

included. Following consultation with a desensitization specialist, patients were premedicated for 3 days (prednisone 40 mg, montelukast 10 mg) and immediately prior to carboplatin (dexamethasone, antihistamine-1 and antihistamine-2 antagonists). Carboplatin was administered in 12 steps under dedicated nursing supervision: Bag1 (1% dose), Bag2 (2.5% of dose), Bag3 (96.5% of dose) were each given in 4 incremental steps. Planned infusion time for steps 1–11 was 15 minutes/step and step 12 was administered at 75ml/hour.

**Results** 30 patients received carboplatin desensitization between 12/2016–01/2019. During their prior HSR 5/30 (16%) had required epinephrine. 19/30 (63%) were seen by an allergist prior to desensitization. 24/30 (80%) received  $\geq 2$  desensitization cycles with median of 3 (range 1–8). During desensitization 11/30 (37%) had breakthrough HSR; 9 of these 11 (81%) were able to receive additional cycles. 2/30 (7%) required epinephrine with 1 patient (3%) transferred to urgent care. No patient required admission for HSR. Reasons for treatment discontinuation were: completed planned treatment (12/30, 40%), disease progression (11/30, 37%), and HSR (5/30, 17%). Median time in chemo unit was 504 minutes (range 335–630).

**Conclusions** 37% had breakthrough HSR despite the 12-step desensitization; however, the majority was able to receive additional platinum desensitization. Our data suggest that outpatient carboplatin desensitization is feasible but repeated HSR can occur. Dedicated nursing care and access to desensitization specialists are required.

## IGCS19-0341

### 318 CORRELATION OF LYMPHOVASCULAR SPACE INVASION AND INVASIVE CIRCULATING TUMOR CELLS IN PATIENTS WITH EPITHELIAL OVARIAN CANCER

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**Objectives** The detection of circulating (CTCs) and invasive circulating tumor cells (iCTCs) in the peripheral blood of women with epithelial ovarian cancer (EOC) has been proven to be feasible and prognostic. The deleterious impact of lymphovascular space invasion (LVSI) has been well-established in various gynecologic malignancies (e.g., vulvar, cervical, endometrial) but has not been extensively evaluated in EOC. The goal of this study is to evaluate the correlation between CTCs, iCTCs and LVSI in patients with EOC.

**Methods** Peripheral blood samples from 85 patients with EOC were assessed for the presence of CTCs and iCTCs using our functional cell adhesion matrix (CAM) enrichment method. The histopathology slides from each patient were reviewed by two gynecologic oncology pathologists for histologic type, grade, presence or absence of LVSI, extent of the LVSI (focal or multifocal) and location (organ site).

**Results** High levels of CTCs and iCTCs were significantly associated with advanced stage but not with grade, debulking status, platinum sensitivity, lymphovascular space invasion, age, or overall survival. High levels of CTCs and iCTCs were positively correlated. Lymphovascular space invasion was significantly associated with decreased overall survival (median: 1194 vs. 2034 days,  $p=0.02$ ) but not with stage, grade, debulking status, platinum sensitivity, median or high levels of CTCs or iCTCs, or age.

**Conclusions** Lymphovascular space invasion is an independent risk factor for women with EOC, but was not associated with levels of circulating tumor cells. These findings suggest that these two circulations have distinct mechanisms by which they contribute to spread of ovarian cancer.

## IGCS19-0100

### 319 RARE OVARIAN CANCER PRESENTATION

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**Objectives** To report an intestinal lymphangitis carcinomatosa related to ovary cancer.

**Methods** The information has been obtained through review of medical records and review of the literature.

**Results** A 57-year-old virgin, deaf-mute patient with cognitive deficit was referred to our hospital with left ovary neoplasia. Abdominal CT showed a 3.5 cm diameter mixed lesion in the left ovary. CA-125 was 74.55. Videolaparoscopy showed lymphatic fluid in the abdominal cavity, lesions in epiploon compatible with metastatic implants. Left oophorectomy was performed. Frozen section revealed undifferentiated malignant neoplasia, not being able to rule out lymphoma. Pathology confirmed the hypothesis of high grade serous ovarian carcinoma. She was readmitted in 2 weeks later with clinical signs of high bowel obstruction. Abdominal CT demonstrated proximal jejunal obstruction. Exploratory laparotomy showed a thickened area of 10 cm in the proximal jejunum, with stenotic enteric lumen, with lesion apparently originating from its submucosal and muscular layers without implants in serosa. Proximal enterectomy, panhysterectomy, omentectomy, and resection of peritoneal implants in hepatic round ligament and bladder were performed. Pathology revealed jejunal stenosis due to an infiltrating tumor, lengthening 7.2 cm. Microscopy showed lymphatic tumoral emboli in mucosa, submucosa and muscularis propria. No serosal involvement was identified. Immunohistochemistry was compatible with metastatic high grade serous ovarian carcinoma.

**Conclusions** Our report demonstrates an unusual pattern of metastasis: embolization of intestinal lymphatic vessels, with subsequent stromal invasion. To the best of our knowledge, this is the first report in the literature of intestinal carcinomatous lymphangitis related to ovary cancer.