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RESEARCH ARTICLE

Hematopathological studies on Feline Panleukopenia with emphasis on bone marrow assessment

J. Thilahayswari[1], K. Nagarajan*, S. Hemalatha and M. Chandrasekar[2]
Department of Veterinary Pathology, Madras Veterinary College, Tamil Nadu Veterinary and Animal Sciences University, Chennai, India
[1]PG Scholar; [2]Resident Veterinary Services Section, Madras Veterinary College, Tamil Nadu Veterinary and Animal Sciences University, Chennai, India

***Address for Correspondence**
K. Nagarajan, Professor, E-mail: nagavet@gmail.com

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ABSTRACT

Feline Panleukopenia Virus (FPV) is a highly contagious viral disease causing severe hematological abnormalities in cats. This study was aimed to evaluate the hematological, biochemical, and bone marrow changes in naturally infected cats and to characterize FPV at the molecular level. A total of 22 cats exhibiting clinical signs of anorexia, pallor of mucus membrane, recumbency, and emaciation were examined. Out of this 7 were subjected to detailed post-mortem evaluation. Hematological analysis revealed significant anemia, leukopenia, and thrombocytopenia in FPV-infected cats, accompanied by hypoglycemia and hypoproteinemia, while liver and kidney parameters remained largely unaffected. Blood smear and bone marrow cytology showed predominant erythroid hypoplasia, myeloid hypoplasia and megakaryocytic hyperplasia, indicating compensatory hematopoietic responses. Necropsy findings included generalized pallor of visceral organs, thickened intestines with mucosal petechiae, mild hepatomegaly, and pulmonary edema. Molecular detection of DNA of FPV from 19 bone marrow samples via PCR targeting the VP2 gene confirmed infection in 15 cats (78.95%). Phylogenetic analysis revealed that the Indian bone marrow isolate clustered closely with other Indian FPV strains, forming a distinct clade separated from strains reported in Europe, Africa, and East Asia, suggesting geographic evolution patterns. These findings highlight the critical role of bone marrow in FPV pathogenesis, the systemic impact of infection, and the regional genetic distinctness of circulating FPV strains in India, which has implications for disease surveillance and vaccine strategies.

Keywords: Bone marrow, cytology, Feline Panleukopenia Virus, hematology, PCR, phylogenetic analysis

INTRODUCTION

Panleukopenia is a hematologic abnormality characterized by a marked reduction in all circulating white blood cell types, most often detected through complete blood count (CBC) evaluation in veterinary practice [1]. Beyond being considered as a hematological abnormality, it is actually a clinical syndrome reflecting systemic viral infection affecting immune and haemopoietic organs [2].Though there are many causes of panleukopenia in cats, the most common and prime cause is Feline parvovirus and the infection is termed as Feline panleukopenia or feline distemper. It is a linear, single-stranded DNA molecule with unique hairpin structures at both ends that is around 5 kb long. It belongs to the Parvoviridae family and the genus Protoparvovirus[3].

Following the virus' initial replication in oropharyngeal lymphoid tissue, it spreads throughout the body through the blood to all tissues, causing viraemia which could be identified clinically after 2–10 days of incubation and diagnosed after 2–7 days post-infection [4]. Significant changes in occur especially to the bone marrow and lymphoid tissue causing severe destruction of whole leukocytes ,thereby substantiating the disease name [5]. Given the central role of bone marrow in hematopoiesis, its evaluation is critical in understanding the severity of FPV infection. This study aims to investigate the presence of parvovirus within the bone marrow and to perform molecular characterization, thereby addressing the existing gaps in knowledge regarding the circulating whole-genome sequences of FPV in India.

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MATERIALS AND METHODS

A total of 22 cats clinically diagnosed with Feline panleukopenia (FPV) were included in this study. These animals were selected based on the presence of clinical signs indicative of panleukopenia, including anorexia, pale mucous membranes, recumbency, hypothermia, and emaciation. Cats of varying ages and both sexes were included to assess age- and sex-related susceptibility. Each cat underwent a detailed

clinical examination to record general health status and specific signs. Seven cats succumbed to death due to late clinical presentation and they were subjected to detailed necropsy examination.

Peripheral blood was collected aseptically from all the cats to perform complete blood counts and serum biochemical parameters using an automated hematology analyzer (BC-2800Vet, Mind ray Medical Instrumentation, China) and automated serum biochemistry analyzer (A15 Random Access Analyzer, Biosystems, Barcelona, Spain). A total of 22 Bone marrow aspirates and biopsy including 15 from live animals and 7 from dead cats were collected from the trocanteric fossa of proximal femur under sedation using standard aseptic techniques for cytological examination and viral detection. Control samples were collected from 6 apparently healthy domestic short haired cats of age ranging from 6 months to 2 years to serve as reference for hematological analyses.

Aspirate cytology smears were stained with Leishman-Giemsa stain as per the standard protocol. Tissue samples were routinely processed, with bone tissue decalcified before embedding. Sections 3–5 µm thick were stained with hematoxylin and eosin (H&E) for routine histopathology[6]. Additionally, special stain—Masson's trichrome were performed to check for myelofibrosis for differentiating collagen and semi-quantifying collagen deposition in the bone marrow.

Out of the 22 bone marrow samples, only 19 were subjected to PCR due to insufficient sample quantity in the remaining cases. DNA was extracted from bone marrow samples (approximately 25 mg of tissue) using the commercially standardized DNeasy Blood and Tissue Kit (QIAGEN, Germany). Samples were subjected to PCR targeting the VP2 gene of Feline parvovirus using the following primers: Forward primer (FPL-FP): 5'–GCT TTA GAT GAT ACT CAT GT–3'; Reverse primer (FPL-RP): 5'–GTA GCT TCA GTA ATA TAG TC–3'.Those samples yielding a positive 698 base pair (bp) band by PCR amplification were further subjected to genetic sequencing. The sequence data were compared with VP2 reference sequences from the NCBI database, and phylogenetic tree was generated using MEGA12 software to analyze the genetic relationships of the isolates of different geographical location.

RESULTS

Sex, age and breed wise incidence

Sex, age and breed wise incidence of feline panleukopenia were tabulated (Table 1) and interpreted diagrammatically (Fig. 1). Out of the 22 cats diagnosed with Feline panleukopenia, male cats were more commonly affected compared to females. Age-wise, the highest proportion of cases was observed in cats younger than 6 months among all age groups, indicating a higher susceptibility among younger animals. Regarding breed distribution, the majority of affected cats were Domestic Shorthaired was affected, suggesting that FPV infection predominantly occurs in the common domestic population rather than

Fig. 1. Bar chart depicting Sex, age and breed wise incidence of feline panleukopenia

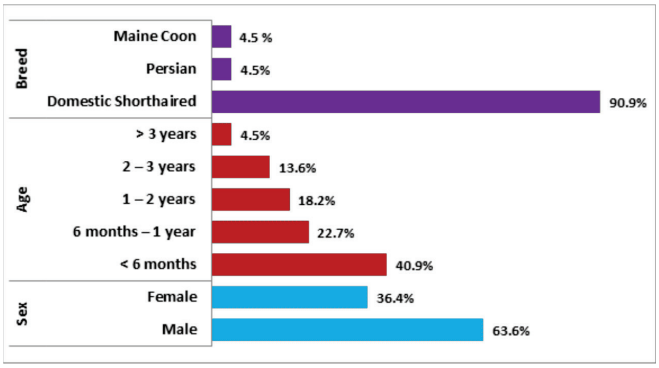

Table 1. Sex, age and breed wise incidence of feline panleukopenia

Parameter	Category	Number of Cats	Percentage (%)
Sex	Male	14	63.6
	Female	8	36.4
Age	< 6 Months	9	40.9
	6 Months – 1 Year	5	22.7
	1 – 2 Years	4	18.2
	2 – 3 Years	3	13.6
	> 3 Years	1	4.5
Breed	Domestic Shorthaired	20	90.9
	Persian	1	4.5
	Maine Coon	1	4.5

** Highly Significant ($p<0.01$); *Significant ($p<0.05$); NS – Not Significant

Table 2. Hematological Comparison between Healthy and FPV-Infected Cats

Parameter	Unit	Group 1 Apparently healthy (n=6)	Group 2 Feline panleukopenia (n=22)	t statistic
Hemoglobin	(g/dl)	11.3±0.41	8.7±0.44	2.97**
PCV	(%)	36.33±2.060	27.58±2.35	1.87^{NS}
RBC	(mil/µl)	7.18±0.34	5.06±0.41	2.29*
WBC	(/cmm)	13633.33±1373.48	6543.18±344.16	10.58**
Platelets	(/cmm)	401916.67±45134.69	242863.64±24939.35	2.98**
Neutrophils	(%)	56±4.03	61.77±2.51	-1.09^{NS}
Neutrophil count	(/cmm)	7715.33±1023.33	2245.63±246.98	7.84**
Lymphocytes	(%)	38±3.68	30.29±2.07	1.74^{NS}
Lymphocyte count	(/cmm)	5771±727.59	1031.38±117.02	9.72**
Monocytes	(%)	3.07±0.55	4.56±0.49	-1.52^{NS}
Monocyte count	(/cmm)	386.07±35.75	160.30±23.04	4.69**
Eosinophils	(%)	2.93±0.65	3.38±0.53	-0.415^{NS}
Eosinophils count	(/cmm)	360.93±54.76	104.88±16.10	6.19**

** Highly significant (p<0.01); *Significant (p<0.05); NS – Not Significant

Table 3. Serum Biochemistry findings comparison between Healthy and FPV-Infected Cats

Parameter	Unit	Group 1 Apparently healthy (n=6)	Group 2 Feline panleukopenia (n=22)	t statistic
Glucose	(mg/dl)	95±3.25	76.27±3.88	2.43*
Total protein	(g/dl)	6.75±0.29	5.15±0.28	2.84**
Albumin	(g/dl)	2.9±0.19	2.81±0.19	0.22^{NS}
BUN	(mg/dl)	23.27±2.05	20.23±1.11	1.27^{NS}
Creatinine	(mg/dl)	0.81±0.09	1.08±0.10	1.33^{NS}
ALT	(mg/dl)	53.33±2.51	46.14±2.95	1.22^{NS}
ALP	(mg/dl)	22.33±3.66	34±4.48	1.31^{NS}

** Highly significant (p<0.01); *Significant (p<0.05); NS – Not Significant

Table 4. Histological and Cytomorphological evaluation of bone marrow

Parameter	Observation	Number of Cats (n = 22)	Percentage (%)
	Normocellular	2	9.1
Marrow Cellularity	Hypocellular	20	90.9
	Hypercellular	-	-
	Normal	-	-
Erythroid Lineage	Hypoplasia	20	90.9
	Hyperplasia	2	9.1
	Normal	-	-
Myeloid Lineage	Hypoplasia	17	77.3
	Hyperplasia	5	22.7
	Normal	8	36.3
Megakaryocytic Lineage	Hyperplasia	10	45.5
	Hypoplasia	4	18.2

purebred cats. The most prominent clinical signs were anorexia, pale mucous membranes, recumbency, hypothermia, and emaciation. Additional findings included vomiting, dyspnea, and absence of pupillary light reflex (PLR). Less frequently, cats exhibited loss of nociception, nystagmus, seizures, and hyperthermia, reflecting the variable severity and systemic impact of Feline panleukopenia.

Haematology and serum biochemistry findings

Comparison between apparently healthy cats and Feline Panleukopenia-infected cats revealed significant alterations in several hematological and serum bichemistry parameters as listed in the table 2 and table 3 respectively. Hemoglobin levels were significantly reduced in FPV-infected cats (8.7±0.44 g/dl) compared to healthy controls (11.3±0.41 g/dl), indicating anemia. Similarly, RBC counts (5.06±0.41 mil/µl) and platelet counts (242863.64±24939.35 /cmm) were significantly lower in infected cats. White blood cell analysis showed a marked decrease in total WBC count (6543.18±344.16/cmm), neutrophil (2245.63±246.98/cmm) and lymphocyte counts (1031.38±117.02/cmm) while the percentages of neutrophils (61.77±2.51%) and lymphocytes (30.29±2.07%) did not differ significantly. Monocyte counts (160.30±23.04 /cmm) were significantly decreased in FPV-infected cats, whereas monocyte percentage (4.56±0.49%) remained unchanged. Eosinophil counts (104.88±16.10/cmm) were also significantly reduced, although eosinophil percentage (3.38±0.58%) did not differ. Overall, these findings demonstrate that Feline Panleukopenia induces severe leukopenia, anemia, and thrombocytopenia, reflecting profound bone marrow suppression and systemic hematological impact in infected cats.

Glucose levels were significantly lower in infected cats compared to healthy controls, indicating potential hypoglycemia. Similarly, total protein levels were significantly reduced in infected cats, suggesting protein loss or impaired synthesis. Other parameters, including albumin, BUN, creatinine, ALT and ALP did not show significant differences between the two groups, indicating that renal and hepatic functions were largely unaffected in this cohort.

Necropsy findings

Necropsy revealed generalized pallor of visceral organs, Mild hepatomegaly and pulmonary edema with predominant lesions in the gastrointestinal tract showing thickened intestines, mucosal petechiae (Fig. 2a) and mesenteric lymphadenopathy (Fig. 2b).

Blood smear and bone marrow examination:

Cats infected with Feline Panleukopenia Virus exhibited anaemia, hypochromasia and acanthocytosis of RBC, leukopenia and thrombocytopenia on peripheral blood smear, reflecting significant hematological compromise. Bone marrow histopathological examination revealed marked hypocellularity (Fig. 2c) with predominance of clear ring shaped adipocytes and reduced hematopoietic cells and no myelofibrosis. Both histopathological and cytological evaluation (Table 4) (Fig.3) showed erythroid hypoplasia (Fig. 2d) predominantly characterized by less number of erythroid cells with dense nucleus and basophilic cytoplasm, suggesting impaired red blood cell production. Myeloid hypoplasia characterized by comparatively less number of myeloid cells with less dense, unlobed/lobed nucleus and pale pink to grey cytoplasm was seen in most of the cases with dysplastic erythroid precursors (Fig. 4a), highlighting compromised granulopoiesis and a mechanism for the observed leukopenia in peripheral blood. A few cases

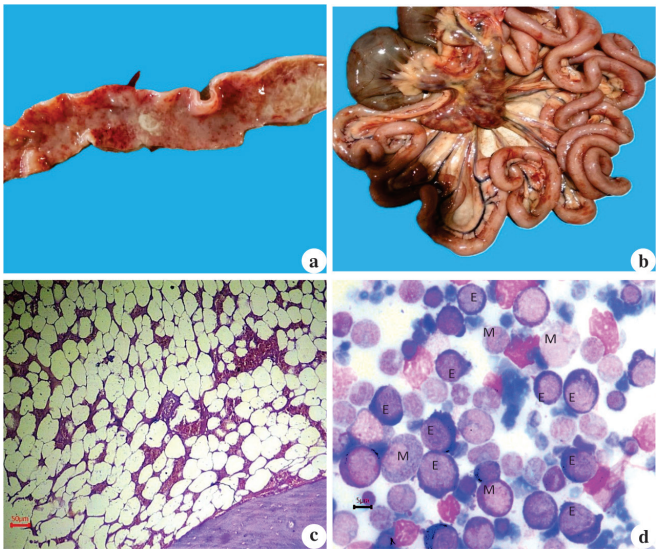

Fig. 2. (a) Intestine- Catarrhal enteritis characterized by excessive mucus coating the mucosa along with multifocal mucosal petechiae; **(b)** Mesentery- Haemorrhagic mesenteric lymphadenopathy characterized by enlarged, dark-red lymph nodes with serosal congestion; **(c)** Bone marrow histopathology- Hypocellular marrow with predominance of clear ring shaped adipocytes and reduced hematopoietic cells; H&E x100; **(d)** Bone marrow cytology- Erythroid cell(E) with basophilic cytoplasm; Myeloid cell(M) with pale pink to grey cytoplasm; Erythroid hypoplasia and myeloid hyperplasia (Leishman-Giemsa x1000).

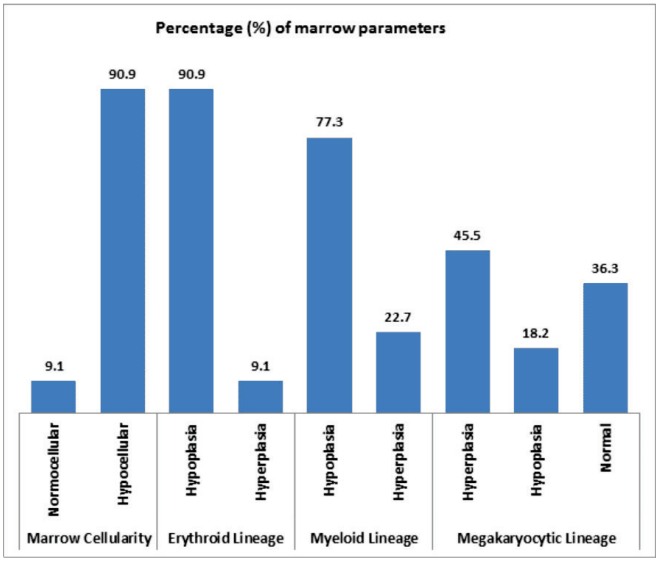

Fig. 3. Histological and Cytomorphological evaluation of bone marrow

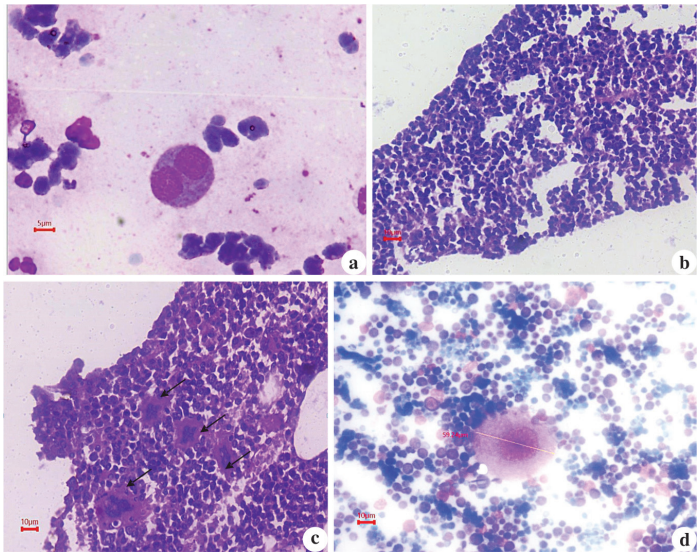

Fig. 4. **(a)** Bone marrow cytology- Binucleate (dysplastic) erythroid cell with basophilic cytoplasm indicative of dysplastic erythropoiesis; LG x1000; **(b)** Bone marrow histopathology- Erythroid hyperplasia and Myeloid hypoplasia- Numerous erythroid cells with dark, dense nucleus and basophilic cytoplasm; H&E x400; **(c)** Bone marrow histopathology- Megakaryocytic hyperplasia (Black arrows) showing >3 megakaryocytes/ low power field; H&E x100; **(d)** Bone marrow cytology- Mature megakaryocyte (59.24 µm) with abundant cytoplasm and multilobulated nucleus (Leishman-Giemsa x400).

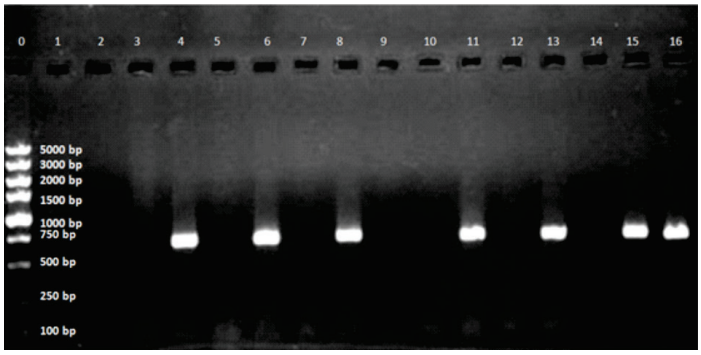

Fig. 5. Feline parvovirus targeting VP2 gene- Lane:0- DNA ladder; Lane:4,6,8,11,13,15- Positive bands at 698 base pairs; Lane:16- Positive control; Lane:2- Negative control

of erythroid hyperplasia (Fig.4b) and myeloid hyperplasia were observed, evident of compensatory erythropoiesis and granulopoiesis representing early infection or recovery stage. A very few cases showed megakaryocytic hyperplasia (Fig.4c), indicating a compensatory response to peripheral thrombocytopenia comparing normal picture (Fig.4d)

Molecular characterisation:

PCR amplification of DNA from bone marrow targeting the VP2 gene produced a specific 698 bp band (Fig.5), confirming FPV in 15 out of 19 samples (78.95%). Phylogenetic analysis (Fig. 6) revealed that the bone marrow isolate clustered closely with Indian FPV strains (OQ266795.1, MK052678.1, MH559110.1), forming a distinct clade

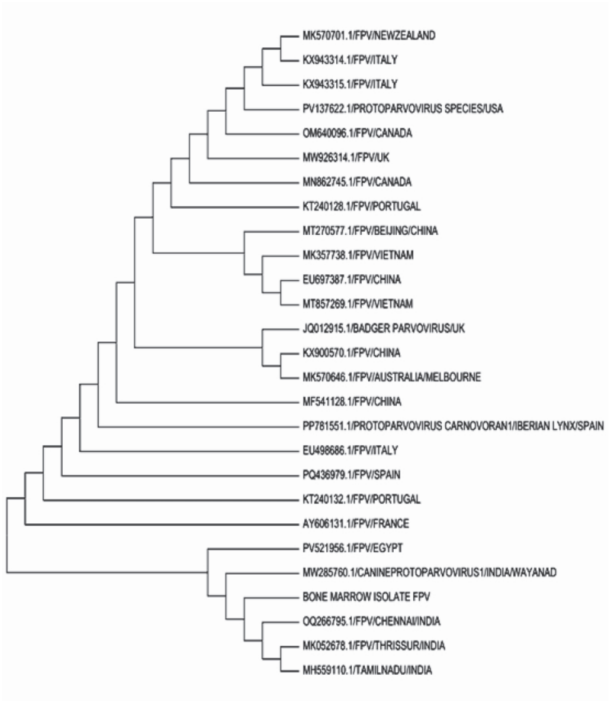

Fig.6. Phylogenetic analysis of feline parvovirus from bone marrow isolate by maximum likelihood method

separated from isolates from Portugal, France, Egypt, China, Vietnam, Australia, and New Zealand. North American and European isolates formed a separate cluster, while other protoparvoviruses appeared as distinct branches, indicating both geographical divergence and the genetic distinctness of the Indian FPV lineage.

DISCUSSION

In the study, cats with Feline Panleukopenia Virus (FPV) infection with significantly reduced hemoglobin, RBC, WBC, and platelet counts, consistent with severe bone marrow suppression align with established literature[5]. The classic pathophysiology involves viral damage to rapidly dividing hematopoietic progenitors in the bone marrow, as demonstrated in vitro studies, exhibiting strong inhibition of colony formation of both myeloid (CFU-GM) and erythroid progenitors[7]. In one study[5] of 187 cats with panleukopenia, leukopenia was detected in 65%, thrombocytopenia in 54% and anemia in 48%. The peripheral cytopenias especially leukopenia and thrombocytopenia are congruent with classical myelogram findings [8], especially immature erythroid and myeloid precursors, in both natural and experimental panleukopenia cases. These data strongly support that bone marrow suppression is a major contributor to the cytopenias seen in peripheral blood of FPV-infected cats.

Hypoglycemia and significant hypoproteinemia has also been reported in FPV-infected cats[5,9,10], including changes in total protein and other metabolic markers reflecting poor nutritional intake, gastrointestinal loss, sepsis, or impaired gluconeogenesis during systemic viral illness.

Some studies[11,12,13] have reported pronounced reductions in albumin in FPV-infected cats which result from compromised intestinal protein absorption and substantial protein leakage into the gut lumen secondary to mucosal epithelial injury [13]. However, in our study, albumin did not differ significantly. This discrepancy may be attributed to variations in the stage of disease at the time of sampling, with many animals possibly being evaluated during the early or recovery phase before substantial protein loss occurred. In addition, differences in disease severity, extent of intestinal villous damage, and hydration status at presentation may have masked hypoalbuminemia. Supportive fluid therapy prior to sampling could also have contributed to the maintenance of near-normal albumin concentrations despite ongoing intestinal pathology.

The absence of significant changes in BUN, creatinine, ALT, and ALP suggests that renal and hepatic functions remained relatively stable in many of the cats in the study. This is consistent with findings in a study, where overt azotemia or marked liver enzyme elevation is not a universal feature[5]. But a report has documented mild to moderate increases in liver enzymes in FPV-infected cats, possibly due to hypoxia, dehydration, or secondary sepsis[12]. Overall, these findings suggest that glucose and protein metabolism were only affected, while liver and kidney function remain relatively stable in the acute phase of infection.

Necropsy findings are in accordance with a study[14] conducted by Kadam *et al.,* including mucosal hemorrhages in the stomach and intestines, enlarged mesenteric lymph nodes, slightly pale bone marrow, and liver congestion in naturally infected cats. These parallels support the notion that systemic hypoxia, bone marrow depletion, and vascular damage are central in feline panleukopenia pathology.

The pronounced erythroid and myeloid hypoplasia aligns closely with the *in vitro* observations where FPV propagates and significantly inhibits colony formation from both myeloid progenitors (CFU-GM) and erythroid progenitors (BFU-E and CFU-E) in feline bone marrow cultures[7]. Another report[8] also documented hypoplastic changes in both erythroid and myeloid series in bone marrow aspirates in an experimental *in vivo* infection study.

Although erythroid and myeloid hypoplasia were dominant in our study, few cases of myeloid hyperplasia were also noticed in some cats suggesting a compensatory or reactive component in marrow response. This is echoed in a large retrospective cytological study[15] in which 46.7% of 152 feline bone marrow samples showed hyperplasia, with granulocytic (50.7%) and erythroid (45.1%) hyperplasia being the most frequent patterns. This substantiates that the feline bone marrow retains a notable capacity for regeneration or reactive proliferation, even during severe systemic stress or infection.

A prominent finding in our study was the frequent megakaryocytic hyperplasia, which may represent a compensatory response to peripheral platelet loss or destruction. While classical FPV literature has mostly focused on erythroid and myeloid suppression, the data on megakaryocytic dynamics remain less well-characterized. Our observations expand on this by suggesting that platelet lineage may mount a regenerative response, perhaps driven by peripheral thrombocytopenia. The variability in the megakaryocytic response may also reflect host-specific factors, such as the timing of sampling, the severity of infection, or the individual capacity for lineage recovery.

The high detection rate of FPV DNA (78.95%) in bone marrow samples strongly suggests that the virus actively invades and persists in hematopoietic tissue, underscoring the bone marrow as an important site for viral replication or reservoir. This observation is in agreement with a study[16] where the presence of FPV genomic material was demonstrated in bone marrow using molecular techniques including conventional PCR, qPCR, and in situ hybridization, thereby supporting the diagnostic value of bone marrow PCR, particularly in cases where faecal or antigen-based assays yield inconclusive results.

Phylogenetic analysis showed that our isolate clusters tightly with other Indian FPV strains (e.g., OQ266795.1, MK052678.1, MH559110.1), forming a well-supported clade- a finding that is consistent with previous molecular epidemiological work in India[17,18].

The clear genetic divergence of this Indian clade from FPV strains reported in Europe, Africa, and East Asia (e.g., Portugal, France, Egypt, China, Vietnam, Australia, New Zealand) suggests distinct geographical evolutionary patterns, possibly driven by regional viral ecology, host population structure, and transmission dynamics. This aligns with broader observations of FPV genetic diversity in southern India, where phylodynamic studies identified novel mutations and potential recombination events in VP2 among cat populations[18]. But this is in contrast to a report of phylogenetic analysis[19] based on the full VP2 gene of FPV which demonstrated close genetic affinity of FPV strains circulating in Mizoram state of India with the non-Indian isolates from Thailand

(MW589472), Italy (MZ508524) and China (OR727315). A recent study[20] also reported novel amino acid mutations in VP2 at positions 303, 441, 554 plus a recombination event, indicating high genetic heterogeneity in feline parvoviruses, warranting the need for genetic sequencing and phylogenetic analysis.

Altogether, our findings support the hypothesis of a regionally circulating FPV lineage in India, which may have co-evolved locally. This has important implications for disease surveillance and vaccine design, since antigenic drift in VP2 could affect viral pathogenicity and the effectiveness of vaccines derived from non-local strains.

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