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CLINICAL CANINE PARVOVIRUS TYPE 2C INFECTION IN A GROUP OF ASIAN SMALL-CLAWED OTTERS (*AONYX CINEREA*)

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Abstract: Despite the occurrence of clinical disease in a wide range of carnivore hosts, only vague accounts of clinical canine parvovirus type 2 (CPV-2) in any otter species have been reported in the literature. Over the course of 25 days, nine Asian small-clawed otters (*Aonyx cinerea*) presented for evaluation of inappetence, lethargy, vomiting, and diarrhea. A diagnosis of canine parvovirus type 2c was made based on electron microscopy, polymerase chain reaction, and DNA sequencing of group fecal samples. Supportive care was provided based on individual clinical assessment and included subcutaneous crystalline fluid therapy, antiemetics, antibiotics, appetite stimulants, and a neuraminidase inhibitor. Five of the nine otters exhibited moderate to severe disease requiring treatment, and one case was fatal despite supportive efforts. In light of this case report, CPV-2 should be recognized as a potential cause of gastrointestinal disease in Asian small-clawed otters.

Key words: *Aonyx cinerea*, Asian small-clawed otter, canine parvovirus, CPV-2, viral enteritis.

BRIEF COMMUNICATION

Canine parvovirus type 2 (CPV-2) causes gastrointestinal disease in a variety of carnivore species and is considered a disease of concern for mustelids. Seropositive North American river otters have been reported; however, little information regarding clinical disease in otters associated with CPV-2 appears in the literature.[1,2,4] This case report describes the presentation, clinical progression, diagnosis, and management of an outbreak of CPV-2 in a group of Asian small-clawed otters (*Aonyx cinerea*).

In August 2010, a female 5-yr-old Asian small-clawed otter housed with eight conspecifics presented for evaluation of progressive loss of appetite, lethargy, and vomiting over 3 days. Physical examination under general anesthesia on day 4 of clinical signs revealed dry oral mucous membranes, enlarged hyperemic tonsils, fair body condition, and a rectal temperature of 103.1°F. A complete blood count and serum chemistry panel were performed, which revealed a relative polycythemia (hematocrit of 58.4%; reference range 28.5–57.2%), hypoalbuminemia (2.0 g/dl, reference range 2.4–4.1 g/dl), and hypochloremia likely secondary to vomiting (108 mEq/L, reference range 110–128 mEq/L).[3] Direct and zinc sulfate fecal analyses revealed no evidence of parasites, and a urinalysis was unremarkable. Intestinal obstruction or foreign material on abdominal radiographs was not evident. Subcutaneous crystalline fluid therapy with 0.45% NaCl and 2.5% dextrose (Hospira Inc., Lake Forest, Illinois 60045, USA), ceftiofur crystalline-free acid (Excede®, Pfizer Inc., New York, New York 10017, USA; 6 mg/kg s.c.), and flunixin meglumine (FlunixiJect™, Butler Schein Animal Health, Dublin, Ohio 43017, USA; 0.8 mg/kg s.c.) were administered. Anesthetic recovery was uneventful, but the animal did not improve clinically.

On day 6 after onset of clinical signs, the animal was immobilized for endoscopic evaluation of the upper gastrointestinal tract. The gastric mucosa appeared diffusely hyperemic, and several white plaques were observed. Evaluation of the small intestine was attempted, but advancement of the endoscope into the duodenum was unsuccessful. A gastric wash sample was submitted for aerobic bacterial culture and cytology, and a rectal swab was submitted for bacterial culture. Subcutaneous fluid therapy consisting of 0.45% NaCl and 2.5% dextrose and metoclopramide (Metoclopramide Inj., USP, Hospira Inc.; 0.2 mg/kg s.c.) were administered as symptomatic treatments. Cytology from the gastric wash revealed squamous epithelial cells and extracellular bacteria without evidence of suppurative inflammation or exfoliative neoplasia.

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Because of the animal's persistent clinical signs, an exploratory laparotomy was performed on day 7 of clinical signs. No gastrointestinal obstruction was found; however, the gastric mucosa was hyperemic, and the gastric wall was slightly thickened. Paint chips were found within the lumen of the stomach and likely represented the white plaques that were observed endoscopically. Both adrenal glands were enlarged. The remainder of the exploratory laparotomy was otherwise unremarkable. Full-thickness biopsies of the stomach were submitted for histopathologic evaluation. Because of concern for heavy metal toxicity, serum and paint chips from the stomach were submitted for zinc levels. The animal was treated empirically with meloxicam (Metacam®, 1.5 mg/ml oral suspension, Boehringer Ingelheim Vetmedica Inc., St. Joseph, Missouri 64506, USA; 0.17 mg orally), ranitidine (Ranitidine tablets 300 mg, USP, Amneal Pharmaceuticals, Hauppauge, New York 11788, USA; 2.8 mg/kg orally), and clarithromycin (Biaxin®, 250 mg/teaspoon, Abbot Laboratories, North Chicago, Illinois 60064, USA; 50 mg/kg orally).

On day 7, a second otter presented for lethargy, inappetence, and vomiting. Over several days, additional otters developed similar clinical signs, and by day 10, five otters were affected. Two separate group fecal samples were submitted for CPV-2 and canine coronavirus polymerase chain reaction (PCR). Both samples were PCR positive for CPV-2, and sequencing indicated VP2 change of 426 glutamic acid, commonly referred to as CPV type 2c. Electron microscopy of pooled fecal samples revealed viral particles consistent in size and morphology with a virus in the *Parvoviridae* family. Biopsies of the stomach from the index case were histologically normal, and the gastric wash culture had no aerobic bacterial growth. There was heavy mixed aerobic and anaerobic growth consistent with normal fecal flora on the rectal swab culture. Levels of zinc in the serum and paint chips from the index animal did not support a diagnosis of zinc toxicosis.

Over a period of 1 mo, all otters in the group exhibited clinical signs consistent with CPV-2 infection, including lethargy, inappetence, anorexia, vomiting, and ataxia related to weakness. Diarrhea was consistently found within the otter enclosure throughout the outbreak; however, determination of the affected individuals was not feasible. Four cases were considered mild in severity, and one case was considered moderate. Four additional cases were considered severe, with persistent clinical signs lasting longer than 7 consecutive days. One of these resulted in mortality after 7 days. All moderate to severe cases were treated supportively with subcutaneous crystalloid fluid therapy containing dextrose, antibiotics, antiemetics, appetite stimulants, H2 blockers, and vitamin B complex as indicated by daily assessment of clinical signs. All otters received oseltamivir phosphate (Tamiflu®; Roche Laboratories Inc., Nutley, New Jersey 07110, USA; 2 mg/kg orally), regardless of clinical signs, beginning on day 12 after the onset of the index case. Quarantine, disinfection, and biosecurity measures were instituted. A summary of clinical signs, duration of illness, and treatment for affected individuals is depicted in Table 1. After the outbreak, serum hemagglutination inhibition antibody titers for CPV-2 on frozen banked serum samples collected before the outbreak were performed for seven of the nine otters. All samples had been obtained within 3.2 yr before the outbreak and had been stored at −80°C before analysis. The results of these titers are also presented in Table 1.

Gross postmortem examination of the deceased otter revealed thin body condition, empty stomach, thickening of the intestinal walls, segmental hyperemia of the intestinal mucosa, and a misshapen spleen with omental adhesions. Histopathology revealed moderate lymphocytic and eosinophilic enteritis with mild crypt necrosis, crypt hyperplasia, and goblet cell hyperplasia. Immunohistochemistry was performed for CPV-2, and in rare intestinal crypts, the cytoplasm of attenuated epithelium had stain uptake, which was lighter than the control.

The clinical signs of CPV-2 in the otters of this report were similar to those seen in other species. Early clinical signs in other species usually consist of depression, inappetence, and vomiting. These were the most common clinical signs observed in affected otters of this outbreak. Because diagnosis of infection was performed on group rather than individual samples, it may be argued that mild signs in some individuals could have been unrelated to CPV-2 infection. Supportive care consisting of subcutaneous fluid therapy, injectable antiemetics, and injectable antibiotics was used in the otters of this case report. Although oseltamivir was used in this case, it has not been shown to be an effective treatment for CPV-2.

Vaccination is the mainstay of CPV-2 prevention in domestic canines, and current recommendations include vaccination as part of a comprehensive preventive care program for *A. cinerea*.[6] Poor seroconversion has been demon-

Table 1. Characteristics of clinical disease exhibited by Asian small-clawed otters (*Aonyx cinerea*) during an outbreak of canine parvovirus type 2c.

Animal	Sex	Age (yr)	Clinical signs[a]					Severity	HI[b] titer	Onset postindex (d)	No. days affected	Duration of illness (d)	Days with clinical signs (%)	Treatments administered[c]
			Inapp.	Vom.	Leth.	Ataxia	Death							
1	M	5	•	—	—	—	—	Mild	N/A	23	1	1	100	Oseltamivir
2	F	5	•	—	•	—	—	Mild	1:80	21	1	1	100	Oseltamivir
3	M	5	•	•	—	—	—	Mild	1:80	16	2	3	67	Oseltamivir
4[d]	M	17	•	•	—	—	—	Mild	1:320	9	7	16	67	Oseltamivir
5	F	10	•	•	—	—	—	Mod	1:20	13	4	14	29	Ceftiofur CFA, ondansetron, oseltamivir
6	F	5	•	•	•	—	—	Severe	1:160	0	13	14	93	SC fluids, ceftiofur CFA, flunixin meglumine, ranitidine, meloxicam, metoclopramide, clarithromycin, oseltamivir
7	F	5	•	•	•	•	—	Severe	1:80	7	16	19	84	Ceftiofur CFA, ondansetron, ranitidine, oseltamivir
8	M	5	•	•	•	•	—	Severe	N/A	7	18	18	100	SC fluids, ceftiofur, diazepam, ondansetron, ranitidine, vitamin B complex, oseltamivir
9	F	5	•	•	•	•	•	Fatal	1:40	9	7	7	100	Ceftiofur CFA, ondansetron, oseltamivir
Group totals (%)	—	—	100	78	56	33	11	—	—	—	—	—	—	—
Group means	—	—	—	—	—	—	—	—	—	—	7.7 ± 6.5	10.3 ± 6.9	—	—

[a] Inapp., inappetence; Vom., vomiting; Leth., lethargy; •, clinical sign present. Diarrhea was observed for the otter group, but determination of affected individuals was not possible.
[b] HI, hemagglutination inhibition assay; N/A, not available. All results were obtained from frozen banked serum collected within 3 yr 2 mo before the outbreak.
[c] SC, subcutaneous; CFA, crystalline free acid. Subcutaneous fluid therapy consisted of administration of 0.45% NaCl with 2.5% dextrose.
[d] Animal was vaccinated before outbreak for canine parvovirus type 2c using modified live or killed vaccine.

strated in North American river otters after administration of killed vaccines for CPV-2,[4] and their efficacy in preventing infection in otters remains questionable. Although modified live CPV-2 vaccines have proven effective against infection in domestic canines,[5] their use is not currently recommended in otters because of the potential for vaccine-induced clinical disease. Further investigation would be necessary to determine whether modified live vaccines for CPV-2 are safe and effective in otters. Only one animal in the group had been previously vaccinated for CPV-2 using modified live or killed vaccines. This otter's titer was the highest of all animals before the outbreak, and it exhibited only mild clinical signs.

This case demonstrates that CPV-2c may cause clinical disease of varying severity in Asian small-clawed otters. To the authors' knowledge, this has not been reported previously. In light of this report, CPV-2 should be considered a differential diagnosis of gastrointestinal disease in Asian small-clawed otters.

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