THE IMPLICATIONS OF CONTAGIOUS ECTHYMA IN BIGHORN SHEEP

by

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Abstract

The documented occurrences and clinical signs of contagious ecthyma in the bighorn sheep of North America are reviewed. The significance and possible consequences of this disease upon individual animals and herd mortality patterns are discussed.

INTRODUCTION

Contagious ecthyma (CE) has long been recognized as a common and ubiquitous disease of domestic sheep. It is well known in the sheep industry of the intermountain regions of western North America and sheep ranchers refer to CE by such terms as sore mouth, scab mouth, or simply, scab. CE is produced by a large DNA virus of the parapox group. The virus predominately affects epithelial tissues lining the oral cavity and the external genitalia. An important characteristic of the CE virus is its ability to persist in the environment in shed scab material. Dried scab material has been found to be infective after 20 years (Livingston and Hardy, 1960).

REPORTS OF CONTAGIOUS ECTHYMA IN BIGHORN SHEEP

CE in bighorn sheep was first documented in the herds of Banff National Park, Alberta, in the Spring of 1953 (Connell, 1954). Cases were seen in the sheep bands of the national parks of western Alberta, eastern British Columbia, and Glacier National Park of northwestern Montana in the following years (Blood, 1971). The extent of the problem in this region was further elucidated by Samuel *et al* (1975). Many of the cases involved bighorn lambs so severely affected that they were destroyed by field personnel.

During 1975, the loss of 5 of 6 lambs and 1 adult ewe from a herd of bighorn sheep in an enclosure in the Lava Beds National Monument of northern California was attributed to CE (Blaisdell, 1976). Serological evidence from southern California indicates that CE is present in the bighorn sheep of the Santa Rosa Mountains (Payson, 1977), although clinical cases have not been documented.

An outbreak of CE was confirmed in a pen of captive Dall sheep (Ovis dalli dalli) in Alaska (Nieland, 1978) and serological results incicate the disease is also present in some of the free-ranging herds (Zarnke, 1980).

During the 1978 hunting season, 7 of 20 rams taken from the Saguache herd of southern Colorado had active clinical lesions of CE. These rams ranged in age from 3 to 7 years. Although no active lesions were seen in 17 rams harvested from this same herd in the 1979 hunting season, 20 of 29 from a ewe-lamb group trapped during January, 1980 had clinical CE (Lance, unpublished data).

Following an outbreak of scabies in the desert bighorn sheep (Ovis canadensis mexicana) of the San Andres refuge in southern New Mexico, CE was confirmed in a group of 33 of these sheep that were captured, dipped in toxaphene, and confined in a large enclosure (Langa, unpublished data).

CE in bighorn sheep is characterized by large (1cm) proliferating growths on the oral mucosa, around the base of the incisor teeth, and over the hard palate. Ulcerated areas also are common on the hard palate and the dorsum of the tongue. Elevated lesions covered with a dried crusty exudate may be present over the nostrils, the mucocutaneous junction of the mouth, and around the eyes. Dried crusts on the eyelids may produce eye lesions through mechanical damage to the cornea. Active lesions have been seen around the horn base of a hunter-killed ram with clinical CE (Lance,

unpublished data). Elevated lesions covered with a crusty exudate may be produced on the external genitalia of both rams and ewes; moreover, ewes may have similar lesions on the teats and udder. Secondary bacterial infections and accompanying complications are frequent.

The lesions of CE develop rapidly and reach a maximum size in three to four weeks. In uncomplicated cases, healing is usually complete in six weeks. As the disease develops in bighorn sheep, intense itching is evident because affected animals constantly rub the lesions against objects or other sheep in an effort to relieve the irritation. Once the lesions have healed in bighorn sheep, small irregular shaped white scars may persist on the muzzle and the mucocutaneous junction for at least six months (Lance, unpublished data). If a reasonable number in a band of sheep are examined, these white scars may be used as a field aid to indicate if CE has been active recently.

IMPACT OF CE ON BIGHORN SHEEP

The first management consideration of CE is the effect on individuals. The primary viral lesion of the mouth and the accompanying secondary bacterial infection may be sufficiently severe to prevent young lambs from feeding normally. Lactating ewes, with painful udder and teat lesions, may not permit lambs to nurse. Although not documented, genital lesions could impair breeding in both rams and ewes.

As a rule, lesions seen in bighorn lambs are much more severe than those in adults. Death in lambs most likely would be due to complications arising from inadequate nutrition secondary to the extensive oral lesions. Although adult mortalities from the primary or secondary effects of CE have been seen, this is the exception rather than the rule. But, if the individual sheep is presently under the stress of other environmental or disease agents, CE may be the final

mortality factor. In harvested rams, the CE lesions may produce a disfigured trophy, esthetically displeasing to most hunters. In herds where CE is known to exist, hunters should be advised to handle diseased animals with caution because CE is transmissible to man and can produce painful, persistent, skin lesions.

The impact of CE on the herd, and its mortality pattern, will vary with the herd age structure, the immune status of individuals, the seasonal social patterns of the sheep, and the presence of other active or latent mortality agents. If CE becomes active after the previous years' lamb crop is 9 to 10 months of age, and no other detrimental environment associated factors or disease agents are present, mortality is likely to be limited. But, if a CE outbreak occurs shortly after lambing season and factors are present which may depress the immune status of individuals (stress due to intraspecific factors, environmental stress, etc.), a considerable loss of lambs may occur.

Presently, the exact distribution of CE in the bighorn sheep herds of North America is unknown. Additionally, questions relating to the mechanisms of the epizootiology of CE outbreaks, such as the one in Colorado, remain to be answered. Was the virus recently introduced or has it been latent in the environment? If it was latent, by what mechanisms did it become active? Once the disease becomes active in a herd, will it continue to be so indefinitely? How is the CE virus in bighorn sheep related to that in domestic sheep? Was the virus active in bighorns on their native range prior to introduction of domestic sheep?

Interestingly, the occurence of CE in northern California, Alaska and New Mexico involved animals in enclosures and/or those recently captured. The Colorado epizootic occurred in the largest herd in the state. This herd is known for its large bands of sheep which congregate in rather limited

range. This outbreak also followed two years of dry range conditions. The occurrences in Alberta were proposed to be associated with animals congregating around salt blocks.

At this time, it appears that the reports of CE in bighorn sheep in recent years have been associated with animals maintained in artificial enclosures, artificially congregated, or, as in the New Mexico case, stressed by trapping, and then put into enclosures. In the Colorado outbreak, the disease occurred in a herd that some management personnel judged to be reaching the limits of habitat and range. It is possible that CE may be an indicator disease of a long term low level stress condition in a population, and symptomatic of other more basic problems in the condition of the herd and their environment.

Literature Cited

Blaisdell, J.: 1976. The Lava Beds bighorn- so who worries. Desert Bighorn Council, 1976, Trans, 50.

Blood, D.: 1971. Contagious ecthyma in Rocky Mountain bighorn sheep. J. Wildl. Mgmt., Vol. 35, No. 2, pp. 270-275.

Connell, R.: 1954. Contagious ecthyma in Rocky Mountain bighorn sheep. Can. J. Comp. Med., Vol. 18, No. 2, pp. 59-60.

Lance, W.: 1980. Unpublished data. Wild Animal Disease Center, Fort Collins, Colorado.

Lange, R.: 1980. Unpublished data. New Mexico Game and Fish, Las Cruces, New Mexico.

Livingston, C.W. and W.T. Hardy: 1960. Longevity of contagious ecthyma virus. J. Am. Vet. Med. Assoc. 137: 651.

Neiland, K.A.: 1978. Contagious ecthyma: A potential wildlife disease problem in Alaska. Alaska Dept. of Fish and Game, Fairbanks, 20 pp.

Payson, J.: 1977. A disease study of free-ranging desert bighorn sheep in the Santa Rosa mountains, Riverside County, California. M.S. Thesis. Univ. of Wyo. 41 pp.

Samuel, W.M., G.A. Chalmers, J.G. Stelfox, H. Loenen and J.J. Thomson: 1975. Contagious ecthyma in bighorn sheep and mountain goat in western Canada. J. Wildl., Dis. 11: 26-31.

Zarnke, R. and K. Neiland: 1979. Research progress report. Serological surveys for natural foci of contagious ecthyma infection. Big game investigations. Alaska Fish and Game, Fairbanks.

QUESTIONS - RESPONSES

Wayne Heimer: I tell you just a comment or two about the history, I've never written about it because I'm not allowed to write about it. There was as you said, a real biological fight and I hung out on the side of the bighorns, not knowing any better. We were told that it would kill sheep like dynamite and 2 of the lambs in the pen up there did die. Things progressed, so we decided we better see what's going on in the wild. We scratched up some old serum from our low quality study area and we went out and got some new. We captured a bunch of sheep from the high quality study area and we had found that it is in our wild sheep. It is present in the high quality study area at a greater frequency, with our small sample sizes, than it is in the low quality population. Neither of those population should have had contact, in recorded history, with domestic sheep.

Bill Lance: This is something Daryll Hebert alluded to in a conversation regarding CE in goats.

Wayne Heimer: While trapping this summer we did catch one ewe with active lesions on her udder which have been subsequently identified as CE. I'm interested in how much different it is the different strain that you have. It doesn't seem to bother except for two lambs in the pen, our sheep all that much. Our highest productive herds have more of it than our lower ones.

Bill Lance: I think the key to this is, that once it is introduced into a herd; you know you start developing some immune individuals and they become exposed, there may be some immunity through the colostrum. This is something that we're going to try to answer later with some sheep we now have in captivity. I think the real problem is throwing this virus into a herd that's never had any history of exposure to it. I wouldn't expect it to be a herd mortality factor year after year after year, but I think if you throw it in cold your going to lose some sheep.

Daryll Hebert: Bill, I was wondering if anybody knew whether this CE virus may be transmitted aerially, or does it have to be direct contact?

Bill Lance: I'm sure it can be transmitted aerially. It doesn't take much to get this virus going, but once it gets going in a group of sheep it goes like wildlife. I put it in a group last year and within 16 days everybody had it. You watch sheep, they rub on each other and really get close.

Wayne Heimer: Another thing that may be of interest is that our disease people up there have tried infecting a caribou calf, I think and a moose calf. They haven't been able to kill them, but I think they are satisfied that they might have grown some lesions.

Bill Lance: This Sawatch strain, we've got it to grow in mule deer, whitetail deer, pronghorn and elk. I tried to get it to go into a bunch of people. We had 20 infected animals under the net and I didn't bother to mention that it is contagious to man. After it was all over I offered them all a steak dinner if anybody would come up with decent lesions. Nobody took me up on it.