Congenital tarsal hyperextension in three cats

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Case Description—3 kittens were examined because of a malformation affecting the hind limbs, resulting in an inability to bear weight or ambulate normally.

Clinical Findings—2 kittens were younger than 6 weeks of age, and 1 was 4 months of age at the time of initial examination. The congenital abnormality was characterized by severe tarsal hyperextension in which weight was borne on the cranial aspect of the tarsus, and the plantar surface of the metatarsus faced dorsally. In 2 kittens, the condition affected both hind limbs, and in the older kitten, the condition was unilateral. In the 2 kittens in which radiographs were obtained, no bone abnormalities were detected. Full-cylinder fiberglass casts were applied and changed weekly to accommodate growth. Owners administered physical therapy after final cast removal.

Treatment and Outcome—Conservative management involving external coaptation and physical therapy led to favorable results in all 3 cats.

Clinical Relevance—Although further studies are needed to determine the etiology of the disorder, affected kittens may be successfully treated with conservative management. Owners should be committed to the necessity for returning cats for serial cast changes, care for pressure sores, and administration of physical therapy after cast removal.

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From Angell Animal Medical Center-Boston because of abnormal conformation involving the hind limbs. The kitten’s parents were known to be anatomically normal. The affected kitten was the only kitten in the litter and had been born with bilateral hind limb abnormalities that had neither progressed nor improved with age. No other physical abnormalities were observed. The owner had researched “twisted leg disease” on the Internet and brought the kitten in for evaluation and possible treatment.

The only musculoskeletal abnormality observed during the initial physical examination was severe bilateral tarsal hyperextension such that the kitten bore weight on the cranial aspect of the tarsus (Figure 1). No palpable bone abnormalities were detected. The tibiotarsal joints could be flexed to an angle of approximately 135° (typical angulation of the tarsus in standing position), but the manipulation required sufficient force to elicit signs of discomfort in the kitten. No neurologic deficits were observed.

Radiographs were not obtained because of the kitten’s age and the assumption that bone ossification would be incomplete. The kitten was sedated with oxymorphone (0.03 mg/kg [0.013 mg/lb], SC), midazolam (0.3 mg/kg [0.13 mg/lb], SC), and propofol (2.0 mg/kg [0.9 mg/lb], IV) to facilitate examination and application of a splint to each hind limb. Fiberglass splints were placed on the lateral aspect of each limb from the mid-tibial level to the digits to maintain the tarsus in an appropriate weight-bearing position. The laterally placed splints were ineffective at maintaining limbs in the proper position and were changed 1 week later so that they were positioned on the cranial aspect of the limbs. This method of splinting was also ineffective. The following week, full-cylinder fiberglass casts were placed on both hind limbs from the level of the proximal portion of the tibia to the digits. Casts were changed weekly to allow for growth. By the third week after cast application, the kitten was able to ambulate while wearing the casts (Figure 2). Pressure sores with purulent discharge developed near the medial and lateral malleoli and were cleansed with a dilute chlorhexidine solution and partially closed with skin staples. The kitten was treated with amoxicillin (22 mg/kg [10 mg/lb], PO, q 12 h) for 7 days.

The cast on the kitten’s left hind limb was maintained for 8 weeks, and the cast on the right hind limb was maintained for 14 weeks. After final cast removal, the owner was instructed to perform physical therapy to increase the range of flexion of the tibiotarsal joints. This entailed forcing the kitten to walk on the hind limbs by manually elevating the forelimbs and by passive manipulation of each tarsal joint. Physical therapy was prescribed for a minimum of 3 to 4 times each day.

Figure 1—Photograph of a 5-week-old kitten that was examined because of a congenital anatomic defect involving the hind limbs. Notice the severe tarsal hyperextension and inward metatarsal rotation resulting in weight being borne on the cranial surface of the tarsi with the plantar surface of the metatarsus facing dorsally.
for 10-minute periods until normal limb position and range of motion in the tarsus were observed. One year after the initial examination, the owner reported that the cat was using both hind limbs well, although the right limb was still disfigured. The owner acknowledged discontinuing physical therapy 1 month after the last appointment.

A 4-week-old female domestic shorthair kitten weighing 0.63 kg (1.4 lb; case 2) was surrendered to the Angell hospital shelter. The kitten was the only kitten in the litter and was the offspring of a mother-son mating. Physical examination revealed severe tarsal hyperextension in both hind limbs. The kitten was able to drag itself with the forelimbs but could not bear weight on the hind limbs.

The kitten was sedated, and full-cylinder fiberglass casts were placed at the level of the proximal portion of the tibia and molded such that the tarsal joints were maintained in a weight-bearing position (approx 135°). Meloxicam (0.1 mg/kg [0.045 mg/lb], SC) was administered for analgesia. Casts were changed every 7 to 10 days to accommodate growth. Between visits, the owner observed the kitten to be ambulatory while wearing the casts. A pressure sore that developed on the left tarsus was managed with cleansing and administration of amoxicillin (22 mg/kg, PO, q 12 h) for 7 days.

Six weeks after the first cast was applied, the cast slipped off the right hind limb, and the owner was instructed to perform physical therapy. At a recheck examination 1 week later, the cat was using the right hind limb well and improvement was observed in the position of the right tarsus. The tarsus had a decreased range of motion (range of flexion, 90°), but the cat was able to properly bear weight on the paw. The cast on the left limb was removed, and physical therapy was continued on both hind limbs. Radiographs of both hind limbs were obtained at 10 weeks of age, and no skeletal abnormalities were observed. The cat was examined every 2 weeks for the next 6 weeks, and improvement was evident at each visit. Sixteen weeks after the initial examination, the cat had a slightly noticeable decrease in flexibility of the left tarsus but was otherwise ambulating normally (Figure 3).

A 4-month-old male kitten (case 3), which had been adopted from an animal shelter 1 month earlier, was examined. The kitten had been surrendered to the shelter with a female littermate that was clinically normal. Severe tarsal hyperextension in the left hind limb was observed with the kitten bearing weight on the cranial surface of the tarsus. The right hind limb was anatomically normal. Radiographs of the affected limb revealed no skeletal abnormalities. After sedation, a fiberglass cast was placed on the left hind limb from the mid-tibial level to the digits. In this kitten, even with sedation, it was not possible to reduce the tarsal hyperextension to 180° of angulation without inducing signs of pain. A single dose of meloxicam (0.1 mg/kg, SC) was administered after application of the cast.

The cast was changed every 7 days. While the kitten was sedated, the tarsal joint was progressively flexed prior to cast reapplication until a weight-bearing angle of approximately 135° was achieved. The cat ambulated well between visits while wearing the cast. After 4 weeks of casting, the tarsus remained at a 135° angle. The cast was removed at this time, and the owner initiated physical therapy. The kitten was examined 3 times during the next 2 months, and use of the limb progressively improved. Although the cat walked on the plantar aspect of the paw appropriately, the tarsus remained in an extended position. At that time, the tarsus could be flexed to an angle of approximately 90° without causing signs of discomfort.

**Discussion**

Congenital hyperextension of the tarsus occurs infrequently in cats and has not been reported in the veterinary literature to our knowledge. The common calcaneal tendon is composed of 5 structures, most importantly the superficial digital flexor tendon and tendon of the gastrocnemius muscle. Other contributing structures are the tendons of the biceps femoris, gracilis, and semitendinosus muscles. Disruptions of the tendons can lead to various clinical signs including plantigrade stance, curling toes, swelling over the tarsus, and severe lameness; methods of diagnosis and
manipulation for these disorders are well known.\(^1\)\(^6\) Talocalcaneal luxation, which results in similar clinical signs to those observed in the cats of this report, has been reported in companion animals.\(^7\) A search of the Internet revealed many nonveterinarian-hosted sites in which “twist-leg cats” or “twisted limbs” in cats were discussed; however, a literature search disclosed no peer-reviewed reports describing the condition.\(^8\)

An anatomically similar condition (ie, clubfoot or talipes equinovarus) has been described in Caucasian human infants. The structural abnormalities in affected infants are similar to those described in the kittens of this report. In humans, physical examination findings include the ankle and heel being positioned in equinus (severe flexion at ankle joint) and varus (heel pointed laterally) configurations, with the front part of the foot supinated (internal rotation) and with metatarsus adductus and calf-muscle atrophy.\(^9\)\(^10\) In affected humans, the defect occurs in both legs approximately 50% of the time and is present at birth. The defect is thought to arise from 3 etiologies. Postural clubfoot develops as a result of intrauterine molding and may be self-correcting or respond to conservative treatment. In other infants, the condition is linked with neurogenic or teratogenic causes and in association with other neuromuscular disorders (eg, spina bifida and arthrogryposis).\(^10\)\(^11\) Most (75%) cases in humans are of unknown etiology, although a genetic relationship involving a single autosomal dominant gene has been proposed.

Breeding histories were not available for the cats of the present report, so determination of a genetic component to the disease was not possible. Two cats were singleton kittens, which should have allowed for more space in the uterus and a decreased likelihood of intrauterine malpositioning. Neither concurrent congenital defects nor neurologic deficits were observed in any of the cats. A primary muscular cause could not be ruled out because muscle biopsy was not performed in any of the cats, but such information would be of interest in future studies.

The treatment of choice in humans is initiation of casting shortly after birth. Other treatments investigated have included methods of soft tissue release (ie, posterior, medial, or plantar tenotomy procedures), as well as application of malleable splints and taping.\(^13\)\(^17\) However, casting has the best outcome and is the least invasive. In the present study, cats responded favorably to conservative treatment that included external coaptation followed by physical therapy. Strict owner adherence to cast care measures and a long-term commitment to returning cats on a weekly basis for reevaluation and cast changes are necessary. The only complication observed in the cats of this study was development of pressure sores associated with the casts, but cleansing, administration of antimicrobials, and skin stapling yielded good outcomes in each instance. Although casting can be challenging in pediatric veterinary patients, initiation of proper tarsal positioning and stretching of the musculotendinous unit of the common calcanean tendon at as young an age as possible appeared to be important. The soft tissues of the third and oldest cat were more difficult to manipulate, necessitating that stretching be staged. Two of the cats had signs of pain during recovery from sedation after application of the first cast, presumably as a result of stretching of the common calcanean tendon and other soft tissues. It appears that treatment of this condition should be initiated as early as possible to take advantage of the favorable fibroelastic properties of soft tissues in and around the joint in young kittens.

A cast was maintained in the first cat for 8 to 14 weeks; the final outcome in that cat was not as cosmetically favorable or functional in 1 of the affected limbs as it was in the other. Failure of the owner to continue rigorous physical therapy beyond 4 weeks after cast removal may have played a role in this outcome. Casts were applied for 6 weeks in the second cat, followed by 10 weeks of physical therapy. Final outcome in that cat was a near-normal range of tarsal motion and function in both hind limbs. In the third cat, casts were applied for only 4 weeks and physical therapy was performed for 8 weeks; that cat had substantial improvement in positioning and use of the limb. Other possible treatments such as tenotomy of the common calcanean tendon and external skeletal fixation were unnecessary. Additionally, the age of the kittens would likely preclude the use of external skeletal fixation because of the absence of bone development. External skeletal fixation alone would also have been difficult in the third cat because the proper weight-bearing angle could not be achieved at the first visit. Surgical correction would likely be reserved for cats that did not respond to conservative management.

Physical therapy was considered essential to the positive outcome in the cats of this report. In older cats, staged stretching at the time of cast applications may be necessary, in addition to physical therapy for an extended period of time. In humans, corrections can be made with casting and physical therapy in children as old as 2 to 3 years of age. Study of a larger number of cats will be necessary to ascertain differences in treatment protocols, outcomes, possible causes, incidence, and prognosis.

References
Effects of deracoxib and aspirin on serum concentrations of thyroxine, 3,5,3'-triiodothyronine, free thyroxine, and thyroid-stimulating hormone in healthy dogs

David L. Panciera et al

Objective—To evaluate the effects of deracoxib and aspirin on serum concentrations of thyroxine (T₄), 3,5,3'-triiodothyronine (T₃), free thyroxine (fT₄), and thyroid-stimulating hormone (TSH) in healthy dogs.

Animals—24 dogs.

Procedure—Dogs were allocated to 1 of 2 groups of 8 dogs each. Dogs received the vehicle used for deracoxib tablets (PO, q 8 h; placebo), aspirin (23 to 25 mg/kg, PO, q 8 h), or deracoxib (1.25 to 1.8 mg/kg, PO, q 24 h) and placebo (PO, q 8 h) for 28 days. Measurement of serum concentrations of T₄, T₃, fT₄, and TSH were performed 7 days before treatment (day –7), on days 14 and 28 of treatment, and 14 days after treatment was discontinued. Plasma total protein, albumin, and globulin concentrations were measured on days –7 and 28.

Results—Mean serum T₄, fT₄, and T₃ concentrations decreased significantly from baseline on days 14 and 28 of treatment in dogs receiving aspirin, compared with those receiving placebo. Mean plasma total protein, albumin, and globulin concentrations on day 28 decreased significantly in dogs receiving aspirin, compared with those receiving placebo. Fourteen days after administration of aspirin was stopped, differences in hormone concentrations were no longer significant. Differences in serum TSH or the free fraction of T₄ were not detected at any time. No significant difference in any of the analytes was detected at any time in dogs treated with deracoxib.

Conclusions and Clinical Relevance—Aspirin had substantial suppressive effects on thyroid hormone concentrations in dogs. Treatment with high dosages of aspirin, but not deracoxib, should be discontinued prior to evaluation of thyroid function. (Am J Vet Res 2006;67:599–603)