FACTORS AFFECTING THE INCIDENCE OF THE PE "SUDDEN DEATH" FORM OF *PHALARIS AQUATICA* POISONING IN SHEEP.

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Abstract

PE "sudden death" is one of three unrelated forms of phalaris poisoning. It has probably occurred in sheep grazing phalaris dominant pastures for many decades, but was not recognised as a specific problem until 1986. It is characterised by the sudden death of large numbers of animals within 12 to 48 hours of their being moved onto a toxic phalaris pasture. It has occurred in several states and tends to be an annual problem in those parts of Australia that have a Mediterranean-type climate. It has occurred on several different cultivars of phalaris. The incidence of PE ‘sudden death" appears to be associated with the following factors: phalaris domination of the pasture mix, moisture stress of the phalaris, repeated frosting of the phalaris, the commencement of the new seasons growth, light patchy rainfall during an otherwise drought period, very short re-shooting phalaris plants, deferred grazing management practices, high stocking rates, insufficient time for sheep to adapt to a toxic pasture, rapid ingestion of the pasture by sheep, and feed deprivation immediately prior to the sheep being placed on the pasture.

Key words: Phalaris, polioencephalomalacic, poisoning, sheep, death.

Introduction

The phalaris toxicity complex comprises three unrelated forms of poisoning. The first is phalaris staggers, the second is cardiac 'sudden death', and the third is PE (polioencephalomalacic) 'sudden death' (1,2,3). Phalaris staggers can affect sheep or cattle and cause nervous symptoms which persist for days, weeks or sometimes months (1,2). Cardiac 'sudden death' can affect sheep (3) and horses (unpublished data). It causes small numbers of animals to suddenly collapse and die when a flock is mustered or disturbed in some other way. PE 'sudden death' affects sheep (3) and results in large numbers dying overnight, within 12 to 48 hours of being moved onto a paddock of phalaris. PE 'sudden death' has been responsible for many outbreaks of phalaris poisoning in NSW, it was first recognised as a distinct syndrome by Bourke and Carrigan (3) following an outbreak at Dubbo NSW in 1986. Outbreaks of PE 'sudden death' have occurred in several states, notably at Geelong in Victoria, Naracoorte in South Australia, and Esperance in Western Australia. PE ‘sudden death’ is as significant a cause of stock losses on phalaris pastures as phalaris ‘staggers’.

The toxin in phalaris responsible for PE ‘sudden death’ is unknown, however the rapid way in which this form of PE develops would suggest that the toxin functions as a direct antagonist of either thiamine or pyridoxine. This could help explain how it is able to act so quickly in compromising the integrity of the blood vessels supplying the central portion of the brain. Failure of these blood vessels to adequately supply oxygen and nutrients to the brain results in rapid brain degeneration, and consequently rapid death.

The following case reports highlight the factors that are frequently associated with outbreaks of this form of phalaris poisoning, an understanding of these factors should ensure management practices are put in place that will minimise the occurrence of this problem.

Results

Case 1. 'Carinya' Ballimore via Dubbo, Central NSW

Phalaris poisoning occurred on a series of small paddocks (6 to 10 ha each) on an alluvial sandy loam soil along a river flat. Soil pH was 5.0 to 5.5. The area had been sown to Sirocco phalaris and was being

managed on a tight rotational grazing system using Merino sheep. The average annual rainfall in this district is 600 mm, and their is no seasonality of rainfall distribution.

The outbreak of 'sudden death' occurred in 1986 which was a drought year. At the end of a day, in late May,1,500 Merino wethers were moved onto a drought affected, frost affected, phalaris dominant paddock. The phalaris pasture was very short but had started to re-shoot in response to a very limited recent fall of rain (8 mm). The phalaris was stocked at 250 sheep/ha. Next morning 4 sheep were recumbent. By that afternoon a number of sheep were lying on their side convulsing. Their limbs were stiff, they were paddling, frothing at the mouth, stretching their necks backwards and had flickering eye movements. In addition about 30 sheep were dead, and by the following day this had increased to 100.

Four weeks later (late June) the drought conditions continued and a mob of 2,500 Merino hoggets were put onto another phalaris pasture, again at about 250 sheep/ha. The phalaris was up to 10 cm high but sparse. Much of the phalaris consisted of dry butts, many of which were pulled up by the sheep and eaten. On the first day they were left on for 3 hours, and on the second day 1 hour, without any problems. On the third day they were put on the phalaris paddock in the morning and were all well when checked 6 hours later. However the following morning 200 were found dead and another 30 were sick. Most of these affected survivors died over the following 24 hours. Their symptoms included depression, mild head tremors, fine body tremors, aimless wandering, apparent blindness, head pressing, a high stepping gait, ataxia, star gazing (opisthotonus), and lateral recumbency with either convulsions or coma. The diagnosis of rapid onset PE was confirmed by the microscopic examination of brain tissues from affected survivors and dead sheep.

Case 2. 'Darrawill North' Geelong, Victoria

The phalaris cultivars Sirosa and Australian were sown on a farm about 15 km west of Geelong. From late 1996 to late 1997 was a drought period in this district. In late May of 1997, a group of approximately 570 sheep were put onto a phalaris dominant pasture, 60 hours later, 122 were found dead and one comatose. The pasture was almost pure phalaris, the plants were about 4 to 6 cm in height and the growth described as sparse, moisture stressed, and frost affected. A diagnosis of PE "sudden death" was confirmed by the results of the microscopic examination of brain tissues from the comatose animal and several dead sheep. A number of other outbreaks of this form of phalaris toxicity occurred on other farms in the Geelong district at this time.

Case 3. 'Waverley' Naracoorte, South Eastern South Australia

The portion of this property involved in outbreaks of phalaris poisoning consisted of a well maintained Holdfast phalaris stand harvested annually for the production of certified seed. The soil type was a black sandy loam, over red clay and ironstone. The site was a slightly elevated, well drained ridge. Soil pH was originally 5.5, but use of alkaline irrigation water had lifted it to 6.5. The pasture was kept very pure and free of all other plants. The dry phalaris trash along each phalaris row was removed by burning. The average annual rainfall in this district is 600 mm and the seasonality of rainfall distribution winter-spring (June to Nov). December to May is usually hot and dry. Supplementary irrigation is used on the phalaris seed block to ensure adequate consistent growth, and adequate seed set and seed development, consequently it is normally only used in the mid to late growing season.

When Merino sheep were given access to this block they were only able to graze pure fresh green phalaris shoots. The sheep were managed on a loose rotational grazing system. The stocking rate was high, and grazing was carried out for short periods only, to control the rate of development of the phalaris plants during the early to mid growing season period. In 1996 problems with 'sudden death' occurred in sheep on this property during the March to June period, when phalaris growth was erratic because of the unreliability of rainfall at that time of the year. Affected sheep were either found dead, or observed to develop symptoms and then to die, during the first 12 to 48 hours after being moved onto the phalaris paddock. The symptoms displayed were as follows: sheep became depressed, stopped grazing, stood about, gradually developed tremors and twitches, then went down into lateral recumbency. Some

developed limb rigidity, paddled, frothed at the mouth and died, while others became comatose before dying. These clinical signs are typical of those normally displayed by sheep affected by PE prior to death.

Case 4 'Oak Marsh Farm', Esperance, Southern Western Australia

The portion of this property involved in phalaris poisoning consisted of 200 ha divided up into 35 ha paddocks and sown down to Sirolan phalaris. These pastures were rotationally grazed by Merino sheep and a portable electric fence was used to facilitate a strip grazing system within each paddock. The stocking rate used was nominally 14.7 DSE/ha. The moveable strip fence created an effective stocking rate of 131 DSE/ha. The average annual rainfall in this district is 600 mm and the seasonality of rainfall distribution is winter- spring (June to November). December to May is usually hot and dry. The soil type was sand over clay, typically 25 to 50 cm deep. The sandy topsoil was water repellent and had a pH of 4.5 to 5.5. The water table was high, water logging was a problem during winter, and salinity was developing.

Outbreaks of 'sudden death' had occurred every year in sheep on these pastures since they were first sown in the winter of 1989, and up until the phalaris was removed by herbicide application in the winter of 1996. These outbreaks always occurred between December and May. That is they occurred when the pastures were phalaris dominant, and when that phalaris was suffering from heat and moisture stress. Usually the phalaris pasture was about 15 to 20 cm high when grazed, and consisted of fresh green and short regrowth plant material The outbreaks commonly occurred about 4 weeks after an isolated fall of rain. Sheep losses ranged from 2 to 51%, with an average figure of 15%. Symptoms of poisoning would first develop within 8 to 12 hours of the sheep being given access to a fresh strip of phalaris pasture, and deaths would follow within 24 to 48 hours. The symptoms observed in affected sheep were as follows: disorientation, wandering away from the mob, depression, walking into electric fences, tremors and twitches, collapse into lateral recumbency with limb paddling and foaming at the mouth, increasingly laboured respiration, convulsions and death. These are the clinical signs associated with PE 'sudden death..

Discussion

The following factors may have contributed to the mortality at Dubbo. The sheep were very hungry when put on the phalaris pasture. The pasture was predominantly phalaris. The phalaris was drought affected and frost affected, but re-shooting after a very limited amount of rain. The sheep were being rotationally grazed using very high stocking rates. The sheep consumed a lot of plant material in a short period of time. It is noteworthy that prior test grazing of several hours duration gave no indication of the potential toxicity of this phalaris pasture.

The following factors may have contributed to the mortality at Geelong. The previous summer had been very dry and the autumn break had arrived late, with only light patchy falls of rain recorded. As a consequence the new seasons growth of phalaris had been slow to come away and this may have meant that it contained a large amount of toxin. There was moisture stress and frost stress upon the pasture. The sheep were grazing new season phalaris shoots for the first time that year and had not had enough time to adapt to them, if the level of toxin in the plant had not been high, then possibly this adaptation process would have occurred without incident. The plant stress associated with the very dry seasonal conditions may have favoured the production of elevated amounts of toxin by the plant.

The following factors appear to have contributed to the mortalities at Naracoorte. The stand of phalaris was pure, the stage of growth grazed was new short green shoots, the time of the year when losses occurred was usually warm and dry but with occasional patchy falls of first seasonal rains. The sheep would have gone onto the phalaris whilst still very hungry (since they were being rotationally grazed and the stocking rate was high), consequently they may have consumed a lot of plant material in a short period of time.

The following factors appear to have contributed to the deaths at Esperance. The stand of phalaris continued to grow when all other pasture species in the mix had ceased to grow and had been eaten out.

The pasture was therefore pure phalaris. The stage of growth being grazed was new short green shoots, the time of the year was dry but there had been occasional light falls of rain that were enough to maintain some phalaris growth. The sheep were being managed under a very tight strip grazing rotational system, this ensured that they were hungry prior to grazing each new strip. In addition, the very high stocking rate would have ensured that each sheep ate ravenously when offered a new strip, in order to obtain fresh green shoots before the others.

Conclusion

The phalaris toxin responsible for the PE form of 'sudden death' is unknown. However it is clear from the above four case studies that it occurs in several phalaris cultivars including: Sirocco, Holdfast, Sirolan, and either Sirosa and or Australian. The level of toxin in the plant appears to increase greatly in response to adverse seasonal conditions, notably soil moisture stress, and possibly frost damage. The amount of toxin seems to be greatest in the new seasonal growth. Set stocked sheep seem capable of successfully detoxifying this phalaris toxin, presumably because they are able to increase their intake of it slowly. It would appear that it is the sudden, rapid ingestion of large amounts of phalaris plant material, at a time when the levels of toxin are rising, or are already high, that kills sheep. Pasture management systems which involve deferred grazing seem to strongly favour outbreaks of phalaris PE ‘sudden death’. Pastures that normally contain a mixture of plant species may experience seasonal periods of phalaris dominance, typically immediately following a very dry summer, and should be grazed with caution during these periods. Test grazing periods of less than 12 to 24 hours will give no indication of the potential toxicity of a phalaris pasture.

References

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