

HEMOLYTIC ANEMIA IN THE BLACK RHINOCEROS--AN UPDATE

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Since a 1981 survey of hemolytic anemia in the black rhinoceros (Diceros bicornis) (3,4), an additional five animals in the United States, Europe and Japan have been located that have undergone episodes of hemolysis. The most recent cases in the United States have been tested with red blood cell enzyme and autoimmune reagents that became available in 1984. Documentation currently exists for 27 hemolytic episodes in 21 animals. Seventeen (80%) died during their initial or recurrent hemolytic crises. As before, three familial groups have been noted, but they do not appear to be related to each other, or account for the majority of the cases. Of the fatal cases, sex ratios (10.7) and wild vs. captive origin (7 wild caught, 10 captive bred) do not appear to be significant factors.

Though leptospirosis is strongly suggested in several cases (in addition to the one reported)(2), a definitive diagnosis common to all cases has not been found. In addition to leptospirosis, copper toxicity, red blood cell enzyme deficiencies and autoimmune hemolytic anemia (AIHA) have all been considered in the differential diagnosis. The latter two etiologies could not be fully evaluated until recent laboratory investigations addressed these possible etiologies.

Immunoematologic procedures centered on the development of a Coomb's reagent specific to the black rhinoceros. An anti-rhinoceros whole sera and an anti-rhinoceros IgG reagent were synthesized and applied to two cases of hemolytic anemia (one fatal) that have occurred in the United States since 1984 as well as normal rhinos. These reactions were negative from normal rhinoceroses and from the animal that survived its hemolytic event. However, there was indication in the fatal case that there was increased membrane coating of the red blood cells with the third component of complement. It was felt that this probably did not represent a primary autoimmune reaction. Osmotic fragility, erythrocyte membrane protein composition, hemoglobin electrophoresis, and hemoglobin stability tests did not distinguish any differences between normal and affected rhinoceroses. (1)

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A second project evaluated 30 erythrocyte enzymes and metabolites in 10 normal rhinos (though one of which was the dam of three affected offspring), and two rhinos during their hemolytic crises. Comparison of the values between these two groups did not identify an enzyme abnormality as a cause for the syndrome, though basic data for possible future comparisons was collected. Additionally, it was noted that significant differences occurred between erythrocyte reduced glutathione and 2,3-diphosphoglycerate concentrations in black rhinoceroses of East African (n=4) and South African (n=3) origin. (5)

All future cases of hemolysis in this species should be evaluated as previously described (leptospirosis, AIHA, etc.), and consideration is being given to additional etiologies to evaluate further hemoglobin structure and stability studies, determination of vitamin E and selenium levels, and more sophisticated measures of red blood cell stability. Such a data base will hopefully serve to identify a common defect that may account for the large number of peracute hemolytic events in this species. Samples from normal and affected black rhinoceroses will continue to be solicited in order to pursue these additional testing procedures.

Bibliography

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