Introduction/Background* Determining the degree of myome-
trial infiltration allows establishing the best therapeutic
approach for each patient as it is an important factor in pre-
nûding nodal metastases.

Few prospective studies comparing the diagnostic perform-
ance 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 accu-

racy. 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 influ-
ence 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 hav-
ing 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 Karlson’s method for both TVS and MRI. During surgery,
inaoperative 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 and
the ultrasound assessment of myometrial infiltration.

Result(s)* Variables such as age older than 65 years old, endo-
metrial thickness determined by ultrasound greater than 15
mm, ultrasound pattern of moderate-abundant vascularization,
definitive G3 histological grade and presence of lymphovascu-
lar invasion in definitive AP study are related to a higher risk
of ultrasound misclassification. The first three variables tend
to cause its underestimation.

Conclusion* When assessing myometrial infiltration by transvag-
nal ultrasound we should remind that there are some confounding
variables which could make us misclassify myometrial infiltration.

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 are
usually recently observed. Both types of EC arise after
menopause when tumor tissue depends on formation of estro-
gens from inactive steroid precursors. In EC, active estrogens
can be formed from circulating estrone sulfate (E1-S) via sulfa-
tase pathway by the sulfatase (STS) and reductive 17β-hydro-
ysteroid 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 differenti-
ated EC. This supports the importance of estrogens in poorly
differentiated (type II) EC. Further studies in tissue samples of
these two types are needed to confirm our findings.

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