

Divergent Clinical Manifestations in Two Cats with FPV and FCoV Coinfection: A Case Report

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Abstract. Concurrent infection of *Feline panleukopenia virus* (FPV) and *Feline coronavirus* (FCoV) is rarely documented in cats and current knowledge is largely limited to isolated case observations. This report describes the clinical, hematological, and molecular features of two cats with confirmed FPV–FCoV coinfection. Two mixed-breed cats presenting with anorexia, vomiting, and diarrhea were physically examined and screened using a rapid test kit. Hematological parameters were evaluated, and molecular assays (PCR/RT-PCR) were performed to confirm the diagnosis. Cat 1 (an 18-month-old male) presented with acute hyperthermia (40.1°C) and severe leukopenia (WBC $0.16 \times 10^9/L$), while Cat 2 (10-month-old female) presented with hypothermia (36.1°C), moderate anemia (RBC $4.7 \times 10^6/\mu L$; HCT 30%), and leukopenia (WBC $1.5 \times 10^9/L$). Molecular assays confirmed FPV and FCoV coinfection in both cats. Although limited by the very small sample size ($n = 2$), these cases illustrate heterogeneous clinical and hematological manifestations of FPV–FCoV coinfection, ranging from profound leukopenia without anemia to combined anemia and leukopenia. These findings may not intended to be generalized, but emphasize the potential immunosuppressive impact of viral coinfection and highlight the importance of molecular confirmation in multiple feline viral infections. This report contributes the preliminary observational data to the limited literature on FPV–FCoV coinfection in cats.

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1 Introduction

Feline panleukopenia virus (FPV) and *Feline coronavirus* (FCoV) are among the most significant viral pathogens frequently reported affecting domestic cats, leading to substantial morbidity and mortality, particularly in regions with high cat population densities and limited access to preventive care [1]. *Feline panleukopenia virus* belongs to the Parvoviridae family and possesses a marked tropism for rapidly dividing cells, such as in the bone marrow and intestinal crypts, resulting in profound leukopenia, severe gastrointestinal signs, and elevated fatality rates, especially in young and unvaccinated cats [2]. Preventive vaccination has proven highly effective in reducing outbreaks, although unprotected populations remain at high risk.

Feline coronavirus (FCoV) is an enveloped RNA virus classified under the genus *Alphacoronavirus*, within the family Coronaviridae. It is related to other enteric alphacoronaviruses, such as canine coronavirus and transmissible gastroenteritis virus [3]. It is also commonly encountered in multi-cat households, catteries, and shelters, where it is efficiently transmitted via fecal-oral routes. Although most FCoV infections are subclinical or result in transient mild diarrhea, a small proportion of cats experience viral mutation within their macrophages, giving rise to the highly pathogenic *Feline infectious peritonitis virus* (FIPV) and subsequent development of feline infectious peritonitis (FIP), which is challenging to diagnose and invariably fatal without aggressive intervention [4].

While single infections with FPV or FCoV are extensively documented, there is still a lack of reports of coinfection in field cases, although the case might be present. Coinfections could significantly exacerbate immune system compromise, aggravate clinical signs, and create considerable diagnostic challenges due to overlapping hematological and symptomatic profiles [1]. The synergistic effect may lead to more severe disease progression and hinder accurate identification, underscoring the need for molecular diagnostic confirmation.

This study describes two cases of cats with confirmed FPV–FCoV coinfection, providing a detailed analysis of their clinical presentations, hematological findings, and molecular diagnostic evidence to highlight the complexities and diagnostic considerations in managing such coinfections in feline practice. The results and discussion hopefully support the United Nations Sustainable Development Goal 3 (Good Health and Well-being), particularly its target to strengthen the capacity for early warning, risk reduction, and management of global health risks. By improving understanding of feline viral coinfections and emphasizing the importance of accurate molecular diagnostics, this work contributes to the One Health framework, which integrates animal, public, and environmental health for disease prevention and control.

2 Materials and Methods

2.1 Subjects

This study includes two cases from two patients who were registered and clinically managed at the veterinary clinic. Case 1: An 18-month-old mixed-breed male cat presented with anorexia, vomiting, and watery bloody diarrhea. Clinical examination revealed fever (40.1°C), lethargy, moderate dehydration (3/5), heart rate 80 bpm, and respiratory rate 68 bpm. The general condition of the cat was poor, with depressed mentation. Case 2: A 10-month-old mixed-breed female cat presented with one week of anorexia, vomiting, and watery diarrhea. The cat was hypothermic (36.1°C), with weak body condition (2/5), tachycardia (160 bpm), tachypnea (40 bpm), and clinical dehydration.

2.2 Methods

Physical and Hematological Examination

Physical examination included assessment of general demeanor, body condition score, rectal temperature, heart rate, respiratory rate, mucous membrane color, and capillary refill time [5]. Hydration status was evaluated based on skin turgor, mucous membrane moisture, and eye position. Gastrointestinal signs, such as vomiting and diarrhea, were recorded, along with appetite and behavioral changes. Body temperature was measured using a digital thermometer, while heart and respiratory rates were determined by auscultation and visual chest movement counts, respectively. Hematological parameters of red blood cells (RBC), hemoglobin (HGB), hematocrit (HCT), white blood cells (WBC), mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) were analyzed by using a hematology analyzer.

Molecular Diagnostic for FPV

Whole blood samples were collected from both cats into EDTA tubes and subjected to DNA extraction using a commercial kit (Zymo Research, USA) according to the manufacturer's protocol. Conventional polymerase chain reaction (PCR) targeting the VP2 gene of FPV was performed using specific primers [6]. Amplification was carried out in a 25 μ L reaction mixture containing extracted DNA, primers, Taq DNA polymerase, and dNTPs. PCR cycling conditions included an initial denaturation at 94°C for 5 minutes, followed by 35 cycles of denaturation (94°C) for 45 seconds, annealing (51°C) for 45 seconds, and extension (72°C) for 30 seconds, and a final elongation step at 72°C for 10 minutes. Amplicons were analyzed by agarose gel electrophoresis (1.5% agarose gel stained with DNA gel stain) and visualized under UV illumination. Samples showing a 237-bp band were considered positive for FPV DNA[7].

Molecular Diagnostic for FCoV

Detection of FCoV RNA was performed using a nested reverse transcriptase polymerase chain reaction (RT-nPCR) method as previously described [8]. Viral RNA was extracted from blood samples using a commercial kit (Zymo Research, USA). The RT-nPCR assay targeted the highly conserved 3'-untranslated region (3'-UTR) of the FCoV genome to ensure broad detection of both enteric and FIP-associated strains. Reverse transcription was carried out using Moloney murine leukemia virus reverse transcriptase, followed by the first round of PCR amplification with primer pairs P205 (sense) and P211 (antisense). The nested PCR employed primers P276 (sense) and P204 (antisense) to amplify a 177-bp fragment specific to the FCoV genome. The PCR products were visualized by electrophoresis on 2% agarose gel. Samples showing a 177-bp band were considered positive for FCoV RNA.

3 Results

Both cats presented with some gastrointestinal signs, including anorexia, vomiting, and diarrhea, but differed in systemic clinical parameters. Cat 1 exhibited fever, moderate dehydration, depressed mentation, and hemorrhagic diarrhea, with an overall poor general condition. In contrast, Cat 2 showed hypothermia, tachycardia, tachypnea, and severe dehydration.

The hematological findings of both cats are summarized in Table 1. Cat 1 showed red blood cell (RBC), hemoglobin (HGB), and hematocrit (HCT) within the normal reference range, as well as MCV, MCH, and MCHC. However, the total white blood cell (WBC) count was markedly decreased. Meanwhile, Cat 2 exhibited lower RBC, HGB, and HCT values compared with Cat 1. The MCV and MCH were higher than the reference values, while MCHC remained within normal limits. The WBC count in Cat 2 was also below the reference interval but higher than that of Cat 1.

Table 1. Hematology results of cats

Parameters	Unit	References [9]	Cat 1	Cat 2
RBC	$10^6/\text{mm}^3$	5-10	9,33	4,7
HGB	g/dL	8-15	13,6	10,2
HCT	%	24-45	40,7	30,0
MCV	fL	41-54	43,6	63,8
MCH	pg	13,5-17,5	14,5	21,7
MCHC	g/dL	31-36	33,3	34,0
WBC	$10^3/\text{mm}^3$	5,5-19,5	0,16	1,5

PCR assays targeting the VP2 gene successfully amplified FPV DNA from both cats, producing the expected amplicon size of approximately 237 bp (Fig. 1a). Similarly, RT-PCR assays targeting the 3'-UTR region of the FCoV genome generated the expected 177 bp fragments in both cases (Fig. 1b). Based on these findings, both cats were molecularly confirmed to be coinfecting with FPV and FCoV.

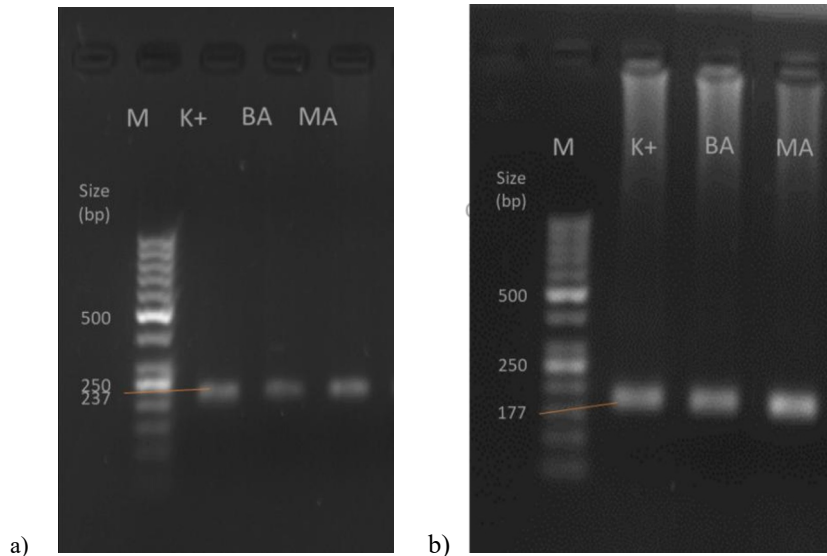


Fig. 1. The final visualization of PCR assays for FPV showed a band at 237 bp (a), and FCoV showed a band at 177 bp (b). Note M: marker; K+: Positive control; BA: Cat 1; MA: Cat 2.

4 Discussion

Infectious diseases remain a major cause of morbidity and mortality in cats worldwide, particularly in environments with high population density, inadequate vaccination coverage, and frequent exposure to infectious agents [4,10]. The complexity of feline infectious diseases is further increased by the potential for concurrent infections. Previous studies revealed significant differences in viral abundance and diversity in the enteric virome of FPV-infected cats compared with clinically healthy cats. There were several viruses known to be highly prevalent as co-pathogens with FPV, i.e, feline astroviruses and feline coronavirus [1]. These findings suggest that FPV infection may occur within a broader context of enteric viral dysbiosis, potentially predisposing affected cats to concurrent or secondary viral infections

This report provides molecular confirmation of two cats with feline panleukopenia virus (FPV) and feline coronavirus (FCoV) coinfection as a strong evidence of concurrent viral infection under field conditions. Both cats were initially suspected of feline panleukopenia based on their clinical signs and hematological profiles. The likelihood of FCoV infection in Cat 1 was initially considered low; however, the veterinarian recommended a multiple rapid test kit, which subsequently confirmed FCoV coinfection. Meanwhile, a slight abdominal distension palpated during examination was found in Cat 2, and it was initially presumed to be due to fat accumulation. However further investigation by the veterinarian revealed ascitic fluid, leading to the diagnosis of concurrent FCoV infection.

From a mechanistic perspective, FPV and FCoV differ markedly in their cellular tropism and immunopathological effects, which may help explain the divergent clinical and hematological outcomes observed in these two cases. FPV primarily targets rapidly dividing cells, including intestinal crypt epithelial cells and bone marrow progenitors, leading to acute gastrointestinal damage and profound leukopenia [2,10]. In contrast, FCoV preferentially infects monocytes and macrophages, where persistent viral replication and immune activation may occur, particularly in cases associated with systemic involvement [4]. Coinfection with these two viruses therefore represents an interaction between acute cytopathic injury (FPV) and immune-mediated or chronic inflammatory processes (FCoV).

The severe leukopenia observed in both cats aligns with FPV-induced bone marrow suppression. The preservation of red cell indices in Cat 1 indicates an acute disease course with limited marrow involvement. In contrast, the concurrent anemia and leukopenia in Cat 2 may reflect a more prolonged or multifactorial pathological process[2,11]. A plausible hypothesis is that pre-existing or concurrent FCoV infection in Cat 2 contributed to chronic immune activation, inflammatory cytokine release, or altered hematopoiesis, thereby exacerbating erythroid suppression or peripheral red cell loss. Although this mechanism cannot be confirmed without cytokine profiling or bone marrow evaluation, it is consistent with the established immunomodulatory effects of FCoV infection [12].

Differences in thermoregulatory responses between the two cats further support the likelihood of divergent systemic inflammatory states. Hyperthermia in Cat 1 is characteristic of acute viral infection with preserved inflammatory responsiveness. In contrast, hypothermia in Cat 2 may indicate systemic compromise, metabolic exhaustion, or dysregulated inflammatory signaling. In other species, hypothermia during severe infection has been linked to advanced disease stages or impaired host responses, suggesting that Cat 2 may have been evaluated at a later or more severe phase of illness. Under viral coinfection, such variability in host response may be amplified, resulting in heterogeneous clinical presentations even among cats infected with the same viral agents [13].

FPV-induced immunosuppression may facilitate FCoV persistence or replication by reducing effective antiviral immune surveillance, potentially through suppression of key cytokines such as IFN-gamma and IL-12 [14]. Conversely, macrophage-associated FCoV infection may alter cytokine environments, for example through IL-6-driven macrophage activation, which could further influence FPV pathogenesis or tissue tropism [15]. Although

these bidirectional interactions remain speculative, they highlight biologically plausible pathways through which viral coinfection could modulate disease expression.

These cases demonstrate that FPV–FCoV coinfection does not result in a uniform clinical phenotype, but instead produces a spectrum of outcomes shaped by viral interactions, host immune status, and disease timing. However, this study has an important limitations since the sample size was extremely small (n=2), so that this report served as a descriptive case-based observation rather than an analytical or inferential study. As such, no statistical analysis was performed, and the findings cannot be generalized to the broader feline population or used to establish causal relationships between FPV–FCoV coinfection and specific clinical or hematological outcomes. The purpose of this report is therefore not to draw definitive conclusions, but to document rare, molecularly confirmed cases and to highlight diagnostic challenges that may warrant further investigation in larger, well-designed studies.

Overall, these cases highlight how FPV–FCoV coinfection may complicate clinical interpretation and patient management. The immunosuppressive effects of FPV could potentially facilitate FCoV replication, worsening disease severity. Even though the sample size is limited, the observations indicate the importance of integrating clinical, hematological, and molecular findings to achieve an accurate diagnosis and to better understand the complexity of feline viral infections. The findings underscore the importance of confirmatory diagnostics in disease management, particularly in cases with atypical presentations. Although limited in number, these cases add valuable insights into feline viral coinfections and emphasize the need for further research to better understand their clinical significance and impact on feline health

In addition, these findings suggest the potential benefit of developing a multiplex diagnostic assay capable of detecting multiple feline viral pathogens with overlapping clinical signs. This approach would improve diagnostic efficiency, guide timely and targeted treatment, and reduce misdiagnosis in clinical practice. This recommendation aligns with the United Nations Sustainable Development Goal 3 (Good Health and Well-being), which emphasizes strengthening diagnostic capacity and early disease detection as part of the global effort to enhance animal and public health within the One Health framework.

5 Conclusion

This report documents two cases of molecularly confirmed coinfection with feline panleukopenia virus and feline coronavirus, each presenting with distinct clinical and hematological features. The first cat exhibited classical signs of panleukopenia, including severe leukopenia and fever, whereas the second cat showed a combination of leukopenia and anemia accompanied by hypothermia and prolonged anorexia. These contrasting manifestations highlight the variability of disease expression in coinfecting cats and suggest that the interaction between FPV and FCoV may result in diverse clinical outcomes.

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