International Journal of Medical and Pharmaceutical Research

Online ISSN-2958-3683 | Print ISSN-2958-3675
Frequency: Bi-Monthly
Available online on: https://ijmpr.in/

Original Article

Histopathological Spectrum of Endometrial Lesions with Correlation to Hormonal Status in Abnormal Uterine Bleeding

Dr Sucheta Yadav[1], Dr Babli[2], Mamta Kannaujia[3], Devendra Kumar[4*]

[1]*Assistant Professor, Department of Pathology, Government Medical College, Budaun*
[2]*Assistant Professor, Department of Pathology, Pt. Neki Ram Sharma Medical College, Bhiwani, Haryana*
[3]*Assistant Professor, Department of Pathology, Government Medical College, Budaun*
[4]*Associate Professor, Department of Community Medicine, Autonomous State Medical College, Firozabad*

Corresponding Author:

Devendra Kumar[4]

Associate Professor, Department of Community Medicine, Autonomous State Medical College, Firozabad, India.

Received: 28-09-2025
Accepted: 13-10-2025
Available online: 30-11-2025

ABSTRACT

Background: Abnormal uterine bleeding (AUB) is a common gynecological complaint with diverse etiologies. Histopathological examination of the endometrium, complemented by hormonal assessment, remains fundamental in determining the underlying cause. **Material & Methods:** This one-year prospective study included 100 women presenting with AUB. Endometrial samples were evaluated histologically, and hormonal profiles were categorized as estrogen-dominant, progesterone-dominant, or anovulatory. Data were analyzed to assess correlations. **Results:** Proliferative endometrium (30%) was the most common finding, followed by secretory (20%) and disordered proliferative patterns (15%). Hyperplasia was noted in 12%, while carcinoma was detected in 2%. Estrogen-dominant status was most prevalent (45%) and strongly associated with proliferative and hyperplastic lesions. Anovulatory status correlated with chronic endometritis and atrophic patterns. **Conclusion:** The most common histopathological findings were proliferative, secretory, and disordered proliferative endometrium, with a significant proportion showing hyperplasia. A clear association was observed between estrogen-dominant hormonal status and proliferative or hyperplastic lesions, while anovulatory states correlated strongly with chronic endometritis and atrophic endometrium.

Keywords*: Abnormal uterine bleeding, Endometrium, Histopathology, Hormonal status, Hyperplasia, Estrogen dominance.*

INTRODUCTION

Abnormal uterine bleeding (AUB) is one of the most frequent gynecological complaints encountered in clinical practice, affecting up to 30–40% of women during their reproductive and perimenopausal years (1). It significantly impairs quality of life, contributes to anemia, and represents a substantial proportion of gynecologic outpatient visits. AUB is a multifactorial condition with etiologies ranging from benign functional disturbances to premalignant and malignant endometrial lesions (2). Given its wide differential diagnosis and variable presentation, accurate evaluation is essential for appropriate management.

The International Federation of Gynecology and Obstetrics (FIGO) introduced the PALM-COEIN classification system, categorizing AUB causes into structural (Polyp, Adenomyosis, Leiomyoma, Malignancy/hyperplasia) and non-structural (Coagulopathy, Ovulatory dysfunction, Endometrial, Iatrogenic, Not yet classified) entities (3). Despite advancements in imaging and hormonal assays, histopathological examination of the endometrium remains the gold standard for diagnosing many of these conditions, particularly atypical hyperplasia and endometrial carcinoma (4). Endometrial sampling is indispensable in women above 40 years and in those with risk factors for endometrial neoplasia.

Endometrial morphology is influenced by multiple systemic and local factors; among these, hormonal status—especially estrogen and progesterone balance—plays a crucial role (5). Normal cyclical endometrial changes are orchestrated by

predictable variations in ovarian hormones. In contrast, hormonal imbalance, particularly unopposed estrogen exposure, can lead to proliferative endometrium, disordered proliferative patterns, endometrial hyperplasia, or neoplasia (6). Progesterone deficiency, common in anovulatory cycles, prevents secretory transformation and induces unpredictable shedding, resulting in dysfunctional uterine bleeding.

In reproductive-age women, AUB is frequently associated with ovulatory dysfunction, polycystic ovarian syndrome, stress, thyroid abnormalities, or obesity—all of which disturb hormonal equilibrium (7). In perimenopausal women, irregular follicular cycles and declining ovarian reserve lead to prolonged anovulation, causing excessive estrogenic stimulation of the endometrium. Conversely, postmenopausal women with AUB raise suspicion of exogenous hormone intake, obesity-related aromatization, or endometrial carcinoma until proven otherwise (8).

Histopathological evaluation often reveals a wide spectrum of lesions, including proliferative endometrium, secretory endometrium, endometrial polyps, chronic endometritis, endometrial hyperplasia, and carcinoma. These lesions have distinct relationships with hormonal milieu. For instance, endometrial hyperplasia—especially the atypical variant—is strongly associated with sustained estrogenic stimulation, tamoxifen therapy, and metabolic syndrome (9). Similarly, endometrial carcinoma, particularly the Type I estrogen-dependent variety, demonstrates a well-recognized association with hyperestrogenic states (10). Understanding these correlations is crucial because early diagnosis significantly improves prognosis and allows for timely initiation of medical or surgical treatment.

Given the rising incidence of lifestyle-related endocrine disorders, late menopause, obesity, and hormone replacement therapy, the pattern of AUB is evolving. A more detailed understanding of the hormonal and histopathological interplay can assist in improving diagnostic accuracy, refining treatment protocols, and identifying high-risk cases earlier. The present study aims to evaluate the histopathological spectrum of endometrial lesions in women presenting with AUB and correlate these findings with their hormonal status, thereby bridging an important gap in current research.

MATERIAL & METHODS

Study Settings

A **hospital-based observational cross-sectional study** was conducted at a tertiary care teaching hospital in North India over a period of one year from September 2024 to August 2025 to evaluate the histopathological spectrum of endometrial lesions among women presenting with abnormal uterine bleeding (AUB) and to correlate these findings with their hormonal status. Women in the reproductive, perimenopausal, and postmenopausal age groups presenting to the Gynecology Outpatient Department or admitted with symptoms of abnormal uterine bleeding and undergoing endometrial sampling were included. A total of **100 endometrial samples** fulfilling the inclusion criteria were included in the study over the one-year duration based on convenient sampling method.

Inclusion Criteria

- Women presenting with any form of abnormal uterine bleeding (menorrhagia, metrorrhagia, polymenorrhea, oligomenorrhea with irregular bleeding, intermenstrual bleeding, postmenopausal bleeding).
- Patients who underwent endometrial biopsy, dilatation and curettage (D&C), or hysterectomy.
- Patients with available hormonal profile reports (estrogen, progesterone, LH, FSH, TSH, prolactin, as applicable).
- Patients willing to provide informed consent.

Exclusion Criteria

- Pregnant women or bleeding due to pregnancy-related causes.
- Patients with insufficient, poorly preserved, or autolyzed tissue samples.
- Patients on anticoagulants or with known bleeding disorders (unless evaluated separately).

Data Collection Procedure

Clinical Data

Relevant clinical details were recorded on a structured proforma, including: Age, parity, BMI, Menstrual history and type of AUB, Duration of symptoms, Medical history (PCOS, thyroid disorder, diabetes, hypertension), Drug history (tamoxifen, hormone therapy) and associated systemic illnesses

Hormonal Profile

Laboratory records were reviewed to collect hormonal assays, as requested by the treating clinician which included: Serum estrogen, Serum progesterone, Luteinizing hormone (LH), Follicle-stimulating hormone (FSH), Thyroid-stimulating hormone (TSH) and Prolactin levels.

Based on these results, patients were categorized into:

- **Normal hormonal profile**

- **Estrogen-dominant state**
- **Progesterone-deficient state**
- **Anovulatory cycle**
- **Thyroid dysfunction**
- **Hyperprolactinemia**, where applicable

Endometrial Sampling and Histopathology
Types of Samples
The study included: Endometrial biopsy samples, Dilatation and curettage (D&C) specimens, Endometrial curettings and Endometrium obtained from hysterectomy specimens All samples were fixed in **10% neutral buffered formalin** for adequate preservation, processed using routine paraffin-embedding technique, sectioned at **3–5 µm thickness** and were stained with **Hematoxylin and Eosin (H&E)**. Special stains (PAS) and immunohistochemistry for **ER and PR** were employed when necessary to aid diagnosis.

Histopathological Evaluation
Microscopic evaluation was conducted independently by experienced pathologists to minimize observer bias.
Endometrial patterns were classified into:

- **Normal physiological patterns:** proliferative, secretory, menstrual
- **Disordered proliferative endometrium**
- **Endometrial hyperplasia:** simple, complex, with or without atypia (as per WHO 2014 classification)
- **Endometrial polyp**
- **Chronic endometritis**
- **Atrophic endometrium**
- **Progestin effect or pill pattern**
- **Endometrial carcinoma** (type I and type II)

Correlation between histopathological findings and hormonal status was performed.

Statistical Analysis
Data were entered into Microsoft Excel and analyzed using **SPSS software version 21.** Descriptive statistics were represented as mean, standard deviation, frequencies, and percentage as applicable. **Chi-square test** was used to evaluate association between hormonal status and histopathological patterns and a **p-value < 0.05** was considered statistically significant

RESULTS

Table 1: Age-wise Distribution of Patients (n=100)

Age Group (years)	Number of Cases	Percentage (%)
20–30	10	10%
31–40	30	30%
41–50	42	42%
>50	18	18%

The age of patients ranged from 20 to 65 years, with the majority belonging to the 41–50 years age group (42%), followed by 31–40 years (30%).

Table 2: Histopathological Spectrum of Endometrial Lesions (n=100)

Lesion Type	Number	Percentage (%)
Proliferative	30	30%
Secretory	20	20%
Disordered Proliferative	15	15%
Hyperplasia	12	12%
Endometrial Polyp	10	10%
Chronic Endometritis	8	8%
Atrophic Endometrium	3	3%
Endometrial Carcinoma	2	2%

The most common histopathological pattern was Proliferative endometrium (30%), followed by Secretory endometrium (20%), Disordered proliferative endometrium (15%), and Endometrial hyperplasia (12%).

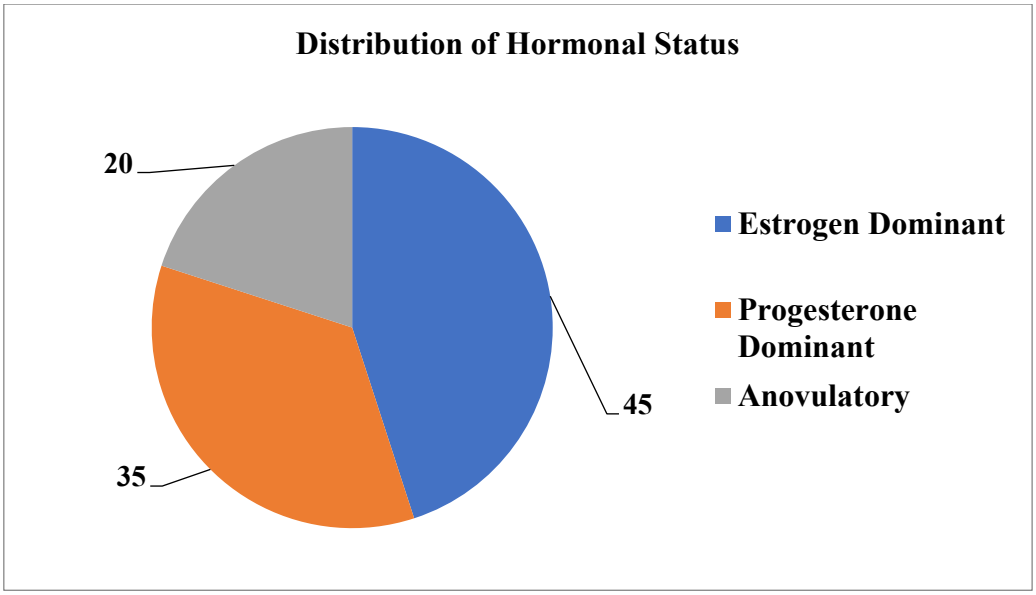

Fig. 1: Distribution of Hormonal Status (n=100)

Based on clinical and laboratory evaluation, hormonal status was categorized into three groups. Estrogen-dominant status was the most prevalent (45%), followed by Progesterone-dominant (35%), and Anovulatory status (20%).

Table 3. Correlation of Endometrial Lesions with Hormonal Status

Lesion Type	Estrogen Dominant	Progesterone Dominant	Anovulatory
Proliferative	20	10	0
Secretory	5	15	0
Disordered Proliferative	10	3	2
Hyperplasia	6	4	2
Polyp	4	5	1
Chronic Endometritis	0	3	5
Atrophic	0	1	2
Carcinoma	0	1	1

A strong correlation was observed between proliferative lesions and estrogen-dominant status (20 cases), secretory lesions with progesterone dominance (15 cases), and chronic endometritis/atrophy with anovulatory cycles (5–2 cases).

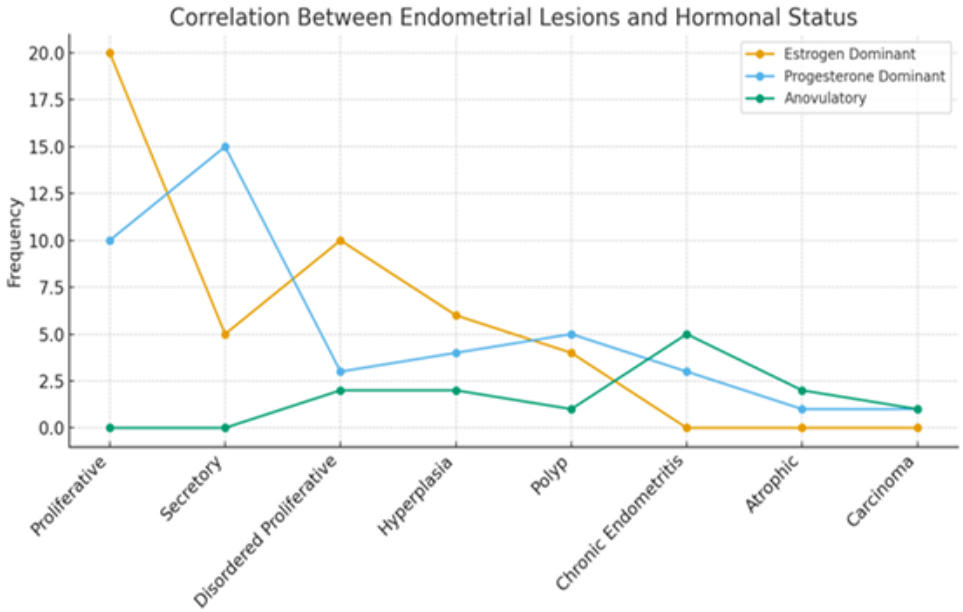

Fig. 2: Correlation of Endometrial Lesions with Hormonal Status

DISCUSSION

In our study, the majority of patients belonged to the 41–50-year age group (42%), consistent with findings by Dangal et al., who reported higher prevalence of AUB in perimenopausal women due to anovulatory cycles and fluctuating hormonal levels (4). The high incidence in this age group is attributable to declining ovarian function, resulting in irregular estrogen and progesterone secretion.The most common histopathological finding in our study was proliferative endometrium (30%), followed by secretory endometrium (20%). These findings are in agreement with studies by Doraiswami et al. (11) and Bhosale et al. (12), who similarly observed proliferative patterns as the predominant diagnosis in AUB. This indicates that many women experiencing AUB may still be undergoing ovulatory cycles but exhibit dysfunctional bleeding.

Disordered proliferative endometrium constituted 15% of cases in the present study. This pattern represents an anovulatory or estrogen-dominant environment, where persistent unopposed estrogen stimulates endometrial proliferation without subsequent progesterone-induced differentiation. Fraser et al. highlighted that unopposed estrogen leads to irregular endometrial growth, resulting in erratic shedding and AUB (2). Our correlation analysis also supported this link, with disordered proliferative lesions strongly associated with estrogen dominance.

Endometrial hyperplasia was identified in 12% of cases, comparable to the 10–15% prevalence reported in other Indian studies (11, 12). According to the WHO classification (6), hyperplasia arises from prolonged estrogenic stimulation, especially in obese, perimenopausal, or anovulatory women. In the present study, hyperplasia showed a significant association with estrogen-dominant hormonal status, reinforcing the etiological role of unopposed estrogen. Reed et al. also reported a strong link between hyperplasia and hyperestrogenic states, particularly in women with metabolic syndrome or chronic anovulation (13).

Endometrial polyps accounted for 10% of cases, a finding in line with the 8–12% reported by Singh et al. (14). Polyps commonly occur in perimenopausal women and may be influenced by tamoxifen therapy or endogenous estrogen stimulation. Their relatively high prevalence reinforces the need for hysteroscopic evaluation in selected cases of AUB. Chronic endometritis was present in 8% of cases, consistent with the 5–15% range observed in previous literature (12). Unlike proliferative and hyperplastic lesions, chronic endometritis exhibited a strong correlation with anovulatory hormonal status, suggesting that persistent low progesterone and irregular cycles may disrupt natural endometrial defense mechanisms.

Atrophic endometrium (3%) and endometrial carcinoma (2%) were less frequent. Atrophic endometrium is an expected finding in postmenopausal women and aligns with global data (4–5%). Although carcinoma was rare in this study, its identification underscores the importance of timely endometrial sampling in women, especially those over 45 years with persistent AUB. Bokhman's dualistic model emphasizes that type I endometrial carcinoma is typically estrogen-dependent and arises in a background of hyperplasia, whereas type II occurs in atrophic endometrium in elderly women (15). Our carcinoma cases corresponded to these classical pathways.

The hormonal correlation component of this study provided valuable insights. Estrogen dominance was the most common hormonal abnormality (45%), similar to patterns described by Speroff et al. (16), who noted that perimenopausal women frequently exhibit estrogen progesterone imbalance. In our data, estrogen dominance correlated with proliferative, disordered proliferative, and hyperplastic lesions, whereas progesterone-dominant status predominated in secretory patterns. Anovulatory hormonal status showed a strong association with chronic endometritis and atrophic endometrium.

Overall, the study highlights the significance of combining histopathological evaluation with hormonal profiling. While histopathology identifies structural and cellular abnormalities, hormonal analysis provides etiological context, guiding appropriate management strategies such as hormone therapy, progestin supplementation, or endometrial ablation. Our findings align well with the FIGO PALM–COEIN system, which emphasizes the dual structural and non-structural causes of AUB (1, 3).

Recommendations

1. Histopathological evaluation should be routinely performed in all women above 40 years presenting with AUB to enable early detection of premalignant and malignant lesions.
2. Hormonal profiling must be included as part of the diagnostic work-up, especially in perimenopausal and anovulatory women, to guide targeted medical management.
3. Patients with estrogen-dominant or anovulatory cycles should receive individualized hormonal therapy to prevent progression to endometrial hyperplasia.
4. Hysteroscopy may be incorporated for improved detection of focal lesions such as polyps and submucous fibroids.

Limitations

1. The study was conducted at a single center, which may limit the generalizability of findings to broader populations.
2. Hormonal assessment was based on single-time measurements and may not fully reflect dynamic hormonal fluctuations across the menstrual cycle.
3. Clinical variables such as BMI, metabolic syndrome, and medication history were not analyzed, although they influence endometrial pathology.
4. Follow-up of patients was not included, restricting evaluation of treatment outcomes or progression of endometrial lesions.

CONCLUSION

In this study the most common histopathological findings were proliferative, secretory, and disordered proliferative endometrium, with a significant proportion showing hyperplasia. A clear association was observed between estrogen-dominant hormonal status and proliferative or hyperplastic lesions, while anovulatory states correlated strongly with chronic endometritis and atrophic endometrium. These findings highlight the pivotal role of hormonal imbalance, particularly unopposed estrogen, in the pathogenesis of AUB.

Conflict of Interest: None

Source of Funding: None

REFERENCES

1. Munro MG, Critchley HOD, Fraser IS. The FIGO classification of causes of abnormal uterine bleeding. *Int J Gynaecol Obstet*. 2018;143(3):393–408.
2. Fraser IS, Critchley HOD, Broder M, Munro MG. The FIGO recommendations on terminologies and definitions for normal and abnormal uterine bleeding. *Hum Reprod Update*. 2007;13(5):467–78.
3. FIGO Working Group on Menstrual Disorders. FIGO classification system (PALM–COEIN) for causes of abnormal uterine bleeding. *Int J Gynaecol Obstet*. 2011;113(1):3–13.
4. Dangal G. A study of endometrium of patients with abnormal uterine bleeding at Chitwan Valley. *Nepal J Obstet Gynaecol*. 2003;1:26–30.
5. Singh S, Dwivedi A, Suneja A, Vaid NB, Mittal S. Clinical and histopathological correlation of abnormal uterine bleeding in perimenopausal women. *J Midlife Health*. 2013;4(1):16–21.
6. Kurman RJ, Carcangiu ML, Herrington CS, Young RH. *WHO Classification of Tumours of Female Reproductive Organs*. 4th ed. Lyon: IARC Press; 2014.
7. Speroff L, Fritz MA. *Clinical Gynecologic Endocrinology and Infertility*. 8th ed. Philadelphia: Lippincott Williams & Wilkins; 2010.
8. Kaunitz AM. Abnormal uterine bleeding in reproductive-aged women. *Obstet Gynecol Clin North Am*. 2014;41:677–86.
9. Reed SD, Newton KM, Clinton WL, Epplein M, Garcia R, Allison KH, et al. Incidence of endometrial hyperplasia. *J Clin Endocrinol Metab*. 2009;94:2424–31.
10. Bokhman JV. Two pathogenetic types of endometrial carcinoma. *Gynecol Oncol*. 1983;15(1):10–17.
11. Doraiswami S, Johnson T, Rao S, Rajkumar A, Vijayaraghavan J, Panicker VK. Study of endometrial pathology in abnormal uterine bleeding. J Obstet Gynaecol India. 2011;61(4):426–30.
12. Bhosale A, Fonseca M, Desai D, Gaikwad P. Evaluation and histopathological correlation of abnormal uterine bleeding in perimenopausal women. J Clin Diagn Res. 2013;7(11):2533–6.
13. Reed SD, Newton KM, Clinton WL, Epplein M, Garcia R, Allison KH, et al. Incidence of endometrial hyperplasia. J Clin Endocrinol Metab. 2009;94:2424–31.
14. Singh S, Best C, Dunn S, Leyland N, Wolfman WL. Abnormal uterine bleeding in pre-menopausal women. J Midlife Health. 2013;4(1):16–21.
15. Bokhman JV. Two pathogenetic types of endometrial carcinoma. Gynecol Oncol. 1983;15(1):10–17.
16. Speroff L, Fritz MA. Clinical Gynecologic Endocrinology and Infertility. 8th ed. Philadelphia: Lippincott Williams & Wilkins; 2010.