Segmental Aplasia of a Uterine Horn (Uterus Unicornis) in a Senepol Beef Cow

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History

A six year old Senepol cow was presented to the Department of Rural Practice, College of Veterinary Medicine, University of Tennessee in March, 1988 because of reproductive failure. According to the history, she had her first calf in July 1984 and a second one in July 1986. Both were born healthy but died later of accidental influences. The cow was flushed as an embryo transfer donor, without success, approximately two months after delivering her second calf. As no estrous was observed after the flushing attempt, the cow was turned out with a bull for pasture breeding. The bull was never seen mounting the cow.

Clinical Findings

The general health of the cow was good. Her body conformation appeared masculine, possibly because she had been open for 20 months.

Rectal palpation revealed a normal left uterine horn. Both ovaries were functioning. The right uterine horn was ligamentous and contained a 4 x 6 cm egg-shaped, very firm mass. This mass was about 4 cm medial to the right ovary.

Pathological-anatomical Findings

The findings from rectal palpation were confirmed on post mortem examination. The egg-shaped, firm structure was a remnant of the tip of the right uterine horn and consisted of a tissue capsule filled with a dry, brown-yellow substance, probably inspissated uterine secretions. This residual uterine horn was connected by the oviduct to the right ovary. There was no connection to the body or left horn of the uterus except for the ligament described above.

Discussion

Segmental aplasia of the uterine horns is the absence of a distinct part of one whole uterine horn.[5] If the aplasia of the horn is partial, between the uterine body and the tip of the horn, then the uterine secretion of the sequestered part cannot drain and therefore fluid accumulates. Depending on the size of the remnant portion of the aplastic horn, the accumulated secretion may feel like an early pregnancy on palpation[1] or like a paraovarian cyst.[5] In this case, the only existing part of the uterine horn was small and the accumulated fluid had inspissated. Therefore, it felt very firm by rectal palpation.

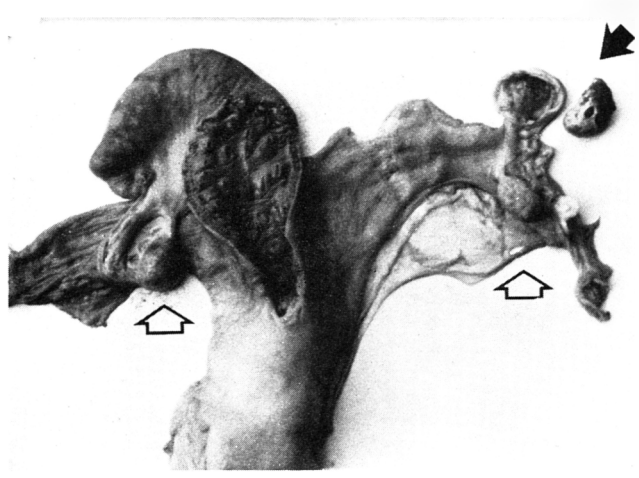

Fig. 1 Uterus unicornis in a six year old Senepol cow. The **black arrow** points to the remnant of the tip of the right uterine horn with its inspissated contents. The **open arrows** indicate the left and right ovary.

The frequency of segmental uterine aplasia (uterus unicornis) is rare, as cited references indicate. Roine (1977)[6] did a study in 2,010 dairy cows, using slaughterhouse material. He found that 0.2% of the cows had a uterus unicornis. Another study done in 6.054 zebu cattle showed 0.15% of the animals (6 heifers and 2 cows) with uteri unicornis.[2]

The segmental uterine aplasia is caused by an autosomal recessive gene.[3,5] The female offsprings with uterine aplasia

have to be homozygous. Therefore, the dam and the sire have to be at least heterozygous carriers of this particular gene. An interesting detail of this case is that the Senepol is a relatively young breed and has a population of only about 15,000 registered cattle. The development of the breed was initiated in St. Croix, U.S. Virgin Islands at the beginning of this century by crossing a Red Poll bull imported from Trinidad with N'Dama cows from Senegal, West Africa.[4] The fact that this particular gene for uterine aplasia exists in this small population of cattle seems to increase the risk that this uterine abnormality could occur much more frequently than in a larger breed. It seems logical that the chance for inbreeding in a small group of animals is greater than in a larger group. But as a recent study shows, the inbreeding coefficient in this small population is not higher than in other livestock breeds.[4] The owner, being aware of the hereditary nature of this abnormality, decided to slaughter this cow.

References

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An investigation of mastitis due to *S agalactiae, S uberis* and *M smegmatis* in a dairy herd

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Subclinical mastitis caused by streptococcal infections affected 27 of 83 cows in a commercial dairy herd. Between three and six weeks after intramammary treatment of these cows with cloxacillin, 16 (59 per cent) of the treated cows developed acute clinical mastitis associated with *Mycobacterium smegmatis*. None of the untreated cows was affected. Infected quarters were moderately hypertrophied and fine clots were present in the milk for three to four weeks. No cows showed systemic signs of illness. Studies carried out over 12 months showed that infected cows shed *M smegmatis* for three to four months and affected quarters remained hypertrophied in all but one cow after 12 months. The mean milk cell count of affected quarters fell slowly from 4,850,000/ml in the acute stage to 810,000/ml five months later and 620,000/ml 12 months later, suggesting that the organism persisted in the udder. The estimated mean loss in lactation yield for cows with *M smegmatis* mastitis was 10·8 per cent. Losses were greatest when the hind quarters were involved (mean 28 per cent for cows with both hind quarters affected). Ten of the 16 affected cows were ultimately culled owing to serious reductions in yield.

Post mortem studies on infertile buffalo bulls: Testicular histology

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Testicular tissues of 22 buffalo bulls (*Bubalus bubalis*) which suffered from three types of infertility were examined histologically. Nine bulls with no sexual libido showed underdeveloped seminiferous tubules; five of them also had various forms of germinal tissue hypoplasia (bilaterally complete, partial or incomplete) and in the other four the seminiferous tubules showed developed layers of germinal epithelium but no complete spermiogenesis. Among 11 bulls which had always produced poor quality semen one suffered from incomplete bilateral testicular hypoplasia and two had simple testicular degeneration; five showed marked testicular degeneration associated with fibrosis of intertubular spaces, and in three there was intertubular fibrosis and tubular stasis. In two bulls in which the quality of semen had deteriorated one showed bilateral partial testicular hypoplasia and the other had bilateral testicular degeneration with unilateral intertubular fibrosis and tubular stasis.