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# Histopathological patterns and molecular alterations in surface epithelial ovarian tumors: A retrospective study from tertiary care centre in central India.

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## Abstract

### Background
Surface epithelial ovarian tumors constitute the most common group of ovarian neoplasms and demonstrate marked heterogeneity in morphology, biological behavior, and molecular profile.

### Objective
To evaluate histopathological patterns of surface epithelial ovarian tumors and correlate them with immunohistochemical and molecular alterations.

### Methods
A retrospective cross-sectional study was conducted at a tertiary care teaching hospital in eastern India. All histologically confirmed surface epithelial ovarian tumors diagnosed between January 2020 and December 2023 were reviewed. Tumors were classified according to the WHO 2020 criteria. Immunohistochemistry panels were applied based on morphological suspicion. Targeted molecular analysis was performed in malignant tumors with adequate tissue to assess TP53, BRCA1/2, KRAS, BRAF, PIK3CA, ARID1A, and CTNNB1 alterations.

### Results
Benign, borderline, and malignant tumours made up 32.8%, 14.4%, and 52.8% of the 180 cases, respectively. The most common type of cancer was high-grade serous carcinoma (61.2%). A TP53 mutation was found in 92% of high-grade serous carcinomas, and it was very similar to the abnormal p53 immunostaining. Eighteen percent of high-grade serous carcinomas had BRCA1/2 mutations. KRAS mutations were primarily identified in mucinous and low-grade serous tumours, whereas ARID1A loss and PIK3CA mutations were prevalent in endometrioid and clear cell carcinomas.

### Conclusion
Different histomorphological patterns of surface epithelial ovarian tumours are closely linked to certain immunohistochemical and molecular changes. Combining morphology with targeted supplementary testing enhances diagnostic precision and enables tailored therapeutic approaches.

### Recommendations
Routine integration of immunohistochemistry with targeted molecular testing is recommended in malignant ovarian tumors.

***Keywords:*** *Ovarian carcinoma; Histopathology; Immunohistochemistry; Tumor protein p53; Breast cancer susceptibility gene; Kirsten rat sarcoma viral oncogene homolog; AT-rich interactive domain-containing protein 1A*
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## Introduction

Ovarian cancer is one of the most common causes of death from gynaecological cancer around the world and in India. This is mostly because it is diagnosed late and there aren't any good screening methods [1,2]. Most ovarian tumours are surface epithelial tumours, which can be anything from benign cystadenomas to very aggressive carcinomas [3].

The World Health Organisation divides surface epithelial tumours into different histological subtypes, such as serous, mucinous, endometrioid, clear cell, and Brenner tumours [4]. These subtypes are now seen as separate biological entities instead of just different forms of the same disease. They have different ways of developing, different molecular drivers, different clinical behaviour, and different responses to treatment [5].

High-grade serous carcinoma is the most common type of cancer that is cancerous. It is marked by a lot of nuclear atypia and almost all TP53 mutations [6,7]. Low-grade serous, mucinous, endometrioid, and clear cell carcinomas, on the other hand, usually have mutations in genes like KRAS, BRAF, ARID1A, PIK3CA, and CTNNB1. These tumours are part of the Type I tumour pathway [8–10].

Histopathological examination is still the most important part of diagnosis, but it can be hard to tell the difference between subtypes because they look so similar. Immunohistochemistry helps solve these problems by showing protein expression patterns that are specific to a lineage and linked to a mutation [11,12]. The growing accessibility of molecular testing has enhanced tumour classification and facilitated targeted therapeutic options, especially for BRCA-mutated tumours [13].

The goal of this study is to look at the histopathological patterns of surface epithelial ovarian tumours and see how they relate to immunohistochemical and molecular changes in a tertiary care setting in India.

## Materials and Methods

### Study Design and Setting

A retrospective cross-sectional study was conducted in the Department of Pathology, Nalanda Medical College and Hospital, Patna, a government-run tertiary referral centre.

### Study Period

Cases diagnosed from **January 2020 to December 2023** were reviewed. Data analysis was performed between January and March 2024.

### Inclusion Criteria

- Surgically resected surface epithelial ovarian tumors
- Adequate tissue for histopathology
- Available clinical records

### Exclusion Criteria

- Germ cell tumors
- Sex cord-stromal tumors
- Metastatic ovarian tumors

### Histopathology and Immunohistochemistry

Slides were reviewed and classified according to the WHO 2020. Immunohistochemistry included WT1, PAX8, p53, ER, PR, HNF1β, Napsin A, β-catenin, CK7, CK20, and CDX2 as indicated.

### Molecular Analysis

Targeted next-generation sequencing was performed in malignant tumors with adequate tissue.

### Statistical Analysis

Descriptive statistics and concordance analysis were applied.

## Results

A total of 180 surface epithelial ovarian tumors were included in the study. Tumors were classified according to the World Health Organization (WHO) 2020 classification. The detailed clinicopathological, immunohistochemical, and molecular findings are presented below with proper citation of all figures and tables.

### Overall Tumor Distribution

Benign tumors constituted 59 cases (32.8%), borderline tumors 26 cases (14.4%), and malignant tumors 95 cases (52.8%) (Table 1). Representative histological features are shown in Figures 1–4.

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**Table 1: Distribution of surface epithelial ovarian tumors.**

| Tumor Category | Number of Cases (n) | Percentage (%) |
|---|---|---|
| Benign | 59 | 32.8 |
| Borderline | 26 | 14.4 |
| Malignant | 95 | 52.8 |

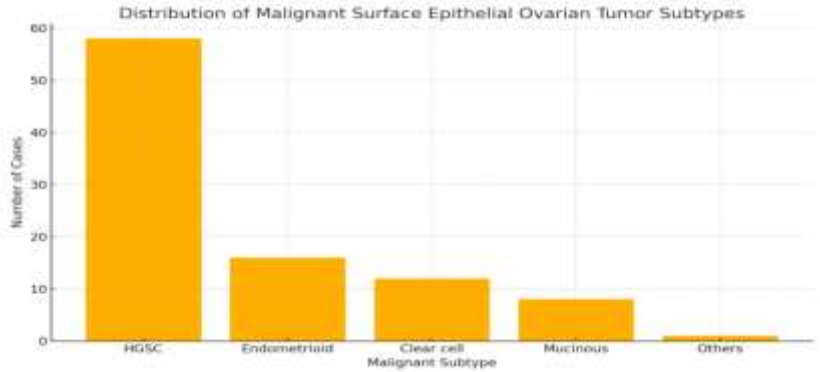


**Figure 1: High-grade serous carcinoma showing complex papillary architecture and marked nuclear atypia (H&E, 40×).**

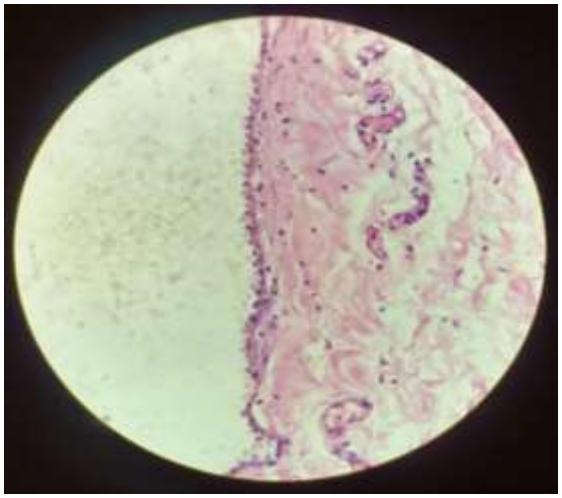

**Figure 2: Serous cystadenoma showing cystic spaces lined by bland cuboidal epithelium (H&E, 40×).**

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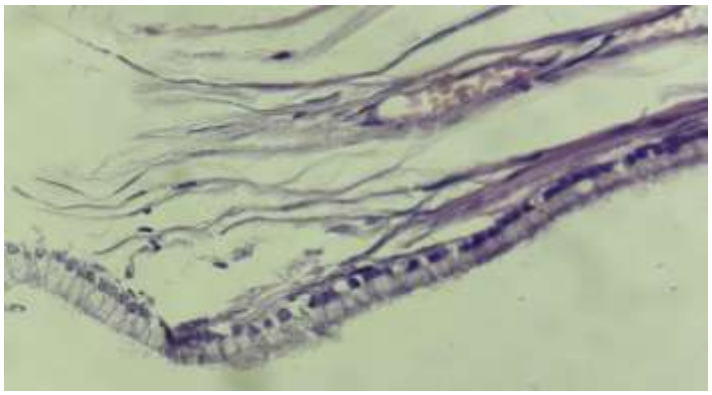

**Figure 3: Mucinous cystadenoma showing tall columnar mucin-secreting epithelium with basal nuclei (H&E, 40×).**

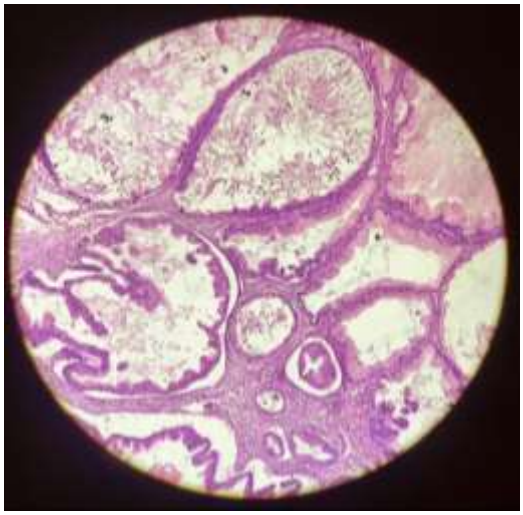

**Figure 4: Mucinous cystadenocarcinoma showing stromal invasion by atypical glands (H&E, 40×).**

### Histological Spectrum of Malignant Tumors

Among malignant tumors (n = 95), high-grade serous carcinoma was the predominant subtype, followed by endometrioid carcinoma, clear cell carcinoma, and mucinous carcinoma (Table 2). High-grade serous carcinomas demonstrated marked nuclear pleomorphism and frequent mitotic activity (Figure 1).

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**Table 2: Histological subtypes of malignant surface epithelial ovarian tumors.**

| Histological Subtype | Number of Cases (n) | Percentage (%) |
|---|---|---|
| High-grade serous carcinoma | 58 | 61.2 |
| Endometrioid carcinoma | 16 | 16.5 |
| Clear cell carcinoma | 11 | 12.1 |
| Mucinous carcinoma | 10 | 10.2 |

## Immunohistochemical and Molecular Findings

Immunohistochemical profiling and targeted molecular analysis showed distinct patterns among tumor subtypes (Table 3). Benign serous tumors exhibited simple epithelial lining without atypia (Figure 2), while benign mucinous tumors showed tall columnar mucin-secreting epithelium (Figure 3). Malignant mucinous tumors demonstrated stromal invasion (Figure 4).

**Table 3: Correlation of histological subtype with immunohistochemical and molecular findings.**

| Tumor Subtype | Key IHC Markers | Major Molecular Alterations | Frequency (%) |
|---|---|---|---|
| High-grade serous carcinoma | WT1+, PAX8+, aberrant p53 | TP53, BRCA1/2 | 92 |
| Endometrioid carcinoma | ER+, PR+, β-catenin | ARID1A, PIK3CA | 68 |
| Clear cell carcinoma | HNF1β+, Napsin A+ | ARID1A, PIK3CA | 72 |
| Mucinous carcinoma | CK7+, CK20+, CDX2 | KRAS | 65 |

## Discussion

This study demonstrates that surface epithelial ovarian tumors represent a biologically heterogeneous group with distinct histopathological and molecular profiles [14–16]. The predominance of high-grade serous carcinoma mirrors global and Indian data and reflects referral bias inherent to tertiary centres [17].

The strong concordance between p53 immunostaining and TP53 mutation confirms the utility of immunohistochemistry as a surrogate marker in routine practice [18]. Identification of BRCA mutations in a subset of tumors highlights opportunities for targeted therapy using PARP inhibitors [19].

Endometrioid and clear cell carcinomas showed characteristic ARID1A and PIK3CA alterations, supporting the concept of endometriosis-associated ovarian carcinogenesis [20–22]. Mucinous tumors demonstrated KRAS mutations, consistent with previously described molecular pathways [23].

## Conclusion

Histopathological evaluation supported by focused immunohistochemistry and selective molecular testing provides an effective framework for accurate classification and therapeutic stratification of surface epithelial ovarian tumors.

## Limitations

- Retrospective design
- Incomplete molecular testing in all cases

## Recommendations

Routine integration of immunohistochemistry with targeted molecular testing is recommended in malignant ovarian tumors.

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## Conflict of Interest

None declared.

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