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MORTALITY IN CAPTIVE BIGHORN SHEEP — CLINICAL, HEMATOLOGICAL, AND PATHOLOGICAL OBSERVATIONS

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Abstract: The onset and course of disease in a captive herd of Rocky Mountain bighorn sheep *(Ovis canadensis)* were studied. Major clinical signs were diarrhea, persistent coughs, mucopurulent nasal discharges, loss of weight, poor pelage appearance, and delayed pelage shedding. Primary pathological findings in 16 of 17 deaths were related to pneumonia. *Mycoplasma arginini, Pasteurella* sp., and *Streptococcus* sp. were isolated and considered probable etiologic agents. Parasites were not considered to be a primary cause of the clinical signs or the lung disease observed. Hematological changes indicated the chronicity of the disease and had prognostic and limited diagnostic value. Amyloidosis was observed in seven animals, suggesting a high susceptibility of the bighorn to secondary amyloidosis.

INTRODUCTION

Objectives of this investigation were to document the onset and course of disease in captive bighorns, seeking clues to possible etiologic agents involved in the lung disease complex; and to determine if blood analysis had prognostic value or could be used to detect the presence of subclinical disease.

Preliminary data from the study have already been published.[7,9,10] This paper contains additional information and a summary of the major findings.

Eleven bighorns were placed in captivity in December 1968. Four lambs were born in 1969, three in 1970, and two in 1971. Data on the 17 deaths occurring during the period from December 1968 to October 1971 are listed in Table 1.

METHODS

The sheep were handled at quarterly intervals from April 1969 to January 1971 to obtain nasal swabs and jugular vein blood samples. Blood parameters were determined by methods previously reported.[9]

Serum samples were subjected to tests for neutralizing antibody to infectious bovine rhinotracheitis (IBR), bovine virus diarrhea (BVD), and parainfluenza 3 (PI3) viruses using standard methods. Isolates were cultured from nasal swabs of live animals, and from lung, liver spleen, kidney, and blood at necropsy. Thoracic fluid and abscesses were also cultured when present.

Fecal samples were examined for signs of parasitism at irregular intervals using sugar flotation and Baerman methods. Specific searches for parasites were also made at necropsy and microscopically.

RESULTS

Clinical Signs

Most clinical signs were previously reported.[8,10] An important clinical sign was periodic diarrhea. It occurred often in January 1969, subsided after several weeks, and then returned with moderate intensity from April to June 1969. Although most sheep suffered from the periodic diarrhea, it was most frequent and severe in only three animals (1, 4, and 7).

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TABLE 1. Primary pathological findings in captive bighorn sheep.

Animal Number	Sex/ Age	Time in Captivity (days)	Primary Pathological Findings
1	F-A*	195	Pneumonia, lung abscesses, lungworms, focal hepatic necrosis
2	M-A	440	Pneumonia, pulmonary edema, pleuritis, necrotic glomerulitis, mycardial necrosis, enteritis, focal hepatitis, amyloidosis
3	F-A	303	Pneumonia
3A	M-A	865	Pneumonia, lung abscesses, amyloidosis
4	F-A	764	Pneumonia, lung abscesses, pleuritis, pericarditis, amyloidosis
4A	M-A	858	Pneumonia, lung absecesses, amyloidosis
4B	F-L	11	Pneumonia, *E. coli* enteritis (presumptive)
5	M-A	485	Pneumonia, amyloidosis
6A	F-L	73	Pneumonia, pleuritis, myositis (from injections?)
6B	F-L	144	Pneumonia, hepatic necrosis, biliary hyperplasia, skeletal muscle degeneration
7	F-A	200	Colitis, pneumonitis, lungworms, focal hepatitis, nephritis
8	F-A	391	Pneumonia, lung abscesses, nephritis
9A	M-L	69	Pneumonia, mucoid enteritis, nephrosis
9B	M-L	41	Pneumonia
9C	F-L	168	Pneumonia, amyloidosis
10	F-A	399	Pneumonia, lung abscesses, pleuritis, nephrosis, amyloidosis
11	F-A	416	Pneumonia, pleuritis, focal hepatic necrosis

* M = male; F = female; L = lamb; A = adult

For several days prior to death, diarrhea was severe in three of four lambs that died (4B, 9A, 9B) and in adults 1, 2, and 7. Diarrhea was not present in other sheep prior to death, and only occurred intermittently and for short durations in some surviving animals after January 1970.

Bacteriology and Virology

Bacterial isolates obtained from nasal swabs and lung tissue are listed in Table 2. Isolates obtained from other tissues were generally inconsistent, similar to those obtained from lung, or negative.

Two attempts to isolate viruses from lung material and other organs were unsuccessful. All serum samples tested were negative for IBR antibodies. Animals 7 and 10 had titers to P13, Animals 2, 5, 7, 10, 11, 3A, and 4A had titers against BVD. Antibody to BVD were present in all samples taken from July 1969 to April 1971 from sheep 3A and 4A. Animal 5 had antibody titers from April 1969 to February 1970. The other sheep with antibody titers had only one or two positive tests in April, August, or September 1969.

TABLE 2. Organisms isolated from living and dead bighorns.

Isolate	Found in Bighorns Number	
	Nasal Swabs	Lungs
Mycoplasma arginini	1A, 3, A3, 4, 4A, 5, 6, 8, 9, 10,11	1, 3, 3A, 4, 4B, 5, 6A, 6B, 8, 9B, 9C
Bacillus sp.	1A, 3A, 4, 4A, 5, 6, 9, 11	9A
Candida sp.	3	———
Corynebacterium sp.	4, 4A	2
Diplicocci	3A, 4A, 9	4, 4B
E. coli	1A, 2, 3A, 4, 4A, 8, 9, 10	3, 3A, 4A, 4B, 6B, 8, 9A, 9B
Klebsiella sp.	4A, 9	8
Mima sp.	1A, 3A, 6, 9	———
Neisseria sp.	1A, 3A, 4, 4A, 5, 9	2, 4A, 5
Pasteurella sp.	———	2, 5, 4A, 4B, 8, 10, 11
Proteus sp.	———	6A, 8
Pseudomonas sp.	4, 4A, 5, 6, 9	6A, 6B, 9A
Serratia sp	———	9A
Shigella sp.	———	9A
Staphylococcus sp.	1A, 3A, 4, 4A, 5, 6, 9	9B
Streptococcus sp.	1A, 2, 3, 3A, 4, 4A, 6, 8, 9, 10	1, 2, 3, 4, 6A, 8, 10, 11

Hematological Changes

Initial blood data from seven of these sheep was reported.[9] The blood parameters from 11 sheep measured after a period of chronic illness are listed in Table 3.

The progressing disease complex in the bighorns was accompanied by a leukocytosis, neutrophilia, lymphopenia, and an eosinopenia. A second hematological trend was the anemia characterized by a decreasing packed cell volume and hemoglobin with no discernible trend in the red cell count. These changes resulted in a decreasing mean cell volume (MCV) and mean cell hemoglobin (MCH), and a relatively constant mean cell hemoglobin concentration (MCHC). Another hematological trend observed was a decreasing serum albumin and an increasing serum globulin resulting in a progressively decreasing albumin/globulin (A/G) ratio.

In these bighorns, the pneumonia complex was probably primary and the hematological changes indicated a continuing course with a poor prognosis. Only animal 7 died from causes other than the pneumonia complex, but blood parameters from that ewe did indicate the presence of chronic illness.

Pathological Changes

The major pathological findings are summarized in Table 1. Lung lesions observed in all cases were similar to those previously reported for five bighorn deaths.[10] The changes can be generally described as a chronic bronchopneumonia with suppuration.

Amyloidosis was observed in seven sheep (41%). The organs involved included the liver, kidneys, spleen, adrenals, heart, and rumen. In some cases, the presence of amyloid was suggested by

TABLE 3. Hematological data from 11 captive bighorns dying after chronic illness.*

Parameter	Animal #2 Sample 10/15/69 Died 2/19/70	#3 9/5/69 10/14/69	#3A 4/20/71 9/20/71	#4 10/13/70 1/8/71	#4A 4/20/71 9/19/71	#5 2/19/70 4/4/70	#6A 7/22/70 8/14/70	#7 6/23/69 6/23/69	#8 10/15/69 12/31/69	#10 10/15/69 1/8/70	#11 10/15/69 1/25/70
WBC**	10,616	14,009	23,500	18,800	30,650	22,974	17,900	12,273	11,197	19,349	12,134
RBC ($x10^6$)	11.20	11.12	6.01	9.67	5.45	9.96	11.87	8.78	11.90	10.60	11.16
MCV (μ^3)	40	39	67	29	65	42	47	49	42	41	44
PCV (%)	45	44	40	28	36	42	55	43	50	43	49
MCHC (%)	39	35	35	33	34	34	24	35	37	36	34
Hb (g/100 ml)	17.4	15.2	13.8	9.1	12.4	14.4	13.2	15.2	18.5	15.2	16.8
MCH ($\mu\mu g$)	15	14	23	10	23	14	11	18	16	14	15
Segmented cells %	81	78	65	72	59	87	74	—	78	64	63
Lymphocytes %	18	16	26	17	25	12	20	—	18	32	35
Band cells %	0	0	1	0	8	1	3	—	1	0	0
Monocytes %	1	3	5	0	1	0	3	—	1	4	2
Eosinophils %	0	4	2	0	12	0	0	—	1	0	0
Basophils %	0	0	1	3	3	0	0	—	0	0	0
T. Protein (g/100 ml)	6.6	6.4	6.9	8.4	5.4	5.8	9.2	10.9	8.7	8.1	7.4
Albumin (g/100 ml)	2.6	4.0	2.7	2.7	2.1	2.5	7.8	4.1	3.1	3.4	3.4
Globulin (g/100 ml)	4.0	2.4	4.2	5.7	3.4	3.8	1.4	6.8	5.6	4.7	4.0
A/G ratio	0.7	1.7	0.6	0.5	0.6	0.5	5.6	0.6	0.6	0.8	0.9

* Initial blood values for 7 of these bighorns were reported by Woolf and Kradel (1970).
**WBC = white blood cells
RBC = red blood cells
MCV = mean corpuscular volume
PCV = packed cell volume
MCHC = mean corpuscular hemoglobin concentration
Hb = hemoglobin
MCH = mean corpuscular hemoglobin

swelling and pallor of the kidneys and liver, but in most tissues the presence was detected only histologically.

Enteritis was diagnosed post mortem in four of six cases in which diarrhea was a terminal sign. No evidence of enteritis was found in the other cases in spite of careful gross and microscopic examination of the gastrointestinal tract. Acid-fast stains were applied to intestinal sections from all animals with a history of diarrhea and no evidence of Johne's disease was found.

DISCUSSION

Diarrhea was a good indicator of illness in these animals and apparently signaled the presence of other disease, particularly in lambs. However, it was observed mainly as a periodic clinical sign and an etiologic diagnosis was not made at necropsy.

Cultures of fecal material resulted in inconclusive and inconsistent isolates of *E. coli* and *Proteus* sp., both in living and dead animals. Parasites, both nematodes and coccidia, were detected in fecal examinations, but the level of parasitism remained rather constant during either presence or absence of diarrhea. Examination of histological sections did not disclose lesions associated with frank parasitism.

The titers against BVD antibody found in some serum samples were in contrast to the absence of diagnostic lesions in the alimentary canal. These results are inconclusive and BVD can neither be incriminated nor ruled out as a possible cause of the diarrhea.

Prophylactic and treatment therapy with antibiotics can readily induce diarrhea in ruminants. Although extensive therapy was applied to these animals, it followed the onset of diarrhea observed early in 1969 and we do not believe it was a causative or contributing factor.

The causes of diarrhea are manyfold and include metabolic disturbances complicating indigestion, especially in ruminants.[6] The diarrhea in these animals may have been a manifestation of this. Low-grade parasitism and the presence of opportunistic bacteria may have been complicating agents.

Hematological changes observed were a general response characteristic of various infections.[2,7] The hematological data was too variable and sampling periods too far apart to determine with accuracy the onset of clinical disease. It is also recognized that the observations served only to better define the state of the subjects at a point in time and that many diseases and physiological aberrations may produce similar changes. However, the data did demonstrate that major changes in blood components can be useful in detecting a pathological process in bighorns and may serve as a relatively good indicator of disease course and prognosis. Also, these findings support previous reports of assumed normal hematological values for bighorns.[3,4,9]

Although normal blood parameters for bighorns still do not have well-defined ranges; a PCV below 48%, hemoglobin below 16g, a WBC count greater than 12,000, a differential count of neutrophils exceeding 65%, and an A/G ratio less than 0.9 might indicate that the animal was suffering from chronic disease. Blood data evaluated on this basis combined with visual signs could be applied to determining the condition of animals captured for transplant operations or confinement for investigations.

The etiology of the lung disease observed in these animals was not determined. The effects of bacteria in pneumonic conditions of animals are always difficult to evaluate. The frequent isolation of *Mycoplasma arginini*[1,10] from nasal swabs and lung tissue appears significant, but the role in the respiratory complex of bighorn sheep remains uncertain. *Pasteurella* sp. and *Streptococcus* sp. were also isolated with frequency and would appear to have played some role in this complex. Other isolates were most likely frank opportunists or contaminants.

These data indicate that at least three potential bacterial pathogens were involved, separately or synergistically, in the pneumonia complex. Whether these bacterial agents are capable of producing pneumonia in the absence of other pathogens, such as viruses, remains to be determined.

It should be reported that these bighorns were maintained in close proximity, but not direct contact, with mouflon sheep and bighorn-mouflon hybrids. What effect, if any, this may have had on the etiology of the disease complex is unknown. The mouflon and hybrid sheep did suffer from the same disease, but not with the frequency observed in the bighorns.

The role of lungworms did not appear significant. Their possible role as a portal of entry for an etiologic agent cannot be denied, but they were not considered significant in the pathogenesis of the disease observed. Lungworms were not observed in many of the lungs and when present, the location of lungworm lesions in the diaphragmatic lobes was in contrast to the primary involvement of the anterior lung lobes.

The amyloidosis frequently observed was considered to be secondary to chronic inflammatory and suppurative processes. However, its occurrence does suggest an unusual susceptibility of bighorns to amyloid formation as previously reported.[5]

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