POLISH GYNECOLOGY

# GINEKOLOGIA POLSKA

Edited since 1922

no 6/vol 96/2025

ORGAN POLSKIEGO TOWARZYSTWA GINEKOLOGÓW I POŁOŻNIKÓW
THE OFFICIAL JOURNAL OF THE POLISH SOCIETY OF GYNECOLOGISTS AND OBSTETRICIANS

IF: 1.0, MNiSW: 40

## ORIGINAL PAPERS

Tubo-ovarian abscess management in our clinic
Necdet Öncü, Ali Buhur, Hüseyin Güray Biçer
433

NMR-based serum metabolomics in patients with low-differentiated serous ovarian cancer
Mateusz M. Klimek, Agnieszka Skorupa, Mateusz Ciszek, Tomasz Cichon, Bartosz Cichon, Lukasz Boguszewicz, Andrzej Witek, Maria Sokol
437

The impact of mismatch repair (MMR), p53, and LCAM-1 immunohistochemical expression on prognosis in low-risk endometrial cancer
Şener Gezer, Büşra Yaprak Bayrak, Müzeyyen Dilşad Eser, Esra Betül Tunce
446

Hysteroscopic isthmocele resection: evaluating quality of life and symptom improvements based on isthmocele volume
Mehmet Faruk Olcenoglu, Merve Olcenoglu, Mesut Onal, Ayse Zehra Ozdemir
454

Association between the triglyceride-glucose index in third trimester pregnant women and neonatal birth weight
Jialei Shen, Wenhui Liu, Kedan Cao, Feng Wang
462

Effects of gestational diabetes mellitus with different birth weight on genetic metabolism of newborns. A retrospective cohort study
Dandan Xia, Huiyan Wang, Yuqi Yang, Wenli Wang
469

Major opinion about motherhood among women with Turner syndrome
Malgorzata Wiecek, Wiktoria Kempinska, Aneta Gawlik-Starzyk
476

Transforming clinical practice in just one year: lessons from external cephalic version success
Maisa Manasar-Dyrbus, Bohdan Seifert, Agnieszka Drosdzol-Cop, Rafal Stojko, Jakub Staniczek
482



GINEKOLOGIA POLSKA 2025, VOL 96, NO 6, PAGES 433–518

ISSN 0017–0011
e-ISSN 2543–6767

VIA MEDICA

Favorite Export

JCR Year 2024

# Ginekologia Polska

Open Access since 2007

ISSN

**0017-0011**

EISSN

**2543-6767**

JCR ABBREVIATION

**GINEKOL POL**

ISO ABBREVIATION

**Ginekol. Pol.**

## Journal information

EDITION

Science Citation Index Expanded (SCIE)

CATEGORY

OBSTETRICS & GYNECOLOGY

| LANGUAGES | REGION | 1ST ELECTRONIC JCR YEAR |
|---|---|---|
| Polish | POLAND | 2010 |

## Publisher information

| PUBLISHER | ADDRESS | PUBLICATION FREQUENCY |
|---|---|---|
| VIA MEDICA | UL SWIETOKRZYSKA 73, 80-180 GDANSK, POLAND | 12 issues/year |

# Journal's performance

## Journal Impact Factor

The Journal Impact Factor (JIF) is a journal-level metric calculated from data indexed in the Web of Science Core Collection. It should be used with careful attention to the many factors that influence citation rates, such as the volume of publication and citations characteristics of the subject area and type of journal. The Journal Impact Factor can complement expert opinion and informed peer review. In the case of academic evaluation for tenure, it is inappropriate to use a journal-level metric as a proxy measure for individual researchers, institutions, or articles. Learn more

| 2024 JOURNAL IMPACT FACTOR | JOURNAL IMPACT FACTOR WITHOUT SELF CITATIONS |
|---|---|
| **1.0** | 0.9 |
| View calculation | View calculation |

### Journal Impact Factor Trend 2024

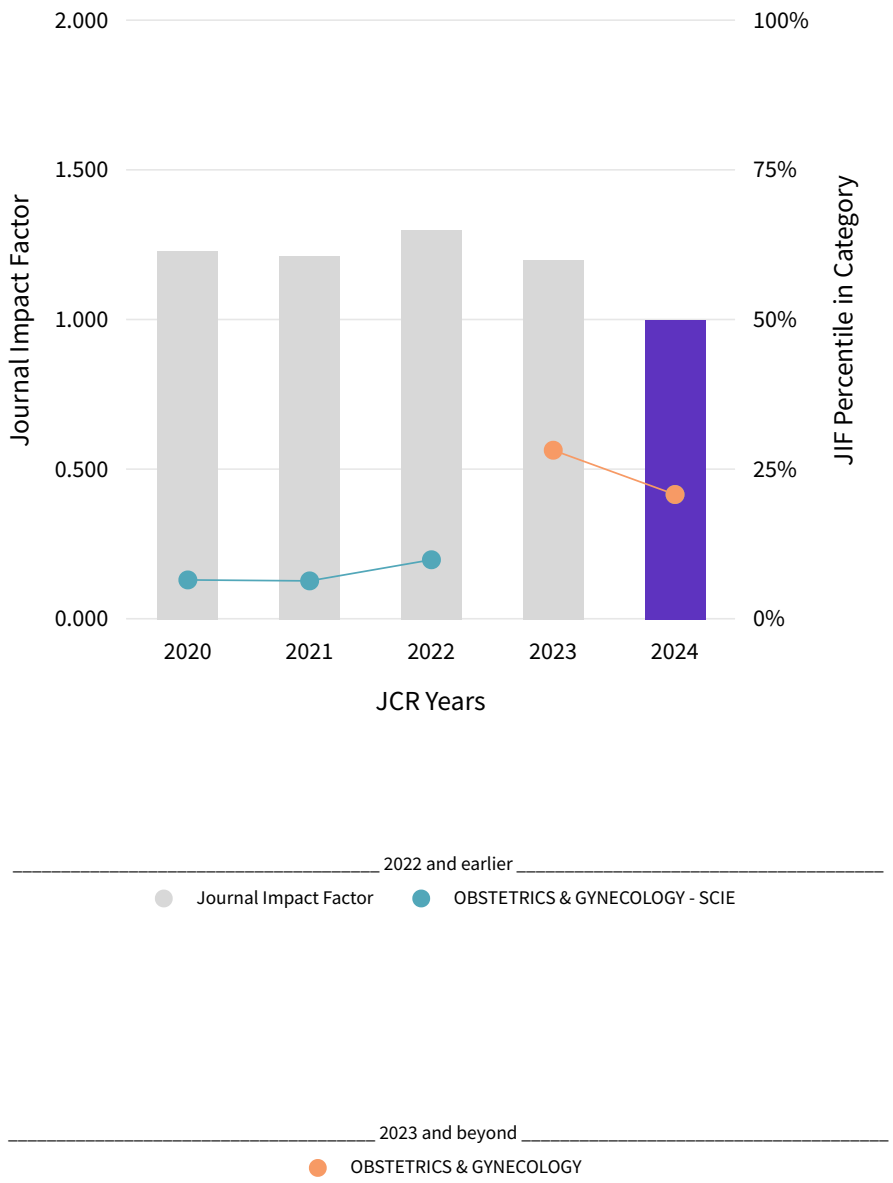
Export

### Journal Impact Factor contributing items

Export

Citable items (340) Citing Sources (203)

| TITLE | CITATION COUNT |
|---|---|
| The impact of COVID-19 pandemic on reproductive intentions among the Poli… | 7 |
| Endometriosis is associated with an increased whole-blood thrombogenicit… | 5 |




View all years

| | |
|---|---|
| Ischemia modified albumin levels in intrauterine growth restriction: levels ar… | 5 |
| Polish Society of Gynecologists and Obstetricians (PTGiP) and Polish Societ… | 5 |
| The Polish Society of Gynecologists and Obstetricians Expert Group… | 5 |
| Validation of the Numerical Anxiety Rating Scale in postpartum females: a… | 5 |
| Alpha-fetoprotein (AFP)-new aspects of a well-known marker in perinatology | 4 |
| Chronic endometritis - is it time to clarify diagnostic criteria? | 4 |
| Correlation between human papillomavirus infection and… | 4 |
| Evaluation of the prevalence of folic acid supplementation before conception an… | 4 |

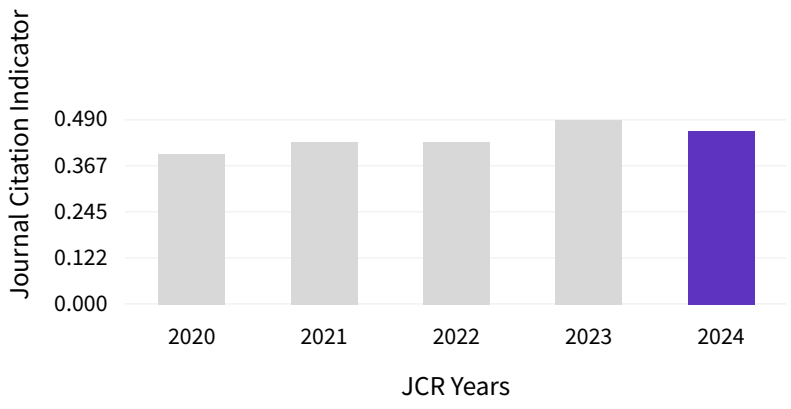
View All in Web of Science

## Journal Citation Indicator (JCI)

Export

**0.46**

The Journal Citation Indicator (JCI) is the average Category Normalized Citation Impact (CNCI) of citable items (articles & reviews) published by a journal over a recent three year period. The average JCI in a category is 1. Journals with a JCI of 1.5 have 50% more citation impact than the average in that category. It may be used alongside other metrics to help you evaluate journals. Learn more



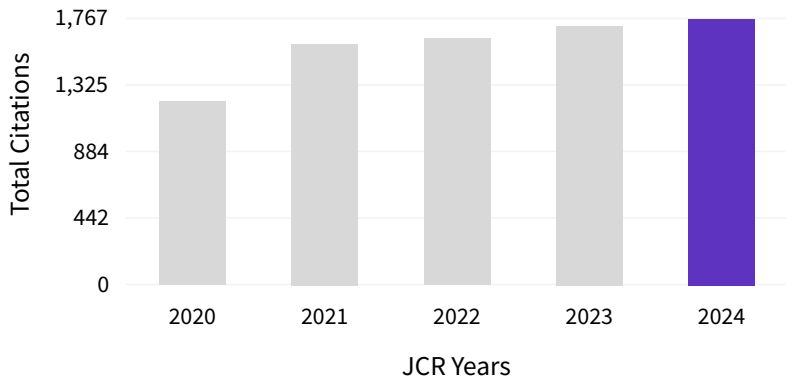

View all years

## Total Citations

Export

**1,767**

The total number of times that a journal has been cited by all journals included in the database in the JCR year. Citations to journals listed in JCR are compiled annually from the JCR years combined database, regardless of which JCR edition lists the journal.




View all years

# Citation distribution

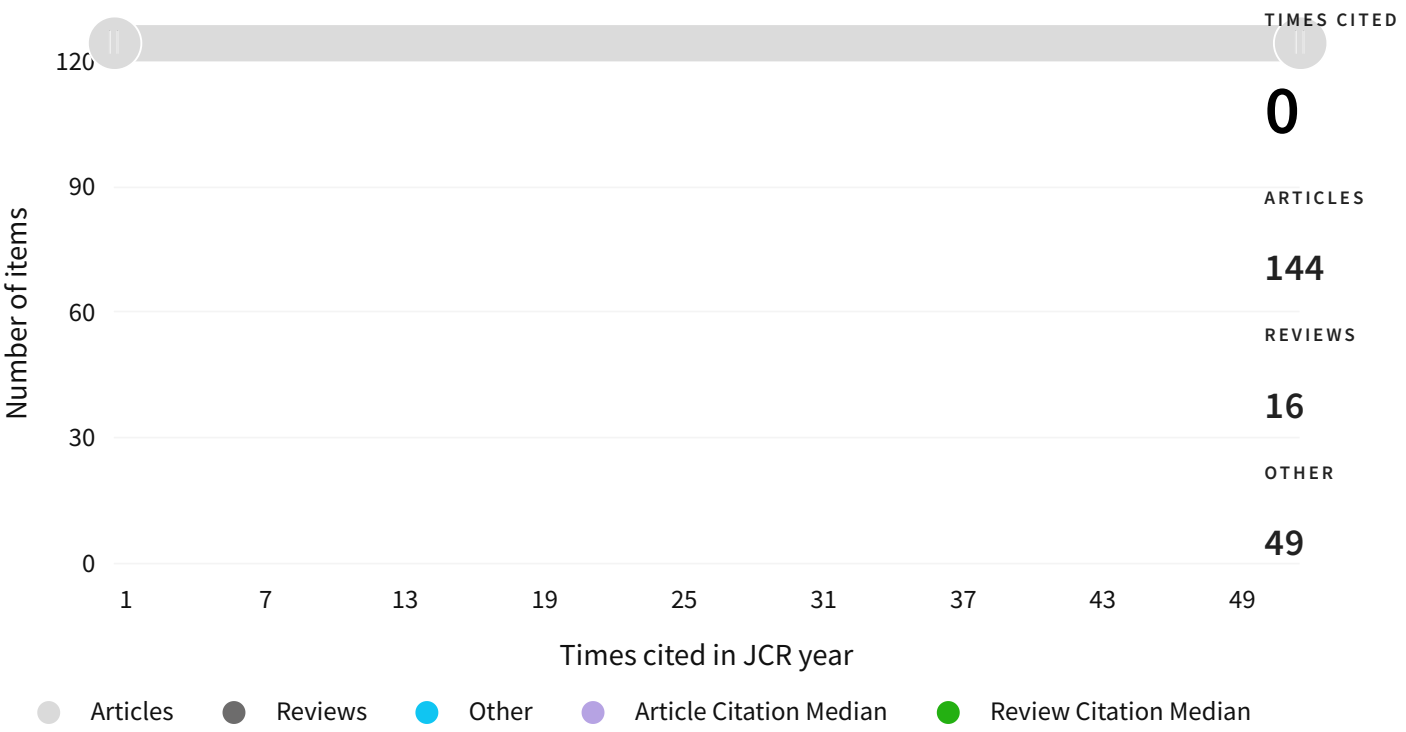
Export

The Citation Distribution shows the frequency with which items published in the year or two years prior were cited in the JCR data year (i.e., the component of the calculation of the JIF). The graph has similar functionality as the JIF Trend graph, including hover-over data descriptions for each data point, and an interactive legend where each data element's legend can be used as a toggle. You can view Articles, Reviews, or Non-Citable (other) items to the JIF numerator. Learn more

ARTICLE CITATION MEDIAN
1

REVIEW CITATION MEDIAN
1

UNLINKED CITATIONS
12




TIMES CITED
0

ARTICLES
144

REVIEWS
16

OTHER
49

# Open Access (OA)

Export

The data included in this tile summarizes the items published in the journal in the JCR data year and in the previous two years. This three-year set of published items is used to provide descriptive analysis of the content and community of the journal. Learn more

## Items

| TOTAL CITABLE | % OF CITABLE OA |
|---|---|
| 427 | 97.89% |

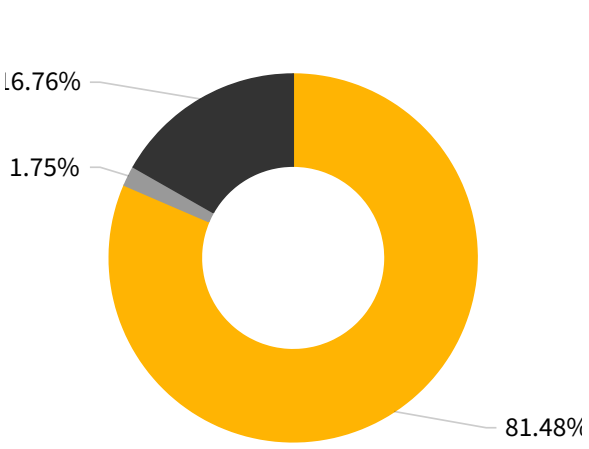
CITABLE

GOLD OPEN ACCESS
418 / 81.48%

SUBSCRIPTION AND FREE TO READ
9 / 1.75%

NON-CITABLE

OTHER (NON CITABLE ITEMS)
86 / 16.76%




## Citations*

| TOTAL CITABLE | % OF CITABLE OA |
|---|---|
| 350 | 96.86% |

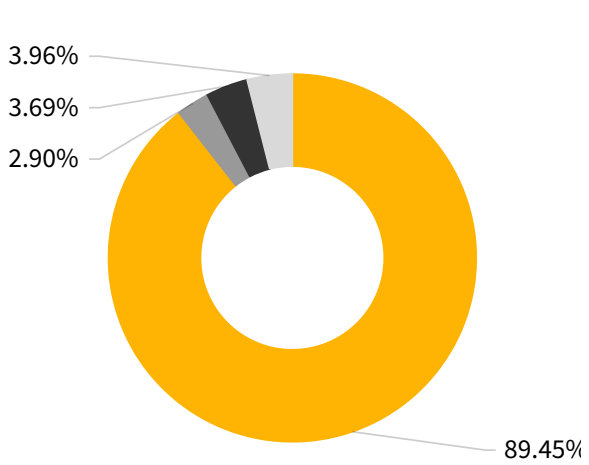
CITABLE

GOLD OPEN ACCESS
339 / 89.45%

SUBSCRIPTION AND FREE TO READ
11 / 2.90%

NON-CITABLE

OTHER (NON CITABLE ITEMS)
14 / 3.69%



UNLINKED CITATIONS

15 / 3.96%

*Citations in 2024 to items published in [2022 - 2024]

# Rank by Journal Impact Factor

Journals within a category are sorted in descending order by Journal Impact Factor (JIF) resulting in the Category Ranking below. A separate rank is shown for each category in which the journal is listed in JCR. Beginning in 2023, ranks are calculated by category.
Learn more

CATEGORY

OBSTETRICS & GYNECOLOGY

**112/141**

| JCR YEAR | JIF RANK | JIF QUARTILE | JIF PERCENTILE |
|---|---|---|---|
| 2024 | 112/141 | Q4 | 20.9 |
| 2023 | 98/136 | Q3 | 28.3 |

**Rank by JIF before 2023 for OBSTETRICS & GYNECOLOGY**

EDITION

Science Citation Index Expanded (SCIE)

| JCR YEAR | JIF RANK | JIF QUARTILE | JIF PERCENTILE |
|---|---|---|---|
| 2022 | 77/85 | Q4 | 10.0 |
| 2021 | 80/85 | Q4 | 6.47 |
| 2020 | 78/83 | Q4 | 6.63 |
| 2019 | 75/82 | Q4 | 9.15 |

# Rank by Journal Citation Indicator (JCI)

Journals within a category are sorted in descending order by Journal Citation Indicator (JCI) resulting in the Category Ranking below. A separate rank is shown for each category in which the journal is listed in JCR. Data for the most recent year is presented at the top of the list, with other years shown in reverse chronological order. Learn more

CATEGORY

OBSTETRICS & GYNECOLOGY

**104/141**

| JCR YEAR | JCI RANK | JCI QUARTILE | JCI PERCENTILE |
|---|---|---|---|
| 2024 | 104/141 | Q3 | 26.60 |
| 2023 | 94/136 | Q3 | 31.25 |
| 2022 | 97/131 | Q3 | 26.34 |
| 2021 | 95/128 | Q3 | 26.17 |
| 2020 | 91/121 | Q4 | 25.21 |
| 2019 | 93/121 | Q4 | 23.55 |
| 2018 | 91/120 | Q4 | 24.58 |

# Citation network

## Cited Half-life

**6.2 years**

The Cited Half-Life is the median age of the items in this journal that were cited in the JCR year. Half of a journal's cited items were published more recently than the cited half-life.

TOTAL NUMBER OF CITES

**1,767**

NON SELF-CITATIONS

**1,681**

SELF-CITATIONS

**86**

## Citing Half-life

**7.8 years**

The Citing Half-Life is the median age of items in other publications cited by this journal in the JCR year.

TOTAL NUMBER OF CITES

**3,346**

NON SELF-CITATIONS

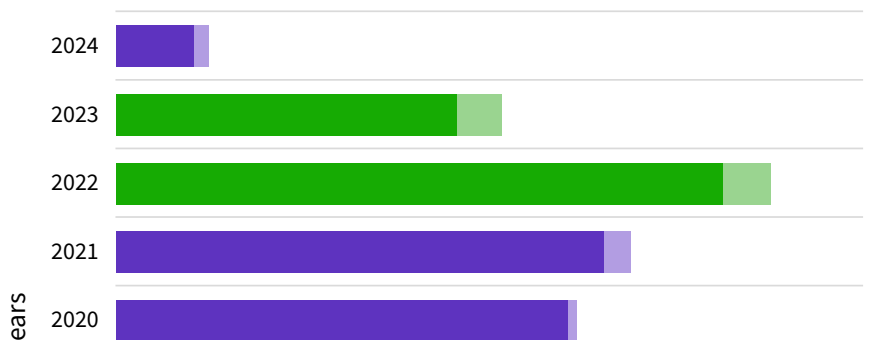
**3,260**

SELF-CITATIONS

**86**

Cited Half-life Data | Citing Half-life Data

Export

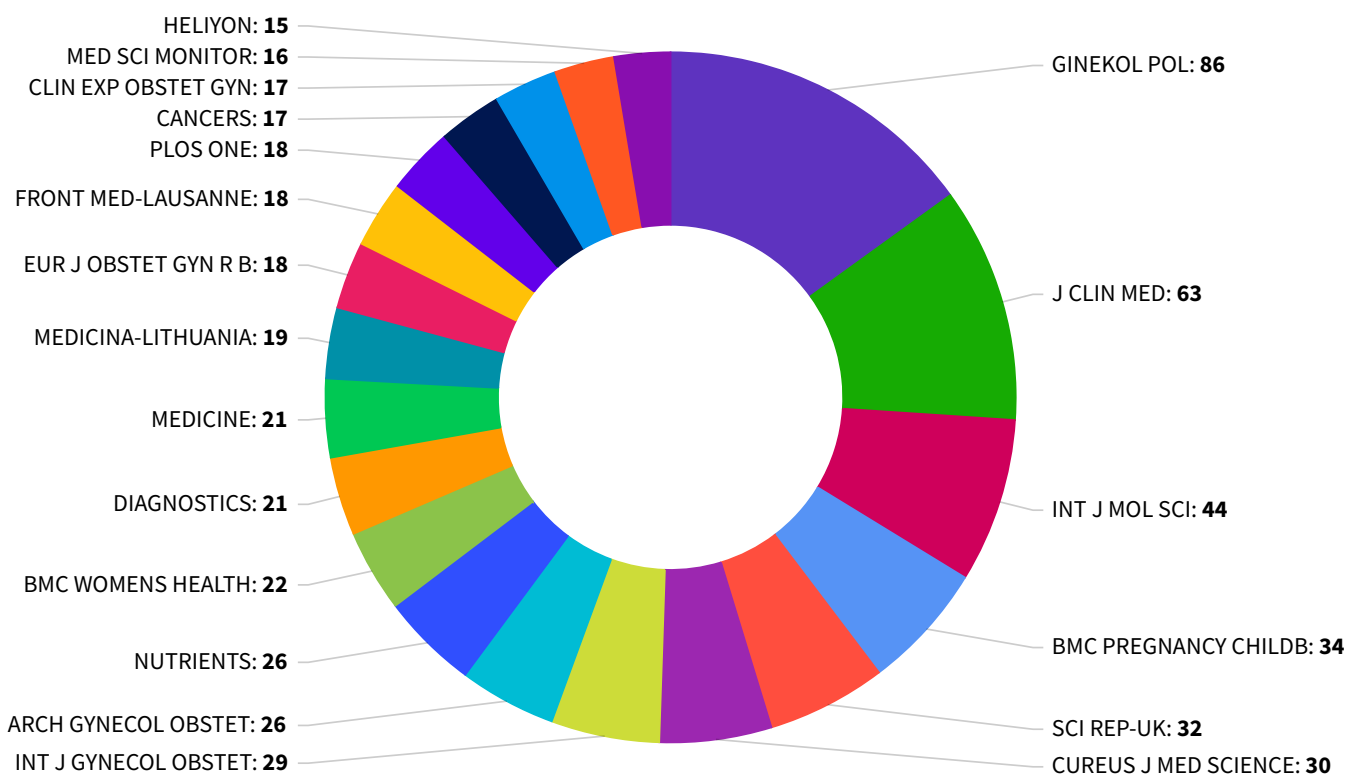



| CITED YEAR | # OF CITES FROM 2024 | CUMULATIVE % | # OF CITING SOURCES |
|---|---|---|---|
| **All years** | **1,767 citations** | **100.00%** | **702 sources** |
| 2024 | 31 citations | 1.75% | 25 sources |
| 2023 | 129 citations | 9.05% | 89 sources |
| 2022 | 219 citations | 21.45% | 140 sources |
| 2021 | 172 citations | 31.18% | 120 sources |
| 2020 | 154 citations | 39.90% | 115 sources |

# Journal Citation Relationships

**Cited Data** Citing Data

Top 20 journals citing GINEKOL POL by number of citations




# Content metrics

## Source data

This tile shows the breakdown of document types published by the journal. Citable Items are Articles and Reviews. For the purposes of calculating JIF, a JCR year considers the publications of that journal in the two prior years. Learn more

**87 total citable items**

| | ARTICLES | REVIEWS | COMBINED(C) | OTHER DOCUMENT TYPES(O) | PERCENTAG |
|---|---|---|---|---|---|
| NUMBER IN JCR YEAR 2024 (A) | 76 | 11 | 87 | 28 | 76% |
| NUMBER OF REFERENCES (B) | 2,811 | 361 | 3,172 | 174 | 95% |
| RATIO (B/A) | 37.0 | 32.8 | 36.5 | 6.2 | |

## Average JIF Percentile

Export

The Average Journal Impact Factor Percentile takes the sum of the JIF Percentile rank for each category under consideration, then calculates the average of those values. Learn more

| ALL CATEGORIES AVERAGE | OBSTETRICS & GYNECOLOGY |
|---|---|
| 20.9 | 20.9 |

## Contributions by organizations

Export

Organizations that have contributed the most papers to the journal in the most recent three-year period. Learn more

| RANK | ORGANIZATION | COUNT |
|---|---|---|
| 1 | MEDICAL UNIVERSITY OF SILESIA | 63 |
| 2 | JAGIELLONIAN UNIVERSITY | 50 |
| 3 | MEDICAL UNIVERSITY OF WARSAW | 48 |
| - | POZNAN UNIVERSITY OF MEDICAL SCIENCES | 48 |
| 5 | MEDICAL UNIVERSITY OF LUBLIN | 37 |
| 6 | MEDICAL | 31 |

## Contributions by country/region

Export

Countries or Regions that have contributed the most papers to the journal in the most recent three-year period. Learn more

| RANK | COUNTRY / REGION | COUNT |
|---|---|---|
| 1 | POLAND | 284 |
| 2 | TURKIYE | 94 |
| 3 | CHINA MAINLAND | 82 |
| 4 | GERMANY (FED REP GER) | 9 |
| 5 | EGYPT | 7 |
| 6 | CZECH REPUBLIC | 6 |
| - | SERBIA | 6 |
| 8 | KAZAKHSTAN | 5 |

# Additional metrics

## Eigenfactor Score

**0.00154**

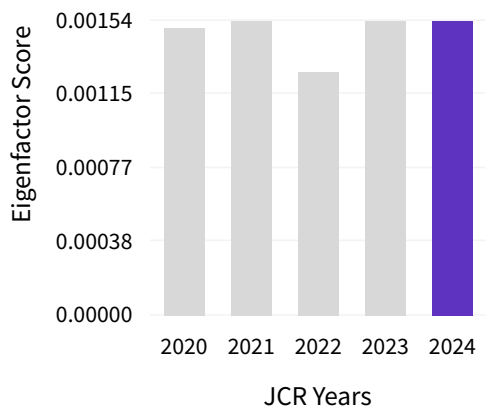
## Normalized Eigenfactor

**0.34526**

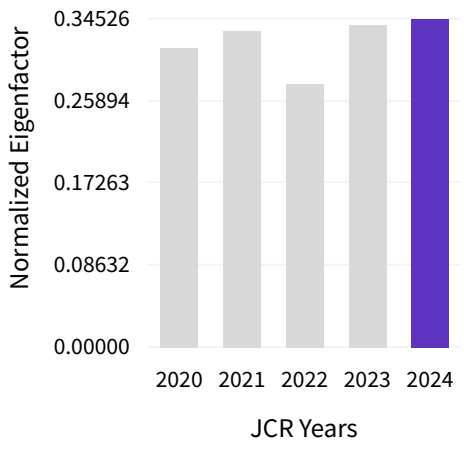
## Article influence score

**0.272**

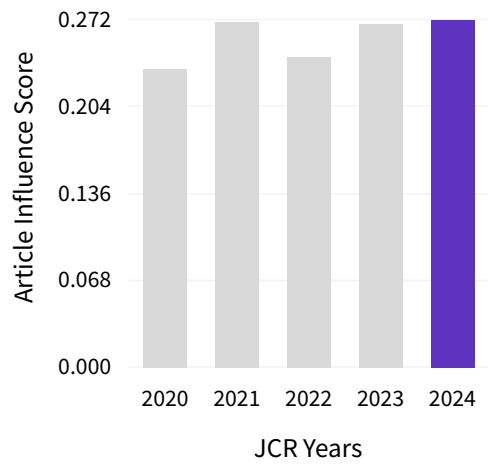
The Eigenfactor Score is a reflection of the density of the network of citations around the journal using 5 years of cited content as cited by the Current Year. It considers both the number of citations and the source of those citations, so that highly cited sources will influence the network more than less cited sources. The Eigenfactor calculation does not include journal self-citations. Learn more




The Normalized Eigenfactor Score is the Eigenfactor score normalized, by rescaling the total number of journals in the JCR each year, so that the average journal has a score of 1. Journals can then be compared and influence measured by their score relative to 1. Learn more




The Article Influence Score normalizes the Eigenfactor Score according to the cumulative size of the cited journal across the prior five years. The mean Article Influence Score for each article is 1.00. A score greater than 1.00 indicates that each article in the journal has above-average influence. Learn more

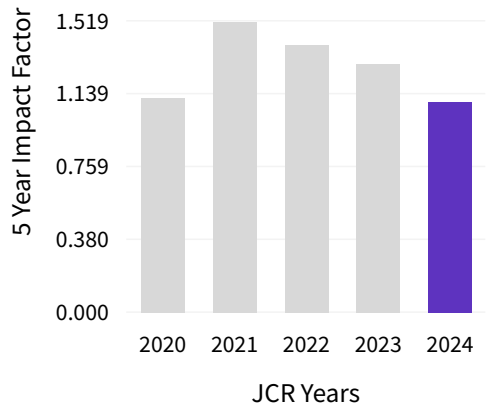



## 5 Year Impact Factor

**1.1**

View Calculation

The 5-year Impact Factor is the average number of times articles from the journal published in the past five years have been cited in the JCR year. It is calculated by dividing the number of citations in the JCR year by the total number of articles published in the five previous years. Learn more

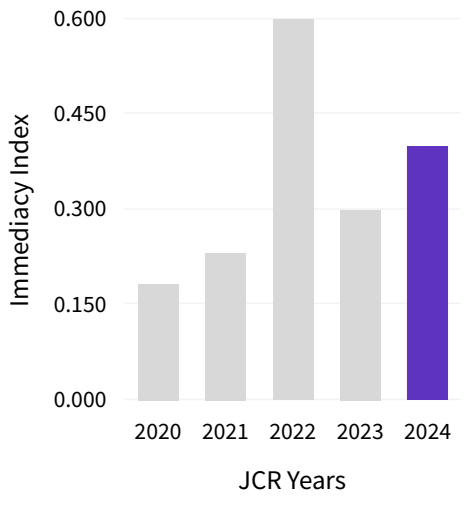



## Immediacy Index

**0.4**

View Calculation

The Immediacy Index is the count of citations in the current year to the journal that reference content in this same year. Journals that have a consistently high Immediacy Index attract citations rapidly. Learn more




© 2025 Clarivate Legal center

Privacy notice

Cookie policy

Terms of Use

Copyright notice

Accessibility

Help

VIA MEDICA Journals

Log In

POLISH GYNECOLOGY
GINEKOLOGIA POLSKA
Edited since 1922
ORGAN POLSKIEGO TOWARZYSTWA GINEKOLOGÓW I POŁOŻNIKÓW
THE OFFICIAL JOURNAL OF THE POLISH SOCIETY OF GYNECOLOGISTS AND OBSTETRICIANS



# Editorial Team

## Editor-in-Chief

**Rafał Stojko** Poland Affiliation

## Past Editor-in-Chief

**Ewa Nowak-Markwitz** Poland Affiliation

**Marek Spaczyński** Poland Affiliation

## Deputy Editor-in-Chief

**Agnieszka Barbara Drosdzol-Cop** Poland Affiliation

## Section Editors

**Paweł Basta** Poland

**Mariusz Bidziński** Poland

**Karol Bielasik** Poland Affiliation

**Wojciech Cnota** Poland Affiliation

**Iwona Gabriel** Poland Affiliation

**Paweł Guzik** Poland Affiliation

**Krzysztof Nowosielski** Poland Affiliation

**Agnieszka Rychlik** Poland

**Maria Szubert** Poland Affiliation

**Sławomir Wożniak** Poland Affiliation

## Publishers Editor

**Karolina Klimek** Poland

## Editorial Advisory Board

**Elizabeth A. Bonney** Affiliation

**Grzegorz Bręborowicz** Poland Affiliation

**Zana Bumbuliene** Lithuania Affiliation

**Gian Carlo di Renzo** Italy Affiliation

**Krzysztof Drews** Poland Affiliation

**Dan Farine** Canada Affiliation

**Sonia Grover** Australia Affiliation

**Moshe Hod** Israel Affiliation

**Grzegorz Jakiel** Poland Affiliation

**Jacques Jani** Affiliation

**Agata Karowicz-Bilińska** Poland Affiliation

**Jan Kotarski** Poland Affiliation

**Vernon J. Louw** Affiliation

**Kypros Nicolaides** United Kingdom Affiliation

**Zuzana Niznanska** Slovakia Affiliation

**Przemysław Oszukowski** Poland Affiliation

**Tomasz Paszkowski** Poland Affiliation

**Ritsuko K. Pooh** Japan Affiliation

**Krzysztof Preis** Poland Affiliation

**Joseph G. Schenker** Germany Affiliation

**Jim G. Thornton** United Kingdom Affiliation

**Mirosław Wielgoś** Poland Affiliation

**Paul Wood** United Kingdom Affiliation

**Sławomir Wołczyński** Poland Affiliation

**Mariusz Zimmer** Poland Affiliation

**Paolo Zola** Italy Affiliation

Copyright © 2023-2025 Via Medica | Regulations | Cookie policy | Privacy Statement | Legal Note

VIA MEDICA

ORIGINAL PAPER / GYNECOLOGY

Ginekologia Polska
2025, vol. 96, no. 6, 446–453
Copyright © 2025 PTGiP
ISSN 0017–0011, e-ISSN 2543–6767

DOI: 10.5603/gpl.101983

# The impact of mismatch repair (MMR), p53, and LCAM-1 immunohistochemical expression on prognosis in low-risk endometrial cancer

**Şener Gezer[1], Büşra Yaprak Bayrak[2], Müzeyyen Dilşad Eser[1], Esra Betül Tunce[2]**

[1]*Department of Obstetrics and Gynecology, Kocaeli University School of Medicine, Kocaeli, Türkiye*
[2]*Department of Pathology, Kocaeli University School of Medicine, Kocaeli, Türkiye*

## ABSTRACT

**Objectives:** To investigate the relationship between mismatch repair (MMR) deficiency, *TP53*, and L1 cell adhesion molecule (L1CAM) immunohistochemical staining and their impact on progression-free survival (PFS) and overall survival (OS) in low-risk endometrial cancer.

**Material and methods:** A total of 253 low-risk endometrial cancer patients were retrospectively screened. Immunohistochemical stains were applied to tumor tissue samples to assess MMR deficiency, *TP53*, and L1CAM expression, and survival analysis were performed.

**Results:** The expected PFS time was 78.6 months for the MMR-proficient group and 70.3 months for the MMR-deficient group ($p = 0.011$). OS was 71.6 months for the MMR-proficient group and 68.2 months for the MMR-deficient group ($p = 0.755$). L1CAM overexpression was associated with a poorer PFS, 62.7 months compared to 77.7 months ($p = 0.039$). However, there was no statistically significant difference in OS, 58.5 months versus 72.1 months, respectively ($p = 0.242$). p53 abnormal (p53-abn) staining was associated with a worse prognosis in terms of PFS, 62.8 months versus 77.7 months ($p = 0.035$), and OS, 43.4 months versus 73 months, respectively ($p < 0.001$), compared to patients with wild-type staining.

No significant statistical relationship was observed in survival times concerning tumor diameter, grade, and lymphadenectomy status. In a multivariate analysis, MMR deficiency emerged as an independent poor prognostic factor for PFS, while p53-abn was identified as an independent poor prognostic factor for OS.

**Conclusions:** p53-abn staining was associated with a poor prognosis for both PFS and OS in low-risk endometrial cancer patients. Meanwhile, MMR deficiency and L1CAM positivity were found to be associated solely with a poorer prognosis for PFS.

**Keywords:** low-risk endometrial cancer; mismatch repair; *TP53*; L1CAM

Ginekologia Polska 2025; 96, 6: 446–453

## INTRODUCTION

Patients with endometrial cancer are typically diagnosed through surgery following preoperative biopsy and imaging. Subsequent adjuvant treatment is determined based on their relapse risk classification. The low-risk category includes patients with grade 1 or 2 endometrioid-type tumors confined to the uterus, with less than half myometrial invasion and minimal or no lymphovascular invasion. These patients generally have a high life expectancy and long disease-free survival, and adjuvant therapy (radiotherapy or chemotherapy) is often unnecessary [1]. Although early-stage endometrial cancer is reportedly associated with survival rates of 85% to 95%, there remains a slight risk of recurrence, and the therapeutic success rates of approximately 40% can negatively impact overall survival (OS) [2–5].

The growing use of molecular classification in clinical practice has enhanced clinicians' understanding of why some patients with low-risk endometrial cancer develop relapse. According to this classification, the low-risk group may include subcategories such as *POLE*-mutated, mismatch repair (MMR)-deficient, *TP53*-mutated, and those with a nonspecific molecular profile, each at varying rates [6, 7]. However, routine molecular profiling for every patient can be expensive. While next-generation sequencing offers

**Corresponding author:**
Şener Gezer
Department of Obstetrics and Gynecology, Kocaeli University School of Medicine, 41380 Kocaeli, Türkiye
e-mail: dr.senergezer@gmail.com

Received: 7.08.2024 Accepted: 5.01.2025 Early publication date: 15.02.2025
This article is available in open access under Creative Common Attribution-Non-Commercial-No Derivatives 4.0 International (CC BY-NC-ND 4.0) license, allowing to download articles and share them with others as long as they credit the authors and the publisher, but without permission to change them in any way or use them commercially.

detailed insights, immunohistochemical (IHC) studies are a more cost-effective alternative. The Proactive Molecular Risk classification tool for Endometrial cancers (ProMisE) reduces costs by using IHC staining for MMR and *TP53*, while still providing comparable survival data [7].

*TP53* regulates the cell cycle, DNA repair, apoptosis, and senescence in response to cellular stress. Loss of p53 function allows cells with genomic damage to survive and divide, promoting tumor formation and progression [8].

The MMR system identifies and repairs DNA replication errors, such as base mismatches and small insertions or deletions, that occur during cell division. MMR is essential for maintaining genome stability. Defects in MMR genes (*e.g.*, *MLH1*, *MSH2*, *MSH6*, and *PMS2*) are associated with microsatellite instability and can lead to colorectal and endometrial cancer [9].

L1 cell adhesion molecule (L1CAM) is a cell adhesion molecule that plays a key role in cell–cell interactions, epithelial-to-mesenchymal transition, and cellular migration. Overexpression of L1CAM is associated with a poor prognosis in patients with endometrial cancer [10–13].

The use of IHC biomarkers such as *TP53*, MMR, L1CAM, estrogen, and progesterone receptors provides useful information in terms of the necessity of adjuvant therapy [14]. Molecular analysis from the PORTEC-3 trial revealed a statistically significant survival benefit for stage I–III endometrial carcinomas with abnormal p53 expression (p53-abn) when treated with combined chemoradiotherapy [15]. However, no clear advantage was observed for MMR-deficient tumors. These tumors are more responsive to immune checkpoint inhibitors due to their high mutational burden and increased production of neoantigens [16]. Although L1CAM expression is associated with a poor prognosis, further research on its impact in low-risk patients is needed.

Studies focused on patients with low-risk endometrial cancer, who typically do not receive adjuvant therapy, are particularly valuable for accurately assessing the prognosis [17]. The present study was designed to evaluate the prognostic value of the IHC markers MMR, *TP53*, and L1CAM in women with low-risk endometrial cancer, in whom the influence of possible confounding factors is minimal.

## MATERIAL AND METHODS

### Study population

This study involved patients with low-risk endometrial cancer who underwent surgery between January 2017 and December 2022 at Kocaeli University Hospital. The patients were retrospectively screened following approval from the ethics committee of Kocaeli University (Approval number: GOKAEK-2022/23.22). Patients with endometrioid-type endometrial cancers, invasion limited to less than half of the myometrium, grades 1 and 2, and no lymphovascular invasion were included in the study population.

The exclusion criteria were non-endometrioid tumor types (*e.g.*, serous, clear cell), grade 3 tumors, myometrial invasion exceeding half of the myometrium, neoadjuvant chemotherapy or radiation therapy, adjuvant chemotherapy or radiation therapy, concomitant malignancies, and loss to follow-up.

For follow-up, patients were scheduled for physical examinations four times during the first 2 years, twice a year for the next 3 years, and annually thereafter. If recurrence was suspected, further evaluations included serum Ca-125 level monitoring and advanced imaging techniques such as magnetic resonance imaging and positron emission tomography scans. Progression-free survival (PFS) was defined as the time from surgery to the occurrence of recurrence or disease progression. OS was defined as the time from surgery to death, with each death considered an event.

### IHC analysis

Four-micrometer sections were prepared from paraffin-embedded, formalin-fixed tissue blocks for each patient. Hematoxylin- and eosin-stained sections were used for histological examination, which was conducted by two experienced pathologists. IHC staining was performed using the following markers: MSH2 (G219-11229, 1:50 dilution; Ventana Medical Systems, Tucson, AZ, USA), MSH6 (SP93, 1:200 dilution; Ventana Medical Systems), PMS2 (A16-4, 1:200 dilution; Ventana Medical Systems), MLH1 (M1, 1:200 dilution; Ventana Medical Systems), *TP53* (DO-7, 1:1000 dilution; DAKO, Glostrup, Denmark), and L1CAM (14.10, 1:100 dilution; BioLegend, San Diego, CA, USA).

MMR deficiency was defined as the complete absence of nuclear staining for at least one MMR protein. L1CAM was considered positive if ≥ 10% of the tumor cells exhibited staining [11]. *TP53* staining was classified as abnormal (p53-abn) in two scenarios: overexpression, referred to as the "all-type" phenotype, or complete absence of expression, referred to as the "null-type" phenotype.

### Statistical analysis

All statistical analyses were conducted using SPSS 20.0 (IBM Corp., Armonk, NY, USA). The assumption of normality was assessed using the Kolmogorov–Smirnov and Shapiro–Wilk tests. Continuous variables, presented as median (interquartile range), were used because of the non-normal distribution of the data. Categorical variables were summarized as counts and percentages. The Mann–Whitney U test was employed to compare continuous variables between groups, and the chi-square test was used to examine associations between categorical variables. Kaplan–Meier

**Table 1. The demographic and clinical characteristics of the patients**

| Variable | n = 253 |
|---|---|
| Age | 58 (53–64) |
| **Age** | |
| < 60 | 145 (52.3%) |
| ≥ 60 | 108 (42.7%) |
| Gravida | 3 (2–5) |
| Parity | 3 (2–4) |
| Body mass index (BMI) | 33 (30–37) |
| **BMI** | |
| < 30 | 60 (23.7%) |
| ≥ 30 | 193 (76.3%) |
| **Menopausal status** | |
| Premenopause | 64 (25.3%) |
| Postmenopause | 189 (74.7%) |
| **Systemic disease** | |
| Hypertension | 149 (58.9%) |
| Diabetes | 94 (37.2%) |
| **Grade** | |
| I | 160 (63.2%) |
| II | 93 (36.8%) |
| **Tumor diameter (cm)** | 3 (2–4) |
| **Tumor diameter** | |
| ≤ 2 cm | 78 (30.8%) |
| > 2 cm | 175 (69.2%) |
| **Lymphadenectomy status** | |
| None | 37 (14.6%) |
| Pelvic | 150 (59.3%) |
| Pelvic and paraaortic | 66 (26.1%) |
| **Pelvic lymph node count** | 19 (13–26) |
| **Paraaortic lymph node count** | 5 (2–8) |
| **Total lymph node count** | 20 (14–26) |

Numerical variables that do not correspond to the normal distribution are shown as Median (25.–75. percentile) and the categorical variables are shown as n (%)

survival analysis with the log-rank test was applied, and multivariate analysis was performed using Cox regression analysis. A p value < 0.05 was considered statistically significant.

## RESULTS

The study enrolled a total of 253 patients with low-risk endometrial cancer for whom both survival data and pathological specimens were accessible. The clinical and pathological characteristics of these patients are summarized in Table 1. The median follow-up time was 38 months. The median PFS was 77.5 months, and the median OS was 71.9 months. Survival analyses for both PFS and OS are presented in Figures 1 and 2.

Of the 253 patients, 72 (28.5%) were found to have at least one MMR deficiency. The distribution of MMR deficiencies was as follows: 20 (7.9%) patients with MSH2 deficiency, 11 (4.3%) with MSH6 deficiency, 56 (22.1%) with MLH1 deficiency, and 52 (20.6%) with PMS2 deficiency. Among the MMR-deficient group, there were four (5.6%) relapses, while in the MMR-proficient group, there was only one (0.5%) relapse. The expected PFS was 78.6 ± 0.3 months (95% CI: 77.8–79.3) in the MMR-proficient group and 70.3 ± 1.7 months (95% CI: 66.8–73.8) in the MMR-deficient group (p = 0.011).

In terms of OS, there were 6 (8.3%) deaths in the MMR-deficient group, whereas 18 (9.9%) deaths occurred in the MMR-proficient group (p = 0.875). The expected OS was 71.6 ± 1.6 months (95% CI: 68.4–74.8) in the MMR-proficient group and 68.2 ± 2.2 months (95% CI: 63.8–72.5) in the MMR-deficient group (p = 0.755).

In the multivariate analysis, MMR deficiency was identified as an independent poor prognostic factor for PFS (HR: 20.654; 95% CI: 1.738–245.475; p = 0.017) (Tab. 2).

Nine (3.6%) patients exhibited p53-abn staining, while the remaining patients had wild-type p53 expression. Among the patients with p53 wild-type expression, four (1.6%) relapses were observed, while in patients with p53-abn staining, one (11.1%) relapse occurred. The expected PFS was 77.7 ± 0.6 months (95% CI: 76.5–78.9) for p53 wild-type patients and 62.8 ± 6.6 (95% CI: 49.8–75.9) for those with p53-abn staining (p = 0.035).

In terms of OS, 19 (7.8%) deaths occurred in the p53 wild-type group, while five (55.6%) deaths occurred in the p53-abn group. The expected OS was 73 ± 1.3 months (95% CI: 70.5–75.5) for p53 wild-type patients and 43.4 ± 8.7 months (95% CI: 26.2–60.6) for those with p53-abn staining (p < 0.001). In the multivariate analysis, p53-abn staining was identified as an independent poor prognostic factor for OS (HR: 7.342; 95% CI: 2.615–20.612; p < 0.001) (Tab. 3).

L1CAM staining was positive in 12 (4.7%) patients. Among the L1CAM-negative group, four (1.7%) relapses occurred, while the L1CAM-positive group had one (8.3%) relapse. The expected PFS was 77.7 ± 0.6 months (95% CI: 76.5–78.9) for L1CAM-negative patients and 62.7 ± 5.8 months (95% CI: 51.2–74.2) for L1CAM-positive patients (p = 0.039).

In the L1CAM-negative group, 22 (9.1%) deaths occurred, while 2 (16.7%) deaths were observed in the L1CAM-positive group. The expected OS was 72.1 ± 1.3 months (95% CI: 69.5–74.8) in the L1CAM-negative group and 58.5 ± 6.7 months (95% CI: 45.3–71.7) in the L1CAM-positive group (p = 0.242).

For Grade 1 patients, the PFS was 78 ± 0.6 months (95% CI: 76.7–79.3), while for Grade 2 patients, the PFS was 73.8 ± 1.2 months (95% CI: 71.4–76.2). However, the difference in PFS between Grade 1 and Grade 2 patients was not statistically significant (p = 0.307).

In terms of OS, Grade 1 patients had an OS of 70.7 ± 1.7 months (95% CI: 67.2–74.2), while Grade 2 patients had an OS of 71.3 ± 1.8 months (95% CI: 67.7–74.9).

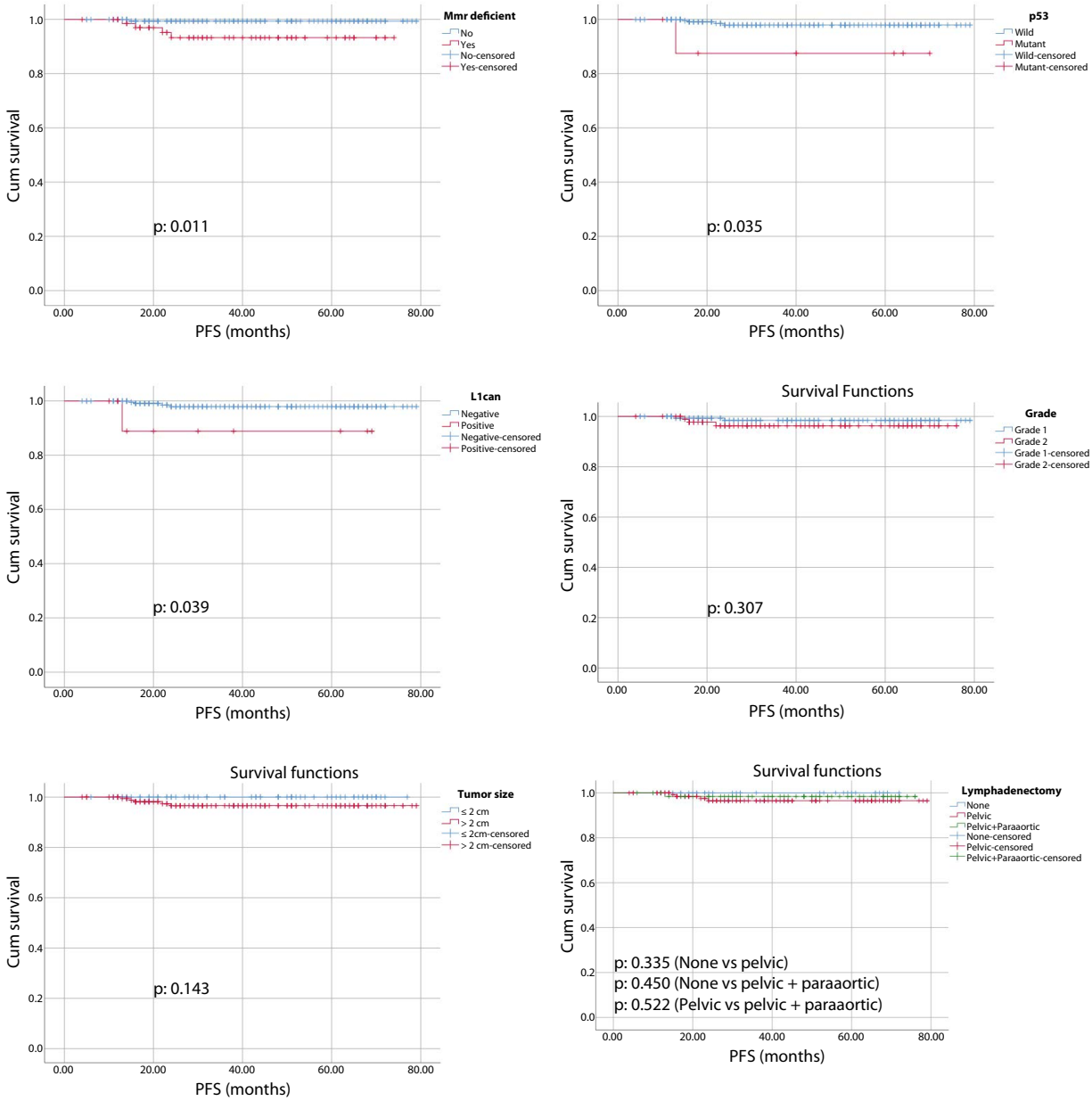



**Figure 1.** Survival analyzes for progression-free survival (PFS)

Similarly, no statistically significant difference in OS was observed between Grade 1 and Grade 2 patients (p = 0.273).

No relapses were observed in patients with a tumor diameter of ≤ 2 cm, whereas five relapses occurred in patients with tumors of >2 cm (p = 0.143). Patients with a tumor diameter of ≤ 2 cm had an OS of 73.4 ± 1.7 months (95% CI: 69.9–76.8), while those with tumors of > 2 cm had an OS of 70.4 ± 1.7 months (95% CI: 66.9–73.9). However, this difference in OS between the two groups was not statistically significant (p = 0.115).

From the perspective of the lymphadenectomy status, no relapses were observed in patients who had never undergone lymphadenectomy. There were four relapses in patients who had undergone only pelvic lymphadenectomy, and one relapse was observed in those who had undergone both pelvic and paraaortic lymphadenectomy. When comparing groups based on lymphadenectomy status — pelvic lymphadenectomy versus no lymphadenectomy (p = 0.335), pelvic and paraaortic lymphadenectomy versus no lymphadenectomy (p = 0.450), and pelvic and paraaortic lymphadenectomy versus pelvic lymphadenectomy (p = 0.522) — no significant statistical differences were found in terms of PFS.

The expected OS in patients who had never undergone lymphadenectomy, those who underwent pelvic lymphadenectomy, and those who underwent pelvic and paraaortic lymphadenectomy were 68.9 ± 2.0 months (95%

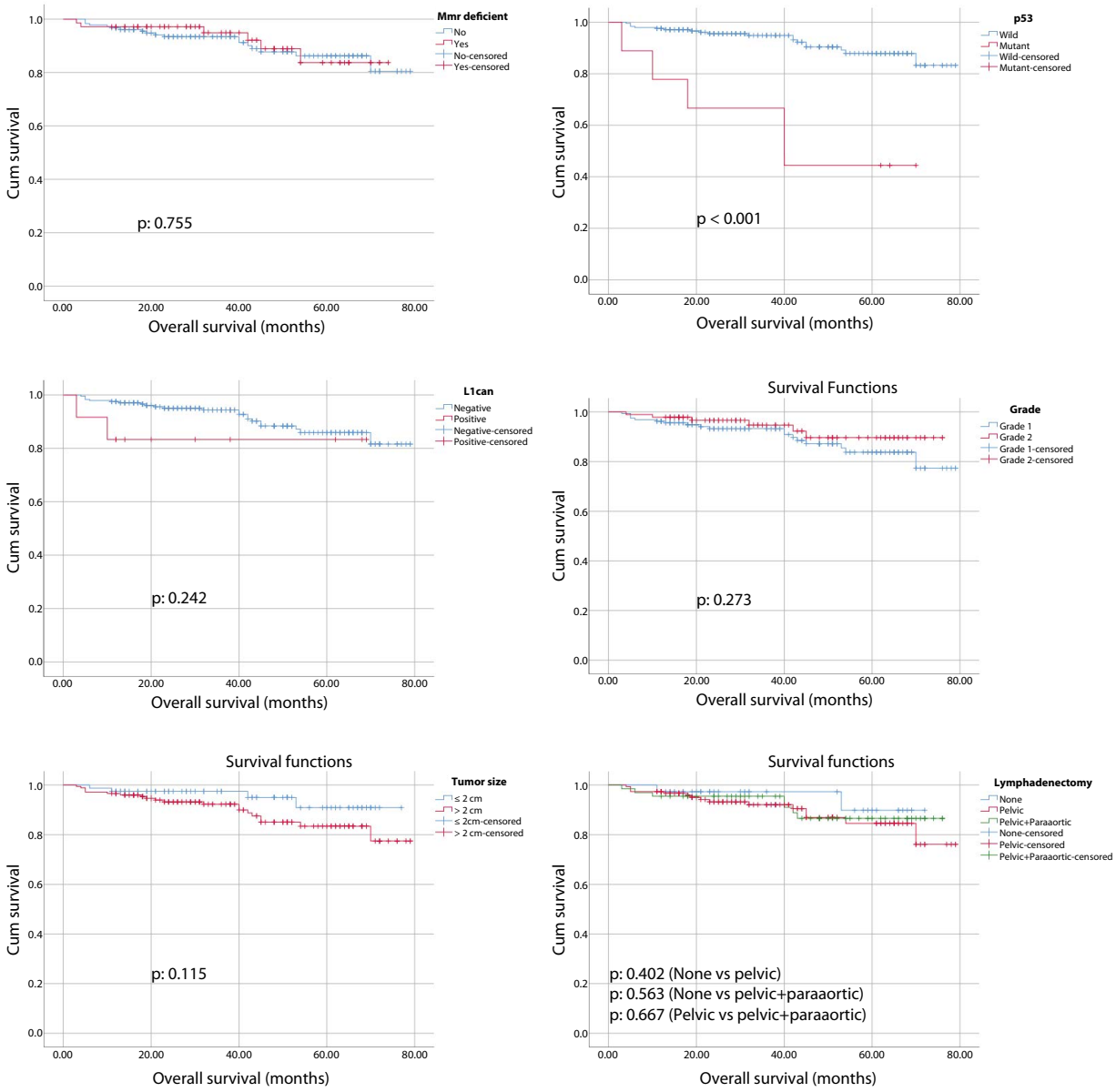



**Figure 2.** Survival analyzes for overall survival (OS)

**Table 2.** Factors associated with progression-free survival (PFS) (n = 253)

| | Univariate analysis | | | Multivariate analysis | | |
|---|---|---|---|---|---|---|
| | HR | 95% CI | p value | HR | 95% CI | p value |
| Gravida | 0.720 | 0.449–1.153 | 0.171 | 0.850 | 0.583–1.240 | 0.400 |
| Hypertension | 0.184 | 0.021–1.643 | 0.130 | 0.207 | 0.019–2.300 | 0.200 |
| Pelvik lymph node count | 0.908 | 0.803–1.027 | 0.125 | 1.654 | 0.275–9.961 | 0.583 |
| Total lymph node count | 0.908 | 0.810–1.018 | 0.099 | 0.496 | 0.080–3.076 | 0.452 |
| Mmr deficient | 9.943 | 1.111–88.971 | **0.040*** | 20.654 | 1.738–245.475 | **0.017*** |
| p53-abn | 7.407 | 0.828–66.289 | 0.073 | 14.357 | 0.960–214.754 | 0.054 |

Variables with a p value below 0.2 were included in the univariate analysis, *: p < 0.05
p53-abn: overexpressed ("all type" phenotype) or totally unexpressed of *TP53* ("null type" phenotype) with Immunohistochemical analysis

**Table 3.** Factors associated with overall survival (OS) (n = 253)

| | Univariate analysis | | | Multivariate analysis | | |
|---|---|---|---|---|---|---|
| | HR | 95% CI | p value | HR | 95% CI | p value |
| Age ≥ 60 | 3.114 | 1.328–7.304 | **0.009*** | 1.813 | 0.656–5.005 | 0.251 |
| Gravida | 1.124 | 1.012–1.249 | **0.029*** | 0.974 | 0.707–1.348 | 0.875 |
| Parity | 1.188 | 1.050–1.346 | **0.006*** | 1.111 | 0.730–1.691 | 0.623 |
| Hypertension | 5.580 | 1.661–18.750 | **0.005*** | 2.798 | 0.744–10.520 | 0.128 |
| Diabetes | 2.979 | 1.318–6.736 | **0.009*** | 1.908 | 0.794–4.585 | 0.148 |
| Tumor size ≥ 2 cm | 2.311 | 0.790–6.763 | 0.126 | 1.396 | 0.455–4.285 | 0.559 |
| p53-abn | 6.852 | 2.549–18.418 | **< 0.001*** | 7.342 | 2.615–20.612 | **< 0.001*** |

Variables with a p value below 0.2 were included in the univariate analysis, *: $p < 0.05$
p53-abn: overexpressed ("all type" phenotype) or totally unexpressed of *TP53* ("null type" phenotype) with Immunohistochemical analysis

CI: 64.8–73), 70.8 ± 1.9 months (95% CI: 67–74.7) (p = 0.402), and 69.7 ± 2.2 months (95% CI: 65.3–74.1) (p = 0.563), respectively. Similarly, no significant differences in OS were observed between patients who underwent pelvic lymphadenectomy and those who underwent pelvic and paraaortic lymphadenectomy (p = 0.667).

## DISCUSSION

Molecular classification and potential cost-effective alternatives, such as ProMisE, have introduced repeatable innovations in the prognostication of endometrial cancer compared with classical histopathological classification. However, relapses, albeit rare, may still occur in low-risk groups. Although IHC studies involving *TP53*, MMR, and L1CAM in early-stage endometrial cancers have been documented in the literature, these studies often include heterogeneous patient groups, such as those with non-endometrioid histologies, Grade 3 tumors, lymphovascular invasion, and those who received adjuvant therapy. Adjuvant therapy may obscure whether MMR deficiency is prognostic or predictive of outcomes [18].

In a population-based study involving 475 patients with endometrioid endometrial cancers, those with MMR deficiency exhibited worse PFS than those with MMR-proficient tumors (median of 24 vs 27 months, respectively) [17]. Multivariate analysis further revealed that MMR deficiency was associated with a higher risk of relapses. However, no significant difference in OS was observed between the MMR groups. A key aspect of this study was the inclusion of 42 (8.8%) patients with lymphovascular invasion and 30 (6.3%) patients who received adjuvant therapy. In the subgroup of patients without lymphovascular invasion and who did not receive adjuvant therapy, a similar relationship between MMR deficiency and recurrence could not be demonstrated (p = 0.16). Additionally, because the lymphadenectomy rate in this study was only 9%, some tumors may have been under-staged.

Stasenko et al. [19] conducted a refined study on ultra-low-risk patients and found no statistically significant difference in recurrence rates between those with and without MMR deficiency. Conversely, a case-control study involving 311 patients with Grade 1 endometrioid endometrial cancer revealed a significantly higher prevalence of MMR deficiency in the relapse group than in the control group [20].

The present study identified MMR deficiency as a worse prognostic factor for PFS and an independent poor prognostic factor for PFS in the multivariate analysis. Moreover, in an earlier study focusing on the low-risk group, we found that MMR deficiency was associated with ovarian metastasis and synchronous gonadal involvement [21].

In studies that included patients from all endometrial cancer risk groups, p53-abn was significantly associated with higher risk classifications and poorer outcomes [22, 23]. Two studies specifically evaluating p53-abn in low-risk endometrial cancer reported no significant differences in PFS or OS [19, 20]. By contrast, the current study found a clear association between p53-abn and worse PFS and OS, identifying it as an independent poor prognostic factor for OS.

L1CAM expression has been shown to be an independent predictor of both PFS and OS in patients with endometrial cancer. Zeimet et al. [12] conducted a study of 657 patients with low-grade endometrial cancer, finding L1CAM positivity in 17% of cases. L1CAM was strongly associated with an increased risk of death (HR: 15.00) and recurrence (HR: 16.33). Additionally, L1CAM staining in tissues from the PORTEC-1 and PORTEC-2 study cohorts confirmed a higher incidence of distant and pelvic nodal recurrence in L1CAM-positive patients [10]. In patients with L1CAM positivity, the risk of distant recurrence increased by a factor of 3.5, while OS decreased by a factor of 2.1. However, in this

study, only 26.9% of the patients were classified as low-risk, and 72.6% of the patients received adjuvant therapy, which may have influenced the outcomes.

In a separate study analyzing TCGA data on uterine cancer, L1CAM was identified as an independent prognostic factor for OS [11]. However, the authors could not confirm a worse effect on survival in Stage I subpopulation. In a large study involving 1,199 patients with endometrial cancer, L1CAM expression was linked to poor outcomes even in stage I cancers [13]. Kommos et al. [24] reported significantly worse PFS (71.8% vs 100%, $p < 0.0001$) and OS (63.8% vs 95.3%, $p < 0.0001$) in L1CAM-positive patients. The study identified L1CAM as an independent prognostic factor in low-risk endometrial cancer and recommended revising the ESGO risk classification to consider only L1CAM-negative patients as low-risk.

A common criticism of these studies is the inclusion of heterogeneous patient groups and the use of adjuvant therapy, which may affect the findings. More specifically, a meta-analysis evaluating patients with stage I endometrial cancer and L1CAM positivity reported worse PFS (HR: 4.11) and OS: (HR 3.62) in L1CAM-positive patients [25]. Notably, only one of the five studies included in this review focused exclusively on pure endometrioid endometrial cancer patients, and even that study included patients with Grade 3 tumors.

In terms of survival, we found no significant differences related to grade, tumor diameter, or lymph node status. These findings are consistent with similar studies in the literature, which also reported no significant association between tumor diameter and survival in early-stage endometrial cancers [26, 27].

The strengths of this study lie in its exclusive focus on low-risk patients, the exclusion of adjuvant therapy that could influence prognosis, and the simultaneous evaluation of all MMR subgroups, *TP53*, and L1CAM IHC prognostic markers. This approach ensures a more precise assessment of the independent prognostic value of these markers in a homogeneous patient population.

The lack of *POLE* analysis can be considered a limitation of the study. However, because PFS and OS are generally good in patients with *POLE* mutation, our study aimed to decipher the relationship between relapse and death using only IHC markers. Other limitations include its retrospective design and the relatively small patient cohort because it was a single-center study.

In conclusion, p53-abn staining was associated with poor PFS and OS in patients with low-risk endometrial cancer, while MMR deficiency and L1CAM positivity were found to be associated solely with poorer PFS.

## Article information and declarations

### *Data availability statement*

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

### *Ethics statement*

Ethics committee of Kocaeli University (Approval number: GOKAEK-2022/23.22).

### *Author contributions*

Ş.G. and B.Y.B. performed the protocol design, and implementation and drafted the manuscript. M.E. and E.B.T. contributed to data analysis, critical revision of the manuscript and final approval.

### *Funding*

This study was supported by the Kocaeli University Scientific Research Projects Coordination Unit (Project Num: TSA-2022-2893).

### *Acknowledgments*

None.

### *Conflicts of interest*

The authors have no conflicts of interest to disclose.

### *Supplementary material*

None.

## REFERENCES

1. Concin N, Matias-Guiu X, Vergote I, et al. ESGO/ESTRO/ESP guidelines for the management of patients with endometrial carcinoma. Int J Gynecol Cancer. 2021; 31(1): 12–39, doi: 10.1136/ijgc-2020-002230, indexed in Pubmed: 33397713.
2. Xu Y, Burmeister C, Hanna RK, et al. Predictors of survival after recurrence in women with early-stage endometrial carcinoma. Int J Gynecol Cancer. 2016; 26(6): 1137–1142, doi: 10.1097/IGC.0000000000000733, indexed in Pubmed: 27206283.
3. Bednarikova M, Vinklerova P, Gottwaldova J, et al. The Clinical Significance of DJ1 and L1CAM Serum Level Monitoring in Patients with Endometrial Cancer. J Clin Med. 2021; 10(12): 2640, doi: 10.3390/jcm10122640, indexed in Pubmed: 34203959.
4. Topfedaisi Ozkan N, Meydanlı MM, Sarı ME, et al. Factors associated with survival after relapse in patients with low-risk endometrial cancer treated with surgery alone. J Gynecol Oncol. 2017; 28(5): e65, doi: 10.3802/jgo.2017.28.e65, indexed in Pubmed: 28657226.
5. Francis SR, Ager BJ, Do OA, et al. Recurrent early stage endometrial cancer: Patterns of recurrence and results of salvage therapy. Gynecol Oncol. 2019; 154(1): 38–44, doi: 10.1016/j.ygyno.2019.04.676, indexed in Pubmed: 31029507.
6. Kandoth C, Schultz N, Cherniack AD, et al. Cancer Genome Atlas Research Network. Integrated genomic characterization of endometrial carcinoma. Nature. 2013; 497(7447): 67–73, doi: 10.1038/nature12113, indexed in Pubmed: 23636398.
7. Talhouk A, McConechy MK, Leung S, et al. A clinically applicable molecular-based classification for endometrial cancers. Br J Cancer. 2015; 113(2): 299–310, doi: 10.1038/bjc.2015.190, indexed in Pubmed: 26172027.
8. Perri F, Pisconti S, Della Vittoria Scarpati G. P53 mutations and cancer: a tight linkage. Ann Transl Med. 2016; 4(24): 522, doi: 10.21037/atm.2016.12.40, indexed in Pubmed: 28149884.

9. Li GM. Mechanisms and functions of DNA mismatch repair. Cell Res. 2008; 18(1): 85–98, doi: 10.1038/cr.2007.115, indexed in Pubmed: 18157157.
10. Bosse T, Nout RA, Stelloo E, et al. L1 cell adhesion molecule is a strong predictor for distant recurrence and overall survival in early stage endometrial cancer: pooled PORTEC trial results. Eur J Cancer. 2014; 50(15): 2602–2610, doi: 10.1016/j.ejca.2014.07.014, indexed in Pubmed: 25126672.
11. Dellinger TH, Smith DD, Ouyang C, et al. L1CAM is an independent predictor of poor survival in endometrial cancer — An analysis of The Cancer Genome Atlas (TCGA). Gynecol Oncol. 2016; 141(2): 336–340, doi: 10.1016/j.ygyno.2016.02.003, indexed in Pubmed: 26861585.
12. Zeimet AG, Reimer D, Huszar M, et al. L1CAM in early-stage type I endometrial cancer: results of a large multicenter evaluation. J Natl Cancer Inst. 2013; 105(15): 1142–1150, doi: 10.1093/jnci/djt144, indexed in Pubmed: 23781004.
13. van der Putten LJM, Visser NCM, van de Vijver K, et al. L1CAM expression in endometrial carcinomas: an ENITEC collaboration study. Br J Cancer. 2016; 115(6): 716–724, doi: 10.1038/bjc.2016.235, indexed in Pubmed: 27505134.
14. Reijnen C, Gogou E, Visser NCM, et al. ENITEC Consortium. Preoperative risk stratification in endometrial cancer (ENDORISK) by a Bayesian network model: A development and validation study. PLoS Med. 2020; 17(5): e1003111, doi: 10.1371/journal.pmed.1003111, indexed in Pubmed: 32413043.
15. León-Castillo A, de Boer SM, Powell ME, et al. TransPORTEC consortium. Molecular Classification of the PORTEC-3 Trial for High-Risk Endometrial Cancer: Impact on Prognosis and Benefit From Adjuvant Therapy. J Clin Oncol. 2020; 38(29): 3388–3397, doi: 10.1200/JCO.20.00549, indexed in Pubmed: 32749941.
16. Le D, Uram J, Wang H, et al. PD-1 blockade in tumors with mismatch-repair deficiency. N Engl J Med. 2015; 372(26): 2509–2520, doi: 10.1056/nejmoa1500596, indexed in Pubmed: 26028255.
17. Kim SR, Pina A, Albert A, et al. Mismatch repair deficiency and prognostic significance in patients with low-risk endometrioid endometrial cancers. Int J Gynecol Cancer. 2020; 30(6): 783–788, doi: 10.1136/ijgc-2019-000910, indexed in Pubmed: 32354793.
18. McMeekin DS, Tritchler DL, Cohn DE, et al. Clinicopathologic significance of mismatch repair defects in endometrial cancer: an NRG oncology/gynecologic oncology group study. J Clin Oncol. 2016; 34(25): 3062–3068, doi: 10.1200/JCO.2016.67.8722, indexed in Pubmed: 27325856.
19. Stasenko M, Feit N, Lee SSK, et al. Clinical patterns and genomic profiling of recurrent ‚ultra-low risk' endometrial cancer. Int J Gynecol Cancer. 2020; 30(6): 717–723, doi: 10.1136/ijgc-2020-001241, indexed in Pubmed: 32376737.
20. Moroney MR, Davies KD, Wilberger AC, et al. Molecular markers in recurrent stage I, grade 1 endometrioid endometrial cancers. Gynecol Oncol. 2019; 153(3): 517–520, doi: 10.1016/j.ygyno.2019.03.100, indexed in Pubmed: 30910249.
21. Gezer Ş, Bayrak BY. Metastatic and synchronous ovarian involvement in low-risk endometrial cancer; clinicopathological analysis with detection of DNA mismatch repair deficiency. Ginekol Pol. 2021; 92(9): 599–606, doi: 10.5603/GP.a2021.0150, indexed in Pubmed: 34541641.
22. Vrede SW, van Weelden WJ, Visser NCM, et al. ENITEC Consortium. Immunohistochemical biomarkers are prognostic relevant in addition to the ESMO-ESGO-ESTRO risk classification in endometrial cancer. Gynecol Oncol. 2021; 161(3): 787–794, doi: 10.1016/j.ygyno.2021.03.031, indexed in Pubmed: 33858677.
23. Talhouk A, McConechy MK, Leung S, et al. Confirmation of ProMisE: A simple, genomics-based clinical classifier for endometrial cancer. Cancer. 2017; 123(5): 802–813, doi: 10.1002/cncr.30496, indexed in Pubmed: 28061006.
24. Kommoss F, Kommoss F, Grevenkamp F, et al. L1CAM: amending the „low-risk" category in endometrial carcinoma. J Cancer Res Clin Oncol. 2017; 143(2): 255–262, doi: 10.1007/s00432-016-2276-3, indexed in Pubmed: 27695947.
25. Giannini A, D'Oria O, Corrado G, et al. The role of L1CAM as predictor of poor prognosis in stage I endometrial cancer: a systematic review and meta-analysis. Arch Gynecol Obstet. 2024; 309(3): 789–799, doi: 10.1007/s00404-023-07149-8, indexed in Pubmed: 37454351.
26. Ureyen I, Karalok A, Turkmen O, et al. Factors predicting recurrence in patients with stage IA endometrioid endometrial cancer: what is the importance of LVSI? Arch Gynecol Obstet. 2020; 301(3): 737–744, doi: 10.1007/s00404-019-05418-z, indexed in Pubmed: 31883046.
27. Ayhan A, Şahin H, Sari ME, et al. Prognostic significance of lymphovascular space invasion in low-risk endometrial cancer. Int J Gynecol Cancer. 2019; 29(3): 505–512, doi: 10.1136/ijgc-2018-000069, indexed in Pubmed: 30665899.