ROBERT H. KOONTZ, DVM

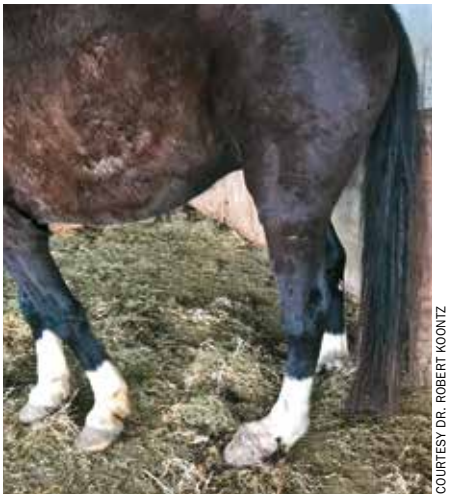
Degenerative Suspensory Ligament Disease

I am at the farm to euthanize a friend. Joe is a 20-something Arabian show horse. He has an impressive show name, but I just liked to think of him as Joe. I thought about how unfair it was that he should have a life-threatening hereditary disease through no fault of his own. His mind is still willing, but his body has betrayed him. Joe has degenerative suspensory ligament disease (DSLD). We had managed it for several years, and just two seasons ago he was still competing at a high level. But now he has more painful days than good ones, and it has become increasingly difficult for him to get up. Today Joe's hindquarters have given up the fight and he is unable to rise.

Degenerative suspensory ligament disease causes chronic suspensory ligament breakdown—this structure runs down the back of the cannon bone and attaches to the sesamoid bones. Without its support the fetlock joints (most commonly the rear) drop below their normal angle. Resulting ligament breakdown, flexor tendon injury, and osteoarthritis in the fetlock, hock, and stifle joints lead to debilitating lameness. The disease is manageable but not curable.

Veterinarians first reported DSLD in 1981 in Peruvian Pasos. Since then, we've learned a number of breeds can develop it, including Arabians, American Saddlebreds, American Quarter Horses, Morgans, Thoroughbreds, and European Warmbloods. We have also learned that it affects more than just the suspensory ligaments; the abnormal accumulation of proteoglycans it causes impacts tendons and other connective tissues, as well.

Proteoglycans are protein complexes that provide structural support to the cells that make up ligaments, tendons, and joints. While some proteoglycans in a ligament provide strength, too many will weaken the ligament. This phenomenon has prompted some researchers to suggest changing the name of the disease to equine systemic proteoglycan accumulation. Whenever a disease has more than one name or changes names, it usually means we do not understand it completely. This is certainly true with DSLD.



COURTESY DR. ROBERT KOONTZ

Notice how DSLD changes the conformation of the entire rear limb and causes hyperextension of the stifle, hock, and fetlock joints.

Researchers believe DSLD to be hereditary, although they have not determined the exact lineage. There has been recent speculation that metabolic diseases such as pituitary pars intermedia dysfunction (PPID) could play a role. While PPID does disrupt the suspensory ligament fibers, it is unclear if there is a cause-and-effect relationship between these conditions or if they just happen to occur at the same time. Because of this possibility, test horses with DSLD for metabolic disease.

Veterinarians diagnose DSLD based primarily on clinical signs. Researchers have been attempting to develop a diagnostic test to identify DSLD before symptoms occur, so owners can avoid breeding horses that carry the genetic disorder, but this is still in the works.

Because there is no cure, management centers around supporting the affected limbs. Mild to moderate exercise every other day has been shown to improve clinical signs. Shoeing to support the suspensory ligament and superficial and deep flexor tendons can also be beneficial.

Because the suspensory ligament is degenerating, the flexor tendons assume more concussive force when the leg hits the ground. This can cause injury to both of these structures. Support wraps while exercising can help prevent injuries. Maintaining these horses in support wraps over long periods, however, might lead to flexor tendon laxity (looseness) and increased risk of injury.

As the suspensory ligament apparatus becomes less effective, the fetlock, hock, and stifle become increasingly hyperextended. This gives the hind limb a straight appearance. The hyperextended fetlock joints can even become subluxated (partially dislocated). Traumatic injury can occur if the fetlock joint hyperextends to the point that the pastern bone strikes the cannon bone. Hyperextension of all three joints leads to abnormal stress on the joint cartilage and, eventually, osteoarthritis.

We can manage osteoarthritis with non-steroidal anti-inflammatory drugs, steroidal joint injections, and/or regenerative medicine. Some veterinarians speculate that we should not treat DSLD-induced osteoarthritis with polysulfated glycosaminoglycan (Adequan) because it may contribute to proteoglycan accumulation in the ligaments.

Like any disease without a cure, a host of "natural remedies" and "nutritional supplements" have popped up, with manufacturers claiming they cure DSLD. To date these supplements have not been scientifically studied for efficacy. Ultimately, we might not be able to cure horses like Joe, but wiser breeding practices could be the key to eradicating this devastating disease.

American Association of Equine Practitioners, 4033 Iron Works Parkway, Lexington, KY 40511 · 859/233-0147 · www.aaep.org