Congenital vulvar deformity in 6 alpacas

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Case Description—6 female alpacas, ranging in age from < 1 day to > 2 years, were examined because of primary owner complaints related to urogenital malformation.

Clinical Findings—in all instances, the vulva was totally or subtotally imperforate. One neonate had failure of passive transfer of immunity and mild azotemia at the time of initial examination. No additional urogenital malformations were detected in any of the alpacas.

Treatment and Outcome—Vulvoplasty performed via local anesthesia was successful in all alpacas. The neonate with failure of passive transfer received a plasma transfusion.Postsurgical wound management was limited to topical medications.

Clinical Relevance—Congenital vulvar deformity in alpacas may result in interference with urine outflow. Neonates with completely imperforate vulva may be brought to veterinarians for examination on an emergency basis. Less severely affected alpacas may be examined later in life with owner complaints ranging from stranguria or dysuria to urogenital malformation. No other primary abnormalities of the urogenital tract in alpacas have been reported, to the authors' knowledge. Vulvoplasty, performed with local anesthesia, resolves obstructed urine flow. Because it is possible that this condition is heritable, affected alpacas, and possibly their sires and dams, should not be used for breeding.

A newborn female alpaca < 12 hours of age was examined at the Graham French Neonatal Intensive Care Unit at New Bolton Center as an emergency admission because of inability to urinate and suspected urogenital abnormality (case 1). The cria had been born earlier on the day of initial examination and was considered normal at that time. When the cria was checked by the owners in the evening, a swelling was noticed in the region of the vulva and the cria was transported to the hospital for evaluation and treatment. Physical examination was unremarkable except for the presence of a large fluid-filled structure at the vulva, and the finding that there was no identifiable vulvar opening (Figure 1). The cria was bright and alert, and no hematologic or plasma biochemical abnormalities were identified. Plasma IgG concentration was considered adequate.

Results of ultrasonographic evaluation of the fluid-filled structure and urogenital tract suggested that the absence of a vulvar opening was the primary abnormality. Fluid, thought to be urine, was detected in the uterine lumen. The diagnosis was imperforate vulva or fusion of the vulvar labia with secondary mild urometra.

The cria was positioned in ventral recumbency and the vulvar skin was aseptically prepared with chlorhexidine scrub and sterile water. Local anesthesia was provided via SC injection of a small (< 2 mL) volume of 2% lidocaine solution. A temporary stoma was created in the vulva by stab incision with a No. 11 scalpel blade. After the procedure, the cria was observed to urinate. On the following day, a permanent stoma was created with the cria positioned, aseptically prepared, and anesthetized locally, similar to the first procedure. The temporary stoma was elongated on midline with a No. 11 scalpel blade, and with mosquito hemostats and digital palpation used to guide the incision, the opening was extended dorsally and ventrally. Care was taken to incise only vulvar tissue. The permanent stoma was created by apposing vestibular mucosa to vulvar skin with 4-0 synthetic absorbable suture material in an interrupted pattern to prevent adhesion formation between, and reposition of, the vulvar lips (Figure 2). Additional treatments included topical wound treatment with a triple-antimicrobial and corticosteroid ointment,7 support with IV administered fluids, and administration of cefotiofur sodium8 (10 mg/kg [4.5 mg/lb], IV, q 6 h). The cria continued to urinate normally and was discharged to the owner 3 days after admission for continued wound management at home. The cria did well clinically, and no additional genitourinary problems were reported.

A 2-day-old cria was examined at the Cummings School of Veterinary Medicine at Tufts University because of stranguria (case 2). Parturition had reportedly been without complications. At the time of initial examination, the cria was bright and alert, mildly febrile (rectal temperature, 39.2°C [102.6°F]), tachycardic (heart rate, 170 beats/min), and tachypneic (respiratory rate, 48 breaths/min). Examination revealed a distended, subtotally imperforate vulva. A small (< 1 mm) opening, through which the cria had been urinating, was observed in a ventral position on the vulvar lips (Figure 1). Except for high serum creatine kinase activity (1,718 U/L), no hematologic or plasma biochemical abnormalities were identified. Plasma IgG concentration was adequate. The vulvar skin was anesthetized with local infiltration of 2% lidocaine solution and surgically opened to restore full patency of the urinary tract. The cria recovered without complications.
A 4-week-old female cria, was examined with a history of persistent watery diarrhea that had been observed for the previous 10 days (case 3). On physical examination, the cria was in good body condition but was mildly tachypneic (respiratory rate, 40 breaths/min). Examination of the urogenital tract revealed evidence of urometra. During hospitalization, the cria passed a moderate amount of pasty diarrhea. Because of this finding, vulvoplasty was postponed so as to limit wound contamination. Ten days after the initial examination, the diarrhea had resolved and the cria was readmitted for a vulvoplasty procedure. A full stream of urine was observed after the procedure, and the cria had an uncomplicated recovery.

A 1-day-old female full sibling to the cria of case 3 was admitted for correction of an imperforate vulva (case 4). The owner had permitted a repeat breeding between the same sire and dam, despite evidence of a congenital vulvar anomaly in case 3. Similar to findings with the cria of case 3, the sibling cria did not have any physical abnormalities other than mild tachypnea (42 breaths/min) and congenital vulvar labial fusion, which led to marked vulvar distension and impaired urination. In this cria, plasma creatinine concentration was high (3.5 mg/dL), and the cria had partial failure of passive transfer (IgG concentration, 125 mg/dL; reference concentration, >1,000 mg/dL). The partial failure of passive transfer was treated with IV administration of alpaca plasma. Vulvoplasty was performed with no complications via local anesthesia, and the cria recovered fully.

Cases 5 and 6 involved alpacas from the same farm as cria 1; the 3 alpacas were reportedly unrelated. The alpacas were examined at the New Bolton Center on the same date with identical complaints of vulvar deformity, stranguria, and perineal staining. In both cases, vulvar labial fusion was apparent but there was sufficient ventral opening to allow for urination (Figure 1). One alpaca was 1 year of age, and the other was 2 years of age. Vulvoplasty was completed without complications on an outpatient basis.

Discussion
To the authors' knowledge, congenital labial fusion has not been reported in alpacas or other South American camelids. Atræsiæ vulvi was reported in 2 camels in 1997, and an incompletely developed vulva has been described in a Friesland heifer. Additionally, posterior vulvar labial fusion was reported as a familial trait in a human female in which the family history was remarkable for a similar congenital defect in 2 aunts and their daughters, suggesting an autosomal dominant trait with incomplete penetrance. Vulvar fusion with a suspected autosomal recessive mode of inheritance has also been reported in a colony of common marmosets. In these marmosets, as was reported in the affected humans and observed in the camelids of

Figure 1—Photographs of the vulvar anatomy of alpacas with imperforate vulvar labia. A—Vulva in alpaca 1 at the time of initial examination. Notice the bubblelike appearance of the imperforate vulva. B—Alpaca 2 at initial examination. Notice the urine passing through a small (<1 mm) opening in the ventral aspect of the vulva and the turgid appearance of the vulva. C—Alpaca 3 at initial examination. In this alpaca, the semi-perforate vulva was not distended with urine. D—Alpaca 5 at initial examination. This is the vulva of a more mature affected alpaca. E—Alpaca 6. Only a small opening is present in the ventral portion of the vulva.

Figure 2—Photograph of case 1 immediately after a vulvoplasty procedure was performed.
this report, only the vulva was affected; there was no evidence of defects in any other portion of the genitourinary tract, such as clitoral hypertrophy, which has been associated with certain adrenogenital syndromes and with exogenous androgen therapy of the dam, sex reversal, or freemartinism and chimerism. Recessive inheritance implies that both parents must carry the undesirable genetic trait, although neither the dam nor the sire will necessarily express the defect phenotypically. If the inheritance pattern for this trait in camelids is similar to that in marmosets, further breeding of either parent or the affected cria may perpetuate spread of the undesirable trait, although the gene may not be expressed in all offspring.

Two of the alpacas of this report had urometra, likely secondary to occlusion of urinary tract outflow associated with the vulvar deformity and resultant backflow of urine through the cervix into the uterus. Dense and nearly complete labial fusion with severe hydroureternephrosis that resolved after incision of the adherent labia has been reported in a single human infant. In another report, stranguria and urethral duplication in an intersex llama cria was described. Distension of the genitourinary tract with urine may be seen in alpacas with vulvar fusion and is likely dependent on the degree of deformity; imperforate vulvar deformities may be encountered by veterinarians as a neonatal emergency, as occurred in case 1, whereas less severely affected animals may be examined for other reasons, ranging from suspected diarrhea with stranguria to deformity in females reaching breeding age.

The evidence suggests that this congenital deformity may have a heritable component in alpacas, on the basis of appearance of the defect in 2 full siblings produced from sequential breedings in separate years. It is possible that environmental or infectious causes play a role, but this seems less likely. For all alpacas of this report, a recommendation was made to owners not to use the affected animals as breeding stock, and in case 3, a specific recommendation was made to not repeat the breeding. Because the reproductive tract appears to be normal in affected alpacas, corrective surgery should be performed to allow for appropriate urine outflow, but breeding of affected females, and potentially their dams and sires, should be discouraged.

References