metformin yielded significantly higher CR rate (39.6%, 21/53) than MA alone (20.4%,10/49, p=0.032, OR 0.347, 95%CI 0.132–0.914). Regarding 30w-CR rate, it was slightly higher in MA/metformin group than control (69.2% vs 57.4%, p=0.167). Nevertheless, the mean treatment time in MA/metformin group was 4 weeks shorter (27 vs 31 weeks). Particularly, the mean weight gain by MA/metformin (2.5kg) was twice lower than MA alone (5.0kg, p=0.014). No intra-group difference was found in rates of recurrence, pregnancy or live birth.

Conclusions MA/metformin may lead to a higher CR rate, shorter treatment time and less weight gain compared with MA alone.

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| 8 | MUTATIONAL ANALYSIS OF CERVICAL CYTOLOGY IN ENDOMETRIAL CANCER: A PROSPECTIVE MULTICENTER TRIAL |

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Objectives Endometrial carcinoma (EC) is traditionally diagnosed by histopathological assessment of endometrial biopsies, leaving up to 22% of patients undiagnosed. This study explores the feasibility of the clinical implementation of detecting EC using mutational analysis of cervical cytology.

Methods This prospective multicentre study included patients that underwent a hysterectomy for histopathologically proven EC or a benign gynecological condition (control group). A Pap brush sample, cervicovaginal self sample, pipelle and hysterectomy specimen were obtained from each patient. A targeted next-generation sequencing panel was used to screen these samples for mutations in eight genes. Diagnostic accuracy was calculated, including sensitivity, specificity and predictive values.

Results Fifty-nine EC patients and 31 control patients were included. In these patients traditional histopathological diagnosis had a sensitivity of 78.9% and a specificity of 100%. For EC patients, 96.6% of surgical specimens contained at least 1 mutation. Blinded mutational analysis of Pap brush samples, self-samples, and pipelle endometrial biopsies yielded a sensitivity of 78.0%, 67.3% and 96.5% with a specificity of 96.8%, 96.8% and 93.5%, respectively. Combining these three methods with histopathological pipelle endometrial biopsies evaluations yielded a sensitivity of 95.8%, 93.0% and 96.5 respectively.

Conclusions This study has shown the potential of mutational analysis of cervical cytology and pipelle endometrial biopsies to improve diagnosis of EC, whether or not in additional to traditional histopathological assessment. Prospective evaluation is required for validation and clinical implementation.

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| 9 | GENOMIC PROFILING OF RECURRENT "ULTRA-LOW RISK" ENDOMETRIAL CANCER |

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Objectives To characterize the genomic alterations in recurrent low-grade, non-invasive endometrioid endometrial carcinomas (EECs).

Methods We retrospectively identified patients with stage IA EEC who underwent primary surgery at our institution, 2/2009–2/2017, and had follow-up of at least 11 months. "Ultra-low risk" was defined as FIGO grade 1/2, non-invasive, and lymphovascular space invasion-negative. DNA extracted from 36 tumors and matched normal tissue/blood was subjected to massively parallel sequencing targeting over 400 cancer-related genes. Microsatellite instability was assessed via MSIsensor.

Results 499 patients with "ultra-low risk" EEC were identified. 14/499 (2.8%) had a recurrence. Median follow-up for non-recurrent cases was 33.0 months (range, 11–116) and for recurrent 50.5 months (range, 11–116). Recurrent patients were older than non-recurrent patients (p=0.016), and had endometriosis identified during pathologic review of specimen more frequently (p=0.015). Other clinical characteristics did not differ.

Mutational profiling of primary tumors from 8 recurrent and 28 non-recurrent patients revealed that most ultra-low risk EECs were microsatellite-stable (7/8, 88% recurrent; 26/28, 93% non-recurrent). Mutational signatures varied widely with no dominant signature identified among either group. *PTEN* and *PIK3CA* were the most frequently mutated genes in both groups. *CTNNB1* hotspot mutations were found in 4/8 (50%) recurrent and 9/28 (33%) non-recurrent EECs (p=0.146).

Conclusions Patients diagnosed with "ultra-low risk" EEC have an excellent prognosis; however, we noted that 2.8% of patients developed a recurrence without identifiable clinical or pathologic risk factors. Genomic profiling did not reveal unique alterations in either group. Further work is needed to elucidate the mechanism/biomarker for recurrence in this "ultra-low risk" population.