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Canine Parvovirus Infection in South American Canids

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SUMMARY

Canine parvovirus (CPV) infections occurred in 5 of 35 South American canids at the Department of Conservation (DC), a breeding facility of the National Zoological Park in Front Royal, Va. The clinical signs were anorexia, lethargy, diarrhea, and vomiting. Three of the affected canids survived and had high hemagglutination-inhibition titers to CPV in the recovery period. Necropsy of the 2 that died revealed extensive necrosis of the intestinal mucosa; CPV particles were observed by electron microscopy in the intestinal contents of both animals. Six of the 30 canids that remained healthy had high hemagglutination-inhibition titers to CPV prior to the episode of illness, indicating earlier subclinical exposure. Pet dogs belonging to DC personnel that were screened as a possible source of the infection had no evidence of disease. All canids (including pet dogs) on the DC grounds were vaccinated repeatedly with a killed feline panleukopenia virus product after the episode, with little or no effect on existing titers.

CANINE PARVOVIRUS (CPV) infection is a well-recognized syndrome in domestic dogs, with worldwide distribution; cases have been seen in the United States,[1-6] Canada,[7] Australia,[8,9] and Great Britain.[10,11] The disease has been described in 1 South American canid, the maned wolf.[12] In the present report, we describe CPV infection in 3 species of South American canids.

Results of studies of hemagglutination-inhibition (HI) titers are described, and possible routes of infection of captive South American canids are discussed.

History

Initially, a CPV-like disease occurred at the National Zoological Park's Department of Conservation (DC), in Front Royal, Va. During the 1st half of September 1979, 3 South American canids—1 bush dog (BD 1; *Speothos venaticus*), 1 maned wolf (MW 1; *Chrysocyon brachyurus*), and 1 crab-eating fox (CEF 1; *Cerdocyon thous*)—developed a disease characterized by anorexia, lethargy, vomiting, and foul-smelling or hemorrhagic diarrhea (Table 1). All 3 animals were hospitalized and were given supportive therapy (antibiotics, fluids, and multiple vitamins). The animals promptly recovered and

TABLE 1—South American Canids Studied in Episode of CPV Infection

Animal	Age (yr)	Sex	Duration of illness
Bush dogs			
BD 1	(9 mo)	M	9/8/1979–9/14/1979
BD 2	3	F	10/2/1979–10/6/1979*
BD 3	2½	M	†
BD 4	5½	M	†
BD 5	3½	F	†
Maned wolves			
MW 1	3	F	9/3/1979–9/8/1979
MW 2	2	M	10/14/1979–10/19/1979*
MW 3	>6	F	†
Crab-eating foxes			
CEF 1	3½	M	9/11/1979–9/14/1979
CEF 2	2½	M	†
CEF 3	2½	F	†

*Died. †Clinically normal.

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were returned to their compounds within 1 week. The initial laboratory findings from these 3 animals were considered to be within normal limits for these species. Blood cultures were negative for bacteria, and fecal culturing revealed normal flora. Fecal flotations were negative for parasites. Approximately 6 weeks after recovery, all 3 animals had high HI titers for CPV (1:640 to 1:5,120), which persisted until early 1980.

On Oct 2, 1979, a 2-year-old female bush dog (BD 2) became ill. Clinical signs were anorexia, diarrhea, and depression. The animal was hospitalized and treated with antibiotics, fluids, and vitamins. Blood and fecal specimens obtained at admission were negative for pathogens. The WBC count was 4,900/mm^3 (normal = 6–10,000/mm^3).[a] During the next 3 days, the count dropped to 800/mm^3. The animal became progressively more depressed and continued to vomit and to have hemorrhagic diarrhea. Despite continued supportive therapy, the bush dog died on the 4th day of hospitalization. Five months earlier, the HI titer for CPV was ≤1:20; serum was not available for a titer determination at the time of illness. Necropsy findings were compatible with CPV infection, which was confirmed by identification of CPV particles on electron microscopy of intestinal contents.

The 2nd maned wolf (MW 2) to develop signs of CPV-like disease had been shipped from Rotterdam to the United States on Sept 12, 1979. After spending 1 night in an animal shelter in New York City, it was placed in the quarantine facilities at the National Zoological Park (NZP). These facilities are in a separate building from the hospital; personnel don fresh coveralls and rubber boots and cross an antiseptic footbath upon entering the quarantine facility. On Oct 4, 1979, the wolf was transported to the hospital for an initial physical examination. At this time, the animal was clinically normal. Since the examination occurred at the same time that BD 2 was in the hospital with hemorrhagic diarrhea, numerous precautions to prevent cross infection were taken: (1) All procedures except radiography were performed in a room where the sick bush dog had not been. (2) Fresh coveralls and rubber boots were donned by clinical personnel before handling the maned wolf. (3) The radiography equipment (which was used on both the bush dog and the maned wolf) was thoroughly disinfected with chlorhexidine,[b] the disinfectant of choice at the time in both the hospital and quarantine facilities. This incident was the only time the maned wolf was out of the quarantine facilities; the exposure in the radiography room was the only known opportunity for exposure to CPV. Ten days later, the animal was found in its quarantine facility in sternal recumbency, unable to rise. We immediately instituted intensive care, which included fluids, antibiotics, vitamins, intestinal protectants, and oral alimentation. The wolf began to have fluid, foul-smelling diarrhea, and vomited the majority of any fluids administered orally. Inasmuch as we suspected CPV infection, 250 ml of whole blood from the recovered maned wolf (MW 1) was administered in an attempt to confer some degree of passive immunity. The urine output of the affected animal dropped to 0.5 ml/hr. Dopamine was administered (5 μg/kg/min, IV) to stimulate urine output. The urine output increased and over the next 24–48 hours the animal seemed to improve and was able to stand briefly. Antibiotic therapy consisted initially of penicillin, followed by carbenicillin and ampicillin. The animal's condition began to deteriorate after 72 hours; diarrhea and vomiting continued, sternal recumbency recurred, and oliguria progressed to anuria. Five days after the initial collapse, the animal died, apparently of renal failure.

The WBC of MW 2 had initially decreased and then increased to normal numbers during the latter period of intensive care. On the day of suspected exposure (10/4), the total WBC count was 10,400/mm^3. On the 1st day of clinical signs (10/14), the count had dropped to 2,500/mm^3. The next day (10/15), the count reached its low point (1,200/mm^3). The counts on these 2 days consisted of approximately 75% lymphocytes. The WBC count for the next 4 days (10/16–10/19) showed an increase to a more normal number of leukocytes (11,000/mm^3 on 10/19), along with the return of adequate numbers of granulocytes (80% segmented cells, 10% band cells, and 10% lymphocytes).

After the death of MW 2, HI titers were determined from serum from this animal. On the date of his presumed exposure, the titer was ≤1:20. At the onset of clinical signs (10 days after exposure), the HI titer was 1:5,120, and it remained at this level until the animal died, 5 days later. Feces obtained from this animal at necropsy were examined by electron microscopy; CPV particles were identified.

Vaccination and HI Titers in Domestic Dogs

After the episode of CPV infection, we began to search for the source of CPV. Conversations with a veterinary practitioner in Front Royal, Va, revealed that he had seen fatal hemorragic diarrhea in pet dogs in town shortly before the 1st episode of diarrhea was seen at the DC. Since there were no serum samples available (all affected animals had died), it was impossible to confirm that illness as CPV infection. There were, however, 14 pet dogs that belonged to the DC staff and which lived on the DC campus. Although these animals were kept separated from NZP animals, we believed it would be advantageous to determine HI titers and then to vaccinate them against CPV infection. The dogs were vaccinated on 10/24/1979 and again 2 weeks later with a killed feline panleukopenia virus (FPLV) vaccine.[c] Before the animals were vaccinated the 1st time, blood was drawn from all 14 animals. Approximately 5 months after the 2nd vaccination, HI titers were determined from 6 randomly selected dogs

[a]Values for clinically normal South American canids in this report compiled from National Zoological Park records.

[b]Nolvasan, Fort Dodge Laboratories, Fort Dodge, Iowa.

[c]Felocine, Norden Laboratories, Lincoln, Neb.

TABLE 2—Hemagglutination-Inhibition Titers for CPV in Domestic Dogs at DC, Front Royal, Va

Dog No.	Titer, by date 10/24/1979	3/27/1980
1	<1:10	<1:10
2	<1:10	<1:10
3	<1:10	<1:10
4	<1:10	1:40
5	<1:10	<1:10
6	<1:10	1:20
7	<1:20	ND
8	<1:10	ND
9	<1:10	ND
10	<1:10	ND
11	<1:10	ND
12	<1:10	ND
13	<1:10	ND
14	<1:10	ND

Vaccination for CPV: 10/24/1979 and 11/8/1979. ND = not determined.

(Table 2). None of the 14 pet dogs kept on the grounds of the DC ever developed signs typical of CPV infection.

Vaccination, HI Titers, and Clinical Histories in South American Canids

The remaining canids at DC were vaccinated with a killed FPLV product[c] shortly after the death of MW 2. Each of the canids was vaccinated on 3 occasions, at 2-week intervals. None of the animals in this series had any evidence of CPV infection. Titers for CPV were determined from serum samples available in the serum bank of the NZP. Some of these samples were obtained prior to the episode of CPV infection at DC. The results are shown in Table 3.

Many of the South American canids that did not have signs of CPV infection had high HI titers. Bush dog 3 had come from Frankfurt, Germany, on 8/8/1979. It had spent 1 night in the same animal shelter in New York City as had MW 2, and then it had been sent to the quarantine facilities at NZP. This animal has never had any signs of CPV infection, nor has it ever been to DC, because it went directly into the main zoo collection after a 30-day quarantine. Although the animal was vaccinated (killed FPLV) on 10/16/1979, 11/3/1979, and 12/4/1979, the initial HI titer was determined from a postquarantine sample (before vaccination and before the animal was moved to the park).

Bush dog 4 also came to us from Frankfurt. It arrived at DC in 1976, 2 years before reports of CPV infection anywhere in the world. This animal has been a resident at DC throughout the episodes of CPV infection. It was vaccinated 3 times in late October and early November of 1979. The HI titer of 1:640 was determined on 3/26/1979, 6 months before the episode of CPV at DC.

Bush dog 5 died of hypothyroidism. It never had any signs of CPV infection. The animal was vaccinated on 10/15/1979 and 12/24/1979. This animal, along with the remaining animals in the series (MW 3, CEF 2, and CEF 3), had been housed at the DC facilities since before the episode of CPV infection.

TABLE 3—Hemagglutination-Inhibition Titers for CPV from Clinically Normal South American Canids

Animal	Date ( ) and titer		
Bush dogs			
	(8/29/1979)	(10/16/1979)	(4/3/1980)
BD 3	1:2,560	1:5,120	1:5,120
	(3/26/1979)	(12/3/1979)	(4/3/1980)
BD 4	1:640	1:640	1:640
	(10/24/1979)	(11/19/1979)	
BD 5	1:640	1:1,280	ND
Maned wolf			
	(4/30/1979)	(10/16/1979)	(4/3/1980)
MW 3	1:160	1:160	1:160
Crab eating foxes			
	(5/12/1979)	(3/27/1980)	
CEF 2	1:20	1:10,240	ND
	(3/27/1980)		
CEF 3	1:2,560		ND

ND = not determined.

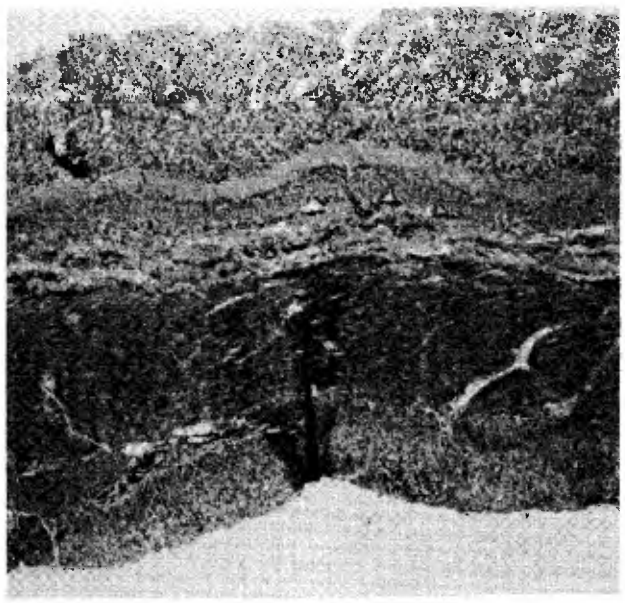

Fig 1—Photomicragraph of duodenum specimen from BD 2, showing extensive loss of villi. H&E stain; × 10.

Pathology

The gross and histopathologic findings in the 2 animals that died (BD 2 and MW 2) were typical of CPV infection.[13,14] The mucosa of the small intestine in both animals and the colon in MW 2 were thrown into thickened hyperemic folds, with segments of the intestine being devoid of epithelium. The mesenteric lymph nodes were enlarged and edematous. Microscopic examination of the small intestine of BD 2 revealed loss of villi, with matting of the remaining lamina propria (Fig 1) and with a paucity of inflammatory cells. Although there was no surface epithelium in the small intestine, there were occasional enterocytes deep in the mucosa. Some of these cells appeared to be regenerating forms, in that they were plump and pleomorphic (Fig 2).

In the intestinal mucosa of MW 2, there was considerable evidence of regeneration of enterocytes, but normal villous architecture had not been reestablished. The lamina propria was thickened and contained irregularly spaced, tortuous glands lined by hypertrophied cells with numerous crypt abscesses (Fig 3).

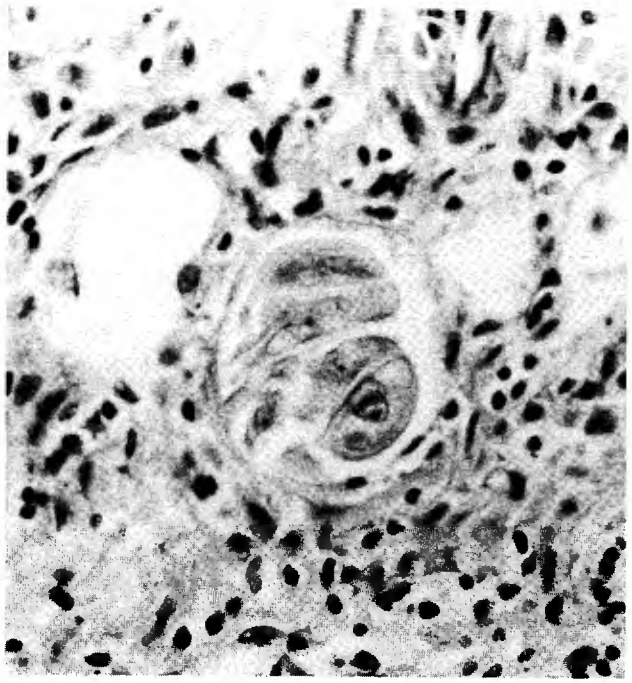

Fig 2—Photomicrograph of jejunal mucosa from BD 2, showing hyperplastic enterocytes in a crypt adjacent to several empty crypts altered by CPV infection. H&E stain; × 160.

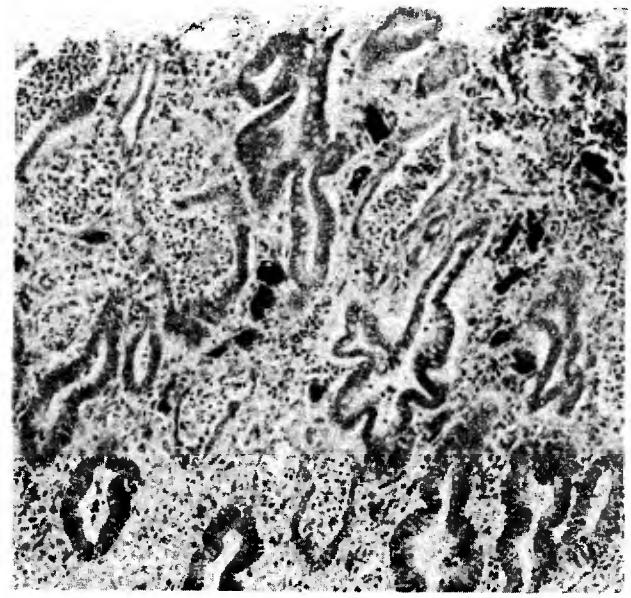

Fig 3—Photomicrograph of duodenum specimen from MW 2, showing tortuous glands and crypt abscesses. H&E stain; × 25.

There were areas of perivascular hemorrhage and necrosis in the cerebrum of MW 2, with numerous thrombi in small vessels, some of which contained fungal hyphae consistent with *Phycomycetes*. Fungal elements were also seen in foci of necrosis in the neuropil, surrounded by inflammatory cells.

Postmortem bacterial cultures from MW 2 revealed *Escherichia coli* in heart blood, spleen, and colon: findings that support the clinical impression of gram-negative sepsis.

Discussion

We assumed that the animals at DC were exposed to CPV at some point before September 1979. Although possible, it is unlikely that the sources of CPV were the pet dogs at DC. None of the 14 dogs tested had appreciable HI titers on 10/24/1979, indicating that they had not been exposed to CPV. (In general, HI titers of greater than 1:256 are considered to be indicative of clinical exposure to CPV.[9,12,15]) These domestic dogs received a series of 2 vaccinations with FPLV vaccine. When HI titers were determined on these animals 5 months later, only 2 dogs had a titer: one at 1:20, the other at 1:40. In general, dogs vaccinated with the killed vaccine product will have an initial titer in the range of 1:40–1:360 but will not have a detectable titer after 3 months.[5] An HI titer of at least 1:80 appears necessary to confer protection against CPV.[5]

The South American canids that survived had high HI titers (1:640–1:5,120) shortly after they recovered. These findings correlate with the situation in domestic dogs, wherein HI titers are generally quite high (1:640–1:10,240) at the time of infection; these titers do not generally increase during convalescence, but they do persist for months.[5] Two of the 3 recovered South American canids had titers persisting 6½ months after infection.

The HI titers in the clinically normal South American canids (BD 3, BD 4, BD 5; MW 3; CEF 2, CEF 3) indicated exposure to CPV at some point before September 1979, the time of the CPV infection at DC. The only animals to die (BD 2, MW 2) had titers of <1:20 before they developed clinical signs, indicating lack of prior exposure.

The regeneration that occurred in the intestine of MW 2, along with the return of a normal WBC count, implies that this animal might have survived the CPV infection had it not developed gram-negative sepsis and mycotic encephalitis.

The fact that the exposure of MW 2 evidently occurred after thorough disinfection of the facilities with chlorhexidine further underscores the resistance of CPV in the environment and the need to use proper disinfectants. After the death of MW 2, the entire hospital area was placed under quarantine, with all entrances and exits equipped with footbaths consisting of 5.25% sodium hypochlorite diluted 1:30 with water. This mixture is the accepted disinfectant[5,9] for areas contaminated by CPV.

None of the affected South American canids had any cardiopulmonary signs of CPV infection, as described in domestic dogs.[16,17] However, all domestic dogs so affected were pups, whereas the South American canids of this report were all adults, with the exception of BD 1, which was 9 months old.

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