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Original Research

Histological Changes in the Deep Branch of the Lateral Palmar Nerve of Horses With Induced Proximal Suspensory Desmitis

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ABSTRACT

Desmitis of the proximal aspect of the suspensory ligament, or *interosseus medius* muscle, of the pelvic or thoracic limb is a commonly diagnosed cause of lameness of performance horses. Despite medical treatments available for horses with proximal suspensory desmitis (PSD), most horses treated medically for PSD of a pelvic limb remain persistently lame; this persistent lameness may be the result of a neuropathy caused by compression of nerves by an enlarged suspensory ligament. Few horses with PSD of a thoracic limb remain persistently lame. Based on the results of reports citing successful treatment of horses chronically lame because of PSD of a pelvic or thoracic limb, by excising a portion of the deep branch of the lateral plantar or palmar nerve (DBLPlN/DBLPaN), we theorized that persistent lameness of horses caused by PSD of a thoracic limb may also be due to compression of nerves that supply the ligament. The aim of this study was to determine if histological signs of compression neuropathy of the DBLPaN are present in horses with PSD in a thoracic limb. To test this hypothesis, we induced PSD by instilling collagenase into the ligament and then examined the DBLPaN after harvesting this nerve 2 months later. We found that the DBLPaNs of all treated limbs showed histologic changes suggestive of nerve compression. We conclude that studies examining the DBLPaN of horses with naturally occurring PSD for histological evidence of neuropathy are warranted.

1. Introduction

Desmitis of the proximal aspect of the suspensory ligament, or *interosseus medius* muscle, of the pelvic or thoracic limb is a commonly diagnosed cause of lameness of performance horses and can occur unilaterally or bilaterally [1,2]. Medical treatments of horses with proximal suspensory desmitis (PSD) include prolonged confinement, periligamentous deposition of a corticosteroid, intralesional injection of autogenous bone marrow or autogenous platelet-rich plasma, shock-wave therapy, and corrective shoeing [3,4]. Despite the large array of medical treatments available for horses with PSD, most horses treated medically for PSD of a pelvic limb remain persistently lame [2–5]. Some of these treatments, however, appear to be highly effective in resolving lameness of most horses with PSD

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of a thoracic limb [2–5]. Failure of horses with PSD of a pelvic limb to respond to medical treatment may be due to neuropathic pain created by compression of the innervation of the suspensory ligament, which may occur as the damaged suspensory ligament enlarges within the canal to which it is confined [6–9].

The suspensory ligament of the pelvic limb is innervated by the lateral and medial plantar metatarsal nerves, which originate from the deep branch of the lateral plantar nerve (DBLPlN) [2]. The proximal aspect of the suspensory ligament of the pelvic limb, along with the distal most segment of the DBLPlN, is rigidly confined within the suspensory canal by the plantar aspect of the third metatarsal bone, the axial borders of the second and fourth metatarsal bones, and fascia plantar to the suspensory ligament [2,3]. Tóth et al [10] examined the DBLPlN of horses determined to be lame on one or both pelvic limbs because of PSD and found histological changes in the nerve suggestive of chronic compression [11]. These changes included proliferation of the endoneural myxomatous matrix, formation of Renaut bodies, and degeneration of myelin.

In a study of 271 horses with PSD of one or both pelvic limbs, 79% of horses were able to return to their previous level of exercise after undergoing plantar fasciotomy and excision of a portion of the DBLPlN of the affected pelvic limb [6]. In another study, 78 of 84 horses (91%) that underwent bilateral neurectomy of the DBLPlN, without plantar fasciotomy, for treatment for bilateral or unilateral PSD, returned to their intended use [7].

The proximal aspect of the suspensory ligament of the thoracic limb is innervated by the lateral and medial palmar metacarpal nerves, which originate from the deep branch of the lateral palmar nerve (DBLPaN) at the level of the proximal end of the fourth metacarpal bone [10,12,13]. The DBLPaN branches from the lateral palmar nerve in the midcarpal region and contains fibers from the ulnar and median nerves [13].

Results of a report citing successful treatment of four horses that remained chronically lame because of PSD of a thoracic limb, by excising a portion of the DBLPaN [14]. Based on this information, we theorized that neural compression may also be a cause of persistent lameness of horses caused by chronic PSD of a thoracic limb. The aim of this study was to determine if histological signs of compression neuropathy of the DBLPaN are present in horses with PSD in a thoracic limb. To test this hypothesis, we induced PSD by instilling collagenase into the ligament and then examined the DBLPaN after harvesting this nerve 2 months later. The aim of this study was to determine if compression neuropathy might be the cause of persistent lameness of horses with PSD of a thoracic limb that do not respond to medical therapy.

2. Materials and Methods

2.1. Horses

Eight mixed-breed horses, weighing 350 to 400 kg and ranging in age from 4 to 13 years, determined to have no evidence of PSD of the thoracic limbs during physical and ultrasonographic examinations, were used for this study. The suspensory ligament of both thoracic limbs was examined ultrasonographically in the longitudinal planes using a 10 MHz, 38-mm linear probe (Mindray-DP-50; Shenzhen Mindray Bio-Medical Electronics Co, Ltd, Shenzhen, China), with the limb fully loaded. The proximal aspect of the suspensory ligaments was considered ultrasonographically normal based on the previously established criteria [15–18]. All horses were determined to be healthy, based on the routine clinical examination and hematologic evaluation, and to be sound, when evaluated for lameness while trotting in a straight line on a hard surface, using a commercially available, inertial sensing device that measures asymmetry of torso motion to objectively quantify the degree of lameness (Lameness Locator, Equinosis, St. Louis, MO). The protocol for this study was approved by the National Autonomous University of México's Institutional Animal Care and Use Committee.

2.2. Induction of PSD

A thoracic limb of each horse was randomly selected for induction of PSD. Each horse was administered phenylbutazone (2.2 mg/kg PO) and detomidine HCl (0.02 mg/kg IV). The proximal, palmar aspect of the limb was prepared for sterile injection of the suspensory ligament, and the site of injection was desensitized by administering local anesthetic solution subcutaneously. Using ultrasound guidance, a solution (0.3 mL) containing 2,500 IU of filter-sterilized collagenase type 1 (Sigma-Aldrich Co, St. Louis, MO) in sterile water was injected through a 22 ga, 3.18 cm (1¼ inch) needle. With the limb bearing weight, the needle was inserted perpendicular to the skin on the palmarolateral surface of the metacarpus and advanced to the center of the proximal portion of the suspensory ligament, 2 cm distal to the base of the fourth metacarpal bone. All injections of collagenase were performed by the first author. A compression bandage extending from the distal aspect of the carpus to the hoof was applied and removed after 2 days. After injection, each horse was confined to a stall without exercise for 2 weeks, then walked in hand for 15 minutes, three times daily for 6 weeks.

2.3. Ultrasonographic Examination

The proximal aspect of the suspensory ligament of both thoracic limbs of each horse was evaluated ultrasonographically, after clipping hair over the ligaments, for thickness and for the presence or absence of long linear parallel echoes, before 2 and 8 weeks after injecting collagenase and 8 weeks after the DBLPaN was excised. Measurements of dorsopalmar thickness (DPT) of the suspensory ligament were made in the longitudinal plane with the horse standing, using a 10 MHz, 38-mm linear probe (Mindray-DP-50), and images were stored for comparison. For each limb, measurements were taken 2 to 4 cm from the base of the fourth metacarpal bone end of the accessory carpal bone that corresponded to the site of injection of collagenase. Scores of echogenicity, 0 to 3, were assigned to images acquired in the longitudinal plane, using a previously described scoring system [15]. A score of 0 was