transplantation and she still suffered from a progressive tumor recurrence and died 5 years later.

Conclusion The potential complexity and heterogeneity of cervical carcinosarcoma contributed to the variety of treatment modality. Such rapidly growing tumor may be responsive to radiotherapy and the role of chemotherapy may also be important, but their expected effects on the sarcomatous component may not be ideal. As a result salvage surgical intervention could be a therapeutic option for such locally advanced diseases.

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417 KRAS MUTANT UTERINE CARCINOMAS

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Background Inhibitors of KRAS mutations (KRASm) disease have shown efficacy in early clinical studies. Data informing about KRASm targeting in endometrial cancer (EC) are lacking.

Methods ECs (n=8336 with various histologies) were queried for presence of actionable mutations (592 genes) and fusions (Whole Transcriptome Sequencing) using Caris Genomic Profiling database. Comparison was done using Fisher-Exact/ChiSquare (p values) and adjusted for multiple tests by Benjamini-Hochberg (q) and Pairwise nonparametric analysis using Wilcoxon Method.

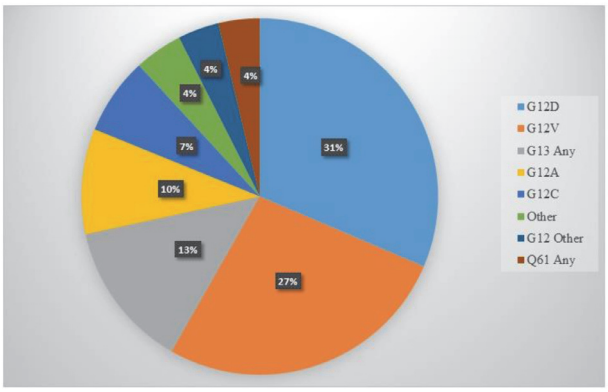
Results a. KRASm is a frequent genotype in Endometrial Cancer.

KRASm were detected in 15.2% of EC cases. Code was most frequently mutated, with G12D (31%) and G12V (27%) being the most common subtypes (figure 1).

b. Biomarkers of immunotherapy response co-occur with KRASm in EC.

MSI-H/dMMR and TMB-H (>10 mt/MB) were seen 36.4% and 42.8% in KRASm and 15.9% and 27.9% in KRASwt, respectively (p>0.05).

c. BRCA1/2 mutations were detected with equal frequency among KRASm and KRASwt. BRCA1/2 mutations were seen in 6% of KRASm vs 4.6% in KRASwt (p=0.033).



Abstract 417 Figure 1 KRAS distribution in entire endometrial cohort

d. KRASm are mutually exclusive of oncogenic fusions. No fusions in FGFR1/3, MET, ALK were detected concurrently with KRASm. Overall, incidence of fusion was extremely low, independent of KRAS status.

Conclusions KRASm EC represents a genomically distinct group of endometrial cancers. Targeted therapy using this biomarker should be explored in clinical trials. Overlap exists with predictors of immunotherapy response, suggesting a possible immunotherapy combination option. Clinical trials to evaluate these strategies are needed.

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418 CLINICAL TRANSLATION OF METHYLATED DNA MARKERS OF ENDOMETRIAL CANCER USING TAMPON-BASED DETECTION

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Objective In tampon samples from women with and without EC, we tested methylated DNA markers (MDMs) for EC originally identified through discovery and validation in tissue.

Methods From 2/2013–8/2019, women ≥45 yrs with abnormal or postmenopausal bleeding or biopsy-proven EC were

Abstract 418 Table 1 Distribution of endometrial cancer (EC) histologies and cross-validated sensitivity by methylated DNA marker panel at 95% specificity in PBS/EDTA tampon buffer (N=57 ECs)

EC histology	Endometrioid	Serous	Carcinosarcoma	Clear cell	Mixed
N	20	23	9	3	2
Sensitivity at 95% specificity % (95% CI)	85% (62-97%)	78% (56-93%)	89% (52-99%)	67% (9-99%)	50% (1-99%)