Proceedings of the
18th Annual Meeting of the
Italian Association of Equine Veterinarians
SIVE

Feb. 3-5, 2012 - Bologna, Italy

Next SIVE Meeting:

Feb. 1-3, 2013 – Arezzo, Italy

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Endometritis in the mare can be divided into acute infectious, chronic infectious or post mating induced endometritis. The most critical component in preventing the disease is rapid physical clearance of uterine contents after foaling and mating. Mares that are unable to clear the by-products of insemination or foaling quickly may develop post mating induced or acute endometritis. If endometritis is not promptly resolved, the infection can become chronic and difficult to treat successfully.

Repeated foaling and breeding can cause anatomical defects such as poor perineal conformation, incompetent vagino-vestibular sphincter, vaginal stretching, incompetent cervix, a pendulous uterus. Prolonged inflammation can lead to degenerative changes such as abnormal uterine contractility, periglandular fibrosis, vascular elastosis, lymphangectasia, scarring and atrophy of endometrial folds or damage to the mucociliary apparatus (Inoue et al. 2000; LeBlanc et al. 1995; LeBlanc et al. 1998; Schoon et al. 1999; Troedsson and Liu 1991). Each of these abnormalities can hinder physical clearance.

Older nulliparous mares that are not mated until 10 or more years of age and those that have repeated embryo recovery attempts also experience delayed uterine clearance, often because of cervical malfunction. The cause leading to an inability to rapidly clear the uterus must be identified and corrected, if possible or treated, if the mare is to carry a foal successfully to term.

**DIAGNOSTICS**

The cause of chronic endometritis can be difficult to identify because clinical signs, ultrasonographic and laboratory findings vary between uterine pathogens. Different bacteria express different virulent factors and have different modes of evading the immune response. This can result in an array of clinical signs, ultrasonographic and laboratory findings. Some bacteria such as *E coli* tenaciously adhere to epithelial surfaces, preventing their physical removal. Others such as *streptococci* stimulate production of an inflammatory exudate, interfering with neutrophil phagocytosis. *Pseudomonas aeruginosa* and some yeast and fungi secrete a biofilm, an adhesive matrix that supports growth and maintenance of bacterial micro-colonies. Biofilms provide inherent resistance to antibiotics and both cellular and humeral immune defenses resulting in persistent, chronic infections even after prolonged antibiotic treatment. (Costerton et al. 1995; Donlan and Costerton 2002; Otto 2006). Some bacteria or fungi form focal plaques that are not identified by routine swab culture techniques, while others don’t produce intrauterine fluid, the “hallmark” of endometritis. The uterine response to a pathogen also contributes to the establishment and chronicity of infection. During acute and subacute endometritis, mucus production by epithelial cells lining the endometrium, (Causey 2007; Causey et al. 2000), is increased while in chronically inflamed endometria, there is loss of the epithelium and mucus blanket and increased opportunities for bacter-
Adhesion (Causey et al. 2008). Changes in the production, elasticity or viscosity of endometrial mucus can interfere with the ability of the muco-ciliary apparatus to remove particulate matter, with sperm migrating to the oviduct or with antibiotic penetration by the endometrium. Because of the many factors affecting fertility, clinicians need to tailor their diagnostics specifically for each individual. Identifying the inciting cause of bacterial endometritis may require more than swabbing the endometrium. Culturing endometrial biopsy tissue or uterine fluids are more sensitive methods for identifying E. coli and other gram negative organisms than culture swab while endometrial cytology identifies twice as many mares with acute inflammation than uterine culture swab (LeBlanc et al. 2007; Nielsen 2005; Riddle et al. 2007). Accumulation of 2 or more cm of uterine fluid during the ovulatory period is a good indicator that a mare is susceptible to mating-induced endometritis and is consistently associated with decreased pregnancy rates. (Barbacini et al. 2003; McKinnon et al. 1988; Pycock and Newcombe 1996). However, intra-uterine fluid is not always associated with bacterial endometritis. Thoroughbred mares from which the bacterial organisms, E. coli, Staphylococcus aureus, Pseudomonas spp., or bacillus were isolated had intra-uterine fluid in less than 40% of the ultrasonographic examinations (17-39% depending on organism) conducted immediately before a uterine culture was obtained. While mares with β-hemolytic Streptococcus, Klebsiella pneumoniae, Enterobacter cloacae, or yeast isolated from their uterus had intra-uterine fluid in 45-55% of the ultrasonographic examinations (Burleson et al. 2010). Pathogens associated with uterine fluid were more likely to have neutrophils on cytology while pathogens not associated with uterine fluid, tended to be negative for neutrophils on cytology.

DEGENERATIVE CHANGES CONTRIBUTING TO ENDOMETRITIS

An adequate blood supply is needed for hormonal signaling, uterine contractility, placentation, and fetono-endometrial interactions. Recent work indicates that blood flow and tissue perfusion is diminished in some subfertile mares and mares with lymphatic cysts within the endometrium (Esteller-Vico et al. 2007; Ferreira et al. 2008; Schoon et al. 1999). The severity of vascular lesions increases with parity and therefore age of the mare. Old (> 15 yr of age) pluriparous mares are 233 times more likely to have moderate to severe vascular elastosis compared to young, nulliparous mares (Liu et al. 2008). Degenerative changes in arterial and venous vessels include elastosis, fibrosis and fibroelastosis of the vessel wall as well as perivascular fibrosis and calcification processes. These lesions can be identified on routine endometrial biopsies if tissue sections are stained with von Giesen stain, a silver stain. Angiosis appears to indirectly reduce fertility through a reduction in endometrial perfusion, and through disturbances in uterine drainage caused by reduced venous return in capillary beds. In reproductively healthy mares, endometrial edema develops physiologically during estrus, resulting in the typical estrous edema of the uterine wall. The edema peaks about 24 h before ovulation and disappears rapidly after ovulation, providing the drainage mechanisms are functionally intact. If drainage is reduced or inflammation persists after mating, a pathological endometrial edema develops that is morphologically characterized by persistent lymphangectasia (Samper 2009). Prolonged endometrial edema results in continuation of inflammatory response and in decreased fertility.

CONCLUSION

A delay in the uterine clearance of inflammatory by-products and semen is the primary cause of infertility in the mare. If not corrected quickly it can result in prolonged inflammation, ulceration of the uterine epithelium leading to bacterial endometritis. Prolonged inflammation and infection in combination with aging produces a number of degenerative changes to the uterus. These changes affect blood flow, uterine contractility and pregnancy maintenance. Diag-
ACKNOWLEDGEMENTS

Portions of this manuscript has previously been published: LeBlanc MM. Advances in the diagnosis and treatment of chronic infectious and post-mating induced endometritis in the mare. Reprod Domest Anim 2010: 45 Suppl 2: 21-27.

REFERENCES


