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Paresis and Death in Elk (*Cervus elaphus*) Due to Lichen Intoxication in Wyoming

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ABSTRACT: During February– April 2004, an estimated 400–500 free-ranging elk (*Cervus elaphus*) developed paresis, became recumbent, and died or were euthanized in the Red Rim Wildlife Habitat Management Area (RRWHMA), Wyoming, USA. Elk were found in sternal recumbency, alert and responsive, but unable to rise. Their condition progressed to lateral recumbency followed by dehydration, obtundation, and death. Gross lesions were limited to degenerative myopathy, with pallor and streaking in skeletal muscles. Microscopically, affected muscles had degenerative lesions of varying duration, severity, and distribution, some with early mineralization and attempts at regeneration. Diagnostic testing ruled out common infectious, inflammatory, toxic, and traumatic causes. Tumbleweed shield lichen (*Xanthoparmelia chlorochroa*) was found in the area and in the rumen of several elk. This lichen was collected and fed to three captive elk. Two of these elk exhibited signs of ataxia, which rapidly progressed to weakness and recumbency after 7 and 10 days on this diet, respectively, and a degenerative myopathy, consistent with lesions observed in the elk affected at RRWHMA, was observed. All remaining elk migrated from the RRWHMA during the spring and no subsequent losses have been documented.

Key words: Elk, lichen, myopathy, paresis, *Xanthoparmelia chlorochroa*.

The Red Rim Wildlife Habitat Management Area (RRWHMA) is a 40–50-mi^2 area (41°42.955′N, 107°28.239′W) in southeastern Wyoming, USA. The area is not typically used by large numbers of elk (*Cervus elaphus*) in the winter, but in the winter of 2003–04, approximately 800 elk were utilizing the area. The region had experienced a drought since 1999 and range conditions were considered moderately to severely overgrazed; conditions noticeably worsened during the time the elk were in the area (M. Smith, personal communication).

On 8 February 2004, two live, but recumbent, elk in the RRWHMA were reported to the Wyoming Game and Fish Department (WGFD). The elk were euthanized via gunshot to the neck and one was submitted to the Wyoming State Veterinary Laboratory (WSVL), Laramie, Wyoming, USA. Two days later two more elk were found; both were euthanized and taken to WSVL. The next week, seven more cases were discovered, and by the following week over 60 cases had been located. New cases continued to be documented through April 2004.

Affected elk displayed progressive weakness and were almost always found unable to rise; most were in sternal recumbency, but in a few advanced cases (shortly before dying) elk were found in lateral recumbency. Fifty-two elk were found entangled in four-strand, 42-inch barbwire fences, apparently unable to jump them. Elk were afebrile, with normal pulse and respiration. They displayed normal mentation as evidenced by their behavior and reaction toward humans. Affected elk did not survive, though most of them would eat and drink if provided forage and water. Animals were generally in fair-to-good nutritional condition. Full diagnostic workups were performed on 12 affected elk and all had elevated serum creatine kinase activity. All but two elk had elevated serum lactate dehydrogenase and aspartate aminotransferase activities. Serum albumin and Ca^{+2} were depressed in

most animals. All exhibited a marked leukocytosis with neutrophilia, interpreted as a stress leukogram (Latimer and Prasse, 2003). Urine was collected from the urinary bladder of six elk at necropsy. Urine specific gravity ranged from 1.000 to 1.010 in these samples and one specimen contained a trace of occult blood. Urine samples otherwise were negative for leukocytes, nitrite, urobilinogen, bilirubin, protein, pH, glucose, epithelial cells, casts, and bacteria.

Standard laboratory testing, history, and clinical signs ruled out known bacterial, viral, parasitic, and common toxic causes of neurologic/muscular syndromes. Over 40 differential diagnoses including chronic wasting disease, infectious and parasitic causes of encephalomyelitis and meningitis, peripheral neuropathies, nutritional myopathies, common toxic myopathies including ionophore toxicosis, and trauma were considered and ruled out.

Findings at necropsy were minimal; some elk had muscle pallor and streaking, particularly of the semitendinosus, semimebranosus, and other muscles of ambulation in pelvic limbs. Histologically, these muscles demonstrated acute degenerative myopathy as evidenced by varying degrees of myofiber degeneration (swelling, hyaline change, and vacuolar change), rupture, and necrosis with mononuclear inflammation of varying severity (Fig. 1). Mineralization was rare within skeletal muscle lesions, but was observed in muscle from two elk. Other histologic findings were unremarkable. Many affected elk had moderately distended large bowels (spiral, transverse, and descending colon and rectum) that were filled with fecal pellets of normal consistency. Discolored (orange-red) urine was found staining the snow in the vicinity of many affected elk. The urinary bladders of affected elk were typically empty, but when not, contained urine of normal (yellow-amber) color.

Four live elk with typical clinical signs were captured in the field and transferred to the WSVL, where they were treated for up to 17 days. All four elk were treated with intravenous fluids (lactated Ringer's), and two received injections of vitamins A, B, D, and E and selenium. One also was

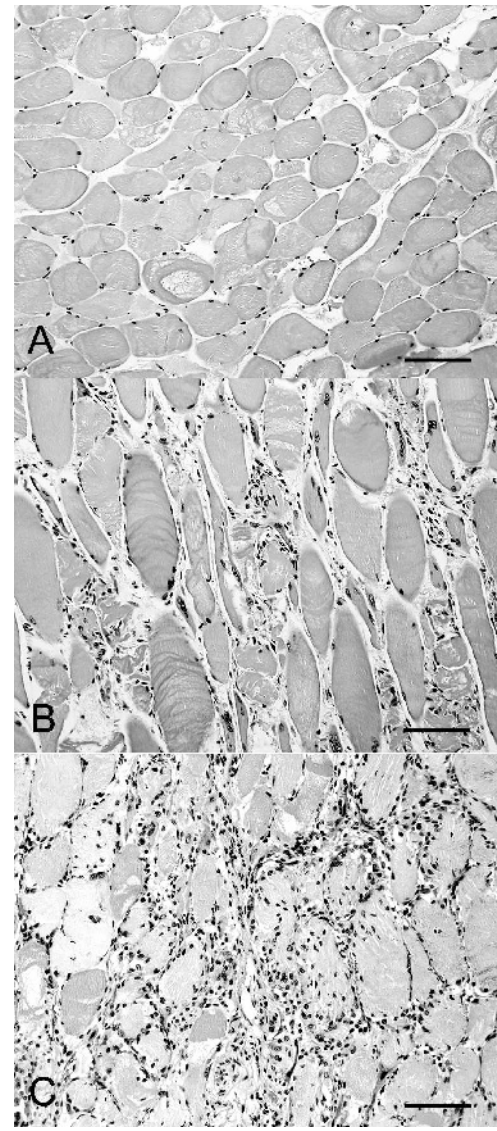

FIGURE 1. Semitendinosus muscle from three affected elk. A. Early lesions demonstrating myofiber swelling, hyaline change with loss of striations, and early vacuolar change. H&E stain. Bar=200 μm. B. More extensive myofiber degeneration and necrosis with early mononuclear inflammation. H&E stain. Bar=100 μm. C. Similar diffuse myofiber degeneration and necrosis with more extensive inflammation. H&E stain. Bar=100 μm.

treated with calcium gluconate, dextrose, phosphorus, magnesium, and potassium. Two elk were treated with anti-inflammatory (flunixin meglumine) and corticosteroid (dexamethasone) drugs. One elk was hoisted up in a sling in an attempt to stimulate ambulation. None responded to any treatment; all either died or were euthanized because of persistent recumbency and inability to rise.

Microscopic examination of rumen contents (J. Reagor, Texas Veterinary Medical Diagnostic Laboratory, College Station, Texas) confirmed that affected elk in the RRWHMA were eating a free-living lichen commonly known as tumbleweed shield lichen (*Xanthoparmelia chlorochroa*). Approximately 50 kg of this lichen was collected at RRWHMA in March of 2004 for use in feeding trials. Three captive elk, translocated from northwestern Wyoming, were fed a diet containing the lichen between 12 March 2004 and 25 March 2004 at the WGFD's Sybille Wildlife Research Unit. For 3 days, the elk were offered a mix of alfalfa and lichen, with the proportion of alfalfa to lichen decreasing daily. On the fourth day, only lichen was offered, which was weighed every 24 hr to determine total amount consumed. The three elk consumed from 1.1 kg to 3.6 kg lichen daily. Four days after feeding lichen, orange-red colored urine was noted in the pen. Two of three elk displayed clinical signs of weakness and paresis at 7 and 10 days after feeding only lichen and were euthanized and examined postmortem. The remaining elk ate little, if any, lichen, showed no clinical signs, and was returned to a normal diet. Gross and histologic examination of tissues of the two euthanized elk revealed degenerative myopathy lesions identical to those of affected elk from the field. Twenty-five elk from the same source housed at the Sybille facility and fed a diet composed entirely of alfalfa hay exhibited no signs of weakness or paresis during and after the 2-wk trial period.

Lichens are composite, symbiotic organisms composed of members from as many as three kingdoms. The dominant partner is a fungus (kingdom Fungi), which cultivates partners that manufacture food by photosynthesis, either algae (kingdom Protista), or cyanobacteria (bluegreen algae; kingdom Monera). The lichen implicated in the current episode is *X. chlorochroa* (Hale, 1990; Syn.: *Parmelia chlorochroa*), tumbleweed shield lichen, a common free-living ground lichen in Wyoming and elsewhere in western North America. Information on lichen as a food item used by elk is limited. Elk were reported to eat *Bryoria trichodes* subsp. *americana*, *Ramalina menziesii*, and *Usnea* spp. (Kufeld, 1973; Marcum, 1980) but we believe that *Usnea barbata* and *Usnea plicata,* as cited, were misidentifications. Marcum (1980) reported an average of 3% volume of lichens occurring in 33% of rumens sampled from a western Montana herd in October and November 1972. Most forage studies of elk do not list lichens; they are probably occasional winter forage, especially at times of stress or limited herbaceous or grassy forages. Schwartz and Mitchell (1945) reported that lichens on the trunks and limbs of trees on the Olympic Peninsula were an important part of Roosevelt elk diets, especially at higher elevations in winter. Nelson and Leege (1982) also noted that elk occasionally consume lichen, particularly arboreal lichens in winter.

Reports of intoxication with *Xanthoparmelia* spp. are sparse. Poisoning of "laboratory animals" fed "an emulsion of usnic acid extracted from a lichen identified as *Parmelia molluscula* Ach" was mentioned as two personal communications (Beath, Wyoming Agricultural Experiment Station, 1960; Huffman, Salina Utah Experiment Station, 1955) in Kingsbury (1964). The remaining fraction, devoid of usnic acid, was not toxic, leading Beath to conclude that usnic acid was the toxic compound, especially since the lichen in question contained approximately 1.6%

usnic acid (as extracted with carbon disulfide). Likely the lichen species identification was incorrect, as *P. molluscula* is not known to occur in North America (Esslinger, 2002), and we believe the investigators may have been working with *X. chlorochroa*. Earlier, Beath et al. (1953) briefly described an intoxication in domestic livestock caused by free-living lichen again identified as *P. molluscula* (probably *X. chlorochroa* also), stating that the lichen was not a serious menace to livestock, and that it caused poisoning in winter when forage was scarce although the lichen was toxic year-round. Described clinical signs in this report included depression and hind-limb ataxia in mild cases and both forelimb and hind-limb paresis in more severe cases. The authors stated that no lesions or characteristic pathology developed, although it is unclear if they examined the muscles. Unlike sheep, cattle continued to eat and drink while recumbent. Subcutaneous injections with strychnine sulfate were stated to be beneficial for affected cattle, but not for sheep. The authors concluded that the toxic principal affected either the spinal cord or cerebrum. A toxic dose was reported to be 1% of an animal's weight for 5 days, or a single dose of 3.6% of an animal's weight. All of these citations appeared to refer to a single investigation by Beath et al. in the 1930s.

There is no record of *X. chlorochroa* or other lichen toxicity occurring in elk or other wild ungulate species, and in fact, some references suggest this lichen may be an important forage source, especially for pronghorn (*Antilocapra americana*) (Mitchell and Smoliak, 1971; Thomas and Rosentreter, 1992a, 1992b). According to Brodo et al. (2001), *X. chlorochroa* may be a reliable indicator of excellent pronghorn habitat and the presence of this lichen has been used to select pronghorn reintroduction sites in the United States in the past. Further, these authors state that this lichen commonly is consumed by pronghorn and may be an especially important forage source in early spring and under drought conditions. A rumen sample of an antelope on winter range in southeastern Idaho contained 51% lichen (Bernt, 1976). Domestic sheep grazed some of the same ranges and were thought to compete with antelope for the same lichens. Anecdotal reports from ranchers in the area indicated that sheep have historically used lichen as winter forage.

The pathogenesis of *X. chlorochroa* toxicosis in the affected elk remains unknown. That usnic acid may play some role seems reasonable, but other contributing or synergistic compounds in the lichen cannot be ruled out, and the molecular and cellular events producing ataxia, paresis, and recumbency are unclear. The degenerative myopathy observed in the majority of clinically affected elk is most consistent with an exertional or ischemic/compression type of injury, because few or no muscle lesions were observed grossly or microscopically in acute field and experimental cases. However, animals that were recumbent for several days or longer demonstrated progressively more severe muscle degeneration and necrosis. In all cases, nonambulatory skeletal muscle, including diaphragm and tongue, were not affected, and in no cases was cardiac muscle affected. A direct toxic myopathy cannot be ruled out entirely, but it seems unlikely given the clinical course of disease and lesion distribution.

The urine discoloration also appears to be unrelated to myopathy and is not consistent with a myoglobinuric nephrosis; no significant lesions were observed in kidneys from any elk microscopically, and urinalysis data fail to support such a nephrosis. Of interest, *X. chlorochroa* was used by Navajo as a source of red dye (Brodo et al., 2001). When soaked in water, the water turns an orange–red, nearly identical to the color of the urine observed near affected elk. We believe the urine discoloration may be caused by excretion of a pigment produced by the

lichen, and further research into this phenomenon is ongoing.

Losses are believed to have begun between 26 January 2004 and 8 February 2004. By the end of April, the WGFD had confirmed 327 cases and estimated total losses were 400–500 elk. Most affected animals were adult females (275 or 84%). This is largely a cow–calf herd, which is presumed to explain the paucity of affected bull elk. Only 31 (9.5%) of the 327 confirmed cases were calves. The post-season ratio in 2003–2004 was 45 calves:100 cows. Thus calves were underrepresented in the die-off (calves were approximately 30% of the herd, but less than 10% of affected animals). Calves may have been less likely to consume the lichen, or may have been more resistant to its effects. Down calves were harder to find than down cows and thus the lower representation may have been because of reduced sightability of calves.

A herd of approximately 400 healthy elk was seen feeding in the area in late January 2004. On 31 December 2003, a recumbent elk was found approximately 11 miles east of the Red Rim outbreak. This animal had signs consistent with the elk from this outbreak and may have been the first affected in the outbreak, but no diagnostic work was attempted on this elk.

Losses ceased when the affected herd moved out of the RRWHMA. No other species, including pronghorn, mule deer (*Odocoileus hemionus*), or resident cattle and horses were affected. However, there were few horses or cattle in the area, and we found no evidence (hoof prints or feces in the lichen) that they had eaten the lichen. The pronghorn herd was estimated at 300–500 animals, but they seemed to use different areas than the elk. There were an estimated 40–60 mule deer on the northeastern part of the area but there was no evidence that they had eaten the lichen. It is possible that other species were not affected because they did not consume the lichen.

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