Twenty Cases of Degenerative Suspensory Ligament Desmitis in Peruvian Paso Horses

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Degenerative suspensory ligament desmitis is a syndrome characterized by chronic, progressive bilateral or quadrilateral lameness that may or may not cause secondary progressive hyperextension of the affected fetlocks. There is no sex-, age-, or work-related predisposition for this syndrome in Peruvian Pasos. Authors' addresses: Starland Veterinary Services, 603 Hayts Road, Ithaca, NY 14850 (Mero); Department of Pathobiology and Population Medicine, Mississippi State University, College of Veterinary Medicine, Mississippi State, MS 39762 (Pool).

1. Introduction

Degenerative suspensory ligament desmitis (DSLD) as a term was first coined by Dr. Jan Young in 1993 in a paper describing the syndrome in several breeds.[1] Sporadic reports of suspensory apparatus failure in Peruvian Pasos and references to their predisposition for this disease have been reported in the literature, but are largely anecdotal.[1–4] The second author first recognized this syndrome in Peruvians in the 1980s in an unpublished paper.[a] That study evaluated clinical and radiographic findings of suspensory apparatus failure in 17 Peruvian Pasos. Gross dissections and histopathological examinations were done on suspensory ligaments from seven of these horses.

Suspensory ligament desmitis commonly occurs in several breeds of horses, and its development in some breeds is associated with athletic function.[3,4] Degenerative disease of the suspensory ligament that spontaneously occurs in breeds other than Peruvian Pasos typically develops in horses that have intense work loads or have sustained a prior suspensory desmitis. They also include aged animals and broodmares.[3–5] Work-related injuries of the suspensory ligament that occurs in athletic horses are primarily restricted to the proximal one-third of the suspensory ligament, the body of the ligament, or in one or both branches.[3–5]

DSLD in Peruvian Pasos is a progressive degeneration primarily affecting the branches of the suspensory ligaments. This disorder causes mild-to-severe lameness either bilaterally or quadrilaterally, has no sex or age predilection, and can occur in the absence of athletic activity. Clinical signs can range from stiffness during gaiting to overt lameness. Mild-to-severe pain can be evoked by palpation of the suspensory ligament branches. There is thickening and enlargement of the ligament particularly in the branches, and there is a positive response to the fetlock flexion test. Historically progressive hyperextension or dropping of the fetlocks toward the ground with or without an overall enlarging of the affected fetlocks are considered the main signs of DSLD.[1–4]

Ultrasonographically, DSLD in Peruvian Pasos is similar to degenerative changes in the suspensory ligaments of other breeds.[b] What is unique to Pe-

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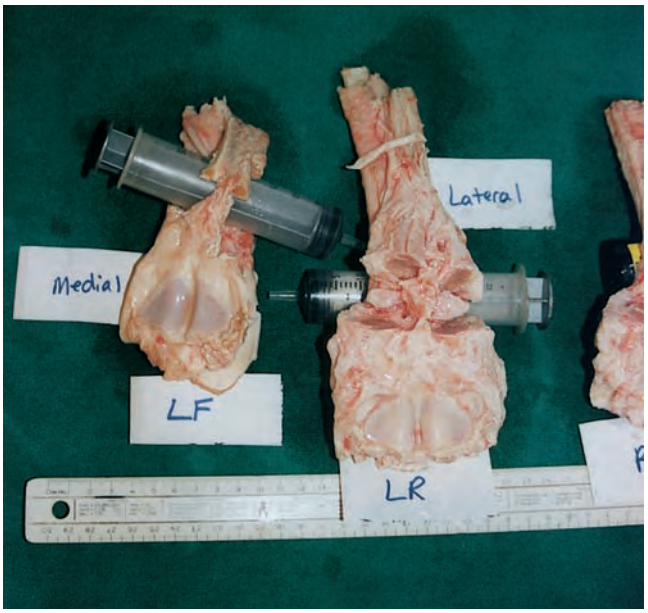


Fig. 1. Thirteen-year-old Peruvian Paso mare. DSLD in all four limbs. Left rear and left front cross-sections through suspensory ligament branches. Left rear was the most affected with grossly enlarged and discolored SL branches.

ruvians is the progressive, continuous enlargement of the suspensory ligament, primarily involving both branches, over time, in more than one limb.[1,b] There is a diffuse loss of echogenicity and poor fiber pattern images noted throughout the affected portions of the ligament.[1,3,4] Discrete hypoechoic lesions in the body or branches are less commonly noted.

Pathologically, DSLD is consistently characterized by degeneration and swelling of collagen bundles that form the fascicular system of the suspensory ligament. Clusters of ligamentocytes, i.e., fibrocytes, in affected collagen bundles undergo degeneration, necrosis, proliferation, or chondroid metaplasia. These progressive changes result in failure of collagen fiber maintenance and loss of tensile strength of collagen bundles. Loss of vascularity and fibrosis of the interstitial tissue between fascicles accompanies coalescence of swollen fascicles. Thickening of the branches of the suspensory ligament are caused by swollen fascicles of cell-poor degenerative collagen, enlarged fascicles of highly cellular fibrillar matrix apparently produced in an abortive attempt at fascicular repair and generalized interstitial and periligamentous fibrosis. As this progressive disorder of collagen injury and ineffective fibrous repair continues, the suspensory ligament becomes progressively inelastic, weak, and enlarged.

The purpose of this paper is to describe DSLD in Peruvian Paso horses clinically, ultrasonographically, and histopathologically. Because of the progressive nature of the disease and the lack of successful treatments to date, no discussion on treatment will be offered.

2. Materials and Methods

Sixteen purebred Peruvian Paso horses were presented to the first author for lameness evaluation. Chief complaints included obscure, recurrent lameness, back pain, overt lameness, marked swellings about the fetlocks, a sinking of the fetlocks towards the ground, and excessive lying down and a reluctance to move. A medical history for all 16 Peruvians was obtained, and all were evaluated clinically by palpation of all four limbs, observance of a baseline lameness, and flexion tests of all four limbs. Diagnostic local anesthesia was employed for three horses to localize the source of pain. The angle of each fetlock during weight bearing was also noted.

Affected limbs of 15 horses were examined by ultrasonography, and results were recorded. For some horses, all four limbs were evaluated using sonograms of normal limbs for comparison. Ultrasonic changes included an increase in the lateral to medial and palmar/plantar to dorsal measurements of the suspensory ligament branches, or the palmar/plantar to dorsal measurement of the body. There

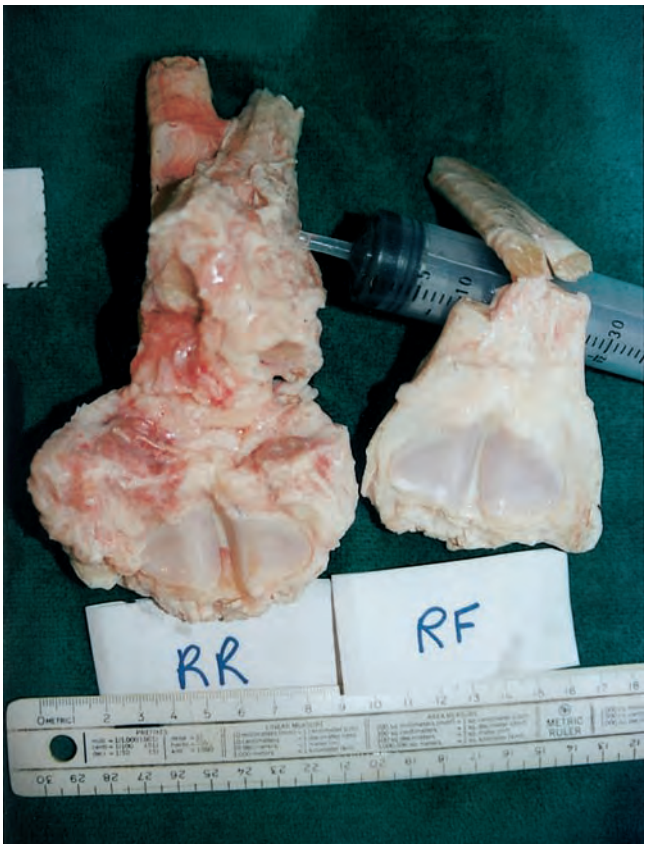


Fig. 2. Thirteen-year-old Peruvian Paso mare (same as Fig 1). The RF most approaches normal size and appearance but on histopathologic examination was also affected. The RR is same as LR—grossly enlarged and discolored.

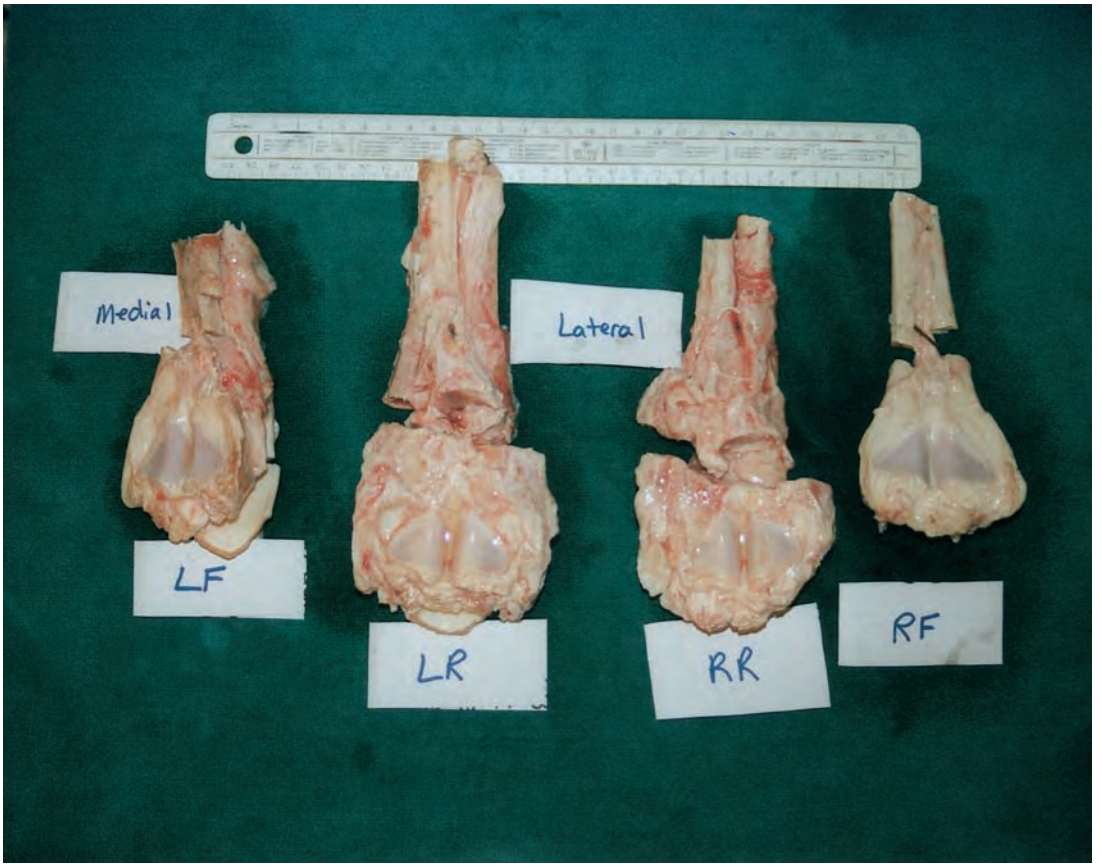


Fig. 3. Same mare as in Figures 1 and 2. All SL shown.

was a diffuse loss of echogenicity. Poor fiber patterns were present throughout the affected portions of the ligament, and in some cases, discrete hypoechoic lesions were observed within the body or branches. In several cases, re-exams, both clinically and ultrasonographically, were performed.

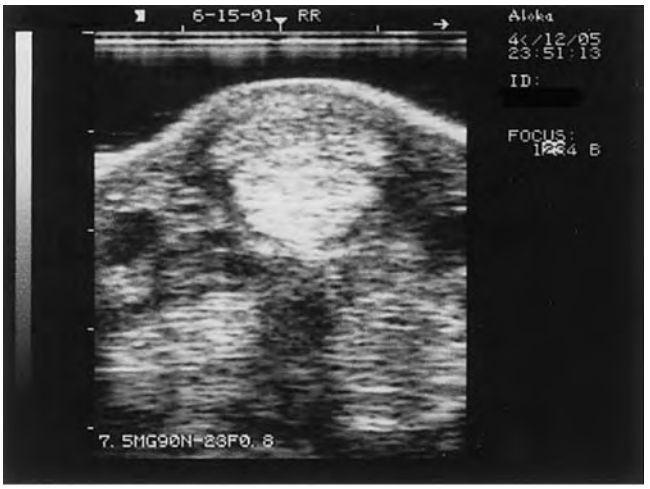
The suspensory ligaments of one Peruvian in this report were dissected and examined by the first author, who made gross measurements of the body and branches of the affected suspensory ligament (Figs. 1–3). The second author examined, by histopathology, selected necropsy specimens removed from affected suspensory ligaments of 7 of the 16 Peruvians that had been clinically evaluated. Recently, specimens from suspensory ligaments of four additional adult Peruvian Pasos with a history of progressive lameness and a clinical diagnosis of DSLD were removed at necropsy and submitted to the second author for inclusion in this study.

3. Results

Of the 20 Peruvians affected with DSLD, 12 horses had involvement in all four limbs, 6 horses had only rear limb involvement, and 2 horses had only the front limbs affected. Thirteen of the 20 horses were mares, 1 was a stallion, and 6 were geldings. The ages of the affected horses ranged from 3.5 to 19 yr of age. Athletic discipline of all 20 cases was noted as follows: broodmare, 5 of 20; pleasure/no work, 8 of 20; pleasure/light work, 5 of 20; or show/moderate work, 2 of 20.

Of the 16 horses examined clinically by the first author, 9 had visibly swollen and enlarged fetlocks and/or visibly enlarged suspensory ligament branches. Six of 16 cases had hyperextended fetlocks. Fourteen of 16 cases had pain on palpation of the suspensory ligament body and branches ranging from mild to severe. Lameness was noted following the AAEP Lameness Grading System of 0-5: grade 0, no apparent lameness; grade 1, lameness difficult to observe; grade 2, difficult to observe at a walk or trot in a straight line; grade 3, consistently observable at a trot at all times; grade 4, obvious lameness; and grade 5, minimal weight-bearing lameness.[6] Thirteen of 16 horses had baseline lameness (range of 1/5–4/5). All horses were lame after flexion of the affected fetlocks, 13 of which exhibited responses of 3–4/5. A high volar nerve block obliterated the baseline lameness in the three horses in which diagnostic anesthesia was employed in the clinical exam.

Ultrasonographically, 12 of 15 horses had enlarged suspensory ligament branches with lateral to medial measurements over 1.1 cm and palmar/plan-



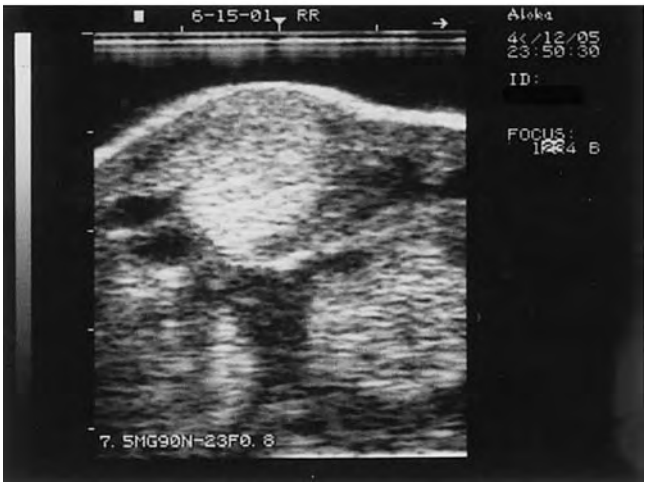
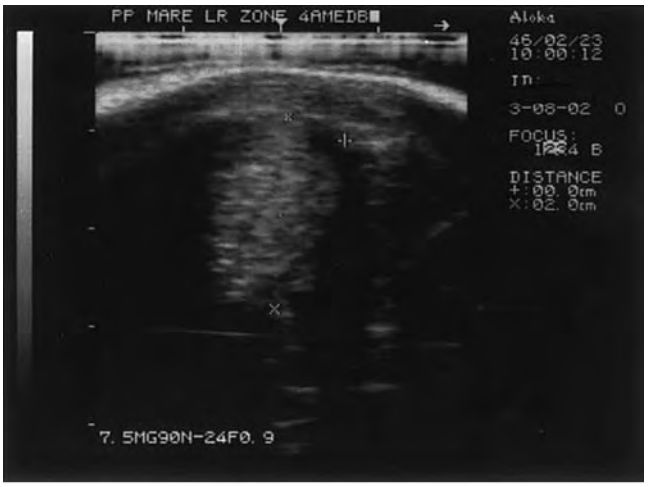


Fig. 4. Eleven-year-old Peruvian Paso gelding. Severely affected both rear limbs. Views in cross section proximal to fetlock (zone 3B/4A) showing enlarged SL branches below SDF and DDF.

tar to dorsal measurements over 0.9 cm (Figs. 4 and 5).[7] Three of 15 horses had enlargement of the suspensory ligament body. Nine of 15 horses had an increase in the hyperechogenicity of the suspensory ligament primarily confined to the branches (Fig. 4). Poor fiber patterns were noted throughout the affected ligaments in 12 of 15 horses. Discrete hypoechoic lesions of various sizes were noted in the branches and/or body in 7 of 15 horses.

Histopathology specimens were available for examination from the branches of the suspensory ligaments of 11 horses that included specimens from 7 horses examined clinically by the first author and from 4 horses examined by the second author. Transverse sections through the distal body of the suspensory ligament were available for five horses. All submitted specimens had lesions that characterize this disorder and differed primarily in the stage, e.g., early, intermediate, or late lesions, and degree of cross-sectional area of involvement of the suspensory ligament branch. In general, branches from suspensory ligaments of the hind limbs had a greater diameter and had more late, chronic, and extensive changes, whereas early, less extensive, and less complex changes were present in the suspensory ligament branches of the forelegs.

Earliest change recognized in the branches of the suspensory ligaments was the presence of clusters of enlarged ligamentocytes, i.e., fibroblasts, located within collagen bundles forming fascicles of dense collagenous tissue. Affected fascicles were not remarkable in size or location within the branch, although the smallest initial lesions were frequently located in fascicles near the center of the branch. Ligamentocytes in the cluster had undergone degeneration, necrosis, or proliferation, or were undergo-



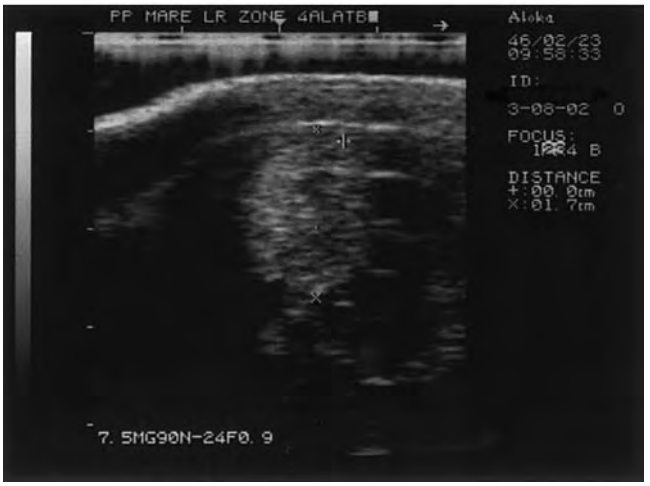


Fig. 5. Same mare as photos showing ultrasounds of the left rear SL medial and lateral branch with lateral to medial measurements, LR lat—1.7 cm; LR med—2.0 cm.

ing chondroid metaplasia. Collagen fibers in affected bundles were less dense and fibrillar. Amorphous matrix not normally observed in densely packed collagen of a control ligament surrounded the metaplastic chondrocytes and partially replaced the fibrillar matrix. As the focal clusters of affected ligamentocytes increased in size and numbers, the fascicles containing those cellular clusters increased in diameter.

In intermediate stage lesions containing numerous enlarged fascicles, there was a concomitant loss of vascularity and increased deposition of fibrous tissue in the slit-like interfascicular interstitial spaces between closely packed fascicles. Adjacent swollen fascicles underwent coalescence to form giant, pale-staining, paucicellular fascicles. Abortive attempts at repair occurred in enlarged hypercellular fascicles in which initial clusters of ligamentocytes had undergone proliferation. However, it seemed that the proliferative ligamentocytes in these hypercellular fascicles failed to form replacement collagen matrix that resembled normal collagenous matrix of unaffected fascicles. The DSLD lesions in the branches of some Peruvian horses in the intermediate stage of this disorder had mixed patterns of pale swollen paucicellular fascicles and swollen hypercellular fascicles beginning to be entrapped in interfascicular fibrosis.

In late stage lesions, periligamentous fibrosis and interfascicular fibrosis predominated. Large, pale, paucicellular, degenerative fascicles in the centers of the branches of the suspensory ligaments were often partially united by dense fibrous tissue that had filled interfascicular spaces. Large caliber blood vessels in these thickened branches were relatively more prominent than capillary-sized vessels. Large areas of periligamentous fibrosis often contained large avascular areas of liquefaction that were filled with mucoid degeneration of connective tissue. The areas of mucoid connective tissue were characterized by acellularity, an almost complete absence of a capillary bed and replacement of fibrillar matrix by an amorphous matrix. Inflammatory cells were essentially absent in all specimens.

4. Discussion

DSLD in Peruvian Paso horses is a chronic progressive disorder in which horses in the early stages of disease can present with mild, obscure signs, or in advanced stages of disease, can present with marked lameness. Some chronically affected horses may also have enlarged, hyperextended fetlocks. Consistent signs encountered during physical examination include pain on palpation of the branches of the suspensory ligament and baseline lameness of various degrees. The majority of horses having a positive fetlock flexion test will exhibit a 4–5/5 post-flexion response for several minutes.

Earlier reports of DSLD in Peruvian Pasos found hind limbs to be most commonly affected, followed by front limb involvement.[1,a] Interestingly, in this series, the frequency of limb involvement for lesions of DSLD was for all four limbs followed by hind limbs and front limbs. In addition, historically, more emphasis has been placed on swelling and hyperextension of the affected fetlocks; however, in this series only about one-half of the horses in this series exhibited those signs. In this series, fetlock swelling and hyperextension were not a consistent enough finding to permit a definitive diagnosis of DSLD in the Peruvian Paso based on their presence alone. Because of the large number of horses in this series that had quadrilateral involvement, it seems reasonable to recommend that all four limbs be examined in any Peruvian Paso that is suspected of having DSLD.

Ultrasound exams of the affected limbs were a useful adjunct to diagnosing DSLD, especially in those horses that were in early-to-middle stage disease and that exhibited only mild lameness or mild pain responses to palpation and/or flexion tests. Ultrasonography proved to be most useful if routinely used to scan all four limbs of an affected horse. In this manner, it was an aid in the proper diagnosis of additional affected limbs. Eighty percent of the Peruvians in this series examined by ultrasonography had enlarged branches of the suspensory ligament and poor fiber patterns in affected areas of the branch. Serial ultrasound exams were also useful in tracking the progressive enlargement of affected branches over time, especially in those cases that initially had only mild enlargement.

The etiopathogenesis of DSLD is unclear. The early lesions appear to be microscopic foci of degenerative of ligamentocytes that are located in collagen bundles forming the fascicular system of the branches and body of the suspensory ligament. The initially affected fascicles are randomly distributed primarily in the central region of the branches of the suspensory ligament. In these early lesions there is no apparent loss of vascularity or other reaction present in the adjacent interfascicular tissue where the capillary blood supply is located. However, there is histological evidence that similar foci of ligamentocyte damage form a mirror image pattern in contiguous fascicles where the intervening capillary bed of the interfascicular space is degenerative or is no longer patent. It is also apparent that small, paucicellular, degenerative fascicles undergo coalescence to form large pale paucicellular giant fascicles following the loss of their respective circumferential vascular beds. Ligamentocytes located in the centers of abnormally large fascicles are far removed from their blood supply in the interfascicular spaces and either degenerate and die or undergo metaplasia to chondrocytes that can survive by anaerobic metabolism. Therefore, based on examination of these specimens, one cannot determine whether decreased vascularity is a primary or secondary factor in the etiopathogenesis of this disorder. The fibrosis and abortive

attempts at collagen repair by hyperplastic ligamentocytes are considered secondary responses involved in the healing process.

The finding of angular atrophy of skeletal muscle fibers indicative of neurogenic atrophy in histological sections taken through the body of the suspensory ligaments of several Peruvians with DSLD should be investigated. However, skeletal muscle atrophy would not be expected to play a role in what seems to be a primary failure of collagen bundles. Although the suspensory ligament was once the interosseous medius muscle in the evolution of the horse, the partial loss of nerve supply to the very small volume of residual skeletal muscle in this primarily ligamentous structure should not be expected to play a role in DSLD of the Peruvian Paso. It is likely that denervation atrophy in these specimens is the result of nerve entrapment from secondary fibrosis rather than a primary nerve or muscle disorder.

The fact that 13 of 20 horses developed this disease with no athletic work at all and that those affected displayed a wide range in age suggests a different etiology for DSLD in Peruvian Pasos than in other breeds. A genetic component seems plausible and warrants further investigation. In conclusion, DSLD should be considered in any Peruvian Paso horse that experiences progressive lameness, pain on palpation of the suspensory ligament branches, and positive fetlock flexion tests in more than one leg, regardless of sex, age, or physical activity.

References and Footnotes

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