J Vet Diagn Invest 5:84-87 (1993)

Naturally occurring selenosis in Wyoming

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Abstract. A review of Wyoming State Veterinary Laboratory records for 1947-1987 revealed no substantiated cases of naturally occurring selenosis. However, older reports attributed thousands of animal deaths to selenium each year in this area. Beginning in August 1988, cases of suspected selenosis and selenium deficiency were solicited from veterinarians and producers by announcements in various statewide livestock publications. As of August 1991, 4 cases (all horses) of naturally occurring selenosis have been confirmed. Clinical signs were most often referable to epithelial damage, e.g., hoof lesions and loss of mane and tail. None involved neurologic signs. Sources of selenium included native range and grass hay.

Selenium (Se) is a group VI element with chemical properties very similar to those of sulfur. In North America, it is most abundant in Cretaceous shales and glacial drift in the Great Plains. Historic accounts of livestock diseases that might have been chronic selenosis date back to Marco Polo.[10] However, the first scientific association of Se with naturally occurring disease(s) was developed in a series of experiments at the Wyoming and South Dakota Agricultural Experiment Stations in the late 1920s.[3] Numerous reports were published during the 1930s and 1940s as a result of these investigations. However, after the essential nature of Se was demonstrated,[11] the focus of most research efforts shifted to Se deficiency. Extensive mortality and teratogenesis among wild waterfowl at the Kesterson Wildlife Refuge in California recently rekindled interest in Se toxicity, especially in the popular press.

Despite anecdotal reports of severe livestock losses in Wyoming during the early 1900s,[10,12] a review of the records of the Wyoming State Veterinary Laboratory (WSVL) from 1947 to 1988 revealed no confirmed cases of selenosis. In 1987, Se deficiency was diagnosed in cattle herds in reputedly seleniferous areas of the Powder River basin. Therefore, a study was undertaken in cooperation with the Wyoming State Chemistry and Bacteriology Laboratory to determine the nature and extent of selenosis in Wyoming livestock.

Materials and methods

Cases of suspected selenosis were solicited through announcements in the Wyoming Veterinary Medical Association newsletter, extension publications, and farm publications. Suggested criteria for inclusion in the study included clinical signs previously described as characteristic of blind staggers (circling, head pressing, ataxia, convulsions), alkali disease (inappetence and ill thrift, hair loss, and hoof abnormalities),[10] and acute selenosis (dyspnea, ataxia, central nervous system depression, polyuria, and diarrhea).[7,8] After 6 mo, these criteria were expanded to include any case that the owner or referring veterinarian felt was associated with consuming seleniferous vegetation.

Samples of blood, serum, liver, and kidney were analyzed for Se by a fluorometric method [Dolan M, Zinn KR, Rottinghaus GE: 1985, TLC, HPLC, GLC and neutron activation determination of Se. Midwest Regional AOAC, June 17-19]. Forage and other environmental samples were analyzed where available from cases with elevated tissue concentrations.

Results

From August 1988 to July 1991, the WSVL analyzed 586 samples for Se. Of the 275 accessions represented by these samples, Se toxicity was considered to be a possible differential diagnosis in 52. The rest were submitted as probable Se deficiencies or else no history or clinical diagnoses were available. Clinical diagnoses were relatively unreliable. In 2 cases, probable iatrogenie Se intoxication in sheep were originally presented to the lab as Se deficiency. In 2 additional cases (1 bovine, 1 ovine), elevated tissue Se concentrations were discovered serendipitously, but the animals' owners refused to cooperate any further as the possibility of naturally induced selenosis did not support pending lawsuits. Thus, for purposes of this study, all cases in which the reported clinical signs or tissue Se concentrations suggested selenosis were investigated, regardless of presenting diagnosis.

Alkali disease

Four cases of naturally occurring alkali disease were diagnosed in horses based on compatible history, clinical signs, and tissue Se concentrations.

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Presented at 34th Annual Meeting of the AAVLD, San Diego, CA, October 27-29, 1991.

Received for publication October 31, 1991.

Case I (10-year-old quarterhorse mare). The mare was placed in a 10-ha native pasture northwest of Casper, Wyoming in June 1989. Although the pasture contained an intermittent stream, the owners hauled municipal water to the horse daily. In addition to various native grasses, the pasture contained several dense strands of *Astragalus bisulcatus*. In late July, the owners noticed a mild forelimb lameness. Approximately 1 week later, in early August, the mare's mane and tail hair fell out, and the owner noticed the coronary bands of all 4 hooves were swollen and hot (Fig. 1). The referring veterinarian reported no response to hoof testers and that the mare was leukopenic; however, a differential white blood cell count was not done. On recommendation of the referring veterinarian, the horse was confined to a dry lot and fed low-Se grass hay.

Blood, serum, and forage samples were collected for Se analysis on August 29. At this time the pasture, which had not been grazed for 1-2 weeks, appeared to be in good condition. Most of the grasses showed evidence of being grazed, but the forbs, especially *A. bisulcatus* did not. Forage (grasses and forbs) Se concentrations varied from 0.1 ppm to 20 ppm on an "as received" basis. Blood and serum contained 1.3 ppm and 1.07 ppm Se, respectively. Follow-up blood samples taken at 30 and 120 days contained 0.86 ppm and 0.16 ppm Se, respectively. By December, the only hoof lesion consisted of a horizontal crack, which continued to grow out with the hoof. At this time, the mare appeared to be sound and competed in several barrel races during the spring of 1990 without any recurrence of lameness.

Case II (mixed breed mare, age unknown). The mare was part of a herd of 8 horses kept in a 20-ha paddock in central Wyoming. The horses were fed grass hay (0.16 ppm Se) and sweet feed (0.11 ppm Se) daily and had well water (0.006 ppm Se) available ad lib. Several varieties of weeds grew in the paddock, but none were available for identification or Se analysis. Clinical signs were similar to those reported in the first case, but no samples were available until several weeks after the onset of signs, after the mare had been separated from the other horses and switched to different forage. At this time, the mare's blood Se concentration was 0.86 ppm; it declined to 0.51 ppm after 30 days then increased to 0.6 ppm after 60 days, when she was returned to the herd. The other 7 animals never showed any signs of disease and had blood Se concentrations of 0.4-0.7 ppm.

Case III (5-year-old quarterhorse mare). The mare was kept in a 480-ha native pasture south of Gillette, Wyoming, with approximately 100 head of cattle. She was known to be a "hard keeper," so the owner occasionally supplemented her diet with whatever commercial sweet feed was on hand. In late August, the owner noticed that the mare was losing condition but attributed this to the horse's history of ill thrift. In September, the mare's mane and tail hair fell out, leaving only short stubble. In early October, the mare was lame and taken to the local veterinarian. At physical examination, the only abnormality noted in addition to emaciation was a horizontal crack around the hoof approximately 1 cm below the coronary band. Blood Se concentration was 0.96 ppm. A sample of forage grasses from the pasture contained 0.13 ppm Se. The mare was confined to a small drylot on grass hay and grain. By February, the blood Se concentration had declined to 0.35 ppm and the mare was gaining weight.

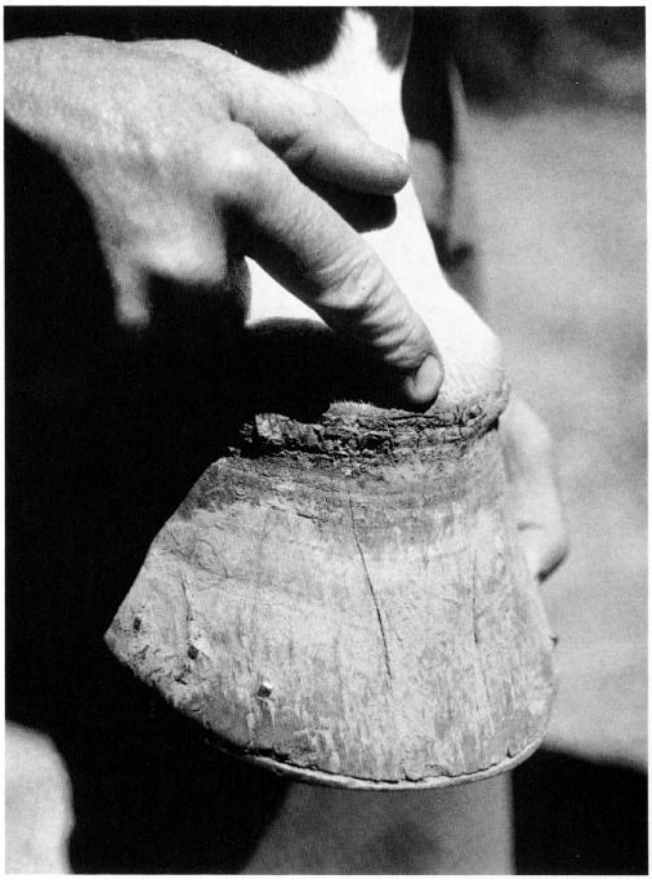

Figure 1. Rear hoof of quarterhorse mare with selenosis approximately 2 weeks after onset of lameness. Note swelling and cracking of coronary band.

Case IV (15-year-old Appaloosa gelding). The gelding, together with 4 other horses, was moved into a native pasture northwest of Laramie, Wyoming, in June 1990. Rainwater was occasionally available in a small pond, but most water was hauled from town. In September, the gelding was noticed to be lame. At that time he was diagnosed as having alkali disease by the local farrier, although no hoof lesions were apparent. The gelding was transferred into a small (30- x 30-m) pen and put on a diet of local grass hay and grain. Shortly after being moved off pasture, the gelding began

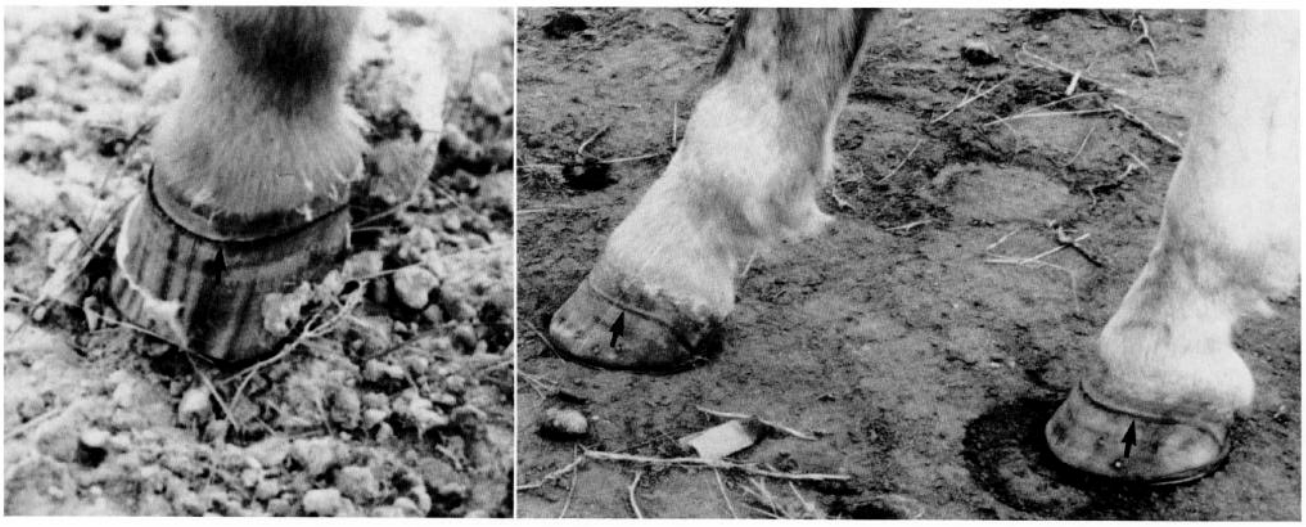

Figure 2. Front hoof of mixed-breed gelding with selenosis approximately 2 months after onset of lameness and hair loss. Note horizontal crack (arrow).

Figure 3. Rear hooves of horse in Fig. 2, 90 days later. Note that cracks (arrows) are progressing outward with hoof growth. At this time, the severity of lameness was beginning to subside.

loosing his mane and tail and developed cracks around the coronary band (Fig. 2). In November, the hay was switched to North Park (Colorado) grass hay. Blood taken in early December contained 1.1 ppm Se and revealed a leukopenia (4,550 white blood cells; 42% lymphocytes, 56% segmented neutrophils). In March, blood Se was 0.5 ppm and the leukogram was normal. At this time, some minor lameness persisted, but the horse had regained condition (Fig. 3). By June, he had completely recovered and was returned to pasture.

Blind staggers

Six cases of bovine neurologic disease, tentatively diagnosed as "blind staggers" by owners or referring veterinarians, were received. In 5 cases, the morphologic diagnosis was polioencephalomalacia (PEM), the sixth was diagnosed as thromboembolic meningoencephalitis. One bull with PEM had elevated blood and liver Se concentrations; however, other affected individuals from the same herd did not. In 4 of the cases diagnosed as PEM, the cattle were drinking high-sulfate water (2,800, 3,000, 4,700, and 6,000 ppm SO,).

Discussion

According to Beath,[10] "Seleniferous plants can produce either acute or chronic poisoning in livestock. The chronic poisoning occurs in two forms, one known as blind staggers and the other as alkali disease." Acute selenosis and the collection of clinical signs referred to as alkali disease have been associated with elevated tissue Se concentrations in field cases[7] and reproduced by feeding seleniferous forages and grains and/or Se salts.[1,8] The condition described by early investigators as blind staggers has never been reproduced by feeding Se compounds.

The clinical signs described for blind staggers[10] closely resemble those of the condition now recognized as PEM.[2] Frequently, the geochemistry associated with selenium accumulator plant species also produces high-sulfate waters. High-sulfate diets have been associated with PEM[4,9] and have been experimentally reproduced by feeding inorganic sulfur compounds.[5] Polioencephalomalacia is a common problem today on many ranches in areas where earlier observations of blind staggers were reported. Many of the older ranchers interviewed during this study referred to any bovine neurologic disease as blind staggers, which they attributed to Se intoxication and which suggests that the condition originally described as blind staggers was in fact PEM and is unrelated to Se toxicity. This hypothesis is further buttressed by the association of high-sulfate waters with PEM and the lack of elevated Se tissue concentrations in the cattle in this study.

Conversely, the condition described as alkali disease does seem to be associated with Se. This survey found only 4 cases of alkali disease in 3 years, indicating that the condition is not as widespread as suggested previously. In 2 instances, only 1 horse of a herd was affected, indicating considerable individual variability in susceptibility to Se toxicity or dietary selection or both. Each of these horses recovered with only supportive care, suggesting that the wasting and mortality reported for alkali disease in the past is the result of an animal's inability to obtain food and water rather than a direct effect of chronic low-level selenosis.

Although only horses were diagnosed with alkali dis-

ease in this study, there were anecdotal reports of similar hoof and hair problems in cattle on neighboring ranches in the past. According to these producers, newly introduced cattle are most susceptible. The problem rarely if ever occurs in locally grown stock,[6] suggesting that animals can develop tolerance to elevated dietary Se, although at present this observation has not been experimentally confirmed and no mechanism has been proposed for the phenomenon.

The results of this study indicate that chronic selenosis, although relatively uncommon, does exist. Millions of acres of rangeland overlay geologic formations conducive to growth of seleniferous forages. The effects of chronic high-level selenium intake on livestock species, however, have been studied little since the 1940s and 1950s, when toxicologic tools to thoroughly evaluate chronic effects such as infertility or immunosuppression typically associated with chronic intoxications did not exist. Until all the effects have been adequately explored, the true impact of Se on animal production cannot be ascertained.

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