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 anorectal 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.

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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 breeding 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 vulo-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 cows 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

diagnosis is prevention, particularly defects impairing structure of function and hence, usefulness of affected animals.

Undesirable inherited traits should be listed on the pedigree or in any advertisement of breeding animals. Epidemiology of genetic defects should consider three possible causes: unknown, suspected, and known. Many genetic problems in cattle have not been clearly identified and defects undoubtedly exist that await description and clarification. A single undesirable recessive or lethal trait can rapidly become a real problem in a breed or group of cattle. Maintaining a recording system is the most efficient and lease expensive method for breed organizations, AI centers and other institutions to monitor undesirable traits. If evidence is convincing for inheritance of the trait, one documented case should be reason enough to label or discard the bull that sired the defective animal. In cases of suspected or unknown etiology, after three similar cases have been collected, we recommend testing and temporary or permanent removal of the bull from service.

A number of congenital defects involving the rectum and anus were reviewed recently.[1] The female reproductive system may be affected with defects of the ovaries, Mullerian ducts derivatives (uterus, cervix and vagina) and vulvo-vestibular area. All of these defects have been reported to stem from genetic causes.[1,3]

The association of anal stenosis and vulvo-vestibular stenosis in Jersey cattle has been reported previously.[2,4] In Kentucky, a Jersey bull sired 22 females affected with constricted tubular reproductive tracts and anal constriction. Calving was difficult.[4] Another report described three herds in which 14 Jersey cows had constriction of the anorectal area and vagina. Maternal and paternal lines of all affected cows were traced to a common ancestor.[2] The pattern of occurrence of this defect appeared to be compatible with simple autosomal recessive inheritance. Cases described in this paper and those described earlier are related.[2] Studies on the inheritance patterns of RVC are in progress.

Dystocia is a major factor affecting perinatal calf viability and good maternal health at parturition is fundamental for subsequent milk production. Therefore, knowledge of the causes of dystocia and its effects are of great importance. Dystocia increases with breed size; in large Friesians about 40% of heifers and 25% of all calvings require assistance.[5] Large-sire breeds produce less dystocia when mated to small breeds, like Jerseys, than when mated to larger breeds. The effects are largely mechanical as the fetus grows proportionally larger relative to pelvic size in large breeds than it does in small breeds.[5,6] The younger the heifer, the higher the dystocia rate. The incidence of feto-pelvic incompatibility is higher in Friesians and Friesian crosses than in other dairy breeds; Jerseys are the lease affected.[6] Any variation in the course of parturition is due to either the dam or the calf;[7] the causes are nongenetic or genetic.

Rectovaginal constriction, a unique congenital defect of female and male Jersey cattle, is characterized by stenosis of the vulvo-vestibular portion of the female genital tract and stenosis of the anal spincter in both sexes. The major lesion appears to be the lack of elasticity of the functional unit of the anorectal and vulvo-vestibular area; this can be explained embryologically. Both the hindgut and genital system enter the cloaca, where the urorectal septum developes and divides the cloaca anteriorly into the primitive urogenital sinus and posteriorly into the anorectal canal. The mutant genes act on parts of the two systems with a common genesis. Embryologically, the defect is ectodermal and mesodermal in origin, the stenosis being ectodermal and the accompanying muscular changes being mesodermal.

Clinically RVC is characterized with difficulty in performing normal vaginal and rectal examination and artificial ensemination, and primary manifestations are dystocia and udder edema. Dystocia in primigravida heifers can be relieved only by severe episiotomy or Caesarian section, usually the latter. Frequently, the udder edema is so severe and persistent that teats crack and may even desquamate, and mastitis and ulceration on the lateral walls of the udder in contact with the thighs are common. RVC is not associated with malformation of the Mullerian ducts (entoderm); that is, the vagina, cervix, and uterus are normal in RVC affected females.

Based on the results of this study, criteria for distinguishing normal Jersey females from RVC affected cattle are: 1) *vagina*—vulva and vestibule circumference greater than 20 cm and the ability of the examiner to reach and palpate the external os of the cervix; 2) *rectum*—anal sphincter circumference greater than 31 cm and the ease with which the examiner's arm can penetrate cranially for a distance of at lease 40 cm to palpate the body and horns of the uterus and ovaries of a nonpregnant animal. We consider these criteria applicable to females at least 15 months old and not older than 3 years, and preferably primigravida. There must be no evidence of episiotomy having been performed.

Differential diagnosis of RVC includes: segmental aplasia of the Mullerian ducts, white heifer disease, freemartinism, immaturity, juvenile genital tract, vulvar stenosis, and scar constriction following parturient trauma. RVC is easily separated from these conditions that are not associated with anal stenosis, and by the fact that bulls are also involved with RVC.

Summary

Rectovaginal constriction (RVC) was diagnosed by veterinary practitioners or cattle breeders in 46 Jersey cows or heifers from 33 farms in Colorado, Florida, Iowa, Idaho, Kansas, Michigan, Minnesota, Missouri, Nebraska, New Hampshire, Nevada, North Carolina, Ohio, Oklahoma, Oregon, South Carolina, Texas, Virginia, Washington and Wisconsin. Thirty-seven of the RVC affected cattle were transported to Kansas State University for futher studies.

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 vestibulor-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 tnd 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.

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Contribution 81-5b-j

This research was supported by the Regional Dairy Cattle Project NC-2. The assistance of the American Jersey Cattle Club is gratefully acknowledged. We particularly thank all the herd owners who supported this study.

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 1 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.

Paper presented at the XI International Congress on Diseases of Cattle, Tel Aviv, Israel, Oct. 20-23, 1980