Research Article

A CROSS-SECTIONAL STUDY ON CLINICAL AND HISTOPATHOLOGICAL PROFILE AND SHORT TERM TREATMENT OUTCOME OF WOMEN WITH ENDOMETRIAL HYPERPLASIA

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ABSTRACT

Background: Endometrial hyperplasia[EH] is a precursor to endometrial carcinoma. Its clinical presentation, risk factors, and outcomes vary across populations, and data from Indian women remain limited. **Objective**: To study the demographic, clinical, histopathological profile of women with EH and to assess short term treatment outcomes. **Methods:** This cross-sectional study included 50 women with histopathologically confirmed endometrial hyperplasia attending the gynecology outpatient department at MAMC & Lok Nayak Hospital, New Delhi, between May 2023 and April 2024. Clinical history, risk factors, ultrasound findings, and histopathology were recorded. Patients were managed with oral medroxyprogesterone acetate (MPA), levonorgestrel-releasing intrauterine system (LNG-IUS), or total hysterectomy with bilateral salpingo-oophorectomy, depending on histology and clinical profile. Outcomes were assessed at 6 months. **Results:** The mean age of participants was 45.7 ± 8.9 years. Most patients were multiparous (84%) and overweight/obese (98%). The predominant symptoms were heavy menstrual bleeding (74%) and frequent cycles (48%). Co morbidities included diabetes (20%), hypertension (16%), and thyroid disorders (16%). Histopathology revealed benign hyperplasia in 82% and atypical hyperplasia in 18%. Atypical hyperplasia cases were significantly older (mean 51.3 vs. 44.4 years; p=0.034). Uterus size was significantly associated with atypical hyperplasia (p=0.026). At 6 months, 91.4% of women treated with MPA or LNG-IUS achieved complete histological regression. **Conclusion:** EH in Indian women predominantly affects multiparous, overweight, and perimenopausal women. Older age and increased uterine size were associated with atypical hyperplasia. Continuous oral progesterone and LNG-IUS were effective in achieving disease regression, underscoring the importance of early diagnosis and management.

KEYWORDS: Endometrial hyperplasia, premalignant, endometrial intraepithelial neoplasia, progesterone therapy, hysterectomy.

INTRODUCTION

Endometrial hyperplasia (EH) represents a spectrum of abnormal endometrial glandular proliferation with variable architectural complexity and cytologic atypia [increased gland-to-stroma ratio]. Historically classified by WHO into four categories (simple/complex, with/without atypia), the contemporary 2014 WHO system simplifies classification into two clinically actionable groups: hyperplasia without atypia and atypical hyperplasia/endometrial intraepithelial neoplasia (EIN). Atypical hyperplasia carrying up to a 28% risk of progression to endometrial carcinoma compared with <1.6% in non-atypical hyperplasia.[1,2]

Globally, endometrial cancer ranks as the sixth most common gynecological malignancy and the most common gynecologic malignancy in high-income countries and its incidence has been rising; in the United States recent estimates reported over 66,000 new cases and 13,000 deaths in 2023, with obesity and metabolic syndromes strongly implicated as a key driver of this trend.[3] Identifying and treating EH prevents malignant progression and reduces the burden of uterine cancer. The global burden of endometrial cancer, in developing nations undergoing rapid epidemiological transition, necessitates improved understanding of precursor lesions. In India, where healthcare access and awareness is limited, many women present late, and therefore characterization of clinical patterns of hyperplasia becomes more critical.

Risk factors of endometrial hyperplasia can be, modifiable (polycystic ovarian syndrome, obesity, diabetes mellitus, hypertension) and non-modifiable (early menarche, late menopause, genetic cause) usually due to unopposed estrogen exposure.[2,4] Identifying risk factors in populations not only helps clinicians tailor preventive strategies but also informs health policy for early detection programs. Several population studies and registries indicate growing incidence of both EH and endometrial carcinoma in multiple regions, attributed to aging populations, rising obesity, delayed childbearing, and metabolic co morbidities. The growing burden has economic and health systems implications.[5,6]

Clinically, it manifests as abnormal uterine bleeding (AUB), which includes heavy, irregular, or postmenopausal bleeding.[7] Earlier detection through evaluation of AUB, targeted imaging, and endometrial sampling (aspiration cytology or hysteroscopy guided biopsy) can permit conservative therapy and reduce surgical morbidity. Many epidemiologic studies are from high-income settings, and there are data gaps from low and middle-income countries where differing reproductive patterns, health seeking behaviors, and resource constraints may alter presentation and management.[5,6]

Management depends on histology: benign hyperplasia responds well to continuous oral or local progesterone therapy, while atypical hyperplasia often requires hysterectomy due to higher malignant potential.[8] In women who desire fertility, guidelines favor the levonorgestrel‑releasing intrauterine system (LNG‑IUS) as first line medical therapy for non‑atypical EH and a compelling option for fertility preserving management of atypical EH in selected cases.[5,6]

However, regional variations in risk factors and treatment responses warrant localized data. This study aimed to evaluate the clinical, demographic, and histopathological profile of women with endometrial hyperplasia, and to assess treatment outcomes over six months in a tertiary care teaching institution in India.

METHODS

This cross-sectional observational study was conducted in the Department of Obstetrics & Gynecology, Maulana Azad Medical College and Lok Nayak Hospital, New Delhi, between May 2023 and April 2024. After obtaining informed written consent, all patients with confirmed histopathology of endometrial hyperplasia attending gynecology outpatient department were included in the study. A detailed medical history and physical examination were conducted, and a proforma was filled out to record demographic information, including age, parity, education, religion, occupation, socioeconomic status, and place of residence, along with their chief complaints, co morbidities. The following information were documented: menstrual cycle details (age at menarche, cycle regularity, number of pads used per day, menopausal history, and post-menopausal bleeding), obstetric history, age at marriage, contraception history, any history of metabolic disorders, family history of gynecological cancers, personal history, history of substance abuse, use of exogenous hormones estrogen, and exercise habits.

A general physical examination, abdominal examination, as well as speculum and bimanual examinations, were performed to assess the size of the uterus and the condition of the cervix. Additionally, ultrasounds was conducted to measure endometrial thickness and to check for the presence of any polyps or growths. Transvaginal ultrasound (TVS) measured endometrial thickness, and endometrial aspiration cytology and PAP smear of cervix performed for all cases in OPD & Histopathology results were collected from the pathology department at MAMC.

Treatment was administered according to hospital protocols based on the latest guidelines for endometrial hyperplasia, following proper counseling of the patient. Most patients with benign hyperplasia received continuous oral MPA, 10–20 mg thrice /day, while others were provided with LNG-IUS, depending on the patient's profile and affordability (LNG-IUS is not available free of cost through government supply). Atypical hyperplasia cases underwent total abdominal hysterectomy with bilateral salpingo-oophorectomy

(TAH+BSO) after a pre-anesthesia evaluation and fitness check. The decision to perform a bilateral salpingo-oophorectomy varied based on the patient's age, whether they were premenopausal or postmenopausal. Treatment outcomes were assessed at six months through transvaginal ultrasound (TVS) and repeat endometrial biopsy to determine whether there was any reversal or persistence of hyperplasia. Data was analyzed to understand the association of risk factors with endometrial hyperplasia in patients. Treatment outcome was histological regression at 6 months.

Sample Size: Using a prevalence estimate of 3% for atypical hyperplasia, with 95% confidence and 5% absolute error, the calculated sample size was 45. Accounting for non-response, 50 women were included.

Statistical Analysis: Categorical variables were expressed as frequencies and percentages, continuous variables as mean ± SD or median with 25th and 75th percentiles (interquartile range) (IQR). The data normality was checked by using Shapiro-Wilk test. The following statistical tests were applied for the results: The association of the variables which were quantitative in nature was analyzed using independent t test. The association of the variables which were qualitative in nature was analyzed using Fisher's exact test as at least one cell had an expected value of less than 5. Multivariate logistic regression was used to find out independent significant risk factors of atypical hyperplasia. Univariate logistic regression was used to find out effect of treatment on outcome. For statistical significance, p value of less than 0.05 was considered statistically significant. The study setting is a tertiary referral center catering to a heterogeneous patient population from both urban and rural areas, thereby offering a representative sample of women from different socioeconomic strata. The data entry was done in the Microsoft EXCEL spreadsheet and the final analysis was done with the use of Statistical Package for Social Sciences (SPSS) software, IBM manufacturer, Chicago, USA, version 25.0.

RESULTS
This study analyzed 50 histopathologically confirmed cases of endometrial hyperplasia, focusing on the demographic, clinical, histopathological, and treatment profiles of the participants attended OPD of Department of Obstetrics & Gynecology, Maulana Azad Medical College and Lok Nayak Hospital, New Delhi in one year.

Table-1 depicts the sociodemographic profile and Clinical Characteristics of the study participants. Age of participants ranged from 30 to 75 years [mean 45.7±8.9 years]. Half (50%) were illiterate, and only 8% had graduate education. The majority were home makers (96%), and 4% working as skilled labor. Socioeconomic distribution showed 50% belonged to the upper-lower class, 20% to lower class.

Table-2 depicts the clinical parameters, Reproductive, Lifestyle, and clinical Examination details of the study participants. The most common complaint was heavy menstrual cycles (74%), and post-menopausal bleeding (18%). The mean age of menarche was 12.44 years, and the mean age of menopause was 48.9 years. Most were multiparous (84%), and the average age at first childbirth was 20 years. A significant proportion had a history of diabetes (20%) and hypertension (16%). Histopathological Examination (HPE) revealed 82% had benign hyperplasia, while 18% had atypical hyperplasia.

Table-3 depicts the investigations findings, Management and Follow-up of the study participants. The mean endometrial thickness on ultrasound was 9.29 mm. Atypical hyperplasia cases had a higher proportion of larger uterus sizes (6-10 weeks) compared to benign hyperplasia. The majority (64%) were treated with oral MPA, followed by hysterectomy (30%) and LNG-IUS (6%). Participants were followed at 6 weeks with clinical examination and repeat endometrial thickness by TVS and histopathology after 6 months. Follow up at 6 weeks, 70% were symptomatic, and at 6 months, 90.6% showed normal histopathology after treatment with MPA and 100% showed normal histopathology after insertion of LNG IUS. The hysterectomy group showed no further symptoms.

Table 4 depicts: Association of Demographic and Clinical Factors with Atypical and Benign Endometrial Hyperplasia. Compared to Benign Hyperplasia group, Atypical Hyperplasia group had comparable distribution of age: p value = 0.181, Mean ± SD age p value = 0.034, education:[p value = 0.319], occupation: [p value = 1]. and socioeconomic status:[p value = 0.429].

Compared to Benign Hyperplasia group, Atypical Hyperplasia group had comparable distribution of chief complaints: post menopausal bleeding [p value = 0.334], HMB [p value-0.214] irregular bleeding (p value = 0.665) and frequent cycles (p value = 0.142), menstrual history:[p value = 0.956] and menopause (p value = 0.093).

Table- 5 depicts -Association of Reproductive, Hormonal, Co morbidity, Familial, and Anthropometric Factors with Atypical and Benign Endometrial Hyperplasia among Participants. Compared to Benign Hyperplasia group, Atypical Hyperplasia group had comparable distribution of parity:(p value = 1), Mean ± SD age at first childbirth (p value = 0.454) and exogenous hormone intake: (p value = 0.331), co morbidity-- No significant past history (p value = 0.705), diabetes (p value = 0.358), hypertension (p value = 0.144), infertility (p value = 1), PCOS (p value = 0.456) and thyroid disorder (p value = 0.144) & BMI (p value = 0.542). Family history of cancer was nil in both groups.

Table-6 Depicts:-Association of Uterine and Endometrial Parameters, Associated Pathologies, and Treatment

Outcomes with Atypical and Benign Endometrial Hyperplasia among Participants. Compared to Benign Hyperplasia group, Atypical Hyperplasia group showed significantly different distribution of uterus size, (p = 0.026) and associated pathology: (p value = 0.476). Compared to the MIRENA group, the MPA group had a comparable distribution of HPE after 6 months: normal (MPA: 90.63% vs. MIRENA: 100%) and benign hyperplasia (9.38% vs. 0%, respectively) (p-value = 1)

Table-7 Depicts multivariate regression analysis, of the factors as independent risk factor of atypical hyperplasia.

On performing multivariate regression, none of the factors was an independent significant risk factor of atypical hyperplasia.

Table 8 depicts: Univariate logistic regression to assess effect of treatment on normal outcome. On performing univariate regression, treatment was not significantly affected outcome (p value=0.908).

Table 1: Distribution of Sociodemographic and Clinical Characteristics of the Study Participants.

AGE	N (%)
35 to 44 years	27 (54.0%)
45 to 54 years	16 (32.0%)
55 to 64 years	4 (8.0%)
65 to 74 years	3 (6.0%)
Mean age ± SD	45.68 ± 8.9
EDUCATION DISTRIBUTION	
Illiterate	25 (50.0%)
Primary School	6 (12.0%)
Middle School	5 (10.0%
Secondary School	5 (10.0%)
Senior Secondary school	5 (10.0%)
Graduate	4 (8.0%)
OCCUPATION	
Housewife	48 (96.0%)
Skilled worker	2 (4.0%)
SOCIOECONOMIC STATUS	
Upper middle	5 (10.0%)
Lower middle	10 (20.0%)
Upper lower	25 (50.0%)
Lower	10 (20.0%)
BMI	**N (%)**
Normal	1 (2.0%)
Overweight	27 (54.0%)
Obesity class I	18 (36.0%)
Obesity class II	4 (8.0%)
COEXISTING PATHOLOGY	
none	43 (86.0%)
Adenomyosis	6 (12.0%)
Polyp	1 (2.0%)

Table 2: Clinical, Reproductive, Lifestyle, and Examination Characteristics of the Study Participants.

CHIEF COMPLAINTS	N (%)
Heavy menstrual cycles	37 (74.0%)
Post menopausal bleeding	9 (18.0%)
Irregular bleeding	10 (20.0%)
Frequent cycles	24 (48.0%)
EXOGENOUS HORMONE INTAKE	**N (%)**
OCP use	1 (2.0%)
Tamoxifen	1 (2.0%)
HISTORY OF SMOKING	**3 (6.0%)**

Table 3: Distribution of Investigations, Management and Follow-up among study Participants.

MENSTRUAL HISTORY	N (%)
Age at Menarche	
<10 years	0 (0.0%)
10-11 years	4 (8.0%)
11-12 years	31 (62.0%)
12-13 years	6 (12.0%)
>13 years	9 (18.0%)
Mean age of Menarche ± SD	12.44 ± 1.03
Age at Menopause	
<45 years	1 (9.0%)
45-50 years	7 (63.6%)
>50 years	3 (27.2%)
Mean age of Menopause ± SD	48.9 ± 2.66
h/o Irregular cycles	5 (10.0%)
OBSTRETICAL HISTORY	
Nulliparous	2(4.0%)
Primipara	6(12.0%)
Multipara	42 (84.0%)
Mean age at first childbirth (years) ± SD	20.02 ± 2.53
Family history of cancer	0 (0.0%)
ASSOCIATED CO MORBIDITY	**N (%)**
Diabetes	10 (20.0%)
Hypertension	8 (16.0%)
Infertility	1 (2.0%)
PCOS	3 (6.0%)
Thyroid disorder	8 (16.0%)
EXERCISE	1 (2.0%)
PER ABDOMEN EXAMINATION OF PARTICIPANTS	
Uterus size on per abdomen	
Normal	36 (72.0%)
6 weeks	3 (6.0%)
8 weeks	2 (4.0%)
10 weeks	2 (4.0%)
12 weeks	5 (10.0%)
14 weeks	1 (2.0%)
18 weeks	1 (2%)

CERVICAL CYTOLOGY (LBC)	N(%)
NILM	50 (100.0%)
HPE DISTRIBUTION	
Benign hyperplasia	41 (82.0%)
Atypical hyperplasia	9 (18.0%)
ENDOMETRIAL THICKNESS ON USG	
Mean ± SD	9.29 ± 2.84 mm
Median (25—75th percentile)	8 (7.25-10.75) mm
Range	5-18 mm

TREATMENT	N (%)
MPA	32 (64.0%)
LNG-IUS	3 (6.0%)
TAH + BSO	15 (30.0%)
FOLLOW UP	
Symptomatic at 6 weeks post treatment	35(70.0%)
Symptomatic at 6 months post treatment	3 (8.5%)
HPE AT 6 MONTHS	
Normal	32 (91.4%)
Benign hyperplasia	3 (8.5%)
Atypical hyperplasia	0 (0.0%)

Table 4: Association of Demographic and Clinical Factors with Atypical and Benign Endometrial Hyperplasia among study participants.

AGE	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
35 to 44 years	3 (33.33%)	24 (58.54%)	27 (54%)	
45 to 54 years	3 (33.33%)	13 (31.71%)	16 (32%)	0.181*
55 to 64 years	2 (22.22%)	2 (4.88%)	4 (8%)	
65 to 74 years	1 (11.11%)	2 (4.88%)	3 (6%)	
Mean ± SD	51.33 ± 11.19	44.44 ± 7.98	45.68 ± 8.92	$0.034^{\dagger}$
Median(25th-75th percentile)	50(42-57)	42(39-49)	42.5(39-50)	**Independent t test, Fisher's exact test**
Range	39-74	35-67	35-74	

EDUCATION	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Illiterate	4 (44.44%)	21 (51.22%)	25 (50%)	
Primary school	0 (0%)	6 (14.63%)	6 (12%)	
Middle school	2 (22.22%)	3 (7.32%)	5 (10%)	
Secondary school	2 (22.22%)	3 (7.32%)	5 (10%)	0.319*
Senior secondary school	1 (11.11%)	4 (9.76%)	5 (10%)	
Graduate	0 (0%)	4 (9.76%)	4 (8%)	
Total	9 (100%)	41 (100%)	50 (100%)	
OCCUPATION	**ATYPICAL HYPERPLASIA(N=9)**	**BENIGN HYPERPLASIA(N=41)**	**TOTAL**	**P VALUE**
Housewife	9 (100%)	39 (95.12%)	48 (96%)	
Skilled worker	0 (0%)	2 (4.88%)	2 (4%)	1*
Total	9 (100%)	41 (100%)	50 (100%)	
SOCIOECONOMIC STATUS	**ATYPICAL HYPERPLASIA (N=9)**	**BENIGN HYPERPLASIA (N=41)**	**TOTAL**	**P VALUE**
Upper middle	0 (0%)	5 (12.20%)	5 (10%)	
Lower middle	1 (11.11%)	9 (21.95%)	10 (20%)	
Upper lower	7 (77.78%)	18 (43.90%)	25 (50%)	0.429*
Lower	1 (11.11%)	9 (21.95%)	10 (20%)	
Total	9 (100%)	41 (100%)	50 (100%)	

CHIEF COMPLAINTS	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Heavy menstrual cycles	5 (55.56%)	32 (78.05%)	37 (74%)	0.214*
Post-menopausal bleeding	3 (33.33%)	6 (14.63%)	9 (18%)	0.334*
Irregular bleeding	1 (11.11%)	9 (21.95%)	10 (20%)	0.665*
Frequent cycles	2 (22.22%)	22 (53.66%)	24 (48%)	0.142*

Table 5: -Association of Reproductive, Hormonal, Comorbid, Familial, and Anthropometric Factors with Atypical and Benign Endometrial Hyperplasia among Participants.

MENSTRUAL HISTORY	ATYPICAL HYPERPLASIA	BENIGN HYPERPLASIA	TOTAL	P VALUE
History of irregular cycles	0 (0%)	5 (12.20%)	5 (10%)	0.57*
Menopause	**4 (44.44%)**	**7 (17.07%)**	**11 (22%)**	**0.093***
Age of menarche (years)				
Mean ± SD	12.11 ± 0.78	12.51 ± 1.08	12.44 ± 1.03	
Median (25th-75th percentile)	12(12-12)	12(12-13)	12(12-13)	$0.296^{\dagger}$
Range	11-14	11-16	11-16	
Age of menopause (years)				

Mean ± SD	49 ± 4.69	48.86 ± 0.9	48.91 ± 2.66	0.956[†]
Median (25th-75th percentile)	51(48.75-51.25)	49(48-49.5)	49(48-50.5)	
Range	42-52	48-50	42-52	
OBSTETRIC HISTORY	**ATYPICAL HYPERPLASIA**	**BENIGN HYPERPLASIA**	**TOTAL**	**P VALUE**
Parity				
Nulliparous	0 (0%)	2 (4.88%)	2 (4%)	1[*]
Primipara	1 (11.11%)	5 (12.20%)	6 (12%)	
Multiparous	8 (88.89%)	34 (82.93%)	42 (84%)	
Age at first childbirth (years)				
Mean ± SD	19.44 ± 1.81	20.15 ± 2.67	20.02 ± 2.53	0.454[†]
Median(25th-75th percentile)	20(18-20)	20(18-22)	20(18-22)	
Range	17-22	16-26	16-26	

EXOGENOUS HORMONE INTAKE	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Nil	8 (88.89%)	40 (97.56%)	48 (96%)	0.331[*]
OCP	1 (11.11%)	0 (0%)	1 (2%)	
Tamoxifen	0 (0%)	1 (2.44%)	1 (2%)	
Total	9 (100%)	41 (100%)	50 (100%)	

CO MORBIDITY	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Not significant past history	5 (55.56%)	27 (65.85%)	32 (64%)	0.705[*]
Diabetes	3 (33.33%)	7 (17.07%)	10 (20%)	0.358[*]
Hypertension	3 (33.33%)	5 (12.20%)	8 (16%)	0.144[*]
Infertility	0 (0%)	1 (2.44%)	1 (2%)	1[*]
PCOS	1 (11.11%)	2 (4.88%)	3 (6%)	0.456[*]
Thyroid disorder	3 (33.33%)	5 (12.20%)	8 (16%)	0.144[*]
FAMILY HISTORY OF CANCER	**ATYPICAL HYPERPLASIA (N=9)**	**BENIGN HYPERPLASIA (N=41)**	**TOTAL**	**P VALUE**
No	9 (100%)	41 (100%)	50 (100%)	NA
Total	9 (100%)	41 (100%)	50 (100%)	

BODY MASS INDEX	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Normal	0 (0%)	1 (2.44%)	1 (2%)	0.542[*]
Overweight	4 (44.44%)	23 (56.10%)	27 (54%)	
Obesity class I	5 (55.56%)	13 (31.71%)	18 (36%)	
Obesity class II	0 (0%)	4 (9.76%)	4 (8%)	
Total	9 (100%)	41 (100%)	50 (100%)	

Table 6:-Association of Uterine and Endometrial Parameters, Associated Pathologies, and Treatment Outcomes with Atypical and Benign Endometrial Hyperplasia among Participants.

UTERUS SIZE	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Normal	5 (55.56%)	31 (75.61%)	36 (72%)	0.026[*]
6 weeks	1 (11.11%)	2 (4.88%)	3 (6%)	
8 weeks	0 (0%)	2 (4.88%)	2 (4%)	
10 weeks	2 (22.22%)	0 (0%)	2 (4%)	
12 weeks	0 (0%)	5 (12.20%)	5 (10%)	
14 weeks	1 (11.11%)	0 (0%)	1 (2%)	

18 weeks	0 (0%)	1 (2.44%)	1 (2%)	
Total	9 (100%)	41 (100%)	50 (100%)	

ASSOCIATED PATHOLOGY	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Nil	6 (66.67%)	37 (90.24%)	43 (86%)	0.100*
Adenomyosis	3 (33.33%)	3 (7.32%)	6 (12%)	
Polyp	0 (0%)	1 (2.44%)	1 (2%)	
Total	9 (100%)	41 (100%)	50 (100%)	
ENDOMETRIAL THICKNESS (MM)	ATYPICAL HYPERPLASIA (N=9)	BENIGN HYPERPLASIA (N=41)	TOTAL	P VALUE
Mean ± SD	10.33 ± 3.04	9.06 ± 2.78	9.29 ± 2.84	0.225†
Median (25th-75th percentile)	10(8-12)	8(7-10)	8(7.25-10.75)	
Range	6-15	5-18	5-18	

Table 7: Multivariate Regression Analysis, of the Factors as Independent Risk Factor of Atypical Hyperplasia.

Variables	Beta coefficient	Standard error	P value	Odds ratio	Odds ratio Lower bound (95% confidence interval)	Odds ratio Upper bound (95% confidence interval)
AGE (YEARS)	0.087	0.045	0.056	1.091	0.998	1.192
UTERUS SIZE						
Normal				1		
6 weeks	2.071	1.297	0.110	7.930	0.625	100.696
8 weeks	-0.360	1.987	0.856	0.697	0.014	34.273
10 weeks	5.431	5.304	0.306	228.287	0.007	7.46E+06
12 weeks	-1.135	1.533	0.459	0.321	0.016	6.493
14 weeks	6.106	12.662	0.630	448.686	7.48494E-09	2.68965E+13
18 weeks	1.721	2.239	0.442	5.593	0.069	450.538

HPE AFTER 6 MONTHS	MPA(N=32)	LNG-IUS(N=3)	TOTAL	P VALUE
Normal	29 (90.63%)	3 (100%)	32 (91.43%)	1*
Benign hyperplasia	3 (9.38%)	0 (0%)	3 (8.57%)	
Total	32 (100%)	3 (100%)	35 (100%)	

Table 8: Univariate logistic regression to assess effect of treatment on normal outcome.

Variable	Beta coefficient	Standard error	P value	Odds ratio	Odds ratio Lower bound (95%)	Odds ratio Upper bound (95%)
Treatment						
MPA				1.000		
LNG-IUS	0.186	2.157	0.908	1.204	0.051	28.484

DISCUSSION

This study reports the clinical, pathological profile and short term treatment outcomes of EH in a tertiary care hospital in Delhi. The findings of this study highlight the clinical spectrum of EH in an Indian cohort, where cultural, demographic, and socioeconomic factors differ from Western populations. Metabolic co morbidities such as obesity, diabetes, and hypertension emphasizes the overlap between lifestyle-related conditions and gynecological disease. The proportion of atypical hyperplasia (18%) in this study is within the range reported globally (10–25%), and underscores the need for careful evaluation when EH is suspected.[9]

In this study of 50 women with histopathologically confirmed EH, the mean age of participants was 45.7 years. The findings consistent with those reported by Farzaneh F. et al., [45.4 years]. and Sajjid et al. while confirming the established association between older age and atypical hyperplasia.[10,11] In the present study, 50% of the participants had no formal schooling, and 38% had completed secondary education or higher. A similar trend was observed in the study by Sajjid et al., where 60% of participants had no formal schooling, while 40% had received some form of education.[11] In the present study, 96% were homemakers and 2% were skilled workers which can be attributed to the fact that

individuals were from lower socioeconomic backgrounds. None of the studies has performed correlation between EH and occupation.

Population based studies and cancer registries have documented increases in the incidence of endometrial carcinoma in many, high-income countries, over the last two to three decades. This upward trend parallels the global obesity epidemic and changes in reproductive behavior (delayed childbearing), which together increase lifetime estrogen exposure and metabolic risk. EH sits at the interface between modifiable risk factors and preventable cancer: timely diagnosis and effective conservative therapy can prevent progression to cancer. EH is amenable to fertility sparing management in younger women, using efficacious medical options like LNG‑IUS, which have lower systemic side effects than oral progestins.[9] In the present study, the histopathological results showed that 82% of the cases had benign hyperplasia, while 18% had atypical hyperplasia. These findings are comparable to studies by: Farzaneh F et al. & Sajjid N et al.[10,11]

The leading presenting complaint was heavy menstrual bleeding(74%), and postmenopausal bleeding(18%). Similar findings were observed in the study by Sajjad N et al. [HMB- 61.1%], Farzaneh F et al. [82.5% AUB], and Munawar S et al. [PMB- 48%].[10,11,12] In the present study, 6% were occasional smokers contrary to a study by Rosen MW et al. [50%].[13] In the present study, 2 % had a history of infertility with history of ovulation induction, and majority of participants were multiparous (84%), comparable with studies by: Sajjid N et al.[11] The predominance of multiparous women contrasts with reports from high-income countries, where nulliparity and infertility are stronger contributors.

In the present study, 12.2% and 33.3% of participants were hypertensive in the benign and atypical hyperplasia group respectively which is comparable to study by Farzaneh F et al.[10] In the present study, 16% of the participants had thyroid disorder which is similar to study by Kothapalli D et al. [27%.] & study by Kumar N A et al. [23%].[14,15]

In the present study, the average age of menarche was found to be 12.44 years. In contrast, Farzaneh F et al. reported 7.1% of their participants experienced early menarche, before age 9year.[10] In the present study, the mean age of menopause was 48.91 ± 2.66 years, with 27.2% of participants experienced menopause after the age of 50. Which is comparable to, Farzaneh F et al.[13.6%] study.[10]

In the present study, all participants (100%) reported no family history of cancer. The lack of family history of cancer in all participants reflects regional differences, as Lynch syndrome–associated hyperplasia is more commonly reported in Western cohorts.

In the present study, 6% of participants had a history of polycystic ovary syndrome (PCOS). which is comparable to study by Farzaneh F et al. [4.3%], while Agarwal R et al., found that 24% had hyperplasia, may be due to their larger sample size.[10,16] In the present study, 23% of participants with benign hyperplasia and 44.4% with atypical hyperplasia were overweight, 41% of individuals with benign hyperplasia and 55% with atypical hyperplasia were obese which signifies the risk factor. In the present study, 2% of participants had a history of oral contraceptive use. Histopathology revealed 18% had atypical hyperplasia, which occurs at an older mean age and associated with increased uterine size. Medical management with continuous oral medroxyprogesterone acetate or LNG-IUS showed high regression rates at 6 months,

In the present study, 17.7% of participants in the benign hyperplasia group and 44.4% in the atypical hyperplasia group had attained menopause which depicts older age group has atypical hyperplasia.

Large cohort studies, including Ryu et al. (2022), show that tamoxifen exposure increases the risk of endometrial pathology including polyps and hyperplasia, and modestly increases endometrial cancer risk. This underlines the need for vigilance and prompt evaluation of AUB in women taking selective estrogen receptor modulators.[17] In the present study, 2% of participants had a history of tamoxifen use which is comparable to Lee M et al.[2.5%].[18] In the present study, 17.0% and 33.3% of participants had diabetes in benign and atypical hyperplasia groups respectively. This is similar to a study by, Farzaneh F et al.[10]

In the present study, the mean endometrial thickness in the benign hyperplasia group is 9.06 ± 2.78 mm, and in the atypical hyperplasia group, it is 10.33 ± 3.04 mm. In the study by Sharif S et al., endometrial thickness of >5 mm with AUB was associated with 5.83% of participants having benign hyperplasia and 2.5% having atypical hyperplasia.[19] In the present study, 2%cases had association of endometrial polyps, 12% of participants had adenomyosis which is high in comparison to study by Garg P et al. [3.9%].[20]

Since 2020, high‑quality trials and systematic reviews have strengthened evidence supporting LNG‑IUS for management of non‑atypical EH and as a fertility‑preserving option in selected atypical cases. Randomized trials and multicenter studies have reported regression rates exceeding 85–90% with LNG‑IUS, often outperforming oral progestins in both efficacy and tolerability.[21] These data have influenced recommendations by professional societies to prioritize intrauterine progestogen delivery when feasible. In the present study both oral progesterone and LNG-IUS are highly effective in inducing regression in benign hyperplasia[78% were given MPA, 90% achieved total regression, Mirena given to7%, 100% regression after 6

months]. The regression rates with progestins were consistent with the findings of Shen et al. [93% regression with LNG-IUS vs 66% with oral therapy], and Kim et al. [>90% complete regression with LNG-IUS] which reinforce that for non‑atypical EH, LNG‑IUS should be strongly considered where available and acceptable. And oral MPA remains a reasonable and accessible option in resource limited settings.[22,23,24] Hysterectomy done for 14% of participants in the benign hyperplasia group in view of older age and high risk of progression to cancer in the future, underscoring their utility as cost-effective options in low-resource environments. Patients with atypical hyperplasia showed a higher mean age and larger uterine size. In the present study all participants belonging to the atypical hyperplasia group underwent hysterectomy as per treatment protocol. The outcome is comparable with other studies [Shen Y et al.].[22]

CONCLUSION
The rising incidence of endometrial carcinoma and the preventable nature of many EH risk factors argue for integrated public health strategies like obesity prevention and management programs, diabetes and hypertension control, reproductive health education, and strengthening primary care pathways that ensure timely gynecological evaluation for AUB.

Endometrial hyperplasia commonly presents in multiparous, overweight, and perimenopausal patients, with AUB as the leading symptom. Older age and uterine enlargement were associated with atypical hyperplasia. In benign endometrial hyperplasia Continuous oral progesterone or LNG-IUS should be offered depending upon the availability, cost, and accessibility of these treatments as both provided high regression rates. Understanding of risk factors play a crucial role. Women should counseled to seek early medical care and be made aware of potential consequences of atypical hyperplasia, the benefits of surgical treatment [hysterectomy, with or without bilateral salpingo-oophorectomy], and potential alternatives if fertility preservation is a concern. The available treatment options for EH have demonstrated good efficacy in reducing the disease burden, managing symptoms, and preventing progression to cancer. Timely treatment and patient-centered care, including surgical and medical management, contribute to improved quality of life and reduced long-term health risks.

Conflict of interest. Nil.

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