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STRUCTURAL MODIFICATIONS OF THE SHEEP LIVER PARENCHYMA IN FLUKE INFESTATION

BY

DIANA GHEŢEU[1], ANCA NEAGU[1], GIANINA COMĂNESCU[1]

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A light microscopical examination was made of the liver tissues în fluke infestation (*Fasciola hepatica*) at sheep.
The microscopic examination revealed the multiple modifications of hepatic tissues in acute and chronic invasion: hemorrhage, vacuolation, necrobiosis of hepatocits and replacing newly formed connective tissue; the chronic form leads to hepatocirros.

Introduction

Sheep and other fluke receptive species almost permanently and concomitantly have numerous species of parasites of which pathogenic action compete at the economic losses (Olteanu, 1973). The cosmopolitan presence of intermediate hosts in their different types of biotops explain the permanent possibilities of flukes infestation in goats, sheep, cattle, small and bigger mammals including human being; a well known epidemic episode has been registrated in France, region of Lyon between 1956 – 1958 (Olteanu,1996). In Romania the first infestation in human being was observed by Nicolae Leon in 1908 (Olteanu, 1996).

The pathogenic action of *Fasciola hepatica* begins with the ingestion of encysted metacercaria (the stage preceding the adult) with vegetation or freshwater (Dulceanu, 1982).

The diagnosis can be made on the gall bladder and bile duct (Olteanu, 1973; Lee,1992) where adult flukes may be observed in chronic invasion (Olteanu,1973). Hemorrhagic (small red) points on Glisson's capsule indicate an acute invasion (the perforation made by young flukes penetrating this capsule) (Dulceanu, 1996; Olteanu, 1973 et al.).

Materials and Methods

The pieces of liver with flukes were fixed for light microscopy in 10 % formaldehyde and Bouin mixture, dehydrated in ethanol and amylic alcohol, embedded in paraffin and sectioned at 5 microns. They were stained with hemalaun-eosine and examined with a NOVEX microscope and photographed with a Canon EOS 1V.

[1] „Al.I. Cuza" University of Iaşi

Results and Discussions

The helminthoma reveals the liver fluke fixed with the oral sucker in the lumen of the biliary radicle: we can see the destruction of the radicle cubical cells (Fig. 1). The internal part of the helminthoma has white blood cells, nearby, collagen fibers, indicating an older lesion (Paul, 1982); the external zone is limited by a fibrillary area (Fig. 1.a) detailed in Fig. 2, where we can see a massive fibrosis, the result of isolation reaction of the host (Paul, 2001). A massive lymphatic infiltration, as a defense reaction (Paul, 2001) to a foreign body of parasitic provenience can be seen in Fig. 3. The parasitic granuloma is immunitary granuloma (Crespeanu, 1992).

Young flukes migrate through the digestive walls to the liver, penetrate Glisson's capsule. Its target is biliary radicles. They can also locate in the lungs (Paul, 2001) and other organs of the host.

The prints of the migration through the liver tissue are marked by destruction of the normal architecture of the liver lobules.

The spines prints (Fig. 4.a) indicate a recent location of the fluke which produced a massive hemorrhage (Fig.4.b) and destruction of the tissue. We can see also a regeneration center limited by connective tissue (Fig. 4.c) nearby vacuolization of cells, first reversible lesion of an aggressed cell (Teleman and others, 1998) that are increased their volume - binucleate hepatocites (Fig. 4).

Some cells near the path of the fluke have increased their metabolism and their hepatin use, so, in HE coloring the liver tissue aspect become similar to a sponge. The cells membrane become more permeable to serum albumin and globulin that will go into the mitochondria so the cytoplasm of the liver cells become granular (Paul, 1987). After that they enter in necrosis (Fig. 5).

In chronic infestation with flukes the general result is hepatocirrosis from which a microscopic aspect (Fig. 6) reveals the disorganized limit of lobule (Fig. 6.a), newly formed blood vessels (Fig. 6...b) and connective replacing all ill areas (Fig. 6.c).

Usually a new (acute) infestation upon the chronic disease (us we discovered in our cases) leads to the death of the animals (Olteanu, 1973).

Conclusions

In their migrations, young flukes destroy the tissues they met with their spines, causing massive hemorrhage. The destructed vessels are immediately replaced but the hepatocits are increasing their metabolism, become vacuolated and finally enter in necrobiosis because of the hipotrophia. The architecture of normal lobule is changed, newly formed connective tissue replace the destructed areas and the final results can be the hepatocirrosis. Regeneration of small liver affected territories is possible after the acute faze of fasciolosis.

In the case of helminthoma, the result of host-parasite relationship, if the parasite is fixed in the gallbladder, can obstruct it. The helminthoma indicate a chronic invasion.

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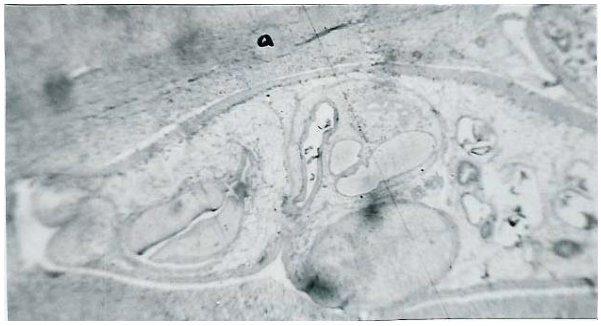

Fig. 1. Helminthoma with *Fasciola hepatica*: it can be observed the destruction of cubical cells of the radicle, a) fibrosis (3,3X4) HE

Fig. 2. Fibrosis - detail (3,3X100) HE

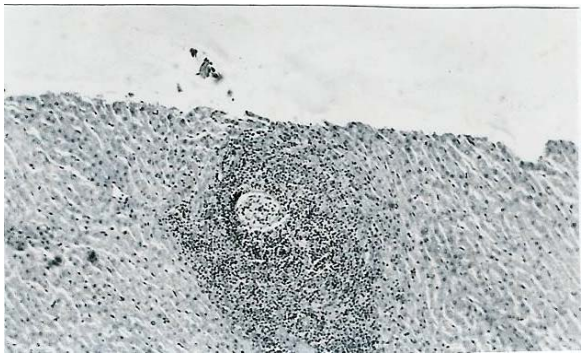

Fig. 3. A massive lymphatic infiltration, as a defense reaction to a foreign body of parasitic provenience (3,3X10) HE

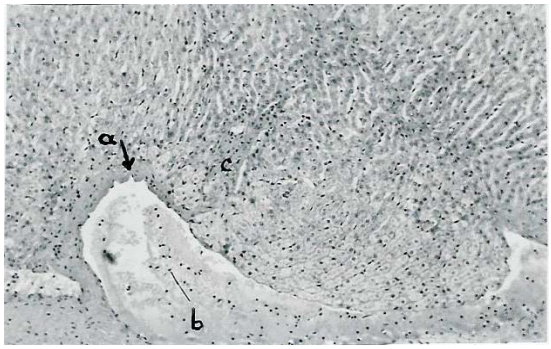

Fig. 4. Modifications liver structure: a) prints of the spines, b) hemorrhage, c) connective tissue limiting a regeneration center, nearby, the vacuolization of hepatocites (3,3X40) HE

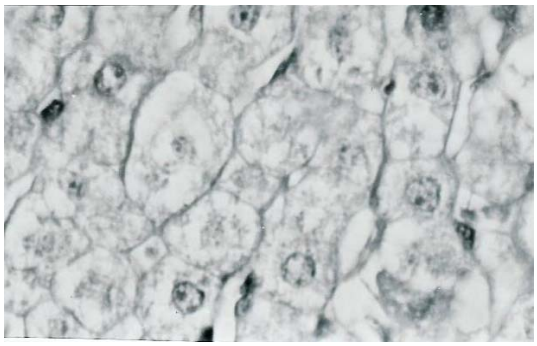

Fig. 5. Vacuolation and necrobiosis of hepatocits (3,3X100) HE

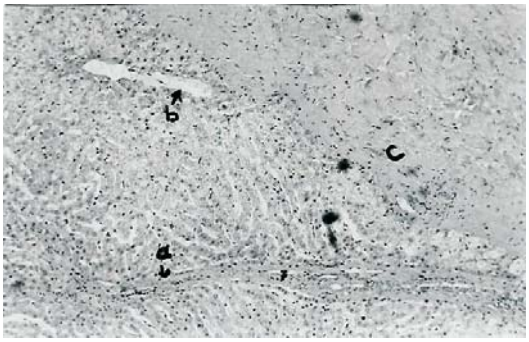

Fig. 6. Hepatocirrosis: a) limit of destructed lobule, b) blood vessel, c) newly formed connective tissue replacing ill areas (3,3X10) HE

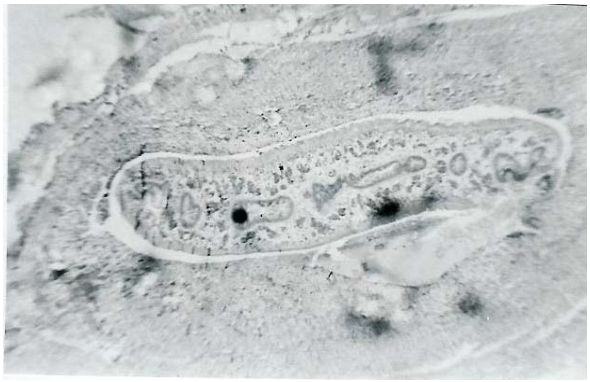

Fig. 7. Helminthoma with *Fasciola hepatica* - cross section (10), HE