Conclusion/Implications It is feasible for chemotherapy completion in older adults with EOC. Age only is not the determinant of chemotherapy completion. Comorbidity and disease status are crucial in determining chemotherapy discontinuation.

Abstracts

PR068/#140 PREVALENCE AND EFFECT OF MALNUTRITION ON SURGICAL AND ONCOLGICAL OUTCOMES IN ADVANCED OVARIAN CANCER PATIENTS
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Introduction In patients with advanced ovarian cancer, malnutrition is a significant concern. It might be associated with poor treatment outcomes. This study aims to determine the prevalence of malnutrition in advanced ovarian cancer patients and investigate the effect of malnutrition on surgical and oncological outcomes for the disease.

Methods 290 advanced ovarian cancer patients (FIGO stage 3–4) who were never diagnosed as ‘malnutrition’ for another reason were enrolled in the study. We determined malnutrition status using the geriatric nutritional risk index (GNRI). Information derived from medical records was gathered, including BMI, treatment complications, and length of hospital stay.

Results This study showed 137 of 290 patients (47.2%) have malnutrition. Anemia and CKD were present concomitant with malnutrition. Malnutrition impacts both surgical and oncological outcomes, including the percentage of optimal surgeries (35.8% in the malnutrition group and 62.7% in the well-nourished group) and the length of hospital stays (malnourished patients stayed longer than the average by three days). It was also found that well-nourished patients had a higher overall survival rate of more than 12.97 months than those with malnutrition patients.

Conclusion/Implications Advanced ovarian cancer patients frequently had malnutrition. Malnutrition reduces optimal surgery rate, may lengthen hospital stays, and may reduce overall survival rates.

PR069/#709 PROGNOSTIC IMPACT OF ERYTHROPOIETIN-STIMULATING AGENTS DURING FRONT-LINE CHEMOTHERAPY IN PATIENTS WITH OVARIAN CANCER: A KOREAN MULTICENTER COHORT STUDY
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Introduction To evaluate whether erythropoiesis-stimulating agents (ESAs) treatment for chemotherapy-induced anemia (CIA) affect progression-free survival (PFS) in patients who received front-line chemotherapy following surgery for ovarian cancer.

Methods We retrospectively reviewed all consecutive patients who received front-line chemotherapy after surgery during 2013–2019 from seven institutions. Patients were divided according to the use of ESA during front-line chemotherapy. Primary endpoint was PFS. The secondary endpoint included occurrence of thromboembolism. A propensity score matching (PSM) analysis was used to compare survival in matched cohorts.

Results Overall, 2,147 patients (433 for ESA and 1,714 for No-ESA) were identified with median follow-up of 44.0 months. ESA group showed significantly higher proportion of stage III/IV disease (81.8% vs 61.1%; P<0.001) and postoperative gross residual (32.3% vs 21.2%; P<0.001) compared to No-ESA group. In multivariable Cox regression, use of ESA did not affect PFS (adjusted hazard ratio, 1.034; 95% confidence interval [CI], 0.891–1.201; P=0.661). The incidence of thromboembolism was 10.2% in the ESA group and 4.6% in the No-ESA group (adjusted odds ratio, 6.581; 95% CI, 3.261–13.281; P<0.001). When comparing the well-matched cohorts after PSM, PFS did not differ between the ESA (median PFS 38 months, range 0.1–77.2 months) and No-ESA group (median PFS 35 months, range 2.2–81.2 months)(P = 0.13, log rank test).

Conclusion/Implications Use of ESA during front-line chemotherapy did not negatively affect PFS in patients with ovarian cancer after surgery but increased risk of thromboembolism.

PR070/#391 PHASE I TRIAL OF TILVISTAMAB, A FUNCTION-BLOCKING ANTIBODY INHIBITING AXL, IN PLATINUM-RESISTANT/REFRACTORY HIGH GRADE SEROUS OVARIAN CANCER (PROC)
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Introduction AXL expression in PROC is associated with a poor prognosis, a mesenchymal (Mes) gene expression molecular subtype (GEMS) and resistance to platinum chemotherapy. Pre-clinically, the function-blocking antibody tilvestamab binds and inhibits AXL tyrosine kinase, reducing AXL expression and downstream signalling.