Walkabout disease in horses

Introduction

'Walkabout' disease, also known as *Crotolaria* poisoning and Kimberley horse disease, affects horses that graze plants of the genus *Crotalaria*. These plants are commonly known as 'rattlepods' and 'Gambia peas'. There are 25 recorded species of *Crotalaria* in the Northern Territory. However, only limited information is available on the potential toxicity of individual species for horses. Therefore, owners should assume that all *Crotalaria* plants are toxic to horses.

Disease occasionally occurs very soon after a horse eats a large amount of *Crotolaria* (acute cases). More commonly, the onset of clinical signs progresses slowly over years, when horses graze the plants in small amounts over time (chronic cases). Walkabout disease is a common cause of mortality in horses in the Territory.

Known toxic species

Crotolaria species are native to Africa. The plants were introduced to Australia as a green manure crop and are now considered a naturalised weed across Northern Australia. They are short-lived annual species that grow commonly in disturbed areas of pasture, including open woodland and grasslands, floodplains and riverbanks in tropical and subtropical regions.

The following *Crotalaria* species are common causes of walkabout disease in horses in the Territory. These images show the wide range in foliage between species, from upright shrubs to ground-dwelling plants.

- *C. crispata* (Kimberley horse poison)
- *C. dissitiflora* (grey rattlepod)
- *C. novae-hollandiae*
- *C. ramosissima*
- *C. retusa* (wedge-leafed rattlepod)
- *C. trifoliastrum*

Figure 1: *Crotalaria* species commonly found in the Territory

C.novae-hollandiae

C.trifoliastrum



C.trifoliastrum

C.crispata

C.crispata

More images and information to identify Top End *Crotolaria* species are available on the NT Herbarium website.

Causes of toxicity

Crotolaria contain toxic substances called pyrrolizidine alkaloids (PAs) that cause liver damage. When horses consume plants containing PAs, the toxic effects are cumulative. If a horse has limited but intermittent access to plants that contain PAs, over time progressive liver damage may cause clinical disease and death. Once PA toxicity affects more than 70% of the liver, the organ is unable to perform normal, vital functions.

The healthy liver works to detoxify chemicals that are normal by-products of digestion, such as ammonia. However, the intoxicated and damaged liver is unable to metabolise and then remove these chemicals from the circulation, so the dramatic signs of 'walkabout' may develop. Stumbling, frenzied galloping, head pressing and collapse are the poisonous effects of ammonia and other metabolic toxins on the brain. This is called hepatic encephalopathy, and is secondary to the initial liver toxicity caused by consuming PAs.

Risk factors

Crotalaria species are not very palatable, so consumption generally occurs only when there is a shortage of edible pasture, or when there is insufficient manufactured roughage provided (such as hay or chaff) causing horses to graze indiscriminately. Poisoning may also occur if *Crotalaria* plants and seedpods are accidentally incorporated into hay.

Horses are 30 to 40 times more susceptible to the toxic effects of PAs than sheep and goats. Cattle are also susceptible, but toxicity is rarely reported in the Territory.

Clinical signs

- Anorexia (off feed)
- Weight loss
- Jaundice (gums and the whites of eyes have a yellow or peach-coloured tinge)
- Horses become dull and depressed
- Muscle tremors, especially of the head and neck
- Frequent yawning
- Head pressing
- Urine may be copper coloured or red
- Attacks of frenzy and violent, uncontrollable galloping
- Difficulty swallowing, horses may stop eating halfway through a mouthful of hay or grass
- Horses often stand with their heads hanging down
- Affected horses appear to be blind and may aimlessly wander, walking in circles or bumping into objects and head-pressing (hence the name 'walkabout disease')
- Dragging of the hind legs, causing the hooves to have worn tips

Diagnosis

A presumptive diagnosis in a live horse is based on clinical signs, blood tests, and known or suspected exposure to the toxic plants. Blood samples from recently affected horses will usually have greatly elevated liver enzymes, particularly one called gamma glutamyltransferase (GGT). This reflects acute liver damage caused by PA toxicity. However, with chronic, long-term exposure, liver cirrhosis occurs. The normal structure of the liver is replaced with fibrous scar tissue, resulting in gradually decreasing function. Blood test results from these horses may show relatively mild changes in liver-specific enzymes and low serum protein because the output of normal products drops when liver function is severely disrupted.

Examination of a liver sample from a horse that has died or been euthanised because of walkabout disease will show distinctive changes typical of PA poisoning. The presence of *Crotalaria* plants in the pasture combined with typical clinical signs and liver damage is highly suggestive of walkabout disease.

In sudden-onset (acute) cases, the liver may be enlarged and fragile to touch, breaking apart when handled. In longer-term (chronic) cases, the liver is shrunken, thickened and has a rubbery texture, owing to replacement of normal liver with scar tissue.

Vital functions of the liver, such as making blood proteins and clotting factors, are interrupted when the liver fails. When there is low protein in the circulation, there may be swelling of abdominal tissues. Small patches of bleeding may be visible in various tissues because the liver is no longer producing enough blood-clotting factors. Jaundice (yellowing) may be visible in all pale body tissues, such as fat and intestinal walls.

Treatment

Liver damage caused by PAs is not reversible. Anecdotal evidence suggests that horses may survive many years with reduced liver function, but may struggle to gain or maintain weight. Some owners choose to use injectable supplements to support liver function, such as Di-isopropylamine Dichloroacetate (DADA) combined with amino acids, as well as a highly digestible but low protein diet to avoid overwhelming the horse's metabolic capacity. Consult your private veterinarian to develop a monitoring and treatment plan for managing long-term liver failure in affected horses.

Prevention

Preventing horses from accessing *Crotolaria* plants is the best protection. During periods of feed shortage, remove horses from paddocks that contain *Crotalaria* plants and feed good quality hay or a combination of hay and grain. If possible, remove all *Crotalaria* species from horse paddocks mechanically, or by using appropriate herbicides.

Ensure that purchased feed is free of contaminants. Insist on machinery wash-downs prior to property entry to avoid introduction of seeds. New horses entering the property should be kept in a separate area such as a yard for at least 4 days before introduction into large paddocks. This allows any seeds to pass through the animal in a confined space so they will not contaminate pasture.

Other sources of toxicity

PAs occur in a number of other plants, including:

- *Senecio sp*. Common names Groundsel, Yellowtop, Rubbish Daisy, native to central Australia
- *Heliotropium sp*. Native to the Tanami and central Australian regions
- *Amsinckia sp*. Common name Hairy Fiddleneck, weed found in central Australia
- *Echium sp*. Common name Paterson's Curse, weed found in central Australia
- *Trichodesma sp*. Common name Cattle Bush, Camel Bush, native found Territory-wide
- *Cynoglossum sp*. Common name Australian Hounds Tongue, native to central Australia

Like *Crotolaria*, most of these plants are not very palatable so animals are unlikely to eat them by choice, unless there is very scarce feed available. Additionally, these species tend to grow sparsely or in isolated pockets so opportunities to graze a toxic quantity are limited. Use the NT Flora website to assist with identification of suspicious pasture plants, or contact your regional livestock biosecurity or weeds officer for further information.

Consumption of *Trichodesma sp* has also been linked with a separate syndrome of toxicity, where affected horses lose weight over a period of time and then develop sudden onset respiratory signs, shortly before dying. Post-mortem examination of these horses reveals extensive lung changes with fibrosis. *Trichodesma sp* plants should be removed or controlled in pasture or yards where horses are kept intensively and where there is little other grazing available.

Toxicity for other stock

Cattle are also susceptible to PA toxicity, but there are age-related differences in sensitivity. Calves are more sensitive and more likely to develop liver disease than older cattle. Cattle are less sensitive than horses, but more sensitive than sheep and goats.

Similar clinical signs occur in cattle and horses. Early signs of disease in cattle may include depression, decreased appetite, diarrhoea and wasting. When liver function fails, cattle will present with nervous system signs including staggering, aimless wandering, collapse and death.

PA toxicity in cattle, while being a well-described condition in Australia and New Zealand, is rarely reported in the Territory. This may reflect different grazing habits of cattle compared with horses, or may reflect different species susceptibility to PA-containing plants in the Territory, compared with elsewhere in the country. However, the same recommendations apply for cattle as for horses, with respect to limiting access to *Crotolaria* and other potentially toxic plant species.