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p53 Immunoexpression in Epithelial Ovarian Carcinomas: A Clinicopathological Correlation Study at tertiary Care Centre

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ABSTRACT

Background: TP53 is one of the most frequently altered tumor suppressor genes in epithelial ovarian carcinoma (EOC), particularly in high-grade serous carcinoma. Immunohistochemistry (IHC) for p53 serves as a practical surrogate for TP53 mutation analysis in routine diagnostic practice, especially in resource-limited settings. Pattern-based interpretation improves correlation with underlying mutational status.
Objective: To evaluate p53 immunoexpression patterns in epithelial ovarian carcinomas and correlate findings with histological subtype, stage, grade, and clinicopathological parameters.
Materials and Methods: This laboratory-based prospective study included 70 histopathologically confirmed cases of epithelial ovarian carcinoma. Formalin-fixed paraffin-embedded sections were subjected to p53 immunohistochemistry using monoclonal antibody (Pathn Situ™, clone PM101). Staining was interpreted using a pattern-based approach and categorized as mutant-type (diffuse overexpression, null pattern, or cytoplasmic staining) or wild-type. Clinicopathological parameters were compared between mutant and wild type p53expression groups.
Results: High-grade serous carcinoma was the most common histological subtype (58.3%). Mutant-type p53 expression was observed in 35 cases (71%), while 14 cases (28.5%) demonstrated wild-type staining. Advanced-stage tumors (Stage III–IV) showed a higher frequency of mutant-type expression (80%). Capsular breach (7.3%) and lymphovascular invasion (5.7%) were more frequently observed in the mutant group; however, perineural invasion and tumor necrosis did not show significant association.
Conclusion: Aberrant p53 immunoexpression was predominantly observed in high-grade and advanced-stage epithelial ovarian carcinomas. Pattern-based p53 evaluation is a useful adjunct in histological type confirmation and clinicopathological correlation in Epithelial Ovarian Carcinoma (EOC).

Keywords: p53; epithelial ovarian carcinoma (EOC); immunohistochemistry (IHC)

INTRODUCTION

Ovarian carcinoma represents a heterogeneous group of neoplasms with diverse morphological and molecular characteristics. Epithelial ovarian carcinomas (EOC) constitute the majority of malignant ovarian tumors and are associated with significant morbidity and mortality, largely due to late-stage presentation. High-grade serous carcinoma (HGSC) is the most common histological subtype and accounts for the majority of ovarian cancer–related deaths. Despite advances in surgical and chemotherapeutic strategies, the overall 5-year survival rate remains suboptimal, particularly in advanced-stage disease.[1]

The pathogenesis of epithelial ovarian carcinoma is closely linked to genetic alterations affecting tumor suppressor genes and pathways regulating cell cycle progression, DNA repair, and apoptosis. Among these, TP53 is one of the most frequently mutated genes in human malignancies and plays a pivotal role in maintaining genomic stability. The TP53 gene, located on chromosome 17p13, encodes the p53 protein, which functions as a transcription factor regulating cell cycle arrest, senescence, and apoptosis in response to cellular stress and DNA damage.

Under normal conditions, p53 mediates G1/S cell cycle arrest through activation of downstream targets such as p21, thereby allowing DNA repair. In cases of irreparable DNA damage, p53 induces apoptosis to prevent propagation of genetically unstable cells. Mutation of TP53 leads to loss of tumor suppressor function, contributing to genomic instability, tumor progression, and aggressive biological behavior. In ovarian carcinoma, particularly high-grade serous carcinoma, TP53 mutation is considered a defining molecular event.

Molecular sequencing remains the gold standard for identifying TP53 mutations; however, its routine use is limited in many settings due to cost and technical constraints. Immunohistochemistry (IHC) for p53 has emerged as a practical surrogate marker for TP53 mutational status. Pattern-based interpretation of p53 immunostaining—recognizing overexpression, null, and cytoplasmic patterns—has demonstrated improved correlation with underlying genetic alterations and enhanced reproducibility in diagnostic practice.

Given the established molecular significance of TP53 in epithelial ovarian carcinoma, evaluation of p53 immunoexpression may provide useful diagnostic and clinicopathological insights. The present study aimed to assess p53 expression patterns in malignant epithelial ovarian neoplasms and to correlate p53 status with histological subtype, grade, stage, and clinical outcome.

MATERIALS AND METHODS

This laboratory-based prospective cohort study was conducted after obtaining approval from the Institutional Ethics Committee (Approval No. OG/3723/23). A total of 70 histopathologically confirmed cases of epithelial ovarian carcinoma were included. Formalin-fixed, paraffin-embedded (FFPE) tissue blocks were retrieved for analysis. Written informed consent was obtained from all participants.

Histopathological Evaluation-Three to five micron-thick sections were cut from FFPE blocks and stained with hematoxylin and eosin (H&E) for morphological evaluation. Tumours were classified according to the 2020 WHO Classification of Female Genital Tumours. Histological subtype, tumour grade, and pathological stage (based on available clinical and surgical records) were documented. Representative tumour sections were selected for immunohistochemical analysis.

Immunohistochemistry-

Immunohistochemistry was performed manually on 2–3 µm thick sections mounted on poly-L-lysine–coated slides. Antigen retrieval was carried out using Tris-EDTA buffer (pH 9.0) in a decloaking chamber at 95–100°C for 20 minutes, followed by cooling at room temperature. The primary antibody used was anti-p53 monoclonal

antibody (PathnSitu™, clone DO-7; dilution 1:100) used according to manufacturer instructions. Sections were incubated with the primary antibody for 60 minutes at room temperature. followed by appropriate secondary antibody. Detection was performed using a polymer-based horseradish peroxidase (HRP) detection system with diaminobenzidine (DAB) as chromogen. Counterstaining was done with hematoxylin. Appropriate external positive and negative controls were included in each staining run. Internal stromal and inflammatory cells served as internal positive controls, p53 expression was interpreted using a pattern-based approach in accordance with contemporary guidelines. Staining patterns were categorized as:

Mutant-type expression:

- Diffuse strong nuclear staining in >70% of tumour cells (overexpression pattern)
- Complete absence of nuclear staining in tumour cells with retained internal control staining (null pattern)
- Cytoplasmic staining with or without nuclear staining

Wild-type expression:

Variable nuclear staining of heterogeneous intensity in tumour cells.

Statistical Analysis

Statistical Analysis was performed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA. Clinicopathological parameters were compared between mutant-type and wild-type p53 expression groups. Categorical variables were analyzed using Chi-square or Fisher's exact test. A p-value <0.05 was considered statistically significant Fisher's exact test was applied where expected cell counts were less than five. A composite adverse outcome variable (recurrence, refractory disease, or mortality) was analyzed in relation to p53 expression status.

RESULTS

A total of 70 histopathologically confirmed epithelial ovarian carcinoma cases were included. Of these, p53 immunohistochemistry was successfully performed and interpretable in 49 cases. In 9 cases, prior neoadjuvant chemotherapy resulted in scant residual tumor, precluding reliable assessment. In 12 cases, repeated section loss during IHC processing prevented evaluation.

Figure 1- Depicts that out of a total of 77 patients, 70 cases were of Epithelial Ovarian Carcinoma (EOC) out of which high grade serous adenocarcinoma was the most common histological type observed in 42 patients. Low grade serous adenocarcinoma was seen in 12 patients. Mucinous cystadenocarcinoma was noted in 10 patients. Clear cell carcinoma was present in 5 patients. while endometrioid carcinoma was the least common and was seen in 1 patient.

Out of Seven (7) Non-Epithelial Ovarian Carcinoma, Germ cell tumor was found in 3 patients, Sex cord stromal tumor was observed in 4 patients

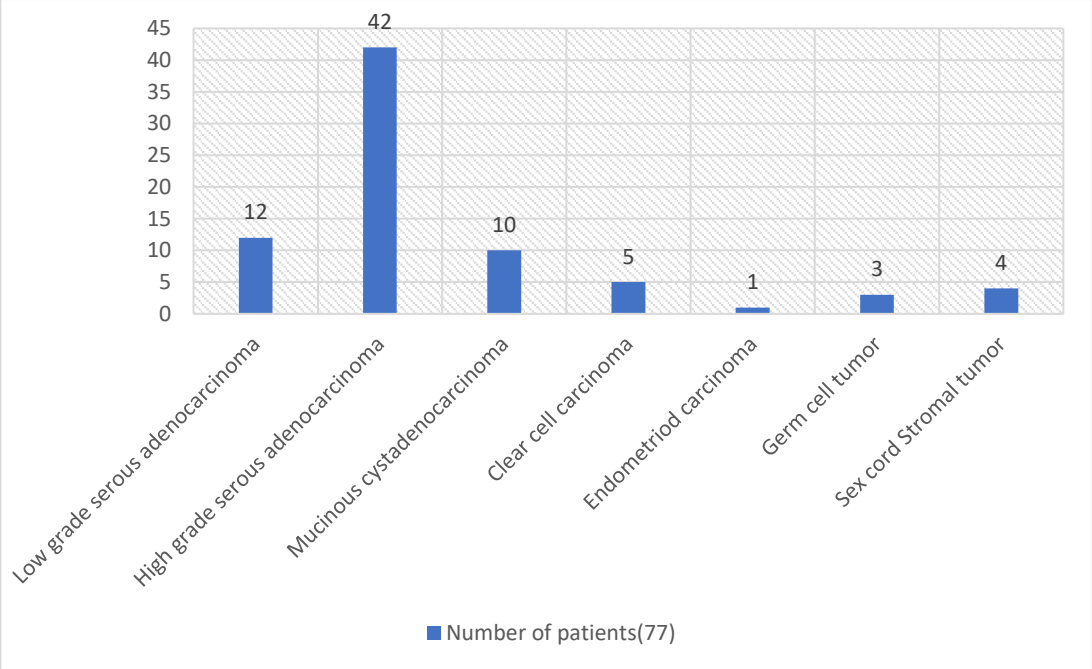

Figure 1- Depicts spread of Malignant ovarian Tumors according to HPE (N=77).

Figure 2- illustrates that among 49 epithelial ovarian carcinoma cases evaluated for p53 expression, mutant type p53 expression was observed in 35 cases constituting 71.4%, whereas wild type p53 expression was seen in 14 cases accounting for 28.5%.

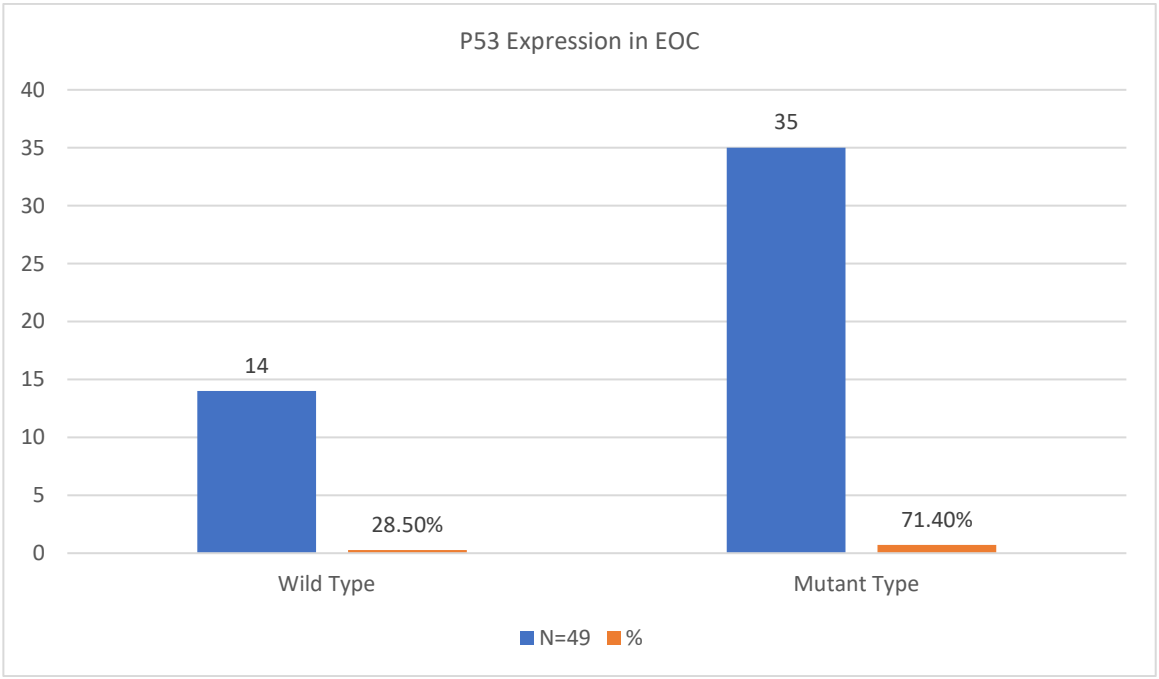

Figure 2- Depicts P53 results in malignant EOC (n=49).

Table 1: Distribution of p53 expression across FIGO Stages

FIGO STAGING	MUTANT TYPE N (%)	WILD TYPE N (%)	P-value
Stage I	3 (8.57%)	7 (50.0%)	0.004
Stage II	4 (11.43%)	2 (14.3%)	
Stage III and above	28 (80.0%)	5 (35.7%)	

The association between FIGO stage and p53 expression was statistically significant (p = 0.004 (two-sided exact test).

The above table illustrates the distribution of p53 expression across FIGO stages among the study participants. Out of the total 35 mutant type cases, 3 cases (8.57%) belonged to Stage I, 4 cases (11.43%) belonged to Stage II, and 28 cases (80.0%) belonged to Stage III and above. Out of the total 14 wild type cases, 7 cases (50.0%) belonged to Stage I, 2 cases (14.3%) belonged to Stage II, and 5 cases (35.7%) belonged to Stage III and above**. (Figure 3)** The association between FIGO stage and p53 expression was statistically significant with p value of 0.004.

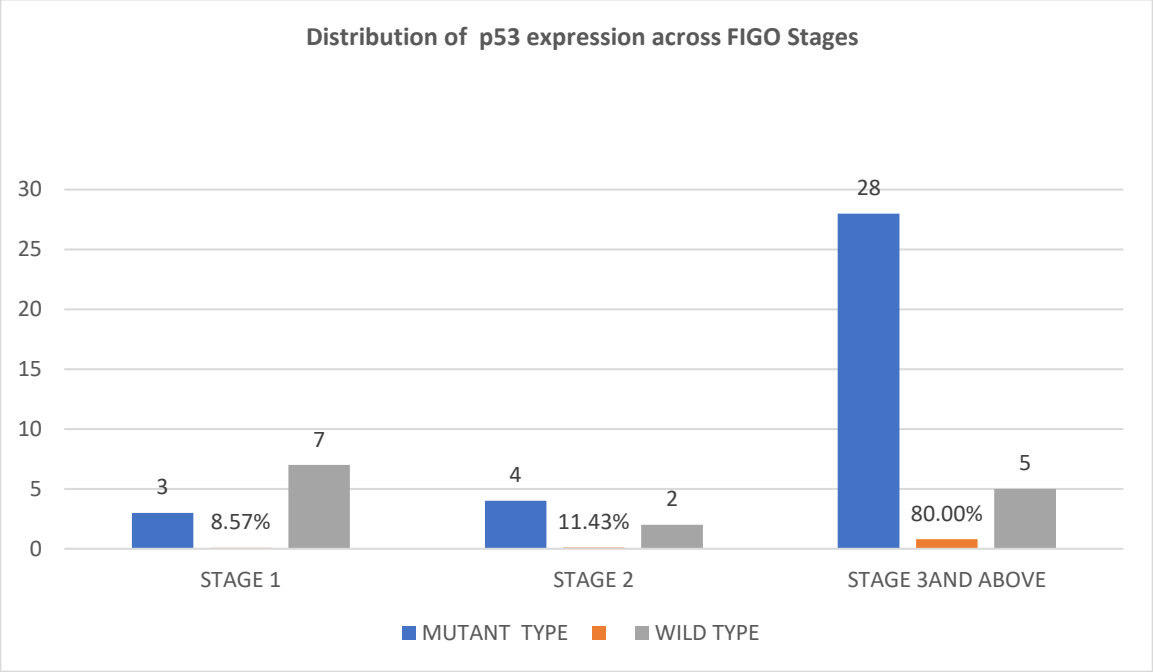

Figure 3- Depicts distribution of p53 expression across FIGO stages

Table 2: Association of p53 staining with high grade histopathological features

High grade features	MUTANT TYPE		WILD TYPE		Fisher's Exact p value
	N	%	N	%	
Lymphovascular invasion					
Absent	33	94.3%	14	100.00%	1.00
Present	2	5.7%	0	0.00%	
Perineural invasion					
Absent	35	100.00%	13	92.86%	0.286
Present	0	0.00%	1	7.14%	
Necrosis					
Absent	23	65.7%	7	50.00%	0.346
Present	12	34.3%	7	50.00%	
Capsular involvement					
Breached	3	8.6%	1	7.14%	1.00
Intact	32	91.4%	13	92.86%	
Tumour Cellularity					
LESS THAN 50%	7	20.0%	0	0.00%	0.170
>=50%	28	80.0%	14	100.0%	

Table 2- Illustrates the association of p53 staining with high grade histopathological features among the study participants. With regard to lymphovascular invasion, absence of lymphovascular invasion was observed in 33 mutant type cases (94.3%) and 14 wild type cases (100.0%), while presence of lymphovascular invasion was noted in 2 mutant type cases (5.7%) and none of the wild type cases (0.0%). The Fisher's exact p

value was 1.00. which was not statistically significant

For perineural invasion, absence was seen in 35 mutant type cases (100.0%) and 13 wild type cases (92.86%), whereas presence was observed in none of the mutant type cases (0.0%) and in 1 wild type case (7.14%). The Fisher's exact p value was 0.286.

For necrosis, absence was present in 23 mutant type cases (65.7%) and 7 wild type cases (50.0%), while necrosis was present in 12 mutant type cases (34.3%) and 7 wild type cases (50.0%). The Fisher's exact p value was 0.346. was not statistically significant

With respect to capsular involvement, breached capsule was noted in 3 mutant type cases (8.6%) and 1 wild type case (7.14%), while intact capsule was observed in 32 mutant type cases (91.4%) and 13 wild type cases (92.86%). The Fisher's exact p value was 1.00. and showed no statistically significant association with p53 expression

For tumour cellularity, less than 50% cellularity was observed in 7 mutant type cases (20.0%) and none of the wild type cases (0.0%), whereas tumour cellularity ≥50% was observed in 28 mutant type cases (80.0%) and 14 wild type cases (100.0%). The Fisher's exact p value was 0.170.

Table 3: Shows follow up of the patients under two groups: Mutant type and Wild Type.

Type of Tumor	Mutant. (n=35)	Wild (n=14)	Fisher's Exact p value
Recurrence (platinum sensitive)	1	0	0.024
Recurrence (Platinum Resistant)	1	1	
Mortality	3	2	
Loss to follow up	2	1	
Refractory	2	0	

Table 3- illustrates the follow-up outcome of patients according to p53 expression pattern. Among the mutant type group comprising 35 cases, recurrence with platinum sensitive disease was observed in 1 case, recurrence with platinum resistant disease in 1 case, mortality in 3 cases, loss to follow up in 2 cases and refractory disease in 2 cases. Among the wild type group comprising 14 cases, recurrence with platinum sensitive disease was not observed in any case, recurrence with platinum resistant disease was noted in 1 case, mortality was observed in 2 cases, loss to follow up in 1 case and refractory disease was not observed. Overall, mutant-type p53 expression showed a statistically significant association with adverse clinical outcomes (recurrence, refractoriness, and mortality) The association showed a p-value of 0.024. **(Figure 4)**

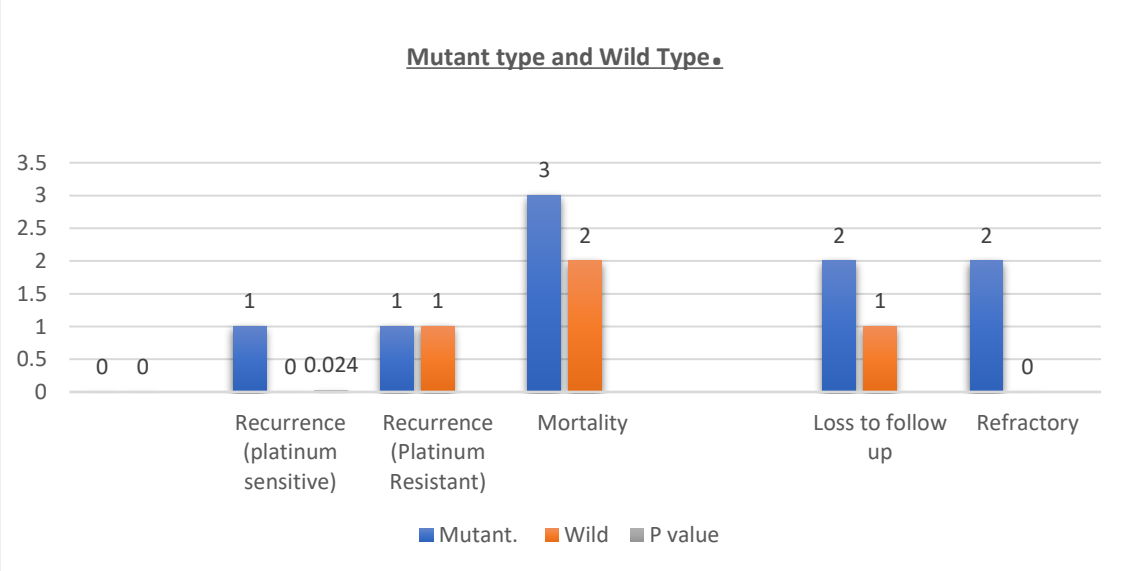

Figure 4- Outcome of patients according to p53 expression pattern

Image plate 1: Section shows hematoxylin and eosin-stained images of epithelial cancers in the ovary in study Cohort

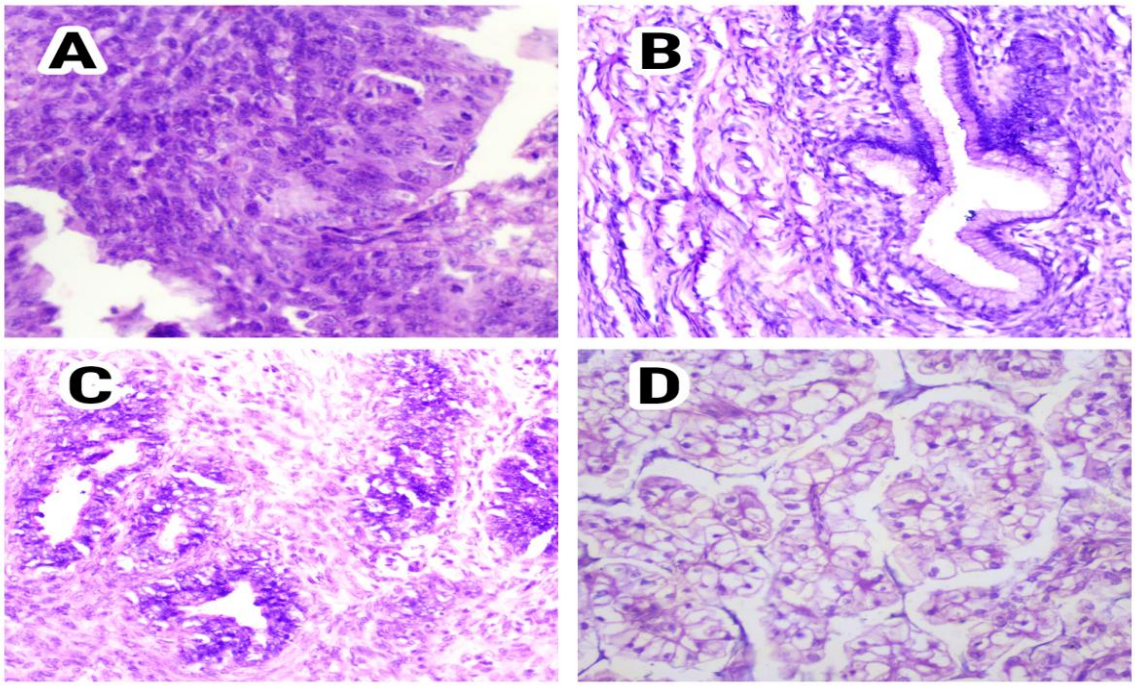

Image plate 1: Section shows hematoxylin and eosin stained images of epithelial cancers in the ovary (A) high-grade serous carcinoma is seen with tumor cells arranged in sheets displaying high nucleocytoplasmic ratio and prominent nucleoli, in B mucinous cancer is seen with tumor cells displaying cytoplasmic mucin, in C endometroid tumor is seen with irregular glands infiltrating the stroma. In D clear cell carcinoma can be seen with cytoplasmic clearing of the tumor cells. (A-D; H&E x200)

Image plate 2: Representative p53 IHC images (wild- mutant type pattern)

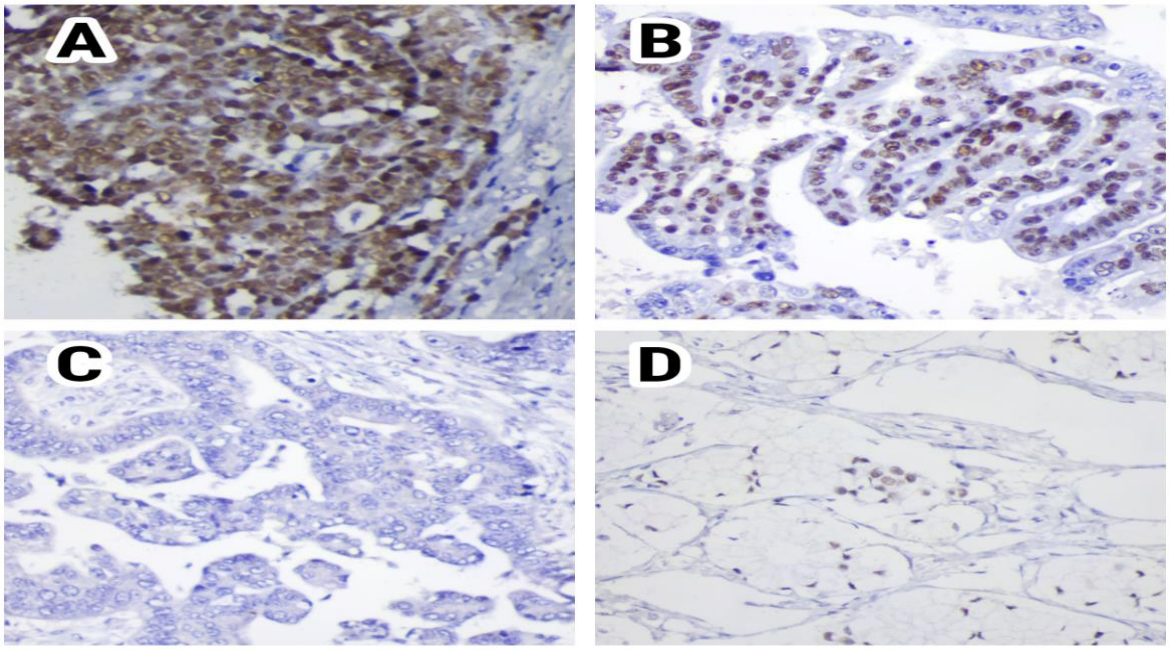

Image plate 2: Section A showing positive expression of p53 in tonsil (Control). Section B from serous carcinoma showing wildtype expression of p53. Section C from high grade serous carcinoma showing null type expression of p53. Section D from mucinous carcinoma showing mutated p53 having nuclear positivity in all tumor cells. (A-D; DAB x200)

DISCUSSION

Epithelial ovarian carcinomas (EOC) represent a heterogeneous group of neoplasms with distinct morphological, immunophenotypic, and molecular characteristics. The major histological subtypes include high-grade serous carcinoma (HGSC), low-grade serous carcinoma, mucinous carcinoma, endometrioid carcinoma, and clear cell carcinoma. Increasing evidence supports that these tumours are biologically distinct entities rather than a single disease spectrum. In the present study, high-grade serous carcinoma constituted the majority of epithelial ovarian malignancies (58.3%), followed by low-grade serous and mucinous carcinomas (16.7% each), while endometrioid carcinoma was least common (1.4%). This distribution is consistent with global epidemiological data demonstrating the predominance of serous carcinoma among epithelial ovarian cancers and in line with Minmin Wang, et al.[2]

The high frequency of HGSC in our cohort is noteworthy, given its established association with TP53 mutation. TP53 is one of the most frequently altered tumour suppressor genes in human malignancies and plays a pivotal role in cell cycle regulation, genomic stability, and apoptosis. In ovarian carcinoma, TP53 mutation is considered a defining molecular event in high-grade serous carcinoma.

We adopted a pattern-based interpretation of p53 immunohistochemistry in accordance with contemporary recommendations, classifying staining as mutant-type (overexpression, complete absence/null pattern, or cytoplasmic staining) versus wild-type. This approach correlates more reliably with underlying TP53 mutation status compared to earlier methods that relied on percentage positivity or intensity grading alone.

In our study, aberrant (mutant-type) p53 expression was observed in 71.4% of cases, while 28.5% demonstrated wild-type staining patterns which align with study by Aswathi R et al.,[3] who reported fifteen (75%) of the twenty malignant tumors were p53 positive, & they were all classified as serous malignancies and our results are also in concordance to Chiesa-Vottero et al.[4] who reported eighty percent of serous ovarian carcinomas are p53 positive, and are also similar with Bilyk et al. [5] and *Köbel et al.*[6] who reported expression of the protein varies according to the degree of differentiation high-grade tumors have p53 positivity throughout . Other studies like Lassus H et al. [7] (59% of malignant carcinoma expressed p53) & Sylvia MT et al.[8.] (63.6% of malignant carcinoma expressed p53) showed somewhat lower positive rates. The variation in p53 positivity reported in literature (ranging from approximately 50% to 80%) may be attributed to differences in scoring criteria, antibody clones used, fixation variables, and interobserver variability

Consistent with established molecular paradigms, mutant-type p53 expression in our study was predominantly observed in high-grade serous carcinomas. Lower frequencies of aberrant expression were noted in other histological subtypes. This observation supports the dualistic model of ovarian carcinogenesis, wherein type II tumours (including HGSC) are characterized by high genomic instability and near-universal TP53 mutations, in contrast to type I tumours (such as low-grade serous, mucinous, and endometrioid carcinomas), which typically harbor alternative molecular alterations.

In our study Stage-wise analysis revealed a higher proportion of mutant-type p53 expression in advanced-stage tumours with p53 mutation (80.0%) [Table 1] Most common p53 expression in Stage 1and II was wild type 50%,14.3% respectively This finding is in concordance with study by Eltabbakh et al.[9] who found that p53 overexpression was associated with advanced stage (P = 0.04), higher grade (P = 0.0003), serous histology (P = 0.0018), and patient age > 61 years (P = 0.013). Buttitta F[10] reported p53 alterations were present only in invasive ovarian carcinomas, and they were detected much more frequently in

tumors characterized by high histological grade (P = 0.01) and advanced-stage disease. However, given the limited sample size and observational design, definitive conclusions regarding prognostic significance should be interpreted cautiously.

Khound R et al.[11] in their study reported that as P53 is overexpressed in high grade serous tumors, it is found to have a prognostic role. In our study, Mutant-type p53 expression showed significant association with adverse clinical outcomes, including recurrence and mortality. A Reles et al.[12] in their study explored the role of p53 mutations in the resistance of ovarian cancer cells to platinum-based chemotherapy in their study they reported Patients with high-grade serous ovarian cancer who harbored p53 mutations tended to have worse responses to platinum therapy and were more likely to experience recurrence after treatment. Hientz K [13]reported Overexpression of mutated p53 with reduced or abolished function is often connected to resistance to standard medications, including cisplatin, Buttitta F[10] in their study for 33 patients reported a strong correlation (P = 0.001) between p53 alterations and response to chemotherapy; only one (14%) of seven patients who had a pathological complete response to antiblastic drugs showed p53 aberrations, whereas 18 (82%) of 22 cases with partial response and all of the four non-responsive patients scored positive for p53 abnormalities. In our study we found statistical association between p53 status and treatment response (p value-0.024), In our study, one patient had platinum sensitive recurrence which had positive p53 expression, out of the platinum resistant recurrence, one patient had positive expression of p53 cells. Mortality was observed in five patients, three were having positive p53 and two had negative p53 expression. Two patients with positive p53 expression were refractory. The heterogeneity of epithelial ovarian carcinomas and the absence of molecular confirmation limit the strength of this inference. Larger studies incorporating molecular sequencing would be required to validate these findings.

From a diagnostic standpoint, p53 immunohistochemistry remains a valuable ancillary tool in routine surgical pathology practice. It aids in confirming the diagnosis of high-grade serous carcinoma, particularly in morphologically ambiguous cases, and assists in distinguishing high-grade from low-grade serous neoplasms. Pattern-based interpretation enhances reproducibility and aligns with current WHO recommendations.

The limitations of our study include its single-center design, relatively small cohort size, and lack of molecular validation of TP53 mutational status. Despite these limitations, our findings reinforce the established role of p53 immunohistochemistry as a surrogate marker for TP53 alterations and highlight its utility in histotype classification and clinicopathological correlation in epithelial ovarian carcinoma.

CONCLUSION

In our study p53 immunohistochemistry demonstrated a high frequency of mutant-type expression in epithelial ovarian carcinomas, particularly in high-grade serous and advanced-stage tumors. A significant association was observed between mutant-type p53 expression and adverse clinical outcomes in our cohort. Pattern-based interpretation of p53 staining serves as a reliable and practical surrogate for TP53 mutational status and is a valuable adjunct in histotype confirmation and clinicopathological correlation in routine diagnostic practice. Further large-scale studies with molecular validation and long-term follow-up are required to better define the prognostic and predictive significance of p53 alterations in epithelial ovarian carcinoma

Authors' Contribution:

Concept, design - SA, ER (Dr Shuchi Agrawal, Eva Raman), definition of intellectual content - SA, ER literature search- SA, ER, PA clinical studies- SA, ER,

PA (Preeti Agrawal) experimental studies- SA, ER, PA, RS data acquisition- SA, ER, PA data analysis- SA, ER statistical analysis-ER, SA manuscript preparation- ER, SA editing - ER, SA, PA, AA manuscript review - ER, SS, PA, AA, SG.

Declaration by Authors

Ethical Approval: Approved. Number: IEC - 2076/Ethics/2023

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Conflict of Interest: The authors declare no conflict of interest.

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