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Pathomorphological Changes in Intestinal Yersiniosis in Cats

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Abstract. Mammalian yersiniosis is of interest to researchers around the world, due to the expansion of the range of spread of the disease. Most publications of previous years concerned productive animals and the spread of the pathogen in environmental objects. Recently, isolated reports have begun to appear about the isolation of pathogens, yersinioses, from unproductive animals and a rather specific course of diseases. The interest of bacteriologists and epidemiologists is based on the increasingly frequent detection of *Y. Enterocolitica* pathogen in the body of so-called companion animals, which are often directly in close contact with the host. To date, the possibility of human infection from a bacterial carrier has already been proven. The purpose of this study was to clarify the main vectors of pathogenesis of spontaneous feline intestinal yersiniosis by investigating changes in the architectonics of parenchymal organs. The materials for the study were the internal organs of cats that died as a result of spontaneous intestinal yersiniosis, the diagnosis of which was established based on the clinical picture and the determination of diagnostic titres of antibodies to standard yersiniosis antigens. It was found that the feline disease is mostly asymptomatic, and due to the affinity of many pathogens of yersiniosis in animals and humans, it becomes both epizootological and epidemiological in nature. The most significant pathomorphological changes were found in the organs of cats with intestinal yersiniosis; in the organs of the gastrointestinal tract (stagnant phenomena, uneven damage to intestinal villi, sometimes crypts of the submucosal layer and vascular walls, active cell proliferation in desquamation areas); in the liver, mainly protein dystrophy, local areas of necrobiosis and cytolysis of hepatocytes; in the kidneys, congestive hyperaemia and the development of haemorrhagic diathesis, signs of vascular fragility and diapedesis bleeding, and in the case of chronisation of the process – a response to vascular endothelial damage in the form of migration of mononuclears to the future focus of inflammation; in the spleen and lymph nodes, delymphotisation of white pulp was observed, in other cases, an increase in the number of secondary lymph nodes with the formation of large reactive centres was detected in the spleen

Keywords: feline yersiniosis, pathomorphology, *Y. enterocolitica,* cellular changes

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INTRODUCTION

Earlier studies have established that intestinal yersiniosis is a zooanthroponotic disease caused by a gram-negative bacterium from the Yersinia genus (Yagüe-Muñoz *et al*, 2019; Maliy, 2016).

The disease is characterised by damage to the gastrointestinal tract, respiratory organs, arthritis, septicopyaemia, infertility in females, the birth of non-viable offspring (Tuompo et al, 2017; Miyata *et al*., 2022). Most reports indicate the dominance, prevalence, and increase in pathogenicity of Y. enterocolitica, among other Yersinia biovars (Orekhova, 2015; Razzuoli *et al*., 2020).

Therefore, when describing the pathoanatomical picture, the greatest attention was paid to intestinal yersiniosis. Furthermore, this is also conditioned upon the important epidemiological significance of the disease. Catarrhal-haemorrhagic gastroenterocolitis, serous peritonitis, synovitis, hepatosis of lesions of female genital organs, less often – other pathologies, especially complications of the underlying disease, are described in the published materials upon the autopsy of the corpses of various animals for intestinal yersiniosis (Remsty & Tennant, 2019). Scientific information about feline yersiniosis is still limited. The study of this problem in cats was influenced by the opinion of scientists J. Remsty and B. Tennant that Y. enterocolitica can be a natural commensal of the autochthonous microflora of this animal species (Remsty & Tennant, 2019). The authors believe that there has recently been a powerful impact on the ecosystem of many biotic and abiotic factors, which adversely affected the environment and one of its components – sapronic microflora, a representative of which is Yersinia. The biological properties of these bacteria were also changed by their pathogenic potential, which led to bacterial carrier and bacteraemia in many animals.

Most of the published research on this issue concerns the study of the biological properties of the pathogen, their contamination of products of animal and plant origin, diseases of productive animals (Marsicano *et al*., 2020; Nesbakken, 2021; McNally *et al*., 2004). But there is a lack of information regarding the pathological manifestation of feline intestinal yersiniosis. Considering the recent trend to popularise the breeding of small domestic animals prone to yersiniosis, or to being bacterial carriers of pathogenic variants of pathogens, the study of the problem of spread and transmission of this disease is truly relevant. Yersiniosis is also dangerous for humans. This fact is confirmed by the estimates of the Center for Disease Control and Prevention of the United States, which were presented in 2016. (Chlebicz & Śliżewska, 2018).

The purpose of this study was to establish characteristic pathoanatomic changes in cats with intestinal yersiniosis, which occurred spontaneously and tended to spread among other domestic animals, within the same farm.

LITERATURE REVIEW

According to existing information, Yersinia enterocolitica of biovars 2, 3, 4 and serotypes O:3 and O:9 are most often isolated in cats, which, as pathogens, are also common to humans (Marsicano *et al*., 2020; Shestakova *et al*., 2005; Wang *et al*., 2013).

Y. enterocolitica produces an enterotoxin, the effect of which is to activate adenylate cyclase of enterocytes, as a result of which the permeability of membranes increases and causes an active release of water and electrolytes – this very process causes the occurrence of diarrhoea and, as a result, causes dehydration. At the same time, the above-mentioned enterotoxin is rather difficult to distinguish from its counterparts (McNally *et al*., 2000). It is also noted that during parasitocoenosis of *Y. enterocolitica*, there is an increase in E prostaglandin, which confirms the role of sensitisation of the body in the development of the disease (Shestakova *et al*., 2005).

According to J. Remsty, B. Tennant, *Y. enterocolitica* most often does not cause a disease with pronounced clinical signs in cats and dogs (Remsty & Tennant, 2019). There is even an assumption that feline Yersinia enterocolitica is a natural intestinal commensal and therefore does not cause clinical manifestations of the disease and these animals can be long-term carriers (Smith, 2016; Cully, 2010).

The disease proceeds in the form of acute diarrhoea with mesodenitis. The causative agent not only causes inflammation of various parts of the intestine but is also capable of affecting parenchymal organs. The pathogen was isolated from cats with chronic peritonitis (Truba & Zon, 2021). Feline intestinal yersiniosis also revealed inflammation of regional lymph nodes (mainly pharyngeal), fever, arthritis, depression and apathy, anorexia and polydipsia, shortness of breath resulting from pneumonia (pulmonary form, rare), oedema, signs of septic shock, blood clotting disorders (McNally *et al*., 2000). A rather ambiguous signal about the disease is the presence of reactive arthritis, which affects the wrists, knee joints, and metatarsals and can turn into a chronic form and worsen even a month after the first episode of short-term diarrhoea (Bohn *et al*., 2019). The scientific literature describes the following changes, for spontaneous canine intestinal yersiniosis, during autopsy: catarrhal-haemorrhagic gastroenterocolitis, serous peritonitis, hepatosis, focal pneumonia, nephrosis, less often – other pathologies, especially for complications of the underlying disease (Maliy, 2016). Premature births, abortions, malformed foetuses, congenital anomalies, or infertility may result from infection with *Yersinia enterocolitica* in the first stage of pregnancy. The degree of severity of the disease differs in animals of the same age group and gender (Grahek-Ogden *et al*., 2007).

A review of the literature on this issue indicates a wide range of damage to organs and systems and does not allow identifying pathognomonic signs of the disease in cats, which substantially reduces the probability of a timely diagnosis.

MATERIALS AND METHODS

The study was conducted in 2017-2021 at the Department of Virology, Pathological Anatomy and Poultry Diseases of Sumy National Agrarian University, and during 2018-2021 – in private clinics of veterinary medicine in the Chernihiv and Sumy Oblasts of Ukraine.

Experimental studies, the results of which are presented in this paper, were based on the principles of moral values and compliance with all bioethical requirements defined by the Law of Ukraine No. 692 "On Humane Treatment of Animals" of 2008. All studies were conducted according to the bioethical requirements of the "Declaration of universal norms of bioethics (UNESCO, 2005) and Directive 2010/63/EU of the European Parliament and of the Council of 22 September 2010 on the protection of animals used for scientific purposes) and designed per Order No. 624 "On Changes to the Composition of the Commission on Bioethical Expertise of Scientific Research" of October 28, 2020.

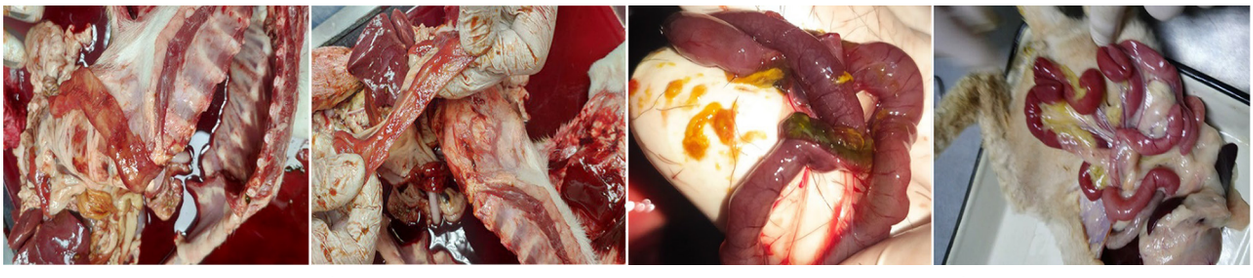
In this paper, pathological material from the corpses of cats was used, which during ex vivo diagnosis gave a positive indirect hemagglutination reaction (IHAR) and an agglutination reaction (AR) with yersiniosis antigens O:3 and O:9 produced by RAO "Biopreparat" (St. Petersburg), and antigens O:3, O:6.30 and O:9 of Ukrainian production, manufactured by the State Enterprise "Veterinary Medicine" of the National Research Centre "Institute of Experimental and Clinical Veterinary Medicine" of the National Academy of Agrarian Sciences of Ukraine (Kharkiv). The IHAR was established according to the macro method, by adding equal doses of antigen-sensitised erythrocytes to successive two-fold dilutions of serum. The finished mixture was left for 2-3 hours at room temperature. The results were evaluated as follows: if the serum contains antibodies to the pathogen that sensitised red blood cells, in this case, antibodies to Y. enterocolitica, hemagglutination will be noticeable, which is evaluated in crosses. The highest serum dilution was taken as the serum antibody titre, which provided hemagglutination by at least two crosses. According to the results of the study, a dilution of 1:200 was considered as the diagnostic titre.

Pathological autopsy and selection of pathological material was performed from four cat corpses according to the general "Rules for the selection of samples of pathological material, blood, feed, water, and their forwarding for laboratory research", which were approved by the head of the veterinary medicine department of the Ministry of Agriculture and Food of Ukraine P.P. Dostoevsky dated April 15, 1997, No. 15-14/111. Different clinical diagnoses were established in animals subjected to autopsy, *in vivo*, but all of them were serologically positive for intestinal yersiniosis.

Pathological material (pieces of the stomach, small and large intestines, lymph nodes, liver, spleen, kidneys) was fixed in a 10% aqueous neutral formalin solution for up to 30 days, then washed in tap water and dehydrated with alcohol solutions of increasing concentration from 40° to 96° for a day in each and double treatment with absolute alcohol (100°) for 12 hours in each solution. The material was embedded in paraffin, forming blocks, from which sections were made on a microtome, which were subsequently stained with Carazzi's haematoxylin for 10 min and with a 1% aqueous solution of eosin for 30 sec. Stained and dried sections were placed in a Canadian balm. The section was examined under a Biolam R 15 light microscope with a primary magnification of 100 and an added magnification of 400 times. The photo was taken using a Digital Camera M 1000 PLAS series LEVENHUK Microscope using a Lenovo G 50-70 with Microsoft 10 software.

RESULTS AND DISCUSSION

Pathoanatomical examination of cats with spontaneous yersiniosis revealed mainly catarrhal enteritis or enterocolitis with single petechial haemorrhages. The mucous membrane with local redness, swelling, without natural shine, with manifestations of folding and loosening. An excessive accumulation of mucus was found in the lumen of the intestine, sometimes with splashes of blood. The vessels of the mesentery are excessively filled with blood, the lymph nodes are enlarged, the features of serous lymphadenitis with hyperaemic areas are visible on the section (Fig. 1 a-d).



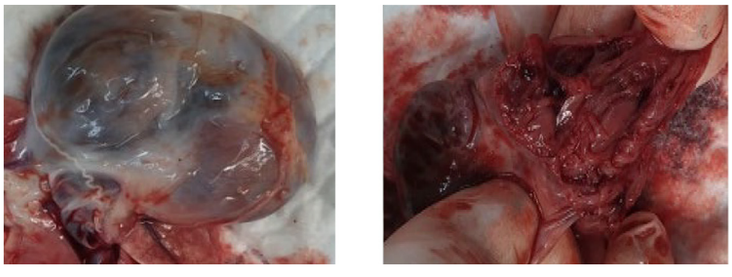
a b c d

***Figure 1**. 1 a-d. Pathoanatomical manifestation of acute enterocolitis with petechial haemorrhages*

Signs of serous peritonitis with polyserositis were observed in the abdominal cavity, which indicated the presence of fibrin flakes. In this case, parenchymatous organs showed signs of local maceration (Fig. 2 a-b). An autopsy of one of the cat's corpses revealed a changed shape of an enlarged heart, a myocardium of flaccid consistency with thinned walls (Fig. 3 a-b).

a b

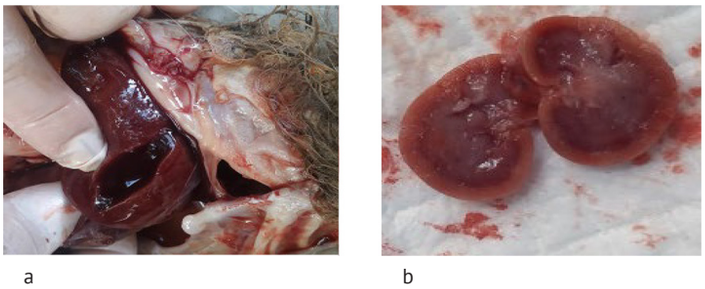
Figure 2. *Serous peritonitis (a) and fibrinous overlays (polyserositis) on the internal organs of a cat (b)*



a b

Figure 3. *Changes in heart shape (a), thinning of the myocardial walls(b)*

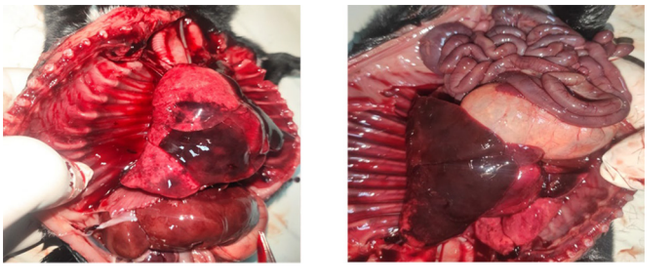
Pathoanatomically, blunted edges, discoloration, and capsule tension were detected in the liver. The colour is heterogeneous, it has a shade from dark cherry to clay, the consistency is loose, the pattern is smoothed on the section, the scraping is moderate. The gallbladder was full of bile in all cases. The kidneys had signs of hyperaemia and granular dystrophy (Fig. 4 a-b).



a b

Figure 4. *Hyperaemia and granular dystrophy of the liver (a) and kidney (b) of a cat*

Examination of the lungs revealed congestive hyperaemia and petechial haemorrhages (Fig. 5 a-b), which occurred due to difficult outflow of blood from the organ due to compression of the heart and compaction of the lungs themselves. Hypoxia occurred in the tissues and the hydrostatic pressure in the vessels increased, as a result of which the permeability of the capillary walls increased, which led to irreversible changes at the micro level. Furthermore, there was a growth of connective tissue in the interalveolar septa, which led to compaction of the lungs and disruption of their functioning.



a b

Figure 5. *Congestive hyperaemia (b) and petechial haemorrhage (a) in the lungs of cats*

At the microscopic level, signs of congestive processes and catarrhal bronchopneumonia were found in the affected areas of the lungs. Histological changes in the liver were characterized by granular dystrophy, with signs of focal necrosis of hepatocytes and liver beams (Fig. 6).

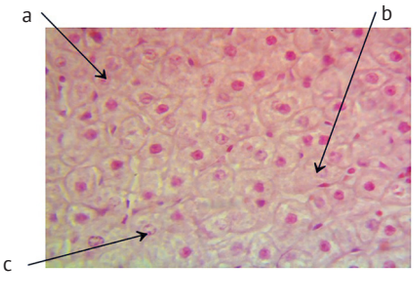
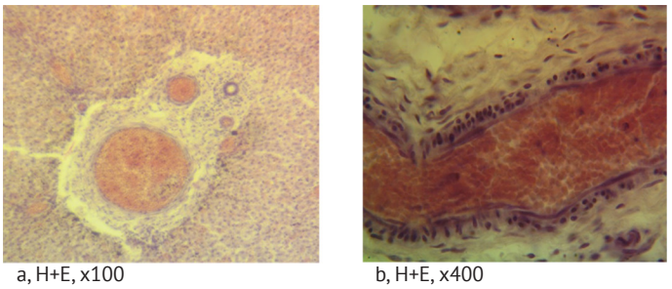


Figure 6. *Granular dystrophy and signs of liver necrosis, H+E, x400*
Note: *a – pyknosis, b – rhexis, c – lysis of hepatocyte nuclei*

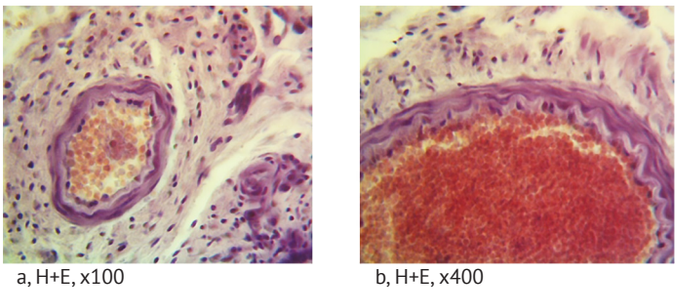
Hyperaemia and perivascular oedema were observed due to effusion of fluid from the liver vessels. A substantial sign of the duration of this process is the pronounced reproduction and proliferation of various populations of macrophages, which are located both in the vascular membranes themselves and in their surrounding space (Fig. 7).



a, H+E, x100 b, H+E, x400

Figure 7. *Hyperaemia of the liver (a) and perivascular oedema (b)*

A change in the morphological state of the endothelium of vessels is quite noticeable, which can affect their anatomical and physiological features, namely rigidity, elasticity, plasticity, etc. Filled with blood, large vessels exert pressure on their smaller representatives in the microcirculatory channel and on the bile ducts of the liver, which provokes their further dysfunction (Fig. 8 a-b).



a, H+E, x100 b, H+E, x400

Figure 8. *Perivascular proliferation (a) on the background of congestive hyperaemia in the liver*

When assessing morphological changes in the kidneys, it should be noted that the infectious process is accompanied by congestive hyperaemia and the development of haemorrhagic diathesis. Foremost, long-standing septic processes destroy the endothelial layer of vessels and the basal membrane, thereby causing vessel fragility and diapedesis bleeding (Fig. 9). However, in the case of chronicity of the process, a response to vascular endothelium damage was observed in the form of migration of mononuclear cells to the future focus of inflammation, which indicates the possible permanent presence of microorganisms in these areas. Furthermore, the limited number of polymorphonuclear leukocytes in these sites indicates that the absence of stimuli for the development of exudative-destructive inflammation (Fig. 10).

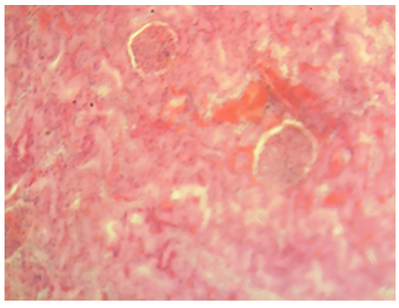


***Figure 9**. Hyperaemia and haemorrhagic diathesis in the kidney, H+E, x100*

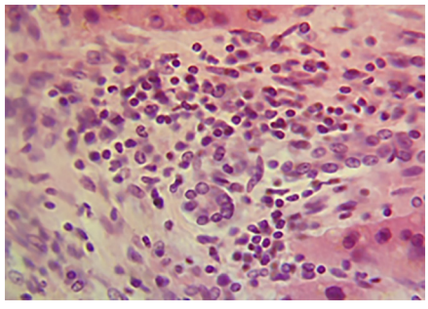


***Figure 10**. Active cellular response in the structures of the brain layer of the kidneys, H+E, x400*

In the lymph nodes, a general decrease in lymphocytes, cellular devastation of the reactive centres of the follicles was observed, which can be a sign of both the chronic course of the disease and the development of secondary immunodeficiency (Fig. 11). Under these conditions, the percentage ratio of stromal elements increased relative to the parenchyma of the organ.

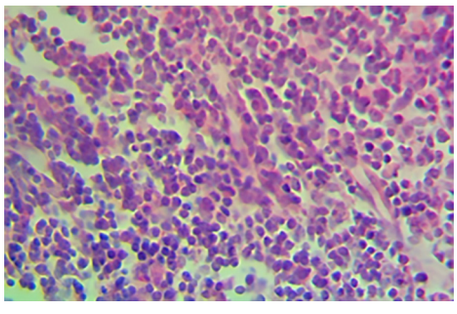


***Figure 11**. Lymph node delymphotisation, H+E, x400*

Morphological changes in the spleen in some cases were characterised by an increase in the number of secondary lymph nodes with the formation of large reactive centres, where numerous blast forms and cells are found in a state of mitosis. The periarterial zone increased insubstantially against the background of a substantial increase in the marginal zone of lymphatic follicles (Fig. 12 a-b).

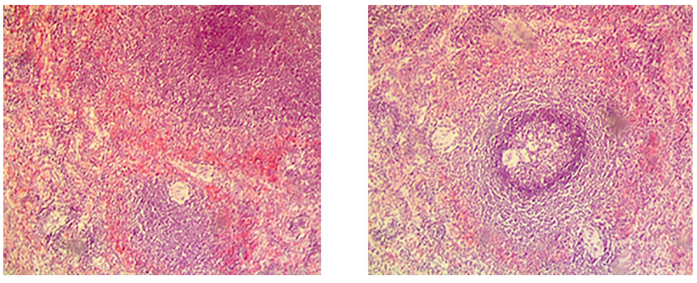
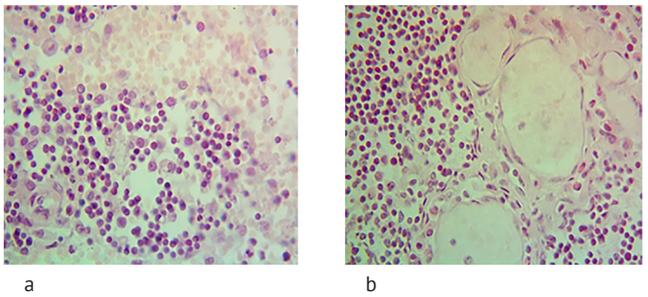


Figure 12. *Hyperplasia of the lymphoid nodule of the spleen (a) with an enlarged reactive centre (b), H+E, x200*

In other cases, pronounced delymphotisation of the white pulp of the organ was found, and in some cases, the formation of small cystic cavities. The nuclei of reticulocytes in such cases were more poorly detected against the background of an increase in the number of haemosiderophages (Fig. 13 a-b).



a b

Figure 13. *Delymphotisation (a) and formation of small cystic cavities (b), H+E, x400*

Changes in the intestine: uneven damage to the villi, less often crypts of the submucosal layer. In certain areas, the destruction of the walls of blood vessels is detected. Active cell proliferation is observed in the foci of damage, especially against the background of desquamative catarrh (Fig. 14).

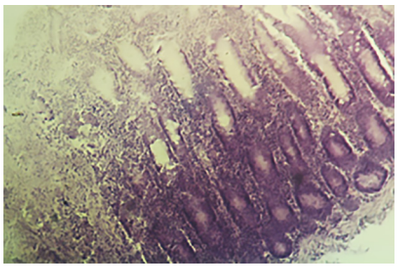
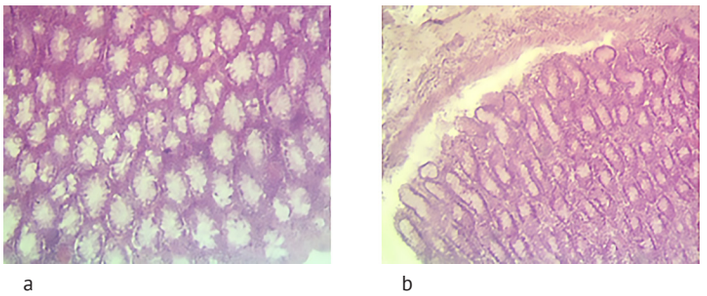


Figure 14. *Uneven damage to the villi of the small intestine*

Goblet cells, mostly crypts, were filled with secretions. Lymphoid nodules and Peyer's patches are swollen in places, hyperplastic due to the active movement of lymphoid elements towards the alterative areas; their content was often depleted (Fig. 15 a-b).



a b

Figure 15. *Hypersecretion of goblet cells (a) script of the small intestine (b), H + E, x200*

Catarrhal desquamative processes were less pronounced in the large intestine. A pronounced reaction was mainly observed on the part of goblet cells, in which mucus accumulated in excessive amounts (Fig. 16).

Figure 16. *Reaction of goblet cells of the large intestine, H+E, x200*

Pathoanatomical studies of cats with spontaneous yersiniosis revealed mainly catarrhal processes on the mucous membranes of the gastrointestinal tract. Petechial haemorrhages were found in the small intestine. Against this background, the intestinal vessels were excessively blood-filled, lymphodenopathy was observed, and the lymph nodes themselves, on the incision, had signs of serous lymphadenitis. According to the testimony of various researchers (Marsicano, *et al*., 2020; Truba & Zon, 2021; Chebanyuk, 2013), catarrhal and haemorrhagic processes with a desquamative tendency are detected in various animals with intestinal yersiniosis, less in the stomach, and mainly in the small and large intestines. Cytoarchitectonic changes in the intestine contained signs of catarrh with uneven villi damage. In isolated cases, damage to the submucosal crypt and vascular walls was found. A rather significant cellular response was also recorded in places where the submucosal layer is disturbed, especially against the background of intense damage. The mucous membranes were swollen and riddled with haemorrhages.

Histological changes in the liver were characterised by granular dystrophy, signs of focal cytolysis of haepatocytes and liver beams. Hyperaemia and perivascular oedema were observed due to effusion of fluid from the liver vessels. The duration of this process is indicated by the pronounced reproduction and proliferation of various macrophage populations, both in the membranes of blood vessels and in the surrounding space. The studies covering intestinal yersiniosis in animals and humans describe active dystrophic-necrotic processes, especially during the pathogenic effect on the liver of *Yersinia enterocolitica* serovars O:9 and O:4 (Borysevich *et al*., 2015; Ochoa & O'Ryan, 2018; Byun *et al*., 2011). In the authors' research, during spontaneous infection of cats with *Yersinia enterocolitica* serovar O:9, similar data were obtained, which indicates a pronounced effect of this serovar on the gastrointestinal tract of animals.

Evaluating the detected morphological changes in the kidneys, it should be noted that the disease causes stagnant hyperaemia and the development of haemorrhagic diathesis. It is probable that bacteremia and toxaemia destroy the vascular endothelial layer and basement membrane, causing vascular fragility and diapedesis bleeding. However, in the case of chronicity of the process, a response to vascular endothelium damage occurs in the form of migration of mononuclear cells to the future focus of inflammation, which indicates the possible permanent presence of microorganisms in these areas. Furthermore, the limited number of polymorphonuclear leukocytes in the specified areas indicates the absence of stimuli for the development of exudative-destructive inflammation. Studies of some scientists describe mainly dystrophic, less often – inflammatory processes in these organs (Smith, 2016; Chebanyuk, 2013; Maliy, 2016). According to the authors, the expansion of the spectrum of lesions may be due to many factors that, for certain reasons, may not be considered due to the lack of anamnestic data and the possibility of diagnostic studies.

In the lymph nodes, a general decrease in lymphocytes, cellular devastation of the reactive centres of the follicles was observed, which can be a sign of both the chronic course of the disease and the development of secondary immunodeficiency.

Morphological changes in the spleen in some cases were characterised by an increase in the number of secondary lymph nodes with the formation of large reactive centres, where numerous blast forms and cells are found in a state of mitosis. The periarterial zone increased insubstantially against the background of a substantial increase in the marginal zone of lymphatic follicles. In other cases, pronounced delymphotisation of the white pulp of the organ was found, and in some cases, the formation of small cystic cavities. The nuclei of reticulocytes in such cases were worse against the background of an increase in the number of haemosiderophages.

It is known that when a bacterial antigen enters the body, a serous-fibrinous inflammation develops in the local tissue, which is initially accompanied by a microphagic (neutrophil) reaction, which is later replaced by a macrophage reaction. Under the conditions of antigenic stimulation (sensitisation), characteristic changes in organs of the lymphoid tissue occur.

Antigenic irritation of any nature in infectious and invasive diseases causes some morphological changes

in the organs of the central and peripheral immune system (Shestakova *et al*., 2005; Razzuoli *et al*., 2020). Therefore, the changes in the relevant organs detected by the authors can be associated with the influence of *Yersinia enterocolitica* on the body of cats.

Immunomorphological changes in the organs of the immune system are known to be characterised by the following reactions of the same type. The micro- and macrophage reaction is accompanied by an increase in the number of neutrophils and macrophages in the sinuses, blood vessels, organ parenchyma, antigen phagocytosis by them, the same changes are present during infection in the lymph nodes, spleen, bone marrow, tonsils, Peyer's plaques, and lungs. That is why the authors chose these organs for research, where active immunogenesis occurs.

The so-called blast reaction of B-lymphocytes (blast transformation) is characterised by an increase in the number of secondary lymphoid follicles with large reactive centres containing B-lymphoblasts in the lymph nodes, spleen, tonsils, Peyer's patches, and lungs. The authors also determined signs of this reaction during pathomorphological studies.

The authors also found the blast reaction of T-lymphocytes, which is accompanied by the expansion of the thymus-dependent zones of the paracortical layer of lymph nodes and the periarterial zone of the lymphatic follicles of the spleen due to the intensive proliferation of T-lymphoblasts, in the corresponding parts of the organs. An increase in the number of secondary lymph nodes in the spleen, tonsils, Peyer's patches (aggregated lymph nodes) was usually pronounced when bacterial antigens were administered. However, the devastation of the lymph nodes, revealed during the study, is considered to be a consequence of impaired immunogenesis.

Thus, the identified changes in the peripheral lymphoid tissue are most inherent in antigenic stimulation (sensitisation) of the body and are manifested by a macrophage reaction, hyperplasia of lymphocytes, with their successive plasmacytic transformation, increased permeability of microvessels, and interstitial oedema. Established changes in the intestine may indicate an enzymopathy of the intestinal glands, and damage to lymphoid nodes is one of the causes of impaired immune response in general.

CONCLUSIONS

1. Intestinal yersiniosis of cats, during a spontaneous course, is patho-anatomically characterised by pronounced catarrhal changes mainly in the small intestine, inflammation of abdominal lymph nodes, stagnant, dystrophic processes in the liver and lungs. In the abdominal cavity, in several cases, signs of serous peritonitis with polyserositis were observed, and in the kidneys – signs of hyperaemia and protein dystrophy, in the lungs – stagnant hyperaemia and petechial haemorrhages.

2. Histological changes in spontaneous intestinal yersiniosis in cats were characterised by hyperaemia and perivascular oedema, granular dystrophy with signs of focal necrosis of hepatocytes and liver beams, in the kidneys – stagnant hyperaemia and the development of haemorrhagic diathesis, in the lymph nodes by lymphotisation, in the spleen, in some cases, an increase in the number of secondary lymph nodes with the formation of large reactive centres, and others with pronounced devastation of the white pulp of the organ. Changes in the intestine were defined as an uneven catarrhal inflammation with partial or complete damage to the villi, sometimes the crypt and the endothelium of the vessels.

Further research will be aimed at developing diagnostic tests for intestinal yersiniosis in small animals on a molecular genetic basis.

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Патоморфологічні зміни за кишкового ієрсиніозу котів

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Анотація. Ієрсиніози ссавців викликають зацікавленість дослідників різних країн світу, у зв'язку з розширенням ареалу розповсюдження хвороби. Переважна кількість публікацій минулих років стосувалась продуктивних тварин та розповсюдження збудника в об'єктах довкілля. В останні роки почали з'являтися поодинокі повідомлення про виділення збудників, ієрсиніозів, від непродуктивних тварин та досить специфічний перебіг хвороб. Зацікавленість бактеріологів та епідеміологів базується на все частішому виявленні збудника *Y. Enterocolitica* в організмі так званих тварин-компаньйонів, які часто безпосередньо знаходяться в тісному контакті з господарем. На сьогоднішній день вже доведена можливість інфікування людини від бактеріоносія. Метою досліджень було з'ясувати головні вектори патогенезу спонтанного кишкового ієрсиніозу котів шляхом вивчення змін в архітектоніці паренхіматозних органів. Матеріалами для досліджень були внутрішні органи котів, які загинули внаслідок спонтанного кишкового ієрсиніозу, діагноз який встановлювали на підставі клінічної картини та визначення діагностичних титрів антитіл до стандартних ієрсиніозних антигенів. Встановлено, що захворювання у котів перебігає переважно безсимптомно, а у зв'язку зі спорідненістю багатьох збудників ієрсиніозу у тварин і людини набуває як епізоотологічного, так і епідеміологічного характеру. Виявлені найбільш суттєві патоморфологічні зміни в органах котів за кишкового ієрсиніозу; в органах шлунково-кишкового тракту (застійні явища, нерівномірне пошкодження ворсинок кишечнику, іноді крипт підслизового шару та стінок судин, активна клітинна проліферація в ділянках десквамації); в печінці – переважно білкові дистрофії, локальні ділянки некробіозу і цитолізу гепатоцитів; в нирках виявляли застійну гіперемію та розвиток геморагічного діатезу, ознаки ламкості судин та діапедезну кровоточивість, а в разі хронізації процесу – відповідь на ушкодження ендотелію судин у вигляді міграції мононуклеарів у майбутнє вогнище запалення; в селезінці та лімфатичних вузлах спостерігалась делімфотизація білої пульпи, в інших випадках в селезінці виявляли збільшення кількості вторинних лімфатичних вузликів з утворенням великих реактивних центрів

Ключові слова: ієрсиніоз у котів, патоморфологія, *Y. enterocolitica*, клітинні зміни