infiltrative. In this study, we investigated the clinical implications of their histological subtypes on survival outcomes.

**Methods** We identified patients with PMOC who had undergone primary surgery between 2003 and 2021. Patients with other types of ovarian cancer or severe comorbidities were excluded. We collected patients’ baseline characteristics, surgical details, and pathological information. Progression-free survival and overall survival were calculated, while prognostic factors were also investigated.

**Results** We included 131 patients in total. The median age was 50 years, and 103 (78.6%) patients had stage I disease. During 55.9 months of median follow-up, there were 27 recurrences and 20 deaths. Among them, 113 patients were classified into 87 (77%) expansile and 26 (23%) infiltrative subtypes after the slide review. Advanced stage, lymph node involvement, and residual tumors after surgery were more common in the infiltrative subtype. The infiltrative group showed worse 5-year progression-free and overall survival rates (figure 1). In multivariate analyses, advanced stage and residual tumor after surgery were associated with worse prognosis, while the infiltrative subtype showed no statistical significance (table 1). In the subgroup analysis of stage I disease, there was no difference in survival between the two groups.

**Conclusion/Implications** In PMOC, the infiltrative histological subtype showed worse prognosis than the expansile subtype, with a higher proportion of advanced-stage tumors. However, it remains uncertain whether infiltrative subtype is an independent prognostic factor.

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**OSBP10-APOE PATHWAY INCREASES RESISTANCE OF PLATINUM AND PARPi BY REGULATING CHOLESTEROL METABOLISM AND DNA DAMAGE REPAIR IN PDX MODEL OF HRD-POSITIVE HGSOC**

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**Introduction** Chemoresistance is a key factor limiting the cure rate of ovarian cancer. Cancer-associated fibroblasts (CAFs) have been shown to be actively involved in cancer progression and chemoresistance. Intratumoral inflammatory environments affect many therapeutic responses, but the underlying mechanisms by which CAF participates in chemoresistance by modulating the tumor’s inflammatory environment are largely unknown.

**Methods** We cultured primary ovarian cancer-associated fibroblasts, using indirect co-culture to study how ovarian cancer cells activate CAF to participate in ovarian cancer platinum-resistant. WB shows activation of the cGAS-STING pathway in CAF, and IHC shows differential expression of STING in cisplatin-sensitive/resistant patients. Cell viability or apoptosis using CCK8 or apoptosis kits, respectively. The STING antagonist H-151 and the STING agonist MSA-2 investigated the contribution of the cGAS-STING pathway to the chemoresistance of ovarian cancer.

**Results** After treatment with cisplatin for ovarian cancer cells, the supernatant was indirectly co-cultured with CAFs, and mRNA sequencing showed that the cGAS-STING-IFNB1 pathway was activated in CAFs. We found that IFNB1 can promote platinum resistance in cancer cells by inhibiting cisplatin-induced DNA damage and promoting HRR. Anti-IFNB1 restores platinum sensitivity. In addition, the expression of STING in the tumor stroma is associated with poor prognosis in patients. In vivo experiments showed that inhibition of STING expression could restore platinum sensitivity, while activating STING could not significantly enhance immune killing ability.

**Conclusion/Implications** Our study shows that activation of the cGAS-STING pathway in CAFs is involved in platinum resistance in ovarian cancer, and STING inhibitors are able to restore ovarian cancer chemotherapy sensitivity.