Explor Anim Med Res.
Vol. 15, Issue 1, 2025
DOI: 10.52635/eamr/15.1.133-139

ISSN 2277-470X (Print), ISSN 2319-247X (Online)
Website: www.animalmedicalresearch.org

Short Communication

ASSESSMENT OF MACROSCOPIC AND MICROSCOPIC LESIONS IN FELINE PANLEUKOPENIA VIRUS INFECTED ORGANS OF CATS

Rebecca Lamuanpuii[1], Kiran J.[2], Kalyan Sarma[1*], Parimal Roychoudhury[3], Chethan G. E.[1], Malsawmsangi[1], Neeraj Thakur[1]

Received 24 July 2024, revised 15 February 2025

ABSTRACT: Feline panleukopenia is a highly contagious and often fatal disease affecting both domestic and wild felines. The current study aimed to investigate the macroscopic and microscopic lesions in various organs of cats with feline panleukopenia. History and clinical examination of the infected cats revealed inappetence/ anorexia, vomiting, diarrhoea, dehydration, respiratory stress and oral lesions. Feline panleukopenia was diagnosed by using rapid antigen detection kit and further confirmation was done by polymerase chain reaction (PCR). During the present study, six kittens did not respond to treatment and succumbed to the disease. A systematic post-mortem examination was performed on the dead animals and macroscopic lesions were recorded. The tissue samples from various vital organs such as the heart, lungs, liver, spleen kidney, intestine and mesenteric lymph node were collected in 10% neutral buffered formalin and processed for histopathological examination. The macroscopic examination revealed enlarged flabby left ventricular wall of heart, pneumonic lungs with infarction, hepatomegaly with distended gallbladder, splenomegaly with congestion, pale enlarged kidneys, enlarged and edematous mesenteric lymph nodes, and thickened and pale intestinal loops with hemorrhage on the mucosal layer. The major microscopic lesions were separation of muscle fibers with loss of cross striations and severe hemorrhages in the heart, severe congestion in the alveolar capillaries with emphysematous areas, degenerating hepatocytes with a mild degree of fatty changes, lymphoid depletion of the spleen, hemorrhages in the interstitial spaces with microthrombus formation in the interstitium and the glomerular tuft of the kidneys, severely affected mucosal layer with sloughed villi with enlarged crypts containing degenerating crypt epithelium, and depletion of lymphoid follicles of mesenteric lymph nodes. Very few reports are available on the pathological aspects of feline panleukopenia and thus the outcome of the present study can yield valuable information to aid in the diagnosis of feline panleukopenia in cats under field conditions.

Keywords: Feline panleukopenia, Cats, PCR, Lesions, Intestine.

Feline panleukopenia, caused by the feline panleukopenia virus (FPV), is one of the most serious diseases in domestic cats, characterized by anorexia, vomiting, diarrhoea, leukopenia, and high mortality in kittens [1]. FPV is closely related to canine parvovirus 2 (CPV-2), currently classified into species, *Carnivore protoparvovirus 1*, in the genus *Protoparvovirus* of the family *Parvoviridae* [2]. Cats younger than one year of age are more prone to feline panleukopenia, but unvaccinated or improperly vaccinated cats of all ages are also infected. The outcome of FPV infection in cats ranges from subclinical to fatal infection with high mortality depending on the cat's age, immune status, and concurrent infections [3]. The virus has an affinity to damage the cells present in the bone marrow, lymphoid organs and intestinal crypt cells, leading to severe symptoms and potentially fatal outcomes. The pathogenesis of FPV and CPV-2 is almost similar. Like CPV-2, FPV enters cells using transferrin receptors [4]. Transmission of infection is by the feco-oral route, and

*[1]Department of Veterinary Medicine, [2]Department of Veterinary Pathology, [3]Department of Veterinary Microbiology, College of Veterinary Sciences and Animal Husbandry, Central Agricultural University (Imphal), Selesih, Aizawl-796015, Mizoram, India. *Corresponding author. e-mail: kalyan_srm@rediffmail.com*

indirectly through contaminated fomites. The major clinical signs of infection are lethargy, inappetence/ anorexia, vocalization, weakness, vomiting, watery to hemorrhagic diarrhea, dehydration, fever or sub-normal temperature, and rapid loss of weight. Some cats develop only anorexia and lethargy, in the absence of vomiting, diarrhoea, or leukopenia [5]. Death usually results from complications relating to dehydration, electrolyte imbalances, hypoglycemia, hemorrhage, viremia, and endotoxemia. Relatively little is known about the infection of cats with FPV infection in some parts of India including Mizoram. Cats are the second most popular companion animal in the Mizoram state of India, and most of the owners never vaccinate their cats for the prevention of feline panleukopenia. The present research was undertaken to study the macroscopic and microscopic changes noticed in naturally occurring FPV infection in cats.

The study

Informed consent was obtained from all cat owners before enrolment in the study. The study protocol was approved by the Institutional Animals Ethics Committee (IAEC) vide letter No. CVSC/CAU/ IAEC/21-22/P-32 dated 16/11/2022. The study was carried out in three different districts of Mizoram viz. Aizawl, Kolasib and Champhai (Fig. 1). Client-owned cats presented to outpatient department (OPD) with a history of lethargy, inappetence/anorexia, vocalization, weakness, vomiting, watery to hemorrhagic diarrhea, dehydration, fever or sub-normal temperature, oral lesions, icterus and rapid loss of weight during the period from November 2022 to December 2023. The samples were collected from Veterinary Clinical Complex (VCC), College of Veterinary Sciences and Animal Husbandry (CVSc&AH), Central Agricultural University (CAU), Selesih, Aizawl, and Government and Private Veterinary Hospitals of Aizawl, Kolasib and Champhai districts. Twenty cats suspected to be affected with feline panleukopenia in each selective area were included in this study. Fecal samples were collected from suspected cases and suspended in 0.5 mL of phosphate buffer saline (1 × PBS; pH 7.3 ± 0.1). The collected fecal samples were stored in screw cap vials at a temperature of -20°C until processing [6]. 2 mL of blood sample was collected from each suspected animal by venipuncture of medial saphenous vein into vials with dipotassium ethylenediaminetetraacetic acid (K2-EDTA) and stored at -20°C until processing. Fecal samples were tested for the presence of FPV antigen by using a commercially available rapid FPV antigen detection kit following the manufacturer's instructions [Feline Panleukopenia Antigen Test Kit (FPV Ag), Cat. No. SDA101B, Secure Diagnostics, Bhopal, India].

A systematic necropsy examination was conducted on six cases which were previously confirmed to be positive for FPV infection and eventually succumbed. Representative tissue samples from different organs such as the heart, lungs, liver, spleen, kidney and intestine were collected in 10% neutral buffered formalin for histopathological analysis and the fresh samples were stored at -80°C. Fixed tissues were processed by routine paraffin embedding technique for histopathological examination [7] and observed under microscope to record the lesions. Total genomic DNA was extracted from all the collected fecal, blood and tissue samples using DNA extraction kit (QIAamp DNA Mini Kit, Cat. No.: 51304, Qiagen, Hilden, Germany) as per manufacturer's instructions. For the detection of FPV, a conventional polymerase chain reaction (PCR) assay was performed by using a previously published primer pair FM-F (5'GCTTTA GATGATACTCATGT3') and FM-R (5'GTAGCT TCAGTAATATAGTC3' targeting 698 bp fragment of VP2 [8]. Amplification was performed with 30 cycles of denaturation at 94°C for 30 sec, primer annealing at 55°C for 2 min, and extension at 72°C for 2 min. Subsequently, the PCR-amplified products were separated on a 1.5% agarose gel in Tris-acetate EDTA buffer and visualized under ultraviolet (UV) light in a gel documentation system (Molecular Imager® Gel Doc™ XR+System, Bio-Rad, USA).

Results and discussion

Out of sixty suspected cases, thirty seven cases were found positive for FPV infection by using a rapid antigen test (Fig. 2). This diagnosis was further confirmed by PCR assay results of fecal, blood and tissue samples which revealed the expected 698 bp product on an agarose gel under UV transillumination (Fig. 3). PCR assay showed positive for different tissues such as heart, lungs, liver, spleen, kidney and intestine indicating viraemia. Feline panleukopenia has been reported in cats from clinical cases in Tamilnadu and a total prevalence of 68.9% was reported [9]. PCR assay has been reported to be a sensitive, specific and rapid technique for detection of FPV as compared to commercially available immunochromatographic strips, haemagglutination (HA), and enzyme linked immunosorbent assay (ELISA) [9, 10]. DNA amplification of FPV from myocardial tissue specimens has been done in cats with cardiomyopathy, and positive results were observed in 32.2% cases [11, 12]. All the

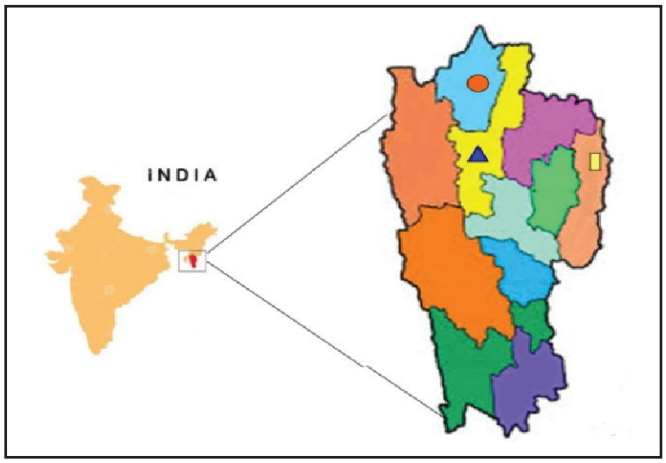

Fig. 1. Study area of Mizoram. [Blue colour triangle indicates the Aizawl district; red circle indicates the Kolasib district and yellow colour rectangle indicates the Champhai district].

Fig. 2. Confirmation of FPV infection in cats by using rapid antigen detection kit.

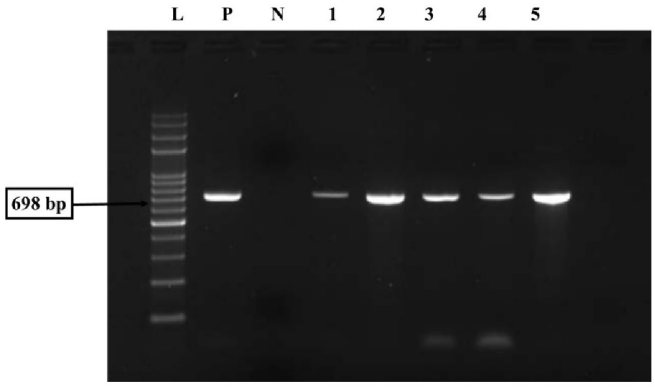

Fig. 3. 1.5% agarose gel electrophoresis of PCR amplified products for the detection of FPV.
Lane L: 100 bp ladder, P: Positive control (698 bp); N: Negative control; 1-5: Positive samples.

cats with gastroenteritis irrespective of FPV infection were treated with symptomatic and supportive therapy consisting of fluid and electrolyte therapy, broad spectrum gut acting antibiotic, antiemetic, antacid as well as hemostatic agent whenever indicated [13, 14]. Unfortunately, six cats with FPV infection did not respond and succumbed to infection after two days of initiation of therapy. The majority of infected cats die from dehydration when the dehydration percentage is over 8%, severe gastroenteritis with vomiting and diarrhoea in addition to panleukopenia with a sharp drop in the level of circulating leukocyte count [15]. The remaining animals showed uneventful recovery.

Grossly, the carcasses of the kittens died due to FPV infection appeared severely dehydrated and emaciated with a pale mucus membrane and rough body coat. The heart showed pale musculature and an enlarged flabby left ventricular wall (Fig. 4A), the lungs were moderately congested with pneumonia (Fig. 4B), and liver was enlarged with blunt edges and distended gallbladder due to accumulation of dark-coloured bile (Fig. 4C). Similarly, the spleen also showed splenomegaly with congestion and discolouration (Fig. 4D). Both the kidneys were pale and enlarged (Fig. 4E). The significant gross lesions were observed in intestine and lymphoid organs. Mesenteric lymph nodes were enlarged and edematous (Fig. 4F). The intestinal lesions revealed thickened and pale intestinal loops with hemorrhage on the mucosal layer (Fig. 4F). The gross lesions observed in the present study corroborate the observation described earlier [16, 17]. Jones *et al*. [18] reported that the lymph nodes appear edematous and hyperemic on macroscopic examination in the initial stages of the disease. FPV has also been associated with various inflammatory and degenerative liver diseases including focal areas of ecchymotic hemorrhages, and coagulative necrosis of hepatocytes [19].

The microscopic lesion of the heart showed separation of muscle fibers, loss of cross striations and severe hemorrhages (Fig. 5A). The pathological lesions in the heart as mentioned above are also described by Meurs *et al.* [12], and reported that the virus can cause cardiomyopathy in kittens due to the presence of rapidly dividing cells. Since FPV preferentially affects cells undergoing rapid mitosis and is comparable in this respect to CPV-2 [13], myocardial infections in very young animals might be expected to occur similarly. Similar to CPV-2 infection, cats surviving FPV induced myocarditis might later develop cardiomyopathy. Chronic myocardial inflammation and the persistence of viral myocarditis may have played a role in the development of myocardial injury or cardiomyopathy in the present study. Histopathology of the lung showed severe congestion of alveolar capillaries with the proliferation of type-II pneumocytes and hemorrhages in the interstitium (Fig. 5B). Emphysematous areas were observed adjacent to the areas of consolidation (Fig. 5C). Bronchiolar lining showed infiltration of inflammatory cells (Fig. 5B). Many authors recorded

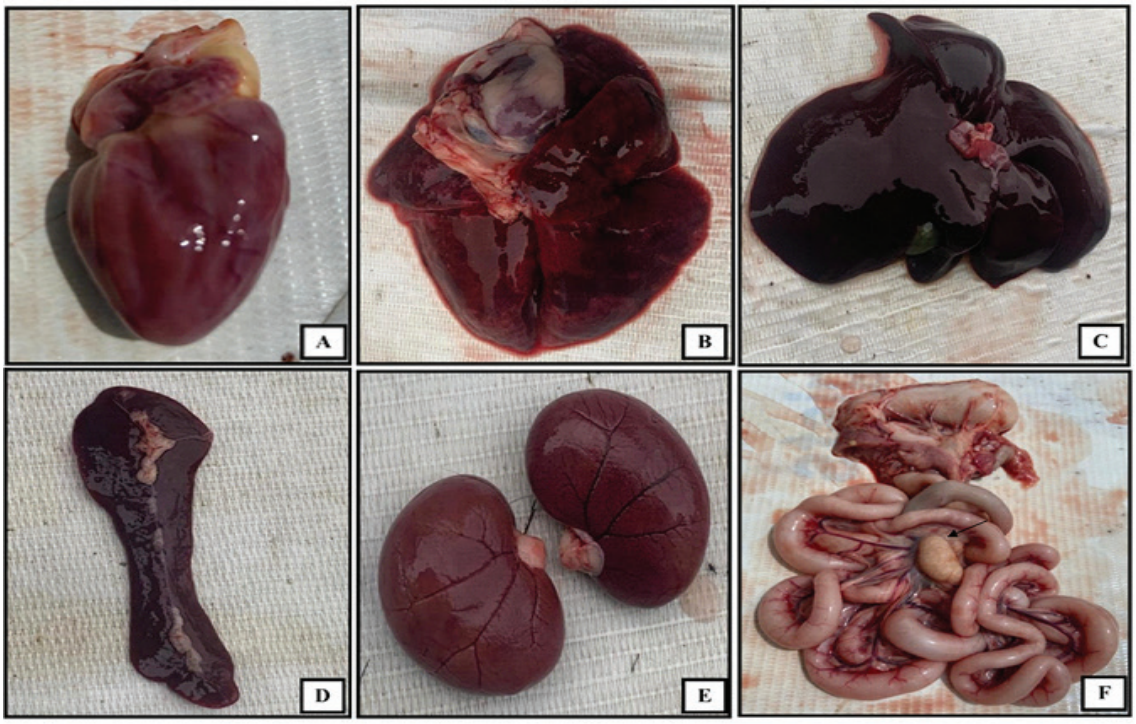

Fig. 4. Gross pathological changes of different organs due to FPV infection in cats. [A: Heart with pale musculature and enlarged left ventricular wall. B: Pneumonic lungs with infarction. C: Hepatomegaly with distended gall bladder. D: Splenomegaly with congestion. E: Pale enlarged kidneys. F: Intestine with pale serosal layer and enlarged mesenteric lymph nodes (arrow)].

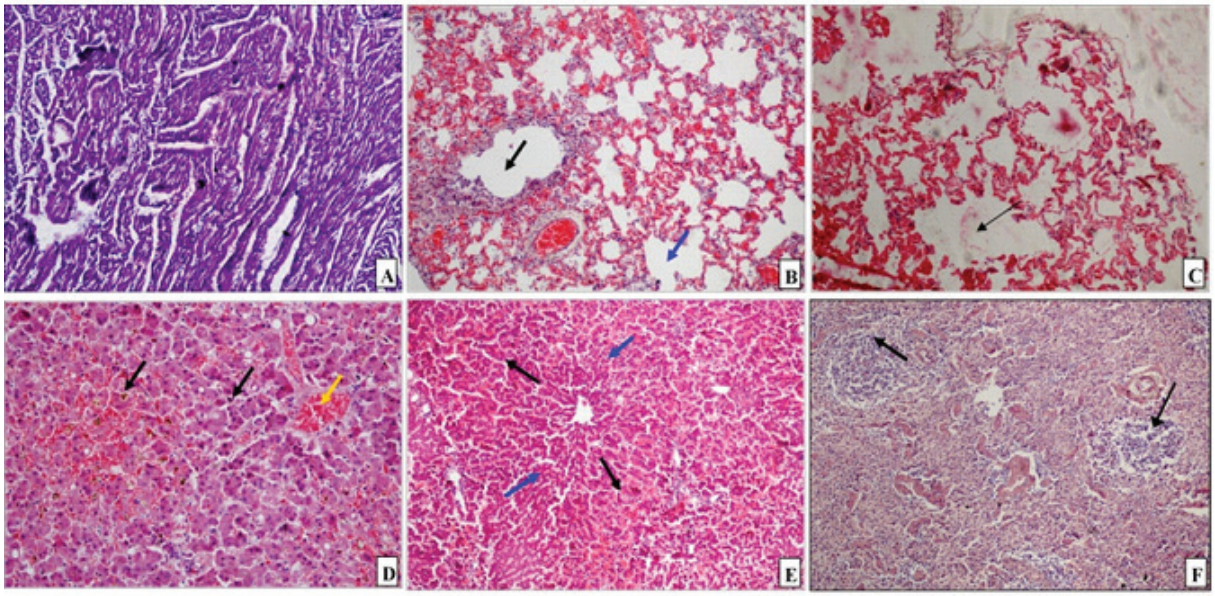

Fig. 5. Microscopic lesions of different organs (heart, lung, liver and spleen) of cats infected with FPV. [A: Separation of muscle fibres, loss of cross striations and severe hemorrhages in the heart, 40X, H&E. B: Lung showing severe congestion of alveolar capillaries with a proliferation of type-II pneumocytes and haemorrhages in the interstitium. Emphysematous areas (Blue arrows) were observed adjacent to the areas of consolidation. Bronchiolar lining showed infiltration of inflammatory cells (Black arrows), especially with mononuclear cells, 20X, H&E. C: Lung showing severe congestion in the alveolar septa with emphysematous areas (black arrows), 20X, H&E. D: Liver showing congestion in sinusoidal spaces with hemosiderin-laden cells (black arrows). The central vein was congested with microthrombus formation (yellow arrow). Degenerating hepatocytes were showing mild degree of fatty change, 40X, H&E. E: Severe sinusoidal congestion in the periportal region was observed (black arrows) in the liver. Degenerating and individualized hepatocytes were seen especially in the centrilobular region (blue arrows). 10X, H&E. F: Severe lymphoid depletion around the central artery of the follicles (arrow) in the spleen, 20X, H&E].

severe pulmonary signs associated with FPV or CPV-2 such as lung haemorrhages or edema or alveolitis [20, 21]. FPV has an affinity for dividing cells and the ability to infect and replicate in endothelial cells [22]. Emphysema is always secondary to obstruction of outflow of air which frequently occurs in carnivores with bronchopneumonia, and is characterized by distention and rupture of alveolar walls. Many viruses infect the respiratory system of cats and may cause pneumonia. Bayati and Akaby [23] reported that the FPV causes pleuropneumonia and proliferative pneumonia. Histologically, liver showed severe sinusoidal congestion in the periportal region with hemosiderin-laden cells (Fig. 5D) and central vein was congested with microthrombus formation (Fig. 5D). Degenerating hepatocytes, especially in the centrilobular region were showing a mild degree of fatty change (Fig. 5E). Singh *et al*. [19] also observed extensive fatty degeneration around the central vein with an increase in the number of Kupffer's cells in parvovirus infection in cats. The generalized systemic infection might be the causative factor for hepatic fatty changes, necrosis, hemorrhages and inflammatory response. In this study, hepatic infiltration of inflammatory cells was intense in portal areas, which is likely linked to the presentation of jaundice in these cats. Spleen showed severe lymphoid depletion around the central artery of the follicles (Fig. 5F). Severe lymphoid depletion of the spleen might be due to severe viral infection in addition to heavily concurrent bacterial infection as shown by Boosinger *et al.* [24] who recorded that the leukocytosis occurs due to complicating secondary bacterial infection.

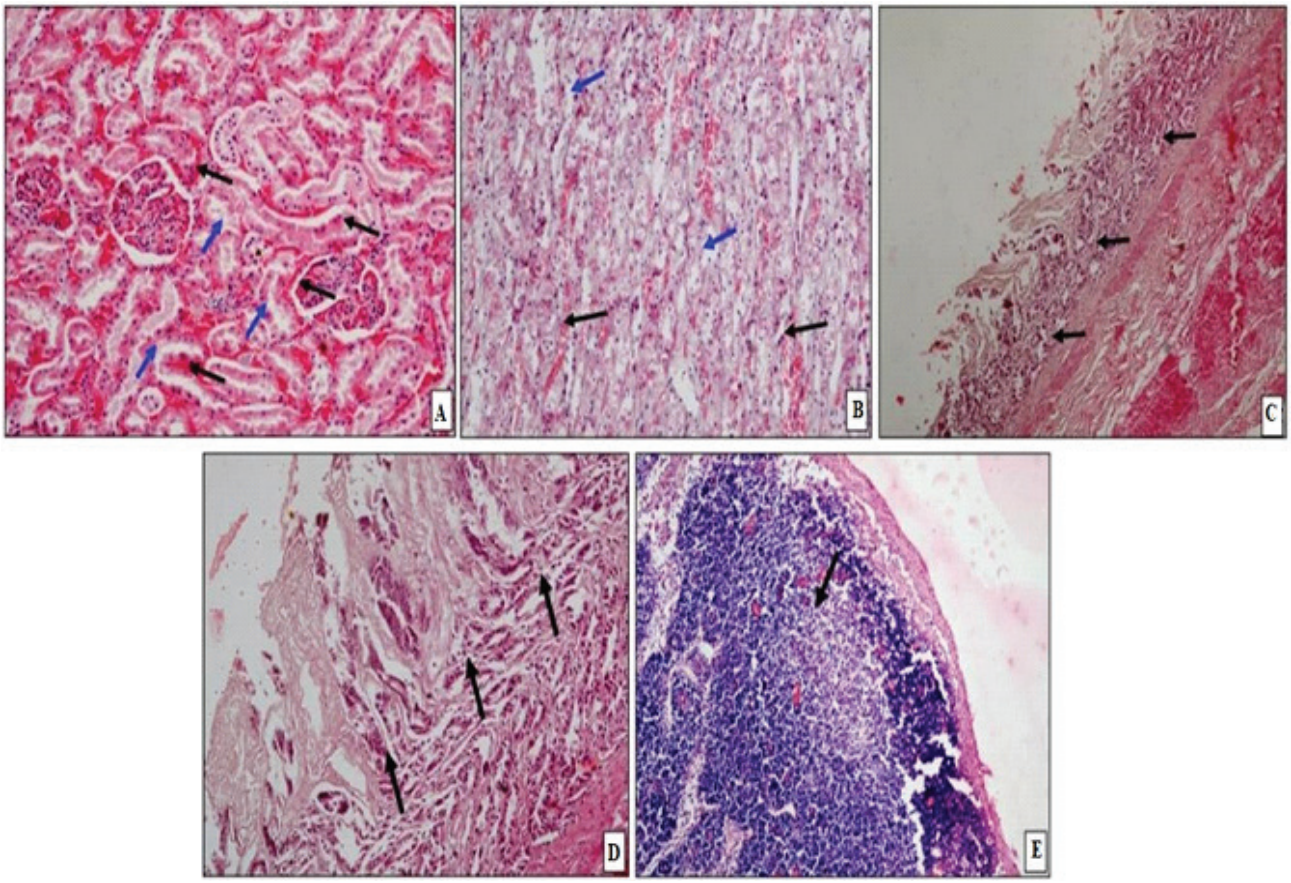

Fig. 6. Microscopic lesions of different organs (kidney, intestine and mesenteric lymph node) of cats infected with FPV. [A: The medullary region of the kidney showing hemorrhages in the interstitial spaces with microthrombus formation in the interstitium and the glomerular tuft (black arrows). Tubular epithelium lost its brush border and underwent coagulative necrosis (blue arrows). In many fields, the tubular lumen was filled with necrotic debris, 40X, H&E. B: The renal cortex showing extensive hemorrhages (black arrows) in the interstitium and severe necrosis of the tubular epithelium (blue arrows), 20X, H&E. C: Intestine showing sloughed-off and necrosed intestinal villi exposing the lamina propria to the lumen. Crypts are dilated with degenerating epithelium (arrows) which are desquamated from the basement membrane, 40X, H&E. D: Intestine showing severely affected mucosal layer with sloughed villi. Lamina propria showing enlarged crypts containing degenerating crypt epithelium (arrow), 10X, H&E. E: Mesenteric lymph node showing depletion of lymphoid follicles (arrow), 20X, H&E].

Microscopically, the section of the kidney showed hemorrhages in the interstitial spaces of the medullary region with microthrombus formation in the interstitium and glomerular tuft (Fig. 6A). Tubular epithelium lost its brush border and underwent coagulative necrosis (Fig. 6A). In many fields, the tubular lumen was filled with necrotic debris (Fig. 6A). The renal cortex showed extensive hemorrhages in the interstitium and severe necrosis of the tubular epithelium (Fig. 6B). The kidney changes noticed in the present study were supported by the observations of Machlachlan *et al*. [25], who also observed necrosis of developing renal tubular cells due to the generalized infection of FPV. Section of intestine showed sloughed-off and necrosed intestinal villi exposing the lamina propria to the lumen (Fig. 6C). Crypts were dilated with degenerating epithelia which are desquamated from the basement membrane (Fig. 6C). The mucosal layer was severely affected with sloughed villi. Lamina propria showed enlarged crypts containing degenerating crypt epithelium (Fig. 6D). A similar finding was also observed by Carlson *et al*. [26], Jones *et al*. [18] and Machlachlan *et al*. [25] in FPV infection in cats. Destruction of intestinal crypt epithelium might be due to mucosal collapse, with contraction and fusion of intestinal villi [27]. In severe acute disease, crypt epithelium can slough completely, leaving only the basement membrane. Therefore, the histological lesions of intestine and lymphoid tissue are considered as pathognomonic for FPV [28]. Mesenteric lymph nodes showed depletion of lymphoid follicles (Fig. 6E). There was marked lymphocyte destruction in mesenteric lymph nodes. Similarly, Boes and Durham [29] reported that immunosuppression in FPV infection occurs directly through lymphocytolysis and indirectly through the depletion of lymphocyte precursors.

Conclusion

In conclusion, the macroscopic and microscopic findings in FPV organs revealed significant pathological changes in intestine, mesenteric lymph node, heart, lungs, liver, kidney and spleen. Very few reports are available on the pathological aspects of FPV infection in cats. Therefore, the outcome of the present study can yield valuable information to aid in the diagnosis of FPV infection in cats under field conditions.

ACKNOWLEDGEMENT

The authors are highly thankful to the Vice Chancellor, Central Agricultural University, Imphal and the Dean, College of Veterinary Sciences and Animal Husbandry, Selesih, Aizawl, Mizoram for providing necessary facilities to carry out this study.

REFERENCES

1. Truyen U, Addie D, Belak S. Feline panleukopenia. ABCD guidelines on prevention and management. J Feline Med Surg. 2009; 11:538–546. doi.org/10.1016/j.jfms.2009.05.002.

2. Cotmore SF, Agbandje-McKenna M, Canuti M, *et al*. ICTV virus taxonomy profile: parvoviridae. J Gen Virol. 2019; 100:367. DOI: 10.1099/jgv.0.001212.

3. Stuetzer B, Hartmann K. Feline parvovirus infection and associated diseases. Vet J. 2014; 201(2):150-155. DOI: 10.1016/j.tvjl.2014.05.027.

4. Goodman LB, Lyi SM, Johnson NC. Binding site on the transferrin receptor for the parvovirus capsid and effects of altered affinity on cell uptake and infection. J Virol. 2010; 84:4969–4978.

5. Kruse BD, Unterer S, Horlacher K. Prognostic factors in cats with feline panleukopenia. J Vet Intern Med. 2010; 24:1271–1276. doi.org/10.1111/j.1939-1676.2010.0604.x.

6. Chethan GE, De UK, Singh MK, *et al*. Antioxidant supplementation during treatment of outpatient dogs with parvovirus enteritis ameliorates oxidative stress and attenuates intestinal injury: A randomized controlled trial. Vet Anim Sci. 2023; 21:100300. doi.org/10.1016/j.vas.2023.100300.

7. Luna LG. Manual of Histologic Staining Methods of the Armed Forces Institute of Pathology. 3rd Edition. New York: McGrawHill; 1968.

8. Mochizuki M, Horiuchi M, Hiragi H, San Gabriel MC, Yasuda N, Uno T. Isolation of canine parvovirus from a cat manifesting clinical signs of feline panleukopenia. J Clin Microbiol. 1996; 34(9):2101–2105. doi.org/10.1128/jcm.34.9.2101-2105.1996.

9. Saranraj P, Manigandan M, Nisha R, Yuvalakshmi L. Molecular detection of feline panleukopenia virus from clinical cases in India. AJVS. 2024; 17(2):27-33. DOI. org/10.37940/AJVS.2024.17.2.4.

10. Raheena KP, Priya PM, Mani BK, Mini M, Pillai UN. Comparison of different diagnostic test to detect feline panleukopenia virus among cats in Kerala, India. Indian J Anim Res. 2017; 51(2):347-349, DOI: 10.0.73.117/ijar.10975.

11. Jayanthi N, Saahithya R, Pazhanivel N, *et al*. Pathomorphological and molecular studies on an outbreak of feline parvo viral infection in a colony of cats. Int J Curr Microbiol App Sci. 2018; 7(6):1800-1806.

12. Meurs KM, Fox PR, Magnon AL, Liu SK, Towbin JA. Molecular screening by polymerase chain reaction detects panleukopenia virs DNA in formalin-fixed hearts from cats with idiopathic cardiomyopathy and myocarditis.

Cardiovasc Pathol. 2000; 9(2):119–126. doi.org/10.1016/S1054-8807(00)00031-4.

13. Rice JK. Successful treatment of feline Panleukopenia: a guideline for Rescuers and Veterinarians, Part I. J Vet Sci Med Diagn. 2017; 6:2, DOI: 10.4172/2325-9590.1000223.

14. Pandey S. Feline panleukopenia infections: Treatment and control in Nepal. EJ-Vetmed. 2022; 2(1)10-14. doi.org/10.24018/ejvetmed.2022.2.1.19.

15. Awad RA, Khalil WK, Attallah AG. Epidemiology and diagnosis of feline panleukopenia virus in Egypt: Clinical and molecular diagnosis in cats. Vet World. 2018; 11:578-584. doi.org/10.14202/vetworld.2018.578-584.

16. Mosallanejad B, Avizeh R, Ghorbanpoor NM. Antigenic detection of Feline Panleukopenia virus (FPV) in diarrhoeic companion cats in Ahvaz area. Iran J Vet Res. 2009; 10(3):289-293.

17. Lane EP, Brettschneider H, Caldwell P, *et al*. Feline panleukopaenia virus in captive non-domestic felids in South Africa. Onderstepoort J Vet Res. 2016; 83(1):a1099. doi: 10.4102/ojvr.v83i1.1099.

18. Jones TC, Hunt DR, King WN. Veterinary Pathology. 6th Edition. Baltimore: Lippincott Williams and Wilkins; 1997. p. 1149-1221.

19. Singh K, Shukla S, Shrivastava N, *et al*. Clinico-histo-ultra pathobiology of parvoviral enteritis of felines in and around Indore, Madhya Pradesh. J Pharm Innov. 2023; 12(12):2019-2027.

20. Robinson WF, Huxtable CR, Pass DA. Canine parvoviral myocarditis: a morphological description of the natural disease. Vet Pathol. 1980; 17(3):282–93. doi.org/10.1177/030098588001700302.

21. Turk J, Miller M, Brown T, *et al*. Coliform septicemia and pulmonary disease associated with canine parvoviral enteritis: 88 cases (1987-1988). J Am Vet Med Assoc. 1990; 196(5):771–3. doi.org/10.2460/javma.1990.196.05.771.

22. Csiza CK, Lahunta AD, Scott FW, Gillespie JH. Pathogenesis of feline panleukopenia virus in susceptible newborn kittens ii. pathology and immunofluorescence. Infect Immun. 1971; 3(6):838-846. DOI: 10.1128/iai.3.6.838-846.1971.

23. Bayati HAA, Akaby SRA. Study of histopathological changes associated with feline pan leukopenia virus infection in naturally infected cats. JCEWU. 2017; 1(26):511-520. doi.org/10.31185/eduj.vol1.iss26.106.

24. Boosinger TR, Rebar AH, DeNicola DB, Boon GD. Bone marrow alterations associated with canine parvoviral enteritis. Vet Pathol. 1982; 19:558, DOI: 10.1177/030098588201900513

25. Machlachlan N, Dubovi EJ, Barthold SW, *et al*. Parvoviridae. In: Machlachlan N, Dubovi EJ, editors. Fenner's Veterinary Virology. London: Elsevier; 2016.

26. Carlson JH, Scott FW, Duncan JR. Feline Panleukopenia. I. Pathogenesis in germfree and specific pathogen-free cats. Vet Pathol. 1977; 14(1):79-88, DOI: 10.1177/030098587701400110.

27. Barrs VR. Feline panleukopenia A re-emergent disease. Vet Clin Small Anim. 2019; 49(4):651-670. doi.org/10.1016/j.cvsm.2019.02.006.

28. Olariu-Jurca I, Coman M, Jurca AO, Stancu A, Lazău A, Avram E. Morphological changes in cat parvovirosys/feline panleucopenia and their relevance in disease histopathological diagnosis. Lucrari Stiintifice: Medicina Veterinara Timisoara. 2011; 44(2):173-178.

29. Boes KM, Durham AC. Bone marrow, blood cells, and the lymphoid/lymphatic system. In: Zachary JF, (editor). Pathologic Basis of Veterinary Disease. 6th Edition. London: Elsevier, London; 2017. 724–804. DOI: 10.1016/B978-0-323-35775-3.00013-8.

Cite this article as: Lalmuanpuii R, Kiran J, Sarma K, Roychoudhury P, Chethan GE, Malsawmsangi, Thakur N. Assessment of macroscopic and microscopic lesions in feline Panleukopenia virus infected organs in cats. Explor Anim Med Res. 2025; 15(1), DOI: 10.52635/eamr/15.1.133-139.