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Original Article

Pathology Section

Expression of E-cadherin in Endometrial Hyperplasia and Endometrioid Endometrial Carcinoma: A Cross-sectional Immunohistochemical Study

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ABSTRACT

Introduction: Endometrial carcinoma is one of the most common gynaecological malignancies and is often preceded by endometrial hyperplasia, representing a spectrum of disease progression. The transition from hyperplasia to carcinoma involves complex molecular mechanisms, among which Epithelial-Mesenchymal Transition (EMT) plays a critical role. E-cadherin, a key cell adhesion molecule, is essential for maintaining epithelial integrity, and its loss has been strongly associated with tumour invasion, metastasis, and dedifferentiation. However, its expression pattern across premalignant and malignant endometrial lesions and its clinicopathological significance remain incompletely understood.

Aim: To evaluate E-cadherin expression in endometrial hyperplasia and endometrioid endometrial carcinoma and to assess its association with histopathological parameters.

Materials and Methods: The present cross-sectional descriptive observational study with retrospective and prospective components was conducted over a period of 19 months from April 2024 to November 2025 in the Department of Pathology at Meenakshi Medical College Hospital and Research Institute, Kanchipuram, Tamil Nadu, India. A total of 38 paraffin-embedded tissue samples were analysed, including endometrial hyperplasia (n=15) and endometrioid endometrial carcinoma (n=23). The clinicopathological parameters studied included age, type of surgical procedure, histological type (hyperplasia with/without atypia and carcinoma), tumour grade, International Federation of Gynecology and Obstetrics (FIGO) stage, Tumour, Node, and Metastasis (TNM) stage, depth of myometrial invasion, and Lymphovascular Invasion (LVI). Immunohistochemical evaluation of E-cadherin expression was performed using a semi-quantitative scoring system based on staining intensity and proportion of positively stained cells. Statistical analysis was carried out using Statistical Package for the Social Sciences (SPSS) version 22, applying Student's t-test and Chi-square/Fisher's exact test, with $p<0.05$ considered statistically significant.

Results: The mean age of patients with endometrial carcinoma was significantly higher than that of patients with endometrial hyperplasia (57.09±10.04 years vs 45.47±8.34 years; p=0.005). Among carcinoma cases (n=23), FIGO Grade 2 was the most common (11/23, 47.83%), with Stage IB predominance (11/23, 47.83%). E-cadherin expression was preserved in all cases of endometrial hyperplasia (15/15, 100%), whereas only three of 23 carcinoma cases (13.04%) showed positive expression. Loss of E-cadherin expression demonstrated significant association with higher tumour grade (p=0.0394), deeper myometrial invasion (p=0.0383), and presence of LVI (p=0.0129).

Conclusion: Loss of E-cadherin expression is significantly associated with adverse pathological features in endometrioid endometrial carcinoma and may serve as a potential indicator of tumour aggressiveness; however, its definitive prognostic role requires validation in larger studies with survival analysis.

Keywords: Cadherin switching, Cell adhesion molecules, Endometrial neoplasia, Epithelial-mesenchymal transition, Histopathological correlation, Immunohistochemical markers, Tumour invasion

INTRODUCTION

Endometrial carcinoma is the most common malignant tumour of the female reproductive tract, with the endometrioid subtype accounting for the majority of cases. It typically develops through a well-recognised sequence beginning with endometrial hyperplasia, which may progress to endometrioid endometrial carcinoma, although the underlying molecular mechanisms governing this transformation remain incompletely understood [1,2]. One of the key processes implicated in this progression is EMT, a biological phenomenon in which epithelial cells lose their polarity and adhesion properties and acquire mesenchymal characteristics, enhancing their migratory and invasive potential [2].

E-cadherin, a transmembrane cell adhesion molecule, plays a critical role in maintaining epithelial integrity and tissue architecture. Loss or reduction of E-cadherin expression disrupts intercellular adhesion and has been strongly associated with tumour invasion, metastasis, and dedifferentiation in various malignancies, including endometrial carcinoma [3-5]. Several studies have demonstrated that decreased E-cadherin expression correlates with higher tumour grade, advanced stage, and aggressive tumour behaviour, highlighting its importance in tumour progression and potential clinical relevance [3-5].

N-cadherin, a mesenchymal adhesion molecule, has been associated with increased migratory and invasive potential of cancer cells. The phenomenon of "cadherin switching," characterised by the loss of E-cadherin and gain of N-cadherin expression, is recognised as a key feature of EMT and tumour progression [6]. In endometrial lesions, studies have demonstrated reduced E-cadherin expression along with increased N-cadherin expression in carcinoma compared to hyperplasia and normal endometrium, supporting its role in malignant transformation and tumour aggressiveness [5,7,8].

Immunohistochemical expression of E-cadherin through the range of hyperplasia (without/with atypia), and EEC could provide substantial insights of tumour progression and prognostic stratification.

Besides, linking their expression with clinicopathological features can elevate the risk evaluation to another level and even suggest the direction of management by means of differentially targeted therapies.

Despite growing evidence on the role of cadherins in tumour progression, limited studies have comprehensively evaluated E-cadherin expression across both premalignant and malignant endometrial lesions within a single study population, particularly in resource-limited settings. Furthermore, the association of E-cadherin expression with key histopathological parameters such as tumour grade, myometrial invasion, and LVI remains inadequately characterised in the Indian context. Therefore, the present study was undertaken to evaluate the immunohistochemical expression of E-cadherin in endometrial hyperplasia and endometrioid endometrial carcinoma, and to assess its association with clinicopathological parameters. The present study aimed to contribute to a better understanding of tumour progression and to explore the potential utility of E-cadherin as an indicator of tumour aggressiveness.

MATERIALS AND METHODS

The present cross-sectional descriptive study with retrospective and prospective components was conducted over a period of 19 months from 30 April 2024 to 30 November 2025 in the Department of Pathology, Meenakshi Medical College Hospital and Research Institute, Kanchipuram, Tamil Nadu, India. The study was conducted in accordance with the principles of the Declaration of Helsinki and was approved by the Institutional Ethics Committee (IEC No: MMCH&RI IEC/ PG/02/MAY/24). Informed consent was obtained from participants where applicable, and patient confidentiality was strictly maintained by anonymising all data.

The study included women aged 35-75 years who underwent total abdominal hysterectomy with or without bilateral salpingo-oophorectomy.

The sample size was determined based on the availability of eligible cases during the study period, as this was a time-bound, Institution-based study; no formal sample size calculation was performed, which is acknowledged as a limitation.

Inclusion and Exclusion criteria: The study included women aged 35-75 years with histopathologically confirmed endometrial hyperplasia (with or without atypia) or endometrioid endometrial carcinoma who underwent endometrial biopsy or total abdominal hysterectomy, with or without bilateral salpingo-oophorectomy. A total of 38 formalin-fixed paraffin-embedded tissue blocks were analysed for E-cadherin expression, comprising 15 cases of endometrial hyperplasia and 23 cases of endometrioid endometrial carcinoma. Cases of non-endometrioid endometrial malignancies, patients who had received prior chemotherapy or radiotherapy, and specimens with inadequate or poorly preserved tissue were excluded.

Study Procedure

Clinicopathological variables including age, procedure performed, tumour location and size, histological type and grade, FIGO stage, TNM stage, depth of myometrial invasion, and LVI were retrieved from departmental records and cancer registries. Tumours were staged according to the FIGO staging system and the TNM classification (Berek JS et al., 2023; AJCC, 2017) [9,10].

From each selected paraffin block, 4-5 µm thick sections were prepared for immunohistochemical analysis. Standard protocol included deparaffinisation, rehydration, heat-induced antigen retrieval, blocking of endogenous peroxidase activity, incubation with primary antibody, application of secondary antibody, and detection using 3,3′-Diaminobenzidine (DAB) chromogen, followed by haematoxylin counterstaining. Positive and negative controls were included in each staining batch. The primary antibody used was E-cadherin-EP6, Rabbit Monoclonal Antibody, manufactured by Pathin Situ, dilution 1:100.

Sections were examined under light microscopy, and E-cadherin immunoreactivity was evaluated using a semi-quantitative scoring system based on staining intensity and the proportion of positively stained cells. The proportion score ranged from 1 (0-25% positive cells) to 4 (76-100% positive cells). The staining intensity was graded from 0 to 3 in the present study. The final Immunoreactive Score (IRS) was calculated and categorised as negative (0-3) and positive (≥4). The scoring approach was based on the IRS system originally described by Remmele and Stegner (1987) [4]. In carcinoma cases, E-cadherin expression was further analysed for its association with histological grade, depth of myometrial invasion, LVI, and FIGO stage.

STATISTICAL ANALYSIS

Data were entered into Microsoft Excel and analysed using Statistical Package for the Social Sciences (SPSS) software, version 22.0 (IBM Corp., Armonk, NY, USA). Continuous variables such as age were expressed as mean±standard deviation, while categorical variables were presented as frequencies and percentages. Comparisons between groups were performed using the Student's t-test for continuous variables and the Chi-square test or Fisher's exact test for categorical variables, as appropriate. The association between E-cadherin expression and clinicopathological parameters such as histological grade, depth of myometrial invasion, LVI, and FIGO stage was analysed using these tests. A p-value of <0.05 was considered statistically significant.

RESULTS

The mean age of patients in the endometrial hyperplasia group (n=15) was 45.47±8.34 years, whereas patients with endometrioid endometrial carcinoma (n=23) were significantly older, with a mean age of 57.09±10.04 years (p=0.005).

Endometrial biopsy was performed in 8/15 cases (53.33%) of hyperplasia and 6/23 cases (26.09%) of carcinoma, while total abdominal hysterectomy with bilateral salpingo-oophorectomy (TAH + BSO) was more frequently performed in carcinoma cases (17/23; 73.91%) compared to hyperplasia (7/15; 46.67%). However, this difference was not statistically significant (p=0.0931). Among hyperplasia cases, 8/15 (53.33%) were without atypia and 7/15 (46.67%) were with atypia [Table/Fig-1].

Parameters		Endometrial hyperplasia (n=15)	Endometrial carcinoma (n=23)	p-value
Age (years)		45.47±8.34	57.09±10.04	0.0050*
Procedure	Endometrial biopsy	8 (53.33%)	6 (26.09%)	0.0931
	TAH + BSO	7 (46.67%)	17 (73.91%)	
Histological Type	Hyperplasia without atypia	8 (53.33%)	-	-
	Hyperplasia with atypia	7 (46.67%)	-	

[Table/Fig-1]: Clinical profile of the study population (N=38).

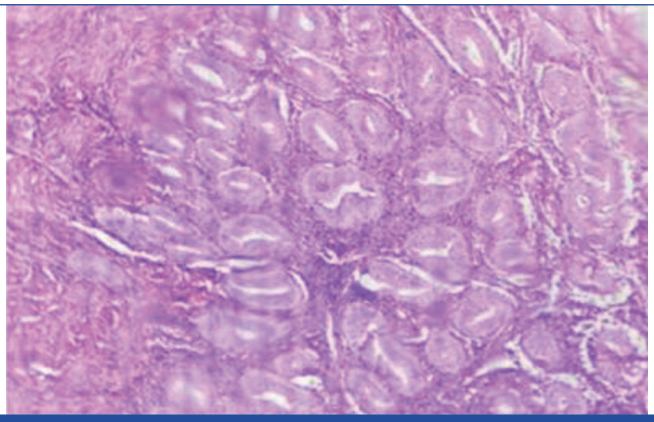
Among the 23 endometrial carcinoma cases, FIGO Grade 2 was the most common 11 (47.83%), followed by Grade 1 in 8 (34.78%) and Grade 3 in 4 (17.39%). FIGO staging revealed Stage IA in 9 (39.13%) Stage IB in 11 (47.83%), and Stage II or above in 3 (13.04%). TNM staging showed pT1aN0Mx in 9 (39.13%), pT1bN0Mx in 11 (47.83%), and pT2N0Mx in 3 (13.04%). Myometrial invasion was <50% in 10 (43.48%) and ≥50% in 13 (56.52%). LVI was present in 15 (65.22%) and absent in 8 (34.78%) [Table/Fig-2].

Haematoxylin and Eosin (H&E), 40× images demonstrate endometrial hyperplasia without atypia showing proliferative glands with preserved architecture and absence of cytological atypia

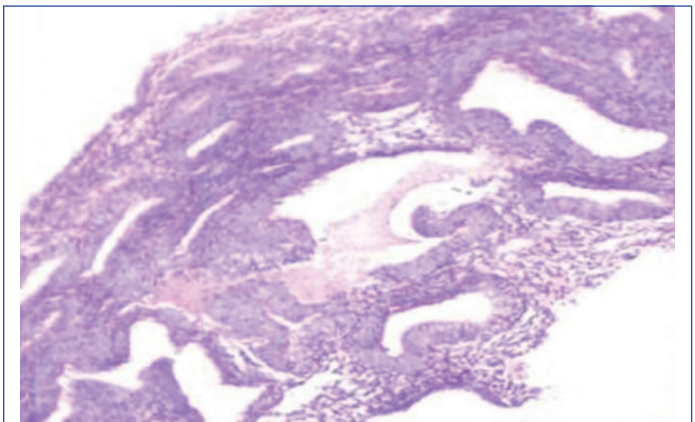
Pathological profile		Endometrial carcinoma
FIGO Grade	Grade 1	8 (34.78%)
	Grade 2	11 (47.83%)
	Grade 3	4 (17.39%)
FIGO Stage	IA	9 (39.13%)
	IB	11 (47.83%)
	II or above	3 (13.04%)
TNM Stage	pT1aN0Mx	9 (39.13%)
	pT1bN0Mx	11 (47.83%)
	pT2N0Mx	3 (13.04%)
Myometrial Invasion	<50%	10 (43.48%)
	≥50%	13 (56.52%)
Lymphovascular Invasion (LVI)	Present	15 (65.22%)
	Absent	8 (34.78%)

[Table/Fig-2]: Pathological profile of participants with endometrial carcinoma (n=23).

[Table/Fig-3], and endometrial hyperplasia with atypia showing glandular crowding with cytological atypia and nuclear stratification [Table/Fig-4]. Endometrioid endometrial carcinoma shows irregular glandular architecture with stromal invasion and cytological atypia [Table/Fig-5]. Immunohistochemistry (IHC), 40× images demonstrate strong membranous E-cadherin expression in glandular epithelial cells in hyperplasia without atypia [Table/Fig-6], moderate membranous expression in hyperplasia with atypia [Table/Fig-7], and reduced to weak membranous E-cadherin expression in neoplastic glandular cells in endometrioid endometrial carcinoma [Table/Fig-8].

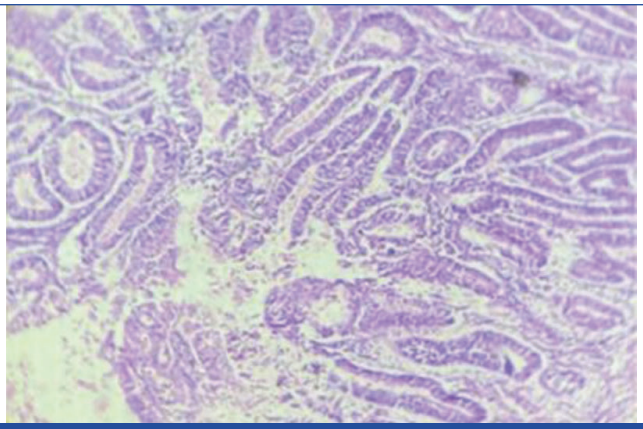


[Table/Fig-3]: Endometrial hyperplasia without atypia (H&E, 40x).



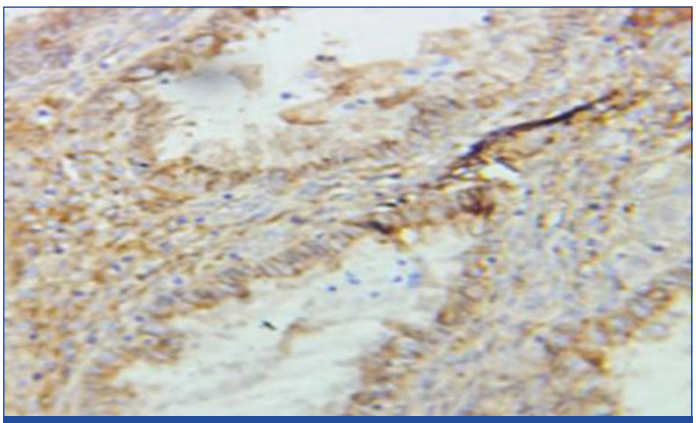
[Table/Fig-4]: Endometrial hyperplasia with atypia (H&E,40x).

E-cadherin expression was positive in all cases of endometrial hyperplasia, including 8/8 (100%) hyperplasia without atypia and 7/7 (100%) hyperplasia with atypia. In contrast, among endometrioid endometrial carcinoma cases, only 3/23 (13.04%) demonstrated positive E-cadherin expression, while 20/23 (86.96%) were negative [Table/Fig-9].

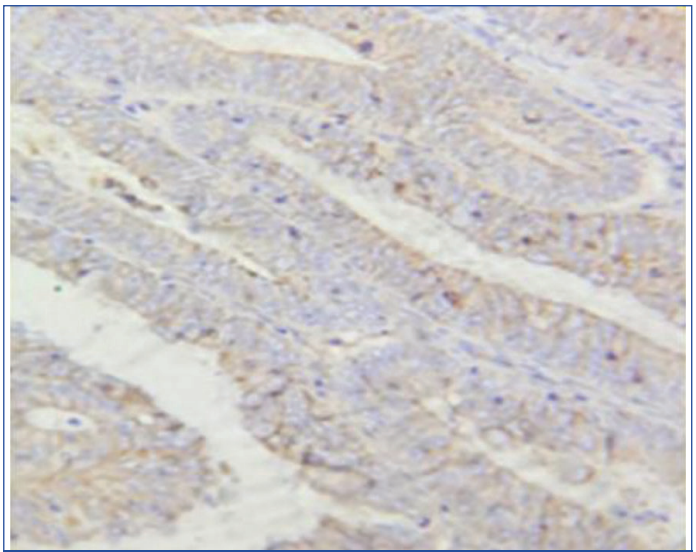


[Table/Fig-5]: Endometrioid endometrial carcinoma (H&E,40x).

[Table/Fig-6]: Endometrial hyperplasia without atypia showing strong membranous E-cadherin staining in the glandular epithelium (IHC, 40x).



[Table/Fig-7]: Endometrial hyperplasia with atypia showing moderate membranous E-cadherin staining in glandular epithelium (IHC, 40x).



[Table/Fig-8]: Endometrioid endometrial carcinoma showing very weak E-cadherin staining in some glandular epithelium (IHC, 40x).

Expression of E-cadherin	Endometrial hyperplasia without atypia (n=8)	Endometrial hyperplasia with atypia (n=7)	Endometrioid endometrial carcinoma (n=23)
Positive n (%)	8 (100%)	7 (100%)	3 (13.04%)
Negative n (%)	0 (0%)	0 (0%)	20 (86.96%)

[Table/Fig-9]: Expression of E-cadherin in endometrial hyperplasia (with and without atypia) and endometrioid endometrial carcinoma.

In histological grade, all E-cadherin positive cases (3/3, 100%) were observed in Grade 1 tumours, whereas none of the Grade 2 or Grade 3 tumours demonstrated E-cadherin positivity. Among E-cadherin negative cases (n=20), 5 (25%) were Grade 1, 11 (55%) were Grade 2, and 4 (20%) were Grade 3. This association between E-cadherin expression and histological grade was statistically significant (p=0.0394).

A significant association was also observed between E-cadherin expression and depth of myometrial invasion (p=0.0383). All E-cadherin positive tumours 3/3 (100%) showed superficial myometrial invasion (<50%), whereas none demonstrated deep invasion (≥50%). In contrast, among E-cadherin negative tumours (n=20), 7 (35%) exhibited <50% invasion, while 13 (65%) showed deep myometrial invasion (≥50%).

Lymphovascular invasion also demonstrated a statistically significant association with E-cadherin expression (p=0.0129). None of the tumours with LVI showed E-cadherin positivity 0/15 (0%), whereas all E-cadherin-positive tumours 3/3 (100%) were observed in cases without LVI. Among E-cadherin negative cases (n=20), 15 (75%) showed LVI, while 5 (25%) showed no evidence of LVI [Table/Fig-10].

Pathological profile		E-cadherin positive n (%) (n=3)	E-cadherin negative n (%) (n=20)	p-value
Histological grade	Grade 1	3 (100%)	5 (25%)	0.0394
	Grade 2	0 (0%)	11 (55%)	
	Grade 3	0 (0%)	4 (20%)	
Myometrial invasion	<50%	3 (100%)	7 (35%)	0.0383
	≥50%	0 (0%)	13 (65%)	
Lymphovascular Invasion (LVI)	Present	0 (0%)	15 (75%)	0.0129
	Absent	3 (100%)	5 (25%)	

[Table/Fig-10]: Association between E-cadherin expression and pathological profile in endometrioid endometrial carcinoma.
p<0.05 , significant

DISCUSSION

Endometrial carcinoma is one of the most common gynaecologic malignancies, ranking among the top female cancers worldwide. Global estimates indicate a substantial annual incidence, with markedly higher age-standardised rates in North America and Europe compared with many low and middle-income regions. In high-income countries, most cases are detected at an early, localised stage, and 5-year survival for localised disease approaches 95%, contributing to a generally favourable prognosis when diagnosed promptly [11,12]. The incidence of endometrial carcinoma is rising globally, largely due to increasing obesity rates and aging populations. Obesity remains the strongest modifiable risk factor, with risk increasing sharply with higher Body Mass Index (BMI), while diabetes, nulliparity, unopposed oestrogen exposure and other hormonal factors also contribute. Although countries such as India have historically reported lower incidence than Western regions, recent registry data indicate a gradual rise linked to lifestyle and demographic transitions. These trends highlight the need for public health strategies focused on reducing obesity and metabolic risk to curb the future burden of endometrial cancer [13-15].

Hypermethylation of E-cadherin in endometrial carcinoma reported by Park JH et al., demonstrated a significantly reduced E-cadherin expression in endometrial carcinoma compared to hyperplasia. Similarly, Carico E et al., showed that E-cadherin (along with α-catenin) expression progressively decreases from normal and hyperplastic endometrium to neoplastic (carcinoma) tissue [16,17]. The association between reduced E-cadherin expression and adverse tumour characteristics has been consistently documented. Park JH et al., reported that loss of E-cadherin correlates with tumour dedifferentiation and metastatic potential in endometrial carcinoma [16]. Frąszczak K et al., demonstrated a significant relationship between lower E-cadherin expression and higher histological grade, reinforcing its role as a marker of tumour aggressiveness [7].

Yalta T et al., reported that E-cadherin expression was significantly lower in non-endometrioid carcinomas than in endometrioid carcinomas and non-neoplastic controls, supporting the same pattern observed in the present study, that loss of E-cadherin increases with malignant transformation [18]. Lewczuk Ł et al., showed that hyperplastic endometrium demonstrated weak cytoplasmic E-cadherin staining, while endometrial carcinoma, especially metastatic and higher-grade disease exhibited markedly reduced or irregular E-cadherin expression. Diminished membranous staining at invasive tumour fronts and metastatic deposits. These findings align with the current study by supporting the role of E-cadherin loss in tumour progression and dissemination [8].

Reduced E-cadherin expression has been consistently associated with adverse pathological features in endometrial carcinoma, particularly higher histological grade. Multiple studies have demonstrated that loss of E-cadherin expression is more frequently observed in poorly differentiated tumours, reflecting tumour dedifferentiation and aggressive biological behaviour. Frąszczak K et al., reported a significant association between decreased E-cadherin expression and higher tumour grade [7], while Holcomb K et al., similarly observed that E-cadherin negative tumours were predominantly high-grade lesions [19]. These findings support the role of E-cadherin as a marker of tumour differentiation and progression.

Several studies have also explored the relationship between E-cadherin expression and depth of myometrial invasion. Decreased expression has been linked to deeper myometrial invasion, suggesting its role in tumour invasiveness and local spread. Sakuragi N et al., demonstrated that tumours with reduced E-cadherin expression showed a greater tendency for deep myometrial infiltration [20]. Similar observations have been reported in other studies, where altered E-cadherin expression was associated with increased tumour invasion and metastatic potential [21]. Although some studies have reported inconsistent or non significant associations between E-cadherin expression and LVI [22], the present study demonstrated a statistically significant association (p=0.0129), suggesting that loss of E-cadherin may contribute to tumour invasiveness and vascular dissemination.

E-cadherin expression has shown variable sensitivity and specificity across studies. Habib F et al., reported a sensitivity of approximately 81.9% but low specificity (28.6%) for detecting endometrial carcinoma, suggesting that while E-cadherin loss is a sensitive indicator of malignancy, it lacks specificity as a standalone diagnostic marker [23].

Limitation(s)

The present study is limited by a smaller sample size, lack of follow-up or survival data, which restricts assessment of prognostic significance. Additionally, absence of multivariate analysis limits the ability to determine independent associations between E-cadherin expression and pathological parameters.

CONCLUSION(S)

E-cadherin expression was significantly reduced in endometrioid endometrial carcinoma compared to endometrial hyperplasia and is strongly associated with adverse pathological features, including

higher tumour grade, deeper myometrial invasion, and LVI. These findings suggest that loss of E-cadherin may serve as a useful prognostic biomarker and indicator of tumour aggressiveness in endometrial carcinoma.

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