Degenerative Polymyopathies in East African Domestic and Wild Animals

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SUMMARY.—Pronounced degeneration and necrosis of skeletal muscle fibres, either of segments or of whole fibres has been observed in East African wild and domestic animals. Interstitial inflammation of variable severity and active regeneration of muscle tissue have also been a constant feature in these lesions.

Introduction

HYALINE degeneration of striated muscle constitutes a distinct pathological change in naturally-occurring muscular disorders of domestic animals. The lesion has been reported with diverse histories of disorders, thus suggesting that several pathological entities exist which probably do not have a common cause. In man, the most important disease involving skeletal muscle is muscular dystrophy which is a genetical syndrome with no counterpart in other animals, except in mice and possibly chickens (Bourne & Galarz, 1963). Myopathies in domestic animals have been widely associated with dietary deficiencies or imbalances of vitamin E and/or selenium (Muth, Olfield, Schubert & Remment, 1959). They are observed predominantly in areas where pregnant ewes and cows are fed rations deficient in either vitamin E and/or selenium and seen in new-born calves and others as old as six months with an average range of one to two months old (Hadlow, 1962). Lambs are usually affected at the age of two to four weeks, but the condition may be apparent from birth up to two to three months (Muth, 1955).

The purpose of this report is to describe muscular degeneration in various East African wild and domestic animals which may have different aetiological agents.

Materials and Methods

Group I : Clinical History and Signs

In Group I, nine black rhinoceros were involved. The animals were caught to be moved from the Parks to different Zoos overseas. During the catching operations the rhinoceros were treated with berenil, ethidium and prothidium to deal with trypanosomiasis which was enzootic. The dosage was 9 g. of each at different intervals. There was no after-effect following treatment with these drugs, with the exception of an occasional abscess at the point of the injection.

After capture the rhinoceros were brought into a waiting compound where one rhinoceros died 20 days later. Babesiosis was diagnosed as the cause of the death. After that, the rest of the rhinoceros were sprayed with delnav (O-Odiethyl phosphorodithiate). Spraying was done for six days. During the spraying period four rhinoceros died, one after two days of spraying, the second after three days of spraying, the third after four days of spraying and the fourth after six days of spraying. The rest of the rhinoceros, except one, died between the sixth and eleventh day after spraying ceased. The clinical history was similar, that is, muscular weakness, unsteady gait, dark urine, collapse and then death.

Acute Granulocytic Leukaemia in a Bitch.—*Concluded.*

None of the eight cases of myeloid leukaemia reported by Meir (1957) included the purpuric syndrome shown in our case. His cases showed non-specific signs of inappetence, weight loss, vomition and, in four of the cases, there was a febrile reaction not responding to antibiotics.

The case of granulocytic leukaemia described in the report of a clinico-pathological conference (1963) showed two episodes of epistaxis which were attributed to thrombocytopenia, but the case differed in some respects both clinically and *post mortem*. The case reported by Medway and Rapp (1962) had episodes of true purpura, and melaena on many occasions over a period of three years. Their case apparently responded to treatment although it subsequently died. The case described by these authors in an addendum to their paper closely resembles the case now reported, particularly in its clinical aspects. There are also many similarities between our case and the condition as described in man (Whitby & Britton, 1947). In previous reports no mention has been made of the splenic and kidney infarction and thrombosis in the oral mucosa, nor has an increase in marrow plasma cells been noted.

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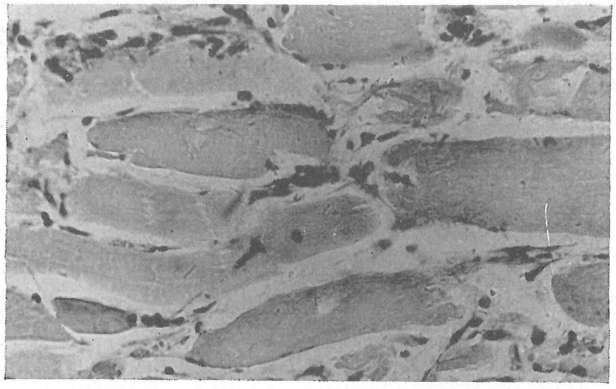

FIG. 1.—Hyaline degeneration of thigh muscle of black rhinoceros. H. & E. ×400.

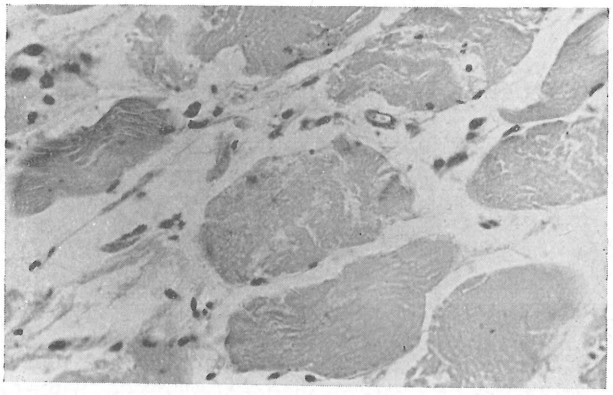

FIG. 2.—Lumpy fragmentation and lysis of necrotic muscle fibres of the quadratus femoris muscle of a cow. H. & E. ×400.

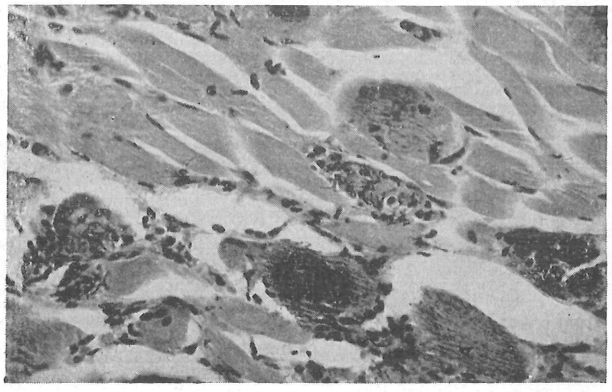

FIG. 3.—A section of thigh muscle of a black rhinoceros. (a) Granular degeneration of muscle fibres. H. & E. ×250.

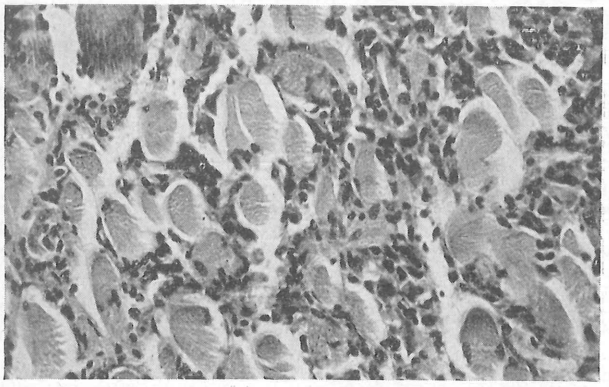

FIG. 4.—A cross-section of necrotic thigh muscle of a black rhinoceros with proliferation of sarcolemmal nuclei. H. & E. ×250.

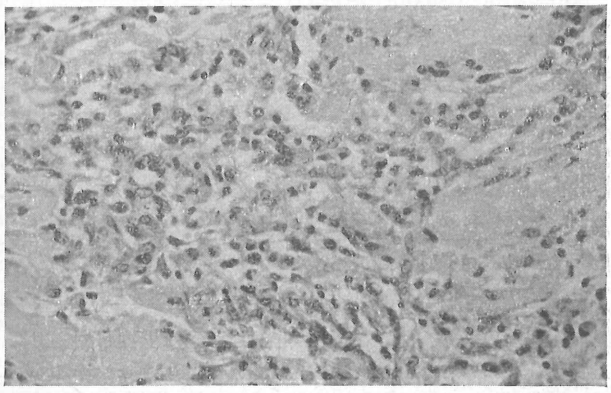

FIG. 5.—Active regeneration of muscle after lysis of necrotic muscle fibres. Section taken from a thigh muscle of a black rhinoceros. H. & E. ×400.

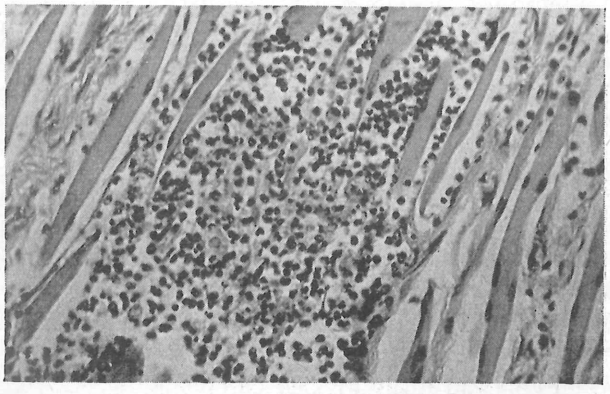

FIG. 6.—Leucocytic infiltration in areas where necrotic muscle fibres had been lysed and in the inter-muscular fibre spaces. Section taken from this of Coke's Hartebeeste. H. & E. ×250.

Gross and Microscopic Lesions

The following tissues were collected and fixed in 10 per cent. neutral formalin: damaged muscle, heart, lung and kidney. Liver was collected from two rhinoceros only, which died after spraying ceased. The sections were stained with haematoxylin and eosin. The histological and staining techniques followed were according to the Manual (1960).

Gross Lesions. At *post-mortem* examination, the only constant lesion was paleness of the skeletal muscle of the hind-legs. The rhinoceros which died early had dark urine and the lungs had alveolar or interstitial emphysema, but those which died after spraying had ceased had apparently normal urine.

Microscopic Lesions

Kidney. There was a coagulative type of necrosis

involving the tubular epithelium, characterised by disintegration of the nuclei. Many nuclei were noted at the karyorrhexis stage of disintegration. Cytoplasm of renal cells varied from granular to homogeneous eosinophilic cytoplasm. There were extensive interstitial haemorrhages, both in the cortex and medulla. In some cases, tubules were filled with erythrocytes. The Bowman's capsule was either filled with a mass of erythrocytes or proteinous exudate. In some animals, hyaline casts were numerous in the tubular lumina.

Liver. In two cases there was marked centrolobular haemorrhagic hepatitis. In some lobules, the hepatic cells had been completely destroyed and replaced by a mass of erythrocytes.

Lungs. The lungs were congested and oedematous. Serous exudate was noted in the alveoli. Alveolar emphysema was common in different lobules of the lungs with broken alveolar walls.

Heart. No specific lesion was noted in this organ.

Skeletal Muscle. The microscopical changes in skeletal muscle varied somewhat from one animal to another, but the essential features were the same in all animals studied.

The morphological alterations comprised granular degeneration, but more commonly waxy or hyaline degeneration. In granular degeneration, segments of the muscle fibres were swollen and stood out in sharp contrast to the adjacent fibres that were displaced by pressure. The protoplasm had been transformed into uniform pale granules.

In hyaline degeneration, the lesions were characterised by change of some or all muscle fibres into a homogeneous acidophilic mass. There were many lumpy cleavages of muscle fibres. Some fibres appeared coarsely granular and basophilic among the acidophilic mass. Occasionally serous exudate with polymorphonuclear leukocytes and lymphocytes were seen in the necrotic debris and between the swollen muscle fibres.

In the rhinoceros which died after six days of spraying there was more interstitial cellular exudate consisting chiefly of macrophages, lymphocytes and a few polymorphonuclear leukocytes. In this case a conspicuous regenerative activity was noted. This was characterised by marked proliferation of muscle nuclei in a linear pattern along the periphery on one side and at the junction of the normal and necrotic part of the fibre. The proliferating muscle nuclei were large, round, vesicular and hyperchromatic with a thin nuclear membrane enclosing two dot-like nucleoli and finely divided chromatin.

In some areas the nuclei were elongated fusiform, or round with a densely-stained nuclear membrane, fine chromatin and two or three large, intensely basophilic, nucleoli.

There was calcification of portions of affected fibres in two rhinoceros. The calcium deposits appeared as blue crystals arranged transversely in the fibres.

Group 2

In this group, different wild animals were submitted to our diagnostic laboratory from different parts of East African National Parks. The animals had died at various times after capture, or they were found dead in the Game Parks. In 1965 our laboratory diagnosed 13 cases of muscular degeneration in wild animals. The animals involved were six wildebeestes, two Thomson's gazelles, two topi, one Hunter's antelope, one buffalo and one Coke's hartebeest.

Gross and Microscopical Lesions

The following tissues were collected and fixed in neutral formalin: damaged muscle, hind-leg muscle, pectoral and cardiac muscle.

Gross Lesions. At *post-mortem* examination, whitish streaks were seen in the affected muscle. The streaks were parallel to the plane of muscle fibre arrangement. They varied in size from one animal to the other and from one muscle to the other in the same animal.

The lesions were found in muscles of the hind-legs in five wildebeestes, in axillary muscles in one wildebeest and in cardiac muscle in three wildebeestes.

In the buffalo and two Thomson's gazelles, the lesions were found only in the muscles of hind-legs. Both legs were involved to the same extent. In the case of the Hunter's antelope the lesion was noted in the pectoral muscles on the left side.

In the topis the lesions were found in the muscles of hind-legs and of the heart.

Microscopical Lesions. Microscopically, the lesions varied from one animal to the other, but the major feature was hyaline degeneration with loss of cross striations. Some muscles had interstitial infiltration of lymphocytes, macrophages and occasional neutrophils. In some lesions there was marked proliferation of the sarcolemmal sheath with increase of hyperchromatic nuclei.

Coke's Hartebeest

This was an adult female which had been captured by a game-trapper. She was kept in a waiting compound with others for eight days. On the ninth day she was found lying down, prostrated and with a fever. She was treated with a broad-spectrum antibiotic but failed to respond. She died two days after the first sign of sickness.

Gross Lesions. There were several pale patches in the skeletal muscles of the hind-limbs. The patches varied greatly in size. The lesions were more extensive bilaterally in the semimembranosus and semitendinosus muscles. Lungs, liver and kidneys appeared congested.

Microscopical Lesions. There was degeneration of muscle fibres. Some fibres were swollen and coarsely granular. In some areas among the damaged fibres there were some which appeared completely normal. The damaged fibres showed a loss of striation and hyaline degeneration. Some fibres appeared brightly eosinophilic with lumpy fragmentation and granular disintegration while others had undergone complete necrosis and disappeared.

The kidneys were very congested and there were numerous hyaline casts in the tubular lumina. The

casts were more numerous in the cortex and scanty in the medulla.

Group 3

In this group there were two bulls that had been brought to our large-animal clinic for treatment, and a dead cow that was brought for diagnosis. One bull had shown paralysis of the hind-limbs for two weeks prior to being brought to the clinic. He failed to respond to broad-spectrum antibiotic treatment and was then destroyed for *post-mortem* examination a week later.

Pathological Lesions

The first animal was in good condition nutritionally. No gross lesions were seen in any of the organs, except in the skeletal muscle of hind-limbs. There was marked muscular degeneration. This completely involved the quadratus femoris muscles bilaterally. The lesions were pale grey in colour, with haemorrhagic necrosis on the periphery. Portions of the muscles on either side were fixed in 10 per cent. buffered formal saline for histological examination.

Microscopically the fibres showed varying stages of muscle degeneration. These ranged from hyaline degeneration and coarse granular degeneration to coagulative necrosis with fragmentation and disappearance of many muscle fibres. Proliferation of sarcolemmal nuclei was very evident, taking a plumb and spindle-shaped appearance. Some sections showed very marked haemorrhage into the degenerating muscle.

The second bull, an adult Ayrshire, was from a farming estate. He was destroyed *in extremis* due to photosensitisation with extensive sloughing and superficial secondary infection. Pathologically, the skin lesions showed severe photosensitisation. It was particularly marked on the lateral aspects of the four limbs, the pectoral region, the perineum and the scrotum. The liver was pale and swollen. The semitendinosus muscle of the left side had groups of white muscle fibres extending for over a foot in length. Histologically, the liver had minute focal necrosis around most of the centrolobular veins. The portal areas showed proliferation of fibroblasts and bile ducts; infiltration of lymphocytes was present in these areas. The microscopic appearance of the muscle fibres was similar to the other bull.

The cow had shown central nervous symptoms before she died. The main lesions, bilaterally, involved the semitendinosus muscles. Each had a channel filled with clotted blood, and the latter surrounded by whitish grey infiltrating parts of muscles. The adjacent muscle groups appeared uninvolved. The histological appearance was similar to the above.

Discussion

Pathological changes in skeletal muscles, similar to those seen in different animals in our laboratory, have been induced experimentally with deficiencies of vitamin E, thiamine, ascorbic acid, vitamin A, and in animals traumatised by a wide variety of physical and chemical agents (Fishback and Fishback, 1932).

Smith, Black-Schaffer and Lasater (1950) reported similar lesions in potassium deficiency in dogs. Gifford and Dalldorf (1951) observed similar lesions in muscles of mice infected wth Coxsackie virus. Ellis (1956) induced hyaline degeneration and regeneration of skeletal muscle by injecting cortisone into rabbits.

In young domestic animals, hyaline degeneration of skeletal muscle has been associated with vitamin E and/or selenium deficiency (Muth *et al.*, 1959). The lesions observed in East African wild animals could have resulted from an unaccustomed muscular exertion during catching operations.

In the cases of rhinoceros the delnav poisoning could have been a possibility. In the other animals, plant toxins in the Parks or on the farms could have contributed to these lesions. Henson, Dollahite, Bridges and Rao (1965) reported myodegeneration in cattle grazing Cassia species. The lesions they observed were similar to the lesions seen in different animals in our laboratory.

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DESTRUCTION OF MOLES

The Minister of Agriculture, having been asked whether he is satisfied that the licensing system for strychnine poison for the destruction of moles adequately controls the use of this poison, said:—

"The retail sale of strychnine for controlling moles is restricted by the Poisons Rules to persons who produce a permit signed by an authorised officer of one of the Agricultural Departments. Permits are issued only to applicants who show that they need strychnine for mole control, and know how to use it. These arrangements are kept under review by my right hon. Friend the Home Secretary, who is responsible for the Poisons Rules, and my right hon. Friend, and we are satisfied that no change is necessary."