

Definitions and Classifications

Lymphoid neoplasms

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Learning outcomes

- Distinguish leukemia and lymphoma as patterns of presentation, not mutually exclusive tumor classes.
- Organize lymphoid neoplasms by lineage and differentiation stage.
- Relate anatomic distribution, secretory effects, and basic laboratory studies to classification.
- Apply the morphology–immunophenotype–genotype–clinical framework.

Names describe where disease is found

- Leukemia and lymphoma originally denoted apparently distinct disorders.
- Overlap in marrow, blood, and tissue involvement blurs that distinction.
- The terminology mainly reflects the usual distribution at presentation.

Leukemia

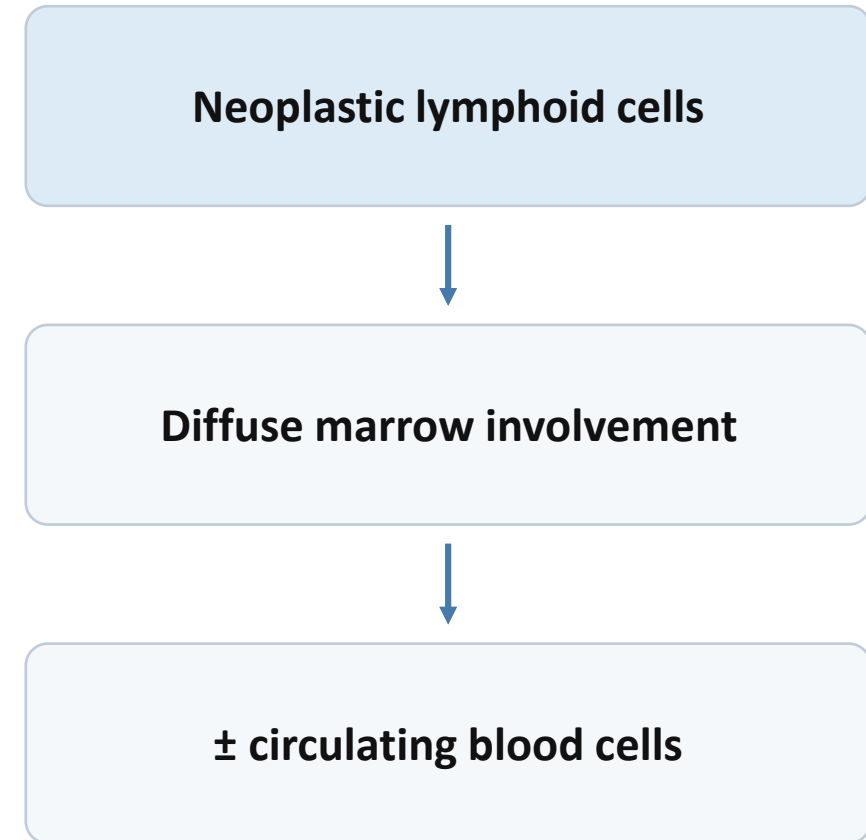
Marrow ± blood

Lymphoma

Tissue mass

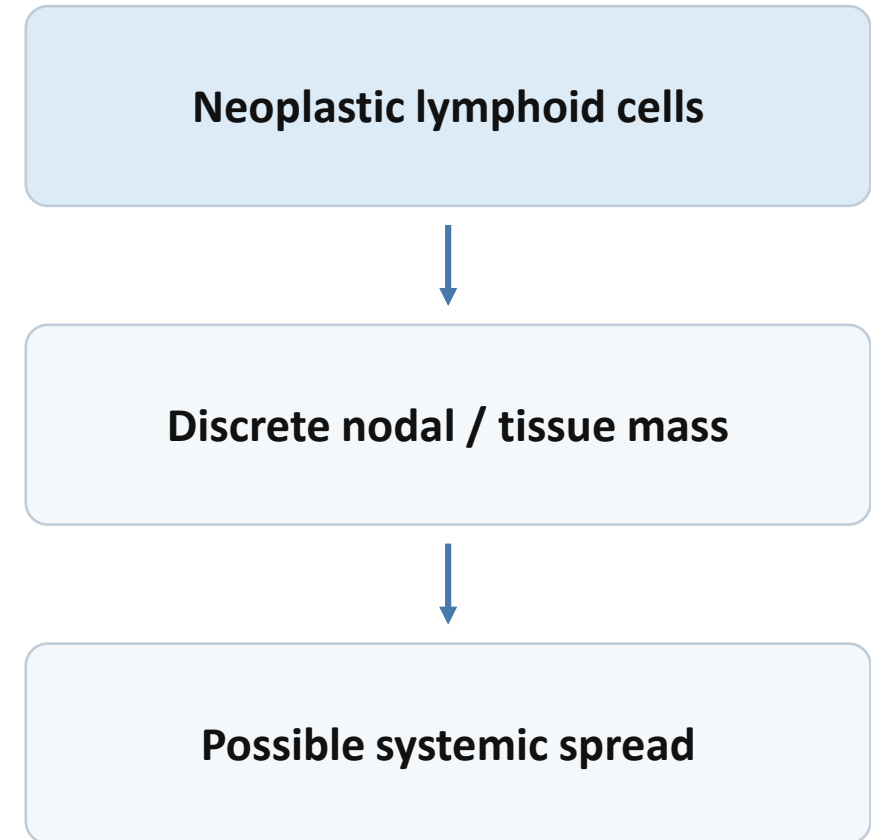
Leukemia: Marrow-centered presentation

- Widespread marrow involvement defines the traditional leukemic pattern.
- Peripheral blood involvement is usual but not obligatory.
- The classification does not require a discrete tissue mass.



Lymphoma: Tissue-mass presentation

- A lymphoma commonly forms a discrete mass of lymphoid cells.
- Lymph nodes are frequent sites, but extranodal organs are also involved.
- The word lymphoma does not exclude simultaneous marrow or blood disease.



Leukemia and lymphoma can be one entity

- Certain named lymphomas acquire substantial circulating tumor cells.
- Tumors identical to leukemias can arise as masses without detectable marrow disease.
- Site of first detection alone cannot establish biologic lineage.

Lymphomatous phase

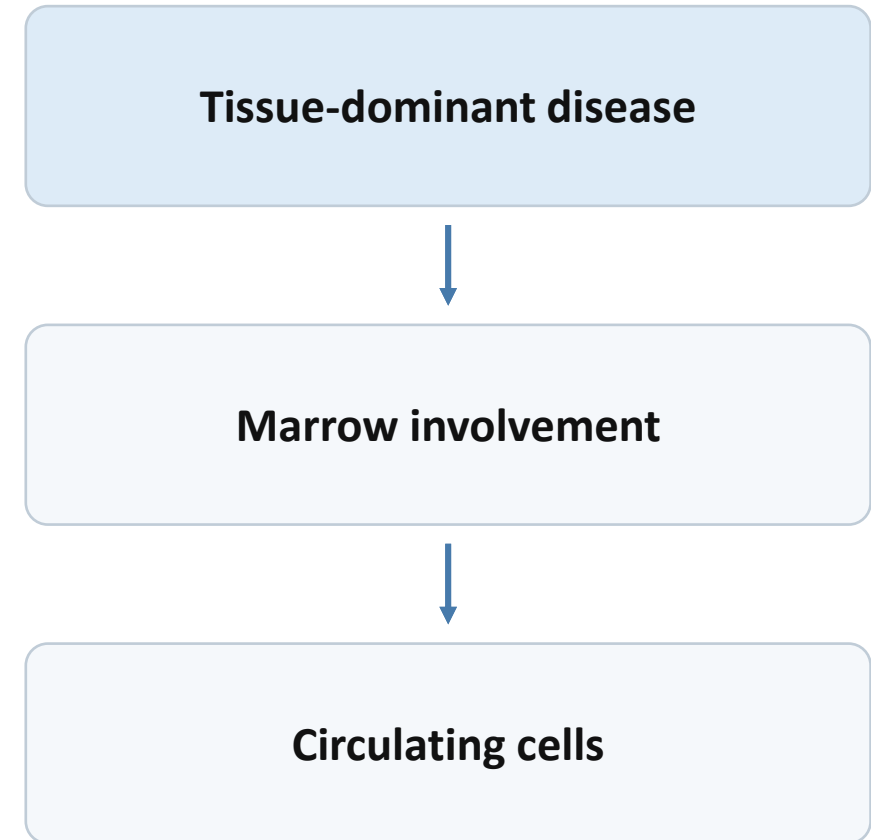
Mass-dominant

Leukemic phase

Marrow / blood-dominant

Changing distribution changes the clinical label

- An initially mass-forming lymphoma can later produce a leukemic presentation.
- This progression is not unusual in incurable lymphomas.
- Reassessment of the same entity must consider all involved compartments.



B-ALL and T-ALL illustrate naming overlap

- B-ALL and T-ALL are precursor-cell neoplasms in the same broad diagnostic family.
- Their names deliberately combine leukemia and lymphoma.
- Distribution and lineage are separate dimensions of classification.

Immature B lineage

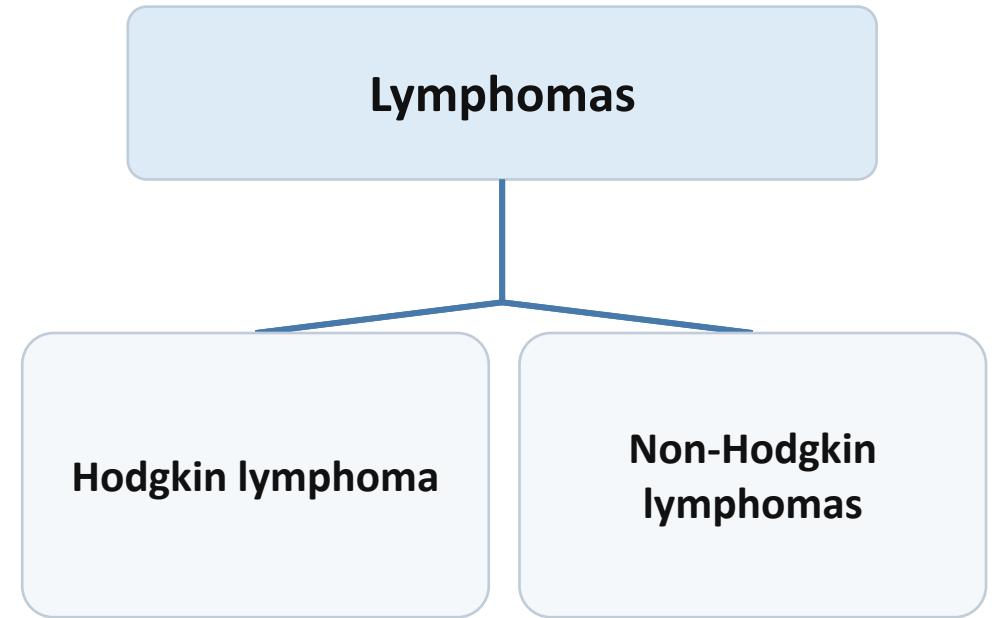
B-ALL / lymphoma

Immature T lineage

T-ALL / lymphoma

Hodgkin and non-Hodgkin lymphomas

- Hodgkin lymphoma is separated because of distinctive pathologic characteristics.
- The other lymphoma groups are conventionally termed non-Hodgkin lymphomas (NHLs).
- The distinction also has clinical and treatment implications.



Non-Hodgkin lymphoma is an umbrella term

- NHL encompasses multiple B-, T-, and NK-lineage neoplasms.
- Individual entities differ in differentiation stage and anatomic behavior.
- An NHL label is not a sufficient final disease classification.

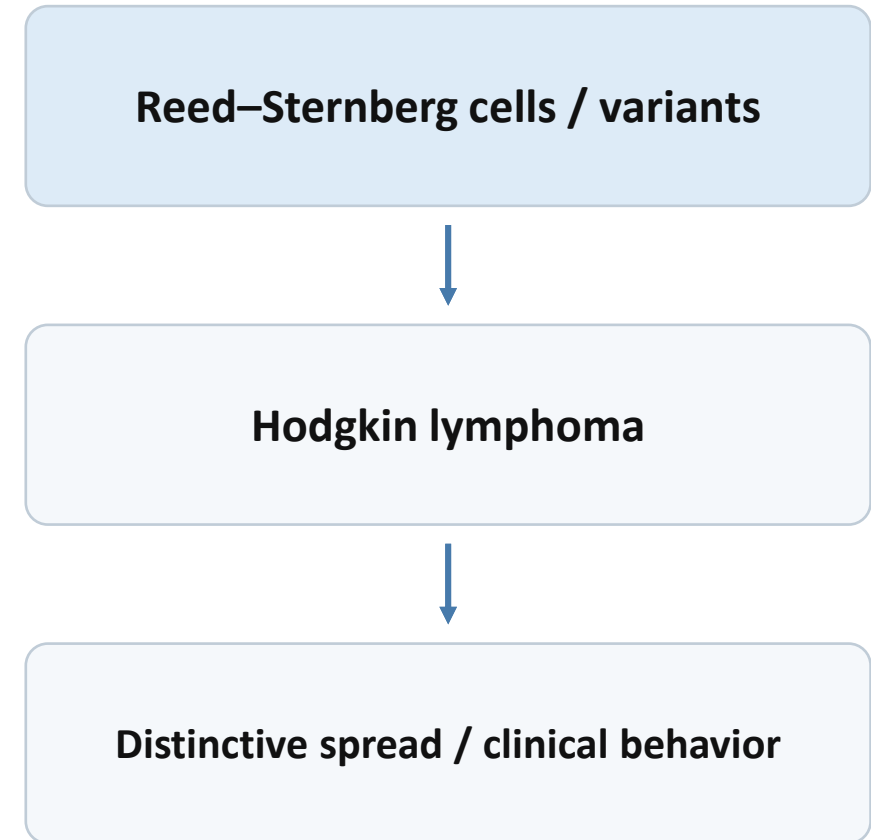
B-cell neoplasms

T-cell neoplasms

NK-cell neoplasms

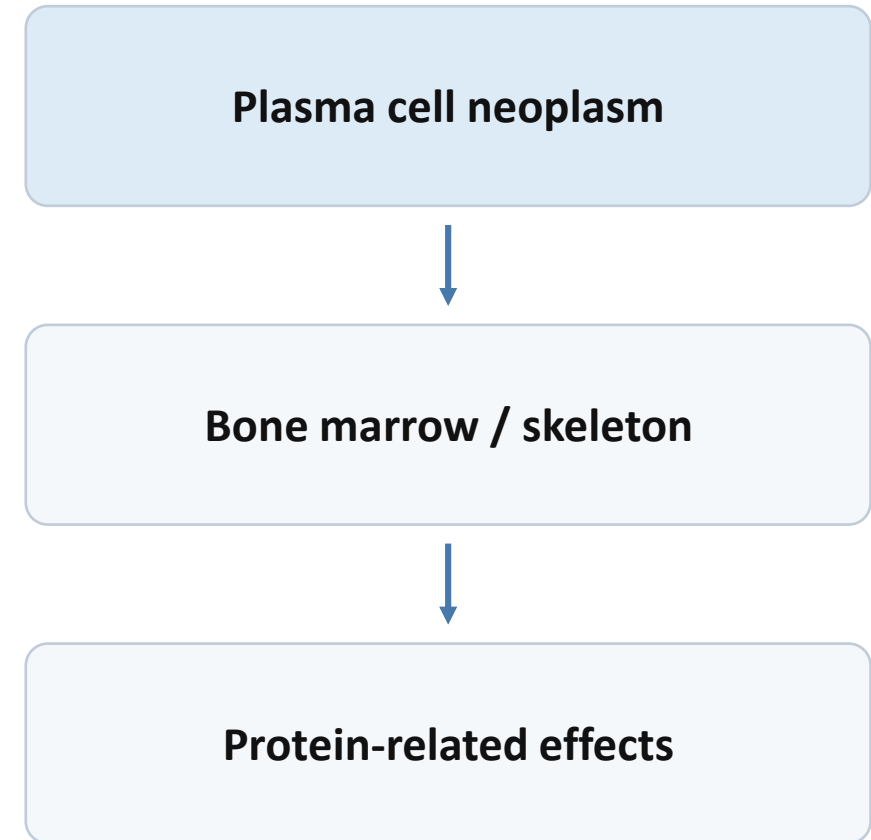
Hodgkin lymphoma is a special family

- The category is organized around Reed–Sternberg cells and their variants.
- Classic subtypes and a nodular lymphocyte-predominant form.
- Its spread pattern differs from many NHLs.



Plasma cell neoplasms form a special group

- Plasma cell tumors most often originate in bone marrow.
- Peripheral blood and lymph nodes are less frequently involved.
- Their clinical consequences often derive from skeletal disease or secreted immunoglobulin.



Three dimensions must be kept separate

- Distribution: Leukemic, nodal, or extranodal.
- Biology: B-, T-, NK-, or Hodgkin-associated tumor lineage.
- Differentiation: Precursor (immature) or peripheral (mature).

Distribution

Lineage

Maturation

Anatomy often predicts first symptoms

- The dominant organ compartment largely determines presentation.
- Nodal enlargement, extranodal dysfunction, marrow failure, or bone destruction are different patterns.
- Secreted proteins and cytokines can generate additional systemic symptoms.

Lymph nodes

Extranodal organs

Marrow

Skeleton

Nodal lymphoma: Painless enlargement

- About two-thirds of NHLs present with enlarged lymph nodes account.
- Virtually all Hodgkin lymphomas present with nodal enlargement.
- The nodes are often nontender; Palpation alone is not a tissue diagnosis.

Non-Hodgkin lymphoma

Often nodal

Hodgkin lymphoma

Typically nodal

Extranodal NHL affects many organs

- The remaining NHLs may present through extranodal involvement.
- Skin, stomach, and brain as examples.
- Organ-specific symptoms may precede recognition of a lymphoid mass.

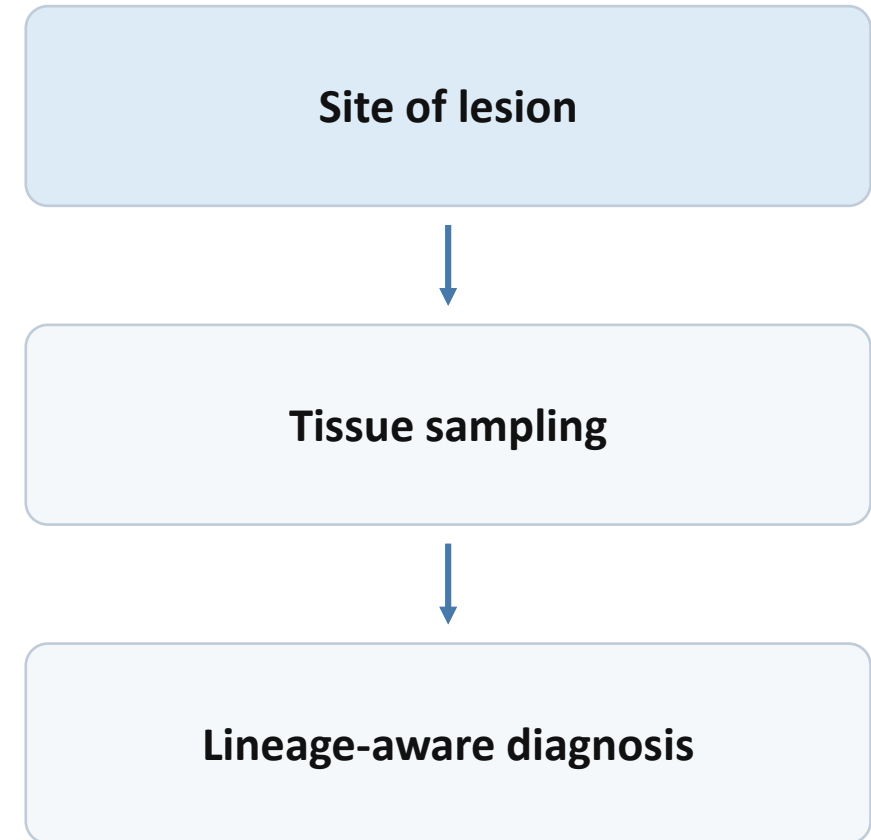
Skin

Stomach

Brain

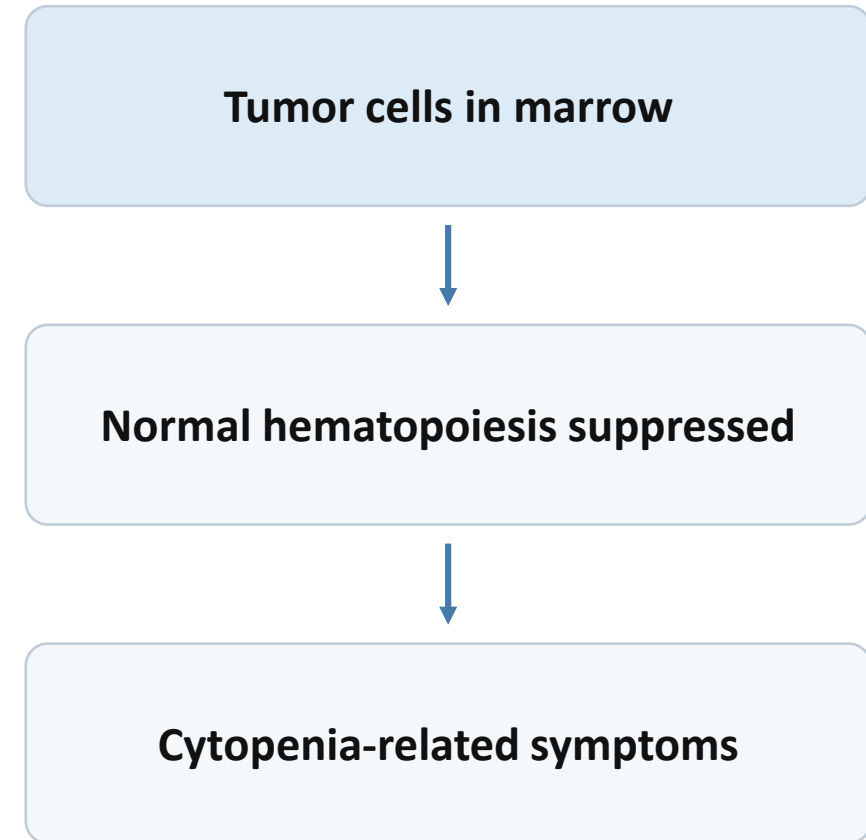
Extranodal location changes the clinical question

- A skin lesion directs attention to cutaneous lymphoid neoplasia.
- A gastric lesion raises the possibility of a primary or secondary lymphoid tumor.
- An intracranial lesion may require evaluation for a lymphoid process.



Leukemia often suppresses hematopoiesis

- Tumor cells occupying marrow compromise normal blood-cell production.
- Clinical manifestations therefore often reflect marrow failure.
- Peripheral blood may show tumor cells but their presence is not obligatory.



Marrow failure produces three clinical signals

- Reduced erythropoiesis can contribute to anemia-related symptoms.
- Reduced normal leukocyte output can predispose to infection.
- Reduced platelet production can increase bleeding risk.

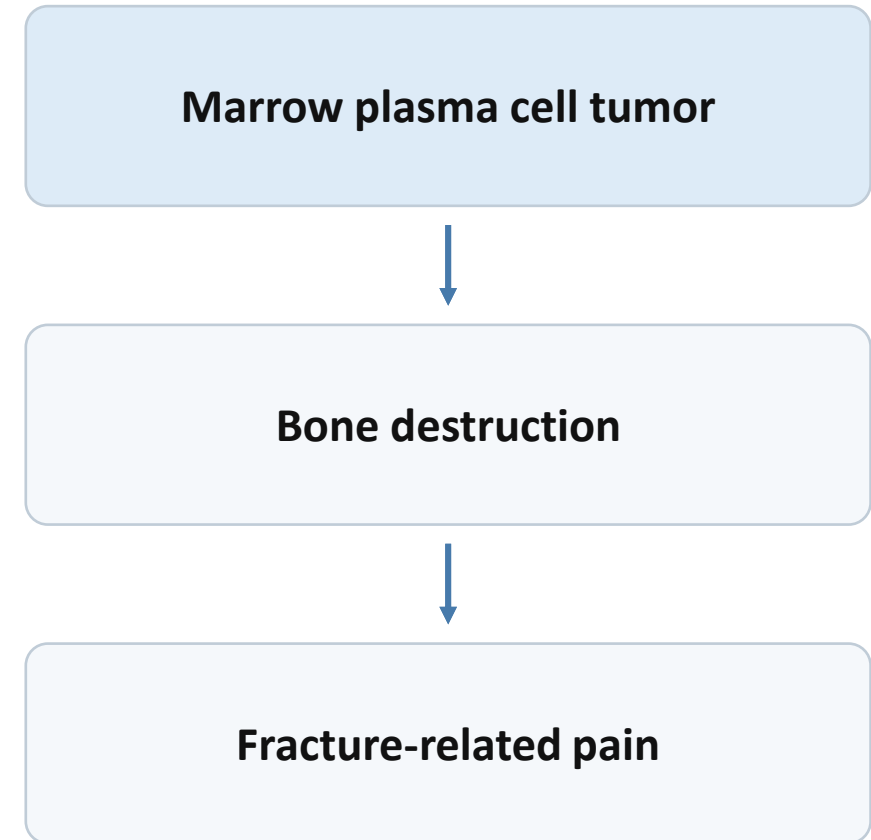
**Erythroid cells →
anemia**

**Normal leukocytes →
infection**

Platelets → bleeding

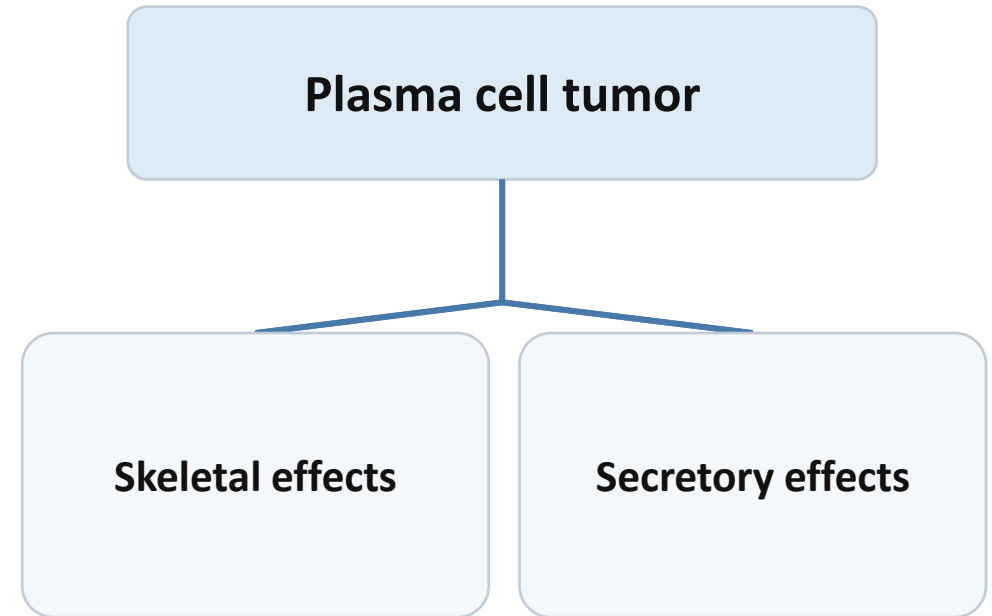
Myeloma damages the skeleton

- Multiple myeloma is the most common plasma cell neoplasm described in this passage.
- Skeletal destruction creates mechanically weakened bone.
- Pain can result from pathologic fractures.



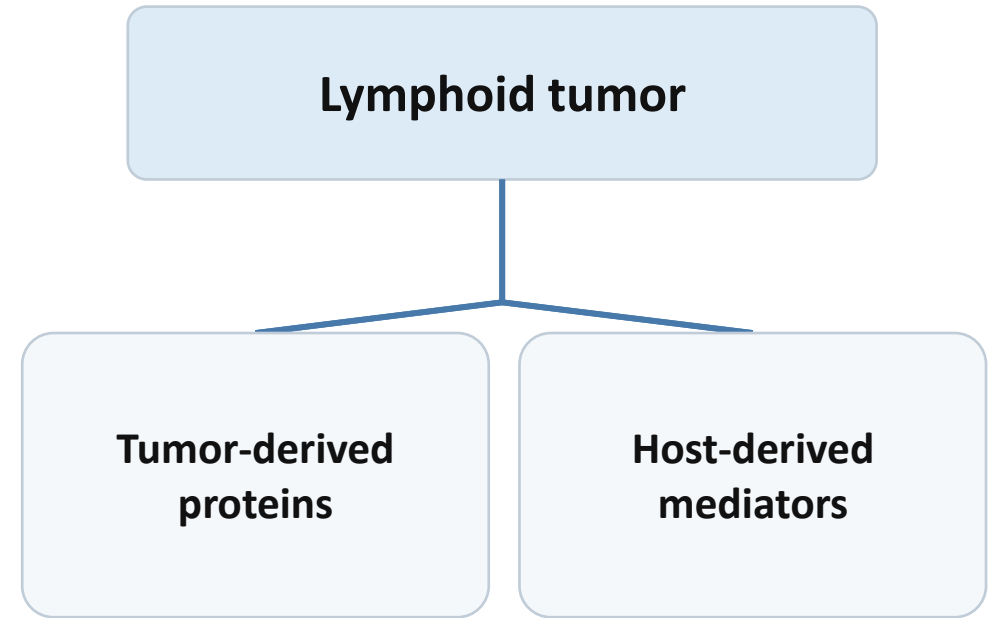
Bone disease and protein disease differ

- Skeletal lesions account for one major group of myeloma-related manifestations.
- Secreted whole immunoglobulins or fragments account for another.
- A single plasma cell neoplasm can cause both.



Secreted products can dominate disease effects

- Not every lymphoma symptom is caused by direct tissue replacement.
- Products made by tumor cells may alter distant organ function.
- Host immune cells responding to tumor can also release clinically important mediators.



Plasma cells may secrete intact or partial Ig

- Plasma cell neoplasms can release whole immunoglobulin molecules.
- Some release immunoglobulin fragments.
- Consequences of the secreted proteins may be central to clinical presentation.

Whole immunoglobulin

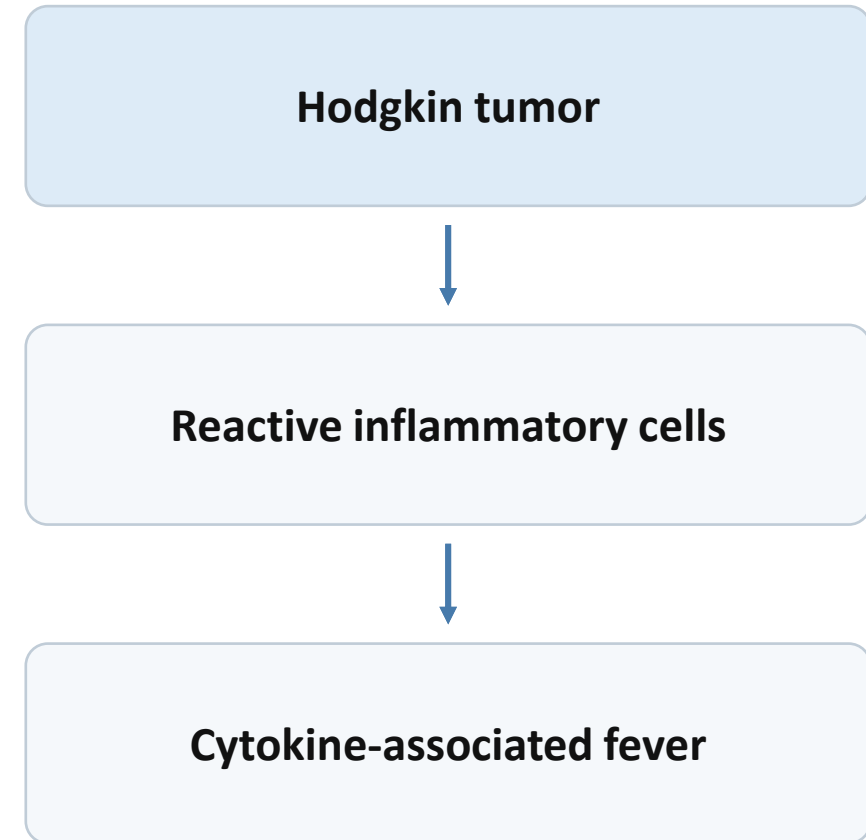
Intact antibody

Ig fragments

Partial protein products

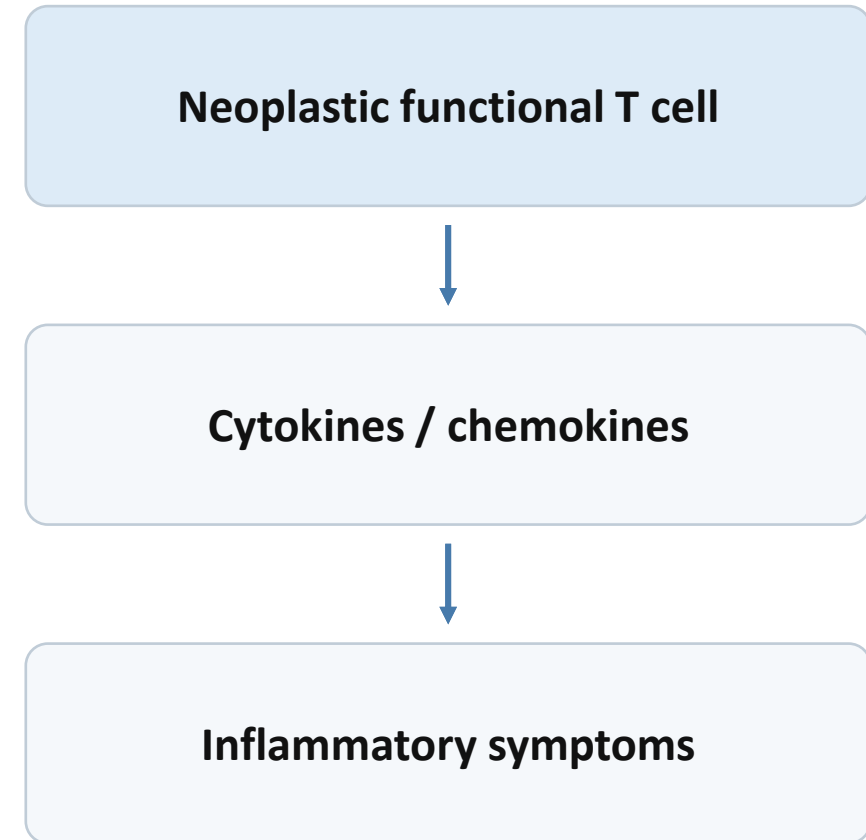
Hodgkin lymphoma: Host-cell cytokines

- Fever can occur in Hodgkin lymphoma.
- The textbook links this systemic symptom to cytokines released by nonneoplastic inflammatory cells.
- The reactive tumor microenvironment can therefore shape the phenotype.



Peripheral T-cell tumors: Tumor cytokines

- Some peripheral T-cell lymphomas retain functional T-cell properties.
- Neoplastic T cells can release proinflammatory cytokines and chemokines.
- This mechanism differs from the reactive-cell cytokines emphasized in Hodgkin lymphoma.



Distinguish mass effect from soluble effects

- Direct effects arise from nodal, organ, marrow, or bone infiltration.
- Indirect effects arise from immunoglobulin or cytokine secretion.
- Both mechanisms may contribute to the same patient's symptoms.

Direct tumor burden

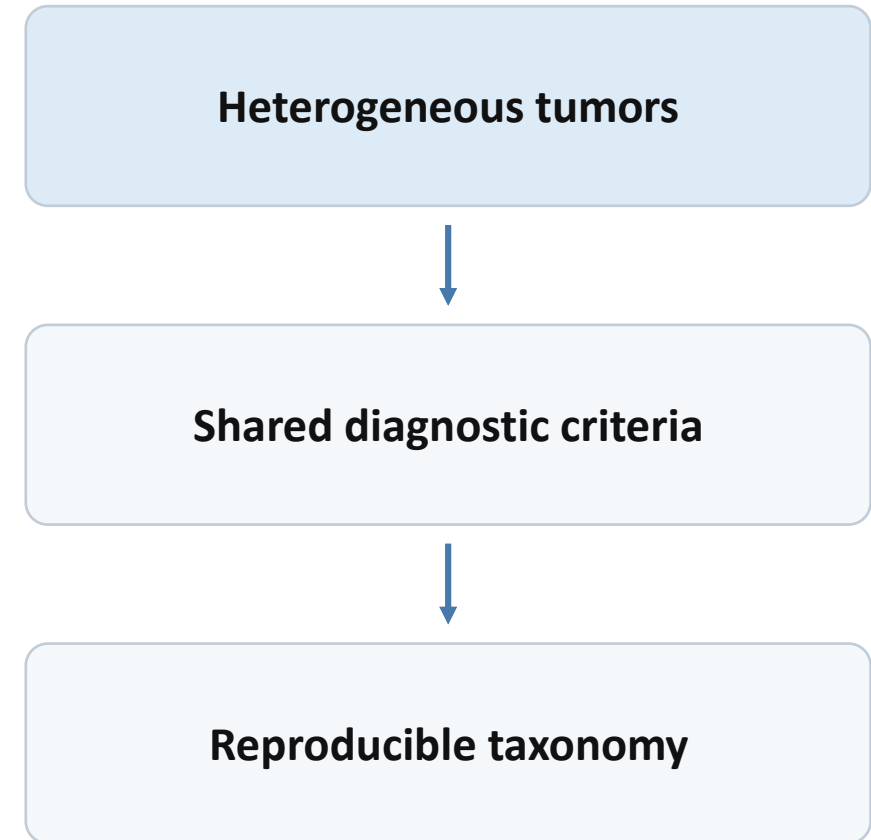
Anatomic effects

Secreted mediators

Systemic effects

Why classification needs a shared system

- Lymphoid neoplasms contain numerous biologically distinct entities.
- Historical classification systems provoked major disagreements.
- Formal classification integrates morphology, differentiation, genetics, and clinical behavior.



WHO and ICC are distinct modern frameworks

- Competing WHO and International Consensus Classification (ICC) systems.
- The chapter teaches the WHO-based framework.
- Differences between classifications should not be silently blended.

WHO

Textbook framework

ICC

Separate consensus system

Four pillars support classification

- Morphology: Observable architecture and cytology.
- Immunophenotype: Proteins indicating lineage or maturation.
- Genotype and clinical findings: Molecular alterations and disease context.

Morphology

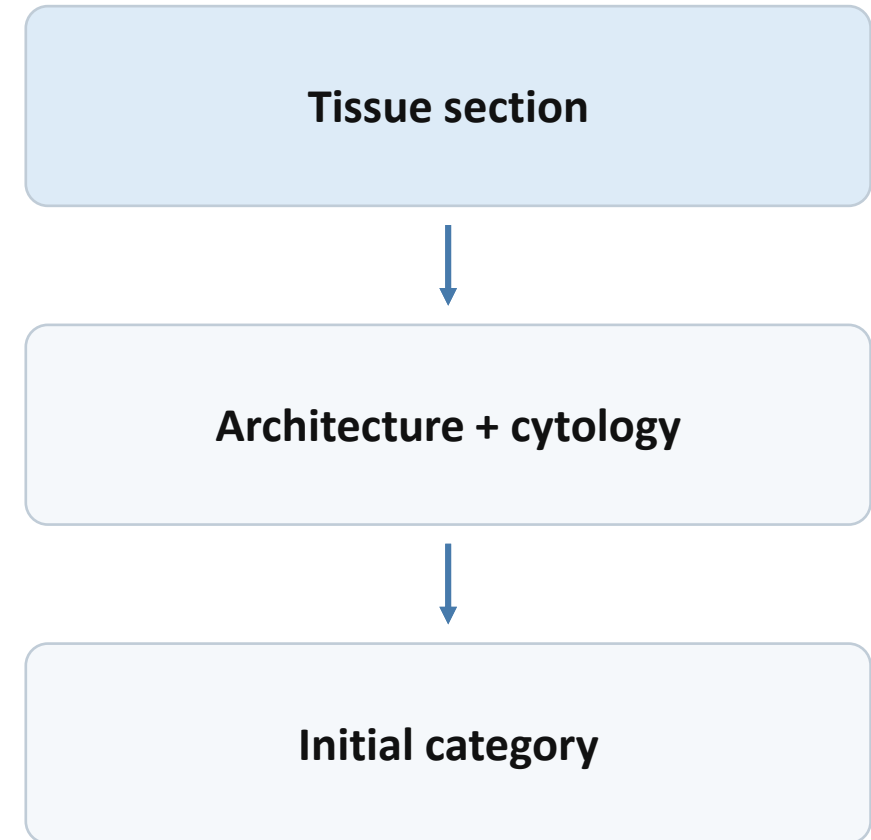
Immunophenotype

Genotype

Clinical context

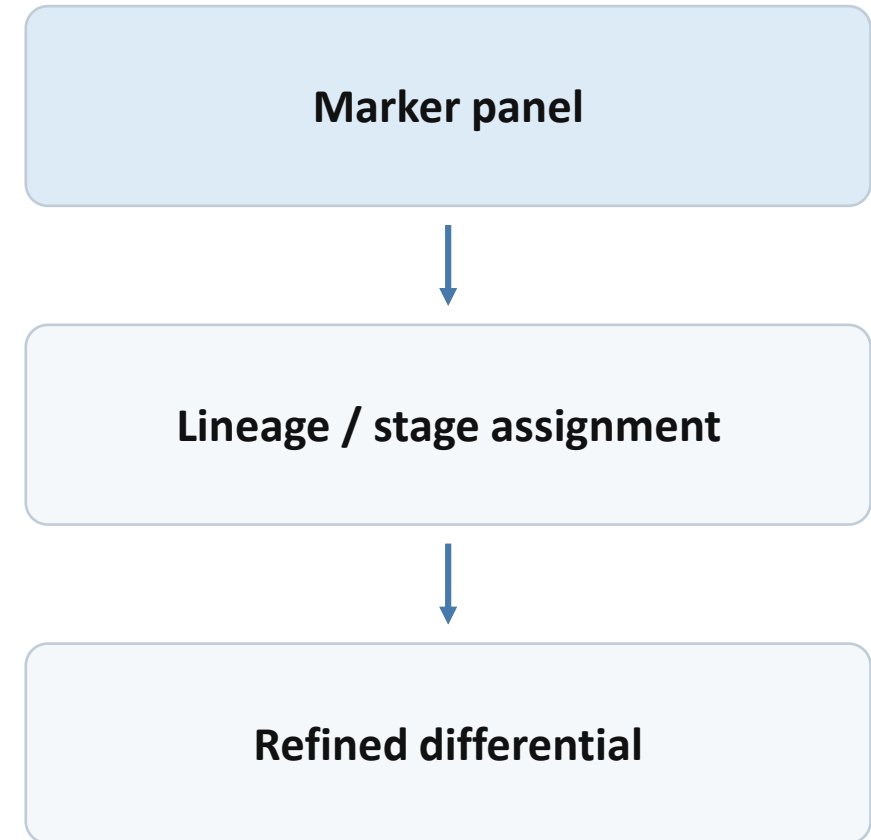
Morphology localizes the diagnostic problem

- Histology reveals tissue infiltration and patterns of architecture.
- Cytologic features help identify the proliferating cell population.
- Morphology is necessary but often not sufficient for final classification.



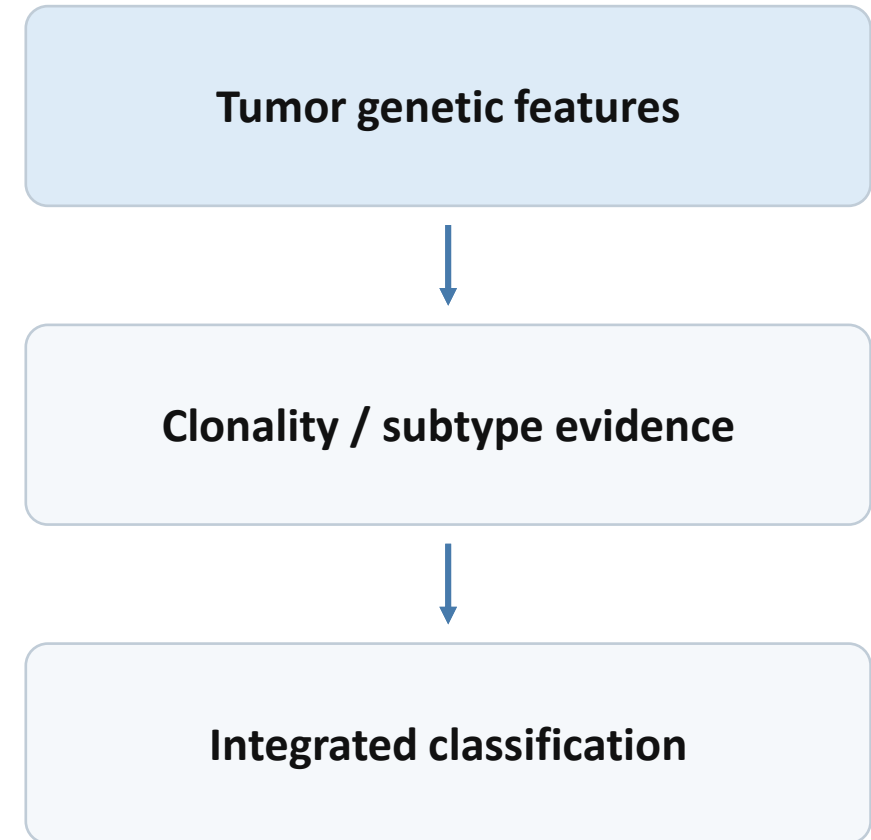
Immunophenotype defines differentiation

- Lineage-associated antigens characterize the neoplastic population.
- Patterns of marker expression are interpreted as a panel.
- The same antigen may occur on more than one normal lineage or maturation stage.



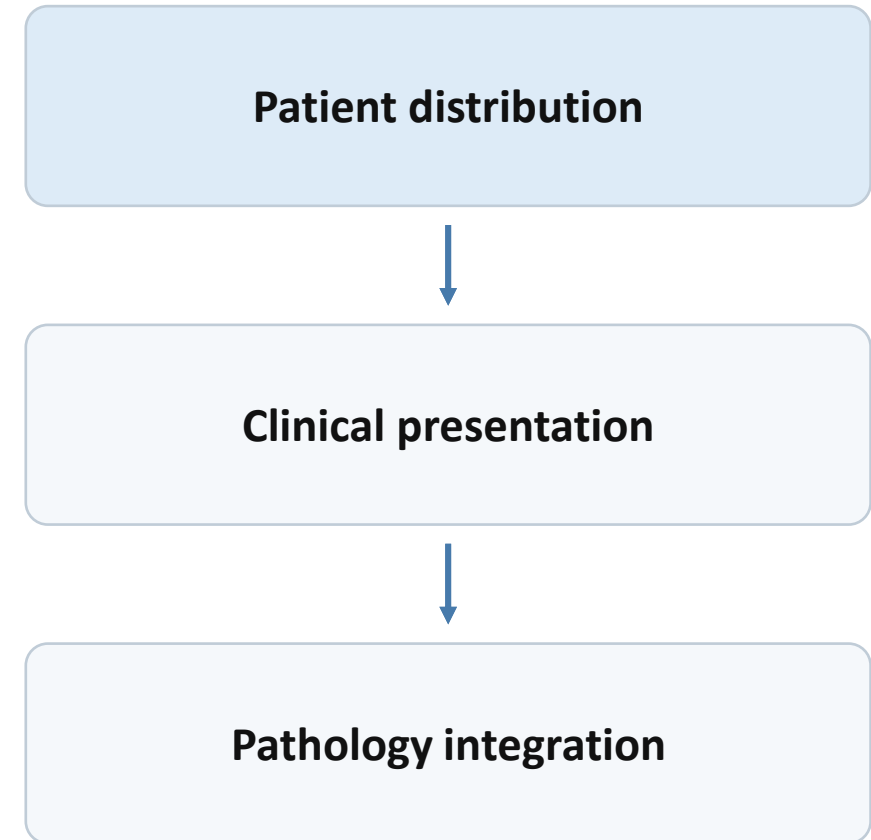
Genotype adds a clonal molecular dimension

- Lymphoid tumors may contain characteristic genetic changes.
- Antigen-receptor rearrangements can serve as clonal signatures.
- Molecular information complements, rather than replaces, tissue diagnosis.



Clinical findings are part of classification

- Presentation as a mass or marrow-centered disease conveys distribution.
- Site of disease provides differential-diagnostic context.
- Clinical information is integrated with laboratory and pathologic findings.



Five broad groups organize lymphoid tumors

- The textbook WHO summary groups tumors by lineage and stage of differentiation.
- Immature and mature B-cell, and immature and mature T/NK-cell, categories are separated.
- Hodgkin lymphoma is retained as a fifth category.

Precursor B-cell

Peripheral B-cell

Precursor T-cell

Peripheral T- / NK-cell

Hodgkin lymphoma

Precursor versus peripheral means immature versus mature

- “Precursor” designates immature lymphoid-cell neoplasms.
- “Peripheral” designates mature B-, T-, or NK-cell neoplasms.
- Stage of normal differentiation is used as an organizing principle.

Precursor

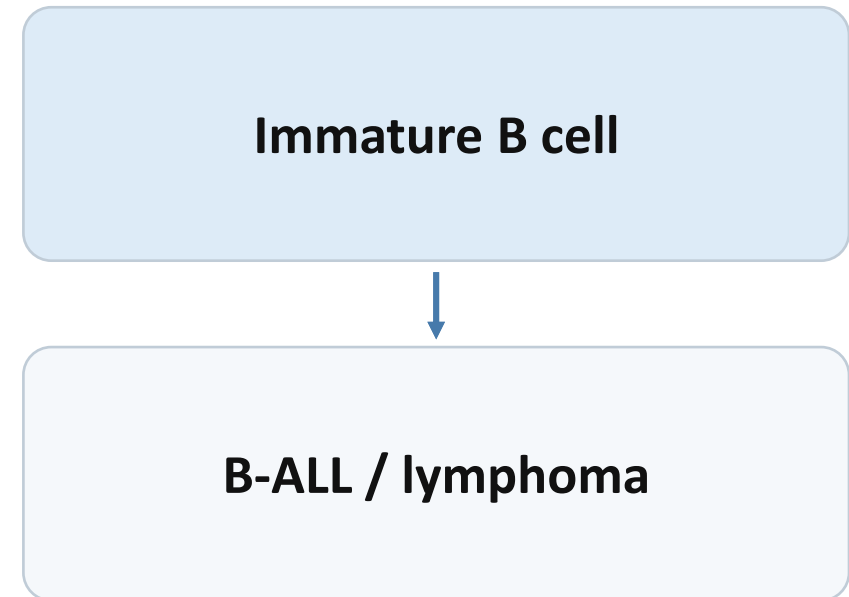
Immature lymphoid cell

Peripheral

Mature lymphoid cell

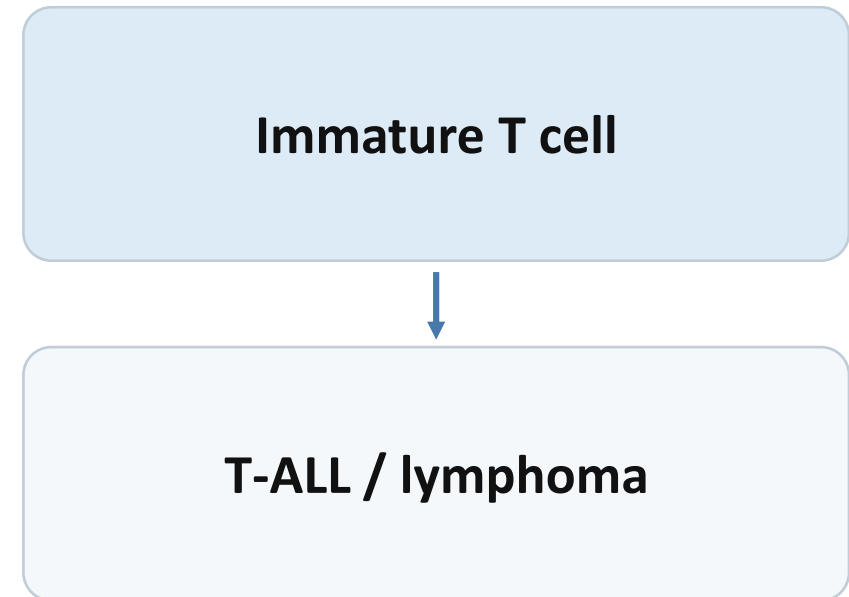
Precursor B-cell category

- The Robbins table lists B-cell acute lymphoblastic leukemia/lymphoma (B-ALL).
- This is a neoplasm of immature B-lineage cells.
- Leukemia and lymphoma presentations belong to the same umbrella entity.



Precursor T-cell category

- The Robbins table lists T-cell acute lymphoblastic leukemia/lymphoma (T-ALL).
- The neoplastic cells are immature T-lineage lymphoblasts.
- This category is distinct from mature peripheral T-cell lymphoma.



The two precursor categories are parallel

- Both are defined by immature lymphoid differentiation.
- B versus T identity establishes the branch.
- A leukemia/lymphoma presentation does not substitute for lineage determination.

B precursor

B-ALL / lymphoma

T precursor

T-ALL / lymphoma

Peripheral B-cell neoplasms: Overview

- This broad family comprises mature B-cell tumors.
- Nodal, splenic, leukemic, and plasma-cell-associated disease names.
- The enumerated examples are not exhaustive.

**Small / leukemic
names**

**Nodal lymphoma
names**

Splenic names

Plasma cell-related

Mature B-cell entities: CLL/SLL and splenic

- Chronic lymphocytic leukemia / small lymphocytic lymphoma is one named entity.
- Splenic B-cell lymphomas and leukemias form a listed group.
- Both illustrate why anatomic presentations coexist within mature B classification.

CLL / SLL

Paired leukemia–lymphoma name

Splenic B neoplasms

Splenic-associated grouping

Mature B-cell entities: Marginal and mantle

- Marginal zone lymphoma is a listed peripheral B-cell neoplasm.
- Mantle cell lymphoma is another distinct listed neoplasm.
- The normal counterpart and distinctive features require entity-specific evaluation.

**Marginal zone
lymphoma**

**Mantle cell
lymphoma**

Mature B-cell entities: Follicular and diffuse

- Follicular lymphoma and diffuse large B-cell lymphoma appear as separate named entities.
- Their names reflect different histologic concepts.
- Classification should not rely on a label fragment in isolation.

Follicular lymphoma

Named entity

Diffuse large B-cell lymphoma

Named entity

Mature B-cell entities: Burkitt and related

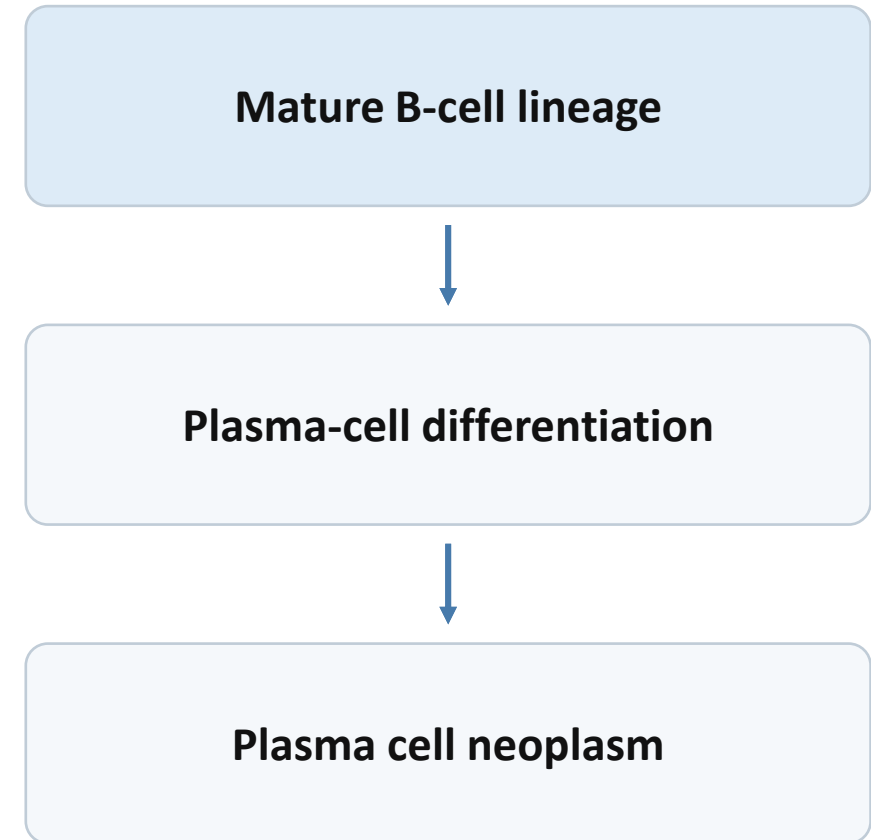
- Burkitt lymphoma is a distinct named peripheral B-cell neoplasm.
- Lymphoplasmacytic lymphoma is separately listed.
- The table enumerates entities without supplying complete diagnostic criteria.

Burkitt lymphoma

**Lymphoplasmacytic
lymphoma**

Plasma cell tumors belong in the B-cell spectrum

- The textbook places plasma cell neoplasms within the peripheral B-cell grouping.
- Plasma cells are terminally differentiated antibody-producing cells.
- Marrow-predominant involvement distinguishes their usual distribution.



Peripheral B-cell family at a glance

- The textbook table names CLL/SLL, splenic groups, marginal zone, and mantle cell lymphoma.
- It also names follicular, diffuse large B-cell, Burkitt, and lymphoplasmacytic lymphoma.
- Plasma cell neoplasms complete this summary list.

CLL / SLL	Splenic B-cell lymphomas
Marginal zone	Mantle cell
Follicular	Diffuse large B-cell
Burkitt	Lymphoplasmacytic
Plasma cell neoplasms	

Peripheral T-cell and NK-cell family

- The broad group includes tumors of mature T cells and NK cells.
- The Robbins examples span blood, skin, nodes, and extranodal sites.
- A site-based label is helpful but does not replace cellular classification.

Leukemic

Cutaneous

Nodal

Extranodal

Mature T-cell leukemic names

- T-cell prolymphocytic leukemia is listed.
- T-large granular lymphocytic leukemia is listed.
- Adult T-cell leukemia/lymphoma is a third named member of this family.

**T-prolymphocytic
leukemia**

**T-large granular
lymphocytic leukemia**

**Adult T-cell
leukemia/lymphoma**

Cutaneous T-cell names

- Mycosis fungoides and Sézary syndrome are each named in the peripheral T/NK grouping.
- These illustrate the importance of skin-related presentation.
- A cutaneous location still requires clinicopathologic evaluation.

Mycosis fungoides

Named cutaneous T-cell tumor

Sézary syndrome

Named cutaneous T-cell tumor

Nodal T-cell lymphoma names

- Peripheral T-cell lymphoma, not otherwise specified (NOS), is listed.
- Anaplastic large-cell lymphoma is separately listed.
- Nodal T-follicular helper cell lymphoma is separately listed.

**Peripheral T-cell
lymphoma, NOS**

**Anaplastic large-cell
lymphoma**

**Nodal T-follicular
helper lymphoma**

Extranodal T-cell lymphoma names

- The table includes enteropathy-associated T-cell lymphoma.
- Subcutaneous panniculitis-like T-cell and hepatosplenic T-cell lymphomas are listed.
- The names signal sites or patterns but are not themselves diagnostic criteria.

Enteropathy-associated

**Subcutaneous
panniculitis-like**

Hepatosplenic T-cell

Mature NK-cell-associated names

- Extranodal NK/T-cell lymphoma is included in this category.
- NK-cell leukemia is separately listed.
- These reflect an NK-associated branch, not a precursor B or T entity.

Extranodal NK/T-cell lymphoma

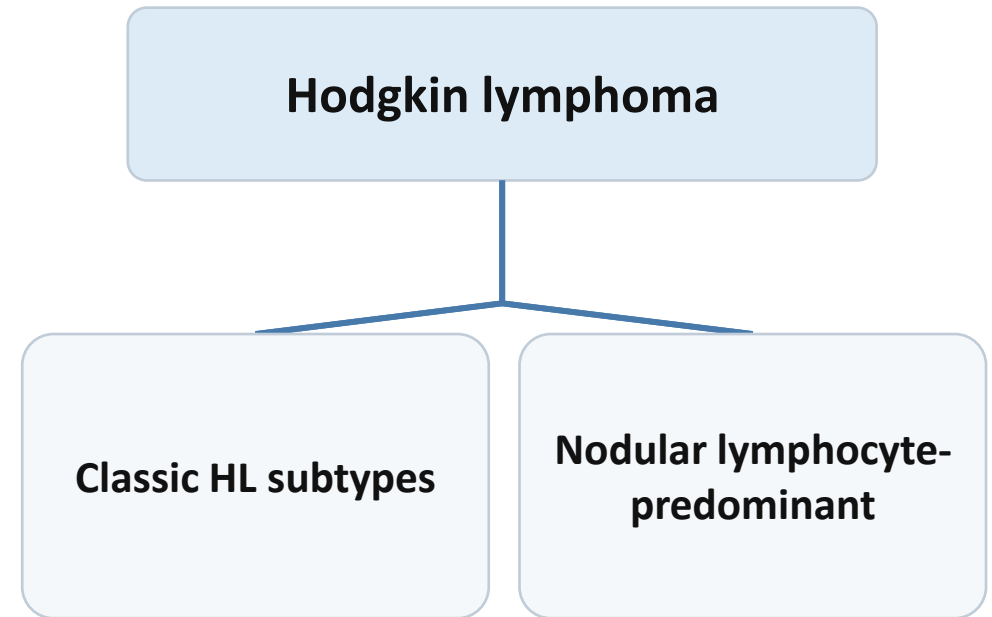
Named entity

NK-cell leukemia

Named entity

Hodgkin lymphoma: Two table branches

- The textbook groups classic Hodgkin lymphoma subtypes together.
- It also lists nodular lymphocyte-predominant disease.
- Terminology can vary among classification frameworks; Retain the source edition when quoting its taxonomy.



Classic Hodgkin lymphoma subtypes

- Nodular sclerosis and mixed cellularity are listed classic subtypes.
- Lymphocyte-rich and lymphocyte-depleted are also listed.
- These are named histopathologic subtypes; Criteria are detailed in the later Hodgkin section.

Nodular sclerosis

Mixed cellularity

Lymphocyte-rich

Lymphocyte-depleted

This table is a teaching summary, not a full atlas

- Robbins Table 13.4 explicitly omits some rare entities.
- Each named subtype demands more detail than a family-level classification slide provides.
- Use one specified classification version for a diagnostic report.

Textbook summary

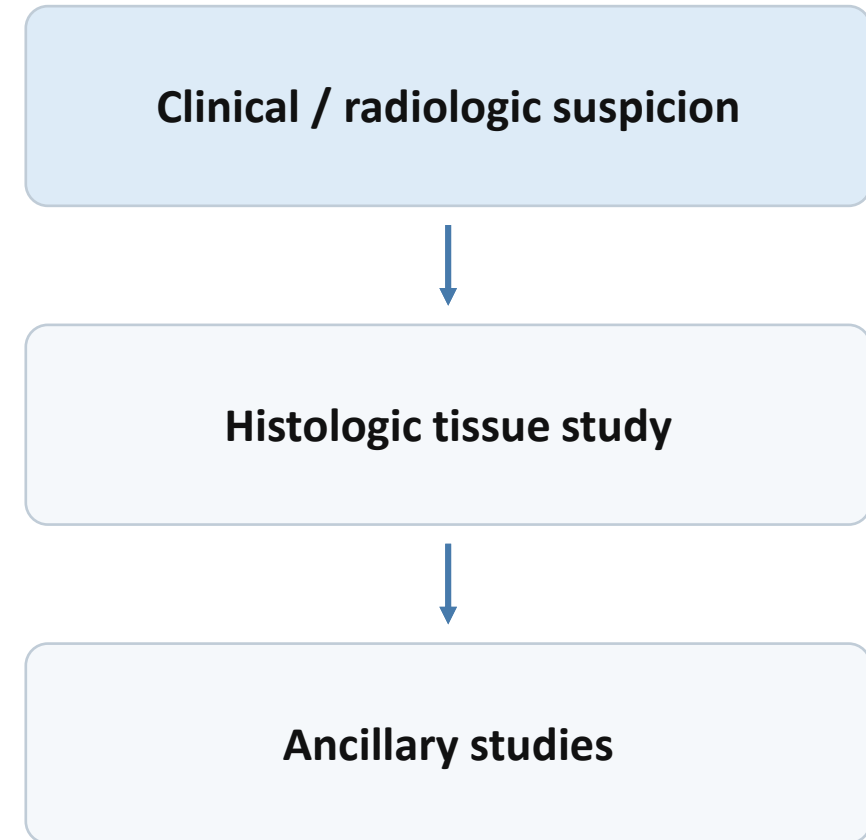
Broad teaching structure

Definitive classification

Entity-specific criteria

Clinical suspicion requires tissue confirmation

- Clinical findings can raise suspicion for a lymphoid neoplasm.
- The chapter states that histologic examination of involved tissue is required for diagnosis.
- Lineage marker and genetic studies are important complementary tests.



Morphology and markers answer different questions

- Histology determines tissue pattern and the nature of the cell proliferation.
- Protein markers support lineage, activation, and differentiation stage.
- Concordant findings yield a more defensible classification than either alone.

Morphology

Architecture + cytology

Immunophenotype

Protein expression

T-cell-associated markers are contextual

- CD3 is associated with thymocytes and mature T cells.
- CD4 marks helper T cells and a subset of thymocytes.
- CD8 marks cytotoxic T cells, some thymocytes, and some NK cells.

CD3

CD4

CD8

CD5 is not absolutely T-cell specific

- CD5 is expressed by T cells.
- A small subset of normal B cells also expresses CD5.
- It must be interpreted alongside a broader panel of markers.

T cells

Common CD5 expression

Subset of B cells

Also CD5 positive

B-cell-associated markers differ by stage

- CD19 marks pre-B cells and mature B cells, but not plasma cells.
- CD20 appears after CD19 during pre-B development and in mature B cells.
- CD79a marks marrow pre-B cells and mature B cells.

CD19

CD20

CD79a

CD10 is present in distinct B-cell stages

- The source lists CD10 on pre-B cells.
- CD10 is also present on germinal center B cells.
- This dual distribution limits interpretation of CD10 as a standalone stage marker.

Pre-B cells

CD10 can be expressed

Germinal center B cells

CD10 can be expressed

CD21 and CD23 add B-cell context

- CD21 is listed on mature B cells and follicular dendritic cells and acts as an EBV receptor.
- CD23 is associated with activated mature B cells.
- Neither should be interpreted without lineage, morphology, and other markers.

CD21

CD23

NK-associated markers have overlap

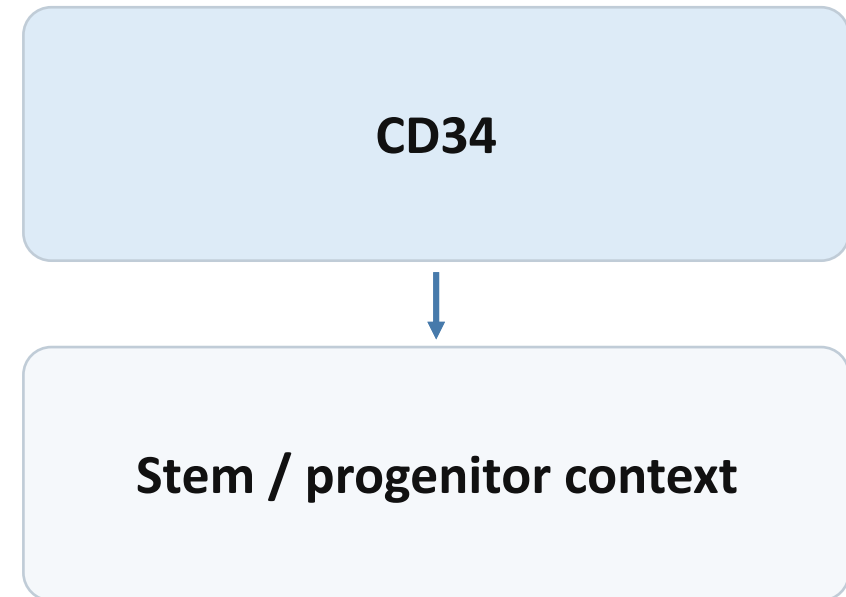
- CD16 is associated with NK cells and granulocytes.
- CD56 is associated with NK cells and a subset of T cells.
- Coexpression and clinical setting are more informative than a single positive stain.

CD16

CD56

CD34 indicates a progenitor compartment

- CD34 on hematopoietic stem cells and many progenitors.
- Its expression informs immaturity but does not, alone, determine lineage.
- The full classification still integrates morphology and other markers.



Reed–Sternberg-associated antigens

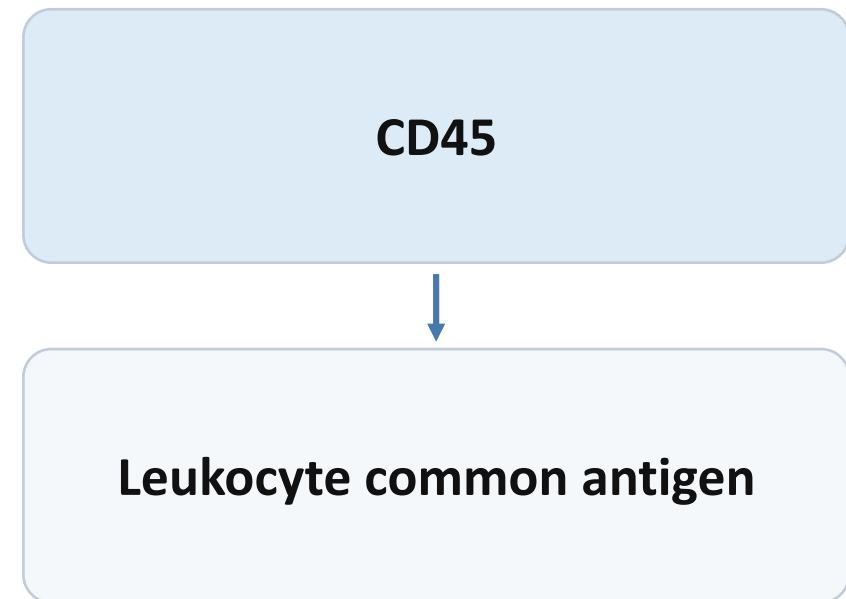
- CD15 is associated with granulocytes and Reed–Sternberg cells or variants.
- CD30 is an activation marker expressed by activated B cells, T cells, and monocytes.
- The textbook also lists CD30 on Reed–Sternberg cells and variants.

CD15

CD30

CD45 is a broad leukocyte marker

- CD45 is also called leukocyte common antigen (LCA).
- The table describes it as present on leukocytes.
- As with all markers, tumor context and staining patterns matter.



Some markers overlap the myeloid lineage

- CD11c occurs on granulocytes, monocytes, macrophages, and hairy-cell leukemias.
- CD13, CD14, CD33, and CD64 occur on differing myeloid or monocytic compartments.
- Lineage assignment requires interpretation of a coordinated panel.

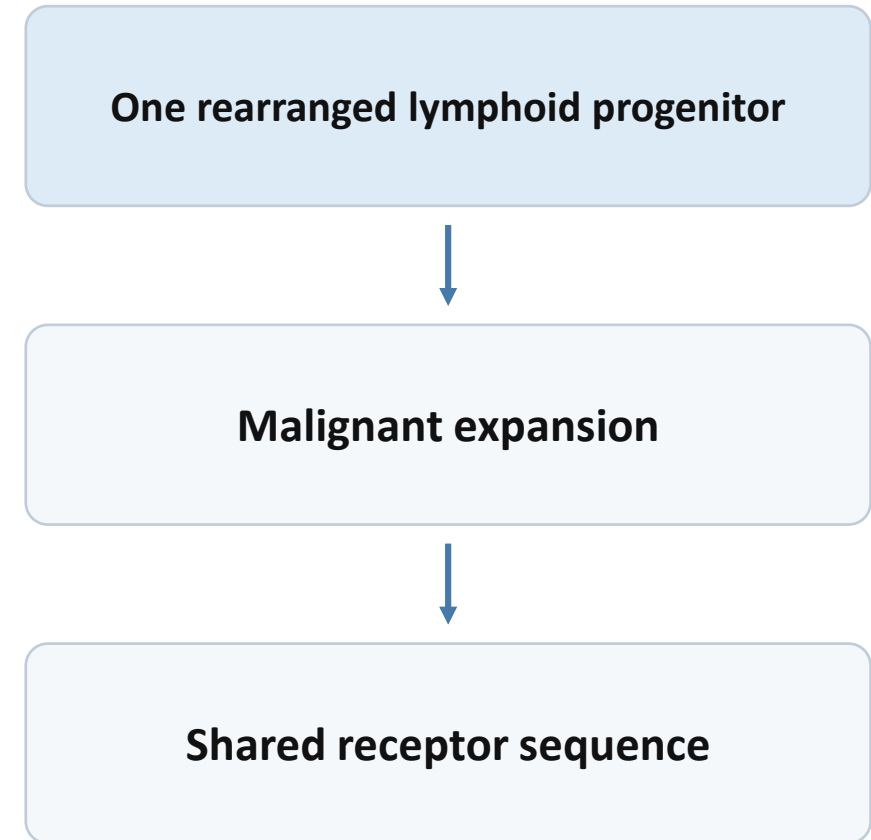
CD11c

CD13 / CD14

CD33 / CD64

A malignant lymphoid clone has a shared receptor

- Antigen receptor gene rearrangement usually precedes malignant transformation.
- Descendant tumor cells therefore inherit the same rearranged receptor sequence.
- This molecular identity provides evidence of clonality.



Polyclonal and monoclonal are different patterns

- Reactive lymphoid responses contain many antigen-receptor rearrangements.
- A malignant clone generally carries one characteristic rearrangement.
- Testing can help distinguish reactive from clonal lymphoid proliferations.

Polyclonal response

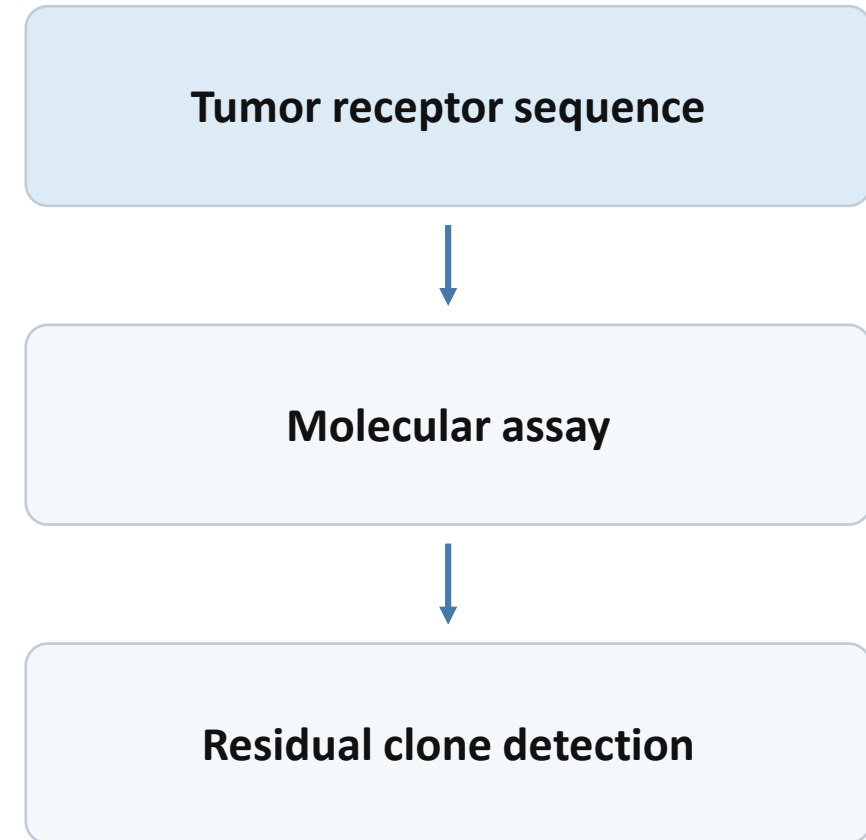
Diverse receptor sequences

Monoclonal tumor

Shared receptor sequence

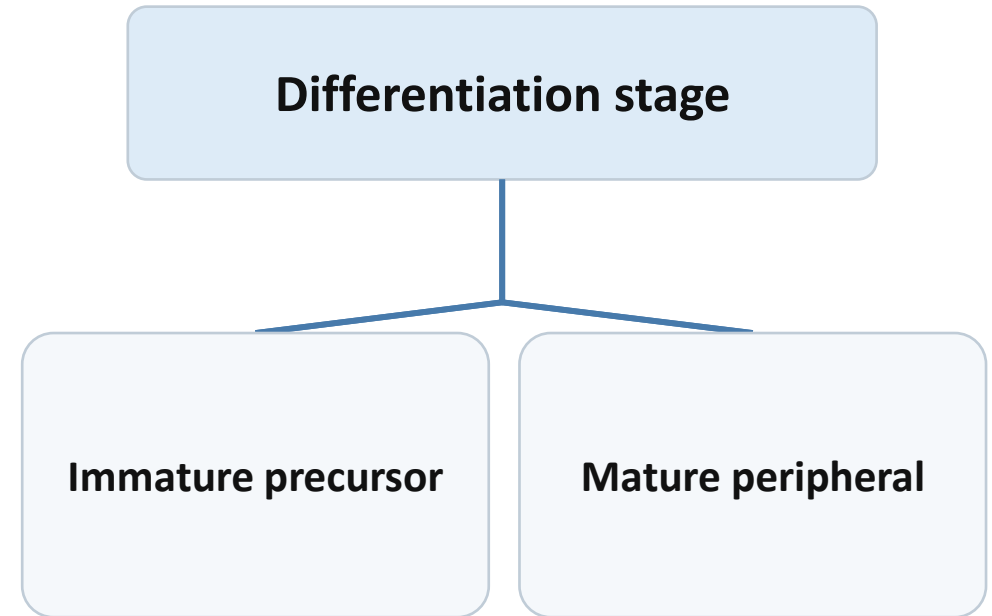
Clonal sequences permit sensitive follow-up

- Each receptor gene rearrangement generates a distinctive DNA sequence.
- That sequence can be used to search for residual malignant lymphocytes.
- Clinical interpretation must link the molecular signal to the original tumor.



Lymphoid tumors resemble stages of development

- Most lymphoid neoplasms resemble recognizable stages of B- or T-cell differentiation.
- This resemblance is central to the classification framework.
- Immature versus mature status is therefore a biologic distinction.



B-cell lineage is most common

- That 85%–90% of lymphoid neoplasms are of B-cell origin.
- Most remaining lymphoid neoplasms arise from T cells.
- NK-cell-origin tumors are rare.

B-cell origin

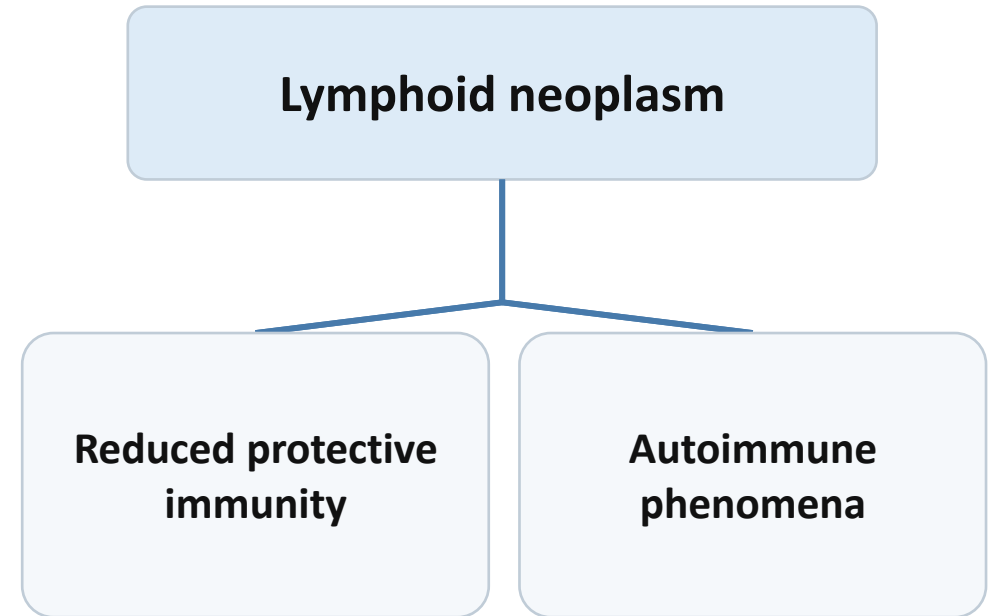
Majority (85%–90%)

T- / NK-cell origin

Smaller fractions

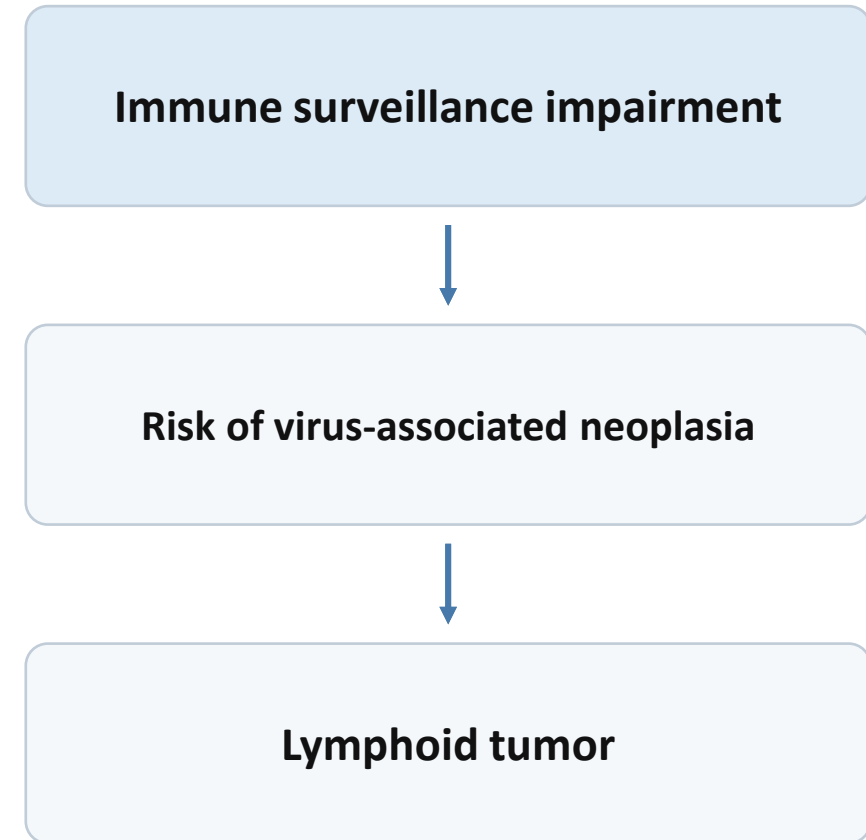
Lymphoid tumors can impair immunity

- Neoplasms may reduce protective immune function, increasing infection susceptibility.
- Some are associated with breakdown of immune tolerance and autoimmunity.
- Both abnormalities may coexist in the same patient.



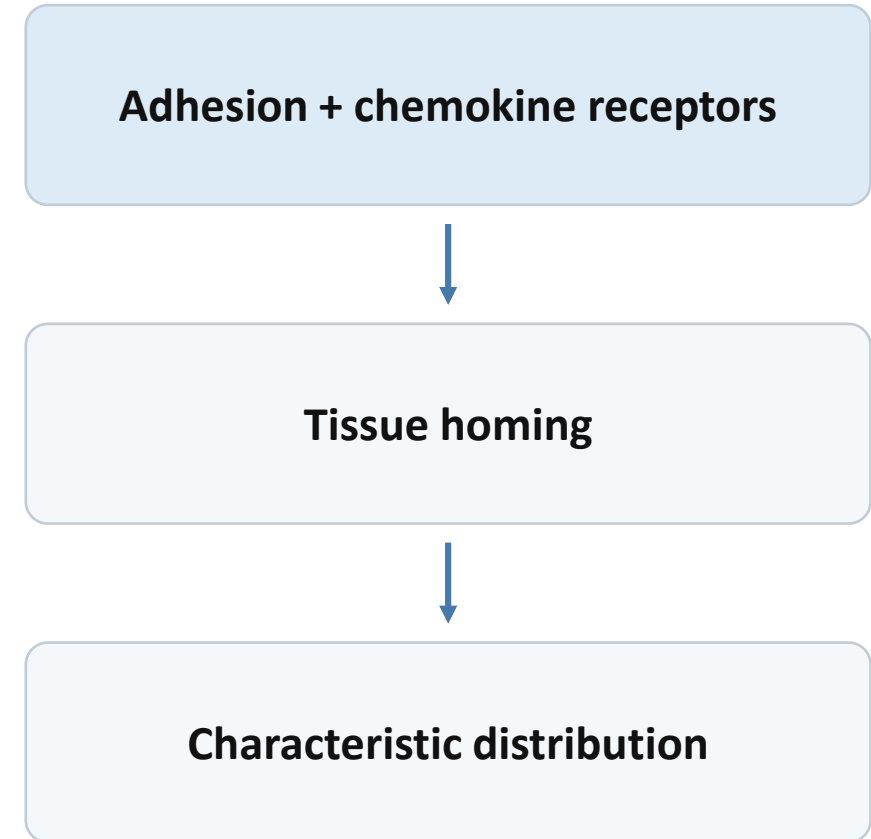
Immunodeficiency also increases lymphoma risk

- Inherited or acquired immunodeficiency increases risk for some lymphoid neoplasms.
- Neoplasms linked to oncogenic viruses such as EBV.
- Immunodeficiency is thus both a possible consequence and a predisposing context.



Neoplastic lymphocytes retain homing programs

- Normal lymphocytes express adhesion molecules and chemokine receptors.
- Neoplastic cells may preserve these trafficking features.
- The retained programs help explain characteristic tissue locations.



Site-specific homing: Two examples

- Follicular lymphoma cells tend to home to lymph-node germinal centers.
- Cutaneous T-cell lymphomas preferentially involve the skin.
- The cell's differentiation program helps explain site predilection.

Follicular lymphoma

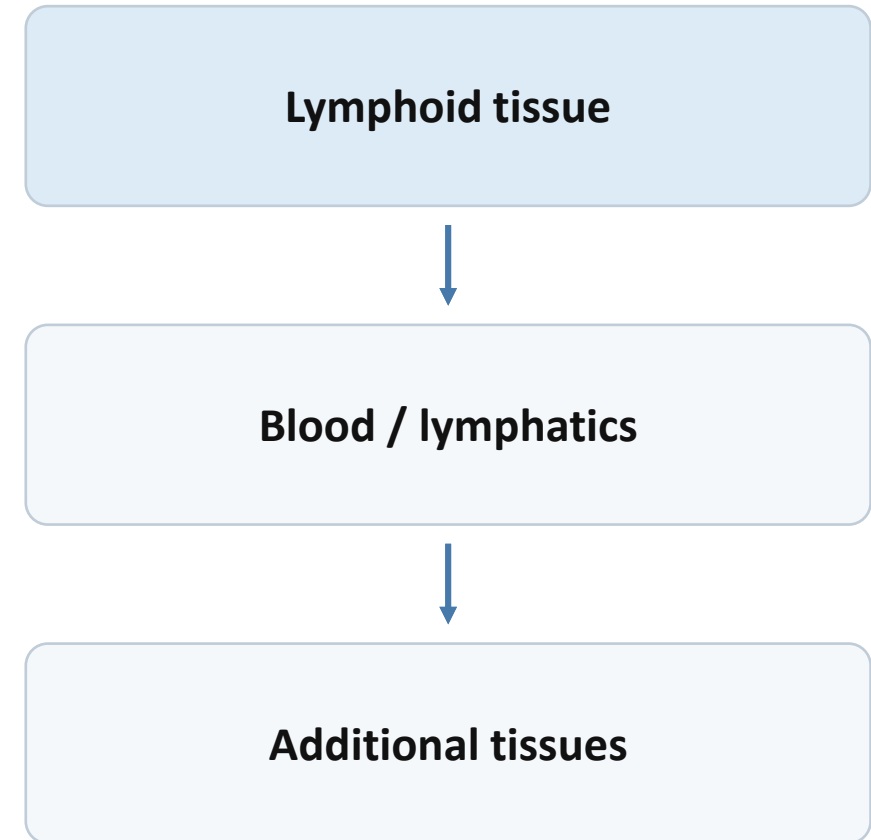
Germinal centers

Cutaneous T-cell lymphoma

Skin

Recirculation explains early dissemination

- Normal lymphocytes circulate between blood and lymphoid tissues.
- Neoplastic B and T cells may recirculate similarly.
- Many lymphoid tumors are already distributed across sites at diagnosis.



Some lymphoid tumors remain localized

- Hodgkin lymphoma may be limited to one nodal group.
- Marginal zone lymphoma may be restricted to a chronically inflamed site.
- These exceptions qualify the general observation of dissemination.

Hodgkin lymphoma

Sometimes one nodal group

Marginal zone lymphoma

Sometimes one inflamed site

Hodgkin and NHL differ in spread pattern

- Hodgkin lymphoma often spreads in a relatively orderly, stepwise pattern.
- Many NHLs disseminate widely and less predictably.
- Patterns overlap; Spread alone does not establish the diagnosis.

Hodgkin lymphoma

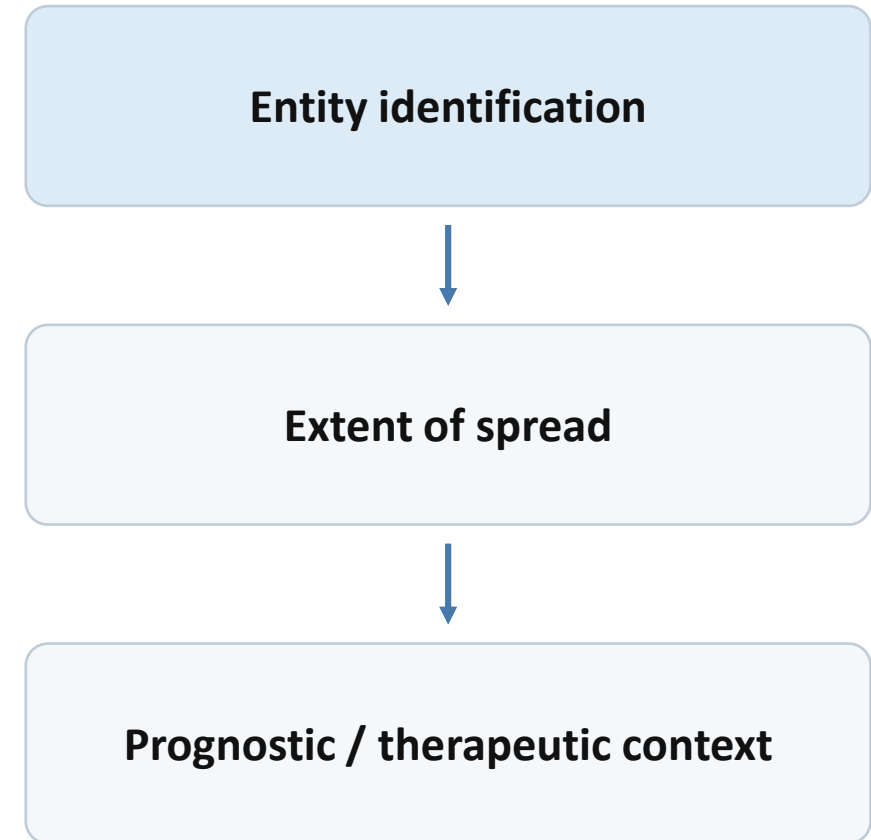
Often orderly / stepwise

Non-Hodgkin lymphoma

Often early / noncontiguous

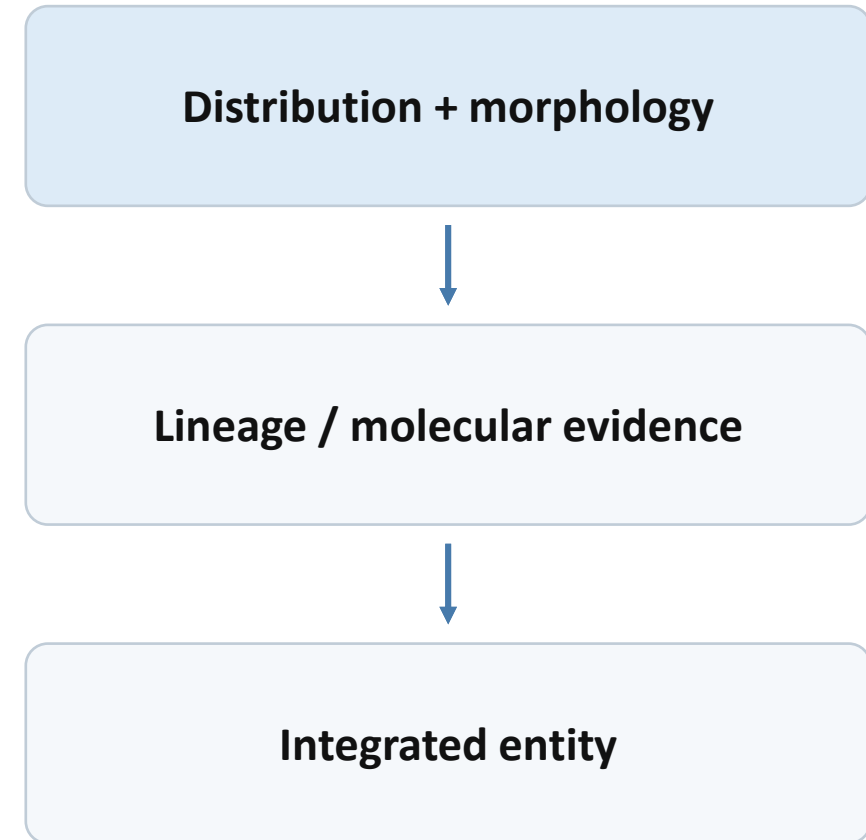
Staging has different decision utility

- Staging conveys prognostic information in lymphoid tumors.
- Its particular therapeutic relevance in Hodgkin lymphoma.
- Tumor identity remains essential when interpreting disease stage.



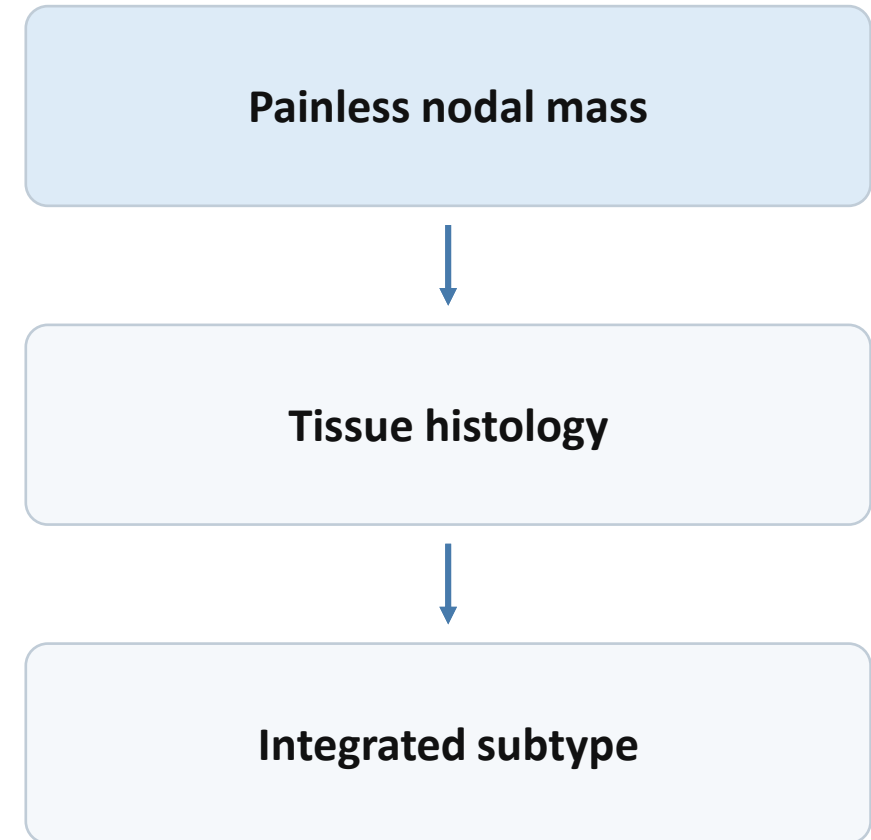
Integrative approach to a lymphoid neoplasm

- Establish the dominant presenting compartment.
- Examine the involved tissue and identify the tumor-cell population.
- Integrate lineage / differentiation markers, molecular findings, and clinical distribution.
- Report a defined entity using one named classification framework.



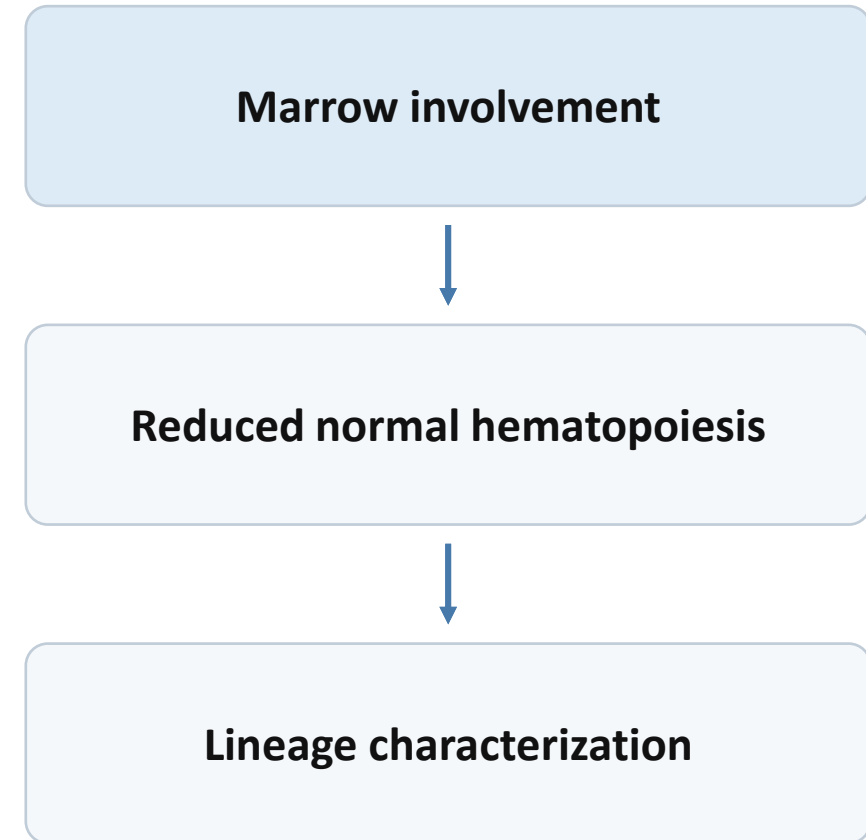
Case: Painless cervical lymphadenopathy

- A persistent, nontender enlarged lymph node suggests, but does not prove, lymphoma.
- Histologic evaluation establishes the presence and pattern of an abnormal infiltrate.
- Immunophenotypic and genetic findings distinguish Hodgkin or specific NHL entities.



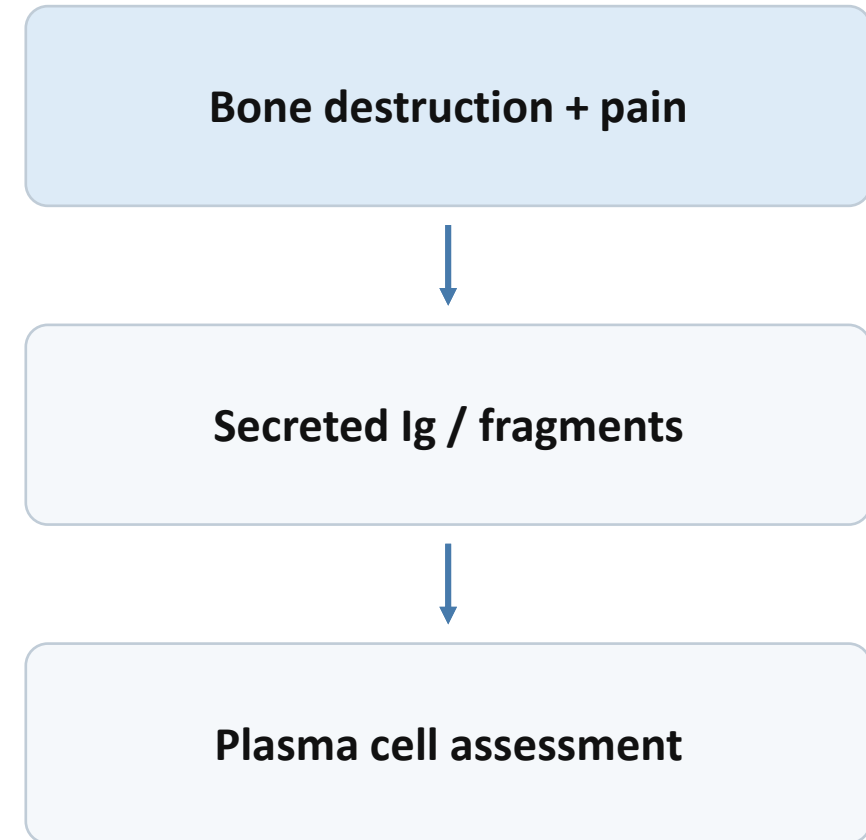
Case: Cytopenias with marrow lymphoid cells

- A marrow-dominant lymphoid neoplasm may present as leukemia.
- Suppression of normal hematopoiesis explains accompanying cytopenic symptoms.
- A circulating tumor-cell count alone does not define the neoplasm's lineage.



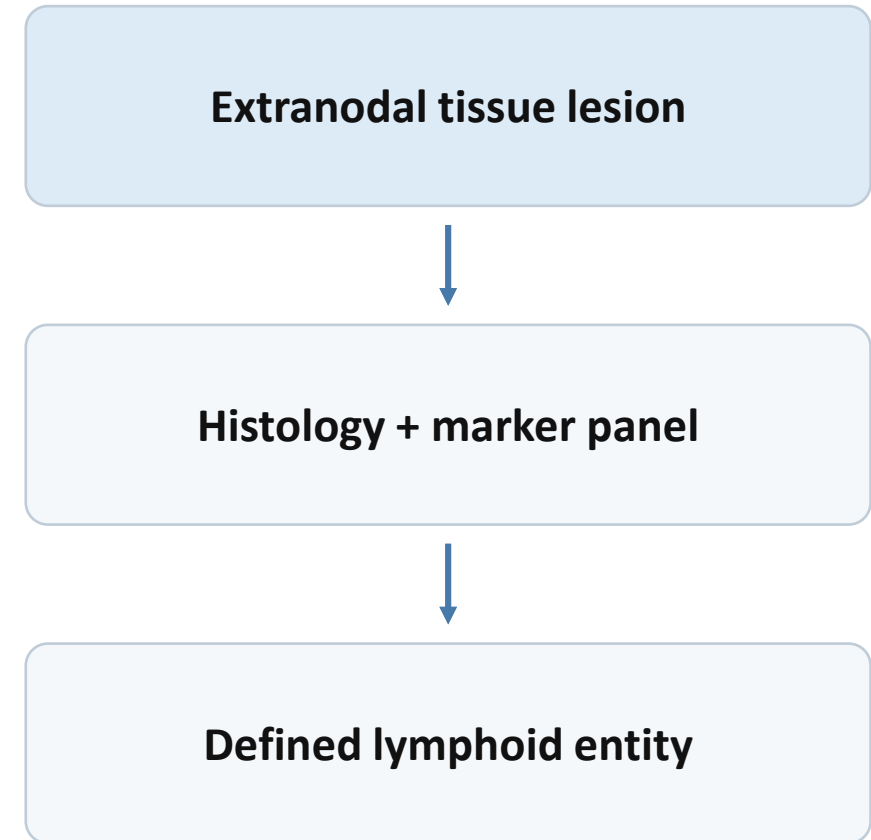
Case: Bone pain and monoclonal protein

- Skeletal destruction and pathologic fractures suggest a plasma cell process.
- Secreted immunoglobulin or fragments may also drive organ dysfunction.
- The combination of anatomic and protein effects focuses classification.



Case: Lesion at an extranodal site

- An extranodal mass must be evaluated according to its involved organ.
- A tissue-defined lymphoma cannot be typed from location alone.
- Morphology, lineage-associated proteins, genetics, and clinical findings are combined.



Diagnostic pitfalls to avoid

- Do not equate “leukemia” with a lineage and “lymphoma” with another lineage.
- Do not diagnose a subtype from one surface antigen or one presenting site.
- Do not treat a summary taxonomy as a complete list of diagnostic criteria.
- Do not combine terminology from WHO and ICC without identifying the chosen edition.

Synthesis: Biology, site, and evidence

- Leukemia and lymphoma describe distribution; The definitive entity captures tumor biology.
- The WHO summary groups lymphoid neoplasms by lineage and differentiation stage.
- Clinical effects arise from distribution, marrow replacement, bone injury, and secreted products.
- Morphology with immunophenotypic, genetic, and clinical context supports classification.

Selected reference

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th edition (2026).
- Section: Lymphoid Neoplasms — Definitions and Classifications.
- WHO-oriented classification overview and immune-cell antigen distributions.
- The complete entity definitions and diagnostic criteria require dedicated classification references.