SKIN LESIONS IN FREE-RANGING BLACK RHINOCEROSES (*DICEROS BICORNIS*) IN ZIMBABWE

Nancy Kock, D.V.M., M.S., and Michael D. Kock, B. Vet. Med., M.P.V.M.

Abstract: Thirty-three biopsies from skin lesions were taken from immobilized black rhinoceroses (*Diceros bicornis*) during capture and translocation in Zimbabwe. The lesions were ulcerated, exudative and crusty and were consistently present in the skin of the ventral neck. Histologically, the ulcerative dermatitis was accompanied by epidermal adult filarial nematodes, intraepidermal necrosis, and dermal granulomas and lymphohistiocytic nodules in some cases.

Key words: Black rhinoceros, *Diceros bicornis,* filarial dermatitis.

INTRODUCTION

Skin lesions have been reported in the black rhinoceros (*Diceros bicornis*) in South Africa[3,8] and Kenya[7,10] and in captive black, white (*Ceratotherium simum*), and Indian rhinoceroses (*Rhinoceros unicornis*).[4] In white rhinoceroses, the skin lesions were directly related to trauma sustained during transport.[4] An exfoliative dermatitis described in a captive Indian rhinoceros in 1926 was associated with *Pityrosporum rhinoserosum,* and in one report, erosive skin lesions in captive black rhinoceroses were suspected to be viral.[4] Early reports on skin lesions in black rhinoceroses from South Africa[3] were attributed to general ability or glandular activity related to breeding; however, in later reports the lesions were associated with filarial nematodes.[3] Parasites from similar lesions in Kenyan black rhinoceroses were identified as *Stephanofilaria* sp., later designated *S. dinniki.*[7]

MATERIALS AND METHODS

To lessen poaching threats from neighboring Zambia, 56 black rhinoceroses were captured and translocated in 1988 by Zimbabwe's Department of National Parks and Wildlife Management (DNPWM) from the Zambezi Valley to internal areas within Zimbabwe.[5] This exercise allowed close inspection of 50 adults (>5 yr old) and juveniles (6 mo–5 yr old), and six calves (<6 mo old). Twenty-seven males and 29 females were examined. Two adult females moved in 1987 from the Zambezi Valley to the lowveld near Bulawayo and one adult male captured in the Zambezi Valley in 1989 were also examined. Lesions were biopsied as logistics permitted. Thirty-three wedge-shaped skin biopsies, measuring about 16 mm on a side and 5–6 mm deep, were taken with #10 scalpel blades from 17 females and 16 males while the rhinoceroses were immobilized with mixtures of narcotics and tranquilizers.[5] Specimens were immediately fixed in 10% phosphate-buffered formalin and later trimmed, embedded in paraffin, cut at 5 μm, and stained with hematoxylin and eosin (H&E). Multiple sections from each lesion were examined by light microscopy. Selected specimens were stained with Ziehl Nielsen, periodic acid–Schiff (PAS), methenamine silver, and Twort bacterial stains.[1]

RESULTS

Fifty-one rhinoceroses (all but six calves and two young adults) had skin lesions that were consistently seen in the skin of the ventral neck. Thirty-five animals had lesions limited to this region. Thirteen had lesions at one other site: seven had involvement of the ventral abdominal region, two of the forelegs, and one each of the pinna, lateral

From the Faculty of Veterinary Science, University of Zimbabwe, Box MP167, Mount Pleasant, Harare, Zimbabwe (N. Kock); the Department of National Parks and Wildlife Management, P.O. Box 8365, Causeway, Harare, Zimbabwe (M. Kock); and International Wildlife Veterinary Services, Box 1413, Orangevale, California, USA (N. and M. Kock).
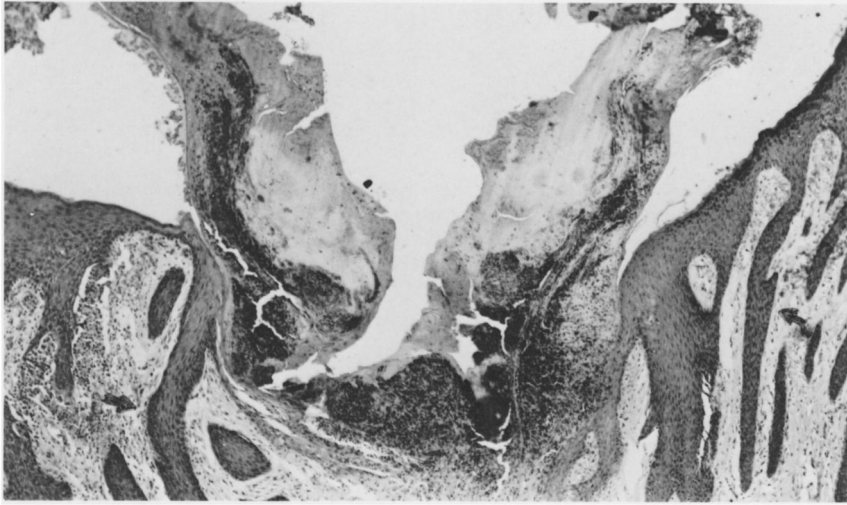
Figure 1. Cutaneous ulcer from a black rhinoceros. Ulcer is covered by a thick layer of serocellular material with rete-ridge formation at the margins (arrows). H&E.

thorax, axilla, and lateral stifle. Of two rhinoceroses with lesions at two sites other than the ventral neck, both had ventral abdominal and lateral thoracic involvement.

The lesions were circumscribed to rectangular and measured from 1 to 10 cm in diameter or from 2 to 10 cm wide and 4 to 18 cm long, respectively. In rhinoceroses with multiple lesion sites, the ventral neck lesions were always the largest. The animals were categorized clinically as having either active or healing lesions. Lesions considered active were ulcerated, exudative, crusty, and erythematous and tended to be large (usually >5 cm wide). Lesions considered healing were smaller, often centrally ulcerated, and surrounded by firm dry raised depigmented skin. Thirty-three of the 51 affected rhinoceroses (65%) had active lesions, and 18 (35%) had healing lesions. Both lowveld rhinoceroses had typical neck lesions, and one animal (also with neck lesions) developed vesicles >1 mo after capture.

Of the 33 biopsies taken, six were from healing lesions (three from the ventral neck and three from the ventral abdomen) and 26 were from active lesions (19 from the ventral neck, two from the forelegs, and one each from the lateral thorax, ventral abdomen, pinna, axilla, and lateral stifle). The remaining biopsy was from the rhinoceros with vesicles. On sectioning the biopsy from the lateral stifle, several 0.5 mm-diameter pale green caseous nodules were discovered.

Histologically, ulcers or erosions were usually covered by thick serocellular layers containing large numbers of degenerating neutrophils and bacteria and extraneous debris (Fig. 1). Acanthosis with rete-ridge formation was usually present at the margins of the ulcers, with fibroplasia and mild superficial dermal necrosis underneath. Intraepidermal foci of necrosis and inflammation (Fig. 2) were evident in 15 of the rhinoceroses with active lesions and two with healing lesions. Some of these intraepidermal foci appeared to be micropustules. Parasites, characteristic of adult filarial nematodes, with body cavities and internal organs surrounded by cuticles, were seen in the epidermis of six rhinoceroses with active lesions that were accompanied by minimal to moderate inflammation (Fig. 3).

Epithelioid granulomas with giant cells and centrally located amorphous eosinophilic material occurred in the dermis in five rhinoceroses with active and one with healing lesions (Fig. 4). Similar granulo-
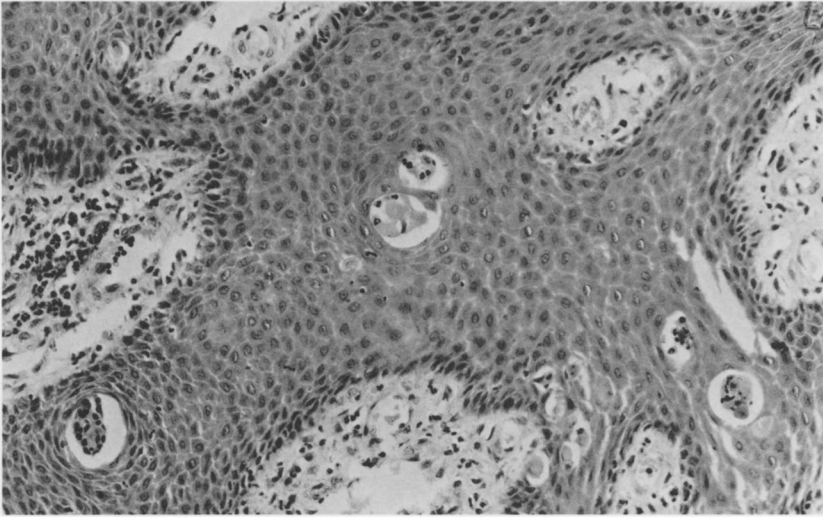
Figure 2. Another field from Figure 1 showing intraepidermal foci of necrosis and inflammation. H&E.

matous inflammation was associated with degenerating filarial nematodes in the biopsy from the lateral stifle (Fig. 5). Multinucleated giant cells, often dermal and perivascular, predominated in the biopsy from the rhinoceros with vesicles. Dermal nodules, composed of lymphocytes and histiocytes, occurred in 10 rhinoceroses with active lesions and in two with healing lesions (Fig. 6). All lesions had various degrees of dermatitis with diffuse infiltrations of neutrophils and eosinophils or with perivascular lymphocytes, plasma cells, and histiocytes. Nine of the biopsies with granulomas and nodular inflammatory aggregates stained with PAS, methenamine silver, Ziehl Nielsen, and Twort stains were negative for any etiological agents.
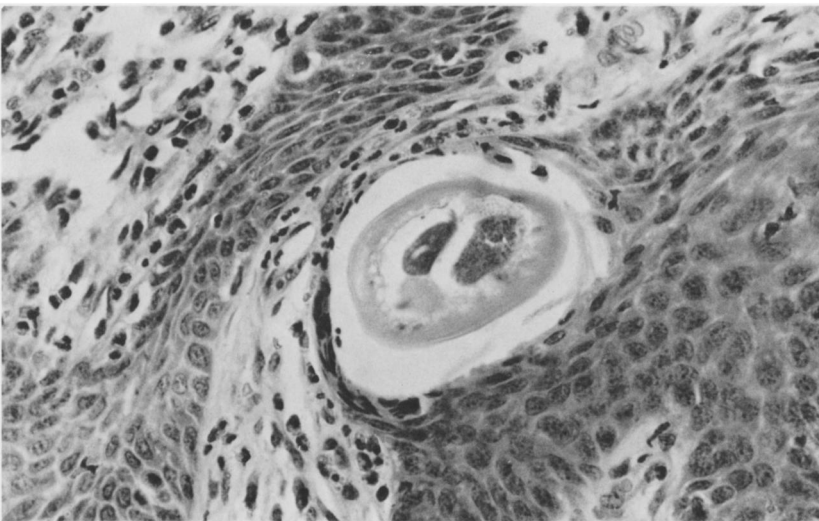
Figure 3. Intraepidermal adult male filarial nematode associated with superficial inflammation from a black rhinoceros. The darkly staining structures within the cuticle are the testis (right) and intestine. H&E.
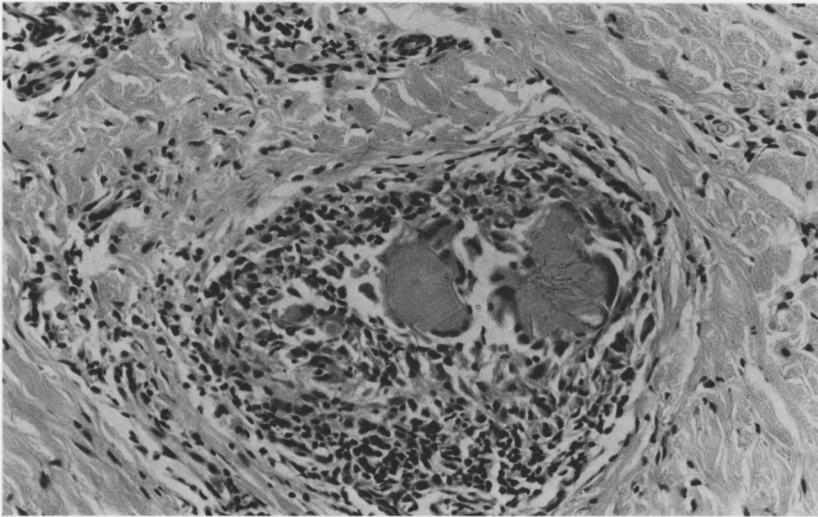
Figure 4. Dermal granuloma from a black rhinoceros. Note amorphous material surrounded by mixed-type inflammatory cells, including multinucleated giant cells. H&E.

DISCUSSION

Acanthosis, rete-ridge formation, and alterations in pigmentation commonly accompany chronic inflammatory conditions of the skin but are not particularly useful diagnostically, merely reflecting chronic dermatitis.[11] Likewise, granulomatous inflammation, when agents are not present, reflects body defense to persistent foreign material and is not specifically diagnostic.[6,11] Lymphoid follicular proliferation likely occurs in situations where persistent antigenic stimulation is encountered, again a nonspecific diagnostic finding. All of these changes in the rhinoceros skin lesions attest to the chronicity of the inflammation but
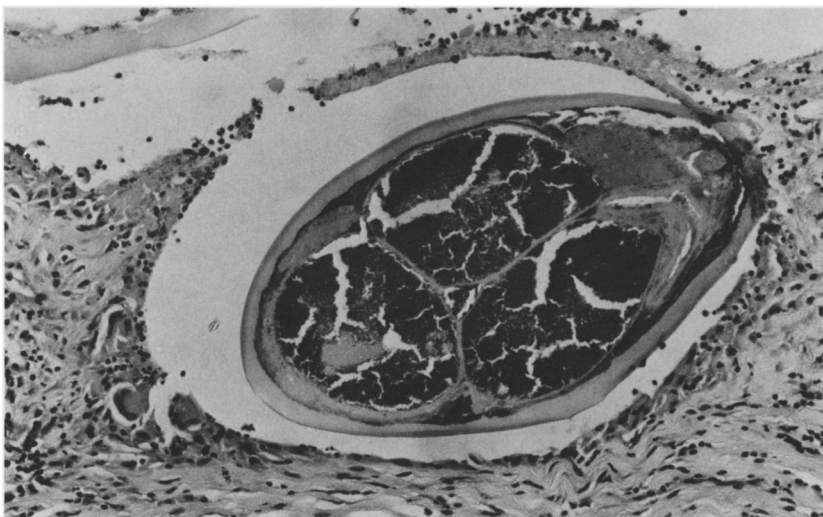
Figure 5. Degenerating female filarial nematode with giant cells at the margins in the dermis of a black rhinoceros. H&E.
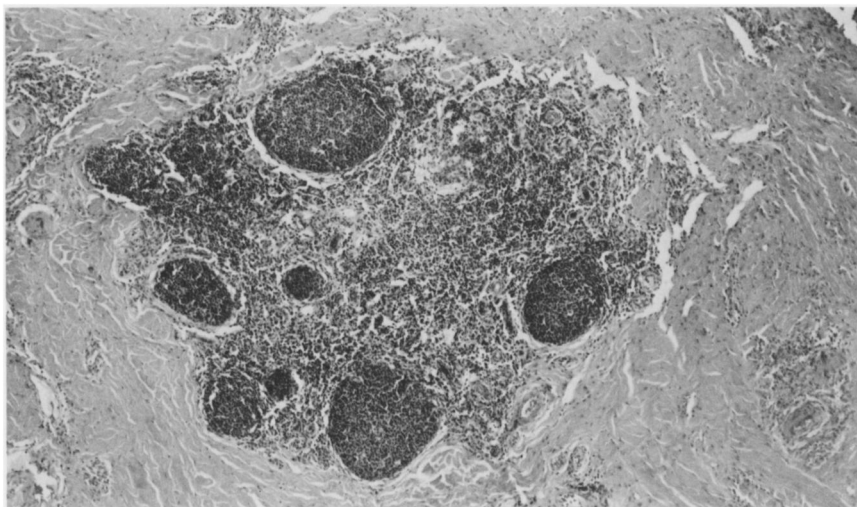
Figure 6. Dermal lymphohistiocytic nodules from a black rhinoceros. H&E.

do not suggest specific causes. Intraepidermal necrosis can be the result of viral and immune-mediated diseases as well as an aspect of any dermatitis in which intracellular or intercellular edema is featured.[11] In the rhinoceros, this lesion is likely a result of edema, given the lack of additional characteristic changes.

Previous reports on the macroscopic changes attributed to active cutaneous filariasis in the black rhinoceros describe foci of exfoliation, crusting, pustules, ulcers, and hemorrhage surrounded by depigmented scarred areas, often with attached larval, nymphal, and adult ticks.[3,7,8,10] The gross lesions in Zimbabwean rhinoceroses described in this study are morphologically comparable but more localized than those reported in South African black rhinoceroses. In addition, the distribution of the lesions in South African rhinoceroses changed with age, with involvement of the withers and lateral neck in young animals, and progressed ventrally and laterally with maturity.[3] Regardless of age, affected Zimbabwean rhinoceroses consistently had lesions in the ventral neck region; only calves and two juveniles had no lesions at all. Seasonal variation of lesion intensity was reported in South African black rhinoceroses, with dormant winter lesions becoming florid during the summer.[8] Pruritus and excoriation probably contribute to the exacerbations. Some workers have not observed any significant seasonal variation in skin lesions.[3] All but three of the Zimbabwean rhinoceroses were examined in the winter, and although most had clearly active lesions, many predominantly healing lesions also had ulcerated foci.

Several parasites of the genus *Stephanofilaria* are responsible for dermatitis in cattle, goats, and Asian buffalo,[9] and preferred feeding sites of parasite insect vectors correspond to typical distribution patterns of lesions. Transmitting insects harbor the parasite forms that develop into the infective third-stage larvae, which are deposited into the skin when the insect feeds. The microfilariae develop in the definitive hosts and are ingested by the vectors during feeding, thus allowing the cycle to continue.[2] Poor body condition and heavy rainfall are thought to predispose to the development or recrudescence of lesions; dormancy of lesions often coincides with dry cool seasons.[9] The primary lesions in the rhinoceroses in this study were likely caused by vector-transmitted filarial parasites, as seen in Kenya and South Africa,[7,10] given the

presence of adult filarial nematodes in some of the biopsies along with the associated eosinophil infiltration. It is unusual, however, that microfilariae and larval forms were never seen in the Zimbabwean animals, although this situation may reflect seasonal variation. The differences in distribution in the lesions in Zimbabwean rhinoceroses compared with those in other African countries may suggest a different vector. The active appearance of many of the lesions is likely the result of pruritus and excoriation.

LITERATURE CITED

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

2. Georgi, J. R. 1985. Parasitology for Veterinarians, 4th ed. W. B. Saunders Co., Philadelphia, Pennsylvania. Pp. 130–133.

3. Hitchins, P. M., and M. E. Keep. 1970. Observations on skin lesions of the black rhinoceros (*Diceros bicornis* Linn.) in the Hluhluwe Game Reserve, Zululand. Lammergeyer 12: 56–65.

4. Jones, D. M., and L. R. Thomsett. 1972. A short review of the diseases of rhinoceros skin with case reports on an exudative dermatitis in the white rhinoceros (*Ceratotherium simum*). Proc. 14th Int. Symp. Dis. Zoo Wild Anim. Pp. 227–231.

5. 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.

6. Robbins, S. L., R. S. Cotran, and V. Kumar. 1984. Pathologic Basis of Disease, 3rd ed. W. B. Saunders Co., Philadelphia, Pennsylvania. Pp. 64–65.

7. Round, M. C. 1964. A new species of *Stephanofilaria* in skin lesions from the black rhinoceros (*Diceros bicornis* L.) in Kenya. J. Helminthol. 38: 87–96.

8. Schulz, K. C. A., and E. B. Kluge. 1960. Dermatitis in the black rhinoceros (*Diceros bicornis*) due to skin filariasis. J. S. Afr. Vet. Med. Assoc. 31: 265–269.

9. Soulsby, E. J. L. 1982. Helminths, Arthropods and Protozoa of Domesticated Animals, 7th ed. Bailliere Tindall, London, U.K. Pp. 320–321.

10. Tremlett, J. G. 1964. Observations on the pathology of lesions associated with *Stephanofilaria dinniki* from the black rhinoceros (*Diceros bicornis*). J. Helminthol. 38: 171–174.

11. Yager, J. A., and D. W. Scott. 1985. The skin and appendages. *In:* Jubb, K. V. F., P. C. Kennedy, and N. Palmer (eds.). Pathology of Domestic Animals, 3rd ed., vol. 2. Academic Press, London, U.K. Pp. 418–421, 489–491.