Abstracts

264 SERUM MARKERS AND CYTOKINES IN PATIENTS WITH OVARIAN CANCER, ENDOMETRIOSIS OR OTHER BENIGN OVARIAN TUMOURS

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Introduction/Background The gold standard of serum tumour markers to detect ovarian cancer is cancer antigen 125 (CA125), human epididymis protein-4 (HE-4) and risk of ovarian malignancy algorithm (ROMA). However there is still need to improve its accuracy. Cytokines play a crucial role in tumour growth and progression according to proangiogenic and immunosuppressive activity. The aim of this study was to investigate the potential use of serum levels of selected cytokines in preoperative diagnosing of adnexal mass.

Methodology The study group consisted of 120 patients: 35 with epithelial ovarian cancer (EOC) and 85 with benign ovarian tumours (24 teratomas, 27 endometriotic and 34 other epithelial). We measured in sera obtained preoperatively the level of CA125, HE-4 and the panel of 6 cytokines: interleukin (IL) 1β, 6, 8, 10, 12, tumour necrosis factor (TNF) using cytometric bead array (CBA) and one chemokine CXCL1/GRO-α by ELISA method.

Results Serum levels of IL-6, IL-8, IL-10 and CXCL1/GRO-α were significantly higher in patients with ovarian cancer (2045 pg/ml; 208; 32; 356 pg/ml, respectively) than in women with benign ovarian tumours (17 pg/ml; 29; 16; 127 pg/ml, respectively). The similar pattern was present with standard ovarian cancer markers – CA125 (959 vs 43 U/ml) and HE-4 (534 vs 51 pmol/l). Other investigated cytokines had similar levels in all groups of patients. Analyzing the differences in the subgroups of women with benign ovarian tumours we didn’t observe any significant except CA125 and IL-8; they were slightly elevated in cases of endometriotic ovarian cysts.

Conclusion Proinflammatory cytokines (IL-6, IL-8), immunosuppressive (IL-10) and CXCL1/GRO-α were elevated in sera of EOC patients what points on their role in cancer development. Moreover, they might be useful in preoperative differential diagnosis of ovarian tumors, as a supplemental markers especially as they were not elevated in cases of endometriosis.

Disclosures All authors report no conflict of interest.

281 REAL-WORLD-DATA ON PLATINUM OUTCOMES AFTER PARP INHIBITORS PROGRESSION IN HIGH GRADE SEROUS OVARIAN CANCER PATIENTS

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Conclusion Although there were differences in demographics and treatment patterns, advanced-stage EOC had poor prognosis and relatively short progression-free-intervals across Asia-Pacific countries with well developed healthcare systems, highlighting the need to develop novel approaches to improve patient outcomes. Variation in the germline BRCA1/2 mutation rates across three datasets is probably due to differences in the composition of the contributing registries (clinic or cohort-based).

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Introduction/Background PARP inhibitors (PARPi) maintenance after Platinum (Pt) based chemotherapy (CT) significantly improves progression-free survival (PARPi-PFS) and PFS after subsequent (ssq) CT (PARPi-PFS2). Data regarding ssq CT is scarce, and PARPi/Pt crossed mechanisms of resistance may impact on outcome of ssq Pt. We provide Real-world-data on this issue.

Methodology
We included HGSOC p treated with ssq CT after progression to maintenance PARPi until 15th Jul 2020 in 3 hospitals. Endpoints were related to this ssq CT: objective response rate (ORR), median(m) progression-free survival (PFS) and overall survival (OS). Multivariate Cox and logistic regression models were adjusted by BRCA status and Pt-free interval (PFI) (6–12 months (mo) vs ≥12 mo). Adjusted hazard ratios (aHR) and odds ratios (aOR) of the risk of progression/death and ORR, respectively, were reported with 95% confidence intervals (CI).

Results
56p were identified (32p BRCAmut; 1p BRIP1mut). 4p (7.1%) received PARPi after 1st line CT, 26 (46.4%) after 2nd line and 26 (46.4%) after ≥3rd line. 34p (60.7%) received olaparib and 22 (39.3%) niraparib. m-PARPi-PFS in the recurrent setting was 7.5 mo (longer in BRCAmut: 10.1 vs 5.5 mo, p 0.03). m-PARPi-PFS2 was 15.8 mo (longer in BRCAmut: 20.9 vs 15.4 mo, p 0.07).

Endpoints regarding ssq CT are shown in table1. ORR to ssq Pt was 33.3% and progression disease without any response 28.6%. ORR in p who received ssq Pt-free CT, ssq Pt with PFI 6–12 mo, and ssq Pt with PFI≥12 mo were 33.3%, 23.8% and 42.8%, respectively. Five complete responses occurred among BRCA mut that had received PARPi in the recurrent setting. mPFS and mOS were significantly longer in the PFI≥12 subgroup vs the others (figure1).

Focusing in p receiving ssq Pt, when adjusting by BRCA status: aOR of ORR in p with PFI≥12 vs 6–12 mo was 0.56 (95% CI: 0.13 – 2.30), aHR of mPFS between these two subgroups was 0.61 (95% CI: 0.30–1.20; p 0.16), and aHR of mOS was 0.20 (95% CI: 0.7–0.61; p 0.005). Results did not change when excluding the 4p who received PARPi as 1st line.

Conclusion A trend towards higher benefit from ssq Pt after PARPi was observed in the PFI≥12 subgroup. Benefit from ssq Pt after PARPi in the PFI 6–12 subgroup was similar to benefit from CT in the non-Pt subgroup. The role of ssq Pt after PARPi in the PFI 6–12 subgroup warrants further research.

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