

Histopathological findings in a cat with feline panleukopenia

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ABSTRACT

Feline panleukopenia (FPL) is a highly contagious and often fatal viral disease of cats, characterized by viral tropism for rapidly dividing cells. This study aimed to document the histopathological changes in various organs of a cat affected with feline panleukopenia virus (FPV). Necropsy was performed on one of the sixty-three confirmed FPL cases presented to the Department of Veterinary Medicine, KVAFSU, Hebbal, Bengaluru. The cat, which succumbed during therapy, was subjected to post-mortem examination and the diagnosis was confirmed by PCR, antigen ELISA and pathognomonic lesions. Sixteen tissue samples were collected and processed for histopathology. Gross lesions included emaciation, segmental enteritis with necrotic mucosa, pulmonary congestion, hepatic hemorrhages, cortical necrosis of kidneys and lymphoid depletion in the spleen. Microscopically, epithelial necrosis, villous atrophy, crypt depletion, goblet cell hyperplasia, lymphoid depletion in spleen and mesenteric nodes, bone marrow appeared pale and hypoplastic and extra-intestinal changes in the liver, lungs, kidneys, heart and cerebellum were observed. These findings confirm the predilection of FPV for mitotically active cells in the intestines, bone marrow and lymphoid tissues, leading to severe immunosuppression and systemic organ damage. The results underscore the importance of strengthening vaccination strategies beyond 12 weeks of age for effective prevention.

Keywords: Feline panleukopenia, histopathology, lymphoid depletion, necropsy

INTRODUCTION

Feline panleukopenia (FPL), caused by feline panleukopenia virus (FPV), is one of the most lethal infectious diseases of cats, with a high morbidity and mortality in unvaccinated populations. The virus replicates in rapidly dividing cells such as intestinal crypts, bone marrow progenitors, lymphoid tissues and the fetal/neonatal cerebellum, resulting in severe enteritis, leukopenia, immunosuppression and neurological deficits in neonates¹⁻³. Although clinical and diagnostic aspects are well-documented, detailed histopathological descriptions from naturally infected cats in India remain scarce. This case study aimed to characterize the histopathological lesions in different organs of a cat that succumbed to FPL.

MATERIALS AND METHODS

One domestic shorthair cat, confirmed positive for FPV by PCR and antigen ELISA (Fig. 1), which succumbed during hospitalization, was subjected to necropsy. Sixteen tissue samples which included the gastrointestinal tract, mesenteric lymph node, spleen, thymus, processing of bone marrow, liver, lungs, kidneys, heart and cerebellum, were collected and fixed in 10% neutral buffered formalin. After 48 hours, tissues were trimmed (3–5 mm), dehydrated in graded alcohols, cleared in xylene and embedded in paraffin. Sections of 4–5 µm thickness were cut using a rotary microtome, mounted on Mayer's albumin-coated slides and stained with hematoxylin and eosin (H&E) following standard protocol⁴. Gross and microscopic lesions were examined under a light microscope and compared with earlier reports on feline panleukopenia.

RESULTS

The gross pathology of the carcass exhibited poor body condition, pale mucous

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membranes and dehydration (Fig. 2). The intestines were gas-filled, congested and hemorrhagic, with segmental necrosis and fibrinous exudates (Fig. 3). The spleen was markedly reduced in size with pale parenchyma. Lungs showed congestion with petechiae, kidneys revealed cortical necrosis and the liver was friable with multifocal hemorrhages (Fig. 4).

Histopathological Findings

The esophagus (Fig. 5) showed squamous epithelial hyperplasia along with epithelial necrosis, reflecting degenerative and proliferative changes. The stomach (Fig. 6a) exhibited goblet cell hyperplasia, mucosal erosion and accumulation of cell debris

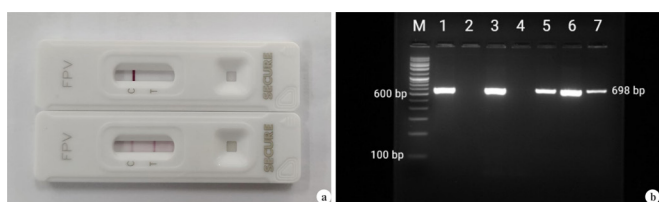


Fig. 1. FPV positive confirmation **a.** FPV-Ag snap test (ELISA) showing positive results. **b.** PCR showing positive with amplicon size of 698bp, Lane M-100bp ladder, Lane 1-positive control, Lane 2-negative control, Lane 3,5,6 & 7-positive clinical samples, Lane 4-negative sample

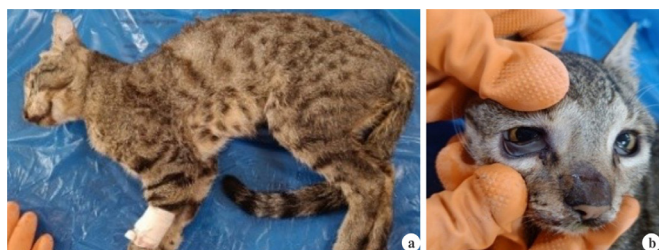


Fig. 2. Low body condition carcass **a.** Severely dehydrated **b.** Pale mucous membranes & sunken eyeballs

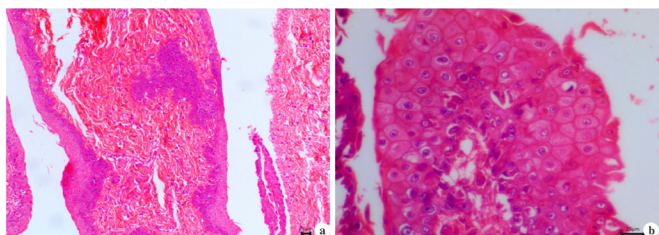


Fig. 5. Section of esophagus showing **a.** Hyperplasia of squamous epithelia **b.** Epithelial necrosis, H&E, (100X & 400X)

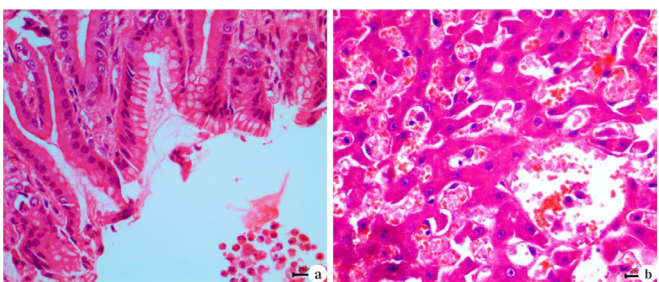


Fig. 6. **a.** Section of stomach showing goblet hyperplasia, erosion of mucosa and cell debris in lumen H & E, 400X **b.** Section of liver showing sinusoidal dilatation and congestion, H & E, 400X



Fig. 4. Gross pathology of Lungs, Kidneys and Liver – **a.** Lungs-hemorrhages with congestion **b.** Kidneys showing cortical necrosis, discoloration & normal medullary region **c.** Liver- hemorrhages and necrosis



Fig. 3. Segmental enteritis in SI- reddened, thickened and edematous, petechial hemorrhages on serosal surfaces

within the lumen, suggestive of severe mucosal injury associated with FPL infection. Hepatic lesions (Fig. 6b) included sinusoidal congestion, Kupffer cell hyperplasia, focal hepatocellular degeneration and portal mononuclear infiltration⁵. The different parts of small intestines and large intestines including duodenum, jejunum, ileum, cecum and colon (Figs. 7, 8) revealed crypt epithelial necrosis, villous blunting and fusion, epithelial denudation, goblet cell hyperplasia and Peyer's patch depletion, along with severe mucosal hemorrhage accompanied by neutrophilic and macrophagic infiltration^[2]. The mesenteric lymph nodes (Fig. 9a) showed lymphoid depletion, follicular necrosis, sinus histiocytosis and plasma cell infiltration, reflecting marked immunosuppression¹. The heart (Fig. 10a) presented focal myocardial hemorrhages; although myocarditis was not grossly evident, microscopic lesions indicated possible subclinical cardiac involvement⁴. The cerebellum (Fig. 10b) showed neuronal degeneration and hypocellularity, consistent with FPV-induced cerebellar hypoplasia in perinatal infections¹. The spleen (Fig. 9b) exhibited white pulp atrophy, lymphoid necrosis, cortical depletion and hemorrhage⁵. Renal tissues (Fig. 10c) revealed tubular degeneration, necrosis and hyaline casts within the renal tubules, with associated interstitial congestion². Bone marrow examination (Fig. 9c) demonstrated severe hypocellularity with aplasia of

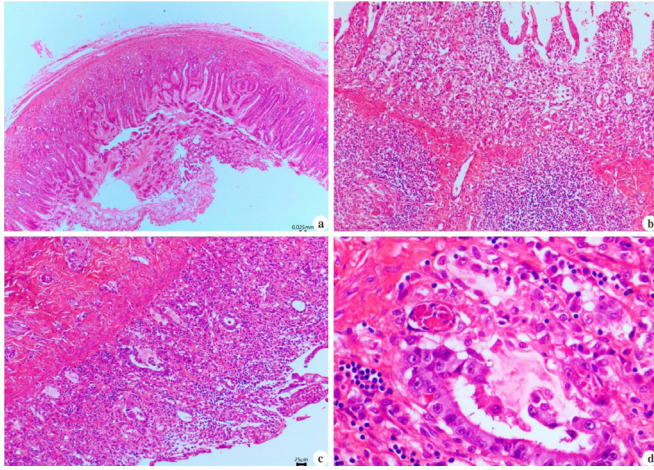


Fig. 7. Histopathological lesions observed in the small intestinal parts of FPV affected cat-**a.** Section of duodenum showing denudation of epithelia, goblet hyperplasia, fusion of villi, H & E, 40X **b.** Section of jejunum depleted payer's patches with granuloma and fusion of villi necrosis of epithelia, H & E, 100X **c.** Section of ileum showing crypt epithelial necrosis & fusion of villi necrosis of epithelia of villi with infiltration of mononuclear cells, H & E, 100X and 400X

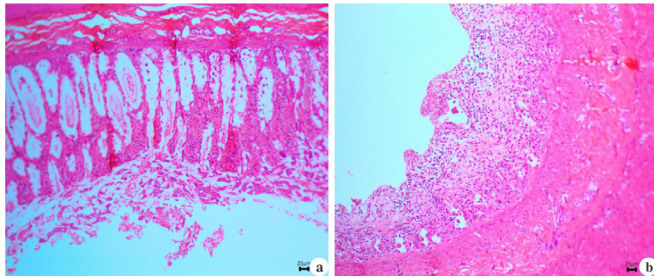


Fig. 8. Histopathological lesions observed in the large intestinal parts of FPV affected cat-**a.** Section of caecum showing dilation of glands, necrosis & sloughing of lining cells **b.** Section of colon showing inflammatory changes in mucosa (H & E, 10X)

myeloid and erythroid series, adipose replacement and reduced mitotic activity, consistent with panleukopenia⁵. Pulmonary changes (Fig. 10d) were characterized by interstitial pneumonia with alveolar edema, congestion and bacterial aggregates, most likely secondary to neutropenia⁷.

DISCUSSION

The histopathological findings confirm FPV's strong tropism for mitotically active tissues, especially intestinal crypts, bone marrow precursors and lymphoid follicles. Intestinal lesions such as villous blunting, crypt necrosis and epithelial sloughing accounted for the severe diarrhea and malabsorption observed clinically, while bone marrow aplasia and lymphoid depletion explained the profound leukopenia and immunosuppression^{2,3}.

Secondary bacterial infections, as evident in pulmonary pneumonia, likely exacerbated the clinical

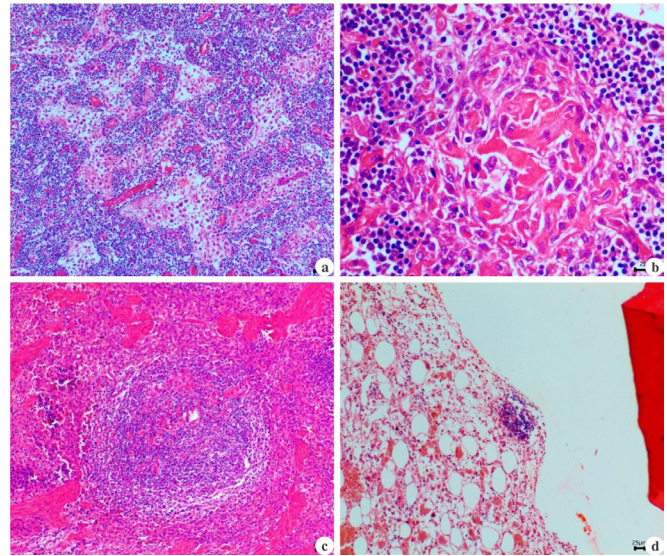


Fig. 9. Histopathological lesions showing immunosuppression in FPV affected cat-**a.** Section of mesenteric lymph node showing hypocellularity, infiltration of plasma cells and granuloma formation within follicle (H & E, 100X and 400X) **b.** Section of spleen showing decreased cellularity, H & E, 100X **c.** Section of bone marrow showing severe hypocellularity, note foci of lymphoid aggregation, H & E, 10X

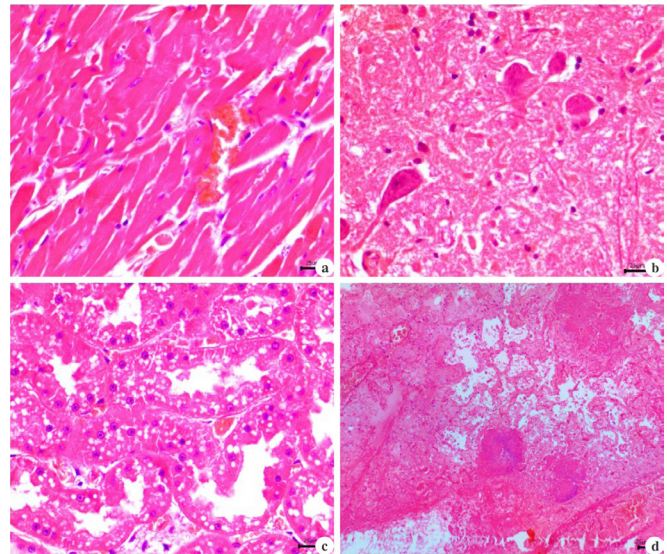


Fig. 10. Histopathological lesions in vital organs due to FPV **a.** Section of heart showing focal hemorrhages, H & E, 400X **b.** Section of cerebellum showing neuronal degeneration and decreased cellularity, H & E, 400X **c.** Section of kidney showing vacuolar degeneration in tubular epithelia, H & E, 400X **d.** Section of lung showing severe pneumonia with secondary bacterial aggregation and edema, H & E, 100X

deterioration⁵. Neural involvement in the cerebellum highlighted the risk of congenital cerebellar hypoplasia in kittens infected in utero¹. In our case cardiac damages were minimal due to the acute stage of the disease but the literature suggests FPV DNA and myocardial inflammation can occur in some advanced cases^{6,7}.

The pathological changes in this cat mirrored those reported globally⁸⁻¹⁰ and reinforced the importance of complete vaccination schedules beyond 12 weeks of age to ensure adequate protection.

CONCLUSION

Histopathological analysis of feline panleukopenia in this case confirmed characteristic lesions in the gastrointestinal tract, bone marrow and lymphoid organs, along with multi-organ involvement including liver, lungs, kidneys, cerebellum and heart. These changes reflect viral replication in rapidly dividing cells and the systemic consequences of immunosuppression. Strengthening vaccination protocols and early supportive interventions remain essential to reduce mortality in cats with FPL.

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