Selenium toxicity in horses following over application of sodium selenate to pasture: a case study

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Case presentation

Four horses on one Hawke's Bay property presented with lameness on 16 August. Two of the four had been seen a month prior with similar symptoms. Of the two initial cases, one was diagnosed with laminitis and removed from pasture and the other with solar bruising. Both made an initial recovery in the weeks following the first visit but relapsed at the same time as the others presented.

History

All horses had been fed a mixture of grass, hay and small amounts of supplementary chaff. They were being fed an oral multi-mineral supplement intermittently. They had intermittent break feeding access to a small part of a hill paddock, and the owner had noted that exposure to this pasture had precipitated one of the horse's signs, leading to an initial diagnosis of laminitis. They had however all been grazing this pasture to some degree over the previous few months.

There were also trade lambs on the property, of which half had been set stocked in the hill paddock and half on bottom flat paddock, along with six other horses and 44 dairy heifers set stocked elsewhere on the property. The only notable animal health events for these stock groups were 13 deaths of suspected clostridial origin in the lambs shortly after a period of confinement and shearing before being let out onto the bottom flat paddock.

Clinical findings

Of the four lame horses presented on the initial visit, three of the four had swollen, oozing and cracking coronary bands with increased digital pulses and lameness in at least one foot. None had any response to hoof testers.

Of the two more chronic cases, (which presented initially a month prior) one that appeared to have solar bruising now had visible cracks on the dorsal hoof wall 5mm distal to the coronary band on both front feet. The other horses had swollen coronary bands on all four feet with serous ooze from cracks in the coronary bands. Two of the four horses had very thin, brittle soles. None of these horses had increased temperatures, reaction to digital pressure with hoof testers or any systemic signs of illness. One had a patch of missing hair over the point of the elbow but otherwise there were no further integumentary changes noted at the time.

Radiographs were taken of front feet of the worst affected horse (Horse La) at the initial visit, but there was no noted rotation of P3 or separation of the hoof wall at this time. Pending laboratory testing, all horses were removed from grass and placed on hay ration with restricted exercise. They were initially treated with the non-steroidal anti-inflammatory drug (NSAID) phenylbutazone to try to control their lameness.

Testing

Blood was taken from the three horses showing similar signs to be run for whole blood selenium and haematology. The selenium results were as displayed in the first results column of table 1.

Table 1. Whole blood and serum selenium results from five horses on the property taken between 16 August 2021 and 19 November 2021.

	Blood (nmol/L)			Serum (nmol/L)
	16 Aug 21	27 Aug 21	8 Nov 21	19 Nov 21
Horse J (Euthanised)	11000	17000	10332	
Horse La (Euthanised)	11000	14000	9567	
Horse T (Survived)	5900		6147	2100
Horse C (Survived)			13969	2500
Horse L (Survived)		23000		
Range	1500–3300			360–1700

Further information

Upon diagnosis of selenium toxicity, further information came to light to reveal the underlying cause. The day prior to the blood results being returned, the owner had revealed a gross over-application of sodium selenate to pasture on a section of the property, 65 times the recommended dosage of 10g/ha. Pasture selenium levels, tested eight weeks post-application, reached 13.6mg/kg dry matter (DM), far exceeding the normal range (up to 1mg/kg DM). Livestock health and pasture management are discussed in the sheep and beef practitioner paper proceedings.

Treatment

All animals were immediately removed from affected pasture. Horses with clinical signs were moved into flat yards with recommendations to source deep, loose sand bedding. Clinically lame horses were continued on NSAID therapy and started on biotin hoof supplements to encourage new hoof growth.

Within the following three months, two of the five horses were euthanised due to pedal bone rotation causing uncontrollable pain. This was despite supportive treatment, pain relief and intensive farriery work. Of the remaining three horses, one never required supportive treatment and the other two had a high degree of farriery intervention, ultimately preventing pedal bone rotation. These two horses, Horse C and Horse T, are clinically unaffected at the time of publication.

Farriery

Farriery interventions were centred around laminitis management providing sole support while also reducing the pull of the deep digital flexor tendon on the pedal bone. All interventions were also aimed at increasing perfusion of the sole to encourage foot growth and increase the sole depth. Horses were casted into heel raises with the soles supported by high density foam.

In the case of Horse La and Horse J, there was not sufficient reduction in pain following the first application of the casts and they were euthanised a few days later. These two horses had radiographic evidence of pedal bone sinking prior to the placement of the casts.

Horse T and C both had their casts replaced once at two weeks and then removed at four weeks. They were then managed with repetitive trimming designed to reduce leverage on the laminae dorsal to the pedal bone. Due to the increased rate of hoof growth, these visits were initially every 1-2 weeks before shifting to every five weeks. Both horses were transferred onto ‘routine’ farrier management by early July 2022, when their hooves had completely grown out, eight months after the initial casting visit by the farrier.

Discussion

This case details the chronic selenium toxicity of five horses, all with varying response following similar exposure to toxic levels of selenium in pasture. A 500kg horse eating 2% of its body weight in dry matter daily, could have been taking in 136mg of Se per day if the pasture selenium levels were 13.6mg Se/kg DM. This far exceeds the reported maximum safe amount of 20mg Se per day (National Research Council 2007).

Selenium toxicity occurs in both acute and chronic forms. It is believed that acute toxicity leads to oxidative tissue damage and cell death, where-as the signs arising due to chronic toxicity may be due to the incorporation of selenium into methionine group amino acids in place of sulphur, altering the structure of these proteins that are important in the formation of keratin (Wilhelm 2010).

In this case, the horses displayed different tolerances to the selenium exposure, which highlights the complicated nature of susceptibility to selenium toxicity. Horse L had the highest serum selenium reading but remained clinically normal throughout the period of the investigation. Horse T was one of the worst affected clinically on initial diagnosis with visible defects in the hoof wall at the level of the coronary band. However, on radiographs there was no rotation or sinking of the pedal bone present and they have ultimately survived to the time of publication. Horse C received intensive farriery treatment but never had pedal bone rotation that progressed past five degrees.

Of the two horses that were ultimately euthanised, Horse La and Horse J, both were clinically painful despite consistent NSAID, paracetamol and intensive farriery. Horse La initially did not have radiographic changes, but within three months of the initial visit, rotation and sinking were severe with the pedal bone approaching the sole of the foot and pain was not able to be controlled. This horse developed bed sores despite being in deep sand bedding.

Horse J was one of the first horses with clinical signs in July, one month after selenium application and quickly following re-introduction to the pasture. There was some clinical improvement when in deep sand but ultimately by the time foot casts were able to be placed there was already severe pedal bone rotation and sinking present and the clinical response to casting wasn't sufficient. Euthanasia was performed with the continued welfare of the horse in mind.

The partnership between the owner of the animals, the farrier and the team of veterinarians allowed for intervention to be sought, which may have prevented the further pedal bone rotation in the case of Horse T and Horse C. It also highlighted the importance of radiographs in forming a full clinical picture of laminitic horses, especially in the face of severe clinical signs. With the benefit of hindsight, once Horse L and Horse J had radiographic changes to the degree they did, a call for euthanasia could have been made earlier, preventing further unnecessary pain.

This case highlights the importance of piecing multiple parts of a clinical picture together and adopting a whole farm approach to diagnosis. The clinical picture with the horses directly led to an investigation into livestock and ultimately saved potentially devastating effects on the downstream food chain that are discussed further in the proceedings for the sheep and beef stream.

References

National Research Council. *Nutrient requirement of horses. 6.* National Academies Press: Washington, DC, USA; 2007

Wilhelm A. *Investigation of the Toxicity and Toxicokinetics of Selenium from the Accumulator Plant Symphyotrichum spathulatum (Western Mountain Aster) in Sheep.* Utah State University; 2010