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SUG 402

GENERAL SURGERY II

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Course code: SUG 402

Course title: General Surgery II

Course credit load: 5 Units

Series 1: Solid Tumours and Neoplasms

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    - Sub Part 1.1.2: Tumour classification and nomenclature
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Introduction

Neoplasia, the abnormal and uncontrolled proliferation of cells, is one of the most important areas of surgical practice. Solid tumours account for the majority of cancer-related deaths worldwide, and the surgeon plays a central role in their diagnosis, staging, and treatment. Understanding the biological differences between benign and malignant tumours, the principles of tumour classification and staging, and the surgical principles that govern oncological resection is fundamental to the practice of General Surgery II.

This Series introduces the core concepts of surgical oncology. We begin with the biology and classification of tumours, then examine the clinical approach to a patient presenting with a tumour. We then survey specific categories of solid tumours: skin and soft tissue, bone and vascular, and lymph node disease. We close with the overarching principles of surgical oncology including curative and palliative surgery and multimodal treatment.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 1.1:** distinguish benign from malignant tumours on biological, clinical, and histological grounds, and describe the classification and staging of tumours,
- **SLO 1.2:** describe a structured approach to the history, examination, and investigation of a patient presenting with a tumour,
- **SLO 1.3:** describe the clinical features and management of common skin tumours, melanoma, and soft tissue sarcomas,
- **SLO 1.4:** describe the clinical features, investigation, and management of primary bone tumours, metastatic bone disease, and vascular tumours,
- **SLO 1.5:** describe the clinical features and management of Hodgkin's lymphoma, non-Hodgkin's lymphoma, and lymphadenopathy, and
- **SLO 1.6:** describe the principles of curative and palliative surgery and multimodal cancer treatment.

1.1 Biology and Classification of Tumours

1.1.1 Benign versus malignant tumours

A benign tumour is a neoplasm that grows slowly, is well-circumscribed, is enclosed in a fibrous capsule, does not invade adjacent tissues, and does not metastasise. Microscopically, the cells resemble the parent tissue (well-differentiated), show minimal mitotic activity, and have no nuclear atypia. Examples include lipoma, fibroma, adenoma, and papilloma. Despite being non-cancerous, benign tumours can cause harm by compression of adjacent structures (for example, a meningioma compressing the brain) or by hormonal secretion (for example, an adrenal adenoma causing Cushing's syndrome).

A malignant tumour (cancer) grows rapidly, invades adjacent tissues, lacks a true capsule, and has the capacity to metastasise (spread to distant sites via lymphatics or blood). Microscopically, malignant cells show pleomorphism (variation in cell size and shape), nuclear atypia, increased and abnormal mitotic figures, loss of normal architecture (dedifferentiation), and prominent nucleoli. The biological hallmarks of cancer include sustained proliferative signalling, evasion of growth suppressors, resistance to apoptosis, replicative immortality, angiogenesis, and invasion and metastasis.

Fill in the blank: A benign tumour is _____ (enclosed in a fibrous capsule) and does not _____. Malignant cells show _____ (variation in cell size and shape) and abnormal mitotic figures on histology.

1.1.2 Tumour classification and nomenclature

Tumours are classified by their tissue of origin. Tumours arising from epithelial tissue are called carcinomas (the most common type of cancer): adenocarcinoma (from glandular epithelium), squamous cell carcinoma (from squamous epithelium), transitional cell carcinoma (from urothelium), and undifferentiated carcinoma. Tumours arising from mesenchymal (connective) tissue are called sarcomas: osteosarcoma (bone), liposarcoma (fat), leiomyosarcoma (smooth muscle), rhabdomyosarcoma (skeletal muscle), and fibrosarcoma (fibrous tissue). Tumours of haematopoietic origin include lymphomas (lymphoid tissue) and leukaemias (bone marrow).

The suffix -oma generally denotes a benign tumour (lipoma, fibroma, adenoma); however, there are important exceptions: melanoma, hepatoma, and lymphoma are all malignant despite the -oma suffix. The suffix -carcinoma and -sarcoma always denote malignancy. Mixed tumours contain more than one cell type (for example, the pleomorphic adenoma of the parotid gland contains both epithelial and mesenchymal elements). Teratomas arise from totipotent germ cells and contain elements of all three germ layers.

Fill in the blank: Tumours arising from epithelial tissue are called _____, while those from mesenchymal tissue are called _____. The suffix -oma generally denotes a _____ tumour, though melanoma and lymphoma are important exceptions.

1.1.3 Staging and grading

Tumour grading describes the degree of differentiation of the tumour cells (how closely they resemble the parent tissue). Grade 1 (well-differentiated: cells closely resemble normal tissue: low-grade: generally better prognosis), Grade 2 (moderately differentiated), and Grade 3 (poorly differentiated: cells bear little resemblance to normal tissue: high-grade: generally worse prognosis). Grade 4 (undifferentiated or anaplastic) is sometimes used. Grading is determined by the pathologist from the histological appearance.

Tumour staging describes the anatomical extent of the disease (how far the cancer has spread). The TNM system is the international standard: T (primary tumour: T1 to T4 by size and local invasion), N (regional lymph node involvement: N0 to N3), and M (distant metastasis: M0 or M1). TNM categories combine to assign an overall stage (I to IV). Staging is the most important prognostic factor and determines treatment: Stage I and II are generally localised and potentially curable by surgery; Stage III has regional spread; Stage IV has distant metastasis and is generally incurable by surgery alone.

Fill in the blank: Tumour _____ describes the degree of differentiation, while tumour _____ describes the anatomical extent of spread. In the TNM system, M1 indicates the presence of distant _____, and the overall stage is _____ (the highest).

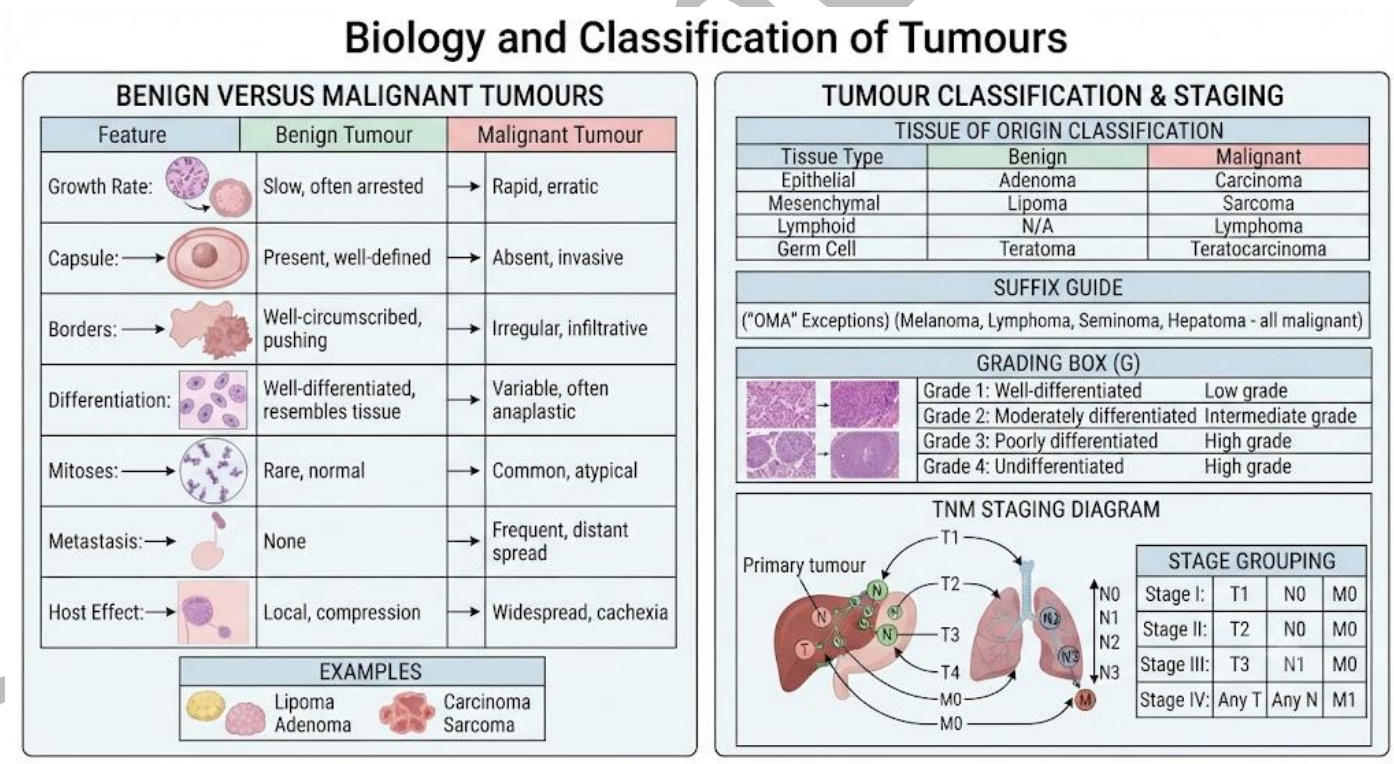

Figure 1.1: Benign versus malignant tumour features, tumour classification and nomenclature, and TNM staging

Pilot questions 1.1

11. Distinguish benign from malignant tumours using six features. (Linked to SLO 1.1)
12. Classify tumours by tissue of origin and give an example of each class. (Linked to SLO 1.1)
13. Explain the suffix rule for tumour nomenclature and give three exceptions. (Linked to SLO 1.1)
14. Describe tumour grading and explain its prognostic significance. (Linked to SLO 1.1)
15. Describe the TNM staging system and state what Stage IV indicates. (Linked to SLO 1.1)

1.2 Clinical Approach to the Patient with a Tumour

1.2.1 History taking in oncology

The oncological history begins with the presenting complaint: the site and duration of the lump or symptom, whether it has changed in size (rapid growth suggests malignancy), and whether it is painful (most malignant lumps are painless until advanced; pain in a bone tumour suggests periosteal invasion or pathological fracture). Key associated symptoms: unexplained weight loss (a cardinal feature of malignancy: above 10 percent body weight in 6 months is significant), night sweats and fever (the B symptoms of lymphoma), haemoptysis (lung cancer), haematuria (renal or bladder cancer), and change in bowel habit (colorectal cancer).

The history must also cover: the patient's age and sex (certain tumours have age and sex predilections: osteosarcoma in adolescents; breast cancer predominantly in women), family history (BRCA1/2 mutations in breast and ovarian cancer; familial adenomatous polyposis in colorectal cancer; Li-Fraumeni syndrome in sarcomas), occupational and environmental exposure (asbestos: mesothelioma; ultraviolet radiation: skin cancer; cigarette smoking: lung, bladder, and oesophageal cancer), and previous cancer treatment (radiation-induced sarcoma is a late complication of radiotherapy).

Fill in the blank: Unexplained weight loss of more than _____ percent of body weight in 6 months is a cardinal feature of malignancy. The B symptoms of lymphoma are unexplained weight loss, night _____, and fever. Asbestos exposure is associated with _____.

1.2.2 Physical examination of a tumour

The structured examination of a lump follows the sequence established in General Surgery I: site and size, shape, surface, edge (well-defined or ill-defined: ill-defined edges suggest infiltration and malignancy), consistency (stony-hard suggests malignancy or calcification), fluctuance, transillumination, pulsatility, reducibility, compressibility, temperature, fixity (to skin: tethering and peau d'orange in breast cancer; to deep structures: fixity to muscle or bone suggests malignant invasion), and regional lymph nodes (enlarged draining lymph nodes suggest metastatic spread or active infection).

The general examination assesses for signs of systemic spread and paraneoplastic manifestations: cachexia (wasting from tumour-induced metabolic effects), anaemia (from chronic disease or

occult bleeding), jaundice (from hepatic metastases or biliary obstruction by tumour), finger clubbing (bronchogenic carcinoma), lymphadenopathy (regional or generalised: Virchow's node: left supraclavicular lymphadenopathy from upper GI malignancy), hepatomegaly (metastatic disease), and ascites (peritoneal carcinomatosis).

Fill in the blank: Ill-defined edges of a lump suggest malignant _____. Tethering of skin to an underlying lump and peau d'orange suggests _____ cancer. Left supraclavicular lymphadenopathy from upper GI malignancy is called _____ node.

1.2.3 Investigations

Investigations in oncology serve three purposes: to confirm the diagnosis (tissue biopsy), to stage the disease (imaging and biochemical markers), and to assess fitness for treatment (baseline organ function). Tissue biopsy types: fine needle aspiration cytology (FNAC: provides cells for cytological analysis: rapid and minimally invasive: does not give histological architecture), core needle biopsy (Tru-Cut: provides a tissue core for histological analysis: preferred for most solid tumours), incisional biopsy (surgical removal of a portion of the tumour), and excisional biopsy (removal of the entire tumour: diagnostic and potentially therapeutic for small accessible lesions).

Staging investigations: CT scan of the chest, abdomen, and pelvis (the most important staging investigation for most solid tumours: assesses primary tumour size, regional lymph nodes, liver and lung metastases), PET-CT (fluorodeoxyglucose PET combined with CT: most sensitive for detecting metabolically active metastases), MRI (soft tissue characterisation and local staging: especially for rectal cancer, sarcomas, and brain tumours), isotope bone scan (detects skeletal metastases), and tumour markers (CEA for colorectal cancer; AFP for hepatocellular carcinoma and germ cell tumours; PSA for prostate cancer; CA-125 for ovarian cancer; CA 19-9 for pancreatic cancer).

Fill in the blank: Core needle biopsy (Tru-Cut) is preferred over FNAC for solid tumours because it provides histological _____ in addition to cytology. CT of the chest, abdomen, and pelvis is the most important _____ investigation for most solid tumours. CEA is a tumour marker for _____ cancer.

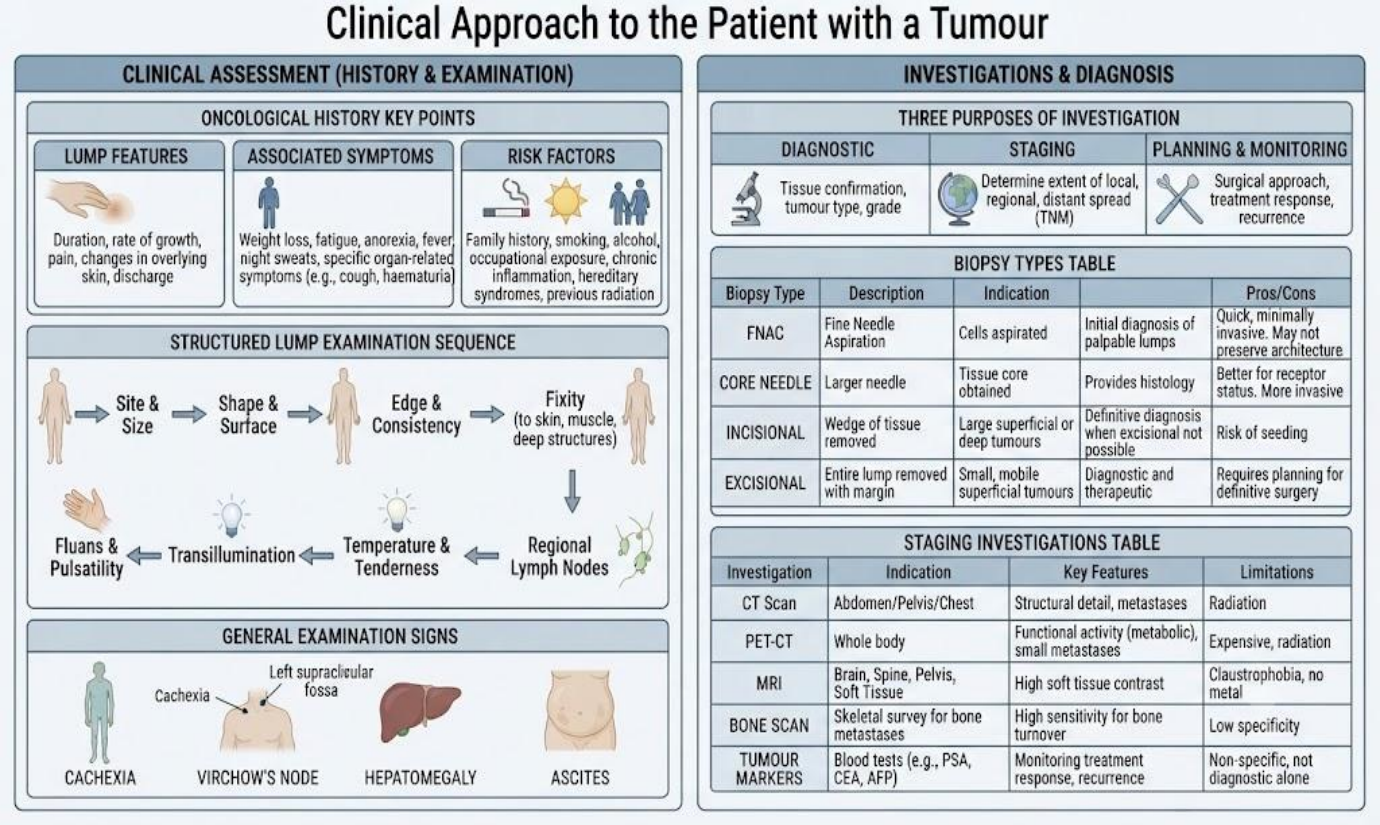

Figure 1.2: Oncological history, lump examination, general examination findings, and staging investigations

Pilot questions 1.2

1. Describe the key features of an oncological history for a patient with a lump. (Linked to SLO 1.2)
2. Describe the B symptoms of lymphoma and explain their significance. (Linked to SLO 1.2)
3. Describe the structured physical examination of a lump. (Linked to SLO 1.2)
4. Distinguish FNAC from core needle biopsy and state the clinical indication for each. (Linked to SLO 1.2)
5. State five staging investigations used in oncology and describe what each assesses. (Linked to SLO 1.2)

1.3 Skin and Soft Tissue Tumours

1.3.1 Benign skin tumours

Common benign skin tumours include: sebaceous (epidermoid) cyst (a blocked sebaceous gland forming a keratin-filled cyst with a central punctum: managed by elective excision; if infected,

drain first then excise), lipoma (a benign tumour of mature adipose tissue: soft, lobulated, mobile, with a slip sign: observe or excise if symptomatic), dermoid cyst (a cystic lesion containing skin appendages such as hair and teeth: external angular dermoid at the lateral eyebrow: excise), and pilomatrixoma (a calcified tumour of hair follicle matrix cells: firm, irregular, often in children: excise).

Keratoacanthoma is a rapidly growing, self-limiting, dome-shaped skin nodule with a central keratin-filled crater that occurs on sun-exposed skin. It closely mimics squamous cell carcinoma both clinically and histologically, making excision mandatory for definitive diagnosis. It typically involutes spontaneously in 3 to 6 months, but because it cannot be reliably distinguished from SCC without excision and histology, all suspected keratoacanthomas should be excised. Solar (actinic) keratoses are pre-malignant scaly lesions on sun-exposed skin in fair-skinned individuals: treated with topical 5-fluorouracil, cryotherapy, or photodynamic therapy.

Fill in the blank: A sebaceous cyst has a visible central _____ and is treated by elective excision. A _____ is a rapidly growing skin nodule that mimics squamous cell carcinoma and must be excised for definitive diagnosis. Solar keratoses are _____ lesions on sun-exposed skin.

1.3.2 Malignant skin tumours

Basal cell carcinoma (BCC) is the most common skin cancer, arising from basal keratinocytes of the epidermis. It occurs predominantly on the face and neck of fair-skinned individuals after prolonged UV exposure. BCC grows slowly, does not metastasise (extremely rarely), but causes significant local tissue destruction (rodent ulcer: the classic appearance is a pearly, telangiectatic nodule with a raised rolled edge and central ulceration). Treatment: surgical excision with clear margins is the standard; alternatives include radiotherapy, Mohs micrographic surgery (for recurrent or high-risk lesions), and topical imiquimod.

Melanoma is the most dangerous skin cancer, arising from melanocytes. Risk factors: fair skin, UV exposure, multiple dysplastic naevi, family history (CDKN2A mutation), and giant congenital naevus. The ABCDE criteria identify suspicious pigmented lesions: Asymmetry, Border irregularity, Colour variation (two or more colours), Diameter above 6 mm, and Evolution (change in size, shape, or colour). Breslow thickness (the depth of invasion in millimetres: measured from the granular cell layer of the epidermis to the deepest tumour cell on histology) is the most important prognostic factor. Treatment: wide local excision with margins determined by Breslow thickness (1 cm for below 1 mm; 2 cm for above 1 mm) and sentinel lymph node biopsy.

Fill in the blank: BCC is the most _____ skin cancer; it presents as a pearly nodule with a raised _____ edge and central ulceration (rodent ulcer). The most important prognostic factor in melanoma is the _____ thickness (depth of invasion in millimetres).

1.3.3 Soft tissue sarcomas

Soft tissue sarcomas (STS) are malignant tumours of mesenchymal origin (fat, muscle, fibrous tissue, nerve, and blood vessels). They are relatively rare, accounting for approximately 1 percent of adult malignancies. Common types: liposarcoma (fat: most common in the retroperitoneum and thigh), leiomyosarcoma (smooth muscle: retroperitoneum and uterus), synovial sarcoma (near joints: young adults: despite the name, does not arise from synovial tissue), malignant fibrous histiocytoma or undifferentiated pleomorphic sarcoma (UPS: most common in elderly patients), and rhabdomyosarcoma (skeletal muscle: the most common soft tissue sarcoma in children).

Clinical features of STS: a deep-seated, painless, enlarging mass (greater than 5 cm in the deep fascia should be regarded as a sarcoma until proven otherwise). Investigation: MRI is the best local staging investigation (defines the relationship of the tumour to neurovascular structures); CT chest for pulmonary metastases (the most common site of distant spread); core needle biopsy for histological diagnosis. Management: wide excision with clear margins (at least 1 to 2 cm of normal tissue: the primary determinant of local recurrence), with or without radiotherapy (pre- or postoperative: reduces local recurrence rates); amputation is rarely necessary with modern limb-sparing techniques.

Fill in the blank: A deep-seated painless mass greater than _____ cm below the deep fascia should be regarded as a sarcoma until proven otherwise. MRI is the best _____ staging investigation for soft tissue sarcomas. The most common soft tissue sarcoma in children is _____.

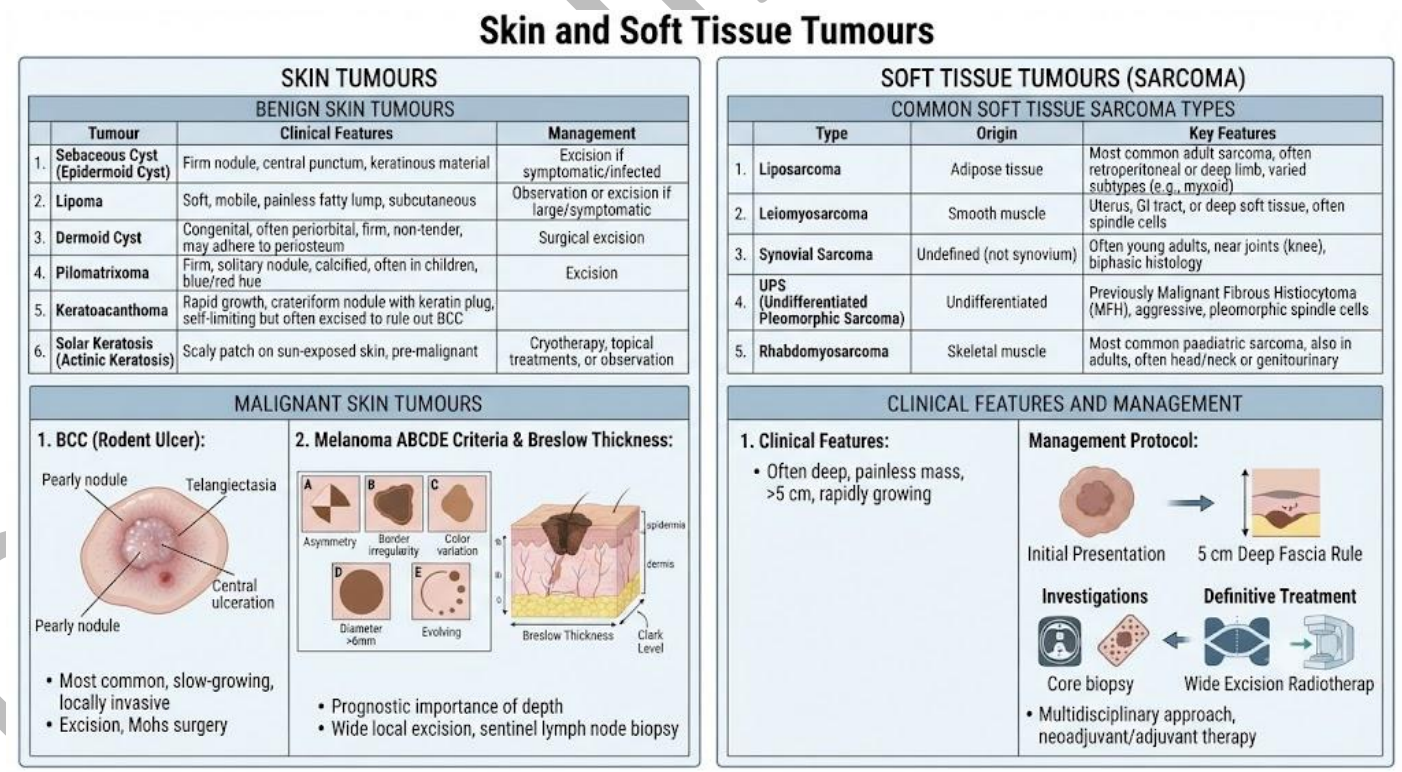

Figure 1.3: Benign skin tumours, malignant skin tumours (BCC and melanoma), and soft tissue sarcomas

Pilot questions 1.3

1. Describe the clinical features and management of basal cell carcinoma. (Linked to SLO 1.3)
2. Describe the ABCDE criteria for melanoma and state the most important prognostic factor. (Linked to SLO 1.3)
3. Describe the surgical treatment of melanoma and explain how Breslow thickness determines margin width. (Linked to SLO 1.3)
4. Describe the clinical features of soft tissue sarcoma and state the investigation of choice for local staging. (Linked to SLO 1.3)
5. Describe the surgical management of soft tissue sarcoma. (Linked to SLO 1.3)

1.4 Bone and Vascular Tumours

1.4.1 Primary bone tumours

Primary bone tumours are classified as benign or malignant and by cell of origin. Common benign bone tumours: osteochondroma (the most common benign bone tumour: a bony exostosis covered by a cartilaginous cap arising from the cortex of long bones near the metaphysis: usually asymptomatic: risk of malignant transformation in hereditary multiple exostoses), enchondroma (cartilaginous tumour within the medullary cavity: common in phalanges: risk of malignant transformation to chondrosarcoma), and giant cell tumour (osteoclastoma: epiphysis of long bones: locally aggressive: treated by curettage and bone grafting or resection).

Osteosarcoma is the most common primary malignant bone tumour, occurring predominantly in adolescents and young adults (peak age 10 to 20 years) at the metaphysis of long bones (distal femur, proximal tibia, and proximal humerus). Presents with pain and swelling around the knee (most common site). X-ray: Codman's triangle (periosteal reaction from tumour elevating the periosteum: a characteristic finding) and the sunburst pattern (tumour bone radiating outward through the cortex). Treatment: neoadjuvant chemotherapy (shrinks the tumour and treats micrometastases) followed by limb-sparing resection and bone reconstruction, then adjuvant chemotherapy. Amputation is reserved for unresectable tumours.

Fill in the blank: Osteosarcoma occurs predominantly in _____ and young adults at the _____ of long bones. The characteristic X-ray findings are Codman's _____ and the sunburst pattern. Treatment involves neoadjuvant _____ followed by limb-sparing resection.

1.4.2 Secondary (metastatic) bone disease

Metastatic bone disease is far more common than primary bone tumours. The most common primary cancers that metastasise to bone are: breast (most common overall), prostate (most common in men: typically produces sclerotic lesions), lung, kidney, and thyroid (mnemonic: BLT with a Kosher Pickle, or Breast, Lung, Thyroid, Kidney, Prostate). Most metastases are

lytic (bone destruction: breast, lung, kidney, thyroid) or mixed; prostate metastases are typically sclerotic (bone formation). Metastatic bone disease presents with bone pain, pathological fracture (fracture through diseased bone from minimal or no trauma), hypercalcaemia (from osteolysis), and spinal cord compression (from vertebral metastasis).

Investigation: isotope bone scan (the most sensitive test for detecting skeletal metastases: shows areas of increased uptake at metastatic sites), plain X-ray (lytic or sclerotic lesions), CT, and MRI (for spinal involvement and cord compression). Management: analgesia (WHO pain ladder: opioids for bone metastasis pain), bisphosphonates (zoledronic acid: inhibit osteoclast activity: reduce skeletal-related events and bone pain), radiotherapy (effective for pain control and for preventing or treating pathological fracture), orthopaedic stabilisation (intramedullary nail for impending or actual long bone fracture), and treatment of the primary tumour.

Fill in the blank: The five most common primaries that metastasise to bone are breast, lung, thyroid, kidney, and _____. Prostate metastases are typically _____ (increased bone density), unlike most other metastases which are lytic. Bisphosphonates (such as _____) inhibit osteoclast activity and reduce bone pain.

1.4.3 Vascular tumours and anomalies

Haemangiomas are benign vascular tumours and the most common tumours of infancy. Capillary haemangiomas (strawberry naevus: bright red, raised, compressible: appear in the first weeks of life, grow rapidly, then involute spontaneously by age 5 to 7 years: observe unless complicated by bleeding, ulceration, airway compromise, or Kasabach-Merritt syndrome). Cavernous haemangiomas are deeper, less bright, do not involute spontaneously, and may require treatment (laser therapy, sclerotherapy, or surgical excision). Port-wine stain (naevus flammeus) is a congenital capillary vascular malformation (not a true haemangioma: does not involute: present at birth: grows with the child: treated with pulsed dye laser).

Angiosarcoma is a rare but aggressive malignant tumour of vascular endothelium, occurring in the skin (scalp and face of elderly patients), breast (after radiotherapy: Stewart-Treves syndrome: angiosarcoma arising in a lymphoedematous arm after mastectomy), liver (associated with vinyl chloride, arsenic, and thorotrast exposure), and soft tissues. It presents as a rapidly enlarging purplish or haemorrhagic skin lesion. Treatment is wide surgical excision with radiotherapy; prognosis is poor because of early haematogenous metastasis. Kaposi's sarcoma (a vascular tumour associated with HHV-8 infection and immunosuppression: particularly HIV/AIDS) presents as purple or brown skin lesions typically on the lower extremities.

Fill in the blank: Capillary haemangiomas (strawberry naevus) typically _____ spontaneously by age 5 to 7 years and are managed by _____ unless complicated. Angiosarcoma of the lymphoedematous arm after mastectomy is called _____ syndrome.

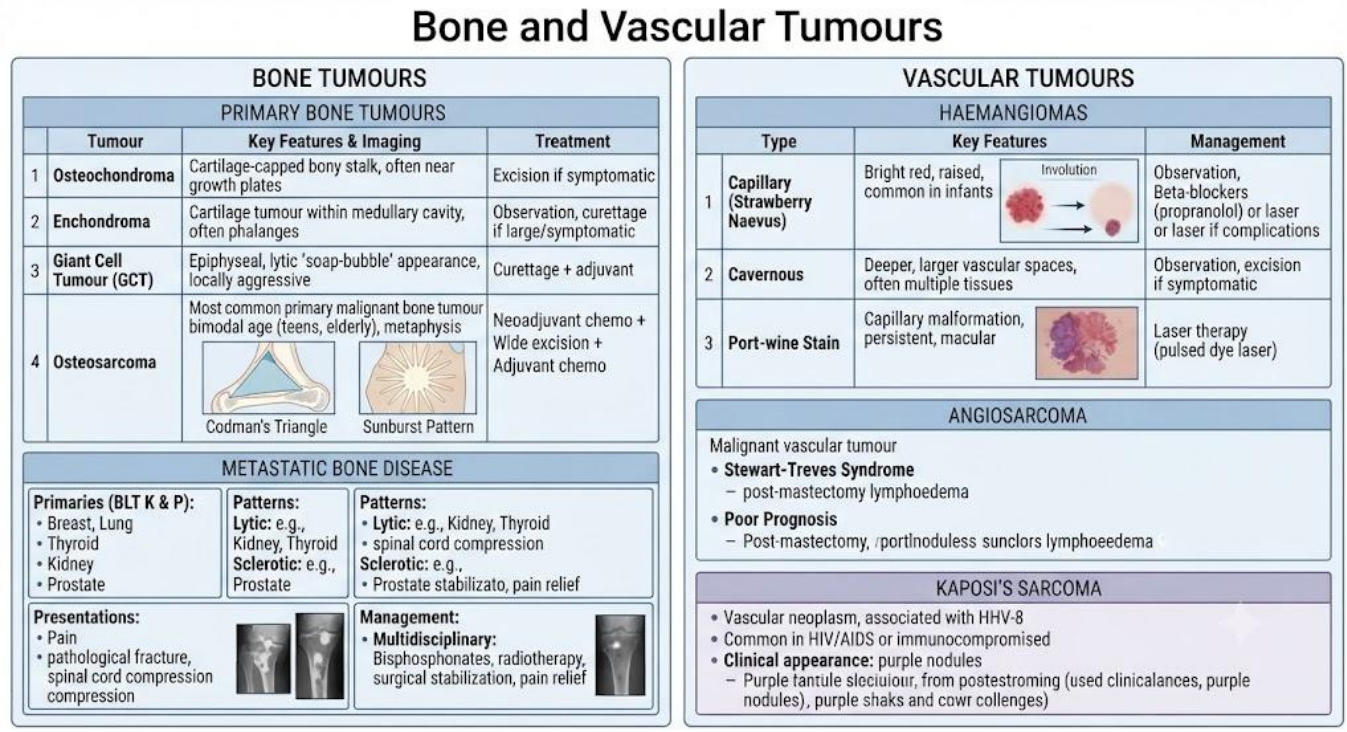

Figure 1.4: Primary bone tumours, metastatic bone disease, and vascular tumours and anomalies

Pilot questions 1.4

- Describe the clinical and radiological features of osteosarcoma. (Linked to SLO 1.4)
- State the five most common primary tumours that metastasise to bone and distinguish lytic from sclerotic metastases. (Linked to SLO 1.4)
- Describe the clinical presentation of metastatic bone disease and state its complications. (Linked to SLO 1.4)
- Describe the natural history of a capillary haemangioma (strawberry naevus) and state when treatment is required. (Linked to SLO 1.4)
- Describe the Stewart-Treves syndrome and explain its pathogenesis. (Linked to SLO 1.4)

1.5 Lymphoma and Lymph Node Disease

1.5.1 Hodgkin's lymphoma

Hodgkin's lymphoma (HL) is a malignancy of lymphoid tissue characterised histologically by the presence of Reed-Sternberg cells (large binucleate or multinucleate cells with prominent owl-eye nucleoli: the pathognomonic cell). It has a bimodal age distribution (first peak in young

adults aged 15 to 35 years; second peak above 55 years) and is associated with Epstein-Barr virus (EBV) infection, particularly the mixed cellularity subtype. The Ann Arbor staging system (Stages I to IV, with A or B suffix for absence or presence of B symptoms) is used.

Clinical features: painless cervical lymphadenopathy (the most common presentation: rubbery, non-tender, discrete cervical nodes), mediastinal lymphadenopathy (on CXR), B symptoms (fever above 38 degrees C, drenching night sweats, and unexplained weight loss of above 10 percent body weight in 6 months), pruritus (diffuse itching: a B-equivalent symptom), alcohol-induced lymph node pain (a classic but rare feature). Investigations: excision biopsy of the node (not FNAC: lymphoma requires the full nodal architecture for Reel-Sternberg cell identification and subtype classification), CT of the chest, abdomen, and pelvis for staging, and PET-CT for treatment response assessment. Treatment: ABVD chemotherapy (doxorubicin, bleomycin, vinblastine, and dacarbazine) is the standard first-line regimen; radiotherapy is added for early-stage disease.

Fill in the blank: The pathognomonic cell of Hodgkin's lymphoma is the _____ cell with prominent owl-eye nucleoli. Hodgkin's lymphoma is staged using the _____ staging system (Stages I to IV). The standard first-line chemotherapy is _____ (doxorubicin, bleomycin, vinblastine, dacarbazine).

1.5.2 Non-Hodgkin's lymphoma

Non-Hodgkin's lymphoma (NHL) is a heterogeneous group of lymphoid malignancies without Reed-Sternberg cells, encompassing more than 60 distinct subtypes. It is classified as B-cell (approximately 85 percent: diffuse large B-cell lymphoma (DLBCL) is the most common: aggressive but potentially curable; follicular lymphoma: indolent but incurable) or T-cell (approximately 15 percent: generally more aggressive). NHL is associated with immunosuppression (HIV/AIDS, post-transplant), chronic infection (Helicobacter pylori: gastric MALT lymphoma), autoimmune disease, and EBV.

Clinical features of NHL differ from HL: NHL more commonly presents with widespread, non-contiguous lymphadenopathy, extranodal disease (stomach: MALT lymphoma; central nervous system; skin; bone marrow: causing cytopenias), and B symptoms. The Ann Arbor staging system is also used for NHL. Treatment: R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, prednisolone) is the standard first-line treatment for DLBCL. Gastric MALT lymphoma (low-grade, associated with H. pylori) may regress with H. pylori eradication therapy alone.

Fill in the blank: NHL is classified as predominantly _____ cell (85 percent) or T-cell (15 percent). The most common aggressive NHL is _____ large B-cell lymphoma (DLBCL). Gastric MALT lymphoma may regress with _____ eradication therapy.

1.5.3 Reactive and metastatic lymphadenopathy

Lymphadenopathy (enlarged lymph nodes) has many causes: reactive (the most common: in response to infection or inflammation), malignant (lymphoma or metastatic cancer), and granulomatous (tuberculosis, sarcoidosis). The clinical approach: localised lymphadenopathy (in the drainage territory of a specific region: suggests infection or malignancy in that region), generalised lymphadenopathy (multiple regions: suggests systemic infection such as infectious mononucleosis or HIV, haematological malignancy, or autoimmune disease). Key clinical features suggesting malignancy: hard or rubbery consistency, non-tender, non-mobile (fixed to adjacent structures), size above 1 cm, progressive enlargement, and absence of signs of infection.

Investigation: blood tests (FBC: lymphocytosis in CLL and viral infections; LDH: elevated in NHL; ESR: elevated in HL; EBV and CMV serology; HIV test), imaging (ultrasound: characterises node architecture; CT for staging; PET-CT for lymphoma), and tissue diagnosis (excision biopsy: the gold standard for lymphoma diagnosis; core needle biopsy acceptable for metastatic lymphadenopathy). Virchow's node (left supraclavicular lymphadenopathy) strongly suggests upper GI or thoracic malignancy: Troisier's sign. Sister Mary Joseph's nodule (periumbilical lymphadenopathy) indicates peritoneal metastases from an abdominal or pelvic primary.

Fill in the blank: Lymphadenopathy with hard, fixed, non-tender nodes above _____ cm suggests malignancy. Virchow's node (left _____ lymphadenopathy) suggests upper GI or thoracic malignancy. Sister Mary Joseph's nodule (periumbilical) indicates _____ metastases.

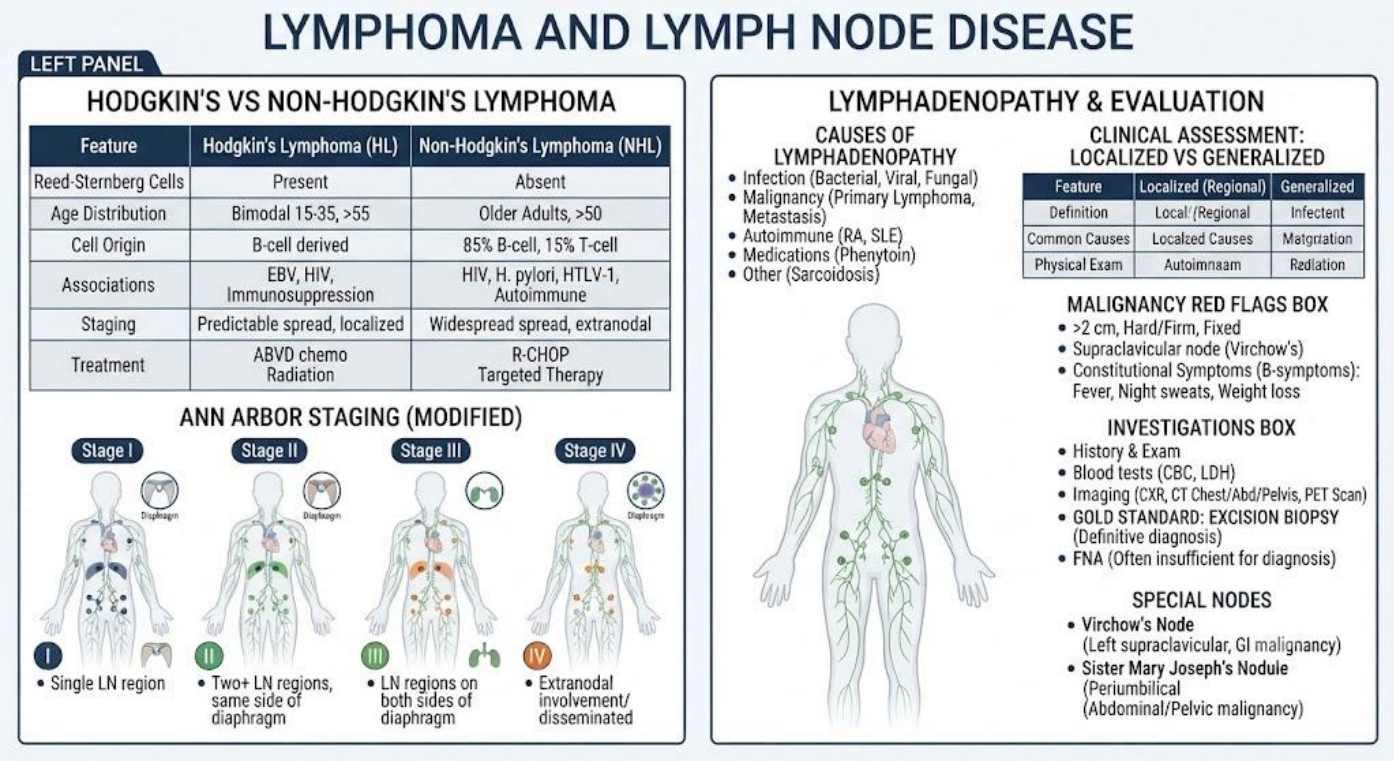

Figure 1.5: Hodgkin's versus non-Hodgkin's lymphoma comparison, Ann Arbor staging, and lymphadenopathy assessment

Pilot questions 1.5

1. Describe the Reed-Sternberg cell and explain its diagnostic significance in Hodgkin's lymphoma. (Linked to SLO 1.5)
2. Describe the clinical features of Hodgkin's lymphoma and state the Ann Arbor staging system. (Linked to SLO 1.5)
3. Distinguish Hodgkin's from non-Hodgkin's lymphoma in terms of clinical features and treatment. (Linked to SLO 1.5)
4. Describe the clinical approach to a patient with generalised lymphadenopathy. (Linked to SLO 1.5)
5. Describe Virchow's node and Sister Mary Joseph's nodule and state the significance of each. (Linked to SLO 1.5)

1.6 Principles of Surgical Oncology

1.6.1 Curative surgical principles

The goal of curative cancer surgery is to remove all macroscopic and microscopic tumour with clear surgical margins (the resection plane must be free of tumour cells: the R classification: R0: no residual tumour: the only potentially curative resection; R1: microscopic residual tumour; R2: macroscopic residual tumour). The principles of curative surgery include: en bloc resection (the primary tumour and its regional lymph nodes are removed together as one specimen without entering the tumour), clear margins (a rim of normal tissue surrounds the tumour on all sides: margin width depends on the tumour type), no-touch technique (the venous drainage is ligated early to prevent haematogenous dissemination during operative manipulation), and lymph node dissection (removal of the regional lymph nodes for staging and to achieve locoregional control).

Sentinel lymph node biopsy (SLNB) is a technique to identify and selectively remove the first draining lymph node from a tumour (the sentinel node) to determine whether regional lymph nodes are involved without performing a full lymph node dissection (which carries risks of lymphoedema and nerve injury). SLNB is now standard for melanoma (Breslow thickness above 0.8 mm) and early-stage breast cancer. The sentinel node is identified by injection of a blue dye and/or a radioactive tracer around the tumour; if the sentinel node is negative for metastasis, the remaining nodes are not removed.

Fill in the blank: An R0 resection means _____ residual tumour. The no-touch technique ligates the _____ drainage early during surgery to prevent haematogenous dissemination. _____ lymph node biopsy identifies the first draining node from a tumour to spare full dissection.

1.6.2 Palliative surgery and supportive care

Palliative surgery aims to relieve symptoms and improve quality of life in patients with incurable cancer, without attempting to cure the disease. Common palliative surgical procedures: bypass

operations (gastrojejunostomy for unresectable gastric cancer causing gastric outlet obstruction; biliary bypass or endoscopic stenting for unresectable pancreatic head cancer causing obstructive jaundice), defunctioning stoma (colostomy for obstructing colonic cancer when primary resection is not possible), debulking or cytoreductive surgery (removal of the majority of the tumour to reduce tumour bulk: used in ovarian cancer: reduces ascites and improves chemotherapy response), and feeding access (gastrostomy or jejunostomy for nutritional support in patients with head and neck or oesophageal cancer unable to swallow).

Supportive care in surgical oncology includes: pain management (WHO analgesic ladder: step 1 paracetamol and NSAIDs; step 2 weak opioids; step 3 strong opioids: morphine or fentanyl; adjuvants: dexamethasone for bone pain; bisphosphonates for bone metastases), nutritional support (enteral nutrition preferred when the gut functions; parenteral nutrition for gut failure), wound care (fungating tumours: odour control with metronidazole dressings; exudate management), and psychological support (breaking bad news: SPIKES framework: counselling and palliative care team involvement).

Fill in the blank: Gastrojejunostomy for unresectable gastric cancer causing gastric outlet obstruction is an example of _____ surgery. The WHO analgesic step 3 for cancer pain uses _____ opioids such as morphine. The SPIKES framework is used for _____ bad news.

1.6.3 Multimodal cancer treatment

Modern cancer treatment is multimodal, combining surgery, chemotherapy, radiotherapy, targeted therapy, and immunotherapy. Neoadjuvant therapy (given before surgery): shrinks the tumour (downstaging), allows assessment of chemosensitivity, treats micrometastases, and may enable organ preservation (for example, neoadjuvant chemoradiotherapy for rectal cancer can achieve a complete pathological response in approximately 15 to 20 percent of patients, potentially avoiding surgery). Adjuvant therapy (given after curative surgery): eradicates residual micrometastases to reduce the risk of systemic recurrence.

Targeted therapy (monoclonal antibodies and small molecule inhibitors that target specific oncogenic pathways): trastuzumab (Herceptin) targets HER2-positive breast cancer; cetuximab targets EGFR in colorectal cancer; imatinib (Gleevec) targets BCR-ABL in CML and c-KIT in GIST (gastrointestinal stromal tumour). Immunotherapy (immune checkpoint inhibitors): pembrolizumab and nivolumab (anti-PD-1 antibodies) have transformed the treatment of melanoma, lung cancer, and renal cell carcinoma by releasing the immune system's natural anti-tumour response. The multidisciplinary team (MDT) meeting (surgeon, oncologist, radiologist, pathologist, specialist nurse) is the standard of care for all cancer treatment decisions.

Fill in the blank: Neoadjuvant therapy is given _____ surgery to shrink the tumour. Trastuzumab (Herceptin) targets _____ in breast cancer. Pembrolizumab and nivolumab are _____ checkpoint inhibitors that target the PD-1 pathway.

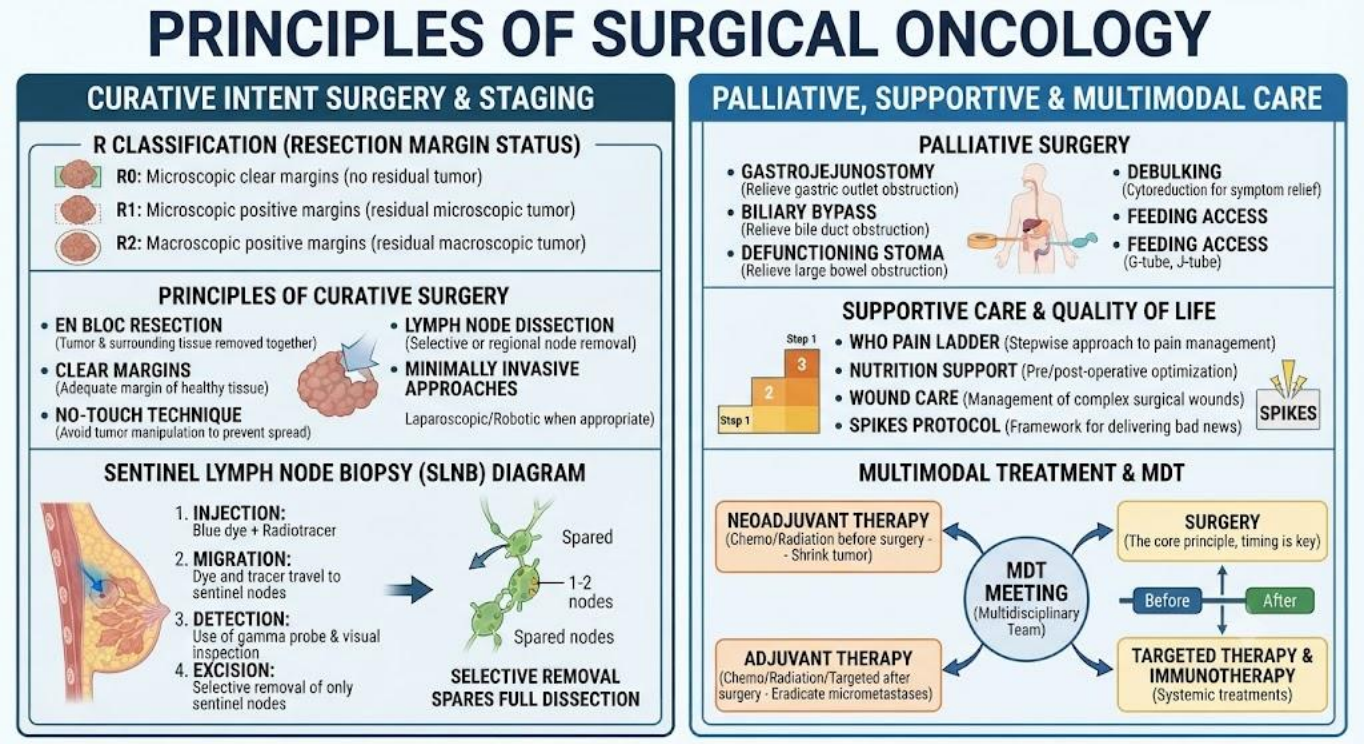

Figure 1.6: Curative surgery principles, sentinel lymph node biopsy, palliative surgery, and multimodal cancer treatment

Pilot questions 1.6

- Describe the R classification for surgical resection margins and state which is the only potentially curative resection. (Linked to SLO 1.6)
- Describe the principles of curative cancer surgery. (Linked to SLO 1.6)
- Describe sentinel lymph node biopsy: the technique, indications, and advantage over full lymph node dissection. (Linked to SLO 1.6)
- Describe three palliative surgical procedures and state the indication for each. (Linked to SLO 1.6)
- Distinguish neoadjuvant from adjuvant therapy and give an example of each. (Linked to SLO 1.6)

Series Summary

This Series covered the core principles of solid tumours and surgical oncology. We distinguished benign from malignant tumours, classified tumours by tissue of origin, and described tumour grading and TNM staging. We examined the structured clinical approach to a tumour: history (weight loss, B symptoms, risk factors), examination (lump characteristics, Virchow's node), and investigations (core biopsy, CT staging, tumour markers). We then surveyed specific tumour categories: skin tumours (BCC, melanoma with ABCDE and Breslow thickness, STS), bone tumours (osteosarcoma: Codman's triangle; metastatic bone disease: BLT kidney prostate), vascular tumours (strawberry naevus involution; angiosarcoma; Stewart-Treves), and lymph node disease (HL: Reed-Sternberg cell and ABVD; NHL: R-CHOP; lymphadenopathy: Virchow's and Sister Mary Joseph's). We closed with surgical oncology principles: R0 resection, en bloc, no-touch, SLNB, palliative procedures, and multimodal treatment.

You should now be able to distinguish benign from malignant tumours and describe staging and grading (SLO 1.1), describe the clinical approach to a tumour (SLO 1.2), describe skin, soft tissue, bone, vascular, and lymph node tumours (SLOs 1.3 to 1.5), and describe the principles of curative and palliative surgical oncology and multimodal treatment (SLO 1.6).

Glossary of Terms

- **Angiosarcoma:** a rare and aggressive malignant tumour of vascular endothelium; Stewart-Treves syndrome refers to angiosarcoma arising in a lymphoedematous arm after mastectomy.
- **Ann Arbor staging:** the staging system for lymphoma: Stage I (single node region), Stage II (two or more regions same side of diaphragm), Stage III (both sides of diaphragm), Stage IV (extranodal involvement); A suffix: no B symptoms; B suffix: B symptoms present.
- **Breslow thickness:** the depth of invasion of a melanoma in millimetres, measured from the granular cell layer of the epidermis to the deepest tumour cell on histology; the most important prognostic factor in melanoma.
- **Codman's triangle:** a radiological finding on plain X-ray in osteosarcoma: a triangular area of new bone formation where the periosteum is lifted from the cortex by the growing tumour; characteristic of osteosarcoma.
- **En bloc resection:** surgical removal of the primary tumour and its regional lymph nodes as a single intact specimen without entering or breaching the tumour; a core principle of curative cancer surgery.
- **R0 resection:** a surgical resection with no residual macroscopic or microscopic tumour at the margins; the only potentially curative surgical resection in cancer surgery.
- **Reed-Sternberg cell:** the pathognomonic cell of Hodgkin's lymphoma: a large binucleate or multinucleate cell with prominent owl-eye nucleoli; its identification requires excision biopsy of the lymph node (not FNAC).

- **Sentinel lymph node biopsy (SLNB):** selective removal of the first draining lymph node (sentinel node) from a tumour, identified by blue dye and radiotracer injection, to determine regional node status without full lymph node dissection.
- **Stewart-Treves syndrome:** angiosarcoma arising in the chronically lymphoedematous arm of a patient who has undergone mastectomy and axillary lymph node dissection; a rare but grave complication.
- **Virchow's node:** an enlarged left supraclavicular lymph node from metastatic upper GI or thoracic malignancy; also called Troisier's sign; an important clinical indicator of advanced abdominal or thoracic cancer.

Concept Check Questions [Series 1]

Objective questions

1. A malignant tumour arising from epithelial tissue is called a _____, while one arising from mesenchymal tissue is called a _____. (Linked to SLO 1.1)
2. In the TNM system, M1 indicates distant _____, and corresponds to Stage _____ disease. (Linked to SLO 1.1)
3. The most important prognostic factor in melanoma is _____ thickness, measured in millimetres from the granular cell layer to the deepest tumour cell. (Linked to SLO 1.3)
4. The pathognomonic cell of Hodgkin's lymphoma is the _____ cell, which has prominent owl-eye nucleoli. (Linked to SLO 1.5)
5. An R0 surgical resection means there is _____ residual tumour and is the only potentially _____ resection. (Linked to SLO 1.6)

Short-answer questions

1. Distinguish benign from malignant tumours using four features. (Linked to SLO 1.1)
2. Describe the ABCDE criteria for identifying a suspicious pigmented skin lesion. (Linked to SLO 1.3)
3. State the five most common primaries that metastasise to bone and distinguish lytic from sclerotic metastases. (Linked to SLO 1.4)
4. Describe the clinical features that suggest malignancy in a patient with lymphadenopathy. (Linked to SLO 1.5)

5. Describe sentinel lymph node biopsy: technique, indications, and advantage over full dissection. (Linked to SLO 1.6)

Long-answer questions

1. Describe the clinical approach (history, examination, and investigations) to a patient presenting with a neck lump that is suspected to be malignant. (Linked to SLO 1.2)
2. Describe the clinical features, investigation, and management of soft tissue sarcoma. (Linked to SLO 1.3)
3. Describe the clinical and radiological features of osteosarcoma and outline its management. (Linked to SLO 1.4)
4. Compare Hodgkin's lymphoma and non-Hodgkin's lymphoma in terms of pathology, clinical features, and treatment. (Linked to SLO 1.5)
5. Describe the principles of curative cancer surgery and explain the significance of the R classification. (Linked to SLO 1.6)

Answers to Concept Check Questions [Series 1]

Objective questions

- Carcinoma; sarcoma.
- Metastasis; IV.
- Breslow.
- Reed-Sternberg.
- No; curative.

Short-answer questions

- Benign: slow growth, encapsulated, well-defined borders, no metastasis. Malignant: rapid growth, no true capsule, infiltrative borders, capacity to metastasise. Microscopically, malignant cells show pleomorphism and nuclear atypia and abnormal mitoses, while benign cells closely resemble normal tissue.
- Asymmetry (one half does not mirror the other), Border irregularity (uneven or notched edges), Colour variation (two or more colours within the lesion), Diameter above 6 mm, and Evolution

(any change in size, shape, or colour over time). Any lesion satisfying one or more ABCDE criteria warrants urgent dermatological or surgical review.

- The five most common: Breast (most common overall), Lung, Thyroid, Kidney, and Prostate (mnemonic: BLT with Kidney and Prostate). Lytic metastases (breast, lung, thyroid, kidney) destroy bone, producing radiolucent areas on X-ray with risk of pathological fracture. Sclerotic metastases (prostate) cause increased bone density (radiodense) with risk of spinal cord compression.
- Hard or rubbery consistency, non-tender, fixed to adjacent structures (not mobile), size above 1 cm, progressive enlargement over weeks, and absence of any signs of infection in the drainage territory. The presence of B symptoms (fever, night sweats, weight loss) further raises the suspicion of lymphoma.
- Technique: inject blue dye and a radioactive tracer (technetium-99m) around the tumour; identify the first draining (sentinel) lymph node using a gamma probe and visualisation of blue staining; remove only this node for histological analysis. Indications: melanoma with Breslow thickness above 0.8 mm; early-stage breast cancer. Advantage: spares the patient a full lymph node dissection (and its complications of lymphoedema and sensory nerve injury) if the sentinel node is negative for metastasis.

Long-answer questions

- **Basic response:** History: age (lymphoma common in young adults; metastatic cancer more common in older patients); duration and rate of growth of the lump (rapid growth: lymphoma or aggressive malignancy); associated symptoms: B symptoms (fever, night sweats, weight loss above 10 percent in 6 months: lymphoma), dysphagia or hoarseness (oesophageal or laryngeal cancer), haemoptysis (lung cancer); family history of cancer; alcohol and smoking history; previous cancer treatment. Examination: site and size of the lump; consistency (rubbery: lymphoma; hard: metastatic cancer); fixity to skin or deep structures; whether the upper border is palpable (if not: extends through inguinal canal or thoracic inlet); other lymph node groups (generalised lymphadenopathy suggests lymphoma or systemic disease); thyroid moves on swallowing; thyroglossal cyst moves on tongue protrusion; hepatosplenomegaly; Virchow's node; oral cavity and laryngoscopy. Investigations: bloods (FBC: lymphocytosis in CLL; LDH: elevated in NHL; EBV and CMV serology; HIV); ultrasound (characterise node: benign features versus malignant: loss of hilum, rounded, peripheral vascularity on Doppler); excision biopsy (gold standard for suspected lymphoma); core needle biopsy (for suspected metastatic disease); CT chest, abdomen, pelvis (staging).

Value-added response: The distinction between excision biopsy and core needle biopsy is critical: lymphoma requires the intact nodal architecture to identify Reed-Sternberg cells and classify the subtype accurately; FNAC is insufficient and core biopsy provides only partial architecture. For suspected metastatic lymph node disease, core needle biopsy is usually sufficient to confirm the primary and histological type.

- **Basic response:** Clinical features: a deep-seated, painless, progressively enlarging mass in the extremity or retroperitoneum, greater than 5 cm below the deep fascia; should be regarded as a sarcoma until proven otherwise. Pain is unusual early but may develop as the tumour enlarges. Investigation: MRI of the affected region (the investigation of choice for local staging: defines the relationship of the tumour to neurovascular structures, the extent of the tumour, and guides surgical planning); CT chest (pulmonary metastases: the most common site of distant spread); core needle biopsy (preferred for histological diagnosis; excisional biopsy risks contaminating the tissue planes and compromising subsequent curative resection). Management: wide excision with clear margins (at least 1 to 2 cm of normal tissue in all directions: the primary determinant of local recurrence rate); pre- or postoperative radiotherapy (reduces local recurrence rates from approximately 30 to below 10 percent); chemotherapy for high-grade sarcomas and rhabdomyosarcoma; limb-sparing techniques have largely replaced amputation.

Value-added response: Unplanned excision of a soft tissue sarcoma (a whoops procedure) is a serious error: when an apparently benign lipoma-like lump is excised without prior imaging or biopsy and turns out to be a sarcoma, the surgical planes are contaminated and subsequent wide re-excision or even amputation may be required. Any soft tissue mass greater than 5 cm and deep to fascia should be imaged with MRI and biopsied before excision.

- **Basic response:** Clinical features: pain and swelling around the knee (most common site: distal femur and proximal tibia) in an adolescent or young adult. The pain is initially activity-related but becomes constant. A palpable, warm, tender mass develops. Systemic features: elevated ESR, LDH, and alkaline phosphatase. Radiological features: X-ray (plain film): Codman's triangle (periosteal reaction from the periosteum being lifted by the growing tumour: a triangular area of new bone formation between the cortex and the elevated periosteum) and the sunburst pattern (spicules of tumour bone radiating outward from the cortex through the periosteum: pathognomonic of osteosarcoma). MRI: defines local extent and relationship to the neurovascular bundle (essential for surgical planning). CT chest: pulmonary metastases (present in approximately 20 percent at diagnosis). Management: neoadjuvant chemotherapy (MAP: methotrexate, doxorubicin, cisplatin: typically 3 cycles before surgery: shrinks the tumour and allows assessment of chemosensitivity from the histological response in the resected specimen) followed by limb-sparing surgical resection with bone reconstruction (endoprosthesis or allograft), then adjuvant chemotherapy (3 further cycles).

Value-added response: The degree of histological necrosis in the resected specimen after neoadjuvant chemotherapy (the histological response: defined as above 90 percent necrosis in the resected tumour: a good response) is a strong prognostic indicator in osteosarcoma: patients with a good histological response have a significantly better 5-year survival (approximately 70 to 80 percent) than those with a poor response (approximately 40 to 50 percent).

- **Basic response:** Hodgkin's lymphoma: pathognomonic Reed-Sternberg cell (binucleate with owl-eye nucleoli); bimodal age distribution (15 to 35 and above 55 years); associated with EBV; typically presents with painless cervical lymphadenopathy; B symptoms; mediastinal lymphadenopathy on CXR; treatment: ABVD chemotherapy plus radiotherapy for early-stage disease; generally good prognosis (5-year survival approximately 85 to 90 percent for early

stages). Non-Hodgkin's lymphoma: no Reed-Sternberg cell; heterogeneous group over 60 subtypes; B-cell 85 percent and T-cell 15 percent; more commonly presents with widespread non-contiguous lymphadenopathy and extranodal disease (stomach: MALT; CNS; skin; bone marrow); associated with immunosuppression, H. pylori, EBV, autoimmune disease; most common aggressive subtype: DLBCL: treated with R-CHOP; indolent subtypes (follicular lymphoma): incurable but slow-growing. Both use Ann Arbor staging (Stages I to IV with A or B suffix for B symptoms).

Value-added response: Excision biopsy is the gold standard for diagnosing both HL and NHL because the Reed-Sternberg cell, which defines HL and determines its subtype, requires the intact nodal architecture for identification. FNAC samples individual cells without preserving architecture and cannot reliably distinguish HL from NHL or classify the subtype; this distinction is critical because HL and NHL are treated differently.

- **Basic response:** R classification: R0 (no residual tumour at the resection margins: microscopically and macroscopically clear: the only potentially curative resection); R1 (microscopic residual tumour at the margins: microscopically positive margin: not curative); R2 (macroscopic residual tumour left behind: not curative). Principles of curative surgery: En bloc resection (the primary tumour and its regional lymph nodes are removed together as a single intact specimen without entering the tumour: breaching the tumour disseminates cancer cells and compromises cure). Clear margins (a rim of histologically normal tissue must surround the tumour on all sides: the required margin width depends on tumour type: 1 to 2 cm for sarcomas; 1 cm for melanoma if Breslow above 1 mm). No-touch technique (the venous drainage from the tumour is ligated before the tumour is manipulated to prevent haematogenous dissemination of tumour cells during surgical handling). Lymph node dissection (regional lymph nodes are removed with the specimen for accurate staging and to achieve locoregional control: positive nodes upstage the tumour and indicate the need for adjuvant systemic therapy).

Value-added response: The concept of surgical margin is evolving with advances in imaging and molecular pathology. In some tumour types (for example, breast-conserving surgery for early breast cancer), a margin of as little as 2 mm is accepted as adequate provided the patient receives radiotherapy. The principle that a positive margin (R1) must always be re-excised where possible remains fundamental: in most solid tumours, a positive margin doubles or triples the local recurrence rate, regardless of adjuvant treatment.

Answers to Pilot Questions [Series 1]

Part 1.1

1. Benign: slow growth, well-encapsulated, well-defined borders, no metastasis, well-differentiated cells resembling parent tissue, rare and normal mitoses. Malignant: rapid growth, no true capsule, ill-defined infiltrative borders, capacity to metastasise, pleomorphic and atypical cells, frequent and abnormal mitoses.

2. Epithelial: carcinoma (example: adenocarcinoma of the colon). Mesenchymal: sarcoma (example: osteosarcoma of the femur). Haematopoietic: lymphoma (example: Hodgkin's lymphoma) and leukaemia. Germ cell: teratoma (example: testicular teratoma). Neural: glioma (example: glioblastoma multiforme).

3. The suffix -oma generally denotes a benign tumour (lipoma, fibroma, adenoma). Three important exceptions where -oma denotes a malignant tumour: melanoma (malignant tumour of melanocytes), hepatoma (hepatocellular carcinoma), and lymphoma (malignant lymphoid tumour). The suffixes -carcinoma and -sarcoma always denote malignancy.

4. Tumour grading describes the degree of differentiation of tumour cells on histological examination. Grade 1 (well-differentiated: cells closely resemble normal parent tissue: low-grade: slower growth and better prognosis). Grade 2 (moderately differentiated). Grade 3 (poorly differentiated: cells bear little resemblance to normal tissue: high-grade: more aggressive and worse prognosis). Grade 4 (undifferentiated or anaplastic). Grading is an independent prognostic factor: high-grade tumours are more likely to metastasise and have higher recurrence rates after treatment.

5. TNM: T describes the primary tumour by size and local invasion (T1 smallest to T4 most locally advanced). N describes regional lymph node involvement (N0: no nodal involvement; N1 to N3: increasing number and extent of involved nodes). M describes distant metastasis (M0: absent; M1: present). Stage IV indicates M1 (distant metastasis) and signifies that the cancer has spread to distant organs (commonly liver, lung, bone, or brain); it is generally not curable by surgery alone and treatment is aimed at prolonging life and relieving symptoms.

Part 1.2

1. Key features: presenting complaint (site, duration, rate of change in size); associated symptoms (unexplained weight loss above 10 percent in 6 months: cardinal feature of malignancy; B symptoms: fever, night sweats, and weight loss: lymphoma; haemoptysis: lung cancer; haematuria: renal or bladder cancer; dysphagia: oesophageal cancer); risk factors (age and sex; family history: BRCA1/2 for breast and ovarian cancer, FAP for colorectal cancer, Li-Fraumeni syndrome for sarcomas; occupational: asbestos for mesothelioma, UV radiation for skin cancer, smoking for lung and bladder and oesophageal cancer); previous cancer treatment (radiation-induced sarcoma after radiotherapy).

2. B symptoms of lymphoma: fever (above 38 degrees C), drenching night sweats, and unexplained weight loss of above 10 percent of body weight in 6 months. Significance: B symptoms indicate more advanced (Stage III or IV) disease and are associated with a worse prognosis compared with Stage A (absence of B symptoms). The presence of B symptoms upgrades the staging suffix from A to B (for example, Stage IIA becomes Stage IIB) and influences the intensity of chemotherapy required.

3. Sequence: site and size (measure in two dimensions), shape (regular or irregular), surface (smooth or nodular), edge (well-defined: benign; ill-defined: infiltrating: malignant), consistency (soft: lipoma; firm: fibroma; hard: calcification; stony-hard: malignancy), fluctuance (fluid-filled), transillumination (cystic), pulsatility (expansile: arterial aneurysm), reducibility (hernia or varicocoele), compressibility (venous anomaly), temperature (warmth: inflammation or vascular tumour), fixity to skin (tethering or peau d'orange: malignancy) and to deep structures (fixed to muscle or bone: malignant invasion), regional lymph nodes (enlarged: metastatic or infective), auscultation (bruit: AVM).

4. FNAC: a fine needle aspirates cells from the lump for cytological analysis; provides information about cell type but not histological architecture; rapid and minimally invasive; indicated for thyroid nodules, breast lumps (as part of triple assessment), and superficial lymph nodes; cannot distinguish in situ from invasive malignancy; insufficient for lymphoma diagnosis. Core needle biopsy (Tru-Cut): a larger cutting needle obtains a tissue core for full histological analysis including architecture; preferred for most solid tumours; can diagnose sarcomas (where grading requires histological architecture), distinguish lymphoma subtypes (architecture needed), assess receptor status in breast cancer, and confirm malignancy with higher specificity than FNAC.

5. CT chest, abdomen, and pelvis: most important staging investigation for most solid tumours: assesses primary tumour dimensions, regional lymph node involvement, hepatic and pulmonary metastases, and peritoneal disease. PET-CT: most sensitive for detecting metabolically active metastases: identifies disease in sites not anatomically enlarged on CT (for example, metabolically active normal-sized lymph nodes); used for lymphoma staging and for assessing treatment response. MRI: superior soft tissue characterisation: local staging for rectal cancer (assesses circumferential resection margin), sarcomas, and brain tumours. Isotope bone scan: sensitive for detecting skeletal metastases: shows areas of increased isotope uptake at metastatic foci throughout the skeleton. Tumour markers: CEA (colorectal cancer), AFP (hepatocellular carcinoma and germ cell tumours), PSA (prostate cancer), CA-125 (ovarian cancer), CA 19-9 (pancreatic cancer).

Part 1.3

1. BCC is the most common skin cancer, arising from basal keratinocytes after prolonged UV exposure in fair-skinned individuals, predominantly on the face and neck. It presents as a pearly, translucent nodule with surface telangiectasia, a raised rolled edge, and central ulceration (rodent ulcer). It grows slowly and invasion of adjacent tissues (nerve, cartilage, bone) occurs without treatment, but haematogenous metastasis is extremely rare. Treatment: surgical excision with histologically clear margins is the standard; alternatives include radiotherapy (for large or periocular lesions), Mohs micrographic surgery (for recurrent or high-risk lesions at critical sites: nose, ears, eyelids), and topical imiquimod (for superficial BCC only).

2. ABCDE: Asymmetry (one half does not mirror the other), Border irregularity (uneven, notched, or poorly defined edges), Colour variation (two or more shades within the lesion: combinations of

brown, black, red, blue, or white), Diameter above 6 mm (though melanomas can be smaller), and Evolution (any change in the lesion over time: growth, shape change, colour change, bleeding, or new symptoms). Breslow thickness (the depth of invasion in millimetres from the granular cell layer of the epidermis to the deepest tumour cell) is the single most important prognostic factor: the deeper the invasion, the higher the risk of lymph node metastasis and systemic spread.

3. Wide local excision with margins determined by Breslow thickness: below 1 mm Breslow: 1 cm excision margin; 1 to 2 mm: 1 to 2 cm margin; above 2 mm: 2 cm margin. Sentinel lymph node biopsy (SLNB) is performed concurrently for melanomas with Breslow thickness above 0.8 mm to determine whether regional lymph nodes are involved without performing a full lymph node dissection. The SLNB result guides further management: negative sentinel node: no further nodal surgery required; positive sentinel node: discussion of completion lymph node dissection or ongoing surveillance. Adjuvant immunotherapy (anti-PD-1: pembrolizumab or nivolumab) is now standard for Stage III and high-risk Stage II melanoma.

4. Clinical features: a deep-seated, painless, progressively enlarging mass, typically greater than 5 cm in the extremity or retroperitoneum, below the deep fascia: this should be regarded as a sarcoma until proven otherwise. The lump is firm and may be warm. Pain is usually absent early but develops as the tumour enlarges and compresses adjacent structures. Investigation of choice for local staging: MRI defines the relationship of the tumour to neurovascular structures, the extent of the compartment involved, and guides surgical planning; it is superior to CT for soft tissue characterisation.

5. Wide excision with clear margins of at least 1 to 2 cm in all directions is the primary treatment: the width of the clear margin is the most important determinant of local recurrence. Radiotherapy (pre- or postoperative) reduces local recurrence rates from approximately 30 percent (surgery alone) to below 10 percent (surgery plus radiotherapy) in high-grade sarcomas. Chemotherapy is used for high-grade sarcomas (particularly rhabdomyosarcoma and Ewing's sarcoma in children and young adults) and for systemic disease. Limb-sparing techniques (wide local excision preserving the limb with reconstruction of bone and soft tissue defects) have largely replaced amputation in most cases; amputation is reserved for tumours encasing major neurovascular structures or where limb-sparing excision would leave a non-functional limb.

Part 1.4

1. Clinical features: pain and swelling around the knee (most common site: distal femur most frequently, then proximal tibia) in an adolescent or young adult aged 10 to 20 years. The pain is initially activity-related then becomes constant; a palpable warm tender mass develops over weeks. Elevated ESR, alkaline phosphatase, and LDH. Radiological features: X-ray: (1) Codman's triangle: a triangular area of periosteal new bone formation between the cortex and the periosteum that has been lifted by the growing tumour: seen at the edge of the tumour on plain X-ray; (2) sunburst pattern: spicules of tumour bone radiating outward from the cortex through the periosteum in a radial pattern: highly characteristic of osteosarcoma. MRI: defines the

intramedullary extent of the tumour, the relationship to the growth plate, and the neurovascular bundle (critical for surgical planning). CT chest: pulmonary metastases (present in approximately 20 percent at diagnosis: the most common site of distant spread).

2. The five most common primaries: Breast (most common overall: approximately 70 percent of metastatic bone disease in women), Lung, Thyroid, Kidney (renal cell carcinoma), and Prostate (most common in men). Mnemonic: BLT with Kidney and Prostate. Lytic metastases: destroy bone (breast, lung, kidney, and thyroid metastases): appear as radiolucent (dark) areas on plain X-ray; risk of pathological fracture is high. Sclerotic metastases: stimulate new bone formation (prostate metastases predominantly; also breast in some cases): appear as radiodense (white) areas on plain X-ray; risk of spinal cord compression from vertebral involvement.

3. Bone pain (the most common presentation: typically constant, worse at night, not relieved by rest: distinguishes from mechanical pain which improves with rest), pathological fracture (fracture through diseased bone after minimal or no trauma: may be the first presentation: commonly at the proximal femur in metastatic breast or prostate cancer), hypercalcaemia (from osteolysis: presents with confusion, constipation, polyuria, nausea, and cardiac arrhythmias: treated with IV saline hydration and bisphosphonates: zoledronic acid), spinal cord compression (from vertebral body metastasis collapsing onto the spinal cord: presents with back pain, leg weakness or paralysis, and bladder and bowel dysfunction: treated with high-dose dexamethasone and urgent radiotherapy or surgical decompression: a surgical emergency).

4. Capillary haemangioma (strawberry naevus): appears in the first weeks of life as a bright red, raised, compressible, well-defined vascular lesion; grows rapidly in the first year then begins to involute spontaneously, with approximately 50 percent involuted by age 5 years and 70 percent by age 7 years; the majority (above 90 percent) involute completely by adolescence leaving minimal or no scar. Management: observe in the majority; treatment is required if: (1) complications arise: bleeding, ulceration, or secondary infection; (2) the lesion threatens the airway (subglottic haemangioma); (3) Kasabach-Merritt syndrome develops (a consumptive coagulopathy from platelet trapping in a large haemangioma); (4) vision is threatened (periorbital haemangioma causing amblyopia); or (5) for cosmetic reasons in adolescence if involution is incomplete. Treatment options: propranolol (oral: now first-line for complicated haemangiomas), laser therapy, or surgical excision.

5. Stewart-Treves syndrome is the development of angiosarcoma in the chronically lymphoedematous arm of a patient who has undergone mastectomy and axillary lymph node dissection for breast cancer. Pathogenesis: the chronic lymphoedema (caused by disruption of the axillary lymphatic drainage) leads to local immunodeficiency (reduced immune surveillance in the oedematous tissue), chronic inflammation, and eventually malignant transformation of the vascular endothelium (angiosarcoma). The latent period between mastectomy and the development of angiosarcoma is typically 5 to 10 years. It presents as a rapidly enlarging purplish or haemorrhagic skin lesion on the affected arm. Prognosis is extremely poor (median survival approximately 18 to 24 months from diagnosis) because of early haematogenous metastasis. Treatment: radical wide excision (often requiring amputation of the arm) and radiotherapy.

Part 1.5

1. The Reed-Sternberg (RS) cell is a large binucleate or multinucleate lymphoid cell with abundant pale cytoplasm, large eosinophilic nucleoli, and a prominent clear halo around each nucleolus, giving the appearance of an owl's eye (the owl-eye appearance). It arises from germinal centre B-cells. Diagnostic significance: the RS cell is the pathognomonic (defining) cell of Hodgkin's lymphoma; its presence (in a background of reactive inflammatory cells including lymphocytes, plasma cells, eosinophils, and neutrophils) is required for the histological diagnosis of HL. Its identification requires excision biopsy of the lymph node (not FNAC, which provides only individual cells without the surrounding reactive background) because the RS cell must be seen in the context of the reactive cellular background to confirm HL.

2. Clinical features: painless, rubbery, non-tender cervical lymphadenopathy (the most common presentation: discrete enlarged nodes in the neck); mediastinal lymphadenopathy on CXR (found in approximately 50 percent of HL at diagnosis: important to assess before anaesthesia as large mediastinal masses risk airway collapse under general anaesthesia); B symptoms (fever above 38 degrees C, drenching night sweats, and unexplained weight loss above 10 percent body weight in 6 months: present in approximately 30 percent of HL: indicate more advanced disease); pruritus (generalised itching: a B-equivalent symptom); alcohol-induced lymph node pain (rare but classic). Ann Arbor Staging: Stage I (single node region); Stage II (two or more regions on the same side of the diaphragm); Stage III (lymph node regions on both sides of the diaphragm); Stage IV (extranodal involvement: bone marrow, liver, lung). A suffix: no B symptoms; B suffix: B symptoms present.

3. Hodgkin's lymphoma: Reed-Sternberg cells present; bimodal age distribution (15 to 35 and above 55 years); typically presents with painless cervical or mediastinal lymphadenopathy; contiguous spread from one node region to adjacent regions; EBV association; treatment: ABVD chemotherapy (doxorubicin, bleomycin, vinblastine, dacarbazine) plus radiotherapy for early-stage disease; generally good prognosis (5-year survival approximately 85 to 90 percent for Stage I to II). Non-Hodgkin's lymphoma: no Reed-Sternberg cells; over 60 subtypes; B-cell 85 percent and T-cell 15 percent; more commonly presents with widespread non-contiguous lymphadenopathy and extranodal disease; associated with immunosuppression, H. pylori, autoimmune disease; treatment: R-CHOP for aggressive DLBCL; H. pylori eradication for gastric MALT lymphoma; prognosis varies widely by subtype (DLBCL: potentially curable; follicular: indolent but incurable).

4. History: duration of lymphadenopathy; associated B symptoms (fever, night sweats, weight loss: lymphoma); symptoms of infection in the drainage territory (pharyngitis: reactive cervical; dental abscess: submandibular); haemoptysis or dysphagia or change in bowel habit (suggesting a primary malignancy with nodal metastasis); risk factors for HIV (immunodeficiency lymphadenopathy). Examination: lymph node characteristics (consistency: rubbery: lymphoma; hard: metastatic cancer; tender: reactive or infective; size; fixed or mobile); hepatosplenomegaly (lymphoma or haematological malignancy); rash (viral exanthem or lymphoma skin involvement); other lymph node groups (generalised: systemic disease). Investigations: FBC (lymphocytosis: CLL or viral; leucocytosis: infection; cytopenias: bone marrow infiltration);

LDH (elevated in NHL); ESR (elevated in HL); EBV, CMV, HIV serology; CT chest, abdomen, pelvis (staging); PET-CT (lymphoma); excision biopsy (gold standard for lymphoma); core needle biopsy (for suspected metastatic nodes).

5. Virchow's node: the left supraclavicular lymph node (also called Troisier's sign when enlarged from malignancy); drains from the thoracic duct (which receives lymph from the entire abdomen and left side of the thorax); enlargement indicates metastatic spread from upper GI malignancy (gastric cancer: the classic association), lung cancer, oesophageal cancer, or lymphoma. It is clinically significant because its presence indicates advanced disease (usually Stage III or IV) that has spread to regional lymph nodes remote from the primary. Sister Mary Joseph's nodule: an enlarged periumbilical lymph node resulting from metastatic spread along the ligamentum teres to the periumbilical lymphatics; it indicates peritoneal carcinomatosis from an intra-abdominal or pelvic primary (most commonly gastric, ovarian, colorectal, or pancreatic cancer); its presence indicates unresectable Stage IV disease.

Part 1.6

- R0: no residual tumour at the resection margins (both macroscopically and microscopically clear): the only potentially curative resection; the margin is confirmed by the pathologist on histological examination of the resection specimen edges. R1: microscopic residual tumour at the margins (the pathologist finds tumour cells at the inked resection margin on histological examination): not curative; associated with significantly higher local recurrence rates; re-excision should be performed where anatomically feasible. R2: macroscopic residual tumour left behind (the surgeon can see or palpate residual tumour at the end of the operation): not curative; typically performed as a palliative debulking procedure (for example, ovarian cancer cytoreduction) to reduce tumour bulk and improve the efficacy of subsequent chemotherapy. R0 is the only potentially curative resection.
- En bloc resection: the primary tumour and its regional lymph nodes are removed together as a single intact specimen without entering or breaching the tumour; breaching the tumour releases tumour cells into the wound and surgical field, dramatically increasing local recurrence and potentially causing dissemination. Clear margins: a rim of histologically normal tissue must surround the tumour on all sides; the required margin width depends on tumour type (sarcomas: 1 to 2 cm; melanoma: 1 to 2 cm depending on Breslow thickness; colorectal cancer: clear circumferential resection margin). No-touch technique: the venous drainage from the tumour is ligated before the tumour is manipulated to prevent haematogenous dissemination of tumour cells during surgical handling. Lymph node dissection: regional lymph nodes are removed with the specimen for accurate pathological staging and to achieve locoregional control; positive nodal status guides adjuvant therapy.
- Technique: a blue dye (isosulphan blue or patent blue V) and a radioactive tracer (technetium-99m-labelled nanocolloid) are injected intradermally or intraparenchymally around the primary tumour. The tracer travels via lymphatic channels to the first draining (sentinel) lymph node. At

surgery, the sentinel node is identified by its blue staining and by using a handheld gamma probe (the node with the highest radioactive count). Only the sentinel node is removed and sent for frozen section or detailed histological analysis. Indications: melanoma with Breslow thickness above 0.8 mm; early-stage breast cancer (clinically node-negative). Advantage over full dissection: if the sentinel node is negative for metastasis (no tumour cells found), the remaining regional nodes are not removed; this spares the patient the complications of full lymph node dissection (lymphoedema: a significant long-term morbidity; sensory nerve injury; wound seroma; restricted range of movement).

- Gastrojejunostomy: a surgical bypass between the stomach and the jejunum; indicated for unresectable gastric or pancreatic cancer causing gastric outlet obstruction; allows the patient to eat and drink and prevents gastric obstruction without removing the tumour. Biliary bypass (hepaticojejunostomy or choledochojejunostomy) or endoscopic biliary stenting: indicated for unresectable pancreatic head cancer, cholangiocarcinoma, or liver hilar metastases causing obstructive jaundice; relieves jaundice, prevents cholangitis, and improves quality of life. Defunctioning colostomy (or stoma): indicated for obstructing left-sided colorectal cancer when primary resection is not possible (peritoneal carcinomatosis or poor patient fitness); diverts the faecal stream and prevents complete bowel obstruction from worsening.
- Neoadjuvant therapy is given before curative surgery: it shrinks the primary tumour (downstaging: making an initially unresectable tumour resectable, or reducing the extent of surgery required), treats micrometastases present at the time of diagnosis, allows assessment of the tumour's sensitivity to chemotherapy (from the histological response in the resected specimen), and may enable organ preservation (example: neoadjuvant chemoradiotherapy for locally advanced rectal cancer can achieve a complete pathological response in approximately 15 to 20 percent of patients, potentially avoiding radical surgery). Adjuvant therapy is given after curative surgery: it eradicates residual micrometastases that remain after surgery (which are too small to detect on imaging but may grow into clinically apparent metastases), reducing the risk of systemic recurrence (example: adjuvant FOLFOX chemotherapy after curative resection of Stage III colorectal cancer reduces the 5-year recurrence rate by approximately 20 percent).

References and Attributions [Series 1]

Textual References

6. Bailey, H. and Love, R. J. M. (2018). Bailey and Love's Short Practice of Surgery (27th ed.). Boca Raton: CRC Press.
7. Townsend, C. M., Beauchamp, R. D., Evers, B. M. and Mattox, K. L. (2017). Sabiston Textbook of Surgery (20th ed.). Philadelphia: Elsevier.
8. Sabel, M. S. (2009). Essentials of Surgical Oncology. Philadelphia: Lippincott Williams and Wilkins.
9. National Comprehensive Cancer Network. (2023). NCCN Clinical Practice Guidelines in Oncology: Soft Tissue Sarcoma. Fort Washington: NCCN.

Figures, Plates, Tables, and Boxes

6. Figure 1.1: Biology and classification of tumours. AI-generated using the prompt: "Two-panel diagram: seven-row benign versus malignant comparison table with examples box; tissue of origin classification table (carcinoma, sarcoma, lymphoma, teratoma), suffix guide box, grading box (Grades 1 to 4), and TNM staging diagram (T, N, M with Stage I to IV). Clean clinical surgical diagram style." Suggested OER search string: "tumour biology benign malignant classification TNM staging grading oncology OER."
7. Figure 1.2: Clinical approach to the patient with a tumour. AI-generated using the prompt: "Two-panel diagram: oncological history key points (lump features, associated symptoms, risk factors), structured lump examination sequence, and general examination signs (cachexia, Virchow's node, hepatomegaly); three purposes of investigation box, biopsy types table (FNAC, core needle, incisional, excisional), and staging investigations table (CT, PET-CT, MRI, bone scan, tumour markers). Clean clinical surgical diagram style." Suggested OER search string: "oncological history tumour examination biopsy staging investigations tumour markers OER."
8. Figure 1.3: Skin and soft tissue tumours. AI-generated using the prompt: "Two-panel diagram: benign skin tumours table (sebaceous cyst, lipoma, dermoid, pilomatrixoma, keratoacanthoma, solar keratosis) and malignant skin tumours box (BCC rodent ulcer and melanoma ABCDE and Breslow thickness); common soft tissue sarcoma types table (liposarcoma, leiomyosarcoma, synovial sarcoma, UPS, rhabdomyosarcoma) and clinical features and management box. Clean clinical surgical diagram style." Suggested OER search string: "basal cell carcinoma melanoma ABCDE Breslow soft tissue sarcoma management OER."
9. Figure 1.4: Bone and vascular tumours. AI-generated using the prompt: "Two-panel diagram: primary bone tumours table (osteochondroma, enchondroma, giant cell tumour, osteosarcoma with Codman's triangle and sunburst pattern and treatment) and metastatic bone disease section (BLT kidney prostate primaries, lytic versus sclerotic, presentations, management);

haemangiomas table (capillary, cavernous, port-wine stain), angiosarcoma box (Stewart-Treves), and Kaposi's sarcoma box. Clean clinical surgical diagram style." Suggested OER search string: "osteosarcoma Codman's triangle metastatic bone disease haemangioma angiosarcoma OER."

10. Figure 1.5: Lymphoma and lymph node disease. AI-generated using the prompt: "Two-panel diagram: Hodgkin's versus non-Hodgkin's lymphoma comparison table (RS cell, age, B/T cell, associations, staging, treatment) and Ann Arbor staging diagram; lymphadenopathy cause categories, clinical assessment (localised versus generalised), malignancy red flags box, investigations box, and special nodes box (Virchow's and Sister Mary Joseph's). Clean clinical surgical diagram style." Suggested OER search string: "Hodgkin lymphoma Reed-Sternberg ABVD non-Hodgkin lymphoma R-CHOP lymphadenopathy OER."
11. Figure 1.6: Principles of surgical oncology. AI-generated using the prompt: "Two-panel diagram: R classification box (R0, R1, R2), curative surgery principles (en bloc, clear margins, no-touch, lymph node dissection), and sentinel lymph node biopsy diagram; palliative surgery box (gastrojejunostomy, biliary bypass, stoma, debulking, feeding access), supportive care box (WHO pain ladder, nutrition, SPIKES), and multimodal treatment diagram (neoadjuvant versus adjuvant, targeted therapy, immunotherapy, MDT meeting). Clean clinical surgical diagram style." Suggested OER search string: "surgical oncology R0 resection sentinel lymph node palliative surgery multimodal treatment OER."

Series 2: Ulcers and Gastrointestinal Pathology

1. **Part 2.1: Classification and Pathophysiology of Ulcers**
    1. Sub Part 2.1.1: Definition and classification of ulcers
    2. Sub Part 2.1.2: Pathophysiology of peptic ulcer disease
    3. Sub Part 2.1.3: Pathological features and healing
2. **Part 2.2: Peptic Ulcer Disease: Clinical Features and Complications**
    1. Sub Part 2.2.1: Clinical features of peptic ulcer disease
    2. Sub Part 2.2.2: Complications of peptic ulcer disease
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3. **Part 2.3: Management of Peptic Ulcer Disease**
    1. Sub Part 2.3.1: Medical management
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4. **Part 2.4: Other Gastrointestinal Ulcers and Conditions**
    1. Sub Part 2.4.1: Stress ulcers and Cushing's and Curling's ulcers
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5. **Part 2.5: Gastrointestinal Pathology: Upper GI**
    1. Sub Part 2.5.1: Gastric carcinoma
    2. Sub Part 2.5.2: Oesophageal pathology
    3. Sub Part 2.5.3: Gastrointestinal stromal tumour
6. **Part 2.6: Gastrointestinal Pathology: Lower GI**
    1. Sub Part 2.6.1: Colorectal carcinoma
    2. Sub Part 2.6.2: Inflammatory bowel disease
    3. Sub Part 2.6.3: Appendiceal and anal pathology

Introduction

Ulcers and gastrointestinal pathology represent a broad and clinically important domain in General Surgery II. Peptic ulcer disease (PUD) remains one of the most common surgical conditions, with complications including perforation, haemorrhage, and obstruction that require urgent intervention. Beyond PUD, the gastrointestinal tract is the site of a wide spectrum of pathological conditions, from oesophageal carcinoma to inflammatory bowel disease, each with distinct clinical presentations and management strategies.

This Series covers ulcers in breadth, beginning with classification and pathophysiology, then examining PUD in clinical and management detail, and addressing other important ulcer types including stress ulcers and leg ulcers. We then survey the major pathological conditions of the upper and lower gastrointestinal tract, from gastric carcinoma and GIST to colorectal carcinoma, inflammatory bowel disease, and anorectal conditions.

Series Learning Outcomes

Upon completing this Series, you should be able to:

1. **SLO 2.1:** classify ulcers, describe the pathophysiology of peptic ulcer disease, and describe the pathological features of ulcer healing,
2. **SLO 2.2:** describe the clinical features, complications, and investigation of peptic ulcer disease,
3. **SLO 2.3:** describe the medical, endoscopic, and surgical management of peptic ulcer disease and its complications,
4. **SLO 2.4:** describe the pathophysiology and management of stress ulcers, leg ulcers, and oropharyngeal ulcers,
5. **SLO 2.5:** describe the clinical features and management of gastric carcinoma, oesophageal pathology, and gastrointestinal stromal tumour, and
6. **SLO 2.6:** describe the clinical features and management of colorectal carcinoma, inflammatory bowel disease, and appendiceal and anal pathology.

2.1 Classification and Pathophysiology of Ulcers

2.1.1 Definition and classification of ulcers

An ulcer is a breach in an epithelial surface that fails to heal in the expected time, resulting in tissue loss that extends below the epithelium into the submucosa, muscularis, or deeper layers. Ulcers are classified by aetiology: peptic (from acid and pepsin: gastric and duodenal), ischaemic (from impaired blood supply: arterial leg ulcers), venous (from venous hypertension: venous leg ulcers), neuropathic (from sensory loss: diabetic foot ulcers), traumatic (from mechanical injury), infective (from specific organisms: tuberculosis: Buruli ulcer: tropical ulcer: syphilis), malignant (ulcerating carcinoma), and autoimmune (Crohn's disease: aphthous ulcers).

Ulcers are also classified by their macroscopic appearance: acute ulcers (shallow, punched-out, with little surrounding inflammation: heal quickly when the causative agent is removed) versus chronic ulcers (deeper, with fibrous walls, surrounding induration, and often a necrotic base: take much longer to heal and may require surgical intervention). The floor of an ulcer may be covered by slough (dead tissue and fibrin), granulation tissue (in a healing ulcer), or show bare bone or tendon (in deep ulcers). The edge of an ulcer provides diagnostic clues: undermined edges (tuberculosis), rolled or heaped-up edges (carcinoma), sloping edges (healing: venous ulcer), and punched-out edges (ischaemic ulcer or syphilis).

Fill in the blank: An ulcer is a breach in an _____ surface that extends below the epithelium and fails to heal. Rolled or heaped-up edges of an ulcer suggest _____. Undermined edges suggest _____.

2.1.2 Pathophysiology of peptic ulcer disease

Peptic ulcer disease (PUD) results from an imbalance between aggressive factors (which damage the mucosa) and defensive factors (which protect it). Aggressive factors: hydrochloric acid (secreted by parietal cells in response to gastrin, histamine, and acetylcholine), pepsin (a proteolytic enzyme activated by acid), Helicobacter pylori (a Gram-negative spiral bacterium: colonises the gastric antrum and body: disrupts the mucous layer, stimulates gastrin release, induces mucosal inflammation, and is present in approximately 95 percent of duodenal ulcers and 70 percent of gastric ulcers), and NSAIDs (inhibit COX-1: reduce prostaglandin synthesis: impair mucosal blood flow, bicarbonate secretion, and mucus production).

Defensive factors: mucous gel layer (traps bicarbonate and creates a pH gradient), bicarbonate secretion (by surface epithelial cells: neutralises acid at the mucosal surface), mucosal blood flow (delivers nutrients and removes acid), prostaglandins (stimulate mucus and bicarbonate secretion and maintain mucosal blood flow: PGE2 and PGI2), and epithelial restitution (rapid migration of cells to cover small defects). H. pylori virulence factors include CagA (a cytotoxin-associated gene A protein: injected into epithelial cells: disrupts cell signalling) and VacA (vacuolating cytotoxin: forms channels in the epithelial membrane and induces apoptosis).

Fill in the blank: H. pylori is present in approximately _____ percent of duodenal ulcers. NSAIDs damage the gastric mucosa by inhibiting _____ and reducing _____ synthesis, which impairs mucosal defence. CagA and VacA are _____ virulence factors.

2.1.3 Pathological features and healing

Microscopically, an acute peptic ulcer shows: loss of epithelium down to the muscularis mucosae or deeper, a surface layer of fibrinopurulent exudate, a zone of granulation tissue (new capillaries and fibroblasts), and a zone of fibrous scarring at the base. A chronic peptic ulcer has: a fibrous base (dense collagenous scar replacing the muscularis propria), smooth muscle hypertrophy at the margins, and chronic inflammatory infiltrate (lymphocytes, plasma cells, and macrophages).

Healing of a peptic ulcer occurs in four stages: elimination of the causative agent (H. pylori eradication or NSAID cessation), regeneration of the epithelium (surface epithelial cells migrate to cover the defect: restitution), formation of granulation tissue (filling the ulcer base with new blood vessels and fibroblasts), and scar formation (collagen replaces the granulation tissue: fibrosis). Complete healing may leave a scar that can cause pyloric stenosis (from scarring at the pylorus) or intestinal stenosis (from scarring in the duodenum or jejunum from peptic disease).

Fill in the blank: Microscopy of a chronic peptic ulcer shows a _____ base, smooth muscle _____ at the margins, and a chronic inflammatory infiltrate. Healing of a peptic ulcer begins with elimination of the _____ agent followed by regeneration of the epithelium.

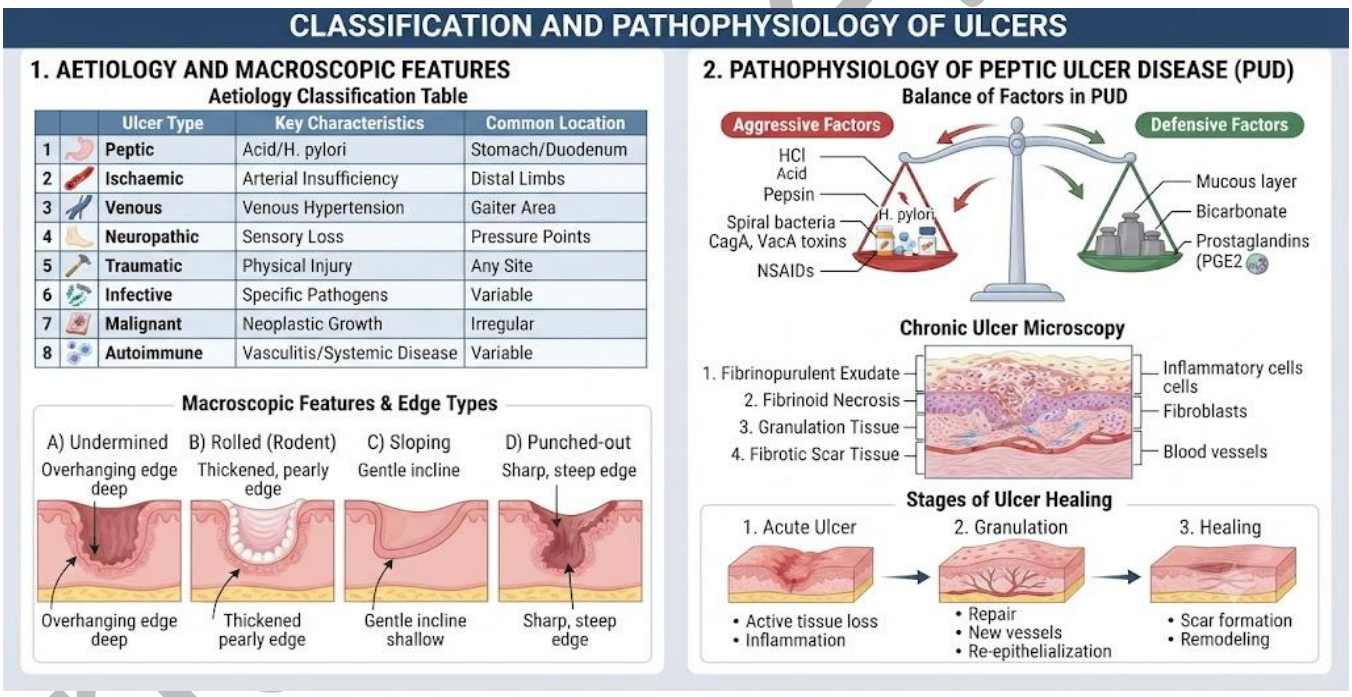

Figure 2.1: Ulcer classification, macroscopic edge types, pathophysiology of PUD, and ulcer healing

Pilot questions 2.1

1. Classify ulcers by aetiology and give an example of each class. (Linked to SLO 2.1)
2. Describe the edge types of ulcers and state the diagnosis each suggests. (Linked to SLO 2.1)

3. Describe the aggressive and defensive factors in peptic ulcer disease. (Linked to SLO 2.1)
4. Describe the role of H. pylori in the pathogenesis of peptic ulcer disease. (Linked to SLO 2.1)
5. Describe the four stages of peptic ulcer healing. (Linked to SLO 2.1)

2.2 Peptic Ulcer Disease: Clinical Features and Complications

2.2.1 Clinical features of peptic ulcer disease

Gastric ulcers present with epigastric pain that is worse after meals (food stimulates acid secretion: aggravates the ulcer) and may be relieved by antacids. The patient may avoid eating due to pain, leading to weight loss. Gastric ulcers are more common in older patients (above 50 years), in women, and in NSAID users. Duodenal ulcers present with epigastric pain that is worse when fasting (2 to 3 hours after meals: the stomach is empty and acid is not buffered by food) and is classically relieved by food or antacids. Duodenal ulcers are more common in young males and are strongly associated with H. pylori (above 95 percent).

Both gastric and duodenal ulcers may be asymptomatic (particularly NSAID-related ulcers: NSAIDs also inhibit the pain response). On examination: epigastric tenderness on palpation is the most common finding. In uncomplicated PUD there are no signs of peritonism. The diagnosis of PUD requires upper GI endoscopy (OGD) for direct visualisation, biopsy (to exclude malignancy in all gastric ulcers and to test for H. pylori: rapid urease test on biopsy), and histological confirmation.

Fill in the blank: Gastric ulcer pain is typically worse _____ meals, while duodenal ulcer pain is worse when _____ (2 to 3 hours after meals). All gastric ulcers require biopsy at OGD to exclude _____.

2.2.2 Complications of peptic ulcer disease

The four major complications of PUD are: haemorrhage (the most common complication: from erosion of a blood vessel in the ulcer base: presents with haematemesis and or melaena: the gastroduodenal artery is most commonly eroded by a posterior duodenal ulcer), perforation (the second most common: sudden severe epigastric pain radiating throughout the abdomen followed by board-like rigidity: generalised peritonitis from gastric acid entering the peritoneal cavity: erect CXR shows free air under the diaphragm in approximately 70 to 80 percent of cases), penetration (the ulcer erodes into an adjacent organ without free perforation: posterior gastric ulcer penetrating into the pancreas causing elevated serum amylase: epigastric pain radiating to the back), and gastric outlet obstruction (from pyloric scarring: projectile non-bilious vomiting: hypochloraemic hypokalaemic metabolic alkalosis from loss of HCl in vomit).

The Forrest classification grades the endoscopic appearance of bleeding peptic ulcers by the risk of re-bleeding: Ia (active arterial spurting: highest risk: 90 percent re-bleeding), Ib (active oozing: 10 to 20 percent), IIa (non-bleeding visible vessel: 50 percent), IIb (adherent clot: 25 to 30 percent), IIc (flat haematin spot: 7 to 10 percent), III (clean ulcer base: lowest risk: 3 to 5 percent re-bleeding). Forrest Ia and IIa require endoscopic haemostasis.

Fill in the blank: The most common complication of PUD is _____ from erosion of a blood vessel. Perforation is confirmed by free air under the _____ on erect CXR (in approximately 70 to 80 percent of cases). Gastric outlet obstruction from pyloric scarring causes a _____ hypokalaemic metabolic alkalosis.

2.2.3 Investigation of peptic ulcer disease

The primary investigation is upper GI endoscopy (OGD): visualises the ulcer, determines its site and size, allows biopsy (mandatory for all gastric ulcers to exclude malignancy; not required for duodenal ulcers which are almost never malignant), and enables endoscopic therapy. H. pylori testing: rapid urease test (CLO test) on antral biopsy (quick and reliable in active disease), histology (most accurate: identifies the organism on biopsy), urea breath test (non-invasive: a radio-labelled urea is ingested: H. pylori urease splits the urea releasing labelled CO2: detected in expired air: used for initial diagnosis and to confirm eradication), and stool antigen test (non-invasive: as accurate as the breath test for eradication confirmation).

Additional investigations: blood tests (FBC: anaemia from chronic blood loss; urea and electrolytes: elevated urea from upper GI bleeding: blood protein digested by gut bacteria; clotting: coagulopathy worsens bleeding), Rockall score (a scoring system to risk-stratify acute upper GI bleeding: combines age, haemodynamic status, comorbidities, and endoscopic findings: total score above 5 predicts high risk of re-bleeding and death), and abdominal X-ray and CT in suspected perforation. Serum gastrin level (fasting) is measured if Zollinger-Ellison syndrome is suspected (ZES: a gastrin-secreting tumour: gastrinoma: of the pancreas or duodenum: causes severe refractory peptic ulceration).

Fill in the blank: All _____ ulcers require biopsy at OGD to exclude malignancy. The urea breath test detects H. pylori by measuring labelled _____ in expired air after ingestion of radio-labelled urea. An elevated fasting serum _____ level suggests Zollinger-Ellison syndrome.

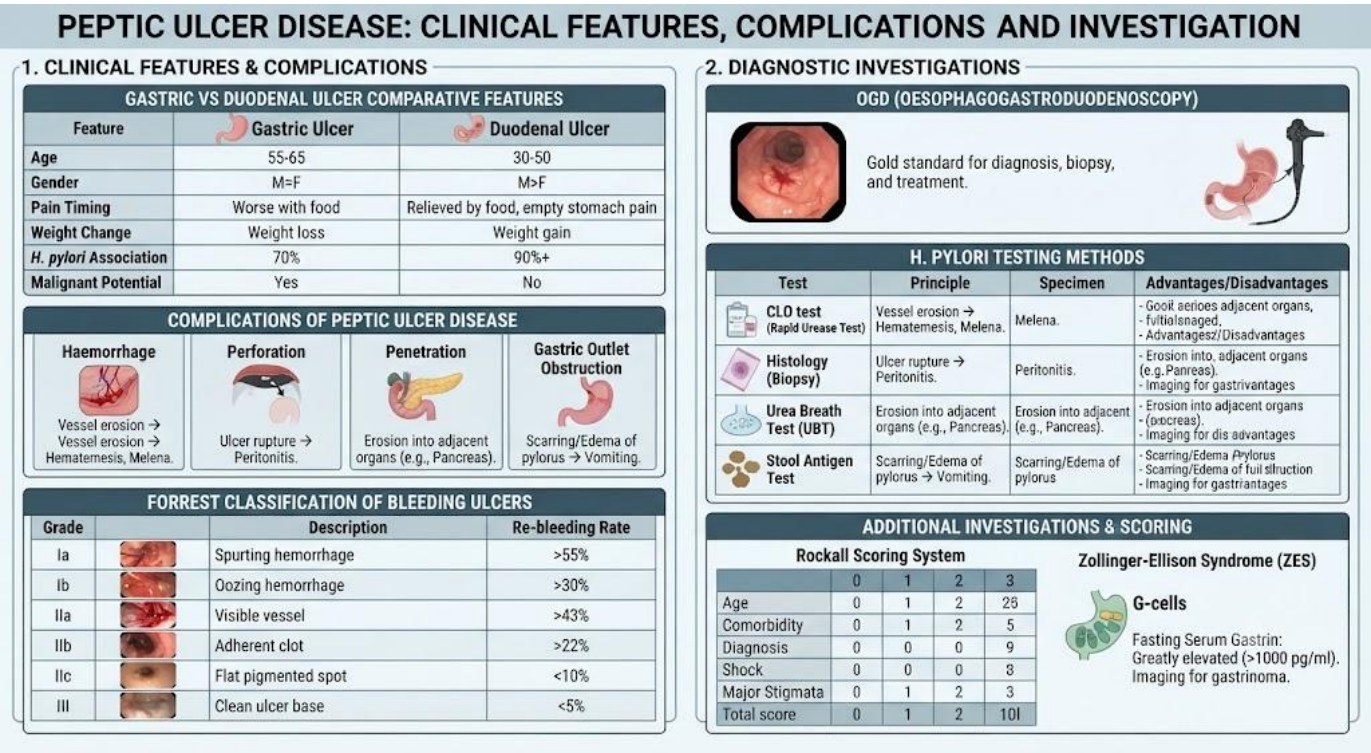

Figure 2.2: Clinical features, complications, Forrest classification, and investigation of peptic ulcer disease

Pilot questions 2.2

- Distinguish the clinical features of gastric ulcer from duodenal ulcer. (Linked to SLO 2.2)
- Describe the four complications of peptic ulcer disease. (Linked to SLO 2.2)
- Describe the Forrest classification and state which grades require endoscopic haemostasis. (Linked to SLO 2.2)
- Describe the H. pylori tests available and state which is used to confirm eradication. (Linked to SLO 2.2)
- Describe the Rockall score and explain its clinical utility. (Linked to SLO 2.2)

2.3 Management of Peptic Ulcer Disease

2.3.1 Medical management

The mainstay of medical management of PUD is acid suppression and H. pylori eradication. Proton pump inhibitors (PPIs: omeprazole, lansoprazole, pantoprazole, rabeprazole, esomeprazole) are the most potent acid suppressants: they irreversibly inhibit the H+/K+ ATPase

(the proton pump) of the parietal cell. H2-receptor antagonists (ranitidine, famotidine) competitively block histamine H2 receptors on the parietal cell: less potent than PPIs. Antacids (magnesium hydroxide, aluminium hydroxide) neutralise gastric acid but provide only temporary symptomatic relief and do not promote healing.

H. pylori eradication is the most important single intervention in PUD caused by H. pylori: eradication heals the ulcer and reduces the recurrence rate from approximately 80 percent per year to less than 5 percent. Standard first-line therapy: triple therapy for 7 to 14 days (a PPI twice daily plus amoxicillin 1 g twice daily plus clarithromycin 500 mg twice daily). Quadruple therapy is used in areas of clarithromycin resistance or for second-line treatment (bismuth-containing quadruple therapy: PPI plus bismuth plus metronidazole plus tetracycline). Confirmation of eradication: urea breath test or stool antigen test at least 4 weeks after completing therapy (and 2 weeks after stopping PPIs).

Fill in the blank: PPIs irreversibly inhibit the _____ ATPase (the proton pump) of the parietal cell. H. pylori triple therapy uses a PPI plus amoxicillin plus _____. Eradication of H. pylori reduces the annual ulcer recurrence rate from approximately _____ percent to less than 5 percent.

2.3.2 Endoscopic management

Endoscopic management is the primary treatment for the complications of PUD: acute upper GI bleeding (Forrest Ia and IIa) and occasionally penetration. Endoscopic haemostasis techniques: adrenaline injection (1:10,000 adrenaline injected into the ulcer base: causes vasoconstriction and tamponade: rapidly achieves initial haemostasis but high re-bleeding rate when used alone), thermal coagulation (heater probe or bipolar electrocautery: coagulates the bleeding vessel), mechanical haemostasis (endoscopic clips: applied across the bleeding vessel: the most effective technique for achieving durable haemostasis), and combination therapy (adrenaline injection plus mechanical or thermal haemostasis: superior to any single modality).

Indications for endoscopy in acute upper GI bleeding: all patients with significant upper GI bleeding (haematemesis, melaena, or haematochezia with haemodynamic instability suggesting an upper GI source) should undergo endoscopy within 24 hours of resuscitation (within 12 hours for high-risk patients: Rockall score above 5, haemodynamic instability despite resuscitation). Endoscopy also allows risk stratification (Forrest classification) to guide further management (low-risk Forrest IIc and III: can be discharged early; high-risk Forrest Ia and IIa: require intensive monitoring and early re-endoscopy if re-bleeding occurs).

Fill in the blank: Endoscopic _____ injection provides initial haemostasis by vasoconstriction but has a high re-bleeding rate when used alone. _____ clips (applied across the bleeding vessel) provide the most effective durable endoscopic haemostasis. High-risk patients (Rockall above _____) should undergo endoscopy within 12 hours.

2.3.3 Surgical management

Surgery for PUD is now required much less commonly since the introduction of PPIs and H. pylori eradication. Indications for surgery in PUD: failed endoscopic haemostasis or re-bleeding after two endoscopic attempts (emergency surgery for bleeding PUD: under-running of the bleeding vessel at operation), perforation (emergency surgery: Graham patch closure of the perforation with omental patch reinforcement and peritoneal lavage), gastric outlet obstruction (elective surgery after medical treatment has failed: gastrojejunostomy or pyloroplasty), and suspicion of malignancy in a non-healing gastric ulcer.

Emergency surgery for perforated peptic ulcer: the patient is resuscitated with IV fluids and IV antibiotics (covering aerobes and anaerobes), a nasogastric tube is passed, and the patient is taken to the operating theatre. At laparotomy: the perforation is identified (most commonly on the anterior wall of the first part of the duodenum or the lesser curve of the stomach), closed with interrupted absorbable sutures, reinforced with an omental patch (Graham patch), and the peritoneal cavity is lavaged with warm saline. Definitive acid-reducing surgery (vagotomy) is rarely performed today as H. pylori eradication and PPIs are curative in the long term.

Fill in the blank: Graham _____ closure reinforces the sutured perforation with an omental patch. Emergency surgery for a bleeding duodenal ulcer involves _____ (suture ligation) of the bleeding vessel. Definitive acid-reducing surgery (vagotomy) is rarely performed today because _____ eradication and PPIs are curative.

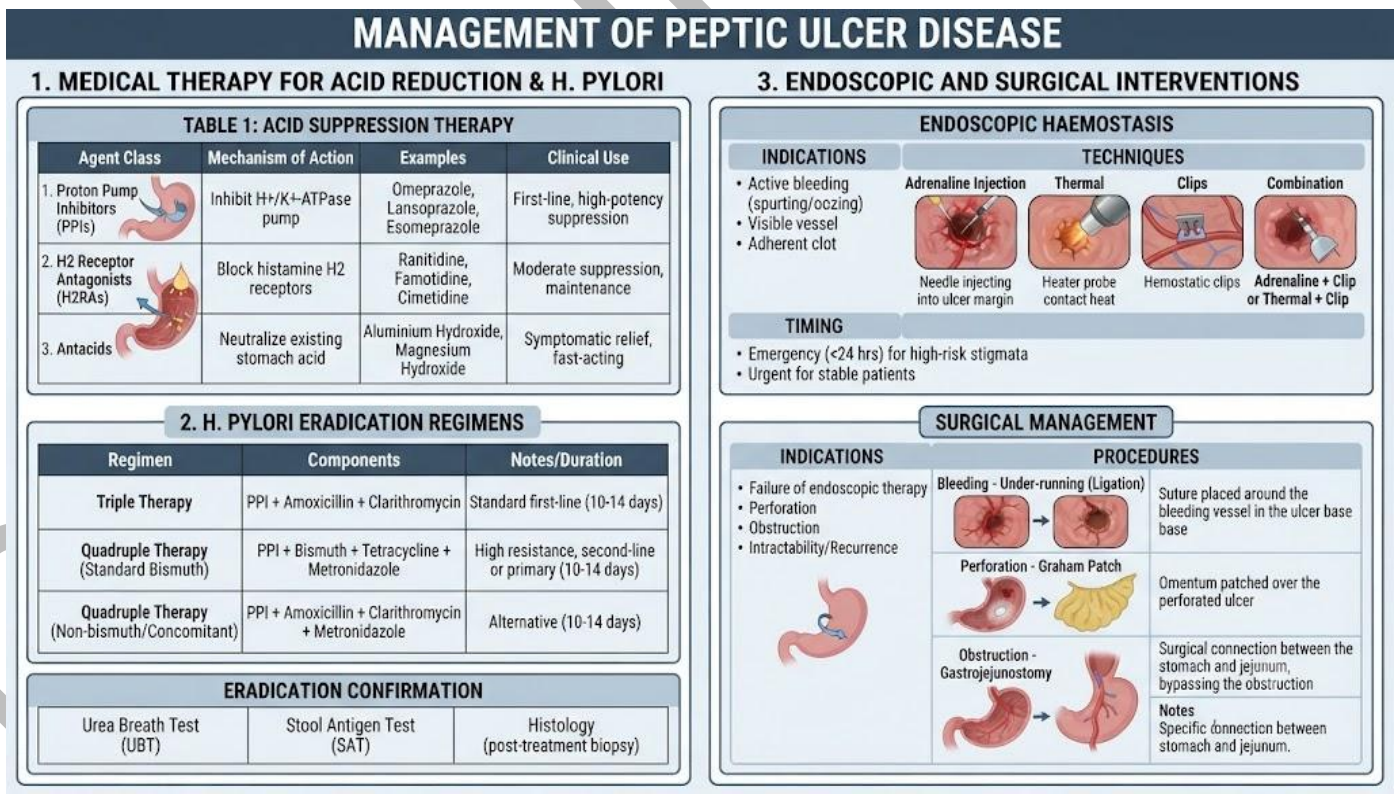

Figure 2.3: Medical management, H. pylori eradication, endoscopic haemostasis, and surgical management of PUD

Pilot questions 2.3

1. Describe the mechanism of action of PPIs and distinguish them from H2-receptor antagonists. (Linked to SLO 2.3)
2. Describe the first-line H. pylori eradication regimen and explain why eradication is the most important intervention. (Linked to SLO 2.3)
3. Describe the endoscopic techniques for haemostasis in a bleeding peptic ulcer. (Linked to SLO 2.3)
4. Describe the surgical management of a perforated peptic ulcer. (Linked to SLO 2.3)
5. State the indications for surgery in peptic ulcer disease. (Linked to SLO 2.3)

2.4 Other Gastrointestinal Ulcers and Conditions

2.4.1 Stress ulcers and Cushing's and Curling's ulcers

Stress ulcers are acute mucosal erosions or ulcerations that develop in critically ill patients (in intensive care: mechanical ventilation, sepsis, major burns, and major trauma are the most common precipitants). The mechanism involves splanchnic vasoconstriction (from the stress response: reduces mucosal blood flow and impairs the mucosal barrier: without adequate mucosal perfusion, the defensive mechanisms fail and acid erodes the mucosa). Stress ulcers are typically multiple, shallow, involving the gastric body and fundus, and are usually clinically silent until they bleed.

Curling's ulcer is a stress ulcer occurring specifically in patients with major burns (above 35 percent total body surface area burns): caused by hypovolaemia and splanchnic hypoperfusion. Cushing's ulcer is a deep, single ulcer in the stomach, duodenum, or oesophagus, occurring in patients with head injuries, brain tumours, or neurosurgical procedures: caused by direct vagal stimulation of parietal cells by the central nervous system, resulting in massive hypersecretion of gastric acid. Prevention of stress ulcers: IV PPIs or H2-receptor antagonists in all critically ill patients on mechanical ventilation or with coagulopathy. Treatment: IV PPI infusion; endoscopic haemostasis for active bleeding; surgery as a last resort.

Fill in the blank: Curling's ulcer is a stress ulcer in patients with major _____ (above 35 percent TBSA). Cushing's ulcer occurs in patients with _____ injuries and is caused by vagal stimulation of parietal cells causing acid _____. Stress ulcer _____ with IV PPIs or H2 blockers is given to all critically ill patients on mechanical ventilation.

2.4.2 Leg ulcers

Leg ulcers are chronic ulcerations on the lower limb below the knee. The three main types are: venous ulcers (the most common: 70 percent of all leg ulcers: from chronic venous hypertension caused by deep vein incompetence or superficial varicose veins: typically in the gaiter area: just above the medial malleolus: shallow, irregular, with sloping edges and a granulating base, surrounded by lipodermatosclerosis and haemosiderin staining), arterial ulcers (from peripheral arterial disease: punched-out, deep, with pale necrotic base, on the toes, heel, or pressure points: associated with absent peripheral pulses and claudication), and neuropathic ulcers (from sensory neuropathy: diabetic foot: painless deep ulcers under pressure points: typically the heel, first metatarsal head, and toes).

Investigations for leg ulcers: ABPI (ankle-brachial pressure index: the ratio of the ankle systolic pressure to the brachial systolic pressure: normal above 0.9; below 0.8 suggests peripheral arterial disease; below 0.5 indicates critical ischaemia: compression bandaging is contraindicated if ABPI is below 0.8 as it worsens arterial flow), duplex ultrasound (assesses venous and arterial insufficiency), blood tests (FBC, HbA1c, lipids, ESR, wound swab for culture). Management: venous ulcers: compression bandaging (four-layer: reduces venous hypertension: the cornerstone of treatment), wound care, and treatment of underlying varicose veins. Arterial ulcers: vascular reconstruction (angioplasty or bypass). Neuropathic ulcers: debridement, off-loading, glycaemic control, and wound care.

Fill in the blank: Venous ulcers are most commonly found in the _____ area (just above the medial malleolus). Compression bandaging for venous ulcers is contraindicated if the ABPI is below _____. Neuropathic (diabetic) ulcers are _____ (the patient feels no pain) and occur at pressure points.

2.4.3 Oral and oropharyngeal ulcers

Aphthous ulcers (canker sores) are the most common oral ulcers: shallow, round or oval, with a yellow-grey base and red erythematous halo, typically on the non-keratinised mucosa (buccal mucosa, floor of mouth, and lateral tongue). They are recurrent, painful, and of uncertain aetiology (possible immunological, stress-related, or nutritional: iron, folate, or vitamin B12 deficiency). Most heal spontaneously in 7 to 14 days. Severe recurrent aphthous ulcers are treated with topical corticosteroids (triamcinolone acetonide in orabase), topical anaesthetics (lidocaine gel), or systemic immunosuppressants in severe cases.

Oral carcinoma (squamous cell carcinoma of the oral cavity) presents as a non-healing ulcer in the oral cavity (most commonly the lateral border of the tongue, floor of the mouth, or buccal mucosa), typically in patients who smoke and drink alcohol (the two most important risk factors: together they have a synergistic effect). It may also present as a red (erythroplakia) or white (leukoplakia) patch: erythroplakia has the highest risk of malignant transformation (above 40 percent). Any oral ulcer that fails to heal after 3 weeks must be biopsied to exclude malignancy. Management: surgical excision with reconstruction, radiotherapy, and chemotherapy depending on stage.

Fill in the blank: Aphthous ulcers are most common on _____ mucosa (buccal mucosa and floor of mouth). Any oral ulcer failing to heal after _____ weeks must be biopsied to exclude malignancy. The two most important risk factors for oral carcinoma are _____ and alcohol.

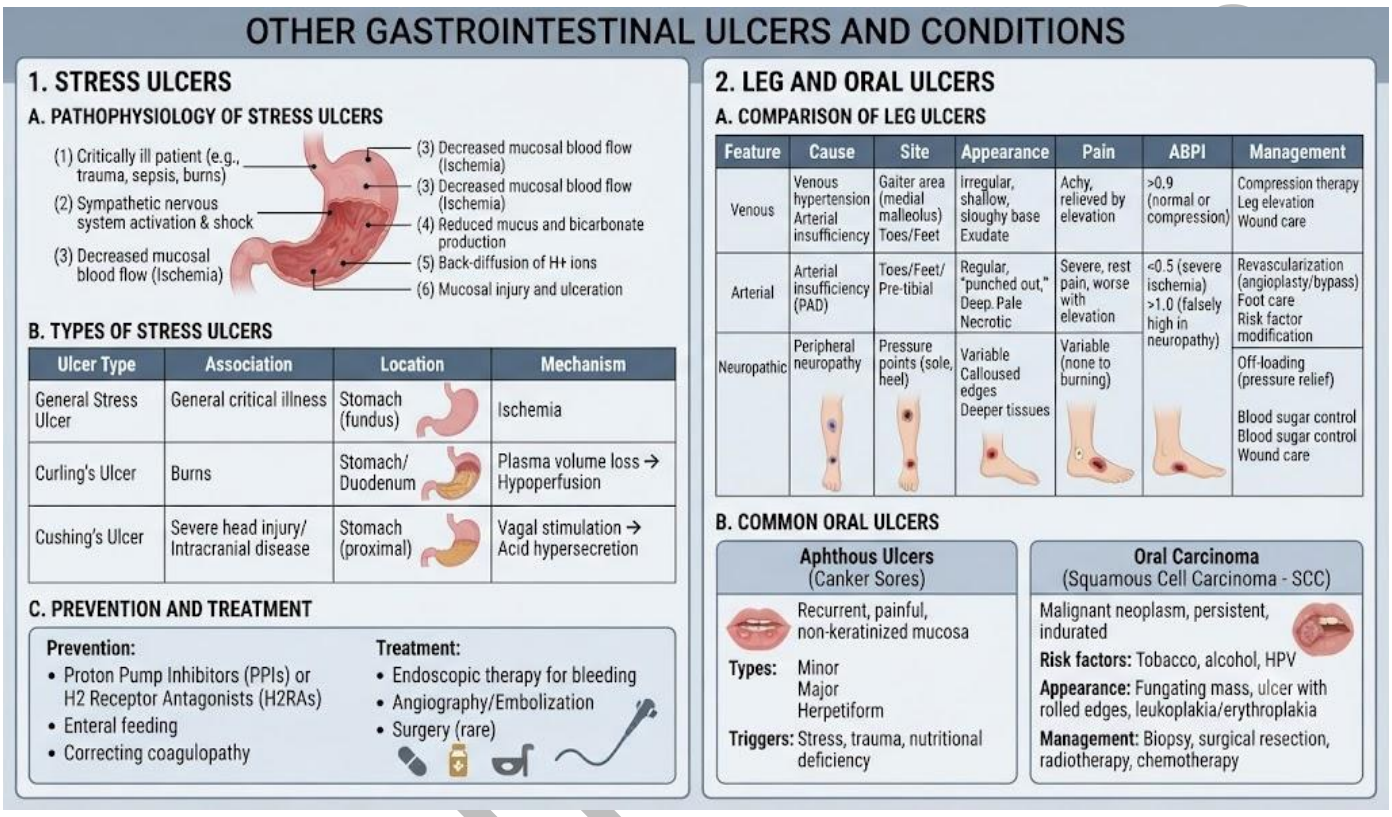

Figure 2.4: Stress ulcers (Curling's and Cushing's), leg ulcer comparison, and oral ulcers

Pilot questions 2.4

6. Describe the pathophysiology of stress ulcers and state who is at highest risk. (Linked to SLO 2.4)
7. Distinguish Curling's from Cushing's ulcer. (Linked to SLO 2.4)
8. Compare venous, arterial, and neuropathic leg ulcers in terms of aetiology, site, appearance, and management. (Linked to SLO 2.4)
9. Explain the significance of ABPI in leg ulcer management. (Linked to SLO 2.4)
10. Describe the clinical features of oral carcinoma and state the rule for biopsy. (Linked to SLO 2.4)

2.5 Gastrointestinal Pathology: Upper GI

2.5.1 Gastric carcinoma

Gastric carcinoma is the most common GI malignancy in Nigeria and one of the most common cancers worldwide. Risk factors: H. pylori infection (the most important modifiable risk factor: causes chronic atrophic gastritis, intestinal metaplasia, and dysplasia: the Correa cascade), dietary factors (smoked, salted, and pickled foods: high salt: nitrosamines), smoking, chronic atrophic gastritis, pernicious anaemia (autoimmune gastritis), previous partial gastrectomy, and family history (hereditary diffuse gastric cancer: CDH1 mutation: E-cadherin gene).

Clinical features: insidious onset with epigastric pain, anorexia, weight loss, and early satiety. Advanced signs: Virchow's node (left supraclavicular lymphadenopathy), Sister Mary Joseph's nodule (periumbilical), Blumer's shelf (mass felt on rectal examination from tumour seeded in the pouch of Douglas), and Krukenberg tumour (ovarian metastasis from transcoelomic spread of mucin-secreting gastric cancer). Staging: OGD with biopsy, CT staging, laparoscopy (to detect peritoneal disease not visible on CT). Treatment: curative radical gastrectomy (subtotal or total) with D2 lymph node dissection; perioperative chemotherapy (FLOT: 5-FU, leucovorin, oxaliplatin, and docetaxel: now the standard perioperative chemotherapy in the UK and increasingly used in Nigerian tertiary centres).

Fill in the blank: The most important modifiable risk factor for gastric carcinoma is _____ infection. Blumer's shelf is a mass felt on _____ examination from tumour seeding in the pouch of Douglas. Perioperative _____ chemotherapy (FLOT) is the standard treatment in combination with radical gastrectomy.

2.5.2 Oesophageal pathology

Oesophageal carcinoma presents with progressive dysphagia (for solids first, then liquids: a red-flag symptom in any adult above 50 years requiring urgent OGD). Two main types: squamous cell carcinoma (upper and middle thirds: associated with smoking, alcohol, and achalasia) and adenocarcinoma (lower third and oesophagogastric junction: arising from Barrett's oesophagus from chronic GORD). Barrett's oesophagus (columnar metaplasia of the lower oesophageal mucosa from chronic acid exposure) is the most important precancerous condition: risk of progression to adenocarcinoma approximately 0.5 percent per year; managed by PPI therapy and endoscopic surveillance.

Other oesophageal conditions: achalasia (failure of relaxation of the lower oesophageal sphincter and absence of peristalsis: caused by degeneration of the Auerbach's plexus: presents with progressive dysphagia and regurgitation of undigested food and nocturnal aspiration: barium swallow shows a bird-beak or rat-tail appearance: treated by Heller's cardiomyotomy or pneumatic dilatation), oesophageal perforation (Boerhaave's syndrome: spontaneous full-thickness tear of the oesophagus from sudden increase in intra-oesophageal pressure from forceful vomiting: severe chest pain and surgical emphysema: extremely high mortality: emergency surgery), and pharyngeal pouch (Zenker's diverticulum: pulsion diverticulum from

the posterior hypopharynx through Killian's dehiscence: presents with dysphagia and regurgitation of undigested food and aspiration: treated by endoscopic or open surgical excision).

Fill in the blank: Barrett's oesophagus is a risk of progression to _____ adenocarcinoma of approximately 0.5 percent per year and is managed by PPI and endoscopic _____. Achalasia shows a _____ or rat-tail appearance on barium swallow and is treated by Heller's cardiomyotomy. Boerhaave's syndrome is spontaneous _____ perforation from forceful vomiting.

2.5.3 Gastrointestinal stromal tumour

Gastrointestinal stromal tumour (GIST) is the most common mesenchymal tumour of the GI tract, arising from the interstitial cells of Cajal (the pacemaker cells of the gut: located in the myenteric plexus). The stomach is the most common site (approximately 60 percent), followed by the small intestine (25 percent). Histologically: spindle cell or epithelioid morphology with positive immunohistochemical staining for CD117 (c-KIT: the tyrosine kinase receptor encoded by the KIT gene: the defining marker of GIST) and DOG1. Approximately 85 percent of GISTs have activating mutations in the KIT gene; approximately 10 percent have PDGFRA mutations.

Clinical features: abdominal pain, GI bleeding (haematemesis or melaena from mucosal ulceration), a palpable abdominal mass, or incidental finding on imaging. Risk stratification (by mitotic count, size, and site) determines prognosis and guides treatment. Management: surgical resection with clear margins (for localised resectable GISTs: R0 resection is the goal: laparoscopic or open depending on site and size), and imatinib (Gleevec: a tyrosine kinase inhibitor targeting c-KIT: for unresectable, metastatic, or high-risk resected GISTs: also used as neoadjuvant therapy to shrink large GISTs before surgery). Imatinib has transformed the treatment of GIST: 5-year survival for resected primary GIST above 90 percent.

Fill in the blank: GISTs arise from the interstitial cells of _____ and are the most common mesenchymal tumour of the GI tract. The defining immunohistochemical marker of GIST is _____ (c-KIT). _____ (Gleevec) is a tyrosine kinase inhibitor used for unresectable or metastatic GIST.

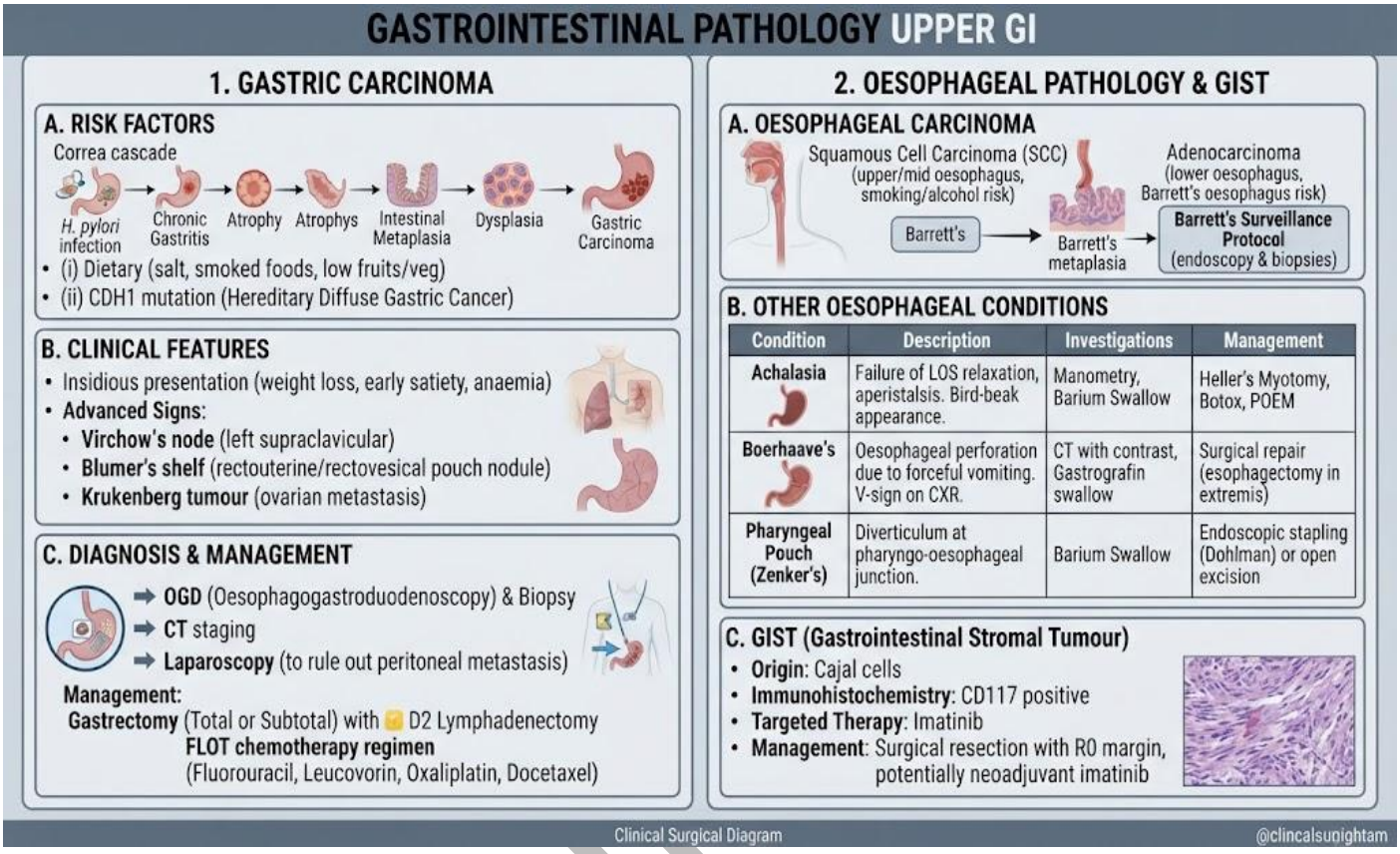

Figure 2.5: Gastric carcinoma, oesophageal pathology, and gastrointestinal stromal tumour

Pilot questions 2.5

11. Describe the clinical features of gastric carcinoma and name four signs of advanced disease. (Linked to SLO 2.5)
12. Describe the Correa cascade and explain how H. pylori causes gastric carcinoma. (Linked to SLO 2.5)
13. Distinguish squamous cell carcinoma from adenocarcinoma of the oesophagus. (Linked to SLO 2.5)
14. Describe the clinical features and treatment of achalasia. (Linked to SLO 2.5)
15. Describe the clinical features and management of GIST. (Linked to SLO 2.5)

2.6 Gastrointestinal Pathology: Lower GI

2.6.1 Colorectal carcinoma

Colorectal carcinoma (CRC) is the most common GI malignancy in high-income countries and is increasing in Nigeria. Risk factors: age (above 50 years), dietary factors (low fibre, high red meat and processed meat), inflammatory bowel disease (Crohn's disease and ulcerative colitis: 10-year

cumulative risk of CRC increases with extent and duration of IBD), genetic syndromes (FAP: familial adenomatous polyposis: APC gene mutation: hundreds of adenomatous polyps: 100 percent risk of CRC if untreated; Lynch syndrome: HNPCC: MLH1 and MSH2 mismatch repair gene mutations: lifetime CRC risk 40 to 80 percent), and previous adenomatous polyps.

Clinical features by site: right-sided CRC (insidious: iron deficiency anaemia from occult blood loss; vague right iliac fossa discomfort; palpable mass; weight loss), left-sided CRC (change in bowel habit: alternating constipation and diarrhoea; bright red or mixed rectal bleeding; progressive large bowel obstruction: the narrower left colon obstructs earlier), rectal CRC (tenesmus: sensation of incomplete evacuation; blood and mucus per rectum; alteration in stool calibre; late: palpable rectal mass on PR examination). Staging: CT of chest, abdomen, and pelvis; MRI rectum for rectal cancer (defines the circumferential resection margin). Surgery: right hemicolectomy for right CRC; sigmoid colectomy or anterior resection for sigmoid and upper rectal CRC; low anterior resection or abdominoperineal resection (APR) for lower rectal CRC with total mesorectal excision (TME).

Fill in the blank: Right-sided CRC typically presents with iron deficiency _____ from occult blood loss. FAP is caused by a mutation in the _____ gene and carries a _____ percent risk of CRC if untreated. The gold standard local staging investigation for rectal cancer is _____ rectum.

2.6.2 Inflammatory bowel disease

Ulcerative colitis (UC) is a chronic relapsing inflammatory disease of the colonic mucosa, beginning at the rectum and extending proximally in a continuous and confluent pattern (no skip lesions). Histologically: superficial mucosal inflammation, crypt abscesses, goblet cell depletion, and pseudopolyps (islands of surviving mucosa in a background of denuded inflamed mucosa). Complications: toxic megacolon (acute dilatation of the colon above 6 cm on AXR: risk of perforation: medical emergency treated with IV corticosteroids and IV fluids: if no improvement in 72 hours: emergency colectomy), haemorrhage, stricture, and colorectal carcinoma (risk increases with extent and duration of disease: pancolitis for more than 10 years has a significant cancer risk).

Crohn's disease (CD) is a chronic transmural inflammatory disease that can affect any part of the GI tract from mouth to anus, most commonly the terminal ileum and colon. It is characterised by skip lesions (areas of normal bowel between areas of disease), transmural inflammation (full thickness: leads to fistulae, strictures, and abscesses), non-caseating granulomas on histology, and a cobblestone appearance of the mucosa on endoscopy (islands of oedematous mucosa separated by deep linear ulcers). Complications: strictures (causing obstruction), fistulae (enteroenteral, enterovesical, enterocutaneous, enterovaginal), abscesses (perianal abscess most common in CD), and perianal disease (fissures, fistulae, and skin tags).

Fill in the blank: Ulcerative colitis begins at the _____ and extends proximally in a _____ pattern (no skip lesions). Crohn's disease is characterised by _____ lesions, transmural inflammation, and non-caseating _____ on histology.

2.6.3 Appendiceal and anal pathology

Acute appendicitis is the most common surgical emergency in young adults. It results from obstruction of the appendiceal lumen (faecolith, lymphoid hyperplasia, or rarely carcinoid tumour), followed by bacterial overgrowth, inflammation, ischaemia, and perforation. Classic presentation: central colicky pain migrating to the right iliac fossa (McBurney's point), anorexia, nausea, low-grade fever, and localised guarding. Investigations: raised WBC and CRP, ultrasound (non-compressible appendix above 6 mm), CT abdomen and pelvis (most accurate). Alvarado score (a clinical scoring tool using symptoms, signs, and lab findings: score above 7 suggests appendicitis). Treatment: laparoscopic or open appendicectomy; IV antibiotics for perforated appendicitis.

Anal conditions: haemorrhoids (dilated submucosal vascular cushions: classified first to fourth degree by prolapse: treated by lifestyle advice, rubber band ligation, or haemorrhoidectomy for third and fourth degree), anal fissure (linear tear distal to the dentate line in the posterior midline: severe pain on defaecation: treated with topical GTN or diltiazem or botulinum toxin or lateral internal sphincterotomy for chronic fissure), perianal abscess (incision and drainage under GA), and anal fistula (a tract connecting the anorectal lumen to the perianal skin: classified by Parks classification relative to the external anal sphincter: fistulotomy for superficial fistulae; seton suture for high fistulae to preserve continence). Colorectal carcinoid tumours (neuroendocrine tumours: most commonly in the appendix: appendiceal carcinoid below 2 cm is treated by appendicectomy alone; above 2 cm requires right hemicolectomy).

Fill in the blank: Acute appendicitis results from obstruction of the appendiceal lumen followed by bacterial overgrowth, inflammation, ischaemia, and _____. An Alvarado score above _____ suggests appendicitis. Haemorrhoids are classified from first to _____ degree by the degree of prolapse.

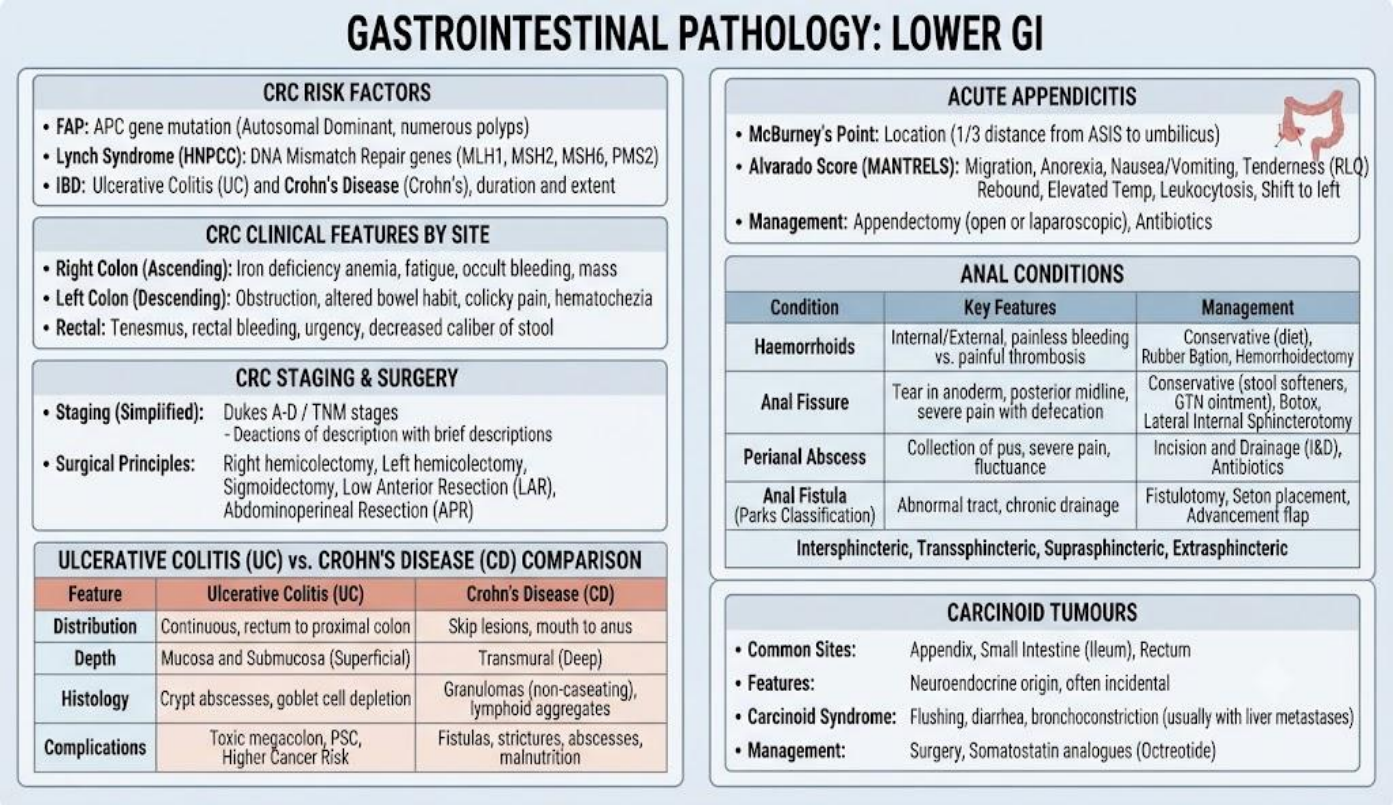

Figure 2.6: Colorectal carcinoma, inflammatory bowel disease comparison, and appendiceal and anal pathology

Pilot questions 2.6

1. Describe the clinical presentation of colorectal carcinoma by tumour site. (Linked to SLO 2.6)
2. Compare ulcerative colitis and Crohn's disease in terms of distribution, depth, histology, and complications. (Linked to SLO 2.6)
3. Describe the classic presentation of acute appendicitis and name the scoring system used to assess it. (Linked to SLO 2.6)
4. Classify haemorrhoids by degree and describe the management of each grade. (Linked to SLO 2.6)
5. Describe the Parks classification of anal fistulae and explain the surgical principle of high fistula management. (Linked to SLO 2.6)

Series Summary

This Series covered ulcers and gastrointestinal pathology in breadth. We classified ulcers by aetiology and macroscopic features, then described the pathophysiology of PUD (aggressive versus defensive factors: H. pylori and NSAIDs), the clinical features and complications of PUD (haemorrhage: Forrest classification; perforation: free air on CXR; gastric outlet obstruction: metabolic alkalosis), and the medical (PPIs and H. pylori triple therapy), endoscopic (clips and thermal therapy), and surgical (Graham patch; under-running) management. We then covered stress ulcers (Curling's: burns; Cushing's: head injury), leg ulcers (venous: gaiter area and compression; arterial: ABPI below 0.8; neuropathic: painless and pressure points), and oral carcinoma (3-week rule for biopsy). Upper GI pathology covered gastric carcinoma (Correa cascade; Virchow's node; D2 gastrectomy; FLOT), oesophageal pathology (Barrett's; SCC versus adenocarcinoma; achalasia; Boerhaave's), and GIST (c-KIT; imatinib). Lower GI pathology covered CRC (FAP and Lynch; TME for rectal), IBD (UC: continuous mucosal; CD: skip transmural granulomatous), and anal conditions.

You should now be able to classify ulcers and describe PUD pathophysiology (SLO 2.1), describe PUD clinical features, complications, and investigations (SLO 2.2), describe PUD management (SLO 2.3), describe stress ulcers, leg ulcers, and oral ulcers (SLO 2.4), describe upper GI pathology (SLO 2.5), and describe lower GI pathology (SLO 2.6).

Glossary of Terms

6. **Alvarado score:** a clinical scoring system for acute appendicitis using migration of pain, anorexia, nausea, right iliac fossa tenderness, rebound tenderness, elevated temperature, leucocytosis, and left shift; a score above 7 suggests appendicitis.
7. **Barrett's oesophagus:** replacement of the normal squamous epithelium of the lower oesophagus by columnar (intestinal-type) epithelium from chronic acid reflux; a pre-malignant condition with approximately 0.5 percent per year risk of oesophageal adenocarcinoma.
8. **Blumer's shelf:** a mass felt on rectal examination in the pouch of Douglas, caused by transcoelomic seeding of gastric or colorectal carcinoma cells into the rectovesical or rectouterine pouch; indicates peritoneal metastasis.
9. **Boerhaave's syndrome:** spontaneous full-thickness perforation of the oesophagus from a sudden increase in intra-oesophageal pressure during forceful vomiting; presents with severe chest and upper abdominal pain and surgical emphysema; high mortality without emergency surgery.

10. **Correa cascade:** the sequence of histological changes in the gastric mucosa leading to intestinal-type gastric carcinoma: normal mucosa to chronic atrophic gastritis to intestinal metaplasia to dysplasia to carcinoma; largely driven by H. pylori infection.
11. **Curling's ulcer:** a stress ulcer occurring in patients with major burns (above 35 percent total body surface area); caused by hypovolaemia and splanchnic hypoperfusion reducing mucosal barrier function.
12. **Cushing's ulcer:** a deep, single peptic ulcer of the stomach, duodenum, or oesophagus occurring in patients with head injuries, brain tumours, or after neurosurgical procedures; caused by direct vagal stimulation of parietal cells causing massive acid hypersecretion.
13. **Forrest classification:** an endoscopic classification of bleeding peptic ulcers by risk of re-bleeding: Ia (active spurting: 90 percent), IIa (visible vessel: 50 percent), IIc (flat spot: 7 percent), III (clean base: 3 percent); Ia and IIa require endoscopic haemostasis.
14. **Graham patch closure:** surgical repair of a perforated peptic ulcer: the perforation is closed with interrupted absorbable sutures and reinforced with a pedicle of omentum sutured over it.
15. **GIST (gastrointestinal stromal tumour):** the most common mesenchymal tumour of the GI tract; arises from interstitial cells of Cajal; characterised by CD117 (c-KIT) positivity; treated by R0 surgical resection and imatinib (a tyrosine kinase inhibitor targeting c-KIT).
16. **Krukenberg tumour:** ovarian metastasis from transcoelomic spread of mucin-secreting gastric carcinoma (or other GI cancers); typically bilateral; histologically shows signet ring cells in the ovarian stroma.
17. **Toxic megacolon:** acute dilatation of the colon above 6 cm diameter (seen on plain AXR) complicating severe ulcerative colitis or Crohn's disease; risk of perforation; medical emergency treated with IV corticosteroids; emergency colectomy if no improvement in 72 hours.

Concept Check Questions [Series 2]

Objective questions

11. Rolled or heaped-up edges of an ulcer suggest _____, while undermined edges suggest tuberculosis. (Linked to SLO 2.1)

12. H. pylori is present in approximately _____ percent of duodenal ulcers. NSAIDs damage the gastric mucosa by inhibiting _____ and reducing prostaglandin synthesis. (Linked to SLO 2.1)
13. The most common complication of peptic ulcer disease is _____ from erosion of a blood vessel in the ulcer base. (Linked to SLO 2.2)
14. The defining immunohistochemical marker of GIST is _____ (c-KIT), and imatinib is a tyrosine kinase inhibitor that targets this receptor. (Linked to SLO 2.5)
15. Ulcerative colitis begins at the _____ and extends proximally without skip lesions, while Crohn's disease is characterised by _____ lesions and transmural inflammation. (Linked to SLO 2.6)

Short-answer questions

1. Describe the aggressive and defensive factors in peptic ulcer disease. (Linked to SLO 2.1)
2. Describe the Forrest classification and state which grades require endoscopic haemostasis. (Linked to SLO 2.2)
3. Describe the first-line H. pylori eradication regimen and explain how eradication is confirmed. (Linked to SLO 2.3)
4. Compare venous, arterial, and neuropathic leg ulcers in terms of site and management. (Linked to SLO 2.4)
5. Describe the Correa cascade and explain how H. pylori causes gastric carcinoma. (Linked to SLO 2.5)

Long-answer questions

1. Describe the clinical features, complications, and investigation of peptic ulcer disease. (Linked to SLO 2.2)
2. Describe the medical and surgical management of a perforated peptic ulcer. (Linked to SLO 2.3)
3. Compare ulcerative colitis and Crohn's disease in terms of distribution, histology, and complications. (Linked to SLO 2.6)

4. Describe the clinical features, staging, and surgical management of colorectal carcinoma. (Linked to SLO 2.6)
5. Describe the clinical approach to a patient with acute upper GI bleeding including risk stratification. (Linked to SLO 2.2 and 2.3)

Answers to Concept Check Questions [Series 2]

Objective questions

1. Carcinoma (malignant ulcer).
2. 95; COX-1.
3. Haemorrhage.
4. CD117.
5. Rectum; skip.

Short-answer questions

6. Aggressive factors: HCl (parietal cells), pepsin (proteolytic), H. pylori (disrupts mucous layer, stimulates gastrin, induces inflammation), NSAIDs (inhibit COX-1, reduce prostaglandins, impair mucosal blood flow and bicarbonate). Defensive factors: mucous gel layer (traps bicarbonate, pH gradient), bicarbonate secretion, mucosal blood flow, prostaglandins (PGE2 and PGI2: stimulate mucus and bicarbonate and blood flow), and epithelial restitution.
7. Forrest Ia: active arterial spurting: 90 percent re-bleeding risk. Ib: active oozing: 10 to 20 percent. IIa: non-bleeding visible vessel: 50 percent. IIb: adherent clot: 25 to 30 percent. IIc: flat haematin spot: 7 to 10 percent. III: clean ulcer base: 3 to 5 percent. Forrest Ia and IIa require endoscopic haemostasis (these have re-bleeding risks above 50 percent and endoscopic treatment significantly reduces re-bleeding and mortality).
8. First-line triple therapy for 7 to 14 days: PPI twice daily (omeprazole 20 mg or equivalent) plus amoxicillin 1 g twice daily plus clarithromycin 500 mg twice daily. Confirmation of eradication: urea breath test or stool antigen test, performed at least 4 weeks after completing antibiotic therapy and at least 2 weeks after stopping PPIs (PPIs suppress H. pylori without eradicating it and can give false-negative results if the test is done while still taking them).
9. Venous ulcers: above medial malleolus (gaiter area); managed with four-layer compression bandaging (after confirming ABPI above 0.8) and wound care. Arterial ulcers: toes, heel, and pressure points; managed with vascular reconstruction (angioplasty or bypass): compression is contraindicated (ABPI below 0.8). Neuropathic ulcers: pressure points (heel, first metatarsal head); managed with debridement, off-loading (special footwear or total contact cast), and glycaemic control.
10. The Correa cascade is the sequence of histological changes from normal gastric mucosa to intestinal-type gastric carcinoma: normal mucosa to H. pylori-induced chronic active

gastritis to multifocal chronic atrophic gastritis to intestinal metaplasia to dysplasia to invasive carcinoma. H. pylori causes this cascade by: (1) directly damaging the mucosal epithelium via CagA and VacA toxins; (2) inducing chronic mucosal inflammation (neutrophils and macrophages); (3) stimulating gastrin secretion (increasing acid output and cell turnover); and (4) promoting oxidative DNA damage in epithelial cells, leading to accumulation of oncogenic mutations over decades.

Long-answer questions

11. **Basic response:** Clinical features: gastric ulcer: epigastric pain worse after meals, weight loss from avoiding food; duodenal ulcer: epigastric pain when fasting 2 to 3 hours after meals, relieved by food; both: epigastric tenderness on palpation; may be asymptomatic (especially NSAID-related). Complications: haemorrhage (most common: haematemesis and melaena: erosion of gastroduodenal artery by posterior duodenal ulcer), perforation (second most common: sudden severe generalised peritonitis: board-like rigidity: free air under diaphragm on erect CXR in 70 to 80 percent), penetration (erodes adjacent organ: posterior gastric ulcer to pancreas: raised amylase and back pain), gastric outlet obstruction (pyloric scarring: projectile non-bilious vomiting: hypochloraemic hypokalaemic metabolic alkalosis). Investigation: OGD (primary: biopsy all gastric ulcers to exclude malignancy; not required for duodenal ulcers; endoscopic haemostasis for bleeding), H. pylori testing (CLO test on antral biopsy; urea breath test for eradication confirmation), Rockall score (risk-stratify upper GI bleeding), FBC and U and E and clotting (anaemia, elevated urea from blood protein digestion, coagulopathy), erect CXR and CT for suspected perforation.

Value-added response: An important practical rule in upper GI bleeding: any patient with haematemesis or melaena should be resuscitated first (IV access, IV fluids, blood transfusion targeting Hb above 7 to 8 g/dL, correct coagulopathy with FFP) before endoscopy; endoscopy on a haemodynamically unstable patient is dangerous and should only proceed once the patient is stabilised. Pre-endoscopy IV erythromycin (a prokinetic: 250 mg IV 30 minutes before OGD) accelerates gastric emptying and improves visibility at endoscopy.

12. **Basic response:** Medical management: resuscitate with IV fluids and IV antibiotics (covering aerobes and anaerobes: cefuroxime plus metronidazole), pass a nasogastric tube for gastric decompression, give IV PPI infusion (reduces gastric acid exposure to the peritoneum during and after repair), and keep the patient nil by mouth. Surgery: laparotomy (or laparoscopy in experienced centres); identify the perforation (most commonly on the anterior wall of the first part of the duodenum or the lesser curvature of the stomach); close the perforation with two or three interrupted absorbable sutures; reinforce with an omental patch (Graham patch: a tongue of omentum sutured over the

closed perforation); lavage the peritoneal cavity thoroughly with warm saline (minimum 3 litres); drain placement is optional. Postoperatively: continue IV PPI, start H. pylori eradication once the patient can eat (to prevent recurrence), and complete the antibiotic course.

Value-added response: Laparoscopic repair of perforated peptic ulcer (laparoscopic Graham patch) is now established as the preferred approach in experienced centres, offering equivalent outcomes to open surgery with significantly lower wound complications, faster recovery, and shorter hospital stay. In Nigeria, open surgery remains more widely practised due to resource constraints, but laparoscopic repair should be offered where the expertise and equipment are available.

13. **Basic response:** Distribution: UC begins at the rectum and extends proximally in a continuous, confluent pattern without skip lesions (the entire colonic mucosa from the rectum upward is involved: from proctitis to left-sided colitis to pancolitis). CD can involve any part of the GI tract from mouth to anus; most commonly affects the terminal ileum and colon; characterised by skip lesions (diseased segments separated by normal bowel). Depth: UC is a mucosal and submucosal disease (inflammation does not penetrate the full thickness of the bowel wall: hence fistulae are uncommon). CD is transmural (full-thickness inflammation: predisposes to fistula formation, sinus tracts, strictures, and abscesses). Histology: UC shows crypt abscesses, goblet cell depletion (mucin depletion), mucosal ulceration, and pseudopolyps (islands of surviving hyperplastic mucosa surrounded by denuded inflamed mucosa). CD shows non-caseating granulomas (the hallmark), cobblestone mucosa (islands of oedematous mucosa separated by deep linear ulcers), and transmural lymphoid aggregates. Complications: UC: toxic megacolon (colon above 6 cm on AXR: perforation risk: IV corticosteroids or emergency colectomy), haemorrhage, stricture, and CRC (risk increases with extent and duration of disease: pancolitis for more than 10 years: significant cancer risk). CD: strictures causing small bowel obstruction, enteroenteral and enterovesical and enterocutaneous fistulae, perianal disease (fissures, fistulae, abscesses, and skin tags: the most common extra-intestinal complication in Nigerian practice), and malabsorption.

Value-added response: The distinction between UC and CD has important therapeutic and prognostic implications: UC can be cured by colectomy (removing the diseased colon eliminates the disease), whereas CD recurs after surgical resection because any part of the GI tract can be affected. This means that surgery in CD is reserved for complications (obstruction, fistula, abscess) and aims to preserve as much bowel as possible, whereas colectomy with ileo-anal pouch (IPAA) can be offered to UC patients who have failed medical therapy or who have longstanding disease with a high cancer risk.

14. **Basic response:** Clinical features by site: right-sided CRC (iron deficiency anaemia from occult blood loss into the large caecum and ascending colon lumen: presents with fatigue

and pallor: vague right iliac fossa discomfort: palpable mass: weight loss). Left-sided CRC (change in bowel habit: alternating constipation and diarrhoea: the narrower left colon obstructs earlier; bright red or mixed bleeding; progressive large bowel obstruction). Rectal CRC (tenesmus: sensation of incomplete evacuation; blood and mucus per rectum; alteration in stool calibre; late: palpable mass on PR examination). Staging: CT of chest, abdomen, and pelvis (primary tumour size and regional lymph nodes and distant metastases: liver and lung); MRI rectum (for rectal cancer: defines the circumferential resection margin (CRM): if tumour is within 1 mm of the CRM: positive margin: requires neoadjuvant chemoradiotherapy to downstage before surgery). Colonoscopy with biopsy (histological confirmation and to assess the rest of the colon). CEA (baseline and postoperative surveillance). Surgery: right hemicolectomy (ileocolic and right colic vessels divided: for right CRC); sigmoid colectomy or anterior resection for sigmoid and upper rectal CRC; low anterior resection with total mesorectal excision (TME) for mid-rectal CRC; abdominoperineal resection (APR) for lower rectal CRC not amenable to sphincter preservation. Adjuvant chemotherapy (FOLFOX or CAPOX) for Stage III CRC reduces recurrence by approximately 20 percent.

Value-added response: Total mesorectal excision (TME) is the critical technical advance that reduced local recurrence rates in rectal cancer from approximately 30 to 40 percent with conventional surgery to below 5 to 10 percent. TME involves sharp dissection in the avascular embryological plane between the visceral and parietal fascia of the mesorectal envelope, excising the rectum together with its complete mesorectal package intact (en bloc: without entering the mesorectal envelope). A positive CRM (tumour within 1 mm of the mesorectal fascia on MRI) is the most important predictor of local recurrence and should prompt neoadjuvant chemoradiotherapy before surgery.

15. **Basic response:** Clinical approach: assessment and resuscitation: ABCDE assessment; establish two large-bore IV cannulae; send blood for FBC, U and E, clotting, blood group and cross-match (4 units); transfuse if Hb below 7 to 8 g/dL or haemodynamically unstable; start IV PPI infusion; pass a nasogastric tube if large haematemesis; catheterise (urine output target above 0.5 mL per kg per hour). Risk stratification: pre-endoscopic Rockall score (uses age, haemodynamic status, and comorbidities: maximum 7 points without endoscopy); patients with a score of 0 (lowest risk) can be discharged and managed as outpatients; patients with higher scores need admission and urgent endoscopy. Endoscopy: within 24 hours for all significant bleeders; within 12 hours for high-risk patients (pre-endoscopic Rockall above 3, haemodynamic instability, active haematemesis). Full Rockall score (adds endoscopic findings to pre-endoscopic score): score above 5 predicts high risk of re-bleeding and death. Endoscopic haemostasis for Forrest Ia and IIa: combination of adrenaline injection plus mechanical clips (or thermal therapy); if endoscopy fails (two attempts), proceed to interventional radiology (transcatheter arterial embolisation: TAE: selectively embolise the gastroduodenal artery or its branches) or surgery (under-running of the bleeding vessel at laparotomy).

Value-added response: Pre-endoscopy IV erythromycin (250 mg IV given 30 to 60 minutes before OGD) acts as a prokinetic, stimulating gastric emptying and clearing the stomach of blood and clot, significantly improving endoscopic visibility and thus the yield of endoscopy. It should be offered to all patients with acute upper GI bleeding who are to undergo emergency OGD.

Answers to Pilot Questions [Series 2]

Part 2.1

1. Peptic: acid and pepsin and H. pylori (example: duodenal ulcer). Ischaemic: impaired arterial blood supply (example: arterial leg ulcer). Venous: venous hypertension (example: venous leg ulcer above medial malleolus). Neuropathic: sensory loss (example: diabetic foot ulcer under pressure points). Traumatic: mechanical injury (example: pressure sore). Infective: specific organisms (example: tuberculosis: Bazin's disease; Buruli ulcer: Mycobacterium ulcerans; syphilitic ulcer: Hunterian chancre). Malignant: ulcerating carcinoma (example: rodent ulcer: BCC). Autoimmune: (example: aphthous ulcer in Crohn's disease).
2. Undermined edges (TB ulcer: the bacterial infection undermines the surrounding skin leaving an overhanging edge). Rolled or heaped-up edges (carcinoma: the proliferating malignant cells pile up at the edge creating a raised everted rim). Sloping edges (healing venous ulcer: the epidermis slopes gradually down from the margin towards the ulcer base as new epithelium grows inward from the edge). Punched-out edges (ischaemic ulcer or syphilitic ulcer: the ulcer edge is vertical and sharply defined as if cut out with a punch, reflecting the sudden cutoff of blood supply or rapid tissue destruction).
3. Aggressive factors: hydrochloric acid (secreted by parietal cells: stimulated by gastrin, histamine, and acetylcholine), pepsin (proteolytic enzyme activated by acid: digests mucosal protein), H. pylori (disrupts the mucous layer, stimulates gastrin release, induces mucosal inflammation, reduces bicarbonate secretion: present in 95 percent of duodenal and 70 percent of gastric ulcers), NSAIDs (inhibit COX-1: reduce prostaglandin synthesis: impair mucosal blood flow and bicarbonate secretion and mucus production). Defensive factors: mucous gel layer (traps bicarbonate, creates a pH gradient between 7 at the cell surface and 1 to 2 in the lumen), bicarbonate secretion (surface epithelial cells), mucosal blood flow (delivers nutrients and removes acid), prostaglandins PGE2 and PGI2 (stimulate mucus and bicarbonate secretion and maintain mucosal blood flow), epithelial restitution (rapid surface epithelial cell migration to cover small defects within minutes to hours).
4. H. pylori is a Gram-negative spiral bacterium that colonises the gastric antrum and body. Mechanisms of mucosal damage: (1) CagA protein (injected into epithelial cells via a

type IV secretion system: disrupts tight junctions, activates oncogenic signalling pathways, and promotes gastric carcinogenesis); (2) VacA toxin (forms channels in epithelial cell membranes, induces vacuolation, promotes apoptosis, and suppresses T-cell function: facilitates immune evasion); (3) urease enzyme (splits urea into ammonia and CO2: ammonia directly damages the mucosa and creates an alkaline microenvironment around the bacterium allowing it to survive in the acid environment); (4) induction of chronic mucosal inflammation (stimulates neutrophil and macrophage infiltration: cytokines including TNF-alpha and IL-8 further damage the mucosa and stimulate acid secretion by promoting gastrin release from G cells).

5. Stage 1: elimination of the causative agent (H. pylori eradication with triple or quadruple therapy; cessation of NSAIDs; acid suppression with PPI). Stage 2: epithelial regeneration by restitution (surface epithelial cells adjacent to the defect migrate rapidly to cover the ulcer base within hours, even before new cell proliferation begins). Stage 3: formation of granulation tissue (new capillary loops and fibroblasts fill the ulcer base with vascular connective tissue over days to weeks). Stage 4: scar formation (collagen is deposited by fibroblasts, replacing the granulation tissue with fibrous scar: this provides tensile strength but can cause pyloric stenosis from scarring at the pylorus or duodenal stenosis in the first part of the duodenum).

Part 2.2

- Gastric ulcer: epigastric pain that is worse after meals (food stimulates acid secretion, aggravating the ulcer); the patient may avoid food and develop weight loss; more common in older patients above 50 years, in women, and in NSAID users; gastric cancer must be excluded by biopsy. Duodenal ulcer: epigastric pain that is worse when fasting 2 to 3 hours after meals (the stomach is empty and acid is not buffered by food or by the duodenal mucus); pain is relieved by eating or antacids; more common in young males; strongly associated with H. pylori (above 95 percent). Both: epigastric tenderness on palpation; may be asymptomatic, especially NSAID-related ulcers (NSAIDs also inhibit prostaglandin-mediated pain signalling).
- Haemorrhage (most common complication: erosion of a blood vessel in the ulcer base; haematemesis and melaena: the gastroduodenal artery is the most commonly eroded vessel: by a posterior duodenal ulcer; classified by the Forrest system). Perforation (second most common: sudden severe epigastric pain rapidly becoming generalised: board-like rigidity from chemical peritonitis: free air under the diaphragm on erect CXR in 70 to 80 percent). Penetration (the ulcer erodes into an adjacent organ without free perforation: posterior gastric ulcer penetrating into the pancreas: elevated serum amylase and epigastric pain radiating to the back). Gastric outlet obstruction (from progressive

pyloric scarring: projectile, profuse, non-bilious vomiting of undigested food: hypochloraemic hypokalaemic metabolic alkalosis from loss of HCl and KCl in vomit).

- Forrest Ia: active arterial spurting: 90 percent re-bleeding risk. Ib: active oozing: 10 to 20 percent. IIa: non-bleeding visible vessel (a pigmented protuberance over the vessel at the ulcer base): 50 percent. IIb: adherent clot overlying the ulcer: 25 to 30 percent. IIc: flat haematin spot on the ulcer base: 7 to 10 percent. III: clean ulcer base without stigmata: 3 to 5 percent. Forrest Ia and IIa require urgent endoscopic haemostasis (re-bleeding risk above 50 percent: endoscopic treatment reduces re-bleeding to approximately 15 to 20 percent and reduces the need for surgery and 30-day mortality). Forrest IIc and III can be discharged early (low re-bleeding risk: no endoscopic therapy required).
- Rapid urease test (CLO test): biopsy of the gastric antrum is placed in a urease-containing medium; H. pylori urease converts urea to ammonia, raising the pH and changing the medium colour: quick and reliable in active untreated disease; false-negative if the patient has recently taken PPIs or antibiotics. Histology: haematoxylin and eosin stain of the gastric biopsy; the most accurate method: identifies the organism directly and assesses the degree of gastritis; can detect H. pylori even when urease test is negative. Urea breath test: non-invasive: the patient drinks radio-labelled urea (C13 or C14): H. pylori urease splits the urea, releasing labelled CO2 detected in the exhaled breath: used for initial diagnosis and to confirm eradication at 4 weeks after therapy and 2 weeks after stopping PPIs. Stool antigen test: non-invasive: H. pylori antigens detected in stool by enzyme immunoassay: as accurate as the breath test for confirming eradication.
- The Rockall score is a validated risk stratification tool for acute upper GI bleeding that combines pre-endoscopic clinical variables and endoscopic findings. Pre-endoscopic variables: age (0: below 60; 1: 60 to 79; 2: above 80), shock (0: no shock; 1: pulse above 100; 2: systolic BP below 100), and comorbidities (0: none; 2: ischaemic heart disease or cardiac failure; 3: renal or hepatic failure or disseminated malignancy). Endoscopic variables: diagnosis (0: Mallory-Weiss or no lesion; 1: PUD or other; 2: malignancy) and stigmata (0: clean base or haematin spot; 2: blood in upper GI tract or visible vessel or adherent clot). Maximum total score: 11. Clinical utility: score of 0 (very low risk: 0 percent re-bleeding and 0 percent mortality: safe for early discharge without inpatient endoscopy); score above 5 (high risk: predicts high re-bleeding rate and significant 30-day mortality: requires ICU-level monitoring and early re-endoscopy if re-bleeding occurs).

Part 2.3

- PPIs (omeprazole, lansoprazole, pantoprazole, rabeprazole, esomeprazole): irreversibly inhibit the H+/K+ ATPase enzyme (the proton pump) of the parietal cell; the most potent acid suppressants available; achieve greater than 90 percent inhibition of acid secretion; once-daily dosing is sufficient because new proton pump molecules must be synthesised before acid secretion can resume; require an acidic environment to be activated (take before meals). H2-receptor antagonists (ranitidine, famotidine): competitively and reversibly block histamine H2 receptors on the parietal cell, reducing acid secretion: less potent than PPIs (achieve approximately 70 percent acid suppression); develop tachyphylaxis (reduced efficacy over time from receptor upregulation).
- First-line triple therapy for 7 to 14 days: PPI (omeprazole 20 mg or equivalent) twice daily plus amoxicillin 1 g twice daily plus clarithromycin 500 mg twice daily. Eradication is the most important intervention because: (1) it heals the ulcer in 90 to 95 percent of cases (versus 50 to 60 percent for acid suppression alone); (2) it prevents recurrence: the annual ulcer recurrence rate falls from approximately 80 percent to less than 5 percent after successful eradication; and (3) it may reduce the long-term risk of gastric carcinoma (by eliminating the Correa cascade driver). Eradication is confirmed by urea breath test or stool antigen test at least 4 weeks after completing antibiotics and 2 weeks after stopping PPIs.
- Adrenaline injection (1:10,000): injected in four quadrants around the ulcer base; achieves initial haemostasis by vasoconstriction and mechanical tamponade from the injection volume; rapid but has a high re-bleeding rate when used alone (approximately 20 percent). Thermal coagulation (heater probe at 20 to 30 J or bipolar electrocautery): directly coagulates the bleeding vessel or the visible vessel; more durable than adrenaline alone. Endoscopic clips (haemostatic clips applied across the bleeding vessel): mechanically compress the vessel, achieving the most durable haemostasis; preferred technique for accessible vessels. Combination therapy (adrenaline injection followed by clip or thermal therapy): superior to any single modality and is the recommended approach for Forrest Ia and IIa lesions.
- Resuscitation: IV fluid resuscitation (normal saline or Hartmann's), blood transfusion (target Hb above 8 g/dL), IV PPI infusion, IV antibiotics (cefuroxime plus metronidazole), nasogastric tube for gastric decompression, nil by mouth. Operative procedure: laparotomy (midline incision); identify the perforation (most commonly on the anterior wall of the first part of the duodenum); perform Graham patch closure (close the perforation with two to three interrupted 2/0 absorbable sutures; suture a tongue of omentum over the closed perforation and fix it in place with further sutures); peritoneal lavage with at least 3 litres of warm saline; drain placement optional. Postoperative: continue IV PPI; start H. pylori eradication therapy when the patient can eat; continue antibiotics for 5 days; start liquids then solid food progressively. Definitive acid-reducing

surgery (vagotomy) is not routinely performed as H. pylori eradication and PPIs are curative in the long term.

- Failed endoscopic haemostasis (two endoscopic attempts have failed to control bleeding: the patient continues to bleed requiring more than 4 units of blood in 24 hours: proceed to angiographic embolisation or laparotomy with under-running of the bleeding vessel). Perforation (peritonitis from perforated peptic ulcer: emergency laparotomy for Graham patch closure). Gastric outlet obstruction from pyloric scarring refractory to 6 to 8 weeks of medical treatment (endoscopic dilatation first; if repeated dilatation fails or stenosis is severe: surgical gastrojejunostomy or pyloroplasty). Suspicion of malignancy in a non-healing gastric ulcer (a gastric ulcer that fails to heal after 8 to 12 weeks of appropriate PPI therapy and two sets of biopsies suggest malignancy: surgical resection).

Part 2.4

- Stress ulcers are acute mucosal erosions or ulcerations developing in critically ill patients in the ICU. Pathophysiology: the physiological stress response (from burns, sepsis, trauma, or mechanical ventilation) causes massive sympathetically mediated splanchnic vasoconstriction (reducing intestinal blood flow by up to 40 to 50 percent of baseline): this impairs the delivery of bicarbonate to the mucosa, reduces prostaglandin synthesis, and depletes the mucosal energy supply, causing the defensive barrier to fail. Without a functioning mucosal barrier, luminal acid erodes the mucosa, causing multiple shallow erosions and ulcers, typically in the gastric body and fundus. Patients at highest risk: those on mechanical ventilation (above 48 hours), those with coagulopathy, those with major burns (Curling's ulcer), and those with severe head injury (Cushing's ulcer).
- Curling's ulcer: stress ulcer occurring specifically in patients with major burns (above 35 percent total body surface area); caused by hypovolaemia and splanchnic hypoperfusion reducing mucosal blood flow and barrier function; typically multiple, shallow, in the gastric fundus and body; presents with bleeding (haematemesis or melaena). Cushing's ulcer: a deep, single ulcer of the stomach, duodenum, or oesophagus, occurring in patients with head injuries, intracranial tumours, or after neurosurgical procedures; caused by direct stimulation of the vagus nerve (or vagal nuclei) by elevated intracranial pressure: this causes massive, unregulated cholinergic (vagal) stimulation of parietal cells, resulting in massive gastric acid hypersecretion (far greater than in ordinary PUD): hence Cushing's ulcers are deeper and more prone to perforation than ordinary stress ulcers.
- Venous ulcer: caused by chronic venous hypertension (from deep vein incompetence, perforating vein incompetence, or superficial varicose veins); site: gaiter area just above

the medial malleolus (the area most affected by the highest venous pressure in the leg); appearance: shallow, irregular, sloping edges, red granulating base, surrounded by lipodermatosclerosis (fibrosis and brown haemosiderin staining of the skin and subcutaneous tissue); management: four-layer compression bandaging (reduces venous hypertension: the cornerstone of treatment: contraindicated if ABPI below 0.8), wound care and debridement, treatment of underlying varicose veins (by endovenous ablation or surgery). Arterial ulcer: caused by peripheral arterial disease (atherosclerosis reducing arterial inflow); site: toes, heel, and bony pressure points (areas furthest from the arterial supply or under the highest pressure); appearance: punched-out, deep, pale or necrotic base, very painful; management: vascular reconstruction (percutaneous transluminal angioplasty for short-segment stenosis; bypass graft for long-segment occlusion): compression is contraindicated. Neuropathic ulcer: caused by sensory neuropathy (most commonly diabetic peripheral neuropathy): the patient cannot feel pain and continues to walk on the ulcerated foot; site: pressure points under the foot (first metatarsal head, heel); appearance: painless, deep, often surrounded by callus; management: debridement of surrounding callus, off-loading (total contact cast or special footwear), glycaemic control, antibiotics for infected ulcers, revascularisation if there is a coexisting arterial component (neuroischaemic ulcer).

- The ankle-brachial pressure index (ABPI) is the ratio of the systolic blood pressure at the ankle (measured by Doppler over the dorsalis pedis or posterior tibial artery) to the systolic blood pressure at the brachial artery: ABPI equals ankle systolic pressure divided by brachial systolic pressure. Clinical utility in leg ulcers: ABPI above 0.9 is normal and indicates adequate arterial inflow: compression bandaging is safe. ABPI 0.8 to 0.9 suggests mild arterial disease: modified compression (reduced pressure) may be used with caution. ABPI below 0.8 indicates significant arterial insufficiency: compression bandaging is contraindicated (it would further reduce already impaired arterial flow to the foot and risk precipitating critical limb ischaemia). ABPI below 0.5 indicates critical limb ischaemia: urgent vascular assessment and revascularisation are required. An ABPI above 1.3 suggests arterial calcification (medial calcinosis: common in diabetic patients) and is an unreliable result (falsely elevated): further investigation with toe-brachial index or duplex ultrasound is needed.
- Oral carcinoma (squamous cell carcinoma of the oral cavity) most commonly presents as a non-healing ulcer on the lateral border of the tongue, floor of the mouth, or buccal mucosa: the ulcer has irregular, rolled (everted) or heaped-up edges, an indurated (hard) base, and may be fixed to underlying structures. It may also present as a red patch (erythroplakia: above 40 percent risk of malignant transformation: the highest risk oral mucosal lesion) or a white patch (leukoplakia: 5 to 10 percent risk of transformation). The two most important risk factors are tobacco smoking and alcohol consumption: together they have a synergistic (multiplicative rather than additive) effect on the risk of

oral carcinoma. The 3-week rule: any oral ulcer that fails to heal within 3 weeks (despite removal of apparent causes) must be biopsied under local or general anaesthesia to exclude malignancy.

Part 2.5

- Clinical features: insidious onset with epigastric pain (dull, chronic), profound anorexia, weight loss above 10 percent body weight in 6 months, and early satiety (the tumour reduces gastric volume and motility). Dysphagia if the tumour involves the gastric cardia or oesophagogastric junction. Four signs of advanced disease: (1) Virchow's node (Troisier's sign: an enlarged, hard, non-tender left supraclavicular lymph node: from metastatic spread via the thoracic duct); (2) Sister Mary Joseph's nodule (a hard periumbilical nodule from transcoelomic seeding of the periumbilical lymphatics); (3) Blumer's shelf (a hard mass felt on rectal examination from tumour deposited in the pouch of Douglas); (4) Krukenberg tumour (bilateral ovarian metastasis from transcoelomic spread of mucin-secreting signet-ring cell gastric cancer: detected on pelvic ultrasound or CT).
- The Correa cascade is the sequence: normal gastric mucosa to H. pylori-induced chronic active gastritis (neutrophil and lymphocyte infiltration) to multifocal atrophic gastritis (gland loss) to intestinal metaplasia (replacement of gastric mucosa with intestinal-type mucosa: goblet cells and Paneth cells) to dysplasia (low-grade then high-grade) to invasive intestinal-type gastric carcinoma. H. pylori drives this cascade by: directly damaging epithelial cells via CagA and VacA toxins; inducing chronic mucosal inflammation with oxidative DNA damage; stimulating hypergastrinaemia (gastrin promotes epithelial cell proliferation); and reducing vitamin C secretion into the gastric lumen (vitamin C is an antioxidant that protects against nitrosamine formation). H. pylori eradication in early stages of the Correa cascade can reverse atrophic gastritis and intestinal metaplasia, potentially preventing progression to cancer.
- SCC: arises from the squamous epithelium lining the upper and middle thirds of the oesophagus (from the cricopharyngeus to the level of the tracheal bifurcation); risk factors: tobacco smoking, alcohol consumption (synergistic with smoking), achalasia (stasis of food and carcinogens in contact with the oesophageal mucosa), tylosis (autosomal dominant palmoplantar hyperkeratosis: rare: strong SCC predisposition), nitrosamine-containing foods, and iron deficiency (Plummer-Vinson syndrome). More common in Africa and Asia. Adenocarcinoma: arises from the glandular (columnar) epithelium of Barrett's oesophagus in the lower third and oesophagogastric junction; risk factors: chronic gastro-oesophageal reflux disease (GORD: the most important), obesity (increases intra-abdominal pressure and GORD), tobacco smoking, and Barrett's

oesophagus (0.5 percent per year risk of adenocarcinoma: requires endoscopic surveillance). More common in Western populations and increasingly common in Nigeria.

- Achalasia is a primary oesophageal motility disorder characterised by: (1) failure of relaxation of the lower oesophageal sphincter (LOS) during swallowing, from degeneration of the inhibitory neurons of the Auerbach's (myenteric) plexus; and (2) absence of progressive peristalsis in the oesophageal body (aperistalsis). Clinical features: progressive dysphagia for both solids and liquids from the outset (unlike carcinoma where dysphagia for liquids comes later: this is because the obstruction in achalasia is functional and affects all food equally), regurgitation of undigested food, nocturnal aspiration (causing recurrent pneumonia), chest pain (from oesophageal spasm), and weight loss. Barium swallow: bird-beak or rat-tail appearance (smooth, symmetric tapering of the distal oesophagus to a point as the non-relaxing LOS prevents the contrast column from passing through). Treatment: Heller's cardiomyotomy (laparoscopic: a longitudinal incision through the LOS muscle fibres down to the mucosa: often combined with a partial fundoplication to prevent GORD) or pneumatic dilatation (endoscopic balloon dilatation of the LOS: effective but carries a 2 to 3 percent risk of oesophageal perforation) or per-oral endoscopic myotomy (POEM: an endoscopic technique).
- Clinical features: abdominal pain (most common: from mucosal ulceration by the tumour), upper GI bleeding (haematemesis or melaena from mucosal ulceration: may be the first presentation), a palpable abdominal mass, or incidental finding on CT or endoscopy. Risk stratification (by mitotic rate, tumour size, and site of origin) determines prognosis: tumours with mitotic rate above 5 per 50 high-power fields, size above 5 cm, and non-gastric site have the highest risk of recurrence. Management: (1) R0 surgical resection (complete excision with clear margins) for localised resectable GISTs: laparoscopic or open depending on site and size: no need for regional lymph node dissection (GISTs rarely spread to lymph nodes: they spread haematogenously to the liver and peritoneum); (2) imatinib (400 mg orally once daily: a selective tyrosine kinase inhibitor targeting c-KIT: has transformed the treatment of advanced and high-risk GIST: 5-year survival for advanced GIST on imatinib approximately 50 to 60 percent compared with approximately 5 percent with surgery alone): used for unresectable or metastatic GIST, as adjuvant therapy for high-risk resected GIST (3 years of treatment after R0 resection), and as neoadjuvant therapy to shrink large or borderline resectable GISTs before surgery.

Part 2.6

1. Right-sided CRC: the caecum and ascending colon have a wide lumen and liquid stool: tumours grow to a large size before causing obstruction. Presents insidiously with: iron deficiency anaemia (from chronic occult blood loss into the lumen: the patient presents with fatigue and pallor rather than rectal bleeding), vague right iliac fossa discomfort or colicky pain (from partial obstruction or bowel wall invasion), a palpable right-sided abdominal mass (in advanced cases), and weight loss. Left-sided CRC: the sigmoid and descending colon have a narrower lumen and formed stool: presents with change in bowel habit (alternating constipation and diarrhoea from a partially obstructing tumour), bright red or mixed blood in the stool, progressive large bowel obstruction (presenting with absolute constipation and abdominal distension), and tenesmus if near the rectum. Rectal CRC: the rectum is accessible on PR examination and sigmoidoscopy: presents with tenesmus (constant sensation of incomplete evacuation), passage of blood and mucus per rectum, alteration in stool calibre (the faecal stream is moulded around the tumour producing a narrow or ribbon-like stool), and in advanced disease a palpable hard mass felt on PR examination (Blumer's shelf may also be felt if the tumour has seeded the pouch of Douglas).

2. Ulcerative colitis: distribution (begins at rectum and extends proximally in a continuous, confluent pattern without skip lesions: proctitis, left-sided colitis, or pancolitis); histology (mucosal and submucosal disease: crypt abscesses, goblet cell depletion, mucosal ulceration, pseudopolyps: islands of surviving hyperplastic mucosa surrounded by denuded inflamed mucosa; no granulomas); complications (toxic megacolon: colon above 6 cm on AXR: risk of perforation: IV steroids or emergency colectomy; haemorrhage; stricture; colorectal carcinoma risk increases with extent and duration: pancolitis for more than 10 years has significant cancer risk; primary sclerosing cholangitis: the most important extra-intestinal manifestation of UC). Crohn's disease: distribution (any part of GI tract from mouth to anus: most commonly terminal ileum and colon: skip lesions: areas of diseased bowel separated by normal bowel); histology (transmural inflammation: full thickness: non-caseating granulomas: the hallmark: cobblestone mucosa: islands of oedematous mucosa separated by deep linear ulcers: fissuring ulcers: transmural lymphoid aggregates); complications (strictures causing small bowel obstruction: most common surgical complication; fistulae: enteroenteral, enterovesical, enterocutaneous, enterovaginal; abscesses: perianal; perianal disease: fissures, fistulae, abscesses, and skin tags: most common in Nigerian patients with CD).

3. Classic presentation: a previously well young adult (peak age 10 to 30 years) with central periumbilical colicky pain that migrates to the right iliac fossa over 4 to 24 hours (as the visceral pain from appendiceal distension becomes somatic pain from parietal peritoneal irritation), accompanied by anorexia (very consistent: a patient who is not anorexic is less likely to have appendicitis), nausea and vomiting (usually after the onset of pain: vomiting before pain makes appendicitis less likely), low-grade fever (above 37.5

degrees C: rarely above 39 degrees C in uncomplicated appendicitis), and localised right iliac fossa guarding and tenderness at McBurney's point. Rovsing's sign (pressure in the left iliac fossa causes pain in the right: suggests right peritoneal inflammation) may be positive. Scoring system: the Alvarado score (also called the MANTRELS score) uses eight clinical and laboratory criteria (migration of pain: 1 point; anorexia: 1; nausea and vomiting: 1; RIF tenderness: 2; rebound tenderness: 1; elevated temperature above 37.3 degrees C: 1; leucocytosis: 2; left shift: 1): maximum 10 points; score 7 to 10: appendicitis very likely and surgical exploration appropriate; score 5 to 6: borderline: further investigation with ultrasound or CT; score below 5: appendicitis unlikely.

4. First degree: bleed only (internal haemorrhoids that bleed on defaecation but do not prolapse below the dentate line): management: dietary advice (high-fibre diet, adequate fluid intake to soften stools), stool softeners, topical preparations (local anaesthetic and anti-inflammatory creams for symptomatic relief). Second degree: prolapse on defaecation but reduce spontaneously: management: rubber band ligation (the most effective outpatient technique for second degree haemorrhoids: a rubber band is applied at the base of the haemorrhoid above the dentate line: causes ischaemic necrosis and the haemorrhoid sloughs off in 5 to 7 days) or injection sclerotherapy (5 percent phenol in arachis oil injected into the submucosa above the haemorrhoid: causes sclerosis and fixation). Third degree: prolapse on defaecation and require manual reduction: management: rubber band ligation (for smaller third degree) or haemorrhoidectomy (surgical excision of the haemorrhoidal pedicles: Milligan-Morgan technique: for larger third degree or failed band ligation). Fourth degree: permanently prolapsed and irreducible: management: haemorrhoidectomy.

5. Parks classification of anal fistulae (based on the relationship of the fistula tract to the external anal sphincter): Intersphincteric (most common: approximately 70 percent: the tract passes between the internal and external sphincters to open in the perianal skin: does not cross the external sphincter); Transsphincteric (approximately 25 percent: the tract passes through both sphincters to open in the ischiorectal fossa: involves part of the external sphincter); Suprasphincteric (rare: the tract passes above the puborectalis then down through the ischiorectal fossa); Extrasphincteric (rarest: the tract bypasses both sphincters entirely: usually from pelvic pathology). Surgical principle for high fistulae: the sphincter muscle is the critical structure for faecal continence: dividing it completely in a single operation (fistulotomy) for high transsphincteric, suprasphincteric, or extrasphincteric fistulae would cause permanent incontinence. Therefore, a seton suture (a thick suture or silastic drain threaded through the fistula tract) is placed to: (1) drain pus and reduce inflammation; (2) cause gradual fibrosis of the external sphincter (if tightened progressively as a cutting seton); or (3) define the anatomy before staged fistulotomy. This preserves sphincter function while treating the fistula.

References and Attributions [Series 2]

Textual References

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Figures, Plates, Tables, and Boxes

- Figure 2.1: Classification and pathophysiology of ulcers. AI-generated using the prompt: "Two-panel diagram: ulcer aetiology classification table (peptic, ischaemic, venous, neuropathic, traumatic, infective, malignant, autoimmune) and macroscopic features and edge types diagram; aggressive versus defensive factors balance diagram (HCl, pepsin, H. pylori with CagA and VacA, NSAIDs versus mucous layer, bicarbonate, prostaglandins), chronic ulcer microscopy box, and healing stages box." Suggested OER search string: "peptic ulcer pathophysiology Helicobacter pylori ulcer classification edge types healing OER."
- Figure 2.2: Clinical features, complications, and investigation of PUD. AI-generated using the prompt: "Two-panel diagram: gastric versus duodenal ulcer clinical features comparison table, complications box (haemorrhage, perforation, penetration, gastric outlet obstruction), and Forrest classification table (Ia to III with re-bleeding rates); OGD box, H. pylori testing table (CLO test, histology, urea breath test, stool antigen), and additional investigations box (Rockall score, fasting gastrin for ZES)." Suggested OER search string: "peptic ulcer complications Forrest classification Rockall score H. pylori testing OER."
- Figure 2.3: Management of PUD. AI-generated using the prompt: "Two-panel diagram: acid suppression table (PPIs, H2 blockers, antacids) and H. pylori eradication box (triple therapy, quadruple therapy, eradication confirmation); endoscopic haemostasis box (adrenaline injection, thermal, clips, combination, timing) and surgical management box

(indications, bleeding under-running, Graham patch, gastrojejunostomy)." Suggested OER search string: "peptic ulcer management PPI H. pylori eradication endoscopic haemostasis Graham patch OER."

- Figure 2.4: Stress ulcers, leg ulcers, and oral ulcers. AI-generated using the prompt: "Two-panel diagram: stress ulcer pathophysiology box and stress ulcer types table (stress ulcer, Curling's, Cushing's) with prevention and treatment; three-type leg ulcer comparison table (venous, arterial, neuropathic) and oral ulcers box (aphthous ulcers and oral carcinoma SCC)." Suggested OER search string: "stress ulcer Curling's Cushing's leg ulcer ABPI venous arterial neuropathic oral carcinoma OER."
- Figure 2.5: Upper GI pathology. AI-generated using the prompt: "Two-panel diagram: gastric carcinoma risk factors box (Correa cascade, CDH1 mutation), clinical features box (Virchow's node, Blumer's shelf, Krukenberg tumour), and management box (OGD, CT, laparoscopy, D2 gastrectomy, FLOT); oesophageal carcinoma box (SCC versus adenocarcinoma, Barrett's surveillance), other oesophageal conditions table (achalasia, Boerhaave's, pharyngeal pouch), and GIST box (CD117, imatinib, R0 resection)." Suggested OER search string: "gastric carcinoma Correa cascade GIST imatinib Barrett's oesophagus achalasia Boerhaave OER."
- Figure 2.6: Lower GI pathology. AI-generated using the prompt: "Two-panel diagram: CRC risk factors box (FAP APC gene and Lynch syndrome HNPCC and IBD), CRC clinical features by site, CRC staging and surgery, and UC versus CD IBD comparison table; acute appendicitis box (McBurney's point, Alvarado score, management) and anal conditions table (haemorrhoids, anal fissure, perianal abscess, anal fistula Parks classification) with carcinoid tumours box." Suggested OER search string: "colorectal carcinoma FAP Lynch syndrome IBD Crohn's UC appendicitis anal fistula Parks OER."

Series 3: Endocrine and Thyroid Disorders

1. **Part 3.1: Thyroid Anatomy, Physiology, and Clinical Assessment**
    1. Sub Part 3.1.1: Surgical anatomy of the thyroid gland
    2. Sub Part 3.1.2: Thyroid hormone synthesis, regulation, and physiology
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6. **Part 3.6: Adrenal and Other Endocrine Surgical Disorders**
    1. Sub Part 3.6.1: Surgical anatomy and disorders of the adrenal gland
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    3. Sub Part 3.6.3: Multiple endocrine neoplasia (MEN) syndromes

Introduction

Endocrine and thyroid disorders form a major component of General Surgery II, requiring an integrated understanding of anatomy, physiology, and surgical technique. The thyroid gland is the most commonly operated endocrine organ, and disorders ranging from simple goitre to thyroid carcinoma present frequently in Nigerian surgical practice, with endemic goitre remaining a significant public health problem in iodine-deficient areas.

This Series covers thyroid anatomy, physiology, and clinical assessment, then examines goitre, hyperthyroidism, hypothyroidism, thyroiditis, and thyroid malignancy in turn. It concludes with the practical surgical management of thyroid and parathyroid disease and a survey of adrenal surgical disorders and the multiple endocrine neoplasia syndromes, which link thyroid and adrenal pathology through shared genetic mechanisms.

Series Learning Outcomes

Upon completing this Series, you should be able to:

- **SLO 3.1:** describe the surgical anatomy, physiology, and clinical assessment of the thyroid gland,
- **SLO 3.2:** classify goitre and describe the investigation of a solitary thyroid nodule,
- **SLO 3.3:** describe the causes, clinical features, and management of hyperthyroidism, including thyroid storm,
- **SLO 3.4:** describe the causes and management of hypothyroidism, thyroiditis, and thyroid carcinoma,
- **SLO 3.5:** describe the indications, types, and complications of thyroid and parathyroid surgery, and
- **SLO 3.6:** describe the surgical disorders of the adrenal gland and the multiple endocrine neoplasia syndromes.

3.1 Thyroid Anatomy, Physiology, and Clinical Assessment

3.1.1 Surgical anatomy of the thyroid gland

The thyroid gland lies in the anterior neck at the level of C5 to T1, consisting of two lateral lobes joined by an isthmus that overlies the second to fourth tracheal rings. It is enclosed by the pretracheal fascia and is closely related to the recurrent laryngeal nerves, which run in the tracheo-oesophageal groove, and the external branch of the superior laryngeal nerve, which lies near the superior pole vessels.

Blood supply is from the superior thyroid artery (a branch of the external carotid) and the inferior thyroid artery (a branch of the thyrocervical trunk). Venous drainage is via the superior,

middle, and inferior thyroid veins. The parathyroid glands, usually four in number, lie posterior to the thyroid capsule and must be identified and preserved during thyroidectomy to avoid hypoparathyroidism.

Fill in the blank: The recurrent laryngeal nerve runs in the _____ groove. The thyroid isthmus overlies tracheal rings _____. There are usually _____ parathyroid glands.

3.1.2 Thyroid hormone synthesis, regulation, and physiology

Thyroid hormone synthesis begins with active iodide trapping by the sodium-iodide symporter, followed by organification (oxidation and binding to tyrosine residues on thyroglobulin) to form monoiodotyrosine and diiodotyrosine, which couple to form triiodothyronine (T3) and thyroxine (T4). T4 is the major secreted hormone but is peripherally converted to the more active T3 by deiodinase enzymes.

Regulation follows the hypothalamic-pituitary-thyroid axis: thyrotropin-releasing hormone (TRH) stimulates thyroid-stimulating hormone (TSH) release, which stimulates thyroid hormone synthesis and release. Negative feedback by T3 and T4 suppresses TSH and TRH. Thyroid hormones regulate basal metabolic rate, thermogenesis, growth, and cardiovascular and neurological function throughout life.

Fill in the blank: The enzyme that traps iodide into the thyroid follicular cell is the _____. TSH is regulated by negative feedback from _____ and _____.

3.1.3 Clinical assessment and thyroid function tests

Clinical assessment includes history (family history, radiation exposure, rate of neck swelling growth, compressive symptoms such as dysphagia, dyspnoea, and hoarseness) and examination (inspection for swelling and movement on swallowing, palpation for consistency, nodularity, tenderness, and mobility, and auscultation for a bruit in hyperthyroidism). Cervical lymphadenopathy and tracheal deviation must be assessed.

Biochemical assessment begins with serum TSH, the most sensitive first-line test, followed by free T4 and T3 when TSH is abnormal. Thyroid autoantibodies (anti-TPO, anti-TSH receptor) support autoimmune diagnoses. Imaging includes ultrasound (first-line for nodules), radioisotope scanning, and fine needle aspiration cytology for suspicious nodules, reported using the Bethesda system.

Fill in the blank: The most sensitive first-line biochemical test of thyroid function is _____. Fine needle aspiration cytology is reported using the _____ system.

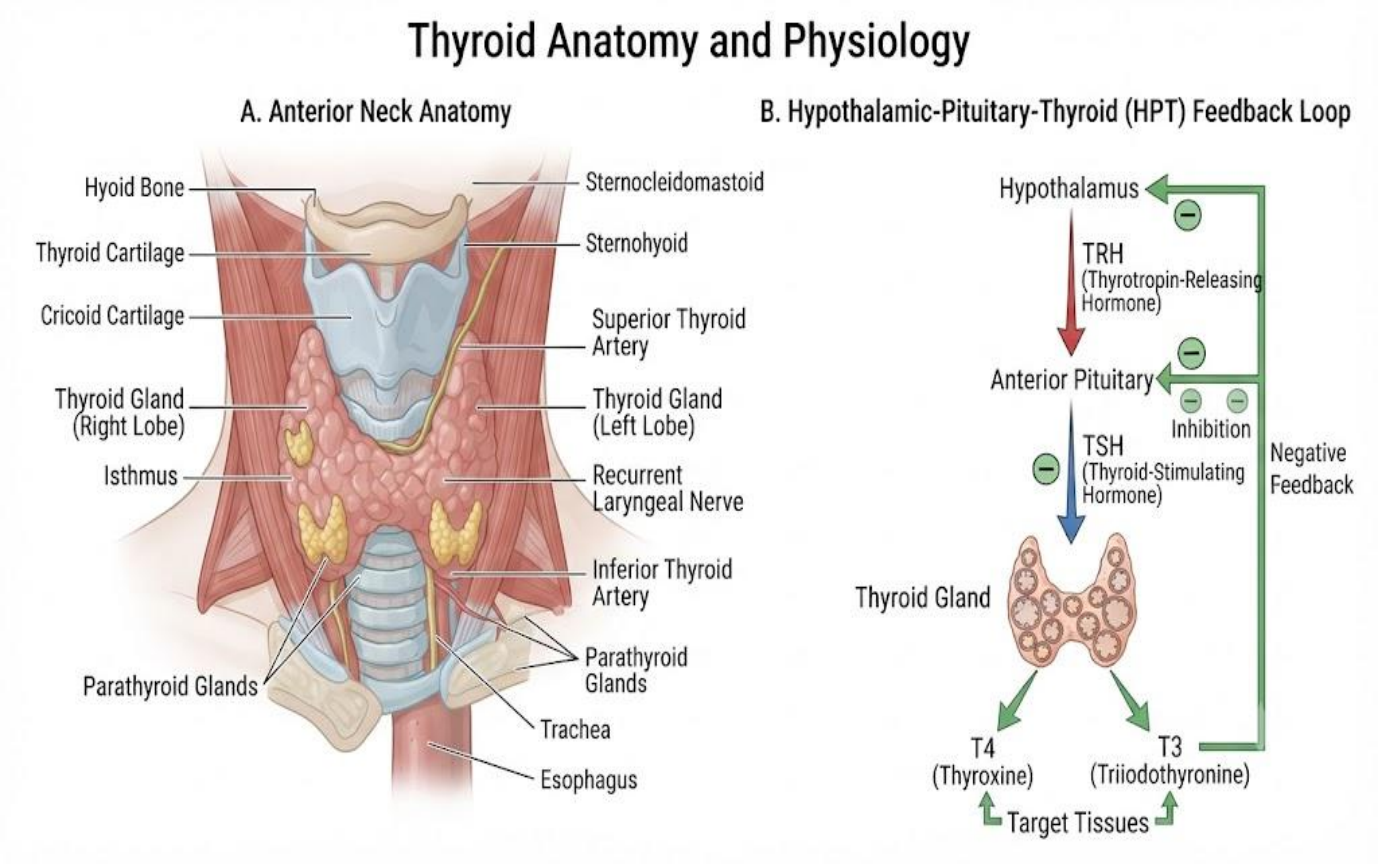

Figure 3.1: Surgical anatomy of the thyroid and the hypothalamic-pituitary-thyroid axis.

Pilot questions 3.1

1. Describe the surgical anatomy of the thyroid gland and its relations to the recurrent laryngeal nerves and parathyroid glands. (Linked to SLO 3.1)
2. Outline the steps of thyroid hormone synthesis from iodide trapping to hormone release. (Linked to SLO 3.1)
3. Describe the hypothalamic-pituitary-thyroid axis and its negative feedback mechanism. (Linked to SLO 3.1)
4. Describe the clinical assessment of a patient presenting with a neck swelling. (Linked to SLO 3.1)
5. Describe the first-line and second-line investigations used to assess thyroid function and nodules. (Linked to SLO 3.1)

3.2 Goitre and Thyroid Nodules

3.2.1 Classification and epidemiology of goitre

A goitre is any enlargement of the thyroid gland, classified as diffuse or nodular, and as toxic (hyperthyroid), euthyroid, or non-toxic. Physiological goitre occurs during puberty and pregnancy from increased demand. Endemic goitre, common in iodine-deficient regions of Nigeria such as parts of the Middle Belt, results from dietary iodine deficiency and remains an important preventable public health problem.

Other causes include autoimmune disease (Graves' disease, Hashimoto's thyroiditis), dyshormonogenesis, and goitrogens in food such as cassava, which contains cyanogenic glycosides that interfere with iodine uptake. Sporadic goitre occurs without a clear cause. Classification guides management: diffuse goitres are often medically managed while large nodular goitres frequently require surgery.

Fill in the blank: Endemic goitre results from dietary _____ deficiency. Cassava is a dietary _____ that can contribute to goitre formation.

3.2.2 Simple and multinodular goitre

Simple goitre is a diffuse, non-toxic enlargement without nodularity, usually euthyroid, and often managed conservatively with observation or iodine supplementation where deficiency is confirmed. Over time, simple goitres may become nodular through repeated cycles of hyperplasia and involution, producing a multinodular goitre (MNG) with nodules of varying size and functional activity.

Multinodular goitre may be euthyroid or progress to toxic MNG with autonomous nodules causing hyperthyroidism. Complications include compressive symptoms (dysphagia, dyspnoea, stridor from tracheal compression), retrosternal extension, and, rarely, malignant transformation. Surgery (usually total or near-total thyroidectomy) is indicated for compressive symptoms, cosmesis, suspicion of malignancy, or toxic MNG.

Fill in the blank: A multinodular goitre with autonomous nodules causing hyperthyroidism is termed _____. Compression of the trachea by a large goitre may cause _____.

3.2.3 Investigation of a solitary thyroid nodule

Every solitary thyroid nodule requires evaluation to exclude malignancy. Serum TSH is checked first: a suppressed TSH suggests a hyperfunctioning nodule, which is investigated with a radioisotope scan rather than biopsy, as hot nodules are rarely malignant. Ultrasound assesses size, composition, margins, and suspicious features such as microcalcifications and irregular margins.

Fine needle aspiration cytology (FNAC) is the investigation of choice for a nodule with a normal or elevated TSH, reported using the Bethesda system (categories I to VI, ranging from non-diagnostic to malignant). Indeterminate cytology (Bethesda III or IV) may require diagnostic lobectomy. Risk factors raising suspicion include age under 20 or over 60, male sex, and prior neck irradiation.

Fill in the blank: A thyroid nodule with a suppressed TSH is investigated with a _____ rather than biopsy. FNAC results are reported using the _____ system.

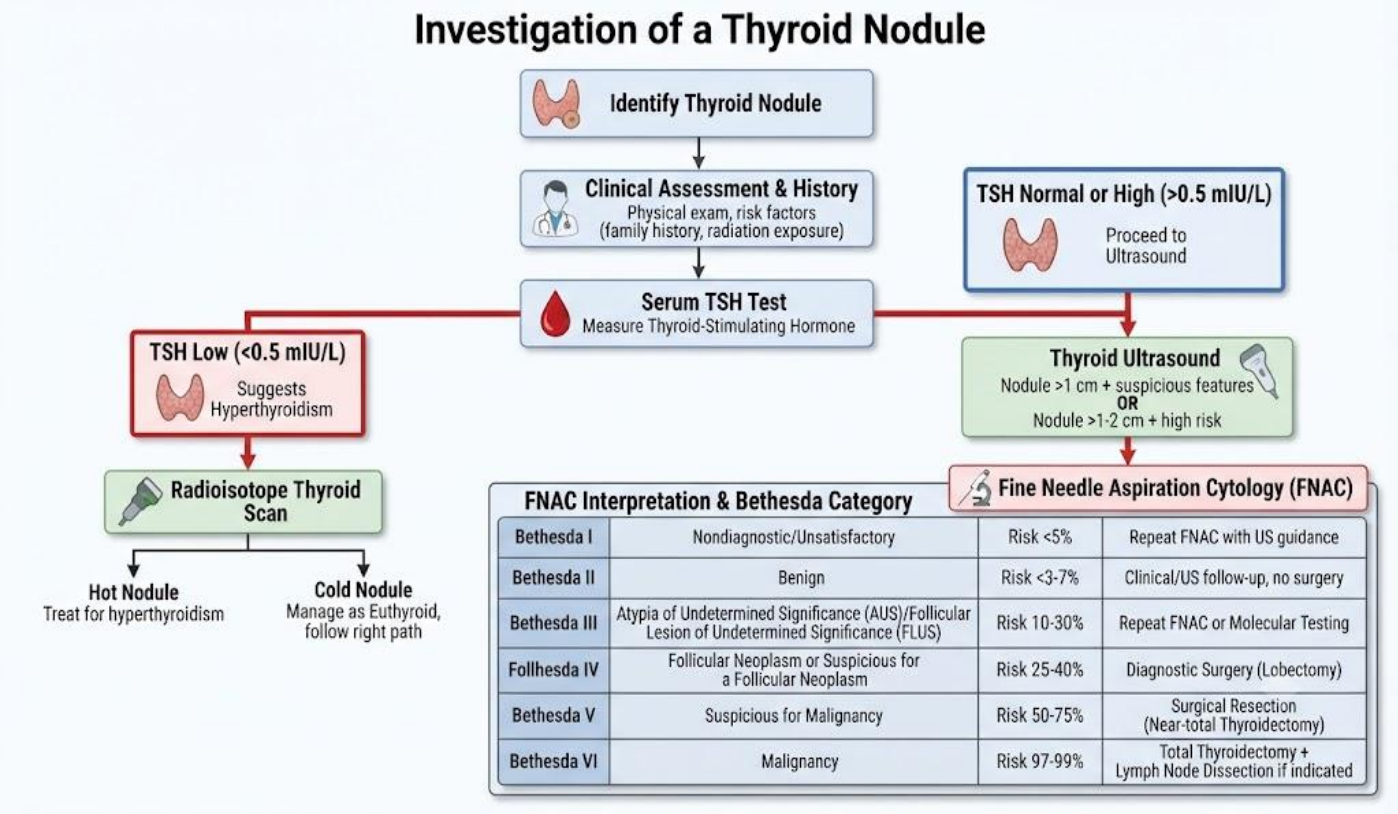

Figure 3.2: Classification of goitre and investigation pathway for a solitary thyroid nodule.

Pilot questions 3.2

6. Classify goitre and describe the causes of endemic goitre in Nigeria. (Linked to SLO 3.2)
7. Distinguish simple goitre from multinodular goitre and describe how the latter arises. (Linked to SLO 3.2)
8. List the indications for surgery in a patient with multinodular goitre. (Linked to SLO 3.2)
9. Describe the stepwise investigation of a solitary thyroid nodule. (Linked to SLO 3.2)
10. Explain the Bethesda system for reporting thyroid FNAC and its clinical implications. (Linked to SLO 3.2)**3.3 Hyperthyroidism**

3.3.1 Graves' disease

Graves' disease is the most common cause of hyperthyroidism, caused by circulating thyroid-stimulating immunoglobulins (TSIs) that bind and activate the TSH receptor. It typically affects women aged 20 to 40 and presents with a diffuse goitre, symptoms of thyrotoxicosis (weight loss, heat intolerance, palpitations, tremor, and anxiety), and specific extra-thyroidal features including ophthalmopathy and pretibial myxoedema.

Management options are antithyroid drugs (carbimazole or propylthiouracil), radioactive iodine ablation, and surgery (total thyroidectomy). Antithyroid drugs are first-line in most patients; surgery is preferred for large goitres, suspicion of malignancy, severe ophthalmopathy, or patient preference, and requires the patient to be rendered euthyroid preoperatively to reduce the risk of thyroid storm.

Fill in the blank: Graves' disease is caused by thyroid-stimulating _____ that activate the TSH receptor. Patients must be rendered _____ before thyroid surgery to reduce the risk of thyroid storm.

3.3.2 Toxic multinodular goitre and toxic adenoma

Toxic multinodular goitre (Plummer's disease) arises when one or more nodules within a longstanding multinodular goitre become autonomously functioning, most commonly in older patients. Cardiac manifestations such as atrial fibrillation may predominate over classic thyrotoxic features. Radioisotope scanning shows patchy uptake with multiple hot and cold areas, distinguishing it from the diffuse uptake of Graves' disease.

Toxic adenoma is a single autonomously functioning nodule causing hyperthyroidism, confirmed by a hot nodule on radioisotope scan with suppression of the remaining gland. Management for both conditions is surgery (usually total thyroidectomy for toxic MNG, or lobectomy for a solitary toxic adenoma) or radioactive iodine, as antithyroid drugs alone rarely achieve remission in autonomous nodules.

Fill in the blank: Toxic multinodular goitre is also known as _____ disease. A single autonomously functioning hot nodule is termed a toxic _____.

3.3.3 Thyrotoxic crisis (thyroid storm)

Thyroid storm is a life-threatening exacerbation of thyrotoxicosis, precipitated by infection, surgery, trauma, or an inadequately prepared thyroidectomy. It presents with severe hyperpyrexia, tachycardia or arrhythmia, agitation or confusion progressing to coma, and cardiac failure. The Burch-Wartofsky scoring system aids diagnosis when the presentation is unclear.

Management is emergency and includes supportive care (cooling, IV fluids, and oxygen), beta-blockers (propranolol) for adrenergic symptoms, high-dose antithyroid drugs (propylthiouracil preferred, as it also blocks peripheral T4 to T3 conversion), iodine solution given after

antithyroid drugs to block hormone release, and hydrocortisone to reduce peripheral conversion and cover relative adrenal insufficiency.

Fill in the blank: Thyroid storm may be precipitated by an inadequately _____ thyroidectomy. The scoring system used to aid diagnosis of thyroid storm is the _____ system.

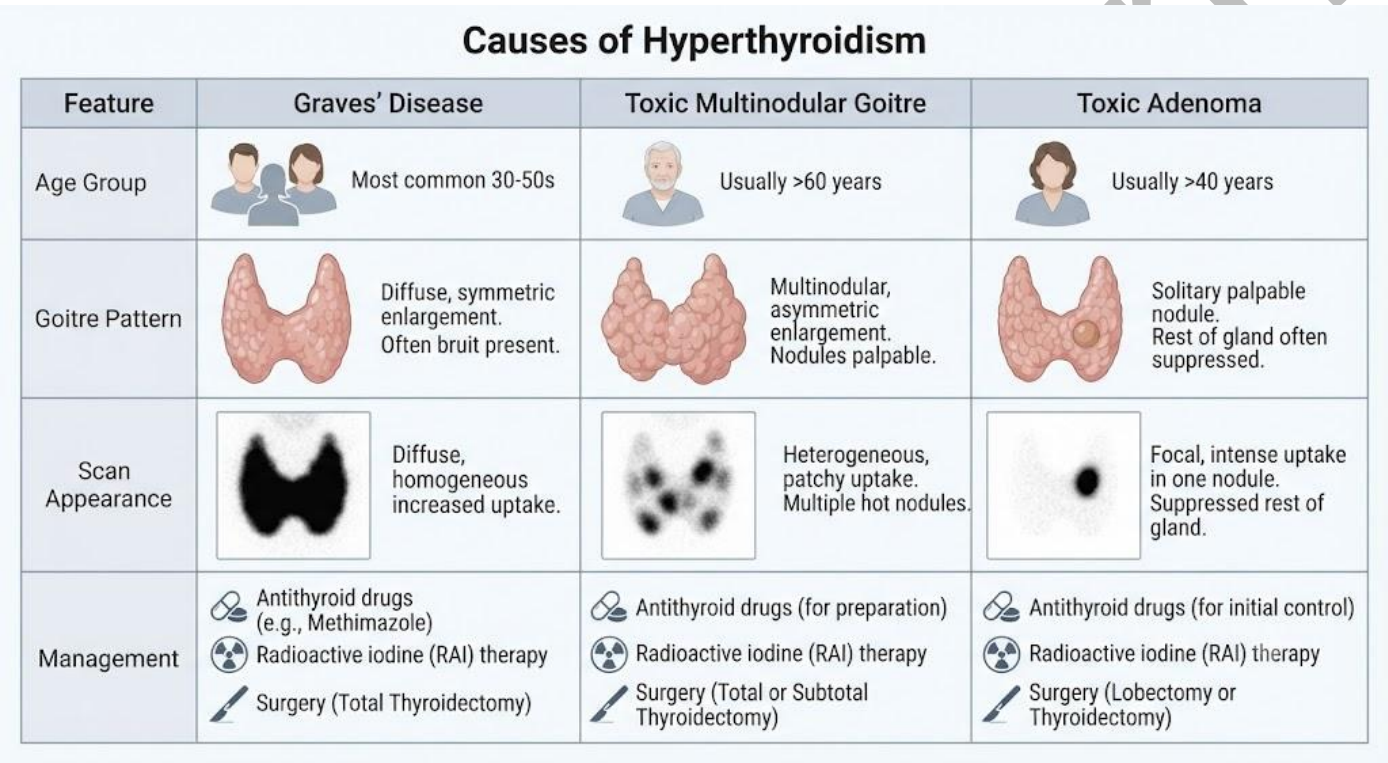

Figure 3.3: Comparison of the causes and management of hyperthyroidism.

Pilot questions 3.3

11. Describe the pathophysiology and clinical features of Graves' disease. (Linked to SLO 3.3)
12. Compare the management options for Graves' disease. (Linked to SLO 3.3)
13. Distinguish toxic multinodular goitre from toxic adenoma in presentation and investigation. (Linked to SLO 3.3)
14. Describe the precipitating factors and clinical features of thyroid storm. (Linked to SLO 3.3)
15. Outline the emergency management of thyroid storm. (Linked to SLO 3.3)

3.4 Hypothyroidism, Thyroiditis, and Thyroid Malignancy

3.4.1 Hypothyroidism and myxoedema

Hypothyroidism most commonly results from autoimmune thyroiditis (Hashimoto's disease), iodine deficiency, prior thyroidectomy, or radioactive iodine treatment. Clinical features include lethargy, cold intolerance, weight gain, dry skin, hair thinning, constipation, and bradycardia. Diagnosis is confirmed by an elevated TSH with a low free T4, and elevated anti-TPO antibodies support an autoimmune cause.

Myxoedema refers to severe, longstanding hypothyroidism with non-pitting oedema from dermal mucopolysaccharide deposition. Myxoedema coma is a rare, life-threatening decompensated state with hypothermia, bradycardia, and reduced consciousness, requiring intravenous levothyroxine, hydrocortisone (to cover coexisting adrenal insufficiency), and supportive care. Treatment of uncomplicated hypothyroidism is lifelong oral levothyroxine replacement.

Fill in the blank: Hypothyroidism is confirmed by an elevated TSH with a low _____. Myxoedema coma requires intravenous levothyroxine and _____ to cover adrenal insufficiency.

3.4.2 Acute, subacute, and chronic thyroiditis

Acute suppurative thyroiditis is a rare bacterial infection presenting with fever and a tender, erythematous swelling, treated with antibiotics and drainage of any abscess. Subacute (de Quervain's) thyroiditis follows a viral illness and presents with a painful, tender thyroid, transient hyperthyroidism followed by hypothyroidism, and a raised erythrocyte sedimentation rate, managed with analgesia and, if severe, corticosteroids.

Hashimoto's thyroiditis is the most common cause of hypothyroidism in iodine-sufficient regions, an autoimmune process with lymphocytic infiltration, a firm rubbery goitre, and high anti-TPO antibody titres, treated with levothyroxine replacement. Riedel's thyroiditis is rare, causing dense fibrosis that mimics anaplastic carcinoma clinically and may require biopsy to exclude malignancy.

Fill in the blank: Subacute thyroiditis is also known as _____ thyroiditis and follows a viral illness. The most common cause of hypothyroidism in iodine-sufficient regions is _____ thyroiditis.

3.4.3 Classification and management of thyroid carcinoma

Thyroid carcinoma is classified as papillary (most common, approximately 80 percent, spreads via lymphatics, good prognosis, associated with prior radiation), follicular (spreads haematogenously, diagnosis requires capsular or vascular invasion not seen on FNAC alone), medullary (arises from parafollicular C cells, secretes calcitonin, associated with MEN 2), and anaplastic (rare, aggressive, poor prognosis, mainly in the elderly).

Management for papillary and follicular (differentiated) carcinoma is total thyroidectomy with or without radioactive iodine ablation and lifelong TSH-suppressive levothyroxine. Medullary carcinoma requires total thyroidectomy with central neck dissection and screening relatives for MEN 2 mutations. Anaplastic carcinoma is usually managed palliatively, as surgery is rarely curative at presentation.

Fill in the blank: The most common type of thyroid carcinoma is _____ carcinoma. Medullary thyroid carcinoma secretes _____ and is associated with MEN _____.

Thyroiditis and Thyroid Carcinoma

A. Thyroiditis Subtypes: Comparison of Cause and Course

Subtype	Cause	Clinical Course	Key Features
1. **Hashimoto's Thyroiditis** (Autoimmune)	Autoimmune destruction (anti-TPO, anti-Tg antibodies)	Gradual progression to hypothyroidism	Diffuse enlargement, lymphocytic infiltration, common in women
2. **Subacute (De Quervain's) Thyroiditis** (Viral)	Post-viral inflammatory response	Acute, self-limiting (weeks to months)	Painful goiter, hyperthyroidism followed by transient hypothyroidism, then euthyroid
3. **Silent (Painless) Thyroiditis** (Autoimmune)	Autoimmune variant, postpartum or sporadic	Triphasic (hyper -> hypo -> euthyroid) over 3-6 months	Painless goiter, similar histology to Hashimoto's
4. **Suppurative (Infective) Thyroiditis** (Bacterial)	Bacterial infection (e.g., Staph aureus), often via a pyriform sinus fistula	Acute, severe infection	Painful, fluctuant mass, fever, requires antibiotics/drainage
5. **Riedel's Thyroiditis** (Fibrous)	Fibrous replacement of gland (IgG4-related?)	Progressive, invasive fibrosis	Rock-hard goiter, compresses trachea/ esophagus, "invasive fibrous thyroiditis"

B. Thyroid Carcinoma: Origin, Spread, and Prognosis

Carcinoma Type		Cell of Origin	Spread Pattern	Prognosis
1. **Papillary Thyroid Carcinoma (PTC)**	Papillary projection (Orphan Annie eyes, psammoma bodies)	Follicular cells	Primarily lymphatic (cervical lymph nodes), slow	Excellent (>95% 10-year survival)
2. **Follicular Thyroid Carcinoma (FTC)**		Follicular cells	Primarily hematogenous (distant sites: lungs, bone), slow	Good (>85% 10-year survival), requires capsular invasion for diagnosis
3. **Medullary Thyroid Carcinoma (MTC)**	C-cells in neuroendocrine pattern, amyloid stroma	Parafollicular C-cells	Both lymphatic and hematogenous, early	Variable (MEN2 variants worse), secretes Calcitonin
4. **Anaplastic Thyroid Carcinoma (ATC)**		Dedifferentiation of follicular cells	Rapid local invasion and distant metastases	Extremely poor (<6 months median survival), rapidly fatal

Figure 3.4: Comparison of thyroiditis subtypes and classification of thyroid carcinoma.

Pilot questions 3.4

1. Describe the causes and clinical features of hypothyroidism and myxoedema coma. (Linked to SLO 3.4)
2. Compare acute suppurative, subacute, and Hashimoto's thyroiditis. (Linked to SLO 3.4)
3. Classify thyroid carcinoma and describe the features of each histological type. (Linked to SLO 3.4)
4. Describe the surgical management of papillary and medullary thyroid carcinoma. (Linked to SLO 3.4)
5. Explain why anaplastic thyroid carcinoma has a poor prognosis. (Linked to SLO 3.4)

3.5 Thyroid and Parathyroid Surgery

3.5.1 Indications and types of thyroidectomy

Indications for thyroidectomy include compressive symptoms, cosmetic concerns, suspected or confirmed malignancy, toxic goitre unresponsive to or unsuitable for medical therapy, and retrosternal extension. Types of thyroidectomy are lobectomy (removal of one lobe, for a diagnostic or unilateral benign nodule), subtotal thyroidectomy, near-total thyroidectomy, and total thyroidectomy (for malignancy, Graves' disease, and bilateral MNG).

Preoperative preparation includes confirming euthyroid status (to avoid intraoperative thyroid storm), vocal cord assessment by indirect laryngoscopy, and imaging to assess retrosternal extension. Informed consent must cover the specific risks of recurrent laryngeal nerve injury, hypoparathyroidism, and bleeding, which are central to the surgical decision-making process.

Fill in the blank: Removal of one thyroid lobe is termed a _____. Preoperative assessment of vocal cord function is by indirect _____.

3.5.2 Complications of thyroid surgery

Early complications include haemorrhage with haematoma formation, which can cause airway compression and is a surgical emergency requiring immediate wound reopening at the bedside, and recurrent laryngeal nerve injury, which causes hoarseness (unilateral) or stridor and airway obstruction (bilateral). Injury to the external branch of the superior laryngeal nerve impairs voice pitch.

Hypoparathyroidism from inadvertent removal or devascularisation of the parathyroid glands causes hypocalcaemia, presenting with perioral tingling, Chvostek's and Trousseau's signs, and, if severe, tetany or laryngospasm, managed with oral or intravenous calcium and vitamin D analogues. Other complications include wound infection, hypertrophic or keloid scarring, and thyroid storm in inadequately prepared patients.

Fill in the blank: Postoperative haematoma causing airway compression requires immediate bedside _____. Hypocalcaemia after thyroidectomy is assessed using Chvostek's and _____ signs.

3.5.3 Parathyroid anatomy and hyperparathyroidism

The parathyroid glands are usually four in number, located posterior to the thyroid lobes, and secrete parathyroid hormone (PTH), which raises serum calcium by increasing bone resorption, renal calcium reabsorption, and activation of vitamin D. Primary hyperparathyroidism is most commonly caused by a single parathyroid adenoma, presenting with hypercalcaemia, described by the mnemonic bones, stones, abdominal groans, and psychiatric moans.

Investigation shows raised serum calcium with an inappropriately raised or normal PTH, localised preoperatively by sestamibi scan and ultrasound. Management is parathyroidectomy for

symptomatic disease, marked hypercalcaemia, renal impairment, or reduced bone density. Secondary hyperparathyroidism (from chronic kidney disease) and tertiary hyperparathyroidism (autonomous secretion after prolonged secondary disease) are also encountered in Nigerian tertiary centres.

Fill in the blank: Primary hyperparathyroidism is most commonly caused by a parathyroid _____. Localisation of a parathyroid adenoma preoperatively uses _____ scan and ultrasound.

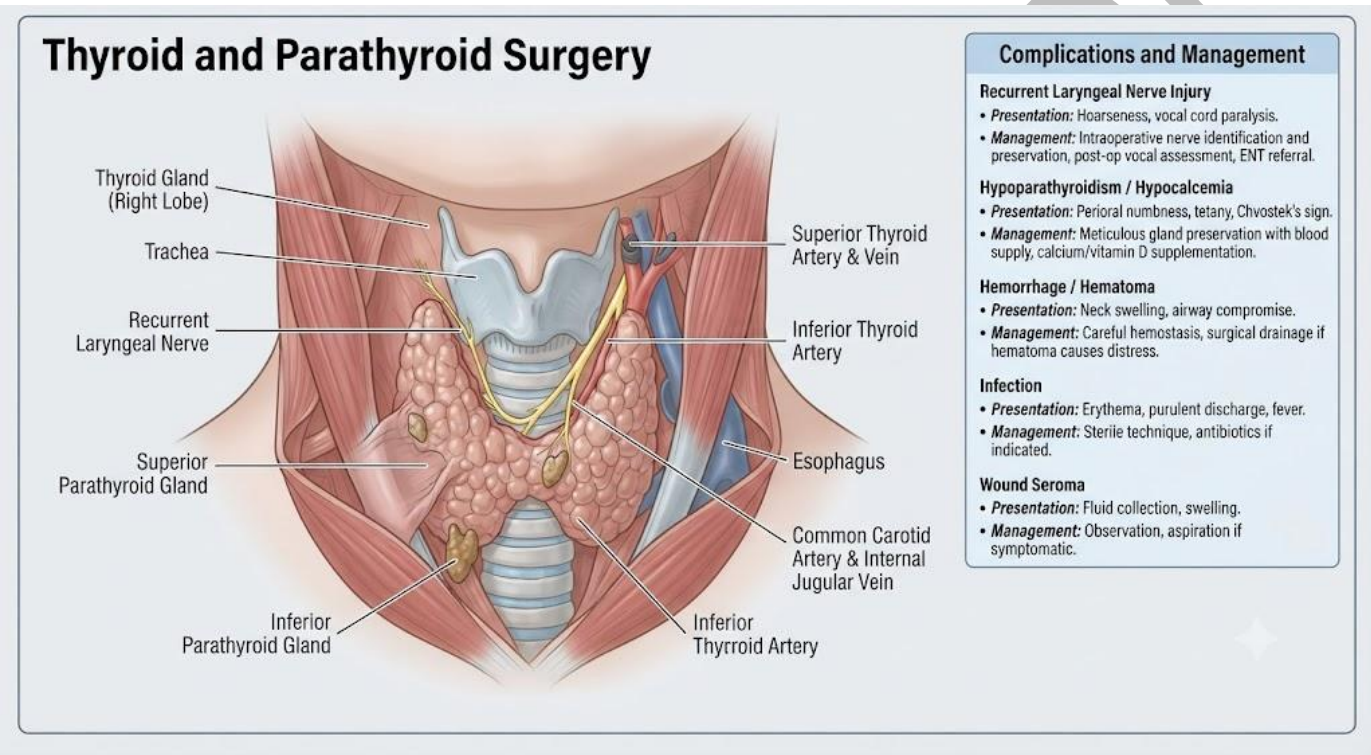

Figure 3.5: Operative anatomy in thyroidectomy and summary of surgical complications.

Pilot questions 3.5

1. List the indications for thyroidectomy and describe the types of thyroidectomy performed. (Linked to SLO 3.5)
2. Describe the preoperative preparation required before thyroid surgery. (Linked to SLO 3.5)
3. Describe the early complications of thyroidectomy and their emergency management. (Linked to SLO 3.5)
4. Describe the clinical features and management of post-thyroidectomy hypocalcaemia. (Linked to SLO 3.5)

5. Describe the clinical features, investigation, and management of primary hyperparathyroidism. (Linked to SLO 3.5)

3.6 Adrenal and Other Endocrine Surgical Disorders

3.6.1 Surgical anatomy and disorders of the adrenal gland

The adrenal glands sit on the superior poles of the kidneys, each with a cortex (secreting cortisol, aldosterone, and androgens in three zones) and a medulla (secreting catecholamines). Cushing's syndrome results from excess cortisol, from an adrenal adenoma, pituitary adenoma (Cushing's disease), or ectopic ACTH secretion, presenting with central obesity, moon face, striae, and hypertension.

Conn's syndrome (primary aldosteronism) results from an aldosterone-secreting adenoma or bilateral adrenal hyperplasia, causing hypertension with hypokalaemia. Investigation uses the aldosterone-renin ratio, and management is adrenalectomy for a unilateral adenoma or spironolactone for bilateral hyperplasia. Adrenal incidentalomas, found on imaging for unrelated reasons, require functional and malignancy screening before deciding on surveillance or surgery.

Fill in the blank: Excess cortisol production causes _____ syndrome. Primary aldosteronism is also known as _____ syndrome.

3.6.2 Phaeochromocytoma

Phaeochromocytoma is a catecholamine-secreting tumour of the adrenal medulla (or extra-adrenal paraganglioma), classically presenting with episodic headache, palpitations, sweating, and severe hypertension, described by the rule of tens (approximately 10 percent bilateral, extra-adrenal, malignant, and familial). Diagnosis is by plasma or urinary metanephrines, followed by CT or MRI for localisation.

Preoperative preparation is essential and life-saving: alpha-blockade (phenoxybenzamine) is started first, followed by beta-blockade only after adequate alpha-blockade to prevent unopposed alpha-adrenergic crisis, alongside volume expansion. Surgery (laparoscopic or open adrenalectomy) follows once blood pressure and heart rate are controlled, as unprepared surgery risks fatal hypertensive crisis.

Fill in the blank: In phaeochromocytoma, _____-blockade must always precede beta-blockade before surgery. Diagnosis of phaeochromocytoma is confirmed by plasma or urinary _____.

3.6.3 Multiple endocrine neoplasia (MEN) syndromes

Multiple endocrine neoplasia syndromes are autosomal dominant disorders causing tumours in multiple endocrine glands. MEN 1 (parathyroid, pituitary, and pancreatic tumours) results from a

MEN1 gene mutation. MEN 2A (medullary thyroid carcinoma, phaeochromocytoma, and primary hyperparathyroidism) and MEN 2B (medullary thyroid carcinoma, phaeochromocytoma, and mucosal neuromas with a marfanoid habitus) result from RET proto-oncogene mutations.

Clinical importance lies in screening: family members of a patient with medullary thyroid carcinoma should undergo RET mutation testing, and confirmed carriers are offered prophylactic thyroidectomy at an age determined by mutation risk category. Any patient with medullary thyroid carcinoma should also be screened for phaeochromocytoma before thyroid surgery, as an unrecognised phaeochromocytoma can precipitate a fatal intraoperative crisis.

Fill in the blank: MEN 2A and MEN 2B result from mutations in the _____ proto-oncogene. A patient with medullary thyroid carcinoma must be screened for _____ before thyroid surgery.

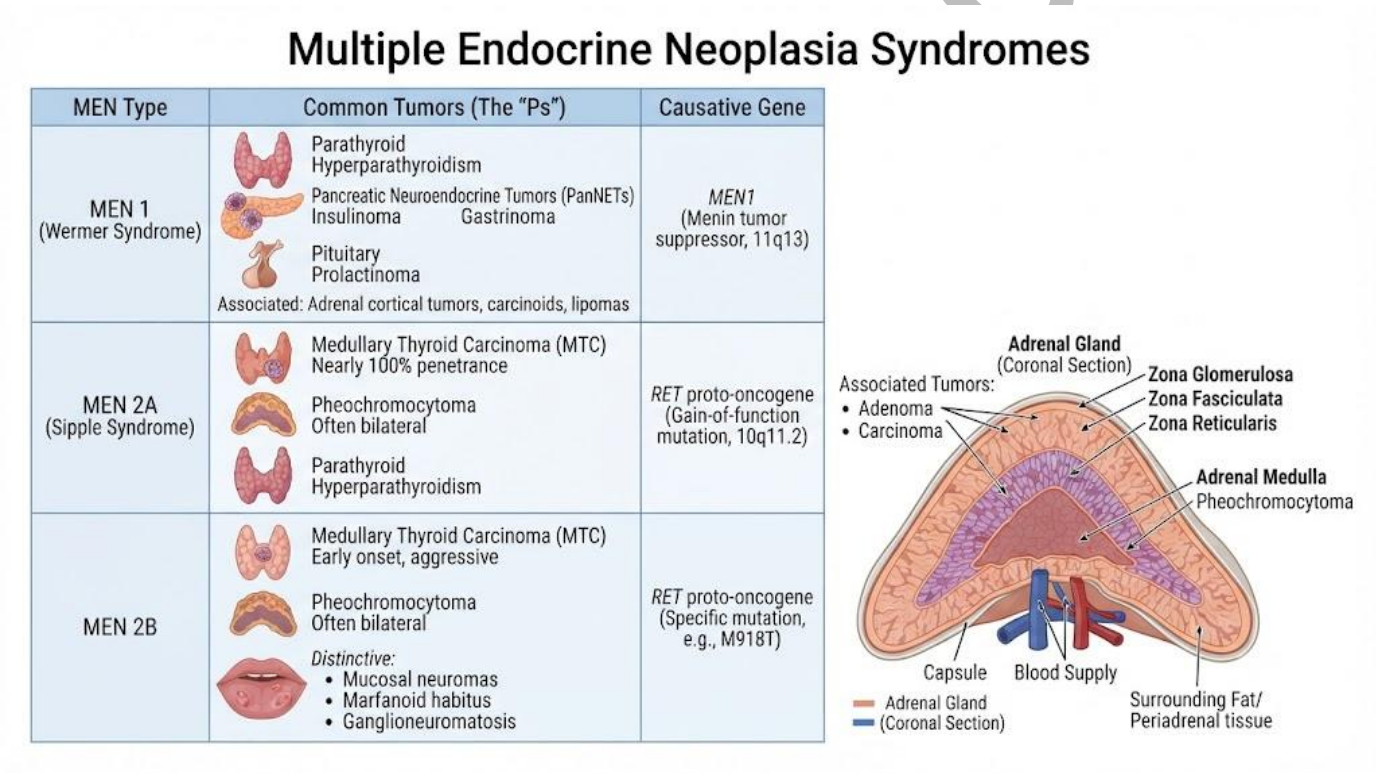

Figure 3.6: Adrenal gland anatomy and classification of multiple endocrine neoplasia syndromes.

Pilot questions 3.6

1. Describe the causes and clinical features of Cushing's syndrome and Conn's syndrome. (Linked to SLO 3.6)
2. Describe the clinical features and diagnosis of phaeochromocytoma. (Linked to SLO 3.6)
3. Explain the correct order of alpha- and beta-blockade before phaeochromocytoma surgery and why it matters. (Linked to SLO 3.6)

4. Compare the tumour associations of MEN 1, MEN 2A, and MEN 2B. (Linked to SLO 3.6)
5. Explain why patients with medullary thyroid carcinoma must be screened for phaeochromocytoma. (Linked to SLO 3.6)

Series Summary

This Series has covered the surgical anatomy, physiology, and clinical assessment of the thyroid gland, the classification and investigation of goitre and thyroid nodules, and the causes and management of hyperthyroidism, hypothyroidism, thyroiditis, and thyroid carcinoma. It has also addressed the indications, technique, and complications of thyroid and parathyroid surgery, together with the surgical disorders of the adrenal gland and the multiple endocrine neoplasia syndromes that link thyroid and adrenal pathology.

You should now be able to assess a patient with a thyroid swelling and investigate it appropriately, distinguish the causes of hyperthyroidism and hypothyroidism and manage thyrotoxic crisis, classify thyroid carcinoma and outline its surgical management, anticipate and manage the complications of thyroid and parathyroid surgery, and recognise the surgical endocrine disorders of the adrenal gland, including safe preoperative preparation for phaeochromocytoma (Linked to SLOs 3.1 to 3.6).

Glossary of Terms

6. **Bethesda system:** a standardised reporting system for thyroid FNAC cytology, with categories from non-diagnostic to malignant, guiding management decisions.
7. **Chvostek's sign:** facial muscle twitching elicited by tapping the facial nerve, a clinical sign of hypocalcaemia.
8. **Euthyroid:** a state of normal thyroid function, neither hyperthyroid nor hypothyroid.
9. **Goitre:** any enlargement of the thyroid gland, whether diffuse or nodular, toxic or non-toxic.
10. **Graves' disease:** an autoimmune cause of hyperthyroidism due to thyroid-stimulating immunoglobulins activating the TSH receptor.
11. **Hashimoto's thyroiditis:** an autoimmune thyroiditis and the most common cause of hypothyroidism in iodine-sufficient regions.

12. **Myxoedema:** severe hypothyroidism with non-pitting oedema from dermal mucopolysaccharide deposition.
13. **Phaeochromocytoma:** a catecholamine-secreting tumour of the adrenal medulla or extra-adrenal paraganglion tissue.
14. **Rule of tens:** the classic teaching that approximately 10 percent of phaeochromocytomas are bilateral, extra-adrenal, malignant, and familial.
15. **Thyroid storm:** a life-threatening exacerbation of thyrotoxicosis with hyperpyrexia, tachyarrhythmia, and altered consciousness.
16. **Toxic multinodular goitre:** a multinodular goitre with autonomously functioning nodules causing hyperthyroidism, also called Plummer's disease.
17. **Trousseau's sign:** carpal spasm induced by inflating a blood pressure cuff above systolic pressure, a clinical sign of hypocalcaemia.

Concept Check Questions [Series 3]

Objective questions

11. The recurrent laryngeal nerve runs in the _____ groove and must be identified during thyroidectomy.
12. Toxic multinodular goitre is also known as _____ disease.
13. The scoring system used to aid diagnosis of thyroid storm is the _____ system.
14. Medullary thyroid carcinoma secretes _____ and is associated with MEN _____.
15. In phaeochromocytoma, _____-blockade must always precede beta-blockade before surgery.

Short-answer questions

1. Describe the hypothalamic-pituitary-thyroid axis and its feedback mechanism. (Linked to SLO 3.1)
2. Describe the stepwise investigation of a solitary thyroid nodule. (Linked to SLO 3.2)
3. Compare toxic multinodular goitre and toxic adenoma. (Linked to SLO 3.3)

4. Describe the clinical features and management of post-thyroidectomy hypocalcaemia. (Linked to SLO 3.5)
5. Compare the tumour associations of MEN 2A and MEN 2B. (Linked to SLO 3.6)

Long-answer questions

1. Describe the causes, clinical features, and management of hyperthyroidism, including the emergency management of thyroid storm. (Linked to SLO 3.3)
2. Classify thyroid carcinoma and describe the surgical management of each histological type. (Linked to SLO 3.4)
3. Describe the indications, types, and complications of thyroidectomy. (Linked to SLO 3.5)
4. Describe the clinical features, diagnosis, and preoperative preparation for phaeochromocytoma surgery. (Linked to SLO 3.6)
5. Compare the causes and clinical presentations of hypothyroidism and hyperthyroidism. (Linked to SLO 3.3, SLO 3.4)

Answers to Concept Check Questions [Series 3]

Objective questions

1. Tracheo-oesophageal.
2. Plummer's.
3. Burch-Wartofsky.
4. Calcitonin; 2.
5. Alpha.

Short-answer questions

6. TRH from the hypothalamus stimulates TSH release from the pituitary, which stimulates thyroid hormone synthesis and release. T3 and T4 negatively feedback to suppress TRH and TSH, maintaining hormone levels within a narrow range.

7. Serum TSH is checked first. A suppressed TSH prompts a radioisotope scan (hot nodules are rarely malignant). A normal or raised TSH prompts ultrasound and FNAC, reported using the Bethesda system.
8. Toxic MNG arises from autonomous nodules within a longstanding goitre, usually in older patients, with patchy uptake on scan. Toxic adenoma is a single hot nodule with suppression of the rest of the gland on scan.
9. Hypocalcaemia presents with perioral tingling and Chvostek's or Trousseau's signs, and if severe, tetany. Management is oral or intravenous calcium with vitamin D analogues depending on severity.
10. MEN 2A causes medullary thyroid carcinoma, phaeochromocytoma, and primary hyperparathyroidism. MEN 2B causes medullary thyroid carcinoma, phaeochromocytoma, and mucosal neuromas with a marfanoid habitus, but not hyperparathyroidism.

Long-answer questions

11. **Basic response:** Hyperthyroidism causes include Graves' disease, toxic MNG, and toxic adenoma, presenting with weight loss, heat intolerance, tremor, and tachycardia. Management is antithyroid drugs, radioactive iodine, or surgery.

Value-added response: Thyroid storm is an emergency precipitated by infection, surgery, or inadequate preparation, managed with cooling, beta-blockers, high-dose propylthiouracil, iodine given after antithyroid drugs, and hydrocortisone.

12. **Basic response:** Papillary and follicular carcinoma are differentiated types managed with total thyroidectomy, radioactive iodine, and TSH suppression. Medullary carcinoma requires total thyroidectomy with central neck dissection.

Value-added response: Anaplastic carcinoma is aggressive and usually managed palliatively. Relatives of medullary carcinoma patients should undergo RET mutation testing to guide prophylactic thyroidectomy timing.

13. **Basic response:** Indications for thyroidectomy include compression, malignancy, cosmesis, and toxic goitre unresponsive to medical therapy. Types range from lobectomy to total thyroidectomy depending on the underlying disease.

Value-added response: Complications include haemorrhage with airway compression requiring emergency bedside reopening, recurrent laryngeal nerve injury, and hypoparathyroidism causing hypocalcaemia, all central to informed consent.

14. **Basic response:** Phaeochromocytoma presents with episodic headache, palpitations, sweating, and hypertension, diagnosed by plasma or urinary metanephrines and localised by CT or MRI.

Value-added response: Preoperative alpha-blockade with phenoxybenzamine must precede beta-blockade to prevent unopposed alpha-adrenergic crisis, alongside volume expansion, before adrenalectomy is undertaken.

15. **Basic response:** Hypothyroidism causes lethargy, cold intolerance, weight gain, and bradycardia, with a raised TSH and low free T4. Hyperthyroidism causes weight loss, heat intolerance, tremor, and tachycardia, with a suppressed TSH.

Value-added response: Hashimoto's thyroiditis is the leading cause of hypothyroidism in iodine-sufficient regions, while Graves' disease is the leading cause of hyperthyroidism; both are autoimmune but with opposite receptor effects.

Answers to Pilot Questions [Series 3]

Part 3.1

1. The thyroid lies anterior to the trachea with two lobes joined by an isthmus. The recurrent laryngeal nerves run in the tracheo-oesophageal groove and the parathyroid glands lie posterior to the thyroid capsule, both requiring careful identification during surgery.
2. Iodide trapping by the sodium-iodide symporter, organification onto thyroglobulin, coupling of MIT and DIT to form T3 and T4, and hormone release, with peripheral conversion of T4 to the more active T3.
3. TRH from the hypothalamus stimulates pituitary TSH release, which stimulates thyroid hormone synthesis. T3 and T4 negatively feedback on the hypothalamus and pituitary to suppress further TRH and TSH release.
4. History covers growth rate, compressive symptoms, and family history. Examination covers inspection, palpation for consistency and mobility, auscultation for a bruit, and assessment of cervical lymph nodes.
5. Serum TSH is first-line, followed by free T4 or T3 if abnormal. Ultrasound is first-line imaging for nodules, followed by FNAC reported using the Bethesda system.

Part 3.2

- Goitre is classified as diffuse or nodular, and toxic, euthyroid, or non-toxic. Endemic goitre results from dietary iodine deficiency, common in iodine-deficient parts of Nigeria, and worsened by goitrogens such as cassava.
- Simple goitre is diffuse and non-toxic. Multinodular goitre develops from repeated hyperplasia and involution cycles, producing nodules of varying size and function.
- Indications include compressive symptoms, cosmetic concerns, suspicion of malignancy, and toxic MNG unresponsive to or unsuitable for medical therapy.
- Serum TSH is checked first. Suppressed TSH prompts a radioisotope scan; normal or raised TSH prompts ultrasound followed by FNAC.
- The Bethesda system reports FNAC in categories I to VI, from non-diagnostic to malignant, guiding whether observation, repeat FNAC, diagnostic lobectomy, or total thyroidectomy is appropriate.

Part 3.3

- Graves' disease is caused by TSIs activating the TSH receptor, causing diffuse goitre, thyrotoxic symptoms, ophthalmopathy, and pretibial myxoedema, typically in women aged 20 to 40.
- Options are antithyroid drugs (first-line), radioactive iodine, and surgery. Surgery suits large goitres, suspected malignancy, severe ophthalmopathy, or patient preference, with preoperative euthyroid control.
- Toxic MNG shows patchy scan uptake in older patients, often with atrial fibrillation. Toxic adenoma shows a single hot nodule with suppression of the rest of the gland.
- Precipitants include infection, surgery, trauma, and inadequately prepared thyroidectomy, presenting with hyperpyrexia, tachyarrhythmia, and altered consciousness.
- Cooling and IV fluids, propranolol, high-dose propylthiouracil, iodine solution after antithyroid drugs, and hydrocortisone for peripheral conversion blockade and adrenal cover.

Part 3.4

- Causes include autoimmune thyroiditis, iodine deficiency, and prior thyroid ablation, with lethargy, cold intolerance, and weight gain. Myxoedema coma adds hypothermia, bradycardia, and reduced consciousness, requiring IV levothyroxine and hydrocortisone.
- Acute suppurative thyroiditis is bacterial with fever and tenderness. Subacute thyroiditis follows viral illness with pain and biphasic thyroid function. Hashimoto's is autoimmune with a firm goitre and high anti-TPO titres.
- Papillary (most common, lymphatic spread), follicular (haematogenous spread), medullary (from C cells, secretes calcitonin, MEN 2), and anaplastic (aggressive, elderly, poor prognosis).
- Papillary carcinoma is managed with total thyroidectomy, with or without radioactive iodine. Medullary carcinoma requires total thyroidectomy with central neck dissection and screening relatives for RET mutations.
- Anaplastic carcinoma presents late in elderly patients with rapid local invasion, making curative surgery rarely possible, so management is usually palliative.

Part 3.5

- Compressive symptoms, cosmetic concerns, suspected malignancy, and toxic goitre unresponsive to medical therapy. Types range from lobectomy to total thyroidectomy depending on the indication.
- Confirming euthyroid status, vocal cord assessment by indirect laryngoscopy, imaging for retrosternal extension, and informed consent covering nerve injury, hypoparathyroidism, and bleeding risks.
- Haemorrhage with airway compression requires immediate bedside wound reopening. Recurrent laryngeal nerve injury causes hoarseness or, if bilateral, airway obstruction requiring urgent airway management.
- Hypocalcaemia presents with perioral tingling, Chvostek's and Trousseau's signs, and tetany if severe, managed with oral or intravenous calcium and vitamin D analogues.
- Hypercalcaemia with an inappropriately raised or normal PTH, localised by sestamibi scan, managed with parathyroidectomy for symptomatic or significant disease.

Part 3.6

1. Cushing's syndrome results from excess cortisol from an adrenal or pituitary adenoma or ectopic ACTH, causing central obesity and hypertension. Conn's syndrome results from excess aldosterone, causing hypertension with hypokalaemia.
2. Episodic headache, palpitations, sweating, and severe hypertension, diagnosed by plasma or urinary metanephrines and localised by CT or MRI.
3. Alpha-blockade with phenoxybenzamine must precede beta-blockade to avoid unopposed alpha-adrenergic vasoconstriction and hypertensive crisis if beta-blockade is given first.
4. MEN 1 involves parathyroid, pituitary, and pancreatic tumours from a MEN1 mutation. MEN 2A and MEN 2B involve medullary thyroid carcinoma and phaeochromocytoma from RET mutations, with MEN 2A adding hyperparathyroidism and MEN 2B adding mucosal neuromas.
5. An unrecognised phaeochromocytoma can cause a fatal intraoperative hypertensive crisis if thyroid surgery proceeds before it is excluded and, if present, adequately treated.

References and Attributions [Series 3]

Textual References

1. Bailey, H. and Love, R. J. M. (2018). Bailey and Love's Short Practice of Surgery (27th ed.). CRC Press.
2. Townsend, C. M., Beauchamp, R. D., Evers, B. M. and Mattox, K. L. (2017). Sabiston Textbook of Surgery (20th ed.). Elsevier.
3. Ross, D. S., Burch, H. B., Cooper, D. S. et al. (2016). American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism. Thyroid, 26(10), 1343 to 1421.
4. Lenders, J. W. M., Duh, Q. Y., Eisenhofer, G. et al. (2014). Phaeochromocytoma and Paraganglioma: An Endocrine Society Clinical Practice Guideline. Journal of Clinical Endocrinology and Metabolism, 99(6), 1915 to 1942.

Figures, Plates, Tables, and Boxes

1. Figure 3.1: Surgical anatomy of the thyroid and the hypothalamic-pituitary-thyroid axis. AI-generated using the prompt: "Create a professional two-panel medical diagram titled Thyroid Anatomy and Physiology. Left panel: anterior neck anatomy showing thyroid lobes, isthmus, thyroid arteries, recurrent laryngeal nerve, and parathyroid glands. Right panel: hypothalamic-pituitary-thyroid feedback loop with labelled hormones and arrows. Clean clinical surgical diagram style."
2. Figure 3.2: Classification of goitre and investigation pathway for a solitary thyroid nodule. AI-generated using the prompt: "Create a professional medical flowchart titled Investigation of a Thyroid Nodule, showing clinical assessment, TSH testing, branching pathways to radioisotope scan or ultrasound and FNAC, and Bethesda category outcomes. Clean clinical surgical diagram style."
3. Figure 3.3: Comparison of the causes and management of hyperthyroidism. AI-generated using the prompt: "Create a professional three-column comparison table diagram titled Causes of Hyperthyroidism, comparing Graves disease, toxic multinodular goitre, and toxic adenoma by age group, goitre pattern, scan appearance, and management. Clean clinical surgical diagram style."
4. Figure 3.4: Comparison of thyroiditis subtypes and classification of thyroid carcinoma. AI-generated using the prompt: "Create a professional two-panel medical diagram titled Thyroiditis and Thyroid Carcinoma, comparing thyroiditis subtypes by cause and course,

and classifying thyroid carcinoma types by origin, spread, and prognosis. Clean clinical surgical diagram style."

5. Figure 3.5: Operative anatomy in thyroidectomy and summary of surgical complications. AI-generated using the prompt: "Create a professional medical diagram titled Thyroid and Parathyroid Surgery, showing the operative field with the recurrent laryngeal nerve and parathyroid glands identified, plus an inset list of complications and management. Clean clinical surgical diagram style."
6. Figure 3.6: Adrenal gland anatomy and classification of multiple endocrine neoplasia syndromes. AI-generated using the prompt: "Create a professional medical diagram titled Multiple Endocrine Neoplasia Syndromes, with a comparison table of MEN 1, MEN 2A, and MEN 2B tumour associations and causative genes, plus a labelled adrenal gland cross-section. Clean clinical surgical diagram style."

Course code: SUG 402

Course title: General Surgery II

Course credit load: 5 Units

Series 4: Abdominal Surgical Conditions

1. **Part 4.1: Abdominal Wall Hernias I: Inguinal and Femoral**
    1. Sub Part 4.1.1: Anatomy of the inguinal and femoral canals
    2. Sub Part 4.1.2: Classification and clinical features of inguinal hernia
    3. Sub Part 4.1.3: Femoral hernia
2. **Part 4.2: Abdominal Wall Hernias II: Other Hernias**
    1. Sub Part 4.2.1: Umbilical and paraumbilical hernia
    2. Sub Part 4.2.2: Incisional and ventral hernia
    3. Sub Part 4.2.3: Rare hernias (obturator, Spigelian, lumbar)
3. **Part 4.3: Complications of Hernias and Principles of Repair**
    1. Sub Part 4.3.1: Irreducibility, obstruction, and strangulation
    2. Sub Part 4.3.2: Emergency management of strangulated hernia
    3. Sub Part 4.3.3: Principles of hernia repair (open and laparoscopic)
4. **Part 4.4: Peritonitis**
    1. Sub Part 4.4.1: Classification and pathophysiology of peritonitis
    2. Sub Part 4.4.2: Clinical features and diagnosis of peritonitis
    3. Sub Part 4.4.3: Management of peritonitis
5. **Part 4.5: Intra-abdominal Abscess and Gangrenous Bowel**
    1. Sub Part 4.5.1: Intra-abdominal and subphrenic abscess
    2. Sub Part 4.5.2: Causes and pathophysiology of bowel gangrene
    3. Sub Part 4.5.3: Management of gangrenous bowel
6. **Part 4.6: Fistula and Related Abdominal Conditions**
    1. Sub Part 4.6.1: Classification and causes of fistula

2. Sub Part 4.6.2: Enterocutaneous fistula
3. Sub Part 4.6.3: Principles of fistula management

Introduction

Abdominal surgical conditions form a core part of General Surgery II, encompassing abdominal wall hernias, peritonitis, intra-abdominal abscess, gangrenous bowel, and fistula. These conditions frequently present as surgical emergencies in Nigerian tertiary centres and require prompt recognition, resuscitation, and often urgent operative intervention to prevent life-threatening complications.

This Series begins with the anatomy and classification of inguinal, femoral, and other abdominal wall hernias, then addresses their complications and principles of repair. It proceeds to peritonitis, intra-abdominal abscess, and gangrenous bowel, concluding with the classification and management of fistula, drawing together the shared emergency principles of resuscitation, source control, and staged surgical management.

Series Learning Outcomes

Upon completing this Series, you should be able to:

6. **SLO 4.1:** describe the anatomy, classification, and clinical features of inguinal and femoral hernia,
7. **SLO 4.2:** describe the classification, presentation, and management of other abdominal wall hernias and their complications,
8. **SLO 4.3:** describe the classification, pathophysiology, and management of peritonitis,
9. **SLO 4.4:** describe the presentation and management of intra-abdominal abscess and gangrenous bowel,
10. **SLO 4.5:** classify fistula and describe the principles of management of enterocutaneous fistula, and
11. **SLO 4.6:** describe the emergency principles applied across hernia, peritonitis, gangrenous bowel, and fistula.

4.1 Abdominal Wall Hernias I: Inguinal and Femoral

4.1.1 Anatomy of the inguinal and femoral canals

The inguinal canal runs from the deep inguinal ring (midpoint of the inguinal ligament) to the superficial inguinal ring (above the pubic tubercle), bounded by the external oblique aponeurosis anteriorly, the conjoint tendon and transversalis fascia posteriorly, the inguinal ligament inferiorly, and the internal oblique and transversus abdominis superiorly. It transmits the spermatic cord in men and the round ligament in women.

The femoral canal lies medial to the femoral vein within the femoral sheath, bounded by the inguinal ligament anteriorly, the pectineal ligament posteriorly, the femoral vein laterally, and the lacunar ligament medially. Its narrow, rigid boundaries explain why femoral hernias have a high risk of strangulation compared with inguinal hernias.

Fill in the blank: The inguinal canal runs from the deep ring to the _____ ring. The femoral canal lies medial to the femoral _____.

4.1.2 Classification and clinical features of inguinal hernia

Inguinal hernias are classified as indirect (through the deep ring, lateral to the inferior epigastric vessels, following the processus vaginalis, more common and often congenital) or direct (through Hesselbach's triangle, medial to the inferior epigastric vessels, from acquired weakness of the posterior wall). Both present as a groin swelling that reduces on lying flat and enlarges with coughing.

Clinical differentiation between indirect and direct hernias is unreliable on examination alone and is often confirmed only at operation. Risk factors include chronic cough, constipation, heavy lifting, prostatism, and connective tissue disorders. Bilateral hernias and a positive family history are common, and elective repair is advised even in asymptomatic reducible hernias to prevent complications.

Fill in the blank: An indirect inguinal hernia passes through the deep ring _____ to the inferior epigastric vessels. A direct inguinal hernia passes through _____ triangle.

4.1.3 Femoral hernia

Femoral hernia is more common in women, presenting as a swelling below and lateral to the pubic tubercle, distinguishing it from an inguinal hernia which lies above and medial to the tubercle. It is often small, may be easily missed, and carries a high risk of irreducibility and strangulation because of the rigid, narrow boundaries of the femoral canal.

Management is surgical repair in all cases because of the strangulation risk, using a low (Lockwood), high (McEvedy), or laparoscopic approach to narrow the femoral canal by approximating the inguinal and pectineal ligaments. Emergency presentation with strangulation is common and requires urgent surgery with assessment of bowel viability.

Fill in the blank: A femoral hernia lies _____ and lateral to the pubic tubercle. Femoral hernias are managed surgically in all cases because of the high risk of _____.

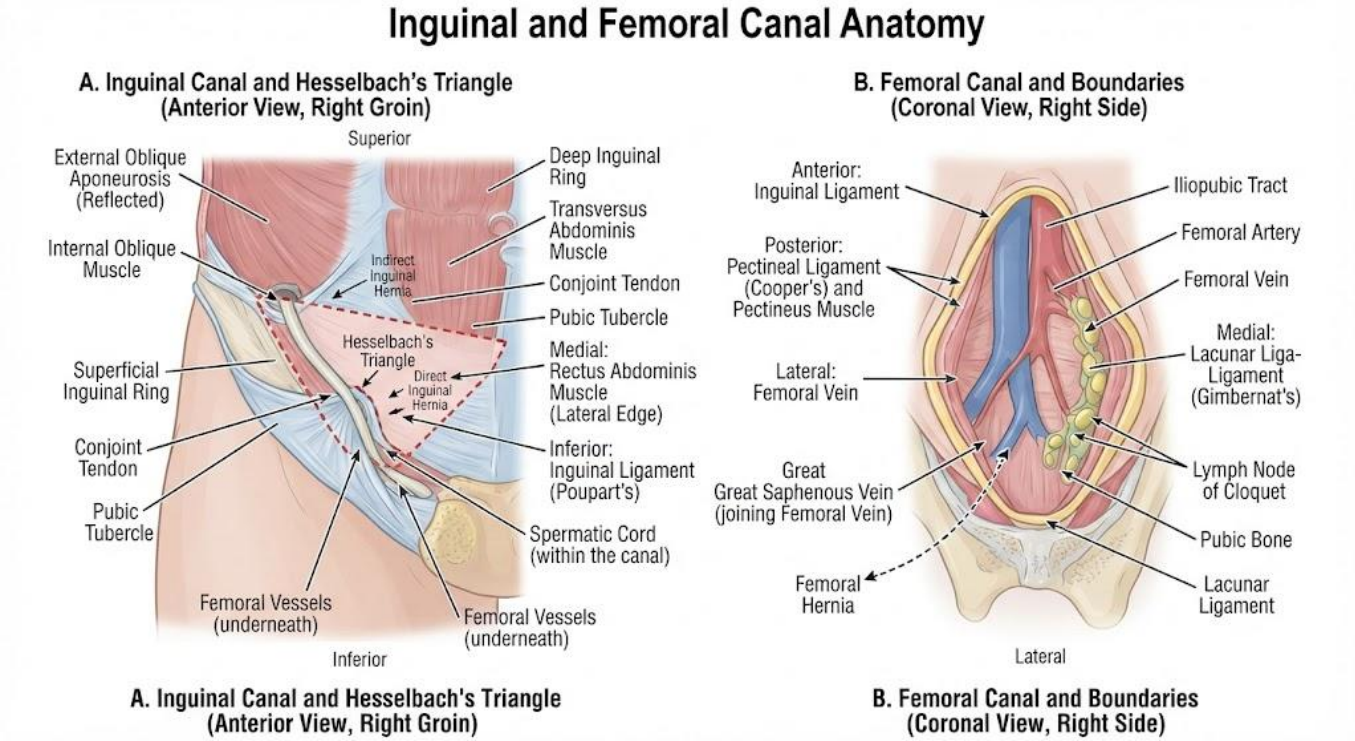

Figure 4.1: Anatomy of the inguinal and femoral canals and hernia classification.

Pilot questions 4.1

11. Describe the anatomy of the inguinal canal and the structures it transmits. (Linked to SLO 4.1)
12. Distinguish indirect from direct inguinal hernia by anatomical route. (Linked to SLO 4.1)
13. Describe the boundaries of the femoral canal and their surgical significance. (Linked to SLO 4.1)
14. Distinguish a femoral hernia from an inguinal hernia on clinical examination. (Linked to SLO 4.1)
15. Explain why femoral hernias are managed surgically in all cases. (Linked to SLO 4.1)

4.2 Abdominal Wall Hernias II: Other Hernias

4.2.1 Umbilical and paraumbilical hernia

Umbilical hernia in infants results from incomplete closure of the umbilical ring and usually resolves spontaneously by age two to three years, so surgery is deferred unless the defect is large or persists beyond age four. Paraumbilical hernia in adults occurs through a defect just above or below the umbilicus, associated with obesity, pregnancy, and raised intra-abdominal pressure.

Paraumbilical hernias in adults have a narrow neck and thick, fatty, often irreducible contents, giving them a relatively high risk of strangulation compared with other ventral hernias. Repair is by open mesh or suture repair (Mayo repair) or laparoscopic technique, generally recommended once diagnosed given the strangulation risk in adults.

Fill in the blank: Infantile umbilical hernia usually resolves spontaneously by age _____ years. Paraumbilical hernias in adults have a relatively high risk of _____ because of their narrow neck.

4.2.2 Incisional and ventral hernia

Incisional hernia occurs through a previous surgical wound, from failure of fascial healing due to wound infection, obesity, poor nutrition, steroid use, malignancy, or a technical error in the original closure. It presents as a bulge at or near the scar, often enlarging over months to years, and can become large and complex with loss of abdominal domain.

Management depends on size and symptoms: small, asymptomatic incisional hernias may be observed, while larger or symptomatic hernias require mesh repair (open or laparoscopic), with component separation techniques used for very large defects. Risk factors for recurrence after repair include obesity, smoking, and inadequate mesh overlap.

Fill in the blank: Incisional hernia occurs through a previous _____ wound. Very large incisional hernias with loss of abdominal domain may require _____ techniques.

4.2.3 Rare hernias (obturator, Spigelian, lumbar)

Obturator hernia passes through the obturator canal, occurring predominantly in thin, elderly women, and often presents with bowel obstruction rather than a palpable swelling because the canal is deep in the pelvis. The Howship-Romberg sign, pain along the medial thigh from obturator nerve compression, is a classic but inconsistent clinical clue.

Spigelian hernia occurs through the linea semilunaris at the lateral edge of the rectus sheath, presenting as a swelling that may be difficult to palpate as it often remains within the abdominal wall layers. Lumbar hernia occurs through the superior (Grynfeltt) or inferior (Petit) lumbar triangles and is rare, usually related to trauma or prior flank surgery.

Fill in the blank: Obturator hernia often presents with bowel _____ rather than a palpable swelling. Pain along the medial thigh from obturator nerve compression is called the _____ sign.

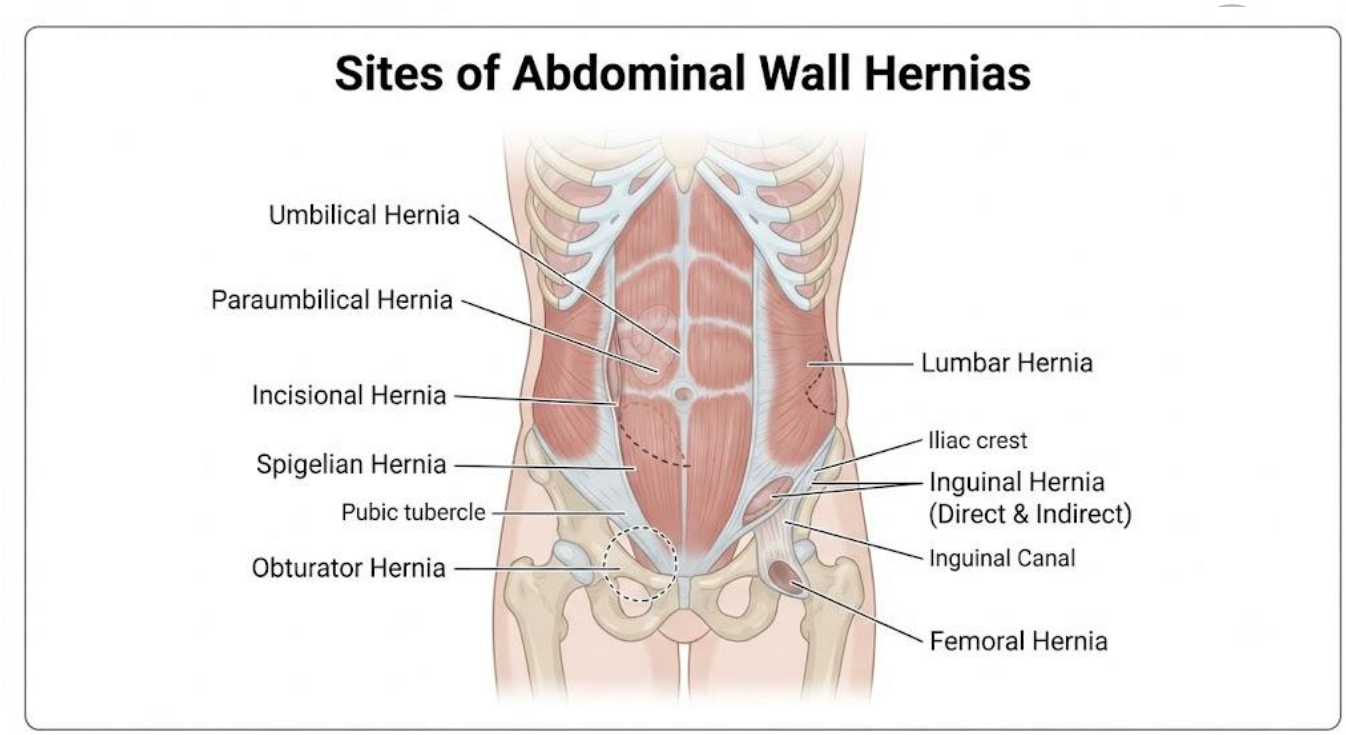

Figure 4.2: Anatomical sites of umbilical, incisional, and rare abdominal wall hernias.

Pilot questions 4.2

1. Describe the natural history and management of infantile umbilical hernia. (Linked to SLO 4.1)
2. Explain why paraumbilical hernias in adults carry a relatively high strangulation risk. (Linked to SLO 4.1)
3. Describe the risk factors and management options for incisional hernia. (Linked to SLO 4.1)
4. Describe the clinical presentation of obturator hernia and the Howship-Romberg sign. (Linked to SLO 4.1)
5. Describe the anatomical routes of Spigelian and lumbar hernias. (Linked to SLO 4.1)

4.3 Complications of Hernias and Principles of Repair

4.3.1 Irreducibility, obstruction, and strangulation

An irreducible hernia cannot be returned to the abdominal cavity, either because of adhesions within the sac or a narrow neck, but retains normal blood supply. Obstructed hernia contains bowel with lumen obstruction but intact blood supply, presenting with colicky abdominal pain, distension, and vomiting in addition to the irreducible swelling.

Strangulated hernia occurs when the blood supply to the herniated content is compromised, a surgical emergency presenting with severe pain, a tense, tender, irreducible swelling, and systemic signs of sepsis as ischaemia progresses to gangrene and perforation. Strangulation risk is highest in femoral and paraumbilical hernias because of their narrow necks.

Fill in the blank: A hernia with intact blood supply but obstructed bowel lumen is termed _____. Strangulation occurs when the _____ supply to herniated contents is compromised.

4.3.2 Emergency management of strangulated hernia

Strangulated hernia requires urgent resuscitation with intravenous fluids, analgesia, nasogastric decompression if obstructed, and broad-spectrum antibiotics, followed by emergency surgery without delay, as any attempt at forceful manual reduction risks reducing non-viable or perforated bowel into the abdomen (reduction en masse).

At operation the hernial sac is opened, the contents assessed for viability (colour, peristalsis, and mesenteric pulsation), and non-viable bowel resected with primary anastomosis or stoma formation depending on contamination. The hernial defect is then repaired, usually without mesh in a contaminated field to reduce infection risk.

Fill in the blank: Forceful manual reduction of a strangulated hernia risks reducing non-viable bowel, known as reduction _____. Mesh is usually avoided in a _____ field to reduce infection risk.

4.3.3 Principles of hernia repair (open and laparoscopic)

Open hernia repair (such as the Lichtenstein tension-free mesh repair for inguinal hernia) reduces the hernia, identifies and preserves the ilioinguinal nerve, and reinforces the posterior wall with a synthetic mesh, performed under local, spinal, or general anaesthesia, and is widely used in Nigerian tertiary centres given its low cost and technical simplicity.

Laparoscopic repair (transabdominal preperitoneal or totally extraperitoneal approach) offers less postoperative pain and faster recovery, particularly useful for bilateral or recurrent hernias, but requires general anaesthesia and specialised equipment and expertise, limiting availability in some centres. Both approaches use mesh reinforcement as the standard of care in elective clean cases.

Fill in the blank: The tension-free open mesh repair technique for inguinal hernia is called the _____ repair. Laparoscopic hernia repair uses the transabdominal preperitoneal or _____ approach.

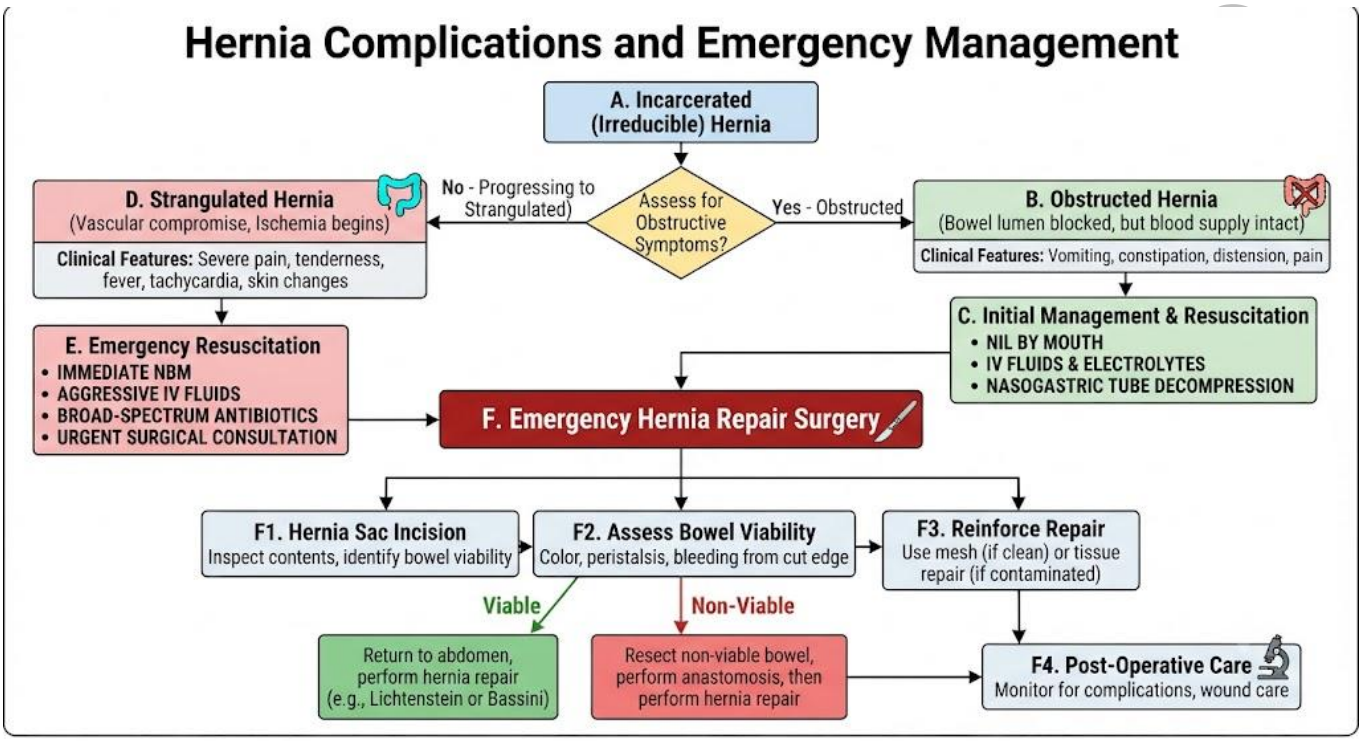

Figure 4.3: Progression of hernia complications and emergency management pathway.

Pilot questions 4.3

1. Distinguish irreducible, obstructed, and strangulated hernia by clinical features. (Linked to SLO 4.2)
2. Describe the emergency management of a strangulated hernia. (Linked to SLO 4.2)
3. Explain the risk of reduction en masse and why forceful reduction should be avoided. (Linked to SLO 4.2)
4. Describe the principles of the Lichtenstein tension-free mesh repair. (Linked to SLO 4.2)
5. Compare open and laparoscopic hernia repair. (Linked to SLO 4.2)

4.4 Peritonitis

4.4.1 Classification and pathophysiology of peritonitis

Peritonitis is inflammation of the peritoneum, classified as primary (spontaneous bacterial, without an intra-abdominal source, seen in cirrhotic ascites and children), secondary (from a perforated viscus, the most common type in surgical practice), or tertiary (persistent or recurrent infection after apparently adequate treatment of secondary peritonitis, often in critically ill patients).

Pathophysiology involves bacterial contamination of the peritoneal cavity triggering an inflammatory response with fluid exudation, fibrin deposition, and localisation attempts by the omentum and adjacent viscera. Uncontrolled spread causes generalised peritonitis with third-space fluid loss, hypovolaemia, and systemic inflammatory response leading to septic shock if untreated.

Fill in the blank: Peritonitis from a perforated viscus is classified as _____ peritonitis. Persistent infection after treatment of secondary peritonitis is termed _____ peritonitis.

4.4.2 Clinical features and diagnosis of peritonitis

Clinical features include severe, diffuse abdominal pain worsened by movement, guarding, rigidity, rebound tenderness, and absent bowel sounds, with the patient often lying still to minimise peritoneal irritation. Systemic features include fever, tachycardia, and, in severe cases, hypotension and signs of septic shock.

Diagnosis is largely clinical, supported by an erect chest radiograph showing free air under the diaphragm in perforation, and abdominal ultrasound or CT to identify the cause and any localised collection. Blood tests show leucocytosis and raised inflammatory markers, and serum lactate helps assess severity and guide resuscitation.

Fill in the blank: Free air under the diaphragm on erect chest radiograph suggests a perforated _____. Serum _____ helps assess the severity of peritonitis and guide resuscitation.

4.4.3 Management of peritonitis

Initial management is resuscitation with intravenous fluids, broad-spectrum antibiotics covering Gram-negative and anaerobic organisms, nasogastric decompression, urinary catheterisation for fluid monitoring, and analgesia, following the principles of the surviving sepsis approach with early source identification.

Definitive management is source control, usually surgical: closure or resection of a perforated viscus, peritoneal lavage to remove contamination, and drainage of collections. A laparostomy (open abdomen) with planned relook may be used in severe cases with ongoing sepsis or an inability to close the abdomen safely at the first operation.

Fill in the blank: Definitive management of secondary peritonitis is surgical _____ control. An open abdomen managed with a planned relook is called a _____.

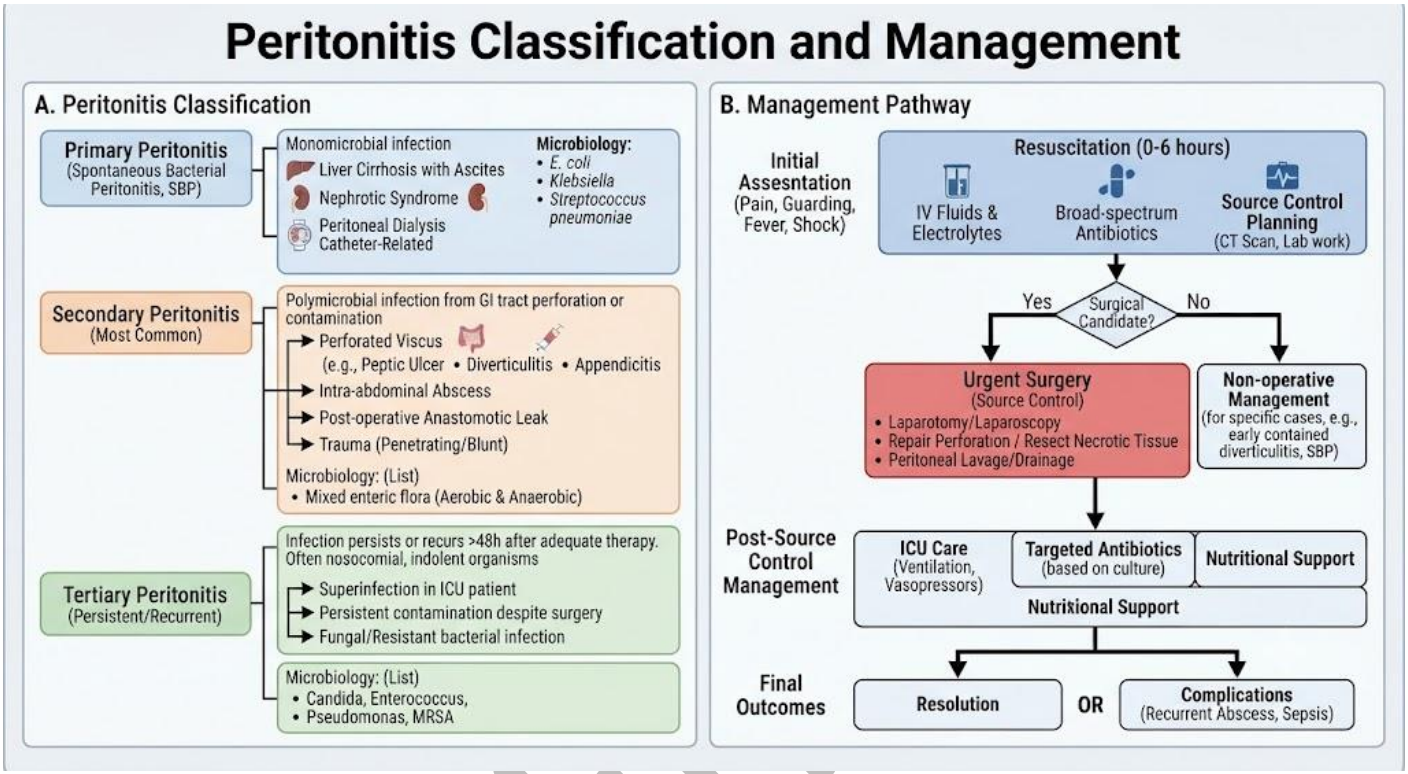

Figure 4.4: Classification of peritonitis and stepwise management pathway.

Pilot questions 4.4

1. Classify peritonitis and give an example cause of each type. (Linked to SLO 4.3)
2. Describe the pathophysiology of generalised peritonitis. (Linked to SLO 4.3)
3. Describe the clinical features of peritonitis on examination. (Linked to SLO 4.3)
4. Describe the initial resuscitation of a patient with peritonitis. (Linked to SLO 4.3)
5. Explain the principle of source control in the management of secondary peritonitis. (Linked to SLO 4.3)

4.5 Intra-abdominal Abscess and Gangrenous Bowel

4.5.1 Intra-abdominal and subphrenic abscess

Intra-abdominal abscess is a localised collection of pus, commonly following perforation, anastomotic leak, or incompletely treated peritonitis, presenting with persistent fever, localised pain, a tender mass, and a swinging pyrexia. Common sites include the pelvis, subphrenic spaces, and paracolic gutters, reflecting the flow of peritoneal fluid.

Subphrenic abscess classically presents with shoulder tip pain from diaphragmatic irritation referred via the phrenic nerve, along with a persistent swinging fever after abdominal surgery. Diagnosis is by CT or ultrasound, and management is percutaneous image-guided drainage where accessible, reserving open surgical drainage for multiloculated or inaccessible collections.

Fill in the blank: Subphrenic abscess classically causes _____ pain from diaphragmatic irritation. The first-line treatment for an accessible intra-abdominal abscess is _____ drainage.

4.5.2 Causes and pathophysiology of bowel gangrene

Bowel gangrene results from irreversible ischaemia, caused by strangulated hernia, volvulus, mesenteric arterial occlusion (embolic or thrombotic), mesenteric venous thrombosis, or intussusception. Ischaemia progresses through mucosal injury to full-thickness necrosis, with loss of peristalsis, dusky discolouration, and eventually perforation with faecal or bowel content contamination.

Acute mesenteric ischaemia classically presents with pain out of proportion to physical findings, often in a patient with atrial fibrillation or atherosclerotic disease, and is a diagnosis requiring a high index of suspicion, as delayed recognition carries very high mortality. Raised lactate and CT angiography aid diagnosis, but definitive diagnosis is often at laparotomy.

Fill in the blank: Acute mesenteric ischaemia classically presents with pain _____ to physical findings. Bowel gangrene may result from strangulated hernia, volvulus, or mesenteric _____ occlusion.

4.5.3 Management of gangrenous bowel

Management begins with resuscitation and broad-spectrum antibiotics, followed by emergency laparotomy to resect all non-viable bowel with margins into healthy, well-perfused tissue. A second-look laparotomy at 24 to 48 hours may be planned when bowel viability is uncertain at the initial operation, to reassess and resect any further non-viable segments.

Reconstruction by primary anastomosis is favoured in a stable patient with a clean field, while stoma formation (resection with end stoma) is preferred in haemodynamically unstable patients or where anastomotic leak risk is high, such as extensive contamination or poor tissue quality, with restoration of continuity planned later.

Fill in the blank: A planned reassessment operation for uncertain bowel viability is called a _____ laparotomy. In unstable patients, resection with an end _____ is preferred over primary anastomosis.

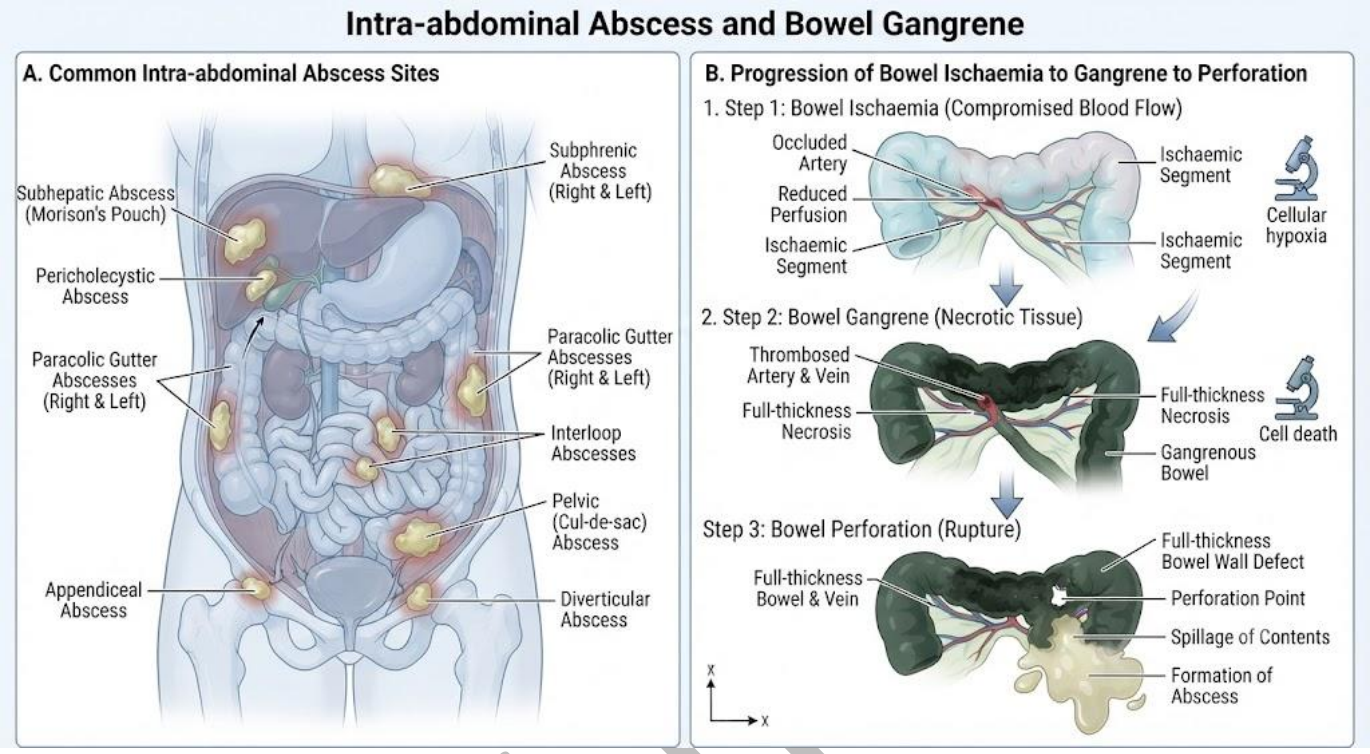

Figure 4.5: Sites of intra-abdominal abscess and pathway from bowel ischaemia to gangrene.

Pilot questions 4.5

1. Describe the clinical features and sites of intra-abdominal abscess. (Linked to SLO 4.4)
2. Describe the clinical presentation and management of subphrenic abscess. (Linked to SLO 4.4)
3. List the causes of bowel gangrene. (Linked to SLO 4.4)
4. Describe the clinical presentation of acute mesenteric ischaemia. (Linked to SLO 4.4)
5. Describe the surgical management of gangrenous bowel, including the role of second-look laparotomy. (Linked to SLO 4.4)

4.6 Fistula and Related Abdominal Conditions

4.6.1 Classification and causes of fistula

A fistula is an abnormal communication between two epithelialised surfaces. Fistulae are classified by anatomical connection (for example, enterocutaneous, enteroenteric, or enterovesical), by output (high output greater than 500 mL per day, or low output), and by

aetiology (congenital, or acquired from surgery, inflammatory bowel disease, malignancy, radiation, or infection such as tuberculosis).

The mnemonic FRIEND (foreign body, radiation, infection or inflammatory bowel disease, epithelialisation, neoplasia, distal obstruction) lists factors that prevent spontaneous fistula closure. Postoperative fistulae, particularly after gastrointestinal surgery with anastomotic leak, are the most common cause encountered in surgical practice.

Fill in the blank: A high-output fistula produces greater than _____ mL per day. The mnemonic listing factors preventing spontaneous fistula closure is _____.

4.6.2 Enterocutaneous fistula

Enterocutaneous fistula connects the bowel to the skin, most commonly following abdominal surgery with anastomotic breakdown, presenting with drainage of intestinal content through the wound, skin excoriation, and, in high-output fistulae, dehydration, electrolyte disturbance, and malnutrition requiring careful metabolic monitoring and correction.

Management follows the SNAP approach: sepsis control (drainage of any collection), nutritional support (often parenteral in high-output fistulae to allow bowel rest), anatomy definition (fistulogram or CT to delineate the tract), and planning definitive surgery, which is typically delayed for three to six months to allow inflammation to settle.

Fill in the blank: Management of enterocutaneous fistula follows the _____ approach. Definitive surgical repair of an enterocutaneous fistula is typically delayed for _____ months.

4.6.3 Principles of fistula management

General principles of fistula management prioritise sepsis control and nutritional optimisation before considering surgery, as most low-output fistulae without a distal obstruction or other adverse factor will close spontaneously with conservative management, typically within four to six weeks.

Surgical management is reserved for fistulae that fail to close spontaneously, or where an adverse factor from the FRIEND mnemonic is present, involving resection of the fistula tract and the diseased bowel segment with primary anastomosis once sepsis is controlled and the patient is nutritionally optimised, in a planned, non-emergency setting.

Fill in the blank: Most low-output fistulae without adverse factors close spontaneously within _____ weeks. Surgical repair of a fistula is undertaken once sepsis is controlled and the patient is nutritionally _____.

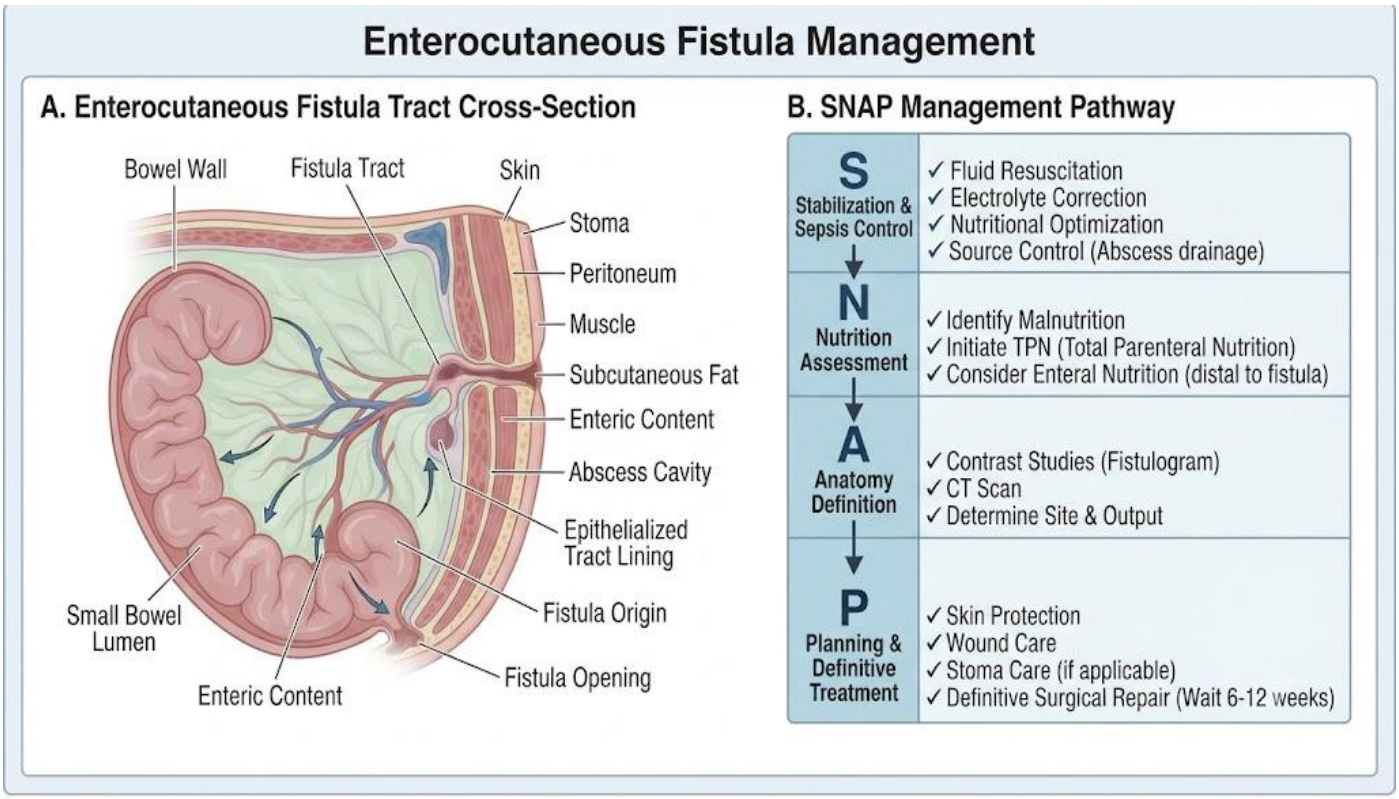

Figure 4.6: Anatomy of enterocutaneous fistula and the SNAP management approach.

Pilot questions 4.6

1. Classify fistula by anatomical connection, output, and aetiology. (Linked to SLO 4.5)
2. Explain the FRIEND mnemonic and its relevance to fistula closure. (Linked to SLO 4.5)
3. Describe the clinical presentation of enterocutaneous fistula. (Linked to SLO 4.5)
4. Describe the SNAP approach to enterocutaneous fistula management. (Linked to SLO 4.5)
5. Explain when surgery is indicated in the management of fistula. (Linked to SLO 4.5)

Series Summary

This Series has covered the anatomy, classification, and management of abdominal wall hernias, from inguinal and femoral hernia to the less common umbilical, incisional, and rare hernias, along with their complications and principles of repair. It has also addressed peritonitis, intra-abdominal abscess, gangrenous bowel, and fistula, drawing together the shared emergency principles of resuscitation, source control, and staged surgical management.

You should now be able to describe the anatomy and classification of the major abdominal wall hernias, recognise and manage hernia complications including strangulation, describe the classification and management of peritonitis, manage intra-abdominal abscess and gangrenous bowel, and classify fistula and describe the principles of its management (Linked to SLOs 4.1 to 4.6).

Glossary of Terms

1. **Enterocutaneous fistula:** an abnormal communication between the bowel and the skin, most commonly following abdominal surgery.
2. **FRIEND mnemonic:** foreign body, radiation, infection or inflammatory bowel disease, epithelialisation, neoplasia, and distal obstruction, factors preventing spontaneous fistula closure.
3. **Hesselbach's triangle:** the anatomical area bounded by the inferior epigastric vessels, rectus sheath, and inguinal ligament, through which direct inguinal hernias pass.
4. **Howship-Romberg sign:** pain along the medial thigh from obturator nerve compression, a classic clinical clue to obturator hernia.
5. **Incisional hernia:** a hernia occurring through a previous surgical wound from failure of fascial healing.
6. **Irreducible hernia:** a hernia that cannot be returned to the abdominal cavity but retains normal blood supply.
7. **Peritonitis:** inflammation of the peritoneum, classified as primary, secondary, or tertiary.
8. **Reduction en masse:** forceful reduction of a strangulated hernia that pushes non-viable or perforated bowel into the abdomen.
9. **SNAP approach:** sepsis control, nutritional support, anatomy definition, and planned surgery, the management approach to enterocutaneous fistula.
10. **Source control:** surgical elimination of the cause of intra-abdominal infection, the definitive management of secondary peritonitis.

11. **Strangulated hernia:** a hernia in which the blood supply to the herniated content is compromised, a surgical emergency.
12. **Subphrenic abscess:** a collection of pus beneath the diaphragm, classically causing referred shoulder tip pain.

Concept Check Questions [Series 4]

Objective questions

- The inguinal canal runs from the deep ring to the _____ ring.
- A hernia with intact blood supply but obstructed bowel lumen is termed _____.
- Free air under the diaphragm on erect chest radiograph suggests a perforated _____.
- Subphrenic abscess classically causes _____ pain from diaphragmatic irritation.
- The mnemonic listing factors preventing spontaneous fistula closure is _____.

Short-answer questions

- Distinguish a femoral hernia from an inguinal hernia on clinical examination. (Linked to SLO 4.1)
- Explain why forceful reduction of a strangulated hernia is dangerous. (Linked to SLO 4.2)
- Describe the clinical features of peritonitis on examination. (Linked to SLO 4.3)
- Describe the clinical presentation of acute mesenteric ischaemia. (Linked to SLO 4.4)
- Describe the SNAP approach to enterocutaneous fistula management. (Linked to SLO 4.5)

Long-answer questions

- Describe the classification and clinical features of inguinal and femoral hernia. (Linked to SLO 4.1)
- Describe the emergency management of a strangulated hernia. (Linked to SLO 4.2)

- Describe the pathophysiology, clinical features, and management of peritonitis. (Linked to SLO 4.3)
- Describe the causes and surgical management of gangrenous bowel. (Linked to SLO 4.4)
- Classify fistula and describe the principles of its management. (Linked to SLO 4.5)

Answers to Concept Check Questions [Series 4]

Objective questions

- Superficial.
- Obstructed hernia.
- Viscus.
- Shoulder tip.
- FRIEND.

Short-answer questions

- Femoral hernia lies below and lateral to the pubic tubercle; inguinal hernia lies above and medial to it. Femoral hernias carry a higher strangulation risk.
- Forceful reduction may push non-viable or perforated bowel into the abdomen (reduction en masse), concealing ongoing ischaemia or contamination and delaying necessary surgery.
- Severe diffuse pain worsened by movement, guarding, rigidity, rebound tenderness, absent bowel sounds, and the patient lying still to minimise peritoneal irritation.
- Pain out of proportion to physical findings, often in a patient with atrial fibrillation or atherosclerotic disease, requiring a high index of suspicion for diagnosis.
- Sepsis control by draining collections, nutritional support often parenteral, anatomy definition by fistulogram or CT, and planned surgery once the patient is optimised.

Long-answer questions

- **Basic response:** Indirect inguinal hernia passes through the deep ring lateral to the inferior epigastric vessels; direct hernia passes through Hesselbach's triangle medially. Femoral hernia lies below and lateral to the pubic tubercle.

Value-added response: Clinical differentiation between indirect and direct hernia is unreliable and often confirmed only at operation; femoral hernias carry the highest strangulation risk of the three.

- **Basic response:** Strangulated hernia requires resuscitation, antibiotics, and emergency surgery without delay, with assessment of bowel viability and resection of any non-viable segment.

Value-added response: Forceful manual reduction should be avoided as it risks reduction en masse, concealing non-viable bowel within the abdomen and delaying diagnosis of ongoing ischaemia.

- **Basic response:** Peritonitis is classified as primary, secondary, or tertiary, presenting with diffuse pain, guarding, and rigidity, and is managed with resuscitation, antibiotics, and surgical source control.

Value-added response: Severe cases may require a laparostomy with planned relook when ongoing sepsis or safe abdominal closure cannot be achieved at the first operation.

- **Basic response:** Gangrenous bowel results from strangulated hernia, volvulus, or mesenteric occlusion, managed by emergency laparotomy with resection of non-viable bowel into healthy tissue.

Value-added response: A second-look laparotomy at 24 to 48 hours may be planned when viability is uncertain, and stoma formation is preferred over primary anastomosis in unstable patients.

- **Basic response:** Fistulae are classified by anatomical connection, output, and aetiology. Management prioritises sepsis control and nutrition before considering surgery.

Value-added response: The FRIEND mnemonic identifies factors preventing spontaneous closure; surgery is reserved for fistulae that fail conservative management or have an adverse factor present.

Answers to Pilot Questions [Series 4]

Part 4.1

1. The inguinal canal runs from the deep to the superficial ring, transmitting the spermatic cord in men and the round ligament in women, bounded by the described muscular and aponeurotic layers.
2. Indirect hernia passes through the deep ring lateral to the inferior epigastric vessels, following the processus vaginalis. Direct hernia passes through Hesselbach's triangle medial to the vessels.
3. The femoral canal is bounded by the inguinal ligament, pectineal ligament, femoral vein, and lacunar ligament, its narrow rigid boundaries predisposing to strangulation.
4. Femoral hernia lies below and lateral to the pubic tubercle, while inguinal hernia lies above and medial to it.
5. Femoral hernias have a high risk of irreducibility and strangulation because of the narrow, rigid boundaries of the femoral canal, so surgery is advised in all cases.

Part 4.2

1. Infantile umbilical hernia usually resolves spontaneously by age two to three years; surgery is deferred unless the defect is large or persists beyond age four.
2. Paraumbilical hernias have a narrow neck and thick, fatty, often irreducible contents, giving a relatively high strangulation risk compared with other ventral hernias.
3. Risk factors include wound infection, obesity, poor nutrition, steroid use, and technical error; management ranges from observation to mesh repair depending on size and symptoms.
4. Obturator hernia often presents with bowel obstruction rather than a swelling, with the Howship-Romberg sign of medial thigh pain from obturator nerve compression.
5. Spigelian hernia occurs through the linea semilunaris; lumbar hernia occurs through the superior (Grynfeltt) or inferior (Petit) lumbar triangles.

Part 4.3

1. Irreducible hernia cannot be returned but has intact blood supply. Obstructed hernia has bowel lumen obstruction with intact blood supply. Strangulated hernia has compromised blood supply, a surgical emergency.

2. Resuscitation with fluids, analgesia, nasogastric decompression, and antibiotics, followed by emergency surgery to assess bowel viability and resect non-viable segments.
3. Forceful reduction risks pushing non-viable or perforated bowel into the abdomen, concealing ongoing ischaemia or contamination and delaying necessary surgery.
4. The Lichtenstein repair reduces the hernia, preserves the ilioinguinal nerve, and reinforces the posterior wall with tension-free synthetic mesh.
5. Open repair is low-cost and technically simple; laparoscopic repair offers less pain and faster recovery but requires general anaesthesia and specialised expertise.

Part 4.4

1. Primary peritonitis has no intra-abdominal source (for example, cirrhotic ascites); secondary peritonitis follows a perforated viscus; tertiary peritonitis is persistent infection after treatment of secondary peritonitis.
2. Bacterial contamination triggers an inflammatory response with exudation and fibrin deposition; uncontrolled spread causes generalised peritonitis and septic shock.
3. Diffuse pain worsened by movement, guarding, rigidity, rebound tenderness, absent bowel sounds, fever, and tachycardia.
4. Intravenous fluids, broad-spectrum antibiotics, nasogastric decompression, urinary catheterisation, and analgesia, following early sepsis management principles.
5. Source control eliminates the cause of infection surgically, by closure or resection of the perforated viscus, lavage, and drainage of collections.

Part 4.5

- Persistent fever, localised pain, a tender mass, and swinging pyrexia, commonly at the pelvis, subphrenic spaces, or paracolic gutters.
- Subphrenic abscess causes shoulder tip pain from phrenic nerve irritation and swinging fever after surgery, managed with percutaneous image-guided drainage where accessible.
- Strangulated hernia, volvulus, mesenteric arterial occlusion, mesenteric venous thrombosis, and intussusception.
- Pain out of proportion to physical findings, often in a patient with atrial fibrillation or atherosclerotic disease, requiring a high index of suspicion.

- Emergency laparotomy with resection of non-viable bowel into healthy tissue, with a second-look laparotomy at 24 to 48 hours when viability is uncertain.

Part 4.6

1. Fistulae are classified by anatomical connection, output (high or low), and aetiology (congenital or acquired).
2. FRIEND stands for foreign body, radiation, infection or inflammatory bowel disease, epithelialisation, neoplasia, and distal obstruction, all factors preventing spontaneous closure.
3. Drainage of intestinal content through the wound, skin excoriation, and, in high-output fistulae, dehydration and electrolyte disturbance.
4. Sepsis control, nutritional support (often parenteral), anatomy definition by fistulogram or CT, and planned surgery once optimised.
5. Surgery is indicated when a fistula fails to close spontaneously or an adverse factor from the FRIEND mnemonic is present.

References and Attributions [Series 4]

Textual References

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7. Townsend, C. M., Beauchamp, R. D., Evers, B. M. and Mattox, K. L. (2017). Sabiston Textbook of Surgery (20th ed.). Elsevier.
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9. Sartelli, M., Chichom-Mefire, A., Labricciosa, F. M. et al. (2017). The Management of Intra-abdominal Infections from a Global Perspective. World Journal of Emergency Surgery, 12, 29.

Figures, Plates, Tables, and Boxes

11. Figure 4.1: Anatomy of the inguinal and femoral canals and hernia classification. AI-generated using the prompt: "Create a professional two-panel medical diagram titled Inguinal and Femoral Canal Anatomy, showing the inguinal canal with deep and superficial rings and Hesselbach's triangle, and the femoral canal with its boundaries. Clean clinical surgical diagram style."
12. Figure 4.2: Anatomical sites of umbilical, incisional, and rare abdominal wall hernias. AI-generated using the prompt: "Create a professional medical diagram titled Sites of Abdominal Wall Hernias, showing an anterior trunk view with labelled markers for umbilical, paraumbilical, incisional, Spigelian, obturator, and lumbar hernia sites. Clean clinical surgical diagram style."
13. Figure 4.3: Progression of hernia complications and emergency management pathway. AI-generated using the prompt: "Create a professional medical flowchart titled Hernia Complications and Emergency Management, showing progression from irreducible to obstructed or strangulated hernia, with resuscitation and operative steps. Clean clinical surgical diagram style."
14. Figure 4.4: Classification of peritonitis and stepwise management pathway. AI-generated using the prompt: "Create a professional two-panel medical diagram titled Peritonitis Classification and Management, showing primary, secondary, and tertiary peritonitis causes, and a management pathway from resuscitation to surgical source control. Clean clinical surgical diagram style."
15. Figure 4.5: Sites of intra-abdominal abscess and pathway from bowel ischaemia to gangrene. AI-generated using the prompt: "Create a professional two-panel medical diagram titled Intra-abdominal Abscess and Bowel Gangrene, showing common abscess sites and the progression from bowel ischaemia to gangrene to perforation. Clean clinical surgical diagram style."
16. Figure 4.6: Anatomy of enterocutaneous fistula and the SNAP management approach. AI-generated using the prompt: "Create a professional medical diagram titled Enterocutaneous Fistula Management, showing a fistula tract cross-section from bowel to skin and a SNAP management pathway box. Clean clinical surgical diagram style."

Series 5: Hepatobiliary and Breast Diseases

1. **Part 5.1: Gallstone Disease**
    1. Sub Part 5.1.1: Formation and epidemiology of gallstones
    2. Sub Part 5.1.2: Biliary colic and acute cholecystitis
    3. Sub Part 5.1.3: Investigation and management of gallstone disease
2. **Part 5.2: Complications of Gallstone Disease**
    1. Sub Part 5.2.1: Choledocholithiasis and obstructive jaundice
    2. Sub Part 5.2.2: Ascending cholangitis
    3. Sub Part 5.2.3: Gallstone pancreatitis and Mirizzi syndrome
3. **Part 5.3: Liver Disease in Surgical Practice**
    1. Sub Part 5.3.1: Liver anatomy and functional assessment
    2. Sub Part 5.3.2: Liver abscess
    3. Sub Part 5.3.3: Benign and malignant liver tumours
4. **Part 5.4: Pancreatic Disease**
    1. Sub Part 5.4.1: Acute pancreatitis
    2. Sub Part 5.4.2: Chronic pancreatitis
    3. Sub Part 5.4.3: Pancreatic carcinoma
5. **Part 5.5: Benign Breast Disease**
    1. Sub Part 5.5.1: Clinical assessment of the breast (triple assessment)
    2. Sub Part 5.5.2: Fibroadenoma and fibrocystic change
    3. Sub Part 5.5.3: Breast infection (mastitis and abscess)
6. **Part 5.6: Breast Malignancy**
    1. Sub Part 5.6.1: Epidemiology and risk factors of breast cancer
    2. Sub Part 5.6.2: Clinical features, staging, and investigation
    3. Sub Part 5.6.3: Management of breast cancer

Introduction

In the last Series, we explored abdominal wall hernias, peritonitis, gangrenous bowel, and fistula, the emergency conditions that so often bring patients to the surgical ward. We now turn to a different but equally important part of General Surgery II: the hepatobiliary system and the breast. Gallstone disease and its complications remain among the most frequent reasons for elective and emergency surgical referral, while breast disease, both benign and malignant, touches the lives of a very large proportion of the women you will care for as surgeons in Nigerian practice.

The aim of this Series is to equip you with the knowledge to recognise, investigate, and manage the common hepatobiliary and pancreatic surgical conditions, alongside the assessment and management of benign and malignant breast disease.

We shall commence this Series by examining gallstone disease, from formation through to biliary colic and acute cholecystitis. Next, we will consider the complications of gallstone disease, including choledocholithiasis, ascending cholangitis, and gallstone pancreatitis. Following that, our attention turns to liver disease in surgical practice, covering functional anatomy, liver abscess, and liver tumours. We will then explore pancreatic disease, from acute and chronic pancreatitis to pancreatic carcinoma. Moving on to the breast, we will examine the triple assessment approach and the common benign breast conditions. Finally, we will conclude by considering the epidemiology, staging, and management of breast cancer.

Series Learning Outcomes

Upon completing this Series, you should be able to:

1. **SLO 5.1:** describe the formation, presentation, and management of gallstone disease,
2. **SLO 5.2:** describe the surgical assessment and management of liver disease, including liver abscess and tumours,
3. **SLO 5.3:** describe the diagnosis and management of acute and chronic pancreatitis and pancreatic carcinoma,
4. **SLO 5.4:** describe the triple assessment approach and management of benign breast disease,
5. **SLO 5.5:** describe the epidemiology, staging, and management of breast cancer, and
6. **SLO 5.6:** describe the complications of gallstone disease and their emergency management.

5.1 Gallstone Disease

5.1.1 Formation and epidemiology of gallstones

Gallstones form when bile becomes supersaturated with cholesterol, bilirubin, or calcium salts, allowing crystals to precipitate and aggregate. Cholesterol stones are the most common type, associated with the classic risk factors summarised as the five Fs: female, fat, forty, fertile, and family history. Pigment stones, either black or brown, form from excess bilirubin, often related to haemolysis or biliary infection.

Epidemiology varies by population, with gallstones common in Western and Nigerian urban populations alike, increasingly linked to rising obesity and dietary change. Most gallstones remain asymptomatic and are found incidentally on ultrasound, with only a minority of patients developing symptoms or complications over their lifetime.

Fill in the blank: The classic risk factors for cholesterol gallstones are summarised by the mnemonic the five _____.

5.1.2 Biliary colic and acute cholecystitis

Biliary colic occurs when a gallstone transiently obstructs the cystic duct, causing right upper quadrant or epigastric pain, often after fatty meals, that resolves as the stone disimpacts, without fever or peritonism. Acute cholecystitis occurs when obstruction persists, causing gallbladder wall inflammation, with continuous pain, fever, and a positive Murphy's sign (arrest of inspiration on palpation of the right upper quadrant).

Differentiating the two is clinically important: biliary colic is managed electively, while acute cholecystitis requires admission, intravenous antibiotics, and early laparoscopic cholecystectomy, ideally within 72 hours of symptom onset, as delayed surgery is associated with increased technical difficulty and complication rates.

Fill in the blank: Biliary colic resolves without fever, while acute cholecystitis presents with a positive _____ sign.

5.1.3 Investigation and management of gallstone disease

Investigation begins with abdominal ultrasound, the first-line test, showing gallstones, gallbladder wall thickening, and pericholecystic fluid in acute cholecystitis. Liver function tests help identify biliary obstruction, and where the common bile duct is dilated or liver function is deranged, magnetic resonance cholangiopancreatography (MRCP) further characterises the biliary tree.

Management of symptomatic gallstone disease is laparoscopic cholecystectomy, the gold standard, offering shorter hospital stay and faster recovery than open surgery. Open cholecystectomy is reserved for cases with dense adhesions, suspected malignancy, or when laparoscopic access is unavailable or unsafe to continue.

Fill in the blank: The first-line imaging investigation for suspected gallstone disease is abdominal _____. The gold standard treatment for symptomatic gallstone disease is laparoscopic _____.

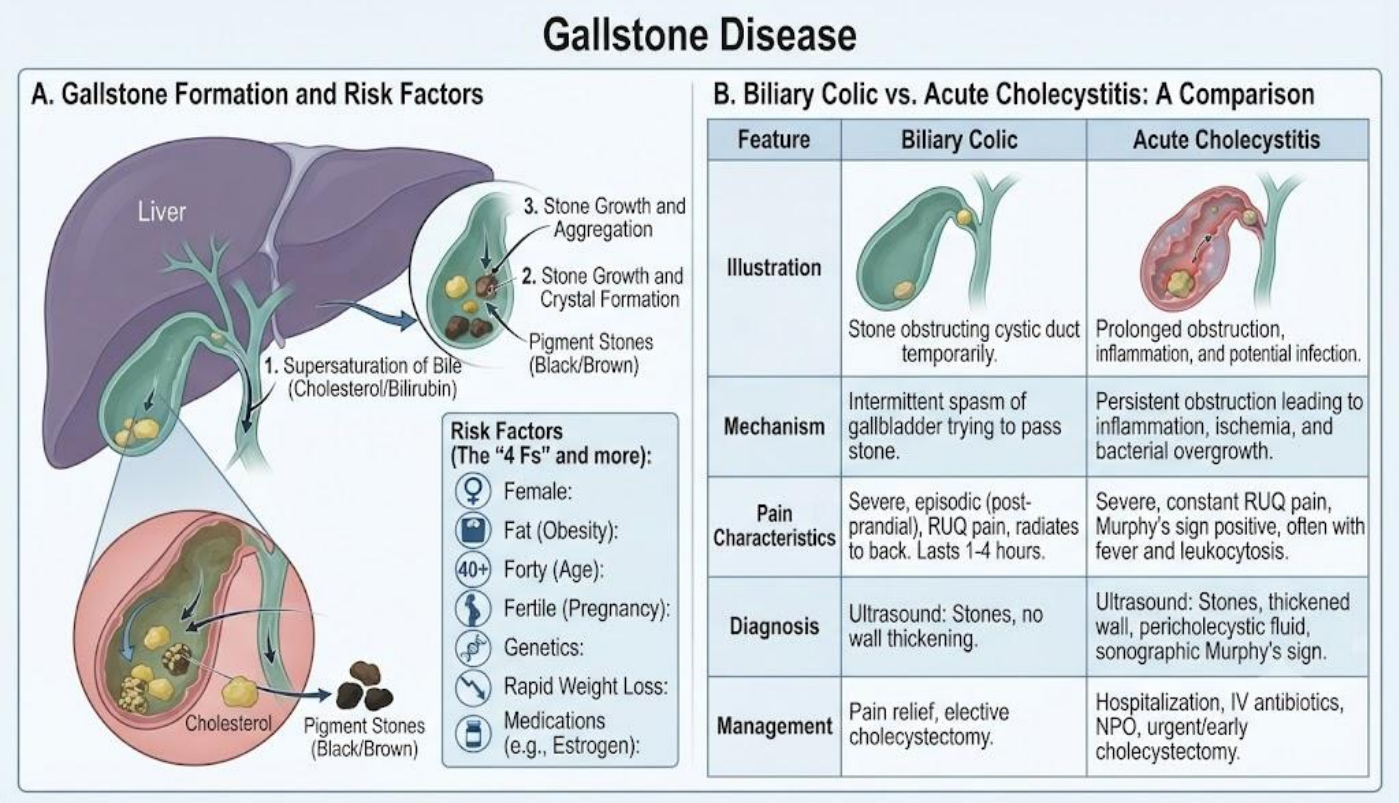

Figure 5.1: Gallstone formation and comparison of biliary colic with acute cholecystitis.

Pilot questions 5.1

1. Describe the types of gallstones and their associated risk factors. (Linked to SLO 5.1)
2. Distinguish biliary colic from acute cholecystitis in presentation and management. (Linked to SLO 5.1)
3. Describe Murphy's sign and its clinical significance. (Linked to SLO 5.1)
4. Describe the first-line investigation of suspected gallstone disease. (Linked to SLO 5.1)
5. Describe the definitive management of symptomatic gallstone disease. (Linked to SLO 5.1)

5.2 Complications of Gallstone Disease

5.2.1 Choledocholithiasis and obstructive jaundice

Choledocholithiasis is the presence of a stone within the common bile duct, causing obstructive jaundice with pale stools, dark urine, and pruritus, and biochemically a raised conjugated bilirubin with a cholestatic pattern of liver function tests (raised alkaline phosphatase and

gamma-glutamyl transferase). Ultrasound may show a dilated common bile duct even when the stone itself is not directly visualised.

Management is endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy and stone extraction, usually followed by interval laparoscopic cholecystectomy to remove the gallbladder and prevent recurrent stone migration, or a combined single-stage laparoscopic approach where local expertise allows.

Fill in the blank: A stone within the common bile duct is termed _____. ERCP with sphincterotomy is used to extract a stone from the common bile duct.

5.2.2 Ascending cholangitis

Ascending cholangitis is a life-threatening infection of the biliary tree secondary to obstruction, classically presenting with Charcot's triad (fever, jaundice, and right upper quadrant pain), and, when severe, Reynolds' pentad adds hypotension and confusion, indicating septic shock and the need for urgent intervention.

Management is emergency resuscitation, broad-spectrum intravenous antibiotics, and urgent biliary decompression, most commonly by ERCP, or percutaneous transhepatic cholangiography when ERCP is unavailable or unsuccessful, followed by interval cholecystectomy once the patient has recovered.

Fill in the blank: The classic triad of fever, jaundice, and right upper quadrant pain in cholangitis is called _____. Adding hypotension and confusion to this triad produces _____.

5.2.3 Gallstone pancreatitis and Mirizzi syndrome

Gallstone pancreatitis occurs when a stone impacts at the ampulla of Vater, obstructing both the biliary and pancreatic ducts and triggering pancreatic enzyme activation and inflammation. It is one of the two most common causes of acute pancreatitis (alongside alcohol) and is managed with supportive care and early ERCP if cholangitis or persistent obstruction is present.

Mirizzi syndrome is extrinsic compression of the common hepatic duct by a large stone impacted in the cystic duct or gallbladder neck, causing obstructive jaundice that can mimic a bile duct tumour on imaging. It is important to recognise preoperatively, as it increases the risk of bile duct injury during cholecystectomy and may require a modified surgical approach.

Fill in the blank: Gallstone pancreatitis occurs when a stone impacts at the _____ of Vater. Mirizzi syndrome is caused by extrinsic compression of the common _____ duct.

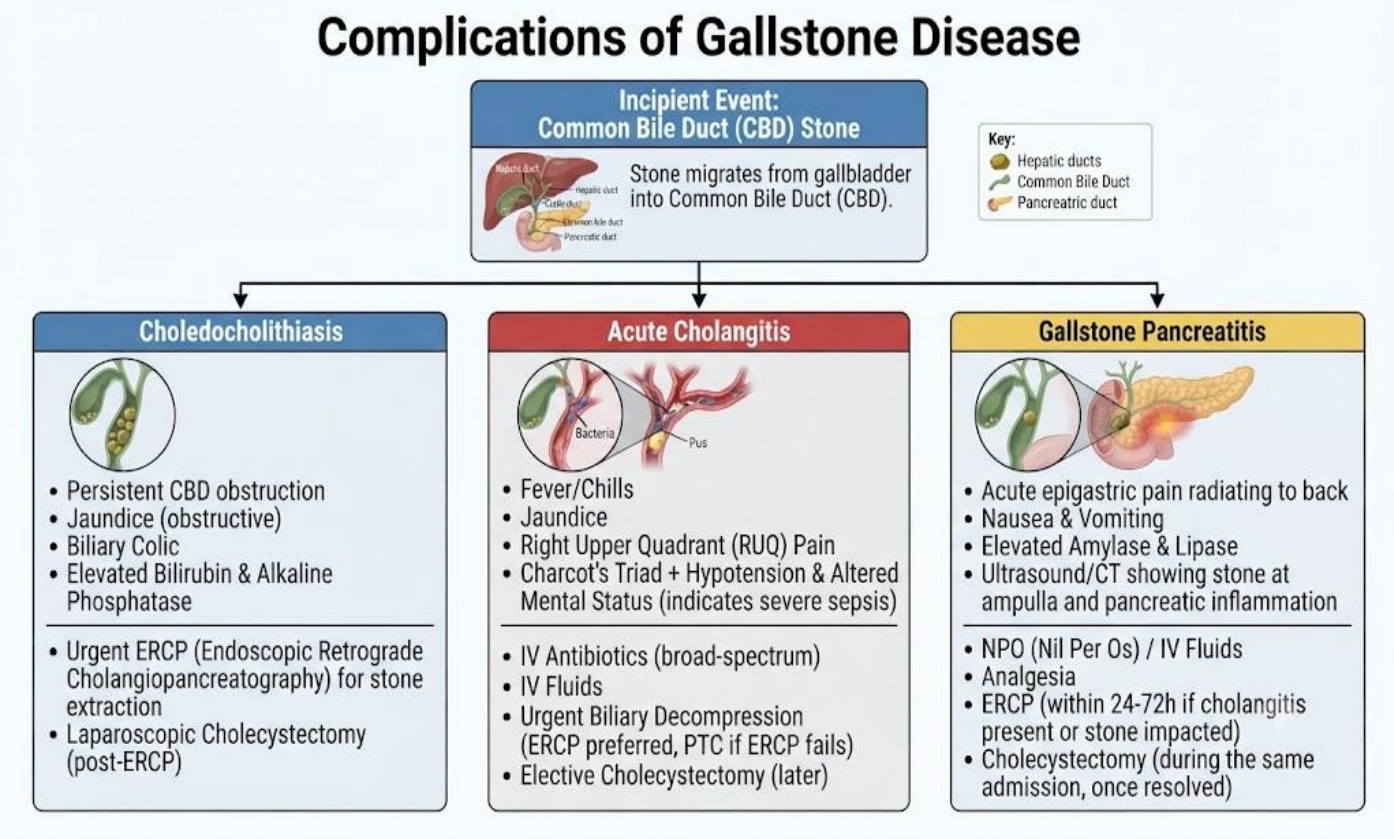

Figure 5.2: Pathways from common bile duct stone to the major complications of gallstone disease.

Pilot questions 5.2

- Describe the clinical and biochemical features of choledocholithiasis. (Linked to SLO 5.1)
- Describe Charcot's triad and Reynolds' pentad and their clinical significance. (Linked to SLO 5.1)
- Describe the emergency management of ascending cholangitis. (Linked to SLO 5.1)
- Explain the mechanism by which gallstones cause acute pancreatitis. (Linked to SLO 5.1)
- Describe Mirizzi syndrome and its surgical significance. (Linked to SLO 5.1)

5.3 Liver Disease in Surgical Practice

5.3.1 Liver anatomy and functional assessment

The liver is divided into eight functional (Couinaud) segments, each with its own vascular inflow, outflow, and biliary drainage, allowing anatomical resections without disrupting adjacent segments. Blood supply is dual, from the hepatic artery (oxygen-rich) and portal vein (nutrient-rich), draining via the hepatic veins into the inferior vena cava.

Functional assessment before liver surgery includes liver function tests, synthetic function markers (albumin and prothrombin time), and, in patients with chronic liver disease, the Child-Pugh score, which grades severity and predicts operative risk, guiding whether major resection is safe.

Fill in the blank: The liver is divided into eight functional segments according to the _____ classification. The _____ score grades the severity of chronic liver disease and predicts operative risk.

5.3.2 Liver abscess

Pyogenic liver abscess most commonly arises from biliary sepsis, portal pyaemia from intra-abdominal infection, or haematogenous spread, presenting with fever, right upper quadrant pain, and tender hepatomegaly. Amoebic liver abscess, caused by Entamoeba histolytica, is common in Nigeria and other tropical regions, often affecting young men with a single right lobe abscess containing anchovy-sauce coloured pus.

Management of pyogenic abscess is percutaneous drainage with antibiotics; amoebic abscess usually responds to metronidazole alone, with drainage reserved for large abscesses, diagnostic uncertainty, or failure to improve with medical therapy. Serology supports the diagnosis of amoebic abscess when the clinical picture is unclear.

Fill in the blank: Amoebic liver abscess is caused by _____. Amoebic liver abscess pus is classically described as _____ coloured.

5.3.3 Benign and malignant liver tumours

Benign liver tumours include haemangioma (most common, usually incidental, managed conservatively), focal nodular hyperplasia, and hepatic adenoma (associated with oral contraceptive use, carrying a risk of rupture and haemorrhage or malignant transformation, particularly in large lesions).

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy, strongly associated with chronic hepatitis B and C infection and cirrhosis, both prevalent in Nigeria, diagnosed by imaging characteristics and raised alpha-fetoprotein. Management depends on tumour stage and liver function, ranging from resection or transplantation to locoregional or systemic therapy.

Fill in the blank: The most common benign liver tumour is _____. Hepatocellular carcinoma is diagnosed with raised _____.

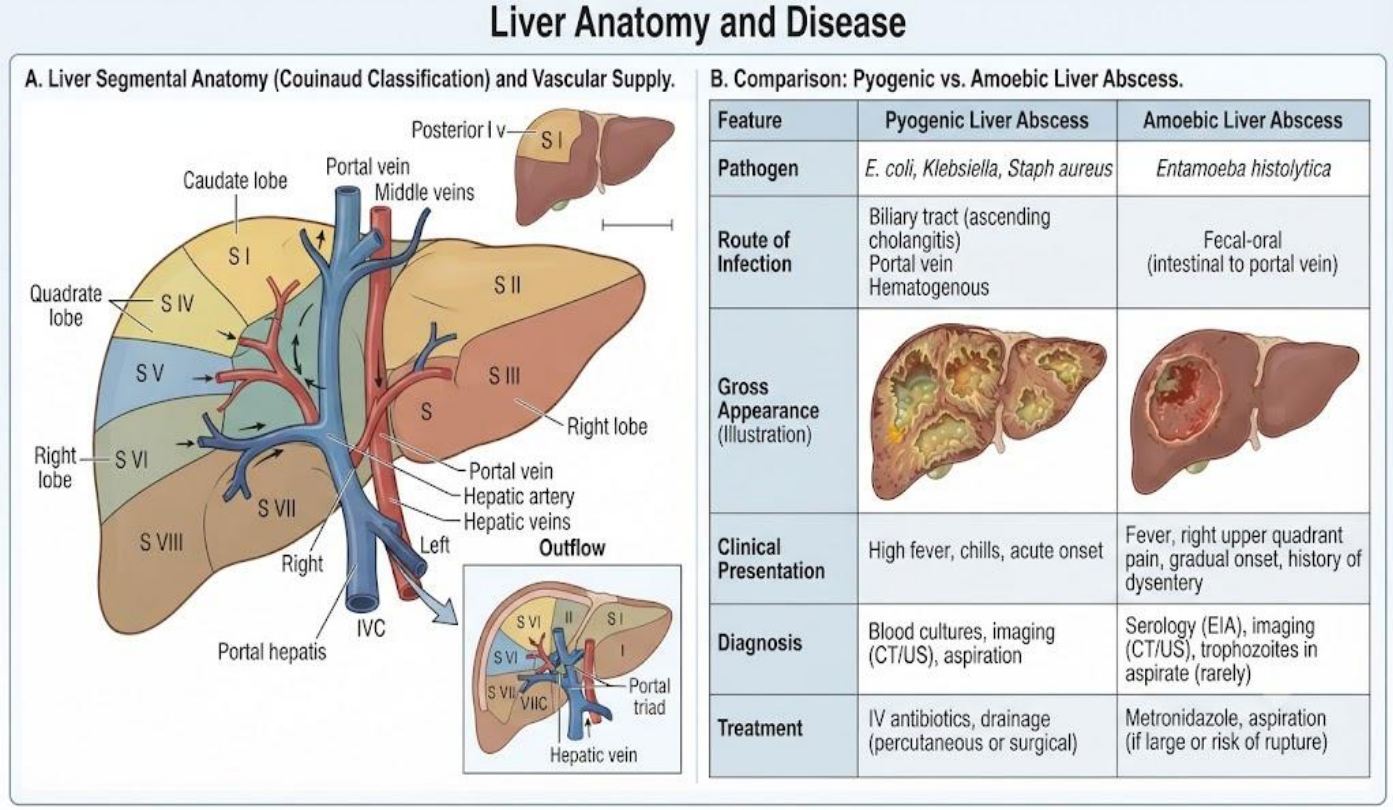

Figure 5.3: Couinaud segmental liver anatomy and comparison of liver abscess types.

Pilot questions 5.3

- Describe the Couinaud segmental anatomy of the liver and its surgical relevance. (Linked to SLO 5.2)
- Describe the Child-Pugh score and its role in assessing operative risk. (Linked to SLO 5.2)
- Compare pyogenic and amoebic liver abscess in cause, presentation, and management. (Linked to SLO 5.2)
- Describe the common benign liver tumours and their management. (Linked to SLO 5.2)
- Describe the risk factors and diagnosis of hepatocellular carcinoma. (Linked to SLO 5.2)

5.4 Pancreatic Disease

5.4.1 Acute pancreatitis

Acute pancreatitis is inflammation of the pancreas, most commonly caused by gallstones or alcohol, presenting with severe epigastric pain radiating to the back, nausea, and vomiting, diagnosed when at least two of three criteria are met: characteristic pain, raised serum amylase or lipase (more than three times normal), and characteristic findings on imaging.

Severity assessment uses scoring systems such as the modified Glasgow-Imrie criteria or APACHE II, assessed within 48 hours, to identify patients at risk of severe disease requiring high-dependency or intensive care. Management is largely supportive: intravenous fluids, analgesia, and early enteral nutrition, with treatment of the underlying cause once stable.

Fill in the blank: Diagnosis of acute pancreatitis requires at least two of three criteria: pain, raised amylase or lipase, and characteristic _____.

5.4.2 Chronic pancreatitis

Chronic pancreatitis is irreversible fibrosis and glandular destruction of the pancreas, most commonly from long-term alcohol use, presenting with recurrent or chronic abdominal pain, and eventually exocrine insufficiency (steatorrhoea and weight loss) and endocrine insufficiency (diabetes mellitus) as functional tissue is progressively lost.

Investigation and management include CT showing pancreatic calcification and duct dilatation, with management focused on analgesia, pancreatic enzyme replacement for exocrine insufficiency, alcohol cessation, and, in selected patients with intractable pain or duct obstruction, endoscopic or surgical drainage procedures.

Fill in the blank: Chronic pancreatitis is most commonly caused by long-term _____ use. Loss of exocrine function in chronic pancreatitis causes _____ and weight loss.

5.4.3 Pancreatic carcinoma

Pancreatic carcinoma most commonly arises in the head of the pancreas, presenting with painless progressive obstructive jaundice, weight loss, and, classically, a palpable non-tender gallbladder (Courvoisier's law: a palpable gallbladder in the presence of jaundice is unlikely to be due to gallstones). Tumours of the body and tail present later, often with pain and weight loss without jaundice.

Management for resectable disease (typically head-of-pancreas tumours without major vascular involvement) is pancreaticoduodenectomy (Whipple's procedure), while most patients present with locally advanced or metastatic disease and are managed with palliative chemotherapy and biliary stenting for symptom relief, reflecting the generally poor prognosis of this cancer.

Fill in the blank: A palpable, non-tender gallbladder in a jaundiced patient is unlikely to be due to gallstones, per _____ law. The surgical procedure for resectable head-of-pancreas cancer is _____.

Criterion	Threshold Value
Age	> 55 years
White blood cell count	> 15 × 10^9/L
Blood glucose	> 10 mmol/L (without diabetes)
Serum urea	> 16 mmol/L
Arterial oxygen	PaO_2 < 8 kPa
Serum calcium	< 2.0 mmol/L
Serum LDH	> 600 IU/L
AST/ALT	> 200 IU/L
Albumin	< 32 g/L

Table 5.4: Modified Glasgow-Imrie criteria for severity assessment of acute pancreatitis.

Pilot questions 5.4

1. Describe the diagnostic criteria for acute pancreatitis. (Linked to SLO 5.3)
2. Describe the purpose and timing of severity scoring in acute pancreatitis. (Linked to SLO 5.3)
3. Describe the clinical features of chronic pancreatitis, including exocrine and endocrine insufficiency. (Linked to SLO 5.3)
4. Describe Courvoisier's law and its clinical significance. (Linked to SLO 5.3)
5. Describe the management of resectable and unresectable pancreatic carcinoma. (Linked to SLO 5.3)

5.5 Benign Breast Disease

5.5.1 Clinical assessment of the breast (triple assessment)

Triple assessment is the standard approach to any new breast symptom, combining clinical examination, imaging (ultrasound for patients under 40, mammography for patients 40 and above, or both), and pathology (core needle biopsy or fine needle aspiration cytology of any discrete lesion), ensuring that no single modality is relied upon alone.

Clinical examination assesses the site, size, consistency, mobility, and skin or nipple changes of any lump, along with axillary and supraclavicular lymph nodes. Red flag features prompting urgent referral include a hard, irregular, fixed lump, skin dimpling or peau d'orange, nipple retraction or bloody discharge, and new axillary lymphadenopathy.

Fill in the blank: Triple assessment combines clinical examination, imaging, and _____. Ultrasound is preferred over mammography as first-line imaging in patients under _____.

5.5.2 Fibroadenoma and fibrocystic change

Fibroadenoma is the most common benign breast lump in young women, presenting as a smooth, firm, highly mobile ('breast mouse') lump confirmed by ultrasound and core biopsy, typically managed conservatively with reassurance and follow-up, as most remain stable or regress, with excision reserved for large, growing, or symptomatic lesions.

Fibrocystic change is a common benign condition in women aged 30 to 50, causing lumpiness and cyclical breast pain related to the menstrual cycle, with palpable cysts that are simple and reassuring on ultrasound. Aspiration relieves symptomatic cysts, and biopsy is reserved for complex or residual masses after aspiration.

Fill in the blank: The most common benign breast lump in young women is _____. A fibroadenoma is classically described as a smooth, firm, and highly _____ lump.

5.5.3 Breast infection (mastitis and abscess)

Lactational mastitis is common in breastfeeding women, caused by milk stasis and Staphylococcus aureus infection, presenting with a red, tender, swollen breast segment and fever, managed with continued breastfeeding or expression, analgesia, and antibiotics (flucloxacillin) if infection is confirmed or symptoms do not settle promptly.

Breast abscess develops if mastitis is inadequately treated, presenting as a fluctuant, tender mass, managed with ultrasound-guided needle aspiration (repeated as needed) as first-line treatment, with incision and drainage reserved for large or multiloculated abscesses that fail to resolve with aspiration alone.

Fill in the blank: Lactational mastitis is most commonly caused by milk stasis and _____ infection. The first-line management of a breast abscess is ultrasound-guided needle _____.

Clinical Feature
Hard, irregular, fixed lump
Skin dimpling or peau d’orange
Nipple retraction or bloody discharge

Clinical Feature
New axillary lymphadenopathy
Age >30 with new discrete lump

Box 5.5: Red flag features of a breast lump requiring urgent referral.

Pilot questions 5.5

1. Describe the components of triple assessment for a new breast symptom. (Linked to SLO 5.4)
2. List the red flag features of a breast lump that warrant urgent referral. (Linked to SLO 5.4)
3. Describe the clinical features and management of fibroadenoma. (Linked to SLO 5.4)
4. Describe the clinical features and management of fibrocystic change. (Linked to SLO 5.4)
5. Describe the presentation and management of lactational mastitis and breast abscess. (Linked to SLO 5.4)

5.6 Breast Malignancy

5.6.1 Epidemiology and risk factors of breast cancer

Breast cancer is the most common cancer in women worldwide and in Nigeria, where patients often present at a later stage than in high-income countries, contributing to poorer outcomes. Risk factors include increasing age, early menarche, late menopause, nulliparity or late first pregnancy, family history, and BRCA1 or BRCA2 mutations.

Genetic risk from BRCA1 and BRCA2 mutations substantially raises lifetime breast and ovarian cancer risk, and genetic counselling and testing are offered to patients with a strong family history, guiding decisions about enhanced surveillance or risk-reducing surgery in appropriately selected patients.

Fill in the blank: The two most significant genetic mutations associated with hereditary breast cancer are _____ and _____.

5.6.2 Clinical features, staging, and investigation

Clinical features of breast cancer include a hard, irregular, fixed lump, skin dimpling, nipple retraction or discharge, and axillary lymphadenopathy, though early cancers may be asymptomatic and detected on screening mammography. Diagnosis follows triple assessment, with core biopsy providing histological type, grade, and receptor status (oestrogen, progesterone, and HER2).

Staging uses the TNM system (tumour size, nodal involvement, and metastasis), supplemented by imaging (chest radiograph, liver ultrasound, and bone scan or CT when metastatic disease is suspected) to guide prognosis and treatment planning, alongside receptor status, which strongly influences systemic therapy choice.

Fill in the blank: Breast cancer staging uses the _____ system, assessing tumour size, nodal involvement, and metastasis.

5.6.3 Management of breast cancer

Surgical management is breast-conserving surgery (wide local excision) with radiotherapy, or mastectomy, depending on tumour size relative to breast size, multifocality, and patient preference, combined with sentinel lymph node biopsy or axillary clearance depending on nodal status.

Adjuvant therapy is tailored to receptor status and risk: chemotherapy for high-risk or triple-negative disease, endocrine therapy (tamoxifen or aromatase inhibitors) for hormone receptor-positive disease, and trastuzumab for HER2-positive disease, reflecting the shift towards receptor-directed, individualised breast cancer treatment.

Fill in the blank: The surgical procedure that removes the whole breast is called _____. Trastuzumab is used in _____-positive breast cancer.

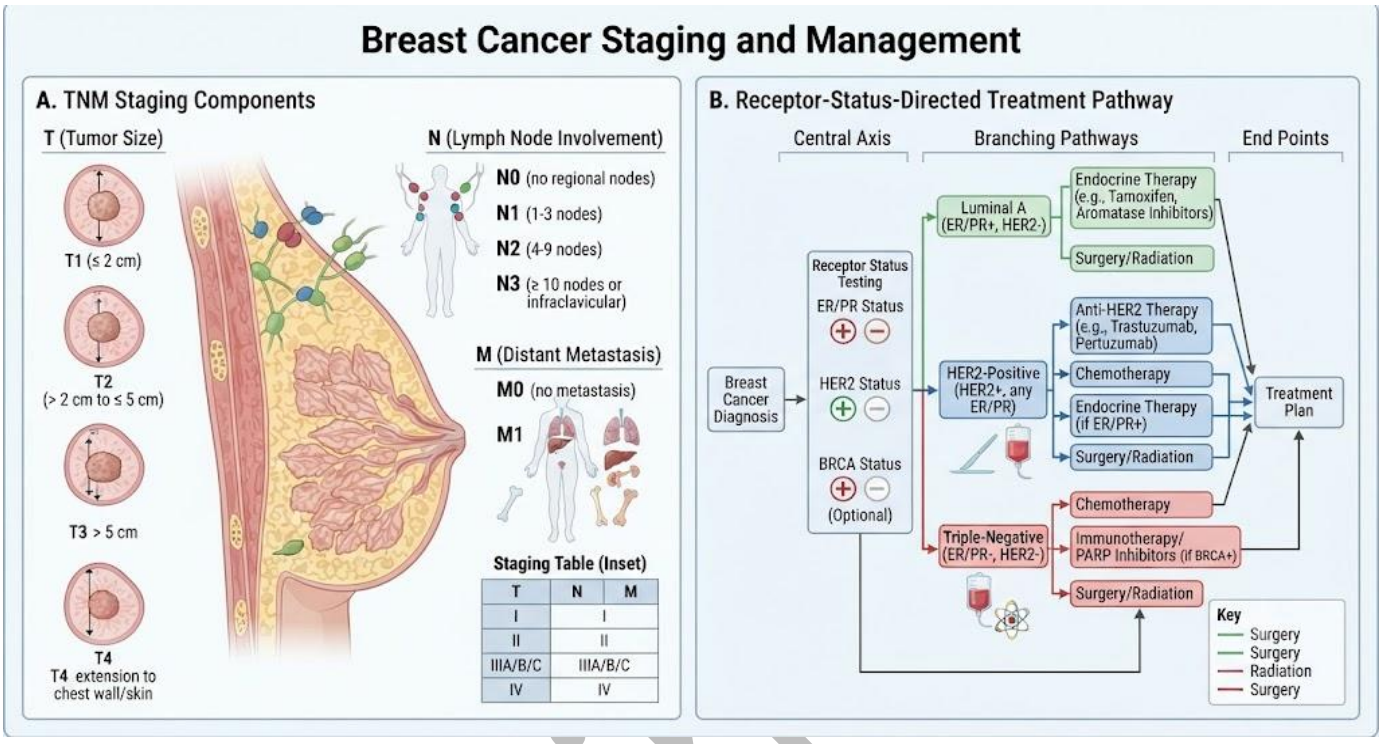

Figure 5.6: TNM staging of breast cancer and receptor-directed management pathway.

Pilot questions 5.6

1. Describe the epidemiology and risk factors for breast cancer. (Linked to SLO 5.5)
2. Describe the role of BRCA1 and BRCA2 mutations in breast cancer risk. (Linked to SLO 5.5)
3. Describe the TNM staging system as applied to breast cancer. (Linked to SLO 5.5)
4. Describe the surgical options for breast cancer and the factors that guide the choice. (Linked to SLO 5.5)
5. Describe how receptor status guides adjuvant therapy in breast cancer. (Linked to SLO 5.5)

Series Summary

This Series has covered gallstone disease from its formation through to biliary colic and acute cholecystitis, then examined its complications, including choledocholithiasis, ascending cholangitis, and gallstone pancreatitis. It went on to address liver disease in surgical practice and

pancreatic disease from acute and chronic pancreatitis to pancreatic carcinoma, before turning to the breast, covering the triple assessment approach, benign breast conditions, and the epidemiology, staging, and management of breast cancer.

You should now be able to describe the presentation and management of gallstone disease and its complications, assess and manage surgical liver disease, diagnose and manage pancreatic disease, apply the triple assessment approach to benign breast disease, and describe the staging and receptor-directed management of breast cancer (Linked to SLOs 5.1 to 5.6).

Glossary of Terms

- **Charcot's triad:** fever, jaundice, and right upper quadrant pain, the classic presentation of ascending cholangitis.
- **Child-Pugh score:** a scoring system grading the severity of chronic liver disease and predicting operative risk.
- **Choledocholithiasis:** the presence of a stone within the common bile duct.
- **Courvoisier's law:** the observation that a palpable, non-tender gallbladder in a jaundiced patient is unlikely to be due to gallstones.
- **Fibroadenoma:** the most common benign breast lump in young women, smooth, firm, and highly mobile.
- **Glasgow-Imrie criteria:** a scoring system used to assess the severity of acute pancreatitis within 48 hours of onset.
- **Mirizzi syndrome:** extrinsic compression of the common hepatic duct by a stone impacted in the cystic duct or gallbladder neck.
- **Murphy's sign:** arrest of inspiration on palpation of the right upper quadrant, a clinical sign of acute cholecystitis.
- **Reynolds' pentad:** Charcot's triad plus hypotension and confusion, indicating severe cholangitis with septic shock.
- **Triple assessment:** the combination of clinical examination, imaging, and pathology used to assess any new breast symptom.
- **Whipple's procedure:** pancreaticoduodenectomy, the surgical resection performed for resectable cancer of the head of the pancreas.
- **Couinaud classification:** the system dividing the liver into eight functional segments, each with independent vascular and biliary supply.

Concept Check Questions [Series 5]

Objective questions

1. Biliary colic resolves without fever, while acute cholecystitis presents with a positive _____ sign.
2. The classic triad of fever, jaundice, and right upper quadrant pain in cholangitis is called _____.
3. Amoebic liver abscess is caused by _____.
4. A palpable, non-tender gallbladder in a jaundiced patient is unlikely to be due to gallstones, per _____ law.
5. The two most significant genetic mutations associated with hereditary breast cancer are _____ and _____.

Short-answer questions

6. Distinguish biliary colic from acute cholecystitis in presentation and management. (Linked to SLO 5.1)
7. Describe the emergency management of ascending cholangitis. (Linked to SLO 5.1)
8. Compare pyogenic and amoebic liver abscess in cause, presentation, and management. (Linked to SLO 5.2)
9. Describe the purpose and timing of severity scoring in acute pancreatitis. (Linked to SLO 5.3)
10. List the red flag features of a breast lump that warrant urgent referral. (Linked to SLO 5.4)

Long-answer questions

11. Describe the presentation, investigation, and management of gallstone disease. (Linked to SLO 5.1)

12. Describe the complications of gallstone disease and their management. (Linked to SLO 5.6)
13. Describe the diagnosis and management of acute pancreatitis. (Linked to SLO 5.3)
14. Describe the triple assessment approach and its application to benign breast disease. (Linked to SLO 5.4)
15. Describe the staging and receptor-directed management of breast cancer. (Linked to SLO 5.5)

Answers to Concept Check Questions [Series 5]

Objective questions

1. Murphy's.
2. Charcot's triad.
3. Entamoeba histolytica.
4. Courvoisier's.
5. BRCA1; BRCA2.

Short-answer questions

6. Biliary colic causes transient pain without fever, resolving as the stone disimpacts, and is managed electively. Acute cholecystitis causes continuous pain, fever, and Murphy's sign, and requires antibiotics and early cholecystectomy.
7. Emergency resuscitation, broad-spectrum antibiotics, and urgent biliary decompression by ERCP or percutaneous transhepatic cholangiography, followed by interval cholecystectomy.
8. Pyogenic abscess arises from biliary sepsis or portal pyaemia and is managed with drainage and antibiotics. Amoebic abscess is caused by Entamoeba histolytica and usually responds to metronidazole alone.
9. Severity scoring (Glasgow-Imrie or APACHE II) is performed within 48 hours to identify patients at risk of severe disease requiring high-dependency or intensive care.

10. A hard, irregular, fixed lump, skin dimpling or peau d'orange, nipple retraction or bloody discharge, and new axillary lymphadenopathy.

Long-answer questions

11. **Basic response:** Gallstone disease presents with biliary colic or acute cholecystitis, diagnosed by ultrasound, and managed with laparoscopic cholecystectomy for symptomatic disease.

Value-added response: Complicated presentations require additional steps: ERCP for choledocholithiasis, urgent decompression for cholangitis, and supportive care with early ERCP for gallstone pancreatitis.

12. **Basic response:** Choledocholithiasis is managed by ERCP with stone extraction. Cholangitis requires emergency decompression. Gallstone pancreatitis is managed supportively with early ERCP if obstruction persists.

Value-added response: Mirizzi syndrome should be recognised preoperatively as it increases the risk of bile duct injury during cholecystectomy and may require a modified surgical approach.

13. **Basic response:** Acute pancreatitis is diagnosed by pain, raised amylase or lipase, and imaging findings, managed with intravenous fluids, analgesia, and early enteral nutrition.

Value-added response: Severity scoring within 48 hours identifies patients needing high-dependency or intensive care, and the underlying cause (gallstones or alcohol) is treated once the patient is stable.

14. **Basic response:** Triple assessment combines clinical examination, imaging, and pathology for any new breast symptom, ensuring no single modality is relied upon alone.

Value-added response: Red flag features such as a hard fixed lump, skin dimpling, or nipple changes should prompt urgent referral, distinguishing them from reassuring features of fibroadenoma or fibrocystic change.

15. **Basic response:** Breast cancer is staged using the TNM system, and management combines surgery (breast-conserving surgery or mastectomy) with adjuvant therapy guided by receptor status.

Value-added response: Receptor-directed therapy includes chemotherapy for high-risk or triple-negative disease, endocrine therapy for hormone receptor-positive disease, and trastuzumab for HER2-positive disease.

Answers to Pilot Questions [Series 5]

Part 5.1

1. Cholesterol stones are most common, linked to the five Fs. Pigment stones form from excess bilirubin, related to haemolysis or infection.
2. Biliary colic is transient without fever, managed electively. Acute cholecystitis is continuous with fever and Murphy's sign, requiring antibiotics and early surgery.
3. Murphy's sign is arrest of inspiration on palpation of the right upper quadrant, indicating acute cholecystitis.
4. Abdominal ultrasound is first-line, showing gallstones, wall thickening, and pericholecystic fluid.
5. Laparoscopic cholecystectomy is the gold standard for symptomatic gallstone disease.

Part 5.2

1. Choledocholithiasis causes obstructive jaundice with pale stools, dark urine, pruritus, and a cholestatic liver function pattern.
2. Charcot's triad is fever, jaundice, and right upper quadrant pain. Reynolds' pentad adds hypotension and confusion, indicating septic shock.
3. Resuscitation, broad-spectrum antibiotics, and urgent biliary decompression by ERCP or percutaneous transhepatic cholangiography.
4. A stone impacts at the ampulla of Vater, obstructing both the biliary and pancreatic ducts and triggering enzyme activation and inflammation.
5. Mirizzi syndrome is extrinsic compression of the common hepatic duct by a cystic duct stone, increasing the risk of bile duct injury at surgery.

Part 5.3

1. The liver has eight independent functional segments (Couinaud classification), each with its own vascular inflow, outflow, and biliary drainage, allowing segment-specific resection.

2. The Child-Pugh score grades chronic liver disease severity using bilirubin, albumin, prothrombin time, ascites, and encephalopathy, predicting operative risk.
3. Pyogenic abscess arises from biliary sepsis or portal pyaemia and is drained with antibiotics. Amoebic abscess is caused by Entamoeba histolytica and usually responds to metronidazole.
4. Haemangioma is the most common, usually incidental. Focal nodular hyperplasia and hepatic adenoma are also benign, with adenoma carrying rupture and malignant transformation risk.
5. Hepatocellular carcinoma is associated with chronic hepatitis B and C and cirrhosis, diagnosed by imaging characteristics and raised alpha-fetoprotein.

Part 5.4

1. At least two of three criteria: characteristic pain, raised amylase or lipase more than three times normal, and characteristic imaging findings.
2. Severity scoring (Glasgow-Imrie or APACHE II) within 48 hours identifies patients at risk of severe disease needing high-dependency or intensive care.
3. Chronic pancreatitis causes recurrent pain, and later steatorrhoea and weight loss (exocrine insufficiency) and diabetes mellitus (endocrine insufficiency).
4. Courvoisier's law states that a palpable, non-tender gallbladder in a jaundiced patient is unlikely to be due to gallstones, suggesting malignant obstruction instead.
5. Resectable disease is managed with pancreaticoduodenectomy (Whipple's procedure); unresectable disease is managed with palliative chemotherapy and biliary stenting.

Part 5.5

1. Clinical examination, imaging (ultrasound or mammography), and pathology (biopsy or cytology), ensuring no single modality is relied upon alone.
2. A hard, irregular, fixed lump, skin dimpling or peau d'orange, nipple retraction or bloody discharge, and new axillary lymphadenopathy.
3. Fibroadenoma is a smooth, firm, highly mobile lump in young women, managed conservatively unless large, growing, or symptomatic.

4. Fibrocystic change causes lumpiness and cyclical pain with simple cysts on ultrasound, managed with aspiration if symptomatic.
5. Mastitis is managed with continued feeding, analgesia, and antibiotics if needed. Breast abscess is managed with ultrasound-guided aspiration, or incision and drainage if it fails.

Part 5.6

1. Increasing age, early menarche, late menopause, nulliparity or late first pregnancy, family history, and BRCA1 or BRCA2 mutations, with later presentation common in Nigeria.
2. BRCA1 and BRCA2 mutations substantially raise lifetime breast and ovarian cancer risk, guiding genetic counselling, surveillance, and risk-reducing surgery decisions.
3. The TNM system stages tumour size, nodal involvement, and metastasis, supplemented by imaging when metastatic disease is suspected.
4. The choice between breast-conserving surgery with radiotherapy and mastectomy depends on tumour size relative to breast size, multifocality, and patient preference.
5. Chemotherapy is used for high-risk or triple-negative disease, endocrine therapy for hormone receptor-positive disease, and trastuzumab for HER2-positive disease.

References and Attributions [Series 5]

Textual References

- Bailey, H. and Love, R. J. M. (2018). Bailey and Love's Short Practice of Surgery (27th ed.). CRC Press.
- Townsend, C. M., Beauchamp, R. D., Evers, B. M. and Mattox, K. L. (2017). Sabiston Textbook of Surgery (20th ed.). Elsevier.
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- Cardoso, F., Kyriakides, S., Ohno, S. et al. (2019). Early Breast Cancer: ESMO Clinical Practice Guidelines. Annals of Oncology, 30(8), 1194 to 1220.

Figures, Plates, Tables, and Boxes

- Figure 5.1: Gallstone formation and comparison of biliary colic with acute cholecystitis. AI-generated using the prompt: "Create a professional two-panel medical diagram titled Gallstone Disease, showing stone formation and risk factors, and a comparison of biliary colic versus acute cholecystitis. Clean clinical surgical diagram style."
- Figure 5.2: Pathways from common bile duct stone to the major complications of gallstone disease. AI-generated using the prompt: "Create a professional medical flowchart titled Complications of Gallstone Disease, branching from a common bile duct stone to choledocholithiasis, cholangitis, and gallstone pancreatitis with their key features and management. Clean clinical surgical diagram style."
- Figure 5.3: Couinaud segmental liver anatomy and comparison of liver abscess types. AI-generated using the prompt: "Create a professional medical diagram titled Liver Anatomy and Disease, showing the eight Couinaud liver segments with vascular inflow and outflow, plus a comparison panel of pyogenic versus amoebic liver abscess. Clean clinical surgical diagram style."
- Table 5.4: Modified Glasgow-Imrie criteria for severity assessment of acute pancreatitis. AI-generated using the prompt: "Create a clean two-column reference table titled Modified Glasgow-Imrie Criteria for Acute Pancreatitis, listing each criterion and its threshold value. Simple clinical reference table style with outside border."
- Box 5.5: Red flag features of a breast lump requiring urgent referral. AI-generated using the prompt: "Create a simple single-column reference box with an outside border titled

Red Flag Features of a Breast Lump, listing five red flag clinical features in a clean bulleted list. Clinical reference box style."

- Figure 5.6: TNM staging of breast cancer and receptor-directed management pathway. AI-generated using the prompt: "Create a professional two-panel medical diagram titled Breast Cancer Staging and Management, showing TNM staging components and a receptor-status-directed treatment pathway. Clean clinical surgical diagram style."