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Emerging Insights into the Pathogenesis and Clinical Management of Canine Endometritis

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Abstract

Endometritis is an important cause of subfertility and infertility in the bitch. The normal canine uterus contains a commensal micro biota; however, disruption of microbial balance or impaired uterine immune function can predispose to inflammation. Unlike metritis, which involves all uterine layers, endometritis is limited to the endometrium and commonly occurs postpartum or following mating. Postpartum endometritis may persist as a clinical or subclinical condition, whereas postmating endometritis results from uterine contamination during breeding combined with inadequate uterine clearance. Persistent inflammation creates a hostile uterine environment that negatively affects sperm survival, fertilization, embryo development, and implantation, ultimately reducing conception rates and litter size. Preventive strategies focus on hygienic breeding and whelping management, although evidence-based non-antimicrobial therapies remain limited. Compared with other domestic species, research on endometritis in the bitch is sparse, highlighting the need for further investigation into its pathogenesis, diagnosis, and targeted treatment strategies.

Canine endometritis: Importance and classification

In the 1950s, researchers noted that acute endometritis in bitches was commonly detected during the initial stage of the luteal phase (Dow, 1959); however, it was subsequently recognized that this condition was often associated with mating or the post-oestrous period, which had not been identified then. The correlation between endometritis and compromised fertility has been recognized only recently (García Mitacek et al., 2017; Gifford et al., 2014). In their latest commentary, England et al. (2021) examined the suspected aetiopathology of postmating and postoestrous endometritis in the bitch and discussed the potential development and maintenance of chronic endometritis. The paucity of comprehensive investigative studies limits accurate determination of the true significance of endometritis as a causative factor in

infertility. Furthermore, the proposed classifications (1) mating-induced endometritis, (2) post-oestrous endometritis, and (3) chronic endometritis remain insufficiently characterized and require further critical evaluation and consensus within the literature. In contrast, postpartum uterine infections typically involve inflammation extending through all layers of the uterine wall; therefore, the condition is more appropriately termed metritis. Postpartum metritis is commonly associated with systemic clinical manifestations, including varying degrees of septicaemia and endotoxaemia, reflecting its potential severity. Although the overall incidence is relatively low, predisposing factors include prolonged parturition, dystocia, retention of foetal membranes, and suboptimal hygienic conditions during the whelping period. Notably, there is a significant gap in the literature regarding the long-term uterine sequelae and the potential persistence of infection or inflammation beyond the acute phase of metritis. Further longitudinal and histopathological studies are warranted to elucidate the chronic reproductive consequences of postpartum uterine infection.

	Prevalence	**Time point**	**Clinical sign**	**Diagnosis**	**Life threatening**
Bitch	Mating-induced endometritis largely relates to age changes, with up to 40% of older bitches having endometrial hyperplasia	Post breeding and Post-partum	Post breeding there is delayed uterine fluid clearance and there may be a post mating vaginal discharge	Persistence of uterine fluid Detected with ultrasound, increased PMNs on endometrial sampling	No

Clinical Signs

Parameter	**Details**
Earliest Sign	Purulent, fetid vulvar discharge (reddish-brown/chocolate; pus mixed with blood)

Parameter	Details
Systemic Signs	Fever, depression, lethargy, dehydration, inappetence
Additional Signs	Tachycardia, panting, vomiting, diarrhea, agalactia
Uterine Changes	Persistent enlarged uterus (delayed involution)
Neonatal Effects	Restlessness, persistent crying due to toxin ingestion via milk; may require hand-feeding
Clinical Suspicion	Any postpartum dam with systemic illness + abnormal vaginal discharge

Pathogenesis

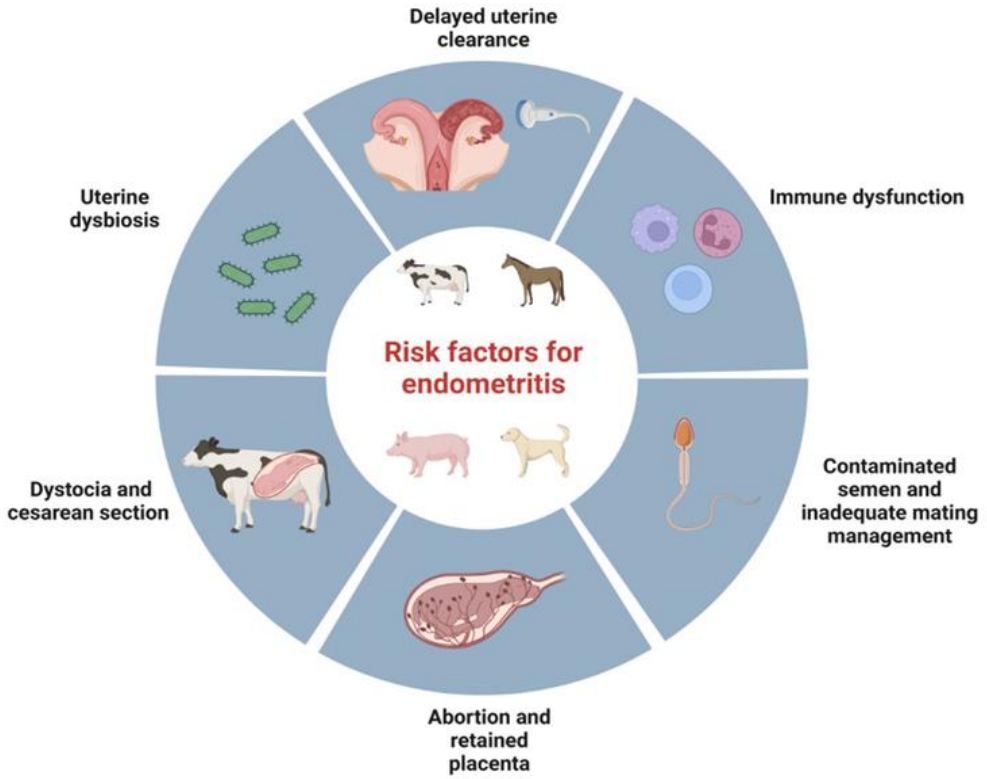
The precise pathogenesis of mating-induced endometritis in the bitch remains incompletely elucidated. However, England et al. (2021) recently proposed a mechanistic hypothesis, particularly in bitches exhibiting concurrent endometrial hyperplasia. They suggested that impaired uterine clearance following breeding may result from partial mechanical obstruction to intrauterine fluid drainage combined with diminished myometrial contractility. This delayed elimination of post-breeding uterine fluid may promote prolonged inflammatory responses, characterized by increased polymorphonuclear leukocyte (PMN) infiltration. Such alterations in the uterine environment were associated with reduced fertility, leading the authors to propose that persistence of inflammatory exudate and cellular infiltration may create a hostile intrauterine milieu that adversely affects embryonic survival at the time of uterine entry. Within the same conceptual framework, England et al. (2021) revisited earlier observations by Dow (1959) and incorporated findings from García Mitacek et al. (2017) and Praderio et al. (2019), suggesting that bacterial contamination of the uterus may also occur via the open cervix during oestrus in the absence of mating. This hypothesis supports the view that both mating-induced endometritis and post-oestrous endometritis may develop in the bitch through overlapping yet potentially distinct pathogenic mechanisms.

Chronic endometritis in the bitch is likely to develop when inflammatory processes initiated during either mating-induced or post-oestrous endometritis fail to resolve and persist throughout the luteal phase and subsequent anoestrus. Prolonged uterine inflammation under sustained progesterone influence may predispose to incomplete immune clearance and continued endometrial pathology.Similarly, bacterial contamination of the uterus during the postpartum period—particularly in cases progressing to metritis—may not always resolve entirely. In such instances, residual endometrial inflammation may remain following clinical recovery, potentially establishing a chronic inflammatory state. This mechanism would be consistent with patterns of persistent uterine infection described in other domestic species.

Despite these plausible pathways, there is currently insufficient evidence quantifying the proportion of chronic endometritis cases that originate from unresolved mating-induced endometritis, post-oestrous endometritis, or postpartum metritis. The absence of longitudinal studies tracking the progression from acute to chronic uterine inflammation represents a significant gap in understanding the epidemiology and long-term reproductive consequences of this condition.

Bacteria	Cow	Mare	Sow	Bitch
Escherichia coli	+	+++	+++	++
Streptococcus sp.	+	+++	+++	++
Staphylococcus sp.	++	++	++	++
Trueperella sp.	+++	–	++	–
Proteus sp.	+	+	+	–
Klebsiella sp.	+	+	++	–
Corynebacterium spp	+	(+)	+	(+)
Pseudomonas spp.	+	+	+	–
Chlamydia spp.	+	+	+	–

(Comparative aspects of bacteria involved in endometritis in domestic animal species (+++ great importance and frequently isolated, ++ moderate involvement, + and (+) rare involvement)



(Schematic presentation of common risk factors associated with endometritis in domestic animals)

Diagnosis

Bitches affected by mating-induced endometritis predominantly present with subfertility, characterized either by failure to conceive or by a reduction in litter size (England, Burgess et al., 2012; Fontaine et al., 2009; Mir et al., 2013). Although the number of available studies is limited and sample sizes remain small, the findings reported to date are noteworthy and warrant further investigation. For instance, Mir et al. (2013) reported that among 14 recently mated bitches that failed to establish pregnancy, histopathological evaluation confirmed the presence of endometritis in four cases (28%). These observations suggest that post-mating uterine inflammation may represent an under recognized contributor to impaired fertility in the bitch.

A subset of bitches diagnosed with mating-induced endometritis concurrently exhibit endometrial hyperplasia (Gifford et al., 2014). This condition can be identified through ultrasonographic evaluation of the uterus, which allows assessment of endometrial thickness and structural alterations (Moxon et al., 2016). In such cases, indirect evidence of endometritis may also be obtained via ultrasonography by demonstrating delayed post-mating intrauterine fluid clearance. More detailed investigations have further documented reduced uterine contractility and increased polymorphonuclear leukocyte (PMN) accumulation following transcervical uterine sampling (England, Burgess et al., 2012; England, Moxon et al., 2012), supporting the presence of an inflammatory response. Definitive diagnosis of both mating-induced and post-oestrous endometritis requires histopathological confirmation through uterine biopsy (Gifford et al., 2014; Mir et al., 2013). Chronic endometritis, characterized by persistence of inflammation throughout the luteal phase and anoestrus into the subsequent oestrous cycle, may be suspected when ultrasonography reveals sustained accumulation of uterine luminal fluid (Lyman et al., 2018). Additionally, chronic inflammatory changes have been identified ultrasonographically in some bitches following post-whelping metritis, suggesting persistence of postpartum uterine infection analogous to that described in other domestic species.

Laboratory Findings	Parameter	Findings
Hematology	Early Stage	May be normal
	Leukogram	Neutrophilia with left shift (may be degenerative)
	Severe Cases	Neutropenia (sepsis)
	Leukocytosis	Marked neutrophilic leukocytosis
	RBC	Anemia of chronic disease

Laboratory Findings	Parameter	Findings
	Changes	
Biochemistry	Serum Proteins	Hyperproteinemia (hypoalbuminemia + hyperglobulinemia)
	Glucose	Hypoglycemia (suggestive of sepsis)
	Renal Parameters	Increased BUN, creatinine
	Coagulation	Coagulopathy in severe septic cases

Differential Diagnoses	Characteristic Features
Normal Lochia	Non-purulent, non-malodorous
Subinvolution of Placental Sites	Hemorrhagic, non-purulent, non-septic discharge
Vaginitis/Cystitis/Urethritis	Scant discharge
Coagulopathy	Prolonged hemorrhagic discharge
Uterine Torsion	Acute abdomen, rapid deterioration
Uterine Rupture/Peritonitis	Acute abdomen, confirmed via ultrasound

Prevention

At present, there is a lack of published evidence outlining validated strategies for the prevention or reduction of endometritis in the bitch. Nevertheless, several theoretical preventive approaches may be considered. Implementation of strict hygienic measures prior to natural mating such as cleansing of the vulva and penis may reduce ascending bacterial contamination. Similarly, the use of artificial insemination techniques under controlled sanitary conditions, along with semen extenders containing appropriate antimicrobial agents, may minimize microbial introduction into the uterine lumen and thereby potentially decrease the incidence of mating-induced endometritis**.** Although exclusion of bitches diagnosed with endometrial hyperplasia from breeding programs could theoretically reduce susceptibility to uterine inflammation, it should be recognized that endometritis has also been documented in unmated bitches. This observation suggests that mechanisms independent of copulation, including spontaneous cervical relaxation and ascending contamination during oestrus, may also contribute to uterine infection.

Therapeutically, it is plausible that administration of ecbolic agents to enhance uterine clearance, anti-inflammatory drugs to modulate the inflammatory response, and immunomodulatory therapies to optimize uterine defence mechanisms could mitigate the effects of bacterial contamination and seminal components. Comparable treatment strategies have been explored in other species, such as the mare, and may warrant investigation in the bitch. With respect to postpartum uterine health, preventive measures should emphasize maintenance of optimal hygiene during whelping, minimization of prolonged or traumatic parturition, and prompt clinical management of retained foetal membranes. Such interventions are likely to reduce bacterial contamination at parturition and consequently lower the incidence of postpartum metritis and secondary endometritis. Future prospective and controlled studies are required to evaluate the efficacy of these preventive and therapeutic strategies and to establish evidence-based guidelines for clinical practice.

Therapy

Research investigating therapeutic strategies for mating-induced endometritis in the bitch remains limited. However, a small number of studies have reported that short-term administration of systemic antimicrobials following mating may be associated with improved conception rates (England, Burgess et al., 2012; England, Moxon et al., 2012; England et al., 2021). The proposed mechanism underlying this apparent “fertility rescue” is based on the anatomical and physiological dynamics of sperm transport. In the absence of pre-existing uterine inflammation at the time of breeding, spermatozoa are believed to be temporarily stored within the distal utero-tubal junction and the uterine tube, where they are relatively protected from the inflammatory processes occurring within the uterine lumen. Under this hypothesis, endometritis develops subsequent to sperm deposition and predominantly affects the uterine environment rather than the oviductal reservoir. Consequently, viable spermatozoa remain available for fertilization. Provided that the inflammatory process is resolved before the arrival of early embryos into the uterine lumen, implantation and maintenance of pregnancy may proceed successfully. The clinical response to antimicrobial therapy in these cases has also contributed to clarifying the likely involvement of bacterial contamination in the pathogenesis of mating-induced endometritis.

Future investigations should prioritize non-antimicrobial therapeutic approaches, particularly in light of concerns regarding antimicrobial stewardship. Strategies aimed at enhancing uterine clearance such as the administration of ecbolic agents may theoretically facilitate removal of inflammatory exudate and retained intrauterine fluid. Nevertheless, robust evidence demonstrating clinical efficacy of such interventions in the bitch is currently lacking. Exploration of alternative agents with bacteriostatic properties, as well as

immunomodulatory therapies, may also represent promising avenues for research.

Another therapeutic concept that has been discussed anecdotally involves the use of progesterone receptor antagonists. By inhibiting progesterone activity, these agents may counteract progesterone-mediated endometrial proliferation and glandular hyperplasia, potentially restoring a more physiologically normal uterine environment. However, controlled studies evaluating their effectiveness in the management of endometritis in the bitch are not yet available. Data regarding the treatment of chronic endometritis are particularly scarce. In one reported case, a combination of pre-breeding systemic antimicrobial therapy, oxytocin administration, and uterine lavage successfully eliminated persistent intrauterine fluid accumulation. Repeated post-breeding treatments were associated with subsequent establishment of pregnancy (Lyman et al., 2018). Beyond isolated reports, however, evidence-based treatment protocols for chronic endometritis in the bitch remain to be established.

Treatment	Category	Details
Initial Stabilization	Fluid Therapy	IV fluids for stabilization
	Antibiotics	Broad-spectrum IV pending culture
Antibiotics (Safe in Nursing Dams)	Cephalexin	20 mg/kg PO
	Amoxicillin–Clavulanic Acid	12.5–13.75 mg/kg PO
Uterine Evacuation	Prostaglandin F2α	0.10–0.20 mg/kg SC
	Oxytocin	5–20 IU IM (effective <48 hrs postpartum)
	Ergonovine	0.2 mg/15 kg IM (caution: uterine rupture risk)
Surgical	Ovario hysterectomy	If medical therapy fails or breeding not desired

Conclusion

Canine endometritis is an important cause of infertility in breeding bitches, commonly associated with bacterial infection during progesterone-dominant diestrus. Progesterone suppresses uterine immunity and promotes glandular secretions, predisposing the endometrium to persistent inflammation. Clinical signs are often subtle, including repeat breeding or mild vulvar discharge, making diagnosis challenging. Accurate detection requires

ultrasonography, cytology, and culture with sensitivity testing to differentiate it from pyometra and related uterine disorders. Targeted antimicrobial therapy combined with ecbolic agents improves uterine clearance. Early diagnosis and appropriate management are essential to restore fertility and prevent recurrence.

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