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Review Report

Epidemiology, Pathogenesis, Diagnosis, Clinical Management, and Prevention of Feline Panleukopenia Virus in Cats

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Feline panleukopenia virus (FPV) is a highly contagious parvovirus that poses a serious threat to both domestic and wild felids. Primarily targeting rapidly dividing cells, FPV leads to marked leukopenia, severe enteritis, and cerebellar hypoplasia in perinatal infections. Transmission occurs mainly via the fecal–oral route, while contaminated objects (fomites) contribute significantly to shelter outbreaks. Clinical suspicion, supported by hematology, biochemistry, antigen testing, polymerase chain reaction, and histopathology, forms the basis of diagnosis. As no antiviral therapy exists, management remains supportive, centered on fluid resuscitation, antimicrobial and antiemetic therapy, nutritional support, and immunomodulation. Prevention relies heavily on timely vaccination with modified live vaccines, reinforced by strict biosecurity and sanitation measures. A coordinated approach that integrates early detection, intensive supportive care, and effective prevention is critical to mitigating the high morbidity and mortality associated with feline panleukopenia.

Keywords: Antivirals, biosecurity, diagnostics, feline panleukopenia, vaccination

INTRODUCTION

Feline panleukopenia virus is a highly contagious and often fatal parvoviral infection that primarily affects domestic and wild felids (Pandey, 2022). Characterized by acute onset of gastrointestinal, hematological, and immunological disturbances, FPV poses a significant threat to feline health worldwide, particularly in unvaccinated populations and densely populated environments such as shelters and catteries (Pacini et al., 2023). As a non-enveloped, single-stranded DNA virus of the Parvoviridae family, FPV exhibits remarkable environmental resistance and a high transmission potential, contributing to frequent outbreaks and substantial morbidity and mortality, especially among kittens (Truyen et al., 2009).

Despite the availability of effective vaccines, feline panleukopenia (FPL) continues to re-emerge in various regions, often due to gaps in immunization coverage, waning maternal immunity, or viral persistence in the environment. The clinical presentation of FPL can be variable, ranging from subclinical infections to severe hemorrhagic gastroenteritis, profound leukopenia, and septic shock (Nohrström et al., 2011). Timely diagnosis and supportive treatment are essential for improving clinical outcomes, but the nonspecific nature of early clinical signs often delays detection.

This integrative review aims to comprehensively synthesize current knowledge on FPV, including its epidemiological trends, molecular pathogenesis, diagnostic modalities, clinical manifestations, and available therapeutic and preventive strategies. A holistic understanding of these aspects is critical for veterinarians, researchers, and public health professionals in improving disease control, guiding evidence-based management, and informing future research directions in feline infectious diseases.

EPIDEMIOLOGY

The epidemiology of FPV encompasses its geographical distribution, risk factors, and seasonal patterns. This virus causes panleukopenia and enteritis in both domestic and wild cats worldwide, with prevalence varying between regions (Pacini et al., 2023). A 2025 meta-analysis reported a global prevalence of 50.30% in domestic cats, with the highest in Germany (15.81%) and the lowest in Korea (2%) (Alessa et al., 2025). A regional survey in Bangladesh reported 40.45% positive cases with a mortality rate of 23.66% (Hossen et al., 2024), whereas clinical data from Vietnam indicated 14.09% positive cases among cats with gastrointestinal disorders (Nguyen and Nguyen, 2025). In Indonesia, a study conducted in Yogyakarta found a prevalence of 24.9%, with a higher risk of infection in cats under one year of age and in unvaccinated individuals (Wafa, 2022).

Feline panleukopenia virus most frequently infects young cats, with a median age of infection of 4 months, and 56.7% of cases occurring in cats under 6 months of age (Litster and Benjanirut, 2014; Ramadhani et al., 2024). Nevertheless, infections also occur in adult cats, including 10.7% of cases in individuals over 5 years old (Truyen et al., 2009). This challenges the assumption that older cats are always protected due to vaccination or prior subclinical infection. In vaccinated cat populations, clinical cases generally occur in individuals that did not receive a booster after 12 weeks of age, although kitten fatalities have also been reported in fully vaccinated households, possibly due to high environmental viral loads (Gore et al., 2006).

Outbreaks are often seasonal, with an increase in cases during summer or breeding seasons, when kitten numbers rise and contact between individuals becomes more frequent. A study by Jenkins et al. (2020) demonstrated that FPV incidence increased by up to 35% during warmer months, particularly in unvaccinated populations. Surges in cases are also common following natural disasters or social disruptions that increase the number of stray cats. For example, after the major floods in Dhaka in 2022, the number of FPV cases doubled within

three weeks, likely due to the growth of the stray cat population and reduced access to veterinary healthcare (Mamun et al., 2023).

PATHOGENESIS OF FELINE PANLEUKOPENIA VIRUS INFECTION

Feline panleukopenia virus is an autonomous single-stranded DNA parvovirus that is highly dependent on the mitotic activity of host cells for replication. Viral replication can only occur when the host cell is in the S-phase of the cell cycle; therefore, FPV exhibits a strong tropism for tissues with high mitotic activity, such as lymphoid tissue, intestinal crypt epithelium, and bone marrow (Stuetzer and Hartmann, 2014). Consequently, infection is most frequently observed in kittens aged 3 to 6 months, when cell division remains highly active (Kruse et al., 2010).

Transmission of FPV typically occurs via the fecal–oral route, with indirect transmission through contaminated fomites representing the primary route of spread (Pandey, 2022). Following entry into the host, the virus initially replicates in the lymphoid tissue of the oropharynx before disseminating hematogenously via cell-free viremia to multiple organs. FPV utilizes the transferrin receptor for cell entry, similar to canine parvovirus (CPV), and selectively replicates in actively dividing tissues (Mannioui et al., 2009). After systemic dissemination, FPV infection causes damage to various target organs. In lymphoid tissue, infection induces necrosis and lymphocytolysis, resulting in lymphopenia and functional immunosuppression. In the bone marrow, the virus infects and destroys early hematopoietic progenitor cells, leading to severe panleukopenia, particularly neutropenia (Laming et al., 2012). Leukopenia is further exacerbated by sequestration of neutrophils within damaged intestinal mucosa. In the gastrointestinal tract, FPV infects crypt epithelial cells, causing villous atrophy, increased mucosal permeability, impaired epithelial regeneration, and reduced nutrient absorption (Jung et al., 2016).

The severity of FPV infection varies widely depending on host age, immune status, and concurrent infections. Subclinical infection is common in immunocompetent adult cats. However, coinfection with other pathogens such as feline enteric coronavirus, *Clostridium piliforme*, *Salmonella* spp., feline leukemia virus, and astrovirus can accelerate epithelial cell turnover and enhance viral replication, thereby exacerbating tissue damage and clinical severity (Wang et al., 2025).

Feline penleukopenia virus infection during pregnancy can have significant consequences. Infection in early gestation may lead to abortion, fetal resorption, congenital anomalies, or infertility, although pregnant queens are often asymptomatic. When infection occurs during late gestation or in neonates under one week of age, the virus targets the cerebellum, particularly Purkinje cells and precursor granule cells in the external granular layer, resulting in cerebellar hypoplasia (Poncelet et al., 2013). This condition is characterized by non-progressive neurological signs such as ataxia, intention tremors, and incoordination, which typically become apparent when kittens begin walking at 2–3 weeks of age. Despite motor deficits, cats with cerebellar hypoplasia retain normal consciousness and appetite, and can live as suitable companion animals.

CLINICAL PRESENTATION OF FELINE PANLEUKOPENIA

The clinical manifestations of FPL are highly variable, depending on the severity of infection, immune status, and age of the affected cat. The disease may present as a mild illness or progress to severe, life-threatening systemic conditions. Common signs include anorexia, fever, vomiting, lethargy, and bloody diarrhea (Ramadhani et al., 2024), almost always accompanied by dehydration ranging from 4% to 12% in severity (Sarmadana et al., 2023). Additional findings may include halitosis, rhinitis, hypersalivation, and, less frequently, oral and ocular abnormalities such as stomatitis, otitis, or mucosal lesions.

In advanced stages, septicemia may lead to circulatory collapse, characterized by weak pulse, pale mucous membranes, and shallow respiration (Litster and Benjanirut, 2014). Respiratory involvement—such as oculonasal discharge, tachypnea, pleural effusion, or lateral recumbency—is typically secondary to bacterial coinfection.

Gastrointestinal signs such as vomiting and bloody diarrhea, reported in 85% and 70% of cases respectively, are consistent with FPV-induced destruction of intestinal crypt epithelial cells. This leads to villous atrophy, malabsorption, and increased intestinal permeability (Kipar et al., 1998; Pirarat et al., 2002). Although these signs may strongly suggest FPL, definitive diagnosis requires laboratory confirmation due to clinical overlap with other enteric diseases such as toxoplasmosis, bacterial sepsis, or intoxication.

Although relatively uncommon, neurological signs are clinically significant. These include ataxia, tremors, seizures, and hypersalivation, most often associated with cerebellar hypoplasia resulting from intrauterine or early neonatal infection (Pfankuche et al., 2018). Such infections interfere with cerebellar neuroblast development, particularly in Purkinje cells, leading to non-progressive motor deficits such as incoordination and intention tremors.

DIAGNOSIS OF FELINE PANLEUKOPENIA

Given the nonspecific nature of its clinical manifestations, further verification through laboratory testing is essential. The most consistent hematologic finding in FPL is leukopenia, particularly neutropenia and lymphopenia. Total leukocyte counts may drop to as low as 50 cells/μL, and toxic band neutrophils may be observed. However, not all infected cats exhibit leukopenia; in one study, only 65% of 187 affected cats had markedly low white blood cell counts (Gülersoy et al., 2023). Severe leukopenia, when present, can be pathognomonic and is associated with a poor prognosis (Irawan et al., 2024). Other hematologic abnormalities may include mild anemia and thrombocytopenia, the latter potentially resulting from bone marrow suppression or disseminated intravascular coagulation (Conner et al., 2015). Hematology is also valuable for monitoring disease progression, with leukocytosis and lymphocytosis often observed during recovery.

Biochemical analysis may reveal hypoalbuminemia, hypoglobulinemia, hypocholesterolemia, and electrolyte disturbances such as hyponatremia or hypernatremia, hypochloremia, and hyperkalemia (Maharani et al., 2025). In severe cases, elevated liver enzymes (AST, ALT), azotemia, hyperbilirubinemia, and glucose imbalances (hypo- or hyperglycemia) may occur (Kolomak, 2023). These findings reflect the multisystemic impact of the virus as well as the consequences of dehydration and sepsis.

Imaging modalities such as abdominal radiography may reveal intestinal dilation, fluid and gas accumulation, and reduced serosal detail, consistent with enteritis. In cases with neurological involvement due to intrauterine or neonatal infection, magnetic resonance imaging (MRI) can demonstrate cerebellar hypoplasia, cerebellar agenesis, or other congenital central nervous system abnormalities, including hydrocephalus, porencephaly, and hydranencephaly (Fortin et al., 2024).

Detection of FPV antigen in feces or rectal swabs can be performed using various commercial diagnostic kits initially developed for CPV. While these tests provide rapid and convenient results, their sensitivity and specificity can vary considerably depending on the kit used and the stage of infection, as viral shedding in feces is typically transient. False-negative results are relatively common, particularly when testing outside the peak shedding period. Conversely, false positives are rare, meaning that a positive result in a clinically affected cat strongly supports a diagnosis of FPL. One of the most commonly used kits is the SNAP Parvo test (IDEXX Laboratories), which is known for high specificity; in one study, 54 of 55 positive results

were confirmed by polymerase chain reaction (PCR) (Jacobson et al., 2021). Another study involving 52 diarrheic cats and 148 healthy cats reported that five different diagnostic kits had sensitivities ranging from 50% to 80% and specificities between 94% and 100% when compared with fecal electron microscopy (Mekaru et al., 2007). However, this study included only 10 FPV-positive cats by electron microscopy, highlighting the need for further research using larger sample sizes, ideally combining real-time PCR and electron microscopy as the gold standard. False positives due to recent vaccination with a modified-live virus are rare, although the likelihood may vary by test type. Among several kits compared (SNAP Parvo, AGEN CPV, and Witness CPV), SNAP Parvo had the lowest risk of post-vaccination false positives (Walter-Weingärtner et al., 2021).

Polymerase chain reaction remains the gold standard for definitive diagnosis of FPL. PCR can detect viral genetic material (DNA) with very high sensitivity and specificity, even in samples containing low viral loads. Real-time PCR not only detects the presence of the virus but can also quantify viral load, which may correlate with disease severity.

For outbreak investigations or vaccination monitoring, serological testing can be performed. However, interpretation is complicated by prior vaccination or natural exposure (Takla et al., 2012). Although the hemagglutination inhibition assay is the reference standard for antibody detection, its application for individual diagnosis is limited. Point-of-care serological kits, such as the ImmunoComb Feline VacciCheck Test Kit, have demonstrated low sensitivity (28–49%) but high specificity, making them more suitable for assessing population-level immunity rather than diagnosing individual cases (Mende et al., 2014).

TREATMENT OF FELINE PANLEUKOPENIA

Treatment of FPL is primarily supportive and symptomatic, as no specific antiviral therapy is currently available. Nevertheless, several studies have explored promising therapeutic candidates. For instance, the use of filgrastim (granulocyte colony-stimulating factor, G-CSF)—originally developed for human medicine—has been clinically evaluated in naturally infected cats, showing restored hematopoietic immunity and achieving a 100% survival rate in a recent study (Klar, 2024). Additionally, recombinant feline interferon-ω (rFeIFN-ω) has been investigated as an antiviral therapy due to its ability to inhibit FPV replication in vitro (de Mari et al., 2004; Gil et al., 2014). However, its clinical efficacy in significantly improving outcomes in infected cats remains inconclusive.

The primary objectives of management are to correct dehydration, prevent secondary infections, restore electrolyte balance, and support immune function and hematopoietic regeneration. Fluid therapy is a top priority, as most cases present with moderate to severe dehydration due to vomiting and diarrhea. Intravenous fluids such as lactated Ringer's solution or 0.9% NaCl are administered at approximately 30 mL/kg body weight/day, adjusted to the degree of dehydration and the patient's overall condition (Hermawan et al., 2023). The addition of 5% dextrose is necessary to address hypoglycemia—particularly in kittens with low energy reserves—with regular blood glucose monitoring during therapy (Priambudi, 2022).

Disruption of the intestinal barrier during FPV infection permits translocation of enteric bacteria into the bloodstream, increasing the risk of bacteremia and sepsis, especially in neutropenic and immunosuppressed patients. Sepsis prevention is therefore crucial, and broad-spectrum antibiotics effective against Gram-negative and anaerobic bacteria are strongly recommended. Agents such as sulfa-trimet and amoxilin clav. Combination therapy involving fluoroquinolones and penicillins—such as enrofloxacin with amoxicillin—is also effective. Other options include amoxicillin/clavulanate, piperacillin with

aminoglycosides, fluoroquinolones, cephalosporins, or piperacillin/tazobactam (Rice, 2017; Porporato et al., 2018; De Marchi et al., 2024). Antibiotics should preferably be administered intravenously to maximize efficacy, with careful consideration of potential side effects.

Clinical signs such as vomiting can be managed with antiemetics including ondansetron, maropitant, or metoclopramide. Fever and systemic inflammation may be controlled with NSAIDs such as tolfenamic acid or corticosteroids such as dexamethasone or methylprednisolone, although their use requires caution due to the risk of immunosuppression (Salih, 2017). Metabolic and tissue regeneration support can be provided via vitamin supplementation, including Introvit-B complex, vitamin B12, or amino acid and vitamin formulations such as Aminoplex and Seloxy AA (Chudow and Adams, 2020). Probiotics can also be used to support treatment. A recent study by Mangiaterra et al. (2024) found that multi-strain probiotics eliminated fecal feline coronavirus (FCoV) viral load in 17 of 25 naturally infected cats after 60 days of treatment. Specific immunoglobulin therapy (IgG, IgM, IgA) has also shown promise, with survival rates of 85% compared to 7% in conventional supportive care, acting via viral neutralization, enhanced phagocytosis, and activation of the cellular immune response (Litster and Benjanirut, 2014).

In cats with hypoproteinemia, plasma or whole blood transfusion may be required to restore oncotic pressure. Plasma transfusion combined with heparin can aid in controlling disseminated intravascular coagulation by supplementing antithrombin III and other essential plasma proteins (de Jonge et al., 1998). Parenteral nutrition should be considered in patients with anorexia, severe vomiting, profuse diarrhea, or persistent hypoproteinemia, ideally administered via a central venous catheter placed in the jugular vein. As an example, in one reported case, supportive therapy consisted of intravenous lactated Ringer's solution at 30 mL/kg body weight/day, hematopoietin (hematodin) 0.1 mL/kg body weight, vitamins and amino acids (Aminoplex) 0.1 mL/kg body weight intramuscularly twice daily, and two antibiotics—enrofloxacin 5 mg/kg body weight/day subcutaneously and amoxicillin 10 mg/kg body weight every two days intramuscularly (Nareswari et al., 2024).

Furthermore, a diet containing high-quality animal protein, moderate fat, and easily digestible carbohydrates is highly recommended. Protein sources such as boiled chicken, turkey, or commercial therapeutic recovery diets support tissue regeneration and immune function. The fat content provides adequate energy without overburdening the digestive system, while easily digestible complex carbohydrates help maintain stable blood glucose levels. The types of therapy commonly used for feline panleukopenia are presented in Table 1.

PREVENTION AND CONTROL OF FELINE PANLEUKOPENIA

The prevention of feline panleukopenia focuses on individual protection through vaccination, environmental management, and strict biosecurity measures, especially in shelters or cattery settings. According to the WSAVA Vaccination Guidelines (2024), vaccination against feline calicivirus (FCV) is classified as a *core vaccine* and is therefore recommended for all cats, unless local regulations specify otherwise. The primary vaccination series should begin at 6–8 weeks of age using a combination vaccine containing FHV-1, FCV, and FPV based on a modified live virus (MLV), followed by boosters every 3–4 weeks until the final dose is administered at or after 16 weeks of age. If vaccination is initiated in cats older than 16 weeks, two doses of MLV administered 3–4 weeks apart are recommended, although a single dose may be sufficient to induce immunity. Following completion of the primary series, a booster should be administered at around 6 months of age or no

later than one year after the last dose given at 16 weeks, to ensure optimal protection.

Table 1. The types of therapy commonly used for feline panleukopenia

Antibiotic	**Doses**
Cefazolin	20–30 mg/kg IV/IM, every 8 hours
Marbofloxacin	2.75–5.5 mg/kg PO, every 24 hours
Sulfadoksin–Trimetoprim (potentiated sulfonamides)	15 mg/kg PO, every 12 hours
Enrofloxacin	5 mg/kg PO, every 24 hours
Amoxicillin	11–22 mg/kg PO, every 8-12 hours
Amoxicillin–Clavulanate	12.5 mg/kg cat PO, every 12 hours
Piperacillin–Tazobactam	50 mg/kg IV, every 6 hours
Gentamicin (aminoglycoside)	5–8 mg/kg IV/IM/SC every 24 hours
Fluoroquinolone (Enrofloxacin)	2.5 mg/kg every 12 hours, or 5 mg/kg every 24 hours (maximum to avoid retinal risk)
Antiemetic	
Maropitant	1 mg/kg SC/IV, every 24 hours (up to 5 days)
Ondansetron	
Metoclopramide	0.2–0.5 mg/kg IV/IM/SC, every 6–8 hours, or CRI 1–2 mg/kg/day
NSAID (Non-Steroidal Anti-Inflammatory Drugs)	
Tolfenamic acid	4 mg/kg PO/SC, every 24 hours
Corticosteroid	
Dexamethasone	0.1–0.3 mg/kg IV/IM, every 24 hours
Methylprednisolone	1–2 mg/kg IV/IM, every 24 hours

IV: Intravenous; IM: Intramuscular;SC: Subcutaneous; PO: Per os; CRI:Constant Rate Infusion
(Rice, 2017; Plumb 2024; MSD, 2025; Ceftiofur, 2025)

For adult cats with low risk of exposure, such as those kept strictly indoors, revaccination every three years is considered adequate to maintain long-term immunity. Vaccination protocols should also take into account specific clinical conditions, for instance avoiding the use of FPV MLV vaccines in pregnant queens or kittens under one month of age, except under high-risk situations such as in shelters. Proper implementation of these vaccination strategies plays a crucial role in reducing the burden of FCV infection in domestic cat populations.

In contrast, cats living in shelters represent a high-risk population due to crowding, frequent turnover, and increased exposure to infectious agents. In such settings, early immunization is essential to provide rapid protection against FCV and other core pathogens. WSAVA Vaccination Guidelines (2024) recommend administering the first dose as early as 4–6 weeks of age, with subsequent boosters every 2–3 weeks until at least 16 weeks of age. In shelters, vaccination upon entry is strongly advised, even for kittens younger than 16 weeks, because maternally derived antibodies (MDA) may interfere with vaccine response, but partial protection is still beneficial in reducing the severity of disease and viral shedding. The use of MLV intranasal vaccines may be considered in this context, as they can stimulate a faster onset of mucosal immunity and may help reduce viral transmission within the population. Strict adherence to vaccination protocols in shelters is therefore critical to mitigate outbreaks and control FCV circulation in high-density feline populations (Rehme et al., 2022; Day et al. 2024). However, their use is

contraindicated in pregnant queens, severely ill cats, or kittens younger than 4 weeks. In such cases, passive preventive alternatives may be considered, such as administration of hyperimmune serum containing high levels of antibodies from donor cats that have been vaccinated or previously exposed to the virus. This serum can be administered subcutaneously or intraperitoneally at a volume of 2 mL and is effective for providing protection for 2–4 weeks if given prior to the onset of clinical signs (Stuetzer and Hartmann, 2014; Friedl et al., 2014). However, serum administration may interfere with the response to active vaccination, and thus vaccination should be delayed for at least three weeks following serum administration. Repeated use of serum is not recommended due to the risk of hypersensitivity reactions (Wiktor et al., 1971).

Feline panleukopenia prevention also involves strict environmental control. Feline panleukopenia virus is highly resistant in the environment and can persist for months on contaminated surfaces. Therefore, thorough disinfection is essential, using agents effective against non-enveloped viruses such as sodium hypochlorite (bleach), accelerated hydrogen peroxide, or potassium peroxymonosulfate (Lin et al., 2020). In shelter settings, additional measures such as isolating cats with gastrointestinal symptoms, separating healthy and sick cats, and implementing quarantine protocols for new arrivals are critical to minimize disease spread.

Furthermore, introducing new kittens into environments previously exposed to FPV is not recommended unless the kittens have completed their vaccination schedule. A comprehensive strategy combining vaccination, biosecurity, environmental hygiene, and owner education can significantly reduce the incidence of FPL in both household and shelter environments.

CONCLUSION

Feline panleukopenia is a severe, highly contagious disease in cats, mainly affecting young and unvaccinated individuals. Supportive care remains the mainstay of treatment, while prevention through vaccination, biosecurity, and disinfection is most effective. Future priorities include developing targeted antivirals, enhancing immunotherapies, and improving rapid diagnostics to reduce mortality and control outbreaks.

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