e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(2); 2272-2276

Original Research Article

# Role of Vaginal and Uterine Microbiome in the Pathogenesis and Recurrence of Pelvic Inflammatory Disease

**Anamika Ranjan[1], Sunita Singh[2], Sangeeta Sinha[3]**

[1]Senior Resident, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India

[2]Assistant Professor, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India

[3]Professor, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India

**Received: 24-12-2025 / Revised: 23-01-2026 / Accepted: 25-02-2026**

**Corresponding Author: Sunita Singh**

**Conflict of interest: Nil**

**Abstract:**

**Background:** Pelvic inflammatory disease (PID) is a major cause of chronic pelvic pain, infertility, ectopic pregnancy, and recurrent reproductive tract morbidity. Emerging evidence suggests that disturbances in the vaginal and uterine microbiome may influence susceptibility, severity, and recurrence of PID.

**Objective:** To evaluate the role of vaginal and uterine microbiome patterns in the pathogenesis and recurrence of PID.

**Methods:** A 12-month prospective observational study was carried out in the obstetrics and gynecology department of a tertiary care facility. One hundred women with PID diagnoses were included in the study. Molecular microbiome profiling and culture were used to investigate endometrial/uterine tissues and vaginal swabs. Patients were monitored for recurrence for a full year after receiving conventional treatment. The chi-square test, t-test, and logistic regression were used to evaluate relationships between microbiome makeup, inflammatory markers, and recurrence.

**Results:** Mixed anaerobic dysbiosis was observed in 38% of women, Gardnerella-rich flora in 26%, and lactobacillus-dominant vaginal microbiota in 24%. Forty-four percent had uterine dysbiosis. During follow-up, 34% of women experienced PID recurrence, which was considerably more common in those with non-Lactobacillus vaginal profiles than in those with Lactobacillus-dominant profiles (31.6% vs. 10.0%, p=0.006). Recurrence was independently linked to uterine dysbiosis (adjusted OR 3.74, 95% CI 1.52–9.18, p=0.004). In dysbiotic groups, elevated inflammatory markers were more common.

**Conclusion:** Vaginal and uterine microbiome imbalance is strongly associated with PID pathogenesis and recurrence. Microbiome-targeted strategies may improve long-term outcomes.

**Keywords:** Vaginal Microbiome, Uterine Microbiome, Pathogens, Recurrence, Anaerobic Dysbiosis, Microbiome-Targeted Strategies.

**DOI:** 10.25258/ijcpr.18.2.372



## Introduction

The endometrium, fallopian tubes, ovaries, and surrounding pelvic structures are all affected by pelvic inflammatory disease (PID), an ascending infection of the upper female genital system. Because it is linked to infertility, ectopic pregnancy, persistent pelvic pain, tubo-ovarian abscess, and repeated hospitalization, it continues to be a significant public health issue. Sexually transmitted infections including Neisseria gonorrhoeae and Chlamydia trachomatis have historically been connected to PID. Nonetheless, mounting data indicates that PID is frequently polymicrobial and may entail a more extensive ecological disruption of the microbiome of the lower and upper vaginal tracts. Lactobacillus species, which produce lactic acid to maintain low vaginal pH and prevent pathogen colonization, frequently dominate the vaginal microbiome in healthy reproductive-age women [1].

Anaerobic organisms like Gardnerella, Prevotella, Atopobium, and Mobiluncus may proliferate when Lactobacillus dominance is lost, resulting in a dysbiotic state akin to bacterial vaginosis. Increased mucosal inflammation, compromised epithelial barrier integrity, and an increased risk of ascending genital tract infection have all been linked to such dysbiosis.

The idea that the uterine cavity is sterile has also been called into question by recent research.

Changes in this uterine microbiome may impact inflammatory reactions, implantation, fertility, and gynecologic disease states. Low-biomass microbial communities have been found within the endometrium. Persistent uterine dysbiosis in PID may be a factor in recurring symptoms, inadequate remission during treatment, and chronic endometritis [2].

Because recurrent inflammatory episodes raise the likelihood of tubal damage and chronic discomfort, recurrence of PID is clinically significant. Women may be more vulnerable to reinfection or relapse if standard antibiotic therapy eliminates dominant pathogens but fails to restore the protective microbial balance. Thus, identifying women at risk for recurrence may be made easier by knowing microbiome characteristics both before and after therapy.

Prospective clinical data assessing the composition of the uterine and vaginal microbiomes in connection to PID recurrence are still scarce in many tertiary care settings, despite increasing global interest. To ascertain whether microbiome profiling might enhance risk management and stratification, local evidence is required [3].

The purpose of this prospective observational study was to evaluate the contribution of uterine and vaginal microbiome patterns to the pathophysiology and recurrence of PID in women who visited a tertiary care hospital during a 12-month period. Characterization of microbial community patterns, association with inflammatory indicators, and recurrence prediction after routine treatment were among the specific goals [4].

## Methods

**Study Design:** Prospective observational study.

**Study Duration:** 12 months.

**Setting:** Department of Obstetrics and Gynaecology, tertiary care teaching hospital.

**Sample Size:** 100 women diagnosed with PID.

### Inclusion Criteria

- Age 18–45 years
- Clinical diagnosis of PID
- Willing for microbiological sampling and follow-up

### Exclusion Criteria

- Pregnancy
- Recent gynecologic surgery
- Antibiotic use within previous 2 weeks
- Known malignancy
- Immunosuppressive disorders

**Statistical Analysis:** Chi-square test, t-test, and multivariate logistic regression.

## Results

**Table 1: Baseline Characteristics**

| Variable | Value |
|---|---|
| Mean age (years) | 28.9 ± 5.6 |
| Married women | 82 (82%) |
| Prior PID episode | 29 (29%) |
| Mean CRP (mg/L) | 14.8 ± 6.2 |
| Chronic pelvic pain at baseline | 40 (40%) |

**Table 2: Vaginal Microbiome and PID Severity**

| Microbiome Pattern | Moderate/Severe PID | Mild PID | p-value |
|---|---|---|---|
| Lactobacillus-dominant | 6 | 18 | |
| Mixed anaerobes | 29 | 9 | |
| Gardnerella-rich | 18 | 8 | |
| Low biomass | 5 | 7 | **0.002** |

**Table 3: Microbiome Pattern and Recurrence**

| Pattern | Recurrence | No Recurrence | p-value |
|---|---|---|---|
| Lactobacillus-dominant (n=24) | 2 | 22 | |
| Non-Lactobacillus (n=76) | 32 | 44 | **0.006** |

**Table 4: Multivariate Predictors of Recurrence**

| Variable | Adjusted OR | 95% CI | p-value |
|---|---|---|---|
| Uterine dysbiosis | 3.74 | 1.52–9.18 | 0.004 |
| Prior PID history | 2.63 | 1.08–6.42 | 0.031 |
| Mixed anaerobic flora | 2.94 | 1.20–7.18 | 0.018 |
| Age >30 years | 1.29 | 0.56–2.98 | 0.54 |

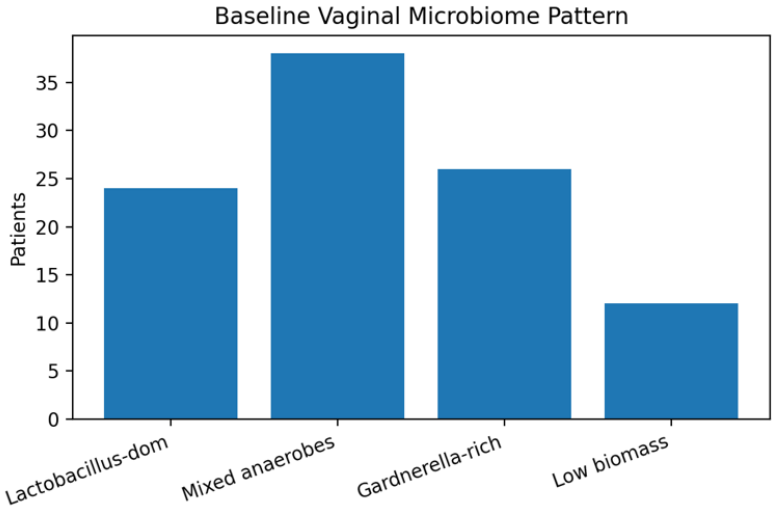


**Figure 1: Baseline vaginal microbiome pattern**

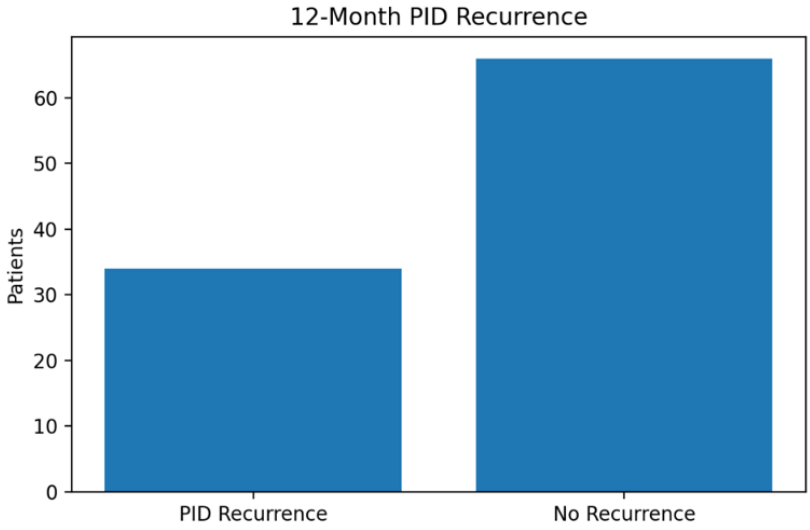


**Figure 2: 12-month PID recurrence**

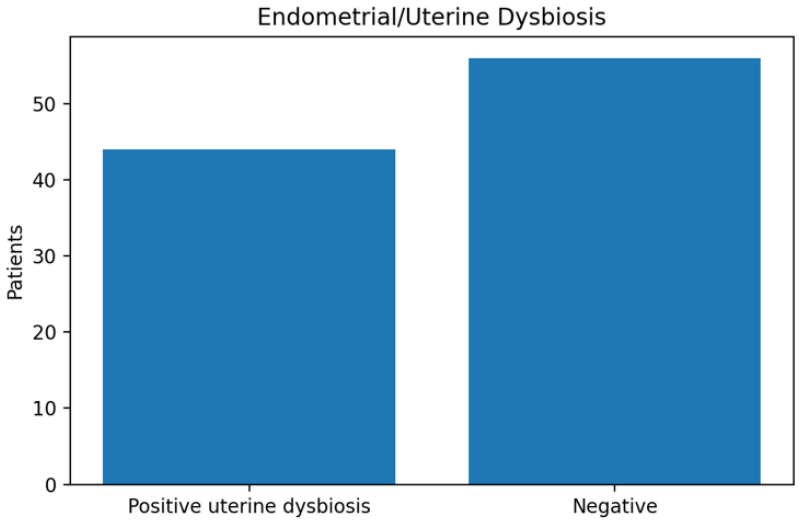


**Figure 3: Endometrial dysbiosis**

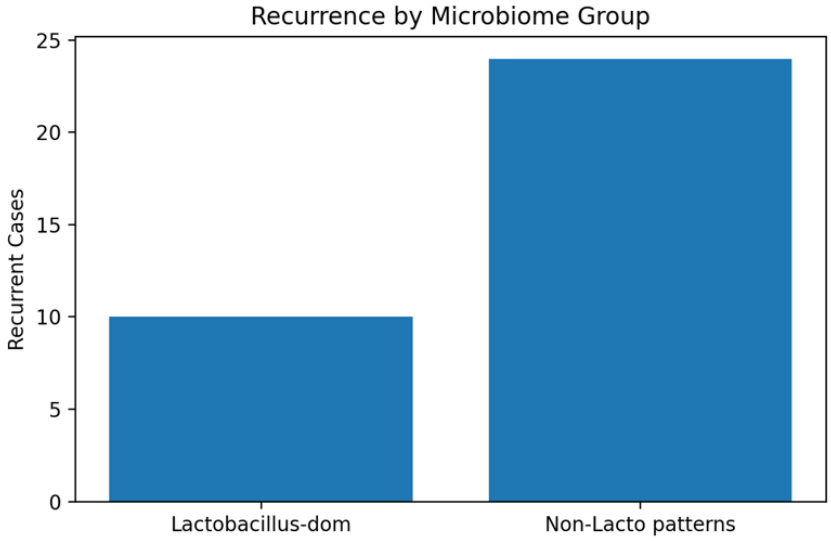


**Figure 4: Recurrence of microbiome group**

## Discussion

This prospective observational study shows a robust correlation between the clinical severity and recurrence of pelvic inflammatory disease and disruptions in the vaginal and uterine flora. Women who had non-Lactobacillus-dominant vaginal communities, especially mixed anaerobic dysbiosis, had considerably higher recurrence rates throughout follow-up and more severe disease at presentation. Furthermore, recurrence was independently predicted by uterine dysbiosis, indicating that a persisting microbial imbalance in the upper vaginal tract may be a factor in the partial resolution following standard therapy. Lactobacillus species, which produce lactic acid, hydrogen peroxide, and bacteriocin-like compounds that prevent pathogenic colonization, usually dominate the healthy vaginal ecology. These organisms support the integrity of the mucosal barrier and keep the pH of the vagina low [5].

The majority of the women in our group showed mixed anaerobic or Gardnerella-rich patterns, with only 25% showing Lactobacillus-dominant flora. This lends credence to the idea that microbial community disruption, rather than a single pathogen infection, is often the cause of PID. Greater disease severity was linked to mixed anaerobic ecosystems. Enzymes and bioactive metabolites that break down mucus, boost epithelial permeability, and intensify local inflammation can be produced by anaerobic organisms. Ascending spread into the fallopian tubes and endometrium is more likely when barrier defenses are compromised. This polymicrobial mechanism contributes to the explanation of why PID can emerge even in the absence of isolated classic sexually transmitted infections [6].

The connection between recurrence and microbiota profile was one of the study's key findings. Recurrence was significantly more prevalent in women with dysbiotic patterns, but it only happened in a small percentage of women with Lactobacillus-dominant flora. While failing to reestablish a stable protective microbiome, standard antibiotics may reduce dominant pathogens and alleviate symptoms. Thus, persistent dysbiosis may increase vulnerability to chronic low-grade endometritis, reinfection, or recurrence. Notably, there is an independent correlation between recurrence and uterine dysbiosis. Although the uterus was once thought to be sterile, low-biomass microbial populations have been found in the endometrium using contemporary genetic techniques. Even if symptoms heal, chronic inflammation may continue when these communities become unbalanced. Clinically, this could show up as recurrent pelvic pain, irregular discharge, irregular menstruation, or recurring episodes of PID [7].

Although infertility and implantation failure were not the main results of the current investigation, uterine microbiota alteration may also have a role. Recurrence was also predicted by prior PID history, which is in line with recognized cumulative risk. Previous occurrences may leave behind endometrial or tubal damage, decreased immunity, and a permanent ecological imbalance that increases the risk of infection in the future. As a result, women who have recurring illness may be a high-risk population that needs additional monitoring. These discoveries have important clinical ramifications. In order to restore the microbial health of the genital tract, PID management may need to move beyond short-term pathogen elimination. Probiotics targeted at restoring Lactobacillus, behavioral counseling, relationship management, post-treatment

microbiome evaluation in specific women, and customized antibiotic regimens covering anaerobes are some possible future therapies [8].

Simpler surrogate markers of dysbiosis may prove helpful in practice, even though routine microbiome sequencing is not yet widely accessible. There are a few restrictions to be aware of. This study had a moderate sample size and was conducted at a single location. Despite precautions, low-biomass uterine collection is susceptible to contamination, and microbiome techniques can differ in sensitivity. It's possible that some minor episodes were overlooked because recurrence was defined clinically. The assessment of temporal microbiome recovery was limited because not all subjects had serial post-treatment sequencing [9].

Notwithstanding these drawbacks, the study offers significant prospective data connecting the pathophysiology and recurrence of PID to uterine and vaginal dysbiosis. Future multicenter research should ascertain whether microbiome-guided therapies might lessen the burden of healthcare, infertility, persistent pelvic pain, and recurrent PID [10].

## Conclusion

According to this prospective observational study, pelvic inflammatory disease development and recurrence are significantly influenced by the vaginal and uterine microbiome. The majority of PID patients had non-Lactobacillus-dominant vaginal flora, such as mixed anaerobic and Gardnerella-rich patterns, which were linked to more severe illness. During follow-up, recurrence rates were considerably lower in women with protective Lactobacillus-dominant microbiota. Recurrent PID was found to be independently linked to uterine dysbiosis, suggesting that microbial imbalance in the upper vaginal tract may continue to cause inflammation even after symptoms have subsided. Recurrence risk was further elevated by prior PID experience, highlighting the cumulative reproductive health impact of recurrent episodes.

These results imply that some patients may not benefit from traditional treatment that only aims to eradicate the pathogen. Restoring a healthy genital tract microbiota through partner treatment, recurrence prevention counseling, optimal antibiotic selection, and even probiotic or microbiome-directed therapies where evidence supports use should be part of future care plans. Clinically, greater monitoring and focused dysbiosis assessment may be beneficial for women with severe or recurrent PID. It is necessary to do research on useful diagnostic instruments and individualized treatment plans.

In conclusion, the pathophysiology and recurrence of PID seem to be significantly influenced by microbiota imbalance. Incorporating microbiome science into gynaecological care may enhance long-term results, lower recurrent infections, and safeguard reproductive-age women's fertility.

## References


1. Han Y, Liu Z, Chen T. Role of Vaginal Microbiota Dysbiosis in Gynecological Diseases and the Potential Interventions. Front Microbiol. 2021;12(June):1–11.
2. Kobayashi H. Gut and reproductive tract microbiota : Insights into the pathogenesis of endometriosis ( Review ). Biomed REPORTS. 2023;19(43):1–12.
3. Vidal A, Kilian AY, Vinayahalingam V, Zagrapan B, Pape J, Karrer T, et al. The Role of Endometrial Microbiota in the Pathogenesis of Chronic Endometritis : A Systematic Review and Meta-Analysis. Biomedicines. 2026;14(871):1–23.
4. Zhang Y, Wei T, Zhao C, Zhang L. The Role of the Vaginal Microbiome in Gynecological Diseases : Mechanistic Insights and Emerging Interventions. Biology (Basel). 2026;15(432):1–29.
5. Iqbal Z, Ibnat A, Altaf H. Role of Vaginal Microbiota in Women ' s Reproductive Health and Diseases. J Multidiscip Stud. 2024;4(7):97–120.
6. Qiuhan Cheng, Siting Lv NY& JW. Microbial regulators of physiological and reproductive health in women of reproductive age : their local , proximal and distal regulatory roles. biofilms and microbiomes. 2025;11(207):1–24.
7. Kim S, Seo H, Rahim A, Tajdozian H. Characteristics of Vaginal Microbiome in Women with Pelvic Inflammatory Disease in Korea. Polish J Microbiol. 2021;70(3):345–57.
8. Kanagasudha BA, B DDV, C VAD. Role of Bacterial Vaginosis in Female Infertility : Chronic Endometritis and Pelvic Inflammatory Disease Journal of Chemical Health Risks. J Chem Heal Risks. 2026;16(2):2509–14.
9. Journal A, Applied OF, Science M. The role of cervicovaginal microbiome homeostasis in the early diagnosis and management of pelvic inflammatory disease. Am J Appl Med Sci. 2026;5101(3):214–25.
10. Gao X, Louwers Y V, Laven JSE, Schoenmakers S. Clinical Relevance of Vaginal and Endometrial Microbiome Investigation in Women with Repeated Implantation Failure and Recurrent Pregnancy Loss. Int J Mol Sci. 2024;25(622):1–23