associated with recurrence-free and disease specific survival ($p<0.05$) while FIGO 2018 stage I substages were not. In the current three-tiered system, disease specific survival for patients with pattern B tumors did not significantly differ from those with pattern C tumors while those with pattern A tumors did (table 2).

Conclusion/Implications These findings highlight the need for future prospective studies to further investigate the prognostic significance of stage I HPV-associated EAC substaging and the inclusion of the binary Silva pattern of invasion classification, which includes LVSI status, as a component of treatment recommendations.

PR050/#373 CLINICOPATHOLOGIC AND GENOMIC ANALYSIS OF UTERINE SEROUS CARCINOMAS ARISING FROM ENDOMETRIAL HYPERPLASIA

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Introduction Uterine serous carcinoma (USC) typically arises from atrophic endometrium but may be associated with hyperplasia in 5–10% of cases. We sought to identify USC with concurrent hyperplasia and define if i) they are molecularly related, and ii) USC associated with hyperplasia are genetically distinct from those without.

Methods Patients diagnosed with USC and hyperplasia on hysterectomy specimen between 1/2014 – 2/2021 were identified. Slides were reviewed by two gynecologic pathologists. Hyperplasia and carcinoma were microdissected separately and subjected to tumor-normal panel sequencing. Results were compared to atrophy-associated USC.

Results Of 291 USCs with clinical sequencing and slides available for review, 10 cases were identified (3%), and eight cases with sufficient tissue were included. Recurrently mutated genes included TP53 (100%), PIK3CA (50%), PPP2R1A (50%), ARID1A (38%), and PTEN (25%). In seven patients (87.5%), USC and hyperplasia were clonally related and shared multiple mutations, including TP53 in 4 (57%) cases. In one clonally related case, USC and TP53 wild-type hyperplasia shared 1 of 11 mutations (PIK3CA hotspot mutation) while being distinct at the copy number level. In the last case, USC and hyperplasia were unrelated at the genetic level, and the hyperplasia was TP53 wild-type. The prevalence of ARID1A mutations was higher in hyperplasia-associated USC compared to atrophy-associated USC (43% vs. 0%, $p=0.02$).

Conclusion/Implications Hyperplasia and USC were clonally related in most cases, commonly harboring TP53 hotspot mutations in both components. ARID1A mutations were more prevalent in hyperplasia- compared to atrophy-associated USC. These results suggest a novel origin of tumorigenesis in this rare subset of endometrial cancers.

PR051/#484 SPATIAL PROFILING OF OVARIAN CLEAR CELL CARCINOMA (OCCC) REVEALS IMMUNE-HOT FEATURES

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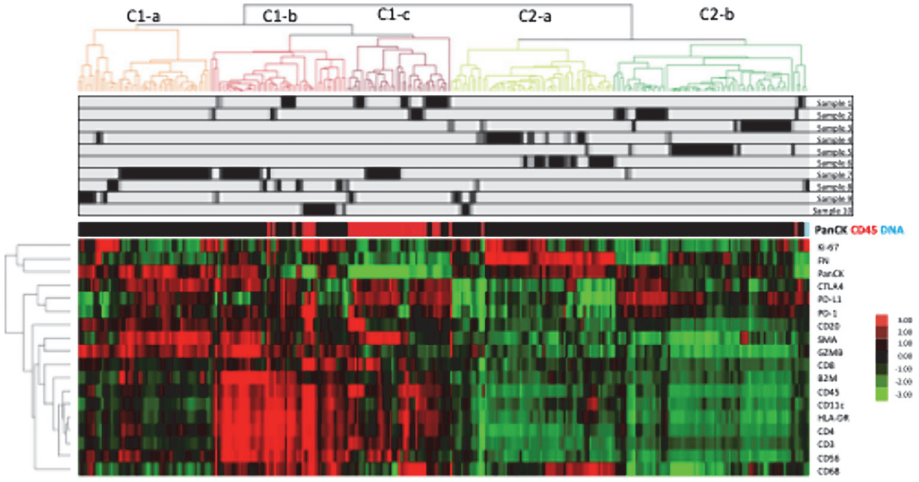
Introduction OCCC has high incidence in Asia with frequent occurrence at early stage but without sufficient data on molecular stratification for high-risk patients. Recently, immune-hot features have been proposed as an indicator for poor prognosis for early-stage OCCC. Specific patterns of intra-tumoral heterogeneity (ITH) associated with immune-hot features need to be defined.

Methods Formalin-fixed paraffine embedded (FFPE) tumor sections from 10 early-stage OCCC patients were included. Digital Spatial Profiling (DSP) of 18 protein targets was conducted by using the nanoString GeoMx system to profile selected regions of interest (ROIs) based on the reference H&E staining morphology. Areas of illumination (AOIs) were defined according to ROI segmentation by the fluorescence signals of visualization markers pan-cytokeratin (PanCK), CD45, or DNA.



Abstract PR051/#484 Figure 1