adjuvant therapy. Local vaginal/pelvic recurrences were more common in the MMRd than MMRi cohort (65% vs. 30%) whereas distant recurrences were more common in MMRi cohort (70% vs. 35%). Post-recurrence survival was higher in MMRd cohort (43.8 vs 20 months; p=0.306). Of those with local recurrences, RT with curative intent was used in the majority (9/15 in MMRd and 7/8 in MMRi). All of those with local recurrences with MMRd tumors salvaged with curative intent RT are alive without disease (9 of 9), whereas only 2 with MMRi tumors are alive without disease (2 of 7).

Conclusions MMRd ECs are more likely to recur locally with high rate of salvage with RT, possibly indicating the increased radiosensitivity in this cohort.

Poster rounds with the professors: Group E4

4/#474 MOLECULAR LANDSCAPE OF ERBB2/HER2 GENE AMPLIFICATION AMONG PATIENTS WITH ENDOMETRIAL CARCINOMA

Dimitrios Nasioudis*, Nawar Latif, Ashley Haggerty, Lori Cory, Sarah Kim, Mark Morgan, Fiona Simpkins, Emily Ko. University of Pennsylvania, Division of Gynecologic Oncology, Philadelphia, USA

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Objectives Investigate the incidence of ERBB2/HER2 gene amplification among patients with endometrial cancer.

Methods The AACR GENIE v12.0 database was accessed and patients with endometrioid (EEC), serous (USC), clear cell (UCC) and carcinosarcoma (UCS) and data on copy-number gene alterations were selected for further analysis. Incidence of ERBB2/HER2 gene amplification was investigated while the genomic profile of patients with ERBB2 amplification was further explored. Data from the OncoKB database was utilized to determine pathogenic gene alterations.

Results A total of 2784 patients were identified; 1648 with EEC (1708 samples), 573 with UPS (593 samples), 389 with USC (389 samples) and 93 with UCC (94 samples). Overall incidence of ERBB2 amplification was 4.5% (n=124); 12% (n=71) for UPS, 8.5% (n=8) for USC, 7.2% (n=28) for USC and 1% (n=17) for EEC. Among samples with ERRB2 amplification, the most prevalent alterations involved the TP53 (91%), PIK3CA (47%), CCNE1 (23%), FBXW7 (24%), MYC (13%), PIK3R1 (17%), KRAS (10%), ARID1A (8%), and ERBB3 (8%) genes. For patients with EEC and ERRB2 amplification, 64.7% (n=11) had a TP53 mutation. However, among patients with EEC and a TP53 mutation (n=304), the overall incidence of ERRB2 amplification was 3.6%.

Conclusions While ERRB2 amplification is frequently encountered among patients with USC, a high incidence was also observed among those with USC, and UCC. For patients with EEC, incidence of ERRB2 amplification is low, especially in the absence of TP53 mutations. Half of tumors with ERRB2 amplification harbored a PIK3CA mutation providing rationale of the combination of transzumab with mTOR/ATK inhibitors in future trials.

5/#274 IS REFLEX MLH1 PROMOTER HYPERMETHYLATION TESTING FOLLOWING MLH1 LOSS BY IMMUNOHISTOCHEMISTRY IN ENDOMETRIAL CARCINOMA BEST PRACTICE?

Anna Plotkin*, Ekaterina Olkhov-Mitsel, Sharon Nofech-Mozes. Sunnybrook Health Sciences Centre, Laboratory Medicine and Molecular Diagnostics, Toronto, Canada

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Objectives Reflex screening of newly diagnosed endometrial carcinomas (EC) was introduced in Ontario for women <70 in 2018 and regardless of age in 2020. MLH1 deficient (MLH1-d) cases by immunohistochemistry (IHC) are further analyzed to detect MLH1 promoter hypermethylation

## Abstracts

### PROGNOSTIC RELEVANCE OF THE MOLECULAR BASED ESMO/ESTRO/ESP2020 RISK CLASSIFICATION IN ENDOMETRIAL CARCINOMA AFTER SURGICAL LYMPH NODE STAGING

1Teresa Prato*, 1Marcel Grube, 1Charlotte Meyer, 1Annika Rohner, 1Suzana Mittelstatt, 2Léa-Louise Volmer, 2Felix Neis, 2Sascha Hoffmann, 2Jürgen Andreas, 2Christina Walter, 2Sara Brucker, 2Amnette Staebler, 2Erfan Oberlechner, 2Bernhard Krämer, 1Stefan Kommos. 1Tuebingen University Hospital, Department of Women’s Health, Tuebingen, Germany; 2Tübingen University Hospital, Institute of Pathology and Neuropathology, Tübingen, Germany

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**Objectives**

The role of endometrial carcinoma (EC) molecular-based risk classification has not yet been fully explored in terms of surgical decision making. Our study aimed to investigate the prognostic relevance of the molecular-based ESMO/ESTRO/ESP2020 risk classification in EC patients after surgical lymph node staging.

**Methods**

Primary EC patients treated at the Tübingen University Women’s Hospital between 2003 and 2016 were identified. Patients without surgical lymph-node staging and FIGO stage IV disease were excluded. Molecular-based risk classification was obtained after POLE sequencing and p53/MMR immunohistochemistry.

**Results**

Molecular, clinical and follow-up data was available in 424 patients. 370(87.3%) cases were endometrioid histotype, grade distribution included 266(62.7%) G1, 73(17.2%) G2 and 85(20.0%) G3 tumors. 338(84.4%) patients were diagnosed with FIGO stage I, 23(5.4%) with stage II and 43 (10.2%) with stage III disease. Molecular classification yielded 123(29.0%) MMRd, 208(49.1%) NSMP, 49(11.5%) p53 abnormal and 44(10.4%) POLE mutated tumors. Positive nodes were reported in 38(9%) patients. Low-risk was assigned in 229(54.0%), intermediate in 76(18.0%), high-intermediate in 46(10.8%) and high-risk in 73(17.2%) cases. In early stage (FIGO I), node negative tumors five year recurrence rates were 3.1% in low risk, 10.9% in intermediate risk, 22.2% in high-intermediate risk and 17.2% in high risk patients (p<0.001).

**Conclusions**

The adverse outcome of early stage high-intermediate and high-risk EC could not be explained by undetected lymphnode involvement in this series. Our findings may aid tailoring surgical EC treatment according to current molecular-based risk classification.