Chapter Seventy-Four

Selenium Poisoning in Cattle

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Selenium is readily absorbed by all plants. However certain soils contain selenium in amounts and forms that render some plants toxic to animals. Plants have been divided into two groups according to their ability to accumulate selenium: (i) primary selenium-accumulators or indicator plants, which include species of the *Astragalus, Haplopappus* and *Stanleya* genera. These plants accumulate low molecular weight, water soluble organic compounds and selenate (Anon., 1980) to give selenium levels of 50 to several thousand mg kg^{-1}; and (ii) non-accumulator plants which include grasses, shrubs, small grains, alfalfa and some forbs. These plants usually accumulate less than 50mg kg^{-1} (generally 5-12mg kg^{-1}) selenium as organic forms in plant proteins. Selenium poisoning in livestock most frequently involves these non-accumulator plants.

Selenium poisoning in livestock has been classified as acute and chronic intoxication. Chronic poisoning has further been divided into two types, alkali disease and blind staggers. Alkali disease has been reproduced experimentally and is associated with the consumption of non-accumulator selenium plants. This form is characterised by loss of hair, overgrown hooves and lameness, loss of weight, and reproductive dysfunction. "Blind staggers", as described in the early literature, is believed to be associated with the grazing of selenium accumulator plants and is characterised by aimless wandering, circling, inability to swallow, and lethargy and death may occur. However, there is some uncertainty that these clinical signs are induced by selenium. A blind staggers-like syndrome in feedlot cattle was described by Jensen *et al.* (1956) as polioencephalomalacia (PEM).

Raisbeck (1982) suggested that diets high in magnesium sulfate may induce polioencephalomalacia in cattle. Indeed, polioencephalomalacia was induced in cattle by feeding diets high in magnesium sulfate (Sadler *et al.*, 1983).

Water from saline seeps in the northern plains (USA) caused the death of cattle (Beath, 1982) reportedly because of its high selenium content (ranchers had associated a blind staggers-like condition in cattle with the consumption of this water). Subsequent surveys of these areas did not reveal high selenium in the forage or water, but it was found that the water contained high levels of sulfates.

The purpose of this study was to: (i) determine if the blind staggers described by Beath (1982) could have been induced by the consumption of high sulfate water rather than selenium; and (ii) compare the clinical and pathologic changes of chronic selenium intoxication, produced by selenium treatment or feeding selenium accumulating or non-accumulating plants, with those of sulfate-induced PEM.

Experimental Induction of the Syndrome

Twenty-five Hereford heifers weighing between 200 and 300kg were divided into five groups. One group of eight heifers were fed 140g of ground *Astragalus praelongus* and 9kg alfalfa hay twice daily. The *A. praelongus* was fed by gavage. The four other groups each contained five heifers and were fed as shown in Table 74.1. The sodium selenate and sodium sulfate were fed by gavage. Selenized alfalfa hay was produced by spraying young growing alfalfa hay with a solution of sodium selenite. The solution was calculated to give a concentration of selenium equal to 15 to 25mg kg^{-1} in the final product. The hay was harvested two to three weeks after the solution was applied and fed *ad libitum*. All calves were observed daily for signs of poisoning and were euthanased and necropsied when signs of intoxication became apparent. Tissues from major organs were collected and fixed in 10% neutral formalin and processed for microscopic examination. Serial sections of the brain were viewed with UV light for evidence of necrosis and selected areas were processed for microscopic examination.

Pathology

Results of the pathological evaluation are shown in Table 74.1. The calves fed sodium sulfate developed clinical signs of PEM while calves fed various sources of selenium developed subacute to chronic selenosis.

Table 74.1. Animal treatments and pathologic findings in experimental animals.

Treatment	Animal no.	Amount fed day^{-1}	Duration of trial (days)	Weight change (kg)	Fate of animal	Chronic Se tox	PE	Cardiac necrosis	C.F.V.	E.S.V.
Astragalus praelongus	5251	140g x 2	23	-87	D	0	0	4	0	1
	5261	140g x 2	13	-50	D	0	0	3	0	±
	5264	140g x 2	23	-78	E	0	0	0	1	±
	5272	140g x 2	74	+34	E	0	0	0	1	1
	5274	140g x 2	11	-79	D	0	0	3	0	2
	5409	62g x 2	49	-123	E	0	0	0	0	2
	5410	42g x 2	28	-29	D	2	0	1	0	2
	5411	62g x 2	11	-63	E	0	0	2	1	1
Sodium selenate	5250	147mg x 2	22	-73	E	0	0	1	0	1
	5254	147mg x 2	126	+57	ECSe	4	0	1	0	4
	5260	147mg x 2	126	+29	ECSe	4	0	1	0	2
	5262	147mg x 2	126	+7	ECSe	3	0	4 C	0	1
	5263	147mg x 2	22	-94	E	0	0	0	0	2
Selenized Hay	5252	3.6kg x 2	281	-55	ECSe	3	0	0	0	2
	5256	3.6kg x 2	282	+54	ECSe	3	0	0	0	5
	5259	3.6kg x 2	297	+272	ECSe	3	0	0	0	1
	5266	3.6kg x 2	307	+95	ECSe	2	0	0	0	1
	5270	3.6kg x 2	307	+218	ECSe	2	0	0	0	3
Sodium sulfate	5245	85g x 2	4		E	0	A	0	0	0
	5246	85g x 2	4		E	0	A	0	0	0
	5247	111g x 2	62	+39	S	0	C	0	0	0
	5257	147g x 2	30	-63	E	0	C	0	0	0
	5267	185g x 2	62	+56	S	0	0	0	0	0
Controls	5255	Nil	92	+136	S	0	0	0	0	0
	5265	Nil	92	+99	S	0	0	0	0	±

0-5, Arbitrary assessment of severity of changes (5=severe); D, Died; E, Euthanased; ECSe, Euthanased with chronic selenosis; S, Slaughtered; C, Chronic; A, Acute; C.F.V., Cytoplasmic Foamy Vacuolation; E.S.V., Empty Spongy Vacuolation in brain.

Astragalus praelongus

In the *A. praelongus* group, all calves lost body weight and showed depression and diarrhoea. One calf also showed signs of chronic selenosis including overgrown hooves and rough hair coat. Two calves (5409, 5411) showed signs of mania. Most had mild to severe thoracic effusion and abomasal ulcers. Three calves had distinct pale mottling of the myocardium, and pulmonary oedema. Histologically there was multifocal myocardial necrosis seen in the walls of both ventricles and in the interventricular septum. The brainstem had mild spongiform change that was most severe near the basal ganglia. Three calves also had mild to moderate cytoplasmic foamy vacuolation of thyroid and pancreatic acinar epithelium which is characteristic of locoweed or swainsonine toxicity. Indeed, the *A. praelongus* was analysed and found to have low amounts of swainsonine.

Sodium Selenate/Selenite

The sodium selenate group demonstrated marked weight loss, depression, and several animals developed chronic selenosis. At necropsy, a few abomasal ulcers were present but no significant gross lesions other than hoof and hair changes were seen. Microscopic lesions included myocardial necrosis similar to that seen in the *A. praelongus* group. Extending from the internal capsule area of the anterior brain stem through to the mid-brain there was vacuolation and spongiform change with neuronal pyknosis and multifocal gliosis. These changes were most severe near the basal ganglia.

All calves in the selenized hay group showed signs of chronic selenosis as described in the previous groups. No obvious lesions were seen at necropsy but the microscopic lesions were similar to the other selenium groups. However, the mild vacuolation near the grey matter extended throughout the spinal cord.

Sodium Sulfate

Two of the calves (5245, 5246) given sodium sulfate for four days were necropsied after developing neurological signs. One became depressed, dyspnoeic and apparently blind. It had difficulty walking and often circled blindly. The other animal was found prostrate with laboured respiration. At necropsy, the brains of both calves were swollen and the cerebellum was coned and partially herniated through the foramen magnum. Both brains, under UV light, had extensive bilateral focal areas of laminar necrosis seen as fluorescence of the cerebral cortical grey matter. Microscopically, the affected brains had extensive areas of laminar cerebrocortical liquefactive necrosis, accumulation of Gitter cells, fine vacuolation of the adjacent neuropil, vacuolation of glial cells and nerve cells, and necrotic nerve cell bodies. There was also mild vascular proliferation with scattered eosinophilic infiltrates. These lesions extended from the frontal pole to the occipital pole and were restricted to the cerebrocortical grey matter.

Both calves had a mild meningitis with accumulations of mononuclear inflammatory cells and eosinophils in the perivascular meninges.

A third calf (5257) developed clinical signs of depression, as indicated by drooping ears and lethargy, and diarrhoea four days after the initial dosage, but made an almost complete recovery with minimal neurological signs. Upon necropsy, the brain had focal areas of chronic necrosis seen as focal fluorescence in the cerebral cortex under UV light. These areas could be seen grossly as focal areas of cerebrocortical discolouration and partial cavitation. Microscopically, there were changes similar to the first two animals with increased liquefaction and cavitation of the grey matter, proliferation of blood vessels and extensive accumulation of phagocytic cells. Several areas of acute coagulative necrosis were also present in the superficial laminae. This calf also had active Wallerian degeneration of the optic pathways from the eye to the optic tracks in the brain.

Calves 5247 and 5267 showed no clinical signs and were necropsied after 62 days on treatment (Table 74.1). Interestingly, calf 5267 had microscopic lesions of chronic cerebrocortical necrosis similar to calf 5257, while calf 5247 was normal grossly and microscopically.

Conclusions

The results reported in this chapter suggest that: (i) regardless of the source, chronic selenium ingestion by cattle results in a disease similar to selenosis, i.e. hoof and hair lesions, myocardial necrosis and mild spongiform change in the spinal cord and brainstem; and (ii) sodium sulfate intoxication in cattle rapidly results in clinical signs consistent with those described in "blind staggers" (PEM).

Although PEM is a non-specific lesion and can be caused by a variety of toxic and nutritional diseases, these data suggest that the PEM associated with selenium intoxication or "blind staggers" is actually a result of sulfate intoxication.

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