

Case Report

Agnesis of the *peroneus tertius* in a foal

F. CALDWELL*, R. TUDOR AND L. NEUWIRTH

Department of Large Animal Medicine, College of Veterinary Medicine, University of Georgia, Athens, Georgia 30602, USA.

Keywords: horse; lameness; reciprocal apparatus; *peroneus tertius*; agnesis

Introduction

Congenital deformities involving tendons and ligaments can be commonly observed in foals. Complete absence or agnesis of a component of the equine distal limb has not previously been reported in the veterinary literature to the author's knowledge. Congenital absence of the *peroneus tertius* muscle in addition to multiple other anatomical abnormalities has been reported in cases of the severe genetic disorder trisomy 13 (Patau's syndrome) in man (Colacino and Pettersen 1978; Aziz 1980).

This report presents an unusual case of agnesis of the *peroneus tertius* in a foal causing an incomplete reciprocal apparatus and clinical lameness.

Case history

A 4-month-old American Quarter Horse colt was presented for evaluation of bilateral hindlimb lameness. At birth the colt exhibited laxity of the flexor tendons and had an angular limb deformity of the hindlimbs characterised as 'windswept' (i.e. tarsal varus of one limb and tarsal valgus of the opposite). These abnormalities had improved by 3 weeks of age. The owner described the colt as moving in an abnormally short-strided manner in the hindlimbs beginning at approximately 4 weeks of age.

Clinical findings

Physical examination

Upon presentation at the University of Georgia Large Animal Teaching Hospital, the colt was bright, alert and responsive. Temperature, heart and respiratory rates were 38°C, 104 beats/min and 36 breaths/min, respectively. The colt had not been weaned from the mare and was large and heavily-muscled. Mild swelling of the distal physes was present in the



Fig 1: The foal with its right stifle flexed and hock extended demonstrating incomplete reciprocal apparatus. Notice the dimpled appearance of the common calcaneal tendon.

third metacarpal and metatarsal bones. The colt was subjectively considered to be very upright in the pasterns and had increased dorsal angulation in the hocks. When observed moving from the side there was a shortened anterior phase to the stride in both hindlimbs. A slight laxity of the common calcaneal tendons bilaterally was observed while viewing the colt moving from behind. When the hindlimbs were examined, the hocks could be extended while the stifles remained in flexion; tension was completely released within the common calcaneal tendons resulting in a dimpled appearance (**Fig 1**). Bilateral rupture of the *peroneus tertius* (PT) tendon was suspected; however, no pain or swelling was palpable over the cranial-lateral aspect of the stifle or tibia.

Diagnostic procedures

An ultrasound examination of both hindlimbs was performed after the colt was sedated with xylazine (0.3 mg/kg bwt i.v.); the PT could not be identified ultrasonographically (L12-5 linear array transducer, ATL 5000)¹ in either limb (**Fig 2**). Hyperextension of the tarsocrural joint and mild tarsometatarsal subchondral bone irregularity consistent with

*Author to whom correspondence should be addressed. Present address: J. T. Vaughan Large Animal Teaching Hospital, 1500 Wire Road, Auburn University, Auburn, Alabama 36849, USA.

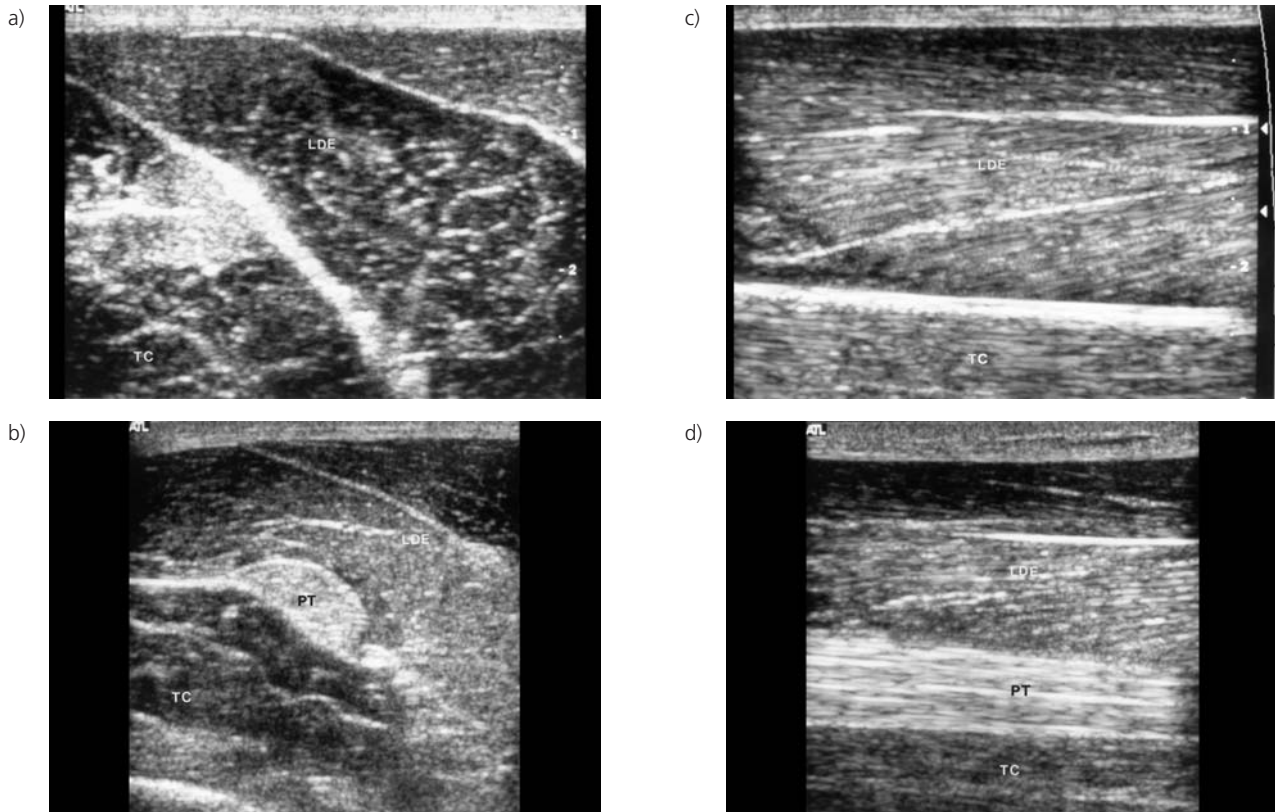


Fig 2: Transverse lateral oblique (a and b) and long lateral oblique (c and d) ultrasonographic images of the cranial aspect of the proximal tibial region of the colt (a and c) compared to a normal horse (b and d). Notice the absence of the peroneus tertius (PT) between the tibialis cranialis (TC) and long digital extensor (LDE) muscles in the colt. On the transverse images medial is to the left and on the sagittal images proximal is to the left.

juvenile osteoarthritis (spavin) were identified on lateral and dorso-lateral plantaro-medial oblique radiographs of the right and left tarsi. On bilateral stifle radiographs the femoral condyles were misshapen and appeared smaller than normal for the colt's size (**Fig 3**).

Treatment and outcome

Because of the uncertainty of his ability to perform athletically, the owner elected to donate the colt to the College of Veterinary Medicine. His development was followed for one year and the abnormal gait did not improve. The horse was subjected to euthanasia at approximately 18 months of age.

Post mortem examination

No evidence of either PT could be found upon gross dissection of the hindlimbs. The long digital extensor muscle did not traverse the lateral femorotibial joint and was found to originate from the deep fascia on the cranial-lateral aspect of the proximal tibia instead of the extensor fossa of the femur. The distal femurs were grossly abnormal, with the most notable features being the absence of extensor fossae, small, flattened femoral condyles, and less prominent lateral trochlear ridges that were short in length.

Discussion

The equine PT is entirely tendinous and originates from the extensor fossa of the lateral femoral condyle with the *M. extensor digitorum longus* (long digital extensor). It forms a sleeve (peroneal manica) distally through which the tendinous part of the *tibialis cranialis* (TC) passes, enclosing and stabilising the TC tendon as it continues across the dorsal



Fig 3: Lateromedial radiograph of the stifle. Notice the small misshapen femoral condyles and absence of the extensor fossa (a) when compared to a normal age-matched foal (b).

aspect of the tarsocrural joint (Sack and Ferraglio 1978; Lohse and Trout 1984; Updike 1984). There are 4 distinct tendons of insertion of the PT, attaching to portions of the central, 3rd, 4th tarsal bones, the 3rd metatarsal bone and the calcaneus (Updike 1984).

The function of the PT is important to the normal reciprocal action of tarsal flexion with stifle flexion. It is the sole cranial component of the reciprocal mechanism and part of the passive stay apparatus, assisting the horse in standing with minimal effort (Trout and Lohse 1981). The PT stretches during extension of the hock, centring the weight on the tarsus and recoiling elastically following break-over. This release of stored elastic energy during the terminal phase of the stride assists with efficient flexion of the hock (Trout and Lohse 1981; Lohse and Trout 1984). Therefore an intact PT is important to the normal combined movement of hock and stifle flexion.

In horses with acute disruption of the PT, swelling and pain can usually be palpated over the cranial-lateral aspect of the tibia. Upon further examination of the limb, the tarsus can be extended while the stifle is flexed and a bunching of the common calcaneal tendon is observed. As the horse is observed from the side at the walk, there is reduced flexion of the hock during the anterior phase of the stride. When observed from behind, there is a brief period of laxity of the common calcaneal tendon as the hindfoot lands. In the more chronic injury, swelling and pain subsides, and gait abnormalities are not as evident as fibrous healing of the PT occurs. This colt demonstrated clinical symptoms of PT disruption without historical or clinical evidence of prior trauma.

The normal PT is visualised on a transverse ultrasonogram as a well-defined, hyperechoic, oval structure between the long digital extensor and the *M. tibialis cranialis* (cranial tibial muscle) (Dik 1993; Léveillé *et al.* 1993). On a longitudinal ultrasonogram, the tendon fibres are aligned in parallel longitudinally along the entire length of the tendon. Most ruptures in adult horses are reported to occur in the mid-body or at the insertion of the tendon (Szabuniewicz and Titus 1967; Koenig *et al.* 2005). Foals usually sustain avulsions at the origin of the tendon, presumably because the immature bone is the weakest portion of the bone/tendon unit (Blikslager and Bristol 1994). Avulsion fragments may originate from the extensor fossa and can be identified as hyperechoic structures with an acoustic shadow distal to the extensor fossa in the proximal segment of the PT. Surgical intervention is not recommended unless there are avulsion fragments present in the lateral femorotibial joint; then, exploratory arthroscopy is recommended to remove fragments to minimise the chances of development of degenerative joint disease in the future (Holcombe and Bertone 1994).

The absence of a normal structural component of the distal limb may be explained by agenesis or aplasia. The authors could not find a report in the veterinary literature documenting any of these possibilities. A congenital unilateral flexural deformity of the hock in a foal caused by an abnormally short PT has been previously reported (Trout and Lohse 1981). It was

postulated that abnormal uterine position could have resulted in the deformity. In this case, surgical resection was used to correct the deformity and allow full hock extension. Four months post operatively the foal had full range of motion and no noticeable signs of lameness when moving.

Several pieces of evidence suggest that agenesis was the cause of this colt's condition. History of an abnormal gait that could have been present since birth and was certainly present at 4 weeks of age suggests the condition may have been congenital. Additionally, the gross abnormalities of the distal femurs on *post mortem* examination are compatible with a congenital condition. Symmetrical trauma without evidence of fibrotic remnants is inconsistent with an acquired condition. It is therefore proposed that an embryological error may have occurred in this foal causing an alteration in the formation of the distal femur. This additionally could have affected the normal development of the supportive structures of the distal limb resulting in the congenital bilateral absence of the PT tendons and the foal's perceived lameness.

Manufacturer's address

¹Philips Medical Systems, Bothell, Washington, USA.

References

- Aziz, M.A. (1980) Anatomical defects in a case of trisomy 13 with a D/D translocation. *Teratology* **22**, 217-227.
- Blikslager, A.T. and Bristol, D.G. (1994) Avulsion of the origin of the *peroneus tertius* tendon in a foal. *J. Am. vet. med. Ass.* **204**, 1483-1485.
- Colacino, S.C. and Pettersen, J.C. (1978) Analysis of the gross anatomical variations found in four cases of trisomy 13. *Am. J. med. Genet.* **2**, 31-50.
- Dik, K.J. (1993) Ultrasonography of the equine crus. *Vet. Radiol.* **34**, 28-34.
- Holcombe, S.J. and Bertone, A.L. (1994) Avulsion fracture of the origin of the extensor *digitorum longus* muscle in a foal. *J. Am. vet. med. Ass.* **204**, 1652-1654.
- Koenig, J., Cruz, A., Genovese, R., Fretz, P. and Trostle, S. (2005) Rupture of the *peroneus tertius* tendon in 27 horses. *Can. vet. J.* **46**, 503-506.
- Léveillé, R., Lindsay, W.A. and Biller, D.S. (1993) Ultrasonographic appearance of ruptured *peroneus tertius* in a horse. *J. Am. vet. med. Ass.* **202**, 1981-1982.
- Lohse, C.L. and Trout, D.R. (1984) Equine limb anatomy: *peroneus tertius* muscle relationships. *Zbl. Vet. Med. C. Anat. Histol. Embryol.* **13**, 313-318.
- Sack, W.O. and Ferraglio, S. (1978) Clinically important structures of the equine hock. *J. Am. vet. med. Ass.* **172**, 277-280.
- Szabuniewicz, M. and Titus, R.S. (1967) Rupture of the *peroneus tertius* in the horse. *Vet. Med. small anim. Clin.* **62**, 993-995.
- Trout, D.R. and Lohse, C.L. (1981) Anatomy and therapeutic resection of the *peroneus tertius* muscle in a foal. *J. Am. vet. med. Ass.* **179**, 247-251.
- Updike, S.J. (1984) Anatomy of the tarsal tendons of the equine *tibialis cranialis* and *peroneus tertius* muscles. *Am. J. vet. Res.* **45**, 1379-1382.