## 788 FACTORS FAVORING THE ERRONEOUS ULTRASOUND CLASSIFICATION OF THE DEGREE OF MYOMETRIAL INFILTRATION IN ENDOMETRIAL CARCINOMA

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**Introduction/Background*** Determining the degree of myometrial infiltration allows establishing the best therapeutic approach for each patient as it is an important factor in predicting nodal metastases.

Few prospective studies comparing the diagnostic performance of transvaginal ultrasound (TVS) and magnetic resonance imaging (MRI) in the preoperative local staging of endometrial carcinoma have been reported. In fact, a recent meta-analysis has shown that both techniques have similar diagnostic accuracy. However, to the best of our knowledge, there has been no prospective comparison of the diagnostic performance of TVS and MRI in the same group of patients with low-grade endometrial cancer.

The aim of this study was to analyse which factors could influence the ultrasound assessment of the myometrial infiltration.

**Methodology** Observational prospective study performed at a single tertiary care centre from 2016 to 2020, comprising 156 consecutive patients diagnosed by endometrial sampling as having an endometrioid grade 1/grade2 endometrial cancer. TVS and MRI were performed prior to surgical staging for assessing MI, which was estimated using subjective examiner's impression and Karlsson's method for both TVS and MRI. During surgery, intraoperative assessment of MI was also performed. Definitive pathological study considered as reference standard.

Univariate logistic regression model has been used to study the association between potential confounding variables adn the ultrasound assessment of myometrial infiltration.

**Result(s)*** Variables such as age older than 65 years old, endometrial thickness determined by ultrasound greater than 15 mm, ultrasound pattern of moderate-abundant vascularization, definitive G3 histological grade and presence of lymphovascular invasion in definitive AP study are related to a higher risk of ultrasound misclassification. The first three variables tend to cause an overestimation fo the MI degree, while the last two tend to cause its underestimation.

**Conclusion*** When assessing myometrial infiltration by transvaginal utlrasound we should remind that there are some counfunding variables which could make us misclasiffy myometrial infiltration.

## 790 IN RL95–2 AND KLE MODEL CELL LINES OF MODERATELY AND POORLY DIFFERENTIATED ENDOMETRIAL CARCINOMA, ESTROGENS CAN BE FORMED VIA THE SULFATASE PATHWAY

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**Introduction/Background*** Endometrial cancer (EC) is the most common gynecological malignancy in the western world. EC has traditionally been divided into type I, which is estrogen dependent, and type II, where associations with estrogens were only recently uncovered. Both types of EC arise after menopause when tumor tissue depends on formation of estrogens from inactive steroid precursors. In EC, active estrogens can be formed from circulating estrone sulfate (E1-S) via sulfatase pathway by the sulfatase (STS) and reductive 17β-hydroxysteroid dehydrogenase type 1 (HSD17B1) enzymes.

**Methodology** In our study, we aimed to investigate the role of estrogens in model cell lines of moderately (type I) and poorly (type II) differentiated EC: RL95-2 and KLE cells, respectively. We evaluated expression of genes involved in E1-S transport, estrogen biosynthesis and oxidative metabolism, and examined cellular uptake of E1-S, formation of estrogens from E1-S, and effects of estrogens on cell proliferation.

**Result(s)*** Gene expression analysis revealed up-regulated expression of several E1-S uptake transporters: *SLCO1A2* (3434-fold), *SLCO1B3* (2302-fold), *SLCO1C1* (381-fold), *SLCO3A1* (19-fold), *SLC22A9* (5-fold), *SLC10A6* (5-fold), and functional studies showed increased E1-S uptake in KLE cells *versus* RL95-2 cells. Higher levels of STS were confirmed in RL95-2 cells, which also better metabolized E1-S to estrone (E1), compared to KLE cells. In KLE cells, disturbed balance in the expression of genes encoding reductive and oxidative HSD17B enzymes enhanced activation of E1 to estradiol (E2), compared to RL95-2 cells, and physiological E1 concentrations stimulated KLE cell proliferation. Additionally, gene expression analysis in KLE *versus* RL95-2 cells indicated increased *CYP1B1* expression (17-fold) as responsible for formation of carcinogenic 4-hydroxycatechols, and down-regulation of genes that encode phase II metabolic enzymes: *COMT* (6-fold), *NQO1* (13-fold), *NQO2* (7-fold), and *GSTP1* (2-fold). This suggested decreased detoxification of carcinogenic metabolites in KLE cells.

**Conclusion*** Our results indicate that in cell lines of type I and type II EC, estrogens can be formed via the sulfatase pathway, and can promote proliferation of poorly differentiated EC. This supports the importance of estrogens in poorly differentiated (type II) EC. Further studies in tissue samples of type II EC are needed to confirm our findings.

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## 791 EFFECTIVENESS AND SAFETY OF LENVATINIB AND PEMBROLIZUMAB (LENPEM) THERAPY FOR ENDOMETRIAL CANCER (EC): RESULTS FROM A RUSSIAN MULTICENTER DATABASE

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