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Detection of Parvovirus DNA Associated with Enteric Disease in a Domestic Ferret (*Mustela putorius furo*): A Case Report

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ABSTRACT

Parvoviruses infect a broad range of vertebrate and invertebrate hosts. In ferrets (*Mustela putorius furo*), the best-characterized parvoviral infection is Aleutian disease virus (ADV), which is classified within the genus *Amdoparvovirus*. Reports of diseases associated with *Protoparvovirus* infection in domestic ferrets are extremely scarce. A 5-year-old domestic ferret was presented with anorexia, depression, gastroenteritis, hyporexia, hypothermia, and persistent hypoglycemia. Despite receiving supportive treatment, the animal died a few hours after admission. Necropsy and histopathological examination revealed severe enteric lesions and inflammatory changes consistent with gastrointestinal disease. A commercial canine parvovirus (CPV) antigen test yielded a positive result, and parvoviral DNA was also detected by conventional PCR using previously published primers. Because sequencing of the PCR amplicon was not performed, definitive identification of the viral species was not possible. Nevertheless, to further assess the specificity of the assay, an *in silico* multiple sequence alignment (MSA) was performed, demonstrating conservation of the primer-binding regions across protoparvoviruses and marked divergence from amdoparvoviruses. This case report describes the detection of parvoviral DNA associated with severe enteric disease in a domestic ferret. Although additional studies, including sequencing, are required to characterize the identity of the detected virus, the findings are consistent with a parvoviral infection.

Keywords: Parvovirus, *Mustelidae*, ferret.

Detección de ADN de parvovirus asociado a una enfermedad entérica en un hurón doméstico (*Mustela putorius furo*): reporte de caso

RESUMEN

Los parvovirus infectan a una amplia variedad de huéspedes vertebrados e invertebrados. En los hurones (*Mustela putoris furo*), la infección mejor caracterizada es la enfermedad de las aleutianas (ADV), clasificada dentro del género Amdoparvovirus. Los casos descritos de enfermedades asociadas a protoparvovirus en hurones domésticos son extremadamente escasos. Se presentó un hurón doméstico de 5 años con anorexia, depresión, gastroenteritis,

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hiporexia, hipotermia e hipoglucemia persistente. A pesar del tratamiento de apoyo, el animal falleció pocas horas después de su ingreso. La necropsia y el examen histopatológico revelaron lesiones entéricas graves con cambios inflamatorios compatibles con una enfermedad gastrointestinal. Una prueba comercial de antígenos del parvovirus dio un resultado positivo, y también se detectó ADN de parvovirus mediante PCR convencional utilizando cebadores publicados anteriormente. Dado que no se llevó a cabo la secuenciación del amplicón de la PCR, no fue posible identificar de forma definitiva la especie viral. No obstante, para evaluar más a fondo la especificidad del ensayo, se realizó un análisis de alineación múltiple (AAM) *in silico* que demostró la conservación de las regiones de unión de los cebadores entre los protoparvovirus y una divergencia sustancial con respecto a los amdoparvovirus. Este informe de caso describe la detección de ADN de parvovirus asociado a una enfermedad entérica grave en un hurón. Aunque se necesitan más estudios, como la secuenciación, para caracterizar la identidad del virus detectado, los hallazgos parecen ser compatibles con una infección por parvovirus.

Palabras clave: parvovirus, *Mustelidae*, hurón.

INTRODUCTION

The family *Parvoviridae* comprises a diverse group of small, non-enveloped viruses with single-stranded DNA genomes that infect a broad range of vertebrate and invertebrate hosts (Cotmore et al., 2019). These viruses are classified into subfamilies according to their host range. *Parvovirinae* comprises the parvoviruses that infect vertebrates, including mammals, birds, fish, and reptiles. Parvoviruses exhibit remarkable host adaptability; indeed, François et al. (2016) identified approximately 81 potential animal hosts through a systematic search for parvovirus-related sequences. The diseases caused by parvoviruses vary according to the viral species and their corresponding host-cell receptor usage. Consequently, although the pathogenesis differs among parvoviruses, these viruses share several pathogenic mechanisms that contribute to tissue damage and the development of clinical disease.

Canine parvovirus (CPV) is among the best-characterized parvoviruses in veterinary medicine, owing to its worldwide distribution, high morbidity and mortality, and major veterinary significance. In ferrets, however, parvoviral infection is most commonly associated with Aleutian disease virus (ADV). Although both ADV and CPV belong to the family *Parvoviridae*, they are classified into different genera, *Amdoparvovirus* and *Protoparvovirus*, respectively. Parvovirus taxonomy is primarily based on the highly conserved nonstructural protein NS1 (Canuti et al., 2022), which also allows the identification of additional genomic distinctions. The *Amdoparvovirus* genome is linear (~4 kb) and contains two major gene cassettes flanked by palindromic terminal sequences. Unlike other parvoviruses, however, the VP1 protein lacks the phospholipase A2 (PLA2) domain (Høeg et al., 2025).

The genus *Amdoparvovirus* includes Aleutian mink disease virus (AMDV), which infects several mustelid species, including the American and European minks (*Neovison vison*, *Mustela lutreola*), stoat (*Mustela erminea*), European pine and stone martens (*Martes martes*, *Martes foina*), and the European and North American river otters (*Lutra lutra*, *Lontra canadensis*). AMDV has also been detected

in other carnivores, including striped skunks (*Mephitis mephitis*), raccoons (*Procyon lotor*), bobcats (*Lynx rufus*), and common genets (*Genetta genetta*) (Franzo et al., 2021). Domestic ferrets (*Mustela putorius furo*) are also susceptible to AMDV infection. Macrophages and other immune cells are the primary target cells of AMDV, and this broad cellular tropism has been associated with the frequent cross-species transmission observed among amdoparvoviruses (Høeg et al., 2025). Consequently, Aleutian disease in mink and other mustelids is typically characterized as an immune complex-mediated disease (Høeg et al., 2025). In domestic ferrets, AMDV infection is often asymptomatic, allowing infected animals to serve as potential reservoirs (Murakami et al., 2001). However, severe disease may also occur, with clinical manifestations including chronic wasting syndrome and neurological disorders (Stevenson et al., 2001).

The genus *Protoparvovirus* includes canine parvovirus (CPV), mink enteritis virus (MEV), and feline parvovirus (FPV), all of which are associated with enteric disease in carnivores such as dogs, raccoons, foxes, and mink. Canine parvovirus type 2 (CPV-2) emerged as a variant of FPV, a virus that has long been recognized as capable of infecting several carnivore species, including mink, raccoons, and foxes (Allison et al., 2012). Cross-species transmission among these hosts has likely contributed to viral host adaptation and host-jumping events. Mink enteritis virus is also closely related to FPV and is nearly genetically identical to FPV (Li et al., 2024).

Unlike amdoparvoviruses, protoparvoviruses possess a unique VP1 region containing a phospholipase A2 (PLA2) domain that is essential for endosomal escape during viral infection. These viruses exhibit a marked tropism for rapidly dividing cells, particularly the intestinal crypt epithelium and hematopoietic tissues, resulting in acute enteritis and leukopenia in susceptible hosts.

Reports of protoparvovirus-associated infections in domestic ferrets remain scarce and outdated. According to Veijalainen (1986), ferrets were considered unlikely to acquire natural infections, although experimental inoculation with FPV or MEV demonstrated susceptibility and induced ataxia in neonatal animals. Similarly, Parrish et al. (1987) reported that MEV failed to replicate in ferrets, CPV replicated poorly, whereas FPV replicated efficiently. Nevertheless, the authors concluded that ferrets do not appear to be naturally susceptible to either MEV or FPV.

Therefore, the present report describes the clinical presentation, pathological findings, and molecular detection of parvoviral DNA in a domestic ferret with severe enteric disease, and discusses the differential diagnosis and limitations associated with definitive viral identification. Because protoparvovirus infections are not routinely considered in the differential diagnosis of ferret enteric disease, and given the increasing popularity of ferrets as companion animals, documenting unusual cases may contribute to improved clinical recognition and epidemiological surveillance.

CASE PRESENTATION

Case History

A 5-year-old female domestic ferret was presented to the Happy Pets Clinic in León, Guanajuato (central Mexico), on January 2, 2024, because of depression

and hyporexia (Figure 1). Signalment, medical history, vaccination status, and husbandry information were obtained at the time of hospital admission. The ferret had been vaccinated 18 months before presentation with a commercial Puppy DP vaccine (distemper and parvovirus) and had also received oral fenbendazole for deworming on the same occasion. According to the owner, the animal was fed a commercial diet formulated for adult ferrets (brand not specified). Although the ferret did not share the household with dogs, it had occasional contact with them. Information regarding previous medical conditions and cohabitation with other animal species was not available.

At presentation, the ferret exhibited gastroenteritis, anorexia, lethargy, hypothermia, and persistent hypoglycemia despite external thermal support and glucose supplementation. A complete physical examination and abdominal ultrasonographic evaluation were subsequently performed. Ultrasonography revealed a structure within the duodenum producing an acoustic shadow that was considered suggestive of a foreign body, although there was no evidence of intestinal obstruction. The presence of a highly compacted food bolus could not be excluded; therefore, serial ultrasonographic monitoring and/or exploratory laparotomy were recommended. Based on these findings, the initial presumptive diagnosis was gastrointestinal disease associated with the presence of a foreign body. Mesenteric lymphadenopathy was also observed.

Because the animal presented with severe diarrhea, a rapid fecal coronavirus antigen test was performed. The commercial CPV/CCV Ag Test Kit (Bionote™), which simultaneously detects canine parvovirus (CPV) and canine coronavirus antigens, yielded an unexpected positive result for parvovirus antigen (Figure 2). Unfortunately, despite receiving supportive treatment, the patient died a few hours after hospital admission.

Necropsy Findings

The carcass of the ferret was received on January 4, 2024, and a complete necropsy was performed on the same day. External examination revealed markedly pale oral and conjunctival mucous membranes, with the perianal region stained by liquid feces. Gross examination of the thoracic cavity showed pronounced costal impressions, pleural adhesions, and non-collapsed lungs. Within the abdominal cavity, the

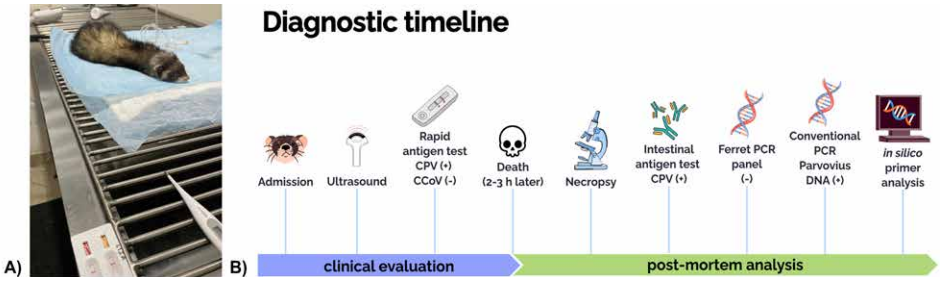

FIGURE 1. A. Five-year-old female domestic ferret (*Mustela putorius furo*) presented for clinical evaluation because of depression and hyporexia. B. Timeline of the diagnostic work-up.

Source: Authors' own work.

FIGURE 2. Rapid antigen tests show a positive result for parvovirus.

Source: Authors' own work.

liver appeared pale with rounded margins. The stomach contained abundant mucus, and the intestinal contents were yellow and mucoid. No macroscopically identifiable pancreatic tissue was observed. Splenomegaly was also noted. Histological examination of the kidney and thyroid gland revealed no apparent pathological alterations.

Microscopic examination demonstrated marked thickening of the alveolar septa due to infiltration by lymphocytes, plasma cells, and histiocytes. In the small intestine, the lamina propria was diffusely expanded by an infiltrate of lymphocytes, plasma cells, and histiocytes. Enterocyte detachment was observed, accompanied by goblet cell hyperplasia, abundant luminal mucus, and mild to moderate villous atrophy. In the liver, hepatocytes contained multiple fine and coarse cytoplasmic vacuoles, while hepatic cords appeared expanded due to sinusoidal congestion (Figure 3). The spleen

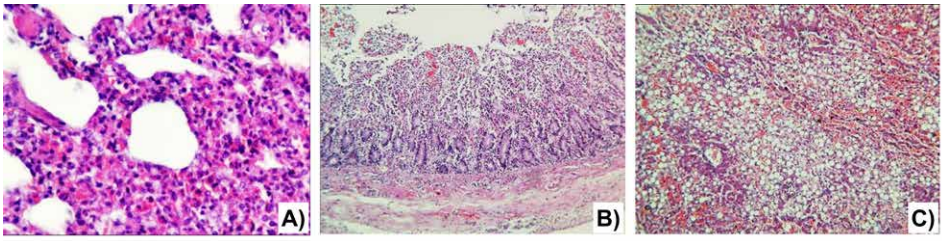

FIGURE 3. Histopathological findings in hematoxylin and eosin-stained tissue sections. A. Lung (40×): marked thickening of the alveolar septa due to infiltration by numerous lymphocytes, plasma cells, and histiocytes. B. Small intestine (10×): diffuse expansion of the lamina propria by lymphocytes, plasma cells, and histiocytes. Enterocyte detachment, goblet cell hyperplasia, abundant mucus production, and mild to moderate villous atrophy are evident. C. Liver (40×): multifocal hepatocellular degeneration characterized by multiple fine and coarse cytoplasmic vacuoles. Hepatic cords are expanded because of sinusoidal congestion.

Source: Authors' own work.

exhibited lymphoid follicular hyperplasia within the white pulp, whereas the lymph nodes showed generalized depletion of lymphoid follicles. In the brain, neurons exhibited peripheral condensation of Nissl substance (chromatolysis), and glial cells surrounding some neurons, consistent with satellitosis, were also observed.

Based on these findings, the pathological diagnosis was severe generalized non-suppurative interstitial pneumonia; moderate generalized lymphoplasmacytic enteritis with multifocal enterocyte detachment, multifocal goblet cell hyperplasia, and mild to moderate villous atrophy; severe multifocal microvesicular and macrovesicular hepatocellular degeneration; moderate multifocal lymphoid hyperplasia; moderate generalized lymphoid atrophy; and mild multifocal neuronal chromatolysis with satellitosis.

Molecular Detection and In Silico Primer-Binding Analysis

Based on the pathological findings, a PCR diagnostic panel for ferret enteric pathogens was performed to test for *Cryptosporidium* spp., *Giardia* spp., ferret coronavirus, canine distemper virus, *Lawsonia intracellularis*, *Campylobacter jejuni*, and *Helicobacter* spp. All results were negative. Additionally, a second antigen test was performed on intestinal contents using a plate-based immunochromatographic assay specific for canine parvovirus antigen, which yielded a positive result.

To confirm this finding, PCR analysis was performed at the Veterinary Faculty Laboratory of Universidad La Salle Bajío. Total DNA was extracted from intestinal tissue using the Invitrogen PureLink RNA/DNA Mini Kit according to the manufacturer's protocol. Endpoint PCR for parvovirus detection was subsequently performed using the Phire Animal Tissue Direct PCR Kit (Thermo Fisher Scientific) following the manufacturer's instructions. The thermal cycling conditions consisted of an initial denaturation at 98°C for 5 min, followed by 40 cycles of denaturation at 98°C for 5 s, annealing at 60°C for 5 s, and extension at 72°C for 20 s, with a final extension step at 72°C for 5 min.

A 337-bp fragment of the parvoviral genome was amplified using the primers described by Hao et al. (2019): forward primer: 5′-AAGACGTGCAAGCGAGTCC-3′ and reverse primer: 5′-GAGCGAAGATAAGCAGCGTAA-3′. The expected amplification product was detected using agarose gel electrophoresis (Figure 4), confirming a positive PCR result for parvoviral DNA with the published primer set. As shown in the electrophoretic profile, an additional DNA fragment of approximately 600 bp was also observed. Because the PCR amplicon was not sequenced, definitive identification of the viral species was not possible.

To further evaluate the specificity of the PCR assay used in this study, an *in silico* analysis of the primer-binding regions was performed using complete genome sequences representing canine parvovirus (CPV), feline parvovirus (FPV), mink enteritis virus (MEV), and Aleutian mink disease virus (AMDV). The accession numbers included in the analysis were as follows: CPV (NC_001539.1, MW539053.1, PV784163.1, PZ162638.1, PZ162637.1); FPV (PP973493.1, OR783315.1, OR198066.1, MT614366.1, MZ362883.1); MEV (MZ044016.1, MZ044015.1, KT899746.1); and AMDV (JN040434.1, K 29428.1, GU269892.1, GU183265.1, GU183264.1). Multiple sequence alignment (MSA) was performed using Clustal Omega and visualized using

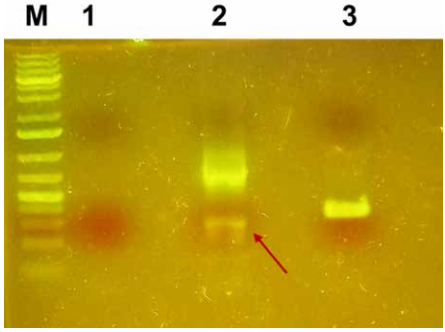

FIGURE 4. Agarose gel electrophoresis of PCR products. M, molecular weight marker; lane 1, negative control; lane 2, patient sample; lane 3, positive control. The arrow indicates the expected 337-bp amplification product observed in lane 2, confirming the presence of parvoviral DNA.

Source: Authors' own work.

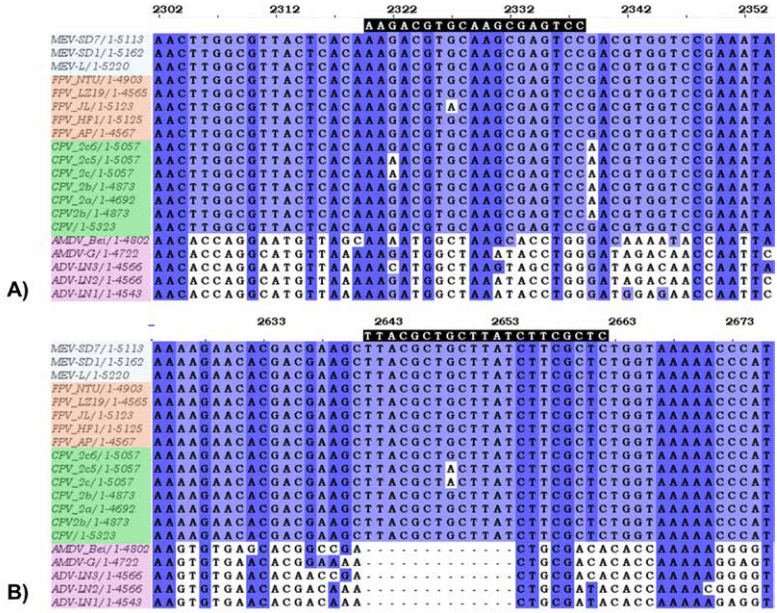

FIGURE 5. Multiple sequence alignment of representative *Protoparvovirus* and *Amdoparvovirus* genomes showing the primer-binding regions targeted by the conventional PCR assay. A. Alignment of the forward primer (CPV-F) binding site. B. Alignment of the reverse primer (CPV-R) binding site. Representative complete genome sequences of CPV, FPV, MEV, and AMDV were retrieved from GenBank and aligned using Clustal Omega. The primer-binding regions are indicated above each alignment. CPV (green), FPV (orange), and MEV (blue) exhibit a high degree of sequence conservation across both primer-binding sites, whereas AMDV (purple) sequences display multiple nucleotide mismatches and insertion/deletion events (gaps), particularly within the reverse primer-binding region.

Source: Authors' own work.

Jalview (Figure 5). The alignment demonstrated that the primer-binding regions were highly conserved among members of the genus *Protoparvovirus*, whereas multiple nucleotide mismatches were observed in the corresponding regions of *Amdoparvovirus*, as summarized in Table 1.

DISCUSSION

This case is noteworthy because ferrets are increasingly maintained as companion animals, and many are kept in households with domestic dogs, increasing the potential for interspecies exposure to canine pathogens. To the best of our knowledge, naturally acquired *Protoparvovirus* infection has not previously been documented in a domestic ferret, making this, to the best of our knowledge, the first reported case. Furthermore, the infection was identified as an incidental finding, suggesting that protoparvovirus infections in ferrets may go unnoticed and underscoring the need for further investigation into its epidemiology, clinical significance, and potential role in cross-species transmission.

Several differential diagnoses were considered based on the clinical presentation and pathological findings, including a gastrointestinal foreign body, canine distemper virus infection, ferret enteric coronavirus infection, Aleutian disease virus (ADV) infection, bacterial enteritis, lymphoma, and insulinoma. Although abdominal ultrasonography initially suggested the presence of a possible duodenal foreign body, the absence of intestinal obstruction together with the rapid progression to severe systemic illness made this diagnosis less likely. Given the acute gastrointestinal presentation, coronavirus infection was also considered; however, the rapid antigen assay yielded a positive result for parvovirus. Although vaccine-derived antigen should be considered when interpreting positive antigen tests, vaccination had been administered approximately 18 months before presentation, making this explanation unlikely. Moreover, the necropsy findings were consistent with an infectious process of probable viral origin. The presence of interstitial pneumonia initially raised suspicion of canine distemper virus infection, whereas the lymphoplasmacytic and catarrhal enteritis was compatible with, although not specific to, coronavirus infection. However, PCR testing yielded negative results for both pathogens, and a second antigen test performed on intestinal contents collected during necropsy also yielded a positive result for canine parvovirus. It should be noted that the antigen assays used in

TABLE 1. Number of nucleotide mismatches between the PCR primers and representative *Protoparvovirus* and *Amdoparvovirus* genome sequences

Virus	Forward mismatches	Reverse mismatches	Predicted amplification
MEV	0	0	Yes
FPV	0-1	0	Yes
CPV	0-1	0-1	Yes
AMDV	10–11	Multiple gaps	No

Source: Authors' own work.

this study were originally developed and validated for canine samples, and their diagnostic performance in ferrets has not been formally established. Consequently, their sensitivity, specificity, and potential cross-reactivity in this species remain unknown, and the results should be interpreted with caution. Nevertheless, the diagnostic interpretation was not based on a single assay but rather on the concordance of multiple independent findings, including two positive antigen assays, molecular detection of parvoviral DNA in intestinal tissue collected at necropsy, compatible clinical and pathological findings, and the exclusion of other common enteric pathogens by PCR. Hepatic lipidosis was interpreted as a secondary consequence of prolonged hyporexia rather than the primary cause of disease.

Aleutian disease also remained an important differential diagnosis because of the gastrointestinal involvement observed. However, no pathological lesions characteristic of this infection were identified. Aleutian disease virus infection is characterized by immune complex deposition within blood vessel walls, leading to multisystemic organ damage, particularly glomerulonephritis and lesions affecting other organs (Une et al., 2000). In the present case, no renal lesions were detected at necropsy, and the moderate generalized lymphoid atrophy observed in the lymph nodes was not accompanied by the systemic plasmacytosis or marked lymphoid hyperplasia typically associated with ADV infection (Blank et al., 2022). Likewise, persistent hypoglycemia could also have been consistent with insulinoma, one of the most common neoplasms affecting adult ferrets; however, this diagnosis did not account for the concurrent gastroenteritis, mesenteric lymphadenopathy, hypothermia, and rapid clinical deterioration observed in this patient.

Protoparvoviruses exhibit a marked tropism for rapidly dividing cells, particularly for intestinal crypt epithelial cells, hematopoietic precursor cells within the bone marrow, and lymphoid tissues. Destruction of the intestinal crypt epithelium results in enterocyte loss, villous atrophy, and excessive mucus production, whereas lymphoid depletion is a well-recognized consequence of viral replication within lymphoid tissues (Mandawat et al., 2025). In the present case, the histopathological findings of mild to moderate villous atrophy, enterocyte detachment, goblet cell hyperplasia, abundant mucus production, generalized lymphoid atrophy, and splenic lymphoid hyperplasia were compatible with the established pathogenesis of CPV infection. Nevertheless, these lesions are not pathognomonic, and the predominance of a lymphoplasmacytic inflammatory infiltrate differed from the classical histopathological pattern of canine parvoviral enteritis, which is typically characterized by severe crypt necrosis, crypt dilation, epithelial collapse, and minimal inflammatory infiltrates. The inflammatory infiltrate observed in this ferret may reflect the stage of disease at the time of death, individual variation in the host inflammatory response, or secondary inflammatory changes resulting from disruption of the intestinal mucosal barrier. Consequently, the histopathological findings should be interpreted cautiously and always in conjunction with the clinical presentation, negative PCR results for other viral pathogens, and positive immunochromatographic detection of parvoviral antigen and molecular detection of parvoviral DNA, rather than as independent diagnostic evidence. Bone marrow was not collected during necropsy;

therefore, histopathological evaluation of this tissue could not be performed. Because protoparvoviruses exhibit marked tropism for rapidly proliferating hematopoietic cells, examination of the bone marrow could have provided additional pathological evidence and should be included in the diagnostic evaluation of future cases.

Canuti et al. (2020) investigated the ecology and cross-species transmission of both amdoparvoviruses and protoparvoviruses among wild carnivores. They reported FPV infections in mink, martens, and otters, a single CPV-2 infection in a mink and another in an otter, as well as an AMDV–FPV coinfection in a mink. No association was found between body condition and viral infection; however, ferrets were not included in that study. Similarly, Ndiana et al. (2021) reported a beech marten that tested positive for CPV-2b, as well as CPV- and FPV-positive Eurasian badgers. These findings support the natural circulation of protoparvoviruses among several mustelid species. To the best of our knowledge, reports of protoparvovirus infection in ferrets remain limited to experimental studies conducted by Veijalainen (1986) and Parrish et al. (1987), which demonstrated susceptibility under experimental conditions, whereas the occurrence of naturally acquired infections remains largely unknown.

The occurrence of protoparvovirus infection in the present ferret is biologically plausible in light of the evolutionary biology of carnivore protoparvoviruses. The host range of these viruses is largely determined by interactions between the viral capsid protein VP2 and the host transferring receptor type 1 (TfR1), which mediates viral entry into susceptible cells (Hueffer et al., 2003; Allison et al., 2014). Relatively few amino acid substitutions in VP2 have been shown to alter receptor-binding affinity, thereby facilitating adaptation to new host species (Ndiana et al., 2021). These evolutionary changes were instrumental in the emergence of CPV from feline panleukopenia virus (FPV) and its subsequent expansion across multiple carnivore hosts (Hueffer et al., 2003; Allison et al., 2014). Therefore, the well-documented capacity of carnivore protoparvoviruses to cross species barriers through adaptive evolution provides a biologically plausible explanation for the occurrence of protoparvovirus infection in a domestic ferret. Although additional epidemiological studies are required to determine the frequency and clinical significance of such infections, the present case suggests that protoparvovirus should be included among the differential diagnoses of acute enteric disease in ferrets, particularly in animals living in close contact with domestic dogs.

Because CPV is highly prevalent and environmentally stable, environmental or laboratory contamination must always be considered when interpreting positive molecular results. However, in the present case, the concordant results obtained from two independent antigen detection assays performed at different stages of the diagnostic investigation, together with the subsequent detection of parvoviral DNA in intestinal tissue collected at necropsy, reduce the likelihood that the molecular findings resulted solely from contamination. Nevertheless, because sequencing of the PCR amplicon was not performed, definitive identification of the viral species was not possible.

Regarding the molecular findings and *in silico* analysis, conventional PCR detected parvovirus-related DNA. The primers used in this study were originally designed for the detection of canine parvovirus and,

therefore, have not been experimentally validated in ferrets. Nevertheless, *in silico* analysis demonstrated that the primer-binding regions were highly conserved among representative members of the genus *Protoparvovirus*. Although experimental validation in ferret samples was beyond the scope of this study, these findings support the likelihood that the assay preferentially amplifies protoparvoviruses rather than amdoparvoviruses. Furthermore, several nucleotide mismatches were located near the 3′ ends of the primers, a region that is critical for efficient primer extension during PCR amplification. Although this analysis does not experimentally validate primer specificity, it provides additional evidence that amplification of amdoparvoviruses with this primer pair would be considerably less likely.

This study has several limitations. First, because sequencing of the PCR amplicon was not performed, definitive identification of the viral species was not possible. Nevertheless, *in silico* analysis of the primer-binding regions demonstrated a high degree of sequence conservation among carnivore protoparvoviruses, whereas substantial sequence divergence was observed among amdoparvoviruses. Despite these findings, the absence of an AMDV-specific molecular assay precludes its complete exclusion. In addition, quantitative PCR (qPCR) was not available, precluding estimation of the viral load. Furthermore, immunohistochemistry or *in situ* hybridization would have enabled localization of viral antigen or nucleic acid within the intestinal lesions, thereby strengthening the pathological association between the detected virus and the observed histopathological changes. These limitations should be taken into account when interpreting the results.

Overall, the molecular and *in silico* findings indicate that the detected amplicon is more likely to be derived from canine parvovirus (CPV), feline panleukopenia virus (FPV), or mink enteritis virus (MEV) than from Aleutian mink disease virus (AMDV). Moreover, the vaccination history, with administration of a modified-live Puppy DP vaccine approximately 18 months before presentation, makes vaccine-derived antigen detection highly unlikely. When interpreted together with the compatible clinical presentation, histopathological findings, negative PCR results for other major viral pathogens, and positive immunochromatographic detection of parvoviral antigen and molecular detection of parvoviral DNA, the evidence supports a protoparvovirus, most likely CPV, as the likely etiological agent in this case. An additional noteworthy finding was the presence of a larger PCR product of approximately 600 bp. Future sequencing of this unexpected amplicon could provide valuable information regarding the presence of genetic variation, a novel mutation, or a previously unrecognized parvovirus strain infecting domestic ferrets.

CONCLUSION

This report describes the clinical and histopathological findings associated with severe enteric disease in a domestic ferret from León, Guanajuato, Mexico. Molecular detection and *in silico* analysis supported the presence of parvoviral DNA, which most likely corresponds to a carnivore protoparvovirus. However, because sequencing was not performed, definitive identification of the viral species was not possible. To the best of the authors' knowledge, this represents one of the first reports of molecular detection of a

protoparvovirus associated with naturally occurring enteric disease in a domestic ferret. Further molecular characterization, particularly through sequencing, is required to clarify the identity of the detected virus and to better define the role of protoparvoviruses in ferret disease. Nevertheless, the present study provides an important precedent by demonstrating that protoparvovirus infection should be considered among the differential diagnoses of enteric disease in domestic ferrets.

ETHICS STATEMENT

Ethical approval was not required for this case report. Written informed consent for the postmortem examination and publication of the findings was obtained from the animal's owner.

CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

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ARTIFICIAL INTELLIGENCE USAGE STATEMENT

No artificial intelligence tools were used in the conduct of this research or in the preparation of this manuscript.

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