EPIDEMIOLOGY AND PATHOLOGY OF HEMORRHAGIC DISEASE IN FREE-RANGING BLACK-TAILED DEER IN CALIFORNIA: 1986-1987

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Abstract: Outbreaks of hemorrhagic disease occurred in blacktailed deer (*Odocoileus hemionus columbianus*) in California in 1986 and 1987. The 1986 outbreak affected deer in Trinity, Tehama, Mendocino, Humboldt, Siskiyou, and Modoc counties, and bluetongue virus type 10 was isolated from one deer in Siskiyou County. The 1987 outbreaks were limited to Lake County and no Orbiviruses were isolated from deer. Gross and microscopic lesions of deer carcasses as well as epidemiologic patterns from both outbreaks were suggestive of bluetongue or epizootic hemorrhagic disease. Deer losses could only be estimated and were not reflected in buck kill, fawn to doe, or buck to doe ratios for reasons described.

Bluetongue (BT) and Epizootic Hemorrhagic Disease (EHD) viruses, closely related members of the genus *Orbivirus*, cause a disease syndrome in white-tailed deer (*Odocoileus virginianus*), black-tailed deer, mule deer (*O. hemionus hemionus*), and pronghorn antelope (*Antilocapra americana*) often referred to as "Hemorrhagic Disease". The term Hemorrhagic Disease (HD) is descriptive of the general pathology seen in animals afflicted with either BT or EHD and epitomizes the difficulty in distinguishing between lesions caused by these viruses. In California, it appears that HD has been endemic for several decades. The first documented evidence of a HD-like syndrome in California was the loss of numerous deer in Modoc County in 1924 by what was at the time thought to be either starvation or foot and mouth disease (Moffitt 1934). This syndrome was later labelled "Modoc Mud Disease" and was attributed to footrot from sheep. Rosen et al. (1951) reviewed footrot in deer and reaffirmed a bacterial etiology although they were never able to reconcile seasonal and pathologic disparities between footrot of sheep and deer. Upon retrospective examination, it appears that the disease described by earlier authors was HD. Indeed, in 1953, shortly after the publication of Rosen's paper, BT virus was isolated in California (McKercher et al. 1953) and since then, 4 serotypes of BT (10, 11, 13, 17) and 2 serotypes of EHD viruses have been isolated from livestock and wildlife in the state (Stott et al. 1985, Jessup 1985).

Outbreaks of HD in wildlife are thought to occur primarily during the late summer and fall when the vector *Culicoides variipennis* is most active (Stott et al. 1985). HD outbreaks in wildlife tend to be sporadic and can be influenced by weather, drought, vector abundance, virus pathogenicity, host densities and reservoir patterns. Further complicating the picture is a lack of consistent pathological signs in animals afflicted with this disease, coexisting viral infections, secondary bacterial infections, and difficulty in isolating orbiviruses.

In 1986, many deer from Siskiyou, Modoc, and Mendocino County were found dead in the span of three months from lesions resembling those of HD. Tissue samples were taken from 17 of these animals and examined. Mortalities were first reported in August in western Tehama County. In late August, deer were found dead in southern Mendocino and Trinity Counties. Losses continued into September and October extending northward up to Humboldt and Siskiyou Counties and eastward across Siskiyou County and the western edge of Modoc County. Locations of deer carcasses (Fig. 1) were recorded and some were perfunctorily examined as decomposition was too advanced to obtain any meaningful information.

From late August through September of 1987, an unusually high number of black-tailed deer carcasses and sick deer were observed near Clearlake in Lake County, California. Carcasses were initially found in northwestern Lake County and the last mortalities were observed in southeastern Lake County (Fig. 2). A majority of the carcasses were found near Borax Lake, an alkaline lake known as a heavy breeding ground for *C. variipennis*. Gross necropsies were done on 18 deer. Tissue samples were taken from four of these animals. The purpose of this paper is to describe the general pathology, epidemiologic pattern, and response of black-tailed deer populations to HD epizootics in the fall of 1986 and 1987 in northwestern California.

METHODS

Representative tissues were taken in 10% buffered formalin, allowed to fix for at least five days, embedded in paraffin, sectioned at 8 microns,

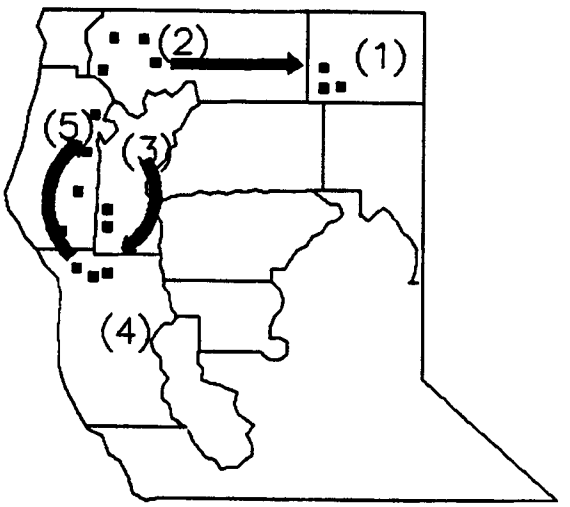

Fig. 1. Northern California counties listed as follows: 1) Modoc, 2) Siskiyou, 3) Trinity, 4) Mendocino, and 5) Humboldt. Arrows indicate the time sequence during which carcasses were found and squares indicate appproximate location of carcasses.

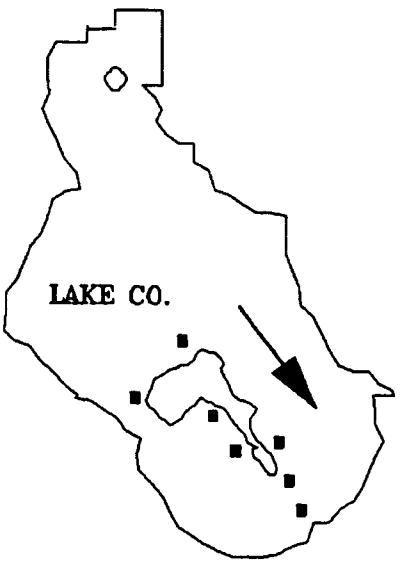

Fig. 2. Lake County. Squares indicate approximate location of carcasses around Clear Lake. Arrow indicates the time sequence during which carcasses were found.

stained with H&E and examined microscopically. Serum, spleen, and bone marrow were collected from relatively fresh carcasses for virus isolation procedures. Virology was done at the California Veterinary Diagnostic Laboratory System and entailed intravenous injection of 10-day old embryonating chicken eggs with a 1:10 dilution of sonicated red blood cells washed in 2% penicillin and streptomycin. If embryo death ensued, the embryo was sub-passaged into Vero monolayer cell cultures, and cytopathic effect read at 7 days. Sera were assayed for antibody to EHD virus (EHDV) and BT virus (BTV) using the agar gel immunodiffusion (AGID) test.

Deer carcasses were counted and losses roughly estimated. California Department of Fish and Game records were examined and buck kills for 1984-88 were compared in each of the affected areas. Additionally, buck to doe and fawn to doe ratios were compared by area and deer herd for these years.

RESULTS

Clinical Signs

All deer affected with HD had similar clinical signs reported by field biologists and local ranchers. These included swollen eyes, attempts to avoid direct sunlight, apparent blindness/disorientation, ataxia, respiratory distress with excessive salivation, foam exuding from the nasal passages and mouth and lack of fear of humans. Most animals affected during both the 1986 and 1987 outbreaks were yearling females and fawns.

Gross Pathology

The most common lesions encountered in carcasses of deer found in Lake County, in 1987, were serous fluid in the thoracic cavity, froth in the trachea, edematous lungs with hemorrhage on the pleural and parietal surfaces, petechiae and echymoses of the epicardium and medial necrosis and hemorrhages of the large elastic arteries (aorta and pulmonary). Often, the thoracic cavity had moderate to large amounts of clear yellow serous fluid. Other lesions noted but not seen with regular frequency were abdominal and pericardial fluid, petechiae in skeletal musculature, and rarely, oral and ruminal ulcers.

In contrast, of 18 deer from a five county area (Siskiyou, Modoc, Mendocino, Trinity and Humboldt) necropsied in 1986, oral necrosis was the most consistent gross lesion closely followed by hemorrhage of the large elastic arteries and heart. Abdominal fluid was present in a number of cases. Most of the deer in both locations were thin and

some were emaciated. Levels of internal and external parasites were similar to those of healthy deer collected in other locations.

Histopathology

The most commonly affected organ in animals from the 1986 outbreak (17 of 18 deer tissues examined) was the heart. Most animals had hemorrhage in the wall of the tunica media of the large arteries with infiltration of neutrophils, perivascular cuffing and inflammation. In rare instances, there was necrosis of the heart muscles. The next most commonly affected organ (12 of 18 samples) was the spleen which demonstrated congestion of the red pulp, and hyperplastic periarterial lymphoid sheaths the centers of which were infiltrated with vacuolated macrophages. Ten of 18 animals had microscopic lesions in the liver ranging from acute multifocal necrosis to hepatic/periportal fibrosis and extramedullary haematopoeisis. Eight of 18 animals had tongue lesions consisting of mucosal and gingival necrosis, perivascular lymphocyte infiltration of the submucosa and swelling of capillary endothelium. Other assorted lesions included mineralization of the salivary glands, petechiae in the skin, and pulmonary congestion, edema and necrosis.

Tissues from the 1987 Lake County outbreak were not examined microscopically.

Virology/Serology

Twenty blood samples were collected in 1986 (12 from Mendocino County, one from Humboldt, and seven from Siskiyou). Of these, three were serologically positive for BTV and EHDV (one from each county) and one each from Siskiyou was positive for BTV and EHDV. BTV-10 was isolated from one animal in Scott Valley (Siskiyou County). Follow-up studies in 1987 revealed 4/35 sera from Siskiyou positive to both EHDV and BTV, 3/35 positive to EHDV only and 1/35 positive for BTV only. One serum from Mendocino was positive for both EHDV and BTV. Isolation attempts for BTV were negative for 12 samples. One of 19 samples collected in Lake County in 1987 was seropositive for BTV and 4/11 for EHDV. No BTV was isolated on 19 attempts and none were made to isolate EHDV. No follow-up samples were collected in 1988. Serology for the northern counties outbreak in 1986 was also sparse and non-descriptive.

Epidemiology

Buck kill for both areas (Lake and the northern counties) during and after the outbreaks were not reduced significantly from other seasons. Similarly, fawn to doe and buck to doe ratios did not differ from other years and other areas in the affected counties both before, during and after the outbreaks.

DISCUSSION

The lesions observed in the deer in 1986 and 1987 are consistent with those described in white-tailed deer experimentally infected with EHDV or BTV. Howerth et al. (1988) noted that the most commonly encountered lesion in deer linfected with BT was necrosis of the oral cavity and hemorrhages seen most often in the abomasum, oral cavity and bladder. Vosdingh et al. (1968) most commonly observed subendocardial hemorrhages, enteritis and lingual hemorrhage in white-tailed deer infected with BTV. Karstad et al. (1961) found that hemorrhage of the myocardium and aorta was were the most common lesion in white-tailed deer infected with EHD. Among these three different experiments, no pathognomonic lesions for BT or EHD in deer could be identified.

The black-tailed deer we studied in 1986 and 1987 all demonstrated one or more of the previously described lesions, however, the frequency of occurrence of a particular lesion differed. None of the deer we necropsied demonstrated hoof lesions, and deer in Modoc and Siskiyou Counties most commonly had oral necrosis followed by hemorrhage in the large arteries while the most common lesion observed in the Lake County deer was hemorrhage of the large elastic artery and pulmonary edema. These difference may be attributable to different viral strains having variable tissue tropism and pathogenicity. Alternatively, these differences could also be an artifact of sampling and may not be reflective of what actually occurred. It appears however that the pathogenesis of the disease in white-tailed deer and black-tailed deer is similar in that both viruses attack vascular endothelium leading to multifocal thrombosis of vessels, hypercoagulation, and DIC (Tsai and Karstad 1973, Howerth et al. 1988). Widespread hemorrhage is a common sign of HD as was the case in our animals. It would be of little surprise that a disease that

degenerates vascular endothelium would affect a wide variety of organ systems and present itself in a variety of forms. It is impossible at this point to determine whether EHDV or BTV were responsible for these outbreaks or whether the difference in preponderance of clinical signs resulted from infection by different viruses.

The lack of virus isolation from our samples could be due to several reasons. The carcasses we sampled were often in advanced stages of degeneration. Bluetongue virus and EHDV do not survive in meat at pH of less than 6.0 (Owen 1964). It is likely that carcass autolysis resulted in acidification of flesh leading to inactivation of the virus. Less likely is that the systems used to isolate virus from the carcasses were inadequate. Although embryonating chicken eggs are (after sheep inoculation) the most sensitive media from which to isolate BTV and EHDV, there have been cases of these viruses not adapting easily to ECE directly from field samples. A third possibility is that the lesions observed were caused by some other virus or infectious agent.

Some of the deer with lesions of HD were malnourished. In the coastal mountain range of California, late summer and early fall are especially trying times for deer populations due to nutritional stress, as most green feed disappears by June. In years of poor rainfall, deer concentrate around remaining water sources. These warm fecal contaminated waters make ideal breeding habitat for the vector of BT, *C. variipennis*. The postulated ecological cycle of HD in California is that many susceptible and reservoir hosts (domestic cattle) gather around water holes, particularly during the dry seasons. *Culicoides variipennis* breed in these water holes and their presence along with the suitable hosts results in effective dissemination of BT and/or EHD. Trapping for *C. variipennis* in some of the affected counties (Mendocino, Modoc, Siskiyou, Lake) in 1987 revealed presence of the vector in all counties. Virus isolation attempts for BTV and EHDV on 517 pools of 20 insects each were negative. The presence of these insects in the outbreak areas does signal the potential for transmission of orbiviruses.

It is impossible to know the number of deer that died during the fall of 1986 and 1987; however, examination of California fish and Game records for buck kill by county for 1984-88 reveals a steady decline in numbers for numerous northern California counties. Specifically, a decline in buck kill is evident between 1986 and 1987, however it is tenuous to attribute this to disease as a similar phenomenon was observed in adjacent counties that did not seem to be affected during the outbreak. Similarly, no distinct pattern could be observed in the buck to doe or fawn to doe ratios between 1986 and 1987 when herds in affected (northern and Lake counties) and non-affected areas were compared. This could be due to the failure to notice carcasses in apparently unaffected counties, inadequate census methods or other factors such as poor forage (1985 being a particularly dry year) that overshadowed the impact of the disease.

Low fodder would not explain the Clear Lake outbreak of 1987 however, as rainfall in 1986 was above the 30 year average. Buck kill, buck/doe ratios and fawn/doe ratios do not seem to indicate that the outbreak had much of an impact on the deer populations during the 1986-87 outbreaks. However, fish and game field personnel, as well as local ranchers in the area are in agreement that there definitely was an abnormal increase in deer mortality in the affected counties during those years. The small numbers of animals sampled during and after the outbreaks preclude any definite conclusions on the seroprevalence of BT or EHD in the affected areas.

The 1986 northern counties and 1987 Lake county outbreaks appeared less severe than those described in pronghorn antelope in Wyoming by Thorne et al. (1988) but indicate that HD can present itself clinically in wild ruminant populations in California. This outbreak revealed to us pitfalls encountered in wildlife disease investigations and it is hoped that more complete information can be gathered during future outbreaks should they occur.

LITERATURE CITED

HOWERTH, E. W., C. E. GREENE, and A. K. PRESTWOOD. 1989. Experimentally induced bluetongue virus infection in white-tailed deer: Coagulation, clinical pathologic, and gross pathologic changes. Am. J. Vet. Res. 49:1906-1913.

JESSUP, D. A. 1985. Epidemiology of two orbiviruses in California's native wild ruminants: Preliminary Report. Pages 53-66 *in* Barber and Jochim, eds. Bluetongue and Related Orbiviruses. Liss, New York.