

pathogenic variant carriers, respectively. The origin of PC is yet unknown. However, as the origin of ovarian cancer probably lies in the Fallopian tube, the Serous Tubal Intraepithelial Carcinoma (STIC) may be the origin of PC as well. In this Individual Patient Data Meta-Analysis (IPDMA), we determine the risk of PC for *BRCA1/2* pathogenic variant carriers with and without STIC at RRSO.

Methodology We performed a systematic search of MEDLINE, EMBASE and Cochrane on studies providing follow-up in *BRCA*-mutation carriers after RRSO. Individual patient data was extracted and the authors of eligible studies were contacted to complete this data. Additionally, we retrospectively collected data from the Radboudumc (Nijmegen, the Netherlands), Kaiser Permanente (San Francisco, USA) and MD Anderson (Houston, USA) of *BRCA1/2* pathogenic variant carriers undergoing RRSO.

Result(s)* After screening, 15 out of 2.151 studies were included. Including the retrospective case series, individual patient data was available for 3.121 women without STIC and 115 women with STIC at RRSO. The median age (range) was 46 (24-80) of women without and 52 (36-77) for women with STIC. The hazard ratio to develop PC after RRSO was 29.3 for women with STIC compared to women without STIC at RRSO ($P < 0.001$) (figure 1). The five-year risk to develop PC was 0.4% (0.2%-0.7%) for women without STIC and 12.6% (5.2%-19.3%) for women with STIC. The respective ten-year risks are 0.9% (0.3-1.4) and 23.5% (10.6-34.5%). Additional sensitivity analyses did not alter the results.

Conclusion* From this IPDMA we conclude that *BRCA* pathogenic variant carriers with a STIC at RRSO are at increased risk to develop PC during follow-up. These results are important for clinical awareness and future research. The question arises whether a STIC should be considered as precursor or early stage ovarian cancer. Larger prospective-multicenter studies are needed to investigate the additional value of staging surgery and/or chemotherapy in case of STIC.

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SURVIVAL IN ADVANCED-STAGE OVARIAN CARCINOMA WITH HIGH VEGF-A EXPRESSION AFTER ADDITION OF BEVACIZUMAB. RESULTS FROM THE GERMAN ICON7 PATIENT COHORT

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Introduction/Background* It has previously been demonstrated that bevacizumab may differentially improve ovarian cancer survival. TCGA classification as well as molecular targets of anti-VEGF therapy were shown to be associated with different levels of bevacizumab efficacy. Translation into individualized treatment options or improved disease outcome was yet hindered by inconsistent results across trials and tissue types. It was the aim of this project to validate retrospective analyses of GOG-0218 derived predictive value of microvessel density (CD31) and tumor VEGF-A.

Methodology CD31 and VEGF-A immunohistochemistry was performed on whole section FFPE tissue samples from the AGO-OVAR11 (ICON7) trial. Patients were stratified into high and low biomarker-expressing subgroups using median cutoffs. The association between biomarker expression and bevacizumab therapy efficacy was evaluated using a proportional Cox regression model. Efficacy endpoints were progression free survival (PFS) and overall survival (OS).

Result(s)* Complete CD31 and VEGF-A immunohistochemical data were available from 387 patients of the German ICON7 trial cohort. Among all biomarker subgroups, only patients with high VEGF-A expression levels had a statistically significant benefit from the addition of bevacizumab to standard chemotherapy. Median PFS and OS of VEGF-A high patients was 23.1 months (95% CI 15.9-30.2) and 64.9 months (median not reached) respectively if bevacizumab was added to standard chemotherapy but only 14.3 (95% CI an 11.2-17) and 47.3 months (95% CI 5.6-36.3) in the control arm. In multivariable analysis, adjusted for age, FIGO and postoperative residual tumor, the anti-angiogenic therapy showed improved PFS (HR: 0.62 [95%CI 0.43-0.89], $p = 0.011$) and OS (HR 0.59 [95%CI 0.39-0.91], $p = 0.02$) among VEGF-A high patients. Patients with low VEGF-A expression levels showed no statistically significant improvement of PFS (HR 0.96 95% CI 0.67-1.38, $p = 0.83$) or OS (HR 1.06 95% CI 0.69-1.62, $p = 0.80$) after addition of bevacizumab. CD31 immunohistochemistry was not predictive for bevacizumab treatment effects in our cohort.

Conclusion* A potential predictive value of VEGF-A expression levels was observed in advanced stage ovarian carcinoma patients from the German ICON7 patient cohort, partly confirming GOG-0218 derived findings. Our results may help to develop biomarker stratified anti-VEGF therapy and hold potential to promote personalized treatment strategies in ovarian carcinoma patients.

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REAL-WORLD OF OVARIAN CANCER TREATMENT OUTCOMES IN NORTHERN PORTUGAL

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Introduction/Background* Epithelial ovarian cancer (EOC) is the fifth most lethal cancer in women in Portugal. Despite advances in surgical and anti-cancer systemic treatment (SACT), EOC overall prognosis remains poor. The objective of this study is to describe SACT outcomes patterns, including target therapies (bevacizumab and PARPi) using real-world data from a comprehensive cancer center, which serves Northern Portugal population.