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CASE REPORT: LOCAL MIX BREED DOGS MORTALITY DUE TO CANINE PARVOVIRUS INFECTION ACCOMPANIED BY *ENTAMOEBA* SPP.

Laporan Kasus: Kematian Anjing Ras Campuran Lokal Akibat Infeksi Parvovirus Disertai *Entamoeba* spp.

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Abstract

Canine parvovirus is caused by canine parvovirus type 2 (CPV-2), which is highly contagious and has a high mortality rate, particularly in young dogs. CPV-2 infection in young dogs is often accompanied by secondary bacterial or parasitic infections, such as *Entamoeba* spp. This case report aimed to determine the cause of death in a four-month-old mixed-breed puppy that exhibited clinical signs of gastrointestinal distress. The clinical signs included anorexia, lethargy, clear vomiting, and bloody diarrhea lasting more than three days. The cause of death was determined through a comprehensive evaluation, including medical history, postmortem examination, histopathology, molecular viral identification, and parasitological and bacteriological examinations. The medical history revealed that the death occurred not only in the examined puppy but also in two other puppies of the same litter. Gross pathology findings revealed cerebral congestion, cardiac enlargement, pulmonary and hepatic hemorrhage, splenomegaly, renal medullary hyperemia, and intestinal hemorrhage. Histopathological findings confirmed edematous myocarditis, lymphoid cell depletion in the spleen, and necrotizing enteritis, which are characteristic of viral infections. Polymerase chain reaction (PCR) test results showed that the puppies were infected with CPV-2, as confirmed by the

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detection of a 910-base-pair amplicon on electrophoresis when compared to controls. *Escherichia coli* was confirmed to have grown from the cultured intestinal isolate, which was confirmed to be part of the normal flora of the intestine. Parasitological examination confirmed the presence of *Entamoeba* spp. in the fecal sample. These findings confirm that the cause of death in the puppy was canine parvovirus complicated by entamoebiasis.

Keywords: CPV-2, Entamoebiasis, Gross Pathology, Parvo, Puppies

Abstrak

Parvo pada anjing disebabkan oleh Canine Parvovirus tipe 2 (CPV-2) yang bersifat sangat menular dan memiliki tingkat mortalitas tinggi, terutama pada anjing usia muda. Infeksi CPV-2 pada anjing muda sering disertai oleh infeksi sekunder bakteri atau parasite, salah satunya adalah *Entamoeba* spp. Laporan kasus ini bertujuan untuk menentukan penyebab kematian seekor anak anjing persilangan berumur empar bulan yang mengalami tanda klinis gangguan pencernaan. Tanda klinis tersebut meliputi anoreksia, lemas, vomitus bening, dan diare berdarah selama lebih tiga hari. Penyebab kematian ditentukan dengan pemeriksaan komprehensif meliputi anamnesis, pemeriksaan post-mortem, histopatologis, identifikasi virus secara molekuler, dan pemeriksaan parasitologi dan bakteriologi. Anamnesis menemukan bahwa kematian tidak hanya terjadi pada anak anjing yang diperiksa namun terjadi pada dua anak anjing lainnya. Temuan patologi anatomi menunjukkan kongesti otak, pembengkakan jantung, hemoragi paru dan hati, splenomegali, hiperemia medula ginjal, serta perdarahan usus. Temuan histopatologi mengkonfirmasi *myocarditis edematosa, deplesi sel limfoid* pada limpa *dan enteritis necrotican* yang merupakan ciri khas infeksi virus. Hasil pemeriksaan Polymerase Chain Reaction (PCR) menunjukkan bahwa anak anjing terinfeksi CPV-2, terkonfirmasi dari temuan amplikon 910 *base pair* pada elektroforesis setelah dibandingkan dengan kontrol. *Escherichia coli* terkonfirmasi tumbuh dari isolat usus yang ditanam, yang terkonfirmasi sebagai flora normal. Pemeriksaan parasitologi mengkonfirmasi *Entamoeba* spp. pada sampel feses. Temuan – temuan pemeriksaan tersebut mengkonfirmasi penyebab kematian anak anjing adalah canine parvovirus disertai entamobiasis.

Kata kunci: Anak anjing, CPV-2, Entamobiasis, Parvo, Patologi Anatomi

INTRODUCTION

The puppy stage (<6 months of age) is a period of life in dogs that is particularly susceptible to infectious diseases. During this stage, the immune system has not yet fully developed, making puppies more susceptible to various diseases (Prittie, 2004). One of the most common clinical signs experienced by puppies is diarrhea. The major risk factors associated with diarrhea in dogs include infectious agents and demographic and immunological factors, such as age, breed, and immune status, with immunosuppressed individuals showing the greatest susceptibility (Aurélien *et al*., 2014; Stavisky *et al*., 2011). Meanwhile, changes in dietary patterns, group-based environmental management, and the presence of other systemic diseases are considered secondary risk factors with moderate levels of evidence (Alves *et al*., 2021; Stavisky *et al*., 2011). The major types of diarrhea in puppies include acute, hemorrhagic, chronic, and recurrent diarrhea, with viral infections, particularly parvovirus infection, being among the most common causes. Bacterial, parasitic, and non-infectious causes also contribute significantly to diarrhea in dogs (Aurélien *et al*., 2014).

Bloody diarrhea in dogs is most commonly associated with canine parvovirus (CPV) infection (Saputri *et al*., 2025). Nevertheless, bloody diarrhea may also result from other causes. *Clostridium perfringens* and *Clostridioides difficile* are frequently associated with bloody diarrhea in dogs. Overgrowth and toxin release by these bacteria play an important role in the

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pathogenesis of acute hemorrhagic diarrhea syndrome (AHDS) (Stavisky *et al*., 2011). *Salmonella* spp. may also be detected in dogs with bloody diarrhea, although their role as a primary cause remains unclear (Stavisky *et al*., 2011). Bloody diarrhea may also be caused by diseases outside the gastrointestinal system, such as poisoning, hypoadrenocorticism (Addison's disease), renal failure, or hepatic failure. These causes are generally ruled out through history taking, physical examination, and laboratory examinations (Gouveia *et al*., 2011).

This case report describes the occurrence of death in three four-month-old puppies, with one puppy used as a necropsy sample. The investigation of the deaths began with history taking and continued through laboratory diagnostic confirmation. None of the puppies had previously received any vaccinations, and all died within 7 days of the onset of clinical signs, including anorexia, vomiting, and bloody diarrhea. No treatment was administered by the owners. Following necropsy, pathological changes were observed in the heart and gastrointestinal tract, suggesting an acute and fatal systemic disease suspected to be associated with an infectious disease. Based on the investigation, the provisional diagnosis of the cause of death was CPV infection, with coccidiosis and bacterial enteritis considered as differential diagnoses. Therefore, this case report aimed to establish the definitive diagnosis of the cause of death in the affected puppy through a comprehensive approach, beginning with history taking and continuing through laboratory examinations. Accurate diagnosis is an essential basis for determining appropriate control and management strategies to prevent similar occurrences.

MATERIALS AND METHODS

Case Animal

A four-month-old male mixed-breed local dog with a brown and white coat was examined. The dog belonged to an owner who kept three puppies in Sumerta Kelod Village, South Denpasar, Denpasar, Bali (protocol number 196/N/25). The puppy died on the fourth day after the onset of clinical signs.

History Taking and Environmental Investigation

To further investigate the case, history taking was conducted through direct interviews with the owner to obtain information regarding the disease history, husbandry system and management practices, vaccination status, treatment history, and environmental conditions that might have contributed as risk factors for the animal's death. Environmental investigation was conducted through direct observation of the puppy's housing conditions by the authors.

Necropsy and Gross Pathological Examination

Necropsy was performed at the Laboratory of Veterinary Pathology, Faculty of Veterinary Medicine (FVM), Udayana University (Unud), to observe gross pathological changes and document them as supporting data. At the time of necropsy, the case animal was already dead. First, the existing clinical manifestations on the body of the case animal were examined and documented before incision. After documentation, the hair around the surgical area was removed. A longitudinal skin incision was then made from the abdominal region toward the head. Subsequently, the thoracic muscles were incised to allow the organs to be examined individually and comprehensively. Samples from each organ were collected and divided into three portions: one portion measuring approximately 1 cm^3 for histopathological examination and two portions measuring approximately 2 cm^3 for virological and bacteriological examinations. Fecal samples were collected from the gastrointestinal tract for parasitological examination.

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Histopathological Examination

Histopathological preparations were performed at the Laboratory of Veterinary Pathology, FVM, Unud. Organ samples showing prominent 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 Laboratory of Veterinary Pathology, and tissue sections were stained with Hematoxylin and Eosin (HE) (Sewoyo *et al*., 2025). The prepared slides were subsequently examined under a microscope for histopathological evaluation.

Molecular Detection of CPV Using Polymerase Chain Reaction (PCR)

CPV infection can be confirmed using PCR, which was performed at the Laboratory of Veterinary Virology, Faculty of Veterinary Medicine, Udayana University. Polymerase Chain Reaction (PCR) is an in-vitro technique used to amplify DNA. This technique synthesizes and amplifies only the desired DNA region selected by the researcher or diagnostician (Mahardika *et al*., 2015). PCR reactions were prepared using template DNA obtained from the case animal (protocol number 196/N/25). The organ sample was subsequently used for viral DNA isolation. DNA isolation was performed using the GeneJET Genomic DNA Purification Kit (Thermo Scientific, Massachusetts, United States), following the manufacturer's instructions. The PCR assay was initiated by preparing the DNA isolate from the case sample (protocol number 121/N/25), master mix containing PCR buffer, dNTPs, $MgCl_2$, and Taq polymerase enzyme, HFORM and VPRM primers specific for CPV, positive-control DNA isolate, negative-control DNA isolate, and distilled water. The amplification products were subsequently analyzed by electrophoresis on a 1% agarose gel at a constant voltage of 100 V for approximately 30 minutes. DNA visualization was then performed using an ultraviolet (UV) transilluminator, and the resulting images were documented (Mahardika *et al*., 2015).

Bacterial Isolation and Identification

To investigate the suspected bacterial enteritis and possible septicemia, bacterial isolation and identification were performed at the Laboratory of Veterinary Bacteriology and Mycology, FVM, Unud. Bacteria were isolated from the heart, spleen, and intestine using Nutrient Agar (NA). After colonies were obtained on the initial NA medium, one colony was selected and subcultured onto fresh NA medium to obtain pure colonies. Bacterial colonies from the NA subculture were subjected to Gram staining. The subcultured colonies were subsequently cultured on selective MacConkey Agar (MCA). In addition, a primary catalase test was performed to determine the ability of the bacteria to produce the catalase enzyme. The subsequent biochemical tests were performed using Triple Sugar Iron Agar (TSIA), Sulfide Indole Motility (SIM), Methyl Red Voges Proskauer (MRVP), Simmons Citrate Agar (SCA), and glucose tests (McVey *et al*., 2013).

Identification of *Entamoeba* spp. Through Direct Smear and Sedimentation Examination

Detection of gastrointestinal protozoa (*Entamoeba* spp.) from fecal samples was performed using direct examination, sedimentation, and saturated salt flotation methods. Protozoal examination was performed using the direct smear method by mixing fecal samples preserved in 10% formalin with physiological saline to form a thin layer on a glass slide. Lugol's iodine was subsequently added to enhance the visualization of the internal morphology of the cysts. The preparations were examined under a microscope at 100–400× magnification (Zajac *et al*., 2021).

A simple distilled-water sedimentation method was also used to concentrate *Entamoeba* spp. Approximately 2 g of fecal sample was homogenized in distilled water to obtain an

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approximately 10% suspension (approximately 30 mL), followed by filtration to remove coarse debris. The filtrate was transferred into a centrifuge tube to approximately three-quarters of its volume and centrifuged at 1,500 rpm for 2–3 minutes. After removal of the supernatant, a small amount of the sediment was collected and homogenized with 1–2 drops of Lugol's iodine on a glass slide. The preparation was covered with a coverslip and examined under a microscope at up to 400× objective magnification to identify *Entamoeba* cysts and trophozoites (Dwinata *et al*., 2017).

RESULTS AND DISCUSSION

Results

Based on the history, the case animal was one of three dogs kept exclusively indoors without access to the outdoor environment and fed commercial dry food twice daily. None of the dogs had a history of medical prophylaxis, including vaccination against canine distemper, canine parvovirus, infectious canine hepatitis, canine parainfluenza, and rabies, or routine anthelmintic administration. All three dogs developed acute clinical manifestations characterized by hemorrhagic diarrhea, emesis, and anorexia for three days. The case recorded a mortality rate of 100%, with two dogs dying on the third day after the onset of clinical signs, while the case dog was presented on the fourth day in a severely lethargic and moribund condition before dying later that afternoon.

Gross pathological examination identified pathological lesions consisting of vascular congestion in the cerebral sulci, multifocal discoloration accompanied by pulmonary hemorrhage, and multisystemic congestion involving the trachea, myocardium, liver, kidneys, spleen, and gastrointestinal tract. Cardiomegaly with a rounded cardiac apex was also observed (Figure 1), whereas the esophagus showed no significant lesions. Histopathological evaluation confirmed systemic inflammation predominantly characterized by mononuclear cell infiltration, consisting of lymphocytes and macrophages. The spectrum of microscopic lesions included meningoencephalitis, edematous myocarditis, hemorrhagic glomerulonephritis, and extensive necroinflammatory lesions comprising necrotizing gastritis, necrotizing enteritis, and colitis throughout the gastrointestinal tract (Figure 2).

Molecular confirmation using PCR followed by agarose gel electrophoresis identified the presence of CPV. Samples from the case animal consistently showed a specific amplicon band measuring 910 base pairs (bp), corresponding to the positive control, whereas no amplification band was detected in the negative control (Figure 3).

Based on colony morphology on Nutrient Agar and MacConkey Agar, confirmed by primary confirmatory tests, biochemical profiles, and carbohydrate fermentation tests, the isolate obtained from the intestine of the case dog was identified as *Escherichia coli*. Microscopic characterization confirmed the isolate as a Gram-negative, short rod-shaped, non-spore-forming bacterium, consistent with the phenotypic criteria described by Dewandaru *et al.* (2019). In this case, *E. coli* was considered part of the normal intestinal flora and was not considered to have contributed to the death of the case dog.

Parasitological examination of the fecal sample using wet mount and sedimentation methods confirmed the presence of *Entamoeba* spp. cysts. Morphological evaluation showed spherical cysts with thin, transparent, and resistant cyst walls. Following Lugol's iodine staining, the preparations showed characteristic brownish cysts, although the internal nuclear structures were not clearly observed (Figure 4).

Discussion

In this case, the mortality rate among the puppies reached 100%, accompanied by emesis and

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bloody diarrhea, with no medical intervention and no history of vaccination. Mortality is influenced by a combination of progressive systemic pathophysiological factors and delayed medical intervention, particularly in young individuals and unvaccinated populations (Kelman *et al.*, 2020). The tropism of CPV for actively dividing cells in the intestinal mucosa and bone marrow induces severe hemorrhagic enteritis and emesis, rapidly resulting in severe dehydration and hypovolemic shock (Bako *et al.*, 2025; Santana *et al.*, 2019). Damage to hematopoietic tissues results in severe leukopenia and immunosuppression, thereby facilitating secondary bacterial translocation, sepsis, and multisystem organ failure (Alves *et al.*, 2021). The high virulence of CPV, extensive transmission, and rapid disease progression, often without specific early clinical manifestations, allow irreversible lesions to develop before medical treatment is initiated (Oleiwi *et al.*, 2025). Without prompt and aggressive supportive therapy, such as intravenous fluid therapy and sepsis management, the combination of acute dehydration, immune system failure, and severe infection may ultimately result in death (Mazzaferro, 2020).

Cardiomegaly accompanied by edematous myocarditis was observed in the pathological findings of this case. This manifestation is primarily associated with the tropism and direct cytopathic effects of the virus on actively dividing cardiomyocytes, particularly in puppies (Saputri *et al.*, 2025; Stancu *et al.*, 2025). Direct infection induces cardiomyocyte destruction and necrosis, which subsequently triggers an acute inflammatory response characterized by diffuse infiltration of mononuclear inflammatory cells and accumulation of interstitial fluid (edema) within the myocardial parenchyma (Ford *et al.*, 2017). The combination of extensive cellular infiltration and interstitial edema causes significant swelling of the myocardial tissue, which is macroscopically manifested as cardiomegaly. This edematous necrotizing myocarditis may progress rapidly, resulting in acute respiratory distress and sudden death (Sime *et al.*, 2015; Souto *et al.*, 2018).

The pathological damage observed in this case was extensive, involving the gastrointestinal tract from the stomach to the large intestine. This damage is associated with the selective tropism of the virus for actively dividing mucosal epithelial cells (Prittie, 2004). The infection produces direct cytopathic effects that result in extensive cellular destruction and necrosis in the stomach, small intestine, and large intestine (Prittie & Medicine, 2004; Schoeman *et al.*, 2013). Destruction of the epithelium and disruption of the mucosal barrier induce a severe local and systemic inflammatory response while facilitating the translocation and colonization of secondary bacteria, thereby exacerbating the inflammatory cascade and expanding tissue necrosis (Schoeman *et al.*, 2013). The synergistic effects of viral cytopathic destruction, the pro-inflammatory cascade, and secondary infection, which are also influenced by the virulence of the CPV strain, collectively contribute to the development of diffuse necroinflammatory lesions throughout the gastrointestinal tract (Schoeman *et al.*, 2013).

In this case, *E. coli* was cultured only from the intestine. *E. coli* was considered part of the normal intestinal flora and was not considered to have contributed to the infection. The role of *E. coli* as a secondary pathogen during CPV infection occurs during the acute phase of disease, particularly after extensive viral destruction of the intestinal epithelium compromises the integrity of the intestinal mucosal barrier (Rzezutka *et al.*, 2003). Damage to the mucosal wall facilitates the translocation of *E. coli*, which may act as an opportunistic bacterium, from the intestinal lumen into the systemic circulation and visceral organs (Rzezutka *et al.*, 2003). Immunosuppression resulting from CPV infection may further increase the pathogenic potential of enterotoxigenic *E. coli* strains carrying the *estI* enterotoxin gene. This interaction may trigger a secondary bacterial infection cascade and exacerbate enteritis, potentially leading to severe sepsis and significantly worsening the prognosis and increasing patient mortality

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(Rzezutka *et al.*, 2003).

Entamoebiasis occurred concurrently with CPV infection in this case. Entamoebiasis is commonly caused by *Entamoeba histolytica*, although several other *Entamoeba* species have also been identified as etiological agents (Al Haider *et al.*, 2026; Ngui *et al.*, 2020). Transmission of these parasites occurs through the fecal–oral route following ingestion of food or water contaminated with *Entamoeba* cysts, as well as through close contact with infected humans or animals (Ngui *et al.*, 2020). Clinically, most *Entamoeba* infections in dog populations are asymptomatic, with dogs acting as carriers without exhibiting significant health disturbances (Ngui *et al.*, 2020). Intestinal amebiasis accompanied by gross morphological lesions and pathognomonic clinical signs is relatively uncommon; however, when clinical manifestations develop, the predominant abnormality is gastrointestinal disturbance characterized by diarrhea (Ngui *et al.*, 2020).

CPV-2 infection can induce significant structural damage to the intestinal epithelium, characterized by crypt abscess formation, mucosal erosion, and severe hemorrhagic enteritis, which markedly increase intestinal permeability and compromise the host's physical barrier function (Li *et al.*, 2025; Zhou *et al.*, 2025). Concurrently, CPV-2 tropism for actively dividing progenitor cells in the bone marrow and lymphoid tissues causes severe systemic immunosuppression characterized by progressive leukopenia (Ashley *et al.*, 2025; Muñoz *et al.*, 2021; Zhou *et al.*, 2025). The combination of impaired mucosal integrity and reduced cellular immune surveillance facilitates the development of an intestinal microenvironment conducive to the adhesion, colonization, and tissue penetration of secondary or opportunistic pathogens, including potential invasion by *Entamoeba* spp. (Li *et al.*, 2025; Muñoz *et al.*, 2021; Yu *et al.*, 2015; Zhou *et al.*, 2025). Invasion by *Entamoeba* spp., which produce cysteine proteinase and amebapores that damage the extracellular matrix as well as mucosal and submucosal cells of the large intestine (Melendez-Lopez, 2007), may further exacerbate the clinical manifestations occurring concurrently with CPV infection.

CONCLUSION AND SUGGESTIONS

Conclusion

This case report demonstrates that the 100% mortality rate in the unvaccinated puppy population was primarily caused by molecularly confirmed CPV infection. CPV induced diffuse necrotizing enteritis and fatal edematous myocarditis. Disruption of the mucosal barrier and severe immunosuppression caused by CPV facilitated opportunistic coinfection with *Entamoeba* spp. Thus, the rapid progression to death, occurring within less than 7 days, was the result of a complex pathophysiological synergy involving viral destruction, cardiovascular dysfunction, and secondary parasitic invasion in a susceptible host without adequate immunological protection.

Suggestions

Animal owners and veterinary practitioners are strongly encouraged to ensure compliance with CPV vaccination protocols and regular antiparasitic administration from an early age, as indoor housing does not guarantee protection from exposure to potentially fatal pathogens. Clinically, the management of hemorrhagic diarrhea should incorporate screening for parasitic coinfections alongside early and aggressive supportive fluid therapy. For further research, genomic sequencing is recommended to identify local CPV strain variants and to investigate the molecular mechanisms underlying the interaction between parvovirus-induced tissue damage and opportunistic parasitic invasion in the gastrointestinal tract.

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REFERENCES

Al Haider, H. I. A., Yakhchali, M., & Tavassoli, M. (2026). Prevalence and associated risk factors of *Entamoeba* infection among humans and dogs in Maysan City, South East of Iraq. *Journal of Parasitic Diseases*. https://doi.org/10.1007/s12639-026-01971-y

Alves, J. C., Jorge, P., & Santos, A. (2021). A survey on the prevalence of diarrhea in a Portuguese population of police working dogs. *BMC Veterinary Research, 17*, 211. https://doi.org/10.1186/s12917-021-02920-y

Ashley, Kencana, G. A. Y., Besung, I. N. K., Dwinata, I. M., & Kardena, I. M. (2025). Complications of canine parvovirus infection with *Streptococcus canis* in a free-roaming mixed breed dog: A case study with anatomical pathology and histopathological. *Buletin Veteriner Udayana, 17*(4), 1177–1185. https://doi.org/10.24843/bulvet.2025.v17.i04.p04

Grellet, A., Chastant-Maillard, S., Robin, C., Feugier, A., Boogaerts, C., Boucraut-Baralon, C., Grandjean, D., & Polack, B. (2014). Risk factors of weaning diarrhea in puppies housed in breeding kennels. *Preventive Veterinary Medicine, 117*(1), 260–265. https://doi.org/10.1016/j.prevetmed.2014.07.016

Bako, E. S., Putra, I. P. C., Winaya, I. B. O., Mahardika, I. G. N. K., & Besung, I. N. K. (2025). Fatal canine parvovirus infection with secondary bacterial and parasitic complications in a puppy: A case report. *Buletin Veteriner Udayana, 17*(4), 1160–1167. https://doi.org/10.24843/bulvet.2025.v17.i04.p13

Dewandaru, R. A., Indarjulianto, S., Yanuartono, Nururrozi, A., Purnamaningsih, H., & Hayati, R. (2019). Diare disebabkan infeksi *Escherichia coli* pada anjing. *Jurnal Ilmu Peternakan dan Veteriner Tropis (Journal of Tropical Animal and Veterinary Science), 9*(2), 38–43. https://doi.org/10.30862/jipvet.v9i2.22

Dwinata, I. M., Apsari, I. A. P., Suratma, N. A., & Oka, I. B. M. (2017). *Modul identifikasi parasit cacing*. Fakultas Kedokteran Hewan, Universitas Udayana.

Ford, J., McEndaffer, L., Renshaw, R., Molesan, A., & Kelly, K. (2017). Parvovirus infection is associated with myocarditis and myocardial fibrosis in young dogs. *Veterinary Pathology, 54*(6), 964–971. https://doi.org/10.1177/0300985817725387

Gouveia, E. M. F., Silva, I. S., Nakazato, G., Araujo, F. R. de, & Chang, M. R. (2011). Experimental infection with enteropathogenic *Escherichia coli* identified by PCR using enteric-coated capsules in boxer pups. *Acta Cirúrgica Brasileira, 26*(2), 144–148. https://doi.org/10.1590/S0102-86502011000200013

Li, H., Li, S., Pan, Y., & Shi, Q. (2025). Decoding canine parvovirus: Biomarkers for diagnosis and advances in vaccine development to address emerging challenges. *Frontiers in Veterinary Science, 12*, 1624275. https://doi.org/10.3389/fvets.2025.1624275

Mahardika, I. G. N. K., Astawa, I. N. M., Kencana, G. A. Y., Suardana, I. B. K., & Suarjana, I. G. K. (2015). *Teknik lab virus*. Udayana University Press.

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https://doi.org/10.24843/bulvet.2026.v18.i04.p30

Meléndez-López, S. G., Herdman, S., Hirata, K., Choi, M.-H., Choe, Y., Craik, C., Caffrey, C. R., Hansell, E., Chávez-Munguía, B., Chen, Y. T., Roush, W. R., McKerrow, J., Eckmann, L., Guo, J., Stanley, S. L., Jr., & Reed, S. L. (2007). Use of recombinant *Entamoeba histolytica* cysteine proteinase 1 to identify a potent inhibitor of amebic invasion in a human colonic model. *Eukaryotic Cell, 6*(7), 1130–1136. https://doi.org/10.1128/EC.00094-07

Muñoz, A. I., Vallejo-Castillo, L., Fragozo, A., Vázquez-Leyva, S., Pavón, L., Pérez-Sánchez, G., Soria-Castro, R., Mellado-Sánchez, G., Cobos-Marin, L., & Pérez-Tapia, S. M. (2021). Increased survival in puppies affected by canine parvovirus type II using an immunomodulator as a therapeutic aid. *Scientific Reports, 11*, 19864. https://doi.org/10.1038/s41598-021-99357-y

Ngui, R., Hassan, N. A., Nordin, N. M. S., Mohd-Shaharuddin, N., Chang, L. Y., Teh, C. S. J., Chua, K. H., Kee, B. P., Hoe, S. Z., & Lim, Y. A. L. (2020). Copro-molecular study of *Entamoeba* infection among the indigenous community in Malaysia: A first report on the species-specific prevalence of *Entamoeba* in dogs. *Acta Tropica, 204*, 105334. https://doi.org/10.1016/j.actatropica.2020.105334

Oleiwi, K. I., Hussein, M. A., Fahad, O. A., & Abdulrazzaq, S. O. (2025). Infection rate of canine parvovirus in dogs presented at private veterinary clinics in Baghdad city. *Open Veterinary Journal, 15*(1), 395–401. https://doi.org/10.5455/OVJ.2025.v15.i1.35

Prittie, J. (2004). Canine parvoviral enteritis: A review of diagnosis, management, and prevention. *Journal of Veterinary Emergency and Critical Care, 14*(3), 167–176. https://doi.org/10.1111/j.1534-6935.2004.04020.x

Rzezutka, A., Osek, J., & Mizak, B. (2003). Canine parvovirus and enterotoxigenic *Escherichia coli* causing the death of a puppy in a kennel. *Bulletin of the Veterinary Institute in Pulawy, 47*(2), 287–291.

Santana, W. de O., Lencina, M. M., Bertolazzi, S., Silveira, S., & Streck, A. F. (2019). Parvovírus canino: Uma abordagem evolutiva e clínica. *Medicina Veterinária (UFRPE), 13*(4), 526–533. https://doi.org/10.26605/medvet-v13n4-3661

Saputri, R. L., Suardana, I. B. K., Putra, I. P. C., Kardena, I. M., & Widyasanti, N. W. H. (2025). Fatal co-infection of canine parvovirus and *Toxocara canis* in a puppy: A case report from Denpasar, Bali. *Buletin Veteriner Udayana, 17*(5), 1657–1674. https://doi.org/10.24843/bulvet.2025.v17.i05.p15

Schoeman, J. P., Goddard, A., & Leisewitz, A. L. (2013). Biomarkers in canine parvovirus enteritis. *New Zealand Veterinary Journal, 61*(4), 217–222. https://doi.org/10.1080/00480169.2013.776451

Sewoyo, P., Permana, R., Putra, I. P., & Suandhika, P. (2025). Pathology of concurrent infection of *Metastrongylus apri* with *Trichuris suis* in a Yorkshire pig. *International Journal of Veterinary and Animal Research, 8*(2), 49–53. https://doi.org/10.5281/zenodo.17068531

Sime, T. A., Powell, L. L., Schildt, J. C., & Olson, E. J. (2015). Parvoviral myocarditis in a 5-week-old Dachshund. *Journal of Veterinary Emergency and Critical Care, 25*(6), 765–769. https://doi.org/10.1111/vec.12347

Souto, E. P. F., Olinda, R. G., Almeida, D. B. B., Rolim, V. M., Driemeier, D., Nobre, V. M. T., Riet-Correa, F., & Dantas, A. F. M. (2018). Surto de parvovirose cardíaca em filhotes de cães no Brasil. *Pesquisa Veterinária Brasileira, 38*(1), 94–98. https://doi.org/10.1590/1678-5150-PVB-4787

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https://doi.org/10.24843/bulvet.2026.v18.i04.p30

Stancu, A., Gros, R.-V., Luca, I., Călugărița, G.-A., Gavrilă, A., & Pașca, A.-S. (2025). Canine cardiac and cardiovascular pathology: Four major life-threatening non-degenerative, non-hereditary conditions. *Veterinary Sciences, 12*(11), 1060. https://doi.org/10.3390/vetsci12111060

Stavisky, J., Radford, A. D., Gaskell, R., Dawson, S., German, A., Parsons, B., Clegg, S., Newman, J., & Pinchbeck, G. (2011). A case-control study of pathogen and lifestyle risk factors for diarrhoea in dogs. *Preventive Veterinary Medicine, 99*(2–4), 185–192. https://doi.org/10.1016/j.prevetmed.2011.02.009

Yu, Q., Yuan, L., Deng, J., & Yang, Q. (2015). Lactobacillus protects the integrity of intestinal epithelial barrier damaged by pathogenic bacteria. *Frontiers in Cellular and Infection Microbiology, 5*, 26. https://doi.org/10.3389/fcimb.2015.00026

Zajac, A. M., Conboy, G. A., Little, S. E., & Reichard, M. V. (2021). *Veterinary clinical parasitology* (9th ed.). Wiley-Blackwell.

Zhou, H., Cui, K., Su, X., Zhang, H., Xiao, B., Li, S., & Yang, B. (2025). Overview of recent advances in canine parvovirus research: Current status and future perspectives. *Microorganisms*, *13*(1), 47. https://doi.org/10.3390/microorganisms13010047

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Figures

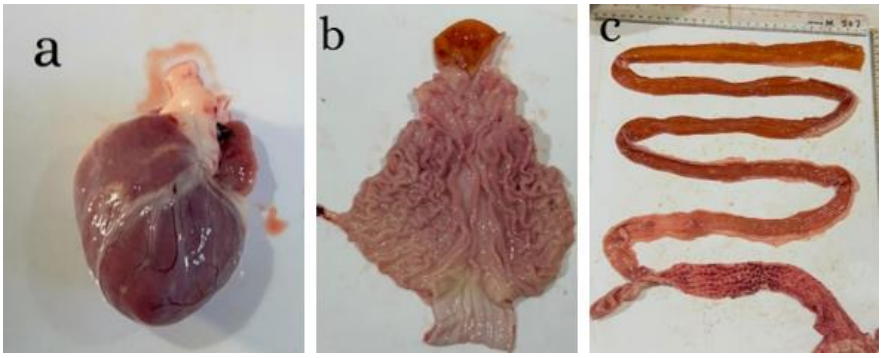

Figure 1. Gross pathological lesions of the visceral organs of the case dog. (a) Cardiomegaly characterized by enlargement of the heart; (b) Hemorrhage in the gastric mucosa; (c) Hemorrhage in the intestinal tract.

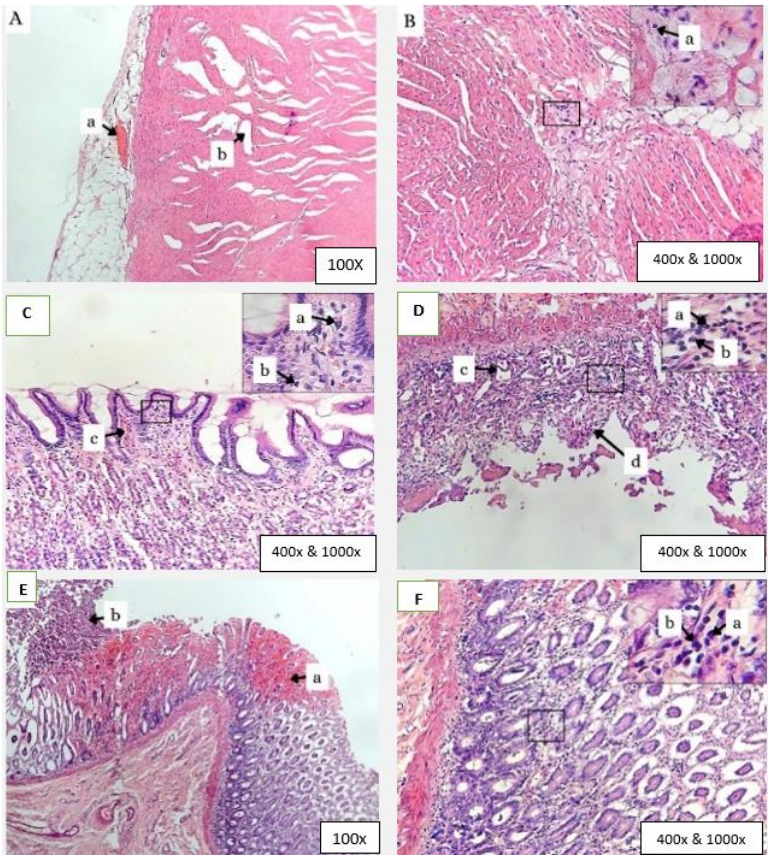

Figure 2. Histopathological findings of the visceral organs of the case dog. (A) Heart (edematous myocarditis): (a) epicardial congestion and (b) intermyocardial edema with infiltration of lymphocytic inflammatory cells. (B) Stomach (gastritis): (a) neutrophilic inflammatory cell infiltration and (b) lymphocytic infiltration in the mucosa, with (c) vascular congestion. (C–D) Small intestine (necrotizing enteritis): (a) lymphocytic infiltration, (b) eosinophilic infiltration, (c) crypt of Lieberkühn necrosis, and (d) villous atrophy. (E–F) Large intestine (colitis): (a) hemorrhage and (b) villous erosion; inflammatory cell infiltration consisting of (a) lymphocytes and (b) neutrophils in the lamina propria. (H&E staining).

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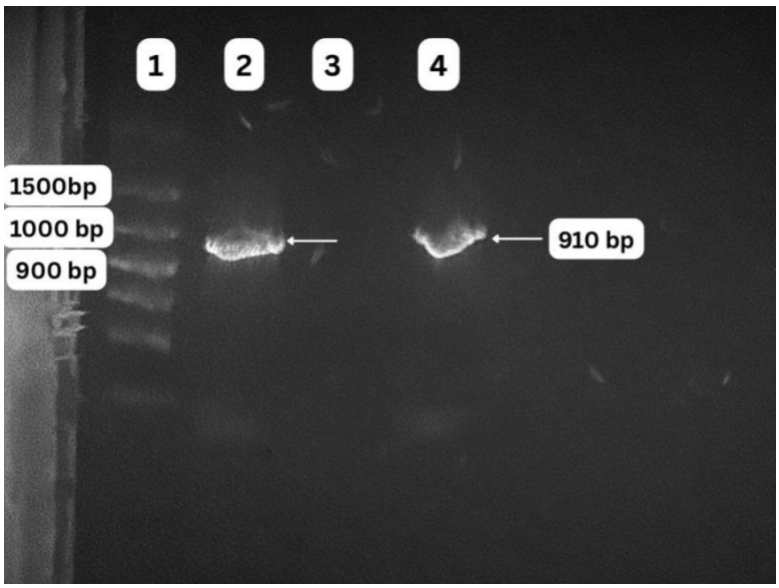

Figure 3. Agarose gel electrophoresis profile of PCR amplification of CPV. (1) DNA molecular marker; (2) Case dog specimen (Protocol No. 196/N/25); (3) Negative control; (4) Positive control.

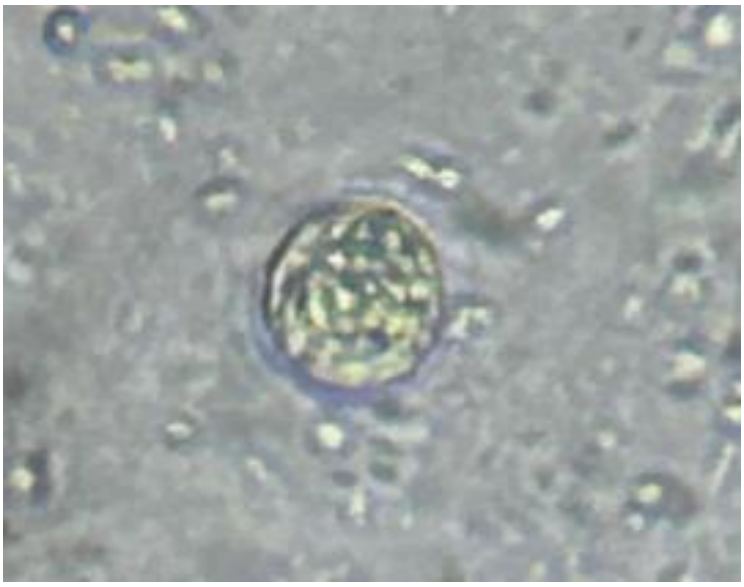

Figure 4. *Entamoeba* spp. cysts from the fecal sample of the case dog stained with Lugol's iodine.

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