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Role of Bacterial Vaginosis in Female Infertility: Chronic Endometritis and Pelvic Inflammatory Disease

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KEYWORDS

Bacterial Vaginosis; Infertility; Chronic Endometritis; Pelvic Inflammatory Disease; Vaginal Microbiome..

ABSTRACT:

Bacterial Vaginosis (BV) is the most prevalent vaginal dysbiosis among women of reproductive age and is increasingly recognized as a contributor to adverse reproductive outcomes, including infertility. Characterized by depletion of protective Lactobacillus species and overgrowth of anaerobic bacteria, BV disrupts vaginal homeostasis and facilitates ascension of pathogenic organisms into the upper genital tract. Chronic Endometritis (CE) and Pelvic Inflammatory Disease (PID), frequently associated with BV- related microbiota, represent key pathological intermediates linking vaginal dysbiosis to impaired fertility. This review synthesizes current evidence on the relationships between BV, CE, PID, and infertility, emphasizing microbiological, inflammatory, and immunological mechanisms. Diagnostic approaches, treatment strategies, and implications for natural conception and assisted reproductive technologies are discussed. Early identification and management of BV and related upper genital tract infections may represent a critical component of preconception care to optimize fertility outcomes.

1.Introduction

The vaginal microbiome plays a fundamental role in maintaining female reproductive health. In most healthy reproductive-aged women, the vaginal ecosystem is dominated by Lactobacillus species, particularly Lactobacillus crispatus, L. gasseri, L. jensenii, and L. iners. These organisms contribute to vaginal defense through lactic acid production, maintenance of low vaginal pH, secretion of antimicrobial compounds, and modulation of mucosal immunity. Disruption of this protective environment predisposes women to vaginal dysbiosis and ascending genital tract infections. [1,3,15] Bacterial vaginosis represents a polymicrobial condition characterized by a reduction in Lactobacillus dominance and an increase in anaerobic bacteria such as Gardnerella vaginalis, Atopobium vaginae, Megasphaera spp., Prevotella spp., and Sneathia spp. BV is frequently asymptomatic, yet it has been consistently associated with increased susceptibility to sexually transmitted infections (STIs), pelvic inflammatory disease, adverse pregnancy outcomes, and infertility. Emerging evidence suggests that BV-related dysbiosis may extend beyond the vagina, influencing endometrial health and tubal integrity. [1,3]. [1,3,14]This review examines contemporary literature exploring the mechanistic and clinical links between BV, chronic endometritis, PID, and infertility, highlighting diagnostic challenges and therapeutic implications.

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2.Bacterial Vaginosis

2.1 Diagnosis and Treatment

BV is commonly diagnosed in clinical practice using the Amsel criteria, which include homogeneous vaginal discharge, elevated vaginal pH (>4.5), presence of clue cells, and a positive amine ("whiff") test. In research settings, the Nugent scoring system remains the gold standard, quantifying bacterial morphotypes on Gram-stained vaginal smears. Molecular diagnostic assays targeting BV-associated bacteria have gained prominence, offering improved sensitivity and reproducibility. [2,25]

Current treatment guidelines recommend antimicrobial therapy for symptomatic BV, most commonly using metronidazole or clindamycin. Newer agents such as secnidazole provide single-dose oral therapy and improved adherence. Despite effective initial treatment, recurrence rates remain high, prompting interest in adjunctive probiotic strategies. Clinical trials have demonstrated that vaginal administration of Lactobacillus crispatus following antibiotic therapy significantly reduces BV recurrence, underscoring the importance of microbiome restoration. [2,24]

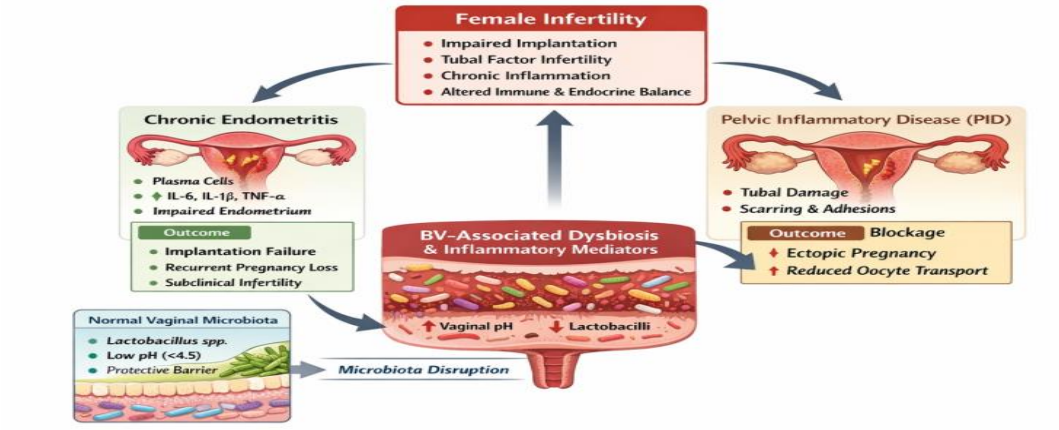
2.2 Bacterial Vaginosis and Infertility

Multiple observational and meta-analytic studies demonstrate a higher prevalence of BV among infertile women, particularly those with tubal factor infertility. BV-associated inflammation is believed to impair fertility through several mechanisms: [12,21]

1. **Inflammatory and Immune Dysregulation:** BV-related bacteria stimulate production of pro-inflammatory cytokines (IL-1β, IL-6, IL-8, TNF-α), leading to mucosal inflammation that may impair sperm function, embryo implantation, and endometrial receptivity. [3,14,27]
2. **Disruption of Cervical Mucus Barrier:** Enzymes such as sialidases and mucinases degrade cervical mucus, facilitating microbial ascension and reducing sperm protection.
3. **Increased STI Susceptibility:** BV significantly increases the risk of *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, *Mycoplasma genitalium*, and viral STIs, all of which are strongly linked to infertility.
4. **Altered Vaginal and Endometrial Microbiota:** Studies in assisted reproduction show that women with Lactobacillus-dominated vaginal and endometrial microbiota have higher implantation and pregnancy rates compared with those exhibiting dysbiosis.

While BV does not consistently reduce live birth rates in assisted reproductive technologies, its association with tubal damage and implantation failure highlights its clinical relevance in infertility evaluation.

Figure 1: Bacterial Vaginosis and Its Role in Female Infertility: Interconnections with Chronic Endometritis and Pelvic Inflammatory Disease



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Note: Schematic illustration depicting the role of Bacterial Vaginosis (BV) in female infertility BV: Bacterial Vaginosis, PID: Pelvic inflammatory disease, IL: Interleukin, TNF-α: Tumor necrosis factor-alpha, Normal vaginal microbiota is dominated by *Lactobacillus* spp., which maintain low vaginal pH and mucosal defense, Chronic inflammation and tubal pathology are major mechanisms linking BV to infertility.

2.3 Chronic Endometritis and Pelvic Inflammatory Disease

Diagnosis and Microbiology

PID and endometritis result from ascending infection of the upper genital tract and frequently coexist. Acute presentations may include pelvic pain, cervical motion tenderness, and abnormal bleeding, whereas chronic endometritis is often asymptomatic or presents with subtle symptoms such as spotting or pelvic discomfort.

Histopathological identification of endometrial stromal plasma cells remains the most reliable diagnostic criterion for CE. Molecular studies reveal that BV-associated anaerobes, along with facultative pathogens such as Escherichia coli, Enterococcus faecalis, Mycoplasma spp., and Chlamydia trachomatis, are commonly detected in CE. [9,10]

Table 1. Latest Evidence - Based Pharmacological Management of BV, Chronic Endometritis and PID with Reproductive Implications

Condition	Drug / Regimen	Mechanism of Action	Route & Duration	Role in Infertility Prevention	References
Bacterial Vaginosis (BV)	**Metronidazole**	Anaerobic antimicrobial targeting BV bacteria	Oral 500 mg bid × 7 d or vaginal gel × 5 d	Standard first-line to reduce BV and ascending spread	Abbe et al., 2023; CDC STI Guidelines
	Clindamycin	Protein synthesis inhibitor, anaerobic coverage	Vaginal cream × 7 d or oral	Alternative first-line; useful in biofilm	Abbe et al., 2023; CDC STI Guidelines
	Secnidazole (single-dose)	Nitroimidazole with longer half-life	Oral single dose	Alternative regimen with similar cure rates	TOL-463 phase data context
	Probiotics / Prebiotics (adjunct)	Microbiome restoration	Oral/Vaginal adjunct	Adjunct to prevent recurrence	Abbe et al., 2023; Int J RSM 2024
Chronic Endometritis	**Doxycycline**	Broad-spectrum antibiotic	Oral 100 mg bid × 14 d	Reduces chronic inflammation, improves implantation	Di Gennaro et al., 2025

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	Azithromycin	Macrolide with intracellular coverage	Oral (clinician-specific regimen)	For resistant or atypical pathogens	Di Gennaro et al., 2025
	Culture-guided therapy	Targeted antimicrobial	As indicated	Tailors to AMR profiles	Di Gennaro et al., 2025
Pelvic Inflammatory Disease (PID)	**Ceftriaxone + Doxycycline + Metronidazole**	Broad gram-neg/anaerobe coverage	Parenteral then oral to complete 14 d	Prevents tubal scarring and infertility	CDC STI Guidelines
	Alternative parenteral (e.g., Clinda + Gentamicin)	Broad spectrum including anaerobes	IV with oral step-down	For severe or complicated PID	CDC STI Guidelines
Recurrent / Resistant Infections	**Adjunct probiotics + extended/metronidazole**	Microbiota modulation	Oral / Vaginal	Reduces recurrence	Abbe et al., 2023; Int J RSM 2024

Notes: BV: Bacterial Vaginosis**,** PID: Pelvic inflammatory disease**,** NSAIDs: Non-steroidal anti-inflammatory drugs**,** Combination antimicrobial therapy is recommended to prevent chronic inflammation and infertility**,** Probiotics should be used as adjuncts, not as monotherapy.

2.4 Endometritis, Pelvic Inflammatory Disease, and Infertility

PID and CE are well-established risk factors for infertility, particularly tubal factor infertility. Repeated or subclinical infections lead to chronic inflammation, fibrosis, and scarring of the fallopian tubes, impairing ovum transport and increasing ectopic pregnancy risk. [18,19]

In women undergoing assisted reproduction, CE is highly prevalent among those with unexplained infertility, recurrent implantation failure, and recurrent pregnancy loss. Treatment of CE with targeted antibiotic regimens has been shown to significantly improve implantation, clinical pregnancy, and live birth rates, reinforcing the causal relationship between endometrial inflammation and infertility.

2.5 Preconception Management and Fertility Optimization

Given the diagnostic challenges and asymptomatic nature of BV and CE, a low threshold for screening is recommended in women with infertility, prior STIs, recurrent pregnancy loss, or repeated IVF failure. Preconception evaluation should include assessment of vaginal and endometrial health, with prompt antimicrobial treatment when infection is confirmed. Emerging strategies integrating microbiome-based diagnostics, probiotics, and personalized antimicrobial therapy may further improve reproductive outcomes. Although routine treatment of asymptomatic BV remains controversial, its potential role in preventing upper genital tract infection warrants further investigation. [7,13,2]

3 Conclusion

Bacterial Vaginosis, chronic endometritis, and pelvic inflammatory disease represent interconnected disorders along a continuum of reproductive tract dysbiosis and inflammation. Through immune activation, microbial ascension, and tissue remodeling, these conditions contribute significantly to female infertility. While current evidence supports aggressive management of symptomatic disease, large prospective studies are needed to clarify the benefits of screening and treating asymptomatic infections. Integrating vaginal and endometrial microbiome assessment into infertility workups may offer new avenues for prevention and treatment, ultimately improving reproductive outcomes.

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