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Histopathological changes of the liver in hepatic parasitosis of animals

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ABSTRACT

The liver is one of the body's primary organs, where you can get information about metabolism. Nutrients enter this organ from the gastrointestinal tract, where the most concentrated and active metabolic processes occur. The liver synthesizes complex organic substances such as proteins and glycogens. Non-acidic and toxic compounds are sterilized in the liver, and complex compounds are broken down into simple compounds. We found pathological changes in the liver in animals with fascioliasis and echinococcosis. In this regard, studying histological changes in this organ has become especially important for us.

Keywords: Liver, Parasite, Histopathology.
Article type: Research Article.

INTRODUCTION

Liver trematodiasis is common in various regions of the Republic of Kazakhstan (Marcos *et al.* 2008; Ibironke & Fasina 2010; Talukder *et al.* 2010; Usip *et al.* 2014, Rana *et al.* 2014; Mukhamejanova *et al.* 2021; Aubakirova *et al.* 2023; Satkanov *et al.* 2024). According to Russian sources (Meiramkulova *et al.* 2017; Darzhigitova *et al.* 2021), foci of liver trematodes still exist in various climatic zones of the country. This is due to the ineffectiveness and untimely implementation of therapeutic and preventive measures, which leads to an increase in the formation of biotopes of intermediate and additional hosts of trematodes in pastures. Some helminthiasis can simultaneously parasitize both animals and humans. Such diseases are called helminthozoanthroponoses. Zoonotic nosologies (fascioliasis; according to WHO and OIE) are recognized as a global biological threat to animal husbandry among the parasitic infestations of animals and humans worldwide. Trematodiasis of the hepatic, biliary, and pancreatic tracts are widespread and cause serious human diseases, often leading to disability and death of patients (Darzhigitova *et al.* 2021; Aubakirova *et al.* 2023). International Agency for Research on Cancer (IARC) stated that the causative agents of trematodes belong to the carcinogens of the first category; that is, there is convincing evidence of carcinogenic effects (Meiramkulova *et al.* 2017). The high incidence of trematodiasis not only in domestic animals but also in humans in the Republic of Kazakhstan is due to a thorough and comprehensive study of their pathogens. Echinococcosis, is a zoonotic progressive parasitosis caused by the tapeworm granulosus. It is a widespread disease. Epizootic and epidemic situations of echinococcosis in Kazakhstan remain difficult;

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therefore, the organization of measures to combat it is an important issue in veterinary medicine. According to medical and veterinary reports, the disease is recorded annually through sporadic conditions. The territories of West Kazakhstan, Turkestan, Kyzylorda, and West Kazakhstan regions are the most unfavorable regarding the prevalence of echinococcosis. In the general structure of the incidence of echinococcosis, the hepatic localization of parasitic vesicles is first associated with the peculiarities of blood flow in the portal vein, that is, up to 85%. Among the lesions of various organs and tissues with echinococcosis, the incidence of liver damage is estimated at 44-84% (Badr & Nasr 2009; Muirson 2011). *Fasciola* causes an inflammatory process, which is a consequence of both mechanical action and tissue irritation by toxins. It inhibits the digestive system and the protective functions of the animal body, which promotes the penetration of pathogens of other etiologies (viruses, bacteria, protozoa) and contributes to mixed diseases. Catarrhal cholangitis first develops at the site of *Fasciola* migration in the bile ducts, followed by biliary cirrhosis. Publications on parasitic liver diseases are limited to data on the study of the spread of parasites, their effects on animal productivity, hematological parameters, and pathoanatomic changes in organs, as well as therapeutic and preventive measures and economic damage in this disease. However, there still needs to be data on a comprehensive study of the effect of liver parasites on the liver at the histological and ultrastructural level. More data on the effects of parasitic liver diseases on morphological changes in the liver of animals determined the choice of the topic and the direction of the study (Hunt & Kind 1997; Shaikh *et al*. 2004; Ansari-Lari & Moazzeni 2006; Coppo *et al.* 2011).

MATERIALS AND METHODS

In 2018-2023, a histological study of part of the liver parenchyma of animals, chitinous and fibrous membranes of echinococcal vesicles was conducted based on the Faculty of Natural Geography of the M. Utemisov West Kazakhstan University. Liver biopsies were obtained during the slaughter. Some 4-6 liver biopsies (including a fibrous capsule) were obtained from each animal with echinococcosis at a distance of 1 cm to 8 cm. The biopsies were placed in a sterile glass jar filled with 76% ethyl alcohol. Histological preparations made by the standard method for examining the condition of liver tissue were stained with hematoxylin-eosin in a Leica Autostainer XL apparatus with histopreparation placed under a cover glass on a Leica CV 5030 apparatus. The stained areas were examined under a Leica light microscope with ten glasses at a magnification of lenses 5, 40, and 100. We have compiled a detailed morphological description of each drug, determining the pathological process's degree of development. The most interesting objects were photographed using the Infinity 1 digital video camera using a visual image analysis system and stored in computer memory.

RESULTS

In animals with fascioliasis, histopathological studies of the liver show characteristic changes in all tissue structures and cellular elements of the liver. Hepatocytes, which comprise the main number of cellular components of the liver, are characterized by a polygonal structure. The cytoplasm has a granular structure and is colored with basic and acidic dyes. The cytolemma is clearly defined, the cells are in close contact with each other. The nucleus of hepatocytes, as a rule, occupies a central position, and fine-grained chromatin nuclei are revealed. Polyploid nuclei are often found, especially with increased dual-core hepatocytes. Histological examination of the liver of sick animals in various combinations showed characteristic changes in the form of dystrophy and necrobiosis of hepatocytes. Separate sections of the liver lobules were formed through a largely destructive process. At the same time, the hepatocytes located in the central part were completely destroyed, hepatocytes with individual signs of destruction were found on the periphery, and areas with fragmentation of the liver plates were also found (Figs. 1-2). The inflammatory process in the area where the hepatic triad is located is especially characteristic when lymphoid cells are concentrated with varying intensity in the surrounding loose connective tissue. First, they surround the intestinal tract of the liver, as well as the intrahepatic arteries and vessels. An inflammatory reaction is detected both in the intestinal bile duct and the liver's excretory duct (Fig. 3). A parallel study of the carbohydrate metabolism of the liver, the central organ of glycogen accumulation, revealed a significant change in histochemical reactions. The liver's pivotal role in glycogen accumulation is underscored by the fact that, in general, a high reaction to glycogen is detected in hepatocytes. However, among the hepatocytes of the liver lobules, there are those that exhibit a weak reaction to glycogen or are even inactive (Fig. 4). The most striking observations are the alterations in the sinusoidal capillaries, the blood vessels between the hepatic plates that transport blood to the central vein. These changes in the liver's microcirculatory channel, including complete bleeding of blood vessels and a slowdown in blood flow, have significant implications (Fig. 5).

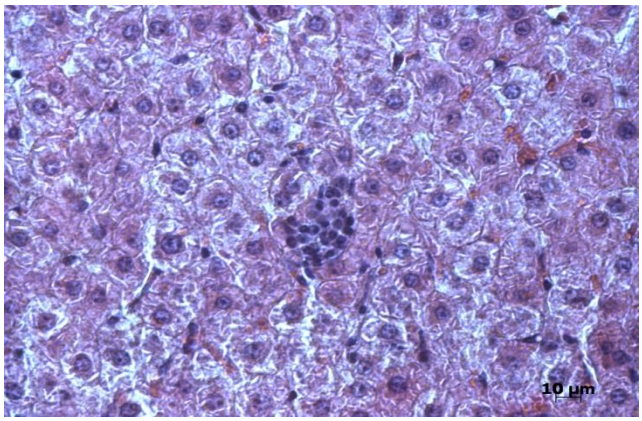

Fig. 1. Destruction of hepatocytes and lymphoid cells in the liver by bovine fascioliasis. Staining with hematoxylin and eosin. Micrography. Ok. 10, vol. 40 63.

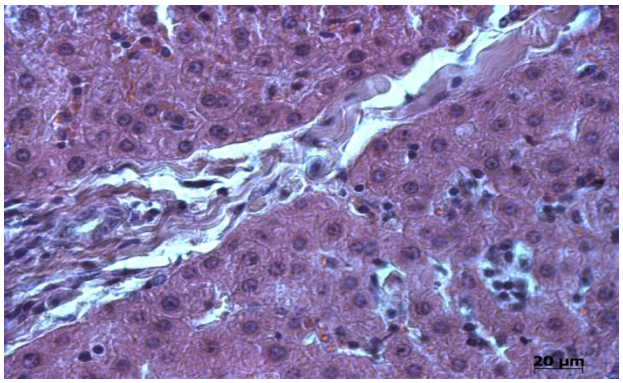

Fig. 2. Destructive area and lymphoid cells in the liver of cattle affected by Dicrocelia. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40.

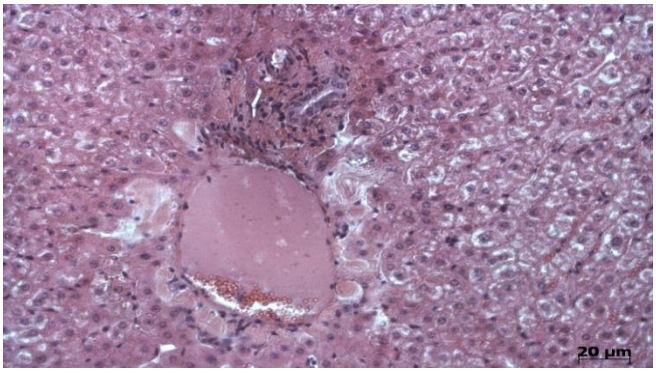

Fig. 3. Inflammatory reaction of the lobular bile ducts of the liver in bovine fascioliasis. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40.

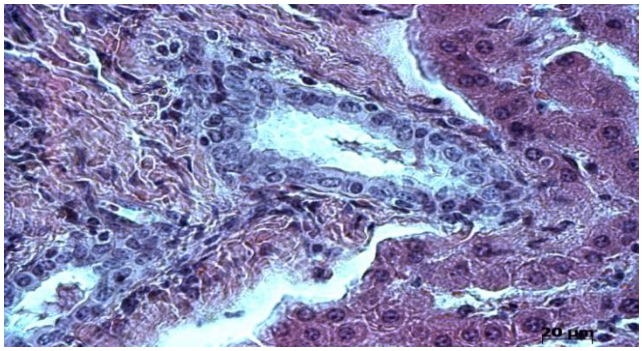

Fig. 4. Weak reaction to glycogen in individual hepatocytes of the liver in bovine fascioliasis. Staining with hematoxylin and eosin. Micrography. McManus's reaction. Ok. 10, about 40.

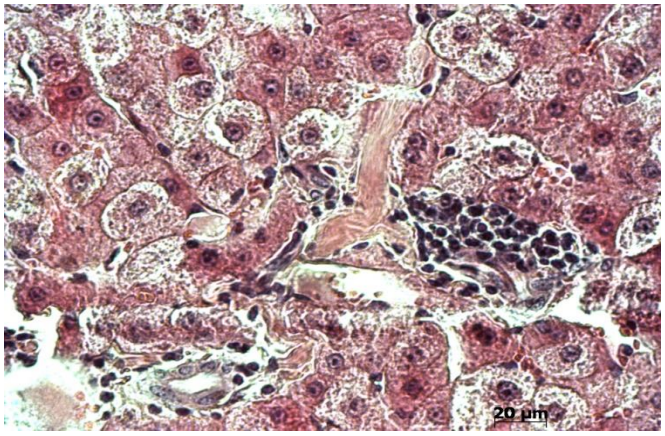

Fig. 5. Expansion of sinusoidal capillaries of the invaded liver of animals. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40

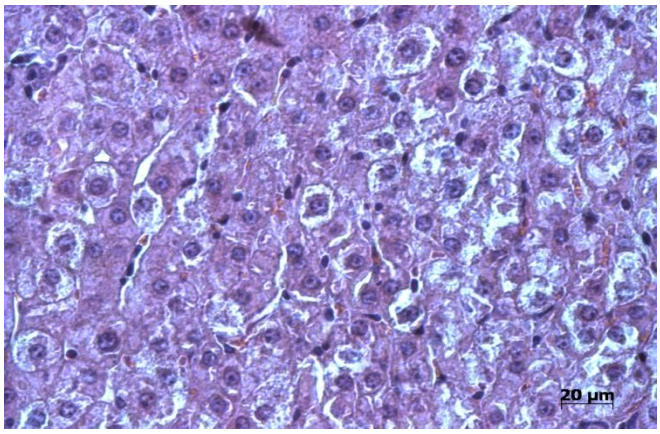

Fig. 6. Macrophages of intravesical sinusoidal capillaries of liver lobules with fascioliasis of animals. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40

Fig. 5 shows that the hepatic plates are separated from each other due to the whole blood of the sinusoidal capillaries. In addition, there is a perisinusoidal edema, an expansion of space. The endothelial cells forming intracinusoidal capillaries carrying mixed blood (arterial and venous) are significantly thickened. Macrophages (Kupffer cells) are found in significant numbers between the endotheliocytes of sinusoidal capillaries or on their surface from the disk space side. They have oval nuclei with homogeneous chromatin. In some places, the number of macrophages located along the sinusoidal capillaries increases significantly (Fig. 6). Another significant finding is the substantial accumulation of blood in the central vein of the liver lobule, accompanied by a noticeable expansion of the vein's lumen (Fig. 7). This observation underscores the importance of our research.

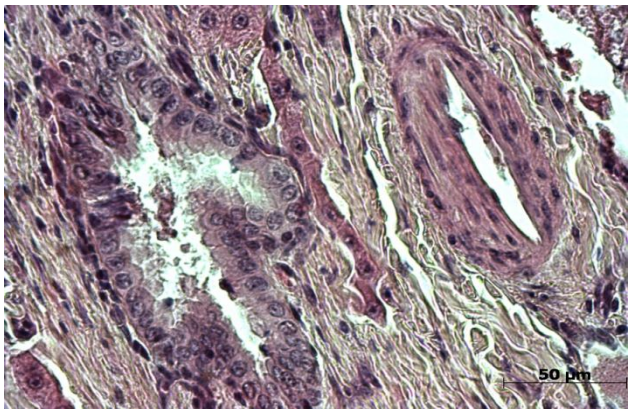

Fig. 7. Hyperemia of the central lobule of the liver of sick animals. Staining with hematoxylin and eosin. Micrography. About 40, about 10.

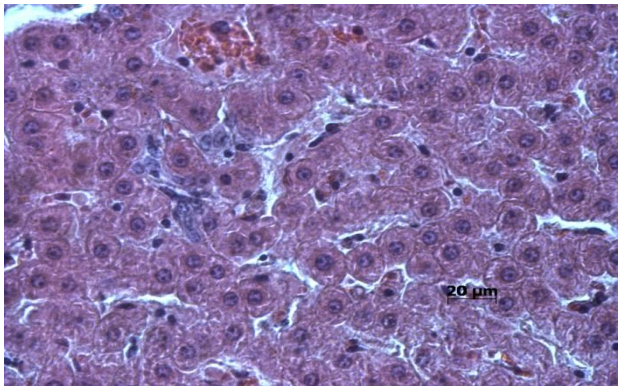

Fig. 8. Macrophages and perivascular infiltration of lymphocytes in the lobule of the liver with fascioliasis of animals. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40.

Due to the slowing of blood flow in dilated vessels, the rheological properties of blood are disrupted by the increased release of leukocytes through the wall of blood vessels (Fig. 8). Damage to the endothelium of blood capillaries causes leukocyte migration, which determines the diapedesis of erythrocytes outside the blood channel (Fig. 9). In general, due to increased exudation of shaped blood elements with impaired blood flow in the blood lobes and the liver, edema occurs with impaired organ function. As the blood flow slows down and the formation of cellular exudate, as well as the production of an inflammatory mediator, a concentration of leukocytes is observed along the significant size of the liver plates and, consequently, along the course of the bile capillaries (Fig. 10). In addition to changes in the microcirculatory channel, the formation of liquid and cellular exudate, and the migration of lymphocytes and macrophages in the liver lobe, lymphoid cells are very densely arranged in rather large clusters (Fig. 11). Simultaneously, the characteristic histological structure of the liver plates is disrupted, indicating a severe impairment of hepatocyte function in this lobule. The presence of considerable lymphoid cell accumulations in some areas further underscores the gravity of the situation (Fig. 12). The main focus of lymphoid accumulation of lymphocytes and macrophages migrates between hepatocytes and along the sinusoidal capillaries. Accordingly, the liver plates lose their characteristic structure when the functional activity of all cellular elements of the liver is impaired. In particular, the liver triad (intestinal artery, vein, and bile ducts), located in loose fibrous connective tissue, undergoes significant changes.

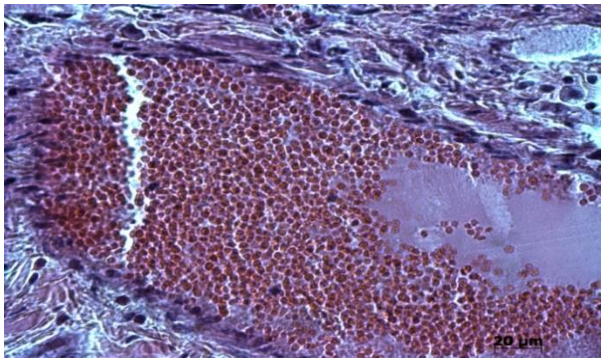

Fig. 9. Diapedesis of erythrocytes outside the capillaries of the lobules of the liver of infected animals. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40.

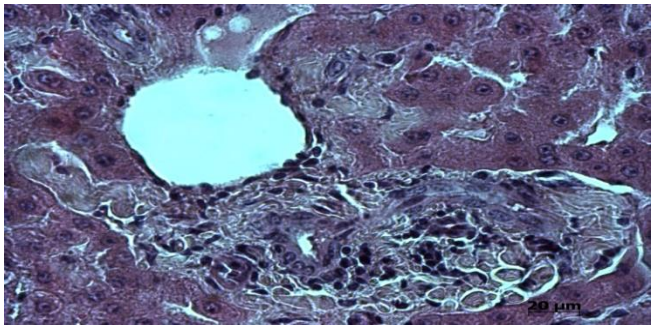

Fig. 10. Infiltration of lymphoid cells through the bile ducts of the liver in animal fascioliasis. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40.

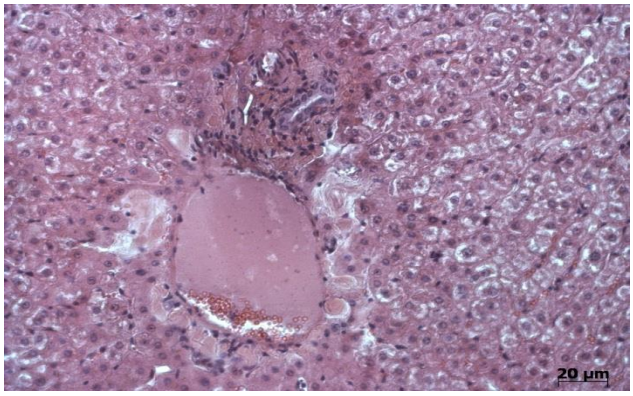

Fig. 11. Accumulation of lymphoid tissue between hepatocytes of the lobule of the liver in fascioliasis of animals. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40.

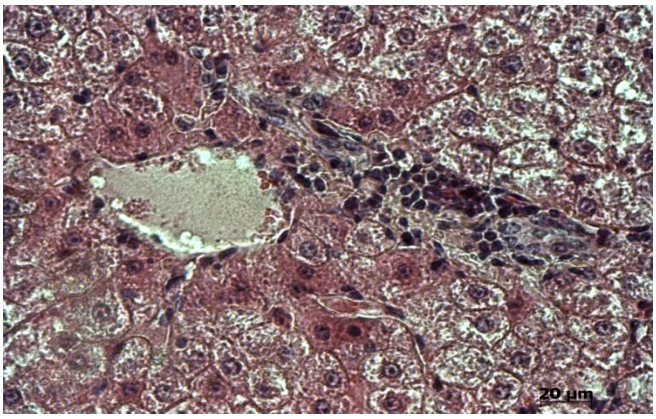

Fig. 12. Destruction of liver cells and proliferation of lymphoid cells in liver lobules. Staining with hematoxylin and eosin. Micrography. Ok. 10, about 40.

The monolayered cubic epithelium lining the intrahepatic bile ducts exhibits a surrounding milieu characterized by diffuse infiltration of lymphocytes and macrophages intermingled with plasma cells, eosinophilic, and neutrophilic leukocytes. Notably, the epithelial cells within the bile ducts manifest a distinct, intense, dark oxyphilic staining. Concurrently, a similar staining intensity is observed in hepatocytes proximal to the biliary tract. These histological alterations in hepatic architecture signify the onset of an inflammatory cascade in fascioliasis. Consequently, fascioliasis in the hepatic and biliary systems elicits a comprehensive array of histopathological changes. These changes commence within the hepatic microvasculature, precipitating disruptions in the host organism's nonspecific and specific immunoprotective mechanisms. According to many researchers, and our studies confirm this, histological changes in the liver are characterized by the following changes if affected by echinococcosis. An irregular distribution pattern is evident, comprising alternating foci exhibiting alterative changes and proliferative reactions within the parenchymal and stromal compartments. These alterations are typified by hepatic lobule atrophy, the proliferation of connective tissue reminiscent of cirrhotic transformation, pronounced perivascular and periductal fibrosis, and the development of granulation tissue featuring abundant multinucleated giant cells. Along with small atrophic hepatocytes, large cells with polychromic cytoplasm are observed. Knowledge of the peculiarities of the growth of the echinococcal bladder of the liver is undoubtedly important for a proper understanding of the principles and details of its surgical treatment. With understanding these features, the surgeon can objectively and correctly choose the appropriate plan for standard and minimally invasive surgical intervention in each specific case of the disease and establish the correct strategy for preventing disease relapses and drug treatment. Upon infiltration into hepatic tissue, the *Echinococcus* larva promptly becomes enveloped by a dense infiltration of mononuclear blood cells. After 26-30 hours, the mononuclears are replaced by peripheral eosinophils with lymphocytes forming an echinococcal follicle. After 4 days, vacuolization is observed in the center of the follicle. On the 7^{th} day, a small parasitic bubble with a liquid and a shell can be detected. The bladder of the parasite exhibits a thin-film structure primarily composed of hyaline during the initial six months of development. Macroscopically, the bladder's internal lining comprises both external (cuticular) and internal (germ) membranes (refer to Fig. 13). Subsequent maturation of the bladder entails

the formation of a capsule derived from the germ membrane, which initiates the protoscolex development process. Mature protoscoleces are observed to float freely within the fluid, resembling "echinococcal sand." Concurrently, a fibrous capsule forms around the bladder's shell itself. This capsule's formation mechanism is predicated upon chronic inflammation evolving within the tissues adjoining the echinococcal bladder. Notably, this inflammation is causally linked to both the parasite's survival and the impact of tissue breakdown products on the surrounding tissues.

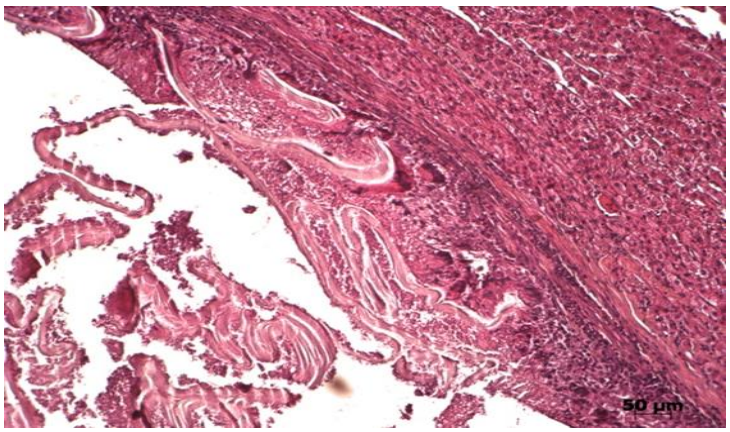

Fig. 13. Staining with hematoxylin and eosin. Biopsy of the echinococcal bladder wall. Magnification of 40 times.

Prolonged growth and compression of liver tissues and their structures, products of *Echinococcus* metabolism, toxic substances secreting the hydatid bladder, and immune reactivity of the body affect the development of structural and morphological changes in the liver (Eguale & Abie 2003). Compression of liver tissue by the hydatid bladder leads to the development of ischemic damage to liver tissue, which leads to a violation of the liver's metabolic function. During the growth process, the parasitic bladder not only squeezes nearby vessels and bile ducts - dystrophic disorders lead to thinning of its walls (Massoud & Vedadi 1983; Modavi & Iseroff 1984; Mahmoud *et al.* 1989). Perforation does not occur until the pressure inside the bladder balances the pressure in the bile duct system. With suppuration of the bladder, a sharp increase in intra-abdominal pressure, its wall ruptures, and the contents of the bladder enter the bile ducts. According to I. J. Deineki (1968), during the larval developmental stage, *Echinococcus* sustains itself by metabolizing substances derived from the fibrous capsule breakdown and neighboring tissues' structural constituents (Swarup & Pachaur 1987; Mas-Coma *et al.* 2005). This assertion supports observations indicating a pronounced reduction followed by the eventual disappearance of necrotic zones within aging echinococcal bladders, concomitant with the depletion of nutrients vital for larval sustenance. Consequently, this process culminates in the vesicles' demise, decomposition, resorption, and calcification, and calcification of the vesicles.

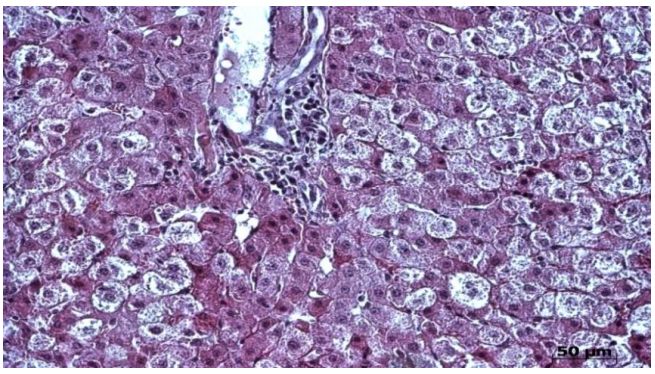

Fig. 14. A biopsy stained with hematoxylin and eosin in the area of an echinococcal cyst. A magnification of 10 times. Fibrous liver tissue, necrobiotic changes, inflammatory infiltration are observed.

The severity of changes in the liver parenchyma of patients with echinococcosis depends on the lesion size. There are fewer of them in solitary uncomplicated vesicles; in multiple echinococcosis, the changed characteristic of diffuse liver damage are observed exhibiting the histological picture of chronic cholestatic hepatitis (Velusamy *et al.* 2002; Okaiyeto *et al.* 2012). When studying the morphological alterations of the liver, significant changes were

revealed in its various parts and in its structure at different distances from the hydatid bladder; the most pronounced changes occurred within 1-4 cm (Figs. 14-16). Thus, the following changes were observed: fibrous liver tissue, necrobiotic changes, inflammatory infiltration, hepatocyte dystrophy, and foci of hemosiderosis of 2-3 degrees. Bile stagnation is detected in the bile capillaries, hypertrophy of elastic membranes in the blood vessels, and often their disintegration. The walls of blood vessels are thickened. The blood vessels of the capsule near the bladder usually expand and fill with thrombotic masses consisting of fibrin and erythrocytes (Fig. 15). Balloon dystrophy, characterized by varying degrees of granulation and vacuolization, is discernible within the cytoplasm of hepatocytes. Upon microscopic examination, the affected cells exhibit a whitish hue, particularly prominent in perinuclear regions. In some places, hepatocyte fatty degeneration is observed, manifested by cell proliferation, acquiring a rounded or oval shape. Fat droplets are located on the periphery of the cell in the cytoplasmic layer. The cytoplasm around the nucleus is preserved as a narrow, homogeneous edge. In some hepatocytes, signs of regenerative hyperplasia are determined by an increase in their size and the formation of large vacuolated nuclei, the number of which is 2-3.

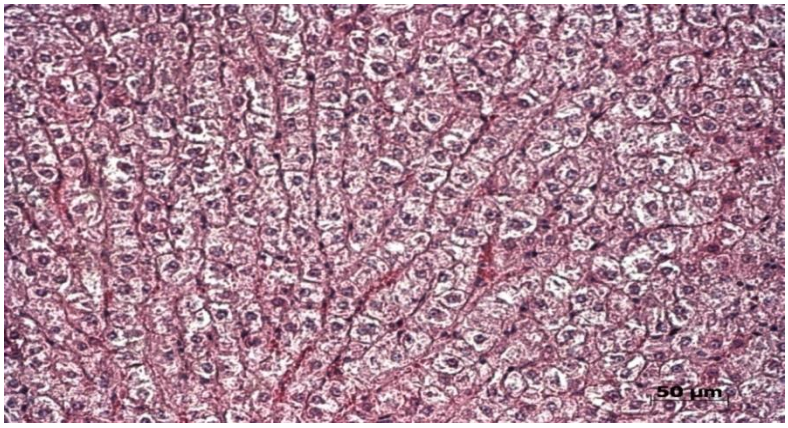

Fig. 15. A biopsy stained with hematoxylin and eosin in the area of the echinococcal bladder. A magnification of 40 times. There is a "balloon" dystrophy of hepatocytes and foci of hemosiderosis of 2-3 degrees and the presence of fibrous changes in liver tissue.

In addition, according to morphology, the liver's normal structure was located in an intact area of the liver. However, in later observations, we observed inflammatory and dystrophic changes in liver tissue even at a distance of 8 cm from the edge of the echinococcal bladder. Infiltrates from elements of lymphoid cells were found in liver tissue around the fibrous capsule. Liver cells are in a state of granular dystrophy. In some places, you can see false lobules, remodeling, or atrophy of the liver trabeculae. The periportal connective tissue grows. Interlobular vessels, arterial and venous branches, and bile ducts remain in place of the former lobules of the liver. In some places, areas of replacement of the liver parenchyma with connective tissue are observed. In areas of acute progressive inflammation, connective tissue has soft, delicate fibers with lymphoid and plasmocyte-type cells.

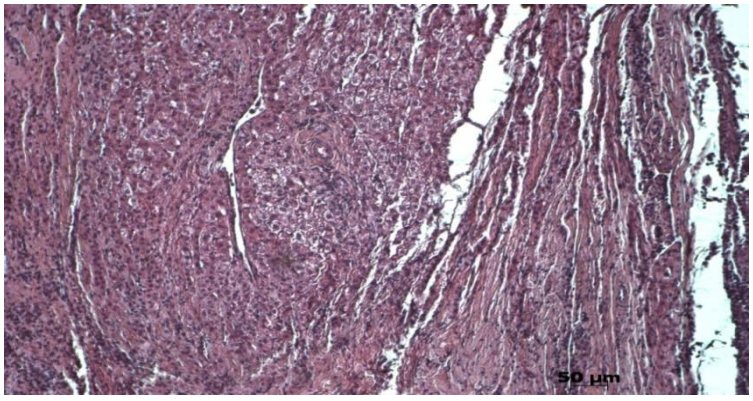

Fig. 16. It is a biopsy stained with hematoxylin and eosin in the area of the echinococcal bladder. A magnification of 40 times. There is a transition of fibrous tissue to healthy tissue, inflammatory infiltration of liver tissue.

When suppressing the inflammatory process, mature connective tissue with a small number of cellular elements between hyalinosis and fibers is revealed. Parenchymal "nodes" (false lobules of the liver) of unequal size are

located between the layers of connective tissue of varying degrees of maturity. In such nodes, liver cells undergo dystrophic changes.

CONCLUSION

Histoathological examinations of the livers of afflicted animals have unveiled distinctive alterations in tissue architecture and cellular constituents indicative of an inflammatory response. These hepatic modifications signify the onset of a comprehensive array of liver and gallbladder changes. The present study shows that proliferation and continuous erosion of the mucous membrane of the bile ducts and damage to liver cells lead to a significant loss of various important substances from the liver.

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