FINAL YEAR MBBS · CLINICAL VIVA

Gynaecology Viva Voce

Common long and short cases for MBBS clinical exams
Based on *Gynaecology by Ten Teachers*

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700 viva questions with model answers · 14 cases

How to use this book

Each chapter is one common examination case. It opens with the typical case you may meet in the exam, followed by 50 questions that examiners commonly ask, from simple to hard: history and examination, diagnosis, investigations, management and complications.

The answers are short model answers, the way an examiner wants to hear them. Read the question, answer it aloud first, then check the model answer. Guidance follows UK practice as taught in Ten Teachers (NICE and RCOG); follow your local hospital protocols where they differ.

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CASE 1

Uterine Fibroids

Typical exam case: A 40-year-old P3 complains of heavy, prolonged periods for 2 years with tiredness. On examination there is a firm, irregular, non-tender pelvic mass equal to a 16-week pregnant uterus. Her haemoglobin is 88 g/L.

HISTORY AND EXAMINATION

Q1. What are fibroids?

> Benign smooth muscle tumours of the uterus (leiomyomas), the most common pelvic tumour in women.

Q2. How common are fibroids?

> Present in up to 40–70% of women by age 50; more common in women of African origin.

Q3. What are the risk factors for fibroids?

> Age 30–50, African ethnicity, nulliparity, obesity, early menarche and family history.

Q4. Which factors reduce fibroid risk?

> Higher parity, use of the combined pill and smoking (but smoking is not advised).

Q5. What are the common symptoms of fibroids?

> Heavy menstrual bleeding, pelvic pressure, urinary frequency, constipation, pain and subfertility. Many are symptom-free.

Q6. Why do submucosal fibroids cause heavy bleeding?

> They enlarge and distort the endometrial surface and interfere with uterine contraction.

Q7. What pressure symptoms can large fibroids cause?

> Urinary frequency, retention, hydronephrosis (ureteric compression), constipation and leg oedema.

Q8. What would you find on abdominal examination?

> A firm, irregular, non-tender mass arising from the pelvis; you cannot get below it.

Q9. What would you find on bimanual examination?

> An enlarged, irregular, firm uterus that moves with the cervix.

Q10. How do you distinguish a fibroid from an ovarian mass clinically?

> A fibroid moves with the cervix and there is no groove between it and the uterus; an ovarian mass may be separate, with a groove.

CLASSIFICATION AND PATHOLOGY

Q11. How are fibroids classified by location?

> Submucosal (into the cavity), intramural (in the wall) and subserosal (under the serosa); some are pedunculated or cervical.

Q12. What is the FIGO classification of fibroids?

> A 0–8 system: type 0 is pedunculated intracavitary, types 1–2 submucosal, 3–4 intramural, 5–7 subserosal and 8 other (e.g. cervical).

Q13. What is the macroscopic appearance of a fibroid?

> A firm, well-defined, whorled white tumour with a pseudocapsule.

Q14. What hormones stimulate fibroid growth?

> Oestrogen and progesterone.

Q15. What happens to fibroids after the menopause?

> They usually shrink.

Q16. What are the types of fibroid degeneration?

> Hyaline (most common), cystic, calcific, red (carneous) and sarcomatous.

Q17. What is red degeneration?

> Haemorrhagic infarction of a fibroid, typically in pregnancy, causing acute pain, low fever and tenderness.

Q18. How rare is sarcomatous change?

> Very rare, less than 0.5%. Suspect if a fibroid grows rapidly after menopause.

INVESTIGATIONS

Q19. What is the first-line investigation?

> Pelvic ultrasound (transabdominal and transvaginal).

Q20. Why check the FBC?

> To detect iron deficiency anaemia from heavy bleeding.

Q21. What is the role of MRI?

> To map fibroids before surgery or embolisation, and to help distinguish fibroids from adenomyosis or sarcoma.

Q22. What is hysteroscopy used for?

> To assess and remove submucosal fibroids and to sample the endometrium.

Q23. When is endometrial sampling needed?

> In women over 45 with abnormal bleeding, or with persistent intermenstrual bleeding or risk factors for endometrial cancer.

Q24. What is a saline infusion sonohysterogram?

> Ultrasound after instilling saline into the uterus to show submucosal fibroids and polyps.

Q25. Why is a pregnancy test done?

> To exclude pregnancy as a cause of an enlarged uterus.

Q26. Why check renal function in large fibroids?

> Ureteric compression can cause hydronephrosis.

Q27. When would you check thyroid function?

> If there are other symptoms suggesting thyroid disease; it is not routine for heavy bleeding.

Q28. Why check the smear history?

> To ensure cervical screening is up to date before surgery.

MANAGEMENT

Q29. How are asymptomatic fibroids managed?

> Reassurance and observation; no treatment is needed.

Q30. What medical treatments reduce heavy bleeding with fibroids?

> LNG-IUS (if the cavity is not distorted), tranexamic acid, NSAIDs, combined pill and progestogens.

Q31. What drug shrinks fibroids before surgery?

> GnRH agonists (e.g. goserelin) for 3–6 months; they also improve anaemia.

Q32. What are the side effects of GnRH agonists?

> Menopausal symptoms and bone loss, so use is limited to about 6 months unless add-back therapy is given.

Q33. What is relugolix combination therapy?

> An oral GnRH antagonist with oestradiol and norethisterone add-back for long-term control of fibroid bleeding.

Q34. Why is ulipristal acetate rarely used now?

> Because of rare but serious liver injury; it is restricted.

Q35. What is hysteroscopic myomectomy?

> Removal of submucosal fibroids through a hysteroscope; best for type 0 and 1 fibroids under 3–5 cm.

Q36. What is myomectomy?

> Surgical removal of fibroids keeping the uterus, by laparotomy or laparoscopy; for women wishing to keep fertility.

Q37. What are the risks of myomectomy?

> Bleeding and transfusion, conversion to hysterectomy, adhesions, recurrence, and uterine rupture in future pregnancy.

Q38. How is bleeding reduced at myomectomy?

> Pre-operative GnRH agonists, vasopressin injection, tourniquets and tranexamic acid.

Q39. What is uterine artery embolisation?

> Blocking the uterine arteries by interventional radiology to shrink fibroids; an option for women who want to avoid surgery.

Q40. What are the complications of uterine artery embolisation?

> Post-embolisation pain and fever, infection, fibroid expulsion, ovarian failure and uncertain effects on fertility.

Q41. What is the definitive treatment?

> Hysterectomy, for women who have completed their family.

Q42. What approach to hysterectomy may be used for a 16-week fibroid uterus?

> Total abdominal hysterectomy, or laparoscopic hysterectomy in skilled hands; vaginal hysterectomy is harder with a large uterus.

Q43. What is the management of red degeneration in pregnancy?

> Conservative: rest, analgesia (paracetamol, opioids) and reassurance; avoid NSAIDs in late pregnancy.

COMPLICATIONS AND COUNSELLING

Q44. What are the complications of fibroids?

> Anaemia, pressure symptoms, degeneration, torsion of a pedunculated fibroid, infertility and, rarely, sarcoma.

Q45. How do fibroids affect fertility?

> Submucosal fibroids reduce implantation and increase miscarriage; removal improves outcomes.

Q46. What are the complications of fibroids in pregnancy?

> Red degeneration, miscarriage, malpresentation, obstructed labour, caesarean section and PPH.

Q47. Should fibroids be removed at caesarean section?

> Usually not, because of heavy bleeding risk, unless pedunculated or blocking closure.

Q48. How would you correct her anaemia before surgery?

> Iron (oral or IV) and GnRH agonist to stop bleeding and allow Hb to rise.

Q49. What is the recurrence risk after myomectomy?

> About 15–30% over 5–10 years.

Q50. What is a recent advance in fibroid treatment?

> Oral GnRH antagonists with add-back (relugolix, linzagolix) and radiofrequency ablation of fibroids.

CASE 2

Uterovaginal Prolapse

Typical exam case: A 58-year-old P6, postmenopausal for 8 years, complains of a 'something coming down' feeling in the vagina for 2 years, worse on standing and at the end of the day. On examination the cervix descends beyond the introitus on straining.

HISTORY AND EXAMINATION

Q1. What is pelvic organ prolapse?

Descent of one or more of the uterus, vaginal vault, anterior or posterior vaginal walls.

Q2. What are the main symptoms?

A lump or bulge in the vagina, dragging discomfort, backache, and bladder, bowel or sexual symptoms.

Q3. What urinary symptoms are linked with prolapse?

Frequency, urgency, incomplete emptying, needing to push the prolapse back to void, and stress incontinence.

Q4. What bowel symptoms are linked?

Constipation, incomplete emptying and needing to press on the vagina or perineum to open the bowels (digitation).

Q5. What are the risk factors for prolapse?

Vaginal births (especially large babies, prolonged second stage, instrumental births), age, menopause, obesity, chronic cough, constipation, heavy lifting and connective tissue disorders.

Q6. Why does menopause increase the risk?

Loss of oestrogen weakens pelvic connective tissue.

Q7. Which position is best for examining prolapse?

Left lateral with a Sims speculum, and also standing if the prolapse is not seen lying down.

Q8. Why is a Sims speculum used?

It retracts one vaginal wall at a time to show the anterior wall, posterior wall and apex separately.

Q9. What else would you look for on examination?

Ulceration of the cervix (decubitus ulcer), vaginal atrophy, stress incontinence on coughing, a pelvic mass and the BMI.

Q10. Why examine the abdomen?

To exclude a pelvic mass or ascites that may push the uterus down.

CLASSIFICATION

Q11. What is a cystocele?

Prolapse of the bladder into the anterior vaginal wall.

Q12. What is a rectocele?

Prolapse of the rectum into the posterior vaginal wall.

Q13. What is an enterocele?

Herniation of small bowel through the pouch of Douglas into the upper posterior vaginal wall.

Q14. What are the traditional degrees of uterine prolapse?

> First degree: cervix within the vagina. Second degree: cervix reaches the introitus. Third degree (procidentia): the uterus lies outside the introitus.

Q15. What is the POP-Q system?

> A standard system measuring points on the anterior wall, posterior wall and apex in relation to the hymen, giving stages 0–4.

Q16. What is a decubitus ulcer?

> An ulcer on the prolapsed cervix from friction and poor blood supply; it must be distinguished from cancer.

Q17. What supports the uterus normally?

> Level 1: uterosacral and cardinal ligaments; level 2: paracolpos attachments to the pelvic side wall; level 3: perineal body and urogenital diaphragm. The levator ani muscles also help.

Q18. What is vault prolapse?

> Prolapse of the top of the vagina after hysterectomy.

INVESTIGATIONS

Q19. Is prolapse diagnosed clinically?

> Yes, mainly by examination.

Q20. What urinary tests are done?

> Urine dipstick and culture, and post-void residual volume by bladder scan.

Q21. When is urodynamic testing done?

> Before surgery if there are mixed urinary symptoms or voiding problems, or if continence surgery is considered.

Q22. Why do you check for occult stress incontinence?

> Correcting the prolapse may unmask stress incontinence; reducing the prolapse during a cough test can show it.

Q23. When is renal ultrasound done?

> In severe procidentia, to look for hydronephrosis from ureteric kinking.

Q24. Why is a decubitus ulcer biopsied?

> If it does not heal with oestrogen and reduction, to exclude cancer.

Q25. What imaging may help complex cases?

> Pelvic floor ultrasound or MRI defecography.

Q26. Which questionnaires are used?

> Validated symptom and quality-of-life questionnaires such as PFDI-20.

Q27. Why is fitness for surgery assessed?

> Many women are older with medical problems.

Q28. Why is a smear history checked?

> To make sure cervical screening is up to date before uterine-sparing surgery.

MANAGEMENT

Q29. How is mild asymptomatic prolapse managed?

> Lifestyle advice: weight loss, treat cough and constipation, avoid heavy lifting, and pelvic floor muscle training.

Q30. What is the first-line treatment for symptomatic prolapse?

> Supervised pelvic floor muscle training for at least 16 weeks, with vaginal oestrogen if there is atrophy.

Q31. What is a vaginal pessary?

> A device (ring, shelf, Gellhorn) placed in the vagina to support the prolapse.

Q32. Who is a pessary suitable for?

> Women unfit for or not wanting surgery, those who have not completed their family, and as a trial before surgery.

Q33. How often is a pessary changed?

> Usually every 4–6 months, with checks for ulceration.

Q34. What are the complications of pessaries?

> Discharge, ulceration, bleeding, expulsion and, rarely, fistula if neglected.

Q35. What is the standard operation for uterine prolapse?

> Vaginal hysterectomy with pelvic floor repair (anterior and/or posterior repair) and vault support.

Q36. What uterus-preserving operations are available?

> Sacrohysteropexy, sacrospinous hysteropexy and the Manchester repair (cervical amputation).

Q37. What is anterior colporrhaphy?

> Repair of the anterior vaginal wall for a cystocele, by plicating the fascia.

Q38. What is posterior colporrhaphy?

> Repair of the posterior vaginal wall for a rectocele.

Q39. What is sacrocolpopexy?

> Fixing the vaginal vault to the sacral promontory with mesh, by abdominal or laparoscopic route, for vault prolapse.

Q40. What is sacrospinous fixation?

> A vaginal operation attaching the vault or cervix to the sacrospinous ligament.

Q41. What is colpocleisis?

> Closing the vagina; for frail women who are no longer sexually active.

Q42. Why is transvaginal mesh now restricted?

> Because of complications such as mesh exposure, pain and dyspareunia; use is limited to specialist settings.

Q43. How is a decubitus ulcer treated before surgery?

> Vaginal oestrogen and reduction with a pessary or packing until it heals.

COMPLICATIONS AND COUNSELLING

Q44. What are the complications of prolapse surgery?

> Bleeding, infection, bladder or bowel injury, urinary retention, new stress incontinence, dyspareunia and recurrence.

Q45. What is the recurrence rate after prolapse surgery?

> About 30% have recurrence, often in another compartment.

Q46. How can prolapse be prevented?

> Pelvic floor exercises in and after pregnancy, avoiding prolonged second stage, healthy weight and treating constipation and cough.

Q47. What advice is given after surgery?

> Avoid heavy lifting and straining for about 6 weeks, treat constipation, and continue pelvic floor exercises.

Q48. Why is prolapse common in Pakistan?

> High parity, births without skilled care, early return to heavy work, and limited access to treatment.

Q49. Should surgery be delayed in a woman wanting more children?

> Yes; use pelvic floor training or a pessary until her family is complete.

Q50. What is a recent advance in prolapse care?

> Laparoscopic sacrohysteropexy and sacrocolpopexy, and greater use of native tissue repairs after mesh restrictions.

CASE 3

Ovarian Mass and Ovarian Cancer

Typical exam case: A 56-year-old postmenopausal woman complains of abdominal bloating, early fullness and weight loss for 3 months. On examination there is ascites and a hard, fixed, irregular pelvic mass.

HISTORY AND EXAMINATION

Q1. What are the symptoms of ovarian cancer?

Persistent bloating, abdominal swelling, early satiety or loss of appetite, pelvic or abdominal pain, urinary urgency or frequency, weight loss and change in bowel habit.

Q2. Why is ovarian cancer often diagnosed late?

Symptoms are vague and are often mistaken for bowel problems.

Q3. What are the risk factors for ovarian cancer?

Age over 50, family history, BRCA1/BRCA2 and Lynch syndrome, nulliparity, infertility, endometriosis, early menarche, late menopause and HRT.

Q4. What factors reduce the risk?

Combined pill use, pregnancy, breastfeeding, tubal ligation and salpingectomy.

Q5. What features on examination suggest malignancy?

A hard, irregular, fixed or bilateral mass, ascites, nodules in the pouch of Douglas, an omental cake, pleural effusion and lymph nodes.

Q6. What features suggest a benign mass?

A smooth, mobile, cystic, unilateral mass in a young woman without ascites.

Q7. Why examine the breasts and lymph nodes?

Breast cancer can spread to the ovary and lymph node spread suggests advanced disease.

Q8. What is Krukenberg tumour?

A metastatic tumour to the ovary, usually from the stomach, with signet-ring cells.

Q9. Why ask about family history?

Two or more relatives with breast or ovarian cancer suggest a BRCA mutation.

Q10. What is Meigs' syndrome?

A benign ovarian fibroma with ascites and pleural effusion, which resolve after removal.

CLASSIFICATION AND PATHOLOGY

Q11. What are the main types of ovarian tumour?

Epithelial (most common), germ cell, sex cord-stromal and metastatic.

Q12. Which is the most common malignant epithelial type?

High-grade serous carcinoma.

Q13. Where is high-grade serous cancer now thought to start?

In the fimbrial end of the fallopian tube.

Q14. Which germ cell tumour is most common in young women?

Mature cystic teratoma (dermoid cyst), which is benign.

Q15. What is a dysgerminoma?

> A malignant germ cell tumour in young women, very sensitive to chemotherapy; LDH may be raised.

Q16. Which tumour markers relate to germ cell tumours?

> AFP (yolk sac tumour), hCG (choriocarcinoma) and LDH (dysgerminoma).

Q17. Which ovarian tumour produces oestrogen?

> Granulosa cell tumour; it can cause postmenopausal bleeding and endometrial hyperplasia. Inhibin is a marker.

Q18. What is a borderline ovarian tumour?

> A tumour with low malignant potential and no stromal invasion; it has an excellent prognosis.

INVESTIGATIONS

Q19. What is the first-line test in primary care?

> Serum CA-125; if 35 IU/mL or more, arrange a pelvic ultrasound.

Q20. Why is CA-125 not specific?

> It is raised in endometriosis, fibroids, PID, pregnancy, liver disease and other cancers.

Q21. What ultrasound features suggest malignancy?

> Multilocular cyst, solid areas, bilateral lesions, ascites and metastases.

Q22. What is the RMI?

> Risk of Malignancy Index = U × M × CA-125, where U is the ultrasound score and M is 3 for postmenopausal women.

Q23. What RMI score needs referral to a gynaecological oncology team?

> An RMI of 250 or more.

Q24. What is the O-RADS or IOTA model?

> Ultrasound-based systems to classify the risk of malignancy in adnexal masses.

Q25. What imaging is done to stage suspected ovarian cancer?

> CT of the chest, abdomen and pelvis.

Q26. Which markers are checked in women under 40 with a complex mass?

> AFP, hCG and LDH for germ cell tumours.

Q27. How is the diagnosis confirmed?

> By histology, from surgery or image-guided biopsy; ascitic fluid cytology may also help.

Q28. Why might CEA be checked?

> A raised CEA with CA-125 may suggest a bowel primary.

MANAGEMENT

Q29. How is ovarian cancer staged?

> Surgically, by the FIGO system (stage I confined to ovaries to stage IV distant spread).

Q30. What is the standard surgery for ovarian cancer?

> Total abdominal hysterectomy, bilateral salpingo-oophorectomy, omentectomy and removal of all visible disease (cytoreduction).

Q31. What is the aim of surgery?

> Complete removal of all visible tumour, as no residual disease gives the best survival.

Q32. What is the standard chemotherapy?

> Carboplatin and paclitaxel.

Q33. What is neoadjuvant chemotherapy?

> Chemotherapy before surgery for advanced disease, to shrink the tumour and allow interval debulking.

Q34. What maintenance treatments are used?

> PARP inhibitors (e.g. olaparib) in BRCA-mutated or HRD-positive cancers, and bevacizumab.

Q35. How is a young woman with early germ cell tumour treated?

> Fertility-sparing surgery (unilateral salpingo-oophorectomy) and chemotherapy (BEP) if needed.

Q36. How is a simple ovarian cyst under 5 cm in a postmenopausal woman managed?

> Repeat ultrasound and CA-125 in 4–6 months; discharge if it resolves or remains small with normal CA-125.

Q37. How is a dermoid cyst managed?

> Laparoscopic ovarian cystectomy, preserving ovarian tissue.

Q38. How is a functional cyst in a young woman managed?

> Most resolve; repeat ultrasound in 6–8 weeks if symptoms or larger than 5 cm.

Q39. How is ovarian torsion managed?

> Urgent laparoscopy to untwist (detort) the ovary and conserve it where possible.

Q40. What are the signs of torsion?

> Sudden severe one-sided pain, nausea and vomiting, and a tender adnexal mass.

Q41. Who should be offered genetic testing?

> All women with non-mucinous epithelial ovarian cancer, for BRCA1/2.

Q42. How can BRCA carriers reduce risk?

> Risk-reducing bilateral salpingo-oophorectomy after completing their family, usually around 35–45 years.

Q43. What is the role of the multidisciplinary team?

> To plan surgery, chemotherapy and palliative care for each patient.

COMPLICATIONS AND COUNSELLING

Q44. What are the complications of advanced ovarian cancer?

> Malignant ascites, bowel obstruction, pleural effusion, VTE and cachexia.

Q45. How is malignant bowel obstruction managed?

> Conservative care with antiemetics, steroids, octreotide, venting and palliative input; surgery in selected cases.

Q46. What is the 5-year survival of advanced ovarian cancer?

> About 30% for stage III–IV, compared with over 90% for stage I.

Q47. Is screening for ovarian cancer recommended in the general population?

> No. The UKCTOCS trial showed no reduction in deaths.

Q48. How is CA-125 used in follow-up?

> A rising CA-125 may show recurrence before symptoms.

Q49. What lifestyle advice reduces risk?

> Combined pill use and opportunistic salpingectomy at the time of other surgery.

Q50. What is a recent advance in ovarian cancer?

> PARP inhibitor maintenance therapy, which has greatly improved survival in BRCA and HRD-positive women, and opportunistic salpingectomy for prevention.

CASE 4

Carcinoma of the Cervix

Typical exam case: A 45-year-old P5 complains of postcoital and intermenstrual bleeding with offensive discharge for 4 months. She has never had a cervical smear. On speculum examination there is a friable, bleeding growth on the cervix.

HISTORY AND EXAMINATION

Q1. What are the symptoms of cervical cancer?

Postcoital bleeding, intermenstrual or postmenopausal bleeding, offensive watery or blood-stained discharge, and pelvic pain. Early disease may cause no symptoms.

Q2. What symptoms suggest advanced disease?

Back or leg pain, leg swelling, urinary or rectal symptoms, weight loss, and uraemia from ureteric blockage.

Q3. What is the main cause of cervical cancer?

Persistent infection with high-risk human papillomavirus, especially types 16 and 18.

Q4. What are the risk factors?

Early first intercourse, multiple partners, smoking, immunosuppression (HIV), high parity, long-term combined pill use and not attending screening.

Q5. Why is smear history important?

Most cervical cancers occur in women who have never been screened or are overdue.

Q6. What would you see on speculum examination?

An exophytic friable growth that bleeds on touch, an ulcer, or a barrel-shaped cervix.

Q7. What would you assess on vaginal and rectal examination?

Spread to the vagina and parametrium; rectal examination assesses parametrial and pelvic side wall involvement.

Q8. Why is a rectovaginal examination important in staging?

It assesses parametrial spread, which decides between surgery and chemoradiation.

Q9. What general signs would you look for?

Anaemia, lymphadenopathy (supraclavicular), leg oedema, and signs of renal failure.

Q10. What is the differential diagnosis of a cervical growth?

Cervical polyp, ectropion, tuberculosis, condylomata and endometrial cancer spreading to the cervix.

PATHOLOGY AND SCREENING

Q11. What are the histological types?

Squamous cell carcinoma (about 80%) and adenocarcinoma (about 15–20%).

Q12. Where do most cervical cancers arise?

In the transformation zone.

Q13. What is CIN?

Cervical intraepithelial neoplasia, a precancerous change graded CIN1–3 by how much of the epithelium is abnormal.

Q14. How does HPV cause cancer?

> The viral proteins E6 and E7 inactivate the tumour suppressors p53 and Rb.

Q15. What is the current UK cervical screening test?

> Primary HPV testing, with cytology only if HPV positive.

Q16. What are the screening intervals in England?

> Every 5 years from 25 to 64 for HPV-negative women (moved from 3-yearly for 25–49).

Q17. Why is screening not started before 25?

> Cervical changes are common and usually regress, and screening younger women leads to overtreatment.

Q18. Which vaccine prevents cervical cancer?

> HPV vaccine (e.g. Gardasil 9), given to girls and boys aged 12–13 in the UK.

INVESTIGATIONS

Q19. How is the diagnosis confirmed?

> By biopsy of the lesion (punch biopsy). A smear is not used to diagnose a visible lesion.

Q20. What is colposcopy?

> Magnified examination of the cervix with acetic acid and iodine to find and biopsy abnormal areas.

Q21. How is cervical cancer staged?

> By the FIGO 2018 system, using examination, imaging and pathology.

Q22. What imaging is used for staging?

> MRI pelvis for local spread and PET-CT or CT for nodes and distant spread.

Q23. What does stage IB mean?

> Cancer confined to the cervix, visible or deeper than 5 mm.

Q24. What does stage IIIB mean?

> Spread to the pelvic side wall or causing hydronephrosis.

Q25. Why check renal function?

> Ureteric obstruction causes hydronephrosis and kidney failure.

Q26. Why check the FBC?

> Bleeding causes anaemia, which reduces the effect of radiotherapy.

Q27. When is cystoscopy or sigmoidoscopy done?

> If bladder or rectal invasion is suspected (stage IVA).

Q28. Why is an HIV test advised?

> HIV increases cervical cancer risk and affects management.

MANAGEMENT

Q29. How is CIN2 or CIN3 treated?

> Large loop excision of the transformation zone (LLETZ).

Q30. How is microinvasive cancer (stage IA1) treated?

> Cone biopsy (fertility-sparing) or simple hysterectomy.

Q31. How is early cervical cancer (IB1–IIA) treated?

> Radical hysterectomy with pelvic lymphadenectomy, or chemoradiation.

Q32. What is a radical hysterectomy?

> Removal of the uterus, cervix, upper vagina and parametrium (Wertheim's operation).

Q33. What is radical trachelectomy?

> Removal of the cervix and parametrium preserving the uterus, for young women with small early tumours who want fertility.

Q34. How is locally advanced cervical cancer (IIB–IVA) treated?

> Chemoradiation: external beam radiotherapy with weekly cisplatin, followed by brachytherapy.

Q35. Why are the ovaries often preserved in young women with squamous cancer?

> Ovarian spread is rare in squamous cancer; preserving ovaries avoids early menopause.

Q36. What did the LACC trial show?

> Minimally invasive radical hysterectomy had worse survival than open surgery for early cervical cancer.

Q37. How is stage IVB disease treated?

> Palliative chemotherapy (cisplatin, paclitaxel, bevacizumab), immunotherapy and symptom control.

Q38. How is heavy bleeding from a cervical tumour controlled?

> Vaginal packing, tranexamic acid, transfusion and urgent radiotherapy.

Q39. How is hydronephrosis managed?

> Ureteric stenting or nephrostomy to protect kidney function.

Q40. What are the side effects of pelvic radiotherapy?

> Diarrhoea, cystitis, vaginal stenosis and dryness, early menopause, fistulas and bowel or bladder damage.

Q41. What is vaginal dilator therapy?

> Regular use of dilators after radiotherapy to prevent vaginal stenosis.

Q42. How is cervical cancer in pregnancy managed?

> By a specialist team; depends on stage and gestation, from delaying treatment until fetal maturity to surgery or chemotherapy in pregnancy.

Q43. What follow-up is done after treatment?

> Regular clinical review for 5 years, looking for recurrence and treatment side effects.

COMPLICATIONS AND COUNSELLING

Q44. What is the commonest cause of death in advanced cervical cancer?

> Renal failure from ureteric obstruction.

Q45. What fistulas can occur?

> Vesicovaginal and rectovaginal fistulas from tumour or radiotherapy.

Q46. What is the 5-year survival for early stage disease?

> Over 90% for stage I, falling to about 15–20% for stage IV.

Q47. What is the WHO cervical cancer elimination strategy?

> 90–70–90 by 2030: 90% of girls vaccinated, 70% of women screened, and 90% of women with disease treated.

Q48. Why is cervical cancer common in Pakistan?

> Limited screening programmes, low HPV vaccine coverage and late presentation.

Q49. What is see-and-treat in low-resource settings?

> Visual inspection with acetic acid (VIA) and immediate treatment with cryotherapy or thermal ablation.

Q50. What is a recent advance in cervical cancer?

> HPV self-sampling for screening and adding immunotherapy (pembrolizumab) to chemoradiation in advanced disease.

CASE 5

Postmenopausal Bleeding and Endometrial Cancer

> **Typical exam case:** A 62-year-old nulliparous woman with diabetes and a BMI of 38 presents with vaginal bleeding 10 years after her menopause.

HISTORY AND EXAMINATION

Q1. What is postmenopausal bleeding?

> Vaginal bleeding occurring 12 months or more after the last menstrual period.

Q2. Why is postmenopausal bleeding important?

> Endometrial cancer must be excluded; it is found in about 5–10% of women with PMB.

Q3. What are the causes of postmenopausal bleeding?

> Atrophic vaginitis or endometritis (most common), endometrial polyps, hyperplasia, endometrial cancer, cervical cancer, HRT and, rarely, oestrogen-secreting ovarian tumours.

Q4. What are the risk factors for endometrial cancer?

> Obesity, nulliparity, late menopause, unopposed oestrogen, tamoxifen, diabetes, PCOS and Lynch syndrome.

Q5. Why does obesity increase the risk?

> Fat tissue converts androgens to oestrogen, giving unopposed oestrogen stimulation of the endometrium.

Q6. Why ask about tamoxifen?

> It acts as an oestrogen on the endometrium and increases the risk of polyps, hyperplasia and cancer.

Q7. What drug history is relevant?

> HRT (especially oestrogen alone), tamoxifen and anticoagulants.

Q8. What family history suggests Lynch syndrome?

> Relatives with colon, endometrial or ovarian cancer, especially at young ages.

Q9. What would you look for on examination?

> Abdominal mass, atrophic vagina, cervical lesion, size and mobility of the uterus, and lymph nodes.

Q10. Why is speculum examination important?

> To find the source of bleeding and exclude cervical or vaginal causes.

PATHOLOGY

Q11. What are the types of endometrial cancer?

> Type 1 (endometrioid, oestrogen-dependent, low grade, better prognosis) and type 2 (serous, clear cell, not oestrogen-related, aggressive).

Q12. What is the precursor of endometrioid cancer?

> Atypical endometrial hyperplasia (endometrial intraepithelial neoplasia).

Q13. What is the risk of cancer with atypical hyperplasia?

> High; up to 40% have a coexisting cancer at hysterectomy.

Q14. What is the molecular classification of endometrial cancer?

> POLE-mutated, mismatch repair deficient, p53-abnormal and no specific molecular profile; it guides prognosis and treatment.

Q15. How does endometrial cancer spread?

> Direct spread into the myometrium and cervix, lymphatic spread to pelvic and para-aortic nodes, and later blood spread.

Q16. Why does endometrial cancer often have a good prognosis?

> It presents early with postmenopausal bleeding, so most are stage I at diagnosis.

Q17. What is the FIGO stage I?

> Tumour confined to the uterine body.

Q18. What does stage III mean?

> Spread to the serosa, adnexa, vagina or lymph nodes.

INVESTIGATIONS

Q19. What is the first-line investigation?

> Transvaginal ultrasound to measure endometrial thickness.

Q20. What endometrial thickness is reassuring?

> 4 mm or less (in women not on HRT) makes cancer unlikely.

Q21. What is done if the endometrium is over 4 mm?

> Endometrial biopsy (Pipelle) or hysteroscopy and biopsy.

Q22. When is hysteroscopy preferred?

> If a Pipelle fails or is non-diagnostic, if a polyp is suspected, or with ongoing bleeding.

Q23. Why is a normal endometrial thickness not reassuring with persistent bleeding?

> Some cancers, especially type 2, can occur with a thin endometrium; recurrent bleeding needs hysteroscopy.

Q24. What imaging is used to stage endometrial cancer?

> MRI pelvis for myometrial invasion and cervical spread, and CT chest, abdomen and pelvis for distant spread.

Q25. Which tumour test is done on all endometrial cancers?

> Mismatch repair (MMR) testing for Lynch syndrome.

Q26. Why is a cervical smear not used to diagnose endometrial cancer?

> It is not sensitive for endometrial disease.

Q27. What bloods are taken?

> FBC, renal function, glucose, and CA-125 in high-risk types.

Q28. Why is fitness for surgery assessed carefully?

> Many women are obese with diabetes, hypertension or heart disease.

MANAGEMENT

Q29. What is the standard treatment of early endometrial cancer?

> Total hysterectomy and bilateral salpingo-oophorectomy, usually laparoscopic.

Q30. Why is laparoscopic surgery preferred?

> Similar survival with less pain, faster recovery and fewer wound problems, especially in obese women.

Q31. What is sentinel lymph node biopsy?

> Removing the first draining nodes using a tracer dye to stage nodes with less morbidity than full lymphadenectomy.

Q32. When is adjuvant treatment given?

> Based on stage, grade and molecular group: vaginal brachytherapy, pelvic radiotherapy or chemotherapy.

Q33. How is atypical hyperplasia treated?

> Total hysterectomy with bilateral salpingo-oophorectomy; if fertility is wanted or unfit for surgery, LNG-IUS or oral progestogens with regular biopsy.

Q34. How is hyperplasia without atypia treated?

> Progestogens, preferably the LNG-IUS, for at least 6 months with follow-up biopsy; address risk factors.

Q35. How is a woman unfit for surgery treated?

> Progestogens (LNG-IUS or oral) or primary radiotherapy.

Q36. How is advanced or recurrent disease treated?

> Chemotherapy (carboplatin and paclitaxel), hormone therapy and immunotherapy for MMR-deficient tumours.

Q37. How is atrophic vaginitis treated once cancer is excluded?

> Topical vaginal oestrogen and lubricants.

Q38. How is an endometrial polyp managed?

> Hysteroscopic removal and histology.

Q39. How is postmenopausal bleeding on HRT managed?

> Assess the HRT regimen; investigate if bleeding is unexpected or persists beyond 6 months of starting.

Q40. Should tamoxifen users have routine scans?

> No; investigate only if they bleed.

Q41. How do you prevent endometrial cancer in women with Lynch syndrome?

> Risk-reducing hysterectomy and BSO after childbearing, usually around 35–40, and daily aspirin.

Q42. What is the role of weight loss?

> It lowers oestrogen levels and recurrence and improves surgical fitness and overall health.

Q43. How long is follow-up after treatment?

> Usually 5 years, with symptom-based review for vaginal bleeding.

COMPLICATIONS AND COUNSELLING

Q44. What is the 5-year survival for stage I endometrial cancer?

> Over 90%.

Q45. What are the complications of surgery in obese women?

> Wound infection, VTE, chest infection and anaesthetic problems.

Q46. What symptoms suggest recurrence?

> Vaginal bleeding, pelvic pain, leg swelling, cough and weight loss.

Q47. What is the link between PCOS and endometrial cancer?

> Anovulation causes unopposed oestrogen, raising the risk of hyperplasia and cancer.

Q48. How can women with PCOS protect their endometrium?

> Ensure at least 3–4 withdrawal bleeds a year with progestogen, the combined pill or an LNG-IUS.

Q49. What is the urgent referral pathway for PMB?

> Women over 55 with PMB are referred on a suspected cancer pathway to be seen within 2 weeks.

Q50. What is a recent advance in endometrial cancer?

> Molecular classification guiding treatment, and immunotherapy (dostarlimab, pembrolizumab) for MMR-deficient tumours.

CASE 6

Heavy Menstrual Bleeding

> **Typical exam case:** A 38-year-old P3 complains of heavy periods for one year, with flooding and clots, needing to change pads every hour. She feels tired. Her cycle is regular and pelvic examination is normal.

HISTORY AND EXAMINATION

Q1. How is heavy menstrual bleeding defined?

> Excessive menstrual blood loss that interferes with a woman's physical, social, emotional or material quality of life.

Q2. What is the normal menstrual blood loss?

> About 30–40 mL; over 80 mL was the old research definition of heavy bleeding.

Q3. What questions assess heaviness?

> Number of pads or tampons used, flooding, clots larger than a coin, double protection, and time off work or school.

Q4. What other menstrual history is needed?

> Cycle length and regularity, duration, intermenstrual or postcoital bleeding, and pain.

Q5. What do intermenstrual and postcoital bleeding suggest?

> Structural or cervical pathology (polyps, ectropion, cervical cancer, infection) that needs examination.

Q6. What medical history is relevant?

> Bleeding disorders, thyroid disease, anticoagulants, liver or kidney disease.

Q7. What suggests a bleeding disorder?

> Heavy periods since menarche, easy bruising, nosebleeds, bleeding after dental work or surgery, and family history.

Q8. What contraception history is relevant?

> Copper IUD can worsen bleeding; hormonal methods may help.

Q9. What would you look for on examination?

> Pallor, thyroid signs, abdominal mass, speculum exam of the cervix, and uterine size on bimanual exam.

Q10. What does an enlarged, boggy, tender uterus suggest?

> Adenomyosis.

CAUSES AND CLASSIFICATION

Q11. What is the PALM-COEIN classification?

> Structural causes: Polyp, Adenomyosis, Leiomyoma, Malignancy/hyperplasia. Non-structural: Coagulopathy, Ovulatory dysfunction, Endometrial, Iatrogenic, Not yet classified.

Q12. What is the most common cause in a woman with a normal pelvis?

> Endometrial dysfunction (formerly dysfunctional uterine bleeding).

Q13. When are ovulatory disorders common?

> At the extremes of reproductive life (adolescence and perimenopause) and in PCOS.

Q14. What is adenomyosis?

> Endometrial tissue within the myometrium, causing heavy painful periods and a bulky uterus.

Q15. Which coagulopathy is most common in women with heavy bleeding?

> Von Willebrand disease.

Q16. What iatrogenic causes are there?

> Anticoagulants, copper IUD and some hormonal treatments.

Q17. Why is age important in the diagnosis?

> Over 45, the risk of endometrial hyperplasia and cancer rises, so endometrial sampling is considered.

Q18. What is the main consequence of heavy bleeding?

> Iron deficiency anaemia.

INVESTIGATIONS

Q19. What blood test is done for all women?

> Full blood count.

Q20. When would you test ferritin?

> If iron deficiency is suspected, though it is not always needed if the FBC shows microcytic anaemia.

Q21. When is a coagulation screen done?

> If heavy bleeding since menarche or history suggests a bleeding disorder.

Q22. When is thyroid function tested?

> Only if there are other features of thyroid disease.

Q23. When is ultrasound done?

> If the uterus is palpable abdominally, examination is abnormal, or there are pressure symptoms or suspected fibroids or adenomyosis.

Q24. When is hysteroscopy recommended?

> If there are suspected submucosal fibroids, polyps or endometrial disease, or risk factors for hyperplasia.

Q25. Who needs an endometrial biopsy?

> Women over 45, those with persistent intermenstrual bleeding, failed treatment, or risk factors such as obesity or PCOS.

Q26. Is routine hormone testing useful?

> No; FSH and LH testing is not needed for heavy bleeding.

Q27. Why is a pregnancy test done?

> To exclude pregnancy-related bleeding.

Q28. What is the role of a cervical smear?

> Check that screening is up to date; a smear is not a test for bleeding.

MANAGEMENT

Q29. What is the first-line treatment for heavy bleeding with no structural cause?

> The levonorgestrel intrauterine system (LNG-IUS).

Q30. How does the LNG-IUS reduce bleeding?

> It releases progestogen locally, thinning the endometrium; bleeding falls by over 90% in many women.

Q31. What are non-hormonal options?

> Tranexamic acid and NSAIDs (mefenamic acid), taken during periods.

Q32. How does tranexamic acid work?

> It is an antifibrinolytic that reduces bleeding by about 50%.

Q33. How do NSAIDs work?

> They reduce prostaglandins, reducing bleeding by about 25% and relieving pain.

Q34. What are other hormonal options?

> Combined hormonal contraception, cyclical or long-acting oral progestogens, and injected progestogens.

Q35. When are GnRH agonists used?

> Short-term, mainly before surgery to stop bleeding and improve anaemia.

Q36. What is endometrial ablation?

> Destruction of the endometrium, e.g. by radiofrequency or thermal balloon, for women who have completed their family.

Q37. Who is ablation suitable for?

> Women with a normal or slightly enlarged uterus who do not want future pregnancy.

Q38. Why is contraception needed after ablation?

> Pregnancy after ablation is dangerous, with high risk of complications.

Q39. When is hysterectomy considered?

> When other treatments fail or are unsuitable and the woman wants definitive treatment with no wish for pregnancy.

Q40. How are polyps managed?

> Hysteroscopic polypectomy.

Q41. How is anaemia treated?

> Oral iron, or IV iron if severe or not tolerated.

Q42. How is acute severe bleeding managed?

> Resuscitation, tranexamic acid, high-dose oral progestogens or the combined pill, and transfusion if needed.

Q43. How is heavy bleeding in von Willebrand disease managed?

> With a haematologist: tranexamic acid, hormonal treatments and desmopressin.

COMPLICATIONS AND COUNSELLING

Q44. What are the effects of heavy bleeding on quality of life?

> Fatigue, missed work or school, social embarrassment and anxiety.

Q45. What are the side effects of the LNG-IUS?

> Irregular spotting in the first 3–6 months, breast tenderness and acne; most women become lighter or stop bleeding.

Q46. What are the risks of hysterectomy?

> Bleeding, infection, bladder or bowel injury, VTE and early ovarian failure in some women.

Q47. What advice is given about iron-rich diet?

> Eat red meat, green leafy vegetables, lentils and fortified cereals, and take iron with vitamin C.

Q48. Why involve the woman in choosing treatment?

> Choice depends on her fertility wishes, side effects and preferences.

Q49. What is the outlook for most women?

> Most respond to the LNG-IUS or medical treatment, and surgery is needed in only a minority.

Q50. What is a recent advance in heavy bleeding treatment?

> Oral GnRH antagonists with add-back therapy for fibroid-related bleeding, and second-generation endometrial ablation devices.

CASE 7

Infertility

> **Typical exam case:** A 30-year-old woman and her 34-year-old husband have been trying to conceive for 2 years. She has regular cycles. They have regular intercourse and no children.

HISTORY AND EXAMINATION

Q1. How is infertility defined?

> Failure to conceive after 1 year of regular unprotected intercourse.

Q2. What is the difference between primary and secondary infertility?

> Primary: never conceived before. Secondary: has conceived before, whatever the outcome.

Q3. When should investigation start earlier than 1 year?

> Woman aged 36 or over, or a known cause such as amenorrhoea, previous pelvic surgery or PID, or male problems.

Q4. What history would you take from the woman?

> Age, menstrual history, previous pregnancies, contraception, PID or STIs, pelvic surgery, smear history, medical problems, drugs, smoking, alcohol and BMI.

Q5. What history would you take from the man?

> Previous children, testicular problems (mumps, undescended testis, surgery), STIs, drugs (anabolic steroids), smoking, alcohol and occupation.

Q6. What symptoms suggest PCOS?

> Irregular periods, hirsutism, acne and weight gain.

Q7. What symptoms suggest endometriosis?

> Painful periods, painful intercourse and chronic pelvic pain.

Q8. What would you look for on examination of the woman?

> BMI, signs of hyperandrogenism, galactorrhoea, thyroid enlargement, pelvic masses, tenderness and fixed uterus.

Q9. Why ask about the frequency of intercourse?

> Intercourse every 2–3 days gives the best chance of pregnancy.

Q10. What lifestyle factors affect fertility?

> Smoking, alcohol, high or low BMI and recreational drugs.

CAUSES

Q11. What are the main causes of infertility?

> Male factor (about 30%), ovulatory disorders (about 25%), tubal damage (about 20%), endometriosis, uterine problems and unexplained (about 25%).

Q12. What is the commonest cause of anovulation?

> Polycystic ovary syndrome.

Q13. What are other causes of anovulation?

> Hyperprolactinaemia, thyroid disease, low weight or excess exercise (hypothalamic), and premature ovarian insufficiency.

Q14. What causes tubal infertility?

> Pelvic infection (chlamydia, gonorrhoea, tuberculosis), previous ectopic, pelvic surgery and endometriosis.

Q15. Why is genital tuberculosis important in Pakistan?

> It is a common cause of tubal and endometrial damage and infertility.

Q16. What uterine factors cause infertility?

> Submucosal fibroids, polyps, uterine anomalies and intrauterine adhesions (Asherman syndrome).

Q17. What are causes of male infertility?

> Low sperm count or motility, varicocele, obstruction, testicular failure, hormonal problems and genetic causes (Klinefelter, Y deletions).

Q18. What is unexplained infertility?

> Infertility with normal ovulation, open tubes and normal semen analysis.

INVESTIGATIONS

Q19. How is ovulation confirmed in a woman with regular cycles?

> Mid-luteal (day 21 of a 28-day cycle) serum progesterone; above 30 nmol/L suggests ovulation.

Q20. What tests are done if cycles are irregular?

> FSH, LH, prolactin, thyroid function and testosterone.

Q21. How is ovarian reserve measured?

> Anti-Müllerian hormone (AMH) and antral follicle count on ultrasound.

Q22. What is the first test for the man?

> Semen analysis, repeated after 3 months if abnormal.

Q23. What are the WHO normal values for semen?

> Volume 1.4 mL or more, concentration 16 million/mL or more, total motility 42% or more, and normal forms 4% or more (WHO 2021).

Q24. How is tubal patency tested in women without risk factors?

> Hysterosalpingography (HSG) or hysterosalpingo-contrast sonography (HyCoSy).

Q25. How are tubes tested in women with risk factors such as PID or endometriosis?

> Laparoscopy and dye test, which can also treat endometriosis or adhesions.

Q26. Why screen for chlamydia before uterine instrumentation?

> To prevent reactivating pelvic infection; give antibiotic prophylaxis if not screened.

Q27. What is the role of pelvic ultrasound?

> To detect fibroids, polyps, ovarian cysts, PCOS and hydrosalpinx.

Q28. Which tests check readiness for pregnancy?

> Rubella immunity and cervical screening status.

MANAGEMENT

Q29. What general advice is given?

> Healthy weight, stop smoking, limit alcohol, folic acid, and intercourse every 2–3 days.

Q30. What is the first-line treatment for anovulation in PCOS?

> Letrozole (or clomifene), with weight loss if BMI is high.

Q31. What is the main risk of ovulation induction?

> Multiple pregnancy; follicle tracking by ultrasound is advised.

Q32. What are second-line options for PCOS?

> Gonadotrophins or laparoscopic ovarian drilling, then IVF.

Q33. How is hyperprolactinaemia treated?

> With a dopamine agonist such as cabergoline or bromocriptine.

Q34. How is hypogonadotrophic hypogonadism treated?

> Pulsatile GnRH or gonadotrophins with LH activity.

Q35. How is tubal disease treated?

> Tubal surgery for mild disease; IVF for severe damage.

Q36. Why are hydrosalpinges removed before IVF?

> They reduce IVF success; salpingectomy or tubal occlusion improves pregnancy rates.

Q37. What is intrauterine insemination (IUI)?

> Placing washed sperm in the uterus around ovulation; used for some couples, such as same-sex couples or sexual problems.

Q38. When is IVF recommended for unexplained infertility?

> After 2 years of trying (NICE), including up to 1 year before investigation.

Q39. What is ICSI?

> Intracytoplasmic sperm injection: one sperm is injected into each egg; used for severe male factor.

Q40. What is surgical sperm retrieval?

> Collecting sperm from the testis or epididymis (TESE, PESA) in azoospermia.

Q41. How does age affect IVF success?

> Success falls sharply after 35, and especially after 40.

Q42. How many embryos are usually transferred?

> One, especially in younger women, to avoid multiple pregnancy.

Q43. What is ovarian hyperstimulation syndrome (OHSS)?

> An exaggerated response to fertility drugs, causing enlarged ovaries, fluid shifts, ascites and risk of thrombosis.

COMPLICATIONS AND COUNSELLING

Q44. What are the risks of IVF?

> OHSS, multiple pregnancy, ectopic pregnancy, procedure complications and emotional stress.

Q45. How is OHSS prevented?

> Antagonist protocols, GnRH agonist trigger, freezing all embryos and careful monitoring.

Q46. What psychological support is needed?

> Infertility causes stress and relationship strain; counselling should be offered.

Q47. What is the chance of natural conception in the first year?

> About 80–85% of couples conceive within a year, and over 90% within 2 years.

Q48. What is fertility preservation?

> Freezing eggs, embryos or ovarian tissue before cancer treatment or for social reasons.

Q49. Why is cultural sensitivity important in Pakistan?

> Infertility is often blamed on the woman; male testing must be done and the couple counselled together.

Q50. What is a recent advance in fertility treatment?

> Letrozole as first-line ovulation induction in PCOS, and vitrification of eggs and embryos with frozen embryo transfer.

CASE 8

Polycystic Ovary Syndrome

> **Typical exam case:** A 24-year-old unmarried woman complains of irregular periods every 2–4 months since menarche, excess facial hair and acne. Her BMI is 32.

HISTORY AND EXAMINATION

Q1. What is PCOS?

A common endocrine condition with ovulatory dysfunction, hyperandrogenism and polycystic ovaries.

Q2. How common is PCOS?

It affects about 8–13% of women of reproductive age; more common in South Asian women.

Q3. What are the typical symptoms?

Irregular or absent periods, hirsutism, acne, hair thinning, weight gain and subfertility.

Q4. What features of hyperandrogenism are seen?

Hirsutism, acne and male-pattern hair loss.

Q5. What is the Ferriman-Gallwey score?

A score of hair growth in 9 body areas to grade hirsutism.

Q6. What is acanthosis nigricans?

Dark, velvety skin in the neck and armpits; a sign of insulin resistance.

Q7. What features suggest an androgen-secreting tumour instead of PCOS?

Rapid onset, virilisation (clitoromegaly, deep voice, male-pattern balding) and very high testosterone.

Q8. What would you ask about family history?

PCOS, type 2 diabetes and heart disease.

Q9. What psychological effects should be asked about?

Depression, anxiety, eating disorders and poor body image.

Q10. Why ask about snoring and daytime sleepiness?

PCOS with obesity increases the risk of obstructive sleep apnoea.

DIAGNOSIS

Q11. What are the Rotterdam criteria for PCOS?

Two of three: oligo- or anovulation, clinical or biochemical hyperandrogenism, and polycystic ovaries on ultrasound, after excluding other causes.

Q12. What are polycystic ovaries on ultrasound?

20 or more follicles per ovary, or an ovarian volume of 10 mL or more (2023 international guideline).

Q13. Why is ultrasound not used for diagnosis within 8 years of menarche?

Multi-follicular ovaries are normal in adolescents.

Q14. What is the role of AMH in PCOS?

Raised AMH can be used instead of ultrasound in adults (2023 guideline).

Q15. What is the underlying problem in PCOS?

Insulin resistance and high LH drive ovarian androgen production, which disrupts follicle development.

Q16. Which conditions must be excluded?

> Thyroid disease, hyperprolactinaemia, non-classical congenital adrenal hyperplasia, Cushing syndrome and androgen-secreting tumours.

Q17. What is non-classical congenital adrenal hyperplasia?

> Partial 21-hydroxylase deficiency causing high androgens; diagnosed by raised 17-hydroxyprogesterone.

Q18. Why is diagnosis important even if symptoms are mild?

> PCOS has long-term risks of diabetes, heart disease and endometrial cancer.

INVESTIGATIONS

Q19. Which hormones are measured?

> Total testosterone (or free androgen index), SHBG, LH, FSH, prolactin, TSH and 17-hydroxyprogesterone.

Q20. What happens to SHBG in PCOS?

> It falls, raising free testosterone.

Q21. What testosterone level suggests a tumour?

> A very high level (e.g. above 5 nmol/L) or rapid rise needs imaging for a tumour.

Q22. Which metabolic tests are done?

> A 75 g OGTT or HbA1c, and a lipid profile.

Q23. How often should glucose be checked?

> Every 1–3 years depending on risk factors.

Q24. Why check BP?

> PCOS raises the risk of hypertension and heart disease.

Q25. When is an endometrial assessment needed?

> If there is prolonged amenorrhoea, abnormal bleeding or thick endometrium, to exclude hyperplasia.

Q26. What is the LH:FSH ratio in PCOS?

> Often raised (above 2:1), but it is not needed for diagnosis.

Q27. Why is a pregnancy test done in amenorrhoea?

> To exclude pregnancy before other tests.

Q28. Which tools screen for depression and anxiety?

> Validated screening questionnaires such as PHQ-9 and GAD-7.

MANAGEMENT

Q29. What is the first-line treatment for overweight women with PCOS?

> Lifestyle changes: diet, exercise and weight loss. A 5–10% weight loss can restore ovulation.

Q30. How are irregular periods treated if pregnancy is not wanted?

> Combined oral contraceptive pill, or cyclical progestogens to give withdrawal bleeds.

Q31. Why must women with PCOS have regular bleeds?

> To protect the endometrium from hyperplasia and cancer caused by unopposed oestrogen.

Q32. How many withdrawal bleeds a year are advised at minimum?

> At least 3–4 a year.

Q33. How is hirsutism treated?

> Cosmetic methods (laser, shaving, waxing), the combined pill, eflornithine cream, and anti-androgens such as spironolactone with contraception.

Q34. Why must anti-androgens be used with contraception?

> They can feminise a male fetus.

Q35. How is acne treated?

> Topical treatments, the combined pill, and antibiotics or isotretinoin under a dermatologist.

Q36. What is the role of metformin?

> It improves insulin resistance, weight and cycle regularity, and is added for metabolic benefit.

Q37. What is first-line treatment for infertility in PCOS?

> Letrozole for ovulation induction.

Q38. What is the second-line treatment for infertility?

> Gonadotrophins or laparoscopic ovarian drilling.

Q39. What is the third-line treatment?

> IVF.

Q40. What is the main risk of gonadotrophins in PCOS?

> Ovarian hyperstimulation syndrome and multiple pregnancy.

Q41. What is the role of anti-obesity drugs?

> GLP-1 agonists may be used for weight loss in women with obesity who are not trying to conceive.

Q42. When is bariatric surgery considered?

> In women with severe obesity who have not responded to other measures.

Q43. What is the role of inositol?

> It may modestly help metabolic features; evidence is limited.

COMPLICATIONS AND COUNSELLING

Q44. What are the long-term risks of PCOS?

> Type 2 diabetes, gestational diabetes, dyslipidaemia, cardiovascular disease, endometrial cancer, sleep apnoea and depression.

Q45. What pregnancy risks are higher in PCOS?

> Miscarriage, gestational diabetes, pre-eclampsia and preterm birth.

Q46. When should GDM screening be done in PCOS?

> Early pregnancy testing in high-risk women, and OGTT at 24–28 weeks.

Q47. Can women with PCOS conceive naturally?

> Many can, especially with weight loss and occasional ovulation; treatment greatly improves chances.

Q48. Is PCOS curable?

> No, but symptoms and risks can be well controlled.

Q49. Why is counselling for an unmarried woman important in Pakistan?

> Concerns about future fertility and stigma are common; she should know that most women with PCOS can have children.

Q50. What is a recent advance in PCOS care?

> The 2023 international evidence-based guideline (AMH for diagnosis, letrozole first line, emphasis on mental health) and GLP-1 agonists for weight.

CASE 9

Ectopic Pregnancy

> **Typical exam case:** A 28-year-old woman with 7 weeks of amenorrhoea presents with left iliac fossa pain and light vaginal spotting. She had pelvic inflammatory disease 3 years ago. Her urine pregnancy test is positive.

HISTORY AND EXAMINATION

Q1. What is an ectopic pregnancy?

> A pregnancy implanted outside the uterine cavity, most often in the fallopian tube.

Q2. Where do most ectopic pregnancies occur?

> In the ampulla of the fallopian tube.

Q3. What are the classic symptoms?

> Amenorrhoea, abdominal or pelvic pain and vaginal bleeding; some women have no symptoms.

Q4. What symptoms suggest rupture?

> Sudden severe pain, shoulder tip pain (blood irritating the diaphragm), fainting, collapse and pain on opening the bowels.

Q5. What are the risk factors?

> Previous ectopic, PID, tubal surgery or sterilisation, IVF, IUD in place at conception, smoking and age over 35.

Q6. Why is shoulder tip pain important?

> It suggests intra-abdominal blood irritating the diaphragm.

Q7. What would you look for on examination?

> Pulse, BP, pallor, abdominal tenderness, guarding, signs of peritonism and adnexal tenderness.

Q8. What is cervical excitation?

> Pain on moving the cervix during vaginal examination; it suggests ectopic pregnancy or PID.

Q9. Why must pelvic examination be gentle?

> Rough examination can rupture an ectopic pregnancy.

Q10. Why should every woman of reproductive age with abdominal pain have a pregnancy test?

> Ectopic pregnancy can present in many ways and must not be missed.

DIAGNOSIS

Q11. What is the main investigation?

> Transvaginal ultrasound.

Q12. What is a pregnancy of unknown location?

> A positive pregnancy test with no pregnancy seen inside or outside the uterus on transvaginal scan.

Q13. What ultrasound findings suggest a tubal ectopic?

> An adnexal mass separate from the ovary (blob or bagel sign), with or without a yolk sac or embryo, and free fluid.

Q14. What is the discriminatory zone?

> The hCG level (around 1500–2000 IU/L) above which an intrauterine pregnancy should normally be seen on transvaginal scan.

Q15. How are serial hCG levels used?

> In a pregnancy of unknown location, hCG is repeated after 48 hours: a rise of over 63% suggests a viable intrauterine pregnancy, a fall of over 50% suggests failing pregnancy, and anything in between suggests possible ectopic.

Q16. What is a heterotopic pregnancy?

> An intrauterine and an ectopic pregnancy at the same time; more common after IVF.

Q17. What are the rare sites of ectopic pregnancy?

> Cervix, caesarean scar, interstitial part of the tube (cornual), ovary and abdomen.

Q18. What is the differential diagnosis?

> Miscarriage, corpus luteum cyst, ovarian torsion, appendicitis, PID and urinary tract infection.

INVESTIGATIONS

Q19. What blood tests are done?

> Serum hCG, FBC, group and save (cross-match if unstable), and renal and liver function before methotrexate.

Q20. Why check blood group?

> RhD-negative women having surgery for ectopic pregnancy need anti-D (250 IU).

Q21. What is the role of serum progesterone?

> A low level suggests a failing pregnancy; it is used in some units for PUL assessment.

Q22. Why are serum progesterone and hCG combined in models?

> Models such as M6 help classify pregnancies of unknown location by risk.

Q23. What does free fluid in the pouch of Douglas suggest?

> Blood from a ruptured or bleeding ectopic.

Q24. When is diagnostic laparoscopy used?

> When the diagnosis is uncertain and the woman has symptoms or signs of bleeding.

Q25. Why test hCG before methotrexate?

> To check eligibility and as a baseline for follow-up.

Q26. What is the role of FAST scan in A&E?

> To detect free fluid in the abdomen in an unstable patient.

Q27. What should be done with tissue removed at surgery?

> Send for histology to confirm the diagnosis and exclude molar pregnancy.

Q28. When is MRI useful?

> For rare sites such as caesarean scar or abdominal pregnancy.

MANAGEMENT

Q29. How is a haemodynamically unstable woman managed?

> Resuscitate and take her straight to theatre for laparoscopy or laparotomy.

Q30. What are the three management options for ectopic pregnancy?

> Expectant, medical (methotrexate) and surgical.

Q31. Who is suitable for expectant management?

> Clinically stable, minimal pain, unruptured ectopic under 35 mm with no heartbeat, and hCG below 1000 IU/L and falling.

Q32. Who is suitable for methotrexate?

> Stable, no significant pain, unruptured ectopic under 35 mm, no visible heartbeat, hCG below 1500 IU/L (can be offered up to 5000), and able to attend follow-up.

Q33. How does methotrexate work?

> It is a folate antagonist that stops dividing trophoblast cells.

Q34. What is the dose of methotrexate?

> 50 mg/m² by IM injection; hCG is checked on days 4 and 7, and a second dose given if the fall is under 15%.

Q35. What are the side effects of methotrexate?

> Abdominal pain, nausea, mouth ulcers, abnormal liver tests and, rarely, bone marrow suppression.

Q36. What advice is given after methotrexate?

> Avoid alcohol, folic acid, NSAIDs and sex until hCG is negative; avoid pregnancy for 3 months.

Q37. When is surgery the first choice?

> Severe pain, ectopic 35 mm or more, visible heartbeat, hCG over 5000 IU/L, rupture or unstable patient.

Q38. What is the preferred surgical approach?

> Laparoscopy.

Q39. What is the preferred operation if the other tube is healthy?

> Salpingectomy (removal of the tube).

Q40. When is salpingotomy considered?

> If the other tube is damaged or absent and she wants future fertility.

Q41. What follow-up is needed after salpingotomy?

> Serial hCG, as about 1 in 5 need further treatment for persistent trophoblast.

Q42. What is given to RhD-negative women having surgery?

> Anti-D immunoglobulin 250 IU.

Q43. How is a caesarean scar ectopic managed?

> By specialists, often with surgical removal or local treatment, because of high bleeding risk.

COMPLICATIONS AND COUNSELLING

Q44. What are the complications of ectopic pregnancy?

> Rupture, haemorrhage, shock, need for transfusion, loss of the tube and death.

Q45. What is the recurrence risk of ectopic pregnancy?

> About 10–15% after one ectopic.

Q46. What advice is given for the next pregnancy?

> Early transvaginal scan at 6–7 weeks to confirm the location.

Q47. What is the chance of future intrauterine pregnancy after ectopic?

> Good; over 60–70% have a later intrauterine pregnancy.

Q48. Why is psychological support important?

> Pregnancy loss can cause grief, anxiety and post-traumatic stress.

Q49. Why is ectopic pregnancy still a cause of maternal death?

> Late diagnosis, missed symptoms and delayed surgery.

Q50. What is a recent advance in ectopic pregnancy care?

> Early pregnancy assessment units, high-resolution transvaginal scanning and the NICE 2019 update allowing methotrexate up to hCG 5000 IU/L.

CASE 10

Miscarriage and Recurrent Miscarriage

> **Typical exam case:** A 33-year-old woman at 9 weeks presents with vaginal bleeding and cramping. She has had three previous miscarriages before 12 weeks.

HISTORY AND EXAMINATION

Q1. What is miscarriage?

> Loss of a pregnancy before 24 weeks.

Q2. How common is miscarriage?

> About 15–20% of recognised pregnancies, mostly before 12 weeks.

Q3. What is recurrent miscarriage?

> Three or more consecutive pregnancy losses (UK RCOG); some guidelines use two.

Q4. What history is important?

> Amount of bleeding, clots or tissue passed, pain, fainting, previous losses and their timing, and medical history.

Q5. What are the types of miscarriage clinically?

> Threatened, inevitable, incomplete, complete, missed (early fetal demise) and septic.

Q6. What is a threatened miscarriage?

> Bleeding with a closed cervix and a viable pregnancy.

Q7. What is an inevitable miscarriage?

> Bleeding with an open cervix; the pregnancy cannot continue.

Q8. What is a missed miscarriage?

> A non-viable pregnancy retained in the uterus, often with no symptoms.

Q9. What are signs of septic miscarriage?

> Fever, offensive discharge, uterine tenderness, tachycardia and shock.

Q10. What is cervical shock?

> Severe bradycardia and hypotension due to products of conception in the cervical canal; removing them with sponge forceps relieves it.

CAUSES

Q11. What is the most common cause of first-trimester miscarriage?

> Chromosomal abnormalities in the embryo, especially trisomies.

Q12. What are the risk factors for miscarriage?

> Older maternal age, previous miscarriage, smoking, alcohol, obesity and uncontrolled diabetes or thyroid disease.

Q13. What are the causes of recurrent miscarriage?

> Antiphospholipid syndrome, parental chromosomal rearrangements, uterine anomalies, endocrine problems and age; many are unexplained.

Q14. Which acquired thrombophilia is linked to recurrent miscarriage?

> Antiphospholipid syndrome.

Q15. What is cervical weakness?

> Painless cervical dilatation causing second-trimester loss.

Q16. Which uterine anomaly is most linked to miscarriage?

> Septate uterus.

Q17. How does age affect miscarriage risk?

> Risk rises steeply after 35, reaching over 50% at 45.

Q18. Do stress, exercise or sex cause miscarriage?

> No. Women should be reassured that these do not cause miscarriage.

INVESTIGATIONS

Q19. What is the key investigation in early pregnancy bleeding?

> Transvaginal ultrasound.

Q20. When is a pregnancy diagnosed as non-viable on ultrasound?

> Crown–rump length 7 mm or more with no heartbeat, or mean sac diameter 25 mm or more with no embryo, confirmed by a second scan or second opinion.

Q21. What is done if the CRL is under 7 mm with no heartbeat?

> Repeat the scan in 7–14 days.

Q22. When are serial hCG levels used?

> In pregnancy of unknown location.

Q23. Which tests are done for recurrent miscarriage?

> Antiphospholipid antibodies (lupus anticoagulant, anticardiolipin, anti-beta2 glycoprotein), pelvic ultrasound (3D) for uterine anomalies, and cytogenetics of pregnancy tissue.

Q24. How is antiphospholipid syndrome confirmed?

> Two positive tests at least 12 weeks apart.

Q25. When is parental karyotyping done?

> If pregnancy tissue shows an unbalanced structural chromosomal abnormality.

Q26. Is routine testing for inherited thrombophilia recommended?

> Not routinely for recurrent first-trimester miscarriage.

Q27. Which hormone tests may be checked?

> Thyroid function and antibodies, and HbA1c if diabetic.

Q28. Why is blood group checked?

> RhD-negative women having surgery need anti-D.

MANAGEMENT

Q29. How is threatened miscarriage managed?

> Reassurance; most continue. Vaginal progesterone is offered if she has had a previous miscarriage (PRISM trial).

Q30. What are the three options for managing miscarriage?

> Expectant, medical and surgical.

Q31. What is expectant management?

> Waiting 7–14 days for natural completion; it works in most women.

Q32. What is medical management of missed miscarriage?

> Mifepristone 200 mg orally, then misoprostol 800 micrograms vaginally 48 hours later.

Q33. What is medical management of incomplete miscarriage?

> Misoprostol 600 micrograms orally or vaginally.

Q34. What is surgical management?

> Manual vacuum aspiration under local anaesthetic or surgical management under general anaesthesia.

Q35. When is surgery recommended?

> Heavy bleeding, infection, unstable patient, or the woman's preference.

Q36. What are the risks of surgical management?

> Bleeding, infection, retained tissue, uterine perforation, cervical damage and intrauterine adhesions.

Q37. How is septic miscarriage managed?

> IV antibiotics, resuscitation and urgent evacuation of the uterus.

Q38. When is anti-D given after miscarriage?

> For surgical management, or medical management after 12 weeks, in RhD-negative women.

Q39. How is antiphospholipid syndrome treated in pregnancy?

> Low-dose aspirin and prophylactic LMWH.

Q40. How is cervical weakness treated?

> Cervical cerclage at 12–14 weeks.

Q41. How is a uterine septum treated?

> Hysteroscopic resection may be offered, though evidence of benefit is mixed.

Q42. What care is given for unexplained recurrent miscarriage?

> Supportive care in a dedicated early pregnancy clinic, with early scans; outcomes are good.

Q43. What is the role of progesterone in recurrent miscarriage?

> Vaginal progesterone is offered to women with bleeding and at least one previous miscarriage.

COMPLICATIONS AND COUNSELLING

Q44. What is the chance of success in the next pregnancy after unexplained recurrent miscarriage?

> About 60–70% have a successful pregnancy with supportive care.

Q45. What psychological effects follow miscarriage?

> Grief, anxiety, depression and post-traumatic stress; support should be offered.

Q46. When can she try again after a miscarriage?

> When she feels ready, once bleeding has stopped; no need to wait longer.

Q47. What lifestyle advice reduces miscarriage risk?

> Stop smoking, avoid alcohol, healthy weight, folic acid and control of medical conditions.

Q48. What is a pregnancy remembrance option?

> Many units offer certificates, keepsakes and sensitive disposal of pregnancy tissue.

Q49. What is the 'graded model' of miscarriage care?

> An approach proposed by the Lancet Miscarriage Matters series (2021): advice and support after the first miscarriage, basic tests after the second, and full investigation after the third.

Q50. What is a recent advance in miscarriage care?

> Mifepristone plus misoprostol for missed miscarriage (MifeMiso trial) and progesterone for threatened miscarriage with previous loss (PRISM trial).

CASE 11

Gestational Trophoblastic Disease (Molar Pregnancy)

Typical exam case: A 20-year-old primigravida at 11 weeks presents with vaginal bleeding and severe vomiting. Her uterus is the size of a 16-week pregnancy and serum hCG is 250,000 IU/L. Ultrasound shows a 'snowstorm' appearance with no fetus.

HISTORY AND EXAMINATION

Q1. What is gestational trophoblastic disease?

A group of conditions arising from abnormal placental (trophoblast) tissue, including hydatidiform mole and gestational trophoblastic neoplasia.

Q2. What are the symptoms of a molar pregnancy?

Vaginal bleeding in early pregnancy, hyperemesis, a uterus larger than dates, and passage of grape-like vesicles.

Q3. Why is vomiting severe in molar pregnancy?

Very high hCG levels.

Q4. What signs of hyperthyroidism may occur?

Tremor, tachycardia and weight loss; hCG stimulates the TSH receptor.

Q5. Why may early pre-eclampsia occur?

Molar pregnancy can cause pre-eclampsia before 20 weeks.

Q6. What are the risk factors for molar pregnancy?

Extremes of maternal age (under 20 and over 40), previous molar pregnancy and Asian ethnicity.

Q7. What might you find on abdominal examination?

A uterus larger than dates with no fetal parts or heart sounds.

Q8. What might you find on pelvic examination?

Bleeding, vesicles in the vagina and enlarged ovaries from theca lutein cysts.

Q9. What are theca lutein cysts?

Large ovarian cysts caused by high hCG; they usually resolve after treatment.

Q10. Is a uterus larger than dates always present?

No. Many moles are now found early on ultrasound with few symptoms.

PATHOLOGY

Q11. What are the two types of hydatidiform mole?

Complete mole and partial mole.

Q12. What is the genetic make-up of a complete mole?

Diploid (46XX usually), with all chromosomes from the father, and no fetal tissue.

Q13. What is the genetic make-up of a partial mole?

Triploid (69 chromosomes): two sets from the father and one from the mother; fetal parts may be present.

Q14. Which type has a higher risk of malignant change?

Complete mole (about 15%) compared with partial mole (about 0.5–1%).

Q15. What is gestational trophoblastic neoplasia (GTN)?

> Persistent or malignant trophoblastic disease: invasive mole, choriocarcinoma, placental site trophoblastic tumour and epithelioid trophoblastic tumour.

Q16. What is choriocarcinoma?

> A highly malignant tumour of trophoblast that spreads by blood, often to the lungs; it can follow any pregnancy.

Q17. Where does choriocarcinoma commonly spread?

> Lungs, vagina, liver and brain.

Q18. How is the diagnosis confirmed?

> By histology of the evacuated tissue.

INVESTIGATIONS

Q19. What is the typical ultrasound appearance of a complete mole?

> A 'snowstorm' or 'bunch of grapes' pattern with no fetus.

Q20. Why is ultrasound less reliable for partial moles?

> Changes may be subtle, with a fetus present; histology is needed.

Q21. What blood tests are done?

> Serum hCG, FBC, group and save, thyroid function, renal and liver function.

Q22. Why is a chest X-ray done?

> To look for lung metastases if GTN is suspected or hCG remains high.

Q23. How is follow-up done after evacuation?

> Serial hCG measurements, registered with a specialist trophoblastic centre.

Q24. How long is hCG follow-up after a complete mole?

> If hCG returns to normal within 56 days, follow-up continues for 6 months from evacuation; otherwise for 6 months after normalisation.

Q25. How long is follow-up after a partial mole?

> Until hCG is normal on two samples 4 weeks apart.

Q26. What hCG pattern suggests GTN?

> A plateau or rise in hCG after evacuation.

Q27. Why is tissue from all failed pregnancies sent for histology in some cases?

> To detect unsuspected molar pregnancies.

Q28. What test is done if choriocarcinoma is suspected after any pregnancy?

> Serum hCG, as abnormal bleeding or unusual metastases after any pregnancy can be choriocarcinoma.

MANAGEMENT

Q29. What is the treatment of a molar pregnancy?

> Suction evacuation of the uterus, preferably under ultrasound guidance.

Q30. Why is medical evacuation avoided for complete moles?

> It may increase the risk of trophoblast spread and GTN.

Q31. Why should oxytocin be avoided before evacuation if possible?

> Contractions may push trophoblast into the circulation.

Q32. Is a second evacuation routinely needed?

> No. Repeat evacuation is only for specific cases, after discussion with the specialist centre.

Q33. Is anti-D needed after evacuation of a mole?

> Yes, for partial moles in RhD-negative women; anti-D is often given in all cases.

Q34. When is GTN treated?

> When hCG plateaus or rises, stays high 6 months after evacuation, or there is choriocarcinoma or metastases.

Q35. How is GTN risk scored?

> Using the FIGO/WHO prognostic score (low risk 0–6, high risk 7 or more).

Q36. What is the treatment of low-risk GTN?

> Single-agent methotrexate with folinic acid rescue.

Q37. What is the treatment of high-risk GTN?

> Combination chemotherapy, such as EMA-CO.

Q38. What is the cure rate of GTN?

> Near 100% for low-risk disease and over 90% for high-risk disease.

Q39. What contraception is advised during hCG follow-up?

> Barrier or hormonal methods, including the combined pill, which can be started straight after evacuation. An IUD should be avoided until hCG is normal.

Q40. When can she try for another pregnancy?

> After hCG follow-up is complete; if she had chemotherapy, wait 1 year.

Q41. What is done after every future pregnancy?

> An hCG check 6–8 weeks after the end of the pregnancy.

Q42. How is hysterectomy used in GTD?

> Rarely; for older women who have completed their family or in resistant disease or bleeding.

Q43. How is hyperthyroidism from a mole managed?

> Beta-blockers before evacuation; it resolves as hCG falls.

COMPLICATIONS AND COUNSELLING

Q44. What are the complications of molar pregnancy?

> Haemorrhage, hyperemesis, pre-eclampsia, hyperthyroidism, theca lutein cysts, trophoblastic embolism and GTN.

Q45. What is the recurrence risk of molar pregnancy?

> About 1 in 80 after one mole, much higher after two.

Q46. Can a woman with GTN have children after chemotherapy?

> Yes. Fertility is usually preserved, with normal pregnancy outcomes.

Q47. Why is follow-up compliance important?

> GTN can be cured if detected early by rising hCG.

Q48. What is the role of national trophoblastic centres?

> They register patients, check hCG by post and arrange treatment (e.g. Charing Cross, Sheffield, Dundee in the UK).

Q49. What is familial recurrent hydatidiform mole?

> A rare inherited condition (NLRP7 or KHDC3L mutations) with repeated complete moles of maternal origin.

Q50. What is a recent advance in GTD?

> Immunotherapy (e.g. pembrolizumab, avelumab) for chemotherapy-resistant GTN.

CASE 12

Endometriosis

Typical exam case: A 27-year-old nulliparous woman complains of painful periods getting worse over 3 years, deep pain during intercourse and difficulty conceiving for 18 months. Pelvic examination shows a fixed, retroverted uterus with tender nodules in the posterior fornix.

HISTORY AND EXAMINATION

Q1. What is endometriosis?

Endometrial-like tissue outside the uterus, causing a chronic inflammatory reaction.

Q2. How common is endometriosis?

About 10% of women of reproductive age.

Q3. What are the typical symptoms?

Painful periods (dysmenorrhoea), deep dyspareunia, chronic pelvic pain, painful bowel opening or urination during periods, and subfertility.

Q4. What is the typical pattern of pain?

Cyclical pain starting before or during periods and getting worse over time.

Q5. Which cyclical symptoms suggest bowel or bladder involvement?

Pain on opening the bowels, rectal bleeding, or blood in the urine during periods.

Q6. What are the risk factors?

Early menarche, short cycles, heavy and long periods, nulliparity, family history and obstruction to menstrual outflow.

Q7. What signs may be found on examination?

Fixed retroverted uterus, tender nodules in the uterosacral ligaments or pouch of Douglas, adnexal masses (endometriomas) and reduced uterine mobility.

Q8. Why is examination often normal?

Superficial peritoneal disease cannot be felt.

Q9. How long is the average delay in diagnosis?

About 8 years.

Q10. Why is it important to ask about the impact on life?

Endometriosis affects work, education, relationships and mental health.

PATHOGENESIS AND CLASSIFICATION

Q11. What is Sampson's theory?

Retrograde menstruation through the tubes deposits endometrial cells in the pelvis.

Q12. What other theories explain endometriosis?

Coelomic metaplasia, lymphatic or vascular spread, and immune dysfunction.

Q13. Where is endometriosis most commonly found?

Ovaries, uterosacral ligaments, pouch of Douglas and pelvic peritoneum.

Q14. What is an endometrioma?

An ovarian cyst filled with old blood (chocolate cyst).

Q15. What is deep endometriosis?

> Disease penetrating more than 5 mm under the peritoneum, often involving bowel, bladder or uterosacral ligaments.

Q16. What is adenomyosis?

> Endometrial tissue within the myometrium; it often coexists with endometriosis.

Q17. How is endometriosis staged?

> The rASRM system (stages I–IV) and the #Enzian classification for deep disease.

Q18. Does disease stage match pain severity?

> No. Mild disease can cause severe pain and vice versa.

INVESTIGATIONS

Q19. What is the first-line imaging test?

> Transvaginal ultrasound.

Q20. What can transvaginal ultrasound detect?

> Endometriomas, deep nodules and bowel involvement in skilled hands; superficial disease is usually not seen.

Q21. What is the appearance of an endometrioma on ultrasound?

> A cyst with uniform low-level 'ground glass' echoes.

Q22. When is MRI used?

> To map deep endometriosis before surgery.

Q23. What is the role of CA-125?

> Not useful for diagnosis; it can be raised but is not specific.

Q24. What is the gold standard for diagnosis?

> Laparoscopy with histology, though imaging is now used for diagnosis in many cases.

Q25. Does a negative scan exclude endometriosis?

> No. Superficial disease can be missed; treat clinically or refer for laparoscopy if symptoms persist.

Q26. What appearances are seen at laparoscopy?

> Powder-burn black lesions, red and white lesions, peritoneal pockets, endometriomas and adhesions.

Q27. What tests are done before surgery for bowel disease?

> MRI and sometimes colonoscopy, with a colorectal surgeon involved.

Q28. What tests help in subfertility assessment?

> AMH for ovarian reserve and tubal patency testing.

MANAGEMENT

Q29. What is the first-line treatment for suspected endometriosis pain?

> Analgesia (paracetamol, NSAIDs) and hormonal treatment with the combined pill or a progestogen.

Q30. Can hormonal treatment be started without laparoscopy?

> Yes. NICE supports empirical treatment if symptoms suggest endometriosis.

Q31. Which progestogens are used?

> Desogestrel, norethisterone, dienogest, the LNG-IUS and the injection.

Q32. How do GnRH agonists help?

> They create a temporary menopause; add-back HRT is given to protect bone and reduce side effects.

Q33. When is surgery indicated?

> Failed medical treatment, endometrioma, deep disease, subfertility and uncertain diagnosis.

Q34. What is the surgical treatment of endometriosis?

> Laparoscopic excision or ablation of lesions and division of adhesions.

Q35. How are endometriomas managed surgically?

> Laparoscopic cystectomy (excision of the cyst wall), which lowers recurrence.

Q36. Why must ovarian surgery be careful in women wanting fertility?

> Cystectomy can reduce ovarian reserve; AMH should be checked first.

Q37. Does hormonal treatment improve fertility?

> No. Hormonal treatment prevents pregnancy and does not improve fertility.

Q38. Does surgery improve fertility in mild endometriosis?

> Yes, excision or ablation of peritoneal disease improves natural pregnancy rates.

Q39. What is the role of IVF?

> For moderate to severe disease, tubal damage or failure of other treatment.

Q40. Who should manage deep endometriosis?

> A specialist endometriosis centre with gynaecologists, colorectal surgeons and urologists.

Q41. What is the place of hysterectomy?

> For women who have completed their family and have severe symptoms; excision of all disease is also needed.

Q42. What is the role of pain management services?

> For chronic pain, with physiotherapy, psychological support and neuropathic agents.

Q43. What is the role of HRT after hysterectomy and BSO for endometriosis?

> Combined HRT is advised; continuous combined preparations lower the risk of reactivation.

COMPLICATIONS AND COUNSELLING

Q44. What are the complications of endometriosis?

> Chronic pain, subfertility, adhesions, bowel or ureteric obstruction and endometrioma rupture.

Q45. What is the cancer risk?

> A small increase in clear cell and endometrioid ovarian cancer.

Q46. What is the recurrence rate after surgery?

> Up to 20–50% within 5 years; hormonal treatment after surgery reduces this.

Q47. Does pregnancy cure endometriosis?

> No, although symptoms may improve during pregnancy.

Q48. What lifestyle support helps?

> Exercise, heat, a healthy diet, pacing activities and support groups.

Q49. Why is early diagnosis important?

> It reduces suffering, improves quality of life and may protect fertility.

Q50. What is a recent advance in endometriosis?

> Oral GnRH antagonists with add-back therapy (relugolix combination therapy) and ultrasound-based non-invasive diagnosis.

CASE 13

Primary Amenorrhoea

> **Typical exam case:** A 17-year-old girl has never had a period. She is of normal height, has normal breast development but scanty pubic and axillary hair. On examination the vagina is short and blind-ending.

HISTORY AND EXAMINATION

Q1. How is primary amenorrhoea defined?

> No periods by age 15 with normal secondary sexual characteristics, or by age 13 with no secondary sexual development.

Q2. What is secondary amenorrhoea?

> No periods for 3–6 months in a woman who previously menstruated (or 6 months with previously irregular periods).

Q3. What history is important?

> Growth and puberty, cyclical pelvic pain, weight change, exercise, eating habits, chronic illness, drugs, headaches, visual problems, galactorrhoea and family history.

Q4. Why ask about cyclical abdominal pain?

> It suggests an outflow obstruction such as imperforate hymen or transverse vaginal septum.

Q5. What is the order of normal puberty in girls?

> Breast development (thelarche), pubic hair (pubarche), growth spurt, then menarche.

Q6. What are Tanner stages?

> A scale (1–5) of breast and pubic hair development.

Q7. What would you examine?

> Height, weight, BMI, Tanner stages, signs of Turner syndrome, hirsutism, thyroid, visual fields, and external genitalia.

Q8. What features suggest Turner syndrome?

> Short stature, webbed neck, wide carrying angle, shield chest, low hairline and absent breast development.

Q9. Why is breast development a useful clinical sign?

> It shows the presence of oestrogen.

Q10. What does absent pubic hair with normal breasts suggest?

> Androgen insensitivity syndrome.

CAUSES

Q11. How are causes of primary amenorrhoea grouped?

> Hypothalamic-pituitary (low FSH), ovarian (high FSH), outflow tract problems, and other endocrine causes.

Q12. What is the most common cause of primary amenorrhoea with absent breast development and high FSH?

> Turner syndrome (45,X).

Q13. What is Mayer-Rokitansky-Küster-Hauser (MRKH) syndrome?

> Congenital absence of the uterus and upper vagina in a 46,XX woman with normal ovaries and breasts.

Q14. What is complete androgen insensitivity syndrome (CAIS)?

> A 46,XY person with resistance to androgens: female appearance, normal breasts, little body hair, blind vagina, no uterus and testes in the abdomen or groin.

Q15. What is the most likely diagnosis in this girl?

> Complete androgen insensitivity syndrome (normal breasts, scanty pubic hair, short blind vagina).

Q16. What is Kallmann syndrome?

> Hypogonadotrophic hypogonadism with loss of smell (anosmia), due to failed migration of GnRH neurons.

Q17. What are causes of hypothalamic amenorrhoea?

> Low weight, eating disorders, excessive exercise, stress and chronic illness.

Q18. What are outflow tract causes?

> Imperforate hymen, transverse vaginal septum, and absent cervix or vagina.

INVESTIGATIONS

Q19. What is the first test?

> A pregnancy test.

Q20. What hormone tests are done?

> FSH, LH, oestradiol, prolactin, TSH and testosterone.

Q21. What does a high FSH show?

> Ovarian failure (e.g. Turner syndrome or premature ovarian insufficiency).

Q22. What does a low or normal FSH with low oestrogen suggest?

> Hypothalamic or pituitary cause.

Q23. What does a high testosterone in the male range suggest?

> Androgen insensitivity syndrome or an androgen-secreting cause.

Q24. What is the key test to tell MRKH from CAIS?

> Karyotype: 46,XX in MRKH and 46,XY in CAIS; testosterone is male-level in CAIS.

Q25. What imaging is done?

> Pelvic ultrasound to check for a uterus and ovaries; MRI if unclear.

Q26. Why is renal imaging done in MRKH?

> Renal anomalies (e.g. absent kidney) are common.

Q27. When is pituitary MRI done?

> If prolactin is high or there are headaches or visual field defects.

Q28. Why is an echocardiogram done in Turner syndrome?

> To look for coarctation of the aorta and bicuspid aortic valve.

MANAGEMENT

Q29. How is an imperforate hymen treated?

> Surgical incision (hymenotomy) to drain the trapped blood (haematocolpos).

Q30. How is CAIS managed?

> Sensitive disclosure, psychological support, gonadectomy after puberty (because of a small cancer risk), oestrogen replacement afterwards, and vaginal dilators.

Q31. Why are the gonads removed in CAIS?

> Testes in abnormal positions carry a risk of germ cell tumour, which rises after puberty.

Q32. How is vaginal length increased in MRKH or CAIS?

> First-line vaginal dilator therapy; surgery (vaginoplasty) if dilators fail.

Q33. Can a woman with MRKH have a genetic child?

> Yes, by IVF with a gestational surrogate, as her ovaries work; uterine transplant is an emerging option.

Q34. How is Turner syndrome managed?

> Growth hormone in childhood, oestrogen to induce puberty, then HRT, and lifelong screening of heart, kidneys, thyroid and hearing.

Q35. What fertility options exist in Turner syndrome?

> Egg donation; pregnancy carries high cardiac risk and needs specialist assessment.

Q36. How is hypothalamic amenorrhoea treated?

> Restoring weight, reducing exercise, managing stress, and HRT to protect bone.

Q37. How is hyperprolactinaemia treated?

> Dopamine agonists such as cabergoline.

Q38. How is Kallmann syndrome treated?

> Hormone replacement for puberty, and gonadotrophins or pulsatile GnRH for fertility.

Q39. Why is HRT important in hypo-oestrogenic women?

> To protect bone density and heart health and to develop secondary sexual features.

Q40. How should the diagnosis be explained to a young woman with CAIS?

> Honestly and sensitively, with a psychologist, in a multidisciplinary team.

Q41. How is a transverse vaginal septum treated?

> Surgical excision in a specialist centre.

Q42. What is the role of a multidisciplinary team?

> Gynaecologists, endocrinologists, psychologists and specialist nurses work together, especially for differences in sex development.

Q43. What is premature ovarian insufficiency?

> Loss of ovarian function before 40, with high FSH and low oestrogen; treated with HRT.

COMPLICATIONS AND COUNSELLING

Q44. What are the long-term risks of untreated low oestrogen?

> Osteoporosis, cardiovascular disease and poor sexual health.

Q45. What are the psychological effects?

> Distress about identity, fertility and relationships; counselling is essential.

Q46. What screening is lifelong in Turner syndrome?

> Blood pressure, aortic size, thyroid, diabetes, hearing, liver function and bone density.

Q47. How common is MRKH?

> About 1 in 4,500–5,000 women.

Q48. Can a woman with CAIS have sexual intercourse?

> Yes, after vaginal dilation if needed.

Q49. Why should puberty be assessed early?

> Delayed diagnosis can cause poor bone health and psychological harm.

Q50. What is a recent advance in amenorrhoea care?

> Uterine transplantation for absolute uterine factor infertility such as MRKH, with successful live births.

CASE 14

Urinary Incontinence and Vesicovaginal Fistula

> **Typical exam case:** A 25-year-old woman from a rural area had an obstructed labour lasting 3 days, ending in a stillbirth 6 weeks ago. Since then she has had constant leakage of urine from the vagina day and night.

HISTORY AND EXAMINATION

Q1. What is urinary incontinence?

> Any involuntary leakage of urine.

Q2. What are the main types of urinary incontinence?

> Stress, urgency, mixed and overflow incontinence, and continuous leakage from a fistula.

Q3. What is stress urinary incontinence?

> Leakage on effort, such as coughing, sneezing or lifting.

Q4. What is urgency urinary incontinence?

> Leakage with a sudden strong urge to pass urine, often part of overactive bladder.

Q5. What does continuous leakage after childbirth suggest?

> A vesicovaginal fistula.

Q6. What is a vesicovaginal fistula?

> An abnormal opening between the bladder and the vagina.

Q7. What is the main cause of VVF in low-income countries?

> Prolonged obstructed labour, causing pressure necrosis of the bladder and vagina.

Q8. What is the main cause of VVF in high-income countries?

> Pelvic surgery, especially hysterectomy, and pelvic radiotherapy.

Q9. What history would you take?

> Details of labour and delivery, timing and type of leakage, faecal leakage, leg weakness, menstruation, and social effects.

Q10. What associated injuries occur with obstetric fistula?

> Rectovaginal fistula, foot drop (nerve injury), vaginal scarring, amenorrhoea and infertility.

DIAGNOSIS AND INVESTIGATIONS

Q11. How is the fistula found on examination?

> Speculum examination in a good light, with a Sims speculum, to see the fistula and urine pooling.

Q12. What is the dye test?

> Methylene blue instilled into the bladder through a catheter; blue staining on vaginal swabs shows a VVF.

Q13. What does clear fluid on the swabs after methylene blue suggest?

> A ureterovaginal fistula.

Q14. How is a ureterovaginal fistula confirmed?

> CT urogram or IV urogram; oral phenazopyridine colours the leakage orange.

Q15. What is the role of cystoscopy?

> To locate the fistula, see its size and relation to the ureteric openings.

Q16. What tests are done for stress or urgency incontinence?

> Urine dipstick and culture, a 3-day bladder diary, post-void residual volume and pelvic examination.

Q17. When are urodynamic studies done?

> Before surgery for stress incontinence, or when the diagnosis is unclear or treatment has failed.

Q18. What does urodynamics show in overactive bladder?

> Detrusor overactivity: involuntary bladder contractions during filling.

Q19. What is a bladder diary?

> A record of fluid intake, voiding times and volumes, and leakage over at least 3 days.

Q20. Why check renal function in obstetric fistula?

> Ureteric injury or obstruction may affect the kidneys.

MANAGEMENT OF FISTULA

Q21. How is a fresh obstetric fistula managed initially?

> Continuous bladder drainage with a catheter for 2–4 weeks; small fistulas may close on their own.

Q22. What general care is needed before repair?

> Nutrition, treatment of infection and anaemia, skin care for urine dermatitis, and physiotherapy.

Q23. When is surgical repair performed?

> Usually once tissues are healthy, often after about 3 months, though early repair is possible in selected cases.

Q24. What are the principles of fistula repair?

> Good exposure, wide mobilisation, tension-free closure in layers, watertight bladder closure and good drainage afterwards.

Q25. What route is commonly used for obstetric fistula repair?

> The vaginal route.

Q26. When is an abdominal route needed?

> High or complex fistulas, ureteric involvement or the need for ureteric reimplantation.

Q27. What is a Martius flap?

> A fat pad from the labium majus placed between bladder and vagina to improve healing.

Q28. How long is the catheter kept after repair?

> About 10–14 days.

Q29. What advice is given after repair?

> Drink plenty of fluids, avoid intercourse for about 3 months, and deliver any future baby by caesarean section.

Q30. What is the success rate of first fistula repair?

> Over 85–90% in experienced centres.

MANAGEMENT OF STRESS AND URGENCY INCONTINENCE

Q31. What is first-line treatment for stress incontinence?

> Supervised pelvic floor muscle training for at least 3 months, with weight loss and lifestyle advice.

Q32. What surgical options exist for stress incontinence?

> Colposuspension, autologous fascial sling, urethral bulking injections and (in restricted settings) mid-urethral tapes.

Q33. Why are mid-urethral mesh tapes restricted in the UK?

> Because of complications such as chronic pain and mesh erosion.

Q34. What drug can be used for stress incontinence if surgery is not wanted?

> Duloxetine, though side effects are common.

Q35. What is first-line treatment for overactive bladder?

> Bladder training for at least 6 weeks, reducing caffeine and adjusting fluid intake.

Q36. What drugs are used for overactive bladder?

> Anticholinergics (e.g. solifenacin, oxybutynin) and mirabegron (a beta-3 agonist).

Q37. Why is oxybutynin avoided in frail older women?

> It can cause confusion and cognitive decline.

Q38. What options exist if drugs fail?

> Botulinum toxin injections into the bladder, sacral nerve stimulation and percutaneous tibial nerve stimulation.

Q39. What is the main side effect of botulinum toxin injection?

> Urinary retention needing self-catheterisation.

Q40. How is overflow incontinence managed?

> Treat the cause (e.g. obstruction or neuropathy) and use intermittent self-catheterisation.

COMPLICATIONS AND COUNSELLING

Q41. What are the social effects of obstetric fistula?

> Constant smell and wetness lead to divorce, isolation, stigma, depression and poverty.

Q42. How can obstetric fistula be prevented?

> Access to skilled birth attendants, use of a partograph, timely caesarean section for obstructed labour, and catheterising women after prolonged labour.

Q43. Why is a catheter placed after a prolonged obstructed labour?

> To rest the bladder and allow small pressure injuries to heal, preventing fistula.

Q44. What advice is given for the next pregnancy after a fistula repair?

> Antenatal care, delivery in hospital and elective caesarean section.

Q45. How common is obstetric fistula worldwide?

> An estimated 0.5–2 million women live with it, mainly in sub-Saharan Africa and South Asia.

Q46. Which organisations help women with fistula in Pakistan?

> Specialist fistula centres, such as Koohi Goth Women's Hospital in Karachi, provide free repair and rehabilitation.

Q47. Why does an obstructed labour cause a fistula?

> The fetal head compresses the bladder and vagina against the pubic bone for hours, cutting off blood supply; the dead tissue sloughs after 3–10 days.

Q48. What is the obstetric fistula 'field injury'?

> Wider damage from obstructed labour: vaginal scarring, urethral loss, nerve injury, pelvic bone changes and infertility, not just a hole.

Q49. What is the role of rehabilitation after repair?

> Psychological support, reintegration into family and community, and training or income-generating skills.

Q50. What is a recent advance in fistula care?

> The WHO and FIGO training curriculum for fistula surgeons, and shorter catheter duration after simple repairs (7 days) shown to be safe.