physiologically achievable concentration (maximum plasma concentration (Cmax) value).

Results Sequencing revealed amplified levels of ERBB2 (17q12), RAF1, c-myc, and ERBB3 (12q13.2) low-level gain. Inhibition of viability was moderate by single agents: Afatinib, binimetinib, JQ1, as shown by inhibitory effect values of 14.4%, 47.8%, 8.8%, respectively at physiologically achievable concentrations (Cmax) of afatinib. Combinations demonstrated increasing inhibitory effect values: 99.7% for Afatinib + binimetinib, and 99.5% for Afatinib + JQ1. Synergy was evidenced for both combinations by a combination index <1 (figure 1).

Conclusions Combined inhibition of pan-ERBB with inhibition of MEK or BET proteins synergistically suppress viability in patient-derived serous EC harboring ERBB amplifications.

Abstract EPV106/#249 Figure 1

EPV106/#249 ENDOMETRIAL CANCER IMMUNOHISTOCHEMICAL RISK STRATIFICATION IN A LARGE UTERINE-CONFINED CANCER SERIES

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Objectives The aim of this study is to assess the clinical reproducibility and the oncological validity of the Endometrial cancer (EC) risk stratification based on the molecular information given by the immunohistochemistry (IHC).

Methods Retrospective IHC analyses were conducted in a large series of 778 pre-operative uterine-confined ECs, studying the presence/absence of MLH1, MSH2, MSH6, to define the mismatch repair (MMR) stable or instable phenotype; the presence of p53 mutations and other molecular features. The molecular profile was correlated with histological, clinical and prognostic EC patients’ data.

Results Based on the IHC, we defined 3 EC populations: MMR stable (MMRs), instable (MMRi) and p53 mutated (p53+) patients. Our result demonstrated that the IHC stratification statistically correlated with the most relevant anatomic-clinical features: FIGO stage (p<0.001), grading (12.5% G3 vs 22.9% vs 95.3% in p53+, p<0.001), histotype (Type II 6.2% in MMRs vs 5.3% in MMRi vs 87.5% in p53+, p<0.001), presence of LVSI (positive in 16.3% in MMRs vs 23.8% in MMRi vs 38.7% in p53+, p<0.001), myometrial invasion and tumor dimension (p=0.003 and p<0.001 respectively). Again, the 3 IHC populations statistically reflected the EC risk class ESGO-ESMO-ESP classification 2020 (p<0.001). These results were confirmed also in Kaplan-Meier curves in terms of over-all survival (OS) and disease-free survival (DFS) (p<0.001) (figure 1).

Conclusions In this large series, we demonstrated that the pragmatic and systematic use of IHC may have an important role to properly stratify, in terms of histological features and clinical outcome, the uterine-confined EC patients.

Objectives Pembrolizumab, an anti–PD-1 antibody, has demonstrated activity as monotherapy and in combination with lenvatinib in patients with previously treated mismatch repair (MMR) deficient and MMR proficient endometrial cancer (EC). ENGOT-en11/GOG-3053/KEYNOTE-B21 (NCT04634877) is a phase 3, randomized, double-blind study of pembrolizumab or pembrolizumab + lenvatinib in patients with newly diagnosed high-risk endometrial cancer.

Abstracts
PITFALLS IN PRE-OPERATIVE PREDICTION OF LYMPH NODE METASTASIS IN EARLY ENDOMETRIAL CANCER

Methods Eligible patients are ≥18 years with newly diagnosed high-risk (stage I/II non-endometrioid or with p53 abnormality and any histology, stage III/IVA), previously untreated EC following surgery with curative intent with no evidence of disease post-operatively. 990 patients will be randomized to receive pembrolizumab 200 mg or placebo Q3W for 6 cycles plus chemotherapy (carboplatin area under the curve [AUC] 5/6 plus paclitaxel 175 mg/m² Q3W or carboplatin AUC 2/2.7 plus paclitaxel 60 mg/m² QW) in stage 1. Patients receive pembrolizumab 400 mg or placebo Q6W for 6 cycles in stage 2. Radiotherapy (external beam radiotherapy [EBRT] and/or brachytherapy) ± radiosensitizing cisplatin 50 mg/m² (days 1 and 29) may be administered after completion of chemotherapy. Randomization is stratified by MMR status (pMMR vs dMMR) and, within pMMR, by planned radiation therapy (cisplatin-EBRT vs EBRT vs no EBRT), histology (endometrioid vs non-endometrioid), and FIGO surgical stage (I/II vs III/IVA). Dual primary endpoints are disease-free survival (DFS; per investigator assessment) and OS. Secondary endpoints include DFS (per BICR), DFS (per investigator assessment) and OS by biomarker status (PD-L1 and tumor mutational burden), safety, and QoL. Enrollment began December 2020 and is ongoing in 28 countries.

Results Not applicable

Conclusions Not applicable

EPV108/#267 ANTHOCYANINS AS A PROSCHTROPHIC Factor IN ENDOMETRIAL CANCER

Abstract EPV109/#274 Table 1

Abstract EPV109/#274

<table>
<thead>
<tr>
<th>Type</th>
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</tr>
<tr>
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</tr>
<tr>
<td>D</td>
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</table>

Notes: At p < 0.05 to compare the correlation between treatment, age of patients, stage Ia, Ib, Ic, tumor differentiation.

EPV109/#274 ANALYSIS OF THE FREQUENCY OF ENDOMETRIAL CANCER STAGE I

Objectives In 2020, according to the National Cancer Registry, endometrial cancer in the structure of cancer took second place (10.9%) after breast cancer. Although the detection and treatment of the disease in the early stages has good prospects, but there are relapses from 2% to 26%, according to various literature.

Methods The analysis of recurrence rate among 968 patients with endometrial cancer and stages of endometrioid type. Recurrences amounted to 68 cases (7.02%). The staging took place according to the 1988 FIGO classification. The following statistical methods were used: standard descriptive, parametric and nonparametric. Differences at p < 0.05 were considered significant.

Results The analysis was performed depending on the characteristics of the tumor process and the type of treatment, the recurrence rate was estimated - see table 1. The average age of patients ranged from 25 to 85 years. The recurrence time was detected, on average, after 36 months ± 15.97 months. In combination treatment, receiving adjuvant radiation therapy, recurrences were most often detected - after 6–18 months ± 13.53 months. Long-term recurrences were detected after a combination of surgical treatment with chemotherapy at 32–