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Navrang

JOGS CHRONICLE

"A JOURNEY THROUGH GYNECOLOGICAL DISORDERS"

THEME

"Dil Chahta Hai Har Naari Ho Swasth Aur Safal...
Banaye Apna Surakshit Ewam Ujjwal Kal... Har Pal"

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प्रेम की पाती

मैं माँ से अब 'अम्मा', बड़ी माँ, नानी बन गई हूँ।

जीवन के सभी उतार चढ़ाव को पार करते हुए,

चेहरे पर प्रेम की झुरियों को लिए आगे बढ़ रही हूँ।

हौसले वही है पर आँखो की चमक कुछ धुधली सी हो गई है।

मेरी कमर कुछ झुकी सी हो गई है।

आजकल कुछ थकान सी महसूस हो रही है।

मन में उमंग तो है पर मनपसंद किससे कुछ भूल रही हूँ।

मेरे पास जीवन अनुभूवों की अनुपम कुँजी है जो,

मैं सबसे लिए संजो रही हूँ।

आओ कुछ पल बैठो मेरे पास,

जीवन के नवरंगों के पिटारे को खोल रही हूँ।

हर रंग में रंगी हुई मैं हिम्मत, उमंग, आशा और दृढ़ता के रंगो को भरते हुए,

प्रोणस्वाथा की नई चुनरी ओढ़ रही हूँ मैं।

– डॉ. रानू जैन

DR. RANU JAIN
Chief Editor
JOGS 2025-26

Menopause is a natural life stage demanding better, individualized care. moving beyond the outdated view of it solely as a medical disease. Through this edition we have tried to cover most challenging diseases of perimenopause and menopause like endometriosis, post menopaual bleeding, osteoporosis and genital malignancies.

This stage in women's life is a roller coaster ride with challenges in both emotional and physical aspects. The key to deal with menopause is support, affection and medical care.

Let us all join our hands together to ease the menopause and life should not be at pause.

Happy Reading

DR. NANDITA BHARTIYA
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JOGS 2025-26

BEST SOCIETY AWARD

OVARIAN CANCERS- AN INSIGHT IN SCREENING

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Introduction- Ovarian cancer is considered as "Silent Killer" in woman's life. There is absolutely no significant symptoms associated with the disease. As per Globacon 2020 the top 5 cancers in woman are affecting Breast, Cervical, Ovary, Lip oral cavity and colorectal .The incidence of ovarian cancer globally is 1.6% with ASR 6.6 /1,00,000 and mortality 6.7% with ASR 4.2 /1,00,000.The incidence of ovarian cancer in India in 3.5% with ASR 6.7/1,00,000 as per Globacon 2020.

Epidemiology- Ovarian cancer is a rising concern, withprojection of approximately incidence of 49,000 new cases in 2025.As per Globacon 2020 in India, the new cases of ovarian cancer were 45,701 and deaths 32,077 .It is now ranking third most common gynecological cancer with diagnosis often late at advanced stage III/IV, demanding better awareness and access to care, especially in rural areas. Late diagnosis is the major issue.

Challenges in developing screening strategies in ovarian cancer-

- No definite pre-invasive or precursor lesion
- Ovary is not accessible like cervix for direct inspection and sampling
- Risk of false positives
- Low prevalence of ovarian carcinoma
- Current screening tests cannot detect ovarian cancer early enough to alter the natural history of the disease

Symptoms of Ovarian Cancer-

- Abdominal bloating,discomfort,abdominal pain
- Change in bowel habits like constipation, diarrhea.
- Increase in abdominal size
- Frequent urination

Screening for Ovarian cancer-

- Symptom index
- Pelvic examination
- CA 125
- TVS

Goff's Symptom index-1

1. Bloating/ increase abdominal size,
2. Feeling full/difficulty eating/loss of appetite
3. Pelvic / abdominal pain

4. Urinary urgency / frequency
5. Weight loss

Persistent (>12 times a month), < 1 year

Sensitivity for early stage disease is 56.7 % & advanced stage disease is 79.5%.

Specificity is 90% for woman above 50 years.

- CA 125-Carcinoma antigen 125 is a glycoprotein , most widely used biomarker in ovarian cancer.CA125 ≥35U/ml is accepted as UL of normal
- Only US Food and Drug Administration (FDA)-approved biomarker for ovarian cancer screening.
- Sensitivity (83%), specificity (59.5%) , positive predictive value (16%); cut off value of 35 U/ml
- May be elevated in fibroids, endometriosis, menstruation and tuberculosis and other cancers (breast, bladder, liver, lungs, pancreas)
- Serial CA125: Positive predictive value of 40% (95% confidence interval = 12.2%, 73.8%) for detecting invasive ovarian cancer.

ROMA- RISK OF OVARIAN MALIGNANCY ALGORITHM2

It was approved by FDA in 2011 and, should be used for the preoperative triage of a woman with a pelvic mass.FDA approved for referral to gynae oncologist, not screening.

Under the ROMA stratification, 3 patient variables are used:

- HE4 levels
- CA125 levels
- Patient's menopausal status

TVS- IOTA Classification3 of 'B' & 'M' rules are good to understand benign & malignant cysts.

Sensitivity: 95%, Specificity: 91% positive likelihood ratio: 10.37, Negative likelihood ratio: 0.06

'B' Rules: Unilocular cysts, Solid components <7 mm, Acoustic shadowing, smooth multilocular <100 mm, no blood flow

'M' Rules: Irregular solid tumour, ascites, at least 4 papillary structures, Irregularmultilocular solid tumour>100 mm

Conclusion-Ovarian cancer screening is recommended in high risk groups (risk of Hereditary cancer syndrome).Do not ignore nonspecific upper GI symptoms in post-menopausal. Women. Combination of serial CA125+ TVS may be beneficial in women with hereditary syndrome till fertility completed. Women with completed family with hereditary syndrome:should think of undergoing prophylactic BSO at 35-40 years of age (BRCA1carriers) & 40-45 years of age (BRCA2, BR1P1, RAD 51C, RAD51D) hysterectomy + BSO for LYNCH syndrome.

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ENDOMETRIAL CANCER – RECENT UPDATES

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Epidemiology and Risk Factors

Endometrial cancer predominantly affects postmenopausal women, with peak incidence between 55 and 65 years of age. Major risk factors include prolonged exposure to unopposed estrogen, obesity, nulliparity, early menarche, late menopause, polycystic ovary syndrome, diabetes mellitus, and hypertension. Use of tamoxifen for breast cancer and hereditary cancer syndromes such as Lynch syndrome significantly increase risk. Protective factors include combined oral contraceptive use, multiparity, and breastfeeding.

Pathogenesis and Classification

Traditionally, endometrial cancer has been classified into two types. Type I tumors are estrogen-dependent, commonly endometrioid adenocarcinomas, low grade, and associated with favorable prognosis. Type II tumors are estrogen-independent, high grade, and include serous carcinoma, clear cell carcinoma, and carcinosarcoma, with poorer outcomes.

Molecular Classification – A Major Advance

A significant breakthrough in understanding endometrial cancer biology came with The Cancer Genome Atlas (TCGA), which proposed a molecular classification dividing tumors into four groups:

1. POLE ultramutated(POLEmut)
2. Microsatellite instability–high
3. p53 abnormal endometrial cancer
4. Non-specific molecular profile(NSMP)

Diagnostic tools- 1. Clinical Evaluation - Postmenopausal bleeding (most common), Abnormal uterine bleeding (peri-/premenopausal), Pelvic pain, discharge (advanced disease)

Risk factors - Obesity, diabetes, PCOS, Unopposed estrogen / tamoxifen,Lynch syndrome, Late menopause, nulliparity.

First-Line Diagnostic Tools- A. Transvaginal Ultrasound (TVUS) Initial test for postmenopausal bleeding

Measures endometrial thickness (ET)

Postmenopausal: ≤4 mm " low risk | >4 mm " biopsy indicated

Premenopausal: limited value (cyclic variation)

Myometrial invasion

Adnexa, free fluid

B. Endometrial Sampling (Gold Standard)

1. Office Endometrial Biopsy (Pipelle)

First-line tissue diagnosis

Sensitivity ~90–95% for global disease but it may miss focal lesions (polyps)

2. Dilatation & Curettage (D&C)

When pipelle inadequate or inconclusive preferably combined with hysteroscopy

Advanced Diagnostic Tools - A. Hysteroscopy

Direct visualization + targeted biopsy

Superior for Focal lesions, polyps & persistent bleeding with negative biopsy

B. Imaging for Staging (Post-Tissue diagnosis)

1. MRI Pelvis (Preferred) for depth of myometrial invasion&Cervical stromal involvement to guide surgical planning

2. CT Scan (Chest/Abdomen/Pelvis) for Lymph node involvement&Distant metastasis used in high-risk or advanced disease management

3. PET-CT reserved for Recurrent disease& unclear metastasis

4. Histopathology & Molecular Diagnostics

A. Histopathology - Tumor type:Endometrioid | Serous | Clear cell | Carcinosarcoma

Grade (FIGO grade 1–3) | LVSI

B. Immunohistochemistry (IHC), p53 (abnormal " aggressive biology), ER/PR (prognostic, hormonal therapy response), Mismatch repair proteins (MLH1, MSH2, MSH6, PMS2)

C. Molecular Classification (TCGA-based) -

- POLE-ultramutated
- MMRd
- p53-abnormal
- NSMP (No specific molecular profile)

Special Situations include -

Young women desiring fertility: Biopsy + MRI mandatory - Lynch syndrome: annual endometrial biopsy

Tamoxifen users: low threshold for biopsy

The 2023 FIGO staging for endometrial cancer.

Stage I – Confined to uterus/ovary

IA1: Non-aggressive histology limited to polyp or endometrium only

IA2: Non-aggressive, <50% myometrial invasion, no/focal LVSI

IA3: Low-grade endometrioid confined to uterus and ovary

IB: Non-aggressive, ≥50% myometrium, no/focal LVSI

IC: Aggressive histology confined to endometrium / polyp

("Non-aggressive" = typically low-grade endometrioid)

Stage II – Cervical stroma / LVSI / aggressive histology

IIA: Cervical stroma invasion (non-aggressive histology)

IIB: Substantial LVSI (non-aggressive histology)

IIC: Aggressive histology with any myometrial invasion

Stage III – Local/regional spread

IIIA: Serosa or adnexal involvement

IIIB: Vagina/parametria or pelvic peritoneum

IIIC1/IIIC2: Pelvic/para-aortic lymph node metastases (micro & macro)

Stage IV – Advanced spread

IVA: Bladder/rectal mucosa invasion

IVB: Extrapelvic peritoneal metastases

IVC: Distant metastases (e.g., lung, liver)

FIGO 2023 STAGING – KEY CHANGES

- Stage I (Uterus confined)

IA: <50% myometrial invasion

IB: ≥50% myometrial invasion

- Stage II - Cervical stromal invasion (no longer split into A/B)
- Stage III -Substage - Definition

IIIA - Serosa/adnexa

IIIB - Parametrial/vaginal
IIIC1 - Pelvic nodes
IIIC2 - Para-aortic nodes
• Stage IV - | IVA | Bladder / bowel mucosa | | IVB | Distant metastasis |

Molecular classification incorporated into staging

Stage-wise & Molecular-based Management

EARLY STAGE (FIGO I–II)

Low-risk - (Stage IA, Grade 1–2, LVSI negative, endometrioid)
Treatment: - Total hysterectomy + BSO, No adjuvant therapy
Sentinel lymph node mapping preferred over full lymphadenectomy

Intermediate risk - (Stage IB G1–2 or IA G3)
Total hysterectomy + BSO
Adjuvant: Vaginal brachytherapy
Molecular impact: POLE-mutated " No adjuvant therapy even if high-grade
p53-abnormal " treat as high-risk

High-risk early stage - (Stage IB G3, Stage II, serous/clear cell, LVSI+)
Molecular Type - Adjuvant Treatment
POLE-mut - Observation or minimal RT
dMMR - RT ± chemotherapy
pMMR (NSMP) - RT + chemotherapy
p53-abn - Chemotherapy + RT**Molecular classification overrides histology**

LOCALLY ADVANCED (Stage III) - Standard Treatment-Surgery (if feasible)
Adjuvant chemotherapy + radiotherapy
Molecular-guided approach
POLE-mut - De-escalation possible
dMMR - Chemo + strong role for immunotherapy
p53-abn - Aggressive chemo + RT

ADVANCED / RECURRENT (Stage III–IV / Recurrent)

First-line therapy is NO longer chemotherapy alone
TREATMENT BY MOLECULAR SUBTYPE
1. dMMR / MSI-H (~30%)
First-line - Carboplatin + Paclitaxel + PD-1 inhibitor, Pembrolizumab / Dostarlimab / Durvalumab
Maintenance
Continue immunotherapy - Dramatic improvement in PFS and OS, Some patients achieve long-term remission
2. pMMR / MSS (~60%)
First-line
Carboplatin + Paclitaxel + PD-1 inhibitor
Maintenance immunotherapy ± PARP inhibitor
After progression
Pembrolizumab + Lenvatinib (established standard)
3. POLE-ultramutated (~10%) - Excellent prognosis, Avoid overtreatment
Even advanced cases may do well with minimal therapy

4. p53-abnormal / Serous-like - Most aggressive subtype, Chemo + Immunotherapy
HER2 positive ⇨ add Trastuzumab
ADCs (trastuzumab deruxtecan)

TARGETED & NOVEL THERAPIES (EMERGING)

HER2 ⇨Trastuzumab, ADCs
PARP ⇨Olaparib (select pMMR)
ADCs ⇨ T-DXd, Disitamab vedotin
Hormonal ⇨ CDK4/6 inhibitors + endocrine therapy (low-grade EC)

MOLECULAR CLASSIFICATION

POLE-ultramutated
~10%
Exonuclease mutation
De-escalate / avoid chemotherapy
dMMR / MSI-H
~30%
Loss of MMR proteins
Good
Immunotherapy sensitive
pMMR / NSMP
~40–50%
No specific mutation
Intermediate
Chemo ± IO
p53-abnormal
~10–15%
TP53 mutation
Poor prognosis
Aggressive multimodal therapy
Histology is secondary to molecular subtype

ESGO–ESTRO–ESP RISK STRATIFICATION

LOW RISK - Stage IA, G1–2, LVSI negative, POLE-mut (any stage I–II)
No adjuvant therapy
INTERMEDIATE RISK - Stage IB G1–2, Stage IA G3, LVSI focal
Vaginal brachytherapy
HIGH-INTERMEDIATE RISK - LVSI substantial, pMMR / dMMR with invasion
EBRT ± chemo
HIGH RISK- Stage III–IVA, Serous / Clear cell, p53-abnormal
Chemo + RT ± Immunotherapy

ADVANCED / RECURRENT DISEASE – CURRENT STANDARD

FIRST-LINE
Molecular Type
First-Line Treatment
dMMR/MSI-H - Chemo + PD-1 inhibitor
pMMR/MSS - Chemo + PD-1 inhibitor
HER2+ serous - Chemo + Trastuzumab
p53-abn - Chemo + IO + RT

Chemo alone is NO longer standard

SECOND-LINE

pMMR: Pembrolizumab + Lenvatinib

dMMR: Single-agent immunotherapy(ADCs & PARP inhibitors " emerging)

INITIAL WORK-UP: **Postmenopausal bleeding/AUB**

Transvaginal USG

(Endometrial thickness >4 mm)⇨ Endometrial biopsy ⇨ Histology + Grade ⇨Mandatory molecular testing (POLE, MMR, p53) ⇨ MRI pelvis ± CT/PET ⇨ FIGO 2023 staging

SURGICAL MANAGEMENT

Confirmed Endometrial Cancer ⇨ TH + BSO ⇨ Sentinel lymph node mapping (Preferred)⇨ Avoid full lymphadenectomy (unless bulky nodes)

ADJUVANT TREATMENT-EARLY STAGE

Stage I–II disease ⇨ Risk stratification (ESGO-ESTRO-ESP + Molecular)

LOW RISK - (Stage IA G1–2 / POLE) - Observation

INTERMEDIATE RISK - (Stage IB G1–2 / IA G3) - Vaginal brachytherapy

HIGH RISK - (Stage IB G3 / II / p53) - Chemotherapy + RT

MOLECULAR GUIDED DECISION MAKING

Molecular subtype identified ⇨ POLE-mut

" De-escalate therapy

" Avoid chemo ⇨ dMMR/MSI-H

" Strong immunotherapy response ⇨ pMMR/NSMP

" Chemo ± Immunotherapy ⇨ p53-abnormal

" Aggressive chemo + RT ± IO

ADVANCED/RECURRENT DISEASE

Stage III–IV / Recurrent EC ⇨ Molecular testing ⇨ First-line treatment ⇨ Carboplatin + Paclitaxel+ PD-1 inhibitor

(Pembrolizumab / Dostarlimab) ⇨ Maintenance immunotherapy

RECURRENT DISEASE- SECOND LINE

Progression after first-line ⇨ Molecular subtype ⇨ dMMR

"Single-agent immunotherapy ⇨ pMMR

" Pembrolizumab + Lenvatinib ⇨ HER2 positive

" ADCs / Trastuzumab

Imaging

MRI is the imaging modality of choice for preoperative assessment of myometrial invasion and cervical involvement. CT scan and PET-CT are used in advanced disease to assess nodal and distant metastasis

Minimally invasive surgery, including laparoscopic and robotic approaches, has become the standard of care for early-stage endometrial cancer. These techniques offer reduced blood loss, lower postoperative morbidity, shorter hospital stay, and faster recovery without compromising oncological outcomes.

Hormonal Therapy

Hormonal therapy plays a role in selected patients with low-grade, hormone receptor–positive tumors. Progestins, aromatase inhibitors, and levonorgestrel-releasing intrauterine systems are used, particularly in fertility-sparing and recurrent settings.

Immunotherapy

Immunotherapy represents one of the most important recent advances. Tumors with mismatch repair deficiency or MSI-H status show excellent response to immune checkpoint inhibitors. These agents have

demonstrated durable responses in recurrent and advanced endometrial cancer and are now an integral part of treatment algorithms.

Targeted Therapy

Targeted therapies such as mTOR inhibitors, anti-angiogenic agents, and HER2-targeted therapy in selected serous carcinomas have expanded treatment options. Combination regimens involving immunotherapy and targeted agents have shown promising results in clinical trials.

Fertility-Sparing Management

Young women with early-stage, low-grade endometrioid carcinoma desiring fertility preservation may be managed conservatively with high-dose progestins or levonorgestrel intrauterine systems under strict surveillance. Assisted reproductive techniques are often employed after disease remission

Conclusion

The management of endometrial cancer has evolved significantly with advances in molecular classification, minimally invasive surgery, sentinel lymph node mapping, immunotherapy, and targeted treatments. These developments have shifted care toward a personalized approach, improving survival outcomes while reducing treatment-related morbidity. Continued integration of molecular profiling into routine clinical practice will further enhance patient-centered care and outcomes in endometrial cancer.

Molecular classification is mandatory

Immunotherapy + chemotherapy is first-line for advanced disease

POLE tumors " de-escalate

dMMR " immunotherapy sensitive

p53-abn " aggressive multimodal therapy

Sentinel node mapping > lymphadenectomy

HYSTERECTOMIES IN WOMEN LIVING WITH HIV THE MANAGEMENT PROTOCOL

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Hysterectomies are common in women living with HIV(HIV-positive women), often for cervicalneoplasia, myoma , heavy bleeding, and other conditions, requiring hysterectomy.

The management protocol for HIV-positive women undergoing a hysterectomyinvolves comprehensive care across the pre-operative, intra-operative, and post-operative phases.It is largely the same as for HIV-negative women, with an emphasis on optimal HIV management (CD4/viral suppression), vigilant intraoperative and postoperative monitoringand potentially minimally invasive techniques, in order to ensure patient safety and minimizing the risk of infection. HIV infection itself is not a contraindication to surgery.

Key management protocol

Preoperative management

- Optimal HIV Status: A detailed pre-operative evaluation, including complete blood count, liver/renal function testsand coagulation profile.

  Serology for HIV and Hepatitis B/C is must as co-infection is common.

  Reviewing recent CD4 count (>200cells/mm3)and viral load is essential . The primary goal is to achieve and maintain viral suppression (undetectable viral load or<200 copies/mL) as it reduces the complication rates comparable to those of HIV-negative individuals.
- Antiretroviral Therapy (ART): The patient should continue their regimen throughout the perioperative period and lifelong
- Comorbidity Management: HIV-positive women often have more comorbidities like diabetes,anemia,hepatitis coinfection, smoking, substance abuse, which need to be managed.
- Antibiotic Prophylaxis: Standard surgical antibiotic prophylaxis should be administered.
- Nutritional status: Nutritional counselling,particulary with regards to low albumin levels to be addressed.
- Informed Consent: The elevated risks and the need for rigorous follow-up to be discussed.
- Counselling and emotional support: The psychological impact of a hysterectomy is addressed, and resources like counseling or support groups may be offered.

Surgical Approach

Abdominal: More common in HIV+ women butminimally invasive hysterectomy (laparoscopic or robot-assisted) is associated with a decreased risk of surgical site infections compared to abdominal hysterectomy.

Cone Biopsy vs Hysterectomy: For early cervical disease, cone biopsy(specially LEEP) isrecommended as the primart treatment for its effectiveness and lower invasiveness but hysterectomy offers a definitive solution for CIS (Carcinoma in Situ).

Various studies havereporteddifferent approaches including abdominal, vaginal, laparoscopic, and robot-assisted. The choice of surgical method depends on the clinical situation and not the women's HIV status.

Overall, with modern medical management and safety protocols,hysterectomies can be performed safely in women living with HIV

Perioperative management

Standard infection control precautions (gloves, masks, protective eyewear for procedures with splashing and safe sharps handling) should be followed by the surgical team and other health care workers. Strict adherence to universal precautions minimizes the occupational exposure risk. Clear protocols for post-exposure prophylaxis (PEP) for healthcare workers in case of accidental exposure must be in place and should be followed immediately, ideally within 2 hours

Postoperative Management

- Complications Monitoring: HIV-infected women may be more likely to experience infectious complications (e.g., surgical site infections, pneumonia, urinary tract infections) and have a longer length of hospital stay. Monitoring for surgical site infections is crucial, as they can occur, sometimes presenting as pelvic abscesses or vaginal cuff cellulitis. Vigilant monitoring is crucial.
- Nutrition: Low preoperative serum albumin levels are associated with increased risk of surgical site infections, highlighting the importance of nutritional support.
- Follow-up Screening: Women with HIV who have had a hysterectomy (especially if the cervix was removed) may still require specific post-operative screening, such as vaginal Pap smears, due to a higher risk of vaginal intraepithelial neoplasia (VAIN). Vaginal cytology with HPV co-testing is recommended for screening

Key Considerations & Risks

- HIV Control: Well-controlled HIV (undetectable viral load, higher CD4 count) significantly reduces surgical risks, asthose of HIV-negative individuals.
- Co-infections: Hepatitis C/B co-infection and substance abuse can increase surgical risks.
- Surgical Site Infections (SSIs): Some studies show higher SSI rates (abscesses, cellulitis) in HIV+ women, linked to lower albumin or abdominal approaches, but minimally invasive methods (laparoscopic) and better albumin levels reduce this risk.
- Vaginal Health: Abnormal Pap smears post-hysterectomy are more common in HIV+ women, requiring continued monitoring.

Conclusion

Hysterectomy in HIVpositive women is a safe and common procedure for women with HIV,particularly for managing HPV- related cervical diseases. Effective procedure when performed with appropriate precautions, thorough pre-operative workup, and consideration of HIV disease control, with modern management, often yield good outcomes.

Key Recommendations

- Optimize HIV status: Ensure viral suppression and good CD4 count before surgery.
- Vaginal Surveillance: Crucial to continue regular vaginal Pap smears (and potentially biopsies) post-hysterectomy to pick VAIN/vaginal cancers.
- Multidisciplinary Care: Involve Gynaecologists, infectious disease specialists, and potentially nutritionists/bone health specialists.
- Medical Evaluation: A thorough medical history, physical exam, and necessary lab/imaging tests (e.g., Pap test, pelvic ultrasound, blood tests) to assess overall health before surgery.
- Emotional Support: The psychological impact of a hysterectomy to be addressed.
- Hormone Management: If ovaries were removed in a premenopausal woman, hormone replacement therapy (HRT) to be discussed and offered to manage menopausal symptoms.
- Follow-up: A follow-up is necessary a few weeks after surgery to monitor recovery progress.

CHALLENGES IN MANAGEMENT OF ENDOMETRIOSIS AND APPROACHES TO ITS MANAGEMENT

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Introduction

Endometriosis is a chronic, estrogen-dependent, inflammatory disease characterized by the presence of endometrial-like glands and stroma outside the uterine cavity. These ectopic implants commonly involve the ovaries, peritoneum, uterosacral ligaments and, in advanced cases, bowel and urinary structures. The condition affects physical, reproductive and psychosocial health, often leading to dysmenorrhoea, dyspareunia, chronic pelvic pain and infertility, significantly diminishing quality of life. Globally, an estimated **10% of women of reproductive age** are affected, translating to over 190 million individuals worldwide.

2. Diagnosis of Endometriosis

Endometriosis presents with a wide spectrum of symptoms, which often overlap with other gynaecological and gastrointestinal disorders. The most frequent symptoms include:

- Dysmenorrhoea (painful menstruation)
- Chronic pelvic pain not confined to menses
- Dyspareunia (especially deep)
- Dyschezia and dysuria, particularly with bowel or bladder involvement
- Infertility, reported in up to 30–50% of affected women
- Other symptoms: fatigue, heavy menstrual bleeding, cyclical shoulder pain, and cyclical bowel/bladder symptoms depending on lesion location.

Clinical suspicion is typically raised based on a detailed menstrual, pain and reproductive history, and physical examination. Findings such as fixed retroverted uterus, uterosacral ligament nodularity, or adnexal masses may be suggestive of deep disease but are not always present.

Radiological Approaches

Transvaginal Ultrasonography (TVS)TVS is typically the first-line imaging modality. It is highly useful for detecting ovarian endometriomas and can suggest deep infiltrating endometriosis (DIE) when performed by experienced operators. However, its sensitivity is lower for peritoneal and subtle lesions.

Magnetic Resonance Imaging (MRI)MRI offers superior contrast resolution and is especially valuable for mapping deep infiltrating disease, including involvement of the rectovaginal septum, bladder and bowel. MRI has high sensitivity and specificity for endometriomas and DIE when protocols are optimized. However, peritoneal lesions remain challenging to identify on imaging alone.

Advanced and Adjunct Techniques Techniques such as transrectal or 3-D ultrasonography, and specialized MRI sequences can improve detection of DIE. Emerging research into artificial intelligence–augmented imaging holds promise for future diagnostic enhancements.

Diagnostic Laparoscopy

Definitive diagnosis has historically relied on laparoscopic visualization and histopathological confirmation. Visual inspection may identify typical red, black or white lesions, with adhesions and endometriomas. Biopsy with

histology provides confirmation, but the absence of histologic proof does not exclude disease if clinical suspicion is high. However, laparoscopy is increasingly considered a **therapeutic procedure** rather than a diagnostic gold standard due to its invasiveness and because many patients are managed empirically based on symptoms and imaging.

There are many Challenges in Diagnosis of endometriosis. Delayed diagnosis, often years from symptom onset. Non-specific symptoms that mimic other disorders. Limited access to specialist imaging and surgical evaluation in many settings. Variability in lesion appearance and distribution.

3. Management of Endometriosis

Medical Management

Medical therapy is primarily aimed at **symptom control and suppression of disease activity,** especially pain.

Indications for Medical Management

- Mild to moderate pain without significant anatomic distortion
- Desire for fertility postponement
- As adjunct to surgical therapy
- Patients unfit for or unwilling to undergo surgery.

Medical Options

- **NSAIDs** for pain control
- **Hormonal therapies** including combined oral contraceptives, progestins (e.g., dienogest), and **GnRH agonists/antagonists** to suppress ovulation and reduce lesion activity
- **Levonorgestrel-releasing intrauterine system (LNG-IUS)** for long-term suppression in select cases
- **Aromatase inhibitors** in refractory cases (often with add-back therapy).

Medical therapies do not cure endometriosis and symptoms often recur upon discontinuation. The choice of regimen should be individualized based on symptom severity, side effects, fertility goals and cost.

Surgical Management

Surgery is indicated when symptoms are **severe or refractory to medical therapy, when there is anatomic disease causing pain or infertility, or when fertility is an immediate goal.**

Surgical Goals

- **Excision or ablation** of endometriotic lesions
- **Adhesiolysis** to restore normal anatomy
- **Ovarian cystectomy** for endometriomas
- Management of DIE involving bowel, bladder, ureters, or other structures
- Fertility-preserving approaches whenever possible.

Conventional Laparoscopy Conventional laparoscopy remains the cornerstone of surgical management due to reduced morbidity compared with laparotomy, with advantages including faster recovery, decreased adhesions and lower infection risk. Excision of lesions and removal of endometriomas are generally preferred over ablation for long-term outcomes.

3D Laparoscopy Three-dimensional (3D) laparoscopy provides enhanced depth perception, potentially improving accuracy of dissection, especially in complex pelvic anatomy. It represents an incremental advance over standard 2D laparoscopy and may aid in meticulous excision of DIE.

Robotic Surgery Robotic-assisted surgery (e.g., using systems such as the da Vinci platform) offers improved ergonomics, **3-D magnification, tremor filtration and increased instrument articulation,** which can be particularly beneficial in complex cases such as DIE involving the bowel, bladder or ureters. Evidence suggests that robotic and conventional laparoscopy achieve **comparable surgical outcomes,** although robotic approaches may involve longer operative times and higher costs.

Studies also indicate potential benefits in preserving ovarian function during cystectomy, though high-quality evidence is still emerging.

Advanced endometriosis, particularly deep infiltrating endometriosis (DIE), frequently results in severe pelvic distortion due to fibrosis, dense adhesions, and obliteration of normal tissue planes. The uterosacral ligaments, rectovaginal septum, pelvic sidewalls, ureters, bladder, and bowel are commonly involved. In such scenarios, **safe surgical management mandates a retroperitoneal approach,** which allows identification and protection of vital structures before disease excision.

Rationale for a Retroperitoneal Approach

Operating in the retroperitoneal space enables the surgeon to:

- Identify and trace the ureter from an **uninvolved segment**
- Visualize major pelvic vessels
- Restore anatomical orientation in a frozen pelvis
- Perform complete excision of disease rather than superficial ablation
- Minimize intraoperative complications such as ureteric, vascular, and bowel injuries

A retroperitoneal strategy is especially critical when the pouch of Douglas is obliterated, adnexa are fixed, or endometriotic nodules infiltrate the uterosacral ligaments or pelvic sidewalls.

Stepwise Surgical Principles for Safe Retroperitoneal Dissection

1. Entry into the Retroperitoneal Space

The retroperitoneum is best accessed through the **paravesical and pararectal spaces,** which serve as anatomical "safe zones":

- Incision of peritoneum lateral to the infundibulopelvic ligament or medial to the external iliac vessels
- Development of the **paravesical space** (between bladder and pelvic sidewall) and **pararectal space** (between uterosacral ligament and internal iliac vessels)
- Gentle blunt and sharp dissection to expose landmarks

Early creation of these spaces restores orientation and facilitates subsequent steps.

2. Ureterolysis: Cornerstone of Safe Surgery

The ureter is frequently displaced medially, encased in fibrosis, or compressed by endometriotic nodules. key principles in safe handling of ureter can be

- Identify the ureter at the **pelvic brim**, where anatomy is usually preserved
- Trace it distally toward the uterine artery crossing
- Perform **circumferential ureterolysis**, freeing it from fibrotic tissue
- Avoid excessive traction or thermal energy near the ureter
- In cases of intrinsic ureteric involvement, collaboration with urology for stenting or segmental resection may be required

Routine ureteric stenting is controversial but may be helpful in select high-risk cases.

3. Pelvic Sidewall Dissection

Endometriosis commonly infiltrates the pelvic sidewall, involving:

- Uterosacral ligaments
- Hypogastric nerves
- Internal iliac vessels and branches

Safe dissection involves:

- Identification of **internal iliac artery and its branches**
- Preservation of autonomic nerves where feasible to reduce postoperative bladder and bowel dysfunction

- Excision of disease while maintaining hemostasis and nerve integrity

4. **Management of Obliterated Pouch of Douglas**

In complete cul-de-sac obliteration:

- Begin dissection laterally, not in the midline
- Mobilize the rectum using retrorectal and pararectal spaces
- Progress medially once safe planes are developed
- Sharp dissection is preferred over blunt tearing to reduce rectal injury

In cases of bowel infiltration, a multidisciplinary approach involving colorectal surgeons is essential.

5. **Handling Deep Infiltrating Endometriosis (DIE)**

DIE nodules may involve:

- Rectovaginal septum
- Bladder
- Ureters
- Parametrium

Principles include:

- Complete excision rather than shaving in symptomatic patients
- Layer-by-layer dissection respecting tissue planes

Informed preoperative consent regarding possible bowel or urinary tract procedures

Follow-up After Surgery

Postoperative follow-up should include:

- Assessment of symptom relief (pain scores, quality of life)
- Monitoring for recurrence of symptoms
- Evaluation of fertility outcomes where relevant
- Imaging as indicated for suspected recurrence
- Consideration of medical therapy to sustain remission.

Reducing Recurrence

Recurrence after surgery is common, with endometrioma or lesion recurrence rates up to 27% or higher within 24 months if no postoperative therapy is used. Postoperative hormonal suppression (e.g., combined oral contraceptives, LNG-IUS, GnRH analogues) has been shown to significantly reduce the risk of recurrence and improve pain outcomes compared with expectant management.

Other strategies include early conception where feasible, long-term suppressive regimens for pain control, and individualized approaches based on severity and fertility goals.

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RECENT ADVANCES IN OVARIAN CANCER

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INTRODUCTION

Ovarian cancer is the 7th most common cancer in women in the world and 8th most common cause of death from cancer in women in the world. Around 313,959 women are diagnosed every year with 207,252 women die of the disease every year around the world[1]. According to GLOBOCON'S 2020 projections,by 2040, the number of women around the world diagnosed with ovarian cancer will rise almost 42% to 445,721. The number of women dying from ovarian cancer each year is projected to increase to 313,617 an increase of over 50% from 2020.

ETIOLOGY

The established risk factors are age and having a family history of the disease.Affected family members constitute a significant risk factor. First degree relatives of proband have a three-seven fold increased risk, often with an early age of onset. The lifetime risk of EOC associated with germline BRCA1 mutation exceeds 50% and that associated with germline BRCA2 mutation ranges from 12-20%.

SCREENING AND PREVENTION

Measurement of CA-125 levels is usually done in adjunction with the imaging. CA-125 is elevated in most of the epithelial ovarian cancers overall, but only half of the earlystage epithelial ovarian cancers[9]. The specificity and positive predictive value found to be higher in postmenopausal women than in premenopausal women. Increased CA-125 levels are also observed in other physiological or benign pathological conditions such as endometriosis, pregnancy, ovarian cysts, inflammatory peritoneal diseases. Hence, other biomarkers are currently being studied to improve specificity for ovarian cancer biomarkers. Human epididymis protein 4 (HE4) is a new biomarker that is currently being evaluated. It is found to be more sensitive for ovarian cancer and found in approximately 100% of serous and endometrioid subtypes. Based on recent studies, a combination of higher CA-125 and HE4 levels are thought to be predictive of malignant ovarian tumours and may serve as a useful diagnostic tool in the future[10]. CA-125 levels can also be used to calculate the risk of malignancy index (RMI), which also utilizes TVUS findings and menopausal status. RMI above 200 is associated with a high risk of malignancy, with a greater than 96 % specificity[8].

The malignancy algorithm (ROMA) risk utilizes a mathematical formula that incorporates HE-4 and CA 125 levels adjusted for pre and post-menopausal status to determine the risk of malignancy[11].

Use of OCP has been associated with a significant reduction in the risk of ovarian cancer. specifically, the risk has been shown to decrease by 10%to 12%after one year of use and by approximately 50%after 5 years of use. Risk reducing salpingo-oophorectomy should be considered for women with germline mutation BRCA1/2, and BRIP1 mutation.

CLINICAL FEATURES:

Epithelial ovarian cancer classically presents with vague persistent gastrointestinal, urologic, or nonacute abdominal/pelvic symptoms, shortness of breath, weight loss, early satiety(bloating, early satiety, discomfort, constipation,vaginal bleeding, indigestion and acid reflux). Ultimately, a pelvic examination or imaging identifies an adnexal mass. While germ cell tumour presents at early age of life.

STAGING OF OVARIAN CARCINOMA

Ovarian cancer is staged according to the 8th edition American Joint Committee of Cancer (AJCC), International Federation of Gynecology and Obstetrics (FIGO) staging system and corresponding Tumor, Node, Metastasis (TNM) classification.

Stage I - Tumor limited to ovaries (one or both) or fallopian tube(s)

- IA - Tumor limited to one ovary (capsule intact) or fallopian tube, no tumor on ovarian or fallopian tube surface; no malignant cells in ascites or peritoneal washings
- IB - Tumor limited to both ovaries (capsules intact) or fallopian tubes; no tumor on ovarian or fallopian tube surface; no malignant cells in ascites or peritoneal washings
- IC - Tumor limited to one or both ovaries or fallopian tubes, with any of the following:
- IC1 Surgical spill
- IC2 Capsule rupture before surgery or tumor on the ovarian or fallopian tube surface
- IC3 Malignant cells in ascites or peritoneal washings

Stage II - Tumor involves one or both ovaries or fallopian tubes with a pelvic extension below pelvic brim or primary peritoneal cancer

- IIA - Extension and/or implants on the uterus and/or fallopian tube(s) and/or ovaries
- IIB - Extension to and/or implants on other pelvic tissues

Stage III - Tumor involves one or both ovaries or fallopian tubes, or primary peritoneal cancer, with microscopically confirmed peritoneal metastasis outside the pelvis and/or metastasis to the retroperitoneal (pelvic and/or para-aortic) lymph nodes

- IIIA1 - Positive retroperitoneal lymph nodes only (histologically confirmed)
- IIIA1i - Metastasis up to and including 10 mm in greatest dimension
- IIIA1ii - Metastasis more than 10 mm in greatest dimension
- IIIA2 Microscopic extrapelvic (above the pelvic brim) peritoneal involvement with or without positive retroperitoneal lymph nodes
- IIIB - Macroscopic peritoneal metastasis beyond pelvis 2 cm or less in greatest dimension with or without metastasis to the retroperitoneal lymph nodes
- IIIC - Macroscopic peritoneal metastasis beyond the pelvis more than 2 cm in greatest dimension with or without metastasis to the retroperitoneal lymph nodes (includes an extension of tumor to the capsule of liver and spleen without parenchymal involvement of either organ)

Stage IV - Distant metastasis, including pleural effusion with positive cytology; liver or splenic parenchymal metastasis; metastasis to extra-abdominal organs (including inguinal lymph nodes and lymph nodes outside the abdominal cavity), and transmural involvement of intestine

- IVA - Pleural effusion with positive cytology
- IVB - Liver or splenic parenchymal metastases; metastases to extra-abdominal organs (including inguinal lymph nodes and lymph nodes outside the abdominal cavity); transmural involvement of intestine

TREATMENT/MANAGEMENT:

SURGERY:

Surgery is the mainstay of treatment in the cases of carcinoma ovary with a goal of R0 or complete resection while the term optimal cytoreduction refers to ≤1 to 2 cm of tumour volume.In the early stages of carcinoma, unilateral

salpingo-oophorectomy will be preferred.However, for advanced-stage ovarian cancer, a debulking surgery comprising hysterectomy/bilateral salpingo-oophorectomy (BSO) has shown better outcomes[4].One of the most powerful independent determinants of improved median survival among patients with stage III or IV ovarian carcinoma is to achieve maximal cytoreduction.Surgical procedures that may be performed in women with ovarian cancer are as follows:

- Surgical staging
- Cytoreductive surgery
- Interval debulking
- Laparoscopic surgery
- Secondary surgery

CHEMOTHERAPY

The use of platinum and taxane basedchemotherapy is the gold standard for adjuvant treatment of advanced EOC. Although these drugs are used for all advanced stage EOCs, they are probably most active in high grade serous and undifferentiated carcinoma.Currently, paclitaxel and carboplatin are administered every 3 weeks as the standard agents for the first-line treatment of advanced ovarian cancer.Postoperative chemotherapy is indicated in all patients with ovarian cancer, except those who have surgical-pathologic stage I disease with low-risk characteristics. Standard postoperative chemotherapy for ovarian cancer is combination therapy with a platinum compound and a taxane (eg, carboplatin and paclitaxel). Additional agents for recurrent disease include the following- liposomal doxorubicin, etopside, topotecan, gemcitabine, vinorelbine, ifosfamide, flurouracil, melphalan, alteramine, bevacizumab, Olaparib, niraparib, rucaparib and pazopanib.

The concept of "dose-dense therapy" is based on the Norton-Simon hypothesis that a shorter interval between the doses of cytotoxic agents is more effective in reducing tumour burden than dose escalation.The Japanese Gynecologic Oncology Group reported the results from a phase III study also proves the same.The median progression-free survival (PFS), the primary endpoint of this study, was substantially improved in the dose-dense treatment group (28 vs 17.2 months, hazard ratio: 0.71, 95% CI: 0.58-0.88, P = 0.0015), and the overall survival (OS) at 3 years was also higher in the dose-dense treatment group (72 · 1%) than in the conventional treatment group (65.1%)[13]

In cases of recurrent ovarian tumour, Platinum free interval [PFI] refers to the interval between the completion of the last platinum-based chemotherapy and the occurrence of relapse[6]. However, platinum sensitivity is generally used to refer to an interval of greater than 6 months between the last platinum-based chemotherapy (PBC) cycle and commencement of subsequent PBC.In such cases we can restart with PBC. The European organization for research and treatment of cancer (EORTC), phase III trial EORTC 55971 recruited women with stage IIIC-IV epithelial ovarian cancer (n=670) and CHORUS trial had a similar recruitment profile with women of stage III A-B besides (n= 550). They showed non-inferiority of median overall survival with neoadjuvant chemotherapy when compared to primary cytoreductive surgery upfront [5].

INTRAPERITONEAL CHEMOTHERAPY

Post operative intra peritoneal chemotherapy [IP] and intravenous [IV] combination therapy for advanced ovarian carcinoma has been studied in four phases III trails by the GOG. Regional therapy takes advantage of both the prolonged confinement with the disease within the peritoneal cavity and the steep dose response relationship observed for most cytotoxic agents. In addition, by exploiting the peritoneal -plasma barrier, the rate of drug clearance is slowed from the peritoneal to systemic compartment and creates a concentration differential favouring the peritoneal cavity.

In hyperthermic intraperitoneal chemotherapy[HIPEC] combines the pharmacokinetic advantage inherent to the intracavitary delivery of certain cytotoxic drugs with the direct cytotoxic effect of hyperthermia. Hyperthermia enhances the tissue penetration of the administered drugs. HIPEC can be administerd by open coliseum technique or by a closed technique.Hyperthermic temperature is monitored using temperature probes placed in bladder, oesophagus and inflow and outflow intra-peritoneal[IP] catheters.

OVARIAN CANCER TARGETED THERAPY

ANTI ANGIOGENESIS THERAPY

Bevacizumab (Avastin) belongs to a class of drugs calledangiogenesis inhibitors.This drug attaches to a protein called VEGF (that signals new blood vessels to form) and slows or stops cancer growth.The most common side effects are hypertension[≥grade 2], gastro-instestinal fistula,perforation. Importantly, gastro-instestinal wall distrubtion [bleeding and impaired wound healing] are black box warnings.

Bevacizumab can also be given with olaparib as maintainence treatment in women whose cancers have a BRCA gene mutation or genomic instability This drug is given as an infusion into the vein (IV) every 2 to 3 weeks.

PARP INHIBITORS

Olaparib (Lynparza), rucaparib (Rubraca), and niraparib (Zejula) are drugs known as a PARP inhibitors [poly {ADP}- ribose polymerase]. PARP enzymes normally help repair damaged DNA inside cells. Mutations in BRCA genes can make it difficult for a cell to repair its DNA. PARP inhibitors can make it even harder for tumour cells with an abnormal BRCA gene to repair damaged DNA, which often leads to the death of these cells. The most common side effect of niraparib, Olaparib and rucaparib are thrombocytopenia[34%], anaemia[18%] and nausea[25%] respectively.

RADIATION ONCOLOGY

In early days, whole abdomen radiation was practiced. however, due to the increased frequency of toxicity and complications, it became non-existent. Adjuvant radiotherapy has not even shown any survival benefit in the early stages of clear cell carcinoma, including a high-risk subset of patients[7].

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DISEASES OF VULVA

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"A single focused examination can prevent years of discomfort –never underestimate vulvar screening"

Introduction

Vulval lesions comprise a broad spectrum of inflammatory, infectious, neoplastic, developmental, and dermatological conditions affecting the external female genitalia. They may present with symptoms such as pruritus, pain, burning, dyspareunia, discharge, ulcers, or be entirely asymptomatic and discovered incidentally. Because many conditions share overlapping clinical features, accurate diagnosis requires a careful history, thorough examination, and, where appropriate, biopsy.

Anatomy Relevant to Vulval Lesions

The vulva includes the mons pubis, labia majora and minora, clitoris, vestibule, urethral meatus, Bartholin and Skene glands, and perineum. It comprises keratinised squamous epithelium externally and non-keratinised epithelium in the vestibule. Rich vascular and lymphatic supply contributes to specific patterns of spread for infections and malignancies.

Classification of Vulval Lesions

1. Congenital / Developmental Lesions

Accessory breast tissue

Epidermal inclusion cysts

Hymenal anomalies

Nevi and vascular malformations

2. Infectious Lesions

a. Viral

Viral infections	Herpes Simplex Virus (HSV 1 & 2)	Human Papillomavirus (HPV)
Clinical features	Painful grouped vesicles → shallow ulcers; dysuria; recurrent pattern.	Condyloma acuminata (genital warts)- : Cauliflower-like papules or plaques.
Diagnosis	PCR swab (most sensitive), clinical appearance.	Visual assessment,Biopsy reserved for atypical pigmented, ulcerated,or uncertain lesions.
Treatment	Oral antivirals (acyclovir/valacyclovir), analgesia.	Podophyllotoxin, imiquimod, cryotherapy, surgical excision.

b. Bacterial

Bacterial	Syphilis (Treponema pallidum)	Chancroid (Haemophilus ducreyi)
Clinical presentation	Painless indurated chancre + lymphadenopathy.	Painful ragged ulcers, tender suppurative inguinal nodes.
Diagnosis	Serology (VDRL/RPR, treponemal tests).	Presumptive clinical diagnosis if all of the following is present 1. one or more painful genital ulcers 2. clinical appearance of ulcer and lymphadenopathy similar to chancroid 3. HCV PCR test and Treponema pallidum microscopy negative.
Treatment	Benzathine Penicillin G	Azithromycin ,Ceftriaxone

c. Fungal

Candidiasis

Symptoms: Pruritus, erythema, fissures, cottage-cheese discharge.

Diagnosis: Microscopy/culture.

Treatment: Topical azoles; oral fluconazole.

d. Parasitic

Pediculosis pubis, scabies - intense pruritus, excoriations.

e. Inflammatory / Dermatological Lesions

Lichen Sclerosis (LS)	Lichen Simplex Chronicus	Lichen Planus	Psoriasis	Contact Dermatitis (Irritant / Allergic)
Chronic inflammatory dermatosis, common in postmenopausal women. Appearance-Porcelain-white plaques, atrophy, "figure-of-eight" pattern around vulva and anus. Symptoms-severe pruritus, pain, dyspareunia Complication-Scarring, architectural distortion, risk of CA vulva Diagnosis- Clinical ± biopsy. Treatment- High-potency topical steroids (clobetasol propionate), long-term maintenance.	Secondary to chronic scratching. Features: Thickened leathery skin, hyperpigmentation. Management:Break itch–scratch cycle; medium- to high-potency steroids.	Autoimmune condition affecting vulva/vagina. Signs: Erosive lesions, Wickham striae, burning pain. Management: Potent topical steroids; systemic therapy for severe disease.	Sharply demarcated erythematous plaques; often non-scaly in intertriginous areas. Topical steroids, vitamin D analogues.	Caused by soaps, pads, tight clothing, topical products. Presents with itching, redness, erosions. Management: Avoid triggers; topical corticosteroids.

4. Cystic Lesions

Bartholin's cyst/abscess – unilateral painful swelling at posterior vestibule.

Epidermoid cysts – firm, round nodules.

Inclusion cysts – post-traumatic or post-episiotomy.

Treatment varies from conservative to surgical excision

5. Premalignant Lesions/Vulvar Intraepithelial Neoplasia (VIN)

Premalignant lesions of vulva are seen in pre-as well as post-menopausal adult womens.These lesions lack a typical clinical presentation and often remain undiagnosed till advanced invasive stages.The 2020 WHO tumour classification divides vulvar lesions into following two categories

Classified into:

HSIL (HPV-related VIN) – Younger women, associated with smoking & oncogenic HPV (16, 18).

dVIN (Differentiated VIN) – Older women, often arises in background of lichen sclerosus; higher malignant potential.

Clinical: White, red, pigmented patches or papules; may be asymptomatic or itchy.

Diagnosis: Colposcopy,Dermoscopy,Biopsy.

Treatment: Wide local excision, Skinning Vulvectomy,laser ablation, imiquimod ,Topical 5FU.

6. Malignant Lesions/Carcinoma of vulva

Carcinoma of the vulva accounts for 3–5% of all female genital tract malignancies. It most commonly affects postmenopausal women, though an increasing proportion of younger patients are affected due to HPV-associated disease. The majority (≈90%) are squamous cell carcinomas.

Epidemiology

Age: Bimodal distribution

Older women: keratinizing SCC, HPV-independent

Younger women: basaloid/warty SCC, HPV-dependent

Risk factors:

HPV 16, 18 (esp. in younger patients)

Vulvar intraepithelial neoplasia (VIN)

Smoking

Immunosuppression (HIV, transplant)

Chronic inflammatory dermatoses (lichen sclerosus ➜ chronic irritation ➜ dVIN SCC)

Older age

Types of Vulvar Cancer

1. Squamous cell carcinoma (90%)
   HPV-associated (warty/basaloid; occurs in younger age group)
   Non-HPV keratinizing type (common in older women; often arises from dVIN)

Others (rare):

2. Melanoma
3. Paget disease (intraepithelial adenocarcinoma)
4. Basal cell carcinoma
5. Bartholin gland carcinoma
6. Sarcoma
7. Adenocarcinoma(Details of majority 3 types of cancers given in text box)

Squamous cell carcinoma (90%)	Melanoma	Paget's Disease
Most common vulvar cancer (≈ 90%). Risk factors: HPV (for HSIL type), chronic inflammation, LS, smoking. Presentation: Persistent ulcer, mass, pruritus, bleeding. Common sites: Labia majora/ minora. Spread: Local extension, lymphatics to inguinal nodes. Diagnosis: Biopsy. Management: Surgery (wide local excision or radical vulvectomy), lymph node assessment; chemoradiation for advanced disease.	Arises from pigmented lesions; aggressive. Requires wide local excision ± sentinel lymph node biopsy.	Red, eczematoid lesion with itching. Often indicates underlying adenocarcinoma. Requires wide excision; recurrence common.

Symptoms and Signs

- Persistent pruritus (most common),Burning, pain, soreness,Discharge or bleeding,
- Nonhealing ulcer, papule, warty growth, or plaque
- Inguinal lymphadenopathy (advanced disease)
- Lesions may be:Ulcerative, exophytic, or infiltrative.Located commonly on labia majora, minora, clitoris, perineum
- Multifocality,Adjacent lichen sclerosus,Lesion size, depth, and palpation for induration,
- Inguinal nodes (size, mobility, tenderness)

Diagnosis

- Clinical Examination

Thorough vulvar inspection under good lighting

Colposcopy may assist in defining lesion margins

Inguinofemoral lymph node assessment

- Biopsy(Gold standard)

Incisional or punch biopsy from thickest/most abnormal area, If multifocal □ multiple biopsies

Document:Histology type,Depth of invasion,Margins (if excisional)

- Imaging

Used for staging and planning lymph node assessment:

MRI pelvis ➜ primary tumor extent

Ultrasound / CT / MRI inguinal nodes

PET-CT if advanced disease/suspicion of metastasis

Differential Diagnosis

- Lichen sclerosus, Lichen planus, Vulvar psoriasis, Chronic infections (HSV), Vulvar intraepithelial neoplasia, Melanoma, Bartholin cyst/tumor.

FIGO Staging (2021 Revision)

Stage I	Tumor confined to vulva/perineum IA: ≤2 cm & stromal invasion ≤1 mm IB: >2 cm or invasion >1 mm
Stage II	Tumour of any size with extension to lower third of vagina, anus with negative nodes
Stage III	Tumour of any size with extension to upper part of adjacent perineal structure or with any number of nonfixed ,non-ulcerated lymph node.Inguinofemoral lymph node involvement IIIA:Tumour of any size with disease extension to upper two third of urethra, upper two thirds of vagina,bladder mucosa or regional lymph node metastasis<5mm IIIB:Regional lymph node metastasis>5mm IIIC:Regional lymph node metastasis with extracapsular spread
Stage IV	Tumour of any size fixed to bone,or fixed,ulcerated lymph node metastasis,or distant metastasis IVA: invasion of upper urethra, bladder, rectum, pelvic bone, or fixed nodes IVB: distant metastasis

Management

Treatment is individualized based on stage, tumor location, size, depth, comorbidities, and node status.

Early-Stage Disease (Stage I–II)

Primary Tumor

Wide local excision or radical local excision. Aim: 1–2 cm gross margin (results in ≥8 mm pathologic margin)

For midline lesions: bilateral nodal assessment usually needed

For lateral lesions (>1 cm from midline): ipsilateral node evaluation

Groin Node Management

Sentinel lymph node biopsy (SLNB) — preferred in selected cases:

Unifocal tumors

Size <4 cm

No clinically suspicious nodes

If SLNB positive ➔ completion inguinofemoral lymphadenectomy

Advanced Disease (Stage III–IVA)

Radical vulvectomy with bilateral inguinofemoral lymphadenectomy

Neoadjuvant chemoradiation to downstage tumors when surgery would be excessively morbid

Adjuvant radiation for:Node positivity,Close/positive margins,LVSI,Large tumors,Metastatic or Recurrent Disease

Radiation Therapy

Indications

- Node positivity
- Margin <8 mm
- Large primary tumors
- Inoperable disease
- Recurrent lesions
- Palliative intent

Follow-Up and Surveillance

- Every 3–6 months for first 2 years
- Every 6–12 months until 5 years
- Annual thereafter

Assess For

- Recurrence (local most common)
- New lesions (HPV or lichen sclerosus background)
- Lymphedema
- Psychosexual complications (very important)

Prognosis depends on:

- Nodal status (strongest predictor)
- Stage at diagnosis
- Margin status
- Tumor size and depth
- Histologic type (dVIN-associated SCC more aggressive)
- Typical 5-yr survival in node-negative cases 70–90% and in node-positive: 25–60%

Carcinoma vulva management requires expertise in detailed vulvar examination, biopsy techniques, appropriate imaging, and correct selection of surgical vs. chemoradiation strategies. The shift toward less radical, function-preserving approaches—especially sentinel lymph node biopsy—has significantly reduced morbidity while maintaining excellent oncologic outcomes.

Approach to a Patient with a Vulval Lesion

History - Duration, progression | Pain, itching, dyspareunia |Discharge or bleeding | Sexual history, STI exposure| Skin disorders, autoimmune disease | Previous treatments | Menopausal status

Examination - Look for colour, borders, moisture, texture, pattern, Presence of ulcers, nodules, plaques, pigmentation, Lymph node assessment, Speculum examination to rule out other genital pathology/malignancy

Investigations

Swabs: HSV PCR, bacterial culture, Candida | Serology for syphilis | Vulvar biopsy (gold standard for atypical, persistent, or pigmented lesions) | Dermatoscopy for pigmented lesions

Management Principles

Treat reversible causes (infections, irritants).

Use biopsies liberally for suspicious, atypical, or persistent lesions.

Provide symptom relief: emollients, antihistamines, appropriate topical steroids.

Address psychosocial impact—many conditions affect sexual function and quality of life.

Follow patients with VIN, LS, and malignancy closely.

Management should ideally be multidisciplinary (dermatologists, gynecologists, oncologists, pathologists, others) - because vulval lesions span dermatologic, infectious, neoplastic and gyn-oncology domain.

Treatment & Surgical Principles

For confirmed invasive disease: radical local excision aiming for tumor-free margins is standard.

In multifocal disease or extensive dermatosis background (e.g., LS), vulvectomy may be needed; radicality can be tailored to preserve function (e.g., avoid unnecessary removal near clitoris, urethra, anus) if feasible.

If excised margins are positive (tumor extends to the cut edge), re-excision is preferred when possible.

For locally advanced disease, a multimodal approach (surgery, radiotherapy, chemotherapy) may be used. Recent guidelines include updated radiotherapy dosimetry, use of image-guided techniques, and caution around toxicity - given morbidity associated with treatment.

Conclusion

Vulval lesions encompass a diverse range of conditions from benign and self-limiting dermatoses to premalignant and malignant disease. A structured clinical approach-beginning with careful assessment, supported by appropriate diagnostic tools such as biopsy-is essential for accurate diagnosis. Early recognition and targeted management not only alleviate symptoms but can prevent serious long-term consequences, including scarring and invasive cancer.

Key Points

- Perform full vulvar examination, Document thoroughly.
- Provide patient education and counselling — about chronic nature of some vulvar diseases, need for maintenance therapy, risk of malignant transformation (in conditions like LS), importance of regular follow-up, and psychological / sexual health support.

Expert eyes, early diagnosis –simple steps that save women from complex vulvar conditions.

EVALUATION OF POST-MENOPAUSAL BLEEDING

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Introduction

Postmenopausal bleeding is a common complaint with a broad differential, which includes both benign and malignant conditions. It is defined as an episode of bleeding occurring 12 months or more after the final menstrual period; however it is recommended that any vaginal bleeding that occurs 6 months after the last period (presumed menopause) should be investigated. Post menopausal bleeding is reported in about 4-11% of women' and it accounts for approximately 5% of gynecologic office visits' postmenopausal bleeding is "endometrial cancer" until proven otherwise, although only 1-14% of such patients will actually have endometrial cancer. Vaginal bleeding is the presenting sign in more than 90% of postmenopausal women with endometrial cancer.

Etiopathology

Postmenopausal bleeding usually attributed to an intrauterine source, but it may arise from the cervix, vagina, vulva and ovaries. The origin of bleeding can also involve non-gynaecologic sites, such as the urethra, bladder, anus/rectum/bowel, or perineum. Possible causes of postmenopausal include endometrial atrophy (most common), endometrial polyps,hyperplasia, estrogen therapy ,cancers(Table - 1). Lack of estrogen causes atrophy of the vagina and endometrium. Inside the uterus, the collapsed and atrophic surfaces of the endometrium contain scanty or no fluid to prevent friction inside the cavity' This leads to the development of micro-erosions of the epithelial surface, with subsequent chronic inflammation. This chronic endometritis is prone to spotting or light bleeding. Some women have genetic predispositions to endometrial cancer, including Lynch syndrome and Cowden disease.

Table 1 Causes of Postmenopausal bleeding

S.NO.	CAUSES
1.	ATROPHY-ENDOMETRIAL OR VAGINAL
2.	ENDOMETRIAL HYPERPLASIA WITH OR WITHOUT ATYPIA
3.	ENDOMETRIAL OR CERVICAL POLYPS
4.	ENDOMETRIAL CANCER
5.	NFECTION-ENDOMETRIAL, CERVICAL OR VAGINAL
6.	DRUGS POST-MENOPAUSAL HORMONE THERAPY, TAMOXIFEN, ANTICOAGULANTS, HERBAL SUPPLEMENTS(PHYTOESTROGEN)
7.	CERVICAL CANCER, VAGINAL OR VULVAR CANCER, OVARIAN CANCER, ESPECIALLY ESTROGEN-SECRETING TUMOUR
8.	FORGOTTEN PESSARY
9.	POST-RADIATION EFFECTS
10.	NON- GYNAECOLOGICAL SUCH AS TRAUMA OR BLEEDING DISORDER.

Evaluation

History

The nature of current bleeding - first episode, persistent or recurrent, duration, frequency, length and quantity of bleeding and its relation with coitus, pain should be elicited. Previous menstrual history, parity, past medical and family history of breast, colon, and endometrial cancer need to be elicited. Women with family history of hereditary non-polyposis colorectal cancer have a lifetime risk of developing endometrial cancer of around 12.10% The presenco t other co-morbid factors such as Diabetes. Hypertension, and use of unopposed Fetrogen thereov should be recorded In addition the women should be asked about history of treatment with Herbal supplements

(Phyloestrogen), Tamoxifen, Tibolone and cyclical/continuous combined HT. Early menarche and late menopause are also associated with increased likelihood of Endometrial hyperplasia / Carcinoma.

Examination - A thorough general and local examination is mandatory. Focus of this is to rule to determine site of bleeding & to rule out vulval, vaginal, cervical and pelvic pathology. Clinical examination should include Speculum examination, Bimanual examination & digital rectal examination.

Investigations - The principal aim of investigation is to identify or exclude endometrial pathology, most notably endometrial carcinoma.

TVS - The initial assessment in all cases of PMB should be using the TVS. Measurement of the endometrium on TVS should include the full double thickness of the endometrium with any content within the endometrial cavity. The American College of Obstetricians and Gynecologists recommend transvaginal ultrasound for initial evaluation4. The endometrial thickness is measured in an anterior-posterior fashion, at the area of endometrial echo of maximal thickness, on a long-axis view of the uterus4. If endometrial thickness >4 mm, hysteroscopic evaluation and endometrial sampling is recommended. An endometrial thickness of less than or equal to 4 mm has a negative predictive value greater than 99% for endometrial carcinoma.

Endometrial Sampling

Indications

1) Findings on ultrasound for which endometrial sampling are indicated include:
   a. A thickened endometrial lining greater than 4 mm
   b. Diffuse or focally increased echogenicity or heterogeneity
   c. The inability to visualize the endometrium adequately
2) Endometrial sampling should also be obtained in patients with persistent or recurrent bleeding, even in thin endometrial echo
3) Asymptomatic postmenopausal women with endometrial thickness> 11 mm (secondary finding) [1]

   The use of 4 mm endometrial thickness as a threshold may miss endometrial cancer for 1 in 339 patients4. The diagnostic accuracy of endometrial sampling correlates positively with the amount of tissue that is collected [7].

Methods of endometrial sampling.

1) Dilation and curettage- has been used for years, now a days isolated d & c should not be used because approximately 60% of curettage specimens sample less than half of the uterine cavity so there is chance of missing pathology [1].
2) Office endometrial biopsy using metal curettes or flexible plastic samplers.
3) Hysteroscopy guided endometrial sampling (Gold standard)

Office endometrial biopsy metal curette or flexible olastic sampler) is a blind sampling method & it may miss foca lesions or intrauterine pathology.it is common for endometrial sampling to result in findings that are insufficient for diagnosis, with rates of samoling failure un to 54%[4]. If sampling was performed first and was inadequate: a follow-up ultrasound may be performed. If a subsequent transvaginal ultrasound shows a thin endometrium, and if bleeding has stopped, no turther evaluation is necessary. For patients with insufficient sampling, or with persistent vaginal bleeding in whom focal lesions may have been missed, additional evaluation should be considered. Hysteroscopy with dilation and curettage or directed biopsy may be warranted in these patients.

MRI - MRI is of value only in Endometrial carcinoma in delineating the size, site and myometrial invasion. Its accuracy is 98% and 90% for myometrial and cervical stromal invasion respectively. The presence of enlarged lymph nodes and cervical involvement can be made out.

Treatment - Treatment of postmenopausal bleeding is focused on the cause; it depends on findings of diagnostic evaluation.

Exclusion of cancer is main objective.

Endometrial Hyperplasia- Mainly depends on histological classification and risk factors (obesity, ovulatory dysfunction, increased age, genetic risk)

New WHO 2014 classification of endometrial hyperplasia Hyperplasia without atypia.

Hyperplasia with atypia or Endometrial Intraepithelial Neoplasia (EIN).

Endometrial hyperplasia without atypia.	Endometrial hyperplasia with atypia/EIN
The risk of progression to invasive malignancy is less than 5%	The risk of progression to invasive malignancy. as high as 27.5% if not treated. Possibility of coexistent endometrial malignancy in atypia/EIN is in around 43% of cases.
Repeat endometrial biopsy every 3-6 months upto 1 year or until biopsy show normal.	Hysterectomy is curative & preferred treatment
Progestin therapy, LNG-IUS (rate of regression upto 90%)	Progestin therapy (LNG-IUS) shown regression upto 75-85%
Hysterectomy if a)No improvement b)Normal endometrium not achieved Atypical hyperplasia or carcinoma develop	Note-Endometrial ablation, morcellation or supracervical hysterectomy should never be performed

Endometrial Malignancy/Endometrial Adenocarcinoma- Hysterectomy with comprehensive staging is recommended. Comprehensive staging consists of total hysterectomy, bilateral salpingo-oophorectomy, pelvic para-aortic lymphadenectomy and collective peritoneal washing for cytology.

Staging allows for appropriate diagnosis, determination of prognosis, and to triage patients for adjuvant therapy appropriately. FIGO committee updated new staging system.

The updated 2023 staging includes the various

histological types, tumour pattern, and molecular classification to better reflect the improved understanding of the complex nature of the several types of endometrial carcinoma and their underlying biological behaviour.

Table 3 Endometrial Cancer risk groups

Risk group	Description
Low risk	Stage IA (G 1-G2) with endometroid type (dMMR and NSMP) and no or focal LVSI. Stage I/II POLEmut for stage III POLEmut cancer.
Intermediate risk	Stage IA G3 with endometrioid type (dMMR and NSMP) and no or focal LVSI. Stage IA non endometrioid type (serous, clear-cell, undifferentiated carcinoma, carcinosarcoma, mixed) and/or p53-abn cancers without myometrial invasion and no or focal LVSI. Stage IB (G1-G2) with endometrioid type(dMMR and NSMP) and or no LVSI. Stage I G1 endometrioid type (dMMR and NSMP) and no or focal LVSI.
High- Intermediate risk	Stage I endometroid type (dMMR and NSMP) any grade and any depth of invasion with substantial LVSI. Stage IB G3 with endometrioid type (dMMR and NSMP) regardless of LVSI. Stage II G1 endometrioid type (dMMR and NSMP) with substantial LVS| Stage II G2-G3 endometrioid type (dMMR and NSMP).
High-Risk	All stages and all histologies with p53-abn and myometrial invasion. All stages with serous or undifferentiated carcinoma including carcinosarcoma with myometrial invasion. All stage Ill and IVA with no residual tumour, regardless of histology and regardless of molecular subtype.

Treatment- Endometrial cancer is surgically staged and pathologically examined, in all stages, the grade of the lesion, the histological types and LVSI must be recorded. If available and feasible molecular classification testing POLEmut, MMRd, NSMP,p53abn) is encouraged in all patients with endometrial cancer for prognostic risk group stratification 9. Treatment of endometrial cancer depends on stage (table 2), histopathological type and grade, and on subgroup of risk classification system (Table 3).

Atrophic vaginitis is a common cause of PMB and is effectively treated with local application of estrogen. Endometrial polyps are best removed during the Hysteroscopic evaluation and sent for HPE. The endometrium should also be sampled and sent for HPE as the likelihood of Endometrial hyperplasia is high (3% of polyps)

Submucous leiomyoma- Hystroscopic removal of leiomyoma, hysterectomy Endometritis or Cervicitis-Antibiotics should be given.

KEY POINTS

1. Postmenopausal bleeding is "endometrial cancer" until proven otherwise, although only 1-14% of such patients will actually have endometrial cancer.
2. Women with PMB with an endometrial thickness of > 4mm should undergo endometrial sampling
3. Hysteroscopy guided endometrial sampling is considered gold standard as it helps in indentifying other endometrial pathology like endometrial polyp, submucous leiomyoma, intrauterine adhesions or other focal pathology.
4. Isolated dilatation and curettage should not be used as the first line method for obtaining endometrial sampling.
5. Women on Tamoxifen with abnormal uterine bleeding should be offered diagnostic hysteroscopy with endometrial sampling as TVS for assessment of endometrium in these women is not useful for triage.
6. Endometrial sampling should also be obtained in patients with persistent or recurrent bleeding, even in thin endometrial echo
7. In patients with endometrial cancer should undergo surgical staging, including hysterectomy ,bilateral salpingo-oophorectomy, peritoneal cytology and sentinel node biopsy or sentinel lymphadenectomy(LNE)
8. In all stages of EC, the grade of the lesion, the histological types and LVS| must be recorded. If available and feasible ,molecular classification testing (POLEmut,MMRd, NSMPp53abn) is encouraged in all patients with endometrial cancer for prognostic risk group stratification8.

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KEYNOTE ON MANAGING OSTEOPOROSIS IN MENOPAUSE

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Osteoporosis in menopause requires a lifelong, multipronged strategy: early risk assessment, bone-friendly lifestyle, targeted pharmacologic therapy, and fall prevention, all tailored to an individual woman's fracture risk.[1][2]

Why menopause drives osteoporosis

- Estrogen falls sharply at menopause, accelerating bone resorption and leading to rapid bone loss over the first 5–10 years after the final period.[1][3]

- One in two postmenopausal women will experience an osteoporotic fracture in her lifetime, often at the spine, hip, or wrist, with major implications for independence and mortality.[2][4]

Risk assessment and diagnosis

- Clinical evaluation should document age, prior fragility fractures, low BMI, family history, smoking, alcohol use, glucocorticoids, rheumatoid arthritis, and secondary causes (thyroid, parathyroid, renal disease, malabsorption).[1][5]

- Bone mineral density (BMD) by DXA of hip and spine plus a fracture-risk tool (such as FRAX) is recommended to classify women as low, moderate, high, or very high fracture risk and to guide treatment thresholds.[6][5]

Core lifestyle and nutritional measures

- Ensure around 1,200 mg/day of elemental calcium (diet plus supplements) and 800–1,000 IU/day of vitamin D for most postmenopausal women, especially those at risk of deficiency.[7][2]

- Regular weight-bearing and resistance exercise (such as brisk walking, stair climbing, dancing, and strength training 2–3 times weekly) improves or maintains BMD and reduces fracture risk when combined with adequate calcium and vitamin D.[8][7]

- Additional measures include avoiding smoking, limiting alcohol, optimizing protein intake, maintaining a healthy weight, and using balance training (e.g., tai chi) to reduce falls.[1][9]

Pharmacologic treatment options

- For women at high or very high fracture risk, first-line antiresorptives typically include oral or intravenous bisphosphonates (e.g., alendronate, risedronate, zoledronic acid) or the RANKL inhibitor denosumab, all of which reduce vertebral and hip fractures.[10][11]

- Anabolic or dual-effect agents (teriparatide, abaloparatide, romosozumab) are recommended for very high-risk women (such as those with multiple or recent vertebral fractures), usually for a limited course followed by an antiresorptive to maintain gains.[10][12]

- Selective estrogen receptor modulators (e.g., raloxifene) and menopausal hormone therapy can be considered in younger postmenopausal women with vasomotor symptoms and moderate fracture risk, balancing benefits against thromboembolic and breast risk profiles.[1][13]

Overview of major drug classes

Class / example	Main role	Key cautions
Bisphosphonates (alendronate, zoledronic acid)	First-line antiresorptives to lower vertebral, non-vertebral, and hip fractures.[10][11]	Renal impairment, rare atypical femur fracture, osteonecrosis of jaw with long

use.[11] |

| Denosumab | Potent antiresorptive, strong hip and spine BMD gains and fracture reduction.[10][14] | Must avoid unplanned stops due to rebound bone loss; ensure follow-on therapy.[10] |

| Teriparatide/abaloparatide| Anabolic agents for very high-risk or multiple vertebral fractures.[12][1] | Limit duration; avoid in some malignancy or hypercalcemia settings.[12] |

| Romosozumab | Dual effect, rapid BMD increases in severe osteoporosis.[10][12] | Avoid in women with prior MI or stroke because of possible CV risk.[12] |

| SERMs (raloxifene) | Vertebral fracture reduction, breast cancer risk reduction.[1][11] | VTE risk, hot flushes.[11]

| Menopausal hormone therapy | Prevents early postmenopausal bone loss, reduces fractures in younger symptomatic women.[1][7] | Not first-line purely for bone in older women; assess VTE, stroke, breast risk.[1] |

Monitoring, duration, and fall prevention

- BMD is generally rechecked every 1–2 years in treated high-risk women, and treatment response is interpreted together with clinical events (new fractures, height loss, back pain suggesting vertebral fracture).[6][2]
- After 3–5 years of oral or 3 years of IV bisphosphonate, reassessment helps decide on continuing versus a "drug holiday" in lower-risk women, while high-risk women typically continue or switch therapy.[10][11]
- Comprehensive fall-prevention includes vision checks, home hazard reduction, appropriate footwear, medication review (sedatives, hypotensives), and referral to physiotherapy or balance programs for frail women.[1][9]

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CERVICAL CANCER SCREENING PROTOCOLS IN THE ERA OF HPV-BASED STRATEGIES: CONSENSUS FROM WHO, ESGO, AND NCCN WITH A FOCUS ON LOW-RESOURCE SETTINGS

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Background and Rationale

Cervical cancer remains a largely preventable cause of morbidity and mortality, yet it continues to disproportionately affect women in low- and middle-income countries where organized screening is limited or absent. Cervical cancer is the fourth most common cancer among women globally, with an estimated 662,000 new cases and 349,000 deaths in 2022, the majority in low-resource regions. Persistent infection with high riskHPV is a necessary cause of cervical cancer, creating a clear target for primary prevention (vaccination) and secondary prevention (screening). The WHO Cervical Cancer Elimination Initiative aims for 70% of women screened with a high-performance test and 90% of those with precancer treated, which requires shifting from opportunistic cytology to organized, HPV-based screening programs.

Guideline Overview: WHO, ESGO, NCCN

WHO guideline framework

The 2021 WHO guideline for screening and treatment of cervical pre-cancer recommends high risk HPV DNA testing as the preferred primary screening method for both the general population and women living with HIV. WHO advises:

- Start age: 30 years for the general population; 25 years for women living with HIV.
- Interval: Every 5–10 years for HPV-negative women in the general population; shorter intervals (3–5 years) for women living with HIV.
- Algorithm: Implement either a screen–triage–treat or screen–treat strategy, adapted to local resources and capacity.

Visual inspection with acetic acid (VIA) and cytology may be done temporarily where quality-assured HPV testing is not yet available, but WHO recommends rapid transition to HPV-based screening because of its higher sensitivity and the challenges of maintaining quality in VIA programs.

ESGO position

The ESGO–EFC position paper on cervical screening recognizes HPV testing as replacing cytology as the primary test in organized programs, given its greater protection against cervical cancer and allowance for longer screening intervals. ESGO highlights:

- Target group: Women 25–65 years in organized, population-based programs with defined intervals and registries.
- Interval: 3–5 years with cytology, 5–7 (up to 10) years with validated high risk HPV tests for HPV-negative women, depending on national policy.
- Triage: Because HPV tests are less specific, triage is mandatory (e.g., cytology, partial genotyping, or other biomarkers) to select women for colposcopy.
- Organization: ESGO strongly emphasizes organized screening, quality assurance benchmarks, and

continuous monitoring of coverage, positivity, colposcopy yield, and cancer incidence.

NCCN perspective

NCCN provides detailed, risk-based algorithms for screening and subsequent management, closely aligned with North American guidance. For average-risk individuals with a cervix:

- Age 25–65:
  - Primary high riskHPV testing every 5 years (preferred where available).
  - Alternatives: Co-testing (cytology + hrHPV) every 5 years or cytology alone every 3 years when HPV testing is unavailable.
- Below 25: Cytology alone at 3-year intervals; HPV testing generally not used for primary screening.

NCCN and related US bodies stress integration with HPV vaccination, recognizing that higher vaccination coverage will ultimately permit later initiation and fewer lifetime screens in vaccinated cohorts, although empirical data are still evolving.

Core Screening Protocol: Average-Risk Women

A harmonized protocol for average-risk women, consistent with WHO, ESGO, and NCCN, can be summarized as follows:

Target population and initiation

- Age at first screen:
  - 25–30 years, with many programs adopting 25 in high-resource settings and 30 in low-resource contexts to optimize cost-effectiveness.
- Upper age limit:
  - 65 years, with possible cessation if there is a documented history of adequate negative screening and no history of CIN2+ in the previous 20 years.

Primary screening test and interval

- Primary test: Validated high-performance high risk HPV DNA test on clinician- or self-collected specimens.
- Interval:
  - HPV-negative: 5–10 years (WHO), often set at 5 years in practice; ESGO suggests 5–7 years; NCCN/US practice typically uses 5 years.
- Alternative strategies when HPV testing unavailable: Cytology every 3 years or VIA at 3–5-year intervals, with clear plans to transition to HPV testing.

Triage of HPV-positive women

Given the lower specificity of HPV testing, triage is essential to determine which HPV-positive women require colposcopy or treatment.

Common triage options include:

- Reflex cytology:
  - HPV-positive samples undergo cytology; women with ASC-US+ are referred for colposcopy.
- Partial genotyping:
  - Immediate colposcopy for HPV16/18 positive; cytology triage for other high risk HPV types.
- VIA:
  - Particularly relevant in low-resource settings, used as a point-of-care triage test with potential for same-day management.

A typical algorithm:

- HPV negative ➜routine recall at 5 years.
- HPV positive, cytology ≥ASC-US or VIA positive ➜ colposcopy or immediate ablation/excision depending on setting and eligibility.
- HPV positive, cytology negative (or VIA negative) ➜ repeat HPV test at 12 months or risk-based reassessment.

Special Populations and Risk Adaptation

Women living with HIV

Women living with HIV (WLHIV) have higher prevalence and persistence of high riskHPV and accelerated progression to high-grade lesions. WHO therefore recommends:

- Start screening at age 25.
- Interval: Every 3 years with HPV testing; shorter intervals post-treatment or after a positive test.
- Algorithm: Screen–triage–treat with HPV primary testing followed by triage (e.g., VIA, cytology, or HPV16/18 genotyping), ensuring robust referral and treatment capacity.

Vaccinated cohorts

ESGO and ACS highlight that the rise of HPV vaccination will ultimately allow fewer lifetime screens and potentially later start age, but current recommendations still largely align with those for unvaccinated cohorts until more outcome data are available. Screening must continue because vaccines do not cover all oncogenic HPV types and many women remain unvaccinated or incompletely vaccinated.

Programmatic Design: Organized vs Opportunistic Screening

Organized screening programs with central registries and systematic invitations have superior effectiveness and equity compared with opportunistic screening. Key components include:

- Defined target population, test, and interval.
- Individual invitations and recalls; tracking of non-attenders.
- Standardized management algorithms and integrated referral networks.
- Ongoing monitoring of participation, positivity, colposcopy rates, CIN2+ detection, and interval cancers.

ESGO explicitly discourages primary HPV testing outside organized programs and recommends against routine double screening (co-testing) because it adds cost without clear benefit over HPV alone when systems are robust.

Screening in Low-Resource Settings

Choice of screening test

In low-resource settings, selection of the screening test must balance performance, cost, infrastructure, and feasibility.

- HPV testing (preferred)
- VIA (interim or adjunct)

Screen–triage–treat vs screen-and-treat

WHO explicitly encourages simplified algorithms to reduce missed opportunities:

- Screen–triage–treat:
  - HPV testing (or VIA) ➜ triage test (e.g., VIA, cytology, genotyping) ➜ treatment of those with positive triage.
  - More targeted but requires multiple steps and robust follow-up systems.
- Screen-and-treat:
  - Women who test positive (e.g., HPV-positive or VIA-positive) receive treatment (e.g., ablative therapy) at the same visit if eligibility criteria are met; colposcopy or excision is reserved for those not eligible.
  - Particularly valuable where loss to follow-up is high and access to colposcopy is limited.

Age range and interval in low-resource settings

WHO recommends focusing limited resources on the age range with the highest yield:

- Target age: 30–49 years (up to 65 where feasible).
- Interval:
  - HPV-based: Every 5–10 years for HPV-negative women; often operationalized as one or two lifetime screens (e.g., at 35 and 45) where resources are severely constrained.
  - VIA: Every 3–5 years, acknowledging lower sensitivity and shorter duration of protection.

Delivery strategies

To achieve high coverage in low-resource settings:

- Community outreach and campaigns:
  - Community health campaigns and mobile clinics can reach under-screened rural populations and have shown promising coverage and cost-effectiveness.
- Task-shifting:
  - Training nurses, midwives, and community health workers to perform VIA, collect HPV samples, and deliver ablative treatment expands capacity.
- Self-sampling:
  - Mailing or providing self-collection kits, or supervised community self-sampling, can reach women who do not attend facilities.
- Integration with other services:
  - Combining cervical screening with maternal health, family planning, or HIV care visits can improve efficiency and uptake.

Treatment options in low-resource contexts

For screen-positive women, WHO recommends:

- Ablative therapies (e.g., cryotherapy, thermal ablation) as first-line where lesions meet ablative eligibility criteria and invasive cancer is not suspected.
- Excisional treatment (e.g., loop electrosurgical excision procedure) or referral to higher-level facilities when ablative criteria are not met or invasive disease is suspected.
- For women living with HIV, closer follow-up and lower thresholds for referral due to higher recurrence risk.

Quality Assurance and Monitoring

High-quality screening demands robust quality assurance at every step:

- Test performance
- Training and certification:
- Indicators:
  - Coverage of target population.
  - HPV positivity or VIA positivity rate.
  - Proportion of screen-positive women completing triage and treatment.
  - CIN2+ detection rates and stage distribution of invasive cancers.
- **Audit:** ESGO stresses auditing the screening histories of women who develop invasive cancer to identify missed opportunities and system weaknesses.

A practical, guideline-concordant protocol might follow:

1. Offer validated high risk HPV testing as the primary screen from age 25–30 to 65.
2. For HPV-negative women, recall in 5 years.
3. For HPV-positive women:
   - Perform reflex cytology (or VIA where cytology is not available).
   - Refer those with abnormal triage (ASC-US+ or VIA-positive) for colposcopy or immediate ablation where appropriate.
   - Repeat HPV testing at 12 months for HPV-positive/triage-negative women.
4. In settings where HPV testing is not yet feasible, implement VIA-based screen-and-treat for women 30–49 every 3–5 years, with clear plans to transition to HPV testing.

Future Directions

Guidelines will continue to evolve as vaccinated cohorts age into the screening pool and as new biomarkers (e.g., dual-stain cytology, methylation markers) and simplified, low-cost HPV assays mature. Adaptive, risk-based strategies will likely refine intervals and ages further, but the unifying principle remains high coverage with a high-performance primary test, supported by robust systems for triage, treatment, and follow-up.