genetic features (POLE mutation [POLEmt], microsatellite
instability high [MSI-H], homologous recombination defect
[HRD], MUC16 mutation [MUC16mt]) showed significant
overlap. In Kaplan-Meier survival analyses, MIS and open sur-
ery brought similar survival outcome in patients with POLEmt,
MSI-H, HRD or MUC16mt. But in POLE wild type, non MSI-H,
non HRD, or MUC16 wild type patients, MIS resulted in shorter
recurrence-free survival (RFS) (p=0.008, 0.015, 0.003, 0.008).
Based on TCGA classification, POLE ultramutated and MSI
hypermutated type had similar prognosis after two surgeries,
while copy-number low type without CTNNB1 mutation and
copy-number high type with TP53 mutation showed more rapid
recovery after MIS (p=0.048 and 0.037). Further analyses were
done to simplify the model. In patients with ≥1 of the 4 features
(POLEmt, MSI-H, HRD or MUC16mt), MIS and open surgery brought
comparable overall survival and RFS (p=0.339 and 0.969); for
patients with none of the features, especially those with
wild type CTNNB1 or TP53 mutation, longer RFS was
observed in open surgery group (p=0.001, <0.001, <0.001,
respectively). All the results of Kaplan-Meier analyses were
verified by Cox regressions.

Conclusion The molecular features of EC are related to
patients’ prognosis after different surgical approaches. MIS
should be recommended in patients with POLEmt, MSI-H,
HRD or MUC16mt for similar survival outcome and less peri-
operative complications compared to open surgery.

Disclosures This work was supported by the National Natural
Science Foundation of China (81972426, 81202041,
81672371, 81874108 and 81802607), Special Projects for
Strengthening Basic Research of Peking University
(BMU2018JC005), National Key Technology R&D Program of
China (2019YFC1005200 and 2019YFC1005201).

The authors have no potential conflict of interest to
disclose.

Introduction/Background Endometrial cancer is the most com-
mon malignancy of the female reproductive tract. Lymph
node metastases are an important prognostic factor in endo-
metrial cancer. Several prognostic factors have been shown to
correlate with lymph node metastases: depth of myometrial
invasion, cervical stroma infiltration, histologic grade of the tumor,
tumor diameter, serous histology, lymphovascular invasion, and
positive peritoneal cytology.

Methodology Finding the pathohistological parameters that
will indicate with greater certainty the possibility of metastases
in the lymph nodes, on the basis of which it will be evaluated
whether such patients should undergo lymphadenectomy or not.
A retrospective analysis of patients with endometrial can-
cer who underwent surgery at the Oncology Institute of Voj-
vodina (Clinic for Operative Oncology – Department of
Gynecology) in the period from 2012 to 2018.The study
included 120 patients who underwent hysterectomy with bilat-
eral adnexectomy and pelvic lymphadenectomy.

Results Among patients who had lymph node metastases, there
were statistically significant more patients (p <0.01) with
endometrial cancer of histological type 2, with depth of myo-
metrial invasion greater than 50%, cervical stroma infiltration,
lymphovascular invasion, and positive peritoneal cytology.

Conclusion Histopathological parameters such as type 2 endo-
metrial cancer, myometrial invasion depth greater than 50%,
cervical stroma infiltration, lymphovascular invasion and posi-
tive peritoneal cytology increase the chances of lymph node
metastases. Tumor size (> 2 cm) as well as histologic grade
did not correlate with a higher incidence of lymph node
metastases. In this study, both the parametral infiltration and
the number of lymph nodes removed have clinical significance,
but not statistical significance.

Abstract 165 Table 1 The stagewise and grade wise numbers

<table>
<thead>
<tr>
<th>STAGE</th>
<th>NUMBER</th>
</tr>
</thead>
<tbody>
<tr>
<td>IA,G1</td>
<td>6</td>
</tr>
<tr>
<td>IA,G2</td>
<td>3</td>
</tr>
<tr>
<td>IA,G3</td>
<td>5</td>
</tr>
<tr>
<td>IB,G1</td>
<td>4</td>
</tr>
<tr>
<td>IB,G2</td>
<td>3</td>
</tr>
<tr>
<td>II</td>
<td>2</td>
</tr>
<tr>
<td>III A</td>
<td>1</td>
</tr>
</tbody>
</table>
The common dose fractionations for VBT were 25 Gy/5#, 24 Gy/4# and 21 Gy/3#; weekly #s. Majority of the prescriptions were to the vaginal mucosa. There was no standard documentation of vaginal shortening. The use of vaginal dilators was scarce.

Conclusion There was a substantial percentage of women who received VBT in the low risk group. A survey among the consultants showed that poor follow up and the lack of patient awareness, as the reason behind this. Patients operated at peripheral centres ended up having VBT in the low risk group. Lower uterine segment involvement seems to be a factor tipping the decision towards VBT. The use of VBT boost after Pelvic Radiotherapy, has been seen in about 88% of the patients without the involvement of cervix, warranting a national consensus guideline for these tumours.

Disclosures We disclose no conflict of interest.

Abstract 165 Figure 1

Percentage of patient receiving only VBT and VBT boost after pelvic RT only VBT: The stage distribution of the patients who underwent only VBT was as follows (figure 2 and table 1).

Abstract 165 Figure 2

The common dose fractionations for VBT were 25 Gy/5 #, 24 Gy/4# and 21 Gy/3#; weekly #s. Majority of the prescriptions were to the vaginal mucosa.

There was no standard documentation of vaginal shortening. The use of vaginal dilators was scarce.

Conclusion There was a substantial percentage of women who received VBT in the low risk group. A survey among the consultants showed that poor follow up and the lack of patient awareness, as the reason behind this. Patients operated at peripheral centres ended up having VBT in the low risk group. Lower uterine segment involvement seems to be a factor tipping the decision towards VBT. The use of VBT boost after Pelvic Radiotherapy, has been seen in about 88% of the patients without the involvement of cervix, warranting a national consensus guideline for these tumours.

Disclosures We disclose no conflict of interest.

Abstract 165 Figure 1

Abstract 254 Figure 1

Int J Gynecol Cancer 2020;30(Suppl 4):A1–A142