HEMOLYTIC ANEMIA IN THE BLACK RHINOCEROS (DICEROS BICORNIS)

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Introduction

Hemolytic anemia may result from a wide variety of causes that lead to the destruction of red blood cells within the vascular system. A previous report of hemolytic anemia in the black rhinoceros suggested an outbreak may have been attributable to leptospirosis (5). Another survey found no common etiology (3), and two cases that temporarily responded to steroids have been described (6). Babesiosis and trypanosomiasis have caused deaths in wild-caught black rhinoceroses from Africa, but their relationship with hemolytic anemia in this species is not clear (4,7,11). Known etiologies of hemolytic anemia in domestic animals have also served as a basis for the investigation into the black rhinoceros hemolytic syndrome.

Case History

An eight-year-old nulliparous female black rhinoceros at the St. Louis Zoo was presented in May 1981 for "blood in the urine." The discoloration was due to the presence of hemoglobin. Due to the animal's rapid deterioration, anesthesia with etorphine was attempted to obtain further diagnostic material. The animal died while anesthetized; anesthesia was most likely complicated by the severe anemia present at that time (PCV 14.5%). The sera were the "port wine" color of the hemoglobinemia. Gross necropsy findings, bacteriology, virology, toxicology and serological results were unremarkable in attempts to identify an etiology (8). Histology revealed chronic iron accumulation in parenchymatous tissues, suggesting a duration of anemia longer than the 48 hours it was clinically evident. Leptospirosis could not be cultured despite specific efforts. Titers for serovars of Leptospirosis interrogans were negative at one laboratory, but were 1:320 for ictohemorrhagica and 1:80 for ballum at another.

Survey Results

The original survey was mailed to 31 American, Canadian, British and Irish zoos that owned black rhinoceroses. Twenty institutions that had kept 98 black rhinoceroses over the previous ten years responded. They reported 33 deaths; 25 in animals greater than one year of age. Eleven of the adult deaths were associated with hemolytic anemia/hemoglobinuria (although in two cases, only an association with "blood in the urine" was noted). Five additional animals that had had nine episodes of hemolytic anemia (three fatal) were found in further contact with two continental European zoos.

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Typically, in the fourteen episodes associated with fatalities, clinical signs preceded death by 24 to 96 hours. Four animals survived previous episodes only to die during subsequent attacks. Where available, antemortem laboratory data were unremarkable besides the hemogram alterations associated with severe anemia; i.e., PCV's of 4.5-14.5 reflected in low erythrocyte and hemoglobin values. Treatments in survivors were supportive and nonspecific--hematinics, vitamin supplementation including vitamin K, antibiotics, corticosteroids in two cases, and a blood transfusion in one. Of the affected animals, ten were female and seven were male. Eleven were zoo bred and six were wild caught. The animals' ages at death ranged from 2 to 22 years (average age 9.6 years). Deaths occurred in February (1), April (2), May (1), June (2), July (3), October (1), November (2) and December (2). Five institutions each had two deaths associated with hemolytic anemia. The deaths of two of these pairs were related in time--an outbreak at the Memphis Zoo where three animals were affected and two died ten days apart, and Metro Toronto Zoo, where deaths of a pair were separated by 24 hours.

Of particular interest was the finding that three of four siblings born to the St. Louis pair had suffered hemolytic episodes; two of them fatal. A fourth sibling is currently unaffected in Frankfurt. A similar grouping occurred in the offspring of a Denver pair. Two of the three siblings were affected and died.

A third familial grouping was noted at the Frankfurt Zoo. There the death of a seventeen-year-old female who had suffered previous hemolytic episodes was associated with hemolysis. Her daughter had two similar episodes, dying during the second; and her granddaughter survived one episode of hemolysis, but later died of nonhemolytic anemia.

Cleveland, Dvur Kralove (Czech.) and Dublin each lost two animals, but at spaced time intervals. A single female died at Columbus. As in many of the cases, the Columbus animal had hemosiderinlike deposits in gastrointestinal and parenchymatous tissues. Bone marrow in that case illustrated active hematopoiesis. Confirmatory laboratory data was unavailable on the Dublin pair. The Dvur Kralove animals had suffered two and three episodes each of hemoglobinuria. The death of the latter animal occurred during anesthesia one month after an episode, and at that time there was "evidence of hemolytic anemia only by histopathologic exam." Titers for serovars of L. interrogans ictohemorrhagica were greatly elevated--up to 1:6400 in one individual and 1:12,800 in the other.

A common toxin or drug exposure could not be found to connect the cases. In the Toronto occurrence there had been an exposure to diaphacinon, a warfarinlike pesticide. Laboratory data and necropsy results found evidence of a hemolytic event, not a bleeding disorder as would be expected with such a compound. A silver stain positive spirochaete was found in the kidney of the male, but could not be definitively identified as leptospirosis. Evidence of leptospiral exposure could not be found in titers collected from adjacent animals and vermin trapped in the area. The female that died at the National Zoo had been on isoniazid for the prophylactic treatment of tuberculosis. An occasional side effect of the drug in man can be hemolytic anemia, but such a correlation in this case is uncertain. In that animal, titers for serovars of Leptospirosis interrogans ranged up to 1:1000 for L. autumnalis.

Discussion

Personal communication and the results of this and previous survey indicate the relative frequency of occurrence of deaths of black rhinoceroses associated with hemolytic anemia. The latter is a symptom and not an etiology. A common agent, if one exists, is not readily apparent at the present time. The massive levels of iron deposition noted in the tissues of the St. Louis and in other cases may also indicate a chronic stage of the problem that clinically appears so peracutely.

Leptospirosis has been suggested in previous reports, and it must be strongly considered in any future cases. High leptospiral titers were evident in Memphis and Dvur Kralove. Unpaired titers are not the most reliable method of diagnosis in domestic animals, but due to the peracute nature of these cases, they are often all that is available. Hopefully, fluorescent antibody techniques for the diagnosis of several serovars of leptospirosis will become available in the future.

Fatal babesiosis and trypanosomiasis have been described in wild black rhinoceroses (7). Although less likely in captive-bred animals, hemic parasites must be ruled out in any episodes of hemolysis. Hemolytic clostridial disease and copper toxicity also deserve attention due to the similarity with hemolytic syndromes in domestic species. Hemolytic anemia responsive to vitamin E therapy has been reported in the owl monkey (Aotus trivirgatus) (10), marmosets (Saguinus labiatus) (2), and other primates (1), and perhaps should be considered in any future rhinoceros cases.

At the present time the possibility of autoimmune hemolytic anemia (AIHA) cannot be fully evaluated. In the St. Louis case, antinuclear antibody (ANA) and rheumatoid factor (RF) tests were employed due to their lesser species specificity. Both were negative, but it would be unsound to rule out the disease on that basis. Hopefully, obtaining normal black rhinoceros sera in the future will allow for the synthesis of the appropriate Coomb's reagent.

The possibility also exists that hemolysis in the black rhinoceros is not due to a single etiology but represents a final common pathway induced by a variety of factors. One model is the glucose-6-phosphatase enzyme deficiency present in certain human populations, although a similar deficiency has not been reported in domestic or exotic animals (9). In that deficiency a normal life span and style may be achieved unless an oxidant or drug exposure induces hemolytic anemia. The prospect for a similar deficiency in the black rhinoceros remains entirely speculative at this time. If blood becomes

available from normal black rhinoceroses, and from any future cases of hemolytic anemia, consideration should be given to its assay for RBC enzyme functions.

Caution must be exercised in the evaluation of present and future cases of hemolytic anemia in the black rhinoceros to establish the relationship of the anemia to death and not overlook other, possibly significant, factors. However, the data currently available indicate a frequent disorder in this species in captivity. Hopefully, further evaluation of normal individuals in addition to those undergoing hemolysis will establish a data base from which to proceed.

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