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CANINE PARVOVIRUS INFECTION IN A LOCAL PUPPY FROM SEMPIDI VILLAGE, BADUNG REGENCY: A CASE REPORT

Laporan Kasus: Infeksi *Canine Parvovirus* pada Anak Anjing Lokal yang Berasal dari Desa Sempidi, Kabupaten Badung

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Abstract

Canine parvovirus (CPV) is a highly contagious viral disease associated with high mortality rates in dogs, particularly in puppies. Clinically, CPV infection manifests in two forms: enteritis and myocarditis. The virus spreads rapidly among dogs through direct contact with infected animals or indirect contact with contaminated feces and fomites. This case report describes a local male dog, approximately two months of age, from Sempidi Village, Mengwi District, Badung Regency, Bali, that presented with lethargy, anorexia, vomiting, refusal to eat and drink, and bloody diarrhea. Gross pathological examination revealed cerebral vascular congestion and meningeal hyperemia, cardiomegaly, multifocal pulmonary discoloration, multifocal discoloration of the liver lobes, dark discoloration of the renal cortex and medulla, splenomegaly, and hemorrhages affecting both the intestinal serosa and mucosa. Histopathological examination demonstrated meningoencephalitis, edematous necrotizing myocarditis, necrohemorrhagic bronchopneumonia, hemorrhagic hepatitis, hemorrhagic splenitis, necrohemorrhagic glomerulonephritis, and necrotizing enteritis. Polymerase chain

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reaction (PCR) testing confirmed CPV infection. Based on the anamnesis, clinical presentation, epidemiological findings, gross and histopathological lesions, and molecular diagnostic results, the dog was diagnosed with canine parvovirus infection.

Keywords: dog, canine parvovirus, gross pathology, histopathology, PCR

Abstrak

Canine parvovirus (CPV) adalah penyakit viral yang menyebabkan kematian tertinggi pada bangsa anjing, terutama pada anak anjing. Berdasarkan manifestasi gejala klinis, terdapat dua bentuk dari CPV yaitu tipe miokarditis dan enteritis. Virus ini sangat menular dan dapat menyebar dari antar anjing melalui kontak langsung atau tidak langsung dengan feses yang terinfeksi. Laporan kasus ini melaporkan seekor anjing lokal berusia ± 2 bulan dengan jenis kelamin jantan berasal dari Desa Sempidi, Kec. Mengwi, Kab Badung, Bali dengantanda klinis lemas, anoreksia, muntah, tidak mau makan dan minum serta diare disertai darah. Perubahan patologi anatomi yang teramati adalah kongesti pada vaskuler otak dan hipermia pada meningens, kardiomegali, perubahan warna tidak merata pada paru-paru, perubahan warna tidak merata pada lobus hati, perubahan warna menggelap pada korteks dan medula ginjal, pembengkakan limpa, serta hemoragi pada serosa dan mukosa usus. Hasil pengamatan histopatologi menunjukan adanya *meningoenchepalitis, myocarditis necroticans et edematosa, bronkopneumonia necroticans et hemmoragica, hepatitis hemorrhagica, splenitis hemorrhagica, glomerulonephritis necroticans et hemmoragica,* dan *enteritis necroticans*. Uji *Polymerase Chain Reaction* (PCR) menunjukkan hasil positif terhadap infeksi CPV. Berdasarkan data anamnesis, tanda klinis, epidemiologi, dan pemeriksaan laboratorium yang telah dilakukan dapat disimpulkan bahwa anjing kasus terinfeksi CPV

Kata kunci: anjing, *canine parvovirus*, patologi anatomi, histopatologi, PCR

INTRODUCTION

Canine parvovirus (CPV) infection is one of the leading causes of mortality in dogs, particularly affecting puppies between six weeks and six months of age (Suartini *et al.*, 2016). Parvoviral disease predominantly affects young dogs. A study by Shima *et al.* (2015) reported that among 204 cases of CPV infection, 60.3% occurred in dogs aged 0–5 months, 27.0% in dogs aged 6–11 months, and 12.7% in dogs older than 11 months. CPV infection was first identified in 1977 in Texas, United States, and subsequently spread worldwide (Tuteja *et al.*, 2022). The virus belongs to the family Parvoviridae, subfamily Parvovirinae, and genus Protoparvovirus. Canine parvoviral disease is caused by viruses within the family Parvoviridae, including Canine parvovirus type 1 (CPV-1), also known as Canine minute virus (MVC) or Canine bocaparvovirus-1, and Canine parvovirus type 2 (CPV-2). However, CPV-2 is recognized as the primary etiological agent of acute hemorrhagic enteritis associated with high morbidity and mortality rates in puppies (Tuteja *et al.*, 2022). CPV-2 is a small, non-enveloped, single-stranded DNA virus with three antigenic variants: CPV-2a, CPV-2b, and CPV-2c. The virus exhibits remarkable environmental stability and is resistant to a wide range of disinfectants (Barrs, 2019).

The incubation period of CPV infection ranges from three to seven days before puppies develop severe clinical disease. The disease progresses rapidly and may result in death within two to three days after the onset of clinical signs (Miranda & Thompson, 2016). Clinical manifestations include depression, loss of appetite, weakness, anorexia, vomiting, pale mucous membranes, high fever during the early stage of the disease that gradually returns to normal, and diarrhea ranging from yellow mucoid feces to hemorrhagic diarrhea with a characteristic foul odor. The severity of clinical manifestations largely depends on the age of the infected

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dog, with younger puppies generally exhibiting more severe disease (Mylonakis *et al.*, 2016). CPV infection presents in two major clinical forms: myocarditis and enteritis. The enteric form is more commonly encountered than the myocardial form. Epidemiological studies have shown that the morbidity rate of the enteric form of CPV infection ranges from 20% to 100%, with mortality rates reaching 50% to 100%. In young, unvaccinated puppies, mortality may reach 100% (Jedaut *et al.*, 2021).

The diagnosis of CPV infection requires specific and sensitive laboratory methods to definitively confirm the presence of the virus. One of the most widely used diagnostic techniques is the polymerase chain reaction (PCR) assay. PCR is an in vitro DNA synthesis and amplification technique capable of generating millions of copies of a target DNA segment within a few hours (Setyawati & Zubaidah, 2021). Compared with rapid immunochromatographic tests, which detect viral antigens and may yield false-negative results when viral shedding in feces is low, PCR offers significantly greater sensitivity, particularly in latent infections or when viral titers are below the detection threshold of conventional diagnostic methods. Furthermore, PCR enables genetic analysis of the viral VP2 gene, which is valuable for variant identification and molecular epidemiological investigations (Nareswari *et al.*, 2016). The present case report aims to describe CPV infection in a puppy from Sempidi Village, Badung Regency, through a comprehensive diagnostic approach that included epidemiological assessment, postmortem gross pathological examination, and PCR testing. In addition, bacteriological, mycological, and parasitological examinations were performed to determine whether concurrent infectious agents were present.

MATERIALS AND METHODS

Case Animal

The case involved a local male dog approximately two months of age originating from a household in Sempidi Village, Mengwi District, Badung Regency, Bali. A total of four dogs were kept at the household, consisting of one dam and three puppies. The dogs were maintained under a free-roaming management system within the household environment. According to the owner, none of the puppies had received vaccination against canine infectious diseases, including canine parvovirus (CPV). Clinical signs observed in the case dog included hemorrhagic diarrhea with a characteristic foul odor, anorexia, refusal to eat and drink, lethargy, and vomiting. Initially, clinical signs appeared in one puppy, and two days later, similar signs were observed in the other two puppies. The first puppy was found dead one day before specimen collection, whereas the case dog died during transportation after experiencing illness for approximately three days. The remaining puppy was also reported to have died the following day.

Epidemiological Assessment

In epidemiological investigations, three major factors should be considered: host, environment, and agent. These factors play critical roles in the occurrence and spread of disease. Disease prevalence within a specific population can be evaluated by calculating morbidity, mortality, and case fatality rate (CFR). Morbidity refers to the number or proportion of animals affected by a disease within a population during a defined period. Mortality refers to the number or proportion of animals that die from a disease relative to the total population at risk. The CFR represents the proportion of animals that die among those diagnosed with the disease. This parameter reflects the severity and lethality of the disease.

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Gross Pathological and Histopathological Examinations

A necropsy was performed to systematically examine organs and tissues and determine the cause of death. The necropsy of the case dog was conducted at the Veterinary Pathology Laboratory, Faculty of Veterinary Medicine, Universitas Udayana. Organ samples exhibiting gross pathological changes were collected and trimmed to approximately 1 × 1 × 1 cm before fixation in 10% Neutral Buffered Formalin (NBF). Routine histopathological processing was performed according to the standard procedures of the Veterinary Pathology Laboratory, and tissue sections were stained with Hematoxylin and Eosin (HE) (Sewoyo *et al.*, 2025). The prepared slides were subsequently examined microscopically for histopathological evaluation.

PCR Assay

Detection of CPV infection was performed using the Polymerase Chain Reaction (PCR) method at the Veterinary Virology Laboratory, Faculty of Veterinary Medicine, Universitas Udayana. The amplified PCR products were subsequently analyzed by electrophoresis on 1% agarose gel under a constant voltage of 100 V for approximately 30 minutes. The DNA bands were then visualized under ultraviolet (UV) illumination using a transilluminator (Sewoyo *et al.*, 2022).

Bacterial Isolation and Identification

Bacteriological examination, including bacterial isolation and identification, was conducted at the Veterinary Bacteriology and Mycology Laboratory, Faculty of Veterinary Medicine, Universitas Udayana. Organ samples were cultured on Nutrient Agar (NA) as a general-purpose growth medium. Colonies growing on NA were subjected to preliminary identification tests, including the catalase test to assess catalase enzyme production and Gram staining to determine bacterial morphology and Gram reaction. The colonies were subsequently inoculated onto Eosin Methylene Blue Agar (EMBA) and incubated for 18–24 hours to evaluate bacterial growth characteristics. Further identification was performed using biochemical tests on Triple Sugar Iron Agar (TSIA), Sulfide Indole Motility (SIM) medium, Methyl Red–Voges Proskauer (MR-VP) medium, Simmons Citrate Agar (SCA), and carbohydrate fermentation tests using glucose (Safika *et al.*, 2023).

Fecal Examination

Fecal samples were collected from the case animal and placed in a urine container containing 10% NBF before being submitted to the Parasitology Laboratory, Faculty of Veterinary Medicine, Universitas Udayana, for examination. Qualitative fecal examination was performed using direct smear, sedimentation concentration, and flotation concentration techniques. In practice, the flotation method is generally more efficient than the sedimentation method because it produces cleaner preparations with less debris. Fecal examination was conducted to rule out gastrointestinal parasitic infections as differential diagnoses that may cause enteritis and hemorrhagic diarrhea in puppies.

RESULTS AND DISCUSSIONS

Results

Based on the epidemiological data, the morbidity rate, mortality rate, and CFR were 75%, 75%, and 100%, respectively. Clinical signs observed in the case dog included hemorrhagic diarrhea with a characteristic foul odor, anorexia, refusal to eat and drink, lethargy, and vomiting. Considering the clinical presentation, rapid disease progression, short survival time, and epidemiological findings, a viral infection was strongly suspected as the primary cause of death.

Gross pathological examination revealed cerebral vascular congestion and meningeal

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hyperemia. The heart showed cardiomegaly with a rounded cardiac apex. The lungs exhibited multifocal dark discoloration in several lobes. The liver was enlarged (hepatomegaly), with rounded lobular margins and multifocal pale discoloration. The spleen was enlarged (splenomegaly), while the kidneys showed diffuse dark discoloration of both the cortex and medulla. The stomach presented serosal hemorrhage and dark discoloration of the gastric mucosa. Hemorrhages were also observed in both the serosal and mucosal layers of the intestine (Figure 1).

Histopathological examination revealed lesions in multiple organs, predominantly characterized by mononuclear inflammatory cell infiltration, supporting a viral etiology (Figure 2). In the brain, meningoencephalitis was observed, characterized by lymphocytic infiltration and congestion in the meninges, as well as gliosis and perivascular edema in the cerebral cortex. The heart showed necrotizing and edematous myocarditis, characterized by myocardial necrosis, lymphocytic infiltration, and interstitial myocardial edema. The lungs exhibited necrohemorrhagic bronchopneumonia, characterized by hemorrhage, congestion, exudate within the bronchiolar lumen, lymphocytic infiltration, and necrosis of the alveolar septa. The liver showed hemorrhagic hepatitis with congestion, hemorrhage, and lymphocytic infiltration in the periportal areas. The kidneys were diagnosed with necrohemorrhagic glomerulonephritis, characterized by glomerular atrophy, congestion, tubular necrosis, and lymphocytic infiltration. The spleen showed hemorrhagic splenitis with lymphoid follicular depletion and hemorrhage. The intestine exhibited necrotizing enteritis, characterized by villous erosion, necrosis of the crypts of Lieberkühn, hemorrhage, and lymphocytic infiltration in the lamina propria.

Definitive confirmation of viral infection was obtained by PCR testing performed at the Veterinary Virology Laboratory. The PCR assay yielded a positive result for CPV infection, indicated by a DNA band corresponding to the positive control at 910 bp following agarose gel electrophoresis and UV visualization. Bacterial isolation and identification revealed the presence of *Escherichia coli* in the intestinal sample. Fecal examination showed no evidence of helminth or other parasitic infestations.

Discussion

cpv infection is an acute, highly contagious infectious disease caused by a virus belonging to the family *Parvoviridae*. CPV primarily targets the gastrointestinal system and most commonly affects young dogs (Sari *et al.*, 2024). The case dog was approximately two months old and had no history of vaccination. Young dogs are highly susceptible to CPV infection due to the presence of a window of susceptibility, during which declining maternal antibodies are insufficient to provide protection, while vaccination administered during this period may not elicit an optimal immune response. Consequently, puppies are at a significantly higher risk of CPV infection (Winaya *et al.*, 2014). Dogs with no vaccination history or incomplete vaccination (without booster doses) have a 10-fold higher risk of CPV infection compared to fully vaccinated dogs (Suartha *et al.*, 2011).

CPV infection is one of the leading causes of death in young dogs. In this study, epidemiological calculations revealed a morbidity rate of 75%, a mortality rate of 75%, and a CFR of 100%. According to Eugster *et al.* (1978), the morbidity of the enteric form may reach 20–100%, with mortality ranging from 50–100%, while in unvaccinated puppies, mortality may reach 100%. Based on the owner's report, similar clinical cases were also observed in neighboring dogs within the same residential area. This finding is consistent with CPV transmission, which occurs both directly through fecal–oral contact with infected dogs and indirectly via oronasal exposure to contaminated fomites. CPV can be shed in feces, urine, saliva, and possibly vomitus. The virus is highly stable and resistant to heat, detergents, alcohol,

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and many disinfectants, and it can persist in the environment for five to seven months (Jedaut *et al.*, 2021).

Gross pathological and histopathological findings in this case indicate systemic viral dissemination and multiorgan involvement, which are characteristic of severe CPV infection. The virus is not confined to the gastrointestinal tract but may also affect multiple organs. Parvovirus replication has been reported in the brain of infected dogs (Zhao *et al.*, 2013). Cells of the external germinal layer of the brain remain mitotically active until approximately 10 weeks of age, providing a permissive environment for viral replication. In systemic infections, myocarditis characterized by degeneration and necrosis of cardiac muscle fibers may also occur, particularly in puppies younger than eight weeks, and may lead to sudden death due to acute heart failure (Lonai *et al.*, 2022).

The spleen showed lymphoid depletion and hemorrhage. Lymphoid follicular depletion reflects viral targeting of rapidly dividing lymphoid cells. Infection of lymphoid tissues results in immunosuppression through direct lymphocytolysis and indirectly via depletion of lymphocyte precursors in the bone marrow (Boes & Durham, 2017). Pulmonary lesions commonly include alveolar edema and inflammatory cell infiltration within alveolar septa, consistent with reports by Sewoyo *et al.* (2022), who described CPV-associated interstitial pneumonia involving alveolar septa. Pulmonary involvement may occur secondary to viremia, leading to vascular injury and respiratory dysfunction.

Other organs such as the liver and kidneys also showed lesions consistent with expanded CPV tropism. The liver often exhibits congestion, while the kidneys may show glomerulonephritis accompanied by lymphocytic infiltration and glomerular atrophy. CPV has been shown to replicate in primary cell cultures derived from the liver, kidneys, heart, spleen, and intestinal epithelium (Sewoyo *et al.*, 2022). These findings indicate that viral dissemination through viremia leads to widespread tissue damage, supporting the concept that CPV is a multisystemic pathogen, particularly in severe infections with poor immune responses (Suartini & Sendow, 2015).

Characteristic lesions of CPV include necrosis and desquamation of the small intestinal epithelium, reflecting viral tropism for rapidly dividing crypt cells (Arpin & Dewantari, 2022). In this case, histopathological examination of the small intestine revealed necrosis of the crypts of Lieberkühn, hemorrhage, and lymphocytic infiltration. Villous structures were also infiltrated by inflammatory cells and showed hemorrhagic changes. Hemorrhage is caused by endothelial dysfunction, which is further exacerbated by viral replication in endothelial cells (Adi, 2021), leading to anemia. Mitotic activity in the crypts of Lieberkühn was absent, and lymphocytic infiltration was present. Necrotic debris mixed with blood is excreted in feces, producing a characteristic foul odor (Sendow *et al.*, 2014).

In severe CPV infection, complete villous atrophy may occur (Goddard & Leisewitz, 2010). Without regeneration from the crypts of Lieberkühn, the intestinal villi lose their absorptive capacity for nutrients and fluids, resulting in diarrhea. This also facilitates secondary bacterial invasion as a portal of entry. Severe villous destruction leads to rapid cessation of digestive function due to acute hemorrhagic inflammation. Massive fluid loss subsequently results in severe dehydration and death (Lubis *et al.*, 2023).

Confirmation of CPV infection was performed using PCR. Viral detection by PCR is based on its high specificity, efficiency, and accuracy. The specificity of PCR lies in its ability to selectively amplify target DNA through repeated cycles. Its high accuracy is attributed to DNA polymerase, which minimizes errors during amplification (Anggisti & Roslim, 2018). Bacterial isolation and identification revealed *Escherichia coli* in the intestinal sample, which is a normal

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commensal flora in the canine gastrointestinal tract (Besung *et al.*, 2019). In CPV infection, damage to intestinal crypts, mucosal necrosis, and loss of intestinal barrier integrity may increase exposure of intestinal tissues to enteric bacteria. As a result, commensal bacteria such as *E. coli* may contribute to the exacerbation of the ongoing enteritis (Mylonakis *et al.*, 2016).

CONCLUSIONS AND SUGGESTIONS

Conclusions

Based on the clinical signs, gross pathological and histopathological findings, as well as laboratory examinations in virology, bacteriology, mycology, and parasitology, it can be concluded that the case dog originating from Sempidi Village, Mengwi District, Badung Regency, was infected with CPV without evidence of concurrent bacterial or parasitic coinfections.

Suggestions

This case report is expected to provide a comprehensive overview of the clinical manifestations, diagnostic approaches, and management strategies of canine parvovirus infection in local dogs. It may serve as a reference for veterinary practitioners and animal owners in managing similar cases in the future.

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Figures

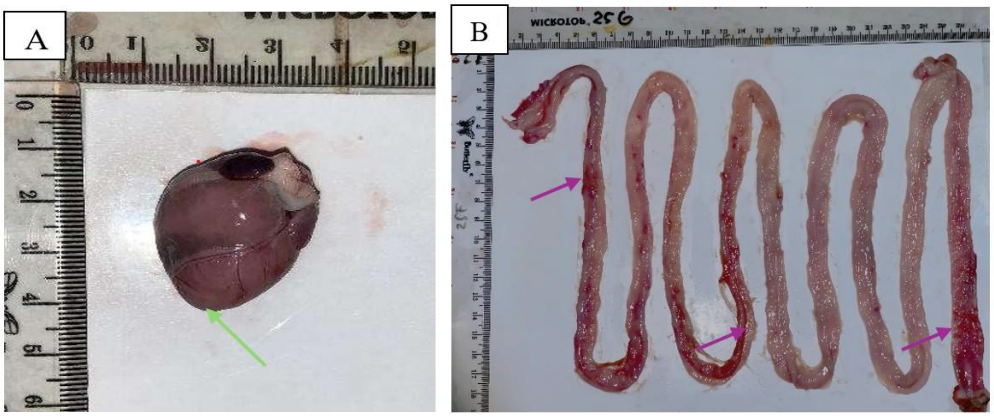

Figure 1. Gross pathological findings showing (A) cardiomegaly with a blunted cardiac apex and (B) hemorrhage along the intestinal mucosa.

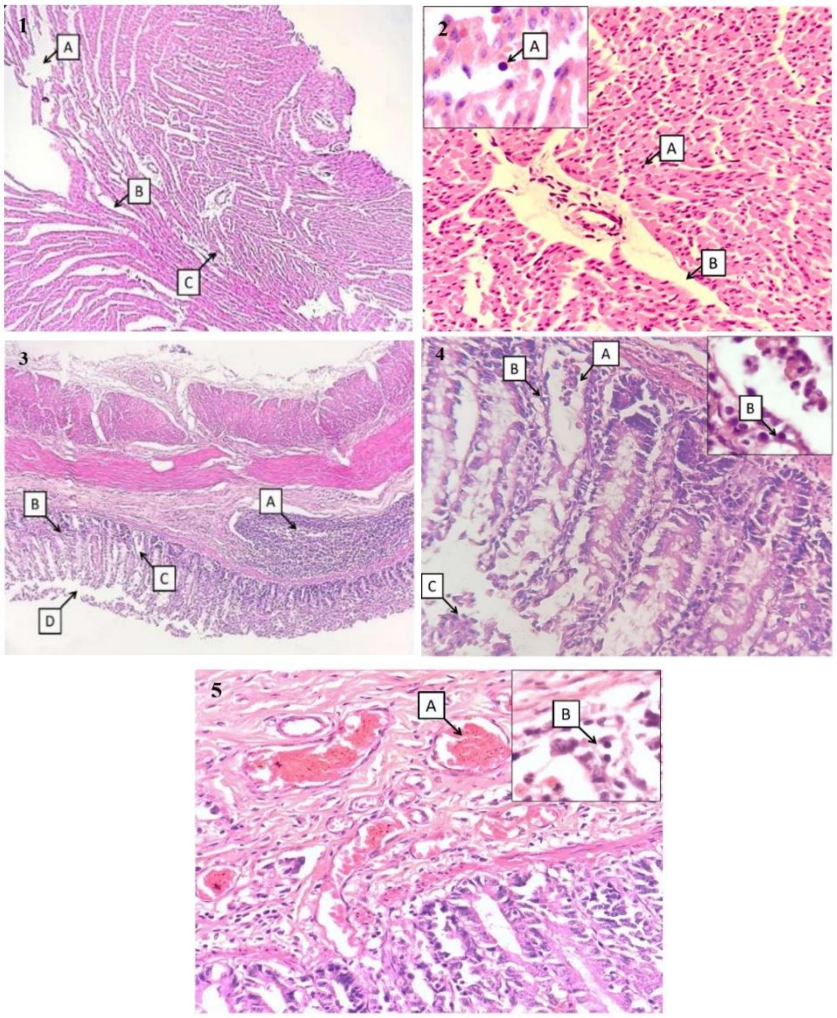

Figure 2. Histopathological lesions in the heart and intestine. The heart (1–2) shows necrotizing and edematous myocarditis characterized by myocardial necrosis, intermyocardial edema, and lymphocytic infiltration. The intestine (3–5) shows necrotizing enteritis characterized by necrosis of Peyer's patches and crypts of Lieberkühn, villous erosion, hemorrhage, and inflammatory cell infiltration. (Hematoxylin-Eosin).

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