Gracilis muscle injury as a cause of lameness in two horses

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EQUINE

- Injury to the gracilis muscle can cause acute, severe lameness in horses.
- Horses with gracilis muscle injuries have a good prognosis for returning to athletic performance after prolonged rest.
- Fibrotic myopathy can develop secondary to gracilis muscle injury.

A 16-year-old female Quarter Horse (horse 1) weighing 470 kg (1,034 lb) was evaluated because of left hind limb lameness of 5 days' duration. The horse was being used for barrel racing. The horse became severely lame after turning around a barrel in a competition 5 days prior to the evaluation and was initially hesitant to bear weight on the left hind limb. On physical examination, the horse had increased digital pulses in the left front foot, digital tendon sheath effusion in the left forelimb, and a soft fluid-filled swelling on the medial aspect of the gracilis muscle belly of the left hind limb. Lameness evaluation revealed a grade 3/5[1] left hind limb lameness. Flexion of the lower portion of the limb did not alter the grade of lameness, but a flexion test performed on the upper portion of the limb increased the degree of lameness to grade 4/5. Perineural diagnostic analgesia was performed to localize the source of lameness. Analgesia of the lower portion of the limb via a low plantar nerve block followed by a peroneal and tibial nerve block did not modify the lameness. Intra-articular analgesia of the left medial and lateral femorotibial, femoropatellar, and hip joints did not change the lameness. Local infiltration of 30 mL of mepivacaine[a] anesthetic into the medial aspect of the gracilis muscle at the site of the fluid-filled swelling resulted in considerable improvement in the left hind limb lameness. Ultrasonographic evaluation of the gracilis muscle of the left hind limb revealed an anechoic fluid space containing fibrous trabeculation within the muscle, which was suggestive of muscle tearing with seroma or hematoma formation (**Fig 1**).

The medial aspect of the gracilis muscle was clipped and aseptically prepared; via needle aspiration, approximately 40 mL of slightly hemorrhagic, serous fluid was obtained. The fluid was drained, and the area was injected locally with triamcinolone[b] (10 mg) and amikacin[c] (100 mg). The owner was instructed to confine the horse to a small run (approx 12 × 30 feet) and hand walk the horse 20 minutes daily for 8 weeks. It was also recommended to apply dimethyl sulfoxide to the region of the gracilis muscle twice daily for 10 days and administer phenylbutazone (2.2 mg/kg [1 mg/lb], PO, q 24 h) for 10 days.

Three months after the diagnosis of gracilis muscle injury was made, the horse was reevaluated. On physical examination, a firm palpable thickening over the medial aspect of the gracilis muscle was detected at the location of the previous injury site. Lameness evaluation revealed that the horse walked with an abnormal gait characterized by a shortened cranial swing phase of the left hind limb and slapping the left hind foot downward just before the end of the cranial phase of every walk stride. This gait was characteristic of horses with fibrotic myopathy. A repeat ultrasonographic examination by use of a 10.0-MHz transducer revealed a bright echogenic fibrous band that originated near the middle aspect of the gracilis muscle and extended distally to incorporate the soft tissue approximately 8 cm distal to the end of the gracilis muscle (**Fig 2**). The fibrous band was 16 mm in thickness in the proximal portion and approximately 19 mm in thickness in the distal aspect.

The horse was sedated with detomidine (0.5 mg, IV) and butorphanol (10 mg, IV). On the caudal aspect of the thigh, the hair was clipped from the region of the stifle joint to the pubic bone over the gracilis and semimembranosus muscles and the skin was aseptically prepared for surgery. Local infiltration of 60 mL of mepivacaine (placed in an inverted U pattern) was administered to desensitize the area prior to surgery. An 8-cm vertical incision was made directly over the palpable fibrotic band located approximately in the ventral third of the gracilis muscle. A large curved hemostat and digital palpation were used to isolate the circumference of the band of scar tissue. The fibrotic band was severed in cross section, and the proximal and distal ends of the fibrotic band were displaced approximately 5 cm. The subcutaneous tissue was apposed by use of size-0 polydioxanone suture in a simple continuous suture pattern, and the skin was apposed by use of size-1 polypropylene suture in an interrupted vertical mattress suture pattern. A rolled gauze stent was sutured over the incision. Perioperatively, phenylbutazone (2.2 mg/kg, PO, q 12 h) and penicillin G potassium (10,000 U/kg [4,545 U/lb], IV, q 6 h) were administered for 3 days. At discharge, the owner was instructed to allow the horse access to a small turnout area for 6 weeks before returning to barrel racing. The horse returned for a reevaluation 3 weeks after surgery. The horse's gait on the left hind limb was unchanged. The owner was instructed to continue the horse's low-level activity in the small turnout area. Eight weeks after surgery, the horse was returned for lameness evaluation; at that time, it had a normal gait with no signs

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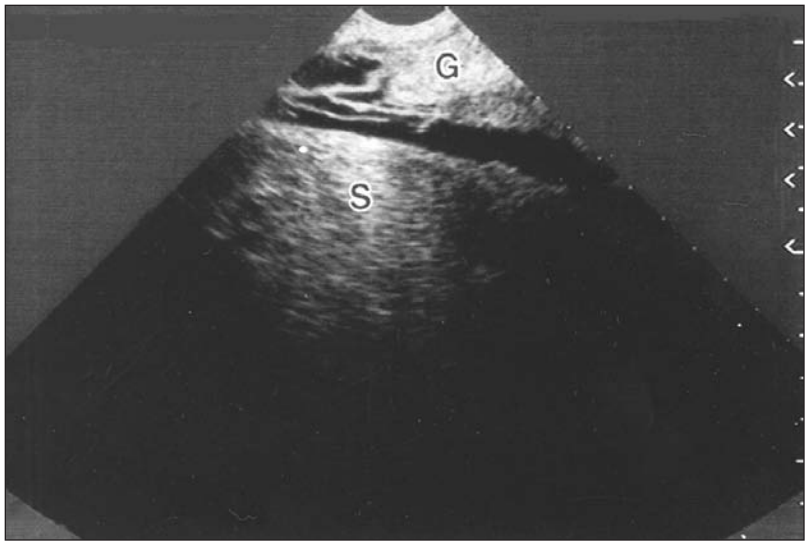

Figure 1—Transverse ultrasonographic image of the caudal aspect of the left thigh of a horse at the time of gracilis muscle injury. Notice that the gracilis muscle (G) has anechoic fluid and fibrous trabeculation at its distal aspect. The semimembranous muscle (S) is seen adjacent to the gracilis.

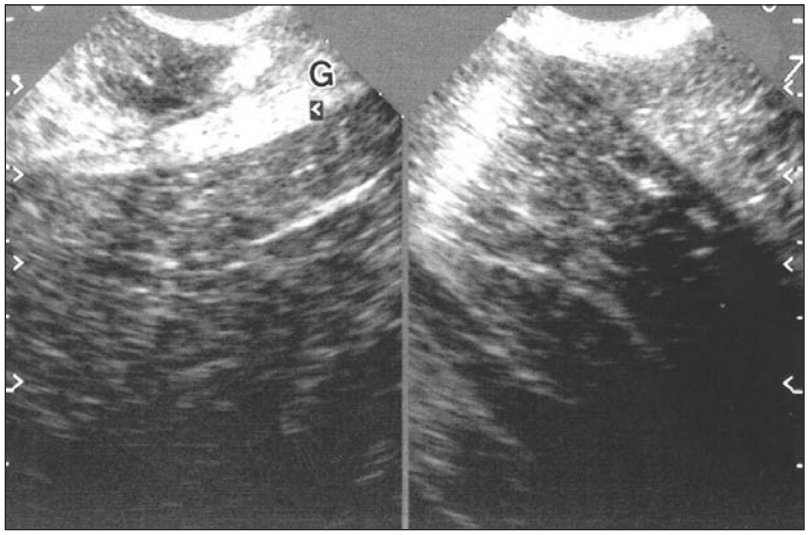

Figure 2—Longitudinal ultrasonographic images of the caudal aspect of the muscles of the left (image to left) and right (image to right) thighs of the horse in Figure 1 three months after the initial injury. Notice the hyperechoic fibrous band (arrow) within the gracilis muscle (G). In both images, the top of the view is oriented toward the tailhead of the horse.

of lameness. Ultrasonographic reevaluation of the gracilis muscle revealed evidence of healing of the gracilis muscle. The previously identified thick fibrous band involving the ventral aspect of the gracilis muscle was no longer visible. The left gracilis muscle had a slight increase in echogenicity, compared with that of the right gracilis muscle. The horse returned to barrel racing activity at its previous level of competition; 2 years after the original injury, there had been no further problems reported.

A 17-year-old female Quarter Horse (horse 2) weighing 489 kg (1,076 lb) was evaluated because of lameness. Two days before this evaluation, the horse was running a barrel race pattern during competition and became severely lame after turning around the second barrel. The horse was lame (grade 4/5) in the right hind limb. Results of an examination with hoof testers were negative, and no palpable abnormalities were evident in the lower aspect of the limb. Heat and signs of pain were detected on palpation of the right gracilis muscle. The horse became violent and kicked when the right gracilis muscle was palpated but had no response to palpation of the left gracilis muscle. Local infiltration of 70 mL of mepivacaine into the right gracilis muscle resulted in notable improvement in lameness, and the horse was subsequently less responsive to palpation of that muscle. Ultrasonographic evaluation revealed a slight increase in echogenicity of the right gracilis, compared with that of the left gracilis muscle; in addition, there was asymmetry in the size and contour of the right gracilis muscle that was subjectively compatible with intramuscular hemorrhage. Results of serum biochemical analyses revealed high **creatine kinase (CK)** activity (620 U/L; reference range, 73 to 450 U/L) and high **aspartate aminotransferase (AST)** activity (1,540 U/L; reference range, 134 to 643 U/L). A diagnosis of gracilis muscle injury was made. The owner was instructed to allow the horse access to a small turnout area for 8 weeks with 20 minutes of hand walking daily. The owner was also instructed to apply dimethyl sulfoxide to the region of the right gracilis muscle twice daily for 10 days. Eight weeks after the initial injury, the horse was reevaluated. Physical examination revealed no signs of pain on palpation of the right gracilis muscle; however, a section (12 × 5 cm) of the gracilis muscle was atrophied, and a depression in the muscle was evident. Lameness evaluation revealed continued right hind limb lameness (grade 2/5). The owner was instructed to continue to allow the horse access to the small turnout area and proceed with hand walking for an additional 8 weeks. Six months after the initial injury, the horse returned to barrel racing competition. At 1.5 years after the initial injury, the horse was competing in barrel races at its previous level of competition.

Muscle injuries are uncommonly reported as a cause of lameness in horses. Injury to the gastrocnemius muscle,[2] muscle strains involving the longissimus and gluteal muscles,[3] and muscle strain of the combined muscles of the caudal region of the thigh (including the semimembranosus, semitendinosus, and biceps femoris[3] muscles) have been reported in horses. To our knowledge, injury solely to the gracilis muscle has not been described as a cause of lameness in horses.

The severity of muscle strain is classified from first to third degree. First-degree muscle strain occurs when the limits of muscle elasticity have been reached but not exceeded and is commonly referred to as a pulled muscle.[3] Second-degree muscle strain is consistent with tearing of muscle fibers and muscle hemorrhage; it occurs when the elasticity of the muscle has been exceeded but the continuity of the muscle is maintained. Third-degree muscle strain is a complete muscle tear and loss of muscle continuity. Ultrasonographically, muscle fiber disruption was identified in horse 1, and intramuscular hemorrhage was detected in both horses of this report. On the basis of muscle strain classification, the horses of this report probably each sustained a second-degree injury to the gracilis muscle; however, compared with the injury in horse 2, the muscle strain in horse 1 appeared more severe on ultrasonographic examination.

Muscle injuries can occur when there is an incoordination of movement with sudden alteration in tension and contraction of muscle fibers. Muscle fatigue will often predispose the muscle to strain by decreasing the elasticity of the muscle fibers.[4] Both horses in this report sustained a gracilis muscle injury during a barrel race competition. Barrel racing is a timed event in which the horse and rider run a cloverleaf pattern, turning 360° around each of 3 barrels, before sprinting to the finish.[5] Located caudal to the sartorius muscle, the gracilis muscle is a wide, quadrilateral muscle that covers the greater aspect of the medial surface of the thigh. It originates on the ventral surface of the pubis caudal to the prepubic tendon and inserts on the medial patellar ligament and medial surface of the tibia. The gracilis muscle functions to adduct the hind limb.[6] Because the injury occurred during the barrel race in each of the horses of this report, it is possible that fatigue played a role in causing the gracilis muscle injury. Both horses ran to the right to reach the first barrel; although both riders reported that the horses were apparently normal prior to the competition, lameness was noticeable as the horses walked immediately after finishing the barrel race pattern. It is also possible that barrel racing activity itself predisposes the gracilis muscle to injury because the competing horse adducts the outside hind limb while turning 360° around barrels during the event. At the Texas Veterinary Medical Teaching Hospital, approximately 150 barrel-racing horses are evaluated because of lameness each year; therefore, in our experience, 2 horses with gracilis muscle injuries is considered uncommon. It may be coincidental that both horses were used for barrel racing. Gracilis muscle injury has been described in horses used in polo competitions[7]; it is possible that this type of injury may be unrelated to the activities of horses. To determine the cause of gracilis muscle injury in horses, a larger sample population would be required.

It is interesting that both horses of this report were female Quarter Horses of similar age. Quarter Horses are most commonly used for western performance events, but geldings are usually preferred.[5] One study[8] described 39 horses with fibrotic myopathy of the semitendinosus muscle. In that study, Quarter Horses (67%) and mares (69%) were overrepresented in the study group, compared with the breeds and sex distribution typical of the overall horse population of that hospital.[7] It is unknown whether female Quarter Horses are predisposed to injuries in the muscles of the caudal aspect of the thigh or whether this is a coincidental finding.

Injuries to muscles in the upper portion of limbs that cause lameness can be a diagnostic challenge. Diagnostic modalities such as radiography and typical peripheral nerve analgesia are not particularly useful in localizing the source of lameness. In the horses of this report, diffuse local infiltration of mepivacaine into the suspected injury site resulted in improvement in lameness and proved to be helpful in the diagnoses. In horse 2, measurements of the activities of CK and AST in serum were helpful in assessing the degree of muscle damage. Serum CK activity is a most sensitive and specific indicator of muscle damage; however, subsequent to muscle damage, the value peaks at approximately 6 hours and declines after 2 to 3 days. Serum AST activity peaks in 24 hours and declines in 7 to 14 days after muscle damage. Horse 2 was evaluated on the third day after injury. At that time, the serum CK activity had most likely reached a peak and was declining (the value at evaluation was just higher than the upper reference limit), whereas the serum AST activity was quite high, compared with reference limits, and therefore more useful diagnostically. Many other reasons could explain the increases in these serum enzyme activities (eg, exertional myopathy and strenuous exercise), but we found the data useful in the diagnosis of muscle injury in the horse with the less severe lesion (as detected ultrasonographically). Ultrasonographic examination was useful in the diagnosis of gracilis muscle tearing in both horses of this report, but was particularly useful for monitoring healing in horse 1. After the initial assessment, horse 2 was not reexamined ultrasonographically because a fibrous band was not palpable at the injury site, and the horse was sound at the time of reevaluation. Thermography or nuclear scintigraphy was not performed in either horse of this report, but such procedures may be of use in identification of injuries to the muscles of the caudal aspect of the thigh in horses.[3]

For the horses of this report, the primary treatment was controlled exercise in a small area with hand walking daily. Access to a small turnout area was recommended to enable the muscle to heal completely without allowing the horse to run; the periods of hand walking were recommended in an attempt to prevent scar tissue and adhesion formation, which nevertheless developed in horse 1. Both horses had similar treatment after injury, but the muscle strain in horse 1 was more severe than that in horse 2, which may have been the reason that horse 1 developed fibrotic myopathy. Fibrotic myopathy is a gait abnormality of the hind limbs of horses that is caused by scar tissue formation or fibrosis within the semitendinosus, semimembranosus, or biceps femoris muscles. There is a report[9] of fibrotic myopathy involving the gracilis muscle in an Arabian horse, but cause of the lesion was not

described. Affected horses have a characteristic gait consisting of an abrupt shortening of the anterior phase of the stride with slapping of the hoof to the ground. Damage to the muscles of the caudal aspect of the thigh may be a result of blunt trauma, falling, or IM administration of injections in that area.[8,10] To our knowledge, there are no other reports of fibrotic myopathy of the gracilis muscle in horses secondary to a work-related injury (as occurred in horse 1 of this report). In horses, fibrotic myopathy has been reported to develop 30 days after blunt trauma to the area,[8] but horse 1 of this report developed the characteristic gait 3 months after straining the muscle. The interval from injury to development of muscle fibrosis may vary with cause of muscle injury.

Three surgical procedures have been described for treatment of fibrotic myopathy, including semitendinosus myotenectomy (transection of the area where the muscle belly and tendon join together),[3] semitendinosus tenotomy at the tibial insertion,[11] and standing semitendinosus myotomy.[8] Incisional complications are common in horses undergoing semitendinosus myotenectomy, and only 1 of 11 horses was free of lameness or gait deficit at follow-up 3 months after surgery in 1 study.[3] In comparison, 4 horses undergoing semitendinosus tenotomy at the tibial insertion had fewer incisional complications, and all those horses returned to athletic use with a normal hind limb gait.[11] Thirty-nine horses with fibrotic myopathy were evaluated after undergoing semitendinosus myotomy (standing surgery), and 29 of 39 horses with follow-up were able to return to their preinjury level of use.[8] Incisional complications were noted in 12 of 32 (38%) of the horses in that study. Although fewer horses undergoing semitendinosus tenotomy were evaluated, it appears that a better outcome with fewer complications is associated with that surgical procedure, compared with the outcome associated with semitendinosus myotomy. In the horses of this report, we performed gracilis myotomy (standing procedure) because of the anatomic variation in gracilis muscle insertion, compared with the uniformity of the insertion of the semitendinosus muscle. The gracilis muscle inserts on the medial patellar ligament and the medial surface of the tibia cranial to the medial femorotibial ligament. At the time, we were uncertain of potential complications that might have developed in association with gracilis tenotomy, such as stifle joint instability. Although only a single case, horse 1 returned to its preinjury level of performance after gracilis myotomy. After surgery, horse 1 retained the abnormal gait typical of fibrotic myopathy for 30 days before returning to normal. The interval after surgery to improvement in gait is variable; improvement may be immediate or occur as much as 2 months after surgery.[3,8,11] Owners of treated horses should be notified that immediate improvement may not occur but that with time, the abnormal gait often does improve after surgical correction.

Both horses of this report with gracilis muscle injuries returned to athletic performance. The time from injury to return to barrel racing was between 5 and 6 months for both horses. These results suggest that horses sustaining a gracilis muscle injury have a good prognosis for returning to athletic use after an adequate period of muscle healing; however, fibrotic myopathy or muscle atrophy could be a complication of the injury, resulting in persistent gait deficits.

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