Research Article

Diagnosis of Feline Panleukopenia and Endoparasite Coinfection in Cats in Bandar Lampung, Indonesia

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Abstract | Feline panleukopenia (FPL) is a highly contagious and immunosuppressive viral disease in cats that is often complicated by secondary infections, such as gastrointestinal endoparasites. This study describes the clinical, hematological, parasitological, and molecular characteristics of PCR-confirmed FPL cases in Bandar Lampung, Indonesia, and examines the potential influence of endoparasite coinfection on clinical severity. Seventeen cats with suspected FPL underwent clinical and hematological assessment, FPV rapid antigen testing, microscopic fecal examination, PCR, and DNA sequencing. Of these, twelve cats (70.6%, 12/17) were confirmed positive for FPL by PCR. Leukopenia was observed in 88.2% (15/17) of clinically suspected cats. Gastrointestinal endoparasite coinfection was identified in 58.3% (7/12) of PCR-confirmed cases, predominantly *Toxocara sp*. and *Dipylidium caninum*. Descriptive assessment indicated that coinfected cats experienced more severe gastrointestinal symptoms, particularly diarrhea and dehydration. Sequencing of two representative samples revealed ≥94% nucleotide identity with FPV strains circulating in East Asia. These findings highlight the limitations of relying on rapid antigen testing alone. The frequent occurrence of gastrointestinal parasitic coinfection and its presence with more severe disease supports the integration of molecular diagnostics and fecal examination, particularly in unvaccinated cats presenting with severe gastrointestinal signs.

Keywords | Coinfection, Endoparasite, Feline panleukopenia, PCR, Sequencing DNA

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INTRODUCTION

Feline Panleukopenia (FPL) is an acute, highly contagious viral disease of cats with substantial morbidity and mortality worldwide. It predominantly affects kittens under one year of age, with mortality rates ranging from 25–90% and infection rates nearing 100% in susceptible populations (Kusumawardhani *et al*., 2019; Mahendra *et al*., 2020; Hermawan *et al*., 2023; Kruse *et al*., 2010). This disease is caused by the Feline Panleukopenia Virus (FPV), a single-stranded, non-enveloped DNA virus that is highly resistant to environmental conditions and capable

of surviving for prolong period outside the host (Stuetzer and Hartmann, 2014). FPV has a global distribution, with high prevalence in regions of dense cat populations and low vaccination coverage (Decaro *et al*., 2010b; Sykes, 2014; Putri *et al*., 2020; Purnamaningsih *et al*., 2022). Transmission of FPV occurs primarily through direct contact with infected cats or their body fluids, including feces, vomit, saliva, and urine. Transmission transmission can also occur through the transplacental route from the mother to the fetus (Stuetzer and Hartmann, 2014). In addition, indirect transmission through mechanical vectors can occur via humans, fleas, and flies, play a significant rol due to the virus's environmental persistance (Mosallanejad *et al*., 2009; Kruse *et al*., 2010; Stuetzer and Hartmann, 2014)

Feline Panleukopenia Virus exhibits a strong tropism for actively dividing cells, especially those in the intestinal epithelium, lymphoid tissue, and bone marrow, causing severe enteritis, leukopenia, immunosuppression, and intestinal mucosal damage (Decaro and Bounavaglia, 2012; Stuetzer and Hartmann, 2014). The initial clinical symptoms include fever, lethargy, anorexia, leukopenia, and dehydration, which then manifest as leukopenia, vomiting, diarrhea, and severe depression and can end in sudden death (Abd-Eldaim *et al*., 2009; Kruse *et al*., 2010; Tinky *et al*., 2015).

Laboratory tests that can be performed to confirm the diagnosis of FPL include hematological examination, antigen and antibody detection, direct red blood cell agglutination, hemagglutination-inhibition test to detect the presence of antibodies in the sample, immunofluorescence antibodies, virus isolation, and viral DNA detection using polymerase chain reaction (PCR) (Sykes, 2014; Stuetzer and Hartmann, 2014; Weese and Evason, 2019). Molecular diagnosis via PCR and real-time PCR enables high-sensitivity, high-specificity differentiation of FPV from *canine parvovirus* (CPV), often using minor groove binder (MGB) probes (Decaro *et al*., 2010a). FPV is characterized by a subacute-to-peracute infection, often accompanied by sudden death (Jacobson *et al*., 2021).

The immunosuppressive condition caused by FPV infection makes cat patients susceptible to secondary infections, including parasitic coinfections. One of the secondary infections that often occurs is coinfection with gastrointestinal endoparasites such as *Toxocara* sp., *Ancylostoma* sp., *Giardia* sp., *Cystoisospora* sp., and *Dipylidium caninum* (Miro *et al*., 2013). These endoparasites are associated with gastrointestinal signs such as diarrhea, vomiting, anorexia, and dehydration, which overlap with FPL clinical manifestation, complicating clinical diagnosis and exacerbating disease severity (Duarte *et al*., 2016). Similar clinical presentation may also be observed in other gastroenteritis disease, further emphasizing the diagnostic challenge (Sykes, 2014; Trotman, 2015).

Several studies indicate a high prevalence of endoparasites in shelter or unvaccinated cats. For example, a study by Gates and Nolan (2009) found that up to 45% of shelter cats were infected with at least one type of endoparasite. This is particularly relevant for FPL patients, most of whom come from environments with poor sanitation and a history of not being vaccinated, which are also high-risk factors for parasitic infestations. Coinfection of FPV with endoparasites can worsen the prognosis and hinder the effectiveness of supportive care therapy. Parasitic infections can exacerbate fluid and nutrient loss, worsen mucosal damage, and impair drug absorption (Robertson *et al*., 2000).

The prognosis generally depends on the severity of the disease, timeliness of diagnosis, and adequacy of the supportive care (Miller *et al*., 2021). Cats with a poor prognosis often experience complications from secondary infections and coinfections (Putri and Wahyuwardani, 2022). Therefore, it is important to conduct a thorough diagnosis, not only to detect the presence of gastrointestinal endoparasites but also to identify the possibility of coinfection.

Bandar Lampung is a region with a rapidly expanding small-animal veterinary sector and a high pet cat density. Based on data from the Lampung Provincial Animal Health and Veterinary Services Office (Disnakkeswan), the cat population has increased year by year over the past five years, mirroring the rise in reported FPL cases. Routine FPL diagnosis in Bandar Lampung has historically relied on clinical signs, hematology, and rapid FPV-Ag tests, with little use of molecular confirmation or systematic screening for gastrointestinal endoparasite coinfection. This diagnostic gap may contribute to misclassification, underestimation of disease severity, and suboptimal treatment strategies. Therefore, integrating molecular FPV detection with systematic parasitological evaluation is essential for accurately characterizing FPL cases in this region.

MATERIALS AND METHODS

Sample collection

Cats were enrolled via purposive sampling, selecting individuals presenting with one or more clinical signs suggestive of FPV infection, including anorexia, lethargy, dehydration, fever (>39.3 °C), vomiting, diarrhea, or halitosis. From an initial pool of 50 cats meeting the clinical case definition, 17 were enrolled based on a sample size calculation for a finite population (Figure 1).

Ethical clearance was obtained from the Animal Research and Ethics Committee, Faculty of Veterinary Medicine, Universitas Gadjah Mada, No: 46/EC-FKH/int./2025.

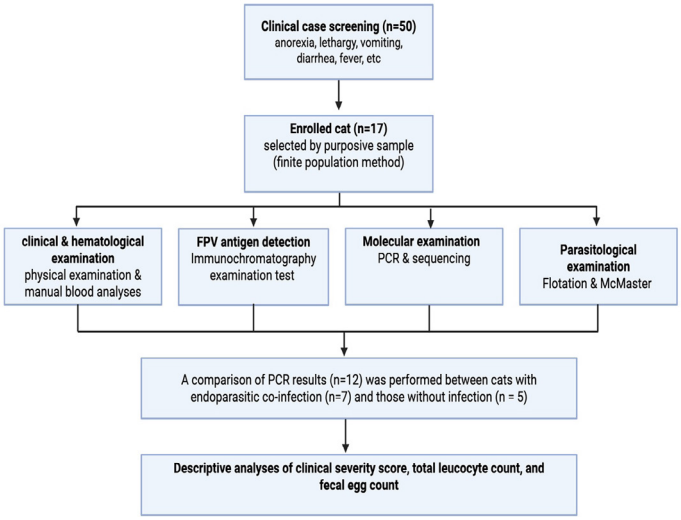

Figure 1: Flowchart of the study design and diagnostic workflow of feline panleukopenia and gastrointestinal endoparasite coinfection.

Clinical and hematological examination

All cats received a standardized clinical assessment comprising history-taking (anamnesis), physical examination, and laboratory tests (Englar, 2017; Indarjulianto *et al*., 2025). The anamnesis included information on vaccination status, deworming history, and environmental conditions (indoor/outdoor). All findings were recorded and analyzed. For hematological evaluation, blood samples were collected from the cephalic or saphenous vein into EDTA tubes and analyzed manually. Hematological analysis was performed manually: total leukocyte counts were obtained using a hemocytometer, and differential counts were evaluated from Wright-Giemsa-stained blood smears. Quality control was ensured through duplicate counts and slide re-evaluation by a second examiner. The results were compared with reference ranges (Tilley and Smith, 2011).

Antigen immunochromatography examination/ antigen rapid test

FPV antigen detection was performed using a commercial immunochromatographic kit (PetX JandG Biotech Ltd., UK). Fresh fecal samples were collected using the sterile applicator provided in the kit and mixed thoroughly with the supplied extraction buffer until a homogenous suspension was obtained. Approximately 3–4 drops of the extracted sample were then dispensed into the sample well of the cassette. The cassette was left undisturbed at room temperature for 5–10 minutes.

The appearance of two distinct colored bands on the control line (C) and test line (T) was interpreted as a positive result, while the presence of only the control line indicated a negative outcome. Tests lacking a visible control line were considered invalid and repeated with a new cassette. All results were recorded immediately after the recommended reading time window to avoid misinterpretation.

Molecular examination

To prevent contamination, separate work areas were maintained for DNA extraction, reagent preparation, amplification, and electrophoresis. Aerosol-resistant tips were used, and each PCR run included positive and negative controls. DNA extraction from blood samples was performed using the gSYNCTM DNA Extraction Kit (Geneaid Biotech Ltd., Taiwan) following the manufacturer's protocol. Amplification of the FPV VP2 gene was conducted using FPV-specific primers designed by Zhang *et al.* (2019), consisting of FPV-F (5′-CATACATGGCAAACAAATAGAGCA-3′) and FPV-R (5′-TGTTTTAAATGGCCCTTGTGTAGA-3′), targeting a 237 bp amplicon. PCR reactions were prepared using KOD Fx Neo polymerase, and amplification was carried out for 30 cycles in a thermal cycler. The cycling conditions were as follows: Initial denaturation at 95 °C for 3 min, followed by 35 cycles of denaturation at 95 °C for 30 s, annealing at 55 °C for 30 s, and extension at 72 °C for 30 s, with a final extension at 72 °C for 5 min. PCR amplicons were separated by electrophoresis on a 2% agarose gel stained with RedSafe, with a DNA ladder and controls included on each gel. Bands were visualized under a UV transilluminator and documented. The two best samples testing positive by PCR were subsequently subjected to bidirectional DNA sequencing at the Integrated Research and Testing Laboratory (LPPT), Universitas Gadjah Mada, Yogyakarta. Sequence identity was confirmed through BLAST analysis using the NCBI database.

Parasite examination

Fresh fecal samples were obtained via rectal swabs using sterile cotton-tipped applicators. Fecal samples were examined microscopically using standard flotation methods, and parasite eggs or proglottids were identified based on morphological keys (Zajac and Conboy, 2012; Bowman, 2014). Quantitative fecal egg counts (EPG) *Toxocara sp*. were determined using the McMaster technique, while *Dipylidium* sp. determined based on presence/absence of proglottid. Fecal samples were homogenized, mixed with flotation solution, and loaded into McMaster counting chambers. Eggs were counted under 100x magnification and multiplied by the standard McMaster factor to obtain EPG values. Fecal egg counts (EPG) were categorized semi-quantitatively as mild (<500 EPG), moderate (500–2000 EPG), or severe (>2000 EPG), based on standard parasitological methods.

RESULTS

Of the 17 cats with clinical suspicion, 12 (70.6%) were PCR-positive for FPV and 5 (29.4%) were PCR-negative (Figure 2). FPV antigen rapid testing detected infection in 10/12 PCR-positive cats (83.3%). Endoparasite coinfection was detected in 7/12 (58.3%) FPV-positive cats, while 5/12 (41.7%) showed no parasites. All PCR-confirmed cats were unvaccinated and had not been dewormed. Clinical severity was classified descriptively based on dehydration status, diarrhea characteristics, and lethargy (Table 1).

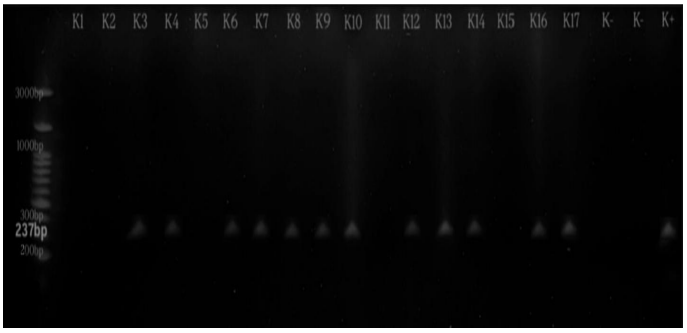

Figure 2: Agarose gel electrophoresis of FPV VP2 gene amplification (237 bp). M: DNA ladder; PC: positive control; NC: negative control; lanes 1–12: clinical samples.

Table 1: Characteristics of cats with PCR-confirmed Feline Panleukopenia (n=12).

Parameter		Number of infected FPL	Percentage (%)
Age (months)	<6	8	66.7
	6-12	4	33.3
Sex	Male	9	75.0
	Female	3	25.0
Leukocyte (cell/mm^3)	<5.500	12	100.0
Antigen rapid test	Positive	10/12	83.3
	Negative	2/12	16.7

Note*: normal leukocyte counts 5,500-19,500 cells/mm^3 (Tilley and Smith, 2011).

Clinical signs observed in the study population are summarized in Table 2. The most prevalent signs were anorexia (100%), lethargy (100%), diarrhea (100%), fever (100%), vomiting (100%), and dehydration (91.7%). Less common findings included halitosis (16.7%), hypersalivation (16.7%), stomatitis (8.3%), nasal discharge (8.3%), and otitis (8.3%). Hematological evaluation revealed leukopenia (<5,500 cells/µL) in all FPV-positive cats.

Gastrointestinal parasite screening was performed in 12 FPV-confirmed cats. Seven cats (58.3%) were positive for endoparasites and five (41.7%) were negative. The parasites detected were *Toxocara* sp. (n = 2), *Dipylidium caninum* (n = 4), and mixed *Toxocara* sp. + *Dipylidium caninum* infection (n= 1). Quantitative McMaster analysis of *Toxocara* sp. showed EPG values ranging from 2600 to 3200 in infected cats. Cats with parasitic coinfection displayed more severe gastrointestinal signs descriptively, including mucoid or hemorrhagic diarrhea, moderate to severe dehydration, anorexia, and profound lethargy (Table 3). The most common parasites identified were *Toxocara* sp. eggs and *Dipylidium caninum* proglottids (Figure 3). Sequencing of two PCR-positive samples produced nucleotide fragments of approximately 180–200 bp. BLAST analysis (Altschul *et al*., 1990) revealed ≥94% identity with FPV sequences to strains from East Asia, especially strain with accession number MN400978 (China) (Figure 4).

Table 2: Clinical symptoms of cats infected with Feline Panleukopenia Virus (FPV) (n=12).

No	Symptoms	Number of infected	Percentage (%)
1	Anorexia	12/12	100
2	Lethargy	12/12	100
3	Fever	12/12	100
4	Vomit	12/12	100
5	Diarrhea	12/12	100
6	Dehydration	11/12	91.7
7	Halitosis	2/12	16.7
8	Hypersalivation	2/12	16.7
9	Stomatitis	1/12	8.3
10	Nasal discharge	1/12	8.3
11	Otitis	1/12	8.3

Table 3: Gastrointestinal endoparasites, Fecal Egg Count (EPG), and Clinical Severity Score in FPV-positive cats (n=12) (EPG terminology clarified; Clinical Severity Score defined as descriptive assessment).

No	Parasite species	n	Mean EPG	Range EPG	% Clinical severity
1	*Toxocara* sp.	2	3025	2850-3200	100
2	*Dipylidium caninum*	4	-	-	100
3	Mixed infection	1	3100	-	100
4	No parasites	5	0	0	0

Note: Clinical severity score was classified based on dehydration (moderate-severe), diarrhea (mucoid or hemorrhagic), and lethargy recorded during physical examination.

DISCUSSION

Feline panleukopenia is a highly contagious and often fatal viral disease characterized by acute systemic illness, severe gastrointestinal involvement, and profound immunosuppression, particularly in young and immunologically naïve cats. In this study, most FPV-positive

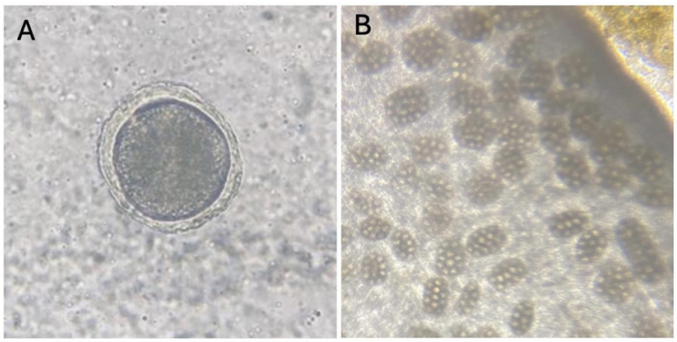

Figure 3: Gastrointestinal endoparasites identified in FPL-positive cats. A) *Toxocara* sp. egg (1,000x magnification). (B) *Dipylidium* sp. (100x magnification).

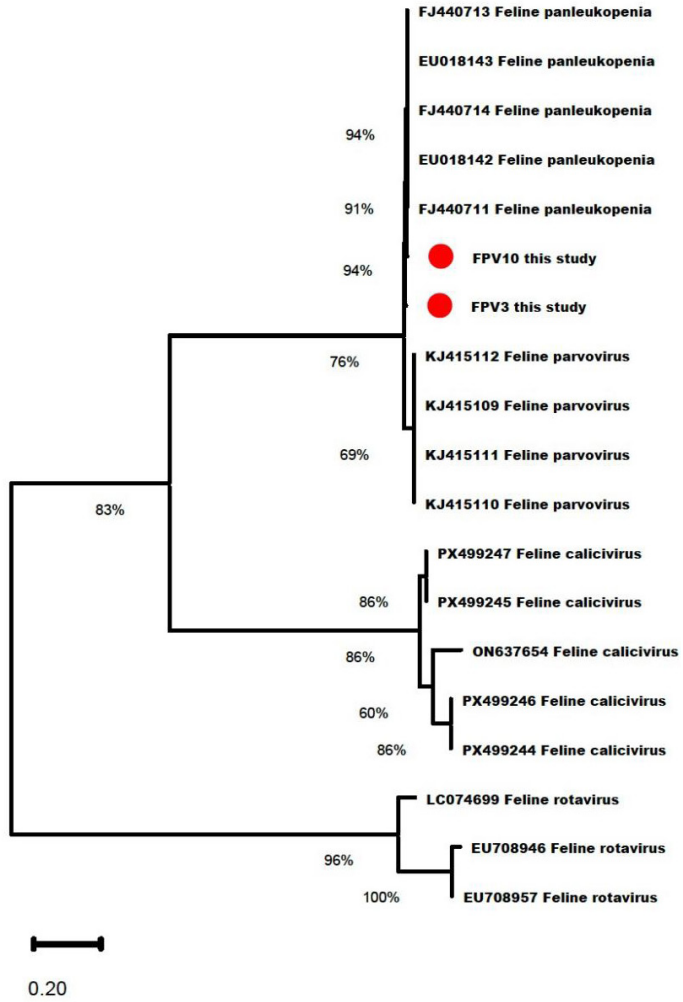

Figure 4: Phylogenetic tree of FPV VP2 gene sequences obtained in this study compared with reference FPV strains from GenBank. The study isolates a cluster closely related to East Asian FPV strains, particularly accession number MN400978 (China). The tree was constructed using the Neighbor-Joining method with 1000 bootstrap replications.

cats were under six months old, consistent with reports identifying kittens as the most susceptible group (Purnamaningsih *et al.*, 2020; Awad *et al.*, 2018; Al-Bayati, 2016; Kruse *et al.*, 2010; Mosallanejad *et al.*, 2009). This vulnerability stems from FPV's tropism for rapidly dividing cells (Sykes, 2014; Imoto *et al.*, 2011; Lian and Chircop, 2016) and the waning of maternally derived antibodies (MDA), creating a period where kittens are neither fully protected nor responsive to vaccination (Day *et al.*, 2016; Baratelli *et al.*, 2020; Claus *et al.*, 2006; Mosallanejad *et al.*, 2009; Jakel *et al.*, 2012). This immunological gap contributes to high morbidity and mortality, with most FPV-related deaths occurring in kittens (Truyen *et al.*, 2009). Additionally, higher total body water in younger animals predisposes them to rapid dehydration and poorer outcomes (Lee and Cohn, 2017).

Although males were slightly more affected than females, the difference was minimal, supporting the notion that sex is not a strong predisposing factor (Islam *et al.*, 2010; Kruse *et al.*, 2010; Awad *et al.*, 2018; Purnamaningsih *et al.*, 2020). Contradictory findings in other studies (Al-Bayati, 2016; Zenad and Radhy, 2020; Isaya *et al.*, 2021) may reflect behavioral differences rather than biological susceptibility. Male cats, especially feral individuals, may be exposed more frequently due to larger roaming ranges and polygynous reproductive behavior (Bengsen *et al.*, 2016; Zhang *et al.*, 2022). Regardless of sex, the durable environmental persistence of FPV facilitates indirect transmission (Rice, 2017; Lamm and Rezabek, 2008; Pandey, 2022), leaving all cats equally vulnerable.

The disease course in this study was characterized by acute illness with prominent gastrointestinal signs, which align with previous reports (Kruse *et al.*, 2010; Sykes, 2014; Moschidou *et al.*, 2011; Awad *et al.*, 2018). The pattern reflects the underlying pathogenesis of FPV, which is characterized by extensive destruction of intestinal crypts, resulting in villus atrophy, malabsorption, increased permeability, and diarrhea (Decaro and Buonavoglia, 2012; Sykes, 2014; Pfankuche *et al.*, 2017; Barrs, 2019; Miller *et al.*, 2021). However, diarrhea could also result from endoparasite infections, including *Giardia* sp., *Toxocara* sp., *Ancylostoma* sp., *Cystoisospora* sp., and *Dipylidium caninum*, all of which can independently impair intestinal function (Gates and Nolan, 2009).

Cats with coinfection appear to exhibit more severe gastrointestinal signs that may be caused by immunosuppression, leading the host to reduce resistance to parasitic infections (Miro *et al.*, 2013). Parasites such as *Toxocara sp.* and *Dipylidium caninum* can intensify the mucosal inflammation, nutrient malabsorption, and systemic immune dysregulation (Bowman *et al.*, 2002; Figueiredo *et al.*, 2010; Traversa *et al.*, 2010). Although statistical comparison was not feasible due to the limited sample size, these findings align with earlier reports indicating that viral–parasitic coinfections worsen clinical outcomes (Mircean *et al.*, 2012).

Leukopenia is the classical hematological profile of FPV infection, presented and mainly as a result of the viral destruction of bone marrow precursor cells (Abd-Eldaim *et al*., 2009; Sykes, 2014; Barrs, 2019; Raj and Haryanto, 2020). The findings align with earlier studies reporting leukopenia in the majority of FPV-positive cats (Mosallanejad *et al*., 2009; Kruse *et al*., 2010; Porporato *et al*., 2018; Purnamaningsih *et al*., 2020; Zenad and Radhy, 2020) that also could be the prognostic indicator since some findings show that it was associated with increased mortality (Kruse *et al*., 2010; Greene, 2012; Barrs, 2019; Miller *et al*., 2021). Parasitic coinfection may also influence leukocyte differentials, as helminth infections are known to induce eosinophilia or reactive neutrophilic responses, potentially modifying the typical hematological profile of FPV infection (Bowman *et al*., 2010; Traversa *et al*., 2010).

Rapid antigen testing demonstrated lower sensitivity than PCR, aligning with previous studies (Mosallanejad *et al*., 2009; Awad *et al*., 2018; Zenad and Radhy, 2020). Sensitivity is affected by shedding dynamics and peaks before clinical signs appear (Sykes, 2014; Miller *et al*., 2021). Low viral load, degraded antigen, improper storage, fecal consistency, and subjective interpretation of faint bands all contribute to false negatives (Esfandiari and Klingeborn, 2000; Decaro *et al*., 2010b; Porporato *et al*., 2018). In contrast, PCR detection using FPV-specific primers (Zhang *et al*., 2019) confirmed infection in a greater number of samples and remains the reference diagnostic method in this case due to the sensitivity (Porporato *et al*., 2018; Awad *et al*., 2018; Raj and Haryanto, 2020; Jacobson *et al*., 2021).

Sequencing of selected PCR-positive samples further validated the assay's specificity. Although phylogenetic analysis was not the primary aim, the high similarity to a Chinese FPV strain suggests genetic relatedness of strains circulating in Bandar Lampung. The global mobility of domestic animals may influence the spread of similar FPV strains across countries (Decaro *et al*., 2010a). Furthermore, the fact that all cats in this study were never dewormed or vaccinated also facilitated the virus's spread, highlighting the urgent need for improved preventive measures. Vaccination programs for owned and community cats, routine deworming protocols integrated into primary veterinary care, structured client education on preventive medicine, and standardized vaccination-deworming policies for individual owners, shelters, and rescue facilities should be more widely promoted and regulated by the government. Establishing these measures could curb the spread of the virus and improve overall feline health in the region.

CONCLUSION

This study confirms that FPL in Bandar Lampung primarily affects young, unvaccinated cats, commonly presenting with leukopenia and severe gastrointestinal signs. PCR demonstrated higher diagnostic sensitivity than antigen rapid testing. Coinfection with gastrointestinal endoparasites was common and descriptively linked to more severe clinical disease. For clinicians, combining PCR with fecal examination should be considered in severe or non-responsive cases, while acknowledging the cost constraints of routine practice.

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NOVELTY STATEMENT

This study provides the first integrated molecular (PCR and sequencing) and parasitological assessment of Feline Panleukopenia in Bandar Lampung, Indonesia, demonstrating a high prevalence of gastrointestinal endoparasite coinfection associated with increased clinical severity. These findings support the integration of molecular diagnostics with routine fecal examination to enhance diagnostic accuracy and patient management.

AUTHORS' CONTRIBUTION

SI and YRN: Conceptualization and supervision. AP, YN, AN, and ADP: Clinical examination. AP, SW, and YRN: Microscopic and molecular analysis. MAA and SW: Hematological analysis. AP and FN: Data analysis and manuscript preparation. All authors reviewed and approved the final manuscript.

Generative AI and AI-assisted technology statement

Artificial intelligence was used solely to improve language and clarity. It was not involved in the study design, data analysis, or interpretation. All scientific content and conclusions are the responsibility of the authors.

Conflict of interest

The authors have declared no conflict of interest.

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