

Association Between Chronic PID and Infertility: A Prospective Study on Tubal and Endometrial Biomarkers (Obstetrics & Gynaecology)Anisha Buddhapriya¹, Anamika Ranjan², Rupam Sinha³¹Senior Resident, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India²Senior Resident, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India³Professor, Department of Obs and Gynae, Patna Medical College and Hospital, Patna, Bihar, India

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Conflict of interest: Nil

Abstract:**Background:** Chronic pelvic inflammatory disease (PID) is an important cause of female infertility through tubal damage, pelvic adhesions, and altered endometrial receptivity. Biomarkers of persistent inflammation may help identify women at higher risk of infertility.**Objective:** To evaluate the association between chronic PID and infertility with emphasis on tubal pathology and endometrial inflammatory biomarkers.**Methods:** Over the course of eight months, a prospective observational study was carried out in the obstetrics and gynecology department of a tertiary care facility. One hundred women were included who had radiological and clinical signs of persistent PID. Endometrial biomarker analysis (IL-6, TNF- α , CRP expression), transvaginal ultrasonography, hysterosalpingography/laparoscopic tubal assessment, infertility status, and detailed reproductive history were all carried out. For six months, the participants' conception results were monitored. The chi-square test, Student's t-test, and logistic regression were employed in the statistical study.**Results:** Infertility was either primary or secondary in 72% of patients. In 72% of cases, tubal anomalies were found, including bilateral tubal block in 42%. Infertile women had considerably higher levels of elevated TNF- α and IL-6 than fertile women ($p=0.011$ and $p=0.004$, respectively). During follow-up, conception rates were lower in women with bilateral tubal block (12% vs. 46%, $p=0.001$). Infertility was independently linked to persistent endometrial inflammation (adjusted OR 3.18, 95% CI 1.34–7.56, $p=0.008$).**Conclusion:** Chronic PID is strongly associated with infertility, particularly through tubal damage and persistent endometrial inflammation. Biomarker assessment may improve early risk stratification and guide fertility-focused management.**Keywords:** Chronic PID, Infertility, Endometrial Inflammation, Conception Rates, Bilateral Tubal Block.**DOI:** 10.25258/ijcpr.18.3.305

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Introduction

Infection and inflammation of the upper female genital system, including the uterus, fallopian tubes, ovaries, and adjacent pelvic tissues, are referred to as pelvic inflammatory disease (PID). A significant portion of women acquire chronic or subclinical PID, which is characterized by continuous low-grade inflammation, pelvic discomfort, irregular menstruation, dyspareunia, and reproductive dysfunction. Acute PID frequently manifests as pain, fever, and vaginal discharge. Chronic PID is still a significant gynecological issue, particularly in low- and middle-income nations where long-term consequences may be exacerbated by delayed diagnosis, recurring STDs, and restricted access to healthcare.

Infertility is one of the most dangerous side effects of long-term PID. Fallopian tube inflammation can result in hydrosalpinx, fibrosis, fimbrial damage, mucosal degradation, and partial or total tubal blockage [1].

Impaired ciliary activity and altered tubal motility can impede ovum transfer and fertilization even in cases where tubes are still patent. Prolonged pelvic adhesions can further impair fertility by distorting tubo-ovarian architecture. Recurrent episodes of PID are linked to an increasing risk of infertility. There is growing evidence that persistent PID may affect endometrial receptivity in addition to tubal factors. Cytokine signaling, implantation molecules, vascular remodeling, and the interface between the

embryo and the endometrium can all be affected by persistent inflammatory activity inside the endometrium. Tumor necrosis factor-alpha (TNF- α), interleukin-6 (IL-6), and C-reactive protein (CRP) are examples of biomarkers that have been investigated as markers of persistent inflammatory activity. Even in cases when traditional imaging is equivocal, elevated levels may detect women with mild endometrial dysfunction [2].

Significant psychological, social, and marital repercussions result from infertility, especially in cultures where having children is highly valued. Therefore, it is crucial to identify preventable causes as soon as possible. In many tertiary care settings, prospective data assessing tubal findings and endometrial biomarkers in women with chronic PID are still scarce, despite the established relationship between PID and infertility. Clinicians may be better able to forecast reproductive outcomes, counsel couples appropriately, and prioritize interventions like antibiotic optimization, hysteroscopic evaluation, tubal surgery, or assisted reproductive techniques if they are aware of the connection between inflammatory biomarkers and structural tubal damage [3].

The Department of Obstetrics and Gynecology conducted this prospective observational study over the course of eight months to evaluate the relationship between infertility and chronic PID in women who visited a tertiary care facility. Determining the prevalence of infertility, describing tubal pathology, assessing endometrial inflammatory biomarkers, and looking at short-term conception results were the specific goals [4].

Methods

Study Design: Prospective observational study.

Study Duration: 8 months.

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

Sample Size: 100 women diagnosed with chronic PID.

Inclusion Criteria

- Age 20–40 years
- Clinical features suggestive of chronic PID for >3 months
- Ultrasonographic or pelvic examination support for PID
- Willing to participate

Exclusion Criteria

- Known ovarian failure
- Male factor infertility as sole cause
- Genital tuberculosis already on treatment
- Pregnancy at enrollment
- Malignancy

Data Collected

- Age, parity, duration of infertility
- Menstrual and sexual history
- Pelvic examination
- HSG/laparoscopic tubal patency assessment
- Endometrial sampling for IL-6, TNF- α , CRP markers
- Follow-up for conception (6 months)

Statistical Analysis: $p < 0.05$ $p < 0.05$ $p < 0.05$.

Chi-square test, t-test, and multivariate logistic regression.

Results

Table 1: Baseline Clinical Characteristics

Variable	Value
Mean age (years)	29.6 \pm 4.8
Primary infertility	54 (54%)
Secondary infertility	18 (18%)
Mean infertility duration (years)	3.2 \pm 1.6
Chronic pelvic pain	62 (62%)

Table 2: Tubal Pathology and Infertility

Tubal Status	Infertile	Fertile	p-value
Normal tubes	14	14	
Unilateral block	22	8	
Bilateral block	36	6	0.001

Table 3: Endometrial Biomarkers by Fertility Status

Biomarker Raised	Infertile (n=72)	Fertile (n=28)	p-value
IL-6	46	8	0.004
TNF- α	41	7	0.011
CRP	30	6	0.049

Table 4: Predictors of Infertility (Multivariate Analysis)

Variable	Adjusted OR	95% CI	p-value
Bilateral tubal block	4.62	1.88–11.36	0.001
Raised IL-6	3.18	1.34–7.56	0.008
Duration of PID >2 years	2.41	1.02–5.66	0.043
Age >30 years	1.52	0.66–3.47	0.29

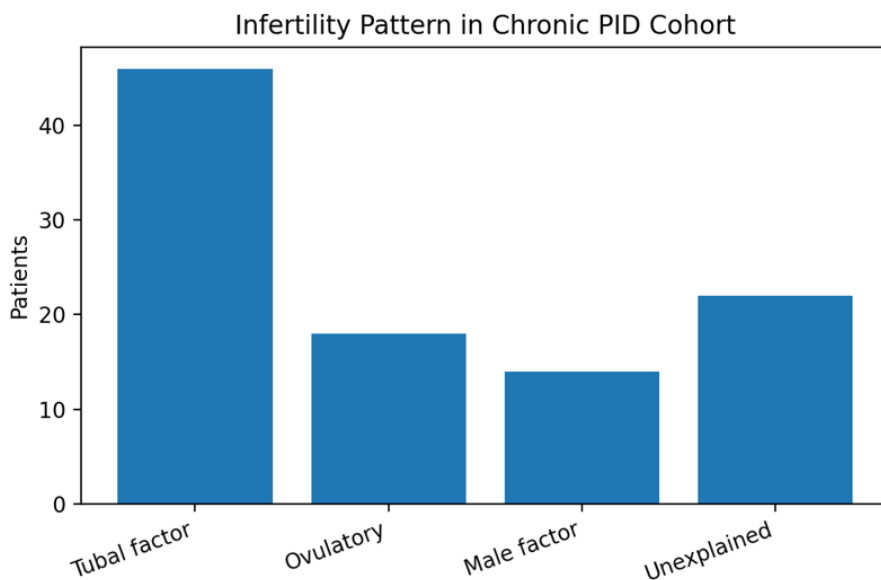


Figure 1: Infertility pattern in chronic PID cohort

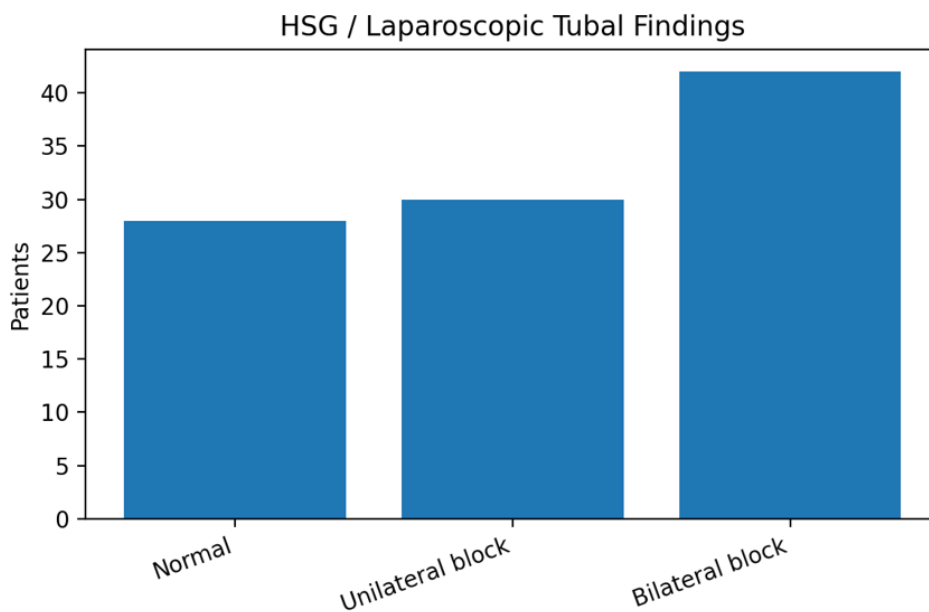


Figure 2: HSG/ Laparoscopic tubal findings

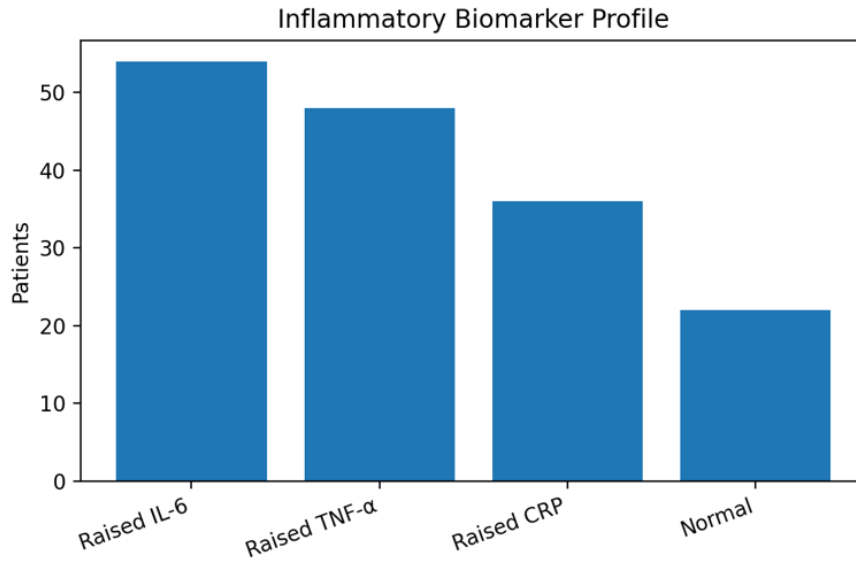


Figure 3: Inflammatory biomarker profile

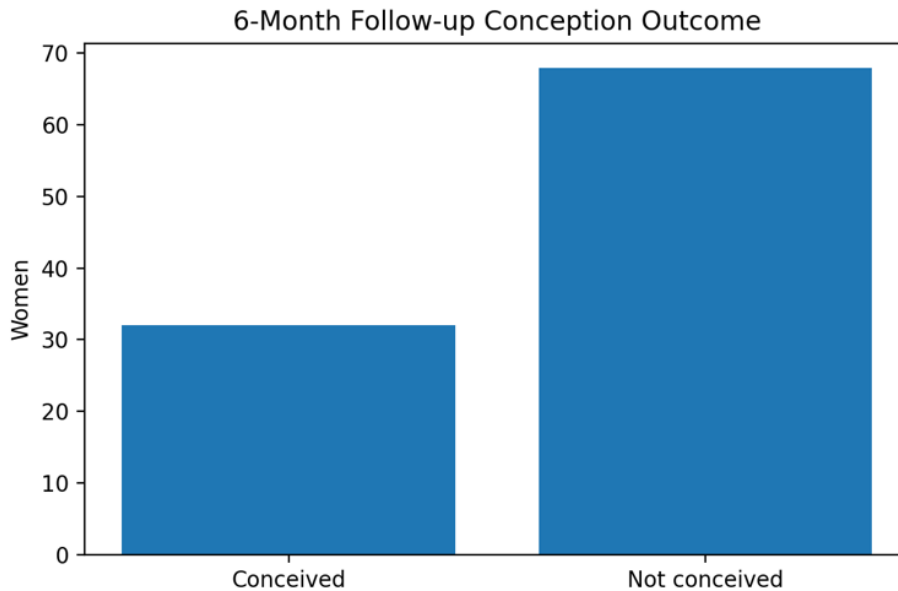


Figure 4: 6-month follow-up conception outcome

Discussion

This prospective observational study shows a robust correlation between infertility and chronic pelvic inflammatory illness, with both persistent endometrial inflammation and structural tubal abnormalities leading to poor reproductive outcomes. Nearly three-quarters of the 100 women in the study had infertility, highlighting the significant reproductive burden of chronic PID in gynecological practice. One important factor that has been identified as connecting infertility and persistent PID is tubal disease. Bilateral tubal block had the highest correlation with infertility, and almost two-thirds of subjects had aberrant tubal findings. This makes biological sense because

continuous or recurrent inflammation affects the sensitive tubal mucosa, kills ciliated epithelium, and encourages fibrosis [5].

Ovum pickup and sperm transport are seriously hampered once fimbrial architecture is altered or luminal blockage occurs. Subfertility and ectopic pregnancy can be made more likely by even partial damage. The clinical importance of tubal damage is further supported by the significant decrease in conception rates among women in our group who had bilateral block.

Crucially, the current study also emphasizes the significance of endometrial inflammatory indicators [6]. Infertile women were substantially more likely

to have elevated TNF- α and IL-6, and even after controlling for covariates, elevated IL-6 was still independently linked to infertility. This implies that diminished endometrial receptivity and tubal blockage are two ways that persistent PID may affect fertility. Angiogenesis, trophoblastic invasion, implantation signaling, and the synchronization of embryonic development with the implantation window can all be disrupted by cytokine imbalance [7].

These results are consistent with the current knowledge that tubal transport, ovulation, sperm function, and a receptive endometrium all depend on an intact reproductive tract for fertility. Because microscopic inflammation and altered endometrial function continue, women with chronic PID may not be able to conceive even when imaging shows tubal patency. Women with infertility that cannot be explained or who experience repeated implantation failure following seemingly normal imaging tests may benefit most from biomarker evaluation. Infertility was also linked to PID symptoms lasting longer than two years. This lends credence to the idea that cumulative reproductive harm results from delayed diagnosis and untreated chronic inflammation. Therefore, it is crucial to identify persistent pelvic pain, dyspareunia, irregular discharge, and menstruation abnormality as soon as possible. Preventable long-term consequences may be decreased by prompt gynecological assessment and public awareness [8].

Our cohort's six-month conception rate was modest; women with normal tubes and lower inflammatory marker burden conceived more frequently, suggesting that structural and biochemical recovery potential influences reproductive prognosis. Women with mild disease may benefit from targeted antimicrobial therapy, adhesiolysis, hysteroscopic evaluation, or ovulation optimization depending on coexisting factors. This study has practical implications. When evaluating infertility in women with suspected chronic PID, careful pelvic examination, and consideration of endometrial inflammation. Incorporating biomarker testing may help identify women who may need an early referral [9].

There are a few restrictions to be aware of. The study had a moderate sample size and was single-center. Standardized cutoffs have not been established universally, and biomarker testing may differ between laboratories. When possible, male factor infertility was ruled out; however, subtle mixed reasons cannot be completely ruled out. The six-month follow-up period may have underestimated the rates of eventual conception. Despite these drawbacks, the study offers significant prospective evidence that tubal injury and persistent endometrial inflammation are the two main routes by which chronic PID is closely associated with infertility.

Future multicenter research should evaluate if early anti-inflammatory or fertility-directed therapies improve outcomes and validate biomarker panels [10].

Conclusion

Among women using a tertiary care gynecology service, this prospective observational study discovered a strong correlation between infertility and chronic pelvic inflammatory illness. Tubal disease, especially bilateral tubal block, was substantially linked to lower conception rates, and infertility was very common in the population. These results validate that structural reproductive tract damage is mostly caused by persistent PID. Additionally, the study showed that infertile women were more likely to have persistent endometrial inflammation, as shown by increased biomarkers including TNF- α and IL-6. This implies that reduced endometrial receptivity, disturbed implantation biology, and tubal blockage may all contribute to infertility in chronic PID. Therefore, persistent microscopic inflammatory alterations may cause infertility in women with normal tubal imaging.

Clinically, our findings support a thorough evaluation of fertility in women with long-term PID, which includes tubal patency testing and, when possible, endometrial inflammatory assessment. To reduce long-term reproductive harm, early PID diagnosis and treatment, quick fertility referral, and prompt management of recurrent symptoms are crucial. While women with milder tubal illness may benefit from focused medicinal or minimally invasive therapies, those with severe tubal disease may benefit from assisted reproductive procedures. To sum up, persistent PID is a significant and perhaps avoidable cause of infertility. Assessing tubal status and endometrial biomarkers together may improve reproductive results, tailor treatment choices, and improve prognosis.

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