**Introduction/Background**

Ovarian cancer ascites is one of the signs of ovarian cancer (OC) metastization in the peritoneum and is found in patients at the time of diagnosis and in disease relapse. This serous fluid is used for the initial cytopathological diagnosis and is frequently drained from the peritoneal cavity at advanced stages only for symptomatic relief (and is discarded). In the ovarian cancer context, the presence of ascites is a unique opportunity to monitor the tumor kinetics during disease progression without additional invasive procedures. The main aim of this work was to evaluate the potential of this usually discarded biological material to evaluate the expression of proteins associated with therapy resistance in ovarian cancer ascites cells during disease progression.

**Methodology**

We received ascites from OC patients at diagnosis (n=7) and during treatment (n=8). After centrifugation, samples were formalin-fixed and embedded in Histogel before standard histological processing. Next, immunocytochemistry was performed to assess the expression of three biomarkers associated with chemoresistance in ovarian cancer (ALDH1, SOX2, and PgP).

**Results**

The majority of our samples had a sufficient number of cells to perform a diversity of histological-based techniques and other molecular studies. The expression of cancer stem cell markers ALDH1 and SOX2 was frequently negative. However, SOX2 and ALDH1 were expressed in samples obtained after chemotherapy. Multi-drug resistance marker (PgP) expression was negative in samples at diagnosis but was found positive, especially in samples from patients with refractory ascites and without clinical response to treatment.

**Conclusion**

The multidimensional potential ovarian cancer ascites as a spontaneous ‘liquid biopsy’ remains underexplored. Our results show that the use of immunocytochemistry to evaluate resistance biomarkers in tumor cells present in ascites drained from patients during treatment has the potential to predict response to treatment.