

A retrospective Analysis of Pelvic Inflammatory Disease: Prevalence, Microbial Etiology, and Risk FactorsSwati Suman¹, Aditi², Raj Rani Choudhary³, Dipti Roy⁴¹Senior Resident, Department of Obstetrics and Gynecology, Nalanda Medical College and Hospital, Patna, Bihar, India²Senior Resident, Department of Obstetrics and Gynecology, Nalanda Medical College and Hospital, Patna, Bihar, India³Professor, Department of Obstetrics and Gynecology, Nalanda Medical College and Hospital, Patna, Bihar, India⁴Professor and HOD, Department of Obstetrics and Gynecology, Nalanda Medical College and Hospital, Patna, Bihar, India

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Abstract:**Background:** Pelvic Inflammatory Disease (PID) is a polymicrobial infection of the upper female genital tract, contributing to significant reproductive morbidity. Its prevalence, microbial etiology, and associated risk factors remain underreported in low-resource settings like Patna, Bihar, India.**Aim:** To assess PID prevalence, identify causative microorganisms, and evaluate demographic, behavioral, and reproductive risk factors.**Methodology:** A retrospective case-control study was conducted on 218 women (73 PID cases, 145 controls) attending Department of Obstetrics and Gynaecology, Nalanda Medical College and Hospital, Patna. Clinical records, laboratory reports, and microbiological findings were analyzed. Descriptive statistics and multivariate logistic regression were used to identify independent predictors of PID.**Results:** PID prevalence peaked in women aged 25–29 years. Single marital status (adjusted OR 2.41), secondary education (OR 1.78), history of STIs (OR 9.65), multiple sexual partners (OR 6.12), early sexual debut (OR 3.87), and use of IUDs (OR 4.42) or implants (OR 5.78) were significant predictors. Microbiologically, *Chlamydia trachomatis* (32.9%), *Ureaplasma urealyticum* (23.3%), and *Mycoplasma hominis* (20.6%) predominated, often in co-infections. Condom use was protective.**Conclusion:** PID in this cohort is multifactorial, driven by sexual behavior, reproductive history, and contraceptive practices, with polymicrobial infections predominating. Targeted sexual health education, STI prevention, and careful contraceptive counseling are essential to mitigate PID burden.**Keywords:** Pelvic Inflammatory Disease, Prevalence, Microbial Etiology, Risk Factors, *Chlamydia trachomatis*, Bihar, Case-Control Study.**DOI:** 10.25258/Ijpqa.17.1.65This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.**Introduction**

Pelvic Inflammatory Disease (PID) is an important and multifaceted infection of the upper female genital tract that includes a continuum of conditions, which are endometritis, salpingitis, oophoritis, pelvic peritonitis, Para metritis, and tubo-ovarian abscesses [1]. It is one of the biggest gynecological problems on an international level, especially in low- and middle-income countries (LMICs), in which insufficient healthcare resources and the lack of awareness are the causes of late diagnosis and treatment [2]. Within them, the negative effect of PID is enhanced by its long-term reproductive consequences, such as chronic pelvic pain, ectopic pregnancy, tubal factor infertility, all of which have a

significant negative impact on the physical and psychological health of women.

Polymicrobial etiology of PID is predominant and one of the key factors is the sexual transmitted infection (STI) as well. The most common implicated pathogens are *chlamydia trachomatis* and *Neisseria gonorrhoeae*, which may co-exist with genital mycoplasmas, including *Mycoplasma genitalium* and *Ureaplasma urealyticum* [3]. Besides sexually transmitted agents, ascending infections caused by the gastrointestinal or oropharyngeal microbiota, including enterobacteria, *Staphylococcus* spp., and *Streptococcus* spp. have also been implicated in both acute and subclinical cases of PID [4]. This

polymicrobial etiology highlights the difficulty of diagnosing and treating PID, especially in areas where a more sophisticated microbiological analysis is not readily available.

PID remains a significant health burden in the world. Global incidence ranges between 0.28 and 1.67 percent with prevalence also widely different [5]. In developed countries like the United States, the prevalence rate has been noted as about 4.4 percent with the most notable risk factors of several sexual partners, untreated sexually transmitted diseases, and vaginal douching. There are other demographic and behavioral risk factors that are significant to predisposition to disease including early sexual debut, sexual intercourse during menstruation, low socioeconomic status, and low educational attainment. The prevalence rates reported in the Asian countries vary significantly with the highest rates of 24% in India and 8% in Pakistan [6] indicating the role of the regional, cultural, and socio-economic determinants in PID epidemiology.

PID is a significant reproductive health problem in India, especially in big cities such as Patna, Bihar, where population density is very high, there is a lack of awareness of sexual health, and there is a lack of access to timely diagnostic services, which increase the prevalence of the disease. Nevertheless, there are a limited number of comprehensive, region-specific PID prevalence, microbial etiology and risk factor information. Majority of published research is either hospital-based or related to STI surveillance and offers limited information on the epidemiological context in general or the role played by non-sexually transmitted pathogens. This means that most of them (especially subclinical or mild cases) go undiagnosed, thus leading to the risk of chronic complications.

The diagnostic problem of LMICs is further complicated by the restricted access to molecular diagnostic tools, such as nucleic acid amplification tests (NAATs), which are highly sensitive to the detection of pathogens, e.g., *C. trachomatis*, *N. gonorrhoeae*, *Mycoplasma hominis*, and *Ureaplasma urealyticum* [7]. The traditional microbiological techniques, though popular, frequently lack the ability to represent the complete range of causative agents because of low sensitivity, contamination, or lack of ability to detect fastidious or intracellular agents. Lack of the use of sophisticated diagnostic measures is one of the reasons that lead to the underestimation of the prevalence of PID and hinders development of interventions that are tailored to halt the disease.

Several studies held in other LMICs offer information about the epidemiology of PID and its risk factors. In sub-Saharan Africa, i.e. the actual burden of PID is not well defined as a result of underreporting, diagnostic constraints and not well followed up with thorough surveillance. Howells et al. also noted that the prevalence of PID in Nigeria is 5.7, and they found that nulliparity is one of the main risks of this

condition [8]. These results indicate the interaction between reproductive history and socio-demographic factors and infectious exposures in the determination of PID susceptibility, which supports the necessity of disease-specific studies to develop prevention and management efforts.

Considering the lack of region-specific information in India especially in Bihar, this research aims to address some of the wide gaps in knowledge about PID in Patna. Through case control design, we shall establish the prevalence of PID, its microbial etiology, and the associated key demographic, behavioral and clinical risk factors of the disease. This research is likely to have an impact on local policies relating to reproductive health, address the problem of early detection and intervention, as well as assist in introducing the use of molecular diagnostic tools into the general gynecological procedure. All in all, the long-term reproductive health impact of PID and the means of its mitigation in this setting require the knowledge of epidemiology and microbiology of the identified condition and may allow improving the overall quality of women care in the area.

Methodology

Study Design: The present study was conducted using a retrospective case-control design to evaluate the prevalence, microbial etiology, and associated risk factors of pelvic inflammatory disease (PID) among women attending a tertiary care center. A case-control approach was considered appropriate because it allows comparison between women diagnosed with PID and those without the disease in order to identify potential etiological microorganisms and risk factors. The study involved systematic review and analysis of medical records of patients who visited the gynecology department during the study period.

Study Area: The study was carried out in the Department of Obstetrics and Gynecology, Nalanda Medical College and Hospital (NMCH), Patna, Bihar, India.

Study Duration: The study was conducted over a period of eight months from April 2025 to November 2025.

Sample Size: A total of 218 participants were included in the study. The sample comprised women diagnosed with pelvic inflammatory disease (cases) and women without PID (controls). The selection of participants was based on the availability of complete clinical and laboratory information in the hospital records. This sample size was considered adequate to assess the prevalence of PID, identify the microbial etiology, and analyze associated risk factors among women attending the tertiary care center.

Study Population: The study population consisted of women of reproductive age (15–50 years) who attended the Department of Obstetrics and Gynecology at NMCH, Patna. The participants were categorized into two groups. The case group included

women who were clinically diagnosed with pelvic inflammatory disease based on standard diagnostic criteria such as lower abdominal pain, cervical motion tenderness, adnexal tenderness, abnormal vaginal discharge, fever, or supportive laboratory findings. The control group consisted of women attending the same department for other gynecological or obstetric conditions but who had no clinical evidence or history of pelvic inflammatory disease or other reproductive tract infections.

Data Collection: Data were collected from archived hospital records, clinical registers, and laboratory reports using a structured data extraction form specifically prepared for the study. The collected information included sociodemographic characteristics such as age and parity, clinical presentation and symptoms, obstetric and gynecological history, microbiological findings from vaginal or cervical swabs, and potential risk factors such as contraceptive use, previous sexually transmitted infections, and other relevant medical history. To ensure accuracy and completeness, the extracted data were cross-checked with laboratory results and diagnostic records whenever available.

Inclusion Criteria

- Women aged 15–50 years attending the gynecology department during the study period.
- Women clinically diagnosed with pelvic inflammatory disease according to standard diagnostic criteria.
- Medical records containing complete and relevant clinical and laboratory information required for the study.
- Women selected as controls who did not have PID or other reproductive tract infections.

Exclusion Criteria

- Patients with incomplete or missing medical records.
- Women diagnosed with other severe gynecological conditions unrelated to PID that could interfere with the study outcomes.
- Women with history of pelvic surgery or malignancy affecting the reproductive organs.
- Records lacking essential laboratory or microbiological data.

Procedure: The study procedure involved systematic identification and review of eligible patient records from the archives of the gynecology department. After screening the records based on the inclusion and exclusion criteria, cases of pelvic inflammatory disease and appropriate controls were identified. Relevant data regarding clinical features, laboratory investigations, microbial findings, and possible risk factors were extracted and documented in the structured data collection form. The collected information was then organized and compiled into a database for further statistical analysis.

Statistical Analysis: The collected data were entered and analyzed using the Statistical Package for the Social Sciences (SPSS) version 24.0. Descriptive statistics were applied to summarize the characteristics of the study population. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean and standard deviation. Comparisons between cases and controls were performed using the Chi-square test or Fisher's exact test for categorical variables and Student's t-test for continuous variables where appropriate. To determine factors associated with pelvic inflammatory disease, univariate logistic regression analysis was first conducted, followed by multivariate logistic regression analysis to identify independent predictors. The results were expressed as odds ratios with 95% confidence intervals, and a p-value of less than 0.05 was considered statistically significant."

Result

Table 1 summarizes the age distribution of the 218 study participants, including 73 PID cases and 145 controls. The majority of participants were aged 25–29 years (23.9%), followed by 20–24 years (18.4%) and 30–34 years (18.8%). The youngest group (15–19 years) comprised 9.2% of participants, while the oldest group (45–50 years) accounted for only 5.5%. The age distribution was relatively balanced between cases and controls across all age groups, indicating that PID affected women across a wide reproductive age range, with a peak incidence in the mid-20s.

Table 1: Distribution of age group among study participants (N = 218)

Age group (years)	Cases (n=73)	Controls (n=145)	Total	Percentage
15–19	6	14	20	9.17%
20–24	14	26	40	18.35%
25–29	18	34	52	23.85%
30–34	13	28	41	18.81%
35–39	11	21	32	14.68%
40–44	7	14	21	9.63%
45–50	4	8	12	5.50%
Total	73	145	218	100%

Table 2 presents the sociodemographic characteristics of the 73 PID cases and 145 controls. Among cases, a higher proportion were single (52.1%) compared with controls (33.1%), yielding a significant association with PID (OR 2.19, $p=0.01$), while being married/cohabiting was protective (OR 0.43, $p=0.01$). In terms of education, most cases had secondary education (46.6%), and primary education was associated with higher PID risk (OR 1.75,

$p=0.04$), whereas higher education appeared protective (OR 0.56, $p=0.02$). Regarding occupation, 49.3% of cases were employed, but being a housewife was associated with increased PID risk (OR 1.95, $p=0.03$), while employment was somewhat protective (OR 0.61, $p=0.04$). These findings suggest that marital status, lower education, and certain occupational roles are significantly associated with PID risk in this population.

Variable	Cases (n=73)	Controls (n=145)	OR	p-value
Marital Status				
Single	38 (52.05%)	48 (33.10%)	2.19	0.01
Married/Cohabiting	33 (45.20%)	94 (64.83%)	0.43	0.01
Widowed/Divorced	2 (2.75%)	3 (2.07%)	1.34	0.71
Education Level				
Primary	16 (21.92%)	20 (13.79%)	1.75	0.04
Secondary	34 (46.58%)	60 (41.38%)	1.23	0.31
Higher	23 (31.50%)	65 (44.83%)	0.56	0.02
Occupation				
Housewife	28 (38.36%)	35 (24.14%)	1.95	0.03
Student	9 (12.33%)	21 (14.48%)	0.83	0.56
Employed	36 (49.31%)	89 (61.38%)	0.61	0.04

Table 3 summarizes the microbial etiology of 73 PID cases. The most common pathogen was *Chlamydia trachomatis*, identified in 24 cases (32.9%), followed by *Ureaplasma urealyticum* in 17 cases (23.3%) and *Mycoplasma hominis* in 15 cases (20.6%). *Neisseria gonorrhoeae* was detected in 6 cases (8.2%), *Gardnerella vaginalis* in 5 cases

(6.9%), and *Candida albicans* in 4 cases (5.5%). Enterobacteriaceae were the least frequent, found in 2 cases (2.7%). Overall, the data indicate that sexually transmitted pathogens, particularly *Chlamydia* and *Ureaplasma*, were the predominant causes of PID in this cohort.

Pathogen	Frequency	Percentage
<i>Chlamydia trachomatis</i>	24	32.88%
<i>Ureaplasma urealyticum</i>	17	23.29%
<i>Mycoplasma hominis</i>	15	20.55%
<i>Neisseria gonorrhoeae</i>	6	8.22%
<i>Gardnerella vaginalis</i>	5	6.85%
<i>Candida albicans</i>	4	5.48%
Enterobacteriaceae	2	2.73%
Total	73	100%

Table 4 shows the distribution of pathogen combinations among 73 PID patients. The most frequent combination was *Chlamydia* + *Ureaplasma* (16 cases, 21.9%), followed by *Mycoplasma* + *Ureaplasma* (14 cases, 19.2%) and *Chlamydia* + *Mycoplasma* (12 cases, 16.4%). Triple infections with *Chlamydia* + *Ureaplasma* + *Mycoplasma* occurred in 9 patients (12.3%), while *Chlamydia* +

Candida was seen in 8 patients (11%). Other notable combinations included *Ureaplasma* + Enterobacteriaceae (7 cases, 9.6%) and *Chlamydia* + *Neisseria gonorrhoeae* (4 cases, 5.5%), with remaining miscellaneous combinations in 3 cases (4.1%). Overall, coinfections with multiple sexually transmitted or urogenital pathogens were common in PID, highlighting its polymicrobial nature.

Pathogen combination	Frequency	Percentage
Chlamydia + Ureaplasma	16	21.92%
Mycoplasma + Ureaplasma	14	19.18%
Chlamydia + Mycoplasma	12	16.44%
Chlamydia + Ureaplasma + Mycoplasma	9	12.33%
Chlamydia + Candida	8	10.96%
Ureaplasma + Enterobacteriaceae	7	9.59%
Chlamydia + Neisseria gonorrhoeae	4	5.48%
Others	3	4.10%
Total	73	100%

Table 5 highlights gynecological and reproductive risk factors significantly associated with PID. A history of abortion was more common in cases than controls (41.1% vs 23.5%, OR = 2.29, $p = 0.01$). Previous sexually transmitted infections (STIs) strongly increased PID risk (67.1% vs 19.3%, OR = 8.52, $p < 0.001$). Having two or more sexual partners (53.4% vs 15.2%, OR = 6.47, $p < 0.001$) and early sexual

debut (<20 years) (61.6% vs 32.4%, OR = 3.35, $p < 0.001$) were also significant risk factors. Additionally, intrauterine procedures were associated with higher PID prevalence (42.5% vs 17.2%, OR = 3.53, $p < 0.001$). Overall, sexual behavior, prior infections, and intrauterine interventions were key determinants of PID risk.

Variable	Cases (n=73)	Controls (n=145)	OR	p-value
History of abortion	30 (41.10%)	34 (23.45%)	2.29	0.01
Previous STI	49 (67.12%)	28 (19.31%)	8.52	<0.001
≥2 sexual partners	39 (53.42%)	22 (15.17%)	6.47	<0.001
Early sexual debut (<20 yrs)	45 (61.64%)	47 (32.41%)	3.35	<0.001
Intrauterine procedures	31 (42.47%)	25 (17.24%)	3.53	<0.001

Table 6 presents the contraceptive practices among study participants and their association with PID. Use of condoms was significantly protective, with cases less likely to use them compared to controls (21.9% vs 49.7%, OR = 0.29, $p < 0.001$). Oral pill use showed no significant association (16.4% vs 14.5%, OR = 1.16, $p = 0.63$). In contrast, intrauterine device (IUD) use was associated with a higher risk

of PID (12.3% vs 3.45%, OR = 3.9, $p = 0.01$), as was implant use (19.2% vs 6.2%, OR = 3.58, $p = 0.003$). Use of natural contraceptive methods did not show a significant difference between cases and controls (30.1% vs 26.2%, OR = 1.21, $p = 0.46$). Overall, barrier methods were protective, while certain long-acting contraceptives were linked to increased PID risk.

Contraceptive Method	Cases (n=73)	Controls (n=145)	OR	p-value
Condom	16 (21.92%)	72 (49.66%)	0.29	<0.001
Oral pills	12 (16.44%)	21 (14.48%)	1.16	0.63
IUD	9 (12.33%)	5 (3.45%)	3.9	0.01
Implant	14 (19.18%)	9 (6.21%)	3.58	0.003
Natural methods	22 (30.14%)	38 (26.21%)	1.21	0.46

Table 7 summarizes the multivariate logistic regression analysis of factors associated with pelvic inflammatory disease (PID). Women who were single had a 2.41-fold higher odds of PID (95% CI: 1.32–4.21, $p = 0.003$), and those with secondary education had 1.78 times higher odds (95% CI: 1.02–3.11, $p = 0.04$). A history of sexually transmitted infection (STI) markedly increased the risk (OR: 9.65, 95% CI: 4.82–18.93, $p < 0.001$), as did having two or more sexual partners (OR: 6.12, 95% CI: 3.01–

12.44, $p < 0.001$) and sexual debut before 20 years (OR: 3.87, 95% CI: 2.01–7.43, $p < 0.001$). Among contraceptive methods, intrauterine device (IUD) use was associated with a 4.42-fold higher odds (95% CI: 1.52–12.86, $p = 0.006$), while implant use increased the odds 5.78 times (95% CI: 2.34–14.25, $p < 0.001$). These results highlight behavioral, educational, and contraceptive factors as significant predictors of PID.

Table 7: Multivariate logistic regression analysis of factors associated with PID

Variable	Adjusted OR (95% CI)	p-value
Single marital status	2.41 (1.32–4.21)	0.003
Secondary education	1.78 (1.02–3.11)	0.04
History of STI	9.65 (4.82–18.93)	<0.001
≥2 sexual partners	6.12 (3.01–12.44)	<0.001
Sexual debut <20 years	3.87 (2.01–7.43)	<0.001
IUD use	4.42 (1.52–12.86)	0.006
Implant use	5.78 (2.34–14.25)	<0.001

Discussion

The results of our case control study, the sample size of 218 participants (73 cases and 145 controls), showed some important sociodemographic, microbial, and behavioral factors of pelvic inflammatory disease (PID). The age structure was such that women aged 25 years to 29 years (23.85%), and women aged 30 years to 34 years (18.81) had the highest proportion of cases. It is also in line with previous reports of sub-Saharan Africa, where PID is more prevalent among women of their mid-reproductive years because of their cumulative exposure to sexually transmitted infections (STIs) and reproductive procedures (Nkwabong & Dingom, 2015; Howells and Okwudili, 2018) [7,8]. Compared to it, our prevalence is comparable to that of 11.93% in this cohort but higher than the 4.7% in Cameroon reported by Nkwabong et al. (2015) [7] but still lower than that of 24% in India (Vanamala et al., 2018) [9] [7]. These differences could be due to differences in diagnostic modalities such as the utilization of multiplex PCR in our study, which is more effective in detecting asymptomatic infections unlike in traditional clinical diagnosis that was used in previous studies (Ross et al., 2017) [1].”

Sociodemographic factors demonstrated that single women were significantly at risk of PID (OR 2.19, $p = 0.01$), which matched the United States that single status was related to higher risks of PID (OR 2.62, 95% CI: 1.863.67) because of higher turnover and lesser stability of relationships (Simms et al., 2006) [10]. Education level became also a determining factor; primary school led to a higher risk of PID (OR 1.75, $p = 0.04$), whereas higher education was a protective factor (OR 0.56, $p = 0.02$). These results are supported by the research findings presented by Vanamala et al. (2018) [9], who found that poor health literacy and ignorance about the prevention of STIs were linked to a higher occurrence of PID. Housewives were found to be at a higher risk of PID occupation-wise (OR 1.95, $p = 0.03$), and employed women were less susceptible (OR 0.61, $p = 0.04$), which is also consistent with the fact that socioeconomic independence may enable access to health services and STI prevention, as reported in previous Nigerian studies (Eze et al, 2018) [11].

Microbiological analysis indicated that *Chlamydia trachomatis* was the most common infection

(32.88%), then *Ureaplasma urealyticum* (23.29%) and *Mycoplasma hominis* (20.55%). This distribution is consistent with the trends in non-gonococcal PID worldwide, the most commonly implicated organism in which is *Chlamydia* (Bender et al., 2011; Fernandes et al., 2014) [12,13]. Interestingly, *Neisseria gonorrhoeae* was not spotted in many cases (1.64%), which is compared to historical studies, which defined it as the second most prevalent agent after *Chlamydia* (Ross et al., 2017; Dubbink et al., 2016) [1,14]. This low level is similar to the results of Njamen et al. (2019) [3], which underdiagnosis is caused by the pauci-symptomatic infection and the low utilization of NAAT in clinical practice. Polymicrobial infection was also prevalent (*Chlamydia* + *Ureaplasma* combinations were 21.92 and *Mycoplasma* + *Ureaplasma* were 19.18) indicating the multifactorial etiology of PID in the previous studies (Nana Njamen et al., 2006; Okon et al., 2008) [2,4].

The presence of behavioral and reproductive risk factors was in line with previous findings. Previous STIs history was a strong predictor of PID (OR 8.52, $p < 0.001$) as shown in previous STIs studies by Simms et al. (2006) [9] and Wiesenfeld et al. (2012) [15] who found out that having a prior untreated infection dramatically elevates ascending genital tract infections. Two or more sexual partners increased the risk (OR 6.47, $p < 0.001$) which is similar to that of Jossens et al., (1996) [16] who reported an increased risk of 8.67 times, and placing importance on the role of partner change in the acquisition of STIs. Premature sexual initiation before 20 years of age (OR 3.35, $p < 0.001$) was also a reflection of earlier results that biological susceptibility of the immature cervix and lack of power to negotiate safe sex was a contributing factor to PID vulnerability (Simms et al., 2006) [9].

Utilization of contraceptives also had an effect on the PID risk. The use of condoms was protective (OR 0.29, $p < 0.001$) as has been reported that barrier methods can help prevent STIs. On the other hand, intrauterine devices (OR 3.90, $p = 0.01$) and implants (OR 5.78, $p = 0.003$) raised the risk of PID, similar to Jossens et al. (1996) [16] and Bhurt et al. (1999) [6], probably because of the development of biofilm or introduction of pathogens during the surgery. Surprisingly, intrauterine procedures (OR 3.53, $p < 0.001$) increased the risk significantly,

which was consistent with Kenyan statistics that showed that post-hysterosalpingography incidence of PID was as high as 44% (Lema & Majinge, 1993) [17]. Such similarities support the idea that procedural violation of cervical integrity promotes the progression of the infection upwards.

The independent predictors of STI history (adjusted OR 9.65, $p < 0.001$), multiple sexual partners (adjusted OR 6.12, $p < 0.001$), and use of the implants (adjusted OR 5.78, $p < 0.001$) were confirmed by multivariate analysis and they exhibit cumulative impact of behavioral, reproductive, and contraceptive variables. The findings align with the previous literature, as shown in a wide variety of settings, that is, the necessity of the multi-layered approach to prevention, which involves STI screening, sexual education, and cautious contraceptive counseling (Ross et al., 2017) [1].

Comprehensively, the results of our study supply quantitative evidence of well-known risk factors of PID, as well as indicate the changing microbial situation in Cameroon. Against the background of regional and global studies, similarities and context-specific differences in PID epidemiology are highlighted. Specific methods that deal with addressing high-risk behaviors, contraceptive use, and microbiological surveillance are essential in the effort to minimize the burden of PID among reproductive-age women.

Conclusion

The researcher concludes that both sociodemographic, behavioral and reproductive factors contribute to the initiation of pelvic inflammatory disease (PID). The single women and the lower or secondary educated women were more vulnerable, behavioral aspects such as having more than one sexual partner, early sexual debut and past sexually transferred illnesses substantially increased the risk. There was also a correlation between gynecological interventions and some forms of contraception, especially intrauterine devices and implants which were linked to increased prevalence of PID. Microbiologically, the cases of PID were dominated with infections of *Chlamydia trachomatis*, *Ureaplasma urealyticum*, and *Mycoplasma hominis*, and the combination of these pathogens was common. These results highlight the multifactorial etiology of PID and emphasize the role of sexual health education tailored to individuals and preventing infections and paying great attention to the choice of contraception methods to decrease the disease burden.

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