ORIGINAL CONTRIBUTION

EXPERIMENTAL ONCOLOGY

https://doi.org/10.15407/exp-oncology.2026.02.133

P. Gordiichuk [1, 2, *], M. Doronina [1], O. Ponomarova [1, 2], M. Gordiichuk [1, 2], D. Shapochka [3], D. Melnyk [1], A. Tereshchenko [4]

[1] Shupyk National University of Healthcare of Ukraine, Kyiv, Ukraine
[2] Communal nonprofit enterprise «Kyiv City Clinical Oncology Center», Kyiv, Ukraine
[3] «Feofaniya» Clinical Hospital, Kyiv, Ukraine
[4] ESC Institute of Biology and Medicine, Kyiv, Ukraine

* Correspondence: E-mail: p_gordijchuk@ukr.net

STUDY OF PREDICTIVE VALUE OF RAD51 EXPRESSION FOR THE EFFECTIVENESS OF PLATINUM-BASED THERAPY OF HIGH-GRADE SEROUS OVARIAN CARCINOMA

Background. The search for novel markers of predictive significance is an important issue in the management of ovarian cancer (OC) patients. Recently, the expression of the RAD51 protein, involved in homologous DNA repair has been shown to be predictive of platinum chemotherapy response in OC patients. **Aim.** To establish the relationship between the level of RAD51 protein expression in patients with high-grade serous ovarian carcinoma (SOC) after neoadjuvant platinum chemotherapy and the frequency of recurrence, disease progression, and relapse-free survival. **Materials and Methods.** RAD51 status was assessed in the archival histological material of 30 patients with SOC of stage III who received perioperative platinum-based chemotherapy. RAD51 expression was analyzed using a double-staining technique in samples containing at least 100 geminin-positive tumor cells. RAD51 status was considered positive if > 10% of geminin-positive nuclei were positive for RAD51. **Results.** In 16 of 30 cases, the tumor had a RAD51–positive status (homologous repair proficient, HRP), and in 14 — RAD51-negative (homologous repair deficient, HRD). We found that the risk of disease progression in patients with RAD51 HRP status was 3.42 times higher compared to patients with RAD51 HRD status (HR = 3.42; 95% CI 1.38—8.46; $p = 0.008$). In SOC patients with RAD51 HRP status, a significantly higher ($p < 0.001$) rate of disease progression over 3 to 12 months of postoperative observation compared to patients with RAD51 HRD status was detected. **Conclusions.** RAD51 expression is predictive for the response of high-grade SOC to platinum therapy.

Keywords: ovarian cancer, RAD51, platinum-containing chemotherapy, potential predictor of platinum sensitivity, homologous repair system.

Ovarian cancer (OC) is associated with high morbidity and mortality due to latent onset, lack of early diagnostic biomarkers, high invasiveness, and a tendency to develop distant metastases [1—4]. The average five-year survival rate is 40%. Approximately 85%—90% of all OCs are of epithelial ori-

Citation: Gordiichuk P, Doronina M, Ponomarova O, Gordiichuk M, Shapochka D, Melnyk D, Tereshchenko A. Study of predictive value of RAD51 expression for the effectiveness of platinum-based therapy of high-grade serous ovarian carcinoma. *Exp Oncol.* 2026; 48(2): 133-138. https://doi.org/10.15407/exp-oncology.2026.02.133.

gin, and approximately 70% of them are poorly differentiated serous adenocarcinoma [5—7]. The standard of treatment consists of surgical cytoreduction and combination therapy with platinum and taxanes. Most women with late-stage epithelial OC are diagnosed with a relapse of the disease, which requires additional treatment [2].

Platinum-containing chemotherapy is the basic treatment of high-grade serous ovarian carcinoma (SOC) at different stages. Platinum compounds induce double-stranded DNA breaks in cells during their replication, which leads to the death of cancer cells.

According to the literature, the most advanced way to repair double-stranded DNA breaks is homologous recombination repair (HRR). It is known that various cancers, including OC, are characterized by a homologous repair deficiency (HRD), leading to incorrect repair of DNA breaks and ultimately to increased tumor sensitivity to platinum compounds and PARP inhibitors [8, 9]. HRD is often caused by loss-of-function mutations in the *BRCA1*, *BRCA2*, *RAD51C*, *RAD51D*, or *PALB2* genes, or by other factors that are still unknown [10].

Approximately 50% of high-grade SOC cases are associated with abnormalities in HRR, including mutations in the *BRCA* genes [11—13].

Genomic testing is used to detect HRD, including analysis of *BRCA* status and assessment of genomic scars, which reflect genomic instability. In turn, genomic instability is the result of HRD [8].

A genomic scar is detected by calculating the HRD index (HRDi) or loss of heterozygosity (LOH) [14]. However, these assays are of high cost and require long processing time. Most importantly, they do not reflect the current functional HRD status of tumors [15], just past events. Therefore, in recent years, functional HRD assays have been developed that target one of its biomarkers, namely, the expression of the RAD51 protein [16—18] predictive of platinum chemotherapy response in OC patients [19].

To date, the clinical validity of RAD51 as a biomarker of HRD has been confirmed [20—23]. The RAD51 test is based on the (in)ability of the RAD51 protein to accumulate at sites of DNA double-strand breaks [16, 20, 24, 25]. It can be performed on formalin-fixed paraffin-embedded (FFPE) tumor tissue samples and includes RAD51/geminin co-immunofluorescence staining. The RAD51 test is of high sensitivity for detecting pathogenic BRCA1/2 variants [26] and high predictive power for response to platinum [27, 28] and PARP inhibitor therapy [25, 29].

The aim of the study was to establish the relationship between the level of RAD51 protein expression in patients with high-grade SOC after neoadjuvant chemotherapy and the rates of relapse, disease progression, and relapse-free survival.

Table 1. **Clinical characteristics of patients stratified by FIGO stage and treatment response to NAC according to RECIST 1.1 criteria (n = 30)**

FIGO stage, n	Response by RECIST 1.1		Average age ± σ, years	Median body mass index ± σ, kg/m^2
	SD	PR		
IIIA, 4	3	1	59.4 ± 6.2	26.9 ± 3.3
IIIB, 2	1	1		
IIIC, 24	11	13		

Notes: NAC — Neoadjuvant chemotherapy; FIGO — International Federation of Gynecology and Obstetrics; RECIST 1.1 — Response Evaluation Criteria for Solid Tumors; PR — partial response; SD — stable disease.

Materials and Methods

The study was performed on archival histological material (paraffin blocks and slides) from 30 patients with high-grade SOC of stage III. The study was reviewed and approved by the Ethics and Academic Integrity Commission of the P.L. Shupyk National University of Health Care of Ukraine (protocol No. 1/2024-4 dated 01.02.2024).

All the patients received neoadjuvant chemotherapy (NAC) and adjuvant chemotherapy (ACT): paclitaxel 175 mg/m^2 followed by cisplatin 75—80 mg/m^2 (or carboplatin AUC 5- 6) intravenously on day 1 every 3 weeks, with a total number of cycles of 6 to 8. Information from archival inpatient and outpatient medical records indicated that all patients had a positive radiological response to NAC (according to RECIST 1.1) (Table 1).

The study was performed on FFPE samples of tumor tissue. 4-μm thick sections were routinely prepared from paraffin blocks. Antigen retrieval and immunostaining were performed using EnVision (Dako, Denmark), primary RAD51 antibodies (monoclonal anti-RAD51, clone 4C11-B4, Sigma-Aldrich, USA), secondary antibodies to RAD51 (an-

ti-mouse IgG(H+L) F(ab`)2 CF568, Sigma-Aldrich, USA), primary geminin antibodies (polyclonal anti-GMNN, HPA049977, Sigma-Aldrich, USA), and secondary antibodies specific for geminin antibodies (anti-rabbit IgG(H+L) F(ab`)2 CF488A, Sigma-Aldrich, USA). The double-staining protocol, allowing for optimal visualization of marker co-localization in the nuclei of tumor cells, was applied. The stained slides were examined using a Leica DM6 fluorescence microscope with LUCIA software at 60x magnification. The cell nuclei exhibited green and blue staining. Green nuclei were classified as geminin-positive, whereas blue nuclei (DAPI-stained) were classified as geminin-negative. Firstly, nuclei positive for geminin were counted, then RAD51 expression was assessed in samples with at least 100 tumor cells positive for geminin, as described in [16, 28]. On average, 5 fields were analyzed per sample. The analyzed tissue area was standardized as a tumor region encompassing 100 geminin-positive nuclei. In such samples, RAD51 expression (red dots) in nuclei positive for geminin was assessed. RAD51 status was considered positive if > 10% of geminin-positive nuclei were positive for RAD51.

Statistical analysis was performed using IBM SPSS Statistics, version 29.0 (SPSS Inc., Chicago, IL, USA). The continuous variables were presented as M ± σ (mean ± standard deviation). The differences between the parametric variables were assessed using the two-tailed Student's *t*-test. Owing to the small sample size, Fisher's exact test was used to compare categorical variables and progression rates. Survival was estimated using the Kaplan–Meier method, and differences between survival curves were evaluated with the log-rank test. The risk of recurrence was calculated using the Cox proportional hazards model and expressed as hazard ratios (HRs) with 95% confidence intervals (CIs). A p-value < 0.05 was considered statistically significant.

Results

The tumor in 16 of 30 patients had RAD51–positive HRP status (53.33%), and in 14 — negative HRD (46.66%) (Figs. 1, 2).

The effectiveness of ACT, depending on the RAD51 expression, was assessed by the patient's overall performance status (ECOG), time to disease progression, and progression-free survival (PFS) (Table 2).

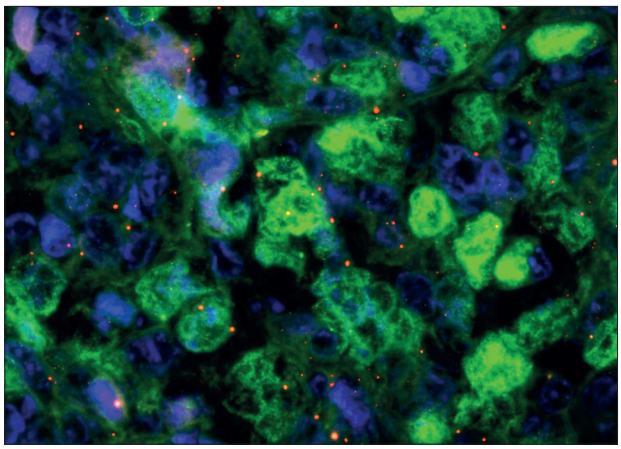

Fig. 1. RAD51-positive nuclei in cells of high-grade SOC

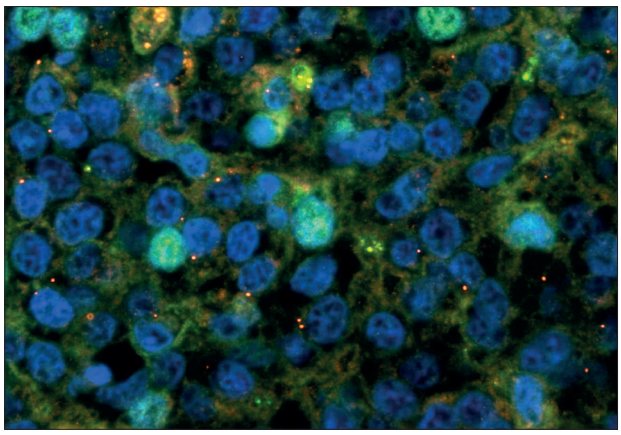

Fig. 2. RAD51-negative nuclei in cells of high-grade SOC

Table 2. **Clinicopathological characteristics of patients (n = 30)**

Indicator	RAD51 positive (proficient > 10%; n = 16)	RAD51 negative (deficient < 10%; n = 14)
IIIA	0	4
IIIB	1	1
IIIC	15	9
ECOG (grade)		
0	4	4
1	9	9
2	3	1
PFS (months)		
≤ 3	6	0
≤ 6	4	2
≤ 12	5	3
< 18	0	1
24	1	1
36	0	1

Notes: ECOG — Eastern Cooperative Oncology Group performance status; PFS — progression-free survival.

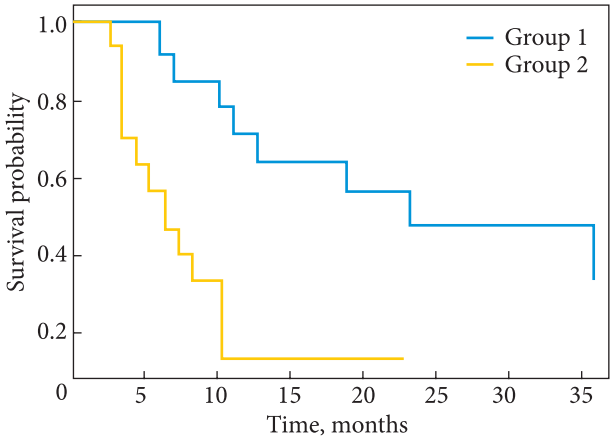

Fig. 3. Kaplan–Meier curves of PFS: Group 1 — RAD51 HRP (n = 16); Group 2 — RAD51 HRD (n = 14)

Among patients with RAD51-positive status, the disease progressed up to 12 months in 93.75% of cases compared to 35.7% of cases with PFS < 12 months in patients with RAD51-negative status (p = 0.0014, Fisher's exact test). Moreover, among patients with RAD51-negative status, in 42.85% of cases, no progression of the disease was evident during the observation period (36 months).

Due to a small sample size, the Cox proportional hazards model was used to assess the risk of disease progression. The proportional hazards assumption was formally tested using the analysis of scaled Schoenfeld residuals (Grambsch–Therneau test, p = 0.14), confirming the adequacy of the model. It was found that the risk of progression in patients with RAD51 HRP status was 3.42 times higher compared to patients with RAD51 HRD status (HR = 3.42; 95% CI 1.38—8.46; p = 0.008).

To assess the PFS, Kaplan–Meier curves were plotted for both groups (Fig. 3). A log-rank test (χ^2 = 12.62, p = 0.00038) confirmed a significant difference between the survival curves. Patients with RAD51 HRD status had a better prognosis in terms of RFS after receiving therapy.

Discussion

Our results indicated that in patients with high-grade SOC of stage III with RAD51 HRP status, despite chemotherapy, the risk of progression was significantly higher compared to patients with RAD51 HRD status.

We found RAD51 expression in 46.7% of our SOC cases, which is slightly higher than the 30%—40% reported in several other studies [16, 28, 30]. This discrepancy likely stems from varying cut-off thresholds across the literature. Following studies that established a >10% threshold as most accurate [16, 28, 29], we defined positive RAD51 status (HRP) as >10% geminin-positive nuclei and negative (HRD) as <10%. Nevertheless, different reference values have also been used in other studies (e.g., >25.89% or >35%) [16, 31]. Consequently, establishing a standardized clinical cut-off for defining RAD51 expression requires further accumulation of clinical data.

Our study results have demonstrated that the HRD functional test can predict a longer period without tumor recurrence (HR = 0.22), which confirms the significant predictive value of RAD51 for the effectiveness of the treatment with platinum drugs. Nevertheless, our study does not fully support the suggestions [28, 31] that RAD51 foci can predict platinum sensitivity with 100% specificity and 100% positive predictive value.

The results show a high probability of platinum sensitivity in SOC with low RAD51 expression levels. However, it should be noted that platinum sensitivity is multifactorial and also depends on drug uptake, DNA damage signaling, nucleotide excision repair, cell cycle checkpoints, cell death pathways, etc. [32].

In this study, we did not find any association between RAD51 expression levels and patient age, BMI, and disease stage, nor have we found such data in other similar studies.

Because our study relied on archival material, we could not assess RAD51 dynamics. Nevertheless, IGH histological analysis offers an accessible, inexpensive, and highly predictive method for evaluating PARP inhibitor efficacy [16, 33, 34], advancing future ovarian cancer research.

Acknowledgments

All authors express their sincere gratitude to the Armed Forces of Ukraine, who ensure our safety and make it possible for us to work.

Conflicts of interest/Financial disclosures

The authors declare no conflict of interest regarding the research, authorship, or publication of this article. This study did not receive any financial support.

REFERENCES

1. Bray F, Laversanne M, Sung H, et al. Global cancer statistics 2022: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. *CA Cancer J Clin.* 2024;74(3):229-263. https://doi.org/10.3322/caac.21834
2. Lheureux S, Gourley C, Vergote I, et al. Epithelial ovarian cancer. *Lancet.* 2019;393(10177):1240-1253. https://doi.org/10.1016/S0140-6736(18)32552-2
3. Eisenhauer EA. Real-world evidence in the treatment of ovarian cancer. *Ann Oncol.* 2017;28(suppl 8):viii61-viii65. https://doi.org/10.1093/annonc/mdx443
4. Wu YB, Li SY, Liu JY, et al. Long non-coding RNA NRSN2-AS1 promotes ovarian cancer progression through targeting PTK2/β-catenin pathway. *Cell Death Dis.* 2023;14(10):696. https://doi.org/10.1038/s41419-023-06214-z
5. Prat J; FIGO Committee on Gynecologic Oncology. FIGO's staging classification for cancer of the ovary, fallopian tube, and peritoneum: Abridged republication. *J Gynecol Oncol.* 2015;26(2):87-89. https://doi.org/10.3802/jgo.2015.26.2.87
6. American Cancer Society: Key statistics for ovarian cancer, 2019. https://www.cancer.org/cancer/ovarian-cancer/about/key-statistics.html
7. Kim J, Park EY, Kim O, et al. Cell origins of high-grade serous ovarian cancer. *Cancers (Basel).* 2018;10(11):433. https://doi.org/10.3390/cancers10110433
8. Pennington KP, Walsh T, Harrell MI, et al. Germline and somatic mutations in homologous recombination genes predict platinum response and survival in ovarian, fallopian tube, and peritoneal carcinomas. *Clin Cancer Res.* 2014;20(3):764-775. https://doi.org/10.1158/1078-0432
9. Norquist BM, Brady MF, Harrell MI, et al. Mutations in homologous recombination genes and outcomes in ovarian carcinoma patients in GOG 218: An NRG Oncology/Gynecologic Oncology Group study. *Clin Cancer Res.* 2018;24(4):777-783. https://doi.org/10.1158/1078-0432.CCR-17-1327
10. Miller RE, Leary A, Scott CL, et al. ESMO recommendations on predictive biomarker testing for homologous recombination deficiency and PARP inhibitor benefit in ovarian cancer. *Ann Oncol.* 2020;31(12):1606-1622. https://doi.org/10.1016/j.annonc.2020.08.2102
11. Konstantinopoulos PA, Ceccaldi R, Shapiro GI, et al. Homologous recombination deficiency: Exploiting the fundamental vulnerability of ovarian cancer. *Cancer Discov.* 2015;5(11):1137-1154. https://doi.org/10.1158/2159-8290.CD-15-0714
12. Rebbeck TR, Mitra N, Wan F, et al. Association of type and location of BRCA1 and BRCA2 mutations with risk of breast and ovarian cancer. *JAMA.* 2015;313(13):1347-1361. https://doi.org/10.1001/jama.2014.5985
13. Frey MK, Pothuri, B. Homologous recombination deficiency (HRD) testing in ovarian cancer clinical practice: A review of the literature. *Gynaecol Oncol Res Pract.* 2017;4:4. https://doi.org/10.1186/s40661-017-0039-8
14. Swisher EM, Lin KK, Oza AM, et al. Rucaparib in relapsed, platinum-sensitive high-grade ovarian carcinoma (ARIEL2 Part 1): An international, multicentre, open-label, phase 2 trial. *Lancet Oncol.* 2017;18(1):75-87. https://doi.org/10.1016/S1470-2045(16)30559-9
15. Fuh K, Mullen M, Blachut B, et al. Homologous recombination deficiency real-time clinical assays, ready or not? *Gynecol Oncol.* 2020;159(3):877-886. https://doi.org/10.1016/j.ygyno.2020.08.035
16. Castroviejo-Bermejo M, Cruz C, Llop-Guevara A, et al. A RAD51 assay feasible in routine tumor samples calls PARP inhibitor response beyond BRCA mutation. *EMBO Mol Med.* 2018;10(12):e9172. https://doi.org/10.15252/emmm.201809172
17. Feng Y, Wang D, Xiong L, et al. Predictive value of RAD51 on the survival and drug responsiveness of ovarian cancer. *Cancer Cell Int.* 2021;21(1):249. https://doi.org/10.1186/s12935-021-01953-5
18. Hill SJ, Decker B, Roberts EA, et al. Prediction of DNA repair inhibitor response in short-term patient-derived ovarian cancer organoids. *Cancer Discov.* 2018;8(11):1404-1421. https://doi.org/10.1158/2159-8290.CD-18-0474
19. Compadre AJ, van Biljon LN, Valentine MC, et al. RAD51 foci as a biomarker predictive of platinum chemotherapy response in ovarian cancer. *Clin Cancer Res.* 2023;29(13):2466-2479. https://doi.org/10.1158/1078-0432.CCR-22-3335
20. Cruz C, Castroviejo-Bermejo M, Gutiérrez-Enríquez S, et al. RAD51 foci as a functional biomarker of homologous recombination repair and PARP inhibitor resistance in germline BRCA-mutated breast cancer. *Ann Oncol.* 2018;29(5):1203-1210. https://doi.org/10.1093/annonc/mdy099
21. van Wijk LM, Kramer CJH, Vermeulen S, et al. The RAD51-FFPE test; Calibration of a functional homologous recombination deficiency test on diagnostic endometrial and ovarian tumor blocks. *Cancers (Basel).* 2021;13(12):2994. https://doi.org/10.3390/cancers13122994
22. van Wijk LM, Nilas AB, Vrieling H, et al. RAD51 as a functional biomarker for homologous recombination deficiency in cancer: A promising addition to the HRD toolbox? *Expert Rev Mol Diagn.* 2022;22(2):185-199. https://doi.org/10.1080/14737159.2022.2020102
23. Byrum AK, Vindigni A, Mosammaparast N. Defining and modulating 'BRCAness'. *Trends Cell Biol.* 2019;29(9):740-751. https://doi.org/10.1016/j.tcb.2019.06.005
24. Llop-Guevara A, Loibl S, Villacampa G, et al. Association of RAD51 with homologous recombination deficiency (HRD) and clinical outcomes in untreated triple-negative breast cancer (TNBC): analysis of the GeparSixto randomized clinical trial. *Ann Oncol.* 2021;32(12):1590-1596. https://doi.org/10.1016/j.annonc.2021.09.003
25. Eikesdal HP, Yndestad S, Elzawahry A, et al. Olaparib monotherapy as primary treatment in unselected triple negative breast cancer. *Ann Oncol.* 2021;32(2):240-249. https://doi.org/10.1016/j.annonc.2020.11.009

26. Blanc-Durand F, Yaniz E, Genestie C, et al. Evaluation of a RAD51 functional assay in advanced ovarian cancer, a GINECO/GINEGEPS study. *J Clin Oncol.* 2021;39(15 Suppl):5513. https://doi.org/10.1200/JCO.2021.39.15_suppl.5513
27. Blanc-Durand F, Yaniz-Galende E, Llop-Guevara A, et al. A RAD51 functional assay as a candidate test for homologous recombination deficiency in ovarian cancer. *Gynecol Oncol*. 2023;171:106-113. https://doi.org/10.1016/j.ygyno.2023.01.026
28. Pellegrino B, Herencia-Ropero A, Llop-Guevara A, et al. Preclinical in vivo validation of the RAD51 test for identification of homologous recombination-deficient tumors and patient stratification. *Cancer Res*. 2022;82(8):1646-1657. https://doi.org/10.1158/0008-5472.CAN-21-2409
29. Graeser M, McCarthy A, Lord CJ, et al. A marker of homologous recombination predicts pathologic complete response to neoadjuvant chemotherapy in primary breast cancer. *Clin Cancer Res*. 2010;16(24):6159-6168. https://doi.org/10.1158/1078-0432.CCR-10-1027
30. Tumiati M, Hietanen S, Hynninen J, et al. A functional homologous recombination assay predicts primary chemotherapy response and long-term survival in ovarian cancer patients. Clin Cancer Res 2018;24(18):4482-4493. https://doi.org/10.15252/emmm.202013366
31. Feng Z, Zhu C, Zhang X, et al. Comprehensive evaluation of genomic and functional assays for homologous recombination deficiency with high-grade epithelial ovarian cancer: Platinum sensitivity and prognosis. *Int J Gynecol Cancer*. 2025;35(1):100031. https://doi.org/10.1016/j.ijgc.2024.100031
32. Wang D, Lippard SJ. Cellular processing of platinum anticancer drugs. *Nat Rev Drug Discov*. 2005;4(4):307-320. https://doi.org/10.1038/nrd1691
33. Gomez-Puerto D, Llop-Guevara A, Cruellas M, et al. Genetic and functional homologous repair deficiency as biomarkers for platinum sensitivity in TNBC: A case report. *Front Oncol*. 2022;12:963728. https://doi.org/10.3389/fonc.2022.963728
34. Kausar MA, Alshammari KF, Alenazi F, et al. RAD51 and PALB2 in precision oncology: Clinical implications for HRD associated breast and ovarian cancers (Review). *Int J Oncol*. 2025;67(2):65. https://doi.org/10.3892/ijo.2025.5771

Submitted: January 29, 2026

П. Гордійчук [1, 2], *М. Дороніна* [1], *О. Пономарьова* [1, 2],
М. Гордійчук [1, 2], *Д. Шапочка* [3], *Д. Мельник* [1], *А. Терещенко* [4]

[1] Національний університет охорони здоров'я України імені П.Л. Шупика, Київ, Україна
[2] КНП «Київський міський клінічний онкологічний центр», Київ, Україна
[3] Клінічна лікарня «Феофанія», Київ, Україна
[4] ННЦ «Інститут біології та медицини», Київ, Україна

ПРОГНОСТИЧНЕ ЗНАЧЕННЯ ЕКСПРЕСІЇ RAD51 У ЛІКУВАННІ ПРЕПАРАТАМИ ПЛАТИНИ ПАЦІЄНТОК ІЗ СЕРОЗНОЮ КАРЦИНОМОЮ ЯЄЧНИКА ВИСОКОГО СТУПЕНЯ ЗЛОЯКІСНОСТІ

Вступ. Рак яєчника характеризується високим рівнем захворюваності та смертності. Висока інвазивність серозної карциноми яєчника високого ступеня злоякісності за відсутності надійних біомаркерів призводить до пізньої діагностики та раннього метастазування, що стає на заваді вибору ефективного медикаментозного лікування. Для раку яєчника характерним є дефіцит системи гомологічної репарації (HRD), де RAD51 відіграє ключову роль у її відновленні. **Мета** роботи — встановити зв'язок між рівнем експресії білка RAD51 у пацієнток з серозною карциномою яєчників високого ступеня злоякісності після неоад'ювантної хіміотерапії препаратами платини з частотою виникнення рецидиву, прогресування захворювання та безрецидивною виживаністю. **Матеріали та методи.** Представлено попередні результати дослідження архівних гістологічних матеріалів 30 хворих на серозну карциному яєчника високого ступеня злоякісності III стадії, які отримували періопераційну хіміотерапію. Статус RAD51 оцінювався імуногістохімічно в зразках, що містили мінімум 100 пухлинних клітин, позитивних на гемінін. Статус RAD51 вважався позитивним, якщо >10% гемінін-позитивних ядер були позитивні на RAD51. **Результати.** У 16 з 30 пацієнток (53,33%) пухлини мали RAD51-позитивний статус HRP (із збереженою функцією гомологічної рекомбінації), у 14 (46,66%) — RAD51-негативний статус HRD (із дефіцитом гомологічної рекомбінації). Встановлено, що ризик прогресування захворювання в пацієнток зі статусом RAD51 HRP був у 3,42 раза вищим порівняно з пацієнтами зі статусом RAD51 HRD (HR = 3,42; 95% ДІ 1,38—8,46; p = 0,008). Отримані результати свідчать, що в пацієнток із серозною карциномою яєчників високого ступеня злоякісності III стадії зі статусом RAD51 HRP, незважаючи на проведену хіміотерапію, спостерігалося статистично значуще ($p < 0,001$) підвищення частоти прогресування захворювання в період від 3 до 12 місяців післяопераційного спостереження порівняно з пацієнтами зі статусом RAD51 HRD. **Висновок.** Експресія RAD51 має вагоме прогностичне значення щодо відповіді хворих на серозну карциному яєчника високого ступеня злоякісності на терапію препаратами платини та потребує проведення додаткових досліджень для рутинного застосування в клінічній практиці.

Ключові слова: рак яєчника, RAD51, платиновмісна хіміотерапія, потенційний предиктор чутливості до платини, система гомологічної репарації.