Enteritis Caused by Type 2c Canine Parvovirus in a 5-Year-Old Dog

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ABSTRACT

Background: Canine parvovirosis, caused by canine parvovirus type 2 (CPV-2), emerged in the 1970s as an important disease affecting dogs, causing severe hemorrhagic gastroenteritis and death. It can occur in any breed, gender, and age; however, puppies of 4 to 12 weeks of age are most commonly afflicted. In 2000 a new variant of the virus, called CPV-2c, was discovered, and has been related to hemorrhagic gastroenteritis in dogs with up to 2 years of age, although some cases have been described in older animals with a full vaccination history. This paper reports a case of enteritis by canine parvovirus type 2c (CPV-2c) in a 5-year-old dog.

Case: At necropsy a pallid oral and conjunctival mucosae were observed. The small intestine showed a very reddish and wrinkled serosa, the wall was thickened, the mucosa was diffusely wrinkled and yellowed with evidenced Peyer plaques and there was no content in the final portion of the intestine. The mesenteric lymph nodes were enlarged and reddish. Multiple suffusions on the serosa of the stomach, and petechiae and subepicardial suffusions in the heart were observed. The histological findings were, collapse of the lamina propria of the small intestine, and fusion of the villi, necrosis of enterocytes, atrophy and the disappearance of crypts, with dilation of remaining crypts showing large rounded nuclei with one or two evident nucleoli, exhibiting accentuated cellular pleomorphism in some cases forming syncytia. In addition, there were bacterial colonies and fibrin adhered to the mucosae. The serosa showed diffuse congestion, marked transmural multifocal hemorrhage, thrombosis and fibrin deposition on the serosa surface. Necrosis of the germinative centers with moderate lymphoid depletion was observed in the lymphoid aggregates of the large intestine. In the bone marrow, spleen and mesenteric lymph node there were accentuated lymphoid depletion, hemorrhage and moderate hemosiderosis. The remaining tissue of the thymus showed accentuated multifocal to coalescent hemorrhage. The anti-parvovirus IHC showed intense immunostaining of the cytoplasm of epithelial cells, mainly in the crypts of the small intestine. In the spleen and lymph node there was intense immunostaining in the lymphocytes of follicular centers. The PCR and sequencing techniques applied to the sample allowed the identification of CPV-2c.

Discussion: Diarrhea in dogs has been associated with a wide variety of viral agents; the canine parvovirus, rotavirus and coronavirus being the main primary pathogens involved. Since CPV-2 emerged at the end of the 1970s this pathogen has gained great importance in the care of dogs and is probably the most common infectious disease of canine species. Shortly after appearing in the canine population, parvovirosis underwent alterations in some of its amino acids, which resulted in new and better adapted viral strains. Studies in which circulating viral strains have been identified have demonstrated the importance of CPV-2c in outbreaks of parvovirosis in previously vaccinated puppies. There are few reports of the detection of CPV-2c in adult dogs. The majority of cases described relate to dogs up to 2½ years of age, one exception being a case involving a 12-year-old dog. This new variant of CPV-2 should be considered as an important pathogen in the diagnosis of causes of sanguinolent diarrhea in adult dogs.

Keywords: CPV-2c, adult dogs, parvovirus, enteric disease.
INTRODUCTION

Canine parvovirosis, caused by canine parvovirus type 2 (CPV-2), emerged in the 1970s as an important disease affecting dogs, causing severe hemorrhagic gastroenteritis and death [1]. It can occur in any breed, gender, and age; however, puppies of 4 to 12 weeks of age are most commonly afflicted [5,11]. The CPV-2 was rapidly replaced with the new antigenic variants CPV-2a and by CPV-2b [7]. In 2000 a new variant of the virus, called CPV-2c [4], was discovered, and has since been reported in several parts of the world, including Brazil [13,14]. This variant has been related to hemorrhagic gastroenteritis in dogs with up to 2 years of age [4,7,8], although some cases have been described in older animals with a full vaccination history [6,7]. The aim of this study was to describe a case of parvovirosis caused by variant 2c (CPV-2c) in a 5-year-old dog, with immunohistochemical and molecular characterization.

CASE

A 5-year-old male mixed breed dog was submitted to necropsy with a history of intense sanguino-salient diarrhea over three days, vomiting, dehydration, marked apathy and anorexia. It was reported that the dog had received three doses of commercial vaccine against parvovirus, canine distemper, adenovirus type 1, adenovirus type 2 and parainfluenza virus at 45, 66 and 87 days of age, however, it received no yearly revaccination.

At necropsy a good physical condition, pallid oral and conjunctival mucosae were observed. The small intestine showed a very reddish and wrinkled serosa (Figure 1a), the wall was thickened, the mucosae was diffusely wrinkled and yellowed (Figure 1b) with evidenced Peyer plaques and there was no content in the final portion of the intestine. The mesenteric lymph nodes were enlarged and reddish. Multiple suffusions on the serosa of the stomach, and petechiae and sub-epicardial suffusions in the heart were observed. Several organs were collected, fixed in 10% formalin, routinely processed for histological examination and stained with hematoxylin and eosin. Feces samples were collected from the rectum for viral detection. The tissue samples were submitted to the immunohistochemical (IHC) technique with anti-parvovirus monoclonal antibody (MCA 2064) by the peroxidase-bound streptavidin-biotin method [12]. The extraction of viral DNA from the fecal sample for the detection of CPV was performed as described previously [2]. The PCR was carried out for the amplification of 583 bp (base pairs) of the VP2 gene (position 4003-4585), using a previously described protocol [4].

The histological findings were, collapse of the lamina propria of the small intestine, and fusion of the villi, necrosis of enterocytes, atrophy and the disappearance of crypts, with dilation of remaining crypts showing large rounded nuclei with one or two evident nucleoli, exhibiting accentuated cellular pleomorphism in some cases forming syncytia (Figure 1c). In addition, there were bacterial colonies and fibrin adhered to the mucosae. The serosa showed diffuse congestion, marked transmural multifocal hemorrhage, thrombosis and fibrin deposition on the serosa surface. Necrosis of the germinative centers with moderate lymphoid depletion was observed in the lymphoid aggregates of the large intestine. In the bone marrow, spleen and mesenteric lymph node there were accentuated lymphoid depletion, hemorrhage and moderate hemosiderosis. The remaining tissue of the thymus showed accentuated multifocal to coalescent hemorrhage.

The anti-parvovirus IHC showed intense immunostaining of the cytoplasm of epithelial cells, mainly in the crypts of the small intestine (Figure 1d). In the spleen and lymph node there was intense immunostaining in the lymphocytes of follicular centers (Figure 1e). In the liver, staining was observed in the cytoplasm of Kupffer cells. In the bone marrow there was staining mainly in the cytoplasm of a few cells (Figure 1f).

The application of the PCR and sequencing techniques detected CPV-2c. The partial amplification of the VP2 gene revealed a single band with the expected size of 583bp.

DISCUSSION

Diarrhea in dogs has been associated with a wide variety of viral agents; the canine parvovirus, rotavirus and coronavirus being the main primary pathogens involved [11]. Since CPV-2 emerged at the end of the 1970s this pathogen has gained great importance in the care of dogs and parvovirosis is probably the most common infectious disease of canine species [11]. Shortly after appearing in the canine population, CPV-2 underwent alterations in some of its amino acids, which resulted in new and better adapted viral strains [4,7].
CPV-2c was detected for the first time in 2000 and rapidly spread to diverse regions of the world [4,8,13,14]. Although cases of parvovirosis are typically associated with puppies of 4 to 12 weeks of age, the period in which a decrease in the maternal antibodies occurs, a case of parvovirosis in a 5-year-old dog is reported herein. The clinical signs, as well as the macroscopic and microscopic findings for this dog are similar to those described for other fatal cases of canine parvovirosis [3,12].

Studies in which circulating viral strains have been identified have demonstrated the importance of CPV-2c in outbreaks of parvovirosis in previously vaccinated puppies [8,13,14]. There are few reports of the detection of CPV-2c in adult dogs [6,7]. The majority of cases described relate to dogs up to 2 ½ years of age, one exception being a case involving a 12-year-old dog [7].

Hemorrhagic gastroenteritis in dogs can sporadically be associated with intestinal infections by type A Clostridium perfringens, parasitism by Ancylostoma caninum [3], and Cryptosporidium parvum [9]. Recently a new canine circovirus (DogCV) was described in the liver of a dog with severe hemorrhagic gastroenteritis, vasculitis, and granulomatous lymphadenitis [10]. DogCV also could be a complicating factor in other canine infectious diseases, as with PCV2 [10].

In conclusion, CPV-2, besides being a principal infectious agent in young dogs, has evolved the new variant 2c which should be considered an important pathogen in the differential diagnosis of the causes of sanguinolent diarrhea.
REFERENCES


