Intersection of the DSIL and the DDFT and Its Relationship to Navicular Syndrome

Kimberly K. Van Wulfen, BS and Robert M. Bowker, VMD, PhD

The microstructure of the intersection between the distal sesamoidean impar ligament and the deep digital flexor tendon suggests a fragility, as it is susceptible to injury caused by extreme tensile and shear forces during locomotion. Such insults can result in the initial cascade of pathophysiological events underlying navicular syndrome. Authors' address: Dept. of Anatomy, College of Veterinary Medicine, Michigan State University, East Lansing, MI 48824-1316. © 1997 AAEP.

1. Introduction
Improved equine foot management and treatment of disease begins with a basic knowledge of the anatomy and physiology of the foot. The suspensory ligaments of the navicular bone, in particular the microscopic anatomy of the distal sesamoidean impar ligament (DSIL), have received little attention from researchers, especially as to how they may relate to such diseases as navicular syndrome. The intersection of the DSIL and dorsal portion of the deep digital flexor tendon (DDFT), which is where they join before inserting onto the third phalanx (P3), represents a potentially important region undergoing stresses in the equine foot. The distal nervous and vascular supply to the navicular bone courses through the DSIL and the intersection. Sensory nerves containing the peptides calcitonin gene-related peptide (CGRP) and substance P (SP) have previously been identified within the DSIL.1,2 Although the functions of these peptides and their receptors in this region are not known, we believe that further study will aid in defining their actions and roles in diseases of the foot.

The purpose of this study was to examine the anatomy of the suspensory ligaments of the navicular bone, focusing on the microscopic anatomy of the intersection of the DSIL and DDFT and how it may relate to navicular syndrome.

2. Materials and Methods
Macroscopic anatomy of the suspensory ligaments of the navicular bone and the DDFT was examined by dissection as the feet were sectioned in various planes on a band saw. The intersection, collateral sesamoidean ligament (CSL), and DDFT both at the flexor surface of the navicular bone and proximal to the CSL were removed and prepared by using routine histological techniques. Nerves were identified using immunochemistry,1,2 gold chloride impregnation techniques,4 and receptor autoradiography.3 The intricate vasculature was studied by flushing the distal limbs with saline and infusing 25% gelatin and 30% India ink. These feet were then frozen, sectioned, and fixed. Tissues of interest were removed, embedded in gelatin, and sectioned at 90 µm. The sections were mounted serially, stained with

NOTES
Methylene Blue and eosin Y, coverslipped, and examined under a microscope.

3. Results

On macroscopic examination, the intersection was found to have parallel fibers of dense connective tissue inserting onto P3, separated by septa of loose connective tissue. Histological sections showed the dense connective tissue fibers and the septa, which were extensive, penetrating the entire DSIL and the dorsal DDFT. Within these septa, immunohistochemistry and gold chloride impregnation revealed the presence of many sensory nerves coursing between the navicular bone and P3. SP receptors were shown to be present in association with the vasculature.

The loose connective tissue septa also contained numerous blood vessels, including novel arteriovenous complexes (AVC's), which consisted of a network of tortuous capillaries connecting two larger vessels. These capillary networks did not seem to supply the surrounding tissue, but they were surrounded by epithelial-like cells. In addition, specific binding of the radiolabeled SP (NK1 receptors) appeared to overlie these arteriovenous complexes.

The proximal suspensory ligament, CSL, consisted of regular dense connective tissue typical of a ligament. However, the major insertion of the CSL was by an attachment of two broad abaxial extensions onto the second phalanx (P2), rather than by attachment solely to the first phalanx (P1), which were represented as thin attachments fused with the joint capsule.

4. Discussion

The anatomy and histology described reveals that the intersection of the DSIL and DDFT is a dynamic region, but possibly quite fragile. The presence of an abundance of nerve fibers and AVC's suggests a major sensory role in the detection and vascular perfusion of tissues in this region, specifically the navicular bone. The distal interphalangeal joint, when forced into dorsiflexion during locomotion, places the navicular bone in a major weight-bearing position, transferring stress from P2 to the suspensory ligaments of the navicular bone. In addition, the pull on the DDFT creates a shear force in the intersection between the DDFT and DSIL. Extreme stresses without proper conditioning could injure microstructures within the intersection, resulting in the initiation of inflammatory processes and tissue damage. An examination of the intersection of 12 midwestern horses affected with navicular syndrome showed changes consistent with this idea. The intersection appeared inflamed, and the DSIL and DDFT were fused more proximally, reducing the distal extent of the navicular bursa. The classic lesions of adhesions between the deep digital flexor tendon and the navicular bone were present only if lesions were also present in the intersection.

The findings that the CSL is attached mainly to P2 argues against the notion that a CSL desmotomy at the level of the proximal interphalangeal joint will potentially be successful surgically in decreasing the stress on the navicular bone in navicular syndrome-affected horses.

The microscopic structure of the intersection of the DSIL and DDFT suggests a complex anatomical arrangement of both sensory and vascular networks and is consistent with our hypothesis that the intersection is the initial site for the pathogenesis underlying navicular syndrome where stress or trauma to this region can account for most pathological observations associated with this devastating disease.

This research was generously supported by The American Quarter Horse Association.

References