Journal of Zoo and Wildlife Medicine 23(2): 230–234, 1992

HEMOSIDEROSIS IN THE BLACK RHINOCEROS (*DICEROS BICORNIS*): A COMPARISON OF FREE-RANGING AND RECENTLY CAPTURED WITH TRANSLOCATED AND CAPTIVE ANIMALS

Nancy Kock, D.V.M., M.S., Chris Foggin, B.V.Sc., Ph.D., Michael D. Kock, B.Vet.Med., M.P.V.M., and Richard Kock, M.A., Vet.M.B.

Abstract: Postmortem material from three zoo-exhibited, 18 translocated, one free-ranging, and six recently caught black rhinoceroses (*Diceros bicornis*) was examined for the presence of generalized hemosiderin deposition as a possible indicator of prior hemolysis. All zoo-exhibited and 72% of the translocated rhinoceroses had generalized hemosiderosis. None of the recently caught or free-ranging rhinoceroses studied had this lesion. Several processes can result in hemosiderosis, and its presence in the captive and translocated animals may not necessarily indicate a hemolytic process; however, the absence of hemosiderosis in free-ranging and recently caught animals suggests that a hemolytic syndrome previously reported in captive black rhinoceroses does not occur under natural conditions.

Key words: Black rhinoceros, *Diceros bicornis,* hemosiderosis, hemolysis.

INTRODUCTION

Since 1986, approximately 300 black rhinoceroses (*Diceros bicornis*) have been translocated by the Department of National Parks and Wildlife Management (DNPWLM) from the Zambian border of Zimbabwe to locations within Zimbabwe to decrease poaching activities.[3,12,13] In the 1970's, there were more than 65,000 wild rhinoceroses in Africa, but fewer than 4,000 can be found in the wild at the present;[7] about 1,000 roam free in Zimbabwe.[25] An idiopathic hemolytic anemia syndrome has been reported in captive black rhinoceroses,[5,8,16–20] with hemosiderosis a predicted feature.[22] The purpose of this study was to examine multiple tissues from Zimbabwean black rhinoceroses for hemosiderin deposition as a possible indicator of this syndrome in wild and relocated rhinoceroses.

From the Faculty of Veterinary Science, University of Zimbabwe, P.O. Box MP167, Mount Pleasant, Harare, Zimbabwe (N. Kock); the Veterinary Research Laboratory, P.O. Box 8101, Causeway, Harare, Zimbabwe (Foggin); the Department of National Parks and Wildlife Management, P.O. Box 8365, Causeway, Harare, Zimbabwe (M. Kock); the Zoological Society of London, Whipsnade Wild Animal Park, Dunstable, Bedfordshire LU6 2LF, England, U.K. (R. Kock); and International Wildlife Veterinary Services, 1850 N. Main Street, Salinas, California 93906, USA (M., N., and R. Kock).

METHODS AND MATERIALS

Formalin-fixed and paraffin-embedded tissues from three captive black rhinoceroses from Whipsnade Zoological Gardens, U.K., and from one free-ranging, six recently captured, and 18 translocated rhinoceroses from Zimbabwe were processed routinely for histologic examination. Tissue sampling was not always uniform or complete. Spleen, liver, kidney, heart, lymph node, and lung were examined in most animals, and gastrointestinal tract, adrenal and thyroid glands, and brain in some. All tissues were stained with hematoxylin and eosin (H&E) and Perl's iron stains[1] and examined for the presence of hemosiderin. Two animals that died shortly after capture were quite old; one was >25 yr old and the other was considered aged, based on dentition (R. du Toit, pers. comm.). The amount of splenic hemosiderin in these two animals and in the one found dead in the wild was considered as normal background for the species. Hemosiderin deposition in the other animals was subjectively judged as normal or excessive based on these reference

Table 1. Incidence of hemosiderosis in free-ranging, recently caught, translocated, and captive black rhinoceroses.

ID no.	Category[a]	Age	Sex[b]	Time[c]	Hemosiderosis	
					Spleen	Other tissues[d]
1	F	Adult	U	0	–	–
2	R	Aged	F	1 day	–	–
3	R	6 yr	F	1 wk	–	–
4	R	25 yr	M	1 wk	–	–
5	R	Calf	F	1 wk	–	–
6	R	Calf	F	1 wk	–	–
7	R	16 mo	F	2 wk	NA[e]	–
8	T	2 yr	M	4 wk	+	–
9	T	Adult	F	5 wk	+	–
10	T	16 mo	F	6 wk	–	–
11	T	U	U	6 wk	NA	L, I
12	T	Adult	F	6 wk	+	L
13	T	Fetus	U	6 wk[f]	–	–
14	T	3 yr	M	6 wk	–	–
15	T	Fetus	U	6 wk[f]	+	L
16	T	Adult	F	6 wk	+	L
17	T	Adult	M	4 mo	NA	L, Lu
18	T	1 wk	M	4 mo[g]	+	L, Lu, I
19	T	Adult	F	5 mo	–	–
20	T	Adult	M	8 mo	+	–
21[h]	T	Adult	F	1 yr	+	L, Lu
22	T	6 yr	F	1 yr	–	–
23	T	Adult	M	2 yr	+	L, I
24	T	Juv	M	2 yr	+	L, Ly
25	T	3 yr	F	2 yr	+	L, Lu, Ly, K, A
26	C	3 yr	F	3 yr[i]	+	L, Ly, I
27	C	6 yr	M	6 yr[i]	+	L, I
28	C	8 yr	M	8 yr[i]	+	L, Lu, K, I, A

[a] F = free ranging; R = recently captured; T = translocated; C = captive.
[b] U = unknown; F = female; M = male.
[c] Time between capture and death.
[d] A = adrenal gland; I = intestine; K = kidney; L = liver; Lu = lung; Ly = lymph node.
[e] Spleen not available for examination.
[f] Time between capture of dam and abortion.
[g] Time between capture of dam and death of neonate.
[h] Dam of animal 18.
[i] Time in captivity.

animals. The presence of hemosiderin in other tissues was scored either positive or negative.

Animals considered "recently caught" died within 2 wk after capture; those considered "translocated" died from 2 wk to about 2 yr after capture. Animals that died within 2 wk of capture were still in confinement at the capture site, awaiting translocation. Those that died after 2 wk were either confined in pens at the translocation sites or had been released into the new habitats.

RESULTS

The data are summarized in Table 1. The one free-ranging and all of the recently caught animals had scanty splenic hemosiderin and none in other tissues. The three zoo-exhibited and 14 of 18 (72%) translocated animals had relatively more splenic hemosiderin (Fig. 1) and often had depo-

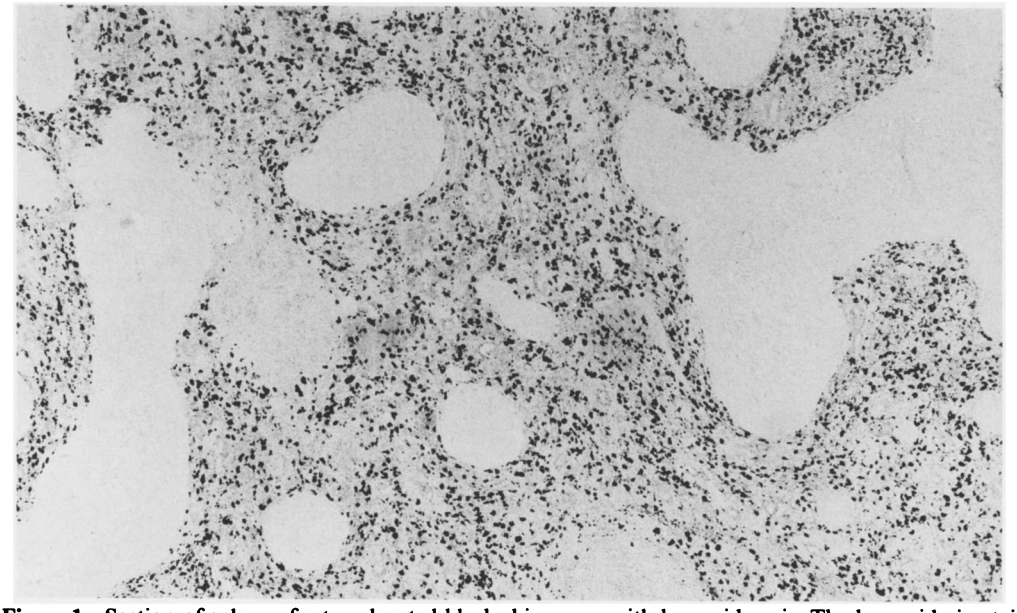

Figure 1. Section of spleen of a translocated black rhinoceros with hemosiderosis. The hemosiderin stains darkly and is scattered throughout in the spleen. Perl's iron stain.

sition in other tissues. After the spleen, the liver (Fig. 2) was the next most commonly affected site, and deposition was diffusely distributed. Variable hemosiderin deposition, often apparently extracellular or within tissue macrophages, in lung, intestine, kidney, lymph nodes, and adrenal glands occurred in animals translocated for longer periods.

In only one of the translocated rhinoceroses was the cause of death possibly associated with overt hemolysis. This animal

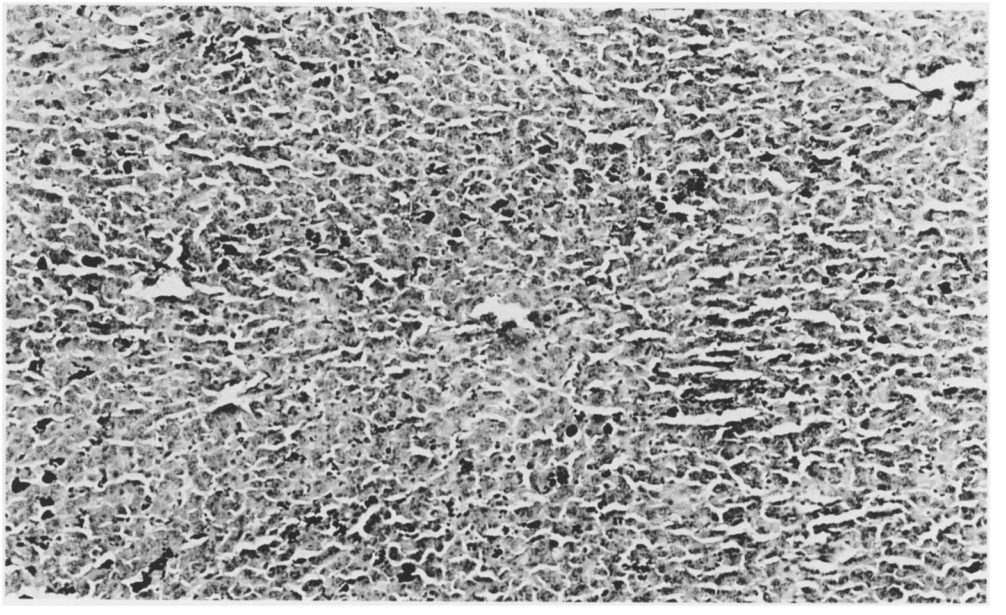

Figure 2. Section of liver of a black rhinoceros with generalized hemosiderosis. Perl's iron stain.

had been translocated about 2 yr before and passed dark reddish-brown urine prior to death. Causes of death in other animals included trauma, aspiration pneumonia, bacterial infections,[15] emaciation, dehydration, and cardiac tamponade due to coronary artery aneurysm rupture.[14] Poor body condition and atrophy of lymphoid tissue were frequently seen in animals with generalized hemosiderosis.

DISCUSSION

The spleen normally degrades senescent red blood cells, storing ferritin in splenic macrophages.[23,24] If ferritin deposition becomes excessive or prolonged, the ferritin forms aggregates of insoluble hemosiderin complexes.[10,11,22–24] Small amounts of hemosiderin in the spleen, therefore, is expected and tends to increase with age. When hemosiderin storage becomes excessive in the spleen or when found in other organs, the condition is called hemosiderosis. Hemosiderin can be seen with routine H&E stains but is emphasized with Perl's iron stain.[10] Hemosiderosis is usually the result of hemolysis, although increased intestinal iron absorption and decreased reuse of iron may also produce this lesion.[23] Increased iron absorption is uncommon in most animal species,[11] and conditions under which iron reutilization is decreased (copper deficiency, cachexia, aplastic anemia, uremia) are usually associated with other signs and lesions in addition to the hemosiderosis.[23] None of the animals with hemosiderosis had tissue pallor or renal disease, and although poor body condition frequently accompanied hemosiderosis in the black rhinoceroses in this study, evidence of true cachexia (emaciation, serous atrophy of fat) was not observed.

None of the animals that died within 2 wk of capture had splenic hemosiderin that qualitatively exceeded the amount in the reference animals, and hemosiderosis did not occur in extrasplenic sites, indicating that underlying mechanisms for generalized hemosiderosis apparently do not occur in free-ranging animals. If hemolysis underlies the hemosiderosis, as expected in the idiopathic hemolytic syndrome of captive black rhinoceroses, a hemolytic syndrome probably does not occur under natural conditions but may develop after capture and translocation. All three animals from Whipsnade Zoological Gardens experienced episodic hemolysis and eventually died of acute anemia following a terminal bout of extensive hemolysis. Possible causes of the hemolytic syndrome in captive black rhinoceroses include leptospirosis,[8] red blood cell parasitism,[2,4,6] red blood cell fragility,[21] red blood cell enzyme abnormalities,[20] immunologic mechanisms,[5] and poor nutrition.[16,19]

Hemosiderosis in the aborted fetus and neonate (animals 15 and 18) suggests the occurrence of an underlying pathologic process in utero or placental crossing of iron pigments from an affected dam. The lymphoid atrophy frequently seen in translocated animals is a possible indicator of chronic stress, which may have predisposed affected animals to infectious diseases[9] and hemolysis. Latent infections with leptospirosis or hematozoa may be triggered with stress in translocated animals.

Of the four translocated animals without hemosiderosis, two died on the same ranch, one 6 wk and the other 5 mo after capture. Some translocation sites may be more suitable than others for this species. Further study of free-ranging and translocation habitats, nutrition, stress reduction, latent infectious diseases, and pathologic conditions of the black rhinoceros is needed.

Acknowledgments: We thank the University of Zimbabwe Research Board and International Wildlife Veterinary Services for funding this project, the director of the DNPWLM for permission to conduct the study, and the technical staff in the Paraclinical Department at the Veterinary Faculty for processing the samples. Raoul du Toit helped with sample and data collection and offered valuable recommendations for the manuscript.

LITERATURE CITED

1. Bancroft, J. D., and A. Stevens (eds.). 1982. Theory and Practice of Histological Techniques, 2nd ed. Churchill Livingstone, Edinburgh, U.K. Pp. 278–296.

2. Bigalke, R. D., M. E. Keep, P. J. Keep, and J. H. Schoeman. 1970. A large *Babesia* sp. and a *Theileria*-like piroplasm of the square-lipped rhino. J. S. Afr. Vet. Med. Assoc. 41: 292–294.

3. Booth, V. F., and A. M. Coetzee. 1988. The capture and relocation of black and white rhinoceros in Zimbabwe. *In:* Nielsen, L., and R.D. Brown (eds.), Translocation of Wild Animals. Wisconsin Humane Society, Milwaukee, Wisconsin, and Caesar Kleberg Wildlife Research Institute, Kingsville, Texas. P. 333.

4. Brocklesby, D. W. 1977. A *Babesia* species in the black rhinoceros. Vet. Rec. 80: 484.

5. Chaplin, H., A. C. Malecek, E. Miller, C. E. Bell, L. S. Gray, and J. L. Hunter. 1986. Acute intravascular hemolytic anemia in the black rhinoceros: hematologic and immunohematologic observations. Am. J. Vet. Res. 47: 1313–1320.

6. Clausen, B. 1981. Survey for trypanosomes in the black rhino. J. Wildl. Dis. 17: 581–586.

7. Cumming, D. 1987. Small population management of black rhinos. Pachyderm 9: 12–19.

8. Douglass, E. M., K. E. Plue, and C. E. Kord. 1980. Hemolytic anemia suggestive of leptospirosis in the black rhinoceros. J. Am. Vet. Med. Assoc. 177: 921–923.

9. Griffin, F. T. 1989. Stress and immunity. *In:* Veterinary Immunology and Immunopathology. Elsevier, Amsterdam, The Netherlands. Pp. 263–312.

10. Jones, T. C., and R. D. Hunt. 1983. Veterinary Pathology, 5th ed. Lea and Febiger, Philadelphia, Pennsylvania. Pp. 84–86.

11. Kaneko, J. J. 1980. Iron metabolism. *In:* Kaneko, J. J. (ed.). Clinical Biochemistry of Domestic Animals, 3rd ed. Academic Press, London, U.K. Pp. 653–667.

12. Kock, M. D., M. La Grange, and R. du Toit. 1990. Chemical immobilization of free-ranging black rhinoceros (*Diceros bicornis*) using combinations of etorphine (M99), fentanyl, and xylazine. J. Zoo Wildl. Med. 21: 155–165.

13. Kock, M. D., R. du Toit, N. Kock, D. Morton, C. Foggin, and B. Paul. 1990. Effects of capture and translocation on biological parameters in free-ranging black rhinoceroses (*Diceros bicornis*) in Zimbabwe. J. Zoo Wildl. Med. 21: 414–424.

14. Kock, N., C. Foggin, M. Kock, P. Trembath, and D. Jessup. 1991. Coronary artery aneurysms in two black rhinoceroses (*Diceros bicornis*) in Zimbabwe. J. Zoo Wildl. Med. 22: 355–358.

15. Kock, N., M. Kock, A. Pawandiwa, and T. Matambo. 1989. Postmortem findings in translocated black rhinoceroses (*Diceros bicornis*) in Zimbabwe. Proc. 31st Int. Congr. Dis. Zoo Wild Anim., Dortmund, Germany. Pp. 275–279.

16. Miller, R. E. 1987. Hemolytic anemia in the black rhino. Pachyderm 9: 26–28.

17. Miller, R. E., and W. J. Boever. 1982. Fatal hemolytic anemia in the black rhinoceros: case report and a survey. J. Am. Vet. Med. Assoc. 181: 1228–1231.

18. Miller, R. E., and W. J. Boever. 1983. Hemolytic anemia in the black rhinoceros (*Diceros bicornis*). Proc. Ann. Meet. Am. Assoc. Zoo Vet. Pp. 51–53.

19. Miller, R. E., H. Chaplin, D. E. Paglia, and W. J. Boever. 1986. Hemolytic anemia in the black rhinoceros—an update. Proc. Annu. Meet. Am. Assoc. Zoo Vet. Pp. 7–8.

20. Paglia, D. E., W. N. Valentine, R. E. Miller, M. Nakatani, and R. A. Brockway. 1986. Acute intravascular hemolysis in the black rhinoceros: erythrocyte enzymes and metabolic intermediates. Am. J. Vet Res. 47: 1321–1325.

21. Paul, B., R. du Toit, S. Lloyd, and A. Mandisodza. 1988. Haematological studies on wild black rhinoceros (*Diceros bicornis*)—evidence of an unstable haemoglobin. J. Zool. (Lond.) 214: 399–405.

22. Robbins, S. L., R. S. Cotran, and J. Kumar (eds.). 1989. Cellular injury and adaptation. *In:* The Pathologic Basis of Disease, 4th ed. W. B. Saunders, London, U.K. Pp. 25–26.

23. Robbins, S.L., R. S. Cotran, and J. Kumar (eds.). 1989. Diseases of red cells and bleeding disorders. *In:* The Pathologic Basis of Disease, 4th ed. W. B. Saunders, London, U.K. Pp. 686–687.

24. Valli, V. E. O. 1985. The hematopoietic system. *In:* Jubb, K. V. F., P. C. Kennedy, and N. Palmer (eds.). Pathology of Domestic Animals, 3rd ed., vol. 3. Academic Press, London, U.K. Pp. 131–167.

25. Zimbabwe Department of National Parks and Wildlife Management. 1992. National Conservation Strategy for the Black Rhinoceros in Zimbabwe. Zimbabwe DNPWLM, Causeway, Harare, Zimbabwe.

Received for publication 24 July 1990.