Poisoning of lambs on mature Bambatsi panic (*Panicum coloratum*) pasture

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Abstract: *In March 2018 the author investigated the deaths of 140 from 1400 Dorper lambs in the Wee Waa district of New South Wales. The deaths occurred after the lambs were introduced to tall, mature Bambatsi panic (*Panicum coloratum*) dominated pasture. The lambs had clinical signs, gross pathology and liver histopathology consistent with saponin toxicity. Photosensitisation was largely absent. Ewes also grazing the pasture were not affected. While the pasture was flowering at the time of the poisoning it is suggested that the lambs preferentially grazed new shoots from recent rain. Grazing management recommendations to avoid similar pasture circumstances were implemented with no further cases in the following 12 months. The lack of photosensitisation and age-related susceptibility is discussed.*

Key words: saponins, photosensitisation

Introduction

Steroidal or lithogenic saponins are a relatively common plant toxin poisoning of ruminants in New South Wales (NSW). Common sources of saponins in NSW include *Panicum* and *Tribulus* spp. Regault (1990) reports a case in sheep grazing Bambatsi panic (*Panicum coloratum*) in the Goondiwindi region of Queensland. McKenzie (2002) lists both Bambatsi panic and Gatton panic (*Megathyrsus maximus*) as sources of saponins, and Bambatsi panic as an important species for poisoning. Both of these grass species are commonly sown in pasture mixes in northern NSW (Harris *et al*. 2014). Saponins cause crystal-associated cholangiohepatopathies and secondary photosensitisation. McKenzie (2002) indicated poisoning commonly involves young ruminants, especially lambs grazing stressed pastures or crops.

This paper reports an investigation into saponin poisoning in black-headed Dorper lambs following introduction to mature Bambatsi panic dominated pasture on a property in the Wee Waa district of NSW in March 2018.

Pasture description

Two adjacent 30 ha paddocks were sown in March 2017 with a Bambatsi panic, Gatton panic and Premier digit grass (*Digitaria eriantha*) mix. Due to conditions at pasture establishment, by the time of poisoning, the pasture consisted of predominantly Bambatsi panic. A small area of *Tribulus* spp. was present in one paddock but largely absent in the other.

At the time of poisoning the pasture was mature, 40–70 cm high and setting seed. Examination of the paddocks showed heaviest grazing of the Gatton panic, followed by Bambatsi panic. The digit grass was least grazed. These observations suggest the sheep had preferentially grazed the Gatton panic, then the Bambatsi panic. In the one paddock where *Tribulus* spp. was present, this had been heavily grazed suggesting that it had also been selectively grazed by the sheep. Perhaps critically, at the time of introduction, there had been 40 mm of rain. As a result, all grasses were actively growing, and new green shoots developing. At the time of paddock inspection, this green regrowth from the base of the plants appeared to have been preferentially grazed. The older longer leaves were generally ungrazed.

Case presentation

The black-headed Dorper lambs were from two mobs, grazing the two adjacent paddocks. One mob consisted of 500 weaned lambs aged 4–8 months. The other mob consisted of 700 Dorper ewes and 900 unweaned lambs aged 4–8 months. The history and grazing management of both 30 ha paddocks was similar and both mobs were introduced to their respective paddocks on the same day.

Deaths were first noticed 10 days after introduction to the pasture with 24 dead lambs. When the deaths commenced was unclear, as the mobs had not been checked since introduction. However, the degree of carcass decomposition suggested the first deaths had occurred 5–8 days after introduction. The sheep were immediately removed from the pasture.

At 13 days after introduction 50 lambs had died and another 80–90 were clinically affected. By 20 days after introduction 90 lambs were dead and 40–50 clinically affected. Deaths continued for two months and eventually almost all clinically affected lambs died (c. 140 lambs). All dead or clinically affected lambs were in the initial clinically affected group. Both mobs of lambs were affected at similar rates. No ewes were observed with clinical signs.

A property investigation was undertaken when deaths were first observed. This included an examination of clinically affected lambs, an autopsy and paddock inspection. Treatment consisted of immediately removing all sheep from the paddocks and placing the lambs in a well-shaded paddock with only dry grasses. The ideal treatment of removing from any direct sunlight was not possible due to the absence of a suitable shed.

Clinically affected lambs showed lethargy, shade seeking and yellowing of mucous membranes. Photosensitisation was not a prominent feature (possibly related to protecting black skin).

The autopsy found extensive yellowing of all tissues and a swollen orange liver. Tissue samples were submitted to the NSW Department of Primary Industries State Veterinary Diagnostic Laboratory. Liver histopathology was consistent with saponin poisoning but pathognomic crystals were not identified. This was potentially due to slight autolysis of samples. No other possible causes of the clinical signs and gross pathology were identified.

Discussion

The classic reported conditions for saponin toxicity of Bambatsi and other panic pastures are grazing short pasture that is actively growing, especially if it subsequently becomes moisture stressed (McKenzie 2002). These were the circumstances for an earlier poisoning incident (10 dead, eight clinically affected from 220 Dorper lambs) that occurred on the same paddocks in December 2017. To manage the risk of future poisonings the owner elected to restrict future grazing by lambs to only mature pastures.

In contrast to these classical circumstances, the March poisonings outlined in this paper demonstrated that poisoning of lambs grazing flowering stands of Bambatsi panic was possible. The observations suggested poisoning can occur if the pasture was actively growing and lambs were able to preferentially graze regrowing shoots. It also demonstrated that poisoning can occur without the pasture showing visible signs of moisture stress.

The role of Gatton panic in this poisoning incident was difficult to quantify. While preferentially grazed, Gatton panic plants were less than one in 20 plants, and as such would have been a small part of the diet. McKenzie (2002) did not categorise Gatton panic as an important species for poisoning. In contrast, Bambatsi panic was categorised as an important species and it was the major component of the grasses observed to have been grazed.

To manage the risk of future poisonings on Bambatsi panic dominated pasture, the following measures were recommended:

- Only graze lambs on mature pasture that is not actively growing;
- Remove sheep from the pasture following rainfall;
- Initially, graze actively growing pasture with ewes to remove green shoots;
- Not grazing lambs until 2–3 weeks after any rain; and
- Grazing lambs at high stock densities to reduce the potential for lambs to preferentially graze any new green shoots.

These measures were successful in preventing poisonings for the following 12 months, but this coincided with severe drought conditions when there were few rainfall events.

The absence of photosensitisation as a prominent clinical sign in a case with significant mortalities was unusual but consistent with the current understanding of secondary photosensitisation as a result of hepatic damage from toxins. This finding suggests it is not photosensitisation that kills most lambs with saponin poisoning but rather liver damage. Moreover, in this case,

the black pigment covering the head of black-headed Dorpers most likely protected the lambs from photosensitisation.

Dowling and McKenzie (1993) summarise photosensitisation as the heightened sensitivity of the skin to sunlight. Secondary photosensitisation occurs when liver damage interferes with the liver's ability to deal with phylloerythrin, a breakdown product of chlorophyll. Phylloerythrin then escapes into the circulatory system and lodges in the skin making it sensitive to sunlight.

Seawright (1989) indicated that this sensitivity damages cellular membranes with enhanced capillary permeability, cell necrosis, vascular occlusion and acute inflammation. The areas of sheep most affected are the ears, eyelids, face lips and vulva. Reddening and oedema result, with rubbing and shade seeking. In lambs, ears droop and the muzzle is swollen. Necrosis of skin can be seen in severe cases.

Bourke (2011) indicated that severity of photosensitisation is not related to individual or species differences in metabolising the offending compounds. Rather tolerance is related to an animal's ability to prevent sunlight from reaching the blood vessels in its skin. Thus variation in tolerance is related to skin thickness, thickness and density of hair or wool coat and the degree of pigmentation in the skin.

The lambs, in this case, were black-headed Dorpers a breed with deep black pigmentation of the head and upper neck. This pigmentation likely prevented the most sensitive areas from being affected with the photosensitisation that is typical of saponin poisoning. However, shade seeking was a prominent feature of the affected lambs.

Further, death in secondary photosensitisation cases is not usually caused by the photo-sensitisation but rather the damage to the liver by the toxin (Seawright 1989). Thus in this case, while the lambs showed few signs of photosensitisation besides shade seeking, the underlying liver damage from the saponins was sufficient to cause death.

The susceptibility of lambs and the absence of poisoning in adult sheep with *Panicum* spp. was noted by Button *et al*. (1987). They also indicated that age-related susceptibility has been used as a management tool for grazing of *P. coloratum*. In contrast, in both the author's experience and Button *et al*. (1987) saponin poisoning from *Tribulus* spp. affects sheep of all ages.

The literature fails to explain this strong age-related susceptibility. In general, younger sheep are more susceptible to toxicities due to differences in ruminant metabolise and general resilience. However, this does not explain the strict immunity of adult animals to *Panicum* spp. poisoning. The selective grazing, in this case, raises the possibility that differences in grazing behaviour may contribute to this age-related susceptibility.

Overall the risk factors for poisoning, in this case, are likely to have been actively growing pasture, the potential for selective grazing of new shoots and grazing of lambs. The grazing management measures outlined earlier may be sufficient to manage these risks, and thus future poisonings on *Panicum* spp. dominated pastures.

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