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ENCEPHALOPATHY IN TWO BLACK RHINOCEROS (DICEROS BICORNIS) CALVES

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Encephalopathy characterized by massive necrosis of gray and white matter and accompanied by marked cerebral edema was noted in two black rhinoceros (Diceros bicornis) calves. Though the two cases differed in several respects, they share many historical and pathologic similarities that warrant joint consideration. Case 1 occurred in 1979 in a two-month-old calf at the Denver Zoological Gardens, and case 2 was a six-month-old animal at the St. Louis Zoo. Both calves were female, dam-raised, and were noted to be moderately depressed 12 hours before being found comatose and unresponsive. Both failed to respond to broad-based treatment and died within 24 hours of the onset of depression. Clinical pathology data collected premortem did not establish a diagnosis in either case.

Gross examination of both brains at necropsy revealed marked discoloration of soft, friable, edematous tissue. Cerebrospinal fluid (CSF) cultures from the Denver animal produced growth of a few colonies of nonhemolytic E. coli, as did cultures of the colon, small intestine and mesenteric lymph nodes. A CSF taken taken from case 2 immediately postmortem was unremarkable in its cell content, and a gram stain was negative. Culture of the CSF yielded a light growth of an alpha-streptococcus and Staphylococcus hominis.

Histologic examination of the brain tissues revealed that the gross lesions reflected extensive necrosis of cerebral gray and white matter in both cases. Extensive spongiform encephalopathy was noted in much of the white matter. Similar, less extensive lesions occurred in the rostral brain stem.

An etiology of the lesions is not readily apparent. Microscopic or cultural findings suggestive of Hemophilus somnus or listeriosis were not found. Mycotoxin assays of the feed in case 1 were negative, as were mouse clostridial enterotoxin assays performed on the stomach contents of case 2. Liver lead levels in case 2 were less than 1 ppm and serum alpha-tocopherol levels were 0.8 ug/dl. Etiologies that remain under consideration for this marked encephalopathy included vitamin E deficiency, polioencephalomalacia, viral encephalitis and possibly, edema disease of swine.

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