Case Report

Management of Incompletely Staged Endometrial Carcinoma: A Case-Based Review

Muhammad Fakhmi, Andi Kurniadi
Department of Obstetrics and Gynecology, Faculty of Medicine Universitas Padjadjaran – Dr. Hasan Sadikin General Hospital Bandung
Corresponding: Muhammad Fakhmi, Email: muhammadfakhmi18@gmail.com
Received: July 7, 2025 | Accepted: March 11, 2026 | Published: March 13, 2026

Abstract
Introduction: Endometrial carcinoma is the most common gynecologic malignancy in developed countries, driven by risk factors such as obesity and unopposed estrogen exposure. Early-stage detection is common due to early symptom onset. Surgical management remains the cornerstone of therapy and serves a dual role in staging the disease. This report presents the initial management of atypical endometrial hyperplasia, evaluation strategies, and treatment planning in a case of advanced-stage endometrial carcinoma with incomplete surgical staging.
Case Illustration: A 50-year-old woman presented with persistent vaginal discharge one week following a supracervical hysterectomy and bilateral salpingo-oophorectomy was referred to Hasan Sadikin Hospital Bandung performed for presumed benign disease. Histopathological and immunohistochemical evaluations confirmed the diagnosis of endometrioid adenocarcinoma of endometrial origin. Although chest imaging was negative for distant metastases, further evaluation suggested local extension consistent with FIGO Stage IIIB. Given the incomplete surgical staging and advanced local disease, the patient was referred for multimodal adjuvant therapy consisting of External Beam Radiotherapy (EBRT) and vaginal brachytherapy.
Conclusion: Total hysterectomy with bilateral salpingo-oophorectomy and appropriate surgical staging per FIGO guidelines represents the standard treatment for endometrial carcinoma. This case highlights that tailored adjuvant radiotherapy (EBRT and brachytherapy) is critical for effective disease management in incompletely staged high-grade or advanced-stage patients.

Key words: Hysterectomy; endometrial carcinoma; management; radiotherapy; incomplete stage endometrial cancer

Manajemen Karsinoma Endometrium dengan Stadium Inkomplit: Tinjauan Berbasis Kasus

Abstrak
Pendahuluan: Karsinoma endometrium merupakan kanker ginekologi yang paling sering terjadi di negara-negara maju, dengan angka kejadian yang terus meningkat secara global. Faktor risiko utama meliputi obesitas, gangguan metabolik, serta paparan estrogen tanpa antagonis progesteron. Deteksi dini sering terjadi karena gejala muncul pada tahap awal penyakit. Penanganan bedah tetap menjadi pilar utama terapi dan sekaligus berperan dalam penentuan stadium penyakit. Laporan ini memaparkan tata laksana awal hiperplasia endometrium atipikal, strategi evaluasi, serta perencanaan terapi pada kasus karsinoma endometrium stadium lanjut dengan staging bedah yang tidak lengkap.
Ilustrasi Kasus: Seorang wanita berusia 50 tahun dengan diagnosis karsinoma endometrium dan riwayat histerektomi supraservikal serta salpingoovarektomi bilateral dirujuk ke Rumah Sakit Hasan Sadikin Bandung dengan keluhan keluarnya lendir berwarna kecokelatan dari vagina yang menetap satu minggu pascaoperasi. Pemeriksaan histopatologi dan imunohistokimia mengonfirmasi diagnosis adenokarsinoma endometrioid yang berasal dari endometrium. Tidak ditemukan metastasis pulmonal pada pemeriksaan radiologi. Pemeriksaan laboratorium menunjukkan anemia ringan, leukositosis, dan trombositosis. Diagnosis akhir yang ditegakkan adalah karsinoma endometrium suspek stadium IIIB menurut FIGO. Pasien kemudian dirujuk untuk menjalani radioterapi eksternal (EBRT) dan brakiterapi vagina sebagai terapi adjuvan.
Kesimpulan: Histerektomi total disertai salpingoovarektomi bilateral dan staging bedah yang sesuai dengan pedoman FIGO merupakan standar penatalaksanaan karsinoma endometrium. Terapi adjuvan seperti brakiterapi vagina dan EBRT berperan penting dalam penanganan kasus dengan staging yang tidak lengkap, seperti yang dijumpai pada pasien ini.

Kata kunci: Histerektomi; karsinoma endometrium; manajemen; radioterapi; stadium inkomplit karsinoma endometrium

 DOI:10.24198/obgynia.v9i1.959

Introduction

Endometrial carcinoma is a neoplasm originating from the endometrial lining and represents the most common gynecologic malignancy in developed countries. The disease typically presents with symptoms at an early stage, allowing the majority of cases to be detected during stage I.[1] Several established risk factors contribute to the pathogenesis of endometrial carcinoma, particularly obesity and components of metabolic syndrome, such as diabetes mellitus and polycystic ovary syndrome (PCOS). Additionally, prolonged exposure to unopposed estrogen, whether originating from estrogen-secreting tumors or estrogen-only hormone replacement therapy without concomitant progestin, is strongly associated with an increased risk of malignancy.[2]

Globally, the incidence of endometrial carcinoma continues to rise. Age-adjusted incidence rates increased by approximately 0.69% annually between 1990 and 2019.[3] As of 2022, endometrial carcinoma ranked as the sixth most common cancer among women, with an estimated 420,368 new cases worldwide.[4] In the United States, the incidence has grown by over 1% annually since the mid-2000s, with a marked rise observed between 2012 and 2021: 0.6% annually among white women and 2–3% among other racial and ethnic groups.[5] This upward trend may be attributed, in part, to a decline in hysterectomy rates for benign indications, which results in a larger population of women retaining their uterus and remaining at risk for endometrial malignancy.[6]

Atypical endometrial hyperplasia (AEH) is widely recognized as the principal precursor lesion of endometrioid adenocarcinoma. Studies indicate that up to 50% of women diagnosed with AEH may harbor concurrent occult endometrial carcinoma.[7] Consequently, definitive surgical management is often recommended to mitigate the risk of progression or missed malignancy.

The standard primary treatment for endometrial carcinoma is total hysterectomy, which concurrently serves as a staging procedure. Surgical staging is vital for prognostic stratification and determining the necessity of adjuvant therapy, such as chemotherapy or radiotherapy.[6] In the present case, a patient initially underwent endometrial curettage revealing hyperplasia, followed by a supracervical hysterectomy. However, final histopathology unexpectedly confirmed endometrial carcinoma. Postoperatively, the patient presented with persistent brownish vaginal discharge, and a working diagnosis of suspected stage IIIB endometrial carcinoma was established. This report discusses the challenges in managing incompletely staged advanced endometrial carcinoma, reviewing diagnostic strategies and therapeutic approaches for incidental malignancy following supracervical procedures.

Case Illustration

A 50-year-old woman, Gravida 1, Para 0 (G1P0A1), was referred from Pertamina Balongan Hospital with a preliminary diagnosis of endometrial carcinoma. The patient presented with complaints of persistent brownish vaginal discharge that developed one week following a supracervical hysterectomy and bilateral salpingo-oophorectomy (BSO), performed on February 13, 2025. She denied abdominal pain, palpable abdominal masses, or malodorous vaginal discharge. However, she reported unintentional weight loss of 4 kg over the preceding month. She reported no bowel or urinary complaints and had no history of chronic comorbidities such as hypertension, diabetes mellitus, asthma, or cardiovascular disease.

The patient had been married for 37 years. Menarche occurred at age 15, with previously regular menstrual cycles every

28 days lasting 5–7 days. The date of her last menstrual period was unknown. A history of oral contraceptive use was noted.

Upon presentation, the patient was alert and hemodynamically stable. Vital signs were within normal limits: blood pressure 118/83 mmHg, heart rate 91 bpm, respiratory rate 20 breaths per minute, and temperature 36.2°C. Anthropometric measurements revealed a weight of 59 kg and height of 152 cm, corresponding to a Body Mass Index (BMI) of 25.5 kg/m² (overweight) and a Body Surface Area (BSA) of 1.58 m². Abdominal examination revealed a soft, flat abdomen without tenderness, muscle guarding, shifting dullness, or palpable masses. Pelvic examination demonstrated normal vulvar and vaginal anatomy. A cervical stump was present and smooth; the uterine corpus was absent due to prior surgery. The adnexal regions were free of palpable masses, and the pouch of Douglas was within normal limits.

The patient's relevant history began with an episode of vaginal bleeding between April and June 2024, for which she underwent endometrial curettage at Pertamina Balongan Hospital. Histopathological analysis on June 26, 2024, revealed complex endometrial glandular hyperplasia with atypia. Regrettably, the patient was lost to follow-up after this procedure.

In January 2025, she experienced a recurrence of symptoms and returned for treatment. A total abdominal hysterectomy with BSO was initially planned; however, intraoperative findings of dense pelvic adhesions necessitated a supracervical hysterectomy with BSO, performed on February 13, 2025. Histopathological examination of the surgical specimen revealed a Grade 2 endometrioid adenocarcinoma with invasion into half the thickness of the myometrium, accompanied by a uterine leiomyoma. No ovarian or tubal abnormalities were reported.

Subsequent evaluation was conducted at Dr. Hasan Sadikin General Hospital (RSHS). Histopathological review on March 11, 2025, confirmed a well-differentiated endometrioid adenocarcinoma. To exclude a primary endocervical malignancy arising from the cervical stump, immunohistochemical staining was performed using markers including vimentin, Ki-67, CK7, and CK20. The tumor cells were negative for p16 and positive for Progesterone Receptor (PR) and Vimentin. This supported a diagnosis of endometrioid adenocarcinoma of endometrial origin rather than a cervical primary.

Figures 1–3 illustrate the findings from oncologic ultrasonography performed at RSHS on May 6, 2025.

Laboratory evaluation on May 7, 2025, using hematological analysis revealed a hemoglobin level of 11.1 g/dL (low), a hematocrit of 36.0% (at the lower limit of normal), and an erythrocyte count of 4.70 million/µL (within the normal range). The

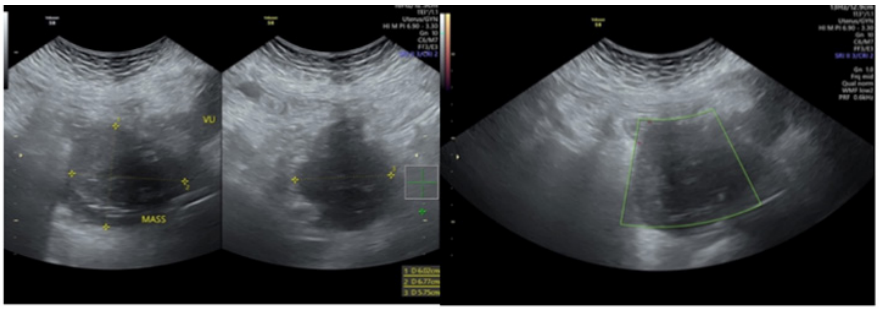

Figure 1 Oncology Ultrasound (1st Part)

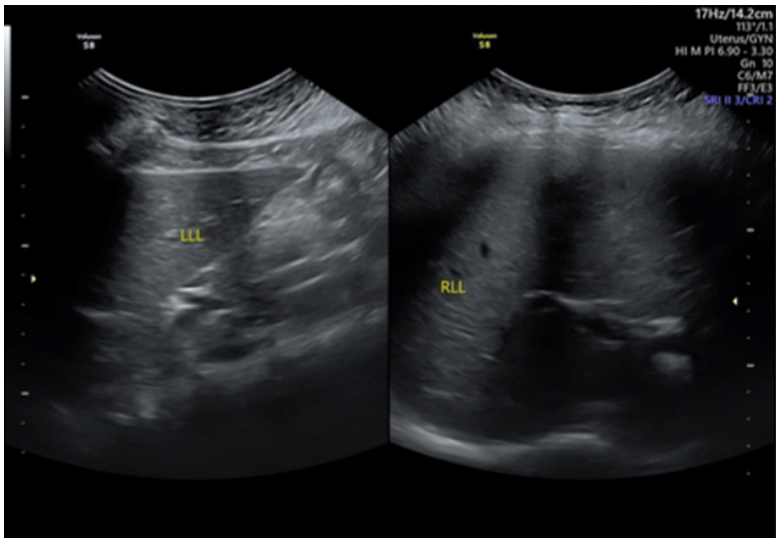

Figure 2 Oncology Ultrasound (2nd Part)

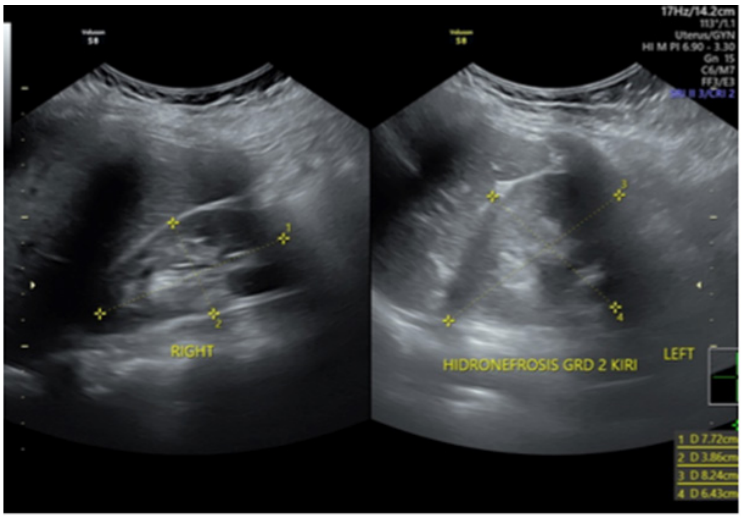

Figure 3 Oncology Ultrasound (3rd Part)

leukocyte count was elevated at 12.67 × 10^3/µL, and a marked thrombocytosis was present, with a platelet count of 634 × 10^3/µL. The differential white cell count showed basophils and eosinophils at 0%, band neutrophils at 0% (low), segmented neutrophils at 67%, lymphocytes at 26%, and monocytes at 7%. A chest radiograph performed on the same date showed no evidence of intrapulmonary metastases or cardiomegaly (Figure 4).

Based on the clinical progression, imaging, and pathological findings, the patient was diagnosed with suspected FIGO Stage IIIB endometrial carcinoma, defined by local extension into the parametrium (Figure 5). Given the incomplete surgical staging (retained cervix) and local advancement, she was referred to the Radiation Oncology department. The treatment plan included external beam radiotherapy (EBRT) combined with vaginal brachytherapy to address the high risk of local recurrence.

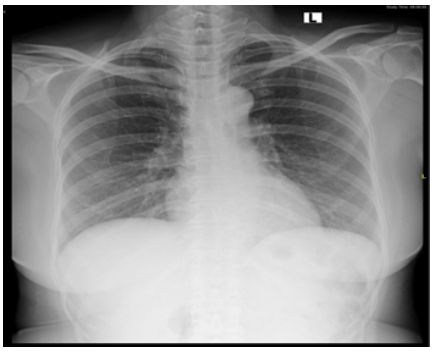

Figure 4 Patient's Chest Radiograph

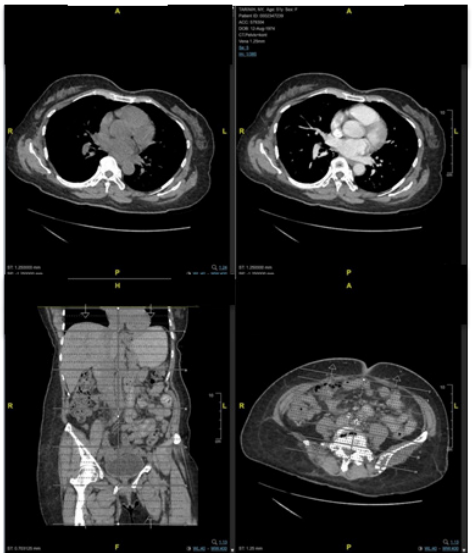

Figure 5 Contrast-Enhanced CT Scan of The Patient Demonstrating Extension of Endometrial Carcinoma and Suggesting Parametrial Involvement

Discussion

Endometrial hyperplasia, particularly its atypical form, is considered the primary precursor lesion to endometrioid adenocarcinoma of the endometrium. In 1983, Bokhman proposed two distinct pathogenetic types of endometrial carcinoma, each influenced by different metabolic and endocrine mechanisms: Type I, estrogen-dependent, and Type II, estrogen-independent. Type I endometrial carcinoma is typically associated with low-grade endometrioid histology and is thought to arise from endometrial hyperplasia as its precursor lesion.

Endometrial hyperplasia is defined as an abnormal proliferation of endometrial glands due to prolonged estrogen stimulation without adequate counteraction by progesterone, a hormonal imbalance often referred to clinically as "unopposed estrogen". This condition is frequently observed in patients with obesity, chronic anovulation, early menarche, late menopause, or estrogen-secreting tumors.[7,8]

Histologically, endometrial hyperplasia is characterized by abnormal glandular proliferation, with an increased gland-to-stroma ratio compared with normal proliferative endometrium, but without invasion into the endometrial stroma. The

diagnosis is confirmed by histopathological examination of tissue obtained through surgical procedures such as endometrial biopsy, curettage, or hysterectomy. Clinical management of endometrial hyperplasia depends on the presence or absence of nuclear atypia, defined by nuclear enlargement, with or without prominent nucleoli. Endometrial hyperplasia without atypia rarely progresses to endometrial carcinoma. In contrast, atypical endometrial hyperplasia is associated with a significantly increased risk of malignancy. Studies have shown that the prevalence of coexisting endometrial carcinoma in patients diagnosed with atypical endometrial hyperplasia can be as high as 50%. Another study reported a prevalence of 32.6% (95% CI: 24.1%–42.4%) for coexisting endometrial carcinoma in women with atypical hyperplasia, with an annual progression rate to cancer of 8.2% (95% CI: 3.9%–17.3%) [9]

Most patients diagnosed with endometrial hyperplasia present with abnormal uterine bleeding, such as postmenopausal bleeding or persistent or recurrent uterine bleeding. Initial clinical assessment should include a detailed history of current symptoms and menstrual history. Definitive diagnosis of endometrial hyperplasia and evaluation for atypia require histological examination of endometrial tissue, which may be obtained through outpatient or inpatient sampling procedures. In the present case, the patient initially presented with abnormal vaginal bleeding lasting one month. She subsequently underwent curettage at Pertamina Balongan Hospital for histopathological evaluation.[7,8]

The management algorithm for endometrial hyperplasia in symptomatic patients typically begins with dilatation and curettage (D&C) to exclude endometrial carcinoma. If the patient desires uterine preservation, initial therapy involves administering medroxyprogesterone acetate (Provera), tailored to the patient's hormonal status—whether premenopausal or postmenopausal—with or without the addition of a levonorgestrel-releasing intrauterine device (LNG-IUD).[9]

A follow-up endometrial biopsy is recommended within 3 to 6 months. If persistent hyperplasia is found, treatment is escalated to high-dose progestin therapy for another three months. Hysterectomy is indicated for persistent disease after hormonal therapy. In patients not desiring future fertility, definitive management with hysterectomy may be pursued immediately. If histologic evaluation shows a normal or atrophic endometrium, continued low-dose Provera therapy is recommended, along with annual surveillance using transvaginal ultrasonography. Ovulation induction may be considered in women seeking fertility.[7-9]

This algorithm underscores the importance of an individualized approach based on reproductive goals and hormonal response. Progestin treatment has shown higher rates of histologic resolution (89%–96%) than expectant management (74%–81%). Progestin therapy is also the mainstay of conservative treatment for atypical endometrial hyperplasia and stage IA endometrioid-type endometrial carcinoma without myometrial invasion. The combination of GnRH analogs, metformin, and hysteroscopic resection with progestin therapy has been reported to enhance overall therapeutic outcomes.[7,10]

Furthermore, studies indicate that progestin therapy reduces the risk of progression to endometrial carcinoma by three- to fivefold in women with atypical hyperplasia. The Royal College of Obstetricians and Gynaecologists (RCOG) recommends total hysterectomy in women with atypical hyperplasia because of the substantial risk of progression to carcinoma. A laparoscopic approach is preferred over laparotomy because it is associated with shorter hospital stays, reduced postoperative pain, and faster

recovery.[11,12]

In postmenopausal women with atypical hyperplasia, total hysterectomy should be performed with bilateral salpingo-oophorectomy. A large cohort study of 7,947 women with atypical hyperplasia reported a cumulative risk of developing endometrial carcinoma of 8% (95% CI: 1.31–14.6%) within 4 years, increasing to 12.4% (95% CI: 3.0–20.8%) within 9 years, and reaching 27.5% (95% CI: 8.6–42.5%) within 19 years. Additionally, concomitant carcinoma has been reported in up to 43% of women undergoing hysterectomy for atypical hyperplasia.[11,12]

Therefore, total hysterectomy is considered the treatment of choice for women diagnosed with atypical endometrial hyperplasia. In the present case, the initial histopathological examination revealed complex endometrial glandular hyperplasia with focal atypia. However, the patient failed to return for follow-up after the diagnosis. According to current recommendations, total hysterectomy would be an appropriate management option, particularly given the patient's age of 50 years and lack of reproductive desire.

Conventional histopathological examination remains a cornerstone in diagnosing endometrial pathology, providing critical information on tumor histomorphologic subtype and grade. Tissue sampling techniques include endometrial curettage, hysteroscopy-assisted curettage, and endometrial biopsy. Although widely used in routine clinical practice, these methods have limitations, particularly discrepancies between preoperative and postoperative findings, which can significantly affect clinical decision-making, especially in treatment planning.13,14 Endometrial carcinoma is traditionally classified into two major types. Type I carcinoma is generally low-grade and estrogen-dependent, frequently arising from underlying endometrial hyperplasia, and is typically associated with a favorable prognosis. These tumors are often diagnosed at an early stage and respond well to surgical and hormonal therapy.

In contrast, Type II carcinoma is high-grade and estrogen-independent, usually arising from atrophic endometrium or precursor lesions, and is more often diagnosed at advanced stages, with a poorer prognosis. Histopathologically, endometrial carcinoma can be classified into several subtypes, the most common being endometrioid carcinoma, serous carcinoma, carcinosarcoma, and clear cell carcinoma. Among these, clear cell and serous carcinomas are Type II or non-endometrioid carcinomas, which, although less common, are notably more aggressive than Type I tumors. Type II carcinomas represent only 10–20% of cases and are less well studied, with most categorized as serous-like.[13,14]

In this case, histopathological and immunohistochemical examinations confirmed the diagnosis of endometrioid adenocarcinoma. According to the International Federation of Gynecology and Obstetrics (FIGO) guidelines, patients with endometrial carcinoma should undergo total hysterectomy, bilateral salpingo-oophorectomy, and surgical staging, including peritoneal washing and lymphadenectomy. Clinical staging requires a comprehensive evaluation, including patient history, physical examination, and radiologic studies (e.g., chest X-ray, intravenous urography, or CT scan), as well as preoperative endometrial biopsy. Although pelvic or peritoneal wash cytology is performed, it has not been shown to significantly affect staging outcomes.[15]

Surgical staging procedures typically include total abdominal hysterectomy or vaginal hysterectomy with laparoscopic assistance, bilateral salpingo-oophorectomy, and pelvic and para-aortic lymph node evaluation. Selective lymphadenectomy may be used in early-stage disease. Although laparotomy was historically the standard

approach, minimally invasive procedures such as laparoscopy or robotic-assisted surgery are increasingly preferred, particularly for tumors confined to the uterus, because they are associated with lower postoperative complication rates, reduced risk of infection, bleeding, and venous thromboembolism, and shorter hospitalization, without compromising oncologic outcomes.[14-16] FIGO also recommends sentinel lymph node biopsy (SLNB) in all patients to assess nodal metastasis. In patients with intermediate- to high-risk disease, either lymphadenectomy or SLNB may be used for accurate staging. In premenopausal women, ovarian preservation may be considered in low-risk cases, as evidence suggests this may improve overall survival in young women with early-stage disease.[14]

The 2023 FIGO staging system introduced new substages within Stage I (IA1, IA2, IA3, IB, IC) and Stage II (IIA, IIB, IIC), based on histologic type, depth of myometrial invasion, and lymphovascular space invasion (LVSI). This updated classification provides more refined stratification based on histologic and anatomic criteria, distinguishing aggressive from nonaggressive histologic types and setting specific thresholds for LVSI and cervical stromal invasion.[16] In the current case, the suspected FIGO stage is IIIB, indicating direct extension or metastasis to the parametrial tissue.[17,18]

The 2021 guidelines of the European Society of Gynaecological Oncology (ESGO) provide comprehensive recommendations for the management of endometrial carcinoma, based on disease stage:[18]

- Stage I: The primary treatment is total hysterectomy with bilateral salpingo-oophorectomy. Lymph node assessment is performed selectively based on risk factors such as tumor grade and depth of myometrial invasion. A minimally invasive surgical approach is recommended whenever feasible.
- Stage II: For tumors that invade the cervical stroma but remain confined to the uterus, radical hysterectomy with bilateral salpingo-oophorectomy and pelvic lymph node dissection is indicated. Adjuvant radiotherapy may be considered based on pathological findings and individual risk assessment.
- Stage III: Characterized by regional or local spread, treatment usually consists of a multimodal approach, including surgery, chemotherapy, and radiotherapy. Surgical resection is tailored to the disease burden, and adjuvant therapy is personalized based on the patient's risk profile.
- Stage IV: In the presence of distant metastases, systemic chemotherapy becomes the mainstay of treatment. Radiotherapy or surgery may be used for symptom control or to manage localized disease, guided by the patient's overall condition and preferences.

Management in Cases of Incomplete Surgical Staging

In patients with incomplete surgical staging, imaging is necessary to assess disease extent. If imaging is negative, patients may be categorized into one of several groups: Stage IA grade 1–2 with myometrial invasion and no lymphovascular space invasion (LVSI), Stage IA grade 3 without myometrial invasion, or Stage IA grade 3 with myometrial invasion but without LVSI. For patients suspected to be Stage IB or with evidence of LVSI, restaging surgery is recommended. The final staging outcome then determines the appropriate adjuvant therapy.

If imaging findings are suspicious or positive and the patient is a suitable surgical candidate, surgery should be performed. If the patient is not eligible for surgery, **a biopsy** should be performed for diagnostic

confirmation. In patients with imaging- or biopsy-confirmed Stage II or higher, further management is guided by stage and clinical condition. Adjuvant therapy for advanced-stage disease (Stage II–IV) often involves a combination of chemotherapy and radiotherapy.[19,20]

In this case, initial histopathology revealed a Grade 2 endometrioid adenocarcinoma. However, clinical findings suggested progression to FIGO Stage IIIB endometrial carcinoma. According to the decision tree, imaging and biopsy may be performed as indicated. The patient refused reoperation following supracervical hysterectomy and bilateral salpingo-oophorectomy, but accepted adjuvant therapy.

Role of Adjuvant Therapy

Adjuvant therapy plays a pivotal role in the comprehensive management of endometrial carcinoma, complementing primary surgical treatment. While surgery remains the primary curative modality, adjuvant chemotherapy, radiotherapy, and hormonal therapy aim to eradicate residual malignant cells. Chemotherapy is generally reserved for advanced disease, while radiotherapy may be used preoperatively or postoperatively. For early-stage disease, whole pelvic radiotherapy and vaginal brachytherapy (VBT) may be employed. Although neither has conclusively demonstrated improved overall survival, both are effective at enhancing local control.[21]

Radiotherapy remains the most commonly used adjuvant modality in endometrial carcinoma. It can be delivered via external beam radiation therapy (EBRT) or vaginal brachytherapy (VBT). In addition to its adjuvant role, radiotherapy may serve as definitive therapy in medically inoperable patients, for local recurrence, or as palliative treatment. In this case, the patient was referred to the Radiation Oncology Department to receive both EBRT and VBT. VBT, because of its targeted delivery to the vaginal cuff and adjacent lymphatics, is associated with low morbidity. In contrast, EBRT may cause acute and long-term complications, including diarrhea, abdominal pain, lower extremity and pelvic lymphedema, enteritis, colitis, and, in severe cases, strictures and fistula formation.[13]

Molecular Profiling and Immunohisto-chemical Assessment

The pathogenesis of endometrial carcinoma involves mutations in key molecular signaling pathways. Dysfunction in these pathways contributes to defective apoptosis, increased cellular proliferation, telomerase activation, and impaired DNA repair. The endometrioid subtype (EEC) is commonly associated with alterations in ARID1A, PTEN, KRAS, and CTNNB1, as well as deficiencies in mismatch repair (MMR) pathways. In contrast, mutations in TP53, HER2, CDKN2A, CCNE1, and FBXW7 are more frequently observed in high-grade endometrioid carcinoma, serous carcinoma, and carcinosarcoma.[22]

Recent advances in the molecular landscape of endometrial carcinoma have led to a refined classification system, enabling better prognostic stratification and guiding personalized treatment. Molecular profiling has revealed that endometrial carcinoma is far more heterogeneous than previously recognized and can be divided into four distinct molecular subgroups according to the Cancer Genome Atlas (TCGA) classification:

1. POLE ultramutated (POLEmut)
2. Microsatellite instability-high (MSI-H) or mismatch repair-deficient (MMRd)
3. Copy number-low / nonspecific molecular profile (CNL/NSMP)
4. Copy number-high / p53 abnormal (CNH/ p53abn)

These molecular subtypes generally align with traditional histological classifications and reflect distinct etiologies and tumorigenic pathways.[12,16]

Immunohistochemical (IHC) analysis is increasingly recommended in several centers because of its cost-effectiveness, sensitivity, specificity, and reproducibility. Techniques such as PCR-based molecular testing—for instance, using five of the eight mononucleotide repeat markers—are available.[23-25]

In this case, the patient underwent an initial biopsy; however, for diagnostic confirmation, IHC testing for vimentin, Ki-67, CK7, and CK20 is recommended.

The integration of molecular classification with the updated FIGO staging system provides a valuable framework for the genetic characterization of endometrial carcinoma, serving as a foundation for precision medicine. Nevertheless, the incorporation of advanced technologies such as next-generation sequencing (NGS) into routine clinical practice remains limited by high cost and the bioinformatics complexity of data analysis.[19]

Conclusions

Atypical endometrial hyperplasia is recognized as the primary precursor lesion of endometrioid adenocarcinoma of the endometrium. Patients diagnosed with atypical endometrial hyperplasia or endometrial carcinoma are recommended to undergo total hysterectomy, bilateral salpingo-oophorectomy, and appropriate surgical staging, including pelvic washing and lymphadenectomy, in accordance with the updated FIGO guidelines. Supracervical (subtotal) hysterectomy is generally not recommended as definitive management for endometrial carcinoma. In patients who have previously undergone supracervical hysterectomy, comprehensive evaluation for residual cervical or parametrial disease is essential to guide further treatment.

Adjuvant therapy, particularly radiotherapy, remains the most commonly used adjunct in endometrial cancer management, either as neoadjuvant (preoperative) or postoperative treatment to reduce tumor burden and recurrence risk. In the present case, the patient had a diagnosis of endometrial carcinoma, with a prior history of supravaginal hysterectomy and bilateral salpingo-oophorectomy, and was subsequently treated with external beam radiation therapy (EBRT) and vaginal brachytherapy as part of her adjuvant therapy regimen.

Conflict of Interest

Authors declare no conflicts of interest

Advice and Thanks (if any)

Not Applicable

References

1. Morice P, Leary A, Creutzberg C, Abu-Rustum N, Darai E. Endometrial cancer. The Lancet. Lancet Publishing Group; 2016 Mar 12;387(10023):1094–108.
2. Lu KH, Broaddus RR. Endometrial Cancer. Longo DL, editor. New England Journal of Medicine. Massachusetts Medical Society; 2020 Nov 19;383(21):2053–64.
3. eBioMedicine. Endometrial cancer: improving management among increasing incidence rates. EBioMedicine. Elsevier B.V.; 2024 May 1;103:105159.
4. Endometrial cancer statistics | World Cancer Research Fund [Internet]. [cited 2025 Jun 1]. Available from: https://www.wcrf.org/preventing-cancer/cancer-statistics/endometrial-cancer-statistics/
5. Key Statistics for Endometrial Cancer | American Cancer Society | American

Cancer Society [Internet]. [cited 2025 Jun 1]. Available from: https://www.cancer.org/cancer/types/endometrial-cancer/about/key-statistics.html?
6. Brooks RA, Fleming GF, Lastra RR, Lee NK, Moroney JW, Son CH, et al. Current recommendations and recent progress in endometrial cancer. CA Cancer J Clin. American Cancer Society; 2019 Jul 1;69(4):258–79.
7. Hermann C, Williams K, Ko EM. Endometrial Hyperplasia. Handbook of Gynecology, Second Edition [Internet]. StatPearls Publishing; 2024 Apr 30 [cited 2025 Jun 7];1029–47. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560693/
8. Uccella S, Zorzato PC, Dababou S, Bosco M, Torella M, Braga A, et al. Conservative Management of Atypical Endometrial Hyperplasia and Early Endometrial Cancer in Childbearing Age Women. Medicina (Lithuania) [Internet]. MDPI; 2022 Sep 1 [cited 2025 Jun 7];58(9). Available from: https://pubmed.ncbi.nlm.nih.gov/36143933/
9. Trimble CL, Kauderer J, Zaino R, Silverberg S, Lim PC, Burke JJ, et al. Concurrent endometrial carcinoma in women with a biopsy diagnosis of atypical endometrial hyperplasia: A gynecologic oncology group study. Cancer [Internet]. Cancer; 2006 Feb 15 [cited 2025 Jun 7];106(4):812–9. Available from: https://pubmed.ncbi.nlm.nih.gov/16400639/
10. Management of Endometrial Hyperplasia Green-top Guideline No. 67RCOG/BSGE Joint Guideline |. 2016.
11. Passarello K, Kurian S, Villanueva V. Endometrial Cancer: An Overview of Pathophysiology, Management, and Care. Semin Oncol Nurs. W.B. Saunders; 2019 Apr 1;35(2):157–65.
12. Alwafai Z, Beck MH, Fazeli S, Gürtler K, KunzYoung Academy of Gynecologic Oncology C, Singhartinger J, et al. Accuracy of endometrial sampling in the diagnosis of endometrial cancer: a multicenter retrospective analysis of the JAGO-NOGGO. BMC Cancer. BioMed Central Ltd; 2024 Dec 1;24(1).
13. Anca-Stanciu MB, Manu A, Olinca MV, Coroleucă C, Comandașu DE, Coroleuca CA, et al. Comprehensive Review of Endometrial Cancer: New Molecular and FIGO Classification and Recent Treatment Changes. Journal of Clinical Medicine 2025, Vol 14, Page 1385. Multidisciplinary Digital Publishing Institute; 2025 Feb 19;14(4):1385.
14. Hoffman BL. Williams Gynecology. 3rd ed. Mc Graw Hill Education; 2012. p. 702–20.
15. Mahdy H, Vadakekut ES, Crotzer D. Endometrial Cancer. StatPearls [Internet]. StatPearls Publishing; 2024 Apr 20 [cited 2025 Jun 4]; Available from: https://www.ncbi.nlm.nih.gov/books/NBK525981/
16. Zhao X, Sun F, Leng N, Zhang X, Zhu Y. The past, present, and future of FIGO staging of endometrial cancer. J Gynecol Oncol [Internet]. J Gynecol Oncol; 2025 [cited 2025 Jun 1];36. Available from: https://pubmed.ncbi.nlm.nih.gov/40350708/
17. Galant N, Krawczyk P, Monist M, Obara A, Gajek Ł, Grenda A, et al. Molecular Classification of Endometrial Cancer and Its Impact on Therapy Selection. Int J Mol Sci. Multidisciplinary Digital Publishing Institute (MDPI); 2024 Jun 1;25(11).
18. Monk BJ, Berman ML, Montz FJ. Adhesions after extensive gynecologic surgery: Clinical significance, etiology, and prevention. Am J Obstet Gynecol [Internet]. Am J Obstet Gynecol; 1994 [cited 2025 Jun 12];170(5):1396–403. Available from: https://pubmed.ncbi.nlm.nih.gov/8178880/
19. Matsuo K, Machida H, Takiuchi T, Garcia-Sayre J, Yessaian AA, Roman LD. Prognosis of women with apparent

stage I endometrial cancer who had supracervical hysterectomy. Gynecol Oncol [Internet]. Academic Press Inc.; 2017 Apr 1 [cited 2025 Jun 4];145(1):41–9. Available from: https://pubmed.ncbi.nlm.nih.gov/28215841/

20. Uterine Neoplasms - Guidelines Detail [Internet]. [cited 2025 Jun 7]. Available from: https://www.nccn.org/guidelines/guidelines-detail?category=1&id=1473
21. Bestvina CM, Fleming GF. Chemotherapy for Endometrial Cancer in Adjuvant and Advanced Disease Settings. Oncologist [Internet]. Oxford University Press (OUP); 2016 Oct 1 [cited 2025 Jun 2];21(10):1250. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC5061541/
22. Kalampokas E, Giannis G, Kalampokas T, Papathanasiou AA, Mitsopoulou D, Tsironi E, et al. Current Approaches to the Management of Patients with Endometrial Cancer. Cancers 2022, Vol 14, Page 4500 [Internet]. Multidisciplinary Digital Publishing Institute; 2022 Sep 16 [cited 2025 Jun 4];14(18):4500. Available from: https://www.mdpi.com/2072-6694/14/18/4500/htm
23. Chiang S, Soslow RA. Updates in diagnostic immunohistochemistry in endometrial carcinoma. Semin Diagn Pathol [Internet]. W.B. Saunders; 2014 [cited 2025 Jun 1];31(3):205–15. Available from: https://pubmed.ncbi.nlm.nih.gov/24951284/
24. Wang M, Hui P. A Timely Update of Immunohistochemistry and Molecular Classification in the Diagnosis and Risk Assessment of Endometrial Carcinomas. Arch Pathol Lab Med [Internet]. Allen Press; 2021 Nov 1 [cited 2025 Jun 1];145(11):1367–78. Available from: https://dx.doi.org/10.5858/arpa.2021-0098-RA
25. Favier A, Varinot J, Uzan C, Duval A, Brocheriou I, Canlorbe G. The Role of Immunohistochemistry Markers in Endometrial Cancer with Mismatch Repair Deficiency: A Systematic Review. Cancers (Basel). MDPI; 2022 Aug 1;14(15).