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Clinical and pathological investigation of feline panleukopenia with respiratory signs: A case report

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Abstract

Feline panleukopenia (FP) in cats may trigger secondary infections, which cause a wide range of clinical symptoms. This study aimed to report a case of two FP-positive cats that showed respiratory disorder. Anamnesis and clinical examination of vital parameters were carried out, as well as further examinations of the complete blood count and rapid FPV-Ag test. Subjects were < 1-year-old Persian cats (UP and CI) that had been treated for three and four consecutive days, respectively, before death. Anamnesis at the first presentation demonstrated that the cats had nasal discharge for more than 4 weeks, loss of appetite, and dehydration without a history of vomiting, but only UP developed paste diarrhea. At first presentation, only UP was lethargic, feverish, and had a lower leukocyte, while CI developed similar conditions after 3 days of treatment. A positive result of the FPV-Ag test was established for both cats. The postmortem examination showed an abnormal intestine, spleen, lungs, and heart. Histopathological examination of the duodenum revealed obstructions of villi and crypt epithelium necrosis. The swollen spleen was observed, along with white pulp atrophy and necrosis. The alveolar walls of the lungs contain inflammatory cells with pinkish exudate, which may be related to the atrophy of myofiber. Overall, this case report details that the FP in cats with respiratory signs had severe leukopenia, elevated fibrinogen, and alterations in multiple organs, highlighting the involvement of secondary issues.

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Introduction

Feline panleukopenia (FP) in cats is a highly contagious and often fatal viral disease, mainly in young cats. Feline Panleukopenia Virus (FPV), a *Pavoviridae* family virus with Canine Parvovirus (CPV-2) and Mink Enteritis Virus (MEV), is the primary causative agent in most cases. However, several studies have found Canine Parvovirus (CPV-2) in cats with FP symptoms (1-4). This virus is highly contagious and remains stable at high temperatures (5). After entering the body, the virus can reach its replication target in the oropharynx 12-18 hours post-infection, then enter the bloodstream within 2-7 days and cause systemic infection. The virus then infects essential organs with the characteristics of actively dividing cells, such as lymphoid tissue, bone marrow and intestinal mucosa (1,6). The mortality rate in the retrospective FP study was significant (80%) even after proper treatment (1,7). Most of the cats died before the three-day course of therapy was finished (8). Low leukocyte or platelet counts during presentation and hospitalization could indicate a diagnosis. Besides the infection, this condition could be worsened by secondary infections (9,10). Feline panleukopenia cats are vulnerable to various secondary infections due to the disease severely compromising the immune system. These secondary infections could be bacterial, viral, or fungal and may lead to a broad spectrum of clinical symptoms. Clinical symptoms of FP are generally related to gastrointestinal problems and

appear around 2-10 days after the virus is transmitted directly through fecal-oral or transferred by the contaminated environment (1,11). In acute conditions, cats experience high fever (40-41^{o}C), weakness, anorexia, vomiting, diarrhea with or without bleeding, and severe dehydration. Hypersalivation and the appearance of ulcers in the oral area accompanied by pale mucosa to jaundice can occur due to bacteremia caused by secondary infection (12). Secondary infection also affects a more severe and broader range of additional symptoms, including respiratory distress (6).

The multifaceted nature of these secondary infections makes managing and treating FP in cats particularly challenging. This study aims to investigate the clinical and pathological outcomes in cats affected by FP who exhibit respiratory symptoms. By focusing on these particular cases, the study aims to uncover the complications and manifestations of FP that contribute to respiratory distress. Understanding these outcomes is crucial as it provides valuable insights into the progression and severity of the disease in a subset of FP-infected cats. This knowledge will be beneficial for identifying alterations in the disease presentation when respiratory signs are present, thereby helping to delineate the complexity of FP. Moreover, the findings from this study will be a reference for disease progression that might be a basis for developing more effective treatment plans, especially for managing further FP with respiratory signs cases.

Materials and methods

Ethical approve

All treatments on animals have been approved by the ethics committee of the Faculty of Veterinary Medicine, Universitas Gadjah Mada (No. 033/EC-FKH/Int./2022). Moreover, the animal owner was asked for consent before any samples of animal dead bodies were taken for research.

Subjects and clinical examination

The included patients were two cats named UP and CI (Persian, below 1 year old, male and female, weighing 1,2 kg and 1,8 kg, respectively) from the UGM Veterinary Medicine Animal Clinic that were admitted in March 2024. Both of them had the same owner and showed signs of anorexia, lethargy, and dehydration, with a history of sneezing and mucous to mucopurulent nasal discharge. The cats did not show vomiting symptoms, but one cat (UP) had paste diarrhea and fever (40°C). Following clinical examination comprising an assessment of the cat's demeanour, vital signs (temperature, heart rate, respiratory rate), hydration status, mucous membrane colour, capillary refill time, and overall body condition, the FPV-Ag test (S&C Biotech, China) and blood profile test were performed (13-15).

The cats were intensively monitored and underwent medication antibiotic combination of enrofloxacin (Kepro Holland, Netherland) 5 mg/kg BW and amoxicillin (Kepro Holland, Netherland) 10 mg/kgBW, as well as NaCl fluid therapy (MJB Pharma, Indonesia), supportive therapy of granulocyte colony-stimulating factor analog drug Filgrastrim (Kalbe, Indonesia) 5 mcg/kg BW and hematodin (Romindo Primavetcom, Indonesia) 10 mg/kgBW for red blood cell development. Unfortunately, UP and CI died on days 3 and 5 of treatment, respectively. Subsequently, cats were necropsied for postmortem examination. Abnormal lungs, spleen, and intestines were taken for further pathological examinations. All procedures carried out on animals are in accordance with standard operating procedures at the animal clinic and animal welfare principles (16).

Hematological analysis

Hematological parameters of red blood cells (RBC), hemoglobin (HGB), hematocrit (HCT), white blood cells (WBCs), fibrinogen, and total plasma protein (TPP) were analyzed manually (17). RBC and total WBC count were carried out using established hemocytometer methods, while hemoglobin was measured by the cyanohemoglobin method. Furthermore, the TPP, including the fibrinogen, was determined manually with the Goldberg refractometer, or TS (total solids) meter (17).

Pathological examination

Organs were first observed in situ or while still in the abdominal cavity. Altered organs of the spleen, lungs, and heart were dissected for in-depth observation. The intestinal lumen was also partially opened to view the condition of the mucosal layer. As previously mentioned, organ specimens were taken about 1 cm^3 in size and placed in 10% formalin for fixation for histopathological examination (18,19).

Results

Anamnesis revealed they had been administered a cat flu drug from a pet shop for three consecutive days without resolution. Neither cat had vaccination or anthelmintic records. The owner reported no vomiting or diarrhea in CI, while UP had paste-like feces. At presentation, both cats were underweight (body condition score 2/5), with mucopurulent nasal discharge. UP showed severe lethargy, fever (40.2°C), and dehydration (capillary refill time >2 seconds). Hematology results on day 0 as seen in table 1 showed low leukocyte (WBC: 0.3 × $10^3/mm^3$) and high fibrinogen level (600 mg/dL) in UP. CI had an initial hemoglobin level of 16.6 g/dL and normal leukocyte count (WBC: 17.95 × $10^3/mm^3$). Serial hematology in CI revealed leukocyte depletion and increased fibrinogen on day 3. UP succumbed on day 3 before a serial hematological examination could be performed, while CI died on day 4 after developing watery diarrhea, hypothermia, and severe leukopenia.

Table 1. Some hematological findings of cats, namely UP and CI

Parameters	UP	CI	
	Day-0	Day-0	Day-3
RBC	8.38	7.03	4.64
HGB	12.09	16.6	10
HCT	32	38	25
WBC	0.3	17.95	0.2
TPP	7.5	7.3	6
Fibrinogen	600	100	800

* RBC: Red Blood Cells (10^6/mm^3), HGB: hemoglobin (g/dL), HCT: hematocrit (%), WBC: White Blood Cells (10^3/mm^3), TPP=Total Protein Plasma (g/dL), Fibrinogen (mg/dL).

Macroscopic examination of the intestines showed hyperemia and diffuse hemorrhage, especially in the duodenum, which was distended and contained mucous fluid mixed with blood. Histopathological examination showed that the villi of the intestines were mainly deformed along with Lieberkhun's crypt epithelium necrosis (Figure 1). Meanwhile, the spleen was moderately enlarged and elongated, with a dark reddish-brown colour. The surface was smooth, and the tissue appeared relatively firm, with no visible nodules, masses, or apparent areas of necrosis. The blunt edges are clearly observed in the ventral and dorsal end. Histopathological examination revealed that the white pulp was expanded, and many areas were seen as space. The center germinal within the follicles suggests reactive changes and an amount of necrotic lymphoid cells (Figure 2), while the red pulp showed congestion.

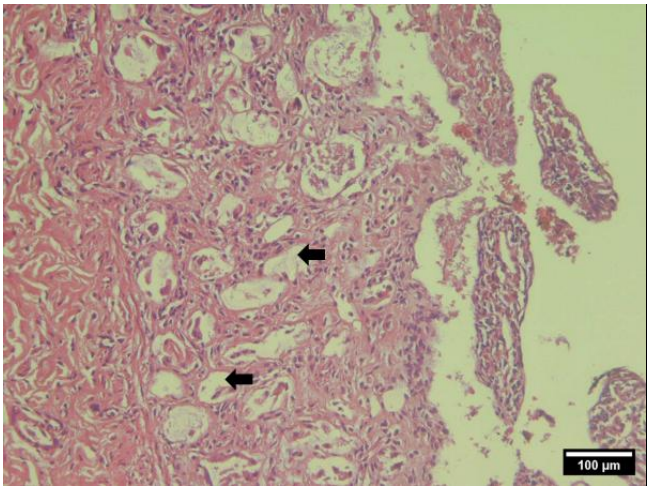

Figure 1: Crypt epithelium necrosis (black arrow) of duodenum of FP cat (magnification 100x, HE staining).

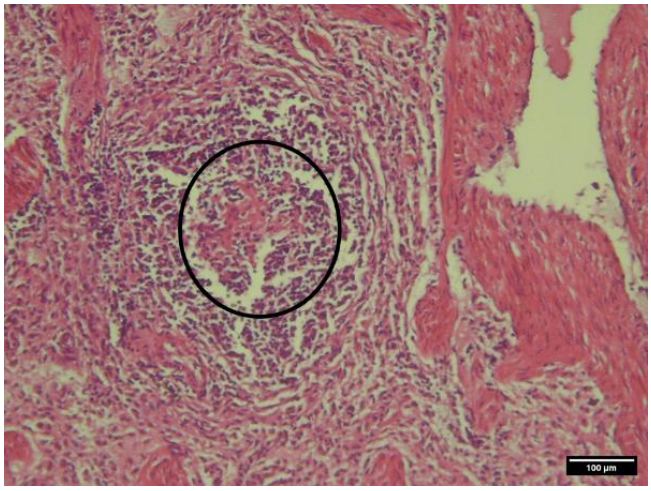

Figure 2: Necrosis of central germinal white pulp (circled area) in the spleen of FP cat (magnification 100x, HE staining).

Pathological examination of the lungs showed congestion but were still floating on the lung float test. The histopathology showed that the alveolar walls (septa) were lined by pneumocytes and contained inflammatory cells, primarily polymorphonuclear cells (Figure 3). The pinkish edematous fluid were seen within the alveolar spaces. The heart was macroscopically appeared blunt with a significant amount of yellowish fatty plaque. Histopathology revealed visible spaces between the myocardium without inflammatory cell infiltration (Figure 4).

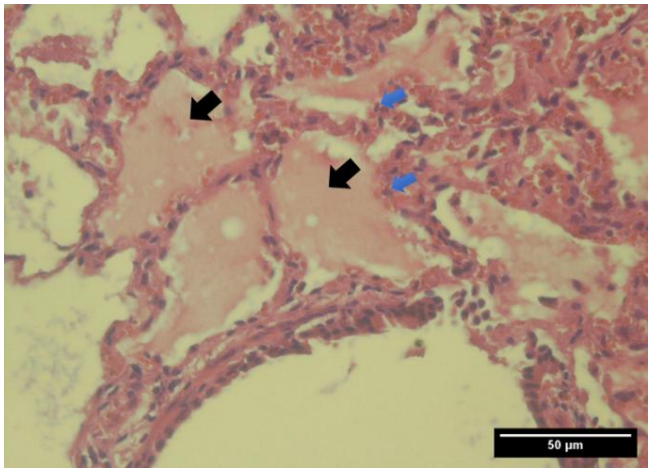

Figure 3: Eosinophilic mass deposit in alveolus indicating pulmonary edema (black arrow) with inflammatory cells infiltration (blue arrow) in the lung's septa (magnification 400x, HE staining).

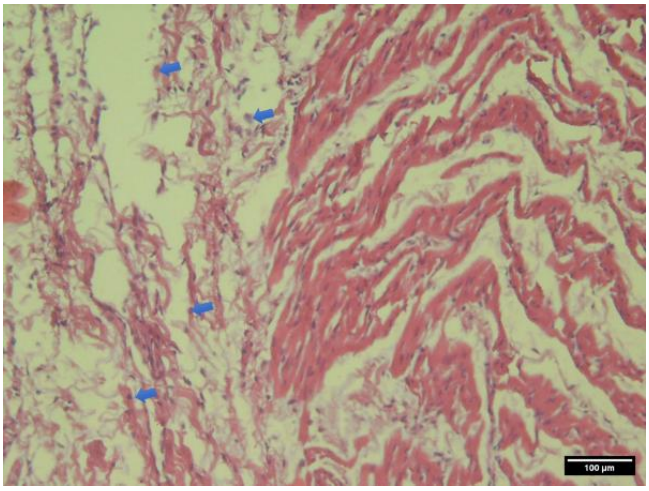

Figure 4: Inflammatory cell infiltration in the pericardium and severe edema between the cardiac muscle fibres (magnification 100x, HE staining).

Discussions

Feline panleukopenia (FP) is known to cause a wide range of clinical symptoms due to its impact on multiple organ systems (12). Cats infected with FP typically exhibit a range of symptoms such as fever, lethargy, anorexia, vomiting, diarrhea (which can be watery or hemorrhagic), dehydration, and neurological signs like cerebellar ataxia mainly if the infections occur during the maternal stage (20-24). Despite the symptoms of the disease in the gastrointestinal tract, central nervous system, and hematopoietic tissue, cats may also experience complications such as secondary infections, contributing to the variability in symptoms and disease severity (10,22,25).

Although CI's hematological parameters on day 0 were within the normal reference range, except for elevated hemoglobin levels, the suspicion of FP remained high due to its group housing and close contact with UP, which could be risk factors for FP (26). Given the highly contagious nature of FP, the rapid FPV antigen (FPV-Ag) test was performed on both cats, despite CI's initially normal hematological profile. The test yielded positive results, confirming FP in both cats.

A serial hematological examination of CI on day 3 revealed a severe leukocyte drop and increased fibrinogen. This indicated the progression of infection during hospitalization, even with a causative and supportive medication. Meanwhile, serial hematology analysis could not be performed on the UP cat because death occurred before the procedure was performed. The elevated fibrinogen levels observed during leukopenia in both cats exceeded the normal reference range, suggesting increased blood coagulation likely associated with disseminated intravascular coagulation (DIC). This condition is commonly triggered by systemic infection or inflammation and corresponds with the severity of clinical signs and poor prognosis (27-29).

The treatment plan for FP generally involves fluid therapy, antibiotics, and supportive therapy such as interferon and granulocyte-colony stimulating factor (30,31). However, the prognosis remains poor, as demonstrated in this study. UP died on day 3 of treatment, while CI deteriorated further, developing watery diarrhea, hypothermia, and severe leukopenia on day 3, culminating in death on day 4. The case outcomes depicted the poor prognosis of FP despite standard treatments. Both cats in the study also deteriorated rapidly, demonstrating the aggressive nature of FP and the limited efficacy of current therapies.

Pathological findings demonstrated that the spleen and intestines were severely affected, consistent with FP's tropism for rapidly dividing cells in these organs (22). The alteration of the intestine was typical in FP patients, as the virus tropism was mainly subjected to highly dividing cells of the crypt (24). These lesions support the definitive diagnosis of FP in both cats, even though the clinical signs when the cats were admitted to the clinic were not typical. The morphological alteration of the spleen and Liebekhun's crypts could be associated with low leukocyte count as the interference of cell differentiation in the lymphoid tissues of the spleen despite the systemic infection that triggered low circulating leukocytes (32,33).

Lesions in the spleen and intestines might be the destructive impact of FP, in accordance with leukopenia and elevated fibrinogen levels, which indicated systemic inflammation. These changes likely weakened the immune system, increasing the cats' susceptibility to secondary infections from bacterial, viral, or fungal pathogens. However, this study did not identify specific causal agents of the respiratory symptoms. Pulmonary edema could have resulted from fluid accumulation due to compromised cardiac function, as suggested by cardiomyopathy or myocardial atrophy observed in one cat (34,35).

Therefore, respiratory signs observed in both cats were likely secondary complications rather than direct effects of FP. While FP pathogenesis does not typically involve the lungs (35-37), secondary bacterial infection might be involved since the disease has an immunosuppressive nature (10,38). The indications of cardiomyopathy which is characterized by a picture of myocardial atrophy in the heart were more likely to cause pulmonary edema in this case. However, the correlation between this condition and FP disease must be clearly explained (39). Notably, respiratory symptoms were reported several weeks prior to the onset of FP signs, suggesting they may have been a predisposing factor. Additionally, co-infection with multiple parvovirus strains, potentially causing genetic recombination and heterogeneity, might have contributed to the respiratory alterations (4,40).

Overall, the occurrence of respiratory signs in FP cases underscores the need for more comprehensive investigations into the FP etiology and potential links to viral and bacterial co-infections. Further study is still needed to identify the possible agents or disease pathogenicity that influence the respiratory system in FP cases, enhance knowledge about disease characteristics, and improve the therapy outcome.

Conclusion

The case highlights the complex clinical presentation of FP, which includes many organ systems. The two cats, UP and CI, presented with symptoms of Feline Panleukopenia (FP), confirmed by a positive FPV-Ag test and severe leukopenia. Despite treatment, both cats died due to the systemic impact of FP, evidenced by histopathological findings showing necrosis in the intestines and the spleen and significant leukocyte depletion. Elevated fibrinogen levels indicated widespread inflammation. Respiratory signs were likely secondary to either concurrent infection or heart failure, as lung edema and myocardial damage were noted.

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Conflict of interest

There is no conflict of interest.

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دراسة سريرية ومرضية لنقص الكريات البيض في القطط المصاحبة للعلامات التنفسية: تقرير حالة

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الخلاصة

قد يؤدي نقص الكريات البيض في القطط إلى حدوث التهابات ثانوية، والتي تسبب مجموعة واسعة من الأعراض السريرية. هدفت هذه الدراسة إلى عرض حالة قطتين إيجابيتين لنقص الكريات البيض أظهرتا اضطرابًا في الجهاز التنفسي. تم اخذ تاريخ الحالة والفحص السريري للمعايير الحيوية، بالإضافة إلى فحوصات أخرى لتعداد الدم الكامل واختبار FPV-Ag السريع. كانت القطط من السلالة الفارسية عمرها اقل من سنة واحدة، تم علاجها لمدة ثلاثة وأربعة أيام متتالية، على التوالي، قبل الوفاة. أظهر تاريخ الحالة في القط الاول أنه كان يعاني إفرازات أنفية لأكثر من ٤ أسابيع، وفقدان الشهية، والجفاف دون تاريخ من القيء، ولكن القط الثاني فقد اصيبت بالإسهال. القط الثاني فقط كان خاملًا ومصابًا بالحمى وكان لديه عدد أقل من كريات الدم البيضاء، بينما القط الاول اظهر علامات مماثلة بعد ٣ أيام من العلاج. تم التوصل إلى نتيجة إيجابية لاختبار FPV-Ag لكلا القطتين. وأظهر الفحص بعد الموت وجود خلل في الأمعاء والطحال والرئتين والقلب. كشف الفحص النسيجي المرضي للاثني عشر عن وجود عوائق في الزغابات ونخر ظهارة القبو. ولوحظ تورم الطحال، إلى جانب ضمور اللب الأبيض ونخره. تحتوي الجدران السنخية للرئتين على خلايا التهابية ذات إفرازات وردية اللون، والتي قد تكون مرتبطة بضمور الألياف العضلية. بشكل عام، يوضح تقرير الحالة هذا أن نقص الكريات البيض في القطط التي تعاني من علامات تنفسية يتسبب في نقص شديد في الكريات البيض، وارتفاع الفيبرينوجين، وتغيرات في أعضاء متعددة، مما يسلط الضوء على تولد مشاكل ثانوية.