A number of different congenital anomalies are known to occur in domestic animals. Several congenital anomalies on digestive and urinary system have been described associated with genetic (transgenes and chromosomal anomalies), or environmental agents (viral infections, toxins, and fertilization techniques)[1, 8, 10, 11].

In this particular anomaly, we suspect that the congenital obstruction of the urachus, which has not previously been reported in the bovine, was the initiating factor in a cascade of events, ultimately resulting in impaired bladder outflow, megacystis, and bladder rupture, with discharge of urinary wastes into the abdominal cavity. During embryogenesis, following the rupture of the cloacal membrane, the proximal part of the allantois dilates to become the urinary bladder[9]. Urine flows to the amniotic sac in early to midgestation via the ureters; however, in later gestation, contraction of the bladder sphincter prevents the further release of urine into the amniotic space and urinary wastes are discharged principally into the allantoic cavity via the urachus[9, 12].

The most common cause of hydrourephrosis is obstruction of urethra and/or urachus in the congenital and experimental environment of the bovine fetus was male at 270 days gestation, was referred to the College of Veterinary Medicine, Kyungpook National University with abdominal distension, arthrogryposis and atresia ani (Fig. 1). This fetus was the first delivery of the two year old Korean native cow by natural breeding in the farm. Any clinical signs, diseases and previous occurrence of malformations or anomalies had not been observed on the dam and other dams or calves in the farm. We couldn’t investigate pedigree because the sire of fetus was already sold out. At necropsy, this fetus had a large amount of ascites, urachal obstruction and marked bladder distention. The ventral surface of the bladder had ruptured and attached to the abdominal wall by fibrinous adhesions. There was bilateral hydrourephrosis with moderate pelvic dilatation and cortical attenuation. The rectum was filled with meconium but the anus was imperforate. The right forelimb was contracted. The cause(s) of these abnormalities could not be determined; however, we believe that developmental abnormalities during embryogenesis may be the result of chromosomal abnormalities.

**NOTE**

Pathology

**Fetal Bladder Outlet Obstruction in a Stillborn Bovine Fetus**

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**ABSTRACT.** A stillborn bovine male fetus with abdominal distention, arthrogryposis and atresia ani was presented for diagnostic evaluation. At necropsy, this fetus had a large amount of ascites, urachal obstruction and marked bladder distention. The ventral surface of the bladder had ruptured and attached to the abdominal wall by fibrinous adhesions. There was bilateral hydrourephrosis with moderate pelvic dilatation and cortical attenuation. The rectum was filled with meconium but the anus was imperforate. The right forelimb was contracted. The cause(s) of these abnormalities could not be determined; however, we believe that developmental abnormalities during embryogenesis may be the result of chromosomal abnormalities.

**KEY WORDS:** congenital urachal obstruction, hydrourephrosis, megacystis.

Ascitic fluid and serum were collected from the fetus and dam, respectively. Elevated levels of creatinine (>10.0 mg/dl) and urea (25.3 mg/dl) were detected in the ascites. Neutralizing antibodies against Akabane virus (AKV), Aino virus (AIV) and Chuzan virus (CHV) were not identified in the serum of dam. Collected tissues were fixed in 10% buffered neutral formalin, embedded in paraffin, sectioned at 4 µm and stained with hematoxylin and eosin for microscopic examination.

The microscopic architecture of the kidney and corticomedullary differentiation was unremarkable. However, there was overall attenuation of the renal parenchyma and progressive dilatation was seen from the tubules through the collecting ducts (Fig. 4). No lesions were observed in brain, brain stem and spinal cord. In skeletal muscle on site of arthrogryposis, non-suppurative inflammation was detected but not prominent.

In this fetus, a multi-systemic clustering of anomalies including atresia ani, megacystis, arthrogryposis, and hydrourephrosis were observed, suggesting a syndrome derived from chromosomal abnormality [1]. Several reports have described that malformation of one portion of the body directly leads to malformation of others [1, 7, 8], and in humans, chromosomal alterations have been identified in up to 40% of anomalous syndromes involving megacystis and concomitant maldevelopment of the gastrointestinal tract [3, 7].

In this particular anomaly, we suspect that the congenital obstruction of the urachus, which has not previously been reported in the bovine, was the initiating factor in a cascade of events, ultimately resulting in impaired bladder outflow, megacystis, and bladder rupture, with discharge of urinary wastes into the abdominal cavity. During embryogenesis, following the rupture of the cloacal membrane, the proximal part of the allantois dilates to become the urinary bladder [9]. Urine flows to the amniotic sac in early to midgestation via the ureters; however, in later gestation, contraction of the bladder sphincter prevents the further release of urine into the amniotic space and urinary wastes are discharged principally into the allantoic cavity via the urachus [9, 12].
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In the experimental sheep model, urachal ligation produced megacystis, bilateral hydronephrosis, and was associated with a high perinatal mortality [6], similar to that seen in this case. In this case, it is likely that obstruction of the urachus, coupled with a competent bladder sphincter, resulted in progressive distention and eventual rupture. The degree of hydronephrosis was likely ameliorated by the megacystis and eventual rupture of the urinary bladder.

Atresia ani has been reported with other fetal abnormalities, e.g. urogenital and skeletal defects in sheep and goats [2, 10]. However, it also has been reported with rectal palpation for pregnancy diagnosis or autosomal recessive trait [8, 10], which could not be ruled out in this case.

Common causes of arthrogryposis-viral (AKV, AIV, CHV), lupinosis, or primary brain lesions were investigated but not identified in either the dam or fetus. In the presence of a normal nervous system, increased intrauterine pressure with mechanical compression of the fetus may cause the anomaly [11], and in this case, fetal megacystis, abdominal enlargement, and resultant malpositioning in utero may have resulted in this lesion.

In this case, we suspect that multiple defects occurring during embryogenesis initiated a chain of events that culminated in the clinical presentation. The presence of a congenitally obstructed urachus resulted in improper bladder outflow, megacystis, and increased pressure within the urinary tract, culminating in hydronephrosis, bladder rupture, and uroperitoneum. The precise contribution and relationship of the atresia ani to the urogenital defects, is unknown. Taken together, however, this report describes unique congenital abnormalities in a bovine fetus affecting the urinary, gastrointestinal, and possibly the musculoskeletal system, and is the first report of congenital urachal obstruction in this species. While the precise cause of these abnormalities could not be determined, we speculate that this constellation of defects likely is the result of chromosomal abnormality in
this particular individual.

REFERENCES