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Pathomorphological and histological changes in the cecum and liver of turkeys affected by histomoniasis

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Histomoniasis of turkeys is one of the most dangerous protozoan diseases of poultry, causing significant economic losses in the poultry industry and characterized by severe lesions of the digestive system and liver. The material for the study consisted of turkeys with clinical signs of histomoniasis obtained from farms of different ownership types in the Odesa region. A total of 56 clinically affected turkeys of the BIG-6 cross, aged 45–60 days, were examined. Among them, 28 birds that had died were subjected to pathological examination with subsequent sampling of liver and cecal tissues for histological analysis. The diagnosis was established based on a comprehensive assessment of clinical signs, pathological changes, and microscopic detection of *Histomonas meleagridis* trophozoites in smear-imprints from the cecum and liver stained following the Romanowsky–Giemsa method, as well as the results of histopathological examination. During the clinical examination of infected turkeys, depression, decreased appetite, progressive emaciation, drooping of the head and wings, and diarrhea with characteristic grayish-yellow droppings were recorded. Post-mortem examination revealed lesions typical of histomoniasis in the ceca and liver, including thickening of the cecal walls with the presence of dense yellowish caseous masses in the lumen, as well as hepatomegaly with numerous round necrotic foci of varying sizes. Histological examination revealed pronounced inflammatory and necrotic changes in the tissues of the ceca and liver. The lesions were characterized by transmural typhlitis, necrotic areas, infiltration by mononuclear cells, and a large number of *H. meleagridis* trophozoites, which were localized both singly and in clusters. In the hepatic parenchyma, multifocal necrotic lesions, degenerative changes in hepatocytes, and inflammatory infiltrates of varying intensity were observed. Prospects for further research are associated with a more in-depth study of the pathogenesis of histomoniasis, morphological changes in various organs of poultry, and the development of effective methods for the prevention and treatment of this parasitic infection.

Keywords: trophozoites; typhlitis; hepatomegaly; necrotic lesions; histopathology; protozoan infection.

Introduction

Histomoniasis (histomonosis), also known as enterohepatitis or “blackhead disease,” is a parasitic disease of domestic poultry caused by the protozoan parasite *Histomonas meleagridis*, a representative of the trichomonad group. The disease is widely distributed and has been reported in poultry production in many countries worldwide (Huber et al., 2005; Abdullah, 2014; Bogach et al., 2024).

Histomonas meleagridis parasitizes various species of birds; however, turkeys are the most susceptible to infection. In this species, the disease often develops in a severe form and is accompanied by high mortality, which may reach 80–100% (Beer et al., 2022). Therefore, considerable attention has been paid to the study of the biology of the pathogen, the immunological mechanisms of host defense in poultry, and the characteristics of the pathological process (Liebhart et al., 2017; Mitra et al., 2018).

The primary sites of parasite localization are the ceca and liver. *Histomonas meleagridis* causes inflammatory and necrotic changes in the ceca with the formation of so-called “cecal cores,” consisting of sloughed tissue and inflammatory cells. Subsequently, the parasite migrates through the portal vein to the liver, where it induces inflammation and the formation of multiple round necrotic lesions in the organ parenchyma, accompanied by impairment of its functional state (Powell et al., 2009; Hess et al., 2015). At the same time, the results of some studies indicate that histomonads may spread beyond their typical sites of localization. According to Singh et al. (2008), the parasite can also localize in the brain, pancreas, heart, lungs, kidneys, and spleen.

Similar systemic lesions have been confirmed by pathomorphological studies, during which turkeys showed hepatomegaly with numerous pale-white nodules, marked thickening of the cecal walls with caseous contents, as well as focal lesions in the kidneys, pancreas, and spleen (Sentíes-Cué et al., 2009).

Clinical signs of infection usually appear 7–14 days after exposure and are characterized by depression, decreased appetite, impaired coordination of movements, and changes in feces. In turkeys, yellow droppings are typical and are considered one of the characteristic clinical manifestations of blackhead disease. During post-mortem examination of turkeys infected with *H. meleagridis*, marked enlargement of the ceca with thickened walls is commonly observed, and their lumen is often filled with fibrinous or caseous material. Hepatomegaly with numerous round necrotic foci is also detected, and in many cases the pathological process may involve a considerable portion of the hepatic tissue (Hess et al., 2006).

Experimental studies have shown that in broiler chickens, pronounced macroscopic lesions of the ceca typical of histomoniasis are already observed during the first and second weeks after infection. Liver lesions in the form of necrotic foci are mainly detected during the second and third weeks after infection, whereas during the first week they are observed only sporadically. Histopathological studies in laying hens revealed more pronounced necrosis and ablation of the cecal epithelium (Lotfi et al., 2014).

In meat-type turkey poults infected with *H. meleagridis*, sudden death may occur by the end of the fifth week without pronounced specific clinical signs. Post-mortem examination reveals severe lesions of the ceca manifested by marked thickening of their walls and filling of the lumen with necrotic or caseous material. In addition, multiple ruptures and areas of necrosis of the ceca have been described, accompanied by the development of localized peritonitis (Grafl et al., 2011).

The characteristics of the immune response also play an important role in the development of the pathological process. In turkeys infected with *H. meleagridis*, a considerable number of parasites migrate from the ceca to the liver, indicating an insufficiently effective local innate immune response in the intestine. As a result, the pathogen actively proliferates in the ceca and spreads throughout the body, reaching the liver, where a prolonged and uncontrolled immune response develops, accompanied by pronounced pathological changes (Powell et al., 2009).

Some studies also indicate the possibility of atypical localization of the parasite. In particular, histomonads have been detected in the bursa of Fabricius, which may suggest alternative routes of infection, including the intracloacal route (Cortes et al., 2004).

Thus, the widespread occurrence of histomoniasis, high mortality among turkeys, the possibility of systemic involvement of the organism, and the increasing number of infections in chickens indicate the relevance of further research on this disease. Particular attention should be paid to the study of the pathogenesis and pathomorphological changes occurring in various organs of poultry during *H. meleagridis* infection, as these changes may significantly influence the course of the disease and its clinical manifestations.

The aim of this study was to investigate pathomorphological and histological changes in the ceca and liver of turkeys infected with *Histomonas meleagridis*.

Materials and methods

All procedures involving animals complied with the current legislation of Ukraine (Article 26 of the Law of Ukraine No. 5456-VI of 16.10.2012 "On the Protection of Animals from Cruel Treatment"), the "General Ethical Principles for Experiments on Animals" adopted by the First National Congress on Bioethics (Kyiv, 2001), and international bioethical standards, including the provisions of the European Convention for the Protection of Vertebrate Animals Used for Experimental and Other Scientific Purposes (Strasbourg, 1985) (Festing & Wilkinson, 2007; Simmonds, 2018; Kabene & Baadel, 2019). The research program was reviewed and approved by the Bioethics Commission of the National Scientific Center "Institute of Experimental and Clinical Veterinary Medicine" in accordance with the established procedure.

The material for the study consisted of turkeys with clinical signs of histomoniasis obtained from farms with different ownership types in the Odesa region. A total of 56 clinically affected turkeys of the BIG-6 cross, aged 45–60 days, were examined. Among them, 28 turkey poults died and were subjected to post-mortem examination. During necropsy, characteristic pathological changes were recorded, and samples from the affected organs, particularly the ceca and liver, were collected for further laboratory analysis.

The diagnosis of histomoniasis was established comprehensively, taking into account epizootiological data, clinical signs, and the results of pathological and laboratory examinations. To confirm the infection, microscopic examination of smear-imprints from the ceca and liver was performed. The smears were stained using the Romanowsky–Giemsa method followed by the detection of *H. meleagridis* trophozoites. Characteristic macroscopic and histopathological changes in internal organs were also taken into account.

During clinical examination, the diseased turkeys exhibited depression, drooping of the head and wings, decreased appetite, and gradual loss of body weight. In most cases, progressive emaciation of the birds was observed. One of the most characteristic clinical signs was diarrhea with the excretion of grayish-yellow droppings.

During necropsy of the dead turkeys, special attention was paid to the examination of the digestive organs and liver. Typical pathological changes characteristic of histomoniasis included marked thickening of the cecal walls with the presence of dense yellowish caseous masses ("cecal cores"). In the liver, enlargement of the organ and the presence of multiple round necrotic foci were recorded. These lesions appeared as so-called "target-like" nodules of yellowish or yellowish-green color with a characteristic depression beneath the surface of the organ, which was particularly evident in severe cases of the disease.

For histological examination, tissue samples from the ceca and liver were collected. The obtained material was fixed in 10% neutral buffered formalin. Subsequent histological processing was carried out according to standard procedures: after fixation, the tissues were dehydrated in alcohols of increasing concentration, cleared in xylene, and embedded in paraffin with a melting point of 56–58 °C to prepare paraffin blocks.

Histological sections 3–4 μm thick were prepared from the paraffin blocks using a microtome (Leica, Germany). The sections were stained with hematoxylin and eosin according to the standard protocol. The histological slides were examined under a light microscope (Leica, Germany) with subsequent photodocumentation of the characteristic morphological changes.

The histological studies were carried out at the Department of Normal and Pathological Morphology, Physiology and Forensic Veterinary Medicine of Odesa State Agrarian University.

During the parasitological examination, no *Eimeria* oocysts, nematodes, or their eggs were detected in the fecal samples. Direct microscopic examination of the cecal contents, as well as analysis of stained preparations, confirmed the presence of the protozoan *H. meleagridis*.

Results

During post-mortem examination of turkeys affected by *H. meleagridis*, pronounced pathological changes of the ceca were observed. The cecal wall was unevenly thickened due to edema, which was most pronounced at the base of the organ. The intestinal lumen contained dense caseous-necrotic masses which, in some cases, were gas-filled and had a sharp unpleasant odor.

The mucous membrane was swollen and hyperemic, with occasional petechial and linear hemorrhages; in some areas it was destroyed, forming ulcerative-necrotic lesions. In the fundal part of the ceca, mushroom-like and nodular formations of various shapes and sizes were observed, clearly protruding above the surface of the mucous membrane. In some cases, these formations merged with each other, forming a continuous "cheese-like" mass, which represents a manifestation of fibrinous inflammation (Fig. 1a).

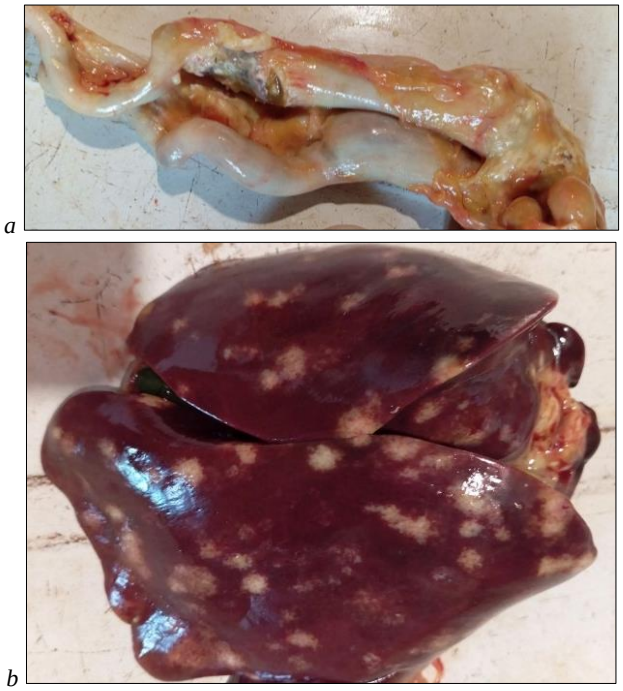

Fig. 1. Pathological changes in the ceca and liver of turkeys with histomoniasis: cecum with a thickened wall and caseous-necrotic masses in the lumen (*a*); liver with multiple round necrotic foci (*b*)

The liver was enlarged, dark red in color, with numerous round grayish-yellow necrotic foci ranging in size from that of a millet seed

to that of a pea, distributed over the entire surface. These lesions had well-defined boundaries with the surrounding unaffected tissue, slightly protruded above the surface of the capsule, and at the same time extended into the depth of the parenchyma. On section, the liver tissue was moist, relatively homogeneous in consistency, and pale brown with a yellowish tint. The edges of the organ were slightly rounded, and the capsule was tense (Fig. 1b).

During histological examination of the cecum of infected turkeys, pronounced pathological changes involving the mucosa and submucosa were observed. The mucosa was markedly thickened and its architectonics was disrupted. Intestinal crypts were deformed, in some areas dilated, with irregular lumens. In some crypts, accumulation of cellular detritus, mucus, and desquamated epithelial cells was observed. The epithelial layer showed dystrophic changes and was partially desquamated.

In the stroma of the mucosa, uneven edema and stasis in the blood vessels were detected (Fig. 2).

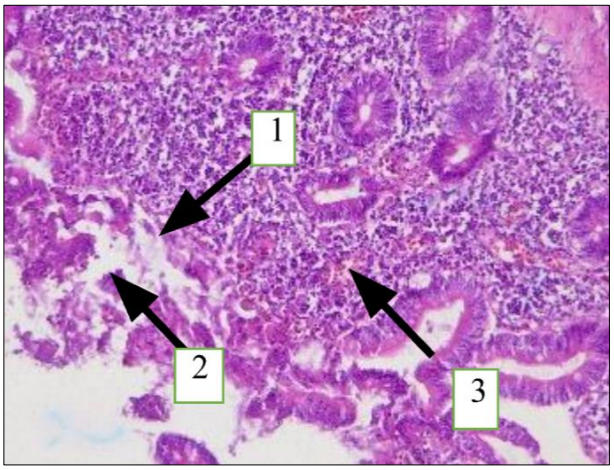

Fig. 2. Diffuse inflammatory infiltrate in the cecal wall of a turkey (×100; hematoxylin and eosin staining): *1* – diffuse inflammatory infiltrate; *2* – stromal edema; *3* – vessel with stasis

The diffuse inflammatory infiltrate was localized between and around the crypts, extending into the submucosa. In the tissues, widening of the intercellular spaces, congestion of the microcirculatory vessels, and areas of destruction with the formation of necrotic masses were observed. The identified morphological changes indicate the development of a subacute inflammatory process with signs of a productive reaction.

At higher magnification, among the cellular detritus and within the infiltrate, round or amoeboid cellular structures with pale eosinophilic cytoplasm and a weakly defined nucleus were observed, morphologically corresponding to trophozoites of *H. meleagridis* (Fig. 3).

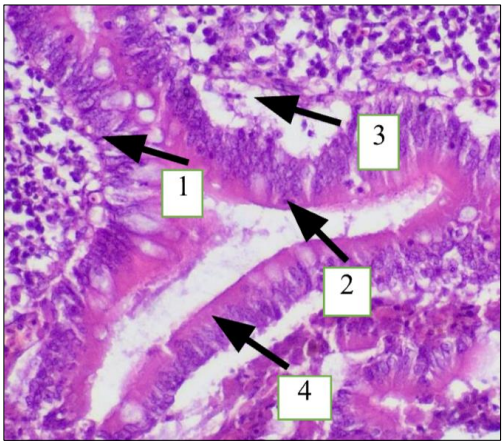

Fig. 3. Destruction of crypts and trophozoites of *H. meleagridis* (×200; hematoxylin–eosin staining): *1* – cecal crypts; *2* – trophozoites of *H. meleagridis*; *3* – inflammatory infiltrate; *4* – necrotic area

In the intestinal wall, trophozoites of *H. meleagridis* are localized both extracellularly and intracellularly (within histiocytes). The parasites have a round or amoeboid shape, basophilic cytoplasm, and occasionally clearly visualized nuclei. They are located among cellular detritus and inflammatory infiltrates. The lamina propria of the mucosa is densely infiltrated with inflammatory cells (Fig. 4).

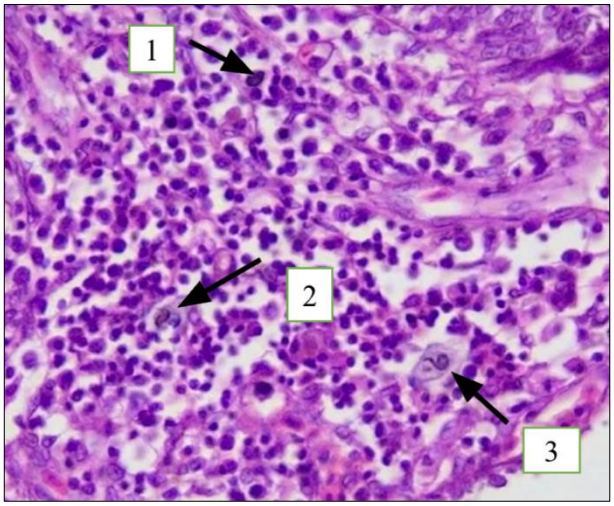

Fig. 4. Cellular composition of the infiltrate and trophozoites of *H. meleagridis* in the cecal wall of a turkey (×400; hematoxylin–eosin staining): *1* – lymphocyte; *2* – segmented neutrophilic granulocyte; *3* – trophozoite of *H. meleagridis*

The infiltrate was predominantly composed of lymphocytes, neutrophils, macrophages, plasma cells, and heterophils, indicating an active inflammatory process. Marked destruction of the intestinal epithelium was observed, manifested by disruption of the integrity of the mucosa, disorganization of the tissue structure, and the formation of necrotic masses. Further histological examination revealed significant thickening of the mucosa and submucosa due to massive lymphocytic infiltration. In the lamina propria of the mucosa, well-formed lymphoid follicles with distinct germinal centers were diffusely distributed, indicating intense antigenic stimulation caused by *H. meleagridis* (Fig. 5).

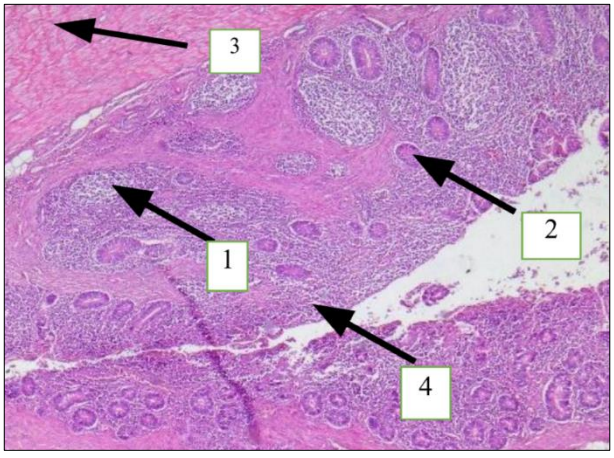

Fig. 5. Lymphoid reaction of the cecal mucosa of a turkey with histomoniasis (×40; hematoxylin–eosin staining): *1* – lymphoid follicles; *2* – intestinal crypts; *3* – muscular layer; *4* – diffuse inflammatory infiltrate

The structure of the intestinal crypts was markedly disrupted; they were separated by inflammatory infiltrates and in some areas compressed or destroyed. The epithelial lining showed signs of desquamation and microerosions caused by the cytopathic effect of the parasite. In the submucosal layer, moderate edema and dilation of blood vessels (hyperemia) were observed, indicating the development of an inflammatory process. The muscular layer of the intestine retained relative integrity; however, the boundary between it and the

mucosa was partially blurred due to the migration of inflammatory cells. The overall histological pattern corresponded to nodular (follicular) typhlitis, which is characterized by a combination of destructive epithelial changes, necrotic processes in the mucosa, and hyperplasia of lymphoid tissue, reflecting a pronounced immune response of the host organism to the parasitic invasion.

Thus, histological examination of the cecum of turkeys infected with *H. meleagridis* revealed pronounced destructive-inflammatory changes characterized by disruption of the mucosal architectonics, deformation of intestinal crypts, stromal edema, and the formation of a diffuse inflammatory infiltrate.

Numerous trophozoites of *H. meleagridis* were detected in the intestinal tissues, localized both extracellularly and intracellularly within histiocytes, confirming their direct involvement in the development of the pathological process.

The inflammatory infiltrate was mainly composed of lymphocytes, neutrophils, macrophages, plasma cells, and heterophils, indicating an active immune response of the host organism to the parasitic invasion. In the chronic course of the disease, the pathological process in the body of turkeys is not limited to lesions of the ceca but spreads to other organs, primarily the liver.

Microscopic examination of liver samples from turkeys naturally infected with *H. meleagridis* revealed profound destruction of the organ parenchyma manifested by pronounced necrotic, inflammatory, and proliferative tissue changes (Fig. 6).

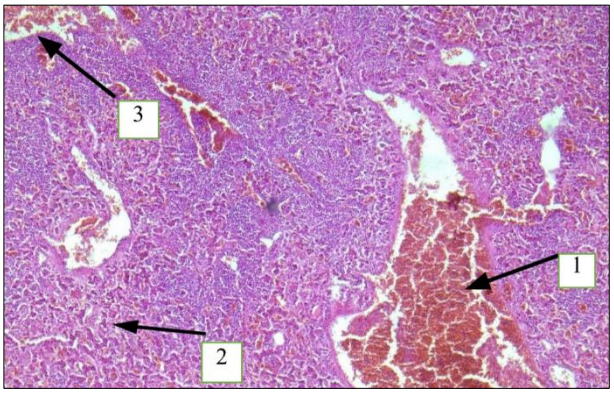

Fig. 6. Focus of necrosis in the liver of a turkey with histomoniasis (×100; hematoxylin–eosin staining): *1* – foci of coagulative necrosis; *2* – inflammatory infiltrate; *3* – preserved liver tissue

Pathomorphological changes were represented by multiple diffusely distributed foci of coagulative necrosis. In the central areas of these foci, complete destruction of hepatocytes was observed. The cytoplasm of the cells exhibited marked eosinophilia, becoming homogeneous or finely granular, while the nuclei were in a state of karyopyknosis, karyorrhexis, or complete dissolution (karyolysis). The architectonics of the hepatic cords within the necrotic zones was completely disrupted, indicating a pronounced cytolytic effect of the pathogen.

A well-defined inflammatory infiltrate formed around the necrotic areas. It was predominantly lymphohistiocytic in nature with the presence of granulocytes (heterophils). This cellular reaction was aimed at localizing the pathological process. Vegetative forms of histomonads were localized at the boundary between necrotic and preserved tissues, appearing as round or oval structures with a characteristic clear zone surrounding the cytoplasm.

In areas distant from the main lesions, the hepatic parenchyma exhibited pronounced granular and vacuolar degeneration. Hepatocytes were enlarged, their contours indistinct, and the sinusoidal capillaries were narrowed due to edema of the parenchyma. Hyperemia of blood vessels and phenomena of stasis were also observed, indicating disturbances of microcirculation and the development of systemic intoxication.

Pronounced alterations of the liver pathomorphological architectonics were observed, characterized by complete disruption of the trabecular structure of the lobules and the development of a diffuse inflammatory process (Fig. 7).

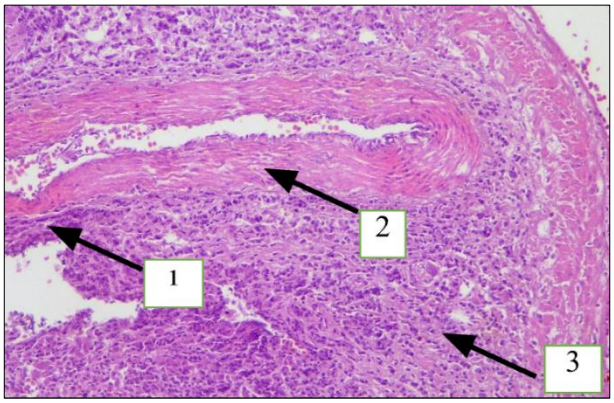

Fig. 7. Massive polymorphocellular infiltration and hemorrhagic syndrome (×200; hematoxylin–eosin staining): *1* – inflammatory infiltrate (polymorphocellular infiltration); *2* – necrotic zone; *3* – hepatocytes

The dominant pathomorphological feature in this histological specimen is diffuse dense polymorphocellular infiltration involving a significant portion of the intertrabecular spaces and sinusoids. The inflammatory infiltrate is represented by a heterogeneous population of cells, predominantly lymphocytes and macrophages, with the presence of heterophils (granulocytes) typical for birds, indicating an active inflammatory reaction. The most intense cellular infiltration is observed in perivascular areas and around a large artery, which may indicate hematogenous spread of the pathogen and the development of a systemic inflammatory response.

In the upper and lower right parts of the section, large structureless areas are observed, filled mainly with hemolyzed erythrocytes and eosinophilic tissue detritus. These changes correspond to zones of hemorrhagic necrosis formed as a result of massive diapedesis of erythrocytes through damaged walls of capillaries and sinusoids under the influence of toxic products of *H. meleagridis* metabolism and inflammatory mediators. In contrast to coagulative necrosis, these areas are characterized by karyolysis and complete destruction of cellular elements, indicating a severe course of the pathological process.

In areas distant from the massive cellular infiltrate, hepatocytes were in a state of pronounced granular and vacuolar degeneration. Their cytoplasm exhibited homogeneous eosinophilia, indicating necrobiotic changes, while the nuclei were swollen with signs of karyopyknosis and karyorrhexis. Some hepatocytes underwent complete lysis, forming foci of cytolysis. As a result of these changes, the normal lobular architecture of the liver was markedly disrupted, leading to the development of pronounced functional insufficiency of the organ.

Alongside the necrobiotic changes, profound disorganization of hepatic blood circulation was observed. A characteristic feature was a pronounced dystonic state of the vascular bed at all levels of the microcirculation (Fig. 8). The lumens of the sinusoids in most hepatic lobules were in a state of persistent dilation. This was caused both by the direct vasoparalytic effect of pathogen metabolites and by mechanical compression of the efferent vessels due to perifocal edema. The sinusoids appeared as optically empty or slit-like spaces containing only a few formed elements, separating the hepatic cords and disrupting their integrity. In areas adjacent to the foci of inflammatory infiltration, numerous zones of blood stasis were observed. These were represented by dilated capillaries with lumens densely filled with erythrocytes that had lost their distinct individual contours. The pronounced eosinophilia of these areas clearly contrasted with the surrounding infiltrated tissue.

Arterial and venous vessels of medium caliber were in a state of marked passive congestion. In the section, a vessel with a well-defined muscular layer was observed, the lumen of which was completely filled with an erythrocytic mass. The endothelial lining of such vessels in some areas showed signs of desquamation, increasing the

permeability of the vascular wall and promoting the leakage of plasma and formed blood elements into the perivascular space.

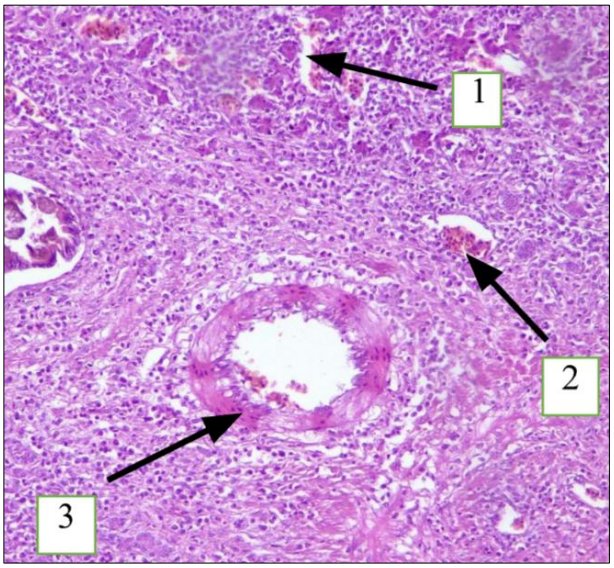

Fig. 8. Disturbances of microcirculation and phenomena of stasis (×200; hematoxylin–eosin staining): *1* – sinusoids; *2* – blood stasis; *3* – congested vessel

Detailed microscopic examination of the hepatic parenchyma at high magnification (×400) revealed that destructive processes involved the majority of hepatocytes, acquiring the character of necrobiosis and focal necrosis (Fig. 9).

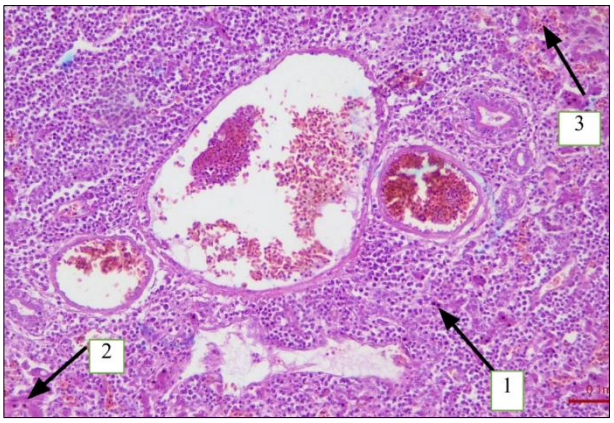

Fig. 9. Dystrophic and necrobiotic changes of hepatocytes (×400; hematoxylin–eosin staining): *1* – hepatocytes with pyknotic nuclei; *2* – hypereosinophilic cytoplasm; *3* – zone of destruction

For most preserved hepatocytes, a state of karyopyknosis was characteristic. The nuclei appeared intensely basophilic, reduced in size, shrunken, with a complete loss of the distinct structure of chromatin and nucleoli. In isolated cases, karyorrhexis was observed (the fragmentation of nuclear material into separate deeply stained fragments). Such changes are irreversible and indicate the terminal stage of cellular activity.

The cytoplasm of affected hepatocytes was characterized by pronounced hypereosinophilia. It acquired an intense pink coloration, becoming homogeneous ("glassy") or finely granular. In some cells, plasmorrhexis was observed (the destruction of the cytoplasmic membrane with the release of cellular contents into the intercellular space). The loss of granularity and swelling of the cytoplasm indicate profound denaturation of intracellular proteins and disruption of osmotic homeostasis under the influence of toxic metabolites of histomonads.

Massive destruction of hepatocytes leads to the formation of zones of tissue destruction. In these areas, cellular boundaries are completely lost, and the tissue is represented by amorphous oxyphilic detritus. Against this background, remnants of nuclei and individual cells of the inflammatory infiltrate (lymphocytes, macrophages) are visualized. The presence of such zones indicates the transition of dystrophic processes into the phase of colliquative or coagulative necrosis, resulting in irreversible loss of the functional capacity of the liver.

Histological examination of liver areas showing signs of disintegration at high magnification (×400) revealed massive involvement of the parenchyma by vegetative forms of the pathogen along with a corresponding reaction of immune defense cells (Fig. 10).

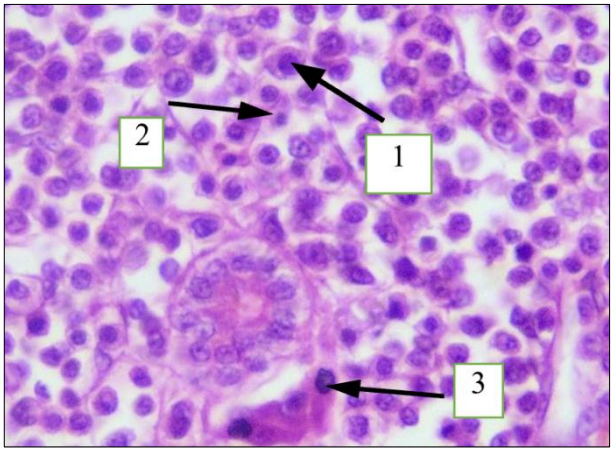

Fig. 10. Massive invasion by trophozoites of *H. meleagridis* (×400; hematoxylin–eosin staining): *1* – vegetative forms of *H. meleagridis*; *2* – lymphocytic infiltration; *3* – dystrophically altered hepatocyte

In the field of view, massive accumulations of histomonad trophozoites are observed. The pathogens have a round or oval shape with a diameter of 8–15 μm. The cytoplasm of the parasites is stained pale pink, while a small, distinctly outlined basophilic nucleus is visible in the center. A characteristic differential feature is the presence of a clear halo surrounding each histomonad, which results from lysis of the surrounding tissues by pathogen enzymes and its motility *in vivo*. The parasites are located both singly and in large conglomerates, completely replacing the destroyed hepatocytes.

The multiplication of the pathogen is accompanied by an intense lymphocytic reaction. Among the vegetative forms of the parasites, numerous small cells with compact, intensely basophilic nuclei are observed (predominantly lymphocytes and activated macrophages).

Among the accumulations of pathogens and the inflammatory infiltrate, remnants of degenerated hepatocytes are observed. The cells are in a state of severe granular degeneration, markedly enlarged in volume, with indistinct contours and vacuolated cytoplasm. The nuclei of these hepatocytes are in a state of karyopyknosis or lysis.

Thus, histomoniasis in turkeys is accompanied by profound destructive changes in the liver parenchyma, manifested by the development of necrosis, massive inflammatory infiltration, and pronounced disturbances of microcirculation. The detection of numerous trophozoites of *H. meleagridis* in zones of tissue destruction indicates active invasion of the pathogen and determines the severe course of the pathological process in the organ.

Discussion

Histomoniasis of turkeys is one of the most dangerous protozoan diseases of poultry that can cause significant economic losses in the poultry industry. The disease has been described in the scientific literature for a long time and continues to remain a relevant problem for the sector (Liebhart et al., 2017).

Previously, the widespread use of preventive and therapeutic drugs significantly limited the spread of histomoniasis in turkeys and other poultry species (Hess et al., 2015).

However, in recent years, cases of the disease caused by *H. meleagridis* have again been reported in flocks of turkeys and chickens. One of the main reasons for this phenomenon is considered to be the ban on the use of antiprotozoal drugs in poultry production

in the EU and the USA, introduced in accordance with international food safety requirements (Phuong et al., 2020; Beer et al., 2022).

According to the literature, the clinical course of histomoniasis in turkeys is characterized by depression, decreased appetite, loss of body weight, and diarrhea with yellowish droppings. In some cases, drooping of the head, ruffled feathers, and cyanosis of the head in young turkey poults are also observed (Majidi et al., 2025).

It is also known that young turkeys are more susceptible to infection with *H. meleagridis*, although a severe course of the disease may also occur in adult birds (McDougald, 2003).

The results of our study confirm the data of previous authors regarding the characteristic pathomorphological changes associated with histomoniasis. During macroscopic examination of the affected organs, thickening of the cecal walls with the formation of grayish-yellow caseous masses ("cecal cores") was observed. In the liver, multiple necrotic foci of various sizes were detected.

Similar pathological changes have also been described in other studies, where histomonads were detected not only in the ceca and liver but also in several other organs. Thus, Karaman et al. (2009) reported the presence of trophozoites in the ceca (100%), liver (91.4%), kidneys (17.1%), spleen (11.4%), proventriculus (5.7%), and bursa of Fabricius (2.8%). PCR analysis in that study also confirmed the presence of the pathogen in various organs, including the lungs (Karaman et al., 2009).

The liver lesions identified in our study reflect the sequential stages of the pathological process. In the early stages, small foci of necrosis and inflammation are observed, which may merge as the invasion progresses, forming larger nodular structures. Similar changes have been described in the work of Senties-Cue et al. (2009), where a correspondence between the macroscopic and histological manifestations of the pathological process in histomoniasis was noted.

According to the results of histopathological analysis, a considerable number of histomonads were detected in the tissues, localized both individually and in grouped clusters. Similar observations are consistent with the findings of McDougald (2005), who emphasized the important role of intense parasitic invasion in the development of necrotic lesions of organs.

A number of studies have also described severe lesions of the ceca characterized by marked thickening of their walls and accumulation of necrotic and caseous material in the lumen. In some cases, ruptures of the cecal walls and the development of localized peritonitis were recorded (Popp et al., 2012; Grafl et al., 2015).

Similar pathomorphological changes have been documented in other regions. Thus, in a study of 140-day-old turkeys of the BUT-6 cross, hepatomegaly with greenish discoloration and fibrotic tissue changes was observed. Histological examination revealed proliferative changes in the hepatic parenchyma and a positive reaction for trophozoites of *H. meleagridis* using periodic acid–Schiff (PAS) staining (Amr Abd El-Wahab et al., 2021).

Histopathological studies by other authors also confirm the presence of necrosis of the ceca and liver as well as a significant number of histomonad trophozoites in the tissues of affected organs (Majidi et al., 2025; Morales-Barrera et al., 2025). In some cases, the parasite may spread beyond its typical sites of localization. Thus, Hauck et al. (2018) reported its detection in the spleen, kidneys, bursa of Fabricius, proventriculus, lungs, pancreas, and crop.

The described changes are also consistent with data from other studies, which reported combined liver lesions manifested as hepatomegaly, steatosis, multifocal necrosis, and granuloma formation in the presence of *H. meleagridis* (Liulin et al., 2023).

The macroscopic and histological changes observed in our study generally correspond to the descriptions provided in the work of Costa et al. (2018), where transmural typhlitis, fibroblast proliferation, neovascularization, and the presence of numerous eosinophilic trophozoites with a diameter of 6–20 µm in the tissues of the ceca were reported. Similar morphological characteristics of the parasite were also described by Gunerhan et al. (2018), who noted that trophozoites have a spherical or oval shape, a size of 8–15 µm, and contain an eccentrically located nucleus. The obtained results confirm the findings of other authors regarding the pathognomonic nature of lesions of the ceca and liver in histomoniasis (Barros et al., 2020; De Barros et al., 2022). At the same time, some studies describe differences in the shape and color of liver lesions. Thus, Emami et al. (2022) reported that the lesions may appear as rounded areas with a greenish center and raised margins.

Differences in the shape and morphology of the lesions may be explained by different stages of the infectious process. In the early stages, they are represented by small foci of necrosis and inflammation, which gradually merge and form larger nodular structures. In the later stages of the disease, massive necrosis of hepatocytes leads to collapse of the hepatic parenchyma and the formation of characteristic saucer-shaped liver lesions (Qudama et al., 2024). Similar changes were also described by Tykałowski et al. (2021), who observed hepatomegaly with rounded ulcerative lesions and the presence of caseous content in the ceca.

Thus, the results of the present study confirm the literature data regarding the characteristic clinical, macroscopic, and histopathological manifestations of histomoniasis in turkeys and indicate the complex nature of the pathogenesis of this disease, which may be accompanied by both local and systemic organ lesions.

Conclusions

In turkeys infected with *Histomonas meleagridis*, pronounced destructive and inflammatory changes develop in the ceca, characterized by disruption of the mucosal architectonics, deformation of the intestinal crypts, stromal edema, and the formation of a diffuse cellular infiltrate. Numerous trophozoites of *H. meleagridis* are detected in the tissues of the ceca, localized both extracellularly and intracellularly within histiocytes, indicating the active involvement of the pathogen in the development of the pathological process.

Histological changes in the liver are characterized by multiple foci of coagulative and hemorrhagic necrosis, massive polymorphocellular infiltration, dystrophic changes in hepatocytes, and disturbances of microcirculation.

Massive accumulation of trophozoites of *H. meleagridis* in the liver parenchyma confirms their active proliferation in the tissues of the organ and indicates a severe course of histomoniasis in turkeys.

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