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Efficacy and safety of Olaparib in cancer treatment: A systematic review

Eficacia y seguridad de olaparib en el tratamiento oncológico: una revisión sistemática

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ABSTRACT

Introduction: Olaparib has become a key treatment for a wide range of solid tumors characterized by defects in homologous recombination repair. Its integration into clinical practice has transformed the management of several malignancies; however, its optimal use requires careful patient selection and appropriate toxicity management, both of which are essential components of oncology pharmacy practice. **Materials and methods:** A systematic review of phase II and III clinical trials published between 2020 and 2025 was conducted using PubMed, Web of Science, and Scopus, in accordance with PRISMA guidelines. Studies evaluating the efficacy and safety of olaparib in advanced breast, ovarian, pancreatic, and prostate cancers were included. **Results:** Seventeen studies were selected for review. Robust evidence from phase III trials supports the use of olaparib in ovarian, pancreatic, and prostate cancers with BRCA mutations. In contrast, evidence in triple-negative breast cancer remains limited and heterogeneous. Combination strategies may improve progression-free survival, although they are associated with increased toxicity. **Conclusions:** Olaparib provides a clear clinical benefit in biomarker-selected populations, particularly in patients with BRCA mutations. From an oncology pharmacy perspective, its use requires the integration of molecular diagnostics, toxicity monitoring, and management of drug–drug interactions. Evidence supporting broader use beyond BRCA-mutated populations remains inconsistent and should be interpreted with caution.

Keywords: BRCA; homologous recombination deficiency; olaparib; PARP inhibitors; mutations.

RESUMEN

Introducción: Olaparib se ha convertido en un tratamiento fundamental en múltiples tumores sólidos con defectos en la reparación por recombinación homóloga. Su incorporación a la práctica clínica ha transformado el abordaje de múltiples neoplasias; su uso óptimo requiere una selección cuidadosa de los pacientes y un control de la toxicidad, que son áreas clave para la práctica farmacéutica. **Material y métodos:** Se realizó una revisión sistemática de ensayos clínicos de fase II y III publicados entre 2020 y 2025 en PubMed, Web of Science y Scopus, siguiendo las directrices PRISMA. Se incluyeron estudios que evaluaban la eficacia y seguridad de olaparib en cánceres avanzados de mama, ovario, páncreas y próstata. **Resultados:** Se seleccionaron 17 estudios para su revisión. La sólida evidencia de los ensayos de fase III respalda el uso de olaparib en los cánceres de ovario, páncreas y próstata con mutación del gen del cáncer de mama. Por el contrario, en cáncer de mama triple negativo, la evidencia es limitada y heterogénea. Las estrategias de combinación pueden mejorar la supervivencia libre de progresión, aunque asociadas a un incremento de toxicidad. **Conclusiones:** Olaparib ofrece un beneficio claro en poblaciones seleccionadas por biomarcadores, en particular en pacientes con mutaciones en el gen del cáncer de mama. Desde una perspectiva farmacéutica, su uso requiere la integración de diagnósticos moleculares, la monitorización de la toxicidad y la gestión de las interacciones farmacológicas. La evidencia que respalda un uso más amplio más allá de las mutaciones en el gen del cáncer de mama sigue siendo inconsistente y debe interpretarse con cautela.

Palabras clave: BRCA; deficiencia en la reparación por recombinación homóloga; olaparib; inhibidores de PARP; mutaciones.

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1. Introduction

Cancer remains a major global health challenge, with a growing burden in recent decades due to population aging and changes in environmental and lifestyle risk factors [1]. Modern oncology has evolved toward a precision medicine approach, integrating molecular information to optimize treatment [2].

This review focuses on four malignancies of high clinical relevance: triplenegative breast cancer (TNBC), ovarian cancer, pancreatic cancer, and prostate cancer. Breast cancer is the most common cancer in women worldwide and a leading cause of mortality, with the worst prognosis observed in its triplenegative subtype [3,4]. Ovarian cancer remains one of the deadliest gynecological tumors due to late diagnosis and the absence of reliable populationlevel screening methods [5,6]. Pancreatic cancer, meanwhile, is characterized by high mortality and a projected increase in incidence [7]. Prostate cancer is the most commonly diagnosed cancer in men in many regions, with reductions in mortality attributed to advances in early detection and treatment [8,9].

Cancer treatment has increasingly incorporated precision medicine, with the development of targeted therapies based on specific molecular alterations [10]. Among these, poly(ADPribose) polymerase (PARP) inhibitors represent a key strategy grounded in synthetic lethality, acting on defects in deoxyribonucleic acid (DNA) repair, particularly in tumors harboring BRCA1/2 mutations [11]. Olaparib, the first approved PARP inhibitor, has demonstrated efficacy across multiple solid tumors, and its use has expanded beyond BRCA mutations to include patients with homologous recombination deficiency (HRD) [12–16]. As an oral inhibitor of PARP1, 2, and 3, it represents a significant advance in precision oncology, with ongoing research aimed at optimizing biomarkerbased patient selection [17].

The growing use of Olaparib poses several challenges, including appropriate patient selection based on molecular biomarkers, management of hematologic toxicity, identification of drug–drug interactions, and patient education. These considerations highlight the need for a clinical practiceoriented synthesis of the evidence to support decisionmaking in realworld settings.

2. Materials and methods

2.1 Primary objective

To evaluate the efficacy and safety of Olaparib in solid tumors (TNBC, ovarian, pancreatic, and prostate), as monotherapy or in combination, in patients with advanced or metastatic disease.

2.2 Secondary objectives

- To analyze the efficacy of Olaparib according to molecular profile, particularly in patients with BRCA1/2 mutations and HRD deficiency.
- To evaluate the impact of combination strategies on clinical outcomes.
- To describe the safety profile and treatmentrelated adverse events.

2.3 Study design

A descriptive, observational systematic review with a qualitative approach was conducted.

2.4 Databases and search terminology

The literature search was conducted in the PubMed, Web of Science, and Scopus databases. The research question was defined using the PICO framework [18,19]. What is the efficacy and safety of Olaparib, as monotherapy or in combination, in adults with triplenegative breast cancer, ovarian cancer, pancreatic cancer, or advanced or metastatic prostate cancer, compared with placebo, standardofcare treatment, or no comparator?

In PubMed, “Olaparib” was combined with cancerspecific MeSH terms (“Breast neoplasms,” “Ovarian neoplasms,” “Prostate neoplasms,” “Pancreatic neoplasms”) and title/abstract terms using Boolean operators, applying filters for “Clinical trial” and “Randomized controlled trial” (n = 125). In Web of Science, the filters “Olaparib,” “PARP Inhibitors Neoplasms,” and Phase III/RCT were used, excluding systematic reviews (n = 57). In Scopus, Olaparib and tumorspecific terms were included with clinical trial filters, excluding systematic reviews (n = 53).

2.5 Inclusion criteria

Phase II or III clinical trials (randomized or nonrandomized), published in English (2020–2025), evaluating the efficacy and safety of Olaparib as monotherapy or in combination (chemotherapy, immunotherapy, or targeted therapy) in adults with advanced/metastatic TNBC, ovarian, pancreatic, or prostate cancer. Studies were considered eligible if they included patients with or without BRCA1/2 mutations, regardless of DNA repair status. The studies evaluated Olaparib in metastatic disease (progression) or as maintenance therapy following a response to chemotherapy without active progression at baseline.

2.6 Exclusion criteria

Costeffectiveness analyses, indirect comparisons, combinations with radiotherapy or neoadjuvant/adjuvant use, other tumor types, PARP inhibitors other than Olaparib, and duplicate reports without additional data.

2.7 Study variables

Efficacy outcomes were defined according to RECIST v1.1 [20]: objective response rate (ORR), disease control rate (DCR), progressionfree survival (PFS), radiographic progressionfree survival (rPFS), overall survival (OS), stable disease (SD), complete response (CR), partial response (PR), disease progression (DP), and duration of response (DR).

Safety was assessed using CTCAE v4.0 [21], including treatmentemergent adverse events (TEAEs) and grade ≥ 3 events.

Methodological quality was assessed using the Joanna Briggs Institute checklist for randomized controlled trials [22,23], and was classified as high (10–13 points), moderate (6–9 points), or low (1–5 points).

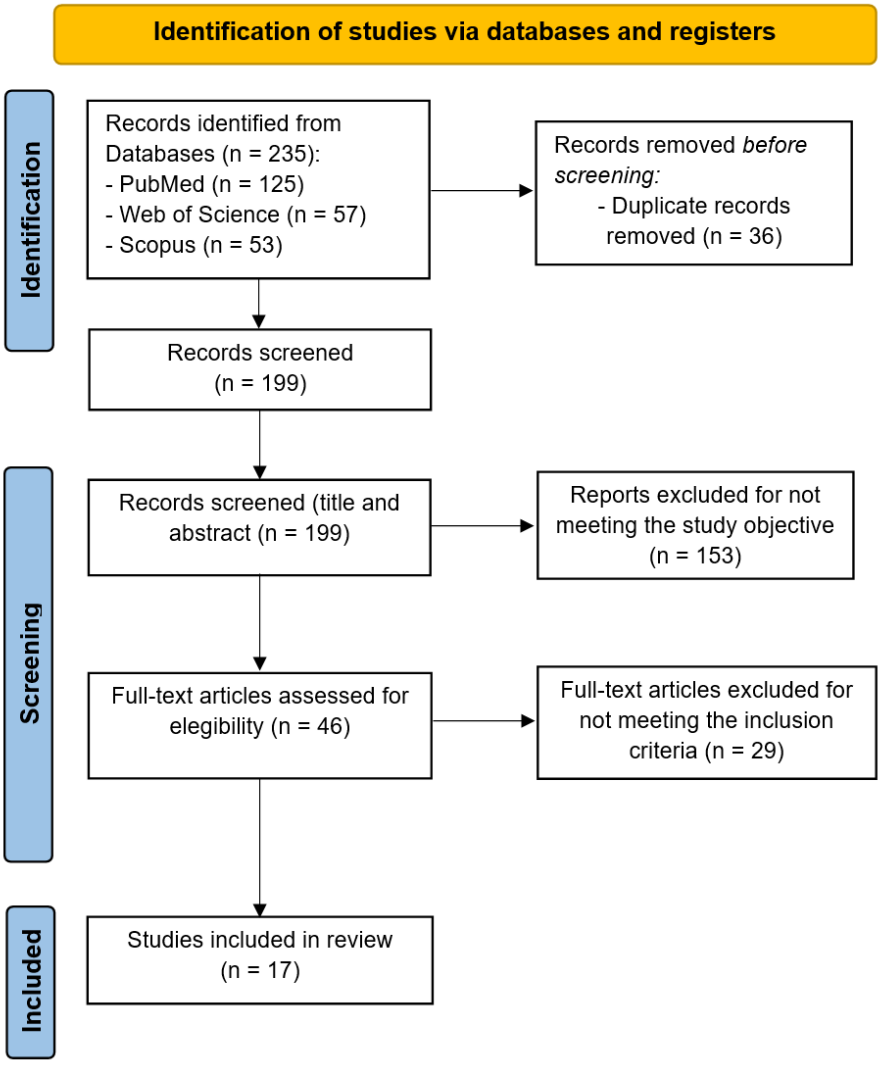
No quantitative synthesis was performed due to considerable clinical and methodological heterogeneity across tumor types, treatment settings (maintenance versus active disease), and therapeutic combinations.

2.8 Selection

A total of 235 records were identified. After removing duplicates (n = 36), 199 were assessed, and 153 were excluded. Review of the full text of 46 articles resulted in 29 exclusions. Finally, 17 studies were included in the qualitative synthesis.

Figure 1 presents the article selection process using a PRISMA (Preferred Reporting Items for Systematic Reviews and MetaAnalyses) flowchart.

Figure 1. Flowchart based on the PRISMA model for article selection.



Source: Author's own creation based on the PRISMA 2020 flowchart.

3. Results and discussion

Table 1 summarizes the characteristics of the 17 studies [24–40]: three on TNBC [24–26], eight on ovarian cancer [27–34], one on pancreatic cancer [35], and five on prostate cancer [36–40].

Regarding treatment strategies, several studies evaluated Olaparib monotherapy [25,28,32,35,36,39], while others assessed combinations with Cediranib [27,30,31,34,37], Abiraterone [38,40], Durvalumab [26,33], Pegylated Liposomal Doxorubicin [29], and Ceralasertib [24].

According to the JBI tool, methodological quality ranged from low to high: two studies were rated as high quality [35,38], most were of moderate quality [26–32,34,36,37,40], and some were of low quality [24,25,33,39]. Phase III trials, such as POLO [35], achieved the highest level of robustness due to adequate randomization and blinding. Lower scores in phase II studies primarily reflect their nonrandomized design, which affects domains such as blinding and allocation concealment, rather than deficiencies in study execution.

The results of the reviewed studies show consistent clinical activity of Olaparib, particularly in tumors with homologous recombination repair defects, with BRCA1/2 mutations being the most robust predictive biomarkers [12,13,15].

In TNBC, available data suggest that Olaparib demonstrates a consistent and welltolerated safety profile and may provide benefit beyond BRCA germline alterations [24–26]. The NOBROLA trial demonstrated activity in HRDpositive patients without BRCA mutations, while studies such as plasmaMATCH and VIOLETTE identified alternative biomarkers, including ATM loss, RAD51 deficiency, and cyclin E1 overexpression [24,25,41–43]. The DORA trial further suggests that platinum sensitivity could serve as a predictive marker of response [26]. Overall, these studies underscore the ongoing search for biomarkers beyond gBRCAm, particularly HRD, although its assessment varies depending on the methods used [44].

In ovarian cancer, Olaparib has demonstrated efficacy as monotherapy and in combination with antiangiogenic, cytotoxic, and immunotherapeutic agents. In platinumresistant disease or following progression on PARP inhibitors, combination strategies aim to overcome resistance [27,30,34]. A previous phase II study demonstrated improved PFS with the combination of Olaparib and Cediranib in platinumsensitive disease [45]; however, these results were not confirmed in the phase III NRGGY004 trial [31]. Studies such as ICON9 showed no benefit in PFS or OS with combination maintenance strategies [46], while NRGGY005 suggests a favorable trend in resistant disease [47]. Conversely, combinations with immunotherapy and antiangiogenic agents may enhance activity in nonBRCA populations, as suggested by the MEDIOLA study, although careful evaluation of the benefit–risk ratio is required [33].

Table 1. Characteristics of the included studies [24–40]

Study data	n	Objective	Conclusions
Ring et al. (2023) [24]. United Kingdom.	75	Phase IIa, open-label, non-randomized, single-arm plasmaMATCH study. Efficacy and safety of Olaparib + Ceralasertib in advanced TNBC (with/ without BRCA/HRR mutations)	ORR: 17.1% (95% CI: 10.4–25.5) PFS: 4.3 months Most common AEs: diarrhea (49%), nausea (48%), and fatigue (45%)
Cortés et al. (2024) [25]. Spain.	6	NOBROLA Phase IIa, open-label, single-arm study. Efficacy and safety of Olaparib monotherapy in advanced NRSC with HRD and without gBRCAm	ORR: 50% (95% CI: 11.8–88.2) PFS: 6.2 months (95% CI: 0.8–15.1) OS: 19.4 months (95% CI: 4.2–34.4) Most common AEs: anemia (83%) and fatigue (50%)
Tan et al. (2024) [26]. Singapore and the United States.	45	Phase II, non-comparative, randomized (1:1), two-arm DORA study: Olaparib (n = 23) and Olaparib in combination with Durvalumab (n = 22). Efficacy of Olaparib ± anti-PD-L1 immunotherapy (Durvalumab), maintenance treatment without CTX in platinum-based QT-sensitive, advanced TNBC	TOS (Olaparib vs. Olaparib in combination with Durvalumab): 44% (95% CI: 23–66) vs. 36% (95% CI: 17–59) PFS: 4.0 months (95% CI: 2.6–6.1) vs. 6.1 months (95% CI: 3.7–10.1) Most common AEs: nausea (48%), fatigue (43%), and anemia (43%) in the Olaparib group vs. anemia (55%) and nausea (50%) in the combination group
Lheureux et al. (2020) [27]. Canada.	34	Phase II, open-label, single-arm EVOLVE study: Cohort 1 (n = 11): platinum-sensitive; Cohort 2 (n = 10): platinum-resistant; Cohort 3 (n = 13): progression to QT and PARP inhibitor Efficacy of Cediranib and Olaparib in ovarian cancer that progressed after treatment with a PARP inhibitor	ORR (cohort 1 vs. cohort 2 vs. cohort 3): 0% vs. 20% vs. 8%. PFS rate at 16 weeks: 55% vs. 50% vs. 39% Most common AEs: diarrhea (68%), nausea (53%), fatigue (44%), and vomiting (44%)

Study data	n	Objective	Conclusions
Penson et al. (2020) [28]. United States.	266	Phase III SOLO3 study, randomized (2:1) to Olaparib (n = 178) or non-platinum-based QT (n = 88) and open-label SOLO3 study. To compare the efficacy of Olaparib monotherapy with non-platinum QT in recurrent platinum-sensitive ovarian cancer and gBRCAm, previously treated with platinum-based QT	ORR (Olaparib vs. QT): 72.2% vs. 51.4% PFS: 13.4 months vs. 9.2 months Most common AEs: nausea: 64.6% vs. 34.2%; fatigue: 52.2% vs. 42.1%; anemia: 51.1% vs. 25.0%
Pérez Fidalgo et al. (2021) [29]. Spain.	31	GEICO-1601/ROLANDO Phase II, single-arm, open-label, non-randomized study. Efficacy of Olaparib with pegylated liposomal doxorubicin in platinum-resistant ovarian cancer, with or without BRCA mutation	ORR: 13% (4 PR). DCR: 77% PFS: 4.2 months (95% CI: 2.7–5.5) OS: 10.8 months (95% CI: 7.1–16.1) Most common AEs: nausea (58%), fatigue (55%), and anemia (55%)
Colombo et al. (2022) [30]. Spain.	123	BAROCCO Phase II, randomized (1:1:1), open-label, three-arm study: Group A (n = 41): QT; Group B (n = 41): Cediranib + Olaparib, continuous regimen; Group C (n = 41): Cediranib + Olaparib, intermittent regimen. Efficacy and safety of Olaparib and Cediranib in platinum-resistant ovarian cancer	PFS (arm A vs. arm B vs. arm C): 3.1 months vs. 5.6 months vs. 3.8 months OS: 9.3 months vs. 11.6 months vs. 9.6 months Most common AEs (continuous vs. intermittent): nausea: 56.1% vs. 48.8%; fatigue: 51.2% vs. 41.5%; anemia: 17.1% vs. 22.0%
Liu et al. (2022) [31]. United States.	565	Phase III NRG-GY004 study, randomized (1:1:1), open-label, three groups: QT (n = 187); Olaparib (n = 189); Olaparib + Cediranib (n = 189). Efficacy and safety of Olaparib and Cediranib vs. QT in platinum-sensitive ovarian cancer	PFS (QT vs. Olaparib vs. combination): 10.3 months vs. 8.2 months vs. 10.4 months Most common AEs (combination): diarrhea (58.2%) and hypertension (38.8%)
Vanderstichele et al. (2022) [32]. Belgium.	160	CLIO Phase II trial randomized (2:1) to receive Olaparib vs. QT, and open-label. Comparing the efficacy of Olaparib vs. QT in recurrent ovarian cancer	Overall ORR (Olaparib vs. QT): 24.3% vs. 28.3%. Overall PFS: 4.8 months (95% CI: 3.2–6.1) vs. 5.7 months (95% CI: 4.0–8.0)
Drew et al. (2024) [33]. Canada and the United Kingdom.	114	MEDIOLA Phase II, non-randomized, open-label, three-cohort study: gBRCAm doublet cohort (n = 51); non-gBRCAm doublet cohort (n = 32); non-gBRCAm triple cohort (n = 31). Efficacy and safety of Olaparib with Durvalumab ± bevacizumab in patients with platinum-sensitive recurrent ovarian cancer who had not previously received PARP inhibitors	ORR (gBRCAm doublet vs. non-gBRCAm doublet vs. non-gBRCAm triple): 92.2% (95% CI: 81.1–97.8) vs. 34.4% (95% CI: 18.6–53.2) vs. 87.1% (95% CI: 70.2–96.4) Most common AEs: nausea: 66.7% vs. 87.5% vs. 74.2%; fatigue: 66.7% vs. 68.8% vs. 54.8%; anemia**:** 51.0% vs. 40.6% vs. 58.1%
Nicum et al. (2024) [34]. United Kingdom.	139	OCTOVA Phase II study randomized (1:1:1), open-label, three groups: Group A (n = 46): CT; Group B (n = 46): Olaparib; Group C (n = 47): Olaparib + Cediranib. Efficacy and safety of Olaparib as monotherapy and in combination with Cediranib in recurrent ovarian cancer	PFS (combination vs. Olaparib vs. QT): 5.4 months vs. 3.7 months vs. 3.9 months ORR: 14% vs. 14% vs. 34% Most common AEs (combination vs. Olaparib): - nausea: 44% vs. 57%; - fatigue: 44% vs. 50%; - anemia: 42% vs. 50%

Study data	n	Objective	Conclusions
Kindler et al. (2022) [35]. United States and Canada.	154	POLO Phase III study randomized (3:2) to Olaparib (n = 92) or placebo (n = 62), double-blind, placebo-controlled. To compare the efficacy and safety of Olaparib in maintenance therapy versus placebo in metastatic pancreatic cancer with gBRCAm that has previously responded to platinum-based QT	PFS (Olaparib vs. placebo): 7.4 months vs. 3.8 months OS: 19.0 months vs. 19.2 months. Most common AEs: fatigue (63%), nausea (49%), and anemia (32%) in the Olaparib group; fatigue (36%) and nausea (24%) in the placebo group
De Bono et al. (2020) [36]. United Kingdom.	387	Phase III PROfound study, randomized (2:1) to Olaparib or control (Enzalutamide or Abiraterone), open-label, two cohorts: -Cohort A (BRCA1/2 or ATM mutations): Olaparib (n = 162) and control (n = 83); -Cohort B (other mutations): Olaparib (n = 94) and control (n = 48). Efficacy of Olaparib vs. control group in mCRPC patients who experienced progression after treatment with Enzalutamide or Abiraterone	- Cohort A: PFS (olaparib vs. control): 7.4 months vs. 3.6 months OS (olaparib vs. control): 18.5 months vs. 15.1 months - General Population (Cohorts A and B): PFS (olaparib vs. control): 5.8 months vs. 3.5 months OS (olaparib vs. control): 17.5 months vs. 14.3 months. - Most common TEAEs (olaparib vs. control): anemia 46% vs. 15%; nausea 41% vs. 19%; fatigue 41% vs. 32%
Kim et al. (2023) [37]. United States.	90	NCI 9984 Phase II, open-label, randomized (1:1) study with two treatment groups: Cediranib + Olaparib (n = 45) and Olaparib alone (n = 45). PFS of Cediranib and Olaparib compared to monotherapy in mCRPC	PFS (combination vs. Olaparib): 8.5 months (95% CI: 5.4–12.0) vs. 4.0 months (95% CI: 3.2–8.5) - Subgroup of patients with HRD: PFS: 10.6 months vs. 3.8 months Most common AEs: nausea: 58% vs. 53%; fatigue: 58% vs. 62%; diarrhea: 56% vs. 51%
Saad et al. (2023) [38]. Canada.	796	Phase III PROpel study, randomized (1:1), double-blind, two-arm: Olaparib + Abiraterone (n = 399) and placebo + Abiraterone (n = 397). PFS of Olaparib and Abiraterone compared with placebo + Abiraterone in mCRPC	PFS (Olaparib + Abiraterone vs. placebo + Abiraterone): 27.6 months (95% CI: 20.5–27.6) vs. 16.6 months (95% CI: 13.9–19.2) OS: 42.1 months vs. 34.7 months Most common AEs: anemia (50%), asthenia (39%), and nausea (31%) in the Olaparib + Abiraterone group; asthenia (30%), hypertension (19%), and anemia (18%) in the placebo + Abiraterone group
Yang et al. (2023) [39]. United States.	30	TAPUR Phase II study (basket trial), non-randomized, single arm. Efficacy of Olaparib in mCRPC and BRCA1/2 mutations	ORR: 69% (95% CI: 51–81%) CR: 58% (95% CI: 37–77%) Most common AEs: anemia (10%), fatigue (10%), and leukopenia (7%)

Study data	n	Objective	Conclusions
Hussain et al. (2024) [40]. United States.	61	BRCAAway Phase II, randomized (1:1:1), open-label, three-arm study: Abiraterone + Prednisone (n = 19); Olaparib (n = 21); and Abiraterone + Prednisone + Olaparib (n = 21). PFS of Abiraterone + Prednisone, Olaparib, or their combination as first-line treatment in mCRPC	PFS (Abiraterone + Prednisone vs. Olaparib vs. Abiraterone + Prednisone + Olaparib): 8.6 months (95% CI: 2.9–17) vs. 14 months vs. 39 months Most common AEs: fatigue: 16% vs. 43% vs. 71%; anemia: 5.3% vs. 29% vs. 24%

Anti-PDL1: antibodies against programmed death-ligand 1; TNBC: triple-negative breast cancer; mCRPC: metastatic castration-resistant prostate cancer; HRD: homologous recombination deficiency; HRR: homologous recombination repair; non-gBRCAm: no germline mutations in BRCA1/2; PARP: poly-ADP-ribose polymerase; PD: disease progression; CH: chemotherapy; OS: overall survival; PFS: progression-free survival; radiological PFS: radiological progression-free survival; DCR: disease control rate; TEAEs: treatment-emergent adverse events.

In pancreatic cancer, Olaparib is a maintenance option for patients with BRCA germline mutations following response to platinumbased chemotherapy, as demonstrated in the POLO study [35]. Although no significant differences in OS were observed, the late divergence of the curves suggests a potential longterm benefit. Realworld data extend its potential application to populations with broader HRD alterations and highlight the prognostic value of inflammatory markers, such as the neutrophiltolymphocyte ratio [48].

In metastatic castrationresistant prostate cancer (mCRPC), Olaparib provides significant clinical benefit in patients with alterations in DNA repair genes, particularly BRCA1/2 and ATM, as evidenced by studies such as PROfound [36]. Its use in combination with hormonal therapies in earlier treatment lines, as in the PROpel study, has shown significant improvements in PFS, including in populations not selected based on molecular criteria [38,40]. The greater benefit observed in selected subgroups reinforces the importance of molecular stratification [36,38,40]. Furthermore, surrogate endpoints such as secondary progressionfree survival (PFS2), time to first subsequent therapy or death, and time to second subsequent therapy or death allow for a more comprehensive assessment of longterm clinical benefit in sequential treatment strategies [49,50].

The safety profile of Olaparib was predictable and manageable, with anemia, nausea, and fatigue being the most common adverse effects. Combination regimens increase toxicity, requiring individualized management, dose adjustments, and close monitoring. From the perspective of oncology pharmacy, these findings highlight the importance of biomarkerbased selection and proactive toxicity management to optimize outcomes with Olaparib.

4. Conclusions

Olaparib is an effective and generally well-tolerated targeted therapy in solid tumors with DNA repair alterations, with the strongest evidence observed in patients with BRCA1/2 mutations and emerging activity in broader HRD-positive populations. However, the use of HRD beyond BRCA remains limited by heterogeneous and non-standardized testing.

In TNBC, ovarian, pancreatic, and prostate cancers, Olaparib demonstrates clinical benefit in both monotherapy and combination therapy, although combination approaches are associated with increased toxicity.

Future research should prioritize standardized biomarker assessment, optimization of combination strategies in molecularly selected patients, and clarification of long-term survival outcomes through extended follow-up and evaluation of treatment sequences.

5. Abbreviations

ATM: Ataxia Telangiectasia Mutated
CR: Complete response
DCR: Disease control rate
DNA: Deoxyribonucleic acid
DR: Duration of response
gBRCAm: Germline mutation in BRCA1/2
HRD: Homologous recombination deficiency
HRR: Homologous recombination repair
JBI: Joanna Briggs Institute
mCRPC: Metastatic castration-resistant prostate cancer
n: Sample size
ORR: Objective response rate
OS: Overall survival
PARP: Poly(ADP-ribose) polymerases
PD: Disease progression
PFS: Progression-free survival
PFS2: Secondary progression-free survival
PR: Partial response
PRISMA: Preferred Reporting Items for Systematic Reviews and Meta-Analyses
rPFS: Radiological progression-free survival
SD: Stable disease
TEAEs: Treatment-emergent adverse events
TNBC: Triple-negative breast cancer

6. Administrative information

6.1 Author contributions:

Silvia Vázquez Gómez: conceptualization.
Silvia Vázquez Gómez, Andrea Portela Sotelo: formal analysis.
Silvia Vázquez Gómez: research.
Silvia Vázquez Gómez, Sandra Caíña López: methodology.
Silvia Vázquez Gómez: project management.
Silvia Vázquez Gómez, Sandra Caíña López, Andrea Portela Sotelo: supervision.
Silvia Vázquez Gómez: validation.
Silvia Vázquez Gómez, Sandra Caíña López, Andrea Portela Sotelo: visualization.
Silvia Vázquez Gómez: drafting – original draft.
Silvia Vázquez Gómez, Sandra Caíña López, Andrea Portela Sotelo: writing – revision and editing.
All authors read and approved the final version of the manuscript.

6.2 Source of research funding

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6.3 Conflict of interest

The authors declare that they have no conflicts of interest.

6.4 Limitations of liability

The authors affirm that the information presented in this manuscript was obtained and analyzed in an ethical and rigorous manner. However, the results and conclusions herein are the sole responsibility of the authors and do not necessarily reflect the views of the journal or its editors. The journal and its editors shall not be held liable for any misuse or misinterpretation of the article's content.

Furthermore, the authors release the journal from liability for any inadvertent errors, omissions, or consequences arising from the publication of this manuscript. The authors assume full responsibility for the originality of the work and for any potential ethical or legal conflicts.

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