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Death of a Wild Wolf from Canine Parvoviral Enteritis

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ABSTRACT: A 9-mo-old female wolf (*Canis lupus*) in the Superior National Forest of Minnesota (USA) died from a canine parvovirus (CPV) infection. This is the first direct evidence that this infection effects free-ranging wild wolves.

Key words: Canine parvovirus, wolf, *Canis lupus*, disease, serology, mortality.

In the U.S., serologic evidence of canine parvovirus (CPV) infection has been found among wild populations of wolves (*Canis lupus*) in Minnesota (Mech et al., 1986), Wisconsin (Wydeven et al., 1995), Alaska (Zarnke and Ballard, 1987), and Montana (Johnson et al., 1994). Evidence also was found that CPV infection may be influencing population changes in wolves in the central Superior National Forest (SNF) of Minnesota through early pup mortality (Mech and Goyal, 1995). However, the only documentation of wolves dying from CPV infection involved 10 captive, 2 to 14 mo-old wolves (Mech et al., 1986). In the present note, we document death of a wild wolf due to CPV infection in the SNF (48°N, 92°W).

Female wolf 409 was captured as a 6-mo-old, 22-kg pup on 29 October 1992, blood-sampled, outfitted with a radio-controlled capture collar (Mech and Gese, 1992), and radio-tracked; her serum tested <1:256 for CPV antibodies (hemagglutination inhibition test); we considered this negative because samples with lower titers were negative by the ELISA test whereas samples with 1:256 were positive (Mech et al., 1986). The animal was recaptured via the capture collar on 8 December 1992, weighed 27 kg, and it again was negative for CPV antibodies.

This wolf was last found alive on 21 January 1993. On 2 February 1993 it was dead and weighed 19.5 kg. The carcass was kept cool, necropsied on 11 February 1993, and found to be dehydrated and in poor condition. The carcass was autolytic. Grossly, the enteric lesions were characterized by a granular consistency over the serosal surface of the terminal portion of the small intestine. There was hyperemia and hemorrhage in the wall of the ileum. The ileal wall was thin, the mucosal surface had a yellow tinge, and contents of the dilated lumen were serosanguinous, bile-stained, and had a putrid odor. The colon contained blood-stained fluid contents. The lungs appeared edematous. No other gross lesions were observed.

The brain, liver, lungs, heart, kidneys, stomach, jejunum, ileum, and colon were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 5 μm and stained with hematoxylin and eosin.

Microscopic lesions were confined to

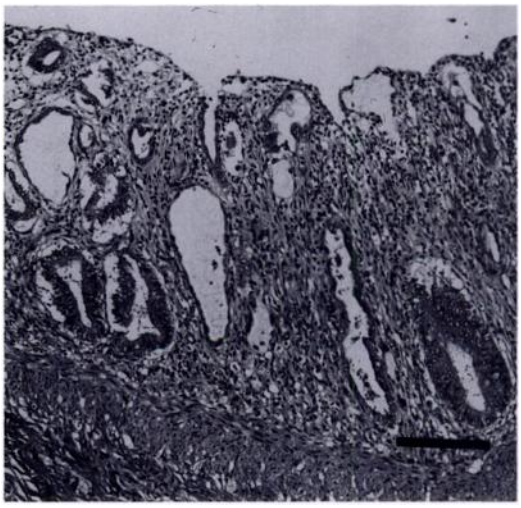


FIGURE 1. Ileal mucosa with loss of villi and superficial enterocytes. The lamina propria is collapsed and contains dilated crypts which are lined by attenuated or hyperplastic enterocytes. H & E. Bar = 100 μm.

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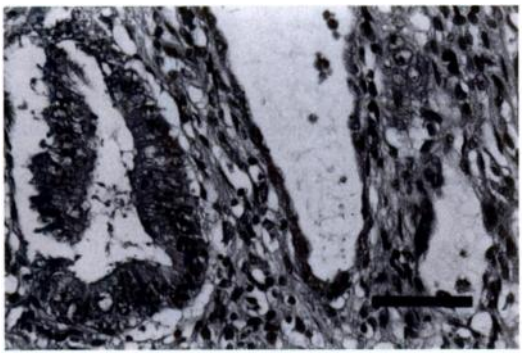


FIGURE 2. Higher magnification of Figure 1. There are hyperplastic enterocytes lining dilated crypts and other dilated crypts lined by attenuated epithelial cells. H & E. Bar = 50 μm.

the terminal portion of the small intestine and were partially obscured by autolytic changes. Sections of ileum had chronic change consisting of dilated intestinal crypts which were lined by enterocytes which were attenuated and flattened (Figs. 1 and 2). Many dilated glandular crypts contained degenerated inflammatory cells which were principally polymorphonuclear leukocytes. Some crypts were lined by proliferating regenerative enterocytes (Figs. 1, 2). The lamina propria was collapsed and had a fibrous appearance due to loss of enterocytes lining the villi. The intestinal surface epithelium was attenuated, and in some areas there was a complete lack of surface enterocytes. Peyer's patches were almost completely devoid of germinal centers and lymphocytes. The ileal lesions were considered characteristic of parvoviral enteritis. Parvoviruses in low numbers were found in the feces by electron microscopy (Muneer et al., 1988).

From this case we conclude that CPV infection can kill wild wolves older than newborn pups, and wild wolves can become infected with CPV during winter. These findings are of value because they represent the first direct documentation of CPV effects on wild wolves.

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