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RESEARCH ARTICLE

PARP inhibitors-associated thrombosis in patients with ovarian cancer: a study of the Spanish Society of Medical Oncology (SEOM) thrombosis and cancer group

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Abstract

Purpose To determine the real-world incidence and predictive factors for venous and arterial thromboembolic events (VTE/AT) in ovarian cancer patients treated with poly-(ADP-ribose) polymerase inhibitors (iPARP).

Methods/patients A multicenter retrospective study involving 329 ovarian cancer patients who initiated iPARP treatment between January 2015 and December 2022. The primary outcome was the incidence of VTE/AT. Secondary outcomes included predictive factors for thrombosis and the impact of thrombosis on overall survival (OS). Data were analyzed using logistic regression and Kaplan–Meier survival analysis.

Results The incidence of VTE/AT was 4.9% (16/329). BRCA2 mutations were significantly more prevalent among patients who developed VTE/AT (56.3% vs. 19.2%; $p < 0.001$). Combined treatment with bevacizumab was significantly associated with a decreased risk of thrombosis (OR: 0.262; 95% CI: 0.095–0.724; $p = 0.010$). No statistically significant differences were observed in the median OS between patients who experienced VTE/ATE (63 months) and those who did not (47 months), with a p value of 0.876.

Conclusions BRCA2 mutations could be a significant predictor for VTE/AT among ovarian cancer patients treated with iPARP. Concomitant treatment with bevacizumab may offer protection against thrombotic events, although a concomitant bias cannot be ruled out. These findings may be of interest when designing future clinical trials in the field of thromboprophylaxis.

Keywords PARP inhibitors · Cancer-related thrombosis · Ovarian cancer

Introduction

The BRCA1 and BRCA2 genes are essential for homologous recombination repair, a precise mechanism for correcting double-strand DNA breaks. Mutations in these genes impair DNA repair, increasing genomic instability and tumor formation [1].

Tumor cells with BRCA mutations can survive by utilizing an alternative repair pathway, non-homologous end joining, where poly-(ADP-ribose) polymerase (PARP) is crucial. PARP inhibitors (iPARPs) block this enzyme, preventing DNA repair in tumor cells while sparing non-mutated ones. This leads to DNA damage accumulation, ultimately causing cell death in BRCA-mutant tumors [1].

iPARPs, including olaparib, niraparib, and rucaparib, are primarily used in ovarian cancer. Clinical trials [2–11] have reported gastrointestinal and hematological toxicities as common adverse effects, with a potential but unclear risk of thromboembolic events (VTE/AT).

Despite the low incidence of thrombosis in clinical trials, real-world data on VTE/AT risk with iPARPs remain limited. Further research is needed to clarify this association and assess the true thrombotic risk in clinical practice.

Extended author information available on the last page of the article

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Material and methods

This study has been sponsored by the SEOM Thrombosis and Cancer Section. It is a retrospective, multicenter study (14 centers). Data from patients with ovarian cancer who initiated iPARP between 01/01/2015 and 31/12/2022 were collected. Selection was independent of tumor stage, type of iPARP, or treatment intent. Participants had to have a minimum follow-up of 6 months (unless this was impossible due to patient demise).

Table 1 Baseline characteristics

Variable	Overall ($n=329$)	VTE/AT ($n=16$)	No VTE/AT ($n=313$)	p value
Age—median (IQR)	62 (55–71)	60 (57–68)	62 (55–71)	0.78
ECOG PS—*n* (%)				0.790
0	154 (46.8%)	6 (37.5%)	148 (47.3%)	
1	147 (44.7%)	9 (56.3%)	138 (44.1%)	
2	24 (7.3%)	1 (6.3%)	23 (7.3%)	
3	4 (1.2%)	0 (0%)	4 (1.3%)	
Disease stage—*n* (%)				0.233
III	161 (49.2%)	5 (31.3%)	156 (50.2%)	
IV	166 (50.8%)	11 (68.8%)	155 (49.8%)	
Khorana score—*n* (%)				0.288
1	251 (76.3%)	10 (62.5%)	241 (77%)	
2	71 (21.6%)	5 (31.3%)	66 (21.1%)	
3	7 (2.1%)	1 (6.3%)	6 (1.9%)	
Oncologic status at iPARP initiation—*n* (%)				0.539
Complete response	92 (28.2%)	5 (31.3%)	87 (28.1%)	
Partial response	149 (45.7%)	7 (43.8%)	142 (45.8%)	
Stable disease	56 (17.2%)	4 (25%)	52 (16.8%)	
Progression	29 (8.9%)	0 (0%)	29 (9.4%)	
Homologous recombination deficiency—*n* (%)				0.114
No	151 (45.9%)	4 (25%)	147 (47%)	
Yes	168 (51.1%)	12 (75%)	156 (49.8%)	
Unknown	10 (3%)	0 (0%)	10 (3.2%)	
BRCA mutation—*n* (%)				0.006
No	175 (53.2%)	6 (37.5%)	169 (54%)	
Yes, BRCA1	74 (22.5%)	1 (6.3%)	73 (23.3%)	
Yes, BRCA2	69 (21%)	9 (56.3%)	60 (19.2%)	
Yes, BRCA1 and BRCA2	1 (0.3%)	0 (0%)	1 (0.3%)	
Unknown	10 (3%)	0 (0%)	10 (3.2%)	
Type of BRCA mutation—*n* (%)				0.512
Somatic	35 (24.3%)	1 (10%)	34 (25.4%)	
Germline	100 (69.4%)	8 (80%)	92 (68.7%)	
Unknown	9 (6.3%)	1 (10%)	8 (6%)	
Treatment setting—*n* (%)				0.713
Maintenance after 1st-line	149 (45.4%)	8 (53.3%)	141 (45%)	
Maintenance after 2nd-line	132 (40.2%)	5 (33.3%)	127 (40.6%)	
Maintenance after 3rd or later	41 (12.5%)	2 (13.3%)	39 (12.5%)	
2nd-line treatment	1 (0.3%)	0 (0%)	1 (0.3%)	
3rd or later lines	5 (1.5%)	0 (0%)	5 (1.6%)	
Type of iPARP—*n* (%)				0.398
Olaparib	160 (48.6%)	10 (62.5%)	150 (47.9%)	
Niraparib	151 (45.9%)	6 (37.5%)	145 (46.3%)	
Rucaparib	18 (5.5%)	0 (0%)	18 (5.8%)	
Concomitant treatment with iPARP—*n* (%)				< 0.001
None	315 (95.7%)	14 (87.5%)	301 (96.2%)	
Bevacizumab	11 (3.3%)	0 (0%)	11 (3.5%)	
Other	3 (0.9%)	2 (12.6%)	1 (0.3%)	

ECOG PS Eastern Cooperative Oncology Group Performance Status, *iPARP* poly(ADP-ribose) polymerase inhibitor, *IQR* interquartile range, *VTE/AT* venous and arterial thromboembolic events

Table 2 Characteristics of VTE/AT events in patients with ovarian cancer and iPARP

Variable	*n* (%)
ECOG PS at VTE/AT diagnosis	
0	7 (43.8%)
1	8 (50%)
2	2 (6.3%)
Cancer status at VTE/AT diagnosis	
Complete response	3 (18.8%)
Partial response	4 (25%)
Stable disease	5 (31.3%)
Progression	4 (25%)
Type of VTE/AT episode	
Pulmonary embolism	4 (25%)
Deep vein thrombosis	5 (31.3%)
Catheter-related thrombosis	2 (12.5%)
Visceral thrombosis	3 (18.8%)
Mixed event (venous and arterial)	1 (6.3%)
Other forms of VTE	1 (6.3%)
Mode of presentation of VTE/AT	
Incidental	10 (62.5%)
Symptomatic	6 (37.5%)
Management setting for VTE/AT	
Outpatient	13 (81.3%)
Hospitalization	3 (18.8%)
VTE/AT treatment	
No anticoagulation	3 (18.8%)
Low molecular weight heparin	13 (81.3%)
Discontinuation of iPARP after VTE/AT	
No	12 (75%)
Yes	4 (25%)
Recurrent thrombosis after initial VTE/AT diagnosis	
No	16 (100%)
Yes	0 (0%)
Hemorrhage after initial VTE/AT diagnosis	
No	16 (100%)
Yes	0 (0%)

ECOG PS Eastern Cooperative Oncology Group Performance Status, *iPARP* poly(ADP-ribose) polymerase inhibitor, *VTE/AT* venous and arterial thromboembolic events

Objective

The primary objective was to calculate the incidence of thrombosis associated with iPARP. Two secondary objectives were defined. The first was to examine the impact of thrombosis on survival among subjects treated with iPARP, while the second was to find predictor variables for the development of VTE/AT.

Statistical analysis

Median and interquartile range (IQR) 25–75 were used to describe quantitative characteristics. Qualitative characteristics were reported by number (n) and percentage (%). Survival analysis was performed using the Kaplan–Meier estimator and log-rank test, calculating the median and 95% confidence intervals (CI) of survival times. To determine predictor variables, multivariate logistic regression models were performed to obtain odds ratios (OR) and 95% CI. Statistical significance was set at a p value of 0.05 and the SPSS 25.0 statistical package (IBM Corporation, Armonk, NY, USA) was used.

Ethics

This study was submitted to the Ethics Committee of each participating center and obtained the corresponding approval prior to its commencement. The processing, communication, and transfer of all personal data complied with the provisions of Organic Law 15/1999, dated December 13, 1999, regarding the protection of personal data and of Organic Law 3/2018, dated December 5, 2018, since it came into force.

Results

Table 1 presents the baseline clinical and molecular characteristics of 329 patients with ovarian cancer treated with iPARP, stratified by the occurrence of VTE/AT. Among these, 16 patients (4.9%) developed VTE/ATE during follow-up, while 313 (95.1%) did not.

The median age at the start of treatment was similar between groups [60 years (IQR 57–68) in the VTE/AT group vs. 62 years (IQR 55–71) in the non-VTE/ATE group; $p = 0.78$]. No statistically significant differences were observed in ECOG performance status, disease stage, Khorana risk score, or oncologic status at the initiation of PARP inhibitors. Homologous recombination deficiency (HRD) was more prevalent among patients who developed VTE/AT (75.0% vs. 49.8%) although this difference did not reach statistical significance ($p = 0.114$).

A statistically significant difference was observed in BRCA mutation status ($p = 0.006$), driven primarily by an overrepresentation of BRCA2 mutations in the VTE/AT group (56.3% vs. 19.2%). No significant differences were

Table 3 Multivariate analysis of predictive factors for VTE/AT events in patients with ovarian cancer and iPARP

Variable	OR (Exp b)	95% CI Lower	95% CI Upper	*p* value
Tumor stage at initiation of iPARP	0.500	0.155	1.611	0.246
ECOG performance status	0.806	0.335	1.944	0.632
Oncologic status at initiation of iPARP	0.791	0.397	1.577	0.506
HRD	0.562	0.182	1.735	0.316
Khorana score	0.511	0.196	1.336	0.171
Systemic treatment modality	1.005	0.511	1.977	0.989
Type of iPARP	1.333	0.449	3.958	0.605
Combined treatment (iPARP + bevacizumab)	0.262	0.095	0.724	0.010

CI confidence interval, *ECOG* Eastern Cooperative Oncology Group, *HRD* homologous recombination deficiency, *iPARP* poly(ADP-ribose) polymerase inhibitors, *OR* odds ratio

identified in BRCA mutation type, treatment setting, or type of iPARP administered. Concomitant treatments were infrequent, but patients with VTE/AT had a higher rate of non-bevacizumab combination therapy (12.6% vs. 0.3%, $p < 0.001$).

Table 2 summarizes the clinical characteristics and management of VTE/AT in patients with ovarian cancer treated with iPARP. Most patients presented with an ECOG performance status of 0 or 1 at the time of diagnosis. Regarding oncologic response, 31.3% of the patients were in stable disease and 25% in partial response. The most common types of events were deep vein thrombosis (DVT) (31.3%) and pulmonary embolism (PE) (25%), followed by visceral thrombosis and catheter-related thrombosis. A majority of events were diagnosed incidentally (62.5%) and managed in the outpatient setting (81.3%). Most patients received low molecular weight heparin (LMWH) as treatment, while 18.8% did not receive anticoagulation. The iPARP was discontinued in 25% of the cases. No patients experienced recurrent thrombosis or hemorrhagic events during follow-up.

Table 3 and Fig. 1 present the results of a multivariate logistic regression analysis evaluating predictive factors for the development of venous and arterial thromboembolic events (VTE/AT) in patients with ovarian cancer treated with iPARP. No statistically significant associations were found for tumor stage, ECOG performance status, oncologic status at treatment initiation, HRD status, Khorana score, systemic treatment modality, or the specific type of iPARP. However, combined treatment with iPARP and bevacizumab was significantly associated with a decreased risk of VTE/AT (OR: 0.262; 95% CI 0.095–0.724; $p = 0.010$).

A Kaplan–Meier survival analysis was conducted to evaluate the impact of VTE/AT on overall survival (OS) in patients with ovarian cancer treated with iPARP inhibitors

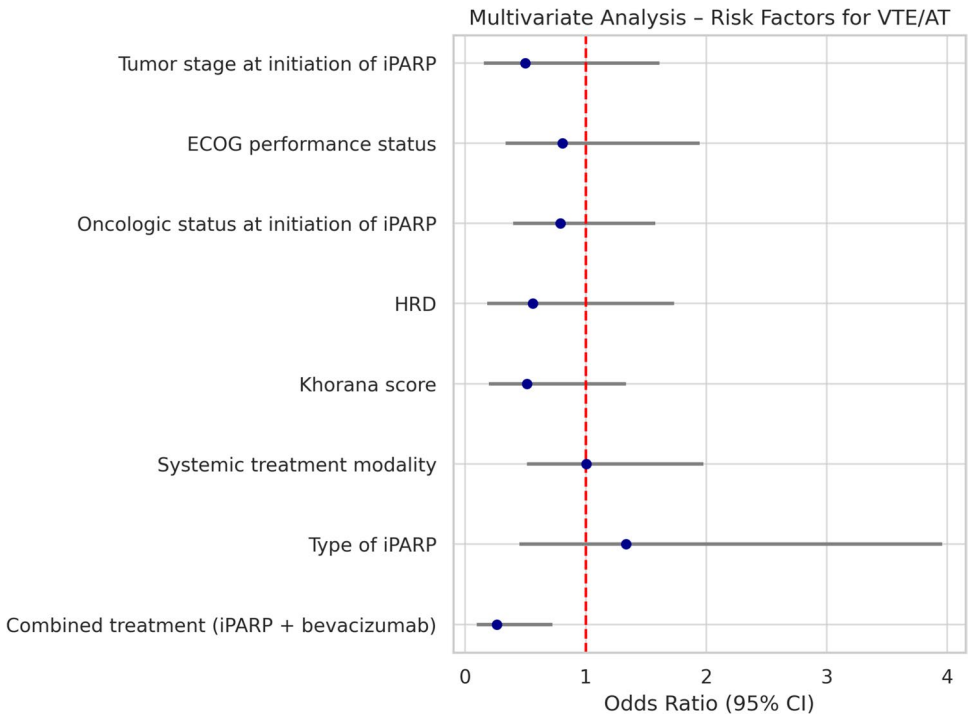

Fig. 1 Forest plot of predictive factors for VTE/AT events in patients with ovarian cancer and iPARP. CI: confidence interval; ECOG: Eastern Cooperative Oncology Group; HRD: homologous recombination deficiency; iPARP: poly(ADP-ribose) polymerase inhibitors

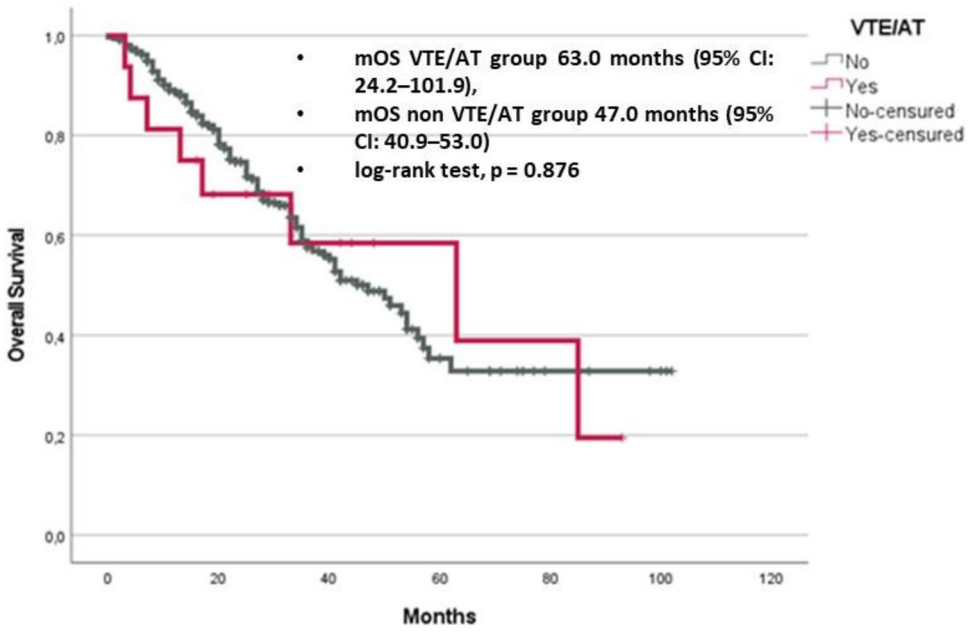

Fig. 2 Survival analysis: Kaplan–Meier curve comparing OS (since initiation iPARP) of ovarian cancer patients treated with iPARP who developed VTE/AT versus those who did not. CI: confidence interval; mOS: median overall survival; VTE/AT: venous and arterial thromboembolic events

(Fig. 2). The median OS in the VTE/AT group was 63.0 months (95% CI: 24.2–101.9), compared to 47.0 months (95% CI: 40.9–53.0) in the group without thromboembolic events. Although the median survival appeared numerically longer in patients who developed VTE/AT, the difference was not statistically significant (log-rank test, $p = 0.876$).

Discussion

Our multicenter retrospective analysis provides valuable real-world evidence regarding the incidence of VTE/AT in ovarian cancer patients undergoing treatment with iPARP. Currently, the available data in the scientific literature on thrombosis associated with iPARP in this population derive exclusively from the pivotal clinical trials that led to the approval of these agents [2–5, 7–11]. In those trials, the reported incidence of VTE/AT ranged from 1 to 3%.

Our findings revealed a VTE/AT incidence of 4.9%, which is consistent with previously reported rates in clinical trials, albeit slightly higher—possibly reflecting differences in patient selection criteria or intensity of follow-up in real-world settings.

An exception to these figures is the PAOLA-1/ENGOT-OV25 trial [6], in which a 6.2% incidence of VTE/AT was reported in the olaparib plus bevacizumab arm, with all events being venous in nature, compared to 3.3% in the bevacizumab-only group, where arterial events predominated.

This finding is of particular interest, as it not only suggests a higher incidence of thromboembolic events in patients receiving bevacizumab, but also a possible shift in the anatomical distribution of the events. However, in our series, combined therapy with iPARP and bevacizumab was associated with a significantly lower risk of thrombosis (OR: 0.262; 95% CI: 0.095–0.724; $p = 0.010$).

We were unable to find a clear explanation for this discrepancy. Probably, the fact that the sample includes only 11 patients treated with bevacizumab (3.3%), none of whom developed VTE/AT, may represent a bias that could account for the results observed. Prior studies, such as that by Carmona-Bayonas et al. using data from the TESEO registry and the CARAVAGGIO trial, reported that antiangiogenic agents are associated with a higher proportion of pulmonary embolism in oncological patients with VTE [12].

Moreover, a meta-analysis conducted by Saerens et al. in patients with ovarian cancer receiving bevacizumab found that the incidence of VTE was reported in nine trials involving 5,121 patients. The absolute risk of VTE was 5.4% in patients treated with bevacizumab compared to 3.7% in those not receiving it (RR: 1.32; 95% CI: 1.02–1.79, $p = 0.04$). The analysis concluded that bevacizumab increases the risk of both arterial and venous thromboembolic events [13].

In addition, it is noteworthy that patients with BRCA2 mutations experienced a higher frequency of thromboembolic events in our cohort. This observation aligns with findings reported by Pérez-Segura et al., who concluded that the presence of BRCA2 mutations is associated with increased plasma levels of thrombosis-related proteins [14].

Conversely, Muñoz AJ et al. conducted a study aiming to assess the incidence of VTE in patients with germline BRCA mutations. According to their results, the risk of VTE

in this population appears to be driven by tumor type, with no significant interaction between germline BRCA mutation status and cancer-associated thrombosis [15].

It is worth noting the high incidence of BRCA1 and BRCA2 mutations in our cohort, with rates of 22.5% and 21%, respectively. These figures appear slightly higher than those reported in the literature, where the combined prevalence is typically estimated at around 20–25% [16]. A possible explanation for this observation may lie in the limited use of iPARP—depending on institutional policies—in patients with homologous recombination proficient (HRP) tumors (i.e., without BRCA mutations or homologous recombination repair defects), particularly as first-line maintenance therapy in advanced ovarian cancer (representing 45.4% of our total cohort). This may have enriched our study population with patients carrying BRCA mutations (43.5% overall).

Given the statistically significant association between BRCA2 mutations and an increased incidence of thromboembolic events, this overrepresentation could have contributed to an amplification of the overall VTE/AT incidence observed in our cohort.

A strength of our study is that, to our knowledge, it is the first to specifically analyze thromboembolic events in ovarian cancer patients receiving iPARP in routine clinical practice. Our literature review identified only one relevant meta-analysis (limited to phase III trials including all solid tumors treated with iPARP), which did not suggest an increased risk of VTE/AT associated with iPARP use [17].

However, another meta-analysis [18] focusing primarily on prostate cancer patients treated with iPARP reported that this drug class is associated with an increased risk of thromboembolic events (OR 1.98; 95% CI 1.06–3.70; $p = 0.030$). The findings from that analysis, together with our results—suggesting a protective role of bevacizumab and a higher prevalence of BRCA2 mutations in patients who developed VTE/AT—may provide a rationale for developing personalized thromboprophylaxis protocols in patients receiving iPARP, both in the general oncologic population and more specifically in those with ovarian cancer.

The main limitation of our study relates to the aforementioned discordance in findings regarding the potential protective effect of bevacizumab and the prothrombotic role that BRCA2 mutations might play. Given the inconsistency of the current evidence, we believe it is prudent to conduct additional studies in real-world patient populations to confirm or refute these observations. Increasing the sample size and conducting prospective analyses may help clarify these uncertainties. Nonetheless, despite these limitations, our findings show that VTE/AT did not significantly impact OS, suggesting that these events may not substantially affect prognosis in ovarian cancer patients treated with iPARP.

Conclusions

In patients with ovarian cancer treated with iPARP, the overall incidence of VTE/AT was moderate (4.9%), with significant associations identified for BRCA2 mutations. Combined treatment with bevacizumab appears to reduce thrombotic risk (however, this result may be biased due to the low number of patients receiving iPARP in combination with bevacizumab included in the study). These findings highlight the importance of individualized thrombotic risk assessment in ovarian cancer patients undergoing iPARP therapy.

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Data availability The authors declare the availability of data analyzed in this study.

Declarations

Conflict of interest The authors have no conflicts of interest related to the development of this research project.

Ethical approval This study was submitted to the Ethics Committee of each participating center and obtained the corresponding approval prior to its commencement. The processing, communication, and transfer of all personal data complied with the provisions of Organic Law 15/1999, dated December 13, 1999, regarding the protection of personal data and of Organic Law 3/2018, dated December 5, 2018, since it came into force.

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