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ORIGINAL ARTICLE

Immunohistochemical Profiles of p53 and ER in Relation to Lesion Type, Tumor Grade, and Pathological Features in Endometrial Cancer

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SUMMARY

Background: **The goal was to assess the pathological measurements of estrogen receptor (ER) and p53 expression in tissues of uterine endometrial carcinoma and their relationship to various clinicopathologic parameters that influence prognosis.**

Methods: **The current study included 40 cases of endometrial carcinoma in total hysterectomy samples obtained from the preservation documents of the Al Azhar University Hospitals along with certain privately operated laboratories between April 2023 and April 2025. Each of the samples was formalin-fixed, subsequently processed and placed inside paraffin blocks. Several serial 5-micron thick sections were extracted from the samples' paraffin blocks; one was stained with hematoxylin and eosin for histopathological analysis to figure out tumor histology, grade, and lymphovascular invasion, whereas the remaining sections were put on positively charged slides and immunostained with mouse monoclonal antibodies against p53, as well as rabbit monoclonal antibodies against ER.**

Results: **The mean age of the study group was 52.20 ± 12.25 years ranging from 38 - 69; 25 (62.5%) had type I lesions and 15 (37.5%) had type II lesions. Twenty-six (65.0%) had wild (normal) IHC: p53 and 14 (35.0%) had mutant (abnormal) IHC: p53. Eleven (27.5%) were negative for IHC: ER and 29 (72.5%) were positive for IHC: ER. There was highly statistically significant association between IHC: p53 and type of lesions (p = 0.001) and statistically significant association between IHC: p53 and grade of pathology (p = 0.007). There were highly statistically significant associations between IHC: ER and lesions (p = 0.001), lymphovascular invasion (p = 0.001) and statistically significant association between IHC: ER and grade of pathology (p = 0.013). There was statistically significant association between IHC: p53 and IHC: ER (p = 0.014).**

Conclusions: **p53 and ER markers were discovered to have a pathological significance in EC. There is a link among these markers and other clinicopathological predictive measures, suggesting that they could serve as possibly beneficial biomarkers.**

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KEYWORDS

endometrial carcinoma, estrogen receptor, immunohistochemistry, p53

INTRODUCTION

Endometrial cancer (EC) is a particularly prevalent gynecological malignant tumor in advanced nations, and its prevalence had been rising in the past few years, causing an enormous medical stress on individuals as well as healthcare facilities [1].

The prognosis of endometrial cancer is determined by the stage, histology, grade, and ethnic origin of the woman. Diagnostic algorithms integrating immunohistochemical indicators and molecular examinations relevant to formalin-fixed, paraffin-embedded tumor tissue have been suggested and evaluated [2].

Estrogen receptor (ERα) is an essential driver of endometrial tumor development, with three main aspects. Estrogen receptor ERα transcriptional activity is influenced by upstream regulators, which can impact EC progression and cell proliferation. ERα and other co-regulators promote the establishment of EC. ERα controls EC advancement, metastasis, and apoptosis through downstream proteins and particular genes [3]. P53 possesses items that serve to combat all of the characteristic features of tumors, that could be ignored or reversed by mutation, suppression, or repression in tumors cells [4]. The accurate determination of p53 IHC is of paramount importance for the molecular EC classification screening algorithm because it has a significant impact on a patient's individual risk estimation as well as the following therapy [5].

The purpose of this study was to assess the pathological measurements of ER, p16, and p53 expression in tissues of uterine endometrial carcinoma and their relationship to various clinicopathologic parameters that influence prognosis.

MATERIALS AND METHODS

The current study included 40 cases of endometrial carcinoma in total hysterectomy samples obtained from the preservation documents of Al Azhar University Hospitals along with certain privately operated laboratories between March 2023 and February 2025. The study was done according to Al Azhar University ethical committee guidelines (approval no. 73-2/2023).

Each of the samples was formalin-fixed, subsequently processed and placed inside paraffin blocks. Several serial 5-micron thick sections were extracted from the samples' paraffin blocks; one was stained with hematoxylin and eosin for histopathological analysis to figure out tumor histology, grade, and lymphovascular invasion, whereas the remaining sections were put on positively charged slides and immunostained with mouse monoclonal antibodies against p53, as well as rabbit monoclonal antibodies against ER.

Clinicopathological data

Included age, clinical presentation, tumor size, and histopathological type and metastasis of lymph nodes. Histopathological data done by hematoxylin and eosin-stained paraffin sections inspection by microscopy to reconsider: EC grading by FIGO grading system [6].

Immunohistochemical assessments

Three Biogenix slides (positively charged) were created from each paraffin block for immunostaining with mouse monoclonal anti-p53 antibody (clone DO-7) and rabbit monoclonal anti-estrogen receptor antibody (clone SP1). Immunohistochemical reactions were performed with the labelled Streptavidin-Biotin2 System-Horseradish Peroxidase (LSAB2 System-HRP). The LSAB2 System, HRP relies on a modified labelled Avidin-Biotin (LAB) approach during which a biotinylated secondary antibody binds to peroxidase-conjugated streptavidin molecules.

Controls

For p53 immunostaining, a section of colonic adenocarcinoma should show diffuse positive nuclear staining in a significant number of neoplastic cells, as shown by Köbel et al. [7] (Figure 1).

For ER immunostaining, normal endometrial glands and stroma were used as a positive control due to their nuclear expression of these receptors (Figure 2) according to Salama et al. [8].

Interpretation

According to Köbel et al. [7], p53 nuclear staining was classified into the following types [6]:

Wild-type (normal) pattern is distinguished by dispersed nuclear staining, mid epithelial (basal sparing) and nearly all normal tissues expressing wild-type expression of p53

Mutated type (abnormal) pattern involved

- Strong and diffuse nuclear staining in 80% of the cells,
- Complete lack of nuclear staining in all cells,
- Or moderate to strong cytoplasmic staining.

Nuclear staining for ER was scored based on Allred score and the intensity and percentage of positive tumor cells according to Odetola et al. [9] as follows:

The intensity of staining (IS) is graded from 0 to 3. A score of zero indicates no staining, one indicates weak staining, two indicates moderate staining, and three indicates strong staining. The percentage of staining (PS) is as follows: nuclear staining levels are classified as 0, < 1%, 1 - 10%, 11 - 33%, 33 - 66%, and 67 - 100%. The total score was determined by the addition of IS and PS. The top possible score is eight:

- Negative (0 and 2)
- Positive (3, 4, 5, 6, 7, and 8)

Table 1. Clinicopathological and histopathological data of the study population.

	No.	%
Age (years)		
≤ 50 years	**14**	**35.0%**
> 50 years	**26**	**65.0%**
Type of lesions		
Type I	**25**	**62.5%**
Endometrioid adenocarcinoma	**25**	**62.5%**
Type II	**15**	**37.5%**
High grade serous carcinoma	**6**	**15.0%**
Clear cell adenocarcinoma	**5**	**12.5%**
Malignant mixed mullerian (carcinosarcoma)	**4**	**10.0%**
Myometrial invasion		
< 50%	**8**	**20.0%**
> 50%	**32**	**80.0%**
Cervical involvement		
Negative	**36**	**90.0%**
Positive	**4**	**10.0%**
Lymphovascular invasion		
Negative	**30**	**75.0%**
Positive	**10**	**25.0%**
IHC: p53		
Wild (normal)	**26**	**65.0%**
Mutant (abnormal)	**14**	**35.0%**
Diffuse nuclear	**8**	**20.0%**
Complete absence (null)	**6**	**15.0%**
Cytoplasmic	**0**	**0.0%**
IHC: ER		
Negative	**11**	**27.5%**
Positive	**29**	**72.5%**

Statistical analyses

Recorded data were analyzed using the Statistical Package for Social Sciences, version 23.0 (IBM SPSS Statistics for Windows; IBM Corp., Armonk, NY, USA). The quantitative data were presented as mean ± standard deviation and ranges when their distribution was parametric (normal) while non-normally distributed variables (non-parametric data) were presented as median with inter-quartile range (IQR). Also, qualitative variables were presented as number and percentages. Data were explored using Student's t-test and chi-squared.

RESULTS

The mean age of the study group was 52.20 ± 12.25 years ranging from 38 - 69; 26 (65.0%) were > 50 years and 14 (35.0%) were ≤ 50 years. Regarding distribution of lesions distribution, 25 (62.5%) of the study group had type (I) lesions and 15 (37.5%) had type (II) lesions. Of type (II) lesions; 6 (15%) had high grade serous carcinoma, 5 (12.5%) had clear cell adenocarcinoma and 4 (10.0%) had malignant mixed Mullerian (carcinosarcoma). Thirty-two (80.0%) of the study group had myometrial invasion > 50% and 8 (20.0%) had myometrial invasion < 50%. Thirty-six (90.0%) of the study group had negative cervical involvement and 4 (10.0%) had positive cervical involvement. Thirty (75.0%) of the study group had negative LVSI and 10 (25.0%) had positive LVSI. Twenty-six (65.0%) of the

Table 2. Association between IHC: p53 and Clinicopathological parameters.

Age	IHC: p53						x^2	p-value	Sig.
	Diffuse nuclear (n = 8)		Complete absence (null) (n = 6)		Wild (normal) (n = 26)				
	No.	%	No.	%	No.	%			
≤ 50 years	2	25.0%	4	66.67%	8	30.77%	2.013	0.354	NS
> 50 years	6	75.0%	2	33.33%	18	69.23%			
Type of lesions									
Endometrioid adenocarcinoma	4	50.0%	1	16.67%	20	76.9%	50.762	0.001	HS
High grade serous carcinoma	2	25.0%	2	33.33%	2	7.7%			
Clear cell adenocarcinoma	2	25.0%	1	16.67%	2	7.7%			
Malignant mixed mullerian (carcinosarcoma)	0	0.0%	2	33.33%	2	7.7%			
Type I	4	50.0%	1	16.67%	20	76.9%	28.568	0.001	HS
Type II	4	50.0%	5	83.33%	6	23.1%			
Myometrial invasion									
< 50%	1	12.5%	3	50.0%	4	15.38%	0.135	0.922	NS
> 50%	7	87.5%	3	50.0%	22	84.62%			
Cervical involvement									
Negative	7	87.5%	6	100.0%	23	88.46%	0.046	0.948	NS
Positive	1	12.5%	0	0	3	11.54%			
Lymphovascular invasion									
Negative	4	50.0%	6	100.0%	20	76.9%	0.362	0.796	NS
Positive	4	50.0%	0	0	6	23.1%			
Grade of pathology									
Grade I	0	0	0	0	2	7.7%	13.535	0.007	S
Grade II	1	12.5%	0	0	14	53.8%			
Grade III	7	87.5%	6	100.0%	10	38.5%			

x^2 - chi-squared test for Number (%) or Fisher's exact test, when appropriate.
NS - Non significant, S - Significant, HS - Highly significant.

study group had wild (normal) IHC: p53 and 14 (35.0%) had mutant (abnormal) IHC: p53 including 8 (20.0%) diffuse nuclear, 6 (15.0%) null and 0 (0.0%) cytoplasmic. Eleven (27.5%) of the study group were negative for IHC: ER and 29 (72.5%) were positive for IHC: ER (Table 1, Figure 3).

There was highly statistically significant association between IHC: p53 and type of lesions (p = 0.001), none had statistically significant association between IHC: p53 and age (p = 0.354), myometrial invasion (p = 0.922), cervical involvement (p = 0.948), lymphovascular invasion (p = 0.796), and statistically significant association between IHC: p53 and grade of pathology (p = 0.007) (Table 2).

There was no statistically significant association between IHC: ER and age (p = 0.421), myometrial invasion (p = 0.851), cervical involvement (p = 0.130), statistically significant association between IHC: ER and type of lesions (p = 0.012), (p = 0.001). There was statistically significant association between IHC: ER and grade of pathology (p = 0.013), highly statistically significant association between IHC: ER and lymphovascular invasion (p = 0.001) (Table 3).

Table 4 shows statistically significant association between IHC: p53 and IHC: ER (p = 0.014).

DISCUSSION

Endometrial cancer is a cancer that develops in the uterine epithelial lining [10]. Endometrial cancer had traditionally been categorized as type 1 or type 2 founded on histological features [11].

The use of biomarkers (such as immunohistochemical

Table 3. Association between IHC: ER and Clinicopathological parameters.

Age	IHC: ER				x^2	p-value	Sig.
	Negative (n = 11)		Positive (n = 29)				
	No.	%	No.	%			
≤ 50 years	4	36.36%	11	37.9%	0.675	0.421	NS
> 50 years	7	63.64%	18	62.1%			
Type of lesions							
Endometrioid adenocarcinoma	10	91.0%	15	51.72%	14.144	0.012	S
High grade serous carcinoma	0	0.0%	6	20.69%			
Clear cell adenocarcinoma	0	0.0%	5	17.24%			
Malignant mixed mullerian (carcinosarcoma)	1	9.0%	3	10.35%			
Type I	10	91.0%	15	51.72%	12.364	0.001	HS
Type II	1	9.0%	14	48.28%			
Myometrial invasion							
> 50%	2	18.18%	6	20.69%	0.017	0.851	NS
< 50%	9	81.82%	23	79.31%			
Cervical involvement							
Negative	9	81.82%	26	89.66%	2.410	0.130	NS
Positive	2	18.18%	3	10.34%			
Lymphovascular invasion							
Negative	9	81.82%	21	72.41%	12.910	0.001	HS
Positive	2	18.18%	8	27.59%			
Grade of pathology							
Grade I	0	0	3	10.34%	9.025	0.013	S
Grade II	1	9.1%	13	44.83%			
Grade III	10	90.9%	13	44.83%			

x^2 - chi-squared test for Number (%) or Fisher's exact test, when appropriate.
NS - Non significant, S - Significant, HS - Highly significant.

Table 4. Association between IHC: p53 and IHC: ER.

IHC: p53	IHC: ER				Total		x2	p-value	Sig.
	Negative		Positive						
	No.	%	No.	%	No.	%			
Diffuse nuclear	2	18.18%	6	20.69%	8	20.0%	7.190	0.014	S
Complete absence (null)	0	0.0%	6	20.69%	6	15.0%			
Wild (normal)	9	81.82%	17	58.62%	26	65.0%			
Total	11	100.0%	29	100.0%	40	100.0%			

x^2 - chi-squared test for Number (%) or Fisher's exact test, when appropriate.
NS - Non significant, S - Significant, HS - Highly significant.

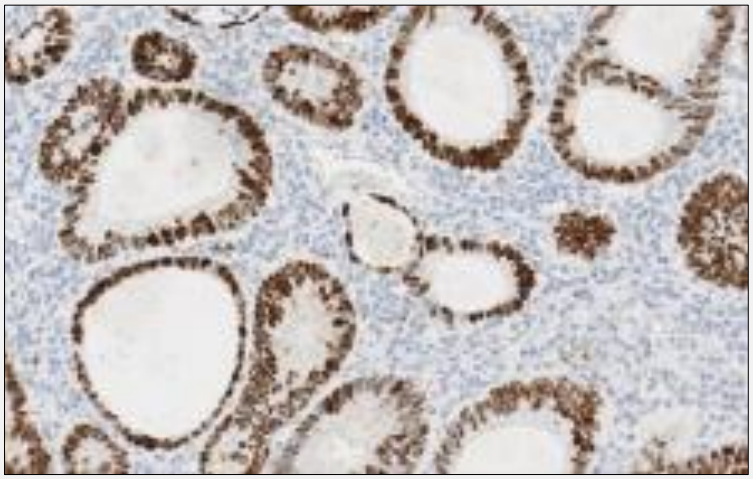
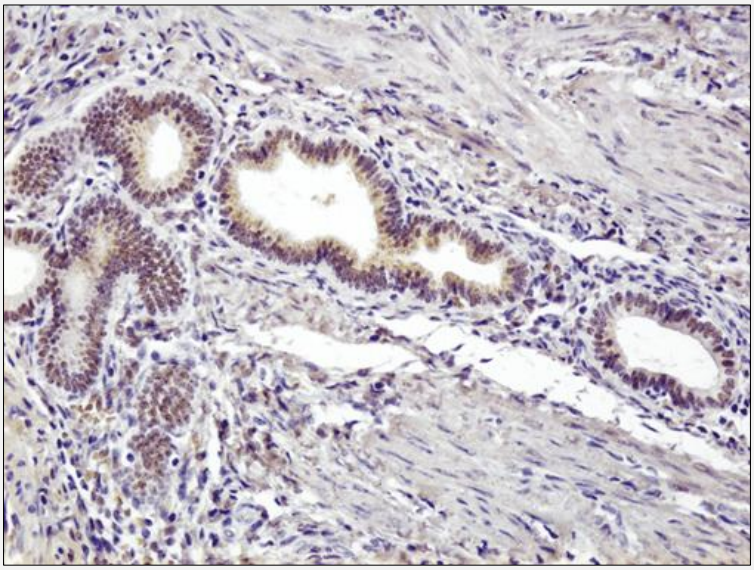


Figure 1. Adenocarcinoma of the colon exhibiting a strong mutated nuclear staining.



Antibody	Species	Clone name	Localization	Catalog number	Ordering code	Quantity	Controls	Isotypes
p53	mouse monoclonal	(DO-7)	nuclear	790 - 2912	05278074001	50 tests	colon adenocarcinoma	IgG1/K
ER	rabbit monoclonal	(SP1)	nuclear	790 - 4324	05278406001	50 tests	breast carcinoma, normal endometrium	IgG

Figure 2. Positive control for ER in cases of invasive mammary carcinomas.

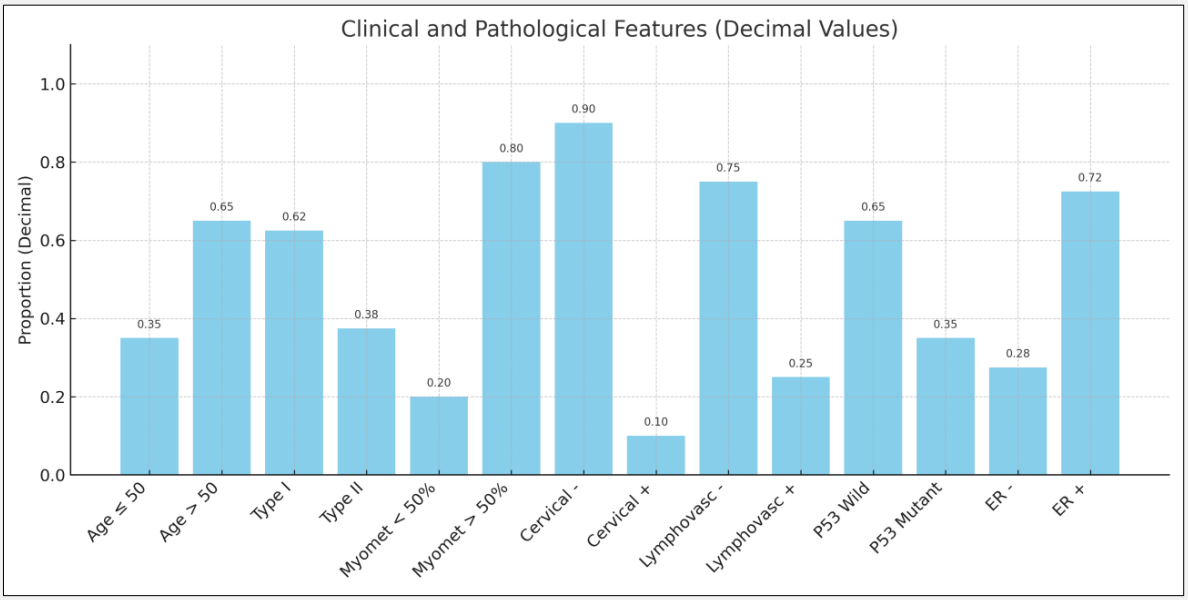


Figure 3. Clinicopathological and histopathological data of the study population.

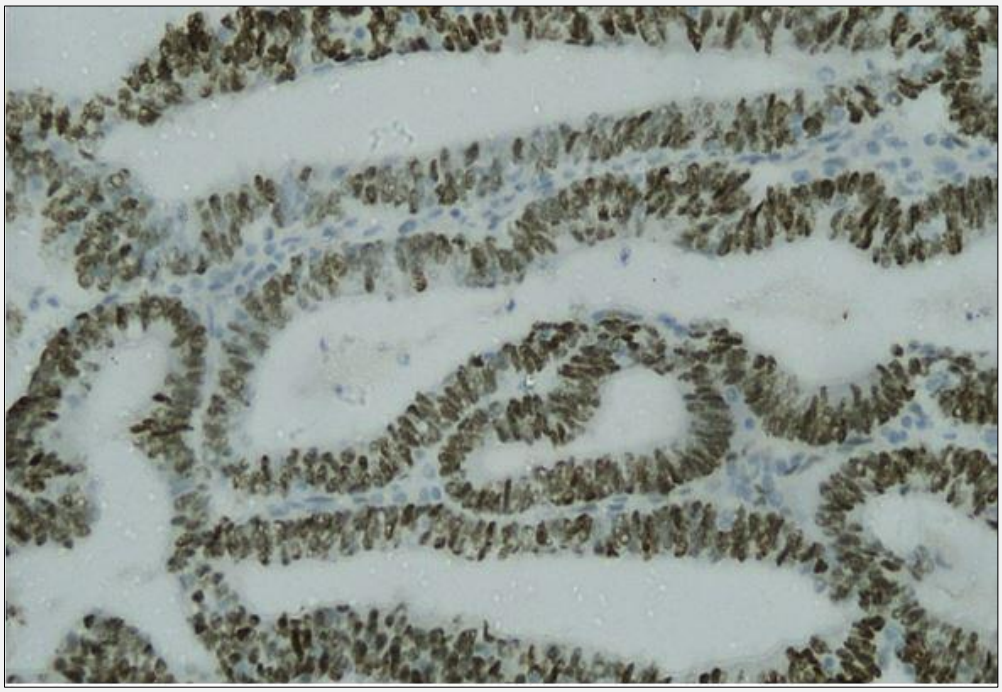


Figure 4. Endometrioid carcinoma well differentiated: IHC staining of ER shows strong nuclear staining of tumor cells (200x).

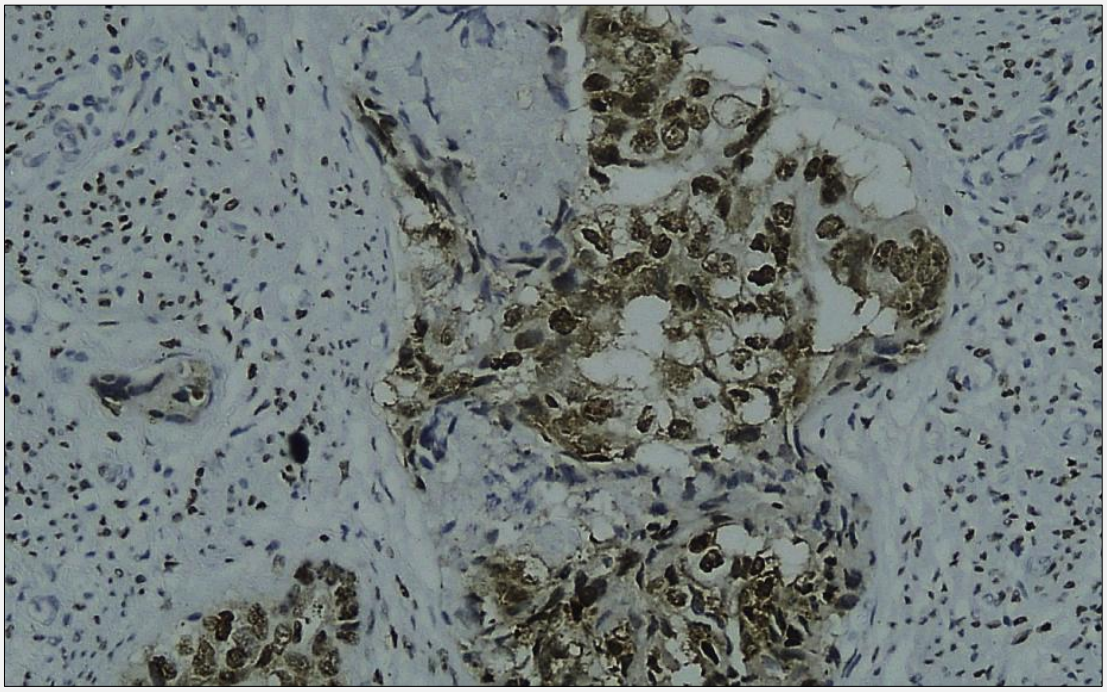


Figure 5. Endometrioid carcinoma moderately differentiated: IHC staining of ER shows Strong nuclear staining of tumor cells (200x).

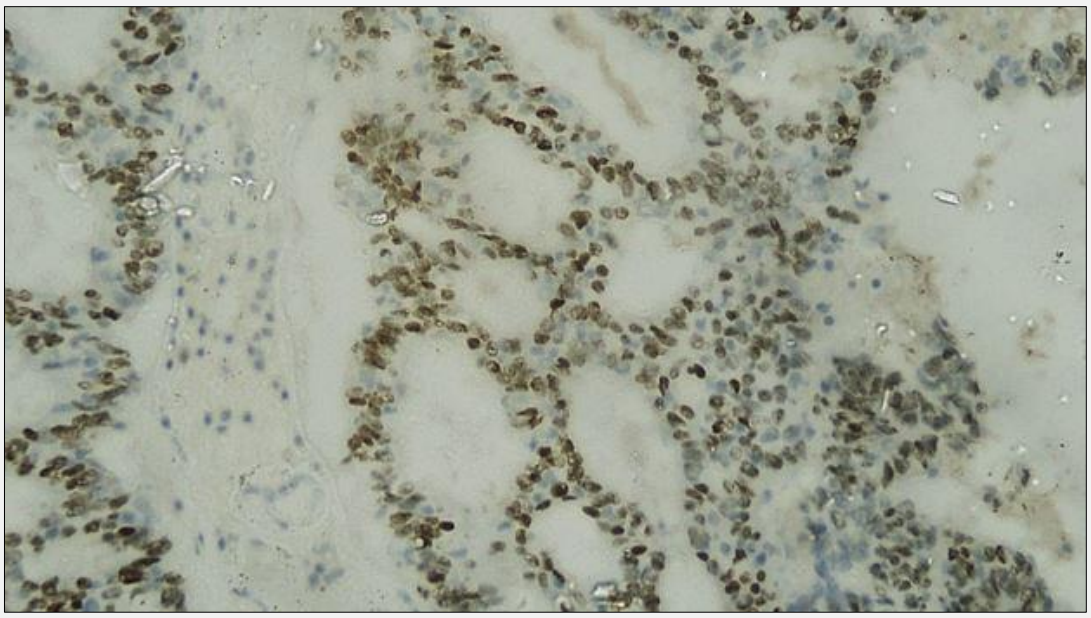


Figure 6. Endometrioid carcinoma: IHC staining of p53 shows nuclear staining of tumor cells (200x).

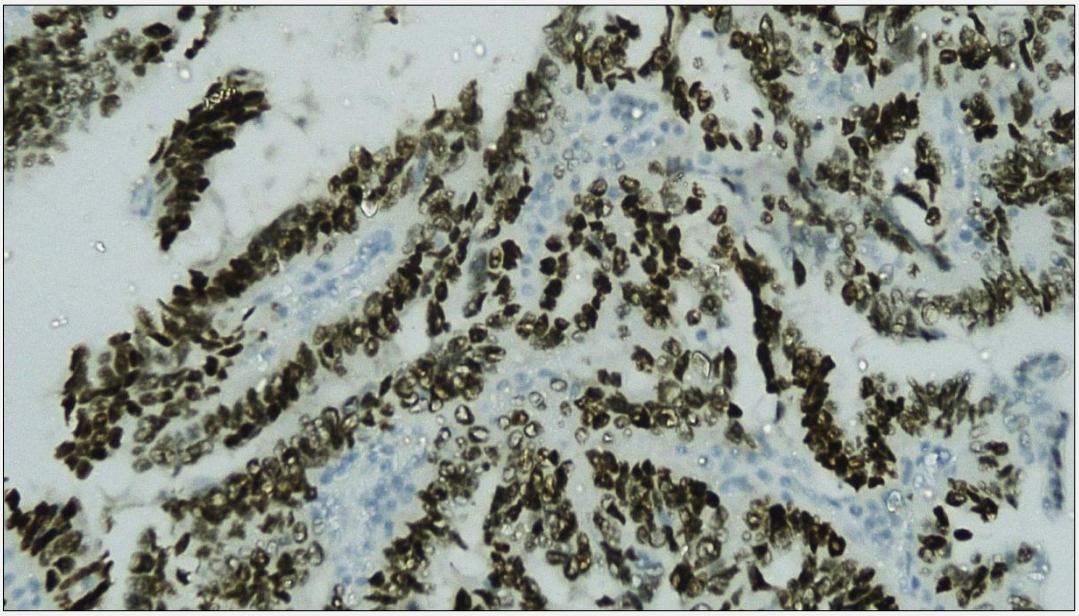


Figure 7. Serous carcinoma of endometrium: IHC staining of p53 shows strong diffuse nuclear staining of tumor cells (200x).

staining) supports pathologists in making diagnoses with greater precision. A past research investigation determined that as much as forty percent of cases originally identified as high-grade endometrial endometrioid adenocarcinomas can be reclassified as uterine serous carcinomas by using an array of four immunostains (p53, p16, estrogen receptor (ER), and mammaglobin) [12].

The present study involved 40 cases of EC with mean age 52.20 ± 12.25 years and 65.0% of cases were > 50 years old. This finding was consistent with Salim et al. [13] who reported that the mean age of EC patients was 58.20 ± 5.17 years. This finding disagreed with Adekanmbi et al. [14] who showed that the mean age of patients was 62.7 ± 11.6 years.

Our study results revealed that 62.5% of the study group had type I lesions and 37.5% had type II lesions. Of type II lesions; 15% had high grade serous carcinoma, 12.5% had clear cell adenocarcinoma and 10.0% had malignant mixed Mullerian (carcinosarcoma). These findings were consistent with Li et al. [15] who revealed that 84.81% of cases had endometrioid carcinoma, 9.49% had carcinosarcoma, 3.16% had serous and 1.27% had clear cell EC.

The current study findings revealed that 80.0% of the study group had myometrial invasion > 50% while 20.0% had myometrial invasion < 50%. Similarly, myometrial invasion was demonstrated in 79.5 % of EG cases of a study by Dane & Bakir [16]. Wang et al. [17] found that 72.8 percent of patients had no or shallow myometrial invasion, while 27.2% had deep myometrial invasion.

Our study results revealed that 10.0% of cases had positive cervical involvement. This finding was supported by Dane & Bakir, [16] who reported that cervical involvement was seen in 18% of the patients. Salama and Khairy, [18] showed that cervical involvement was positive in 19.0% of patients.

In the present study, 25.0% of cases had positive LVSI. This finding was consistent with Salama and Khairy, [18] who revealed that 29% of EC cases were positive for lymphovascular invasion. However, Sun et al. [19] reported that only 8.3% of EC cases were positive for LVSI.

The present study findings revealed that 65.0% of the study group had wild (normal) IHC: p53 and 35.0% had mutant (abnormal) IHC: p53 including 20.0% diffuse nuclear, and 15.0% null. These findings agreed with Tresa et al. [20] who reported that 57.1% of cases had wild type p53 and 42.8% had mutant p53. Another study found that 28% of cases harbored a mutation in *TP53* [21]. These findings disagreed with Jia et al. [22] who found that 23.6% of cases had p53 wild-type, whereas 76.4 % showed abnormal p53 staining.

Our findings revealed that 27.5% of the study group were negative for IHC: ER while 72.5% were positive for IHC: ER. Similarly, another study found that 60.7% of EC cases were ER positive [23] these findings disagreed with

Odetola et al. [9] who found that 29.5% of EC cases were ER-positive.

The current study results showed highly statistically significant association between IHC: p53 and type of lesions (p = 0.001). A previous study investigated TP53 mutations in EC. TP53 mutations were found in 28% of endometrial carcinomas. In endometrioid endometrial carcinomas and serous endometrial carcinomas, TP53 mutation was found in 15% and 8% of cases, respectively. They suggested that even when present in endometrioid endometrial carcinomas (EECs), the biological impact of TP53 mutations may differ between EECs and serous endometrial carcinomas (SECs) [21].

The present study findings revealed statistically significant association between IHC: p53 and grade of pathology (p = 0.007). Other research results showed that p53 was significantly correlated with high-grade tumors [24].

Our findings revealed highly statistically significant association between IHC: ER and lesions (p = 0.001). The positivity of ER in type 1 endometrial cancer was significantly higher than that in type 2 endometrial cancer (p = 0.0001) according to Shen et al. [25].

The current study results showed highly statistically significant association between IHC: ER and lymphovascular invasion (p = 0.001).

The present study results revealed statistically significant association between IHC: ER and grade of pathology (p = 0.013). Similarly, a past study found statistically significant correlation between positive ER expression and grade I - II tumors [26]. This finding disagreed with Shivakumar et al., who found no significant association between ER and grade of pathology (p = 0.423) [24].

The present study results revealed statistically significant association between IHC: p53 and IHC: ER (p = 0.014). Another study found a significant association between p53 expression and estrogen receptor status in breast carcinoma. Nevertheless, the root causes of this connection are not clear. Mutant p53 may affect ER-mediated transcriptional activity, potentially impacting the development and progress of ER-positive breast cancer [27].

This finding disagreed with Maeda et al. [28] who investigated the mechanism of (ER) loss and the condition of the p53 pathway EC. They found no significant association between the p53 pathway and ER status. Another study found no statistically significant association between ER and p53 [29].

P53 and ER markers were discovered to have a pathological significance in EC. Our results emphasize the importance of using a set of histomorphology-based markers to sub classify EC throughout regular surveillance. It also emphasizes the link among these markers and other clinicopathological predictive measures, suggesting that they could serve as possible beneficial biomarkers. Further studies using larger numbers of participants are required for confirming the results obtained from this investigation.

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Declaration of Interest:

None.

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