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CASE REPORT: CANINE PARVOVIRUS WITH CONCURRENT HELMINTHIASIS IN A DOG IN PEGUYANGAN, DENPASAR CITY, BALI

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Abstract

Canine Parvovirus (CPV) is a highly contagious viral infection caused by a member of the Parvoviridae family and is a major cause of death in puppies under six months of age. In addition to the viral infection, nematode worms were also present, which are suspected to exacerbate the condition and suppress the immune system (immunosuppressive effect). This study aimed to determine the cause of death in a Miniature Pomeranian dog showing clinical signs of acute gastroenteritis. The subject was a 2-month-old male dog presenting with lethargy, loss of appetite, and foul-smelling bloody diarrhea. The methods included anamnesis, clinical observation, epidemiological investigation, gross pathological examination, histopathology, virological testing using PCR, fecal parasitological examination, and bacterial isolation and identification. Epidemiological analysis showed a morbidity rate of 60%, a mortality rate of 40%, and a case fatality rate (CFR) of 66% in the surrounding environment of the case. Gross pathology and histopathological examinations revealed various inflammatory lesions. PCR testing confirmed the presence of Canine Parvovirus infection. Parasitological examination revealed eggs and two adults *Toxocara canis* worms, as well as two adult Ancylostoma caninum worms, with a mild level of infection. Bacterial identification in the intestines revealed only Escherichia coli as normal flora.

Keywords: Canine Parvovirus, *Toxocara canis*, *Ancylostoma caninum*, PCR.

INTRODUCTION

The large number of people who keep dogs is attributable to the multifunctional roles these animals play, both as household guardians and as contributors to mental well-being through their stress-reducing effects. Given their loyalty and high intelligence, dogs require intensive management and health monitoring (Sardjana & Kusumawati, 2004). One significant health threat is Canine Parvovirus disease, caused by an infectious agent in the family Parvoviridae. Historically, the virus was first detected in 1977 in Texas, United States, and subsequently

spread worldwide, affecting numerous countries (Spindel et al., 2018). Infection caused by Canine Parvovirus type 2 (CPV-2) is among the most highly transmissible and potentially fatal viral diseases in dogs (Purnamasari et al., 2015). This disease is a major cause of mortality in canine populations due to its high transmissibility. Clinically, the highest susceptibility is observed in puppies under six months of age (Prittie, 2004). Annual data indicate that the risk of infection increases significantly in puppies younger than two months and in those that have not been vaccinated (Suartha et al., 2011).

Canine parvovirus disease is a pathologic condition caused by a parvovirus in the family Parvoviridae. CPV infection presents a broad clinical spectrum; it not only invades the gastrointestinal tract but may also attack myocardial tissue, potentially leading to sudden death in puppies (Hermawan et al., 2022). Epidemiologically, the enteritis form is more prevalent than the myocarditis form, with the highest incidence observed in puppies older than two months (Purnamasari et al., 2015). Although dogs of all age groups are susceptible to type 2 infection affecting the gastrointestinal system (enteritis), the most pathognomonic clinical signs are vomiting and hemorrhagic diarrhea with a distinctive odor (Jedaut et al., 2021). These clinical findings are consistent with the study conducted by (Winaya et al., 2014) in Denpasar, which confirmed that CPV predominantly infects young dogs. Systemic manifestations accompanying the infection include fever, lethargy, anorexia, and changes in fecal consistency, initially presenting as yellowish or grayish feces with a foul odor, which later progress to blackish bloody diarrhea.

Parasitic infections may also accompany Parvovirus disease. In clinical practice, the initial diagnosis of helminthiasis is made through a symptomatic approach, primarily focusing on diarrhea and macroscopic fecal evaluation. In this case, the dog was found to be infected with Toxocara canis and Ancylostoma caninum. The objective of this article is to comprehensively report the findings from anamnesis, clinical manifestations, and epidemiological review of this case. The report focuses on the analysis of anatomical pathology and histopathological changes, supported by diagnostic test results from Polymerase Chain Reaction (PCR) techniques, routine fecal examination, and bacterial isolation procedures.

MATERIALS AND METHODS

Research Object

The research subject was a male Mini Pomeranian dog, identified under protocol number 200/N/25. The dog, named Cloud, was two months old and weighed 2 kg. It belonged to a client residing in North Denpasar, Bali. According to the owner, the dog died on October 28, 2025. A necropsy was performed immediately at the Veterinary Pathology Laboratory, Faculty of Veterinary Medicine, Udayana University, to facilitate further pathological investigation.

Anamnesis and Epidemiology

Anamnesis data and the epidemiological profile of the disease were collected through interviews with the owner and observation of the animal's housing environment. Based on the medical history, the dog had been adopted two weeks before the onset of hemorrhagic diarrhea and had not received any vaccination or deworming. Clinical manifestations were first observed four days before death, characterized by serous nasal discharge, lethargy, and watery diarrhea. During the final two days before death, the clinical condition progressively worsened to hemorrhagic diarrhea with a liquid consistency and a fishy odor, accompanied by weakness and intolerance to oral fluid intake. Epidemiological analysis of the at-risk population at the case location was determined by calculating morbidity, mortality, and the Case Fatality Rate (CFR). Morbidity was calculated as the ratio of the number of sick animals to the total at-risk

population, while mortality was determined as the ratio of the number of deaths to the at-risk population. The CFR was calculated by dividing the number of deaths by the number of sick animals, with each parameter expressed as a percentage.

Anatomical and Histopathological Pathology

An anatomical pathology evaluation was performed via necropsy on the case dog. Lesioned organs were collected in 1 × 1 × 1 cm sections and fixed in 10% Neutral Buffered Formaldehyde (NBF) for a minimum of 24 hours. Histopathological slide preparation began with trimming, followed by processing in a tissue processor. The subsequent procedures included dehydration, clearing, and infiltration using graded alcohol solutions (70% to absolute), toluene, and paraffin, all within a single-day cycle. Following the blocking stage using an embedding set, the samples were sectioned at 5 microns using a microtome. The tissue sections were placed on glass slides, heated on a hot plate for 2 hours, and incubated at 37°C for 24 hours. The final stages included Hematoxylin–Eosin (HE) staining and mounting. Microscopic evaluation was performed using a binocular light microscope at magnifications of 40×, 100×, 400×, and 1000×, with photomicrographic documentation for histopathological analysis.

Virological Examination

To identify the causative agent and confirm the diagnosis of Canine Parvovirus (CPV) infection in the dog, laboratory testing was performed using the Polymerase Chain Reaction (PCR) method. The procedure was conducted at the Veterinary Virology Laboratory of the Faculty of Veterinary Medicine, Udayana University.

Approximately 1 g of organ samples from the brain, heart, spleen, and intestine. The eluted DNA was collected into a new Eppendorf tube, and the cartridge could be stored for future use. The final DNA volume was approximately 200 µl, and only about 1 µl was required for a single PCR reaction. The primers used for Parvo detection consisted of a forward primer (HMForm) and a reverse primer (VPRM). The PCR reaction used MyTaq HS Red Master Mix, which already contained loading dye, combined with sample DNA and distilled water (aq. bidest.). The electrophoresis process began with the preparation of a 1% agarose gel. Electrophoresis was run for approximately 25 minutes, with checks every 5 minutes to confirm proper band migration. The gel was then photographed for documentation.

Bacteriological Examination

Bacterial isolation and identification procedures were performed at the Veterinary Bacteriology and Mycology Laboratory, Faculty of Veterinary Medicine, Udayana University, to detect secondary bacterial pathogens in the case subject. The organ specimens analyzed were the liver, lungs, and small intestine. The isolation protocol used Nutrient Agar (NA) as a general-purpose medium and MacConkey Agar (MCA) as a selective medium. This was followed by Gram staining and a catalase test. If Gram-negative bacteria were identified, additional biochemical testing was performed. Biochemical tests were performed by inoculating MCA colonies onto specific media, including Triple Sugar Iron Agar (TSIA), Sulphide Indole Motility (SIM) medium, Simmons Citrate Agar (SCA), Methyl Red Voges–Proskauer (MRVP) medium, and carbohydrate fermentation testing (glucose).

Parasitological Examination

Parasitological examination was performed using routine fecal analysis, including native smears, sedimentation, and flotation. Egg per Gram (EPG) counts were determined using the Stoll method, and preserved worm specimens were prepared.

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Data Analysis

This study employed a qualitative descriptive approach to comprehensively analyze the case dog's findings, including clinical manifestations, anatomical and pathological changes, and histopathological features. In addition, epidemiological analysis and laboratory isolation and identification procedures were integrated to determine the causative infectious agent.

RESULTS AND DISCUSSION

Results

Based on the data analysis, the epidemiological parameters indicated a morbidity rate of 60%, a mortality rate of 40%, and a Case Fatality Rate (CFR) of 66%. Gross pathological findings included hyperemia and congestion of the brain; hemorrhages in the trachea, esophagus, heart, lungs, and pancreas; swelling and hemorrhage of the kidneys and spleen; yellowish discoloration of the gastric mucosa; hemorrhage and necrosis of the intestines; and the presence of Toxocara canis and Ancylostoma caninum worms (Figure 1). Histopathological observations revealed meningoencephalitis, esophagitis, edematous tracheitis, hemorrhagic and necrotizing bronchopneumonia, edematous myocarditis, glomerulonephritis, hepatitis, splenitis, pancreatic congestion, gastritis, hemorrhagic and necrotizing enteritis, and colitis (Figure 2).

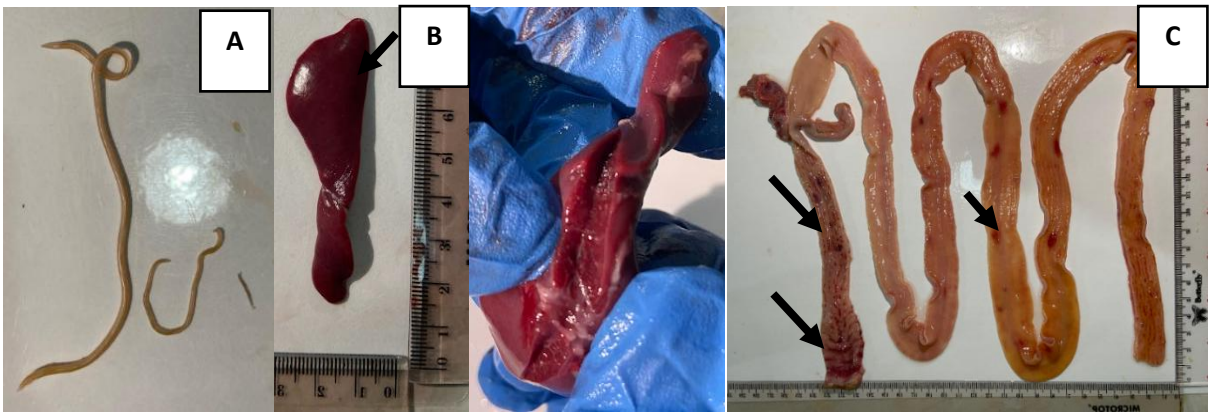

Figure 1. Anatomical Pathology: (A) Worms in the intestine; (B) The spleen is enlarged and darker in color; (C) The intestines show extensive inflammation

Examination at the Virology Laboratory using the PCR (Polymerase Chain Reaction) test showed a positive result, as evidenced by a DNA band aligned with the positive control in electrophoresis (Figure 3). These laboratory findings confirmed that the case dog (protocol number 200/N/25) was infected with Canine Parvovirus.

The results of bacterial isolation and identification on NA and MCA media, Gram staining, catalase test, TSIA, SIM, MR, VP, SCA, and glucose test are presented in Table 1 and Figure 4. The findings indicated that Escherichia coli (E. coli) was part of the normal intestinal flora.

Routine fecal examination using the flotation method revealed Toxocara canis and Ancylostoma caninum eggs. Preserved specimens from necropsy confirmed four adult worms, comprising two Toxocara canis and two Ancylostoma caninum. The Stoll method detected 100 Ancylostoma caninum eggs per gram of feces. Unembryonated Toxocara canis eggs are round, brown, with thick, polygonal walls and a pitted surface (Joesoef & Larasati, 2025). The size of T. canis eggs is approximately 80 × 75 µm (Koesdarto et al., 2001). Ancylostoma caninum eggs are oval, with thin, two-layered walls, measuring approximately 56–75 × 34–47 µm.

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When passed in feces, the eggs are already at the segmented stage, consisting of 8–16 cells (Ahada et al., 2020). Adult Toxocara canis worms measure 7–11 cm in length, possess three lips and narrow alae at the anterior end, and have a finger-like posterior end (Hadi, 2019). Adult A. caninum worms are relatively small and hook-shaped; males measure approximately 11–13 mm and are equipped with spicules, while females measure 14–21 mm. The buccal capsule contains tooth-like structures that function in attachment and blood feeding (Levine, 1994).

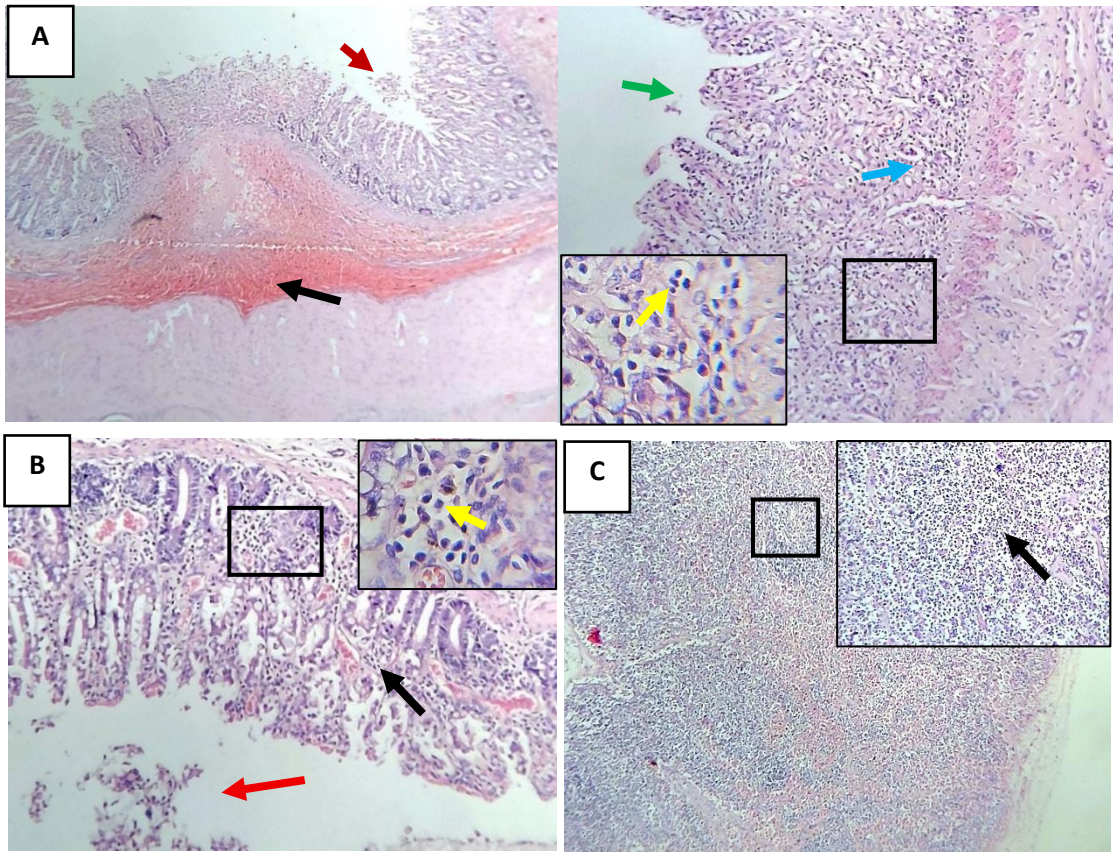

Figure 2. Histopatologi; (A) Enteritis hemorrhagis et necroticans; (B) Colitis; (C) Splenitis

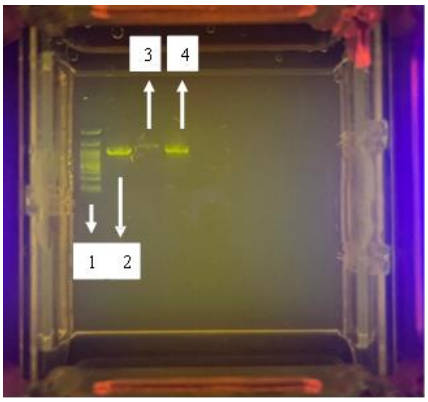

Figure 3. PCR Results: (1) DNA Marker / DNA Ladder; (2) Specimen with Protocol Number 200/N/25; (3) Negative Control; (4) Positive Control

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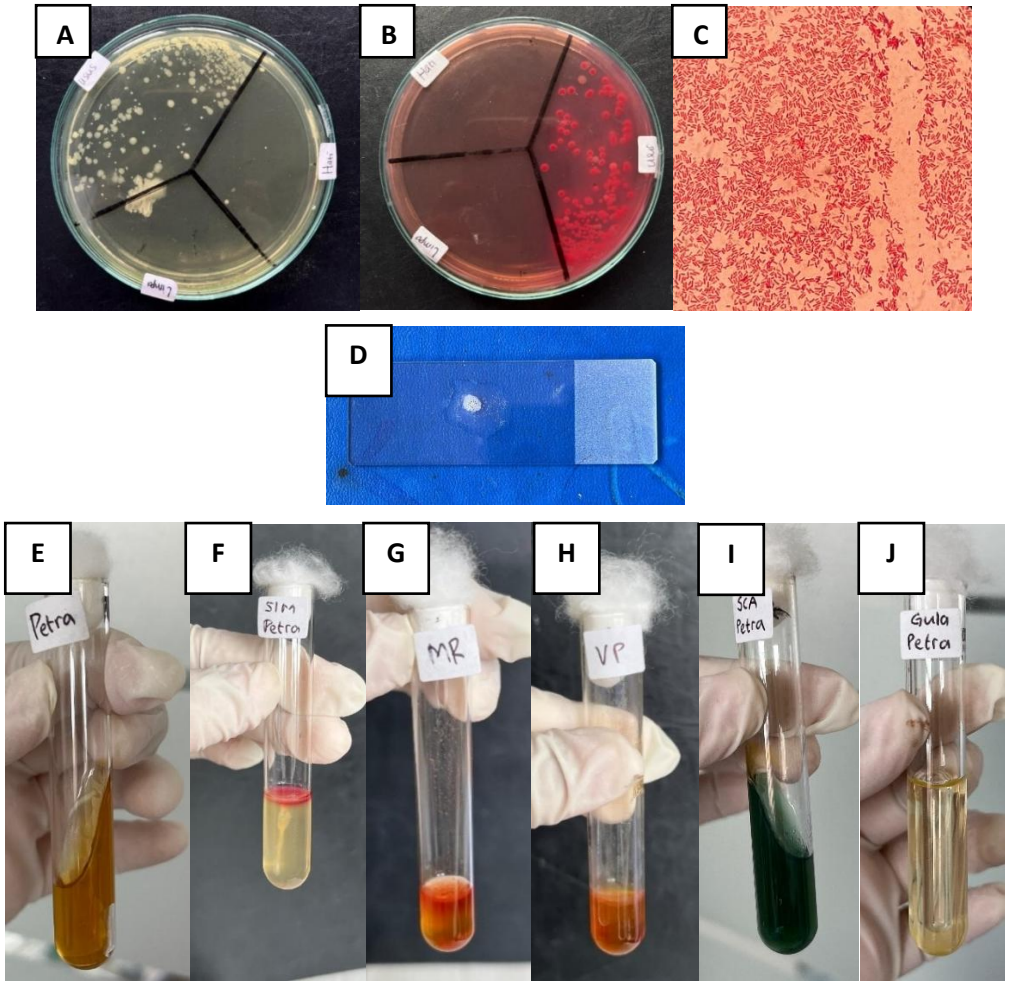

Figure 4. Bacterial Isolation Results: (A) Nutrient Agar; (B) MacConkey Agar; (C) Gram Staining; (D) Catalase Test – Positive; (E) TSIA – Positive; (F) SIM – Positive; (G) MR (Methyl Red) – Positive; (H) VP (Voges–Proskauer) – Negative; (I) SCA (Simmons Citrate Agar) – Negative; (J) Glucose Test – Positive.

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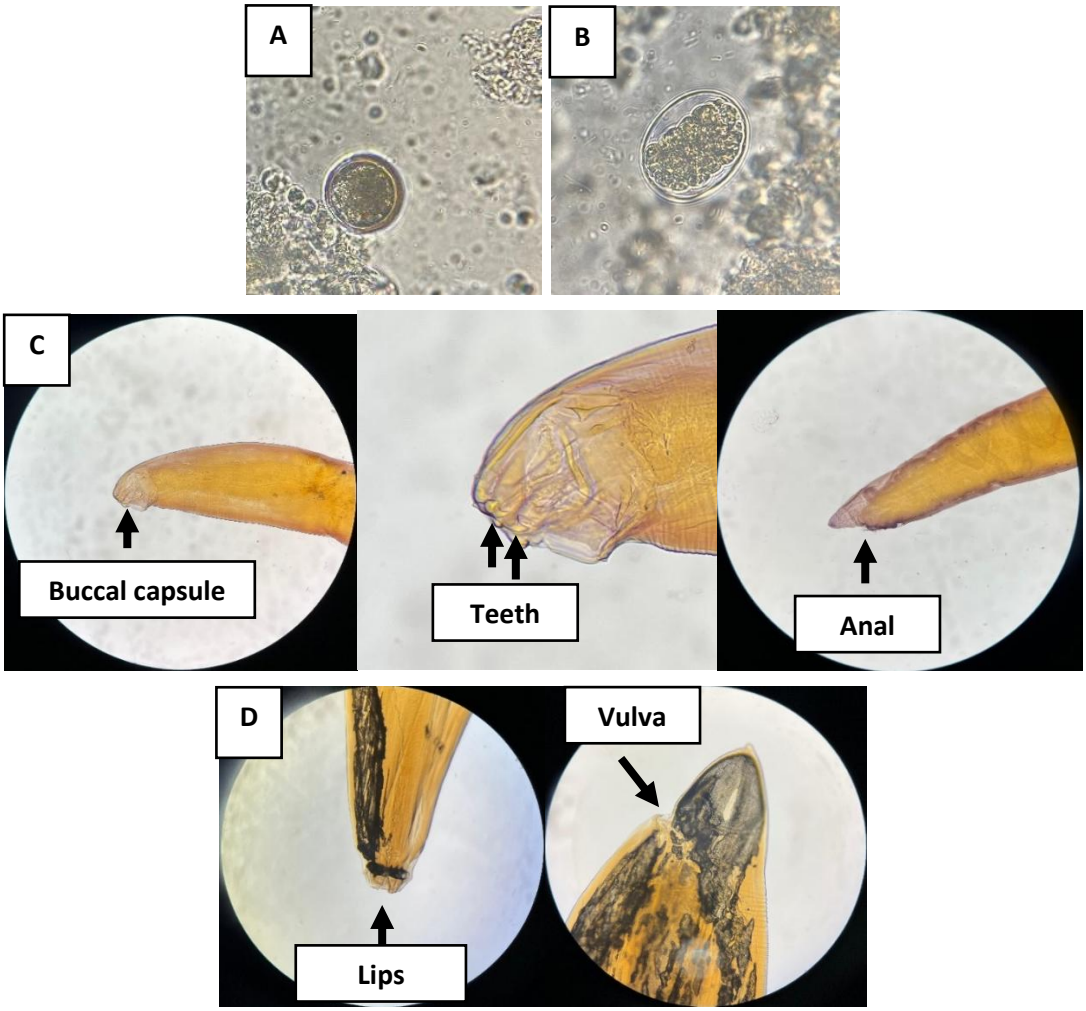

Figure 5. (A) Toxocara canis egg; (B) Ancylostoma caninum egg; (C) Ancylostoma caninum adult worm; (D) Toxocara canis adult worm.

Discussion

Canine parvoviral disease is caused by Canine parvovirus type 2 (CPV-2). It is one of the most contagious and deadly viral diseases in dogs (Purnamasari et al., 2015). The virus remains stable over a wide pH range and can survive heat at 60°C for 1 hour. CPV lacks an outer envelope and is resistant to certain chemicals. However, it can be destroyed by substances such as 1% formalin, certain disinfectants, and ultraviolet light. Following the identification of CPV as a highly resilient pathogen, its emergence was first reported in the United States. It affected dogs presenting with enteritis and/or myocarditis. Subsequently, CPV gained widespread attention after detection in countries such as Australia, Europe, Canada, the United Kingdom, and New Zealand. Natural transmission of CPV occurs through direct contact with infected dogs or ingestion of contaminated feed. The virus is shed in canine feces, urine, and saliva. There is also potential transmission through vomitus (Sendow, 2003).

Since CPV has spread worldwide, veterinarians diagnose parvoviral disease by taking a history, assessing symptoms, performing a physical examination, and conducting laboratory tests. These tests include rapid kits, blood work, ELISA, PCR, and fluorescent antibody tests (Uzuegbu, 2015). In this case, PCR was used to confirm CPV infection. Regarding clinical presentation, early manifestations of CPV infection are generally characterized by depression, anorexia, and fever, which typically progress within one to two days to vomiting and

hemorrhagic diarrhea (Jaya et al., 2022). For the case subject, a dog, the clinical course began four days before death with serous nasal discharge, lethargy, and watery diarrhea, then advanced during the terminal two days to profuse hemorrhagic diarrhea with a characteristic foul odor, adynamia, and intolerance of oral fluid intake.

Epidemiologically, the virus predominantly affects puppies aged 2–6 months (Sardjana & Kusumawati, 2004). Pathologically, CPV-2 infection is classified into two main clinical forms: myocarditis and enteritis. The enteric form is characterized by vomiting and bloody diarrhea with a distinctive fecal odor (Jedaut et al., 2021). Infection is more prevalent in puppies older than 2 months (Purnamasari et al., 2015). The high incidence in young animals is closely associated with an abundance of actively dividing cells, which serve as targets for viral replication (Sendow, 2003). Because the dog showed vomiting, bloody diarrhea with a strong odor, and was two months old, it was diagnosed with the enteric form of Canine Parvovirus.

Building upon the diagnosis, gross pathological examination of the intestines revealed hemorrhage and an intestinal mucosal surface covered with thick yellowish-brown mucus (Figure 1C). In the enteric form, typical gross findings include intestinal hemorrhage, gallbladder enlargement, gastric petechiae, narrowing of the intestinal lumen, and an intestinal mucosal surface containing granular serous fluid to thick yellow-brown mucus. Gastric erosion also occurs (Jedaut et al., 2021). Under a microscope, the small intestine showed white blood cell buildup, damaged and shortened finger-like projections, bleeding, dead tissue in the digestive crypts, and damage to the lining. These showed that the intestine was badly inflamed and damaged.

Histopathological changes in the large intestine (Figure 2.B) included lymphocytic infiltration (yellow arrows), congestion (black arrows), and villous erosion (red arrows), suggesting colitis. The virus has a predilection for lymphoid tissues, resulting in lymphocyte depletion, and for the intestinal tract, causing crypt necrosis and destruction of the small-intestinal villi (Jedaut et al., 2021). Canine Parvovirus infects intestinal epithelial cells, leading to shortening of the intestinal villi. Fragments of necrotic cells mix with blood and are excreted in the feces, producing the characteristic foul odor (Sendow, 2003). Gross pathological examination of the spleen revealed organ enlargement and a darker coloration compared with normal (Figure 1B). Histopathological examination of the spleen (Figure 2C) demonstrated proliferation of lymphoid cells (black arrows), indicating splenitis.

In a dog, two adult worms, Toxocara canis and Ancylostoma were found in the intestine, and Toxocara canis and Ancylostoma caninum eggs were detected on fecal examination. The Ancylostoma caninum egg per gram (EPG) count was 100. The number of eggs per gram of feces (EPG) is a parameter used to assess the severity of helminth infection in the host. Based on the criteria that fewer than 5,000 Ancylostoma eggs indicate a mild infection (Bayou et al., 2025), the level of infestation in this case was classified as mild. Parasitic diseases in dogs generally have a low mortality rate (Savitri et al., 2020). The dog had likely been infected with parasites, which subsequently weakened its immune system. As a result, this weakened state presumably facilitated infection with Canine Parvovirus and accelerated death due to the accompanying immunosuppressive state.

Puppies are highly susceptible to infection with T. canis eggs, particularly because transmission often occurs via the intrauterine route (Savitri et al., 2020). Infection typically begins when puppies ingest infective eggs from the environment. Once inside the host, the eggs develop into second-stage larvae that migrate and form cysts in somatic tissues (Suroiyah et al., 2018). In female dogs, these somatic tissue cysts can later serve as a source of intrauterine infection during pregnancy, as larvae can penetrate the placenta and migrate to the fetus. Consequently, after birth, larvae are commonly found in the lungs of puppies (Savitri et al., 2020). In addition

to intrauterine transmission, T. canis can be transmitted via the transmammary route. Infection originates in the mammary glands and placenta. Larvae migrate and are transmitted to puppies during nursing. Eggs or larvae in colostrum can infect puppies during lactation (Supraptini, 2013). Larval migration may damage organs such as the liver and lungs (Savitri et al., 2020).

Ancylostoma caninum is a nematode parasite, a blood-sucking intestinal worm. The worms attach to the intestinal mucosa and frequently change attachment sites, leaving persistent bleeding wounds. They produce anticoagulant substances that prevent blood clotting at the site of attachment (Ananda et al., 2022). The life cycle of A. caninum begins when eggs passed in the feces hatch into first-stage larvae within 1–2 days under moist environmental conditions. Over approximately one week, these larvae develop into infective third-stage larvae capable of infecting susceptible hosts. Following this developmental period, the cutaneous penetration phase of Ancylostoma spp. larvae typically cause dermatitis in the interdigital regions, the feet, and occasionally the abdominal area (Ahada et al., 2020).

In this case, no evidence of additional bacterial infection was identified. Examination at the Veterinary Bacteriology and Mycology Laboratory, Faculty of Veterinary Medicine, Udayana University, revealed only Escherichia coli in the intestine, which remains part of the normal flora. This was supported by the observation that bacterial colony growth was non-septic and limited to the intestinal sample, with no colonies detected in other organs. In general, E. coli is recognized as part of the normal gastrointestinal flora of mammals (Rahayu et al., 2018) (Rahayu et al., 2018). However, this bacterium may become pathogenic if its population increases significantly or if it spreads beyond its normal site of colonization. Damage to the intestinal epithelium can predispose to secondary infection by normal intestinal flora, including Escherichia coli. Dissemination of the bacteria and their endotoxins into the bloodstream may lead to coliform septicemia, which can progress to septic shock and ultimately result in death (Sewoyo et al., 2022).

CONCLUSIONS AND SUGGESTIONS

Conclusions

This research concludes that the death of the two-month-old Mini Pomeranian puppy was caused by a Canine Parvovirus (CPV) infection, which was exacerbated by a concurrent intestinal parasitic infection (helminthiasis).

Suggestions

Viral and parasitic infections in dogs can be prevented through complete vaccination and routine deworming.

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REFERENCES

Ahada A.H.U, Kusuma I.D, & Yesica R. (2020). Case Report: Ancylostoma caninum, Trichuris vulpis, and Ctenocephalides canis Parasitic Infestation in Dogs. *Media Kedokteran Hewan*, *31*(3).

Hadi A.M. (2019). Morphological study of Toxocara canis (Werner, 1788) worms by scanning electron microscope. *International Journal of Biosciences*, *14*(3), 293–298.

Jedaut F.A, Rohi N.K, & Simarmata Y.T.R.M.R. (2021). Literature Review of Canine Parvovirus in Dogs. *Jurnal Veteriner Nusantara*, *4*(2), 1–6.

Suroiyah F.A, Hastutiek P, Yudhana A, Sunarso A, Purnama M.T.E, & Praja R.N. (2018). Prevalence of Toxocara cati Infection in Pet Cats in Banyuwangi District. *Jurnal Medik Veteriner*, *1*(3), 99–104.

Sendow I. (2003). Canine Parvovirus in Dogs. *Wartazoa*, *13*(2), 56–64.

Purnamasari I.A.A, Berata I.K, & Kardena I.M. (2015). Histopathological Study of Intestinal and Cardiac Organs in Dogs Infected with Parvovirus. *Buletin Veteriner Udayana*, *7*(2), 99–104.

Winaya I.B.O, Berata I.K, Adi A.A.A.M, & Kardena I.M. (2014). Pathological Aspects of Parvovirus Infection in Puppies in Denpasar City. *Jurnal Kedokteran Hewan*, *8*(2), 85–89.

Suartha I.K, Mustikawa D, Erawan I.G.M.K, & Widyastuti S.K. (2011). Prevalence of Parvovirus Disease in Dogs in Denpasar. *Journal Veteriner*, *12*(3), 235–240.

Sardjana I.K.W, & Kusumawati D. (2004). Treatment of Parvovirus Infection in Dogs. *Berkelanjutan Penelitian Hayati*, *10*, 81–83.

Jaya I.M.A.M, Putriningsih P.A.S, & Soma I.G. (2022). Case Report: Ancylostoma caninum, Trichuris vulpis, and Ctenocephalides canis Parasitic Infestation in Dogs. *Buletin Veteriner Udayana*, *14*(1), 43–49.

Hermawan I.P, Darantika G, & Lonai N.B. (2022). Case Study: Canine Parvovirus in a Boston Terrier at Lingka Satwa Animal Care. *Jurnal Vitek Bidang Kedokteran Hewan*, *12*(1).

Prittie J. (2004). Canine parvoviral enteritis: A review of diagnosis, management, and prevention. *Journal of Veterinary Emergency and Critical Care*, *14*(3), 167–176.

Supraptini J. (2013). Cases of Toxocariasis in Dogs at the Teaching Veterinary Hospital, Airlangga University. *Media Vetmedika: Journal of Veterinary Clinics*, *2*(1), 18–24.

Joesoef J.A, & Larasati G.W.K.D. (2025). Case Report: Toxocara canis Parasitic Infestation in a Dog. *Jurnal Veteriner Nusantara*, *8*(2).

Bayou K, Getachew T, & Bersissa K. (2025). Epidemiology of Gastrointestinal Parasites of Dogs in Four Districts of Central Ethiopia: Prevalence and Risk Factors. *PLoS One*, *20*(1).

Ananda M.D, Anthara M.S, Erawan I.G.M.K, & Jayanti P.D. (2022). *Case Report: Hookworm (Ancylostomiasis) Infection Causing Ascites in a Four-Month-Old Pomeranian Crossbreed Dog*. *11*(4), 507–518.

Spindel M.E, Krecic M.R, Slater M.R, & Vigil N. (2018). Evaluation of a Community's Risk of Canine Parvovirus and GIS Mapping of Animal Shelter Intakes. *Journal of Applied Animal Welfare Science*, *21*(4), 362–374.

Levine N.D. (1994). *Veterinary Parasitology Textbook*. Gadjah Mada University Press.

Uzuegbu O.M. (2015). *Case Report on Suspected Parvoviral Enteritis in a Dog*. VeterinaryTeaching Hospital, Michael Okpora University of Agriculture.

Sewoyo P.S, Winaya I.B.O, Berata I.K, Adi A.A.A.M, Dayanti M.D, Grahadi R, & Takariyanti D.N. (2022). Case Report: Clinicopathological Findings in a Puppy with Enteritis and Myocarditis Due to Canine Parvovirus Infection. *Buletin Veteriner Udayana*, *14*(6), 693–704.

Savitri R.C, Oktaviana V, & Fikri F. (2020). Toxocara canis Infection in Local Dogs in Banyuwangi. *Jurnal Medik Veteriner*, *3*(1), 127–131.

Koesdarto S, Subekti S, & Setiawan H. (2001). Control Model of Bovine Toxocariasis Infection Cycle Using Fractionated Essential Oil of Temuireng Rhizome (Curcuma aeruginosa Roxb.). *JPMC*, *2*, 114–122.

Rahayu W.P, Nurjanah, & Komalasari E. (2018). *Escherichia coli: Pathogenesis, Analysis and Risk Assessment*. IPB Press.