

A Case of Feline Infectious Peritonitis in Erzurum Province: Macroscopic and Microscopic Findings

A Case of Feline Infectious Peritonitis

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Abstract: In this case report, a male Tekir cat was brought to Atatürk University Veterinary Faculty Animal Hospital with complaints of loss of appetite and weakness. Feline infectious peritonitis (FIP) was detected with the rapid diagnostic test kit and the cat died despite treatment. At necropsy, multifocal foci were found in the liver, kidney, lung, heart and brain tissues. Microscopic examination revealed granulomatous foci in the lung, liver and kidney, as well as interstitial nephritis in the kidney. Inflammatory cell infiltrates were detected in the epicardium layer of the heart, necrosis in Peyer's patches in the ileum layer of the intestine, and necrosis in the center of the follicles in the spleen. There was meningoencephalitis in the brain. In this case report, macroscopic and microscopic findings seen in tissues and organs of FIP disease provided support to the literature on the diagnosis of FIP disease.

Keywords: Cat, Coronavirus, Histopathology, Macroscopic, Peritonitis

INTRODUCTION

Feline infectious peritonitis (FIP) was first described as a specific disease for cats in 1963 by Dr Jean Holzworth and colleagues (1). FIP is typically observed in cat shelters and cat houses, as cats who live in crowded situations have a higher prevalence of this virus (2).

Feline coronavirus (FCoV) is a virus from the Coronaviridae family that is found in many cat populations worldwide. Coronaviruses are relatively large, enveloped, positive-sense, single-stranded RNA viruses. They show a high rate of mutation during replication and therefore exist in clusters consisting of genetically

distinct populations. It has been determined that a large part of cats in the world are infected with FCoV. FCoV is generally an enteric virus, and infection often does not result in clinical signs or only causes weight loss due to enteritis (3-7). However, a small number of cats infected with FCoV may develop a high-fatality disease with vasculitis called FIP (8). The development of lesions caused by FIP is triggered by activated, virus-infected monocytes. Together with the general activation of venous endothelial cells, monocytes induce granulomatous phlebitis, which can occur in various organs, and since this is specific to iFIP disease, it is

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considered a lesion different from other diseases (5).

Depending on the response of the diseased cat's immune system, FIP can be divided into two forms: effusive and non-effusive (9). The effusive form is characterized by peritonitis, pleuritis and diffuse vasculitis. For this reason, fluid rich in fibrin and protein leaks out of the vessel. Exudative fibrinous serositis in the abdominal and chest cavity is a common finding in the effusive form (10). The non-effusive form, also called dry FIP, has a chronic progressive course. It may often take weeks or even months after the initial infection for the symptoms of the disease to develop. The characteristic lesions of dry FIP are granulomas observed in the liver, kidneys, mesenteric lymph nodes, especially the colon and ileocecal valve. Although approximately 60% of cats with non-effusive FIP develop ocular and central nervous system damage, approximately 40% have abdominal lesions that may or may not be associated with ocular, central nervous system lesions. In this case, FIP was evaluated pathologically (10).

CASE PRESENTATION

This case constituted by a 21 month-old male Tekir cat brought to Atatürk University Faculty of Veterinary Medicine with complaints of loss of appetite and weakness. During the physical examination of the Tekir cat, it was determined that its body temperature was 38.6 °C, its mucosa was anemic and it had generalized lymphadenopathy. In the hematological

examination, lymphopenia, in the biochemical examination, an increase in total protein, hypoalbuminemia and albumin-globulin ratio were detected as 0.18, while creatinine, ALT, AST and ALP values were found to be among the reference values. Considering the albumin-globulin ratio and clinical findings, a diagnosis of FIP was made. Death occurred within a few hours after clinical diagnosis was made with a rapid diagnostic test kit. In accordance with the owner's request, systemic necropsy of the deceased cat was performed and the tissue samples taken were fixed in 10% formaldehyde solution and then embedded in paraffin by tissue processing. Sections of 4 µ thick normal slides were taken from paraffin blocks with a microtome and were stained with hematoxylin-eosin and evaluated under a light microscope.

Clinical examination of the cat revealed loss of appetite, tachycardia, and increased respiratory rate. It was observed that the number of lymphocytes decreased in the blood table of the infected cat. During systemic necropsy, when the abdominal cavity was opened, a small amount of postmortem fluid accumulation was observed in the abdomen. Multifocal granulomatous foci the size of a pinhead were observed macroscopically in the liver (Fig. 1a). Macroscopically, multifocal yellow nodular structures were noted in the kidney (Fig. 1b). Multifocal gray lesions were also found on the lungs (Fig. 1c) and heart (Fig. 1d). There were multifocal gray colored foci in the gyri of the brain (Fig. 1e) and spleen (Fig. 1f).

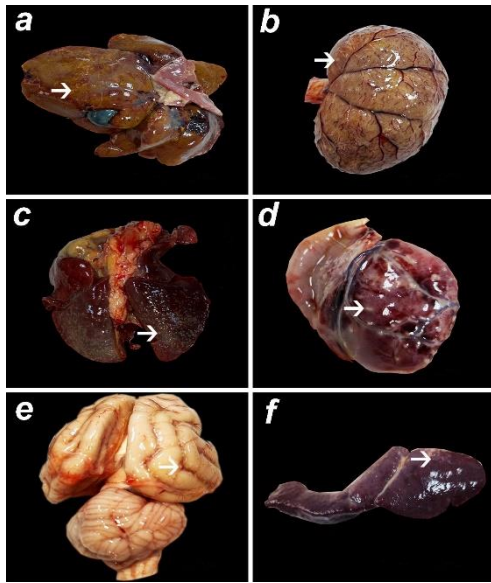


Figure 1. Multifocal lesions (arrow), in the liver (a), kidney (b), lungs (c), heart (d), brain (e) and spleen (f).

Histopathological examination revealed severe parenchymal damage in the liver along with granulomatous foci and congestive vessels (Fig. 2a).

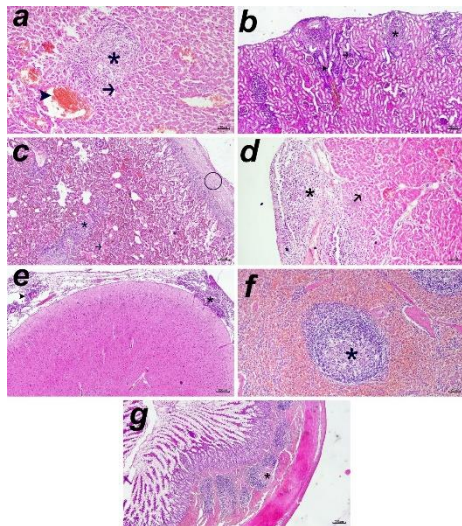


Figure 2. Granulomatous foci (asterisk), necrosis in hepatocytes (arrow) and hyperemia in the vessels (arrowhead) in liver tissue (a). Granulomatous foci (asterisk), interstitial nephritis (arrow) and dilatation of tubules (arrowhead), kidney (b).

Granulomatous foci (asterisk), thickening of the pleura (circle) and edema (arrow), lung (c). Pericarditis (asterisk) and necrosis of myositis (arrow), heart (d). Perivascular mononuclear cell infiltration (arrowhead) and meningitis (asterisk), brain (e). Necrosis of follicles (asterisk), spleen (f). Necrosis (asterisk) in Payer's plaques, intestine (f). H&E, Bar: 70 and 200 μ m.

Histopathologically, interstitial nephritis was observed in the kidney, including intense lymphohistiocytic cell infiltration in the cortex and mononuclear cell infiltration between the tubules. Interstitial nephritis was observed between the tubules including mononuclear cell infiltration and intense lymphohistiocytic cell infiltration in the cortex (Fig. 2b). Edema and thickening due to fibrin and inflammatory cell infiltration were observed in the lung pleura. When the histopathological examination of the grey-white foci detected macroscopically on the tissue surface was performed, it was noted that these structures were granulomatous structures (Fig. 2c). There was endocarditis and necrosis in heart (Fig. 2d). Meningitis was observed in the brain, including intense histiocytic, lymphocytic and plasmocytic cell infiltrates in the meninges (Fig. 2e). There was necrosis in the center of the lymph follicles in the spleen (Fig. 2f) and in the Peyer's patches in the ileum (Fig. 2g).

DISCUSSION and CONCLUSION

Postmortem diagnosis of FIP by anatomopathology is considered the gold

standard for etiological diagnosis. In fact, the key factors are the macroscopic and microscopic lesions typically observed associated with positivity (11). In this case report, the post-necropsy macroscopic and microscopic findings of a Tekir cat that died of FIP disease in Erzurum and its contribution to the diagnosis of the disease are presented. Dry FIP, as the name suggests, is a form in which thoracic and abdominal effusions are absent or too minimal to be detected outside of necropsy. Eye and/or central nervous system involvement is predominant in 60% of cats with dry FIP (10). In the presented case, the presence of a small amount of fluid in the abdominal cavity was attributed to post-mortem fluid accumulation, and the central nervous system involvement of the disease was also supported by the meningitis observed especially in the brain. Many tissues and organs, especially the liver and kidney, are affected by FIP disease (12,13). In the dry form of the disease, it is characterized by granulomatous involvement of parenchymatous organs such as kidneys, mesenteric lymph nodes, intestinal wall, and liver. In the presented case, granulomatous structures were observed in the kidney and liver (14). Diffuse or multifocal interstitial nephritis is also observed in the disease (15). In the presented case, multifocal areas of interstitial nephritis were observed. Cats with FIP often show both T and B cell reductions in lymphatic tissues. Although mesenteric lymph nodes and pericardial mediastinal tissues, including the thymus

and cranial mediastinal lymph nodes, often exhibit granulomatous necrotizing lesions, the lymphoid cell reduction is not greater than that in the spleen (16). In the presented case, when we look at the necrosis that started centrally in the spleen follicles, it was observed that this situation was reflected in the hemogram table as a decrease in the number of lymphocytes. Although small granulomas are observed in the pleura and underlying lung parenchyma in the disease, (10) it has been reported that edema and lymphoplasmocytic cell infiltration are observed in the lung (17). In the presented case, it was observed that the pleura thickened along with edema in the lung, and small focal granulomatous areas were observed, which was consistent with the literature. In FIP, although rare, cardiac lesions in the epicardium characterized macroscopically and microscopically by fibrinous epicarditis have been described (18). In the presented case, multifocal foci were observed on the epicardium and microscopic examination revealed areas of lymphoplasmocytic cell infiltration.

As a result, this case presented was defined as a FIP case that was examined together with macroscopic and microscopic findings under the leadership of previous studies and could contribute to the literature.

CONFLICT of INTEREST

There is no conflict of interest between the authors.

REFERENCES

1. Holzworth J. Some important disorders of cats. *The Cornell Veterinarian*. 1963;53: 157-160.

2. Klein-Richers U, Hartmann K, Hofmann-Lehmann R, Unterer S, Bergmann M, Rieger A, Felten S. Prevalence of feline coronavirus shedding in German catteries and associated risk factors. *Viruses*. 2020;12(9):1000.
3. Pedersen NC, Boyle JF, Floyd K, Fudge A, Barker J. An enteric coronavirus infection of cats and its relationship to feline infectious peritonitis. *Am J Vet Res*. 1981;42: 368–377.
4. Hayashi T, Watabe Y, Nakayama H, Fujiwara K. Enteritis due to feline infectious peritonitis virus. *Jpn J Vet Res*. 1982;44: 97–106.
5. Addie DD, Jarrett O. A study of naturally occurring feline coronavirus infections in kittens. *Vet Rec*. 1992;130:133–137.
6. Kipar A, Kremendahl J, Addie DD, Leukert W, Grant CK, Reinacher M. Fatal enteritis associated with coronavirus infection in cats. *J Comp Pathol*. 1998; 119:1–14.
7. Addie DD, Jarrett O. Use of a reverse-transcriptase polymerase chain reaction for monitoring the shedding of feline coronavirus by healthy cats. *Vet Rec*. 2001;148: 649–653.
8. Kipar A, May H, Menger S, Weber M, Leukert W, Reinacher M. Morphologic features and development of granulomatous vasculitis in feline infectious peritonitis. *Vet Pathol*, 2005; 42: 321–33.
9. Addie D D, Covell-Ritchie J, Jarrett O, & Fosbery M. Rapid resolution of non-effusive feline infectious peritonitis uveitis with an oral adenosine nucleoside analogue and feline interferon omega. *Viruses*, 2020; 12 (11): 1216.
10. Pedersen N C. A review of feline infectious peritonitis virus infection: 1963–2008. *J Feline Med Surg*, 2009; 11(4): 225-258.
11. Pedersen NC, An update on feline infectious peritonitis: diagnostics and therapeutics. *Vet J*, 2014; 201(2): 133-141.
12. Barlough JE, Stoddart CA. Cats and coronaviruses *J Am Vet Med Assoc* 1988; 193: 796–800.
13. Pedersen NC. An overview of feline enteric coronavirus and infectious peritonitis virus infections *Feline Pract*. 1995; 23: 7–20.
14. Montali RJ, Strandberg JD. Extraperitoneal lesions in feline infectious peritonitis, *Vet Pathol*. 1972;9:109–121.,
15. Pedersen N.C. Feline infectious peritonitis. Something old, something new, *Feline Pract*. 1976;6: 42–51.
16. Stranieri A, Scavone D, Paltrinieri S, Giordano A, Bonsembiante F, Ferro S, Lauzi S. Concordance between histology, immunohistochemistry, and RT-PCR in the diagnosis of feline infectious peritonitis. *Pathogens*. 2014;9(10): 852.
17. Kipar A, Köhler K, Leukert W, Reinacher M. A comparison of lymphatic tissues from cats with spontaneous feline infectious peritonitis (FIP), cats with FIP virus infection but no FIP, and cats with no infection. *Journal of Comparative Pathology*. 2001; 125(2-3): 182-191.
18. Hanedan, B, Timurkan M, O, Aydin H, Altun S, Comakli S, Yanar KE. pleural effusion associated with feline infectious peritonitis in a kitten: molecular and histopathological investigation. *Journal of Applied Biological Sciences*. 2022; 16(1): 62-69.
19. Oliveira LB, Susta L, Rech RR, Howerth EW. Pathology in practice. effusive FIP with fibrinous epicarditis in a cat. *J. Am. Vet. Med. Assoc*. 2014;245:899-901.