Clinical Observations in Rectovaginal Constriction in Jersey Cattle

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Most genetic defects in cattle are easily diagnosed in neonates; however, some are not detected until later when the animal is examined or clinical problems such as infertility or dystocia occur. Clinical experience with such a defect in Jersey cattle—constriction of the anus and vulvo-vestibular area, rectovaginal constriction (RVC)—is reported here.

Materials and Methods

Reports of Jersey heifers and cows with dystocia or breeding problems as received by the American Jersey Cattle Club or veterinarians and cattle owners, were part of a long-term study of the nature, cause effect and frequency of genetic defects in cattle. Included were clinical descriptions from practicing veterinarians, herd owners, and veterinarians at artificial insemination centers. Reproductive tracts with attached rectums were sent to Kansas State University (KSU) for examination. The majority of cows or heifers were first examined on the farm by veterinarians or herd owners. Later, affected animals were transported to KSU for further study where they were kept in dry lots and on pasture in a group. Blood samples from affected cows were taken by veterinarians at the farms of origin or at KSU and shipped to the Department of Animal Science, Texas A & M University for parentage verification. Pedigrees of affected heifers and cows were provided by owners or the American Jersey Cattle Club.

The reproductive tract and ano-rectal area of affected cattle were examined clinically at KSU. Normal Jersey females of breeding age were distinguished from those affected with RVC by the cervix, body of horns of uterus, and ovaries being palpable per rectum, having a vulval circumference greater than 20 cm and the external os of the cervix palpable per vaginam.

All cows affected with RVC arriving at KSU had previously either undergone episiotomy or caesarian section for relief of dystocia. They were rebred by natural service or artificial insemination. Parturition of experimental animals was closely followed and vaginal examination with a plastic or glass speculum was used to predict pending parturition. The udder was also examined at this time. Fetal head or legs were allowed to enter the vaginal cavity, the amniotic sac allowed to rupture, and abdominal contraction observed before obstetrical procedural decisions were made. These were based on manual examination and palpation of the vulvo-vestibular and vagina diameter of the birth canal and occurrence of full labor. Episiotomy or Caesarian section via left flank was performed if the clinician considered normal vaginal delivery was not possible. Postparturient recovery was monitored and abnormal clinical signs noted. The udder was examined prepartum, during partum and postpartum.

Two normal control Jersey heifers of different lineage were obtained, one each from Kansas and Texas. These heifers were kept with the RVC cattle, bred, and managed in the same manner.

Reproductive tracts with attached rectums shipped to KSU were examined grossly and descriptions recorded. In addition, veterinarians at artificial insemination centers found several bulls affected with anal stenosis which had sired calves affected with RVC or had sired calves with cows which were taken by Caesarian section at KSU. A bull affected with anal stenosis was loaned to KSU for further study.
Results

History—Combined anal and vulvo-vestibular stenosis (RVC) was reported in 46 females in 33 Jersey herds from Colorado, Florida, Idaho, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, New Hampshire, Nevada, North Carolina, Ohio, Oklahoma, Oregon, South Carolina, Texas, Virginia, Washington, and Wisconsin. The common complaint was a tight anus that prevented or made difficult artificial delivery or pregnancy examination. Episiotomy or Caesarian section to relieve dystocia had been performed on all affected cows. Attending veterinarians reported 9 episiotomies and 28 Caesarian sections had been performed on 37 Jersey cows affected with RVC. Nine heifers reportedly affected with RVC were transported to KSU for further study and are included in this report.

Clinical Signs—Rectovaginal constriction was characterized by stenosis of the anal opening. The anal sphincter diameter was limited and prevented complete rectal examination. Extra digital force applied to the anal sphincter allowed rectal entrance to the middle of the forearm. In some cattle only 3 or 4 fingers up to the knuckles could be inserted through the anal sphincter. Circumference measurement of maximally expanded anal sphincters in RVC cattle ranged from 14.5 to 31.0 cm. The major finding was non-elasticity of the anal opening.

Four Jersey bulls were reported by AI veterinarians as being affected with anal stenosis. The Jersey bulls used in the KSU breeding herd had anal stenosis: rectal examination up to the lower midarm only was possible.

Vaginal examination was carried out on all cows or heifers submitted alive to KSU. Speculum examination revealed an occasional dorso-ventral septum in the caudal part of the vagina just cranial to the vestibular ring. Remnants of a hymen were common. Length of the vulvar cleft was usually about 5 cm. The vulvo-vestibular area was narrow and funnel-like in RVC affected cattle. The major finding was non-elasticity of the vulvo-vestibular area. Usually 3 to 4 fingers could be inserted into the vulvo-vestibular area against considerable resistance from a fibrous, nonelastic, broad vestibular ring. Diameter of the vulvo-vestibular area was less in cows with a fibrous tissue ring circling the area immediately cranial or caudal to the urethral orifice.

The most serious clinical problem encountered in RVC Jersey cattle was dystocia. Preparation for parturition was normal, pelvic ligaments relaxed, the cervix dilated, and fetuses were usually presented in anterior presentation with head and feet located within the vaginal cavity, but the inelastic funnel-like vulvo-vestibular area did not relax. Manual vaginal examination in some cows permitted three to four fingers only to be introduced into the vestibular area; in other, hand and arm could be inserted. A clenched fist (25 cm), however, could not be withdrawn from the vagina through the vestibular ring and vulvo-vestibular area. Caesarian section via left flank was performed to relieve dystocia due to nonrelaxation of the vulvo-vestibular area in all except two cows. Episiotomy was performed on two cows. One calf suffered prolonged dystocia caused by the calf being retained by the inelastic vestibular ring; the calf was delivered dead following forced extraction. Necropsy of the neonatal calf revealed six fractured ribs and other injuries. In the second case, episiotomy was done also because the calf was retained by the vestibular ring. Both control heifers kept under the same conditions delivered live calves unassisted.

In addition to dystocia, 27 RVC cattle had severe udder edema that usually began two to three weeks prior to calving and became so severe and persistent that teats cracked and even desquamated. Mastitis and ulceration of the lateral walls of the udder in contact with the thighs were common.

Four bulls were reported by veterinarians at AI centers as having anal stenosis. The bull loaned to KSU had anal stenosis.

Discussion

Congenital defects, defined as abnormalities of structure or function present at birth, may affect a single structure or function. Genetic variation is the means of evolution and the tool of animal breeders. Not all variations are favorable; many are unfavorable and result in congenital defects and may be highly important economically. Although economic losses in the entire dairy industry due to defects are less than losses due to diseases stemming from nutritional or infectious causes, congenital defects may cause heavy economic losses.

Few defects have clear-cut genetic causes; some are caused by environmental factors (teratogens) and others may result from complex genetic-environmental interactions. Teratogenic agents involved in congenital defects tend to follow seasonal patterns and known stressful conditions; they may be linked to maternal disease rather than following a familiar pattern. Hereditary defects are pathological or pathophysiological results of mutant genes. Many congenital defects follow simple Mendelian inheritance, most stem from homozygosity of a simple autosomal recessive gene or other monofactorial inheritance patterns. Congenital defects also may be inherited polygenically either with or without a threshold.

Identifying genetic causes is difficult, but several procedures are available. The congenitally defective offspring is the diagnostic entity, and all genetic procedures are based on the assumption that genetic diseases run in families or that more closely related animals are most likely to receive the same copy of mutant genes. Hereditary congenital defects such as simple autosomal recessives occur in typical intergenerational patterns and intragenerational frequencies. Identifying the cause may be difficult, but failing to do so results in diagnostic and control problems. If the defect is genetic, breeding programs need to be changed; if it is environmentally induced, herd management needs to be changed. The primary purpose of etiological and physical

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The examination of cattle for rectovaginal constriction (RVC) is a critical aspect of modern veterinary practice, particularly in breeds like Jersey cattle, where the incidences of this condition have been documented. RVC affects both male and female cattle, and its diagnosis is essential for maintaining herd health and productivity.

RVC is characterized by a narrowing of the rectum and vagina, often resulting in dystocia, or difficulties during parturition. The condition is typically diagnosed through a combination of physical examination and genetic testing, with a focus on identifying genetic predispositions.

The epidemiology of RVC indicates that it is more prevalent in certain breeds and regions. Jersey cattle, in particular, have been shown to have a higher incidence of RVC compared to other breeds. This can be attributed to both genetic and environmental factors, highlighting the importance of both selective breeding and improved husbandry practices.

Genetic testing is particularly effective in identifying carriers and affected animals, allowing breeders to make informed decisions about breeding practices. This is crucial for minimizing the incidence of RVC in affected herds and improving overall cattle health.

In summary, rectovaginal constriction is a significant issue in the bovine industry, requiring a multidisciplinary approach that includes genetic screening, clinical examination, and herd management strategies. Continued research and collaboration within the veterinary community are essential for furthering our understanding of this condition and developing effective management practices.

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RVC, a congenital defect of female and male Jersey cattle, is characterized by stenosis of the vulvo-vestibular portion of the female reproductive tract and stenosis of the anal sphincter in both sexes. Parturition is abnormal due to inadequate relaxation of the vestibular ring and vestibulo-vulvar area. Dystocia in primigravida heifers can be relieved only by episiotomy or Caesarian section. Furthermore, udder edema in many RVC heifers or cows is so severe and persistent that it results in mastitis and loss of quarters. Rectovaginal constriction is a genetic defect most likely stemming from homozygosity of a simple autosomal recessive gene.

References

Contribution 81-5b-j
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Intravenous High Potassium Therapy for Diarrheic Calves

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Summary
During the course of diarrhea in neonatal calves, a number of intracellular and extracellular fluid and electrolyte changes occur. Effective therapy should be directed towards restoration of whole body balance, not treatment of changes only in the vascular compartment. Although hyperkalemia is present during acute diarrhea, there is loss of potassium from cells and a whole-body potassium deficit. Intracellular-extracellular potassium imbalance causes detrimental changes in the membrane's potential in many tissues. Restoration of a more normal membrane potential requires replacing the intracellular potassium deficient and correcting the extracellular hyperkalemia. This is accomplished by initiating the movement of potassium in the cells and addition of potassium to the extracellular pool via the replacement therapy.

In dehydrate-diarrheic-acidotic calves with elevated plasma potassium concentrations 8.5 ± 0.7 mEq/l, the administration of I liter of fluid containing 23 mEq/l of potassium in 60 minutes resulted in a decrease in plasma potassium concentration to 6.7 ± 0.2 mEq/l, while intracellular potassium concentration increased from a diarrheic concentration of 147 ± 4 to 153 ± 4 following therapy. This occurs because of the inclusion of both glucose and bicarbonate in the therapy: glucose augments potassium flux into the intracellular pool and bicarbonate causes a transmembrane potassium-hydrogen ion exchange. Potassium continues to decrease towards normal as long as the extracellular glucose concentration is elevated. Bicarbonate increased both intracellular and extracellular pH during and following therapy. The immediate change was 0.1 pH units in both pools.

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