ORIGINAL ARTICLE

# The Role of Immunohistochemistry in Hyperplastic Endometrial Disorders and Endometrial Carcinoma Cases: A Cross Sectional Observational Study

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**Abstract:**
*Background:* Endometrial hyperplasia refers to a variety of morphological endometrial changes. Atypical hyperplasia can progress to endometrial carcinoma. In carcinogenic lesions, IHC aids in the diagnosis, therapeutic response and prognosis. *Aim:* To evaluate the expression of ER,PR,p53 and Ki67 by immunohistochemistry in hyperplastic endometrial disorders and endometrial carcinoma. *Material and Methods:* Present study was single-center, cross sectional study, conducted on specimens of endometrial biopsies/curettage, hysterectomy specimens, received at department of pathology. IHC was performed on EH as well as on EC specimens.IHC markers p53, ER, PR and Ki67 were done. *Results:* In present study, 86 patients with endometrial disorders were included, mean age of patients was 47.44 ± 10.01 years. The commonest diagnosis reported in the present study was typical hyperplasia in 57 (66.3%) of patients, followed by endometrial carcinoma in 16 (18.6%) patients and atypical hyperplasia in 13 (15.1%) patients. Of these 16 carcinoma cases, there were cases of endometriosis adenocarcinoma (15.1%) and 3 cases of clear cell adenocarcinoma (3.5%). The expression of p53 was present in 3 (3.5%) patients. ER and PR expression was present among 82 (95.3%) of patients. Ki67 expression was < 10% in 34 patients, 10-20% in 40 cases and >20% in 12 cases. We reported significant ($p<0.0001$) difference in distribution of patients according to diagnosis and expression of all IHC parameters. *Conclusion:* The immune histo chemical (IHC) expression so markers including p 53, estrogenreceptor(ER),progesteronereceptor(PR)andproliferationmarkerKi67 are recommended in patients with hyperplastic endometrial disorders.

**Keywords:** Endometrial Carcinoma, Hyperplastic Endometrial Disorders, Immunohistochemistry.

## Introduction:

Endometrial hyperplasia refers to a variety of morphological endometrial changes, manifesting clinically as abnormal uterine bleeding. It is distinguished by a higher endometrial gland to stroma ratio than normal proliferative endometrium. Endometrial hyperplasia is classified into two types: typical hyperplasia and atypical hyperplasia. Atypical hyperplasia has the potential to progress to endometrial carcinoma. [1,2] Endometrial carcinoma is a common gynaecologic cancer, particularly in developing countries. Endometrial carcinoma is classified into two types: type 1 and type 2. Type 1 tumours are low grade tumours, whereas type 2 tumours are high grade aggressive tumour's. Haematoxylin and eosin staining has long been considered the gold standard technique for morphologically interpreting endometrial hyperplasia.[3] However, many new techniques are now available to aid in the diagnosis and differentiation of lesions with overlapping histological features. Immunohistochemistry (IHC), detects protein expression and allows cells in a given sample to be differentiated. In carcinogenic lesions, IHC aids in the diagnosis, therapeutic response and prognosis.[4] Present study was aimed to evaluate the expression of ER, PR, p53 and Ki67 by immunohistochemistry

## Material & Methods:

Present study was single-center, cross sectional study, conducted in the department of pathology. The duration of prospective cases was 2 years (from August 2020 to

August 2022) & Retrospective- 1 year (July 2019 to July 2020). Study approval was obtained from institutional ethical committee. All endometrial specimens including biopsies, curettage & hysterectomy specimens received in department of pathology were included in the study and inadequate specimens were excluded from the study. Patient's clinical details such as history taking (Detailed menstrual, medical and family history, presence of post coital, inter menstrual or post-menopausal bleeding, associated dysmenorrhea or premenstrual symptoms, history of intake of hormones, contraceptive pills or device, history of iron therapy or blood transfusion), clinical assessment findings, ultrasonography findings & any previous histopathological reports were noted. Received specimens of endometrium were fixed in 10% formalin for 12-24 hours. After fixation, paraffin blocks were made and4 -5 micron thick sections were cut. Slides were stained with hematoxylin and eosin stain and studied histopathologically. In retrospective cases, relevant paraffin embedded tissue blocks were retrieved from the departmental archives. IHC was done using appropriate monoclonal antibodies. IHC markers p53, ER, PR and Ki67 were included in this study. The primary antibodies used were ER (Clone EP1; Dako) (Control used- Normal breast tissue), PR (Clone PgR636; Dako) (Control used- Normal breast tissue), p53 (Clone DO-7; Dako) (Control used- High grade serous ovarian carcinoma) and Ki67 (Clone MIB-1; Dako). The scoring of ER, PR and p53 (Aberrant expression with strong nuclear staining) were done in as positive and negative while percentage labelling index was followed for Ki67. Relevant clinical data was correlated with histopathological studies. The collected data was coded and entered in Microsoft Excel sheet, and analyzed using SPSS (Statistical Package for social sciences) version 26.0 software. The results were presented in a tabular format. For qualitative data, various rates, ratios and percentages were calculated. For quantitative data Mean/Median, SD etc. were calculated. Independent T test and paired t-test were used to compare the quantitative data. P-value <0.0001 considered as significant.

**Results:**

In the present study, 86 patients with endometrial disorders were included, mean age of patients was 47.44 ± 10.01 years, ranging between 21 to 71 years.The hormonal history of progesteronewas present in 50 (58.1%) out of 86 patients. Common chief complaint was menorrhagia (43%), followed by postmenopausal bleeding (30.2%), polymennorhea (9.3%), andfibroid and oligomenorrhea (in association with PCOS which is related to endometrial carcinoma)5 (5.8%) each. 63 (73.3%) underwent endometrial curettage and remaining 23 (26.7%) underwent hysterectomy. The commonest diagnosis reported in the present study was typical hyperplasia shown in 57 (66.3%) of patients, followed by endometrial carcinoma in 16 (18.6%) patients and atypical hyperplasia in 13 (15.1%) patients. Of 16 carcinoma cases, 13 cases were hysterectomy specimens and remaining 3 cases were received as dilatation and curettage specimens followed by hysterectomythat was did in some other health centre. Of these 16 carcinoma cases, 13 cases were of endometrioid adenocarcinoma (15.1%) and 3 cases of clear cell adenocarcinoma (3.5%).

Table 1: Diagnosis

| Diagnosis | Frequency | Percentage |
|---|---|---|
| Typical Hyperplasia | 57 | 66.3 |
| Atypical Hyperplasia | 13 | 15.1 |
| Endometrial carcinoma | 16 | 18.6 |
| • Endometriosis adenocarcinoma | 13 | 15.1 |
| • Clear cell adenocarcinoma | 3 | 3.5 |

The immunohistochemical (IHC) expressions of markers including p53, estrogen receptor (ER), progesterone receptor (PR) and proliferation marker Ki67 were analyzed. The expression of p53 was present in 3 (3.5%) patients. ER and PR expression was present among 82 (95.3%) of patients. Ki67 expression was < 10% in 34 patients, 10-20% in 40 cases and >20% in 12 cases.

Table 2: IHC expression

| IHC expression | Frequency | Percentage |
|---|---|---|
| p53 | 3 | 3.5 |
| ER | 82 | 95.3 |
| PR | 82 | 95.3 |
| Ki67 expression | | |
| <10% | 34 | 39.5 |
| 10-20% | 40 | 46.5 |
| >20% | 12 | 14 |

We reported significant (p<0.0001) difference in distribution of patients according to diagnosis and expression of all IHC parameters including p53, ER, PR and Ki67. All 3 clear cell carcinoma cases had a high p53 expression, while the expression of ER and PR was negative in these cases with a Ki67 value of >20%. In contrast, 13 cases with endometriosis adenocarcinoma,

Ki67 expression was between 10-20% in 4 cases, >20% in nine cases.

Table 3: Association of diagnosis with IHC expression

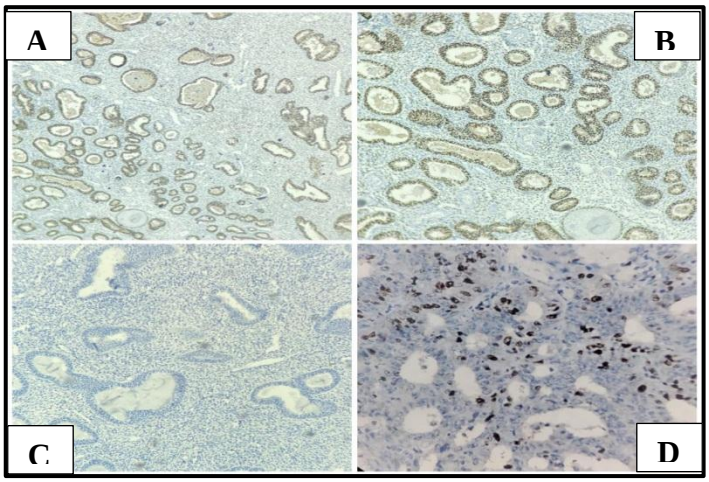
| | | | Typical Hyperplasia | Atypical Hyperplasia | Endometriosis adenocarcinoma | Clear cell adenocarcinoma | P value |
|---|---|---|---|---|---|---|---|
| p53 | Negative | N | 57 | 13 | 13 | 0 | <0.001* |
| | | % | 100 | 100 | 100 | 0 | |
| | Positive | N | 0 | 0 | 0 | 3 | |
| | | % | 0 | 0 | 0 | 100 | |
| ER | Negative | N | 0 | 0 | 1 | 3 | |
| | | % | 0 | 0 | 7.7 | 100 | |
| | Positive | N | 57 | 13 | 12 | 0 | <0.001* |
| | | % | 100 | 100 | 92.3 | 0 | |
| PR | Negative | N | 0 | 0 | 1 | 3 | |
| | | % | 0 | 0 | 7.7 | 100 | |
| | Positive | N | 57 | 13 | 12 | 0 | <0.001* |
| | | % | 100 | 100 | 92.3 | 0 | |
| Ki67 | <10% | N | 32 | 2 | 0 | 0 | |
| | | % | 37.2 | 2.3 | 0 | 0 | |
| | 10-20 % | N | 25 | 11 | 4 | 0 | |
| | | % | 29.1 | 12.8 | 4.7 | 0 | |
| | >20% | N | 0 | 0 | 9 | 3 | |
| | | % | 0 | 0 | 10.5 | 3.4 | <0.001* |

Table 4: Endometrial carcinoma as per histological type with IHC expression

| | | Histological type | | | |
|---|---|---|---|---|---|
| | | Endometriosis adenocarcinoma | | Clear cell adenocarcinoma | |
| | | N | % | N | % |
| p53 | Negative | 13 | 100 | 0 | 0 |
| | Positive | 0 | 0 | 3 | 100 |
| ER | Negative | 1 | 7.7 | 3 | 100 |
| | Positive | 12 | 92.3 | 0 | 0 |
| PR | Negative | 1 | 7.7 | 3 | 100 |
| | Positive | 12 | 92.3 | 0 | 0 |
| Ki67 | <10% | 0 | 0 | 0 | 0 |
| | 10-20% | 4 | 30.8 | 0 | 0 |
| | >20% | 9 | 69.2 | 3 | 100 |

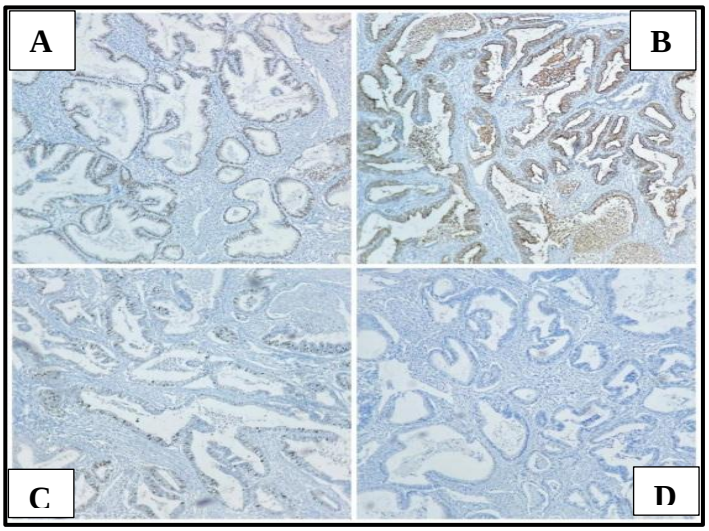
All 3 clear cell carcinoma patients had positive expression of p53, while the expression of ER and PR was negative in these patients with a ki67 value of >20% in all the cases. In contrast, the patients with endometriosis adenocarcinoma, p53 expression was negative in all 13 patients, Ki67 expression was between 10-20% in 4 cases and more than 20% in 9 patients. The expression of ER and PR was positive in 12 (92.3%) patients each out of 13.

**Figure 1- A)Positive ER expression in atypical hyperplasia, B) Positive PR expression in atypical hyperplasia, C) No expression of p53 in atypical hyperplasia, D) Ki67 10-20% in atypical hyperplasia**



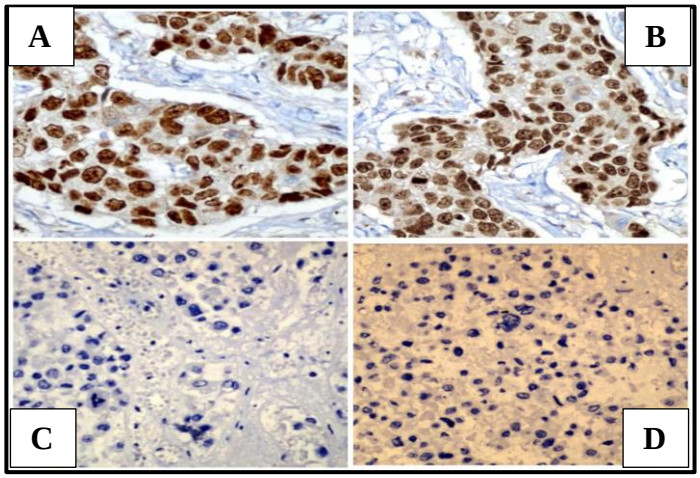

There was only 1 endometriosis adenocarcinoma case reported with negative ER and PR. This case had Ki67 value more than 20% and was graded under grade II. All 8 patients with histological grade 1 had positive expression of ER and PR, while the expression was negative in all for p53.

**Figure 2- A)Positive ER expression in endometriosis adenocarcinoma (Type 1), B) Positive PR expression in endometriosis adenocarcinoma (Type 1), C)Ki67 >20% in endometriosis adenocarcinoma (Type 1), D) No expression of p53 in endometriosis adenocarcinoma**



The values of Ki67 were between 10%-20% in 4 cases and more than 20% in the remaining 4 cases. All 5 cases of grade 2 endometrial carcinoma had negative expression of p53 and only 1 case had ER, PR negative. Ki67 was more than 20% in all the cases of grade 2 endometrial carcinoma. While in grade 3 patients, the expression of p53 was positive in all 3 patients, and expressions of ER and PR were negative. The values of Ki67 were more than 20% in all the cases.

**Figure 3- A) Positive expression of p53 in Clear cell adenocarcinoma (Type 2), B) Ki67 >20% in Clear cell adenocarcinoma (Type 2), C) No expression of ER in Clear cell adenocarcinoma, D) No expression of PR in Clear cell adenocarcinoma**




**Discussion:**

The most common gynecological malignancy is endometrial cancer. After breast, lung, and colorectal cancers, it is the fourth most prevalent cancer in women. [5]Endometrial hyperplasia is a lesion that develops before endometrial cancer. A 1–3 percent chance exists that hyperplasia will develop into cancer. Compared to simple or complex hyperplasia, atypical hyperplasia is associated with a higher risk of developing cancer; 30 to 40 percent of patients with atypical hyperplasia also have concurrent adenocarcinomas.[6] Two types of endometrial cancer are typically recognized. More than 70% of cases are Type I, which is the most prevalent form. Endometriosis adenocarcinomas, or type I tumor, are linked to unopposed estrogenic stimulation. These tumors typically have a low grade. High-grade tumors with the histologic types papillary, serous, or clear cell are more likely to be type II tumors. They have a bad prognosis and a high risk of metastasizing and relapsing. Only 10% of endometrial cancers are Type II, but it is responsible for 40% of deaths from the disease. [6,7] Endometriosis type 1 carcinoma is frequently preceded by atypical hyperplasia and is frequently associated with mutations that up regulate PI3K/AKT signaling. Serous type 2 carcinoma is linked to serous endometrial intraepithelial carcinoma, with TP53 mutations are the most common. [8,9]

Immunohistochemistry (IHC), works on the basis of detecting a specific protein within a cell using a commercially available antibody. After that, the antigen-antibody complex is stained and examined under a light microscope.[10] In carcinogenic lesions, IHC aids in the diagnosis, therapeutic response and prognosis.TP53 mutation, loss of hormone receptors, and Ki67 are three molecular biomarkers that have demonstrated independent prognostic value in endometrial cancer, focusing on survival and/or risk of lymph node metastases. [12,13] It was seen that the most common presentation of PCOS is abnormal menstruation such as oligomenorrhoea, amenorrhoea, or dysfunctional uterine bleeding. In 1949, it was reported that PCOS was associated with endometrial cancer.[11]In the present study, the majority37 (43%) of patients had menorrhagia as their chief complaints, followed by postmenopausal bleeding 26 (30.2%), polymenorrhea8 (9.3%), 5 cases of fibroid and 5 cases of oligomenorrhea (5.8% each) , 2 cases of infertility (2.3%), 2 cases of ovarian tumor (2. 3%) and 1 (1.2%) case of breast carcinoma.. 50 cases of endometrial carcinoma were evaluated by Kaur J et al, [14]pre-menopausal bleeding and discharge appeared in 5 (10%) cases, while post-menopausal discharge was seen in 5 (10%) cases. An endometrial tissue sample is necessary for the accurate diagnosis of endometrial cancer. Endometrial hyperplasia (EH) without atypia, atypical endometrial hyperplasia/endometrial intraepithelial neoplastic, endometriosis adenocarcinoma, uterine serous carcinoma, and clear cell carcinoma were the most prevalent types of EH and endometrial carcinomas reported by Masjeed NMA et al. [12]Kaur J. et al., [14] noted that endometriosis was the most prevalent histopathological pattern of endometrial carcinoma, occurring in 42 (84%) patients and non-endometriosis in 8 (16%) patients. In present study, similar findings that is endometriosis carcinomas were seen in 13 cases and remaining 3 were clear cell carcinoma . Hematoxylin and eosin (H&E) staining of endometrial sections was used to differentiate between benign and atypical endometrial hyperplasia using specific microscopic criteria; however, the subjective differences between the observers may be noticeable in such histopathology evaluation, particularly in the determination of atypical type. Therefore, specific immunohistochemical markers are required to confirm the diagnosis and to pinpoint endometrial hyperplasia cases that are at risk for developing into cancer. [15,16] Undifferentiated endometrial carcinoma is known to be distinguished by ER, PR, p53, and Ki67.[12]Estrogen receptor is expressed by most endometrial cancers (60–

70%). Endometrial tumours that are estrogen-receptor-positive typically have a better prognosis. Despite this, there is frequently a mismatch between the clinical course of the disease and oestrogen receptor expression. [17] The proliferation and expansion of tumor cells are typically correlated with the non- histone nuclear protein Ki67. Since a higher rate of proliferation is one of the characteristics of cancer cells, this makes Ki67 an excellent marker. The proliferative index of endometrial malignant tumors can be used to detect this proliferation, which is correlated with the aggressiveness of these tumors (Ki67). [15,18] While the p53 gene is typically overexpressed in type II carcinomas, the PTEN gene is frequently altered in type I carcinomas. Endometrial carcinomas with poor differentiation, no steroid receptors, and poor prognosis have mutations or increased expression of the p53 tumour suppressor gene in 10-48 percent of cases. Even in its early stages, anti-oncogene p53 mutations may be present in 45–85 percent of serous endometrial cancers. [19,20] Only 3.5% of patients in the current study had p53 expression. This is significant. the expression of p53 was significantly positive only in clear cell carcinoma and negative in other diagnoses, including typical, atypical hyperplasia, and endometriosis adenocarcinoma. ER and PR expression was present in 95.3% of patients; their expressions were significantly positive among all histological diagnoses except clear cell carcinoma, while Ki67 expression was less than 10% in 39.5% cases, between 10- 20% in 46.5% cases and more than 20% in 14% cases. In comparison to the findings of the current study, Suthipintawong C et al.,[21] reported a slightly lower number of patients with positive and negative ER and PR expression. In their study, 76.9 percent of participants had positive ER results, and 72.3% had positive PR results. Ki-67 revealed that 38 cases (58.5%) had nuclear staining that was less than 35 percent positive and 27 cases (41.5%) had nuclear staining that was greater than 35 percent positive. The rates of Ki67 were lower and the expression of p53 was higher, with p53 being positive in 47.7% of cases. However, in our study Ki67 was found to be higher in cases of endometrial carcinomas. In a study by Kaur J. et al., [14] lower levels of ER (64%) and PR (60 %) expression were also noted. According to the diagnosis and grades by histopathology, the expression of ER and PR was significantly changed. Additionally, they reported that, in cases of well-differentiated carcinoma, 14.3% of them had mildly positive ER expression, 28.6% had moderately positive ER expression, and 46.4% had strongly positive ER expression. 66.7 percent of mild and 16.7 percent of moderate ER positivity were found in moderately differentiated carcinoma. According to Masjeed NMA et al.,[12] ER expression was more prevalent in EH without atypia (92.85%) than in AEH (85.71%) and endometrial carcinoma (60.71%). Compared to AEH (85.71 percent) and endometrial carcinoma, EH without atypia (90 percent) exhibited higher levels of PR expression (64.28 percent ). In a study by Alnuaimy S et al, [15] patients with endometrial adenocarcinoma were found to have the highest expression of Ki67, with most cases falling between 1 and 25 percent. Ki67 was reported to express itself at its highest level in postmenopausal women over the age of 50.

**Conclusion:**

Identification of atypia in endometrial hyperplasia is important so that appropriate measures can be taken to prevent the progression of the condition into carcinoma. The expression of ER, PR was seen in cases of endometrial hyperplasia and endometriosis adenocarcinoma (type1) without the expression of p53. In contrast, p53 was seen to positive with negative expression of ER and PR in cases of Clear cell carcinoma (type 2). Ki67 was less than 10% in typical hyperplasia, between 10-20% in atypical hyperplasia and more than 20% in cases of carcinomas 3) Distinguishing between typical and atypical hyperplasia is necessary as the latter is a precancerous lesion and requires appropriate intervention. High ER, PR along with low expression of p53 was seen in type 1 endometriosis carcinoma as compared to low ER, PR and high p53 intype2 carcinoma.

**Conflict of Interest:** Nil

**Source of funding:** Nil

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**How to cite this article:** Nishtha Danu, Kunda Jagadale, Smita Mulay, Anuja Mohan Patil, R.C. Nimbargi and Anish Jagadale,
The Role of Immunohistochemistry in Hyperplastic Endometrial Disorders and Endometrial Carcinoma Cases: A Cross Sectional Observational Study.
Walawalkar International Medical Journal 2023;10(1):17-23.
http://www.wimjournal.com.

Received date: 06/05/2024 Revised date: 11/07/2024 Accepted date: 12/07/2023