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Evaluation of Granulomatous and Vascular Lesions in Feline Infectious Peritonitis

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ABSTRACT

Feline Infectious Peritonitis (FIP) is a fatal systemic viral disease that affects all cat breeds and has two distinc forms: effusive and non-effusive. In the effusive form, there is a common type of vasculitis that causes extravasation of fibrin-rich fluid, resulting in the accumulation of yellow exudative fluid in the body cavities. In the present study, we aimed to evaluate the organ distribution and severity of the pathological alterations associated with granuloma and vasculitis. It also seeks to describe FIPV antigen localization in FIP lesions. For this purpose, necropsies were performed on eight cats suspected of having died from FIP at the Faculty of Veterinary Medicine, Kırıkkale University. Tissue samples were routinely processed for immunohistochemical analysis. FIPV antigen was detected using immunoperoxidase staining, and the immunopositivity of vasculitic and granulomatous lesions in various organs was evaluated semi-quantitatively for each tissue sample. In effusive FIP cases, peritoneal fluid accumulation was significantly more common, and the lesions were characterized by granulomas progressing through the serosa of the intestine, kidney and liver. Vasculitis lesions, usually affecting small and medium-sized vessels, were characterized by endothelial hypertrophy and swelling, edema and hyalinization of the muscular layer, and adventitial neutrophil leukocyte and macrophage infiltration. Granulomas were characterized by microscopic findings with dense infiltration of macrophages and lymphocytes around a few thrombotic and/or degenerative vessels in the center. In the examined cases, FIPV antigen immunopositivity varied according to the organ involvement in each case, but was frequently concentrated around the periphery rather than in the center of vasculitis and granulomatous lesions.

INTRODUCTION

Feline infectious peritonitis (FIP) is a fatal systemic viral infection caused by Feline Corona Virus (FCoV) that affects especially domestic and group-living cats and can be seen in all cats (Foley et al., 1997; Kipar and Meli, 2014). It is an enveloped, single-stranded RNA virus belonging to the Alphacoronavirus genus within the Coronaviridae family and has two different pathotypes, Feline Infectious Peritonitis Virus (FIPV) and Feline Enteric Corona Virus (FECV) (Gonzáles et al., 2003; Payne, 2017). While the highest disease rate is seen in cats between the ages of 3 months and 2 years, it has been reported that this rate increases especially in male, pure breed and non-neutered cats (Rohrbach et al., 2001; Worthing et al., 2012).

While FCoV replicates in enterocytes, and subsequently a subclinical infection period develops. The most widely accepted theory for the development of FIP infection is that the virus mutates and spreads throughout the body after gaining the ability to infect macrophages and monocytes (Herrewegh et al., 1998; Pedersen, 2014). With the systemic spread and replication of FIPV, the expression of some cytokines increases, which facilitates the interaction of activated monocytes in small and medium-sized vascular endothelial cells (Kipar et al., 2006; Regan et al., 2008; Takano et al., 2009). In activated monocytes, the expression of matrix metalloproteinase-9 enzyme increases, which leads to disruption of endothelial function and allows monocytes to exit the vessel and spread to surrounding tissues (Pedersen, 2009; Takano et

al., 2007). In the effusive form of feline infectious peritonitis, there is a diffuse vasculitis picture, which results in the leakage of fibrin-rich fluid out of the vessels, resulting in the accumulation of yellow, fibrin-containing exudate in the abdominal and thoracic cavity (Andrew, 2000). While fluid does not accumulate in the cavities in the non-effusive form, meningitis and obstructive hydrocephalus, associated ataxia, loss of reflexes in the eyes and nystagmus may occur in the effusive form (Montali and Strandberg, 1972). In the present study, we aimed to evaluate the distribution and severity of granulomatous lesions and vasculitis and to define FIPV immunolocalizations in effusive FIP cases.

MATERIALS AND METHODS

Sampling Methodology

The material of the study consisted of clinically suspected FIP cats that were routinely brought to Kırıkkale University, Faculty of Veterinary Medicine, Department of Pathology for necropsy and histopathologic diagnosis (Table 1). After systemic necropsy of 8 cats, tissue samples were fixed in 10% buffered formaldehyde for 48 hours for pathologic examination.

Table 1. Table of breed, sex, age and disease form of FIP disease cases that routinely visited Kırıkkale University, Faculty of Veterinary Medicine, Department of Pathology

Case number	Species	Age	Sex	Form of the disease
1	Tabby	2,5 years	Famale	Effusive
2	Tabby	10 months	Male	Effusive
3	Tabby	7 months	Male	Effusive
4	Tabby	1 year	Male	Effusive
5	Tabby	10 months	Famale	Non-Effusive
6	Tabby	6 months	Famale	Effusive
7	Tabby	2 years	Male	Effusive
8	Yellow Tabby	8 years	Male	Effusive

Histopathologic Examinations

Liver, kidney, lymph node, spleen, intestine, lung and heart tissues were routinely processed on a tissue processing device after trimming and embedded in paraffin. From the paraffin blocks, 4 µm thick sections were taken on positively charged slides and kept in an oven at 37°C for 24 hours. The sections were deparaffinized for routine staining and then rehydrated and stained with hematoxylin and eosin. At the last step, the tissues were again processed through alcohol and xylene series and covered with entellan. In microscopic evaluation, lesions characterized by granulomas and vasculitis were assessed and scored semiquantitatively.

FIPV Specific Immunoperoxidase Test

4 µm thick tissue sections were taken and kept overnight in an oven at 37°C and then deparaffinized in xylene, graded alcohol series for five minutes each and washed in phosphate buffer solution (PBS) and then placed in citrate buffer (pH 6.0) solution. The slides were subjected to antigen retrieval in a microwave oven at 1200 W for 20 minutes. After removing from the microwave, the cooled slides were kept in PBS followed by 0.3% H2O2 for 15 minutes and then again in PBS solution. To prevent nonspecific staining, 1/100 diluted FIPV antibody (FIPV3-70 SantaCruz) was dropped on the tissue sections, which were protein blocked for 7 minutes, without washing and incubated in an oven at 37°C for 2 hours. At the end of the incubation period, tissue sections were washed with PBS to remove the antibody. After 30 minutes incubation in biotin-labeled secondary antibody, the tissues were washed again with PBS. Then they were incubated in streptavidin peroxidase solution for 30 minutes, washed again and stained with DAB chromogen. Finally, the hematoxylin stained tissues were washed under tap water, dehydrated and covered with entellan. For the evaluation of FIPV immunopositivity in tissues, photographs were taken at 20x objective magnification from 5 different areas with vasculitis and granulomatous lesions and scored semiquantitatively as described below. 0=No staining, 1=1-10% area staining, 2=10-30% area staining, 3=30-50% area staining, 4=50-100% area staining.

RESULTS

Necropsy Findings

Although cachectic appearance and jaundice were prominent in most of the cats on general external examination, it was noted that the skin lost its elasticity and they were dehydrated. Yellow-colored cloudy fluid (Figure 1A) was observed in the abdominal cavity in 6 cases and in the thoracic cavity in one of the effusive form FIP cases examined in the study. In cases with abdominal effusion, granulomas and pyogranulomas of varying sizes were observed on the serosal surfaces of the affected organs (liver, kidney, intestines) (Figure 1B, C). In some cases, fibrin-induced adhesions between organs were present. In general, mesenteric lymph nodes were enlarged. Mesenterium was hyperemic and covered with pyogranulomas. The liver was swollen and pale in appearance. Kidneys were icteric or pale in almost all cases (Figure 1C). In 1 case with exudate accumulation in the thorax, the lungs were pale and edematous with fibrin deposits and adhesions between the lobes and with the pericardium.

Histopathologic Findings

Although the distribution of lesions in the organs differed according to effusive or non effusive FIP (Figure 2), in the affected abdominal cavity organs, the serosa of the intestine, liver and kidney was thickened with dense fibrinous, neutrophil leukocyte and macrophage infiltration. In most of the cases, increased cellular

infiltration of macrophages, lymphocytes and plasma cells was observed in the submucosa layer of the small intestine and around the vessels. In the liver, dense granulomas with necrotic changes were observed in most areas. Hepatocytes were swollen, remark cords were irregular and bile stasis and biliary pigments were observed in the sinusoids. In the kidneys, diffuse or multifocal mononuclear cell infiltrations and necrotic granulomas were observed. In the spleen, inflammatory changes limited to the capsule on initial examination were observed to transform into granulomas with macrophages in the center and lymphocytes around it, extending into the subcapsular region in severe cases. In the lungs, especially in cases with pleural effusion, edema and fibrinous effusion in the subpleural, bronchial and bronchiolar lumens and mononuclear cell infiltration of varying severity around these bronchioles were observed. In the heart, mononuclear cell infiltration in the pericardium, phlebitis characterized by media necrosis and thrombophlebitis were noted. The vessel walls were thickened and there was a dense fibrin deposition in the vessel lumens.

Immunohistochemical Findings

In the examined cases, immunopositivity scores in tissues stained with FIPV antibody varied depending on the organ (Figure 3), but were frequently observed in regions with granulomatous lesions and vasculitis. In the intestines, viral antigen positivity was detected in the submucosa, while the serosa showed stronger immunopositivity, particularly surrounding granulomas. High immunopositivity scores were also noted around granulomas in the mesentery. In the liver, positive staining was observed in areas of mononuclear cell infiltration in the capsular and subcapsular regions, as well as in inflammatory cells localized in perivascular areas. In the kidneys, immunopositivity was especially prominent in the regions extending from the renal capsule to the subcapsular area, particularly in association with granulomatous lesions. In contrast, positivity scores in the lungs and heart were lower than those observed in other organs. In the brain, immunopositive staining was concentrated in the leptomeninges. Notably, even in cases presenting with the wet form of the disease, immunopositivity was evident. In the retina, positive immunoreactions were observed in macrophage-rich inflammatory infiltrates near the optic nerve. In this study, immunopositive findings were evaluated based on the density of staining around vasculitic and granulomatous lesions, with the results summarized in Tables 2 and 3.

Table 2. Immunopositivity of tissues stained with FIPV3-70 antibody around vasculitis

Case no	Vasculitis								
	Mesentery	**Liver**	**Kidney**	**Lung**	**Heart**	**Leptomeninges**	**Eye**	**Spleen**	**Other**
1	3	1	2	1	2	1	2	1	LN2, UB3
2	3	1	2	2	0	0	0	3	LN2
3	2	3	1	2	0	0	0	2	
4	3	2	3	2	0	2	2	2	
5	1	NE	NE	NE	NE	2	0	1	
6	4	4	2	0	4	0	2	NE	
7	0	0	0	2	2	0	0	1	
8	0	0	0	0	0	0	0	NE	

LN: Lymph node, UB: Urinary bladder, NE: Not examined

Table 3. Granulomatous inflammation immunopositivity of tissues stained with FIPV3-70 antibody

Case no	Granulomatous Inflammation								
	Mesentery	**Liver**	**Kidney**	**Lung**	**Heart**	**Leptomeninks**	**Eye**	**Spleen**	**Other**
1	4	1	4	2	2	3	3	2	LN3, UB3
2	3	1	3	0	0	0	0	3	LN3
3	2	4	1	3	0	0	0	2	
4	3	2	4	2	0	2	2	2	
5	1	NE	NE	NE	NE	1	0	1	
6	4	4	3	0	2	0	2	NE	
7	0	0	0	3	2	0	0	1	
8	0	0	0	2	0	0	0	NE	

LN: Lymph node, UB: Urinary bladder, NE: Not examined

DISCUSSION AND CONCLUSION

The mechanisms of pathogenesis, fluid effusion and granuloma formation in feline infectious peritonitis are not fully understood (Pedersen, 2009). The poor prognosis of FIP cases reaching clinics and the fact that there is still no clear treatment, except for antiviral molecules in clinical trials, seems to be directly related to the complex pathogenesis of the disease. For this reason, much of the research on FIP has focused on the relationship between the different mutational pathotypes of the virus and host immunity. In the effusive form of FIP, which results in death, abdominal effusion is known to be more common than thoracic effusion (Pedersen and Boyle, 1980). In a study of 25 cases, it was revealed that the development of the FIP form was closely related to the immune response of the host, and effusive form was observed in 64 of the cases (Pedersen, 2014). In the present study, it was similarly shown that effusive form developed in 7 of 8 cases, and serofibrinous effusions were frequently abdominal. It is also a matter of debate as to which organ lesions the fluid leaking into the body cavities mostly originates from. In the effusive form, the virus adheres to the parietal and visceral layers of the peritoneum and damages the serosa epithelial cells in the initial stages of the infection, which are the physical barrier, and the lesions associated with necrotic phlebitis that develop in the advanced stages constitute the main cause of fluid passage into the body cavities (Kipar et al., 2005). Here, it is thought that fluid passage into the abdominal cavity may be due to increased vascular permeability in the liver capsule and parenchyma. In cases of non-infectious ascites, increased portal hypertension and intravascular pressure in the liver and transudate passage through Glisson's capsule into the abdominal cavity support this view (Angelo and Kurzrock, 2007). In FIP, increased permeability of the vessel wall directly results in leakage of fibrin and blood plasma from the serous capsule into the abdominal cavity. This occurs to a lesser extent in the inflamed serosa of the mesenterium and intestinal serosa, which is normal in the absence of functional blood circulation in these organs and tissues. Therefore, while planning drug protocols for the treatment of FIP, it may be beneficial to combine strategies to reduce vasculitis and FIPV-related vascular leakage, especially in the liver. On the other hand, there is still insufficient information on how vasculitis develops in FIP and how effective it is in granuloma formation and fibrinous fluid effusion.

Clinical disease in cats develops when the virus gains the ability to replicate in monocytes/macrophages (Francisco et al., 2016). The virus, which is transported to the target organs via macrophages leaving the bloodstream, localizes in the reticuloendothelial system and perivascular areas of many organs after the infected macrophages leave the bloodstream and the virus gains the ability to enter the tissues. In all this situation, the virus attracts antibodies, the complement system is activated, and more neutrophils and macrophages come to these areas. As a result, typical granulomatous/ pyogranulomatous changes occur (Nafe, 1984; Pedersen, 1995). In this study, the localization of FIPV in granulomatous lesions was mostly localized around granulomatous foci, with immunopositivity in monocyte/macrophage cytoplasm in the vessel lumen. Interestingly, in severe lesions with necrotic vasculitis and hyalinization of the vessel wall, low-intensity levels of FIPV were observed. This seems to support the role of mediators such as Th1 cytokines, cell adhesion molecules and VEGF, which may have a direct viral invasion effect at the onset of vascular lesions but are more activated in advanced vessel wall damage and fluid leakage. Further studies are needed on the aforementioned vascular damage and the pathogenesis of fluid exudation in FIP and the effect of mediators.

Typical FIP vasculitis is a phlebitis mediated mostly by activated virus-infected monocytes, a few neutrophils and T-cells (Kipar et al., 2005). Based on these features, FIP vasculitis can be distinguished from immune-mediated vasculitis (Kipar et al., 2005). However, in acute cases, necrosis of the vessels could be observed and it was even observed that it occurred in vessels where inflammatory reactions were previously detected, confirming that the disease may have a multiphasic nature (Boudreaux et al., 1989; Hayashi et al., 1977; Weiss et al., 1980). Thus, due to the morphologic features of acute vascular lesions, evidence has been provided that type III hypersensitivity reaction contributes to the pathogenesis of FIP in some cases (Boudreaux et al., 1989; Montali and Stranberg, 1972; Weiss et al., 1980). Perivascular granulomatous lesions due to FIP infection have been associated with immunopathogenic mechanisms such as type III and type IV hypersensitivity reactions (Jacobse-Geels et al., 1982; Paltrinieri et al., 1998; Pedersen and Boyle, 1980). Type III hypersensitivity is seen when antigens bind to antibodies and the resulting immune complexes are deposited on the vessel walls (Wills-Karp, 2008). Type IV hypersensitivity is a late hypersensitivity reaction caused by excessive stimulation of T-cells and macrophages, which also contributes to granuloma formation (Benacerraf and Levine, 1962). Vasculitis resulting from monocyte activation was supported by the identification of increased vascular permeability and induction of effusions following the release of vascular endothelial growth factor (VEGF) by FIPV-infected monocytes.

Whether the cat develops the disease in effusive or non-effusive form depends on an insufficiently activated cellular immunity or a strong humoral response. Although often described as distinct syndromes, effusive and non-effusive FIP are caused by an excessive inflammatory response following disease-specific vasculitis and pyogranulomatous lesions. The effusive form is more common than the non-effusive form, and cases of both are common (Pedersen and Boyle, 1980). In general, the balance between cellular and humoral immune response is a critical determinant in infected animals. In part, a more vigorous T cell immune response is thought to cause dry form FIP (Pedersen, 2009; 2014).

In this study, necropsies of naturally infected cats with FIPV were performed and the tissues were stained with histopathological and immunohistochemical immunostaining after the necessary procedures and the lesions caused by the virus in the organs were identified. When the results of the study were evaluated, vasculitis was characterized by endothelial hypertrophy and endothelial swelling in small vessels, while in medium-sized vessels it was characterized by media necrosis and adventitial inflammatory cell infiltration and fibrin deposition in the lumen.

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Ethical Declaration

Since the study material was routine specimens brought to Kırıkkale University, Faculty of Veterinary Medicine, Department of Pathology, no ethics committee was required.

Conflict of Interest

The authors declare that they have no competing interests.

Authorship contributions

Concept, Design, Data Collection or Processing, Analysis or Interpretation, Literature Search, and Writing took placed by O.K. and T.S.Y.

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