Paterson's Curse and Horse Health

PYRROLIZIDINOSIS IN HORSES

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INTRODUCTION

Paterson's Curse *(Echium plantagineum)* is a plant that is toxic to horses due to a compound contained in the plant known as 'pyrolizidine alkaloid'. In fact, Paterson's Curse contains up to 10 individual types of pyrolizidine alkaloid (PA) toxins. When horses eat Paterson's Curse the toxins react within the body causing cells to die, preventing normal cell activity and damaging blood vessel tissue. The liver is highly susceptible to damage from the PA toxins, but cells in the entire body are affected.

Horses may react differently to Paterson's Curse due to the various PA toxins and the different ways they can attack the body tissue. The two recognised forms are an acute form (less common) where death may occur over 2-3 weeks and the more common chronic form where death and symptoms may take months to display themselves, even after the horse is removed from the plant.

The effects of Paterson's Curse relates to the quantity of Paterson's Curse available, the quantity of other pasture feed available, the length of time the animal is exposed to the plant and the growth stage of the plant at the time of grazing. While some studies indicate conflicting views, it is generally believed that Paterson's Curse is more highly toxic at the rosette and maturing stage till flowering, where it may peak. Toxicity may then decline as the plant matures and decays. Hay and feed by-product that accidentally contains Paterson's Curse still retains its toxic capabilities.

Paterson's Curse becomes a major problem following a season characterised by a lack of pasture, such as occurs after drought or fire. At the return to a normal season there is little initial competition and the Paterson's Curse germinates unchallenged and may become the dominant species. As the rosette stage of Paterson's Curse is the first green pick, it is almost impossible for horses not to have some intake if pasture grazing. It should also be recognised that grazing Paterson's Curse over a number of years does cause gradual and accumulative liver damage that can be exacerbated in a year where Paterson's Curse has flourished.

Other species are also susceptible to alkaloid toxicity but the order of susceptibility is highest in horses with cattle, sheep and goats having a decreasing level of susceptibility.

TYPE A—ACUTE FORM

History and Clinical Signs

Death may be sudden—occurring within two to three weeks with little prior evidence of illness. The clinical signs may include a mild change in behaviour, especially becoming quieter. A change in feeding behaviour, like eating less or being selective, respiratory distress and fluid build up in the abdomen.

On post-mortem the liver appears enlarged with a dark bluish black colour and mild hydrothorax and ascites.

Diagnosis

This toxin can affect the liver, lung and the kidney but has a predilection for liver cell damage. Hence a comprehensive liver function test (supported by a history of PC exposure—noting that the history can occur over a number of years and clinical signs), is the most likely and practical diagnostic tool. A whole blood sample should be collected for a full liver function test and this may need to be repeated to establish if the profile is increasing or declining.

Treatment

Due to the acute nature of the disease, little successful treatment can be undertaken and death may occur without an adequate response period. Intensive hospital treatment may prevent death. Restrict access immediately to the plant and reduce the workload on the liver by supporting with a diet low in protein, high in carbohydrate with vitamin supplement. Restrict any chemical intake that is processed via the liver and seek professional advice.

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TYPE B—CHRONIC FORM

History and Clinical Signs

In the chronic form, death may occur after an extended period or not at all if successful intervention occurs. Th