

## Observations on the Pathology of Lesions Associated with *Stephanofilaria dinniki* Round, 1964 from the Black Rhinoceros (*Diceros bicornis*)

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In South Africa, a filarioid infestation has been associated with lesions in the skin of the black rhinoceros (Schulz and Kluge, 1960). Similar lesions had been reported earlier from Kenya, as consistently occurring in these animals but their aetiology was unknown (Spinage, 1960). Recently the opportunity occurred to examine lesion material from four black rhinoceros located in the Royal Tsavo National Park Kenya. From this material adult helminths were recovered and identified (Round, 1964), and further pathological studies made. In addition, one rhinoceros showed evidence of an otitis.

### PATHOLOGY

#### (a) *Skin lesion*

##### *Macroscopic observations*

In the black rhinoceros, the consistent lesion is situated on each flank behind the point of the elbow. However, lesions may occur elsewhere, especially on the limbs. The extent of the lesions varied, but the largest observed were 15-20 cm. in diameter, and the smallest between 5-7 cm. The lesion was characterized by erosions, ulcerations and crust formation; the central area was depressed and the periphery raised above the level of normal skin. The average thickness of unaffected portions of skin from over the last two ribs was 1 cm. The lesion area was between 2-3 cm. thick, and on section was seen to have epithelial damage in the centre, and thickening on the periphery. In one rhinoceros, the skin adjacent to and posterior to the flank lesions on both sides indicated the possibility of areas of recovery from infestation.

### HISTOLOGY

For convenience the skin and lesion areas have been described as follows :—

- (1) Normal skin ;
- (2) Centre of lesion ;
- (3) Periphery of lesion.

(1) *Normal skin*

This was taken from the flank behind the last rib and was about 1 cm. thick. The superficial layer consisted of keratin, deep to which was stratified epithelium. The cells were typical of this structure with occasional vacuolation of the nuclei and well-defined stratum granulosum and stratum germinativum; pigment could be readily seen in this layer. The stratified epithelial layer formed only a thin portion of the total thickness of the skin. The dermal layer consisted of collagen connective tissue considerably denser than that occurring in domestic animals. In the dermis, islands of serous-type (apocrine) sweat glands could be identified. Hair follicles, sebaceous glands and adipose tissue were not seen. The vascular bed appeared normal.

(2) *Centre of lesion* (Plate I, Fig. 1)

There were extensive areas of necrosis with almost complete obliteration of the epidermal layers, and replacement by inspissated exudate and debris. Occasional "islands" of stratified epithelium could be identified. Underlying the necrotic area, there was considerable increase in granulation tissue and young cellular fibrous tissue. Perivascular accumulation of mononuclear cells, mainly of the macrophage type was present, varying in extent from partial to complete cuffing. In the middle layers of the dermis, the apocrine sweat glands were flattened in appearance. In the deep layers of the dermis, foci of mononuclear and eosinophil cells could be seen associated with the small blood vessels.

No adult helminths or free microfilariae could be identified.

(3) *Periphery of lesion* (Plate I, Fig. 2 and Plate II, Fig. 3)

Superficially there was an eosinophilic exudate containing inflammatory cells mainly polymorphonuclear and cellular debris. The epidermis showed proliferation, hyperplasia of the stratum malpighii with elongated rete pegs penetrating deeply into the dermis. The rete pegs were seen cut in different planes and this emphasized their irregularity and branching. The basophil stratum germinativum cells showed numerous mitotic figures and were several layers deep with nuclei partly overlapping. Within the epidermal layer, adult helminth tissue was identified in spaces lined with flattened epithelial cells. The internal structures of the helminth were visible and within the uteri of the females, microfilariae were seen. No free microfilariae could be identified. In addition, "vacuoles" in the epidermis containing eosinophilic keratin-type material and cellular debris were noted. Between the rete pegs, the superficial layers of collagen had been replaced by fibroblastic tissue, with oedema in some areas. A diffuse mononuclear reaction was also present.

Deep to the projection of the epidermal cells, there was an increase in the vascular bed with some mononuclear infiltration often associated with blood vessels.

In lesion material from one animal numerous foci of heavy concentrations of mononuclear cells were seen deep in the dermis. The cells were lymphocytes, macrophages and plasma cells. In addition eosinophils were evident. There was an increase in the vascularity of the area, but little indication of a fibroblastic reaction. In the centre of many of the foci, degenerating helminth tissue could be seen, with a tendency in some instances to early calcification. Foreign body-type giant cells were occasionally noted in some foci.

(b) *Ear lesion*

*Macroscopic observations*

The auditory meatus contained a mass of thick sebaceous-like material which completely filled the ear. On section, the epithelium appeared intact but there was a suggestion of an otitis. As the presence of helminths was not suspected, fresh material was not retained.

HISTOLOGY (Plate II, Fig. 4)

Examination of the material showed changes similar to those recorded from the periphery of the flank lesion. Adult helminth tissue could be identified in the epidermal zone. The epidermal cells showed proliferation and hyperplasia with elongated rete pegs. The stratum corneum was not evident.

The sebaceous glands had wide ducts which on cross-section suggested cyst formation. However it is assumed that these structures are normal for this part of the body. They were partly filled with inspissated laminated eosinophilic staining material. Similarly the ceruminous glands had ducts which on section were wide and filled with an homogeneous eosinophilic staining material. Both types of glands exhibited evidence of increased secretion.

There was an increase in the vascularity of the affected area. The slight mononuclear cell reaction was diffuse in the superficial layers, but tended to be associated more with the blood vessels in the deeper layers. Eosinophils were few in number.

No free microfilariae could be identified in any part of the material examined.

## CONCLUSIONS

The changes occurring in the affected portions of the skin of the black rhinoceros varied. In the centre of the lesions, a subacute inflammation was seen. There was necrosis and almost complete obliteration of the epidermal layers. There was no evidence to indicate the presence of helminths in this zone. However, the periphery of the lesions was characterized by acanthosis with penetration of the deeper tissues by long papillary processes. There was an increase in vascularity, and a mononuclear and eosinophil reaction in the dermis. Perivascular cuffing was frequent.

Adult helminths were only found at the periphery of the lesion, and were usually contained within the area of hyperplastic epidermis.

In the ear, the reaction was entirely chronic, with some increase in secretory activity of the glands.

Evidence is produced that the pathology of lesions associated with *Stephanofilaria dinniki* in the black rhinoceros in Kenya, closely resembles that described in South Africa. It has not been possible to compare lesion material collected at different seasons of the year, but this paper describes that recovered during July and September. In the main, it appears to conform to the intermediary stage mentioned by Schulz and Kluge.

Although the filariid occurring in the ear is, as yet, unidentified, the pathology is similar to that of the skin lesion. There is the suggestion, therefore, that the infestation also may be associated with *Stephanofilaria dinniki*.

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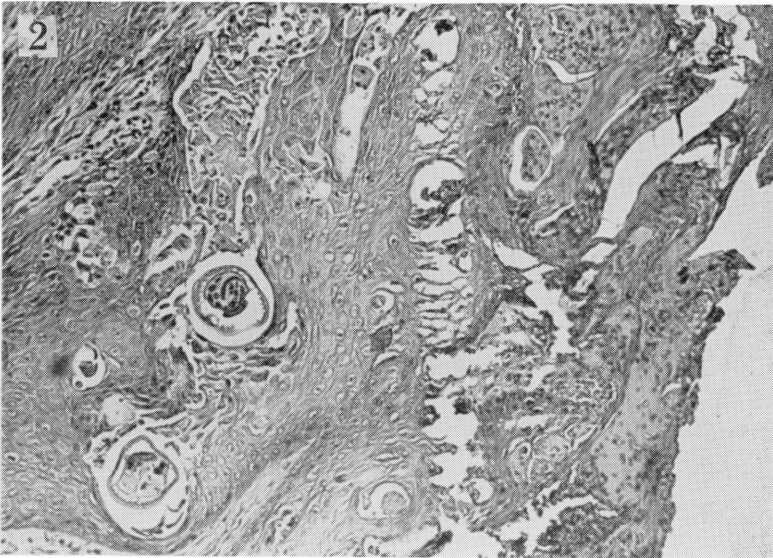
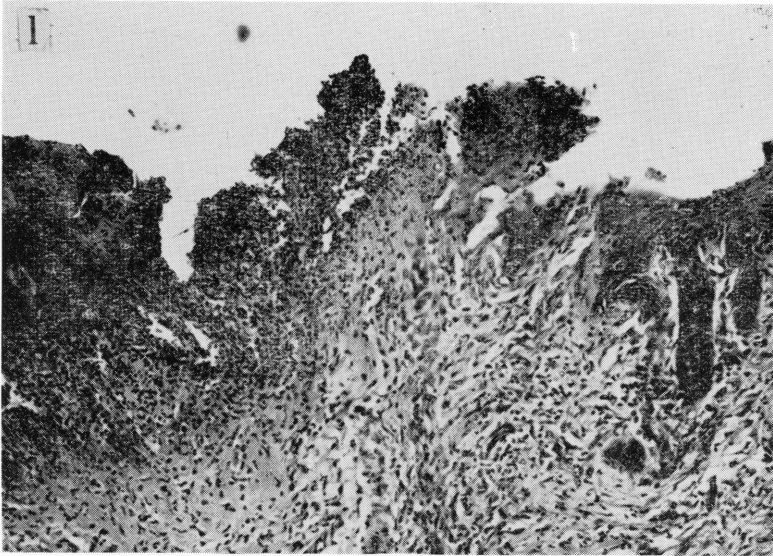


PLATE I

Fig. 1.—Centre of lesion. Extensive necrosis, subacute inflammation and almost complete obliteration of the epidermis. Fig. 2.—Periphery of lesion. Proliferation of epidermal cells. Adult *S. dinniki* evident within. (Both Figs.  $\times 66$ ).

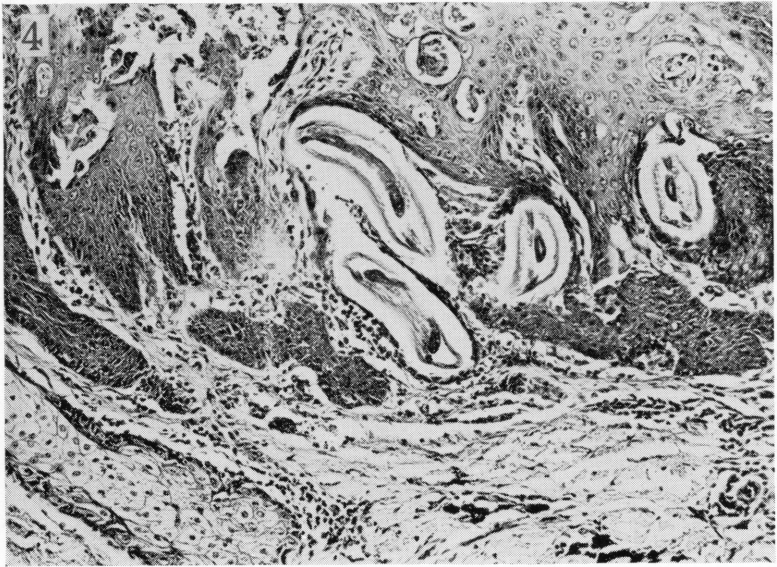
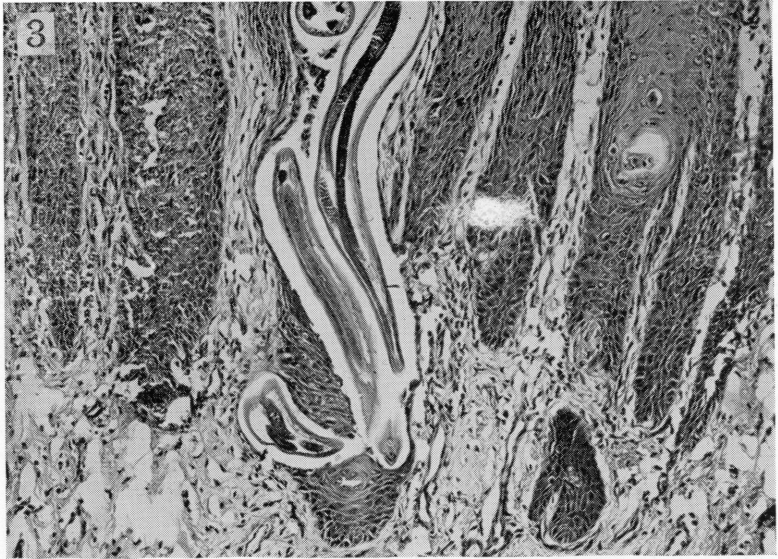


PLATE II

Fig. 3.—Periphery of lesion. Acanthosis and penetration of the deeper tissues by long papillary processes. Adult *S. dinniki* evident. Fig. 4.—Ear lesion. Hyperplasia of epidermal cells. Filariids present. (Both Figs.  $\times 66$ ).