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BILATERAL TESTICULAR DEGENERATION IN A WILD MULE DEER (*Odocoileus hemionus*)

A three-year-old male mule deer (*Odocoileus hemionus*) with polished antlers was collected on November 23, 1968 in Mesa Verde National Park, Colorado. This animal exhibited marked hypogonadism. Testis weights were 3.8 gm left and 11.3 gm right, compared with an average of 41.4 gm per testis for three other animals of the same age that were collected on the same date. No abnormality of urethra, epididymides or tunica albu-

ginea was noted on gross examination. Testes were of the same shape and resilience as normal. Testes and portions of epididymides were fixed in 10% neutral buffered formalin. Specimens were bisected longitudinally and a sample block taken from the area adjacent to the bisection line. Blocks were double embedded in celloidin and paraffin; sectioned at six microns and stained with hematoxylin and eosin.

Histological Observations

Microscopic examination revealed tubular appearances characteristic of testicular degeneration in ruminants (Jubb and Kennedy, 1963. Pathology of Domestic Animals. Volume I. Academic Press, New York. p. 360). Normal tubules were

present also, apparently producing viable spermatozoa (Fig. 1). Tubules with only luminal border deterioration and absence of spermatids and spermatozoa were noted. Progressive tubular degeneration occurred from vacuolation and associated

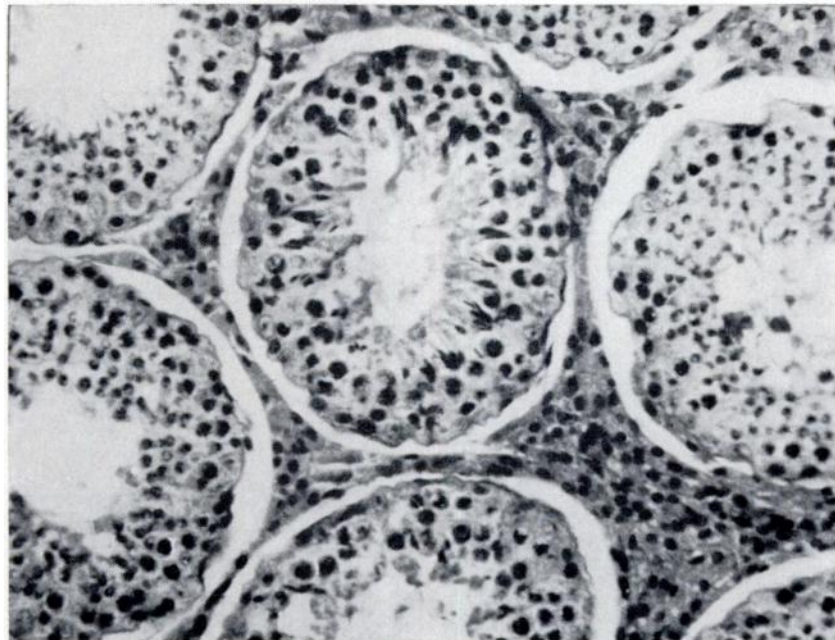


FIGURE 1. Normal tubules from right testis of mule deer exhibiting hypogonadism.

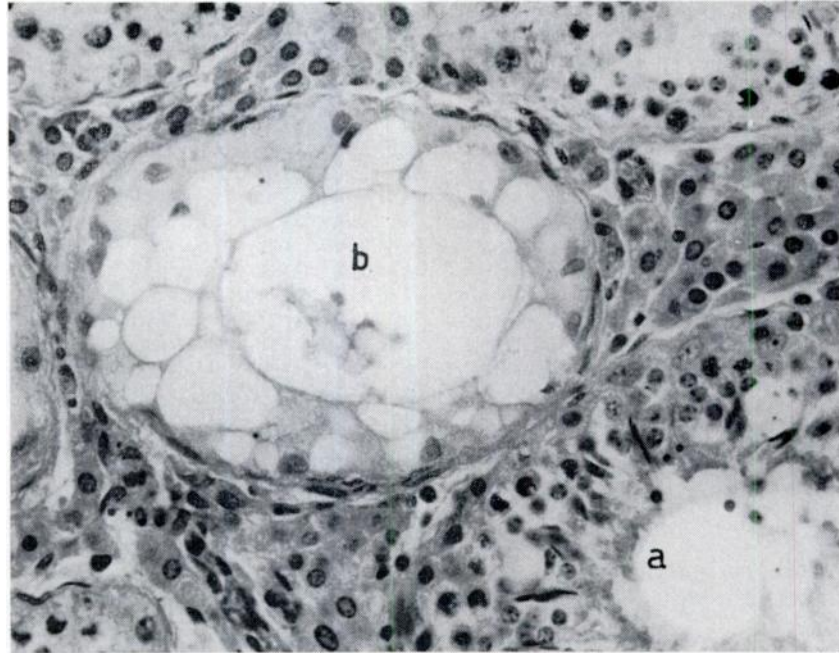


FIGURE 2. Stages of tubular degeneration: (a) luminal necrosis and (b) vacuolation.

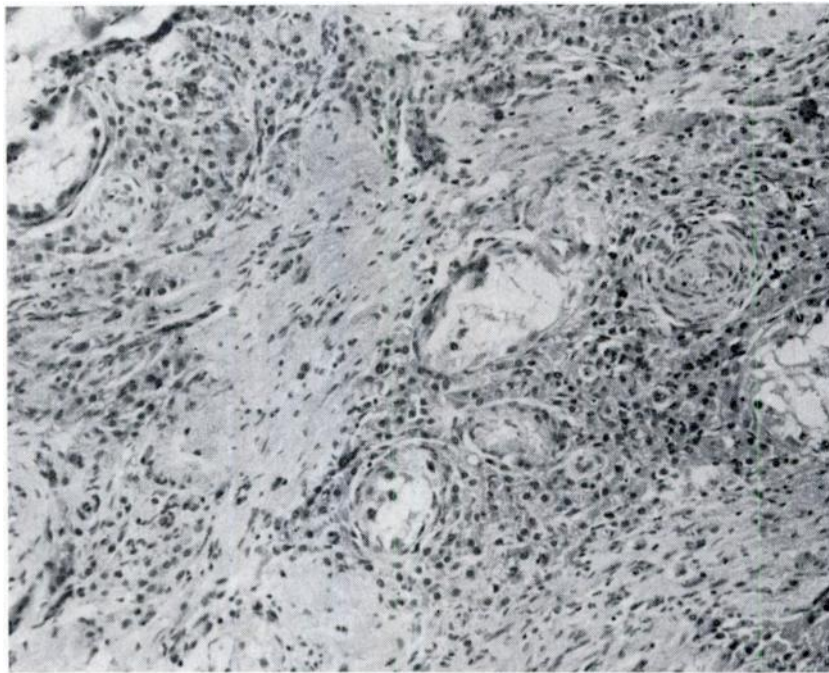


FIGURE 3. Connective tissue stroma among degenerative tubules.

thickening of basement membrane (Fig. 2) to necrosis with mineralization. Phagocytic activity of giant cells and fibrotic replacement occurred. In some tubules deterioration may have been secondary to stenosis followed by stagnation and mineralization of luminal contents. Early stenotic changes were present in some tubules containing abortive spermatogenic cells including some multinucleated cells. Qualitatively the process of degeneration appeared to be the same in both right and left testis. An analysis of ten microscope fields at 30X indicated that normal tubules were more common in the left testis (15 per 100 tubules examined) than

the right (6 per 100 tubules examined). This implied that the process is either more rapid or more advanced in the right testis.

Extensive fibrosis was present in the peripheral regions of the testes. Connective tissue stroma extended into the center of the specimen (Fig. 3). Moderate focal eosinophilia and local plasma cell accumulations were present. Interstitial tissue appeared to be moderately hyperplastic when compared to normal deer. Production of androgens was presumably adequate for antler maturation. Increased thickness of arterial walls apparently was part of the general tissue response.

Discussion

Testicular atrophy in white-tailed deer (*O. virginianus*) has been described by Taylor, Thomas and Marburger (1964, Am. J. Vet. Res. 25: 179-185) in which cellular proliferation was absent in the seminal epithelium and few scattered foci of Leydig cells were present. This condition was associated with failure of the antler to mineralize completely and to shed its velvet. Genital hypoplasia in white-tailed deer has been reported by Marburger, Robinson and Thomas (1967, J. Mammal. 48: 674-676) in which testicular interstitial tissue was well developed but tubules were lacking in seminiferous epithelium. Normal antler development occurs in the latter case.

The condition under consideration differs from both the hypoplasia and atrophy because of the presence of normal tubules, foci of eosinophils and extensive fibrous tissue replacement. The relative

Leydig hyperplasia present distinguishes the degenerative testis of this mule deer from the atrophic condition in white-tailed deer.

Testicular degeneration in domestic ungulates has a variety of causes but the changes in testicular morphology are essentially the same (Jubb and Kennedy, 1963). Common etiological factors include nutritional deficiency, epididymal obstruction and local or systemic infection. The good body condition and antlers which compared favorably in beam diameter and density with normal animals implied that the disorder was not nutritional. No microscopic evidence of obstruction nor abnormality of the epididymes could be noted. Focal eosinophilia and plasma cell accumulations suggest a chronic infection in the testes of this animal.

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