Endometritis

Managing Persistent Post-Breeding Endometritis

Igor F. Canisso, DVM, MSc, PhD[a], Jamie Stewart, DVM, MS[a], Marco A. Coutinho da Silva, DVM, MSc, PhD[b,*]

KEYWORDS

• Mare • Endometritis • Post-Breeding endometritis • Uterine lavage • Intrauterine fluid

KEY POINTS

- Susceptible mares have impaired uterine defense mechanisms.
- Persistent post-breeding induced endometritis is characterized by abnormal imbalance in pro-inflammatory and anti-inflammatory cytokines.
- Diagnosis can be made through endometrial histology, culture, cytology, and/or ultrasound; each of which have differing advantages and disadvantages.
- The most common management strategies include combinations of therapeutic techniques such as uterine lavage and ecbolic treatments that enhance drainage of the persistent uterine fluid.

INTRODUCTION

Mares have been classified as susceptible and resistant to endometritis based on their ability to clear bacteria from the reproductive tract following experimental inoculation through the cervix.[1–6] Clinically, mares are classified as susceptible to endometritis based on the persistent presence of intra-uterine fluid accumulation by 24 to 48 hours post-breeding.[7] For years, it was thought that post-breeding endometritis was solely caused by infectious agents, particularly bacteria.[5,8] However, a seminal study demonstrated that estrous mares infused with either bacteria (*Streptococcus zooepidemicus*) or spermatozoa presented similar number of neutrophils in the uterine fluid at 4 hours post uterine infusion.[9] The results of this study established the concept that post-breeding endometritis is a normal physiologic, transient inflammatory response

[a] Department of Veterinary Clinical Medicine, College of Veterinary Medicine, University of Illinois Urbana-Champaign, 2001 S Lincoln Ave, Urbana, IL 61822, USA; [b] Department of Veterinary Clinical Sciences, College of Veterinary Medicine, The Ohio State University, 601 Vernon Tharp St, Columbus, OH 43210, USA
* Corresponding author.
E-mail address: marco.dasilva@cvm.osu.edu

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that serves the purpose to clear the uterus from excess of sperm cells, seminal plasma, debris and bacterial contaminants.[5,10]

Persistent post-breeding endometritis is a prolonged (ie, longer than 48 h) inflammatory response of the endometrium caused by spermatozoa and is estimated to affect approximately 10% to 15% of mares.[7] In some cases, bacterial contamination may also be present. Therefore, the objectives of this article are to discuss some of the most relevant literature concerning persistent post-breeding endometritis and the authors' clinical experiences managing this condition in ambulatory practice and referring hospitals.

UTERINE DEFENSE MECHANISMS

Reproductive Tract Barriers to Prevent Uterine Infection

Vulva, vestibule-vagina sphincter, and cervix represent three physical barriers or seals in the mare reproductive tract to prevent uterine infections. If one of these barriers is not functional, the mare will be prone to pneumovagina and pneumometra (physometra), causing irritation of the uterus and potential aspiration of bacteria. The vulva, an outward barrier against infection, should have perfectly apposed lips and be vertically oriented withe 2/3 of the vulvar length located below the pelvic brim and 1/3 of the vulvar length above the pelvic brim (**Fig. 1**).

The vestibule-vagina sphincter is the only barrier that remains functional while the mare is in estrus and helps to prevent aspiration of air and debris into the cranial vagina. However, it is also a common site for lacerations during foaling, and its integrity and competency should be assessed during breeding soundness examinations. When performing vaginoscopy, the sphincter should offer some resistance to the passage of the speculum. While advancing or retracting the vaginal speculum, the sphincter should close like curtains in front of the opening of the speculum. Another method consists of physically separating the vulvar lips with hands and fingers as wide as possible, and listening for any noise of air being aspirated into the cranial vagina, which would denote an incompetent vestibule-vagina sphincter. Additionally, if the vestibule-vaginal sphincter is incompetent, the operator may be able to directly visualize the vagina and cervix (**Fig. 2**). The cervix should be free from lacerations and adhesions, relaxed and open during estrus, and be tight and closed during diestrus and pregnancy.

Uterine Immune Response

Following intrauterine deposition of semen, there is initiation of a complement system which is responsible for chemotaxis and activation of polymorphonuclear neutrophils (PMNs) to the uterine lumen as soon as 30 minutes after insemination.[5,11] The activated PMNs then function to remove bacteria, debris, and sperm cells from the uterus through phagocytosis.[3,12] Neutrophil extracellular traps (NETs) (ie, extracellular fibers of DNA) also begin to form and appear to play a major role in the uterine clearance post-breeding by binding to pathogens but not sperm.[13] Rebordão and collaborators[14] found that mares suffering from *S zooepidemicus* or *E. coli* endometritis formed NETs *in vivo*, demonstrating a potential complementary mechanism by which mares can resist endometritis.[14] Further work is still warranted to determine if and how impaired NET formation occurs in mares susceptible to infectious endometritis. While NETs may play a crucial role in preventing bacterial endometritis, they also release inflammatory mediators that some speculate could lead to endometrial degenerative disease.[15] Overstimulation of NET formation and/or a decrease in NET degradation have been postulated as potential causes for increased endometrial fibrosis and