Fascia lata autograft for treatment of congenital cranial cruciate ligament deficiency in a llama

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- Cranial cruciate ligament instability should be considered in neonatal llamas with hind limb lameness.
- Placement of a fascia lata autograft appears to be a useful technique in neonatal llamas with cranial cruciate ligament deficiency.

An adult female llama gave birth to a live, 11.4-kg male cria while under observation at the veterinary teaching hospital at Kansas State University. Parturition was unassisted and uncomplicated. The cria stood and nursed within 2 hours after birth, but ambulated abnormally on its right hind limb. The stifle was abducted, and the cranial phase of stride was shortened. The cria would assume an abnormal crouch position when laying down, preferring to lay on its left side. A cranial drawer sign (approx 3.0 cm of movement) could be elicited during palpation of the limb, and marked laxity of the stifle was evident. The patella was in its normal position, and range of motion of the patella was normal. In comparison, results of palpation of the left stifle were normal, and the left stifle had a normal range of motion.

Radiographic images of both stifles were obtained. The proximal end of the right tibia was displaced cranial and proximal to the distal end of the femur (Fig 1). On a survey radiograph, the lateral aspect of the femorotibial joint appeared wider than normal. On a radiograph obtained while abducting and outwardly rotating the tarsus, medial displacement of the proximal end of the tibia was evident. The right tibia had an irregular proximal physis, narrowed tibial tuberosity, and shortened, blunt intercondylar eminences. On the basis of results of physical and radiographic examination, a diagnosis of cranial cruciate ligament instability and lateral collateral ligament laxity with congenital malformation of the tibia was made.

A transarticular splint was applied. The splint incorporated a 6.4-mm-diameter aluminum rod and extended from the midfemoral to the midmetatarsal region. The splint was configured similar to a Thomas' splint, except that the limb was not placed in traction. The purpose of the splint was to permit weightbearing on the limb, yet limit motion of the stifle until the cria had matured enough to be considered a good candidate for general anesthesia and surgery.

When the cria was 10 days old, atropine sulfate (0.04 mg/kg, SC) and butorphanol tartrate (0.01 mg/kg, SC) were administered, and general anesthesia was induced with isoflurane administered via a face mask. Anesthesia was maintained with isoflurane administered via an orotracheal tube, using a semiclosed circle system. The right hind limb was prepared for surgery, and a 10-cm-long curvilinear incision was made on the cranialateral aspect of the limb, starting in the midfemoral region and ending at the distal aspect of the tibial tuberosity. Arthrotomy of the femorotibial joint was performed, and a thin (approx 2 mm in diameter), loose tissue was identified running along the normal path of the cranial cruciate ligament. This tissue was resected and submitted for histologic examination. The lateral collateral ligament appeared grossly normal, but laxity could be demonstrated by manipulating the joint. A 6-cm-long, 1-cm-wide segment of the fascia lata was collected, leaving the fascial attachment to the tibial tuberosity intact. The strip of...
Deformities of the long bones account for approximately 60% of all congenital defects in camelids.¹,² Congenital deformities known to affect the stifle of camelids include patellar luxation, malformation of the femur, and femorotibial valgus.³,⁴ Although cranial cruciate ligament disruption caused by forced manual extraction of a calf has been observed, the cria in this report had an uncomplicated birthing.³ Therefore, the cranial cruciate ligament anomaly was thought to be a congenital defect. Results of histologic evaluation supported this hypothesis.

Cranial cruciate ligament rupture occurs sporadically in horses and cattle, and treatment has met with limited success.⁶,⁷ Cruciate ligament injury is common in dogs, and numerous surgical techniques for stabilization have been described.¹⁰ Extracapsular stabilization methods often involve placing a heavy suture from the lateral fabella to the tibial crest or distal part of the patellar tendon. Intracapsular stabilization methods involve placement of synthetic materials or autografts (fascia lata or patellar tendon) through the stifle to replace the damaged cranial cruciate ligament. Transposition of the fibular head also is described as a means of stabilizing the stifle joint in dogs.

The anatomy of the stifle in camelids is similar to that of dogs. Llamas have a single patellar tendon, and the femorotibial joint is not separated by a tissue septum as is the case in horses and cattle. Unlike dogs,
however, llamas do not have fabellae caudal to the femoral condyles, and the fibular head is small and fused to the tibia. The absence of fabellae precluded use of an extracapsular suture to stabilize the stifle in this llama. The small fused fibular head precluded use of the fibular head transposition technique. Therefore, intracapsular placement of a fascia lata autograft was used to stabilize the joint. Intracapsular techniques have been advocated for stabilization of the stifle in larger animals.\textsuperscript{7,8,10} Additionally, the graft is readily available and easily placed in the proper location. The procedure is quick, which meant that the cria did not have to be anesthetized for a prolonged period.

Unfortunately, because of the tibial plateau abnormality, anatomic alignment of the stifle could not be established at surgery. However, long-term joint stability was achieved, and the cria returned to near-normal ambulation. The cria's tendency to lay on its left side and not assume a normal crouched position may have been associated with stifle discomfort. However, intermittent attempts to modify this behavior by administering phenylbutazone, butorphanol tartrate, or flunixin meglumine failed. Remodeling of the proximal end of the tibia in response to altered weightbearing was remarkable. The stability, range of motion, and function of the stifle were maintained, despite development of mild osteoarthritis.

Because cranial cruciate ligament deficiency and tibial dysplasia have not, to our knowledge, been previously reported in camelids, it is not known whether these deformities are heritable. The owners have obtained 3 other crias from the dam (2 before and 1 after this cria was born), and none have had musculoskeletal defects. This llama has birthed 1 apparently normal cria. To our knowledge, cruciate ligament aplasia has not been recognized as a heritable disorder in other domestic species.

**References**