COMPARATIVE PATHOLOGY OF THE GENITALIA OF THE BULL, RAM AND BUCK

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Introduction

There are important differences in the occurrence and severity of disorders of the male genitalia of domestic ruminants, and the reasons for this are often unclear. Careful clinical observation with follow-up pathology, and structured research may help explain some of these differences, and perhaps suggest avenues for treatment and control.

Intersexuality

Hermaphroditism associated with polledness seems only to occur in the goat, in which it is inherited as a recessive character. The condition essentially represents an XX sex reversal, and affected individuals vary from being near normal but sterile males to phenotypic females with an enlarged clitoris and abdominal testes. Causally, an autosomal gene that has an Y influence is on, or closely linked to the chromosome that causes polling. Thus although the polled gene is dominant, and heterozygotes are hornless and with differentiated genitalia, homozygous XX goats are hermaphrodites. [1,2]

External genitalia

In regard to the external genitalia, a severe ulcerative balanitis in rams (but more often in wethers) parallels a less severe balanitis that occurs in bulls[3,4] but not steers. Whereas in bulls the more severe lesions involve ulceration of much of the preputial orifice with bleeding, oedema and perhaps abscessation and myiasis, in wethers lesions may progress to necrosis and sloughing of the anterior prepuce (so called "sheath rot") with concurrent necrotising balanitis. In both species the cause is multi-factorial, common factors being a high plane of nutrition and the presence of *Corynebacterium renale*. It was suggested that the occurrence of posthitis in bulls and not steers might have been due to genital injury from homosexual activity in bulls – thereby facilitating infection. Unlike a breed-specific severe balanitis described in Border Leicester rams in Australia[5], breed susceptibility to posthitis does not appear to be a factor in bulls. Clearly, an understanding of pathogenesis requires examination of hormonal influences, perhaps including the role of phyto-oestrogens or myco-oestrogens.

Herpesvirus balanitis, which in the bull, as in most species causes discrete ulcers, may lead in the buck to extensive necrosis of both penis and prepuce.[6] Lesions in bucks vary from discrete 2-4mm diameter ulcers on preputial epithelium to severe necrosis with suppuration and secondary bacterial infection of prepuce and penis, and phimosis. Intranuclear eosinophilic inclusions are easily found at the edges of ulcers. Stress associated with herding and transport of bucks, is considered the explanation for the severity of lesions. Herpetic balanitis appears not to be described in the ram.

Persistence of the penile frenulum is well recognised in the bull, occurs less often in the buck, and seems to be unrecognised in the ram. The frenulum persists when glabrous epithelia of the free part of the penis and the prepuce do not completely separate. Such separation is normally complete by 10-12 months in *Bos taurus* bull*s* and by 15 months in *Bos indicus* bulls.[7] Although in the boar there appears to be some breed predilection for persistent frenulum, there is no clear evidence in any species that the defect is inherited so surgical correction is appropriate. Primary neoplasia of the penis is not seen in the ram or buck but fibropapilloma is a not uncommon tumour in young bulls.

Testis and epididymis

Diseases of the testis and epididymis likewise differ between species. It is of interest that in bucks, but apparently not in the bull or ram, testicular hypoplasia may be segmental - perhaps involving grouped seminiferous tubules in an otherwise normal testis. Segmental testicular hypoplasia in goats, seems unrelated to polledness, and is characterised grossly by pale radiating streaks or a broader pale zone, in a testis that is smaller or of comparable size to the opposite, normal, testis. Microscopically, there is an abrupt transition from normal tubules to small, "Sertoli cell only" tubules which are extensively vacuolated. [6,8] An inherited basis for testicular hypoplasia as demonstrated in one breed of cattle, and suspected in others – including those of *Bos indicus* breed, does not seem to occur in the buck, but may occur in rams in association with monorchidism and cryptorchidism in affected flocks.

Whereas *Brucella abortus* and *Br melitensis* in the bull and buck, respectively, target the testis, *Br ovis* in the ram causes epididymitis - any orchitis being secondary, and mostly the result of intraductal spread. Studies on the pathogenesis of genital brucellosis in both bulls and rams indicate a haematogenous spread of the causal organism to the testis or epididymis, respectively, but it is unclear what factors then operate to facilitate proliferation and persistence of brucellae in these organs. Possibly the greater virulence of *Br abortus* (and *Br melitensis*) is the explanation; whereas in the bull *Br abortus* in the lumen of seminiferous tubules directly causes necrosis, *Br ovis* in the epididymis of the ram is regarded as a low-grade, "humble" pathogen. Lesions, overwhelmingly in the tail of the epididymis, develop slowly. There is hyperplasia and hydropic degeneration of ductal epithelium, which together with concurrent fibrosis, leads ultimately to extravasation of spermatozoa then formation of spermatic granuloma – essentially an autoimmune lesion which becomes increasingly conspicuous because of the large volume of mature spermatozoa present.
As with brucellosis, there are also important species differences in regard to susceptibility to *Actinobacillus seminis* and related strains of the so-called gram-negative pleomorphic organisms. Although there are reports of *A seminis* epididymitis in the bull and buck, these reports were of isolated cases rather than flock infection as occurs in rams – in which infection ascends from the prepuce. Epidemiological factors, rather than inherent species susceptibility, appear to explain these differences.[9]

Accessory sex glands

Perhaps the major contrast in genital disease of male domestic ruminants involves the accessory sex glands (ASG). A reversible enlargement of the bulbourethral glands, and to a lesser extent other ASG occurs in wethers, but not rams grazed on certain strains of clovers (*Trifolium protense, T repens, T subterraneum*) that possess high oestrogenic potency. Wethers may show signs even after a few weeks on such pastures and the epithelial changes may be so severe as to cause urethral obstruction, rectal prolapse and even death. Injection of wethers with hormonal growth promotants (HGP) appears to have a similar effect.[10] It is of comparative interest that changes of comparable magnitude are not described in other ruminant species grazing the same pastures or receiving HGP. Nevertheless epithelial hyperplasia and squamous metaplasia are detectable histologically in the prostates of bulls and goats injected with diethyl stilboestrol.[11] Possibly, similar close (histopathological) scrutiny of male ruminants consuming food contaminated with myco-oestrogens may also reveal changes in ASG and other genitalia; boars receiving zearalenone have a reversible atrophy of genitalia, cessation of spermatogenesis, and diminished libido.[12]
Extensive studies of bulls in several countries have revealed seminal vesiculitis as a common lesion which may contribute to reduced fertility and persistence of infection in a herd.[13,14,15] In cattle populations known to be free of brucellosis, continued prevalence of seminal vesiculitis, with spread of infection via ducts to the ampulla and epididymis, has been causally related to a high incidence of anomalous ASG development in the bull.[16,17] It is of comparative interest that detailed examination of ASG of 845 and 1000 rams and bucks, respectively,[6,18] revealed few of the ASG anomalies previously described in bulls. Furthermore, in the same studies, seminal vesiculitis was never detected on gross examination in rams or bucks, and in rams was only confirmed histologically in animals infected with *Br ovis* – thereby supporting the link in the bull, between anomalous development, infection and inflammation.

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