



UDC 619:616.3-002-091:636.8
DOI: 10.31548/ujvs.13(4).2022.35-41

Microscopic changes in the spleen due to feline infectious peritonitis

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Abstract. The relevance of the study is that pathological and morphological changes with feline infectious peritonitis have been studied by few authors and are not fully described. The purpose of this study was to investigate the effect of the causative agent of infectious peritonitis on the structure of the spleen in cats. The paper highlights the results of histological studies of sections obtained from distinct parts of the spleen of cats of different ages who died from mixed (26 animals) and dry (7 animals) forms of infectious peritonitis. Sections were stained with haematoxylin and eosin according to the generally accepted method. The paper describes the details of microscopic changes in the spleen in dry and mixed forms of feline infectious peritonitis. It was found that these changes are not affected by the form of the disease but are characterized by features depending on the duration of its course. In cats in which the disease lasted up to three weeks before death, the red pulp of the spleen was unevenly swollen, infiltrated by lymphocytes and monocytes, in some places contained foci of necrotic cells, and red blood cells were absent. Changes in the white pulp were represented by hyperplasia of lymphoid nodules. These nodules were of varied sizes and were located eccentrically relative to the central arteries. There are no distinct lymphoid nodules around part of the central arteries. On the surface of the capsule, fibrinous-necrotic overlays are present in places, under which there is no mesothelium, and the capsule is infiltrated with lymphocytes and monocytes. In other areas, mesotheliocytes underwent distinct metaplasia – from flat cells, they turned into columnar cells. In some areas of the spleen, some animals have no serous membrane. In cats with the disease lasting over three weeks, the red pulp is noticeably more swollen, and the lymphoid nodules are single and small. Other microscopic changes were the same as in animals that were ill for less than three weeks. The results of the study are of practical value for pathologists, as well as for scientists investigating the pathogenesis of feline infectious peritonitis

Keywords: coronavirus, macroscopic changes, histological studies, lymphoid nodules, metaplasia

Suggested Citation:

Lisova, V., & Kotliarov, E. (2022). Microscopic changes in the spleen due to feline infectious peritonitis. *Ukrainian Journal of Veterinary Sciences*, 13(4), 35-41.

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Introduction

Coronaviruses in humans and animals cause diseases of the respiratory system, gastrointestinal tract and central nervous system (Enjuanes *et al.*, 2006; Boley *et al.*, 2020). They can adapt to new environments through mutations and recombination with relative ease, and therefore are programmed to effectively change the host range and tissue tropism (Li *et al.*, 2006; Graham & Baric, 2010; Li, 2013). One of the diseases caused by coronaviruses is feline infectious peritonitis, a disease diagnosed in cats worldwide (Andrew, 2000). Currently, feline infectious peritonitis is considered as a viral immunological disease (Kipar *et al.*, 2001).

The coronaviruses affecting this animal species exist in the form of two pathogenic types – low-virulent, called feline intestinal coronaviruses, and virulent – feline infectious peritonitis viruses. The first pathogenic type dominates, since infection with feline coronaviruses occurs by faecal-oral route and pathogens, first of all, enter the intestines. Therewith, the disease does not progress, or manifests itself only with mild enteritis. However, in 4-5% of adult cats and 5-10% of kittens, at some point, after exposure to coronaviruses, infectious peritonitis develops (Addie *et al.*, 2009), as a result of de novo emergence of highly virulent coronaviruses that are formed from low-virulent coronaviruses through mutations in the body of a particular infected cat. This usually occurs without horizontal transmission of the pathogen (Vennema *et al.*, 1998).

In vitro studies have proved that the expansion of the spectrum of cellular pathogenicity of feline coronaviruses, which ensures the growth of their virulence, occurs as a result of efficient and stable replication in macrophages, and not as a result of the expansion of the ability to penetrate the cell (Stoddart *et al.*, 1989).

Presently, it is known that the infectious peritonitis virus is a mutation of the feline intestinal coronavirus, leading to the formation of numerous infectious peritonitis viruses (Stoddart *et al.*, 1989; Vennema *et al.*, 1998). Mutations of enteric coronavirus to infectious peritonitis virus occur frequently and relatively mildly, and probably, occur in a unique and highly variable region of the feline enteric coronavirus genome. At the end of the last century, it was established that infectious peritonitis viruses evolved as deletion mutations of feline intestinal coronaviruses (Poland *et al.*, 1996; Vennema *et al.*, 1998). To date, two genotypes of feline coronavirus are known: feline type I coronavirus, which prevails in the field (Addie *et al.*, 2003; Li *et al.*, 2019) and feline type II coronavirus, which emerged as a result of recombination between feline coronavirus type I and canine coronavirus (Decaro *et al.*, 2008; Terada *et al.*, 2014).

Both serotypes of feline coronavirus can cause infectious peritonitis, but cat populations are certainly dominated by type I coronaviruses, with the number of seropositive animals reaching 98%. Therewith, feline type I coronaviruses induce higher antibody titres than type II coronaviruses and are more likely to cause intestinal diseases and infectious peritonitis. Therewith, a higher prevalence of type II coronaviruses was reported, which ranged from 10% to more than 30% in cats with infectious peritonitis, and sometimes mixed infection with type I and II coronaviruses (Kipar *et al.*, 2014).

According to Levy *et al.* (1999), in 70% of cases, the disease is registered in animals under the age of 1 year.

According to Addie & Jarrett (1998), infectious peritonitis affects cats of all ages, but typically 50% of affected cats are under 2 years of age. At the same time, McReynolds & Macy (1997) emphasize that all cats are susceptible to infection with infectious peritonitis, but the clinical manifestation of the disease is most often observed in young cats aged 3 months to 3 years. Furthermore, it was established that this disease is more common in non-neutered cats compared to neutered ones (Pesteanu-Somogyi *et al.*, 2006; Hartmann, 2000). The incidence of infectious peritonitis is not affected by the age of cats, their gender, methods, and methods of raising and keeping, as well as the presence or absence of concomitant diseases (Foley *et al.*, 1998).

Based on pathological and morphological changes, there are three forms of infectious peritonitis: wet, dry, and mixed forms. In case of the latter, changes inherent in both dry and wet infectious peritonitis are recorded. The wet form of the disease is characterized by polyserositis (chest and abdominal effusion) and vasculitis, and the dry form by granulomatous lesions of various organs (Kipar *et al.*, 2005).

In infectious peritonitis, the pathogenesis of the disease and pathological and morphological changes are understudied (Kipar *et al.*, 2014). In this regard, the purpose of this study was to establish pathological and morphological changes in the spleen of cats in mixed and dry forms of infectious peritonitis.

Materials and Methods

The study was conducted during 2019-2022 at the Faculty of Veterinary Medicine of the National University of Life and Environmental Sciences of Ukraine (Kyiv). As a result, a histological examination of the spleen was performed from 26 cadavers of cats that died from a mixed form of infectious peritonitis, 7 cadavers of animals that died from a dry form of this disease, and 5 cadavers of cats (control) that died as a result of lethal injuries. All cats were of different breeds and ages.

The autopsy of the cadavers of all cats was performed by partial evisceration in the dorsal position (Zon *et al.*, 2009). According to this method, after external examination of the cadaver, skin removal and examination of subcutaneous tissue, skeletal muscles and somatic lymph nodes, all organs of the oral, neck, and chest cavities were removed in a single complex, after which they were examined, paying attention to macroscopic changes visible to the naked eye. Organs of the gastrointestinal tract were separated from the abdominal and pelvic cavities as a single complex, and all macroscopic changes visible to the naked eye were recorded. Together with the organs of the gastrointestinal tract, the mesentery, mesenteric lymph nodes, and pancreas were removed and examined. Macroscopic changes in the liver, spleen, kidneys with ureters, adrenal glands, and bladder were removed from each cat's corpse separately, and genitals were removed and investigated in a single complex.

During the autopsy, pieces of the spleen were selected for histological studies. From each spleen, at least 5 pieces were obtained from various parts of the organ – from its central and peripheral parts. In cats that died from infectious peritonitis, areas of the organ were necessarily selected for histological studies, both covered from the surface with fibrinous-necrotic overlays, and without them.

All pieces of spleen were fixed in a 10% aqueous solution of formalin (pH 7.2-7.4) for a week, dehydrated at room temperature in a series of ethanol of increasing concentration (60°, 70°, 80°, 96°, and 100°) and kept in each of them for one day. Subsequently, they were transferred to a mixture of 100° ethanol and chloroform in a 1:1 ratio, where they were kept for one day at room temperature. Next, these pieces were placed in chloroform, in which they were also kept for one day at room temperature, and subsequently, they were kept in a thermostat TSO-80 (Ukraine) in a mixture of chloroform and paraffin in a 1:1 ratio at 37°C. Then, in the same thermostat, they were kept in liquid paraffin at 56°C. After that, pieces of spleen were poured into paraffin. Histological sections were made using a sledge microtome MS-2 (Ukraine), pasted onto glass slides with a mixture of egg albumin and glycerol in a 1:1 ratio, and then the sections were stained with Carazzi haematoxylin and eosin (Goralskij *et al.*, 2011). During staining, the sections were deparaffinized in xylene for 7 min and passed through 100° and 70° ethanol, keeping in each of them for 5 min. Further, the sections were stained with Carazzi's haematoxylin. Subsequently, they were stained with a 0.5% aqueous solution of eosin, dehydrated in 70° and 100° ethanol, clarified in xylene, and embedded in Canadian balsam. The manufactured histological preparations were investigated and photographed using an MC 100 LED microscope (Austria).

Results and Discussion

During the autopsy, it was found that macroscopic changes in the spleen of all cats that died from mixed and dry forms

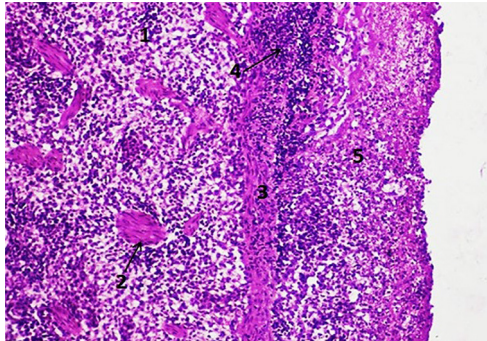


Figure 1. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 50
Note: 1 – red pulp; 2 – trabeculae; 3 – capsule; 4 – infiltration of the capsule by lymphocytes and monocytes; 5 – fibrinous-necrotic overlays

Therewith, macrophages were not detected, which indicates the absence of transformation of monocytes into macrophages, which could be explained by both the lack of appropriate stimulation and damage to the mechanisms of the monocytes themselves responsible for such transformation.

Mesothelium under fibrinous-necrotic overlays was absent, and the spleen capsule in these areas was infiltrated by lymphocytes and monocytes (Fig. 1). Other researchers have only stated the presence of such overlays, but have not described their composition, namely intact lymphocytes and monocytes and destruction of mesotheliocytes under such overlays (Kipar & Meli, 2014).

of infectious peritonitis are similar, but have certain features, depending on the duration of the disease. Thus, in animals that were ill for less than 3 weeks before the onset of death, fibrinous-necrotic overlays of varied sizes, shapes, and localization were found on the surface of the organ. Analogous fibrinous-necrotic overlays on the surfaces of various internal organs of cats that died from a dry form of infectious peritonitis were described by other authors (Wolfe & Gricsemer, 1966; Kipar *et al.*, 2005). Compared to animals in control, the pulp of the spleen was somewhat pale.

In animals that were ill for more than 3 weeks before death, apart from fibrinous-necrotic overlays on the surface of the spleen and paler pulp, compared to cats in the control, atrophy of this organ was established. Macroscopically, such atrophy was manifested by a flaccid, sometimes (in 7 animals, or 21.2% of cases) slightly wrinkled capsule and flaccid pulp.

The results of histological studies of the spleen of cats who died from infectious peritonitis show that microscopic changes in this organ were similar in animal corpses, regardless of the form of the disease, age, and gender. However, these changes varied depending on the duration of the disease before death.

In animals that were sick for up to 3 weeks before the onset of death, fibrinous-necrotic overlays were found on the surface of the capsule in places (Fig. 1).

A small number of unchanged lymphocytes and monocytes were found in these fibrinous-necrotic overlays (Fig. 2).

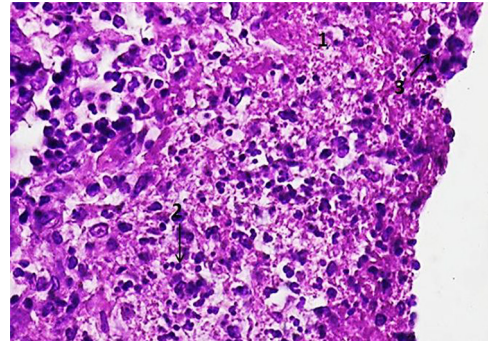


Figure 2. Fibrinous-necrotic overlays on the capsule of the spleen of a cat that had infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 400
Note: 1 – fibrinous-necrotic masses; 2 – lymphocytes; 3 – monocyte

At the same time, it was established that the capsule of the spleen is focally or diffusely infiltrated by lymphocytes and monocytes under fibrinous-necrotic overlays. Focal infiltration in the spleen capsule revealed foci of a fairly dense accumulation of these cells, and diffuse infiltration found single lymphocytes and monocytes located remotely from each other.

In the wet form of infectious peritonitis, the cell infiltrate is represented mainly by neutrophils and macrophages and a small number of T-lymphocytes with plasma cells (Marioni-Henry *et al.*, 2004). However, neutrophil infiltration is inherent in bacterial infections, and therefore

it can be registered in cats with infectious peritonitis complicated by bacterial pathogens. During bacteriological studies, Wolfe & Gricsemer (1966) found out that from patients with feline infectious peritonitis with infiltration of affected tissues and organs by macrophages, neutrophils, lymphocytes and plasma cells, a variety of bacterial microflora is secreted – non-haemolytic staphylococcus, haemolytic streptococcus, enterococci, and *E. coli*.

Other authors have found that with the dry form of infectious peritonitis, the cellular infiltrate consists of macrophages and B-lymphocytes and impurities of plasma

cells (Pedersen, 2009). Microscopic studies found that it is infiltration by monocytes (macrophage precursors) and lymphocytes that occurs in both dry and mixed forms of this disease.

In areas of the organ surface without fibrinous-necrotic overlays, significant microscopic changes were detected in the serous membrane and in the spleen capsule, although these morphological formations stayed unchanged in some areas of the organ. Spleen capsule – swollen (Fig. 3).

Therewith, there is no swelling in the trabeculae in all forms of the disease under study (Fig. 4).

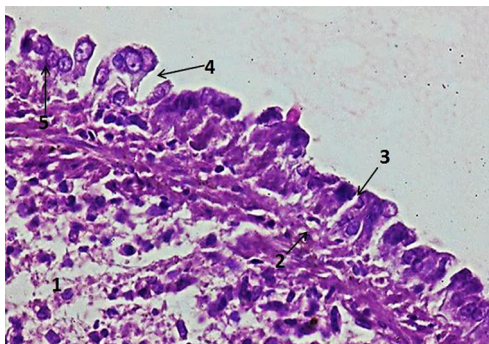


Figure 3. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 400
Note: 1 – swelling of the red pulp; 2 – swelling of the connective tissue capsule; 3 – discomplexation of columnar mesotheliocytes; 4 – separation of mesotheliocytes from the spleen capsule; 5 – destruction of mesotheliocytes

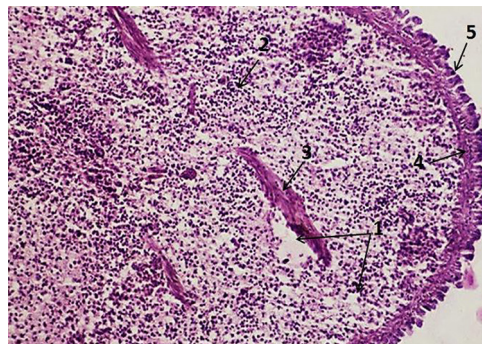


Figure 4. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 50
Note: 1 – swelling of the red pulp; 2 – infiltration of red pulp by lymphocytes and monocytes; 3 – trabeculae; 4 – connective tissue capsule; 5 – columnar mesothelium

Mesotheliocytes of the serous membrane in most areas of the spleen underwent pronounced metaplasia – from flat cells, the long axis of which is oriented parallel to the surface of the capsule of the organ, they turned into cubic and columnar cells (Fig. 3).

The authors of this study believe that this is a reflection of the stages of such metaplasia: initially, due to a gradual increase in the volume of the cytoplasm, cubic-shaped cells are formed, and with a subsequent increase in the volume of the cytoplasm, these cells acquire a columnar shape.

The nuclei of mesotheliocytes that underwent metaplasia noticeably increased in size. They, both in cubic and columnar cells, acquired a rounded or somewhat oval

shape, contained one, rarely two nucleoli. A significant part of such nuclei was occupied by heterochromatin, which, according to modern concepts (Thayer *et al.*, 2022), reflects an increase in the number of active sites of transcription. Such activation of the mesotheliocyte genome indicates an increase in their functional or synthetic activity. Arguably, there is an intensification of the processes of synthesis of cytoplasmic components, as a result of which a noticeable increase in cell volume is observed. Some altered mesothelial cells were destroyed (Figs. 3, 5).

In the spleen of 7 cats, small foci of complete destruction of the organ capsule were recorded on the part not covered with mesothelium (Fig. 6).

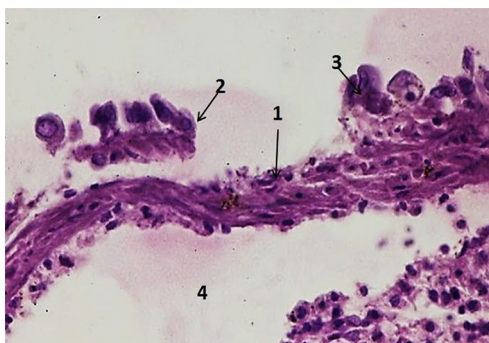


Figure 5. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 400
Note: 1 – capsule not covered by mesothelium; 2 – a layer of mesotheliocytes near the outer surface of the capsule; 3 – destruction of mesothelium cells on the surface of the capsule; 4 – subcapsular swelling

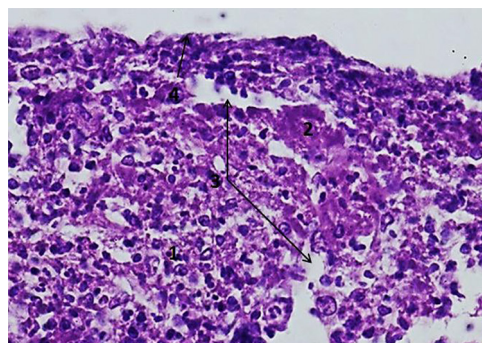


Figure 6. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 400
Note: Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 400

Dyscomplexation of the metaplastic cells of the mesothelium and separation of individual mesotheliocytes or their small groups from the spleen capsule was often found (Fig. 3). Such changes indicate considerable violations of cellular contacts. In some places, entire layers of mesotheliocytes were separated from the capsule (Fig. 5). This can be caused by both destruction of intercellular contacts and damage to cell adhesion molecules.

In the red pulp of the spleen, irregularly located foci of marked oedema were found. No red blood cells were observed in the red pulp. Instead, uneven infiltration by lymphocytes and monocytes and foci of necrotic cells were observed (Figs. 1, 3-6). It was not possible to identify which cells of the red pulp of the spleen were necrotic.

Apart from clear changes in the red pulp, hyperplasia of the lymphoid nodules of the spleen was found. Therewith, lymphoid nodules in the spleen of cats that had infectious peritonitis for less than 3 weeks had varied sizes and were located eccentrically relative to the central arteries. These arteries were localized not in the central

part of the lymphoid nodules, which is characteristic of control cats, but in the peripheral areas of these lymphoid formations.

Notably, the red pulp of the spleen around the lymphoid nodules is infiltrated by a noticeably larger number of lymphocytes and monocytes compared to its more distant areas. Therewith, there were no distinct lymphoid nodules around individual central arteries (Figs. 7, 8).

Necrosis and destruction of the walls were found in the central arteries without distinct lymphoid nodules (Fig. 9).

Perhaps such damage to the walls of these arteries led to the fact that lymphoid nodules did not form near them. On the other hand, such changes could be caused by the action of some factor(s) that caused both the absence of lymphoid nodules and damage to the walls of the central arteries.

In cats in which the disease lasted for more than 3 weeks and less than 3 weeks before death, the microscopic changes in the spleen were similar, but they had some features. The red pulp was noticeably more swollen, and the lymphoid nodules were few and small or were not detected at all (Fig. 10).

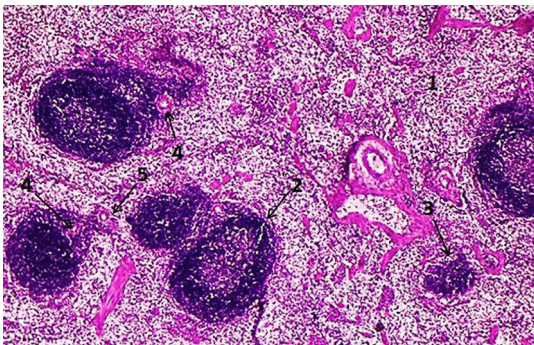


Figure 7. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 50
Note: 1 – red pulp; 2 – a large lymphoid nodule; 3 – a small lymphoid nodule; 4 – the central artery in the peripheral part of the lymphoid nodule; 5 – central artery without a distinct lymphoid nodule

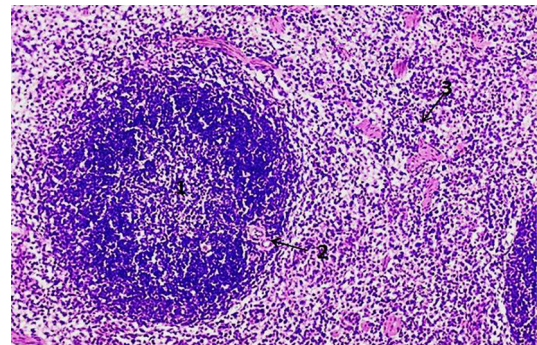


Figure 8. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 100
Note: 1 – a large lymphoid nodule; 2 – the central artery in the peripheral part of the lymphoid nodule; 3 – infiltration of the red pulp by lymphocytes and monocytes

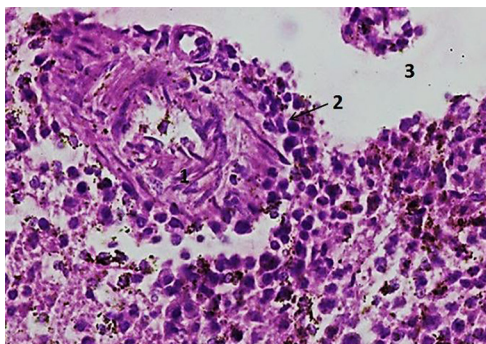


Figure 9. Spleen of a cat with infectious peritonitis for less than 3 weeks. Carazzi's haematoxylin and eosin, x 400
Note: 1 – necrosis and destruction of the wall of the central artery; 2 – a small number of lymphocytes and monocytes near the central artery; 3 – oedema

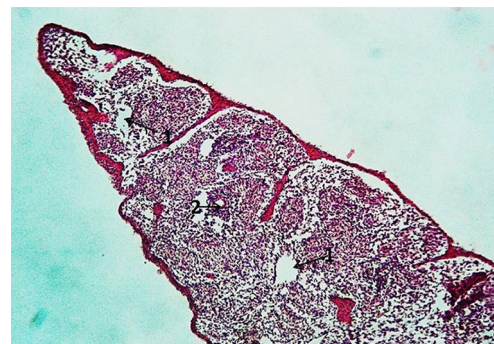


Figure 10. Spleen of a cat with infectious peritonitis for more than 3 weeks. Carazzi's haematoxylin and eosin, x 30
Note: 1 – pronounced swelling of the red pulp; 2 – a small lymphoid nodule with an enlarged central artery

The described microscopic changes suggest that a long course of infectious peritonitis leads to considerable depletion of the immune system in sick cats. Comparable microscopic changes were observed in animals that had infectious peritonitis for less than 3 weeks.

In general, detailed histological studies of the spleen of cats with infectious peritonitis have not previously been conducted abroad (Thayer *et al.*, 2022). In Ukraine, microscopic changes in the spleen of cats in wet and dry forms of infectious peritonitis are described only in one scientific

paper by Halania (2020). The author found that in case of the wet form of the disease, there is a reduction in white pulp. Analogous changes in the white pulp according to the results of pre-microscopic studies were established in cats in which the disease lasted more than 3 weeks. In the same paper, it is indicated that with the dry form of infectious peritonitis, there is intense blood filling of the red pulp and hyperplasia of lymphoid nodules due to T-lymphocytes.

In the red pulp of the spleen of cats whose disease lasted up to 3 weeks before death, the absence of red blood cells, foci of oedema of assorted sizes, uneven infiltration by lymphocytes, monocytes, and small foci of cell necrosis were found, and in the white pulp – hyperplasia of lymphoid nodules.

Since Halania (2020) did not consider the duration of cat disease before death, the author may have studied animals that had a wet form of infectious peritonitis for more than 3 weeks before death, and a dry form for up to 3 weeks.

Various changes in the red pulp in the dry form of the disease could be caused by changes in some properties of the causative agent of the disease, which is characterized by numerous and rapidly emerging mutations (Poland *et al.*, 1996; Vennema *et al.*, 1998).

Thus, the conducted studies indicate that with infectious peritonitis, microscopic changes in the spleen of cats depend on the duration of the course of the disease until death. In the initial stages of the disease, the disappearance of red blood cells from the red pulp, mesotheliocyte metaplasia, and signs of activation of the immune system in the form of hyperplasia of lymphoid nodules

are recorded. With the further development of the disease, changes in the red pulp and mesothelium persist, and the lymphoid nodules atrophy.

Conclusions

Microscopic changes in the spleen of cats with infectious peritonitis did not depend on the form of the disease, but slightly differed depending on the duration of its course.

In cats in which the disease lasted up to 3 weeks before death, the red pulp of the spleen showed the absence of red blood cells, foci of oedema, uneven infiltration by lymphocytes and monocytes, and foci of necrotic cells.

The number of lymphoid nodules of the spleen increased, and necrosis and destruction of their walls were recorded in part of the central arteries.

In the serous membrane of the spleen, the absence of mesothelium under foci of fibrinous-necrotic overlays and metaplasia of mesothelial cells in other areas was established. The nature of mesotheliocyte metaplasia on the surface of the same spleen is different. In some areas of the organ, mesotheliocytes turn from flat cells into cubic cells, and in other areas they are columnar cells. Such metaplasia in the spleen is established in all cats with various forms of the disease.

In cats in which the disease lasted more than 3 weeks, the red pulp is noticeably more swollen, and the lymphoid nodules are few and small, which indicates the exhaustion of the potential of the immune system.

The prospects of the research involve further detailed investigation of pathological and morphological changes upon feline infectious peritonitis in other organs and tissues.

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Мікроскопічні зміни в селезінці за інфекційного перитоніту котів

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Анотація. Актуальність дослідження зумовлена тим, що патоморфологічні зміни при інфекційному перитоніті у котів вивчалися небагатьма авторами і описані недостатньо повно. Мета роботи полягала у вивченні впливу збудника інфекційного перитоніту на будову селезінки в котів. У роботі висвітлено результати гістологічних досліджень зрізів, одержаних із різних ділянок селезінки котів різного віку, що загинули від змішаної (26 тварин) і сухої (7 тварин) форм інфекційного перитоніту. Зрізи фарбували гематоксиліном та еозином за загальноприйнятою методикою. У роботі детально описано мікроскопічні зміни в селезінці за сухої і змішаної форм інфекційного перитоніту котів. З'ясовано, що на ці зміни не впливає форма хвороби, але відзначаються особливостями залежно від тривалості її перебігу. У котів, в яких хвороба до настання смерті тривала до трьох тижнів, червона пульпа селезінки нерівномірно набрякла, інфільтрована лімфоцитами й моноцитами, подекуди містила вогнища некротизованих клітин, а еритроцити відсутні. Зміни білої пульпи представлені гіперплазією лімфоїдних вузликів. Ці вузлики мали різні розміри та були розташовані ексцентрично відносно центральних артерій. Навколо частини центральних артерій виразні лімфоїдні вузлики відсутні. На поверхні капсули місцями наявні фібринозно-некротичні накладання, під якими мезотелій відсутній, а капсула інфільтрована лімфоцитами та моноцитами. На інших ділянках мезотеліоцити зазнали виразної метаплазії – з плоских клітин вони перетворилися на стовпчасті клітини. На окремих ділянках селезінки у деяких тварин серозна оболонка відсутня. В котів, у яких хвороба тривала більше трьох тижнів, червона пульпа помітно більш набрякла, а лімфоїдні вузлики – поодинокі та невеликих розмірів. Інші мікроскопічні зміни були такими ж, як і у тварин, що хворіли менше трьох тижнів. Результати дослідження мають практичну цінність для лікарів-патоморфологів, а також для науковців, які вивчають патогенез інфекційного перитоніту котів

Ключові слова: коронавірус, макроскопічні зміни, гістологічні дослідження, лімфоїдні вузлики, метаплазія