OCCURRENCE OF RUMENITIS IN A SUPPLEMENTARY FED WHITE-TAILED DEER HERD

Authors: WOOLF, A., and KRADEL, D.

Source: Journal of Wildlife Diseases, 13(3) : 281-285

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-13.3.281

OCCURRENCE OF RUMENITIS IN A SUPPLEMENTARY FED WHITE-TAILED DEER HERD

A. WOOLF, Research Director, Rachelwood Wildlife Research Preserve,
R.D. #1, New Florence, Pennsylvania 15944, USA

D. KRADEL, Animal Disease Laboratory, Department of Veterinary Science,
The Pennsylvania State University, University Park, Pennsylvania 16802, USA

Abstract: Rumenitis was observed with increased frequency in a herd of white-tailed deer (*Odocoileus virginianus*) maintained on a high carbohydrate supplemental ration. Healing rumen scars were found in 4.4% (n=225) of animals examined in 1973; 24.1% (n=278) in 1974; and 42.5% (n=308) in 1975. The lesions often involved nearly the entire ventral blind sac of the rumen. Histopathologic studies did not define the etiologic agent and invasion by either fungi or *Fusiformis necrophorus* was not a prominent feature in the cases examined. Recovery appeared to be complete and the disease was not considered to be an important herd mortality factor.

INTRODUCTION

Chemical rumenitis attributed to diet and sometimes complicated by secondary bacterial (*Fusiformis necrophorus*) and fungal agents often has been reported in domestic ruminants. This paper describes the occurrence of rumenitis in a large, captive, white-tailed deer (*Odocoileus virginianus*) herd. The problem may have been associated with a high level of supplemental grain feeding. However, only the paper by Wobeser and Runge[3] has described a similar syndrome in white-tailed deer.

METHODS

The herd's habitat was previously described by Woolf *et al.*[4] White-tailed deer harvested for herd control from September to December in 1973, 1974 and 1975 were brought to a central processing station for examination. Rumens were flushed with water and the entire organ examined for gross lesions. Representative lesions were preserved in 10% formalin and histologic sections prepared using standard techniques. The tissues were cut at 6 μm and stained with hematoxylin and eosin. Selected cases were prepared with Brown-Brenn stain for bacteria and Gridley's stain for fungi. The pH of rumen fluid was measured from a sample of 61 animals using a Bechman Electromate battery operated pH meter.

RESULTS

Prevalence

Rumens examined were classified as normal, rumenitis, or scars. Rumenitis implied the presence of active lesions, although in some cases, healed or healing scars also were present. Scars represented healed areas assumed to represent prior rumenitis. The prevalence of scars has significantly increased each year (Table 1). The prevalence of active rumenitis increased significantly in 1974 then declined in 1975, but nevertheless was present at a higher frequency than observed in 1973. Active or healed lesions were found in all sex and age classes with no significant differences in frequency of occurrence.

TABLE 1. Occurrence of rumenitis and rumen scars in white-tailed deer harvested at Rachelwood Research Preserve, 1973-1975.

	1973	1974	1975
Number examined	225	278	308
Rumenitis (%)	0.4	6.8	1.6
Scars (%)	4.4	24.1	42.5

Gross Description of Lesions

Lesions most frequently occurred in the posterior ventral blind sac, but did occur in all regions of the ventral sac. Only a few times were lesions found in the posterior dorsal blind sac. When viewed on the serosal surface, active rumenitis appeared as well-demarcated, circular, red, granular and dry areas ranging in size from 2 cm to confluent lesions involving nearly the entire ventral blind sac (Fig. 1). When viewed from the mucosal surface the areas were dry and hemorrhagic with the papillae either matted or denuded. Necrotic debris and forage particles frequently adhered to the involved mucosa (Fig. 2).

Healing or healed areas were irregularly circular, white, and contracted. Normal papillae were absent. Areas surrounding the frank scars either lacked papillae or the papillae were atrophied, flat, and white (Fig. 3). The areas of scarring could be detected through the serosal surface because of obvious thinness of the wall and a somewhat translucent appearance. Fibrous adhesions to the omentum and fibrous tags were present on the serosa of many cases.

FIGURE 1. Typical location and serosal appearance of active rumenitis found in white-tailed deer.

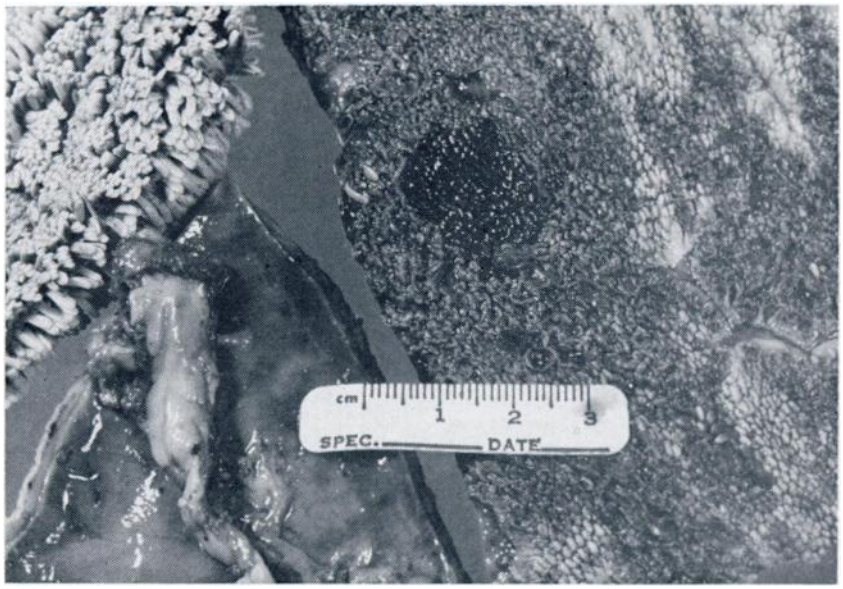

FIGURE 2. Mucosal surface with active rumenitis. Hemorrhagic, edematous serosa lower left, and normal mucosa top left.

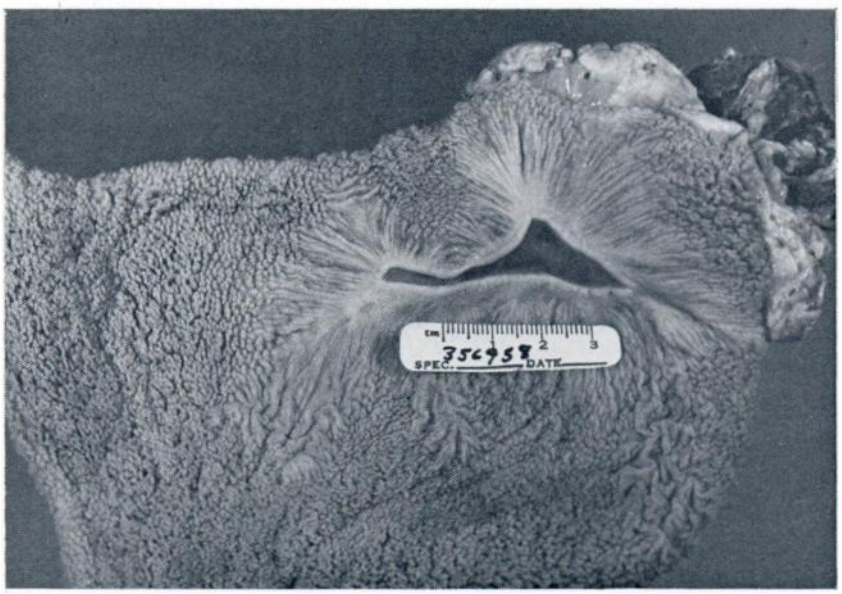

FIGURE 3. Healing lesion of rumenitis on mucosal surface of white-tailed deer rumen.

Histopathology

Mucosal vesiculation, epithelial denudation, fibrinohemorrhagic inflammation, necrosis, fibrous tissue proliferation, and occasional calcification were observed to varying degrees in the rumens. In severe cases the inflammatory changes occurred from the mucosa to the serosal surface. The inflammatory infiltrates were of mixed cell type and many eosinophils were present. In the deeper rumenal layers, inflammatory cells predominated along the connective tissue planes. Necrotic vasculitis and perivascular cuffing occasionally was observed.

No prominent bacterial or mycotic flora were observed. Mixed bacteria and rare mycelial elements were present. These were predominantly on the mucosal surface or in the superficial necrotic tissue. Bacteria morphologically suggestive of *F. necrophorus* or elements of typical mucormycosis were not present.

Rumen pH and Food Habits

The rumen pH of 61 deer ranged from 5.1 to 6.5 with a mean of 5.7. Values were from animals of all sex and age classes and time of sampling ranged from about 1000 h. to 1900 h. The samples included deer both with and without rumenitis. Because these were post-mortem determinations, correlation of rumen pH with time of feeding, diet, and presence of lesions was not considered appropriate.

Food habits determined by rumen content analysis were not obtained in 1975. Hubert[1] analyzed the rumen contents of 45 deer collected during the period September to November, 1973 and found 69.3% supplemental feed by volume in September (n=15) and 47.0% in October-November (n=27). In 1974 (unpublished Rachelwood data), respective values were 33.3% (n=10) and 40.7% (n=20). Visual estimates of per cent supplemental feed in the diet were made in 1975 and did not appear to differ from the previously determined values. The ingredients composing the supplemental feed has been published.[4]

DISCUSSION

While a clear relationship was not established between supplemental feed intake and prevalence of rumenitis in the herd, the high carbohydrate supplemental diet is highly suspicious as the primary cause of the observed lesions. Attempts were not made to correlate quantity or presence of supplemental feed in the rumen with prevalence of lesions since rumen content analysis only represent intake at one point in time. The supplemental feed is provided *ad libitum* year-round and food habits studies indicate a high degree of utilization.

Gross appearance of the active and healed lesions are similar to that described by Jubb and Kennedy[2] for chemical rumenitis complicated with *E. necrophorus* or various fungi (mucormycosis). On the other hand neither of these complicating agents were demonstrated in this study and the presence of many eosinophils is not typical of rumenitis as previously described.[2,3] Similarly, the liver lesions often associated with complicated rumenitis were not observed in these deer.

Eosinophils sometimes are associated with parasitic migration or tissue reaction in which histamine is released. In the authors' experience eosinophils are very common in the alimentary tract of ruminants and seem to respond to various inflammatory stimulants. Certainly none of these circumstances were established in this study, nor could they be eliminated.

The varying prevalence of active rumenitis observed each year is also unexplained. The supplemental diet presently used has been fed since June, 1972 and rumenitis was not observed until 1973. In support of the hypothesis of grain as a possible etiologic agent causing the rumenitis is the increased availability of feed through more feeders established after 1973. It is also not possible to eliminate the possibility that the increasing prevalence of rumen scars is due to the accumulation of deer who previously have experienced active rumenitis.

Rumenitis in domestic animals caused by grain ingestion is usually of minor importance and complete recovery is usually obtained.[2] Wobeser and Runge[3] believed that rumenitis-rumen overload could be a serious disease factor. In this study only one case was found with severe rumen perforation and resulting peritonitis that could have eventually led to the animal's death. However, the ultimate significance of a high prevalence of rumenitis in supplementary fed deer herds remains unknown and should be considered a potential problem in supplemental feeding programs.

LITERATURE CITED

1. HUBERT, G. F. JR. 1974. Food habits of a large, confined white-tailed deer herd provided with supplemental feed. M.S. Thesis. Colorado State University, Fort Collins, Colorado. 181 p.
2. JUBB, K. V. F. and P. C. KENNEDY. 1970. The Forestomachs. 2:50-64 In: *Pathology of Domestic Animals.* Academic Press. New York, New York.
3. WOBESER, G. and W. RUNGE. 1975. Rumen overload and rumenitis in white-tailed deer. J. Wildl. Manage. 39: 596-600.
4. WOOLF, A., D. KRADEL and H. ROTHENBACHER. 1976. Prevalence of renal urolithiasis in a large, captive white-tailed deer herd. J. Wildl. Dis. 12: 306-309.

Received for publication 8 June 1976