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# Epidermal Growth Factor Receptor (EGFR) Mutations in Rare Histological Subtypes of Ovarian Cancer

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## Abstract

### Background

Ovarian cancer comprises a heterogeneous group of malignancies with distinct molecular profiles across histological subtypes. While the molecular landscape of high-grade serous ovarian carcinoma (HGSOC) has been extensively investigated, data regarding rare epithelial and non-epithelial ovarian tumors remain limited. The clinical relevance of epidermal growth factor receptor (EGFR) alterations in ovarian cancer is still uncertain, particularly in uncommon histological subtypes. This study aimed to investigate the presence of clinically relevant *EGFR* mutations in rare ovarian cancer subtypes.

### Methods

A retrospective molecular analysis was performed on 24 formalin-fixed paraffin-embedded ovarian tumor tissue samples collected between 2010 and 2022. The cohort included five mucinous ovarian carcinomas, five clear cell ovarian carcinomas, five non-epithelial ovarian tumors, and nine low-grade serous ovarian carcinomas (LGSOCs). DNA was extracted from tumor tissue containing at least 25% tumor cellularity. EGFR mutation analysis was performed using the Cobas® EGFR Mutation Test v2 (Roche Molecular Systems Inc., Branchburg, New Jersey, USA), a real-time PCR-based assay capable of detecting 42 predefined mutations across exons 18-21.

### Results

All 24 samples yielded valid molecular results, with no assay failures or quality-control abnormalities. No *EGFR* mutations were detected in any of the analyzed specimens. Specifically, no alterations were identified in exons 18, 19, 20, or 21, including G719X, exon 19 deletions, S768I, T790M, exon 20 insertions, L858R, or L861Q. *EGFR* mutation status was uniformly negative across all investigated histological subtypes, including mucinous, clear cell, non-epithelial, and low-grade serous ovarian tumors. Results were concordant across independent analytical runs.

### Conclusion

In this cohort of rare ovarian cancer histological subtypes, no activating *EGFR* mutations were identified. These findings support the limited role of *EGFR* mutations in the molecular pathogenesis of these ovarian tumors and suggest that routine *EGFR* mutation testing is unlikely to provide substantial therapeutic benefit. Larger studies incorporating broader genomic profiling are warranted to identify alternative molecular alterations with potential prognostic or therapeutic relevance.

**Categories:** Obstetrics/Gynecology, Oncology
**Keywords:** egfr mutations, molecular pathways, ovarian cancer, precision oncology, prognostic factors

## Introduction

Ovarian cancer is the leading cause of death among gynecologic malignancies worldwide, mostly because of its silent clinical course and late diagnosis in the majority of patients [1,2]. Despite recent advances in surgical techniques, perioperative management, and systemic treatment, including platinum-based chemotherapy and targeted agents, overall survival rates have improved only modestly over the last decades

[2,3]. This high mortality rate highlights the need for better patient stratification and a more personalized therapeutic approach based on tumor biology rather than on the "one-size-fits-all" strategy [1,3]. Epithelial ovarian cancer (EOC) accounts for the majority of ovarian tumors and consists of a diverse population of tumor subtypes with different histopathological, molecular, and clinical features [1,3]. Although high-grade serous ovarian carcinoma (HGSOC) is the most prevalent and well-studied subtype, it fails to adequately capture the wide range of ovarian cancer biology [4]. Other histological subtypes, including mucinous, clear cell, low-grade serous ovarian carcinoma (LGSOC) and non-epithelial ovarian tumors constitute clinically distinct subgroups of ovarian carcinoma [4,5]. These tumors often display unique patterns of disease progression, differential responses to chemotherapy, and survival outcomes compared to HGSOC, complicating their optimal treatment (5). Crucially, rare ovarian cancer subtypes are either excluded or underrepresented in large randomized clinical trials, creating a lack of high-quality evidence [5,6]. Consequently, therapeutic approaches are often extrapolated from HGSOC, despite the fundamental biological differences between distinct histological subtypes (Atallah et al., 2023). This extrapolation may lead to suboptimal management and underscores the need for disease-specific unique molecular drivers and relevant biomarkers [1,6].

Prognostic and predictive biomarkers are crucial in current clinical practice as they provide insights into the disease outcomes and guide therapeutic decisions [1,6]. Typically, prognostic factors most commonly used include FIGO stage, tumor grade at diagnosis, the extent of residual disease following cytoreductive surgery, and clinical characteristics including age and performance status [1,3]. However, the prognostic significance of these factors depends markedly on histology, especially in rare ovarian cancer subtypes, thus rendering them less universally useful [1]. Recently, a greater focus has been placed on the identification of molecular and genetic biomarkers to facilitate advanced prognostic determination and therapeutic response prediction [6,7]. New technologies for molecular profiling studies have highlighted the vital function of oncogenic signaling pathways, growth factor receptors, and DNA repair pathways in the pathogenesis and treatment resistance of ovarian cancer [4,7]. These molecular changes have been shown to modulate tumor aggressiveness, resistance to platinum-based chemotherapy, and the susceptibility to targeted therapies [1,7]. Nonetheless, most molecular studies are confined to HGSOC, leaving the molecular landscape of rare histological subtypes largely under-defined [6,7]. Recent studies indicate that the other histologies may possess different molecular profiles [4,6]. The limited understanding of the molecular background of non-serous histological subtypes can hinder the development of personalized treatment strategies [4,7]. Comprehensive molecular characterization of these subtypes is essential to provide clinically meaningful prognostic and predictive biomarkers [3,5,6]. Thus, the present study aims to investigate molecular alterations with potential predictive and prognostic value in rare histological subtypes of ovarian cancer [5,6]. Our goal is to better characterize the molecular background of these histological subtypes.

## Materials And Methods

A total of 24 formalin-fixed paraffin-embedded (FFPE) ovarian tumor tissue samples were included in this study. All samples were collected and processed during routine diagnostic procedures -salpingo-oophorectomy or biopsy- at the Oncology Department of Alexandra University Hospital, between 2010 and 2022. All patients met the inclusion criteria of histologically confirmed LGSOC, serous, mucinous, and other non-epithelial ovarian carcinomas. This study was designed as an exploratory pilot study, and the sample size was determined based on the availability of clinical specimens during the study period, aiming to provide preliminary data on epidermal growth factor receptor (*EGFR)* mutation prevalence in our population. All tissue specimens had been previously embedded in paraffin blocks and were selected retrospectively for molecular analysis. The study cohort consisted of rare histological subtypes of ovarian cancer, including five cases of mucinous ovarian carcinoma, five cases of clear cell ovarian carcinoma, five cases of non-epithelial ovarian tumors, and nine cases of LGSOC. The study was performed in accordance with the ethical standards described in the Declaration of Helsinki, and all the eligible patients provided written informed consent.

From each paraffin block, one histological section of 5 μm thickness was obtained and placed into a 1.5 mL microcentrifuge tube with a secure cap. All samples were transferred to the research laboratories of the Hellenic Anticancer Institute, where they were stored at -80°C until further processing. Only FFPE sections containing at least 25% tumor cellularity, as assessed by histopathological review, were included in the analysis to ensure adequate tumor DNA representation.

DNA extraction was performed using the Cobas DNA Sample Preparation Kit (Roche Molecular Systems Inc., Mannheim, Germany), according to the manufacturer's instructions. Before extraction, tissue sections underwent a standard deparaffinization procedure using xylene and ethanol washes, followed by tissue digestion with Proteinase K. The extracted genomic DNA was eluted in a final volume of elution buffer and stored at -20°C until analysis.

Quantitative assessment of the isolated DNA was carried out using the Qubit® Fluorometer (Thermo Fisher Scientific; Waltham, Massachusetts, USA) with the Qubit® dsDNA High Sensitivity Assay Kit (Thermo Fisher Scientific; Waltham, Massachusetts, USA), following the manufacturer's protocol. DNA concentration and purity were evaluated to ensure suitability for downstream molecular testing. DNA samples were subsequently diluted to a standardized working concentration of 2 ng/μL, according to predefined dilution

formulas provided by the assay protocol.

Molecular analysis focused on the qualitative detection of mutations in the epidermal growth factor receptor (*EGFR*) gene. *EGFR* mutation testing was performed using the Cobas® EGFR Mutation Test v2 (Roche Molecular Systems Inc., Branchburg, New Jersey, USA), which is based on real-time polymerase chain reaction (PCR) technology. This assay enables the detection of 42 predefined *EGFR* mutations located in exons 18, 19, 20, and 21, including point mutations, deletions, and insertions.

PCR amplification and mutation detection were carried out using the Cobas® z 480 analyzer (Roche Molecular Systems Inc., Rotkreuz, Switzerland). Each reaction included appropriate positive and negative controls to ensure assay validity. Fluorescence signals were measured in real time across multiple optical channels, and data analysis was performed automatically by the Cobas® z 480 software. Results were considered valid only when all internal controls met predefined quality criteria.

# Results

## Study cohort and sample adequacy

A total of 24 FFPE ovarian tumor tissue samples were included in the molecular analysis. All specimens met predefined inclusion criteria regarding tissue quality, DNA concentration, and tumor content, with tumor content exceeding 25% in all analyzed sections. DNA extraction and downstream molecular analysis were successfully completed for every sample, with no exclusions due to insufficient material or technical failure. The study cohort comprised rare histological subtypes of ovarian cancer, including mucinous ovarian carcinoma, clear cell ovarian carcinoma, non-epithelial ovarian tumors, and LGSOC. All samples were subjected to *EGFR* mutation analysis targeting exons 18, 19, 20, and 21 (Figure *1*).

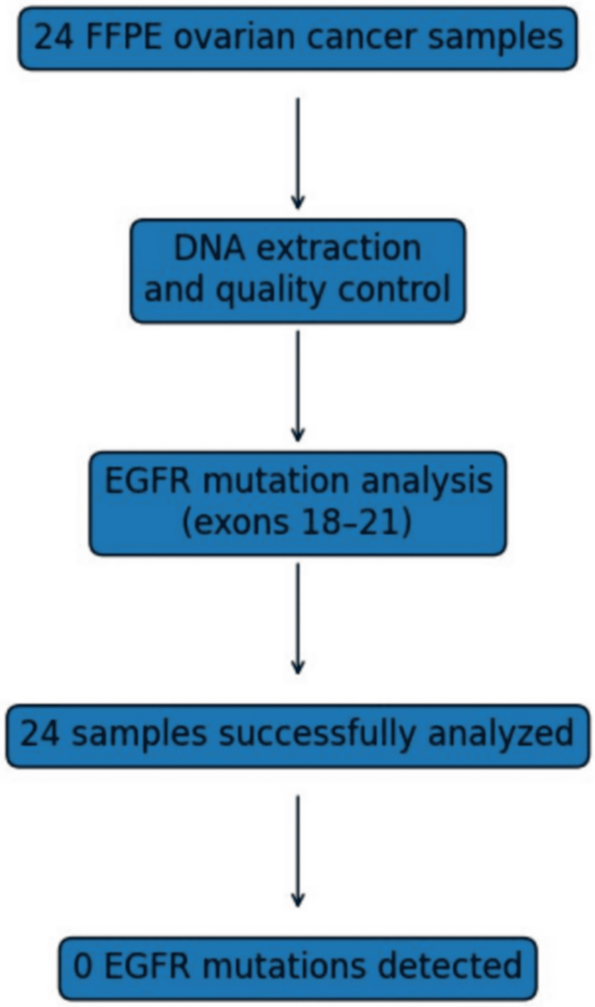



**FIGURE 1: Flow diagram summarizing the study design and EGFR mutation testing results in 24 ovarian cancer FFPE samples.**

FFPE: formalin-fixed paraffin-embedded; EGFR: epidermal growth factor receptor

## Assay performance and quality control

*EGFR* mutation testing was performed using the Cobas® EGFR Tissue Test v2 on the Cobas® 4800 System. All analytical runs were classified as valid according to the assay's internal quality control criteria. Both mutant (positive) and negative controls were included in each run and yielded valid results, confirming appropriate assay performance and analytical reliability. No assay flags, invalid runs, or control failures were observed throughout the testing process. Reagent integrity, amplification curves, and fluorescence signal thresholds were within acceptable limits for all runs, ensuring consistency, and reproducibility of the molecular analysis.

## EGFR mutation analysis

No *EGFR* mutations were detected in any of the analyzed ovarian cancer tissue samples. Specifically, all 24 specimens were reported as "No Mutation Detected" across all targeted *EGFR* mutation groups included in the assay panel. The analysis covered a total of 42 predefined *EGFR* mutations, including point mutations, deletions, and insertions across exons 18-21. No pathogenic or likely pathogenic alterations were identified in exon 18 (including G719X substitutions), exon 19 (including all exon 19 deletion variants), exon 20 (including S768I, T790M, and exon 20 insertions), or exon 21 (including L858R and L861Q substitutions). For all specimens, Result 1 indicated the absence of detectable *EGFR* mutations, while Result 2 was reported as "Not Applicable," consistent with a uniformly negative mutation status. No borderline, equivocal, or indeterminate results were observed (Figure *2*).

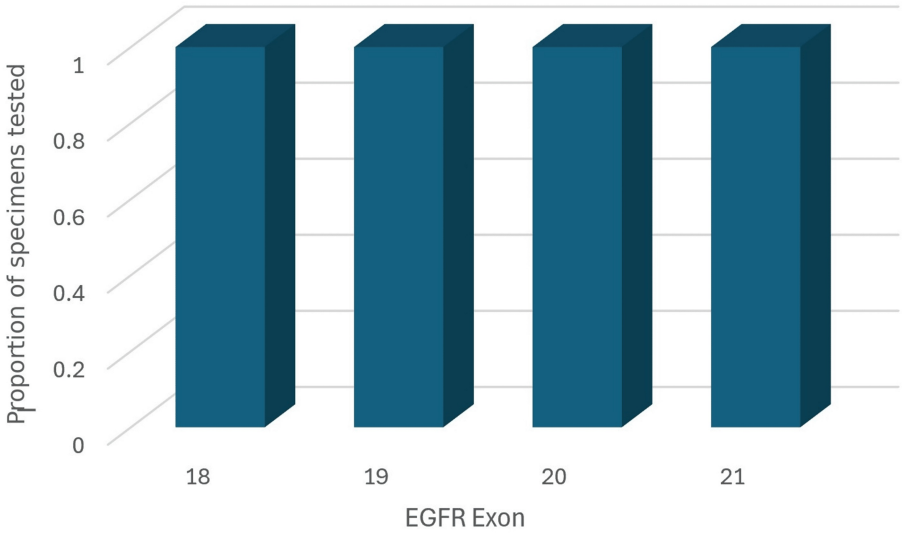



**FIGURE 2: EGFR mutation analysis results for exons 18, 19, 20, and 21. Result 1 indicates the absence of detectable EGFR mutations, whereas Result 2 corresponds to the "Not Applicable" output reported by the assay. No borderline, equivocal, or indeterminate results were observed.**

EGFR: epidermal growth factor receptor

## Results by histological subtype

*EGFR* mutation analysis yielded uniformly negative results across all histological subtypes included in the study (Figure 3). Samples derived from mucinous ovarian carcinoma showed no detectable *EGFR* mutations in any of the analyzed exons. Similarly, all clear cell ovarian carcinoma samples demonstrated an absence of EGFR alterations. Non-epithelial ovarian tumor specimens also showed no evidence of *EGFR* mutations, and none of the LGSOC samples harbored detectable *EGFR* gene alterations. No histological subtype included in the cohort demonstrated even a single *EGFR* mutation event.

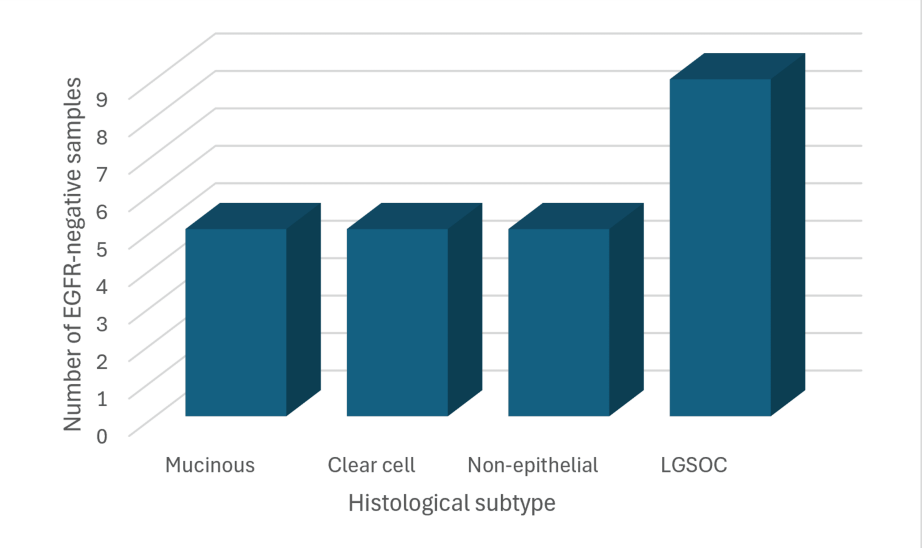



**FIGURE 3: Distribution of EGFR mutation-negative samples across ovarian cancer histological subtypes.**

EGFR: epidermal growth factor receptor

## Reproducibility and inter-run consistency

The *EGFR* mutation testing was performed over multiple independent analytical runs on different dates. All samples yielded concordant results across analytical runs. No deviations between runs were encountered, and all were negative for *EGFR* mutation irrespective of the run date and batch. The reproducibility and the analytical robustness of the testing process are validated by the reliable performance of assay controls from all runs. The consistent performance of the assay controls across all runs supported the validity of the negative results. In summary, extensive *EGFR* mutation analysis of 24 FFPE ovarian cancer tissue samples showed absence of any identified *EGFR* mutations in exons 18-21. All samples were analyzed, all assay controls were performed as intended, and all results were analytically consistent. These results confirm no detectable *EGFR* mutations were identified in the study cohort of uncommon ovarian cancer histological subtypes assayed using the Cobas® EGFR Tissue Test v2.

## Discussion

Ovarian cancer is a biologically heterogeneous group of malignancies, with unique molecular drivers among different histological subtypes [8,9]. HGSOC has already been well studied at a molecular level, but other histological subtypes are often underrepresented in large genomic datasets. In this regard, the potential oncogenic role of *EGFR* mutation in ovarian cancer has been controversial. We performed extensive *EGFR* mutation analysis in 24 ovarian cancer tissue samples that included mucinous carcinoma, clear cell carcinoma, non-epithelial ovarian tumors, and LGSOC. No activating *EGFR* mutations were detected in exons 18, 19, 20, and 21. These results contribute to the molecular characterization of these subtypes and are consistent with previous studies reporting a low prevalence or absence of activating *EGFR* mutations in rare ovarian cancer subtypes and are consistent with published primary studies demonstrating low or absence of *EGFR* mutations within ovarian carcinoma [10,11].

Early sequence-based studies of the molecular landscape of ovarian cancer demonstrated that activating *EGFR* mutations, which are well-established drivers in non-small cell lung cancer, are uncommon in ovarian tumors [11]. In a seminal study evaluating *EGFR* mutation status in ovarian carcinoma, Schilder et al. reported a complete absence of activating *EGFR* mutations despite detectable EGFR protein expression in a subset of cases [11]. Similarly, Lassus et al. found no activating *EGFR* mutations in ovarian cancer samples, highlighting a fundamental biological difference between ovarian and lung carcinogenesis [10]. The uniformly negative *EGFR* mutation status observed in our cohort is therefore consistent with the existing data.

An important distinction highlighted in the literature is the difference between *EGFR* expression and *EGFR* mutation status. Several original studies have demonstrated that *EGFR* overexpression or gene amplification may be present in ovarian cancer without corresponding activating mutations (Desmedt et al., 2008) [12]. This discrepancy may partly explain the rationale for evaluating EGFR-targeted therapies in ovarian cancer despite the absence of biomarkers of response. Our findings further support the notion that EGFR expression alone does not necessarily indicate the presence of an EGFR mutation-driven oncogenic pathway. EGFR-driven oncogenic pathway in ovarian cancer.

When examined by histological subtype, our results are concordant with the already established subtype-specific molecular profile. Mucinous ovarian carcinoma is recognized as a molecularly distinct entity, most commonly characterized by *KRAS* mutations and alterations in the MAPK signaling pathway. Multiple previous genomic studies have demonstrated that *EGFR* mutations are rare or absent in mucinous ovarian carcinoma [12]. The absence of *EGFR* mutations in all mucinous carcinoma samples analyzed in the present study further supports these findings and suggests that *EGFR* mutation testing has limited diagnostic or therapeutic value in this subtype.

Clear cell ovarian carcinoma is similarly characterized by a distinct molecular landscape, with frequent mutations in ARID1A, PIK3CA, and components of the PI3K/AKT pathway [13,14]. Large-scale genomic analyses have consistently shown that *EGFR* mutations are not a characteristic feature of clear cell ovarian carcinoma [13,14]. Our results, demonstrating no *EGFR* mutations in clear cell carcinoma samples, further support these observations and emphasize the importance of focusing molecular testing strategies on pathways with established relevance in this subtype.

LGSOC represents another molecularly distinct entity, driven predominantly by aberrations in the MAPK signaling cascade, particularly *KRAS* and *BRAF* mutations. Original studies have distinguished LGSOC from high-grade serous carcinoma, with distinct clinical behavior and molecular drivers [15]. The absence of *EGFR* mutations in all LGSOC samples analyzed in our study is consistent with the existing data.

Non-epithelial ovarian tumors are a heterogeneous group of rare neoplasms with varying molecular features. Although few studies explore the prevalence of *EGFR* mutations in these tumors, existing evidence supports the idea that EGFR has a modest role in this group [9]. The absence of *EGFR* mutations in non-epithelial ovarian tumors in our cohort is consistent with this observation that alternative pathways may be more prominent in their tumorigenesis. The clinical potential of *EGFR* mutation testing in ovarian cancer has been investigated by several prospective treatment trials. Phase II trials evaluating EGFR tyrosine kinase inhibitors (e.g., gefitinib and erlotinib) in ovarian cancer showed no clinical benefit, even in patients with *EGFR* overexpression [16]. Later correlative analyses indicated that this lack of activating *EGFR* mutations

was the main reason for this treatment failure. The critical investigation of targeted therapy failures in ovarian cancer has highlighted the need to consider selecting the proper biomarker when developing targeted therapy approaches [17]. Molecular analysis of the current study provides further evidence to these clinical observations.

From a clinical perspective, our findings demonstrate the importance of histology-based molecular testing. Instead of using uniform molecular panels across all ovarian cancer subtypes, testing should focus on genes and pathways that have been shown to be relevant to each histological entity. Frameworks of precision oncology have begun to prioritize a rational selection of biomarkers, which may preclude unnecessary testing [18].

### Study limitations

Our study is characterized by certain limitations. Firstly, the small sample size limits the ability to detect very rare EGFR mutation events. It limits the statistical power and the ability to generalize the results to the entire population of patients with endometrial cancer. In addition, our analysis is confined to EGFR mutations and did not consider other genomic mutations that may contribute to tumor pathogenesis or represent potential therapeutic targets. Thus, further studies incorporating broader genomic profiling approaches may help elucidate the molecular drivers of these subtypes. The samples were sourced from a single center, potentially introducing geographical bias. The testing methods employed were focused on identifying known mutation types, which may overlook rarer or novel variants. Future multi-center studies with a larger and more diverse cohort are necessary to validate these preliminary results and further minimize bias.

Despite these limitations, the present study makes a substantial contribution to the existing literature by providing a comprehensive assessment of EGFR mutation status in a group of ovarian cancer using a clinically approved, evidence-based assay. The absence of any EGFR mutation in all considered samples is consistent with the available molecular studies suggesting that the reflex testing of EGFR mutations in clinical and functional settings offers little if any therapeutic benefit. In conclusion, the findings suggest that activating EGFR mutations are unlikely to represent a frequent molecular event in the rare ovarian cancer histological subtypes examined in this study.

## Conclusions

We defined the *EGFR* mutation status in rare epithelial and non-epithelial subtypes of ovarian cancer. No activating EGFR mutations were identified in exons 18-21 among the analyzed samples, as evaluated by a real-time PCR assay. These findings are in agreement with the existing evidence supporting the minor role of EGFR-mediated oncogenesis in ovarian tumors. While *EGFR* mutation status constitutes a cornerstone of targeted therapy in non-small cell lung cancer, clinical trials have shown limited efficacy of EGFR-targeted agents in ovarian cancer and suggest alternative biomarkers to guide treatment. Importantly, our study provides further insight into the molecular characteristics of tumors where evidence remains scarce and highlights the lack of clinical benefit of routine *EGFR* mutation testing. In conclusion, our study identifies the presence of specific *EGFR* mutations in a subset of patients with endometrial cancer, suggesting a potential role for targeted molecular evaluation. However, due to the small sample size and the exploratory nature of this research, these findings should be interpreted with caution. Further large-scale clinical trials are warranted to establish the definitive clinical relevance of these mutations and their impact on personalized treatment strategies.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

**Concept and design:** Anthi-Maria Papahliou, Constantinos G. Zografos, Garyfalia Bletsa, Flora Zagouri

**Acquisition, analysis, or interpretation of data:** Anthi-Maria Papahliou, Eleni Zografos, Ekaterini Domali, Flora Zagouri, Kallirroi Goula, Amalia Sofianidi

**Drafting of the manuscript:** Anthi-Maria Papahliou, Constantinos G. Zografos, Garyfalia Bletsa, Flora Zagouri, Kallirroi Goula, Amalia Sofianidi

**Critical review of the manuscript for important intellectual content:** Anthi-Maria Papahliou, Eleni Zografos, Ekaterini Domali, Flora Zagouri

**Supervision:** Flora Zagouri

### Disclosures

**Human subjects:** Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Scientific Council, General Hospital 'Alexandra', Athens, Greece issued approval 712/22-11-2022. **Animal subjects:** All authors have confirmed that this study did not involve animal subjects or tissue. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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