Review paper

Immunohistochemical and molecular diagnostics tailoring personalised ovarian cancer therapy

Adam Michał Kowalewski[1,2], Łukasz Szylberg[1,3]

[1]Department of Tumour Pathology, Oncology Centre, Prof. Franciszek Lukaszczyk Memorial Hospital, Bydgoszcz, Poland
[2]Faculty of Medicine, Bydgoszcz University of Science and Technology, Bydgoszcz, Poland
[3]Department of Obstetrics, Gynecology and Oncology, Collegium Medicum in Bydgoszcz, Nicolaus Copernicus University in Torun, Bydgoszcz, Poland

Ovarian cancer remains the most lethal gynaecological malignancy, and it exhibits substantial histologic and molecular heterogeneity. Traditional systemic therapies have produced only modest survival gains. Integration of immunohistochemical (IHC) and molecular diagnostics has shifted care toward biomarker-driven personalised treatment. This review presents current IHC approaches for histotype classification and the most important actionable protein markers (FRα, HER2, TROP2, MMR proteins, and PD-L1), together with molecular testing for *BRCA1/2*, homologous recombination deficiency (HRD) status, and circulating tumour DNA (ctDNA) monitoring. These diagnostics now direct poly(ADP-ribose) polymerase inhibitors maintenance in HRD-positive disease and antibody-drug conjugates such as mirvetuximab soravtansine in FRα-high platinum-resistant tumours. However, important limitations remain: intratumoral heterogeneity, variable assay performance, lack of standardisation, and unequal access to testing worldwide. We also discuss how best to combine multiple biomarkers and outline future directions, such as AI-assisted digital pathology, composite predictive models, and ctDNA-guided adaptive strategies, to refine patient selection and improve long-term outcomes.

Key words: ovarian cancer, precision oncology, homologous recombination deficiency, folate receptor α, PARP inhibitors, antibody-drug conjugates.

Introduction

Ovarian cancer is the most lethal gynaecological malignancy, with 5-year overall survival rates remaining at approximately 50% even after optimal cytoreductive surgery and platinum-based chemotherapy [1, 2]. The disease is highly heterogeneous. High-grade serous carcinoma (HGSC) comprises roughly 70% of cases, while clear-cell, endometrioid, low-grade serous, and mucinous subtypes each harbour unique molecular drivers, clinical trajectories, and treatment susceptibilities. For decades, therapy depended on empiric platinum-based regimens offering only limited long-term benefit. Precision oncology has transformed this approach by enabling biomarker-directed treatment using poly(ADP-ribose) polymerase inhibitors (PARPi), antibody-drug conjugates (ADC), and immune checkpoint inhibitors in appropriately selected patients [1, 3]. Immunohistochemical (IHC) panels provide rapid histotype classification and protein-level information, complemented by next-generation sequencing (NGS), genomic scar assays, and circulating tumour DNA (ctDNA) to identify actionable alterations, including *BRCA1/2* mutations and homologous recombination deficiency (HRD).

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This review integrates contemporary evidence on IHC-based surrogates and molecular testing to inform personalised management of advanced epithelial ovarian cancer. Emphasis is given to clinically established biomarkers (*BRCA1/2*), HRD status, folate receptor α (FRα/folate receptor 1 [FOLR1]), HER2, TROP2, mismatch repair proteins, and PD-L1 and their practical application in therapeutic pathways. While HGSC and endometrioid carcinomas are the primary focus given recent therapeutic successes in these subtypes, rarer histologies and real-world challenges (assay standardisation, tissue adequacy, and disparities in access) are also considered.

As of 2026, PARPi maintenance therapy is the standard of care for patients with *BRCA*-mutated or HRD-positive tumours, and mirvetuximab soravtansine has been approved for platinum-resistant, FRα-high disease [4, 5]. Novel ADC directed against HER2 and TROP2, along with emerging combination strategies involving anti-angiogenic agents and immunotherapy, are further advancing the field [6, 7]. Nonetheless, most patients continue to present with advanced-stage disease, tumours exhibit marked intratumoral heterogeneity, and resistance mechanisms emerge rapidly. Robust diagnostic tools are therefore critical for optimal patient selection, avoidance of futile therapy, and promotion of equitable care across diverse settings [8, 9]. We hope this review gives pathologists and oncologists a clear, practical overview based on the latest guidelines and major trial results.

Immunohistochemical diagnostics: histotyping and actionable protein markers

Accurate histopathological subtyping is fundamental to ovarian cancer management, as histotype directly informs prognosis, germline testing, and therapeutic selection. Updated World Health Organisation criteria, combined with validated IHC algorithms, now yield > 95% concordance with molecular classification [10, 11]. A four-marker panel (WT1, p53, Napsin A, and progesterone receptor) correctly assigns the major subtypes in approximately 88% of cases; incorporation of p16, *ARID1A*, HNF1β, and β-catenin further improves the diagnostic accuracy [10].

In HGSC, diffuse nuclear WT1 positivity together with abnormal p53 staining (either overexpression or complete null pattern) provides a reliable surrogate for the near-universal *TP53* mutations. Clear-cell carcinomas are identified by Napsin A and HNF1β expression, frequently with *ARID1A* loss (in approximately 50% of cases). Endometrioid carcinomas are typically WT1 negative and may exhibit nuclear β-catenin accumulation in the presence of *CTNNB1* mutations. These IHC distinctions carry direct therapeutic relevance: clear-cell and mucinous histotypes show limited responsiveness to platinum agents and PARPi but may derive benefit from emerging targeted therapies.

In addition to histotyping, IHC serves as a cost-effective surrogate for key molecular alterations. Loss of BRCA1 protein expression correlates with promoter hypermethylation or biallelic inactivation and can triage patients for germline or somatic *BRCA* testing, although its sensitivity is approximately 80% [11]. Mismatch repair protein IHC (MLH1, PMS2, MSH2, MSH6) identifies deficiency in roughly 3% of ovarian carcinomas, flagging Lynch syndrome carriers and potential candidates for pembrolizumab [10]. Most critically, several protein biomarkers now directly guide antibody-drug conjugate eligibility. FRα expression is assessed by validated FRα (FOLR1) IHC, with ≥ 75% of tumour cells showing moderate to strong

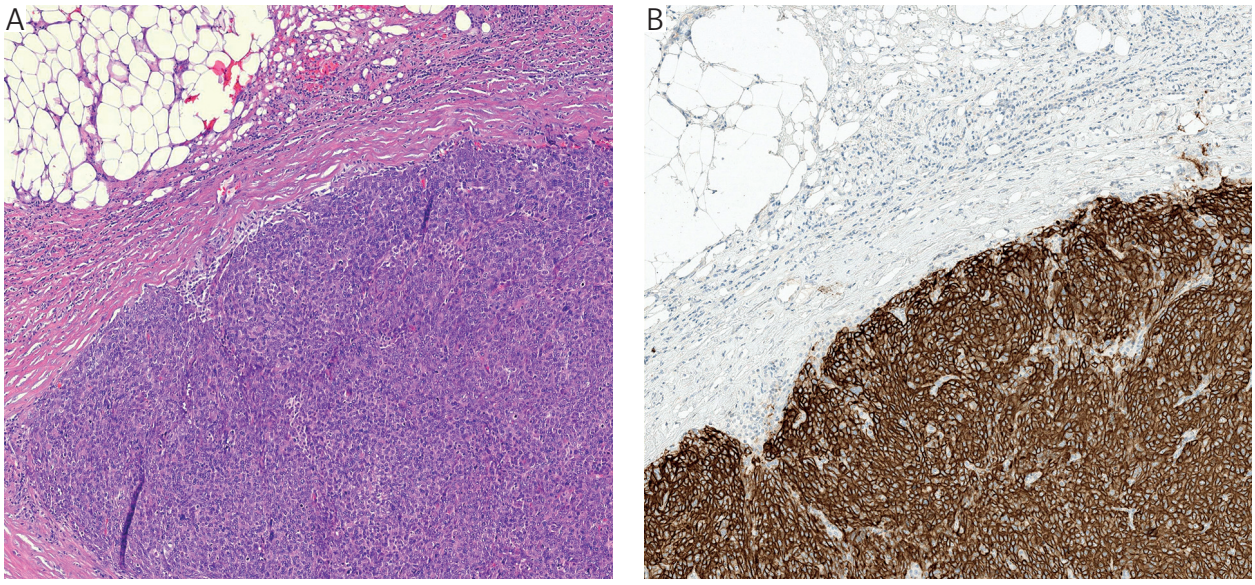

Figure 1. Representative folate receptor 1 positivity in high-grade serous ovarian cancer

On the left is H&E staining. On the right is folate receptor 1 positive immunohistochemistry demonstrating strong membranous staining.

Table I. Major ovarian cancer histotypes and associated immunohistochemical markers

Histotype	Prevalence	Key IHC markers	Prognostic/therapeutic notes
High-grade serous	~70%	PAX8 +, WT1+, p53 abnormal, p16 diffuse	HRD common, PARPi, mirvetuximab
Clear cell	~10%	Napsin A+, HNF1β +, ARID1A–	Platinum-resistant, emerging targeted options
Endometrioid	~10%	WT1–, ER/PR +, β-catenin nuclear	MMRd in ~20%, Lynch syndrome screening
Low-grade serous	~5%	PAX8 +, WT1+, p53 wild-type, p16 negative or patchy	MAPK pathway, limited PARPi benefit
Mucinous	~3%	CK7+, CK20+, CDX2+, p16 patchy	*KRAS/TP53*, poor chemotherapy response

HRD – homologous recombination deficiency, IHC – immunohistochemical, MMRd – mismatch repair, PARPi – poly(ADP-ribose) polymerase inhibitors

membranous staining qualifying for mirvetuximab soravtansine [12]. Representative FOLR1 positivity in HGSC is presented in Figure 1. HER2 is scored according to modified gastric cancer criteria, TROP2 on a 0–3+ scale, and PD-L1 (22C3 or SP263 clones) is evaluated despite its limited predictive value for monotherapy [13]. Table I summarises the major epithelial ovarian cancer histotypes, their characteristic IHC profiles, and associated therapeutic implications.

Molecular diagnostics: genomic instability and homologous recombination deficiency assessment

Homologous recombination deficiency is the most clinically actionable genomic feature in ovarian cancer. Germline or somatic *BRCA1/2* mutations are identified in 15–20% of HGSC, while an additional approximately 30% of tumours exhibit *BRCA*-wild-type HRD driven by promoter methylation, RAD51C/D or PALB2 alterations, or other mechanisms. These defects generate characteristic genomic scars (including loss of heterozygosity, telomeric allelic imbalance, and large-scale state transitions) that are quantified by composite genomic instability scores (GIS) in clinically validated assays.

Current guidelines (ESMO, NCCN, ESGO–ESMO–ESP) recommend universal germline and somatic *BRCA1/2* testing at diagnosis for advanced disease, with reflex HRD assessment in *BRCA*-wild-type cases [5, 9, 11]. Clinically validated HRD assays measuring genomic instability score (GIS ≥ 42) or loss of heterozygosity remain the reference standards [14, 15]. Positive homologous recombination deficiency status strongly predicts benefit from PARPi maintenance therapy: in the SOLO1 trial, olaparib extended median progression-free survival by 13.8–56 months in *BRCA*-mutated patients [16], while PRIMA and PAOLA-1 confirmed similar gains across broader HRD-positive cohorts [17, 18]. Tumours lacking HRD derive minimal or no benefit, underscoring the importance of precise testing.

Liquid biopsy with ctDNA provides a dynamic dimension. Tumour-informed assays can detect *TP53* or *BRCA* reversion mutations that restore homologous recombination proficiency and mediate PARPi resistance, often months before radiographic progression. Serial ctDNA monitoring also outperforms CA-125 for detecting minimal residual disease after surgery or chemotherapy. Expanded NGS panels now routinely include non-*BRCA* homologous recombination repair genes, although the predictive value of many non-*BRCA* alterations for PARPi response still requires cautious interpretation [15].

Biomarker-driven targeted therapies: PARP inhibitors and antibody-drug conjugates

Poly(ADP-ribose) polymerase inhibitors exploit synthetic lethality in HRD-deficient cells by trapping PARP on DNA and inducing lethal double-strand breaks. Olaparib, niraparib, and rucaparib are approved as first-line maintenance therapy in *BRCA*-mutated or HRD-positive ovarian cancer [19, 20]. Their combination with bevacizumab further prolongs progression-free survival in HRD-positive patients [21]. Reversion mutations and other resistance mechanisms can be monitored through ctDNA analysis, enabling timely transition to alternative agents [22].

Antibody-drug conjugates have substantially improved outcomes in platinum-resistant disease. In the phase 3 MIRASOL trial, mirvetuximab soravtansine demonstrated superiority over investigator's choice chemotherapy in FRα-high tumours, achieving an objective response rate of 42% vs. 16% and an overall survival benefit [23]. Trastuzumab deruxtecan has received accelerated approval for HER2-positive (IHC 3+ or 2+ fluorescence *in situ* hybridization amplified)) solid tumours, including ovarian cancer (DESTINY-PanTumor02: ORR 45%) [24]. TROP2-directed ADC such as sacituzumab govitecan and datopotamab deruxtecan have shown promising activity in heavily pretreated patients [12].

Table II. Clinically actionable biomarkers, companion diagnostics, and approved therapies for ovarian cancer

Biomarker	Test method	Prevalence (HGSC)	Therapy	Key trial evidence
BRCA1/2 mut	Germline/somatic NGS	15–20%	Olaparib, niraparib maintenance	SOLO1, PRIMA
HRD (GIS ≥ 42)	Clinically validated HRD assay (GIS or LOH-based)	~50%	Niraparib, olaparib + bevacizumab	PAOLA-1, PRIMA
FRα high	FOLR1 IHC	35–50%	Mirvetuximab soravtansine	MIRASOL
HER2 3+ or amp	IHC + FISH	5–15%	Trastuzumab deruxtecan	DESTINY-PanTumor02
dMMR/MSI-high	MMR IHC or NGS	~3%	Pembrolizumab	KEYNOTE-158 [26]
TROP2+	IHC	> 40%	Sacituzumab govitecan (trials)	Phase 2 ongoing (NCT06028932)

dMMR – mismatch repair deficient, FISH – fluorescence in situ hybridization, FOLR1 – folate receptor 1, GIS – genomic instability scores, HGSC – high-grade serous carcinoma, HRD – homologous recombination deficiency, IHC – immunohistochemical, MSI – microsatellite instability, NGS – next-generation sequencing

A large multicentre Polish cohort of 229 patients with platinum-resistant epithelial ovarian cancer identified FOLR1 positivity in 50.7% (95% CI: 44.2–57.2) of cases using validated FRα IHC. High expression (> 85% of cells with moderate-to-strong membranous staining) was present in 40% of tumours, with approximately 18% falling into the borderline range (65–85%). This prevalence is modestly lower than in some international HGSC series that applied less stringent cut-offs, but it aligns closely with studies using identical criteria, underscoring the influence of histologic heterogeneity and assay standardisation [25]. Table II summarises the major biomarkers, companion diagnostics, and approved therapies as of 2026.

Biomarkers for immunotherapy and multimodal integration

Ovarian cancer is generally regarded as an immunologically "cold" tumour, with low tumour mutational burden and limited tumour-infiltrating lymphocytes. PD-L1 expression is observed in 10–25% of cases but correlates only modestly with response to single-agent immune checkpoint inhibitors [27]. The clearest benefit is confined to the small subset (~3%) of mismatch repair-deficient (dMMR)/microsatellite instability (MSI)-high or *POLE*-ultramutated tumours, which show durable responses to pembrolizumab or dostarlimab [28, 29]. Ongoing phase 3 trials are evaluating PARPi-immunotherapy combinations that exploit PARPi-induced neoantigen formation and PD-L1 upregulation. Early signals of synergy appear particularly promising in *BRCA*/HRD-positive cohorts [30].

PD-L1 IHC, although the most widely available predictive tool, has well-recognised limitations. Four Food and Drug Administration approved assays use distinct antibody clones (22C3, SP142, 28-8, SP263) on separate platforms, each employing non-interchangeable scoring algorithms, including tumour proportion score, combined positive score, and immune cell score, together with variable clinical cut-offs. Intratumoural heterogeneity frequently leads to misclassification, and small biopsies often underestimate expression relative to resection specimens [31]. These issues have stimulated interest in refined sampling strategies (multiple core biopsies), pre-analytical optimisations (e.g. PNGase F deglycosylation), and complementary technologies such as multiplex immunofluorescence, quantitative immunofluorescence, AI-assisted digital pathology, and liquid biopsy readouts (ctDNA, soluble PD-L1, PD-L1-positive circulating tumour cells) [32]. Composite models that integrate PD-L1 with tumor mutational burden (TMB), MSI status, tumour-infiltrating lymphocytes, and neoantigen load consistently outperform PD-L1 alone [27, 33]. Multimodal biomarker integration, combining IHC, NGS, ctDNA, and functional assays such as RAD51 foci formation, has become routine practice in molecular tumour boards [8, 14, 33].

Future directions

Over the past 10 years we have seen several important conceptual advances. Homologous recombination deficiency has become a useful predictive biomarker well beyond *BRCA1/2* mutations, synthetic lethality with PARP inhibitors has been clinically validated, and antibody-drug conjugate technology has matured to the point where it no longer depends on classical oncogenic drivers. Immunohistochemistry has broadened access to biomarker testing, while ctDNA has introduced real-time pharmacodynamic monitoring. Refinements in PD-L1 assessment, including digital pathology, multiplex imaging, and composite biomarker panels, continue to improve patient selection for immunotherapy [3, 21].

Significant gaps remain. Homologous recombination deficiency scoring is not yet globally harmon-

ised, PARPi resistance mechanisms are mechanistically diverse, and immunotherapy response rates stay low outside dMMR subsets [14]. Histotype-specific therapies for clear-cell and mucinous carcinomas remain limited, and equitable access to NGS and HRD testing is far from universal. Additional population-specific biomarker data (FOLR1, PD-L1) from underrepresented regions are urgently needed.

Methodological priorities include assay harmonisation, prospective validation of composite signatures (HRD + FRα + TMB + PD-L1), AI-assisted digital pathology, and spatial multi-omics [34]. Clinical priorities centre on ctDNA-guided adaptive designs, rational combinations (PARPi + ADC + immunotherapy), and dedicated basket trials for rare histotypes.

Conclusions

Several next steps are poised to advance the field. International head-to-head trials comparing commercial HRD platforms with functional assays will be essential to resolve standardisation gaps and establish the most accurate, reproducible predictors of PARPi benefit across populations. Prospective registration of ctDNA-driven adaptive treatment algorithms should be prioritised to evaluate their ability to support real-time therapeutic adjustments and detect emerging resistance well before radiographic progression. In parallel, the development and validation of low-cost IHC- and polymerase chain reaction based surrogates will be necessary to extend precision diagnostics to resource-limited settings without compromising reliability. Single-cell and spatial multi-omics studies will be critical to identify novel therapeutic targets, such as B7-H4 and claudin-6, while providing high-resolution insight into intratumoural heterogeneity and subtype-specific resistance mechanisms [21, 23, 24]. Finally, the establishment of comprehensive real-world evidence registries that systematically track outcomes of biomarker-directed care across diverse populations and healthcare systems will strengthen the generalisability of current strategies in everyday practice. Taken together, these developments should allow us to select patients more precisely, postpone or overcome resistance, and ultimately improve long-term survival across all subtypes of ovarian cancer.

Disclosures

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4. Conflicts of interest: None.

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Address for correspondence

Adam Michał Kowalewski, MD, PhD
Faculty of Medicine
Bydgoszcz University of Science and Technology
Bydgoszcz, Poland
e-mail: adam.kowalewski@pbs.edu.pl