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Some insights into canine azoospermia – histological results from ten cases
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Azoospermia is a common yet not well-understood problem in male dogs. The diagnosis is usually confirmed by repeated semen analyses showing a lack of sperm, and the prognosis is generally poor. Detailed information on testicular level obtained by biopsies, as regularly performed in men with azoospermia of unclear aetiology, is widely missing for the dog. However, this is essential to understand the underlying cause. The aim of this study was to get some further insights into canine azoospermia by investigating hormonal, bacteriological, genetic, and histological changes in dogs with proven azoospermia. Ten dogs with confirmed azoospermia were included (Collie n=3, Cairn Terrier, Danish Spitz, miniature poodle, Coton de Tulear, Welsh Corgi Pembroke, Icelandic Sheepdog, Labrador Retriever n=1). From all dogs, fractionated semen and blood samples were obtained. Alkaline phosphatase was determined from cell-free seminal plasma and bacteriological examination was performed from semen. Blood samples were used for determination of concentrations of LH, testosterone and oestradiol-17β (E2) and karyotyping. Biopsies from both testes were taken under general anaesthesia for further histopathological characterisation. A minimum of four slides from different paraffin blocks was evaluated from each dog from the respective testis. To assess the stage of spermatogenesis, 50 round tubules were evaluated blinded according to the most developed germ cell observed. In these slides, six testicular inflammatory parameters (thickness of basal membrane, presence of fibrosis, immune cells, shrinkage of lumen, abnormal mitotic patterns, and vacuoles) were also assessed. Additionally, a morphometric evaluation of the testicular tissue was performed to identify the percentage distribution of the three compartments (seminiferous tubules, lymph and blood vessels, and interstitial tissue) individually for each left and right testis and compared to normospermic control dogs (n=5). The mean age at time of diagnosis was 6.2±2.1 years ranging form four to ten years old. Seven dogs had sired successfully before with a mean of 2.3±1.5 years since last successful mating. The andrological examination showed that nine dogs had smaller testis (n=5) or testis within the lower reference (n=4) compared to what was expected for the respective sizes of dogs. The histopathological examination showed that 4 dogs had signs of generalized autoimmune orchitis with early arrest of spermatogenesis, i.e. SCO (Sertoli-cell only) or spermatogonia only, and 6 dogs had late arrest of spermatogenesis. Of these, 2 showed generalized inflammatory changes and 3 showed focal inflammatory changes. The morphometry of the azoospermic testes revealed a significantly lower percentage of seminiferous tubules (59.42%±13.60 to 88.81%±1.85, p<0.05), a significantly higher percentage of interstitial tissue (39.51%±13.42 to 10.72%±0.40, p<0.05) and a significantly higher percentage of vessels (1.07%±0.40 to 0.47%±0.08, p<0.05) in azoospermia samples. No significant difference was identified between the morphometry of the left and right azoospermic testes. Levels of LH, testosterone and E2 were found to be within the normal reference interval. Our results indicate that testicular inflammatory changes are a common reason/consequence in randomly selected azoospermia patients that can only be identified by testicular biopsies confirming the relevance of this diagnostic approach.