Palmar Digital Neurectomy for the Treatment of Heel Pain

(How Pacman Lost His Nerve)

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Introduction

Navicular disease is the most common cause of lameness in horses and is estimated to be the cause of 1/3 of all chronic forelimb lameness cases. [2] Navicular disease is defined as pain related to the navicular bone, bursa, deep digital flexor tendon, collateral sesamoidean ligament, distal sesamoidean impar ligament or the distal interphalangeal joint.[1] Due to the close proximity of these structures, abnormalities of only one of these structures is unlikely.[7] Since the pain is often not solely originating from the navicular bone itself, many prefer the term navicular syndrome over navicular disease. Diagnosis based on imaging is somewhat unreliable and is accompanied by localizing the lameness with perineural anesthesia. There are multiple theories behind the pathogenesis of navicular syndrome, including biomechanical stress, osteoarthritis/inflammation and circulatory disturbances.[1] With multiple etiologies, there are a variety of treatment options aimed at each possible route of pathogenesis.

History and Presentation

A 12-year-old, Quarter Horse gelding that presented to Mississippi State University College of Veterinary Medicine Equine Surgery Department, on August 25, 2019, for a refractory right front limb lameness. He is performance horse that competes in barrel racing competitions. He had a history of refractory lameness that was previously managed with intra-articular therapies by his primary care veterinarian.

On physical exam, all vitals were within normal limits. There was a bony swelling at the caudolateral aspect of the left carpus, in the area of the accessory carpal bone, that was not hot or painful on palpation. On lameness examination, while trotting in a straight line on concrete, a slight head nod consistent with a right forelimb lameness was noted. When trotting in a circle to the right, there was a head nod indicative of a right forelimb lameness, as well as a mild head

nod when trotted in a circle to the left. Additionally, when traveling, a short, choppy stride with high head carriage that is suggestive of foot pain. Overall, he was graded to have a three out of five right forelimb lameness.

Pathophysiology

One mechanism of pathogenesis that results in navicular syndrome is biomechanical stress. The deep digital flexor tendon uses the navicular bone as a fulcrum to provide the angulation to attach to the coffin bone or third phalanx. Pressure between the flexor surface of the navicular bone and the deep digital flexor tendon causes degenerative change and remodeling of the bone.[5] This occurs more frequently with improper trimming and shoeing that changes the angle of the horses' hoof, which in turn changes the pressure put on the navicular bone by the deep digital flexor tendon. It is also influenced by the horse's natural limb and hoof conformation, with upright confirmation, small hoof size, negative palmar angle, low heels with long toe or underrun heels being predisposing factors.[5] Breed predispositions include Quarter Horses, Thoroughbreds and Warmbloods.[5]

Another mechanism is vascular compromise to the bone due to thrombosis of the arterioles supplying the distal border of the navicular bone.[1] Necrosis and pain can occur due to an ischemic event to cause the clinical signs of navicular syndrome. The final theory is inflammation and osteoarthritic changes to the navicular bone.[5] Changes to the navicular bone including fibrocartilage of the flexor surface, subchondral bone, medullary cavity and synovium of the navicular bursa are similar to changes documented in osteoarthritis of other joints.[5] These changes occur due to either abnormal forces on normal structures or normal forces on abnormal structures.

Regardless of pathogenesis, navicular syndrome most often presents as a chronic forelimb lameness that is refractory to treatment as it progresses.[2] Although it is often exhibited as a unilateral lameness, if the affected foot is blocked with perineural anesthesia, it can be exhibited on the contralateral limb. The lameness exhibited by horses with navicular syndrome worsens over time or remains constant if managed properly rather than resolving due to the irreversible changes to the bone including damage to the fibrocartilage on the flexor surface.

Diagnostic Approach

The diagnosis of navicular syndrome begins with localizing the lameness to the palmar aspect of the foot using perineural anesthesia.[5] When attempting to diagnose navicular syndrome by placing a palmar digital nerve block, injecting as low as possible in the heel as well as using a small amount of anesthetic is recommended to increase specificity.[2] This nerve block is placed by using between 1-2ml of mepivacaine subcutaneously over the palpable nerve bundle with the needle pointing distally.[4] The efficacy of the block can also be tested by checking superficial sensation in the area of the heel bulbs. Most, if not all, horses with navicular syndrome will improve significantly with a palmar digital nerve block but if adhesions between the deep digital flexor tendon and navicular bursa or accessory nerve supply are present the lameness may persist.[5] Another localizing tool is the use of hoof testers; navicular syndrome will cause pain when hoof testers are applied across the heel or the frog.[5]

Radiographic imaging is commonly used as a screening tool for navicular changes within the bone, but a major disadvantage is that it does not allow for evaluation of the surrounding soft tissue structures.[5] Although it is used to evaluate the bone, a 40% change in bone density is required before degenerative changes can be identified with radiographs.[2] In order to evaluate the navicular bone and due to its anatomic location and shape, radiographic evaluation requires a

minimum of lateromedial, 60 degree dorsoproximal-palmarodistal oblique and palmaroproximal-palmarodistal (skyline) views.[2] Radiographic abnormalities noted with navicular syndrome include sclerosis, loss of corticomedullary distinction, enthesiophyte formation, fracture of the navicular bone, calcification of the deep digital flexor tendon, cyst like lesions and enlarged synovial invaginations.[4]

Ultrasound can also be used in diagnosis of navicular syndrome with regard to soft tissue changes; however, it is more technically challenging and depends on experience and image quality.[4] Nuclear scintigraphy, also known as bone scan, is more sensitive when identifying cause of lameness but less specific.[5] Computed tomography (CT) is the best modality to assess structure of the navicular bone such as pathology within the cortex and trabeculae.[2] When evaluating synovial invaginations, CT is more sensitive for identifying number, depth, and shape when compared to radiography.[3]

The navicular bone is often not the primary abnormality identified in navicular syndrome; this is why magnetic resonance imaging is now considered the gold standard for diagnosis.[2] The most common injury in navicular syndrome is deep digital flexor tendon injury which is seen with MRI; magnetic resonance imaging is also more sensitive when identifying cyst like lesions within the navicular bone.[5] A difficulty with interpreting MRI in horses with navicular syndrome is determining the primary abnormality but with the newer understanding that multiple abnormalities most likely contribute to foot pain this distinction may not be important.[2]

Treatment and Management

Conservative therapy for the treatment of navicular syndrome includes rest, anti-inflammatory medication, and corrective shoeing.[7] Rest for 3-4 weeks followed by an ascending exercise plan is an accepted practice, but if an injury to the deep digital flexor tendon is

identified, a longer period of rest is required.[4] Correcting hoof trimming and shoeing is the treatment to reduce biomechanical stress on the navicular apparatus. The goals of corrective shoeing are to balance the hoof, correct the hoof-pastern-axis, protect the palmar aspect of the hoof and to improve break-over.[5] In addition to trimming, three types of shoes have been used including central support and natural balance shoes.[4] Central support shoes are similar to heart bar shoes and take pressure off the heel and apply it to the frog during loading while natural balance shoes are similar to normal shoes but are rounded at the toe to move the break-over point backwards.[4]

Systemic non-steroidal anti-inflammatory medications are the long-term palliative care option most commonly used in treating chronic navicular syndrome.[4] Intra synovial injections of glucocorticoids with or without hyaluronate into the distal interphalangeal joint or navicular bursa are commonly used in the beginning stages of navicular syndrome but usually have short term effects.[4] Systemic osteoarthritis medications such as sodium hyaluronate, polysulfated glycosaminoglycans, glucosamine, and chondroitin have also been used to varying degrees of success.[5] Another medical treatment option is bisphosphonates, a treatment approved by the Food and Drug Administration in 2014 for the use in horses; they are historically used in humans to treat osteoporosis.[5] Bisphosphonates bind to bone mineral and prevent bone resorption by suppressing osteoclast activity.[6] They are most effective when treating bony lesions identified with sensitive imaging such as navicular bone cysts.[4] Clodronic acid (a bisphosphonate) is reported to have decreased lameness by two points on the lameness scale in 55% of horses in one study.[1] A less common medical treatment option is the administration of Isoxuprine to treat the vascular compromise theory since it is a vasodilator and decreases blood viscosity and platelet

aggregation.[5] Extracorporeal shock wave therapy has also been used to provide analgesia and assist in bone remodeling with conflicting reports of efficacy.[4]

Surgical treatment of navicular syndrome, a palmar digital neurectomy procedure desensitizes the heel and sole.[5] Palmar digital neurectomy procedures are not without complications; return of sensation, neuroma formation and secondary injury due to desensitization of the foot are all possible sequelae.[5] This procedure is not permanent, the nerves will regrow and the disease process will continue to progress.[7]

Case Outcome

To diagnose the patient's lameness, a right palmar digital nerve block was performed with 2 ml of carbocaine over each nerve branch. Loss of skin sensation was confirmed before further lameness evaluation; the right front lameness improved by 85%. Radiographs had previously been taken at the primary care veterinarian and were not repeated. Computed tomography was performed under total venous anesthesia, cross sectional images of both distal forelimbs were obtained. Images of the right forelimb revealed an increased number of synovial invaginations within the navicular bone and a small focal triangular defect along the distal border consistent with a subchondral bone cyst. New bone formation along the proximal aspect of the navicular bone as well as enlargement of the distal sesamoidean ligaments with tissue edema was observed. Images of the left distal forelimb exhibited mild enlargement of the synovial invaginations indicating mild degeneration. These findings are consistent with moderate navicular degeneration with the right navicular bone being more severely affected.

Due to previous unsuccessful medical management, owners elected for a bilateral palmar digital neurectomy procedure. An approximately 4 cm section of each nerve was removed. Sterile wraps were placed on both front limbs and recovery from anesthesia was uneventful.

While in hospital, the patient received intravenous flunixin meglumine for analgesia as well as trimethoprim sulfadiazine and gentamicin for prevention of peri-operative infection. His legs were wrapped to keep surgical sites clean and dry. He was discharged with trimethoprim sulfadiazine to be continued at home as well as 3 days of flunixin meglumine. His bandages were changed every 4-5 days until the sutures were removed and he remained in standing wraps during stall confinement. After discharge, the patient was confined to a stall for 4 weeks with an ascending exercise plan to return to normal activity by 8 weeks post operatively. It was recommended that he be shoed in wedge shoes with pads to provide sole support and sole protection due to desensitization of the foot. It is important to monitor for incisional swelling or discharge, as well as checking daily for any sole puncture wounds. He is doing well at home and returned to barrel racing successfully.

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