| **Core/ Non-core** | **Element name** | **Values** | **Commentary** | **Implementation notes** |
| --- | --- | --- | --- | --- |
| Core | Operative procedure | Single selection value list: • Simple hysterectomy • Radical hysterectomy • Other (specify) |  |  |
| Core | Attached anatomical structures | Multi select value list (choose all that apply) • Vaginal cuff • Left ovary • Right ovary • Left fallopian tube • Right fallopian tube • Parametria |  |  |
| Core | Accompanying specimens | Multi select value list (choose all that apply) • None submitted • Peritoneal biopsies • Omentum • Lymph nodes • Other (specify) |  |  |
| Non-core | Tumour site | Multi select value list (choose all that apply) • Fundus  • Body  • Isthmus/lower uterine segment | There may be an association between lower uterine segment/isthmic tumours and Lynch syndrome.1,2 References  1 Westin SN, Lacour RA, Urbauer DL, Luthra R, Bodurka DC, Lu KH and Broaddus RR (2008). Carcinoma of the lower uterine segment: a newly described association with Lynch syndrome. J Clin Oncol 26(36):5965-5971. 2 Masuda K, Banno K and Yanokura M et al (2011). Carcinoma of the Lower Uterine Segment (LUS): Clinico-pathological characteristics and association with Lynch Syndrome. Curr Genomics 12:25-29. |  |
| Non-core | Maximum tumour dimension | Numeric: \_\_\_mm | There is a significant correlation between primary tumour diameter >20 mm and peritoneal failure. This does not yet reach III-2 evidence level.1 References 1 Mariani A, Webb MJ, Keeney GL, Aletti G and Podratz KC (2003). Endometrial cancer: predictors of peritoneal failure. Gyneco Oncol 89:236-242. |  |
| Core | Histological tumour type | Multi select value list (choose all that apply) • Endometrioid carcinoma • Mucinous carcinoma • Serous endometrial intraepithelial carcinoma (SEIC) • Serous carcinoma • Clear cell carcinoma • Mixed cell adenocarcinoma  • Undifferentiated carcinoma • Dedifferentiated carcinoma • Neuroendocrine carcinoma (specify subtype) • Carcinosarcoma | Endometrial carcinomas should be typed according to the 2014 World Health Organisation (WHO) Classification.1 Accurate typing is necessary in both biopsies and resection specimens. Diagnosis of aggressive tumours such as serous carcinoma, clear cell carcinoma, carcinosarcoma, undifferentiated carcinoma and grade 3 endometrioid adenocarcinoma will usually result in full surgical staging including pelvic and para-aortic lymphadenectomy and omentectomy.  Mucinous adenocarcinoma refers to a subtype of endometrial adenocarcinoma in which more than 50% of the tumour cells contain intracytoplasmic mucin. Many endometrioid adenocarcinomas contain focal mucinous areas and endometrioid and mucinous adenocarcinomas form part of a spectrum. Although carcinosarcomas (malignant mixed Müllerian tumours) are still classified as mixed epithelial and mesenchymal tumours in the 2014 WHO Classification,1 their behaviour is similar to other high grade endometrial carcinomas and they are treated in the same way as aggressive endometrial carcinomas. Carcinosarcomas are believed to be epithelial neoplasms that have undergone sarcomatous metaplasia, the epithelial elements being the ‘driving force’.  The 2014 WHO classification of endometrial carcinomas (see below) now includes serous endometrial intraepithelial carcinoma (serous EIC).1 Even in the absence of demonstrable stromal invasion, malignant cells can shed from serous EIC and metastasise widely to extra-uterine sites. Neuroendocrine tumours are also a new addition to the 2014 WHO Classification.1 They are rare primary uterine neoplasms and the diagnosis should be confirmed immunohistochemically, although some small cell neuroendocrine carcinomas may not express neuroendocrine markers (see note on **ancillary studies**). Neuroendocrine neoplasms of the endometrium are divided into low-grade neuroendocrine tumour (carcinoid tumour) which is extremely rare and high-grade neuroendocrine carcinoma (small cell and large cell neuroendocrine carcinoma) which is more common but also rare. Large cell neuroendocrine carcinoma should demonstrate a neuroendocrine growth pattern in at least part of the tumour, and show expression of one or more neuroendocrine markers (chromogranin, synaptophysin, CD56, PGP9.5) in >10% of the tumour. Undifferentiated carcinoma2,3 is defined by WHO as a ‘malignant epithelial neoplasm with no differentiation’,1 and may show immunohistochemical evidence of epithelial differentiation in only occasional tumour cells (see notes on ancillary studies). Dedifferentiated carcinoma4 is defined as an undifferentiated carcinoma that contains a second component of either FIGO grade 1 or 2 endometrioid adenocarcinoma; in such cases, it is believed that the undifferentiated component develops as a result of dedifferentiation in the low-grade endometrioid component.  Mixed carcinomas must contain two or more different histological types of endometrial carcinoma recognisable on H&E-stained sections. At least one of the subtypes must be a type II tumour and the second component, according to the 2014 WHO Classification,1 must comprise at least 5% of the neoplasm. The most common mixture is endometrioid and serous carcinoma. Immunohistochemistry may assist in confirming the presence of a second, morphologically distinct subtype. All subtypes should be specified in the histopathology report, even if <5% of the neoplasm is composed of type II tumour, because the behaviour of these tumours is determined by the highest grade component.1   In cases where there is no residual tumour in the hysterectomy specimen or where there is a significant discrepancy between the reported tumour type in the biopsy and that in the hysterectomy, it may be necessary to review the prior biopsy. If high-risk/aggressive variants of carcinoma e.g. serous carcinoma, carcinosarcoma etc., are confirmed in the endometrial biopsy but are not identified in the final hysterectomy specimen, the carcinoma should be categorised according to the worst histology.   Adequate sampling of the tumour is required (minimum of 4 blocks) to allow meaningful assessment of this data item.   List WHO entities.  References 1 Kurman RJ, Carcangiu ML, Herrington CS and Young RH (2014). WHO classification of tumours of the female reproductive organs. IARC press, Lyon. 2 Silva EG, Deavers MT and Malpica A (2007). Undifferentiated carcinoma of the endometrium: a review. Pathology 39(1):134-138. 3 Tafe LJ, Garg K, Chew I, TornosA. C and Soslow R (2010). Endometrial and ovarian carcinomas with undifferentiated components: clinically aggressive and frequently underrecognized neoplasms. Mod Pathol 23(6):781-789. 4 Silva EG, Deavers MT, Bodurka DC and Malpica A (2006). Association of low-grade endometrioid carcinoma of the uterus and ovary with undifferentiated carcinoma: a new type of dedifferentiated carcinoma? Int J. Gynecol Pathology 25(1):52-58. | Note that permission to publish the WHO classification of tumours may be needed in your implementation. It is advisable to check with the International Agency on Cancer research (IARC) |
| Non-core | Carcinosarcoma | Numeric: \_\_\_\_% epithelial  AND  Numeric: \_\_\_\_% sarcomatous AND record whether the sarcomatoid component is: • Homologous • Heterologous | A recent study has shown that the presence of heterologous elements in stage I carcinosarcomas is an important adverse prognostic feature; this does not yet reach III-2 evidence level.1  References 1 Ferguson SE, Tornos C, Hummer A, Barakat R and Soslow R (2007). Prognostic features of surgical stage I uterine carcinosarcoma. Am J Surg Pathol 31(11):1653-1661. | record only if selected as a tumour type above. |
| Core | Histological grade | Single selection value list: • Grade 1  • Grade 2  • Grade 3 • Not gradeable  • Not applicable | The FIGO grading system for endometrioid adenocarcinomas of the uterine corpus is based on the following architectural features:1  Grade 1: 5% or less non-squamous solid growth pattern Grade 2: 6% to 50% non-squamous solid growth pattern Grade 3: >50% non-squamous solid growth pattern  Notable nuclear atypia, which exceeds that which is routinely expected for the architectural grade, increases the tumour grade by 1. Notable nuclear atypia should be present in >50% of the tumour.2  In addition, the following guidelines should be used in grading: (1) Non-gland forming squamous elements should be disregarded for grading purposes. (2) Endometrioid and mucinous carcinomas should be graded using the FIGO grading system. (3) Serous, clear cell and undifferentiated carcinomas and carcinosarcomas are not graded but are regarded as high grade neoplasms.3 When the dataset is being completed, these should be designated as “not applicable” for histologic grade. (4) In mixed carcinomas, the highest grade should be assigned.  In general, if there is a discrepancy between the grade of an endometrioid adenocarcinoma in the pre-operative biopsy and the final resection specimen, the final histological tumour grade should be based on findings in the hysterectomy specimen, which usually contains a larger volume of tumour for assessment. This is particularly important if the hysterectomy specimen contains abundant low-grade tumour and the biopsy showed grade 3 endometrioid adenocarcinoma. In this specific situation, application of the guidelines for FIGO grading may result in the tumour being downgraded, although this will not always be the case; for example, where the biopsy contained abundant grade 3 endometrioid adenocarcinoma and the hysterectomy a limited amount of low-grade tumour, the final diagnosis might still be grade 3 endometrioid adenocarcinoma.   References 1 Creasman W, Odicino F and Maisonneuve P et al (2001). Carcinoma of the corpus uteri: FIGO Annual Report. J Epidemiol Biostat 6:45-86. 2 Zaino RJ, Kurman RJ, Diana KL and Morrow CP (1991). The utility of the revised International Federation of Gynecology and Obstetrics histologic grading of endometrial adenocarcinoma using a defined nuclear grading system. A Gynecologic Oncology Group study. Cancer 75(1):81-86. 3 FIGO Committee on Gynecological Cancer (2009). Revised FIGO staging for carcinoma of the vulva, cervix and endometrium. Int. J. Gynecol. Obstet. 105:103-104. |  |
| Core | Myometrial invasion | Single selection value list: • None • <50% • ≥ 50% | Depth of invasion should be measured from the endomyometrial junction (not the surface of exophytic tumours) to the deepest focus of tumour invasion. Measurement of the depth of invasion may be rendered difficult by irregularity of the endomyometrial junction, polypoid tumour growth, intramural leiomyomas, adenomyosis and uncommonly by smooth muscle metaplasia within polypoid neoplasms.1 Deep myometrial invasion has repeatedly been shown to be an important poor prognostic indicator in endometrial carcinoma. This is an independent predictor of haematogenous dissemination by endometrial carcinoma and it is therefore an important determinant of adjuvant therapy.2  References 1 Ali A, Black D and Soslow R (2007). Difficulties in Assessing the Depth of Myometrial Invasion in Endometrial Carcinoma Int J. Gynecol Pathology 26:155-123. 2 Mariani A, Webb MJ, Keeney GL, Calori G and Podratz KC (2001). Hematogenous dissemination in corpus cancer. Gynecol Oncol 80:233−238. |  |
| Non-core | Percentage of myometrium infiltrated by carcinoma | Numeric: \_\_\_\_% | Tumour-free distance (to the uterine serosa) and percentage of myometrium infiltrated are independent prognostic factors for lymph node metastasis in endometrial carcinoma but studies do not reach level III-2 evidence.1  The percentage of myometrium infiltrated by carcinoma is defined as the percentage of myometrium involved as determined by the depth of myometrial invasion from the endomyometrial junction to the deepest focus of invasive carcinoma in comparison to the overall myometrial thickness.   References 1 Kondalsamy-Chennakesavan S, van Vugt S, Sanday K, Nicklin J, Land R, Perrin L, Crandon A and Obermair A (2010). Evaluation of tumor-free distance and depth of myometrial invasion as prognostic factors for lymph node metastases in endometrial cancer. Int J Gynecol Cancer 20:1217-1221. | Applicable is <50% or ≥ 50% is chosen for myometrial invasion above |
| Non-core | Distance of myoinvasive tumour to serosa | Numeric: \_\_\_\_mm | Tumour-free distance and percentage of myometrium infiltrated are independent prognostic factors for lymph node metastasis in endometrial carcinoma; studies do not reach level III-2 evidence.1  References 1 Kondalsamy-Chennakesavan S, van Vugt S, Sanday K, Nicklin J, Land R, Perrin L, Crandon A and Obermair A (2010). Evaluation of tumor-free distance and depth of myometrial invasion as prognostic factors for lymph node metastases in endometrial cancer. Int J Gynecol Cancer 20:1217-1221. |  |
| Core | Lymphovascular invasion | Single selection value list: • Present (specify site) • Not identified  • Indeterminate | Lymphovascular invasion is a predictor of tumour recurrence and lymph node metastasis.1 However, lymphovascular space invasion does not alter the tumour stage. For example, if an endometrial adenocarcinoma is confined to the inner half of the myometrium but shows lymphovascular invasion in the outer half of the myometrium, this should still be staged as FIGO 1A. Similarly lymphovascular invasion alone in cervical, parametrial or para-ovarian vessels does not upstage the tumour. There is an increased incidence of vascular pseudoinvasion in laparoscopic hysterectomy specimens associated with the use of an intrauterine balloon manipulator.1,2  References 1 Watari H, Todo Y, Takeda M, Ebina Y, Yamamoto R and Sakuragi N (2005). Lymph\_vascular space invasion and number of positive para\_aortic node groups predict survival in node\_positive patients with endometrial cancer. Gynecol Oncol 96:651−657. 2 Logani S, Herdman AV, Little JV and Moller KA (2008). Vascular "pseudo invasion" in laparoscopic hysterectomy specimens: a diagnostic pitfall. Am J Surg Pathol 32:560-565. |  |
| Non-core | Cervical surface or crypt involvement | Single selection value list: • Present • Not identified  • Indeterminate | Not necessary for staging but some oncologists administer vault brachytherapy if this is identified. Level III-2 evidence currently not available. |  |
| Core | Cervical stromal invasion | Single selection value list: • Present • Not identified  • Indeterminate | Cervical stromal infiltration by endometrial carcinoma is associated with a risk of recurrence and is a predictor of pelvic lymph node metastases.1,2   References  1 Mariani A, Webb MJ, Keeney GL and Podratz KC (2001). Routes of lymphatic spread: a study of 112 consecutive patients with endometrial cancer. Gynecol Oncol 81:100−104. 2 Fanning J, Alvarez PM, Tsukada Y and Piver MS (1991). Prognostic significance of the extent of cervical involvement by endometrial cancer. Gynecol Oncol 40:46−47. |  |
| Non-core | Distance of tumour to cervical resection margins | Numeric: \_\_\_\_mm | Close margins may indicate a need for vault brachytherapy. Vascular invasion at cervical resection margin should be documented but does not upstage the tumour. |  |
| Core | Vagina | Single selection value list: • Involved • Not involved  • Not applicable |  |  |
| Core | Omentum | Single selection value list: • Involved • Not involved  • Not applicable |  |  |
| Core | Peritoneal biopsy/biopsies | Single selection value list: • Involved • Not involved  • Not applicable |  |  |
| Core | Uterine serosa | Single selection value list: • Involved • Not involved  • Indeterminate | Carcinoma should penetrate through the serosa in order to be classified as serosal involvement. Involvement of the serosa (FIGO stage IIIA) carries a higher risk of locoregional recurrence than does adnexal involvement (also FIGO stage IIIA).1   References 1 Jobsen JJ, Naudin Ten Cate L, Lybeert ML, Scholten A, van der Steen-Banasik EM, van der Palen J, Stenfert Kroese MC, Slot A, Schutter EM and Siesling S (2011). Outcome of Endometrial Cancer Stage IIIA with Adnexa or Serosal Involvement Only. Obstet Gynecol Int.:doi: 10.1155/2011/962518. |  |
| Core | Parametria | Single selection value list: • Involved • Not involved  • Not applicable | Most hysterectomies for endometrial cancer will be simple hysterectomies and will not have parametrial resections. Endometrial carcinomas with parametrial invasion are staged as FIGO IIIB. Although not an independent prognostic indicator, parametrial involvement by direct extension is a poor prognostic factor and also correlates with other poor prognostic factors. The presence of lymphovascular invasion in parametrial tissues should be documented but does not constitute parametrial involvement.1,2  References 1 Sato R, Jobo T and Kuramoto H (2003). Parametrial spread is a prognostic factor in endometrial carcinoma. Eur J Gynaecol Oncol 24:241−245. 2 Yura Y, Tauchi K, Koshiyama M, Konishi I, Yura S and Mori T et al (1996). Parametrial involvement in endometrial carcinomas: its incidence and correlation with other histological parameters. Gynecol Oncol 63:114−119. |  |
| Core | Adnexa | Single selection value list: • Involved • Not involved  • Not applicable | FIGO staging is based on tumour involvement of either the fallopian tube or ovary (stage IIIA). Especially with low-grade endometrioid adenocarcinomas, involvement of the uterine corpus and adnexa may indicate synchronous, independent neoplasms rather than metastasis from the endometrium to the adnexa; a variety of pathological parameters is useful in the distinction between synchronous independent and metastatic neoplasms. As for other sites in the gynaecological tract in which lymphovascular invasion by endometrial adenocarcinoma may be identified e.g., myometrium and parametrial tissue, the identification of lymphovascular space invasion alone in adnexal structures does not alter the tumour stage i.e. endometrial carcinoma should not be upstaged if there is vascular involvement in the adnexa in the absence of tumour outside of vascular channels. |  |
| Non-core | Background endometrium | Multi select value list (choose all that apply) • Cyclical • Atrophic • Polyp/s  • Hormone effect  • Hyperplasia without atypia • Atypical hyperplasia/Endometrial intraepithelial neoplasia | The appearance of the background endometrium and the presence of abnormalities such as hyperplasia or polyps, should be documented. |  |
| Non-core | Peritoneal cytology | Single selection value list: • Positive • Negative • Atypical/suspicious • Not submitted | This data item is not necessary for staging but there is lack of consensus in the literature regarding the prognostic significance of positive peritoneal washings in the absence of other evidence of extrauterine spread. A recommendation is made by FIGO and Union for International Cancer Control (UICC) to record positive peritoneal washings without altering the tumour stage.1,2  References 1 Grimshaw RN, Tupper WC, Fraser RC, Tompkins MG and Jeffrey JF (1990). Prognostic value of peritoneal cytology in endometrial carcinoma. Gynecol Oncol 36:97−100. 2 Fadare O, Mariappan MR, Hileeto D, Wang S, McAlpine JN and Rimm DL (2005). Upstaging based solely on positive peritoneal washing does not affect outcome in endometrial cancer. Mod Pathol 18:673−680. |  |
| Core | Lymph node status | Single selection value list: • Involved • Not involved  • Not applicable | Pelvic and para-aortic node status should be recorded separately since this affects tumour stage. Pelvic node involvement without para-aortic involvement is stage IIIC1 while para-aortic node involvement is stage IIIC2.1,2   Note that micrometastases (greater than 0.2 mm but not greater than 2.0 mm in diameter) are regarded as lymph node involvement and N1mi or N2mi while metastases greater than 2.0 mm in maximum dimension are classified as N1a or N2a. Isolated Tumour Cells (ITCs), in common with TNM8 staging practices at other tumour sites, are regarded as node negative (N0(i+)).  The number of nodes involved and the site of involvement is prognostically important and may direct adjuvant treatment.  References 1 Morrow CP, Bundy BN, Kurman RJ, Creasman WT, Heller P and Homesley HD et al (1991). Relationship between surgical\_pathological risk factors and outcome in clinical stage I and II carcinoma of the endometrium: a Gynecologic Oncology Group study. Gynecol Oncol 40:55−65. 2 Hoekstra AV, Kim RJ, Small W Jr, Rademaker AW, Helenowsky IB and Singh DK et al (2009). FIGO stage IIIC endometrial carcinoma: Prognostic factors and outcomes. Gynecol Oncol 114:273−278. |  |
| Non-core | Left pelvic number retrieved | Numeric: \_\_\_\_ |  | Applicable if involved selected above. |
| Non-core | Left pelvic number involved | Numeric: \_\_\_\_ |  | Applicable if involved selected above. |
| Non-core | Right pelvic number retrieved | Numeric: \_\_\_\_ |  | Applicable if involved selected above. |
| Non-core | Right pelvic number involved | Numeric: \_\_\_\_ |  | Applicable if involved selected above. |
| Non-core | Para-aortic number retrieved | Numeric: \_\_\_\_ |  | Applicable if involved selected above. |
| Non-core | Para-aortic number involved | Numeric: \_\_\_\_ |  | Applicable if involved selected above. |
| Non-core | Extra-nodal spread | Single selection value list: • Present • Not identified • Not applicable |  | Applicable if involved selected above. |
| Core | Histologically confirmed distant metastases | Single selection value list: • Present • Not identified • Indeterminate |  |  |
|  | ANCILLARY STUDIES |  | Immunohistochemistry may be useful in certain diagnostic scenarios. For example, a panel of markers (ER, PR, vimentin, CEA, p16) may be useful in the distinction between a primary endometrial and cervical adenocarcinoma.1-2 Other markers (ER, PR, p53, p16, PTEN, IMP3) may be useful in the distinction between an endometrioid and a serous adenocarcinoma.3-4 p53 and p16 may help to highlight serous EIC and distinguish this from surface atypias which can mimic it. Immunohistochemistry for mismatch repair proteins (MLH1, MSH2, MSH6, PMS2) may be useful in helping to establish whether endometrial carcinomas are associated with underlying mismatch repair gene abnormalities and Lynch syndrome (hereditary non-polyposis colorectal cancer) . 5-6  Undifferentiated endometrial carcinomas are often only focally, but characteristically intensely, positive with broad spectrum cytokeratins, CK18 and epithelial membrane antigen (EMA). This may be useful in the distinction from an undifferentiated sarcoma or other neoplasms and may also help to establish a diagnosis of dedifferentiated carcinoma when a component of low-grade endometrioid adenocarcinoma is present.7-9 Some undifferentiated carcinomas exhibit focal expression of neuroendocrine markers.10   High-grade neuroendocrine carcinomas are usually positive with the neuroendocrine markers chromogranin, synaptophysin, CD56 and PGP9.5. Some small cell neuroendocrine markers are negative with these markers but usually at least one is positive. Large cell neuroendocrine carcinomas should be positive with at least one of these markers in >10% of tumour cells.   Different morphological subtypes of endometrial adenocarcinoma are associated with distinct molecular abnormalities. However, at present molecular analysis has little role in diagnosis or as an independent prognostic or predictive factor. However, this may change in the future and it is likely that targeted therapies will be developed against carcinomas exhibiting specific molecular abnormalities.  References 1 Castrillon DH, Lee KR and Nucci MR (2002). Distinction between endometrial and endocervical adenocarcinoma: an immunohistochemical study. Int J Gynecol Pathol 21:4-10. 2 McCluggage WG, Sumathi VP, McBride HA and Patterson A (2002). A panel of immunohistochemical stains, including carcinoembryonic antigen, vimentin, and estrogen receptor, aids the distinction between primary endometrial and endocervical adenocarcinomas. Int J Gynecol Pathol 21:11-15. 3 McCluggage WG (2007). Immunohistochemistry as a diagnostic aid in cervical pathology. Pathology 39(1):97-111. 4 Yemelyanova A, Hongxiu J, Shih I, Wang T, Wu L and Ronnett B (2009). Utility of p16 expression for distinction of uterine serous carcinomas from endometrial endometrioid and endocervical adenocarcinomas. Am J Surg Pathol 33:1504-1514. 5 Walsh MD, Cummings MC, Buchanan DD, Dambacher WM, Arnold S, McKeone AS and Byrnes R et al (2008). Molecular, pathologic, and clinical features of early-onset endometrial cancer: identifying presumptive Lynch syndrome patients. Clin. Cancer Res. 14(6):1692-1700. 6 Garg K and Soslow RA (2009). Lynch syndrome (hereditary non-polyposis colorectal cancer) and endometrial carcinoma. J Clin Pathol 62:679-684. 7 Silva EG, Deavers MT, Bodurka DC and Malpica A (2006). Association of low-grade endometrioid carcinoma of the uterus and ovary with undifferentiated carcinoma: a new type of dedifferentiated carcinoma? Int J. Gynecol Pathology 25(1):52-58. 8 Silva EG, Deavers MT and Malpica A (2007). Undifferentiated carcinoma of the endometrium: a review. Pathology 39(1):134-138. 9 Tafe LJ, Garg K, Chew I, TornosA. C and Soslow R (2010). Endometrial and ovarian carcinomas with undifferentiated components: clinically aggressive and frequently underrecognized neoplasms. Mod Pathol 23(6):781-789. 10 Taraif SH, Deavers MT, Malpica A and Silva EG (2009). The significance of neuroendocrine expression in undifferentiated carcinoma of the endometrium. Int J. Gynecol Pathology 28(2):142-147. | Heading |
| Non-core | Immunohistochemical markers | Text |  |  |
| Non-core | Molecular data | Text |  | Heading |
|  | PROVISIONAL PATHOLOGICAL STAGING PRE-MDTM |  |  | Heading  Note: MDMT = Multidisciplinary management team |
| Core | Provisional FIGO stage (2009) | Per FIGO staging values 2009. | Staging is provisional since final stage should be determined at multidisciplinary team/tumour board meeting when all relevant clinical and radiological information is available.1,2 Since serous EIC is regarded as a type of endometrial carcinoma, it is staged as FIGO stage IA (T1a).  The reference document TNM Supplement: A commentary on uniform use, 4th Edition (C Wittekind editor) may be of assistance when staging.3   References 1 FIGO Committee on Gynecological Cancer (2009). Revised FIGO staging for carcinoma of the vulva, cervix and endometrium. Int. J. Gynecol. Obstet. 105:103-104. 2 Amin MB, Edge SB and Greene FL et al (eds) (2017). AJCC Cancer Staging Manual. 8th ed., Springer, New York. 3 Wittekind C (ed) (2012). TNM Supplement : A Commentary on Uniform Use, The Union for International Cancer Control (UICC), Wiley-Blackwell. |  |
| Non-core | Pathological staging (TNM 8th ed.) | Per AJCC 8th edition |  | Note that permission to publish the AJCC cancer staging tables may be needed in your implementation. It is advisable to check with AJCC. |