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Evaluating the Therapeutic Efficacy of PARP Inhibition in BRCA2-Mutated Ovarian Cancer

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Abstract. This paper concentrates on BRCA2 (BRCA2 DNA Repair Associated)-mutated ovarian cancer, PARP1 inhibition, and the therapeutic efficacy of PARP inhibitors. For the molecular mechanism, BRCA2 mutation leads to homologous recombination repair deficiency, which increases dependence of tumor cells on alternative DNA damage response pathways. This vulnerability, combined with PARP inhibition, creates synthetic lethality. Clinical trials such as SOLO1, SOLO2, and SOLO3 demonstrate clinical evidence showing benefit from olaparib in BRCA-mutated ovarian cancer. Despite significant initial efficacy, cancer cells can still develop resistance. Main resistance-associated mechanisms are BRCA2 reversion, HR restoration, replication fork protection, and drug efflux, which limit the durability of treatments. Findings of this review have multiple implications for clinical practices and future directions. BRCA2 is a valuable therapeutic biomarker: BRCA2 status can provide a basis for determining if patients are more likely to benefit from PARP inhibition treatment. Additionally, PARP inhibition is clinically effective, but initial effectiveness does not mean to be durable, so the clinical efficacy of PARP inhibition should also be evaluated by duration of benefit and development of resistance. Currently, many clinical trials studied pooled populations of BRCA1/2-mutated ovarian cancer patients, but more specific evidence related to BRCA2 is likely to be needed, because BRCA2-specific clinical efficacy cannot be separated in those trials. In the future, trials are expected to analyze BRCA2-mutated patients separate with whole BRCA-mutated groups; focus on better biomarker-based patient selection; and develop rational combination strategies to delay or reverse PARPi resistance.

Keywords: BRCA2, PARPi, Ovarian cancer, therapeutic efficacy

1. Introduction

Currently, ovarian cancer (OC) is one of the most severe gynecologic malignant tumors. Many patients are diagnosed at the advanced stage of the disease [1]. Standard treatments depend on both debulking surgery and platinum-based chemotherapy [2]. However, most of the patients relapse after being treated, especially in late-stage disease [2]. High-grade serous ovarian cancer (HGSOC) is the most important ovarian cancer subtype, and genomic instability is one of its essential characteristics. The TCGA data indicate that approximately 50% of HGSOC exhibit homologous recombination deficiency (HRD) [3]. HRD is a common molecular feature of HGSOC and is frequently associated

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with alterations in BRCA1/2 and other homologous recombination repair (HRR) related genes [3]. BRCA2 is a key tumor suppressor involved in the repair of DNA double-strand breaks (DSBs) through HRR, and BRCA2 mutations can lead to HRD [4]. Pathogenic BRCA2 mutations can impair HR repair, leading to HRD. PARP is a DNA repair enzyme, activated by recognizing DNA damage fragments and promoting DNA damage repair. Therefore, tumor cells are more dependent on alternative DNA repair pathways, so inhibiting PARP may create synthetic lethality which PARP inhibition a plausible therapeutic strategy [4, 5].

BRCA2 deficiency limits cancer cells from repairing certain DNA-related damage efficiently. Then, PARP inhibition could further increase DNA damage or replication-related stress. The coexistence of BRCA2 deficiency and PARP inhibition can induce synthetic lethality by simultaneously compromising homologous recombination and PARP-mediated DNA damage responses, resulting in selective death of BRCA2-deficient tumor cells. This strategy has clear research progress. Early studies have shown that BRCA-deficient cells have selective sensitivity to PARP inhibition [6]. PARP inhibitors (PARPi) subsequently entered clinical development in OC and have been tested in multiple randomized controlled trials [7]. After that, clinical benefits were demonstrated. For example, the SOLO2 data shown that in the trial of 19.1 versus 5.5 months progression-free survival (PFS) (hazard ratio [HR] = 0·30, $p<0·0001$) [8]. Nevertheless, the functions of PARPi are not limited to first-line maintenance treatment. According to SOLO2, for patients with platinum-sensitive recurrent OC and germline BRCA mutation, olaparib maintenance can generate further apparent PFS benefits and demonstrate the trend of long-term OS benefits [9]. For the more valuable SOLO3 data, the treatment group with olaparib versus the treatment group with nonplatinum chemotherapy showed that: Objective Response Rate (ORR): 72.2% versus 51.4%; PFS: HR = 0.62 (13.4 vs 9.2 months) [10]. Although PARPi provides substantial benefits in BRCA mutation-related OC, not all of the patients respond to it, and resistance is common. A group of patients exhibited primary resistance, meaning that they did not respond apparently in the beginning; furthermore, another group of patients exhibited acquired resistance, which means that PARPi were effective initially, but disease progressed afterward [11]. One of the most common and important mechanisms of PARPi resistance is restoration of BRCA2 function through secondary or reversion mutations. By using BRCA2-mutant PEO1 ovarian cancer cells, different PARPi exposures may contribute to different resistance mechanisms, including functional BRCA2 restoration, drug efflux, Ataxia Telangiectasia and Rad3-related protein / Checkpoint Kinase 1 (ATR/CHK1) activation, and replication fork stabilization [11].

Evaluating PARP inhibition in BRCA2-mutated OC cannot be concluded by whether effective or not but is truly defined by a nuanced assessment of how effective, in which clinical setting, and for how long—measured via PFS, OS response rate, and resistance. In this paper, the first part will be the molecular mechanisms of the chain from BRCA2 mutation to HR deficiency to PARP inhibition and then to synthetic lethality. The second part will be the evaluation of the clinical efficacy of olaparib or other PARPi, including PFS, OS, ORR, and safety in BRCA2-associated OC. Next, this paper will research deeply into BRCA2 reversion mutations, HR restoration, replication fork protection, and drug efflux. Eventually, this paper will answer to what extent PARP inhibition is an effective therapeutic strategy for BRCA2-mutated ovarian cancer.

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2. Molecular mechanisms

2.1. BRCA2 mutation and homologous recombination repair deficiency

The tumor suppressor gene BRCA2 encodes a protein involved in DNA double-strand break repair. One of the most crucial functions of BRCA2 is participating in homologous recombination repair (HRR), which is a relatively high-fidelity DNA repair mechanism. Thus, BRCA2 loss-of-function can disrupt HRR [3]. BRCA2 pathogenic mutations can impair HR and generate an HR-deficient phenotype. In HR normal cells, when DNA breaks, cells activate the HRR mechanism. After repair, DNA can be restored exactly as it was, ensuring genome stability without base loss or rearrangement. However, in BRCA2-mutated cells (defective cells), HRR is impaired when DNA is damaged, leading to genome instability. The status of BRCA determines the efficacy of PARPi: BRCA2 mutation (PEO1) makes it sensitive, but BRCA2 restoration (PEO4) makes it drug-resistant. For the same patient source, excluding individual differences, the only variable is BRCA2 [4].

2.2. BRCA2 mutation, HRR deficiency, and PARP1-mediated DNA damage response

PARP1, a major DNA damage sensor, recognizes DNA lesions; participates in DNA damage response and repair; and can catalyze polyADP-ribosylation (PARylation). When BRCA2 is normal, HRR remains functional. DNA damage caused by PARP inhibition, which interferes with DNA damage repair, can still be handled by other available repair mechanisms or HR. Yet, when BRCA2 is mutated, HR repair is defective. Combining with PARP inhibition, both mechanisms can further compromise DNA damage handling and increase replication stress. Two repair vulnerabilities existing simultaneously generate synthetic lethality, leading to a decrease in tumor-cell survival.

2.3. Preclinical evidence specifically from BRCA2-mutated ovarian cancer

PEO1 and PEO4 are key cell models in the research of OC PARPi drug resistance. Their basic value is that these two cell lines come from the same OC patient but are completely different in their response to BRCA2 status and PARPi. PEO1 is an OC cell line in which BRCA2 is mutated, and HR is deficient. PEO1 is sensitive to PARPi. On the other hand, PEO4 is derived from the same patient, but BRCA2 appears to have a reversion mutation, and HR function is partially restored. Then PEO4 appears drug-resistant. The difference in PARPi response among the two models can support the relation between the status of BRCA2 and sensitivity to PARPi [4]. These observations provide the foundation for clinical evidence in Chapter 3.

3. Clinical efficacy of PARP inhibition in BRCA2-mutated ovarian cancer

3.1. Newly diagnosed advanced ovarian cancer: SOLO1

SOLO1 is a randomized, double-blind, and placebo-controlled phase III clinical trial. This research involved newly diagnosed advanced OC (FIGO stage III or IV) patients with mutations in BRCA1/2, who achieved complete or partial clinical remission after receiving platinum-based first-line chemotherapy. In the trials, grouped patients were randomly assigned in a 2:1 ratio to the olaparib (300mg twice daily) maintenance treatment group or the placebo group [12].

In the overall BRCA-mutated population, olaparib significantly reduced the risk of disease progression or death (HR = 0.30; 95% CI, 0.23-0.41), with median PFS not reached versus 13.8

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months in the placebo group. Subgroup analysis based on the BRCA mutation types has shown a greater PFS benefit in the BRCA2-mutated subgroup (HR = 0.20; 95% CI, 0,10-0,37) compared with the BRCA1-mutated subgroup (HR = 0.41; 95% CI, 0.30-0.56). However, the sample size of the BRCA2 subgroup was relatively similar, and the original research did not conduct a direct statistical comparison between BRCA1 and BRCA2, so we cannot conclude that the therapeutic effect of BRCA2 patients is significantly better than that of BRCA1. The results also showed that in the olaparib group, ORR was 43%, and the complete response (CR) rate was 28%; in the placebo group, ORR was 23%, and the CR rate was 12% [12].

With the extension of the follow-up, the OS analysis has shown that the OS hazard ratio (HR) is 0.55 (95% CI, 0.40–0.76) and 7-year survival rates are 67.0% in the olaparib group and 46.5% in the placebo group. Whereas DiSilvestro et al. specified P = 0.0004, the initially defined statistical significance threshold was P < 0.0001. Thus, olaparib maintenance therapy is clinically meaningful for OS improvement in BRCA-mutated OC patients, but it is not statistically significant according to the prespecified threshold [13]. In the 7-year follow-up of the SOLO1 trial, the safety signal of olaparib is consistent with previous reports; no new safety signal was observed. The most common adverse events (AEs) during long-term follow-up include nausea, fatigue/asthenia, vomiting, and anemia. Therefore, the most common AE of grade ≥3 was anemia [13].

3.2. Platinum-sensitive recurrent disease: SOLO2 and SOLO3

SOLO2 is a randomized, double-blind, and placebo-controlled phase III clinical trial, aiming to evaluate the curative effect of olaparib maintenance treatment in platinum-sensitive relapsed OC patients who carried BRCA1/2 mutations and achieved complete or partial remission after the last line of platinum chemotherapy. Patient samples were randomly assigned in a 2:1 ratio to the olaparib maintenance treatment group (300mg twice daily) or the placebo group.

The PFS results have shown that the median PFS of the olaparib group was 19.1 months; the median PFS of the placebo group was 5.5 months; and the HR was 0.30 (95% CI, 0.22–0.41; P < 0.0001). SOLO1 and SOLO2 jointly show that the benefits of PARP inhibition do not only appear at the newly diagnosed maintenance treatment but also appear in the platinum-sensitive recurrent setting [14].

SOLO3 trial further explained whether PARPi still has a benefit compared with clinically available nonplatinum single-agent chemotherapy. SOLO3 is a randomized and open-label phase III clinical trial. The sample included patients with OC who carried germline BRCA1/2 mutations, were sensitive to platinum, but had relapsed. Also, they have received be equal or greater than two prior platinum-based chemotherapy lines.

The results have shown that the ORR of the olaparib group was 72.2% and the ORR of the chemotherapy group was 51.4% (OR = 2.53; P = 0.002), and PFS of the olaparib group was 13.4 months and PFS of the chemotherapy group was 9.2 months (HR = 0.62; P = 0.013). Thus, for the patients with BRCA-mutated platinum-sensitive recurrent OC who have received multi-line treatment, olaparib monotherapy outperformed nonplatinum chemotherapy in both ORR and PFS [10].

In the SOLO3 trial, the safety signal of olaparib is consistent with the established safety profile; no new safety signal was observed. The most common AEs included nausea, fatigue/asthenia, vomiting, and anemia. The incidence of AEs in the two groups was generally similar, but the proportion of treatment interruption was higher in the chemotherapy group (20% vs 7%) [10].

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3.3. Germline vs somatic BRCA: evidence supplement

ORZORA trial further extended the application evidence of PARPi in BRCA-mutated OC, extending the scope of analysis from germline BRCA (gBRCA) to somatic BRCA (sBRCA). The patients involved in the ORZORA trial were platinum-sensitive relapsed OC patients, and they were divided into three groups by gene mutation types: germline BRCA mutations (gBRCAm), somatic BRCA mutations (sBRCAm), and non-BRCA homologous recombination repair mutations (non-BRCA HRRm). Patients received olaparib maintenance treatment. This was a single-arm study, so it could not demonstrate olaparib superiority against placebo like SOLO1 or SOLO2.

The results showed that, for the BRCAm cohort, median OS = 46.8 months; for sBRCAm, median OS = 43.2 months; and for gBRCAm, median OS = 47.4 months. No new safety signals were reported.

The ORZORA findings suggest that the clinical activity in both germline and somatic BRCAm populations was sustained, although the single-arm design limited direct comparative interpretation [9].

3.4. Clinical evidence synthesis

Among newly diagnosed patients with advanced OC carrying BRCA1/2 mutation and responding to platinum chemotherapy, olaparib maintenance treatment brings strong and durable PFS benefit. According to the subgroup analysis, the BRCA2 subgroup particularly gained a larger numerical effect. 7 years of follow-up observed long-term OS signal, although the preset statistical significance threshold was not reached.

In the recurrent stage, SOLO2 demonstrated a strong PFS benefit of olaparib maintenance treatment among patients with BRCA1/2 mutations and platinum-sensitive recurrence. SOLO3 changed the control of PARPi from a placebo treatment to an active chemotherapy drug treatment. In comparison with clinically available nonplatinum single-agent chemotherapy, olaparib has exhibited benefits in both ORR and PFS.

ORZORA has expanded the population benefiting from gBRCAm to sBRCAm. Olaparib has exhibited clinical activity in both populations, although single-arm limitations prevented this from becoming a definite conclusion.

Current clinical evidence support PARP inhibition is a strong therapeutic strategy for treating BRCA-mutated OC, including BRCA2-mutated patients. However, it is worth to notice that the strength of BRCA2 evidence varies between different studies because many trials have enrolled BRCA1 and BRCA2 patients together.

4. Resistance to PARP inhibition in BRCA2-mutated ovarian cancer

A BRCA2 reversion refers to a secondary somatic cell mutation in the BRCA2 gene during treatment with PARPi. The original BRCA2 mutation leads to loss of functional BRCA2, making HRR defective and PARPi sensitive in this case. However, during treatment, secondary mutations or reversion mutations may appear, partially or completely restoring BRCA2 function, so HRR recovers, reducing PARPi sensitivity. Thus, BRCA2 mutations are not only the basis of PARPi sensitivity but also become a critical entry point for acquired drug resistance. According to Huang et al, when PEO1 OC cells were treated with a higher initial dose and short-term PARPi, they acquired resistance by rapidly restoring BRCA2 and upregulating drug excretion activity (PEO1/OlaJR). On the contrary, PEO1 cells with a lower initial dose and long-term PARPi treatment restored the HR

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repair capability by non-BRCA2-dependent ways, demonstrating that BRCA2 reversion also relates to the treatment plan [11]. From a clinical evidence perspective, a systematic analysis done by Darabi et al expanded this mechanism to clinical patients for multiple tumors [15].

PARPi resistance may not only come from BRCA2 reversion, and the HR pathway can potentially be restored through other alterations. HR pathways can be activated in the following ways: other HR-related gene alterations may restore HR function, compensate for defective repair, and reduce dependence on PARP-mediated repair. Therefore, the relationship between BRCA2 status and PARPi sensitivity is dynamic but not fixed permanently [5].

Double-stranded DNA needs to separate into two single strands to form a fork structure during DNA replication – this is a replication fork. In addition to involvement in HR repair, BRCA2 also relates to protection of stalled replication forks. In BRCA2-deficient cells, replication fork protection is impaired, and PARPi further exacerbate replication-associated stress. DNA replication problems keep accumulating, and this usually increases cell death. However, some specific tumor cells are able to restore or protect stalled replication forks. Even though HR is not completely recovered, cells may still tolerate replication-associated damage, which leads to PARPi resistance [6].

Besides HR repair and replication fork protection, tumor cells are also likely to weaken the cytotoxicity of PARPi by reducing the drug concentration inside the cells. Certain tumor cells may reduce intracellular PARPi concentration through increased expression or activity of drug-efflux transporters, decreasing the effective drug exposure and PARPi cytotoxicity, eventually leading to drug resistance [5, 6].

In summary, the essence of BRCA2 reversion is restoring HR; the essence of HR restoration is restoring DNA repair capacity; the essence of replication fork protection is enhancing resistance to replication stress; and the essence of drug efflux is reducing drug exposure. All mechanisms can be concluded that PARPi resistance is multifunctional, and resistance does not necessarily mean that the original BRCA2 mutations are irrelevant to treatment sensitivity; however, tumor cells can evolve multiple ways to bypass this vulnerability created by BRCA2 deficiency.

5. Limitations and future perspectives

5.1. Limitations

The subjects of studies are all BRCA1/2-mutated samples but not specifically BRCA2-mutated only in many critical trials. The subjects of key trials such as SOLO1, SOLO2, and SOLO3 were all OC patients with BRCA1/2 mutations, no sufficient statistical power to independently demonstrate the specific efficacy of BRCA2. Thus, when interpreting the aforementioned clinical trial data, the evidence boundary needs to be recognized: some clinical evidence supporting PARP inhibition comes from pooled BRCA1/2 populations, which means that BRCA2-specific efficacy cannot be directly inferred from pooled results.

PARPi initially works, but acquired resistance can occur, so long-term disease control remains a challenge, especially with BRCA2 reversion or HR restoration, leading to loss of PARPi sensitivity.

For safety, although PARPi has a manageable safety profile, anemia, fatigue, nausea, and many other treatment-related AEs may affect treatment adherence or quality. This is necessary to consider when using PARPi as a long-term maintenance treatment strategy.

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5.2. Future directions

Although the current patient screening strategy focused on BRCA mutation status is relatively effective, there are obvious limitations. Future research can focus on better biomarker-based patient selection, involving BRCA status, HRD status, HRR-related biomarkers, and molecular characteristics associated with PARPi response or resistance.

In addition, combination strategies are also regarded as a promising field. Since single-agent PARPi effectiveness is limited by BRCA2 reversion mutations, HR restoration, replication fork protection, and multiple resistance mechanisms, rational combination therapies may delay or reverse the occurrence of drug resistance. Rational combination therapies should be based on a deep understanding of resistance mechanisms, such as combining an ATR inhibitor to prevent the replication forks' stabilized pathway. However, these combination strategies still need to have their safety and efficacy verified through clinical trials.

Future research should characterize BRCA2-mutated patients separately, rather than relying on pooled BRCA1/2 analyses exclusively. Besides, studies should figure out whether different BRCA2 mutation types are associated with different PARPi responses or resistance patterns. These studies may help to achieve more accurate patient stratification and individualized treatment, promoting more accurate management in the clinical application of PARPi in BRCA2-mutated OC.

6. Conclusion

Rather than curing BRCA2-mutated OC, PARP inhibition is an effective therapeutic strategy for certain cancers, particularly for disease control and prolongation of progression-free survival. When BRCA2 deficiency appears, HR repair is compromised, creating synthetic lethality with PARP inhibition. SOLO1, SOLO2, and SOLO3 can prove the existence of clinical benefits. However, the clinical benefit is limited by acquired resistance that limits durability of response and leads to disease progression. PARP inhibitors are effective, but their efficacy is not permanent, and the strength of specific evidence for BRCA2 remains limited. Future progress is likely to depend on recognizing patients who are most likely to benefit, detecting resistance earlier, and strategies able to maintain or restore PARPi sensitivity.

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