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# Fatal Myocarditis Caused by Canine Parvovirus in a Litter of One-Day-Old German Shepherd Puppies: Molecular and Histopathological Findings

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## ABSTRACT

Canine parvovirus (CPV) is a highly contagious disease, typically causing haemorrhagic enteritis in dogs. The myocardial form, though well-documented historically, is now rare in vaccinated canine populations. This case report describes the first confirmed outbreak of acute parvoviral myocarditis in one-day-old German shepherd puppies in Iran. A litter of six neonates from a reportedly vaccinated primiparous bitch presented with acute lethargy, refusal to nurse, hypothermia and respiratory distress. Despite supportive medical care, all puppies died within hours. Postmortem examination revealed cardiomegaly with pale myocardial streaks, pulmonary consolidation and thymic haemorrhage. Histopathological analysis of heart tissue identified lymphoplasmacytic myocarditis with intranuclear inclusion bodies. Polymerase chain reaction (PCR) performed on cardiac tissue confirmed the presence of CPV. This case underscores that the cardiac form of CPV remains a threat to neonates, likely due to in utero or perinatal infection in the absence of maternal immunity. This case also serves as a reminder that CPV myocarditis can have a delayed clinical course, with heart failure and death occurring weeks to months after initial infection. Although CPV myocarditis can have a delayed clinical course, with heart failure and death occurring weeks to months after initial infection, this case underscores that the cardiac form of CPV remains a threat to neonates, likely due to in utero or perinatal infection in the absence of maternal immunity. It highlights the critical importance of ensuring complete and timely vaccination of breeding bitches and the need for vigilance regarding this rare but fatal manifestation of CPV.

## 1 | Introduction

Canine parvovirus (CPV), a single-stranded DNA virus, is a major cause of morbidity and mortality in dogs worldwide (Goddard and Leisewitz 2010). Following its emergence in the late 1970s, CPV-2 and its subsequent variants (CPV-2a, 2b, 2c) have primarily been associated with severe haemorrhagic enteritis (Miranda and Thompson 2016). CPV is transmitted via the faecal-oral route and has a tropism for rapidly dividing cells, such as those in the intestinal crypts, lymphoid tissues and, in neonates, the myocardial cells (Mietzsch et al. 2019).

Two distinct clinical forms exist: the common intestinal form and the less frequent myocardial form. Myocardial CPV is now

rare due to widespread vaccination but occurs following transplacental or perinatal infection of puppies from non-immune or inadequately vaccinated dams (Mazzaferro 2020). Infection in the last 2 weeks of gestation or the first 2 weeks of life can lead to viral replication in cardiomyocytes, resulting in necrotising myocarditis, which often presents as acute death or heart failure in puppies aged 3–8 weeks (Ford et al. 2017) (Buonavoglia et al. 2001).

While the intestinal form of CPV is routinely diagnosed in Iran, the myocardial form has been scarcely reported. This case report documents the first molecularly and histopathologically confirmed incidence of acute fatal myocarditis caused by CPV in a litter of one-day-old German shepherd puppies, highlighting a persistent risk in neonatal canine populations.

## 2 | Materials and Methods

### 2.1 | Case History

Six one-day-old German Shepherd puppies were presented to a veterinary clinic within 2 h of birth. The puppies were lethargic, unwilling to nurse and hypothermic. The owner reported that the dam appeared healthy throughout gestation with no observed signs of anorexia, vomiting or diarrhoea. It is noted that such signs can be non-specific and associated with pregnancy itself. Following the death of the puppies, the dam was not tested for CPV antigen or antibodies. Therefore, her infection status at the time of whelping remains unconfirmed.

The dam was reportedly vaccinated against core pathogens, including CPV. However, formal medical records documenting the specific vaccine brand, dates of administration (including completion of the initial puppy series and the most recent booster), and the administrator (veterinarian vs. owner) were not available for review. This information was based on owner's report. The owner reported an uncomplicated whelping. All puppies appeared vigorous, nursed normally and were active immediately after birth. No other litters from this dam or dogs in the household were reported affected.

### 2.2 | Clinical and Postmortem Examination

A complete physical examination was performed, assessing vital parameters including rectal temperature, heart rate and respiratory rate. Supportive care, including fluid therapy and warming, was initiated. Despite intervention, all puppies succumbed within 1 h. A complete necropsy was performed on all carcasses. Tissue samples from the heart, lungs, liver, thymus, spleen, stomach, intestine and kidneys were collected and fixed in 10% neutral buffered formalin for histopathological processing.

### 2.3 | Histopathology

Formalin-fixed tissues were processed routinely, embedded in paraffin, sectioned at 4–5 μm, and stained with haematoxylin and eosin (H&E) for light microscopic examination.

### 2.4 | Molecular Detection

DNA was extracted from fresh heart tissue using a commercial DNA extraction kit (Sina Pure DNA, Sina Col Co., Iran), according to the manufacturer's instructions. Conventional PCR was performed using primers Hfor (5′-CAGGTGATGAATTTGCTACA-3′) and Hrev (5′-CATTTGGATAAACTGGTGGT-3′), which target a 630 bp fragment of the VP2 capsid protein gene of CPV-2 (Prittie 2004). The thermal profile consisted of initial denaturation at 95°C for 5 min; 35 cycles of denaturation at 94°C for 30 s, annealing at 61°C for 1 min and extension at 72°C for 45 s; followed by a final extension at 72°C for 10 min. PCR products were visualised on a 1.5% agarose gel.

## 3 | Results

### 3.1 | Clinical Findings

Upon presentation, the puppies were profoundly lethargic, dehydrated and hypothermic (rectal temperature < 32°C). Bradycardia (heart rate < 100/min) and respiratory distress with paradoxical breathing were noted. A precise percentage of dehydration was not quantified during the brief physical examination prior to death; however, the mucous membranes were moist, and skin tenting was not remarked upon in the clinical notes. No evidence of diarrhoea, vomiting or other gastrointestinal signs was observed in the brief period before death.

### 3.2 | Gross Pathology

Necropsy revealed findings consistent with acute heart failure and systemic hypoperfusion. Key lesions included:

The heart was globally enlarged (four-chamber cardiomegaly) with pale linear streaking visible on the epicardial and myocardial surfaces

- **Cardiovascular system**: The heart was globally enlarged (four-chamber cardiomegaly) with pale linear streaking visible on the epicardial and myocardial surfaces. A postmortem clot was present in the left ventricle.
- **Respiratory system**: The trachea contained frothy fluid. The right lung lobes were consolidated, dark red and sank in formalin, indicating severe acute pneumonia.
- **Lymphoid system**: The thymus was diffusely haemorrhagic. The spleen was mildly enlarged with multiple capsular hematomas.
- **Other organs**: The liver was moderately congested with ecchymotic haemorrhages. Petechiae were observed on the gastric mucosa. The bone marrow was gelatinous and bright red. The intestines and kidneys appeared grossly normal.

### 3.3 | Histopathology

Microscopic examination confirmed the gross observations and provided a definitive diagnosis:

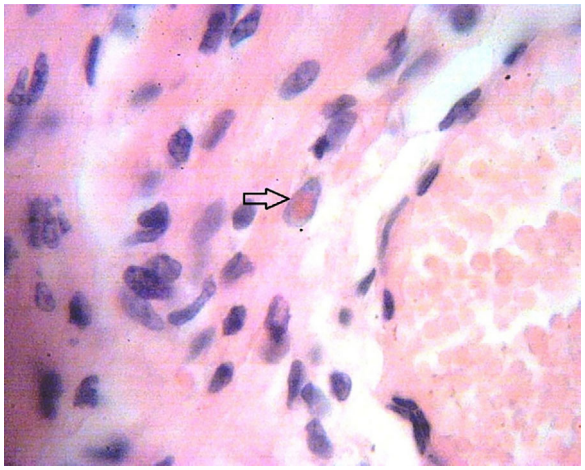

**FIGURE 1** | Pseudocytoplasmic intranuclear inclusion body in a heart myocyte (arrow). H&E staining ×2000.

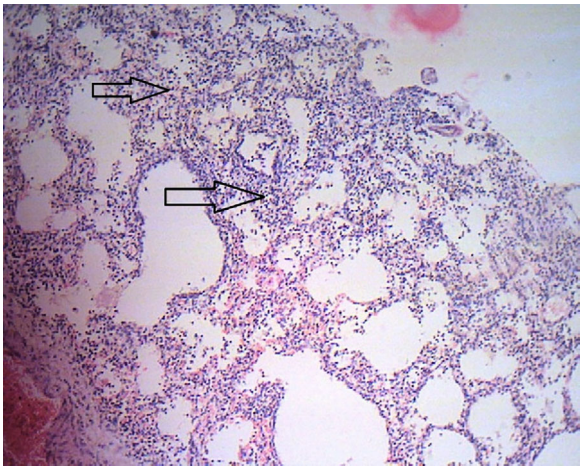

**FIGURE 3** | Alveolar septal thickening and interstitial thickening due to mononuclear infiltration in the lung (arrows). H&E staining ×800.

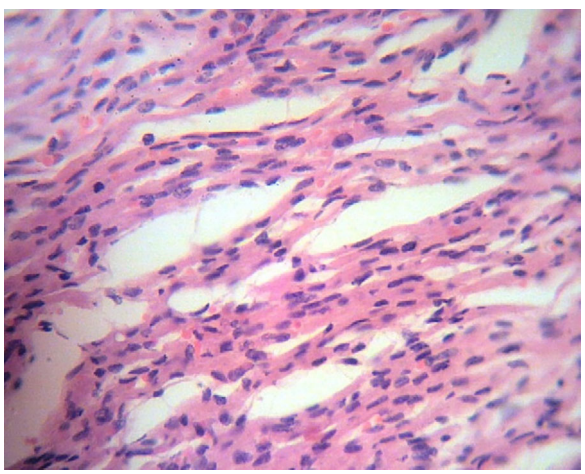

**FIGURE 2** | Oedema and congestion in the myocardium. H&E staining ×800.

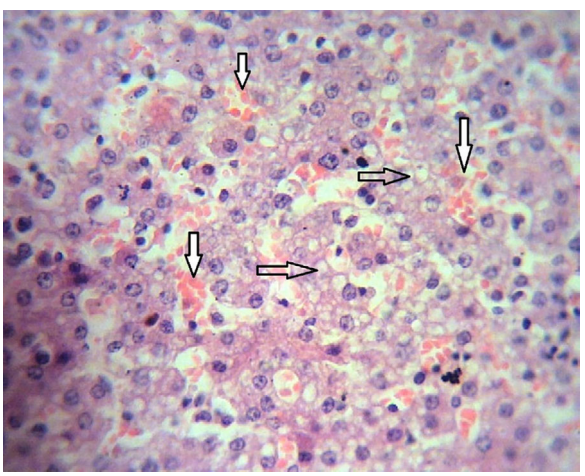

**FIGURE 4** | Hyperaemia (vertical white arrows) and fatty change (horizontal transparent arrows) in the liver. H&E staining ×800.

- **Heart**: There was severe, multifocal lymphoplasmacytic myocarditis with oedema and separation of myofibres. Intranuclear inclusion bodies were readily identified within cardiomyocytes (Figures 1 and 2).
- **Lungs**: Changes included diffuse alveolar septal thickening due to mononuclear cell infiltration, hyperaemia and bronchial epithelial necrosis with neutrophils in the airways (Figure 3).
- **Liver**: Centrilobular congestion and marked fatty change in hepatocytes were present (Figure 4).
- **Thymus**: Severe cortical collapse and atrophy, with prominent Hassall's corpuscles and vascular congestion.
- **Intestine**: Notably, the intestinal mucosa and crypt architecture were within normal limits, with no evidence of enteritis.
- **Spleen and kidneys**: The spleen exhibited mild lymphoid hyperplasia and hemosiderin-laden macrophages. The kidneys were histologically unremarkable.

### 3.4 | Molecular Detection

PCR analysis of cardiac tissue from all six puppies yielded a 630 bp amplicon, confirming the presence of canine parvovirus DNA.

## 4 | Discussion

This report provides a definitive diagnosis of acute parvoviral myocarditis in one-day-old puppies, a rare occurrence in modern veterinary practice. The diagnosis was supported by the classic history, the absence of enteritis, the characteristic gross and histopathological lesions in the heart and the molecular confirmation of CPV in cardiac tissue.

The absence of intestinal lesions is a hallmark of the myocardial form, as the tropism of the virus is dictated by the mitotic activity of the host's cells at the time of infection (Pollock and Coyne 1993) (Mylonakis et al. 2016). In neonates, cardiomyocytes are still undergoing division, making them susceptible to CPV infection, whereas intestinal crypt cells become the primary target in older

puppies (Parker et al. 2001). Our findings align with Ford et al. (Decaro and Buonavoglia 2012), who demonstrated a strong association between CPV infection and myocarditis in young dogs, with viral DNA detectable in the myocardium.

The source of infection in this litter is strongly suggestive of in utero or perinatal transmission from the dam, as the puppies died within 2 h of birth, precluding significant post-whelping environmental exposure. While canine parvovirus myocarditis is most commonly diagnosed in puppies aged 3–8 weeks, presentation at parturition is exceptionally rare. To our knowledge, represents one of the earliest documented cases of fatal CPV myocarditis, underscoring a particularly severe manifestation of transplacental infection.

The histopathological findings, severe, diffuse, necrotising myocarditis with intranuclear inclusion bodies within cardiomyocytes, are characteristic of the direct cytopathic effect of CPV. While these changes are highly suggestive, they are not pathognomonic for CPV; the definitive diagnosis rests on the correlation with molecular detection of the viral genome, as performed in this case.

Although the dam was reported as vaccinated, her primiparous status and young age raise questions about the completeness of her immunisation or the robustness of her immune response, potentially leading to inadequate passive immunity transfer to the puppies (Mylonakis et al. 2016). This case echoes historical reports where myocarditis was a common cause of sudden death in litters from unvaccinated dams (Sykes 2014).

The concurrent finding of acute pneumonia and thymic atrophy is consistent with a systemic viral infection. Thymic lymphoid depletion is a well-known effect of CPV, as the virus replicates in lymphoid precursors (Mietzsch et al. 2019).

This case has several important implications. First, it confirms that the myocardial form of CPV has not been eradicated and should remain a differential diagnosis for acute neonatal death, even in puppies from reportedly vaccinated dams. Second, it underscores the paramount importance of ensuring proper vaccination protocols for breeding animals. Bitches should receive booster vaccinations prior to mating to ensure high levels of maternally derived antibodies are passed to the puppies (Miranda et al. 2015). Finally, it highlights the value of postmortem examination and ancillary testing (PCR and histopathology) in establishing a definitive diagnosis for sudden death in neonates.

## 5 | Conclusion

We describe the first confirmed case of fatal CPV myocarditis in one-day-old puppies in Iran. This report serves as a critical reminder that despite effective vaccines, the cardiac form of CPV can still occur, with devastating consequences. Veterinarians and breeders must be aware of this possibility and prioritise complete and timely vaccination of breeding bitches. In cases of acute neonatal death, a thorough postmortem investigation with histopathology and molecular diagnostics is essential for accurate diagnosis and informed kennel management.

### Author Contributions

**Seyedhosein Jarolmasjed**: methodology, conceptualisation, data curation, formal analysis, investigation. **Mohammadreza Ghorani**: methodology, conceptualisation, data curation, investigation, writing – original draft. **Amir Ali Shahbazfar**: formal analysis, supervision, validation, project administration. **Hamed Sepehr**: formal analysis, supervision, validation, project administration. **Seyed Hossein Zamzam**: writing – review and editing, supervision. **Sina Soleimani**: methodology, writing – review and editing, supervision. All authors read and approved the final manuscript.


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The authors declare no conflicts of interest.

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Data are available on request by contacting the corresponding authors.

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