Mixed endometrial carcinoma refers to rare endometrial tumours that are comprised of two or more distinct histotypes, at least one of which is serous or clear cell. Limited data is available on the recurrence rates for mixed epithelial endometrial carcinoma, as it comprises a relatively understudied subtype of endometrial cancer. The aim of this study is to evaluate the epidemiology, treatment outcomes and survival rates of patients with mixed endometrial carcinoma.

**Methodology** Medical records of the patients diagnosed with mixed endometrial carcinoma between March 2010 and January 2020 were reviewed retrospectively. Clinicopathological variables and treatment strategies were assessed, and overall survival (OS) and disease-free survival (DFS) rates were evaluated.

**Results** A total of 34 patients were included in the study. Histology of endometrioid and serous component was found in 26 (76.5%) patients, followed by serous and clear cell components (5/34, 14.7%) and a mixture of endometrioid, serous and clear cell components (3/34, 8.8%). The median age was 70 years (range 52–84), and median follow-up time was 55 months. Most patients (70%) were treated with laparoscopy. Overall, the 5-year disease-free survival rate (DFS) and the 5-year overall survival rate (OS) were 50.4% and 52.4%, respectively. Advanced disease stage was found to be independently correlated with worse 5-year disease-free survival (DFS) and overall survival (OS) rates (p < .001).

**Conclusion** The management of mixed epithelial endometrial carcinoma presents several challenges for clinicians and researchers that need to be addressed to improve oncologic outcomes. Accurate and early diagnosis plays a fundamental role to determine the appropriate treatment plan. Improved diagnostic techniques, such as molecular profiling and imaging technologies, as well as identification of specific biomarkers associated with the distinct features of the tumour, can help clinicians effectively stratify the patients and tailor treatment accordingly. Undoubtedly, the implementation of molecular analysis will offer further diagnostic and management insights.

**Disclosures** No relevant conflict of interest disclosures for any of the co-authors.

Oncological quality of open, laparoscopic, and robotic surgery in early-stage endometrial cancer, a nationwide, population-based cancer registry study in Taiwan

**Methodology** Data were extracted from the American Association for Cancer Research’s (AACR) Project Genomics Evidence Neoplasia Information Exchange (GENIE) database version 13.1 via cBioPortal (http://genie.bioportal.org). We queried this database for uterine sarcoma samples and analyzed frequencies of pathogenic gene variants (PGVs) for which targeted therapies are currently available or in clinical trials for other cancer types. These included PGVs associated with homologous recombination deficiency (HRD): ATM, ATRD1A, ATRX, BRCA1, BRCA2, BRD1, BRIP1, BLM, BAP1, CHEK1, CHEK2, FANCA, FANC, FANCD2, FANCE, FANCF, FANCG, FANCL, MRE11, NBN, PALB2, RAD50, RAD51B, RAD51C, RAD51D, WRN; PGVs associated with the MAP-kinase signaling pathway: BRAF (V600E), KRAS, NRAS, E121X; PGVs associated with mismatch repair (MMR): MSH2, MSH3, MSH6, MLH1, MLH3, MSH2, EPCAM; PGVs in other genes: PTEN, PIK3CA MTOR, CDKN2A, CDKN2B, and ERBB2 (amplification).

**Results** A total of 704 uterine sarcoma tumor samples from 680 patients were included for analysis. At least one somatic PGV was observed in an HRD associated gene in 27.8% (196/704) of all tumors, with the most common PGVs observed in ATRX (109/645, 16.9%), BRCA2 (27/652, 4.1%) and RAD51B (16/433, 3.7%). At least one somatic PGV was observed in an MMR associated gene in 3.0% (21/704) of all tumors, with the most common PGVs observed in MSH6 (6/648, 0.9%) and MSH2 (5/633, 0.8%). At least one somatic PGV was observed in a MAP-kinase associated gene in 2.8% (20/704) of all tumors, with the most common PGVs observed in KRAS (14/680, 2.1%). The highest frequencies of other targetable PGVs were observed in PTEN (89/677, 13.1%) and PIK3CA (16/680, 2.4%).

**Conclusion** The high rate of PGVs in HRD genes and PTEN in uterine sarcoma tumor samples suggests the need for clinical trials evaluating the efficacy of genetically targeted therapeutics for this patient population.

**Disclosures** No relevant conflict of interest disclosures for any of the co-authors.