Methods Patients who underwent second-look surgery either with or without consolidation HIPEC after having a complete or partial response to primary cytoreductive surgery and adjuvant platinum-based chemotherapy between January 1991 and December 2003 were identified. The 10-year progression-free survival (PFS), overall survival (OS), and toxicity within post-operative 28 days were investigated.

Results The 10-year PFS and OS were significantly longer in the HIPEC group compared with the control group (PFS, 53.6% vs. 34.9%, p = 0.009; OS, 57.0% vs. 34.5%, p = 0.025). In a subgroup of patients with stage III, the HIPEC group showed significantly longer 10-year PFS and OS compared with the control group (PFS, 42.6% vs. 14.8%, log-rank p < 0.001; OS, 46.7% vs. 19.6%, p = 0.036). Patients who underwent HIPEC with paclitaxel showed a longer PFS and OS trend compared with subjects who underwent HIPEC with carboplatin. The more common adverse events in the HIPEC group were thrombocytopenia, elevated liver enzymes, and wound complications.

Conclusions The consolidation HIPEC demonstrated a significant improvement in 10-year PFS and OS with acceptable toxicity in patients with primary epithelial ovarian cancer. Further randomized controlled trials are warranted to confirm these results.

**EP211/#794** OPTIMUM SELECTION CRITERIA FOR SECONDARY CYTOREDUCTIVE SURGERY IN PATIENTS WITH RECURRENT OVARIAN CANCER: A MULTICENTER STUDY

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Objectives Optimum selection criteria for secondary cytoreductive surgery (SCS) in recurrent ovarian cancer is often dependent on the multiple confounding factors. This study aimed to evaluate the survival outcomes of recurrent ovarian cancer and investigated the factors identifying patients most likely benefit from the SCS.

Methods We retrospectively reviewed medical records of recurrent ovarian cancer patients from 5 referral hospitals in Korea from 2010 to 2021. Recurrent characteristics, treatment methods and potential factors for survivals were evaluated between the chemotherapy and surgery groups.

Results A total of 670 patients with recurrent ovarian cancer were identified. The patient’s median age was 55 (24–83) and 88.1% of patients had initial stage III/IV disease. Of all patients, 215 (32.1%) patients received SCS for the disease recurrence and others received 2nd line chemotherapy. The median survival was 85 months (95% CI, 65.0 – 105.0) in chemotherapy group and the median survival time was not reached in SCS group (p<0.001). Among the patients received SCS, only patients received complete resection showed improved survival. Patients with any gross residual disease after SCS had no survival benefit compared to patients received chemotherapy (p=0.942). In multivariate cox analysis, residual disease at primary surgery, PFI, recurrent sites, ascites and SCS was significant prognostic factors for the survival. Meanwhile, predicting factor for complete resection after SCS was only recurrent sites (≤3 lesions or regional carcinomatosis, P<0.001).

Conclusions Platinum-sensitive recurrence with limited regional diseases (< 3 regions or limited carcinomatosis without ascites) can be considered as optimum criteria for SCS in recurrent ovarian cancer.

**EP212/#1141** CLINICAL AND GENOMIC LANDSCAPE OF OVARIAN CLEAR CELL CARCINOMA

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Objectives The genetic landscape of Ovarian clear cell carcinoma (OCCC) is poorly described. We sought to identify genomic characterization of OCCC and correlate findings with clinical outcomes.

Methods We performed a multi-center prospective clinical sequencing program of OCCC patients (n=95) using tumor-normal massively parallel sequencing that included 688 cancer-related genes, and comprehensively analyze the clinical and genomic characteristics of OCCC.

Results In the 95 samples, the most frequently mutated genes were ARID1A(61.1%), PIK3CA(61.1%), TP53(24.2%), MUC16(22.1%), KMT2C(20%), KMT2C, MECOM, SMARCA4, PDGFRB and CDC27 were significantly related to platinum resistance (P<0.05). The Progression-free survival (PFS) was shorter among patients with tumors harboring ARID2, CDK2, CUL4A, DAXX and DDR1 mutations (P<0.05) compared to patients without these mutations. The overall survival (OS) was significantly shorter among patients harboring CASP8, IDH2, LZTR1, MDM4 and PI3KR2 mutations (P<0.05). The OS was longer among patients with tumors harboring RYR2 (P<0.05) and driver gene POLE (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations. Patients with POLE mutation showed extremely high TMB. An increasing trend of CD8+ cytotoxic T lymphocytes (CTL) (P<0.05) mutations.