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ELAEOPHOROSIS IN BARBARY SHEEP AND MULE DEER FROM THE TEXAS PANHANDLE

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Abstract: Adult *Elaeophora schneideri* were recovered from the common carotid artery and its branches in 14 of 14 mule deer, *Odocoileus hemionus,* and 3 of 9 Barbary sheep or aoudads, *Ammotragus lervia,* from Palo Duro Canyon in the Texas Panhandle. Gross cutaneous lesions attributable to elaeophorosis in the Barbary sheep varied from small circumscribed scars up to 10 cm in diameter usually on the poll or orbital region to extensive proliferative irregular encrustations on the frontal, temporal and orbital regions, sometimes extending to the ears and muzzle. Individual lesions varied from slate-gray scarred areas to brown proliferative edematous and hyperemic encrustations, sometimes with depigmented pustules a few millimeters in diameter. Microscopic lesions ranged from granulation tissue to severe pyogranulomatous reactions with neutrophils, eosinophils, lymphocytes and plasma cells as the primary infiltration. Foreign body giant cells and/or microfilariae were not observed. Microscopic changes in the carotid arteries and their branches were limited to small villous projections on the intimal surface apparently resulting from medial hyperplasia. Cutaneous lesions attributable to elaeophorosis were not observed in mule deer. Histopathologic lesions in the carotid arteries of mule deer were similar to those observed in Barbary sheep. The comparative pathology of elaeophorosis in various hosts is reviewed and discussed in terms of its pathology in Barbary sheep. The potential ramifications of this infection on the expanding aoudad population in the southwestern United States require that elaeophorosis be considered in the management of this species, particularly in areas with sympatric mule deer populations.

INTRODUCTION

From 1949 through 1951, the Texas Parks and Wildlife Department released 268 mule deer, *Odocoileus hemionus,* in Palo Duro Canyon to augment a small remnant population.[2] Forty-four aoudads or Barbary sheep, *Ammotragus lervia,* an exotic species native to North Africa, were introduced into the canyon in 1957-58.[3] Palo Duro Canyon, a deep reentrant gorge, about 80.5 km long by 17.5 km in maximum width, located in the otherwise flat, featureless central Texas Panhandle, was formed by the erosive action of the Prairie Dog Town Fork of the Red River. The steep bluffs of the caprock escarpment form the canyon walls overlooking the rolling canyon floor 183-244 m below. Canyon topography is highly variable and includes mesas, ledges, benches and ridge fingers. The vegetation reflects the topographic diversity and includes mesic species along the stream bottoms as well as typical shortgrass prairie and semiarid plants.[8] Good forage and suitable habitat account for the increasing wild ungulate populations in the estimated 200,000 ha area of the Palo Duro.

The present population of Barbary sheep is now estimated to be in excess of 1,600 animals while the mule deer population is thought to exceed 6,000

animals[2,3] and, in recent years, income from hunting camps has become a very important economic component to many local ranching operations. Cattle graze the canyon floor and less demanding slopes within mule deer and Barbary sheep ranges as well as the High Plains adjacent to the canyon. Aside from a few small herds of white-tailed deer, *Odocoileus virginianus,* there are no other wild or domestic ruminants in the area. The purpose of this paper is to document the first recognized occurrence of elaeophorosis in Barbary sheep and to eludicate certain aspects of the pathology of the disease in this host.

MATERIALS AND METHODS

Nine Barbary sheep and 14 mule deer from the Dry Creek study area located in Palo Duro Canyon, Armstrong County, Texas were examined for elaeophorosis during the November hunting seasons from 1977 through 1979. Approximately 2 cm square sections from representative areas of head lesions were excised and preserved in 10% formalin for later histologic examination. Lesions were swabbed with sterile cotton applicators and inoculated on Sabouraud's glucose agar slants incubated at room temperature. The entire head and neck of 6 Barbary sheep and 14 mule deer were transported to the laboratory for necropsy. The carotid arteries and their secondary branches were excised and examined for *Elaeophora schneideri.* Nematodes were fixed in glacial acetic acid and stored in 70% ethyl alcohol with 5% glycerine. Pieces of carotid arteries from infected mule deer and Barbary sheep were fixed in 10% formalin for later histologic examination. Sections of skin lesions and carotid arteries were cut at 4 to 6 μm and stained with hematoxylin and eosin, Gram's stain, Giemsa and/or Verhoff's iron hematoxylin and van Gieson's stains. Thin sections of epon-embedded material were cut from formalized material for electron microscopy. Suspensions of minced tissue were also negatively stained after spraying formvar-coated grids for examination for virus particles.

RESULTS

For the past 4 years, several researchers have been involved in the populations dynamics, food habits, behavior and parasites and diseases of wild ungulates, principally the aoudad, in the canyon. During the course of these studies, we noted on several occasions the presence of small to sometimes extensive encrusted scabbing lesions on the muzzle, face, ears and crown of Barbary sheep. These were usually considered by local ranchers, hunters and guides to be scars resulting from fighting. In 1977, we had the opportunity to examine in the field a mature female which had severe proliferative lesions on the muzzle, gums and face extending on the crown to the base of the horns and involving the poll and ears. Initially, a viral etiology for these lesions was suspected since they clinically resembled contagious ecthyma or sheep pox. Histologic examination at both the light and electron microscope levels failed to produce evidence of inclusion bodies or virus particles. However, a focal to diffuse inflammatory response and presence of yeast buds and fungal hyphae suggested a possible dermatophycosis. Cultures of material from a second animal showing extensive lesions and two additional less severely affected heads obtained in the 1978 hunting season revealed *Trichophyton* sp. and *Candida albicans* as well as *Staphylococcus albus* in only one of the less severely affected animals. Only *S. albus* was cultured and observed in the more severely affected animal. Examination of the carotid artery and its branches in two of the above hosts, including the more severely affected animal, collected in 1978 revealed a number of specimens of *E. schneideri.* Subsequently, the recovery of this nematode in 14 of 14 mule deer examined from the same area and reevaluation of the histopathology of

this infection suggested a diagnosis of elaeophorosis as the causative agent for this condition in Barbary sheep. Of 5 aoudad heads examined during the 1979 hunting season, one host had slight scarring on the crown at the base of the right horn indicative of an older healed lesion, and another had an irregular, active, extensively proliferating lesion over much of the crown, extending unilaterally to the area surrounding the right eye. On necropsy of the head, 13 adult *E. schneideri* were removed from the internal carotid arteries of the latter host. The remaining three animals did not show any clinical manifestations of elaeophorosis and the carotid arteries of these, as well as the fourth Barbary sheep with older healed scars, were not infected with *E. schneideri*.

Gross cutaneous lesions in Barbary sheep varied from small circumscribed slate-gray scars, 5 to 10 cm in diameter on the poll and/or orbital region of the head, to extensive proliferative irregular encrustations on the frontal, temporal and/or orbital regions, sometimes extending to the ears and muzzle (Figs. 1, 2, 3). These lesions were often, though not always, unilateral. The most severely affected animal examined in 1977 had extensive slate-gray to brown lesions on the muzzle, lips, left eyelid and left ear (Figs. 1, 4). Lesions on the frontal and temporal areas, poll and crown were superficially dry, but certain regions of lighter depigmented plaque-like pustules a few mm in diameter as well as larger pale papillomas were noted on the muzzle. There was general alopecia over

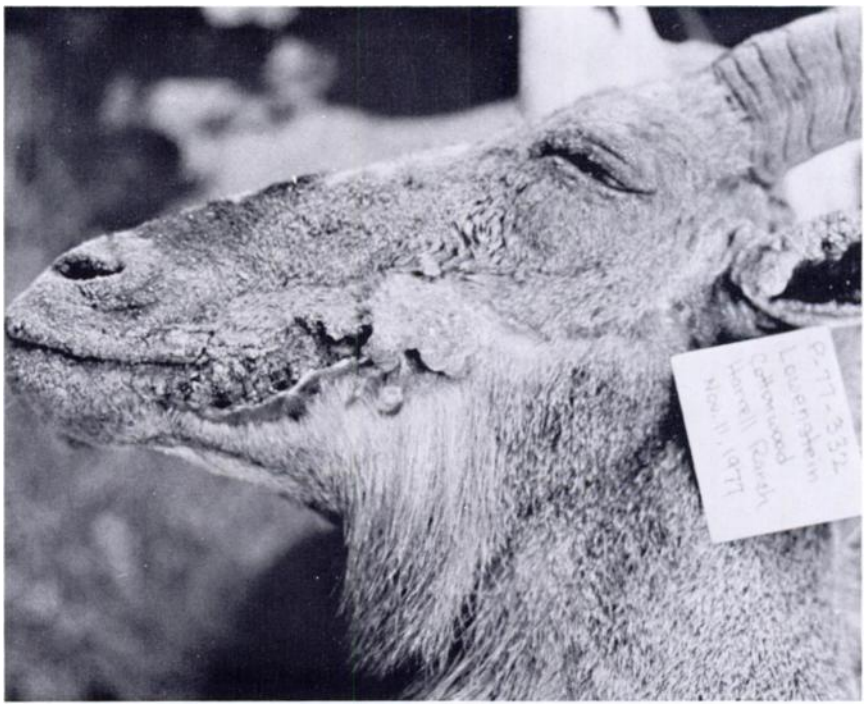

FIGURE 1. Suppurative and proliferative muzzle, orbital and ear lesions of a mature ewe Barbary sheep from Palo Duro Canyon, Texas. This animal also exhibited active and healed lesions on the poll and crown.

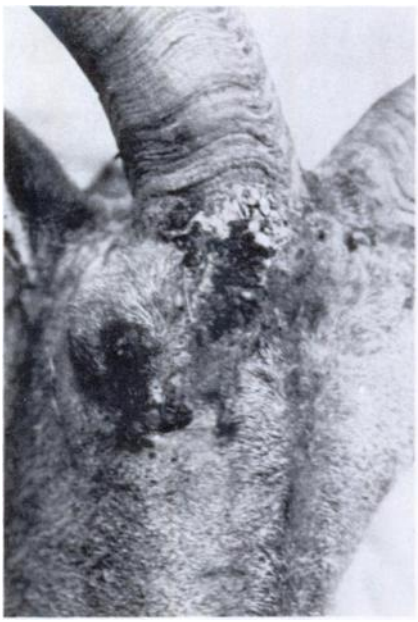

FIGURE 2. Active lesions on the crown, temporal and orbital regions of a Barbary sheep infected with *Elaeophora schneideri*. Note deterioration of the right horn sheath.

FIGURE 3. Encrusted proliferative lesions due to *E. schneideri* in the crown and orbital region. Note lesions extending into right horn sheath.

much of the affected area and some regions were edematous with a serous exudate oozing from beneath the proliferated epithelium (Fig. 1). The lips were edematous and hyperemic. The above lesions were unilateral involving the left ear and eyelid and confluent with the remainder of the poll, crown and muzzle (Fig. 1). The gums and tongue were unaffected. Lesions were noted only on the head. Feet and legs were unaffected. Field examination of this female, estimated to be approximately 8 years old, revealed little other than extreme emaciation.

Similar, but slightly less extensive, lesions were seen on two additional aoudads examined in 1978 and 1979, respectively. Both these animals had more distinctly confluent lesions involving the poll, crown, frontal and orbital areas. There was an indication of deterioration of the horn sheath extending as much as a centimeter from the base (Figs. 2, 3). Although the lesions extended unilaterally into the orbital region, the corneas in both hosts appeared normal. The lesions appeared as dry encrusted scars in one aoudad (Fig. 3), while in the second animal, it was more proliferative with pronounced suppuration in certain areas (Fig. 2). In general, the lesions in both hosts consisted of a greatly thickened epidermis, slate-gray discoloration, alopecia and a dry, scaly surface. Thirteen and four reproductively mature *E. schneideri* were removed from the common carotid artery at the bifurcation of the external carotid arteries in these two hosts, respectively. In the former host, adult nematodes were unilateral and found on the side showing

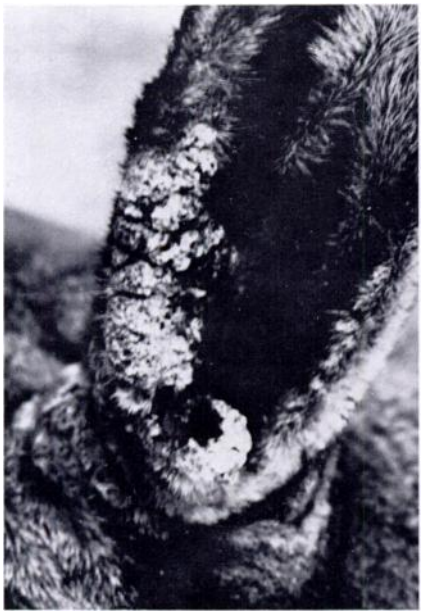

FIGURE 4. Active lesions of *E. schneideri* on the left ear of a Barbary sheep. Same animal as Figure 1.

the lesions, while the nematodes were removed from both carotid arteries of the second animal. Grossly, very small mammalations were observed on the intimal surface of infected carotids of both hosts. Additionally, 6 adult *E. schneideri* were removed from a second less severely affected animal in 1979.

During the hunting seasons of 1976 and 1977, prior to attempts at elucidating the etiology of facial lesions, 6 of 27 additional hunter-killed Barbary sheep examined in the field in conjunction with other studies and ranging in age from 6 months to 10 years demonstrated similar encrusted lesions. These were usually only one to several centimeters in diameter, had a dry, scaly surface and sometimes had partial regrowth of hair. It is speculated that these apparently represent partially to almost completely healed scars resulting from previous experiences with elaeophorosis.

Gross lesions of the skin of the head were not observed in any of the 14 infected mule deer examined. Infection levels ranged from 1 to 25 nematodes in an individual host. Small villous projections, similar to those observed in Barbary sheep, were noted in the internal carotid arteries of more heavily infected hosts.

Microscopic examination of skin and subcutaneous tissues from infected Barbary sheep with active suppurating lesions revealed that the epithelial surface consisted of an eosinophilic mass of amorphous serous fluid, necrotic cells and dark brown pigment granules. Hyperkeratosis with a hyperplastic pigmented squamous epithelium often thrown up in folds and long, spike-like projections was present in the more proliferative lesions. In some areas there was extensive loss of the surface epithelium to the level of the hypodermis. There was extensive edema in the hyperplastic stratum granulosum and hypodermis with a cellular infiltration consisting mainly of neutrophils, eosinophils, plasma cells and lymphocytes. Usually, the most intense inflammatory response was noted in the reticular layer of the dermis and consisted of dense accumulations of neutrophils and eosinophils with a few epithelioid cells and pigment-laden histiocytes. Foreign body giant cells and microfilariae were not observed in these lesions. There was severe vacuolation of the arterial adventitia with a periarteritic infiltrate of eosinophils, neutrophils and lymphocytes. Sebaceous glands and hair follicles were filled with serous fluid and surrounded by an inflammatory response of eosinophils, lymphocytes and plasma cells. In some of the more intense lesions, a few gram-positive cocci, fungal hyphae and yeast cells were observed.

Microscopically, these lesions varied from the intense, acute inflammatory response described above to healed granulation tissue from older scars. The

latter were characterized by little or no inflammatory response and absence of foreign body giant cells or microfilariae.

Microscopic changes in the carotid arteries were limited to small villous projections of the intimal surface apparently resulting from medial hyperplasia. Fibroplastic proliferation of the tunica media was without an accompanying inflammatory response. This appeared to be the same response observed in the carotid arteries of infected mule deer.

DISCUSSION

The comparative pathology of elaeophorosis in elk and mule deer is well documented.[1,4] The disease is usually subclinical or produces only minor arterial and/or appendage lesions in mule deer.[1] The parasite appears to be common in mule deer from the southwest, and this host species is the suspected reservoir of infection for other native, domestic and exotic species.[1] Elaeophorosis is highly pathogenic for elk, *Cervis canadensis*, in New Mexico, Arizona and Colorado, causing blindness, gangrene of the muzzle and ears and other deformities as well as neurological involvement. In elk, the disease primarily affects the cephalic arterial system resulting in circulatory impairment producing secondary ischemic damage to the brain, eyes, optic nerves, muzzle and other tissues of the head. Massive hyperplasia and proliferative intimal, subintimal and medial changes leading to occlusion and thrombosis of the carotid arteries is the common course in this host.[1] In some years, elaeophorosis accounts for significant elk calf mortality in some herds (C.P. Hibler, 1979, Pers. Commun.).

Although skin lesions were not observed, intimal proliferation of the leptomeningeal arteries, cerebrocortical neuronal necrosis, brain hemorrhage and blindness due to elaeophorosis are reported from moose, *Alces alces*, in Montana.[9] Likewise, impaired function of the mylohyoideus muscles was associated with and possibly caused by *E. schneideri* in a white-tailed deer, *Odocoileus virginianus*, in the southeastern United States.[6] Observations on mule deer from the Texas Panhandle substantiates the lack of significant skin lesions associated with elaeophorosis in this host. Fibroplastic proliferative changes of the media causing villous projections on the intimal surface of the common carotid arteries were the only lesions observed in the present study. Unfortunately, it was not possible to perform thorough necropsies on any of the mule deer or Barbary sheep examined in this study. Thus, the extent of involvement of the iliac, tibial and common digital arteries, as reported in other hosts, remains to be elucidated.

Lesions due to elaeophorosis in domestic sheep consist of raw, purulent encrustations on the poll and temporal region which sometimes extend over the head to involve the eyelids, nostrils, mouth and lips. Occasional lesions occur on the thorax, abdomen and hind feet.[5]

Recently, unilateral tumerous masses on the temporal, frontal and orbital regions and the feet of 4 sika deer, *Cervus nippon*, were reported in Texas.[7] Two to 4 mature *E. schneideri* were found in the external maxillary and external carotid arteries, and although there was intimal proliferation of the arteries, these were not occluded as observed in elk. Microfilariae and foreign body giant cells around degenerating microfilariae were observed in the epidermal lesions.

Head lesions in Barbary sheep described herein are similar to, but more extensive than, those described for domestic sheep. Also, there was little damage to the cephalic arterial trunk in contrast to the extensive medial and intimal involvement reported in elk.[4] This, coupled with the occurrence of only mature nematodes in Barbary sheep and absence of microfilariae in skin lesions which also often occurs in domestic sheep, indicates a similar pathogenesis

for skin lesions described in both hosts. Although microfilariae are conjectured as the cause for the initial irritant producing skin lesions,[5] they are generally few in number and frequently cannot be found in histological sections of lesions from domestic sheep. Also, there is evidence that microfilariae in elk may be present in skin biopsies after a patent infection develops, but may disappear shortly thereafter.[4] A similar condition could occur in Barbary sheep. Alternatively, other factors such as unfertilized ova may serve as the initial irritant for the skin lesions (C.P. Hibler, 1979, Pers. Commun.).

While the present study emphasizes the nature and extent of skin lesions due to elaeophorosis in Barbary sheep from the Texas Panhandle, further studies involving thorough necropsies, especially examining the brain, eye and optic nerves, splanchnic vascular tree and femoral arteries, are warranted to fully elucidate the ramifications of elaeophorosis in this host. Since a large number of the Barbary sheep examined from the Palo Duro Canyon have lesions attributable to elaeophorosis, and in some cases these lesions are so extensive to cause the head to be disgarded by the hunter as a trophy, it is now indicated that this disease must be considered as a factor in management of the species. Certainly, further studies on the pathology, distribution, vectors and epizootiology of elaeophorosis in the Barbary sheep are warranted.

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LITERATURE CITED

1. DAVIES, R.B. 1979. The ecology of *Elaeophora schneideri* in Vermejo Park, New Mexico. Ph.D. Disser., Colo. St. Univ., Fort Collins, 236 pp.
2. DeARMENT, R. 1971. Reaction and adaptability of introduced aoudad sheep. Final Report. Fed. Aid Proj. No. W-45-R-21. Texas Parks and Wildl. Dept., Austin. 20 pp.
3. EVANS, P.K. 1967. The aoudad sheep, an exotic introduced in the Palo Duro Canyon of Texas. Proc. Southwestern Assoc. Game and Fish Comm. Conf. 21: 183-188.
4. HIBLER, C.P. and J.L. ADCOCK. 1971. Elaeophorosis. In: *Parasitic Diseases of Wild Mammals.* J. Davis and R.C. Anderson (eds.). Iowa State Univ. Press. 1st Ed. pp. 263-278.
5. KEMPER, H.E. 1957. Filarial dermatosis of sheep. J. Am. Vet. Med. Ass. 130: 220-224.
6. PRESTWOOD, A.K. and T.R. RIDGEWAY. 1972. Elaeophorosis in white-tailed deer of the southeastern USA: Case report and distribution. J. Wildl. Dis. 8: 233-236.

7. ROBINSON, R.M., L.P. JONES, T.J. GALVIN and G.M. HARWELL. 1978. Elaeophorosis in sika deer in Texas. J. Wildl. Dis. 14: 137-141.
8. ROWELL, C. 1967. Vascular Plants of the Texas Panhandle and South Plains. Ph.D. Disser. Okla. State Univ., Stillwater, 217 pp.
9. WORLEY, D.E. and K.R. GREER. 1972. Elaeophorosis in moose from Montana. J. Wildl. Dis. 8: 242-244.

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