# ROTAVIRUS (REOVIRUS-LIKE) INFECTION OF NEONATAL RUMINANTS IN A ZOO NURSERY

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# ROTAVIRUS (REOVIRUS-LIKE) INFECTION OF NEONATAL RUMINANTS IN A ZOO NURSERY

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*Abstract:* An outbreak of a pneumoenteric disease occurred in neonates in a zoo nursery. Four of seven affected animals died. Rotaviruses were observed in the feces of an affected 4-day old impala *(Aepyceros melampus),* a Thomson's gazelle *(Gazella thomsonii)* and an addax *(Addax nasomaculatus).* Encapsulated *Escherichia coli* also were isolated from the feces. The recovered rotaviruses were antigenically related to bovine rotavirus. A bovine rotavirus vaccine was given orally and no adverse effects were noted.

## INTRODUCTION

Rotaviruses, also referred to as reovirus-like agents have been incriminated as the cause of diarrhea in calves,[6] piglets,[5] foals,[4] suckling mice,[8] rabbits,[2] monkeys[3] and human infants.[1] Diarrhea has been reproduced by experimental inoculation of rotaviruses in calves and piglets. Rotaviruses first replicate in the epithelial cells of the small intestinal villi, resulting in some denuding. This initial viral-induced damage to the intestinal epithelium probably results in an exacerbation of the lesion by the normal bacterial flora. Mebus and co-workers[7] have shown that experimental infection with rotaviruses induce only a mild transitory diarrhea in gnotobiotic calves while the rate of mortality is high in field cases.

To our knowledge there have been no reports on rotavirus infections in zoo animals. The following represents a description of the clinical and laboratory findings of an outbreak of diarrhea in a zoo nursery.

## CLINICAL HISTORY

Eight infants, including impala *(Aepyceros melampus),* addax *(Addax nasomaculatus),* Thomson's gazelle *(Gazella thomsonii),* Grant's gazelle *(Gazella subgutterosa),* were placed in the zoo nursery within the first few days of their lives because: (1) the weather was extremely cold and wet during this time (January) and (2) some of them were rejected by their dams. The nursery was overcrowded in January and sick animals could not be isolated. All affected infants were between 1-7 days of age. The course of the disease was acute, some dying within 12 h. after onset of signs. The first signs were a moderate fever, anorexia, clear mucoid nasal discharge, excessive salivation and a yellowish, watery diarrhea. In most cases death appeared to be due to respiratory arrest. Of the eight infants, four succumbed to the infection, three recovered and one remained healthy.

Three animals (one impala, one addax, one Thomson's gazelle) 4-6 days old were presented dead for necropsy.

## NECROPSY AND LABORATORY FINDINGS

**Gross Lesions:** The nasopharyngeal and tracheal mucosa were hyperemic and, in some cases, petechial hemor-

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rhages were noted on the tracheal mucosa. The thoracic cavity and the pericardial sac contained a slightly to moderately increased amount of straw-colored fluid with no fibrin except for one case in which a few fibrin strands were noted in the pericardial sac. The lungs usually were edematous with prominent interstitia. Livers were congested. The intestines contained a yellowish, watery ingesta. The mucosa in the lower parts of the small intestine was hyperemic. The mesenteric as well as retropharyngeal lymph nodes were swollen. The umbilici were normal.

**Microscopic Lesions:** All three animals examined had pneumonia — two had an interstitial type and one had a purulent type. Lesions in various lymph nodes ranged from edema and congestion to a purulent lymphadenitis with proliferation of macrophages. The intestines were not available for microscopic examination.

**Microbiological Results:** *Escherichia coli,* considered encapsulated on the basis of the colony appearance on Tergitol medium, [3] was isolated from the feces of all three animals. No pathogenic bacteria were isolated from any somatic organs. Contaminants, such as *Proteus sp.* and non-encapsulated *E. coli,* also were recovered from feces as well as some somatic organs. A *Salmonella sp.* was not isolated.

Fecal material was clarified by low speed centrifugation (1000 × g) and then pelleted by centrifugation at 30,000 xg for 1 h. Pellets were negatively stained with 4% phosphotungstic acid and examined by electron microscopy. Viral particles of similar morphology were observed in the feces of all three animals (Figure 1). The viruses were spherical, measuring about 60 nm in diameter. Peripheral projections were prominent and appeared to be profiles of empty capsomeres. Most of the viral particles observed did not have an extra capsid. The particles were identified morphologically as rotaviruses. Cytopathic viruses were not isolated from the feces or somatic organs (lung, liver, spleen) using primary fetal bovine kidney cell cultures. The partially purified fecal pellet containing the rotaviruses was subjected to immunelectronmicroscopy.[9] Rotaviruses from the impala, Thomson's gazelle and addax were incubated overnight with an antiserum against bovine rotavirus. [4] When this preparation was viewed in the electron microscope, a definite coating of the viral surface with proteinaceous (antibody) material was noted (Figure 2).

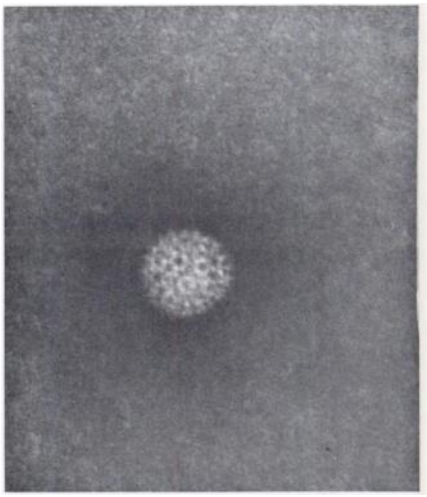

**FIGURE 1. Electronmicrograph of a negatively stained rotavirus. Diameter: 60 nm.**

## TREATMENT

The animals were given supportive treatment with various preparations such as ampicillin, chloromycetin, expectorants, liver iron preparations, Biosol-

[3] Difco Laboratories, Detroit, Michigan.

[4] Supplied by Norden Laboratories, Lincoln, Nebraska 68501, USA.

M[R],[5] electrolyte fluids and glucose. The course of the disease was very acute (about 12 h.) and was not markedly altered by treatment. After a diagnosis of a rotavirus infection was established, the remaining four animals were vaccinated orally with a bovine rotavirus vaccine (Scourvac-Reo[6]).

An additional six infants of various species were subsequently vaccinated orally with Scourvac-Reo upon entry into the nursery. Half the regular bovine dose was used and no adverse effects were noted. No additional cases developed following vaccination.

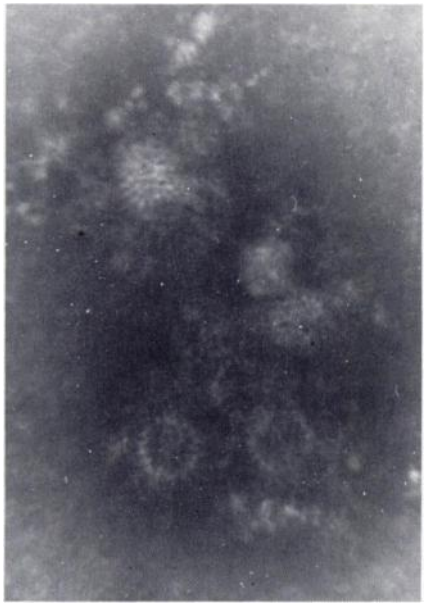

FIGURE 2: Electronmicrograph of rotaviruses coated with bovine rotavirus antiserum.

## DISCUSSION

The source of the virus in this outbreak was not established. In bovines, the adult animals, most likely the dams, are thought to be asymptomatic carriers of rotaviruses and excrete them in the feces. The extremely cold and wet weather conditions could have been stress factors responsible for increased shedding of the virus from carriers. Once the virus was introduced in the nursery it certainly may have spread horizontally since it was overcrowded and isolation facilities for sick animals were not available.

The lesions induced in these three animals probably were the result of both a rotavirus and *E. coli* infection. We have not examined organs other than the intestines for rotaviruses by electron microscopy but rotaviruses have been demonstrated in the lungs of calves.[7] Experiments in gnotobiotic calves have revealed that rotaviruses alone destroy epithelial cells in the lower small intestine. However, the severity of the infection and the rate of mortality were increased if enteric bacteria, such as encapsulated *E. coli* also were present.[7]

Based on the immunelectronmicroscopic results, the virus recovered from these animals appears to be antigenically related to the bovine rotavirus. Woode *et al.*[9] also have shown antigenic relationship between the bovine rotaviruses and those of equine, porcine and murine origin.

No further clinical cases were observed after vaccination with bovine rotavirus vaccine. However, no firm conclusions on the effectiveness of the vaccine can be drawn from this finding since the peak of the epizootic had passed at the time of vaccination.

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[5] The Upjohn Company, Kalamazoo, Michigan, USA.

[6] Norden Laboratories, Lincoln, Nebraska, USA.

## LITERATURE CITED

1. BISHOP, R.F., G.P. DAVIDSON, I.H. HOLMES and B.J. RUCK. 1973. Virus Particles in Epithelial Cells of Duodenal Mucosa from Children with Acute Non-Bacterial Gastroenteritis. Lancet ii: 1281-1283.
2. BRYDEN, A.S., M.E. THOULESS and T.H. FLEWETT. 1976. Rotavirus in Rabbits. Vet. Rec., 99: 323.
3. ELS, H.J. and G. LECASTSAS. 1972. Morphological Studies on Simian Virus SA 11 and the Related "O" Agent. J. Gen. Virol., 17: 129-132.
4. FLEWETT, T.H., A.S. BRYDEN and H.A. DAVIS. 1975. Virus Diarrhea in Foals and Other Animals. Vet. Rec., 96: 477.
5. LECCE, J.G., M.W. KING and R. MOCK. 1976. Reovirus-like Agent Associated with Fatal Diarrhea in Neonatal Pigs. Inf. & Immunity, 19: 816-825.
6. MEBUS, C.A., N.R. UNDERDAHL, M.B. RHODES and M.J. TWIEHAUS. 1969. Diarrhea (Scours): Reproduced With a Virus From a Field Outbreak. Univ. Nebr. Res. Bull. 233: 1-10.
7. ——, E.L. STAIR, N.R. UNDERDAHL and M.J. TWIEHAUS. 1971. Pathology of Neonatal Calf Diarrhea Induced by a Reo-like Virus. Vet. Path., 8: 490-505.
8. MUCH, D.H. and I. ZAJAC. 1972. Purification and Characterization of Epizootic Diarrhea of Infant Mice Virus. Inf. & Immunity, 6: 1019-1024.
9. WOODE, G.N., J.C. BRIDGES, J.M. JONES, T.H. FLEWETT, A.S. BRYDEN, H.A. DAVIES and G.B. WHITE. 1976. Morphological and Antigenic Relationship Between Viruses (Rotaviruses) From Acute Gastroenteritis of Children, Calves, Piglets, Mice and Foals. Inf. & Immunity, 14: 804-810.

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