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Histaminosis of Turkeys in Kazakhstan

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Resume

Histomonosis - is a common protozoal disease of birds in poultry farms of Kazakhstan.

Typical macroscopic and histological changes are found in the cecum and liver, specifically diphtheritic, sometimes fibrinous-hemorrhagic typhlitis, necrotizing hepatitis, catarrhal-desquamativeduodenitis, croup-diphtheritic diffuse or focal esophagitis, focal diphtheritic gastritis, congestive hyperemia and parenchymal dystrophy of the liver and kidneys, increasing glands of the esophagus, anemia of the visible mucous membranes, exhaustion. Histological changes are characterized by the formation of lymphoid-histiocyte infiltrates around histomonad.

Keywords: Histomonosis; Pathmorphology; Histological Examination; Enter hepatitis; Black Head; Parasitology.

Introduction

The problem of elimination of poultry diseases is paramount importance in the further development of industrial poultry farming and the receipt of dietary food products in all countries of the world. It is noted that the annual loss of birds from various diseases in the USA is 375 million dollars, in England - 117 million [1].

An important obstacle in the development of poultry farming is invasive diseases, among which a special place is occupied by histomonosis. In the literature, this disease is described under different names: typhlogopathy, histomonosis, "black head", moniliasis, "liver decay", enterohepatitis. Such a large number of synonymsexplained when we study etiology of this disease.

The first literary report on histominosis appeared in 1893, which indicated the contagious character of the disease and its acceptance for a variety of cholera. Many studies have been devoted to the study of this disease [2].

In Russia, histomonosis was first recorded in 1913 in the Transcaucasia and in the former Novgorod province. Later histomonosis was registered in the farms of the northern Caucasus, and in the Rostov region, Ukraine and other places [3]. M.A. Artemichev on the basis of his studies came to the conclusion that the causative agent of histomonosis is the flagellate protozoan Histomonasmeleagridis, and the eggs and larvae of the helminthHeterakisgallinarum contribute to the spread of this disease.

Histomonosis is a protozoal disease of birds, caused by the protozoan Histomonasmeleagridis of the family Trichomonadidae. The existence of this microorganism is closely related to the nematode of the cecum Heterakisgallinarum and several species of earthworms that are common in the soil on poultry grounds. Histomonads are found in epithelial cells of the intestinal tract of helminths that have just appeared. The mechanism of infection of helminth eggs with histomonads has not yet been studied. Earthworms can serve as intermediate hosts in which the larvae of heterocydes hatch from eggs. Young helminths live in tissues in an invasive state [4].

Thus, the earthworm serves as a means of collecting and concentrating the eggs of heterokerids from the soil of the walks. On the ground, the climatic conditions and the type of soil favor the survival of heterokerids and earthworms, which should be taken into account when attempts are being made to solve the problems of histomonosid relapses.

There is a possibility of direct invasion of turkeys with ingestion of viable histomonads from fresh feces. Nevertheless, the very fragile nature of these microorganisms makes this path of infection unlikely. Histomonads can`t survive beyond their host for more than a few minutes, unless they are protected by an egg of heterokerid or an earthworm.

Chicken, turkey, guinea fowl, quail, ducks and geese are affected by histomonosis. This disease occurs among pheasants and peacocks. Turkeys are considered the most receptive host. The turkey is more often get sick at the age of two weeks to 3 months, but young male birds are affected as well. Turkeys can suffer from histomonosis throughout their life. Adult sick turkeys can distribute histomonosis, which secrete a large number of parasites with feces. Chickens, being permanent carriers of heterokis, also infect turkeys. The source of turkey`s contamination are also contaminated litter, inventory, transport, packaging, hatching eggs, shoes, clothing of maintenance personnel. Despite the improved methods of keeping birds, the availability of effective medicines, histomonosis remains a problem disease in poultry farms for growing chickens, turkeys and other birds. The emergence and spread of histomonosis is facilitated by unsanitary conditions of keeping and inadequate feeding of birds [5].

Currently, histomonosis is registered in all countries and causes tangible economic damage to farms. The economic importance of the disease can`t be overestimated [6]. Reduced productivity as a consequence of the disease and the costs of chemotherapy increase these losses. The continued spread of this disease among birds causes the need to deepen research in many areas related to epizootology, pathogenesis, clinical and morphological and biological indicators [7].

Material and methodology: The research was carried out at the Department of Biosafety of the Kazakh National Agrarian University. The material for our studies was a pathological material taken from 65 spontaneously diseased turkeys, killed by histomonosis in “OrdabasyKus” LLP in the South Kazakhstan region. Age of dead and killed patients with histomonosis of birds ranged from 3 weeks to 4 months. The diagnosis for histomonosis was made taking into account data of epizootology, clinic, pathoanatomical autopsy and, subsequently, confirmed histologically. In order to exclude diseases similar to histomonosis, bacterioscopic and bacteriological studies were carried out in individual cases. As a control material from 3 healthy turkeys was used. All birds were subjected to a detailed pathoanatomical autopsy with detailed logging of each case. The material for histological examination was fixed in a 10% solution of neutral formalin and Carnoy fluid.

For histological examination, pieces were taken from the organs: goitre, glandular and muscular stomach, small intestine, cecum; Liver, spleen, kidney, heart, lungs, pectoral muscles; brain. Celloid, paraffin and frozen sections were prepared. The sections were stained with conventional and some histochemical methods: hematoxylin-eosin, Van Gyzon, Schick reaction, Sudan III, and others. Ultrathin sections were made on a semi-automatic (HEOTION ERM 3100) and a freezing microtome. Histological micro-preparations were studied with a binocular microscope MBI-6 under different magnifications.

Resultsofthestudy: From April to May 2014, "OrdabasyKus" LLP got sick turkeys with a significant case.

To determine the distribution of histomonosis in the South Kazakhstan region, the data of the regional Department of Veterinary Medicine for the last three years (2012-2014) were analyzed. According to the Department of Veterinary Medicine, infectious diseases have been registered in 6047 birds in the last 3 years, including histomorphosis in 789, which is 10.6%. The highest incidence rate of turkey histomoniasis was registered in 2014 compared to the previous years (11.9%). The results are shown in **Table 1.**

Table 1 Distribution of histomonosis of turkeys in the South Kazakhstan region

№	Years	Were diseased, total	Ofthemfellillwithhistomonosis	
			quantity	B %
1	2012	1121	125	11,1
2	2013	1202	130	10,8
3	2014	1201	143	11,9
	Total:	6047	789	10,6

Usually, histomonosis in birds is noted in the summer. In large poultry farms, histomonosis is recorded at any time of the year. Due to the fact that the disease manifests itself in a warm, rainy period, it is especially observed from May to August. According to the results of our research, out of 780 turkeys belonging to OrdabasyKus LLP in 2014, 144 cases of histomonosis (18.5%) developed. The peak of the disease was noted in April, May, June. The results of the studies are given in **Table 2.**

Table 2 Seasonal dynamics of the histomonosis of turkeys in "OrdabasyKus" LLP in 2014					
№	Month of the year	Examined feces	Infected		Detected histominoses
			Quantity	%	
1.	January	50	-	-	-
2.	February	50	-	-	-
3.	Match	65	4	6,25	3
4.	April	180	28	35,0	41
5.	May	180	24	30,0	35
6.	June	180	26	32,5	28
7.	July	65	8	12,3	8
8.	August	80	9	11,3	21
9.	September	80	18	22,5	15
10.	October	50	24	48,0	13
11.	November	50	3	6,0	3
12.	December	50	-	-	-
	Total:	780	144	18,5	14

In "OrdabasyKus" LLP the disease was noted in young birds, out of 330 tested turkeys, 65 were sick, i.e. 19.7%. Among the diseased birds, the greatest percentage was observed among 60-70 days compared to 10-20 day and 100-110 day-old turkeys. The results of the studies are given in **Table 3.**

Table 3 Age dynamics of the histomonosis of turkeys in" OrdabasyKus" LLP					
№	Age of chickens (Days)	Examined Turkeys (number)	Fell ill		Average number of Parasites (in 20 f.v.m.)
			Number	%	
1.	10-20	30	-	-	-
2.	20-30	30	2	6,7	1
3.	30-40	30	2	6,7	1
4.	40-50	30	5	16,7	5
5.	50-60	30	11	36,6	11
6.	60-70	30	16	53,3	16
7.	70-80	30	9	30,0	11
8.	80-90	30	8	26,6	9
9.	90-100	30	6	20,0	8
10	100-110	30	3	10,0	5
11	110-120	30	3	10,0	4
	Total:	330	65	19,7	6,5

Note: f.v.m. - field of view a microscope

Main clinical signs: impaired coordination of movement, decreased appetite. The sick turkeys plaintively squeaked, felt thirsty, trembled, they felt lethargic, ruffled feathers, after 2-3 days, yellow-green diarrhea with mucus appeared,

sometimes it was with an admixture of blood. They also observed drowsiness, drooping wings, swinging gait, closed eyes, lowered or close to the body or under the wing of the head **(Figure 1).**

A. **B.**

Figure 1.Healthy (A) and patients with histomonosis (B) turkeys.

In patients turkeys, a blackening of the scalp, caused by its congestive hyperemia,i.e. cyanosis, was noted.

It is because of this sign that the disease is called "black head". Sick turkeys began to lose weight and by the end of the second week they looked drained **(Figure 2).**

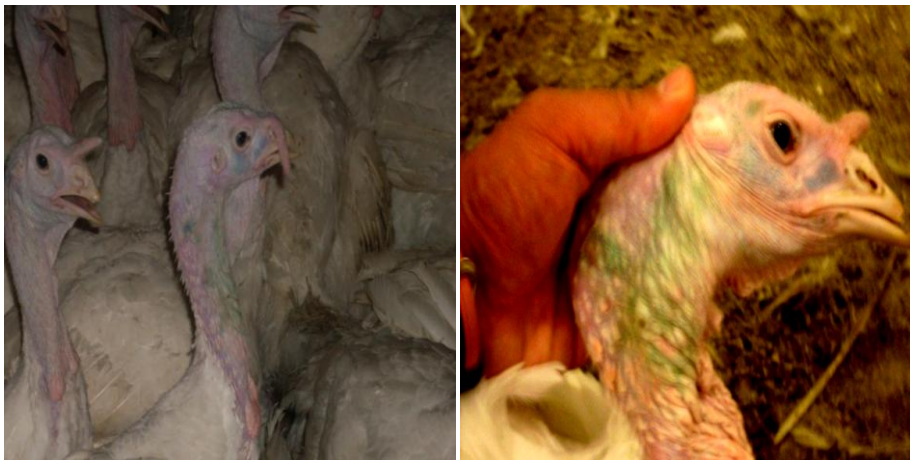


Figure 2.Cyanosis of the scalp.

Then they began to appear paralysis and paresis of separate groups of muscles and limbs. The bird was becoming weak and weak. And on the farm began a massive death of turkeys.

The corpse of a turkey is below average fatness. Visible mucous membranes are pale **(Figure 3).**

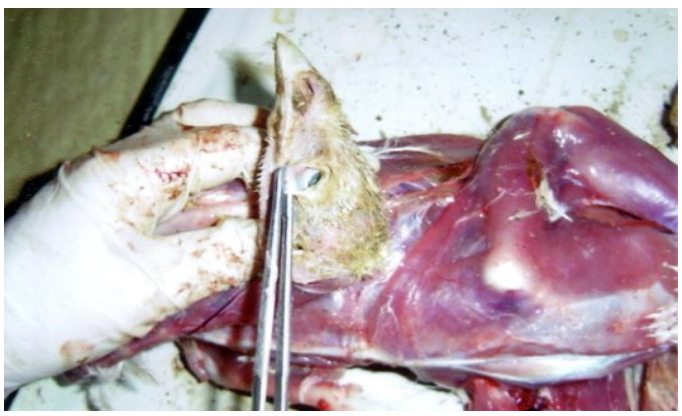


Figure 3.Corpse of turkey.Anemia of the conjunctiva.

The feathers are ruffled, dull, dirty, around the anus are stained with liquid feces of unpleasant odor, fatness is below average, the eyes are sunken. Subcutaneous tissue and serous membranes are dry **(Figure 4).**

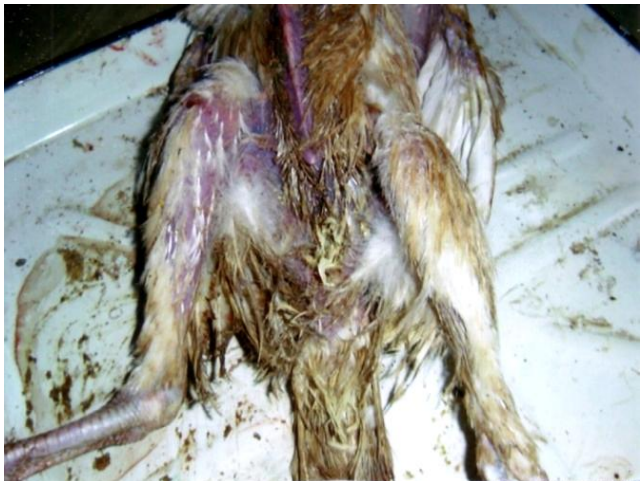
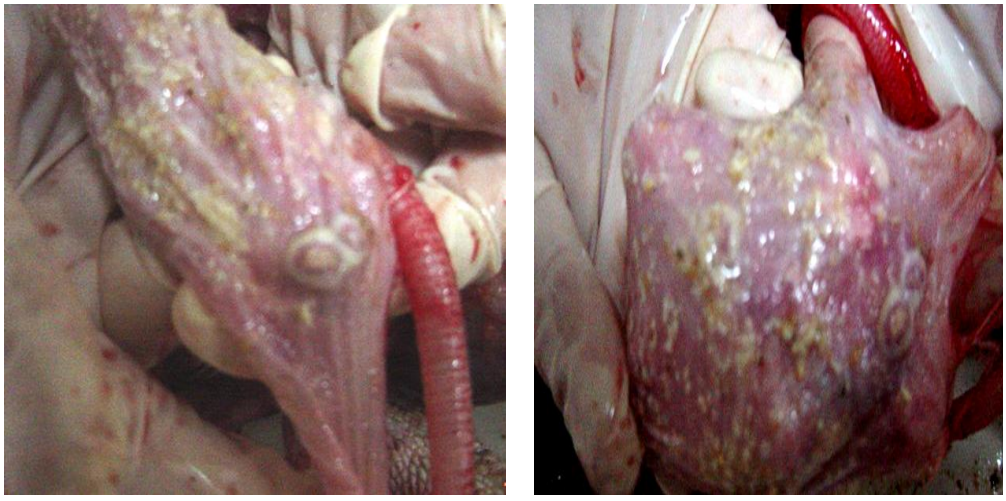


Figure 4.Contamination of the feathers around the anus by the feces.

Trachea - mucous membrane slightly swollen, intercellular blood vessels full-blooded. The lungs are slightly enlarged, testy, from the surface and on a pink incision. Air-borne bags are transparent.

Esophagus - mucous pale pink, granular, small-pimply. More often in the lower part of the esophagus there were isolated, larger, gray foci covered with fibrin flakes, in 12 cases the foci were multiple (Figure 5, A and B).



A. **B.**

Figure 5. Esophagus. On the mucosa there are single (A) and multiple foci (B) of diphtheritic inflammation

The goiter was stretched, its contents were semi-liquid and with gases.

The glandular stomach was without features, the papillae of the glands were weakly expressed, and covered with mucus from above.

Muscular stomach - no special features

Small intestine - the mucosa of the duodenum is thickened, swollen, reddened, covered with mucus, lean and iliac often in the form of narrow tubes, lumen narrowed, mucous membrane of grayish color, without features.

The thick part of the intestine is a mucous membrane of pale pink color, swollen, reddened with small punctate hemorrhages.

The liver is enlarged, the edges are dulled, unevenly colored, mosaic: the dark red color alternates with light brown, or mostly it is light brown in color, flabby consistency. On its surface there are foci of grayish-yellow color, from several foci to numerous, mostly with a pea and more. In relation to the total surface, they are located on the same level, or

slightly sway, have, as it were, meandering edges, sometimes merge with one another, on the cut also visible in the depth of the parenchyma **(Figures 6).**

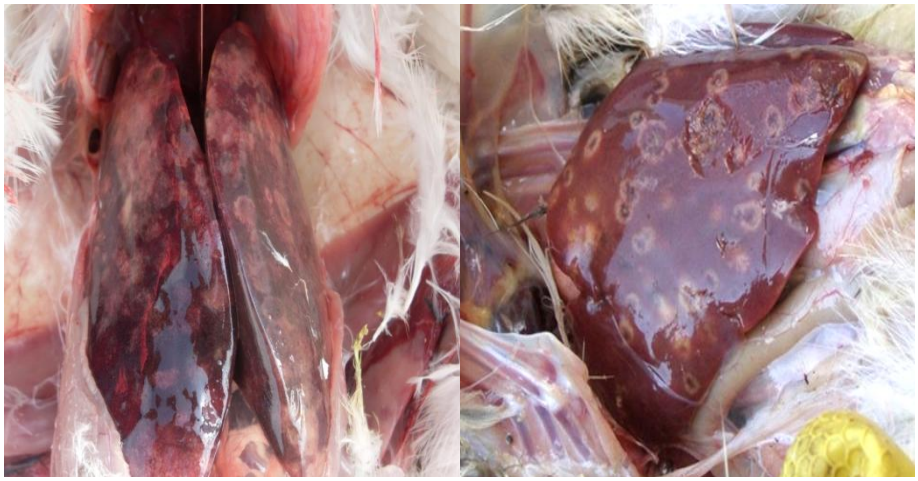
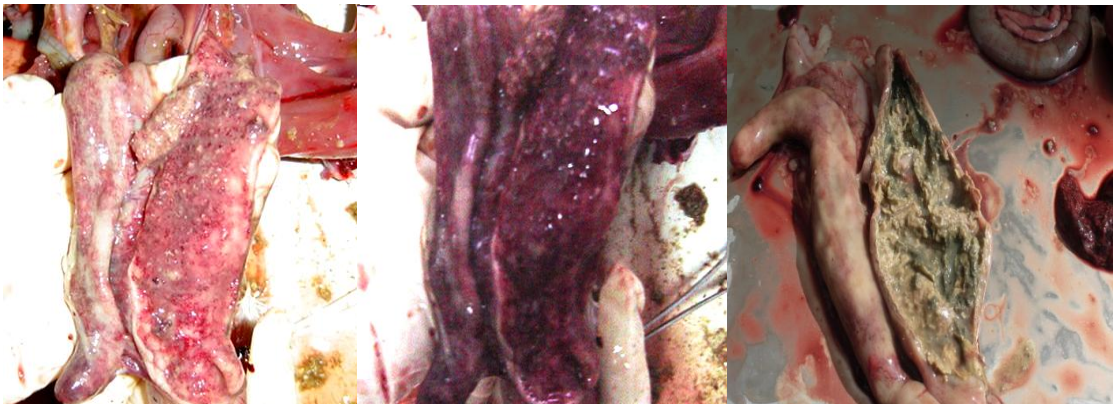


Figure 6.The liver. Enlarged, there are sharply delimited foci of necrosis pressed into the parenchyma.

Cecum - greatly enlarged in volume, serous membranes and mesentery sharply reddened, swollen, dull, in some cases there is glueing of cecum with each other with pressure they tear.

The mucous membrane is strongly swollen, dull, red in color, with hemorrhages, covered with curdled grayish overlays that are difficult to remove from the surface, located diffusely, or in the form of foci.

In some cases, the contents of the cecum are dirty, darker, coffee-colored, with an unpleasant odor (B). And in another part of the cases, the caecum in the cecum contained a confirmed fibrinous mass of yellowish color (B) (Figure 7).



A. **B.** **C.**

Figure 7. Cecum. The mucous membrane is light red (A), the color of coffee (B), with yellowish fibrin (C).

The rectum - mucous slightly reddened, the contents are often light brown in color, with an unpleasant odor.

Kidneys - either mosaic (red color alternating with light brown), or for the most part yellowish-brown color.

The brain is without features.

Pathological diagnosis: diphtheritic, sometimes fibrinous-hemorrhagic tiflitis, necrotizing hepatitis, catarrhal-desquamativeduodenitis, croupous diphtheritic diffuse or focal esophagitis, focal diphtheritic gastritis, congestive hyperemia and parenchymal dystrophy of the liver and kidneys, enlargement of esophagus glands, anemia of visible mucous membranes, exhaustion . For clarity, we give below tables that show data on the pathoanatomical picture.

Table 4. Frequency of pathological processes in the internal organs of turkeys that died from histomonosis									
Types of pathological changes	Heard	lungs	lever	kidneys	spleen	stomach	Small intestine	cecum	Esophagus
Hemorrhages	+	-	+	+	+	+	+	+	+
Hyperemia	-	+	+	+	-	-	+	+	-
Inflammation	-	-	-	-	-	+	+	+	+
Dystrophy	+	-	+	+	-	-	-	-	-
Necrosis	-	-	+	-	-	-	-	+	+

Table 4 significant changes in the histomonosis of turkeys are observed in the cecum and liver. Then, by descending in the esophagus, small intestine, kidneys, and the spleen there are no much changes.

Table 5. Pathological changes in turkeys which died from histomonosis			
Pathological changes	Investigated Corpses, quantity	Pathologoanatomical changes were found	
		quantity	B %
Diphtheric tiflitis	65	50	77
Fibrinous-hemorrhagic tiflitis	65	14	22
Foci necrosis in the liver	65	65	100
Catarrhal and desquamative duodenitis	65	65	100
Diphtheritic esophagitis	65	43	66
Focal diphtheritic gastritis	65	29	44
Congestive hyperemia of the liver and kidneys	65	65	100
Parenchymal dystrophy of the liver and kidneys	65	65	100
General anemia	65	65	100
Exhaustion	65	65	100

In the analysis of Table 5, we can note that diphtheria tiflitis, focal necrosis in the liver, diphtheria esophagitis, catarrhal-desquamativeduodenitis, general anemia and malnutrition were observed more often than others in the analysis of tablets.

Thus, analysis of autopsy results shows that the main pathoanatomical changes in the histomonose of turkeys develop in the blind intestines and in the liver. In all cases of the disease, we observed a lesion of cecum as the same time. The changes were expressed in the diffuse thickening of the wall of the affected bowel, accompanied by croup-diphtheritic inflammation of the mucous membrane.

The mucous membranes of the eyes and oral cavity were pale.

Changes in other organs were not natural and did not have the characteristic features for this disease.

At histological examination, as well as on autopsy, the main changes were found in the blind intestines and liver.

In the cecum, the changes determining the sharp wall thickening were in the submucosa layer. They were characterized by focal and diffuse cell clusters consisting of lymphoid-histiocytic elements. Cellular infiltrates were also found in the intermuscular connective tissue. In different parts of the mucosa, the degree of manifestation of changes was not the same. In some places only catarrhal and desquamative phenomena were observed, while in others all the mucous membrane, sometimes almost to the muscular layer, was in a state of dry necrosis. In the areas of necrosis, clumps and chromatin grains of decaying cell nuclei were detected **(Figure 8).**

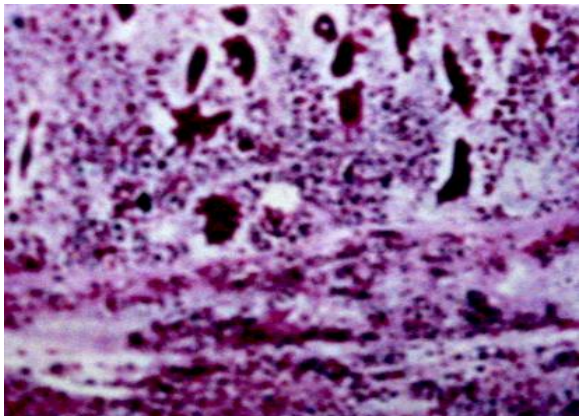


Figure 8.Histomonades in sections of the cecum.

At the heart of histological changes in the liver, the leading place was occupied by dystrophic, necrotic and inflammatory processes of the productive type. In the liver, foci of necrosis were located along the periphery of the lobules among the growing granulation tissue. Inflammatory cell clusters were focal and diffuse. Infiltrates consist mainly of lymphoid cells and histiocytes. Proliferative processes develop in the interlobular connective tissue.

The tissue form of histomonads in the affected wall of the cecum and liver was found in the form of rounded formations, 8-18 μm in diameter, well stained with the Schiff reagent, without a clearly expressed intracellular structure. Often, such histomonads were surrounded by an enlightened rim **(Figure 9).**

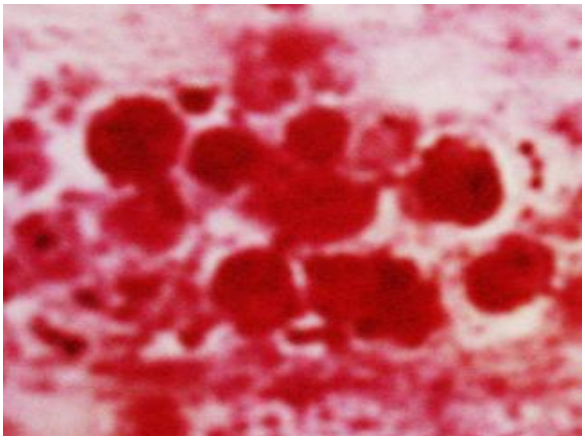


Figure 9.Histomonades, painted with Schiff-reagent.

In the smears-prints stained with hematoxylin-eosin, objects of predominantly oval form, 15 to 18 microns in length, surrounded by an unvarnished zone of lysed liver tissue were identified. Against the background of brightly lilac homogeneous protoplasm, one nucleus of oval form stained with hematoxylin was located in the center. As a result of hematoxylin-eosin staining, histomonad nuclei were identified **(Figure 10).**

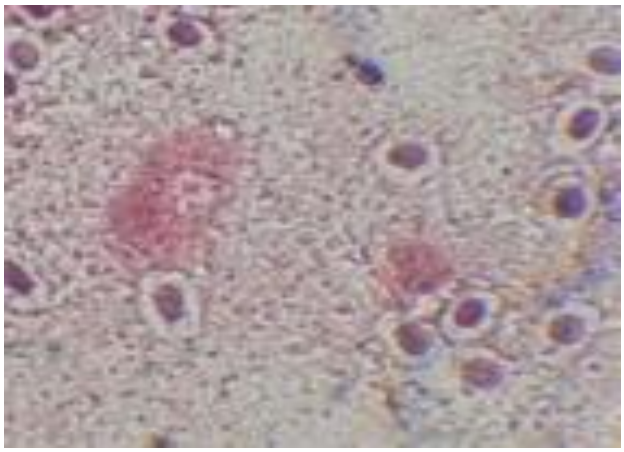


Figure 10.Liver imprint.Stained with haematoxylin-eosin, 40x10.

Conclusion

When diagnosing histomonosis to identify parasites, sections from the cecum and liver should preferably be stained with Schiff-reagent and microscopy of the smears of fingerprints of altered foci of the liver with staining them with hematoxylin-eosin.

The main preventive measures for histomonosis arefull-fledged feeding, observance of sanitary and preventive measures, thorough disinfection and disinfection, and compliance with sanitary and hygienic requirements for poultry care.

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