Rotaviral Enteritis in a Raccoon

Authors: Hamir, A. N., Morin, M., and Rupprecht, C. E.

Source: Journal of Wildlife Diseases, 26(2) : 262-264

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-26.2.262

Journal of Wildlife Diseases, 26(2), 1990, pp. 262–264

Rotaviral Enteritis in a Raccoon

A. N. Hamir,[1] M. Morin,[2] and C. E. Rupprecht,[3] [1] Laboratory of Large Animal Pathology, University of Pennsylvania, School of Veterinary Medicine, New Bolton Center, Kennett Square, Pennsylvania 19348, USA; [2] Faculty of Veterinary Medicine, University of Montreal, Saint-Hyacinthe, Quebec, Canada J2S 7C6; [3] Wistar Institute of Anatomy & Biology, Rabies Research Unit, Philadelphia, Pennsylvania 19104, USA

ABSTRACT: A hand-reared raccoon (*Procyon lotor*) kit had severe diarrhea and died within 24 hr. Gross and histopathologic findings were compatible with a diagnosis of viral enteritis. The immunoperoxidase test revealed rotavirus group A antigen in the intestinal mucosa. This is the first record of rotaviral enteritis in a raccoon.

Key words: Rotavirus enteritis, raccoon, *Procyon lotor*, case report, rotavirus group A antigen, immunoperoxidase test.

Although rotavirus infections are reported from the intestinal tract of many species of mammals and birds (Barker and Van Dreumel, 1985), they have not been documented in the raccoon (*Procyon lotor*). Herein, we report a fatal case of rotavirus enteritis in a raccoon kit.

A 6-wk-old female raccoon kit was hand-reared together with its litter-mate after their dam was killed when hit by an automobile. At the time of the accident, the kits were approximately 2-wk-old. Both were hand-reared for the next 4 wk on an artificial diet containing milk replacer for puppies (Esbilac, Pet-Ag Inc., 201 Keyes Avenue, Hampshire, Illinois 60140, USA), and strawberry preserves (Kraft, Inc., Glenview, Illinois 60025, USA). Both kits were clinically normal until 24 hr prior to the death of one of them. The day before its death, the female kit was given a meal, but was noticed to be sick after finishing the meal. Over the next 6 to 12 hr, she showed profuse watery diarrhea, became dehydrated and died. The carcass was presented to the Laboratory of Large Animal Pathology (University of Pennsylvania, New Bolton Center, Kennett Square, Pennsylvania 19348, USA) for necropsy. Three days later, the second kit developed similar clinical signs and died. This kit was not available for necropsy.

At postmortem examination clear mucoid material was observed around the mouth. The animal was in a fair to good nutritional state (determined by the amount of body fat), but was markedly dehydrated and had pale mucous membranes. The significant gross lesions were confined to the alimentary tract. Both the stomach and the intestine were moderately distended with clear watery contents which were mucoid in the stomach. The wall of the posterior two-thirds of the small intestines was extremely thin and the fluid and gas bubbles present within the lumen were easily seen. The mucosal surface of the affected intestinal segments was extremely pale.

Representative samples of organs (heart, lung, liver, kidney, spleen, lymph nodes, stomach, intestines, adrenals, urinary bladder and brain) from this carcass were fixed in 10% buffered formalin. Blocks of tissues were embedded in paraffin, cut at 6 μm and stained with hematoxylin and eosin for light microscopy. Since the kits were obtained from a raccoon rabies epizootic area, the brain was submitted to the Wistar Institute (36th Street, Philadelphia, Pennsylvania 19104, USA) for fluorescent antibody testing for rabies virus diagnosis. Paraffin sections of small intestines were submitted to the Faculty of Veterinary Medicine (University of Montreal, Quebec, Canada J2S 7C6) for detection of rotavirus and coronavirus antigens (transmissible gastroenteritis of pigs; bovine rotavirus group A and coronavirus of calves) by using the avidin-biotin immunoperoxidase technique (Immunotags by Immunon, Troy, Michigan 48210, USA). The primary viral antibodies were produced by the Institute Armand Frappier (Laval, Quebec, Canada H7N 4Z3).

Histopathologic examination of the tis-

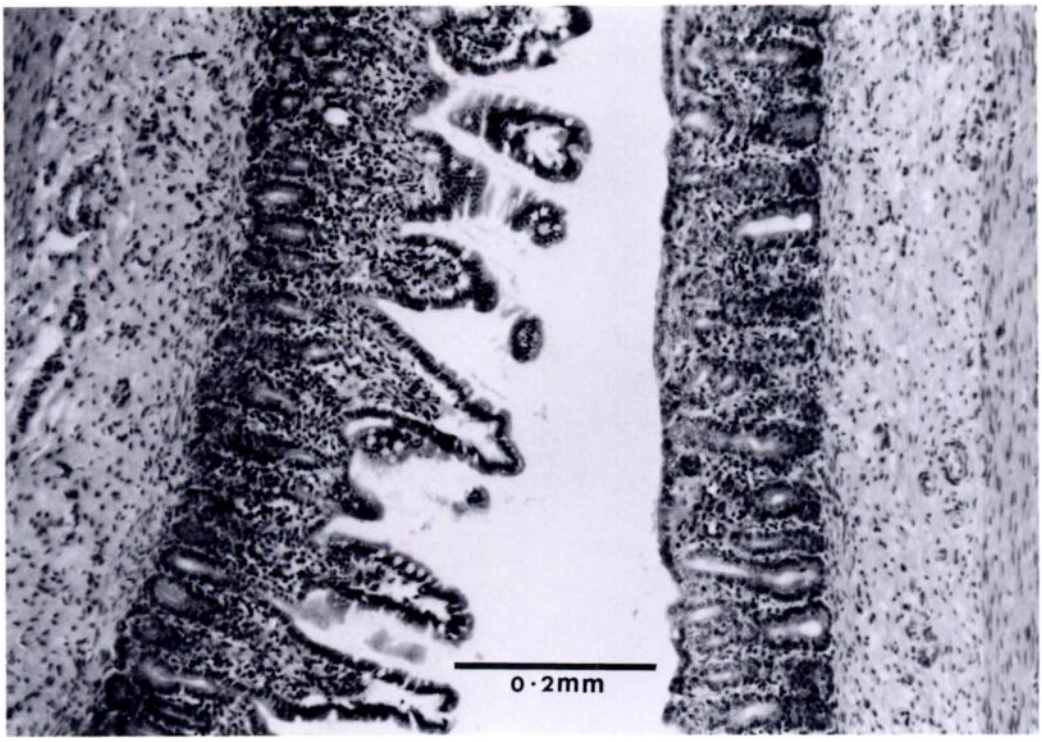

FIGURE 1. Small intestines of a raccoon kit showing markedly atrophic mucosa on the right and relatively normal mucosa on the left. H&E. Scale bar = 200 μm.

sues revealed lesions that were confined to the grossly affected parts of the small intestines. The lesions were characterized by marked atrophy of the mucosal layer and exhibited loss of villi (Fig. 1). The surviving villi were markedly shortened and were covered by cuboidal to flattened enterocytes which had clear to multivacuolated cytoplasm and poorly developed microvilli on their surface.

The immunoperoxidase test was positive for rotavirus group A antigen which was observed within enterocytes at the tips of the atrophic villi (Fig. 2). The test was negative for coronaviruses. The brain tissue was negative for rabies.

In young raccoons that are captured for the purpose of translocation, infectious viral gastroenteritis appears to be a relatively common clinical finding (Nettles et al., 1980). However, the only reported etiologic agent in such cases has been parvovirus (Waller, 1940; Nettles et al., 1980). The presently described case of rotavirus enteritis is the first reported case in a raccoon.

Rotaviruses are widespread among populations of most species of mammals and they are resistant to the external environment (Barker and Van Dreumel, 1985). In this case, the raccoon kits had not been exposed to other raccoons subsequent to when they were orphaned. However, they were attended by a person who had close contact with dead animals (postmortem technician). Since some strains of the rotaviruses are pathogenic across species (Barker and Van Dreumel, 1985), these raccoon kits may have contracted the infection from other infected animals through their handler.

The gross and microscopic lesions seen in this raccoon kit were similar to those that have been described for rotavirus infections in other species (Barker and Van Dreumel, 1985). In swine, rotaviruses can

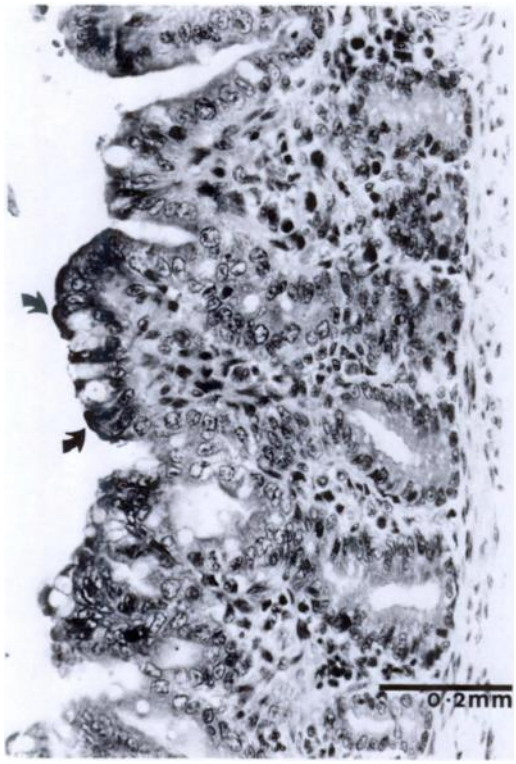

FIGURE 2. Intestinal mucosa of a raccoon kit showing rotavirus positive antigen in enterocytes at the surface of an atrophic villus (arrows). Immunoperoxidase (avidin-biotin) stain. Scale bar = 200 μm.

cause fatal diarrhea in piglets if they are weaned or if the dam does not have rotavirus specific antibodies in her milk (Woode, 1986). The raccoon kits in the present case faced similar circumstances when they were orphaned and may have suffered similar consequences. Regardless, it appears that rotavirus group A, probably derived from a non-raccoon host, was responsible for the diarrhea and death of these raccoon kits. This infection should be considered in situations involved with hand-rearing raccoon kits.

This research was sponsored in part by a grant from the Commonwealth of Pennsylvania, Department of Agriculture.

LITERATURE CITED

BARKER, I. K., AND A. A. VAN DREUMEL. 1985. The alimentary system. *In* Pathology of domestic animals, 3rd ed., Vol. 2. Academic Press, Orlando, Florida, pp. 2–203.

NETTLES, V. F., J. E. PEARSON, G. A. GUSTAFSON, AND J. L. BLUE. 1980. Parvovirus infection in translocated raccoons. Journal of the American Veterinary Medical Association 177: 787–789.

WALLER, E. F. 1940. Infectious gastroenteritis in raccoons (*Procyon lotor*). Journal of the American Veterinary Medical Association 96: 266–269.

WOODE, G. N. 1986. Porcine rotavirus infection. *In* Diseases of swine, 6th ed., A. D. Leman, B. Straw, R. D. Glock, W. L. Mengeling, R. H. C. Penny, and E. Scholl. (eds.). Iowa State University Press, Ames, Iowa, pp. 368–382.

Received for publication 3 August 1989.