WHITE LINE DISEASE IN HORSES

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look into potential causes of white line disease, before turning to diagnosis and treatment for a condition that can be fatal if secondary to another disease

WHITE line disease (WLD) describes a keratolytic process that causes separation of the inner hoof wall between the stratum medium and the stratum internum (Kuwano et al, 2002; O'Grady, 2002).

Once separation between the non-pigmented horn at this junction occurs, opportunistic bacteria and fungi can move in and infect the area.

It was first described by Redden (1990) and since this time a definitive link has yet to be established with an infectious agent.

Aetiology

The aetiology of WLD is unknown. Redding (2012) reports no age, sex, or breed predilection, but this contradicts the study by Kuwano (1999), which found incidence increased significantly with age. WLD can affect a single foot, or multiple, barefoot or shod, but once present on a yard, it does not necessarily mean more than one horse will be affected by it (O'Grady, 2011; Redding, 2012). No single cause has been proven although many contributing factors have been proposed (Redding, 2012).

Excessive moisture has been suggested to contribute to a horse suffering from WLD (O'Grady, 2011). It is thought allowing the moisture to soften the feet allows dirt and debris into a separation, tract or fissure, leading to secondary infection (O'Grady, 2002). WLD is often seen more in wet, humid areas, but this is not always the case as incidences have been seen in hot, dry areas (O'Grady, 2002). Dry hooves are more likely to crack, and therefore allow pathogens a route of entry (O'Grady, 2002 and 2011).

Poor hygiene and management as a cause of WLD has been proposed but this theory is not supported by the limited evidence base (O'Grady, 2011; Redding, 2012). The condition is seen in well-managed yards along with yards with poorer hygiene (O'Grady, 2002).

Significant evidence shows infectious organisms are involved; bacteria, fungi or a combination of the two are commonly isolated, but whether they are primary pathogens or opportunistic secondary infections is unknown (O'Grady, 2002; Redding, 2012). The main and most successful treatment option is debridement, which has been proven to be effective in a high number of cases (O'Grady, 2002; Redding, 2012). This suggests pathogens are perhaps not the primary cause.

Conformation faults are thought to put mechanical stress on the inner hoof wall (O'Grady, 2011). This is then thought to lead to the separation of the hoof wall and WLD. Bad conformation that provides mechanical stress includes long toes and low heels or club feet. These conformation faults are thought to compromise the blood supply associated with the crena marginalis of the third phalanx (O'Grady, 2002 and 2011). Cases of chronic laminitis, where integration between the sole/wall junction is lost, provides a route of entry for pathogens (Kuwano et al, 2002; O'Grady, 2002).

Higami (1999) reported on a high incidence of WLD at a riding stable keeping approximately 100 horses. After investigation it was attributed to feeding low-zinc and low-copper diets. Nelson (1984) reported abnormal hoof growth and hoof pain in ruminants with zinc deficiency.

Diagnosis

Diagnosis is often made by careful clinical examination of the foot. The white line (sole/wall junction) often has more of a chalky appearance and is wider, softer and more obvious visually in the early stages of the disease (Redding, 2012). This separation may first be noticed during a routine visit by the farrier. O'Grady (2002) reports the soles may become increasingly flat and more tender with occasional heat.

Looking at the inner hoof wall, which lies dorsal to the sole/wall junction will generally reveal a separation filled with white/grey powdery horn material (Redding, 2012). The extent of the cavity can be explored using a probe to look at the depth of the cavity (O'Grady, 2011; Redding, 2012). It has been suggested if lameness is present, the horse should be given a full lameness examination involving nerve blocks to localise the area of lameness and radiographs to confirm (O'Grady, 2002 and 2011).

Radiographs can be very useful for diagnosing WLD as they allow the clinician to identify the degree of separation (Redding, 2012; O'Grady, 2002). Radiographs allow any rotation of the pedal bone to be highlighted and evaluated (Redding, 2012). It is recommended dorso-palmar/plantar and latero-medial views are taken to allow the clinician to identify the extent of the separation and help the clinician and farrier work together for appropriate treatment (Redding, 2012).

It is important WLD is differentiated from laminitis and radiographs are important in making this distinction. In WLD the separation seen in the foot will originate from the solar surface whereas in laminitis the separation starts in the dermal lamellae and continues distally (Redding, 2012).

Laboratory testing is unproductive. Cultures have been used, but they generally prove aerobic cultures tend to be opportunistic organisms and anaerobic cultures are negative (O'Grady, 2002; Redding, 2012). In most circumstances, populations are a mixture of bacteria and fungi (Redding, 2012). An aseptic technique for culture has been suggested where a hole is drilled in the front of the hoof wall in the most proximal area of the separation (O'Grady, 2002). This was completed in five horses with WLD where all five tested negative for bacteria, but fungal culture was positive culturing a number of different fungi (Redding, 2012). This technique is thought to have limitations and doesn't show a lot more than we already know.

Treatment

Hoof wall resection is the mainstay of veterinary treatment. Removal of the hoof wall to expose any diseased horn, followed by debridement of all associated tracts, is necessary to prevent extension of the disease process (O'Grady, 2002).

The lead author prefers using a loop hoof knife and hoof cutters, but has used an oscillating saw where larger resections of redundant wall need to be performed. Resection should continue to the margins where healthy tissue and solid horn is located. It is uncommon to encounter marked haemorrhage during resection; a small amount of blood is not uncommon from the outer margins of resection.

Antimicrobials do not have any place in WLD; their use is not justified without appropriate resection of diseased horn. Numerous topical treatments have been suggested; two per cent iodine (often with dimethyl sulfoxide for deeper penetration), hydrogen peroxide, copper sulphate, Venice turpentine and merthiolate are popular. However, they are frequently overused, leading to brittle and often crumbly horn. Phenols, formaldehyde and strong concentrations of iodine are generally contraindicated as they have the potential to damage the sensitive structures of the foot.

In the lead author's opinion, the best course after initial diagnosis and resection is repeat examination and subsequent fortnightly debridement. Resolution occurs when the resection has grown out, although regular farriery and examination is essential to monitor recurrence of tract and fissure formation. Turner (1996) discusses reconstruction of the hoof wall deficit with a combination composition of acrylic and antibiotic. It is the lead author's opinion the hoof wall deficit in WLD should not be filled. The acrylic reconstruction has the potential to seal infection and weaken the adjacent hoof wall because it does not allow air to circulate to the diseased area protecting the bacteria or fungi within.

Therapeutic shoeing is an essential part of WLD management. The aim is to unload and protect the resected area of the hoof wall. If the size of resection is extensive or rotation of the pedal bone is evident on radiographs, a heart-bar shoe should be placed to lock down the foot (prevent expansion or compression of the hoof capsule) and allow weight bearing to be distributed away from the hoof wall to the frog. Shoes should be fitted so breakover is under the toe toward the apex of the frog, to remove the "lever arm"
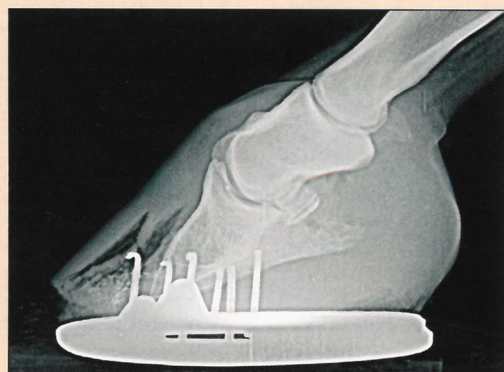
ABSTRACT

White line disease (WLD) describes a keratolytic process that causes separation of the inner hoof wall between the stratum medium and the stratum internum.

The aetiology of WLD is unknown. No single cause has been proven although many contributing factors have been proposed. Excessive moisture has been suggested to contribute. Poor hygiene and management as a cause of WLD has been proposed, but this theory is not supported by the limited evidence base. There is significant evidence to show infectious organisms are involved; bacteria, fungi or a combination of the two are commonly isolated, but whether they are primary pathogens or opportunistic secondary infections is unknown. Conformation faults, such as long toes and low heels or club feet, are thought to put mechanical stress on the inner hoof wall by compromising the blood supply associated with the crena marginalis of the third phalanx.

Diagnosis usually involves looking at the clinical signs. Radiographs can be a very useful diagnostic tool as they allow the clinician to identify the degree of the separation.

Hoof wall resection is the mainstay of veterinary treatment. Removal of the hoof wall to expose any diseased horn, followed by debridement of all associated tracts, is necessary to prevent extension of the disease process. For uncomplicated cases of WLD where it is the primary disease process the prognosis for full recovery is excellent. If WLD occurs secondary to another disease process, such as laminitis or where rotation of the pedal bone has occurred, the prognosis for full recovery is guarded. Recovery times can be lengthy and commitment from the owner is essential; the closer the deficit is to the coronary band the longer the recovery period.

Keywords: white line disease, hoof wall resection, remedial farriery, foot imbalance, laminitis

Figure 1. Latero-medial radiograph showing extensive white line disease.

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Figure 2. The foot in Figure 1 after resection.

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THERAPEUTIC ADVANCES IN CONGESTIVE HEART FAILURE

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highlights recent publications studying the area of feline heart failure and identifies future directions of possible research into treatments

CATS with heart failure are poorly served by published evidence regarding the most effective therapy. Until recently only a single conference abstract from 2003, as yet unpublished, detailed a prospective comparative study on different therapeutic agent effects[1].

However, this may be changing. In recent years, attention has been paid to pimobendan following its successful use and strong evidence base in dogs; novel therapies such as the heart rate modifier ivabradine, have been investigated, and the recent emergence of a cat-specific angiotensin-II receptor blocker agent (ARB) - telmisartan - opens up the possibility of studies into its use in cats with heart disease.

Pimobendan

Pimobendan is a phosphodiesterase-III inhibitor (PDE-III), classed as an inodilator due to its dual action positive inotropic and vasodilatory effects. However, it is a wide-acting drug with several additional actions[2,3]. Its inotropic effects are mainly mediated by calcium sensitisation (and, to a lesser extent, PDE-III inhibition) in the myocardium, and vasodilatory effects by PDE-III inhibition in vascular smooth muscle cells, mainly in arteries and arterioles, that reduces cardiac after load.

Crucially, it increases cardiac output without a concurrent increase in myocardial oxygen demand and, despite effects on calcium cycling, is non-arrhythmogenic. It additionally acts as a positive lusitrope, increasing the rate of relaxation of the myocardium following contraction, thereby allowing a greater diastolic filling time. The inotropy, vasodilation and lusitropy underpin its profound positive effect on cardiac output.

It also displays anti-cytokine effects, especially on NFκB, which is raised in congestive heart failure (CHF) and thought to significantly contribute to cardiac cachexia. It shows anti-thrombotic effects, inhibiting platelet aggregation, and anti-endotoxin effects[2].

The use of pimobendan in dogs with CHF due to mitral valve disease and dilated cardiomyopathy has been shown to offer marked improvements in survival time and quality of life, and studies into its use (in conjunction with furosemide) represent some of the most robust evidence available in the whole of veterinary medicine (for example, the QUEST study)[4,5].

However, its use in cats was thought to be generally contraindicated due to the different nature of their heart disease.

The majority of CHF cases in cats is due to cardiomyopathy, with hypertrophic cardiomyopathy (HCM) by far the most common, representing around 60 per cent to 80 per cent of cases[6].

CHF due to HCM has been classically characterised as primarily due to diastolic failure, with a combination of insufficient diastolic volume (due to mural hypertrophy) and diastolic filling time (due to tachycardia from sympathetic upregulation) to maintain adequate cardiac output. Because of this, it was unclear pimobendan would benefit this disease process.

It was feared in HCM cases it may lead to end-systolic luminal obliteration, and in cats with hypertrophic outflow tract obstruction (HOCM) its use would worsen the condition – either by ejecting blood with greater force through a hypertrophy-narrowed outflow tract, or exacerbate anterior motion of the mitral valve, thereby impeding outflow[2].

While its use in feline dilated cardiomyopathy (DCM) and, possibly, restrictive cardiomyopathy may have been justifiable on the grounds that this represents a systolic failure, HCM/HOCM cats were considered unsuitable targets by many.

However, this was not universal, especially following studies that showed cats with HCM also suffer from systolic dysfunction[7]. Further, it was thought the lusitropic effect of pimobendan could improve HCM diastolic function.

Work has now provided evidence for the beneficial use of pimobendan in cats with HCM, DCM and, to a lesser extent, HOCM. A retrospective case-control study by Reina-Doreste et al showed a significant survival benefit for cats with CHF due to HCM and HOCM treated with pimobendan versus those not given pimobendan[8]. Cats, mostly mixed domestic breed, were divided into either a pimobendan (n=27, receiving median dose 0.25mg/kg q12h) or non-pimobendan (n=27) group, with all cats receiving concurrent furosemide, most also receiving an angiotensin-converting enzyme (ACE) inhibitor, and a small number a beta-blocker.

No differences existed between groups on severity of echocardiographic findings, arrhythmia presence, thromboembolism incidence or furosemide dosage.

The pimobendan group showed a significantly greater median survival time (MST) of 626 days compared to 103 days for the non-pimobendan group (p=0.024) and there was no influence of type of cardiomyopathy (HCM or HOCM), or other medications being administered, on survival.

The authors concluded the study gave strong evidence for the use of pimobendan in cats with CHF due to HCM, and limited evidence ⇨ page 16

Figure 3. Dorsopalmar radiograph showing white line disease affecting from the lateral quarter to toe.

Figure 4. The foot in Figure 3 after hoof wall resection.

Figure 5. The foot in Figure 3 after debridement of necrotic tissue.

at the toe (O'Grady, 2002). In some cases the placement of nails or shoes cannot be done safely; these cases can be treated with aluminium shoes glued to the hoof or, in some animals, wooden shoes can be attached (O'Grady, 2009).

Zinc and copper supplementation should be considered in those suffering from WLD.

The National Research Council (1989) reports zinc requirement as 40mg/kg of dry matter. However, Lewis (1995) and Breedveld (1988) both report lower zinc requirements of 15mg/kg and 18mg/kg respectively.

The differences most probably reflect variation in feeding and exercise regimes; those fed alfalfa with a high calcium content may disrupt zinc absorption; performance horses are more likely to generate greater stresses on the foot and this could explain the difference in zinc requirements between Higami (1999) and Breedveld (1988). A balanced and adequate diet is important; supplements containing biotin and methionine can help by providing the substrate for building healthy horn but have no significant effect on WLD (Higami, 1999).

Environmental management after resection is important and focuses on keeping the feet as dry as possible. Keep bedding clean and dry, and do not turn out to pasture after or during rain or wet weather conditions. The use of topical calcium stearate/zinc oxide sprays applied to the hoof can help to maintain a dry environment.

Prevention of WLD is difficult as the aetiology in unknown. However, regular trimming of the foot by a farrier with exploration of abnormal areas and careful balancing of the feet may prevent separation of the white line and offer protection to the foot (O'Grady, 2003). Trimming or resetting shoes regularly will correct imbalances and reduce strain to specific areas of the hoof capsule.

Good hoof hygiene is important; picking feet out daily will remove gross contamination.

Prognosis

For uncomplicated cases of WLD where it is the primary disease process the prognosis for full recovery is excellent (Oke, 2003), but it often requires persistence.

If WLD occurs secondary to another disease process, such as laminitis or where rotation of the pedal bone has occurred, the prognosis for full recovery is guarded. Recovery times can be lengthy and commitment from the owner is essential – the closer the deficit is to the coronary band the longer the recovery period. Some deficits that extend near the coronary band can take approximately 10 months to grow out completely, assuming there are no complications.

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