(160 mg orally, daily) plus metformin (500 mg orally, three times a day).

Results The complete remission rate, disease-free survival (DFS) rate and pregnancy rate had no significant difference between two treatment groups. However, the DFS rate was higher in the metformin plus MA group than in the MA-only group in non-obese (body mass index<28 kg/m^2) patients with EAH (hazard ratio [HR] = 2.677, 95% confidence interval [CI] = 1.066–6.727; Log Rank P = 0.029), while had a lower tendency in obese patients with EAH (HR = 0.224, 95% CI = 0.045–1.223; Log Rank P = 0.062). According to cox proportional hazards regression analysis, undergoing assisted reproductive treatment (HR = 2.358, 95% CI = 1.069–5.204; Log Rank P = 0.034) was identified as an independent risk factor for recurrence, whereas younger patients were found to have a higher probability to achieve pregnancy (HR = 0.568, 95% CI = 0.332–0.973; Log Rank P = 0.039).

Conclusion/Implications Currently, there is no sufficient evidence to support that the utilization of metformin plus MA can significantly improve the prognosis of patients with EAH or EEC compared to MA monotherapy. However, obese patients with endometrial lesions may benefit from the addition of metformin.

PR029/#227 ROBOT-ASSISTED VERSUS CONVENTIONAL LAPAROSCOPIC SURGERY FOR ENDOMETRIAL CANCER: LONG-TERM COMPARISON OF OUTCOMES

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Introduction There is a lack of multi-institutional large-volume and long-term follow-up data on comparisons between robot-assisted surgery and conventional laparoscopic surgery. This study compared the surgical and long-term survival outcomes between patients who underwent robot-assisted or conventional laparoscopic surgery for endometrial cancer.

Methods We retrospectively reviewed the data of patients from five large academic institutions who underwent either robot-assisted or conventional laparoscopic surgery for the treatment of endometrial cancer between 2012 and 2017, ensuring at least 5 years of potential follow-up. Intra- and postoperative outcomes, long-term disease-free survival, and overall survival were compared.

Results The study cohort included 1,003 unselected patients: 551 and 452 patients received conventional laparoscopic and robot-assisted surgery, respectively. The median follow-up duration was 57 months. Postoperative complications were significantly less likely to occur in the robot-assisted surgery group than in the laparoscopic surgery group (7.74% vs. 13.79%, P = 0.002). There were no significant differences in survival: 5-year disease-free survival was 91.2% versus 90.0% (P = 0.628) and overall survival was 97.9% versus 96.8% (P = 0.285) in the robot-assisted and laparoscopic surgery cohorts, respectively. Cox proportional hazard regression models demonstrated that the mode of surgery was not associated with disease-free survival (hazard ratio, 0.897; confidence interval, 0.563–1.429) or overall survival (hazard ratio, 0.791; confidence interval, 0.330–1.895) after adjusting for confounding factors.

Conclusion/Implications Robot-assisted surgery for endometrial cancer is associated with similar long-term survival outcomes but fewer postoperative complications as compared to conventional laparoscopic surgery.

PR030/#559 PAX2 IS REGULATED BY ESTROGEN/PROGESTERONE THROUGH PROMOTER METHYLATION IN ENDOMETRIOID ADENOCARCINOMA AND TAKE THE IMPORTANT ROLE IN CARCINOGENESIS VIA THE AKT/MTOR SIGNALING PATHWAY

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Introduction PAX2 (Paired box 2) and PTEN inactivation were reportedly as important biomarkers for endometrioid intraepithelial neoplasia (EIN) and Endometrioid endometrial carcinoma (EEC). However, though PTEN was extensively studied, the role of PAX2 in EEC carcinogenesis still remains unclear.

Methods Public databases and clinical paired paraffin-embedded tissues were used to analyze PAX2 expression in EEC. Cell function tests and mouse xenograft models were utilized to study the biological functions of PAX2. Pyrosequencing and demethylating drug 5-Aza-dc were used to verify promoter methylation in clinical tissues and cell lines, respectively. The mechanism underlying the regulatory effect of estrogen (E2) and progesterone (P4) on PAX2 expression was investigated by receptor block assay and double luciferase reporter assay.

Results PAX2 expression was significantly down-regulated in EIN and EEC tissues, its overexpression inhibited EEC cell malignant behaviors in vivo and in vitro and inhibited the AKT/mTOR signaling pathway. PAX2 inactivation in EEC was related to promoter methylation, and its expression was regulated by E2 and P4 through their receptors via promoter methylation.

Conclusion/Implications Our findings elucidated the expression and function of PAX2 in EEC and firstly provided hitherto undocumented evidence of the underlying molecular mechanisms. PAX2 expression is suppressed by estrogen prompting its methylation through estrogen receptor. Furthermore, PAX2 regulates the AKT/mTOR signaling pathway to influence EEC progression.

PR031/#378 A PREDICTIVE MODEL FOR LYMPH NODE METASTASIS USING A TUMOR LOCATION IN PRESUMED EARLY-STAGE ENDOMETRIOID ENDOMETRIAL CANCER PATIENTS

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