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.

**IGCS20_1454**

**417 KRAS MUTANT UTERINE CARCINOMAS**

1K Kilowski*, 2M Dietrich, 3J Xiu, 1J Baca, 4T Herzog, 5M Kom, 1R Holloway, 6AdventHealth Cancer Institute, USA; 2Florida Cancer Specialists and Research Institute, USA; 3Cars Life Sciences, Medical Affairs, USA; 4University of Cincinnati Medical Center, USA

10.1136/ijgc-2020-IGCS.362

**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/Chi-Square (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).

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.

**IGCS20_1455**

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

1J Bakkum-Gamez*, 2M Sherman, 3S Slettedahl, 3D Mahoney, 4M Lemens, 5S Laughlin-Tommaso, 6M Hopkins, 7A VanOosten, 8V Shridhar, 9W Taylor, 10J Staub, 11X Cao, 12P Fooe, 13M Clarke, 14K Burger, 15C Berger, 16M McClintch, 17K Doering, 18JK Schoolmeester, 19S Ken, 20N Wentzensen, 21D Ahquist, 22J Kisel, 23Mayo Clinic, USA; 24Mayo Clinic, USA; 25National Cancer Institute, USA; 26Hospital Pathology Associates, USA

10.1136/ijgc-2020-IGCS.363

**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