Severe Hoof Deformities in Free-Ranging Elk in Western Washington State

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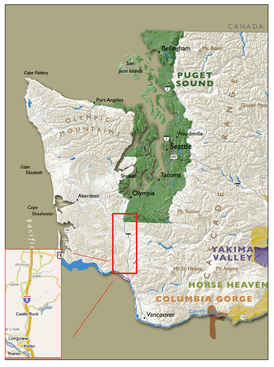
Abstract

Free-ranging Roosevelt elk (*Cervus elaphus roosevelti*) in South Western Washington state are presenting with severely overgrown and deformed claws, and often marked emaciation, the etiology of which has not yet been elucidated. Although reports of deformed hooves in elk have occurred sporadically in SW Washington for over a decade, the number and geographical distribution of these reports increased dramatically in 2008. During the winter of 2009 we undertook an investigation to better characterize the lesions and examine possible etiologies. Agency biologists surveyed groups of elk and interviewed landowners to estimate the prevalence and distribution of affected elk and to plot locations of positive cases on maps. Findings during the winter of 2008-2009 showed that approximately 80% of all herds observed had between 30 to 90% of animals affected with a wide variation in the severity of each case. Affected animals represented all age and sex classes. In this study five affected cow elk from three locations, and three apparently unaffected cow elk from one location were euthanized by gunshot. In all elk complete necropsies were performed and lower limbs were collected. On specimens we performed distal limb radiology, routine histopathology, hepatic trace mineral concentration, bacteriology of select tissues, virus isolation of key viscera, and parasitology of feces was performed. Necropsy, radiology, histopathology, and parasitology were in all cases unremarkable and failed to identify an underlying cause of hoof deformity. Hepatic selenium and copper levels interestingly, were severely deficient based on domestic livestock normal values. A variety of aerobic and anaerobic organisms were cultured from the hoof lesion, including *Dichelobacter nodosus*, an environmental organism, but also a known cause of infectious foot rot in domestic sheep and cattle.

The severe overgrowth and marked deformity of hooves from this cohort of free-ranging Roosevelt elk is unique. As it is not associated with underlying systemic disease, an etiology of toxicity, nutritional deficiency, or potentially infectious etiologies are all considered. Potential etiologies include recurrent laminitis, ergot toxicity, fescue toxicity, secondary conditions associated with severe copper and potentially severe selenium deficiency, or other infectious and toxic causes that may induce regional vasculopathy in the distal limbs. Distantly, a genetic etiology is considered.

Study Area



Herds of wild Roosevelt and Roosevelt-Rocky Mountain hybrid (*Cervus elaphus nelsoni*) elk were surveyed from Chehalis to Longview Washington (Department of Fish and Wildlife Region 5). The investigation covered both public and private lands including areas West and East of interstate I-5 (Fig. 1). Most affected individuals and herds were found within low-lying wet areas West of the I-5 corridor in the Cowlitz River Basin. Habitat in which affected herds were found included cultivated land, natural wild habitat, and pastures grazed by domestic livestock. Exposure to domestic livestock included contact with horses, cattle, and sheep.

Fig. 1.

Study Design

The goal of the current study is to collect **10 grossly normal** elk and **10 elk with abnormal hooves** from within the study area over a period of 1-2 years. In the spring of 2009 we euthanized by gun shot, and collected 3 healthy elk and 5 elk with deformed hooves from depredation areas within the study site. Collected elk were all females representing both adults and subadults. All animals were immediately necropsied and tissues, blood, and feces were collected. Our goal is to determine the overall health status of both seemingly healthy and affected elk and to hopefully identify probable causes of or predisposing factors for the hoof deformity.

On all elk the following was performed:

1. Complete necropsy (Fig. 2)
2. Histopathology of all viscera
3. Histopathology of normal and abnormal hooves
4. Trace mineral and selenium analysis (liver)
5. Radiology of normal and deformed hooves and distal limbs
6. Bacteriology of normal and abnormal hooves
7. Virology from the fresh tissue pool
8. Parasitology of feces

Fig. 2.

Findings

Field observations:

Collected animals included both adult and sub-adult female elk. In the affected herds examined, as many as 30% and up to 90% of animals had some degree of hoof deformity, which included all age and sex classes. Most affected animals had some loss of body condition, with occasional animals quite severely emaciated. Interestingly, affected elk had involvement of either one or both forelimbs or one or both hind limbs, but never a combination of fore and hind limbs, nor were all 4 limbs involved in any animal. Typically both claws per affected limb were deformed, though severity of the lesions on the same foot varied considerably.

Study animals:

Elk #	Sex	Age	Collection Site	Lesion + Location
1	Female	adult	Gould Farm	Right /left fore hooves deformed
2	Female	adult	Gould Farm	Right/left hind hooves deformed
3	Female	adult	Keatley Farm	Left hind solar abscess
4	Female	adult	Trickle Creek	Normal
5	Female	adult	Trickle Creek	Normal
6	Female	adult	Trickle Creek	Normal
7	Female	adult	Fenn Farm	Right/left hind hooves deformed
8	Female	sub-adult	Fenn Farm	Right/left fore hooves deformed with solar abscess

Radiographic examination:

Distal limbs of normal elk and selected abnormal limbs were radiographed and no significant bony abnormalities were detected. Cases with extensive deformity and/or solar abscesses of sloughed hoof walls had mild to moderate periosteal proliferation and osteomyelitis associated with P3 and similar mild reactions in the interphalangeal joint P2-P3 (Fig. 11). No primary or significant bone or joint disease was detected in any of the study animals.

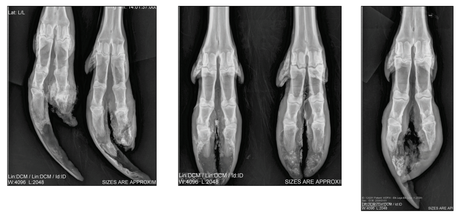


Fig. 11. Radiographs of moderately to severely deformed, and sloughed claws. No significant bone or joint lesions are detected in any animal, with the exception of periosteal proliferation secondary to exposure of P3 and osteomyelitis.

Bacteriology :

Sections of coronet, heel bulb, and interdigital space from six elk were cultured for aerobic and anaerobic bacteria. Five of six cases grew anaerobic organism *Dichelobacter nodosus* (formerly *Bacteroides*) which was definitively identified by sequencing 16-S RNA by PCR. One of 6 cases, with a deep solar abscess, additionally cultured positive for *Archanobacterium pyogenes*. No significant aerobic bacteria or fungi were isolated.

Virology:

Virus isolation of the fresh visceral tissue pool was performed on all eight cases (University of Wyoming) and no viruses were cultured.
PCR for epizootic hemorrhagic disease (EHD) and blue tongue virus (BTV) are currently pending on whole blood to detect current viremia.
Serology for a panel of significant viruses including EHD, BTV, and bovine viral diarrhea virus (BVDV) is pending on serum.

Parasitology:

Feces from six of eight cases were analyzed by fecal flotation, Baermann test (for lung worms), and liver fluke sedimentation. Parasitism was found to be mild to moderate in most animals with predominantly typical nematode parasites detected. Liver flukes were not detected.

Dictyocaulus sp. (1 of 6)
Capillaria sp. (3 of 6)
Strongyles sp. (3 of 6)
Trichuris sp. (1 of 6)
Coccidia (2 of 6)

Necropsy :

In the eight elk collected, three elk were considered clinically normal and five had grossly deformed claws. In all animals, no significant gross underlying disease, pathogenesis, or additional significant lesions were detected. Elk with poor body condition had moderate to severe serous atrophy of bone marrow fat (Fig. 3) consistent with chronic negative energy balance. Shown are representative hooves from collected elk which varied markedly in the severity and character of the deformity. Fig. 4 represent hooves from a grossly and clinically normal elk, in contrast to an elk with mildly overgrown claws (Fig. 5) and elk with severely overgrown and deformed claws (Fig.6, 7a/b/c). Deformities included excessive hoof length and medial and dorsal twisting of the claw (Fig. 6, 7a/b/c). Several elk had one or more claws in which the hoof wall was completely sloughed, or in which the claw was fractured mid-hoof and the proximal hoof wall and coronet remained intact (Fig. 6, 7a/b/c). The hoof walls of deformed claws varied markedly from smooth, to rough and irregular with ridges and incomplete rings. In some cases the hooves had deep cavitations of the sole (Fig.8, 9a/c), near the coronary area, or within the hoof wall mid-claw (Fig.6). Cavitations were typically very deep with extensive lamellar necrosis and foreign material contamination, and exposure of P3. Soft tissue lesions of the distal limb were limited in all cases, with occasional chronic ulceration of the heel bulbs and interdigital space (Fig. 10) that may have been secondary to abnormal ambulation and rubbing from the adjacent claw. In all cases the hoof deformity was much more severe and far more chronic than the soft tissue lesions.

Fig. 3. Serous atrophy of bone marrow fat detected in affected elk with poor body condition. Interestingly, this lesion was inconsistent, and some animals with severely deformed hooves had maintained adequate body condition with normal bone marrow.

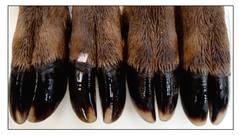


Fig. 4. Three of five collected elk had grossly normal. hooves

Fig. 5. Mildly overgrown claws

Fig. 6. Severely overgrown claws with cavitation of otherwise intact hoof wall (arrow), and completely sloughed claws with exposure of phalange (P)3.

Fig. 7a. Severely overgrown and deformed claws with mid-hoof fracture of the hoof wall, leaving the coronet and proximal hoof wall intact, and complete sloughage of the hoof wall . Dorsal view.

Fig. 7b. Ventral view of the same hooves. Severely deformed claws with sloughed and fractured claws. Ulceration and irregularity of the heel bulb.

Fig. 7c. Cross-sectional view of the overgrown claw showing relatively normal association of hoof wall and lamina to P3 with no joint involvement.

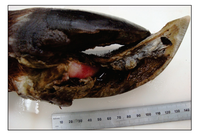


Fig. 8. Overgrown claws with extensive cavitation and necrosis of the sole, and subsequent exposure of P3.

Fig. 9a. Mildly overgrown claws with extensive solar cavitation, complete laminar necrosis, and exposure of P3. Ventral view.

Fig. 9b. Dorsal view of the same hoof showing relatively normal and intact hoof wall with elongation of the toe, limited deformity of the claw, and lacking irregularity of the keratin.

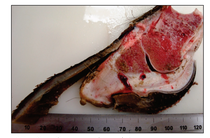


Fig. 9c. Cross section of the same hoof showing extensive loss of the sole and necrosis of the lamina with exposure of P3.

Fig. 10. Deformed hoof with interdigital and heel ulceration and dermatitis.

Histopathology:

Histopathological examination of formalin fixed tissues representative of all viscera from each elk was performed. Tissues included decalcified and de-keratinized sections of bone and hooves. A myriad of mild to moderate, and likely incidental lesions were detected among the eight collected elk. No underlying systemic diseases, infections, or degenerative conditions were detected in any animal. Importantly, lesions causative of, or related to, abnormal hoof growth were not detected. Sections of keratinized hoof wall, lamina, the heel bulb, and haired skin of the coronet and interdigital space in most elk (6 of 8) had mild to moderate lymphocytic-plasmacytic perivasculitis (Fig. 12). Hooves with cavitating lesions additionally had sub-acute thrombosis of arterioles in the lamina and diffuse pleocellular laminitis (Fig. 13a and b). Thrombosis was not seen in cases in which the hoof wall was still intact. Lesions seen more commonly are listed (Tab. 2) and most are interpreted as incidental lesions likely of limited clinical significance, and likely unrelated to the hoof lesions. Keratin was normally organized (Fig. 14) and all cases lacked significant coronet lesions.

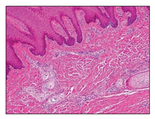


Fig. 12. Mild lymphocytic-plasmacytic perivasculitis of the coronet.

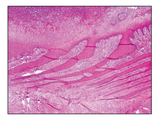


Fig. 13a. Lamellae are generally organized, with varying degrees of pleocellular laminitis.

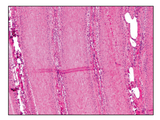


Fig. 13b. Pleocellular laminitis.

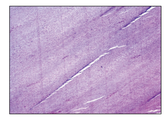


Fig. 14. Keratin tubules. comprising the hoof wall are organized.

Tab. 2. Lesions noted histologically in tissues from all eight study elk:

- Hepatic portal fibrosis, multifocal, chronic moderate with early bridging and biliary hyperplasia (3 of 8 cases)
- Perivascuilitis, lymphocytic, multifocal, random, chronic, mild meninges, brain, heart, GI tract (4 of 8 cases)
- Myocardial degeneration, non-suppurative, subacute, moderate (1 of 8)
- Glomerulonephritis, mesangial-proliferative, lymphocytic-plasmacytic, chronic, mild (1 of 8 cases)
- Renal interstitial nephritis, lymphocytic-plasmacytic, chronic, mild (4 of 8 cases)
- Random visceral abscesses, chronic, mild, pulmonary, lingual, and renal (3 of 8 cases)
- Skeletal muscle sarcocystosis, multifocal, chronic, moderate (8 of 8 cases)

Toxicology:

Heavy metal, trace mineral, and selenium levels were measured from fresh liver in all eight cases including a 9th freshly dead yearling cow elk found with severely deformed hooves and emaciation at one survey site. Normal values were adapted from approximate reference ranges currently used for adult cattle. All elk were consistently and markedly deficient in copper and selenium, with slight elevation of cobalt levels.

Minerals	Range of Results	Reference Range Elk (µg/g)	Reference Range Bovine (µg/g)
Copper	1.6 – 20.0 (Mean 6.15)	20-120	20-120
Selenium	0.06-0.41 (Mean 0.18)		0.25-1.4
Cobalt	0.06-0.12 (Mean 0.08)		0.03-0.08
Zinc	27-100 (Mean 44.9)		23-80
Manganese	2.9-4.3 (Mean 3.7)		2-6
Molybdenum	0.44-1.3 (Mean 1.1)		0.14-1.4

Summary

Severe overgrowth and deformity of the hooves in the Cowlitz river basin Roosevelt elk population has been sporadically noted in individual animals for the last decades. However, recently the incidence of affected elk and the severity of cases has seemingly increased to involve most herds surveyed within this study area, affecting up to as many as 90% of elk in some herds. Cases of overgrown and deformed hooves similar to this have been occasionally recorded throughout the US in populations of wild deer, moose, and bighorn sheep. The cause of hoof deformity in these other herds and species has never been definitively elucidated, though several predisposing factors have been suggested. These factors include severe copper deficiency, selenium toxicosis, chronic ingestion of high carbohydrate diets, chronic ingestion of a predominantly alfalfa diet (with or without recurrent laminitis), exposure to infectious viruses such as epizootic hemorrhagic disease (EHD) or blue tongue virus (BTV), and change of behavior and habitat that include limited ambulation or ambulation on soft soils which may prevent normal hoof wear.

In our study population we found that hoof deformity affected either the fore or rear pair of limbs and did not appear to be restricted to particular age or sex classes, indicating that even severe lesions could develop rapidly enough to affect animals under one year of age. Lesions in the study animals varied markedly from extensive overgrowth and deformity of an otherwise intact hoof, to complete sloughing or breakage of abnormal hooves and hoof walls. Additionally, some hooves had extensive necrotizing and cavitating lesions of the sole or hoof wall. Focal hoof necrosis could be indicative of local infection, or weakness or abnormality in the keratin or underlying lamina that would focally devitalize the hoof or predispose more fragile foci to trauma and infection. In the majority of cases, the keratin of the hoof wall and sole was thick, well organized, and very hard, similar to hoof keratin in unaffected hooves of the same animal, or the hooves of normal elk collected from the study site. Histologically, keratin of the hoof wall and sole and the germinal epithelium of the coronet was well organized and no significant primary lesions were detected, making the likelihood of a developmental or genetic keratin disorder less likely.

In all cases soft tissue lesions, which would be most indicative of a primary infectious foot rot, were lacking or minimal. Bacterial culture of affected hooves was positive for *Dichelobacter nodosus*, a known cause of infectious foot rot in domestic livestock; however this environmental pathogen can be readily isolated from soil and would be an anticipated opportunistic pathogen in any foot condition. Therefore, a primary foot rot should be interpreted with some degree of caution.

Most animals had some degree of soft tissue and lamellar perivasculitis that was predominantly lymphocytic-plasmacytic. Though this lesion may be due to distal limb inflammation and chronic trauma, causes of vasculopathy are considered. Of particular interest are causes of vasculopathy that may compromise normal peripheral blood flow, induce aberrant neovascularization in the hoof or lamellae, or result in random thrombosis of these structures and consequently alter normal hoof growth and integrity. Causes of vasculopathy considered are recurrent laminitis, EHD, BTV, and ergot or fescue toxicity. Serology and PCR to determine viral exposure or current viremia in the study elk are currently pending.

Possibly our most important finding in this study is marked copper and selenium deficiency in this population of elk. Though certainly this area has been historically deprived of copper and selenium, perhaps changes in habitat, encroachment of farmed fields and livestock, emergence of new plant populations, significant dietary changes, or behavioral changes, may have favored the recent development of severe hoof lesions. Copper in particular is known to be vitally important for proper bone and keratin development. A. Flynn described in1977, populations of Alaskan moose with similar severely overgrowth of hooves. The cause of this lesion, described as "slipper foot", was not definitively determined; however, affected moose were found to be significantly deficient in copper. Copper deficiency in domestic cattle is known to be associated with an increased incidence of foot rot, heel cracks, and sole abscesses. Whether copper deficiency alone can induce or predispose hoof deformity in the Cowlitz basin elk, or if copper deficiency is one contributor to a multi-factorial problem, remains yet to be determined.

A more extensive understanding of the habitat and the principal diet of these herds, as well as the movement within the home range, is necessary to completely understand the dynamics of nutritional deficiencies, toxicities, systemic infections, potential exposures to toxic fungi, and to determine what influence movement, soil substrate, or behavioral changes might play in the pathogenesis of these hoof lesions.

References , Funding, and Acknowledgement

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