in the team. All of the surgeries were carried out by trainees in Gynaecological Oncology and supervised by a specialist. The qualifications for a total hysterectomy with bilateral salpingo-oophorectomy and lymph node staging were based on ESGO recommendations. Two groups were compared in the following fields: age, BMI, time of the procedure, total number of dissected pelvic and paraaortic lymph nodes, complications, postoperative hospital stay.

**Result(s)** Most of the results obtained were similar in both groups. The average age of the participants in both groups was 64.57 years (laparoscopy: 63.48 years, open surgery: 66.35 years), BMI was 31.49 kg/m² (31.67 vs 31.25 kg/m²). The quantity of dissected lymph nodes during laparoscopy was 21.2 pelvic nodes and 12.4 paraaortic nodes per procedure and respectively 23.76 and 12.9 for laparotomy. The rate of complications was equal in both groups (n=6). Significant differences were observed in two areas. The time of surgery was longer in a laparoscopic group (181 minutes) vs. 141 minutes for open surgery. The postoperative stay took 3 days after the minimally invasive procedure and 6 days after laparotomy.

**Conclusion** The outcome of surgical treatment performed by trainees was comparable in both groups. The minimally invasive surgery group had many benefits as quick recovery, great precision of surgery with an adequate oncological accuracy. The longer duration of laparoscopic procedures is a result of a learning curve.

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**Survival Analysis of Endometrial Cancer. Our Experience in Las Palmas de Gran Canaria**


**Introduction/Background** Self-evaluation and analysis of healthcare practice in a Gynecology Oncology unit represents an advance in the strategy to improve the quality of cancer care, in this case, endometrial cancer, allowing to obtain relevant data on the quality of care practice.

**Methodology** Retrospective descriptive study carried out in CHUIML. We included patients diagnosed with endometrial cancer during the study period 2012-2016. We analysed epidemiological variables, histological type, type of treatment, final FIGO stage and 5-year overall survival.

**Result(s)** A total of 498 patients were diagnosed with endometrial cancer during this period. Mean age of 64.3 years (range 29-89 years) and a mean BMI of 33.5. 60.2% of the patients were diagnosed in stage IA; 48.7% of which were grade 1 and 2. Type I (endometroid and mucinous) accounted for 74.3% versus 25.7% type II. 40.8% of the patients were classified as low risk presenting a 5-year survival of 99%. Patients with intermediate risk represented 5.9% and had a 96% 5-year survival. Patients with intermediate-high risk (19.7%) had a 5-year survival of 88%. The patients included in the high-risk group (28.2%), had a 5-year survival of 62%, these differences were statistically significant.

We analyzed survival according to the histological type, finding a 5-year overall survival rate of 91.9% in type I, while it was 60.8% for type II, these differences were statistically significant.

**Conclusion** The data analyzed in our Gynecology Oncology Unit are similar to those exposed in the literature, there is an important difference in terms of 5-year survival between the low and high risk groups, 99% vs 62% respectively.

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**Abstracts**

**TERTIARY LYMPHOID STRUCTURES AS MARKERS OF ANTI-TUMOR IMMUNITY WITH INDEPENDENT PROGNOSTIC VALUE IN THE PORTEC-3 TRIAL OF HIGH-RISK ENDOMETRIAL CANCER**

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**Introduction** Tertiary lymphoid structures (TLS) are ectopic lymphoid tissues that form in and around cancers. TLS consist of a germinal centre (GC) with proliferating B-cells and follicular dendritic cells (FDCs), as well as a peripheral T-cell zone. Local and systemic B- and T-cell responses are thought to be initiated and maintained at the TLS. Here, we analysed whether TLS were associated with anti-tumour immunity and a reduced risk of recurrence in endometrial cancer (EC).

**Methodology** TLS were quantified by an expert pathologist (TB) on H&E-stained tumour slides of the cancer genome atlas uterus cancer cohort (TCGA UCEC), and by immunohistochemistry (IHC) on tumour slides from the PORTEC-3 trial biobank. Time to recurrence analysis were performed according to Kaplan-Meier’s method, using log-rank tests and Cox’ proportional hazards models, including prespecified multivariable analysis with clinico-pathological and molecular risk factors.

**Result(s)** Differential gene expression analysis of TLS-positive and TLS-negative cases from TCGA UCEC identified, among others, L1-cell adhesion molecule (L1CAM) (figure 1A). IHC on tumour slides from the PORTEC-3 trial biobank. Time to recurrence analysis were performed according to Kaplan-Meier’s method, using log-rank tests and Cox’ proportional hazards models, including prespecified multivariable analysis with clinico-pathological and molecular risk factors.
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A. Differential gene expression of TLS-negative vs. TLS positive cases in the TCGA uterine cancer cohort.

B. Tertiary lymphoid structures visualised by H&E and L1CAM staining in endometrial cancer

Abstract 482 Figure 1  Identification of L1CAM as a marker of mature TLS in endometrial cancer

Abstract 482 Figure 2  Relation between TLS, CD8+ cel density and molecular classifiers in high risk endometrical cancer
prognostic impact was independent of clinicopathological and molecular factors (adjusted HR 0.32 95%CI 0.14-0.74, p=0.0077).

Conclusion* L1CAM identifies tertiary lymphoid structures with germinal centres. Our data suggest a pivotal role of TLS in the risk of recurrence of EC. L1CAM IHC is simple, available across many study cohorts and could be readily implemented as biomarker of TLS in future trials and clinical care.

**Abstracts**

**486 CLINICOPATHOLOGICAL CHARACTERISTICS OF WOMEN WITH CTNNB1-MUTATED ENDOMETRIAL CANCER**

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**Introduction/Background*** The molecular characterisation of endometrial cancer (EC) represents a step towards personalised management. The current ESGO-ESTRO-ESP guidelines classify EC into four groups: POLE mutated (POLEmut), p53 abnormal (p53abn), mismatch repair deficient (MMRd) and the largest group of no specific mutational profile (NSMP). Women with NSMP tumours generally have a good prognosis, but if disease recurs, the prognosis tends to be poor. A proposed additional molecular classifier to improve the risk assessment are mutations of catenin beta 1 (CTNNB1). The aim of this study was to assess the clinicopathological characteristics of women with CTNNB1-mutated tumours for further risk assessment.

**Methodology** This prospective observational study included women diagnosed with endometrial cancer between January 2020 – March 2021 at the University Medical Centre Maribor, Slovenia. Immunohistochemical (IHC) staining was used to evaluate the expression of p53 and mismatch repair proteins MLH, MSH2, MSH6 and PMS2. Sanger sequencing of exons 9, 13 and 14 was used to determine the POLE status and of exon 3 for CTNNB1 status. Statistical analysis was performed using IBM SPSS version 23. Descriptive statistics were calculated for numerical variables. Chi-Square (χ²) test was used to evaluate the relationship between CTNNB1 status and the tumour stage, depth of tumour invasion and lymph node involvement.

**Result(s)** Out of 45 women included in the study, 5 (11.1%) were found to have a mutation in the exon 3 of CTNNB1, 2 women in D32V (40%), 2 women in S32C (40%) and 1 woman in S37P (20%). Among them, 4 women (80%) were classified as NSMP and 1 (20%) as p53abn. Moreover, 2 women (40%) were diagnosed with early stage (FIGO I-II) and 3 (60%) with advanced stage (FIGO IIIa or more) EC. CTNNB1 status was not correlated with lymph-node involvement (p>.418) and myometrial (p>.802) or lympho-vascular space invasion (p>.855).

**Conclusion** CTNNB1 testing could be used for further classification of molecularly undefined EC. Especially in the NSMP group, this could provide more information about the disease biology and lead to better management of women. Further evaluation of the long-term impact of CTNNB1 mutations on recurrence-free survival and overall survival is needed.

**497 ENDOMETRIAL HYPERPLASIA: RISK OF COEXISTENCE AND PROGRESSION TO ENDOMETRIAL CARCINOMA. RETROSPECTIVE COHORT STUDY**

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**Introduction/Background*** Endometrial hyperplasia (EH) is characterized by an irregular proliferation of the endometrial glands with an increased gland/stroma ratio compared to the proliferative endometrium. The risk of malignancy depends on the presence of the atypia. The purpose of this study was to determine incidence of concomitant endometrial carcinoma (EC) and the risk of malignancy of the disease.

**Methodology** Retrospective cohort study was performed It includes a total of 120 patients diagnosed of EH by endometrial biopsy at Hospital Universitario 12 de Octubre between January 2015 to January 2020. The epidemiological and clinical characteristics of the patients were analysed. The incidence of concomitant EC was investigated in patients in whom hysterectomy was performed. Also in cases of expectant management and/or medical treatment, the rate of regression, persistence an progression of EH was studied.

**Result(s)** According to the criteria of the 2014 WHO classification, 70.8% of cases were EH without atypia and 29.2% with atypia. The mean age at diagnosis was 48.9 years and 71.7% of the patients were premenopausal.. The most frequent symptom was abnormal uterine bleeding and the most ultrasound finding was pathological endometrial thickness (52.5%), followed. A suspected endometrial polyp (21.7%). Hysterectomy was performed in 25% of the total cases. In this group of patients, the incidence of concomitant EC was 33.3%, all of them in EH with atypia. In the group of patients with medical or expectant treatment, after a mean follow-up time of 25 months, a regression of the disease was observed in 95.4% and a progression to EC in 2.3%.

**Conclusion** Although EH is a benign disease entity, with a high regression-cure rate, its risk of coexistence with EC is not negligible, especially in cases where atypia is observed.

**501 LOX1 AND NALP3: FROM IMMUNE TOLERANCE DISRUPTION IN PREGNANCY COMPLICATIONS TO IMMUNE ESCAPE IN ENDOMETRIAL CANCER**

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