Cystic endometrial hyperplasia with hydrometra and inappropriate lactation in a Nigerian dwarf goat, a case report
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Cystic endometrial hyperplasia (CEH) is caused by an increase in number and size of endometrial glands due to progesterone (P4) stimulation of the uterus at an inappropriate time in the reproductive cycle. This condition might result in fluid secretion by the glands into the uterine lumen, causing hydrometra, mucometra, or pyometra. This pathology has been well described in dogs, but is rare in other domestic animals. In dogs, CEH is the result of P4 acting on the endometrium in diestrus, after stimulation by estrogen during the proestrus period. This causes proliferation of the endometrium while the cervix remains closed under the influence of P4. In small ruminants, it is possible that prolonged estrogen stimulation is involved similar to the pathology in the bitch. In the doe, elevated estrogen levels may result from follicular cysts, granulosa cell tumors, pituitary adenomas, or ingestion of estrogen-containing plants. In this case, an 8-year-old Nigerian dwarf goat doe was presented to the University of Georgia Veterinary Teaching Hospital for a two-week duration of pelvic limb paresis, and abnormal mammary gland development. Physical examination revealed a fully distended udder with milk expressible from both teats. The doe was diagnosed with *Parelaphostrongylus tenuis*, by analysis of cerebrospinal fluid, and treatment with flunixin meglumine and fenbendazole was initiated. The *P. tenuis* infection was the likely cause of the neurological signs; however, it did not explain the inappropriate lactation. Transabdominal ultrasound examination revealed multiple loops of fluid-distended uterine horn, and an increased area of echogenicity on the left ovary, consistent with luteal tissue. Based upon the ultrasonographic findings, pseudopregnancy and hydrometra (cloud burst) was the primary differential for the inappropriate lactation. The doe was administered two doses of 5 mg of dinoprost, three days apart in an attempt to produce a sustained luteolytic effect. A small amount of vaginal discharge was observed approximately 24 hours after the first injection; however, there was no reduction in the uterine size over the next 48 hours, evidenced by ultrasonography. The goat was humanely euthanized using a sodium pentobarbital injection due to worsening of neurologic disease, and was subsequently necropsied. Gross reproductive findings included severe distension of the uterine horns with a slightly cloudy uterine fluid containing scattered small caseous precipitate. Histologically the endometrium was hypercellular with luminal papillary projections, and contained cystic structures lined with a single layer of tall columnar epithelium. Scattered collections of eosinophils and dilated glands containing neutrophils and bacteria were seen throughout the lamina propria. The ovaries contained a follicular cyst and multiple corpora lutea (CL). It is hypothesized that this pathology might be the result of CEH due to continuous estrogen stimulation from a follicular cyst. Moreover, lack of a luteolytic signal (possibly influenced by endometrial pathology), could cause persistence of the CL and prolongation of P4 stimulation to the endometrium, which might also be a contributing factor for CEH in this doe. Cystic endometrial hyperplasia may have subsequently predisposed this doe to hydrometra and pseudopregnancy, leading to inappropriate mammary development and lactation. Hydrometra is not commonly combined with CEH. Based on these findings CEH should be considered as a differential diagnosis in does with hydrometra.

**Keywords:** Cystic endometrial hyperplasia, pseudopregnancy, hydrometra, inappropriate lactation.

**Reference**