were accumulated between the cysticercus wall and the capsule. In later stages of infection, tissue reaction was more intense, with characteristics of a strong inflammatory reaction and the appearance of parasitic granuloma (Fig. 1). Viable cysts were surrounded by dense fibrous connective tissue capsules with numerous histiocytes and lymphocytes. Plasma cells and eosinophils were less abundant, giant cells and macrophages were very rare. Muscle fibres surrounding the cysts were showing degeneration. In site of dead and disintegrating cyst, there was a centrally-lying necrotic area enveloped with a dense sheath of connective tissue, with few lymphocytes, plasma cells and giant cells. Such necrotic foci were more abundant in organs of reinfected calves.

In lymph nodes & spleen, host response to infection was characterised by hyperplasia of lymphatic nodules, proliferation of reticuloendothelial cells, mitotic activity and increased number of macrophages, (Fig. II). These changes were coupled by a clear transformation of reticulo-endothelial and lymphoid cells into plasmoblasts. This transformation process was accompanied by a strong pyroninophilic reactivity in the cytoplasm of these cells. Maximum increase in number of pyroninophilic cells (Plasmoblasts) was observed 45-60 days post-infection (p.i.). At this stage, both mature and immature plasma cells were abundant, whereas at 130 days p.i. mature plasma cells were more apparent (Fig. III).

The lamina propria of the intestine displayed an increased number of mast cells and eosinophils. In later stages of infection, the number of these cells decreased and eventually became very rare.

Histochemical studies revealed various decreases

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in glycogen content of liver and affected muscles. In early stages of infection, there was a considerable decrease in glycogen content of hepatic cells, whereas 60 days post infection, their glycogen content was still less than that of control animals, (Fig IV). In calf No. 12, killed 130 days post infection, the glycogen content of hepatic cells was very close to normal. Muscle fibers around the cyst capsule also showed marked decrease in glycogen content. At the same time, intense accumulation of glycogen granules was observed at the subcutaneous layer of the cysts.

### Discussion

Basic pathomorphological reactions in bovine cysticercosis were observed in the liver, skeletal muscles, heart, lung, intestine, lymph nodes and spleen. These changes in animals experimentally infected with the cysticerci of *Taenia saginata* appear to be due to an inflammatory reaction of an allergic nature. In the early stages of infection, the tissue damages were due to the mechanical trauma caused by the migrating parasites, while in later stages they were caused by the allergic reaction of the cysts on the host tissue.

During the early stages of invasion the inflammatory reaction was characterized by cellular infiltration and haemorrhagic lesions. At this time polymorphonuclear leukocytes surround the thin connective tissue capsule forming around the cysticerci. In later stages, the inflammatory reaction was characterized by a marked increase of eosinophilic leukocytes. The commencing granulation tissue on the cysts wall was consisting of lymphocytes, neutrophils, giant cells and fibroblasts. These inflammatory reactions with the development of granulation tissue around the sites of cysticerci indicate an immunomorphological response of the host (Ershov and Naymichva, 1970). Atrophy of cardiac and skeletal muscle fibers around the cysticerci was believed to be due to pressure imposed on them by the parasite. Character of the host tissue reaction to *C. bovis* and its gradual development suggests a reaction to the live parasite. It was observed that, the mode of development of the cysticerci depends on the intensity of the host's tissue reaction, i.e. the stronger the reaction, the less advanced the development of the cysticerci. This agrees with the findings of Silverman & Hulland (1960), and Silverman (1971). Thus the immune response to cysticercosis appear to be a form of delayed hypersensitivity reaction which results in an increased production of lymphocytes, macrophages and the appearance of parasitic granu-

loma. Such cellular reaction with the cysticerci may directly lead to their destruction or tend to limit their development.

The transformation process of lymphoid cells into plasmoblasts with the strong pyroninophilic reactivity observed in lymph nodes and spleen, appear to be directly related to the immunomorphological state of the infected animal. The plasma cells are said to be the main antibody-producing cells. The dynamic state of this reactivity coincides with the high titre and increased globulin fractions in serum of calves being infected with *C. bovis*, (Gallie and Sewell, 1974). An increased eosinophilic oxidation and mast cell accumulation in organs of infected animals was also observed. According to Astafaev (1975), such reactions in parasitic infestation are indicative of the allergic response of the host to the invasion.

Histochemical studies showed that together with histological changes in affected muscles and liver, glycogen content in these organs decreased at varying levels. Up to sixty days post-infection, levels of glycogen content in hepatic cells of infected calves was less than that of control ones. However, gradual normalization, was observed during later stages of infection. The decrease in glycogen of the above mentioned organs may be attributed to the pathogenic effect of the parasite on the liver glycogen synthesis, and partially due to decreased fermentative and absorptive activities of the gastrointestinal tract.

### Summary

Histopathological and histochemical studies on experimental bovine cysticercosis revealed that, host response to infection with *Cysticercus bovis* was an inflammatory reaction manifested by cellular infiltration and the formation of parasitic granuloma. The reaction varied according to the stage of infection and was accompanied by obvious histochemical changes in tissues and organs of infected animals. Distinct immunomorphological changes were also observed in lymph nodes and spleen.

### References

- Astafaev, B.A. (1975): *Meditcina*, Moscow.
- Ershov, V.S. and Nayminchiva, M.I. (1970): *Gelmint Seleskokhoz. Jevot. (Itogi Nayk)*, 5-41.
- Gallie, G.J. and Swell, M.M.H. (1974): *Trop. Anim. Hlth. Prod.* 6, 163-171.
- Ivranova, V.G., Mosina, S.K., and Ivranova, G.V. (1965): *Uchep. Zap Kazan. Vet. Inst.*, 96, 218-223.
- Kozakiewicz, B. (1977): *Acta Parasit.*, 24, 34, 357-369.

Machnicka, B., Sliac, J., Zdrska, Z. Schramlova, J., Hulinska, D. and Sterba, J. (1977): *Acta Parasit.*, 25, 55-62.

Mosina, S.K. (1965): *Uchep. Zap. Kazan. Vet. Inst.*, 94, 123-126.

Pearse, A.G.E. (1968): *Histochemistry, Theoretical and Applied*. Little, Brown and Co., London.

Silverman, P.H. (1954): *Ann. Trop. Med. Parasit.*, 48, 207-215.

Silverman, P.H. and Hulland, T.J. (1961): *Res. Vet. Sci.*, 2, 3, 248-252.

Sliac, J. (1970): *The Morphology and Pathogenesis of the Bladderworms: Cysticercus cellulosae and Cysticercus bovis*. Academia Praque, Praque, 144 p.



Fig 1: Granuloma in heart muscle of a calf 60 days after infection.

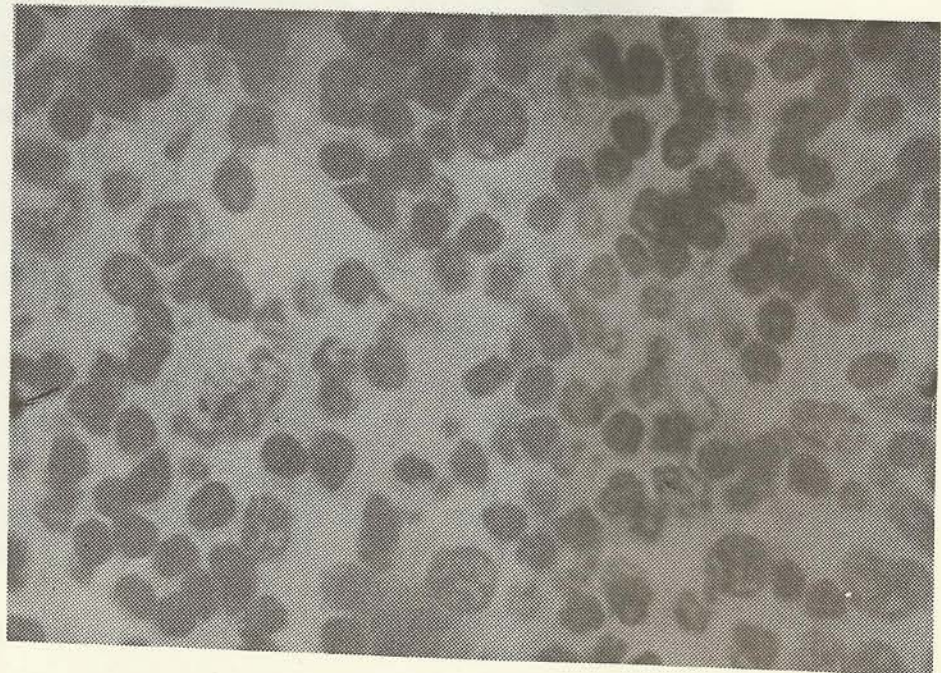


Fig 2: Section of a lymph node showing increased number of macrophages and plasmoblasts.

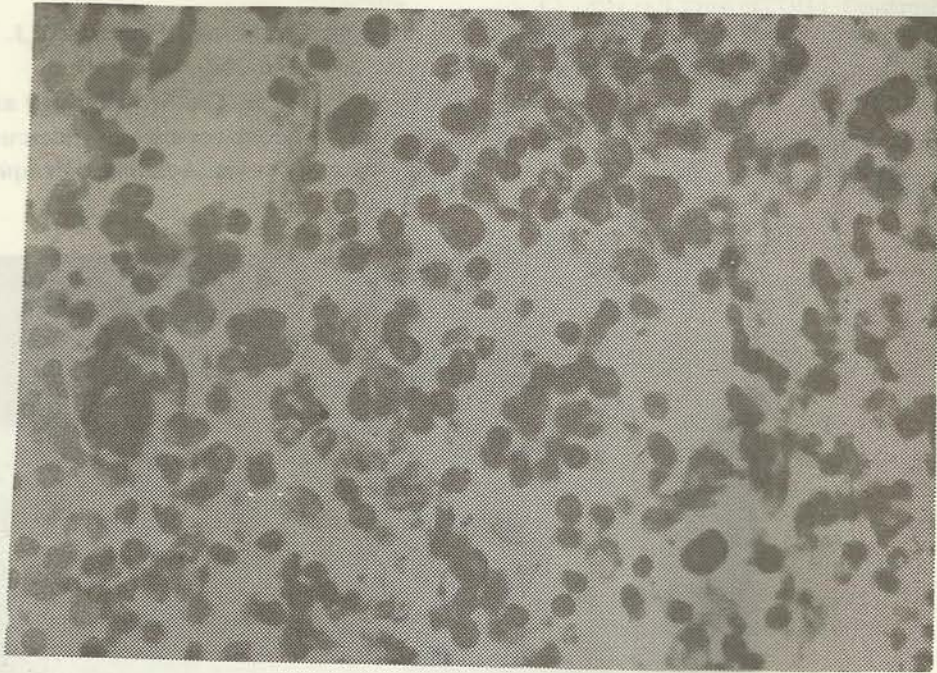


Fig 3: Accumulation of plasma cells in the sinuses of a spleen of an infected calf.

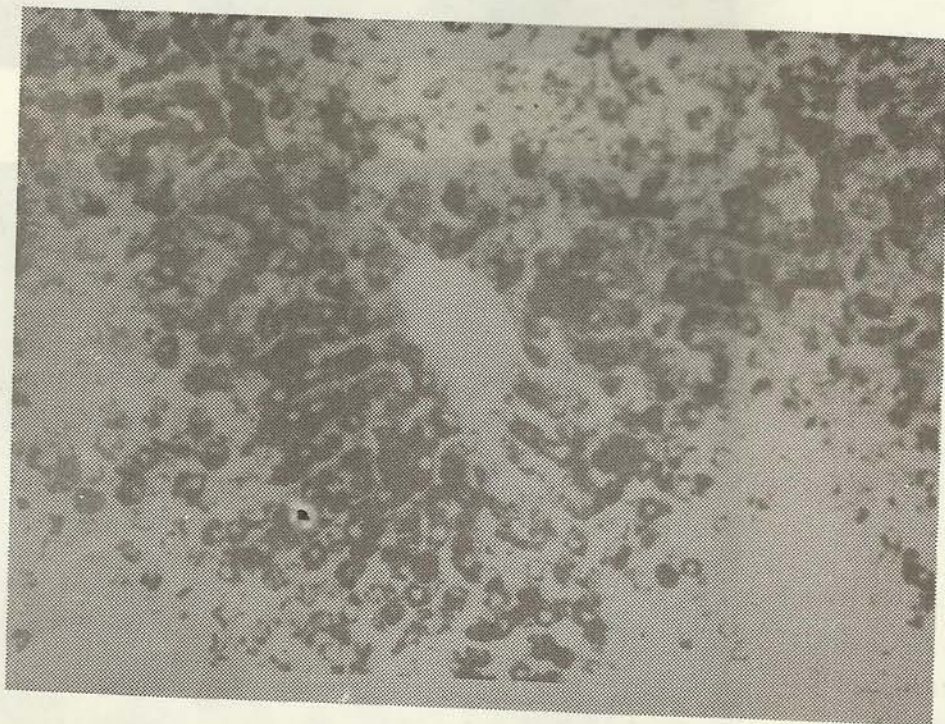


Fig 4: Decreased glycogen content of hepatic cells of an infected calf.