Persistent Cloaca and Caudal Spinal Agenesis in Calves: Three Cases

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Abstract. Three newborn calves were affected by caudal spinal dysgenesis or agenesis (coccygeal vertebra) and persistent cloaca. The cloacas were lined by a mixture of transitional and colonic epithelium. The vertebral column of one calf was internalized into the cloaca. The association of persistent cloaca and caudal spinal anomalies is thought to be related to cell loss in the caudal cell mass, which affects caudal spinal column formation and cleavage of the cloaca by the urorectal septum. This association is well documented in humans and has been reported in Manx cats. By extrapolation, it appears likely to exist also in neonatal calves. Identification of cloacal lesions in neonatal animals should be cause for careful examination of the vertebral column because many of the associated spinal lesions are occult.

Key words: Cattle; caudal spinal dysgenesis and agenesis; congenital malformations; persistent cloaca.

This report documents three cases of persistent cloaca with agenesis of the caudal spinal column in calves.

Calf No. 1 was an incontinent newborn phenotypically male Holstein calf with no tail and lacking haired skin ventral to the anus. The owner elected euthanasia because of a heart murmur. This abnormal calf appears to have been unique within this herd.

At necropsy, the tail was absent caudal to the sacro-coccygeal junction. The anus ended approximately 4 cm inside the body. There was no penis within the prepuce, and only the right testis was in the scrotum. No urethra was present. A persistent cloaca, a 30- x 20- x 15-cm pouch in the pelvic inlet, received the termination of the descending colon and the right ureter. There was no left ureter, and the left kidney was hypoplastic (1 cm diameter). The right kidney had modest dilation of the renal pelvis. There was no left testicle. Within the cloaca, there was a haired surface covering malformed vertebral bodies. These vertebrae were directly continuous with the caudal sacral vertebrae.

Microscopically, the mucosa of the cloaca was a mixture of transitional and glandular columnar epithelia interspersed with foci of dysplastic epithelium and adenomatous foci. Mixed inflammatory cells were in the subjacent connective tissue. Between the haired skin and the disorganized bone of the internalized tail, there was a dense sheet of primitive mesenchyme.

Calf No. 2 was a newborn phenotypically female Holstein calf delivered manually following dystocia. The calf lacked a tail and had an imperforate anus that ended 2 cm from the surface. Feces were present in the vagina, and there was microphthalmia of the left eye. The owner elected euthanasia because of a heart murmur. As with Calf No. 1, this calf appeared to be unique in this herd, with no epidemiologic significance.

At necropsy, the tail was absent caudal to the junction of the sacral and coccygeal vertebrae. The descending colon and both ureters emptied into a cloaca (20 x 10 x 10 cm), which exited the pelvic inlet via a 4-cm-diameter vagina. No uterus or gonads were present. The left kidney was hypoplastic (4 x 2 cm; 50% smaller than the right kidney). There was mild bilateral dilation of the renal pelvis. The microscopy of this lesion was comparable microscopically to that described for Calf No. 1, except that dysplasia and adenomatous foci were not present.

Calf No. 3 was a Limousin heifer calf born without a tail, anus, or vulva. A cloaca was diagnosed ultrasonographically and confirmed during surgical correction. Within 1 week after surgery, colonic impaction with secondary megacolon and inability to empty the urinary bladder were diagnosed, and the animal was euthanatized. As with the other abnormal calves, this calf appeared to be unique in the herd, without epidemiologic significance.

The vertebral column and spinal cord of this animal ended abruptly at the sacro-coccygeal junction. The urethra, cervix, and distended impacted colon all emptied into the cloaca. Microscopically, the cloaca was extensively ulcerated without an apparent epithelium. Mixed inflammatory cells were in the subjacent connective tissue.

Persistent cloaca, often occurring with a body wall defect and cloacal exstrophy ("eversion"), is rare. In humans, it is frequently associated with caudal spinal dysraphism. Careful examination reveals some caudal spinal anomaly, usually occult lipomeningocele, in all individuals with persistent cloaca. An analogous situation exists in Manx cats, wherein selective breeding for anury/brachyury (lack or shortening of the tail) has resulted in an increased incidence of meningomyelocele, caudal spina bifida, and myelodysplasia. Affected Manx cats occasionally have a concurrent persistent cloaca.

Several hypothetical explanations for the association between persistent cloaca and caudal spinal agenesis have been previously proposed. The most common theme among these is a variant of the wedge mechanism, by which persistent cloaca is thought to arise. In the uncomplicated wedge mechanism, the ectodermal and endodermal layers of the cloacal membrane are hypothesized to be overdeveloped. This overdevelopment is speculated to prohibit migration of the wedges of mesoderm that split the cloacal membrane into a trilaminar structure. As the embryo overgrows its lateral and caudal aspects, creating lateral and caudal embryonic folds, tension is created on the persistent bilaminar cloacal membrane. Without the mesodermal ingrowth, the mem-
brane ruptures prematurely under the tension of folding. The premature rupture of the cloacal membrane prohibits the caudal migration of the urorectal septum. Although the mechanisms by which urorectal septal migration might be inhibited are unknown, loss of properly timed cellular migration signals from the trilaminar cloacal membrane has been presumed. Without urorectal septal migration, cleavage of the cloaca into a ventral urogenital sinus and a dorsal rectum does not occur, resulting in persistent cloaca.

The linkage of persistent cloaca induced by the wedge mechanism to caudal spinal anomalies could be as follows. If substantial cell loss occurred in the caudal cell mass immediately prior to the migration of mesoderm into the cloacal membrane, mesodermal wedges might never form and the same sequence of events regarding cloacal persistence would occur. The caudal spinal cord and column form via secondary neurulation within the caudal cell mass prior to the cleavage of the cloaca by the urorectal septum. Thus, cell loss in the caudal cell mass could explain abnormal development of the caudal spinal column and abnormal cleavage of the cloaca.

In severe cases of cloacal extrophy through the ventral abdominal wall in humans, it has been hypothesized that as the cloaca sinks ventrally through the abdominal wall defect, the overlying notocord is mechanically pulled ventrally, decreasing differentiation cues from the notocord to the dorsal cells that will form the caudal vertebral column and spinal cord. If this hypothesis is correct, marked cell loss in the caudal cell mass could result in ventral deviation of the notocord and overlying structures with possible spinal column formation in an internal direction, as observed in calf No. 1.

Cell loss in the caudal cell mass is thus a logical, albeit unproven, proximate explanation for the association of caudal spinal anomalies and persistent cloaca. The question of the etiology of the cell loss is thus raised. There are no data to guide speculation on the specific cause of cell loss, if it indeed occurred, in these calves. It seems logical that a variety of viral, chemical, and other documented causes of bovine malformations could be involved in any given case. Additional diagnostic procedures that might have aided in determining the role of specific etiologic agents were not performed in these cases for economic reasons.

The renal hypoplasia seen in the two Holstein calves has not been reported in humans in association with persistent cloaca or caudal spinal dysraphism. Kidney formation in the urogenital ridges is induced by migration of the metanephric diverticulum from the urogenital sinus. This migration may be disrupted in cases of persistent cloaca. The heart murmurs that lead to the euthanasia of the three calves were presumably physiologic; no evidence of cardiac dysfunction was found in any of these calves.

In summary, caudal spinal column anomalies and persistent cloaca appear, by extrapolation, to be likely to occur in all species. Because many of the spinal lesions in humans are occult lipomeningoceles, veterinary pathologists should be aware of this association and carefully examine the spinal column in cases where cloacal division is anomalous.

References


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