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Endometritis in Mares

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Recent scientific observations have had a profound impact on the way endometritis is managed in mares. These observations are:

- mares susceptible to endometritis have an impaired ability to physically clear fluid from the uterus
- sperm triggers a normal physiological inflammatory reaction within the uterus
- oxytocin promotes rapid and complete uterine clearance.

This issue of *Large Animal Veterinary Rounds* reviews the etiology and pathophysiology of endometritis, describes the mechanisms of uterine defense, and analyzes the options for diagnosis and treatment based on the observations outlined above.

Introduction

Endometritis is defined as an inflammation of the endometrium that can be acute or chronic, infectious or noninfectious. It is, reportedly, the third most common problem faced by equine veterinarians¹ and the primary cause of infertility and sub-fertility in horses.² The presence of cellular debris, inflammatory products, and microorganisms in the uterus creates an unsuitable environment for the embryo. As well, the release of prostaglandin- $F_{2\alpha}$ ($PGF_{2\alpha}$) during the inflammatory process may cause premature luteolysis impeding the establishment of pregnancy. Moreover, endometritis is associated with early fetal death due to interference with placentation. Based on the etiology and pathophysiology, endometritis can be divided into four groups: • sexually-transmitted diseases; • chronic infectious endometritis; • persistent mating-induced endometritis; and • endometrosis.³

Sexually-transmitted diseases (STD): Contagious equine endometritis (CEM) caused by *Taylorella equigenitalis* is a true STD resulting in cervicitis, vaginitis, and endometritis. A copious mucopurulent vaginal discharge usually develops within a week after breeding with an asymptomatic infected carrier stallion. However, a more insidious form with minimal clinical signs has been recently recognized in Europe.⁴ In North America, rigorous control measures have proven efficacious and CEM has not been diagnosed since the late 1970s. In addition to CEM, it has been suggested that genital infections with *Pseudomonas aeruginosa* and *Klebsiella pneumoniae* may be sexually transmitted.

Chronic infectious endometritis: Uterine infection is commonly the result of contamination by fecal and genital opportunistic flora. Mares with functional uterine defense mechanisms are capable of eliminating infections, while those with impaired uterine defenses develop chronic infectious endometritis. The most common microorganisms involved in infectious endometritis are aerobic bacteria. *Streptococcus zooepidemicus* is responsible for approximately 65% of the cases while *Escherichia coli*, *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa* account for approximately 10%. Other aerobic and anaerobic bacteria, fungi, and yeasts have been occasionally implicated as causes of endometritis.

Persistent mating-induced endometritis (PMIE): Recently, it has been demonstrated that sperm have a complement-dependent chemoattractant effect on neutrophils. Insemination of mares with semen free



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of bacteria produces an inflammatory response that is similar to, or greater than, that observed after intrauterine infusion with bacteria or after natural breeding.^{5,6} Sperm deposition into the uterus triggers a normal physiological inflammatory response that may be necessary for clearing excess semen before the embryo reaches the uterus 5 to 6 days after ovulation.⁷ However, mares with impaired uterine defense mechanisms are not capable of resolving the inflammation triggered by sperm and develop PMIE.

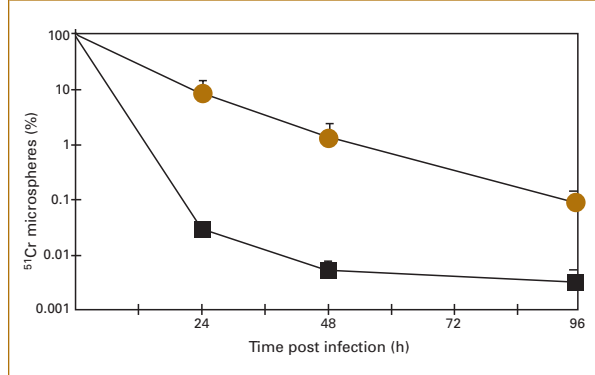
Endometritis: Chronic, degenerative changes of the endometrium such as periglandular fibrosis, lymphatic stasis, and glandular dilation may result from repeated uterine inflammation, but the condition has also been observed in older mares without any known history of endometritis, suggesting that degenerative fibrosis of the endometrium may sometimes be the result of aging rather than inflammation. Based on the possibility of a non-inflammatory cause of the disease, the term “endometrosis” rather than “degenerative endometritis” should be used to describe this condition.²

Uterine defense mechanisms and susceptibility to endometritis

In the 1960s, it was recognized that young maiden mares could eliminate bacteria within 72 to 96 hours after experimental infusion into the uterus, while older multiparous mares were unable to eliminate the microorganisms and became chronically infected. These observations led to the concept of resistance and susceptibility to endometritis. Susceptibility is essentially the failure of uterine defense mechanisms to spontaneously resolve the inflammatory process and eliminate concurrent infection.

Uterine defense is complex and involves the vulva-vagina-cervix physical barrier, humoral and cellular immune responses, and a muscular contractility response for physical clearance from the uterus. Uterine defenses are enhanced during estrus and invasive uterine manipulations are preferably conducted only during this period of the cycle. The mare's endometrium is considered part of the mucosal immune system because of its ability to produce and secrete immunoglobulins. Passive diffusion of immunoglobulins from the peripheral circulation into the uterine lumen also occurs, but this contribution is less significant. Immunoglobulins are present in glandular and luminal epithelium and secretions, and in the uterine interstitium; IgA predominates as it does on other body surfaces. The main leukocyte involved in the uterine cellular immune response is the polymorphonuclear (PMN) neutrophil. The number of neutrophils in the uterine fluid increases dramatically within hours after infection and remains increased for days. Neutrophil migration to the uterus is initiated by the release of chemotactic factors from the site of inflammation; complement, prostaglandins, leukotriene, histamine, and some enzymes may act as chemoattractants. The process of opsonization also plays a central role in the uterine defense mechanism by

Figure 1: Impaired uterine clearance is the single most important factor determining susceptibility to endometritis in mares. Evaluation of uterine clearance after intrauterine infusion of bacteria and radiolabeled microspheres in resistant (■) and susceptible (●) mares during estrus demonstrated that clearance was delayed in susceptible mares.⁸ (Adapted with permission)

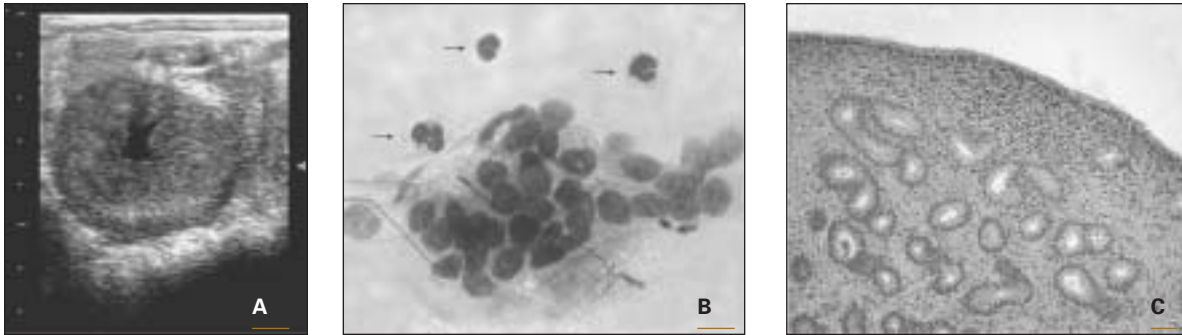


enhancing neutrophil phagocytosis. Opsonins attach to the surface of microorganisms providing binding sites for neutrophils to attack the organism; complement and IgG are the major opsonins in uterine fluid.

Studies on immunoglobulins and the functional ability of neutrophils and opsonins in the uterus of susceptible mares have not identified the presence of an impaired immune response. Instead, uterine muscular response and mechanical drainage of the uterus have been recognized as major contributors to the uterine defense mechanism. Uterine contractions are necessary for physical clearance of fluid, inflammatory debris, and bacteria via cervical and lymphatic drainage. Myometrial contractility is mediated by hormonal (oxytocin and PGF_{2α}) and neuronal (autonomic neurotransmitter) interactions. Oxytocin and possibly, neurotransmitter release due to sexual stimulation, as well as intrauterine PGF_{2α} release during inflammation, trigger myometrial contractions resulting in uterine clearance after breeding or infection. In the 1990s, it was demonstrated that susceptible mares have an impaired ability to physically clear the uterus. Uterine clearance markers are promptly eliminated after infusion into the uterus of resistant mares, while negligible amounts of markers are eliminated in susceptible mares (Figure 1).^{8,9} It has since been established that impaired uterine clearance is the single most important factor determining susceptibility to endometritis.³

Myometrial activity does not differ between susceptible and resistant mares when there is no uterine infection. However, with infection, there is an initial increase in uterine activity (frequency and intensity of myometrial contractions) that remains increased for up to 20 hours in resistant mares, but subsides within 6 to 10 hours in susceptible mares.¹⁰ The myometrial dysfunction in susceptible mares may be related to reduced numbers of α-adrenergic receptors, the unresponsiveness of receptors, or a deficiency in signal translation. It has

Figure 2: Endometritis is diagnosed by ultrasonography, and by evaluation of endometrial cytology and histopathology. Signs of endometritis: A. Intrauterine fluid accumulation during diestrus (scale: 10 mm); B. Presence of PMN neutrophils (arrows) in association with endometrial cells on cytology samples (1000x magnification, Diff Quick® stain) and C. Interstitial leukocyte infiltration upon histopathological exam (200x magnification, HE).



been demonstrated that stimulation of α_2 -adrenergic receptors results in myometrial contraction in resistant, but not in susceptible mares, and α_1 - and α_2 -adrenergic agonists enhance uterine contractility after oxytocin treatment only in resistant mares.^{11,12} A deficiency in myometrial oxytocin receptors is unlikely because uterine clearance and intrauterine pressure are similar in resistant and susceptible mares after oxytocin treatment.^{13,14} Nevertheless, susceptible mares show a greater proportion of aberrant uterine contractions after oxytocin treatment (ie, simultaneous uterine horn and body contractions or reverse contractions), suggesting a dysfunction in electrical signaling among myometrial cells.¹¹ In addition, clearance in susceptible mares may be more difficult due to a greater ventral displacement of the uterus compared to resistant mares.¹⁵

Diagnosis

The diagnosis of endometritis must be based on the presence of inflammation. Endometritis may occur in the presence or absence of uterine infection; therefore, the isolation of bacteria from the uterus does not prove the presence of endometritis, nor does the failure to isolate bacteria eliminate this diagnosis. History and the main predisposing factors (age, parity, and poor perineal/vulval conformation) should be considered. Ultrasonography has proven a valuable diagnostic tool since it has been demonstrated that intrauterine fluid accumulation during diestrus is associated with endometritis (Figure 2A). Moreover, PMIE can be diagnosed based solely on detecting fluid (any amount) in the uterus using 2 ultrasound examinations, one between 4 to 12 hours and one at 24 hours post-breeding.^{4,16,17} The clinical exam should also include an evaluation of endometrial cytology, culture, and histopathology.

The evaluation of endometrial cytology is a simple, quick, and inexpensive method to determine endometrial health. Samples for cytology are collected using a guarded swab and

smears can be fixed with methanol or stained immediately with common hematological stains (eg, Diff Quick®). Because the presence of neutrophils is a hallmark of inflammation, their presence in uterine samples is definitive evidence of endometritis (Figure 2B). However, there is no standard method to interpret the results of endometrial cytology. The presence of >5 neutrophils in 10 microscopic fields (400x), >2 neutrophils in 5 fields, or a proportion of neutrophils to endometrial cells > 1:10 or 1:40 are suggested as positive signs of endometritis. With practice it becomes easier to distinguish normal from abnormal cytological samples without the need to count absolute numbers of cells.¹⁸ Generally, only positive cytology samples are submitted for culture; the same swab used for cytology can be submitted, provided sterile slides are used to make the smears. Gram staining may be helpful in selecting an antibiotic for treatment before culture results are available.

Endometrial histopathology is indicated for any nonpregnant mare with suspected uterine disease. It is especially important for the diagnosis of chronic endometritis and endometriosis, and is the best prognostic tool to gauge future fertility. Endometrial samples are taken using uterine biopsy forceps with an “alligator jaw;” the samples are fixed, preferably in Bouin’s fixative, and the slides are stained with HE (hematoxylin and eosin). Interstitial leukocyte infiltration is the pathognomonic sign of endometrial inflammation, with neutrophils predominating in the acute process and lymphocytes in chronic cases (Figure 2C). Periglandular fibrosis, lymphatic stasis, and endometrial cysts are characteristic signs of endometriosis. Endometrial histopathological specimens are usually classified as normal (Grade I), mildly or moderately inflamed (Grades IIa or IIb), and severely inflamed/degenerated (Grade III). The probability of carrying a foal to term decreases as the grading increases (80%-90%, 50%-80%, 10%-50%, and <10% for Grades I, IIa, IIb, and III, respectively).¹⁹

Treatment

Predisposing anatomical causes such as abnormal perineal and vulval conformation, cervical or recto-vestibular lacerations, pneumovagina, and urine pooling must be corrected by surgical techniques as a first step in resolving endometritis. Sexually transmitted diseases and infectious endometritis require the elimination of etiologic organisms using antimicrobials, while PMIE may only require methods to enhance natural uterine defenses (uterine lavage and uterotonics). Indiscriminate use of antibiotics for PMIE prevention or treatment is not justified and is strongly discouraged.^{4,17} Endometriosis can be treated by curettage (either physical or chemical), but regardless of treatment, the prognosis is poor. Even in the absence of active inflammation, mares identified as being susceptible to endometritis should always be bred using minimum contamination techniques.

Antimicrobial therapy

The majority of uterine infections in mares are limited to the endometrium and an intrauterine infusion of antimicrobials is the most common approach for treatment, even though the use of intrauterine infusion of most antimicrobials is off-label. Uterine infusions are performed daily for 3 to 7 days during estrus, when natural uterine defenses are more effective, preferably after uterine lavage. Infusions should be avoided beyond 2 days after ovulation due to increasing progesterone levels and a consequent decrease in uterine defense mechanism efficiency.¹⁶ Uniform distribution of antimicrobials within the uterus does not seem to be crucial. The infusion of volumes that are more likely to result in reflux through the cervix does not ensure homogeneous distribution throughout the lumen; small volume infusions (30 to 50 mL) are preferred over larger volumes. The difficulties involved in identifying the etiologic agent and predicting antibiotic sensitivity during the short breeding season make broad-spectrum antibiotics the first choice for treatment of infectious endometritis. Ampicillin (1 to 3 g), effective against *Streptococcus zooepidemicus* and many strains of *Escherichia coli*, is a good choice while waiting for culture results. Other broad-spectrum antibiotics commonly used include ceftiofur (1 g), ticarcillin (6 g), carbenicillin (2 to 6 g), and drug combinations such as penicillin (3 to 5 x 10⁶ IU)/gentamicin (1 to 3 g) and penicillin/neomycin (3 to 4 g)/ polymixin B (1 x 10⁴ to 10⁶ IU).²⁰ However, the use of drug combinations is discouraged by some authors because there is often drug incompatibility (the same is true for combinations of antibiotics with antiseptics). In Canada, gentamicin and amikacin (2 g) are the only antibiotics labeled for intrauterine infusion in mares. Aminoglycosides should be buffered by mixing

equal volumes of antibiotic and a 7.5% sodium bicarbonate solution before use. Tetracycline formulations are not recommended for uterine infusion because they are extremely irritating.

Systemic antibiotic treatment should be considered in the following cases:

- when the clinical examination suggests that deeper layers of the uterus are infected or inflamed
- if the mare shows systemic signs of illness in conjunction with uterine disease
- when recontamination of the uterus is of great concern,
- when repeated uterine infusions are not practical.

Systemic therapy can be performed during diestrus and the more sustained tissue levels achieved may be more effective than intermittent high tissue levels resulting from uterine infusion.

Common systemic antibiotics include trimethoprim-sulfa, ampicillin, penicillin, and gentamicin, in the same doses as indicated for any systemic infection. Drug interactions resulting in antagonism, selection of resistant microorganisms (drug resistance), and superinfection, are responsible for the failure of antibiotic treatments. Drug resistance may follow inadequate doses or insufficient repetition of treatment, while superinfection is the consequence of treatment for one microorganism resulting in the replacement of that organism with another organism, often more difficult to treat. The ultimate superinfection problem is yeast or fungal overgrowth. These infections are difficult to treat and may produce marked damage to the uterus. Uterine infusions of antiseptics (0.05 to 0.1% povidone-iodine solution, 2% vinegar solution) and specific antimicrobial drugs (Amphotericin B, 50 to 250 mg; Clotrimazole, 300 to 600 mg) for periods up to 12 days are recommended for treatment of these infections.²⁰

Uterine lavage

Recognition of the importance of mechanical uterine clearance in the pathophysiology of uterine inflammation was responsible for the introduction of uterine lavage as a therapy for endometritis. Lavage should precede antimicrobial uterine infusion for treatment of infectious endometritis and, in combination with uterotonic treatment, is the main therapy for PMIE.^{4,16,17} For PMIE, the uterus should be examined by ultrasound between 4 and 12 hours after breeding and a lavage should be performed if fluid is seen. Early treatment during this time period is preferred before bacteria begin the logarithmic phase of growth. The mare should be rechecked 24 hours after breeding and treated again if fluid accumulation persists. If ultrasound evaluations are not possible, a uterine lavage should be performed between 4 and 12 hours after breeding as a preventive

measure. One to 2 L of warm saline are infused by gravity and recovered by siphoning using a large bore catheter that is retained in the uterus by a cuff. The process is repeated until the recovered effluent is clear and transrectal massage of the uterus is not necessary. Treatment with oxytocin should follow to ensure complete elimination of the infused fluid.

Uterotonics

Because of the importance of physical clearance of the uterus in resistance to endometritis, the administration of uterotonics is an ideal therapy. Uterotonics result in rapid and complete elimination of fluid, inflammatory debris, and microorganisms from the uterus. Oxytocin and PGF_{2α} stimulate uterine contractions, but oxytocin is preferred because it more effectively promotes uterine clearance and provides a shorter stimulus to smooth muscle contraction. Intrauterine pressure increases almost immediately when oxytocin is administered (either IM or IV) during any stage of the estrous cycle, early postpartum, or anestrus. Pressure peaks within 10 minutes after treatment and subsides within 20 to 30 minutes.^{14,21} Oxytocin induces a dose-dependent response and 20 IU is the recommended treatment dose; there is no advantage and there may be adverse effects with higher doses.^{4,16,17} After treatment with 20 IU, > 90% of intrauterine content is eliminated within 15 to 30 minutes.¹³ For the treatment of PMIE, oxytocin can be administered alone, but it is preferably given right after uterine lavage. If fluid accumulation persists in the uterus after uterine lavage at 24 hours post-breeding, oxytocin treatments can be repeated every 2 to 12 hours for up to 3 days after ovulation.¹⁶ If antibiotic infusion is used, it should be performed at least 30 minutes after oxytocin treatment.

Sedatives and anti-inflammatories

Sedatives with α -adrenergic agonist action (xylazine and detomidine) should be used when mares need to be sedated before treatment for endometritis. These sedatives induce an increase in intrauterine pressure and potentiate the effects of oxytocin. On the other hand, treatment with acepromazine (α -adrenergic antagonist) should be avoided because it causes a reduction in the number of uterine contractions after oxytocin treatment.^{11,12} It is also important to note that mares receiving treatment with prostaglandin inhibitors (phenylbutazone) or β_2 -adrenergic agonists (clenbuterol) should be treated with oxytocin after breeding because these drugs decrease uterine contractility. Exposure of mares to breeding stimuli (stallion call, visual contact with the stallion, active teasing) is associated with oxytocin release by the pituitary gland and the rapid onset of myometrial contractions. In most intensively managed stud farms, mares are teased only

once a day and have no further contact with stallions; therefore, it may be beneficial to permit more interaction between the stallion and mares, especially for mares susceptible to endometritis.^{22,23}

Conclusion

Recent research has emphasized that failure of the ability to physically clear inflammatory products and bacteria from the uterus is an important predisposing factor to equine endometritis. Endometritis is diagnosed by uterine ultrasonography, endometrial cytology, and histopathology. Various forms of endometritis are recognized and appropriate treatment is based on the etiology and pathophysiology of the disease. Treatment generally involves intrauterine antibiotic infusion, uterine lavage, and administration of uterotonic agents.

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Dr. Albert Barth graduated in 1971 and received an MVetSci degree in Theriogenology in 1978 from the WCVm. He has been a professor at the WCVm since 1978 and is a Diplomate of the American College of Theriogenologists.

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Abstract of Interest

Oxytocin enhances clearance of radiocolloid from the uterine lumen of reproductively normal mares and mares susceptible to endometritis.

LEBLANC MM, NEUWIRTH L, MAURAGIS D, KLAPSTEIN E, TRAN T.
 The effects of oxytocin on the percentage of technetium 99m albumin colloid (99mTc-microAA), cleared from the uterine lumen was measured in 13 mares. Scintigraphy was performed during 4 consecutive oestrous cycles, on Day 3 of oestrus during Cycles one and 3 and 48 h after ovulation during Cycles 2 and 4. Oxytocin (20 iu) was given i.v. after the initial scintigraphy image during Cycles 3 and 4. Seven multiparous mares (Group 1) were classified as 'susceptible' and 6 mares (2 nulliparous and 4 multiparous; Group 2) were classified as 'resistant' to endometritis. All mares cleared > 90% of 99mTc-microAA within 30 min of oxytocin injection. When no drug was given, Group 1 mares cleared negligible amounts of radiocolloid (< 5%) and Group 2 mares cleared 50-80%. No mares showed signs of colic after oxytocin administration. It is concluded that oxytocin enhances uterine clearance of radiocolloid and may be useful for treating mares exhibiting impaired uterine clearance.
Equine Vet J 1994;26(4):279-282.

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