International Journal of Medical and Health Research
www.medicalsciencejournal.com
ISSN: 2454-9142
Received: 30-01-2026, Accepted: 01-03-2026, Published: 16-03-2026
Volume 12, Issue 1, 2026, Page No. 88-91

Decoding the Uterine -ovarian link: A Histomorphological study of endometrial hyperplasia and ovarian changes

Dr. Suma Hadimani[1*], Dr. Navya B N[2], Dr. Bharathi M[2], Dr. Anjali Rao[3], Dr. Sathyavathi R Alva[4]
[1] Department of Pathology, KVG Medical College and Hospital, Sullia, Karnataka, India
[2] Professor, Department of Pathology, KVG Medical College and Hospital, Sullia, Karnataka, India
[3] Associate Professor, Department of Pathology, KVG Medical College and Hospital, Sullia, Karnataka, India
[4] Head and Professor, Department of Pathology, KVG Medical College and Hospital, Sullia, Karnataka, India
Corresponding Author: Dr. Suma Hadimani

Abstract
Background: Endometrial hyperplasia is defined as a thickening of the endometrium, characterized by an increased gland-to-stroma ratio and proliferation of glands that are irregular in size and shape. It is primarily caused by prolonged exposure to unopposed estrogen. The exact source of this hormonal imbalance is still under investigation, with the ovarian stroma emerging as a possible contributor that may enhance estrogen production. Studying these histomorphological changes in both the endometrium and ovary may help to elucidate the hormonal interplay between these two organs in endometrial hyperplasia.
Objectives: To evaluate the histopathological patterns of endometrial hyperplasia in and to identify and categorize ovarian stromal changes (such as stromal hyperplasia, luteinization, or hyperthecosis) in the same patients and to assess the association endometrial hyperplasia and specific ovarian stromal changes.
Materials: This retrospective study included 53 women diagnosed with endometrial hyperplasia on total hysterectomy specimens with unilateral or bilateral salpingo-oophorectomy, received in the Department of Pathology at our institute from January 2023 to January 2025. Endometrial findings were correlated with histomorphological changes in the ovaries.
Results: Most patients were between 45 and 50 years of age. Of the 53 cases, 49 (92. 45%) were classified as hyperplasia without atypia and 4 (7. 54%) as atypical hyperplasia. Ovarian stromal changes were observed in 46 cases (86. 79%), while 7 cases (13. 2%) showed no notable changes. Among the 46 cases with ovarian alterations, the following changes were noted (some in combination)-Stromal hyperplasia (which was Major finding): 75% (40 cases), Stromal luteinization: 67% (36 cases), Stromal thecosis: 7. 5% (4 cases), Interstitial glands:5% (8 cases), Serous cysts: 43% (23 cases), Mucinous cysts: 9% (5 cases), Follicular cysts: 56% (30 cases).
Conclusion: This study support the hypothesis that the ovary, particularly the stroma, may play a contributory role in the pathogenesis of endometrial hyperplasia through unopposed estrogen production.

Keywords: Endometrium, hyperplasia, ovary, stroma

Introduction
Endometrial hyperplasia is defined as thickening of endometrium with a proliferation of irregularly sized and shaped glands and an increased gland-to-stroma ratio [1]. It is caused by increased exposure to estrogens and a relative deficiency of progesterone- referred to as "unopposed estrogen. " [2]
Most cases are self-limiting and are effectively managed with progestagen therapy, historically referred to as “chemical curettage. ” However, a subset may progress to atypical hyperplasia, which is recognized as a precursor lesion to endometrial adenocarcinoma. Additionally, patients who do not respond to hormonal therapy often require definitive surgical management in the form of abdominal hysterectomy with unilateral or bilateral salpingo-oophorectomy as part of the treatment protocol [3, 4].
Incidence is rising due to increase in risk factors like obesity, diabetes, or other related illnesses [5]. Precise origin of this hormonal imbalance remains a subject of ongoing debate. One potential contributor is the ovarian stroma, which may undergo subtle morphological changes capable of increasing estrogen production [6-8].
Although the endocrine relationship between ovary and endometrium is well established, histopathological correlation studies are limited [9]. Understanding the histomorphological changes in both the endometrium and ovaries can help us to understand the uterine-ovarian hormonal interplay underlying endometrial hyperplasia [10].
This study is performed to assess various patterns of ovarian changes and their relation to endometrial hyperplasia.

Aims and objectives
1. To evaluate the histopathological patterns of endometrial hyperplasia.
2. To identify and categorize ovarian stromal changes (such as stromal hyperplasia, luteinization, or hyperthecosis) in the same patients.
3. To assess the association of endometrial hyperplasia with ovarian stromal changes.

Materials and Methods
Study design: Retrospective study
Duration: Two-years period (January 2023 to 2025)
Specimens showing histopathological features of endometrial hyperplasia in patients undergoing total abdominal hysterectomy with bilateral/unilateral salpingo-oophorectomy were selected randomly.

Specimens showing malignant lesions in endometrium or ovary, patients with prior history of hormonal therapy or chemotherapy and inadequate tissue preservation or poorly processed slides were excluded.

Sample Collection and methodology

Total hysterectomy specimens evaluated and sections taken at 1 cm intervals from endomyometrium. Representative sections from ovaries were given and routine histopathological processing was done. All slides are stained with H&E staining.

Endometrial hyperplasia is classified according to the WHO 2020 classification as:[11]

1. Hyperplasia without atypia
2. Atypical hyperplasia.

Endometrial Parameters	Ovarian Parameters
Gland-to-stroma ratio	Stromal cellularity and edema
Architecture of glands and stroma	Stromal hyperplasia
Glandular lining	Stromal luteinization or thecosis
Nuclear stratification	Follicular and luteal cysts or other
Mitotic activity	Surface inclusion cysts and abortive follicles
Presence of nuclear atypia	Tumor tissue with morphological features

Microscopic Parameters Evaluated

Statistical analysis: was done by using SPSS software, version 20 numerical data were compared by using 2-tailed t-test and all the categorical data were compared by using Chi square test.

For statistical significance, p-value of less than 0. 05 was considered.

Results

Table 1: Distribution of Patients in Different Age Groups (n=53)

Age	Number	Percentage
<40	5	9
40-45	7	13
46-50	33	62
>50	8	15

Among 53 patients with endometrial hyperplasia, 9% (5 patients) were below 40 years, 13% (7 patients) were within the age group of 40 to 45 years, 62% (33 patients) were within the age group of 46 to 50 years, and the rest 15% (8 patients) were in the age group of above 50 years (Table 1).

Table 2: Distribution of the Different Types of Endometrial Hyperplasia

Endometrial Hyperplasia Without Atypia	Atypical Endometrial Hyperplasia
49	4

Among 53 patients, 92% (49) had features of simple hyperplasia without atypia;7. 5% (4) had features atypical endometrial hyperplasia. (Table 2)

Table 3: Ovarian Changes Are Significantly Associated with Endometrial Hyperplasia

Ovarian Changes	Number of cases	Percentage of cases
Non-specific stromal hyperplasia	40	75
Luteinization	36	67
Thecosis of stroma	4	7
PCOS and focal granulosa cell hyperplasia	0	0
Interstitial glands.	8	15
Follicular cyst	30	56
Corpus luteal cyst	27	50
Endometriosis	2	3
Stromal Odema	4	7
Simple serous cyst	23	43
Mucinous cystadenoma	5	9

Table 4: Percentage Distribution of Subtle Ovarian Stromal Changes

	Ovarian Change	No Ovarian Change
Endometrial hyperplasia	47	6

Out of the 53 total cases having endometrial hyperplasia, 47 cases (88. 6%) had one or more subtle ovarian stromal changes and 6 cases (11. 3%) lacked the ovarian stromal changes. (Table. 3)

The distribution of subtle ovarian stromal changes in the study population revealed that non-specific stromal hyperplasia was the most common finding, observed in 40 cases (75%). Luteinization was noted in 36 cases (67%), making it the second most frequent change. Follicular cysts were identified in 30 cases (56%), followed by corpus luteal cysts in 27 cases (50%). Simple serous cysts were present in 23 cases (43%). Interstitial glands were observed in 8 cases (15%). Less frequent findings included mucinous cystadenoma in 5 cases (9%), thecosis of stroma in 4 cases (7%), and stromal edema in 4 cases (7%). Endometriosis was identified in only 2 cases (3%). Notably, no cases of PCOS with focal granulosa cell hyperplasia were observed in the present study. (Table4)

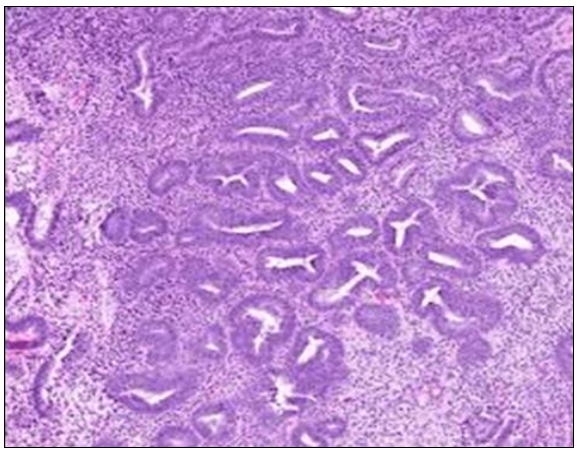


Fig 1: Simple hyperplasia without atypia (H&E, X40)

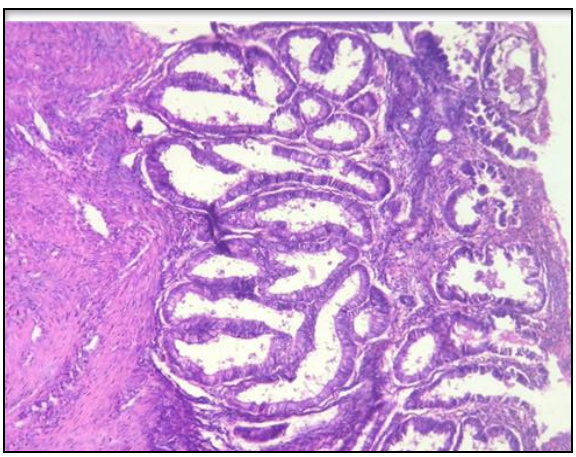


Fig 2: Hyperplasia With Atypia (H&E, X40)

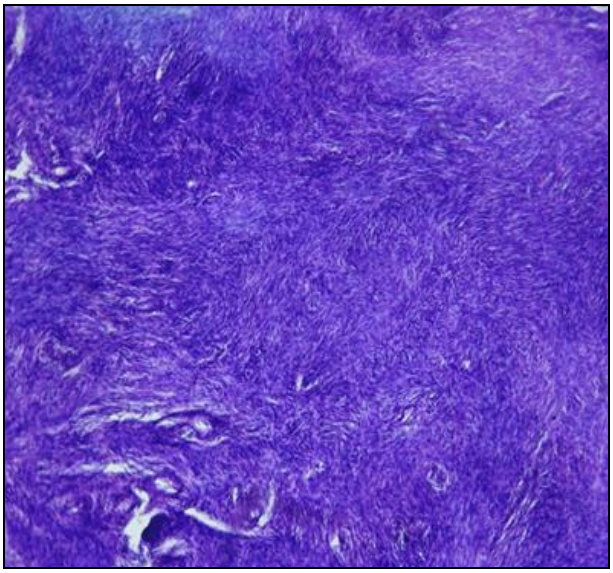


Fig 3: Non-specific Stromal Hyperplasia (H&E, X40)

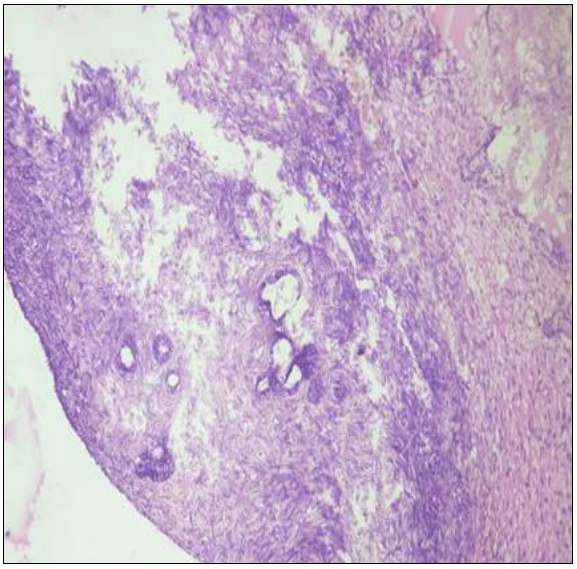


Fig 4: Interstitial Glands in Ovarian Stroma (H&E, X40)

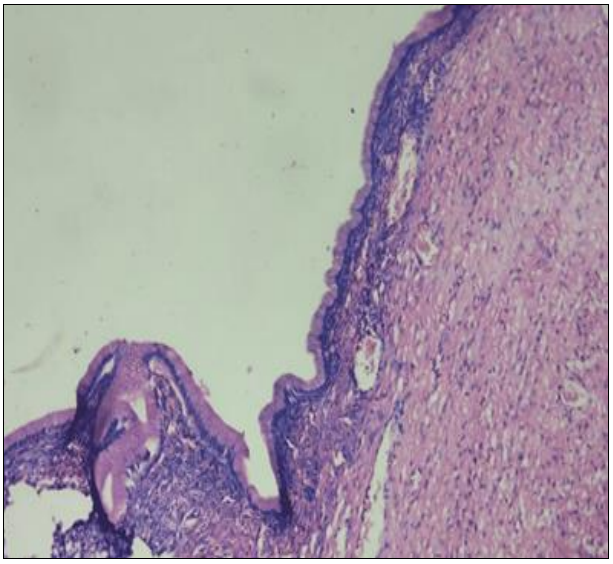


Fig 5: Mucinous cystadenoma (H&E, X40)

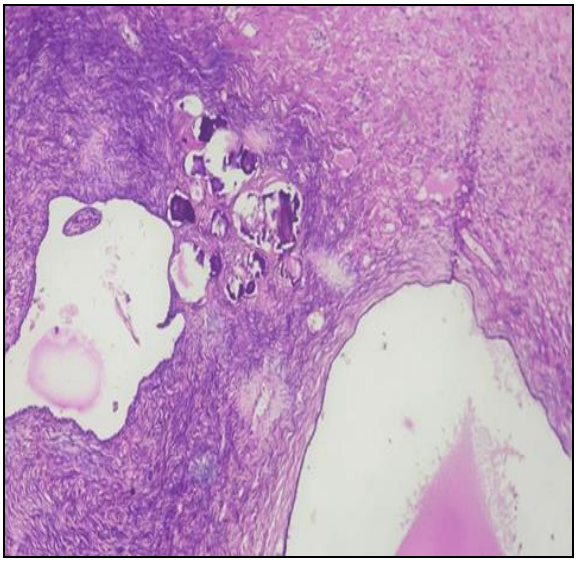
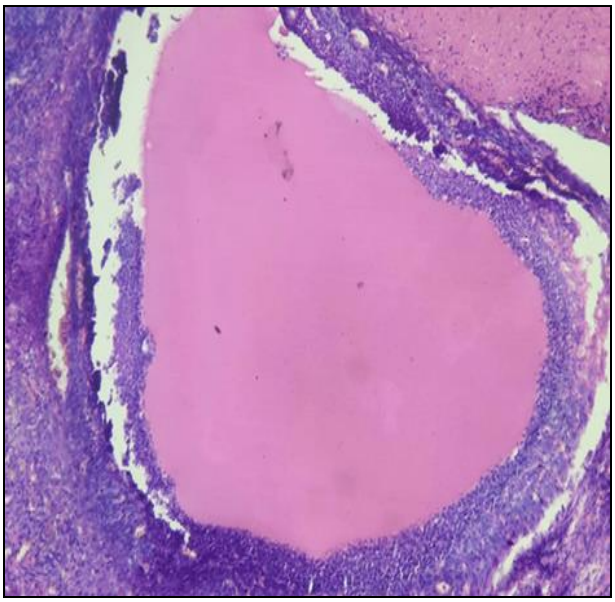


Fig 6: Serous Cystadenoma with Psomommatous Calcification (H&E, X40)



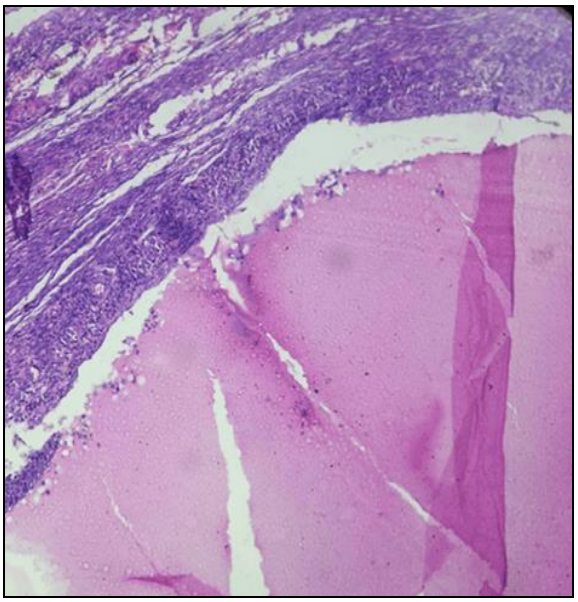


Fig 7: Follicular cyst (H&E, X40)

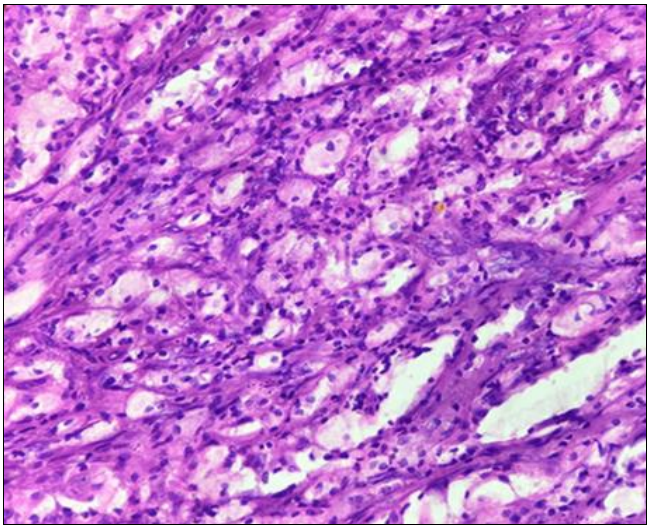


Fig 8: Luteinization of stroma (H&E, X40)

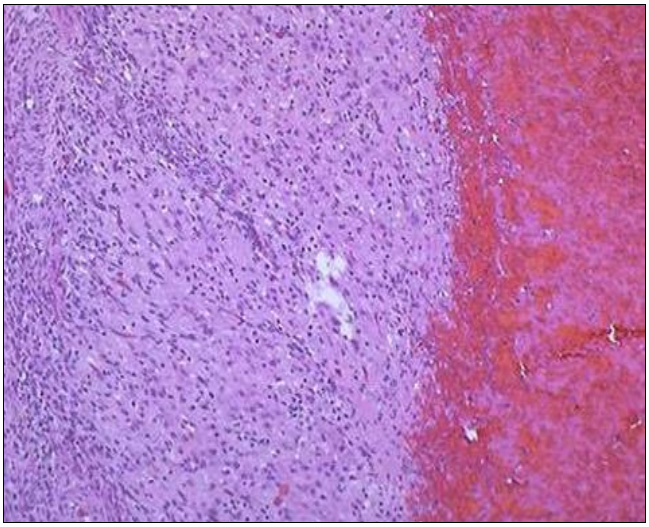


Fig 9: Hemorrhagic corpus luteal cyst (H&E, X40)

Discussion

In this study, the cases of endometrial hyperplasia were within the age range of 39 to 57 years and majority (62%) are a predilection for 45-50 years. Most of the endometrial hyperplasia cases in this study showed the ovarian stromal changes (86. 7%). Non-specific stromal hyperplasia was the commonest observation among different ovarian changes.

In this study, the cases of endometrial hyperplasia were within the age range of 39 to 57 years and majority (62%) are a predilection for 45-50 years. Most of the endometrial hyperplasia cases in this study showed the ovarian stromal changes (86. 7%). Non-specific stromal hyperplasia was the commonest observation among different ovarian changes. Thecomatosis, luteinization of stroma, interstitial glands, follicular cysts, hemorrhagic cysts etc., were the other different observations, either singly or in combination. These findings suggest that various stromal changes—occurring in different combinations and proportion, may contribute to an unopposed hyperestrogenic state, which could play a key role in the pathogenesis of endometrial hyperplasia. We found that simple endometrial hyperplasia without atypia is the commonest entity to produce uncontrolled meno-metrorrhagia leading to hysterectomy.

In study by, Ghosh S *et al* [6]. also -Non-specific stromal hyperplasia was the commonest observation among different ovarian changes., indicating that various stromal changes in various combinations and different proportions may be a reason behind unopposed hyperestrogenic state in cases of endometrial hyperplasia. Farber *et al* [12]. And Aiman *et al* [13], also studied ovarian stromal cells and their luteinization and told that increase functional activity of luteinized stromal cells often produces significant quantities of steroid hormones

Conclusion

Endometrial hyperplasia is primarily linked to prolonged stimulation of the endometrium by unopposed estrogen; however, the precise origin of this hormonal imbalance remains uncertain. The ovarian stroma has been proposed as a potential source, with subtle histological alterations possibly contributing to excess estrogen production. In this study, a significant correlation was observed between ovarian stromal changes and the presence of endometrial hyperplasia.

Limitations

This is a tertiary hospital-based study having very short period and limited cases, and therefore biased result is possible.

References

1. Mutter GL. Endometrial hyperplasia and neoplasia. Modern Pathology,2000:13:285-297.
2. Lobo RA. Endometrial hyperplasia. Journal of Clinical Endocrinology and Metabolism,2008:93:S161-S162.
3. Kim JJ, Kurita T, Bulun SE. Progesterone action in endometrium. Endocrine Reviews,2013:34:130-171.
4. Zaino R, Carinelli SG, Ellenson LH. Tumours of the Uterine Corpus: Epithelial Tumors and Precursors. WHO Press,2014:125-126.
5. Baak JP, *et al*. Prognostic markers in endometrial hyperplasia. International Journal of Gynecological Pathology,2005:24:1.
6. Ghosh S, Mukherjee S, Bandopadhyay G. A Study to Correlate Endometrial Hyperplasia with Ovarian Stromal Change. Recent Advances in Pathology and Laboratory Medicine,2018:4:1-5.
7. Speroff L, Fritz MA. Clinical Gynecologic Endocrinology and Infertility. 2011.
8. Rotterdam ESHRE ASRM PCOS Consensus Group. Revised consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertility and Sterility,2004:81:19-25.
9. Evans P, *et al*. Ovarian morphology in endometrial hyperplasia. Histopathology,2011:59:1114-1122.
10. Giannopoulos T, *et al*. Ovarian changes in hyperplasia. European Journal of Obstetrics Gynecology and Reproductive Biology,2012:164:181-186.
11. World Health Organization Classification of Tumours Editorial Board. World Health Organization Classification of Tumours Female Genital Tumours. IARC Press,2020.
12. Farber M, Facog D, Philip R, *et al*. Hyperthecosis syndrome. Obstetrics and Gynecology,1974:44:1-157.
13. Aiman J, Nalick RH, Facog J, *et al*. The origin of androgen and estrogen in a virilized postmenopausal woman with bilateral benign cystic teratomas. Obstetrics and Gynecology,1977:49:6.