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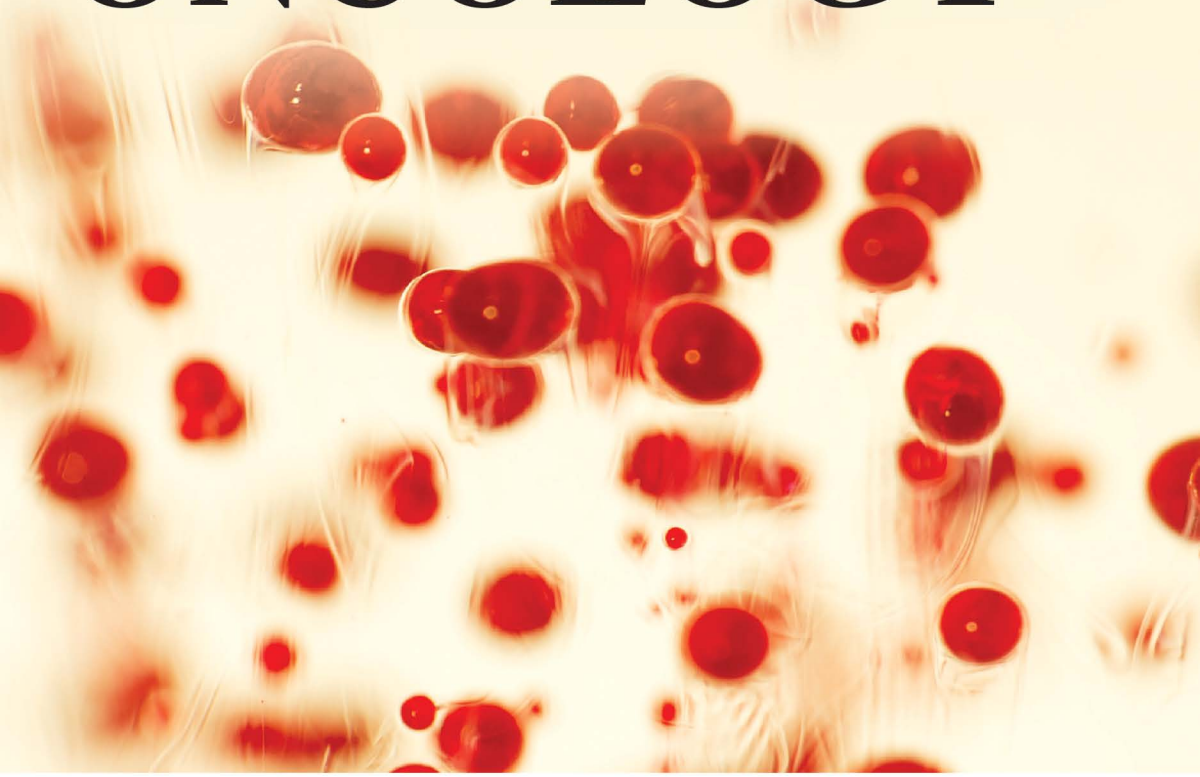

RK Jena
HP Pati

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CHAPTER 3

Diagnosis, Risk Stratification, and MRD in Hematological Malignancies

HP Pati, Neha Singh

INTRODUCTION

The hematological malignancies constitute a large group of cancers involving different hematopoietic cell lineages such as myeloid, lymphoid (T and B cells) of either primitive stem cell or of mature cell origin. They mainly consist of the acute myeloid and acute lymphoid leukemia, chronic leukemias such as chronic myeloid leukemia (CML) and other myeloproliferative neoplasm, myelodysplastic neoplasm (MDS), Hodgkin and non-Hodgkin lymphomas (NHL), and the plasma cell (PC) neoplasms.

Although the diagnosis and classification of these tumors were earlier based primarily on morphologic features, supplemented by cytochemical and immunohistochemical studies, diagnosis of hematological malignancies now requires a complex battery of specialized tools that include immunophenotyping, cytogenetic, and molecular genetic studies.[1] In this chapter, we will mainly discuss the key points about diagnosis, risk stratification, and minimal residual disease (MRD) assessment of acute leukemias (ALs), myeloma, and lymphoma particularly chronic lymphocytic leukemia. The chapters on MDS and CML in this monogram will include their respective diagnostic and risk stratification strategies.

ACUTE LEUKEMIA

Acute leukemia is a highly heterogeneous disease, comprising of acute myeloid leukemia (AML), acute lymphoblastic leukemia (ALL), acute undifferentiated leukemia (AUL), and mixed phenotype acute leukemia (MPAL).

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Diagnosis

All patients suspected with AL should be investigated from basic tests to high-end tests as follows:

- *Complete blood count and peripheral smear (PS) examination*: AL patients present with either leukocytosis or cytopenias or leukoerythroblastic picture. Blasts may or may not be seen in the PS and may be granular or agranular. Patients of AL may show thrombocytopenia, eosinophilia, monocytosis, and morphologic dysplasia such as presence of pseudo-Pelger–Huët neutrophils, etc. Abnormal promyelocytes are a classical feature of acute promyelocytic leukemia (APML) and warrant prompt action in diagnosis and treatment as the tissue thromboplastins released from the abnormal promyelocytes may precipitate disseminated intravascular coagulation (DIC) with deranged coagulation test parameters.[2]
- *Bone marrow (BM) morphology and cytochemistry*: BM biopsy in acute leukemias may be hypercellular or occasionally hypocellular with scattered blast clusters. A dry tap on BM aspiration may be indicative of a packed BM due to marked proliferation of blasts or marrow fibrosis. Hence, any comments on cellularity should be reserved for BM biopsy, where at least 10 marrow spaces have been examined. The blast cutoff of >20% is required for diagnosis of AL. However, lower number of blasts is sufficient if associated with recurrent genetic abnormalities as per the World Health Organization (WHO), 2022 revised guideline, except in AML with *BCR:ABL1*, AML with CEBPA mutation and AML-myelodysplasia related.[3] Features suggestive of myelodysplasia, often associated with AML with myelodysplasia-related changes (same as in MDS) have been described in **Box 1 and Figures 1 to 3**, which point toward the possibility of progression of AL from MDS. Presence of faggots that are bundles of Auer rods **(Fig. 4)** are mostly seen in APML.

Cytochemical stains such as myeloperoxidase (MPO), periodic acid–Schiff (PAS) and nonspecific esterase (NSE) should be routinely applied in all AL cases. MPO is a very important cytochemical stain as MPO positivity >3% in blasts helps in differentiating myeloid and lymphoid lineage blasts **(Fig. 5)**. NSE helps in highlighting the AMLs with monocytic differentiation **(Fig. 6)**. PAS stain shows block positivity in few ALLs and coarse globular positivity in AML-M6 and/or MDS **(Figs. 7A and B)**. Perl stain is used for assessment of the BM iron stores and also to highlight the ringed sideroblasts, which are seen in conditions such as sideroblastic anemia, MDS with ringed sideroblasts, and some AML. Although cytochemistry gives a preliminary clue to the diagnosis of ALs, further lineage typing needs immunophenotyping by flow cytometry (FC).[1]

Flow cytometry has become an important test for the diagnosis, sub-classification, and post-treatment monitoring of ALs. Multiparameter FC (MFC) measures simultaneously and rapidly numerous surface and/or

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BOX 1 **Morphologic manifestations of dysplasia in peripheral blood and bone marrow.**

Dyserythropoiesis:
- Nuclear budding
- Internuclear bridging
- Karyorrhexis
- Multinuclearity
- Megaloblastoid changes
- Ring sideroblasts
- Vacuolization
- Periodic acid–Schiff (PAS) positivity

Dysgranulopoiesis:
- Small or unusually large size
- Nuclear hyposegmentation (pseudo-Pelger-Huët)
- Nuclear hypersegmentation
- Decreased granules; agranularity
- Pseudo-Chediak-Higashi granules
- Dohle bodies
- Auer rods

Dysmegakaryopoiesis:
- Micromegakaryocytes
- Nuclear hypolobation
- Multinucleation

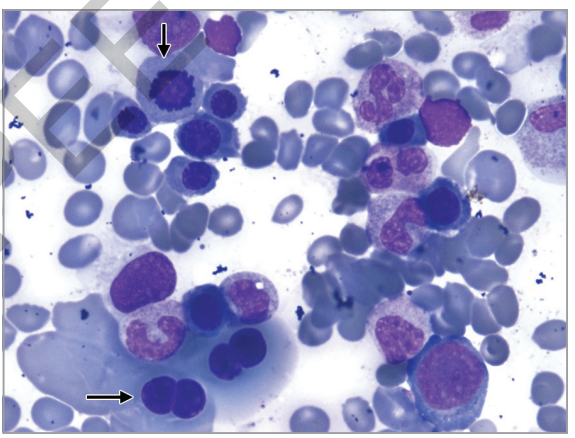

FIG. 1: Dysplastic erythroid cells in the form of binucleation and karyorrhexis.

intracytoplasmic markers on a single cell, allowing accurate phenotypic characterization of different cell populations. Antibodies attached with different fluorochromes allow recognition of different antigens on the same cell with multicolor FC. It can be done in different samples, such as peripheral blood (PB), BM, fine needle aspirates, effusions, etc. FC analysis can

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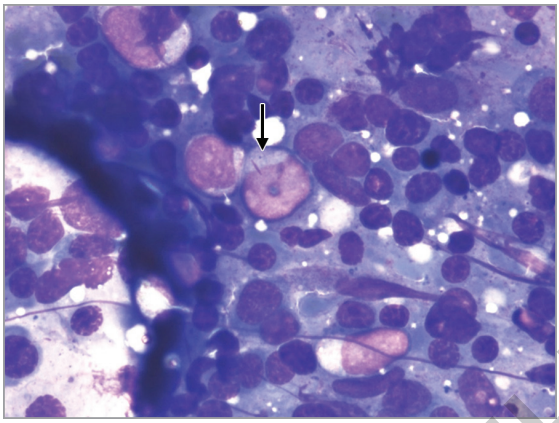

FIG. 2: Auer rods in acute myeloid leukemia (AML) blast.

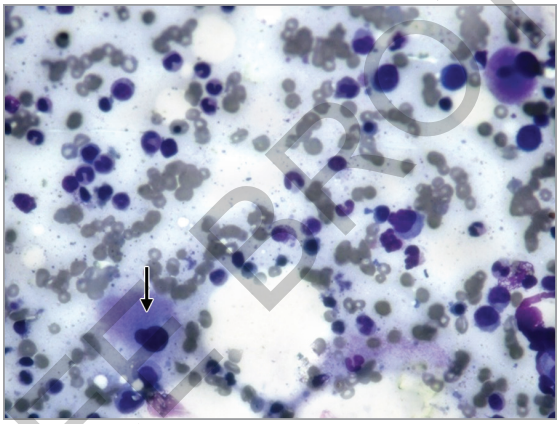

FIG. 3: Hypolobated dysplastic megakaryocytes.

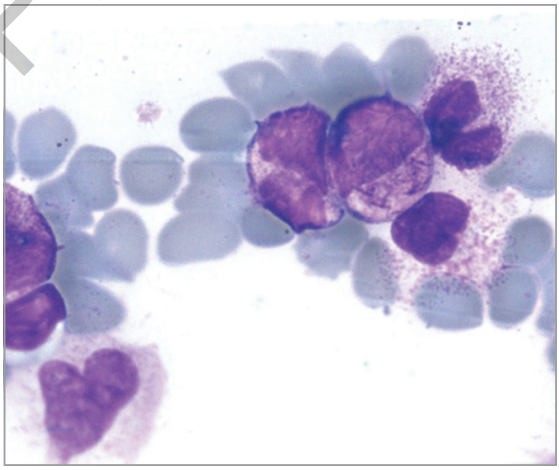

FIG. 4: Faggots (bundles of Auer rods) and hypergranular promyelocytes in acute promyelocytic leukemia (APML).

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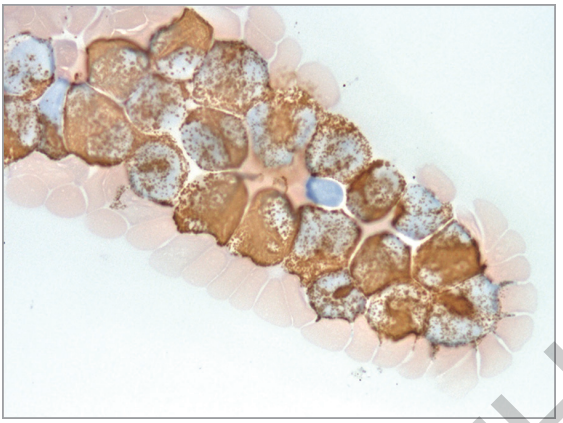

FIG. 5: Myeloperoxidase stain in acute myeloid leukemia with maturation.

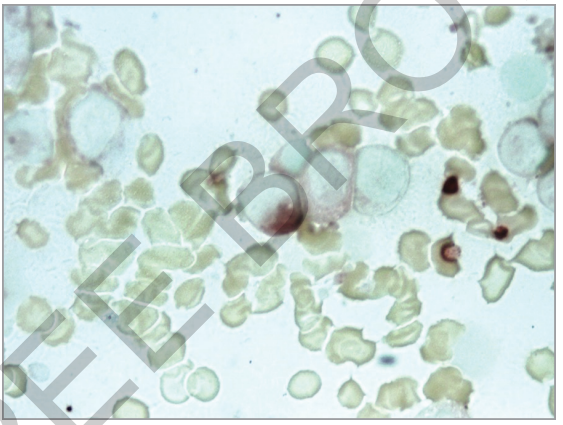

FIG. 6: Nonspecific esterase stain in acute monoblastic leukemia.

precisely differentiate between B- and T-cell malignancies, between mature (peripheral) and precursor cells, and determine the myeloid, lymphoid, or mixed phenotype lineages.

Acute lymphoblastic leukemias are identified as B-cell acute lymphoblastic leukemia (B-ALL), depending upon expression of B-lineage antigens such as CD19, CD10, cytoCD22, and cytoCD79a or T-cell acute lymphoblastic leukemia (T-ALL) with T-cell antigens such as cytoCD3 (lineage-specific), CD7, CD5, CD1a, etc. Negative CD5 is a characteristic of early-T precursor lymphoblastic leukemia.[4] MPO positivity is one of the most important criteria for defining AMLs, however, presence of other myeloid antigens such as CD13, CD33, CD117 and monocytic antigens such as CD14, CD64, CD11c, etc., helps in further subtyping of AMLs.

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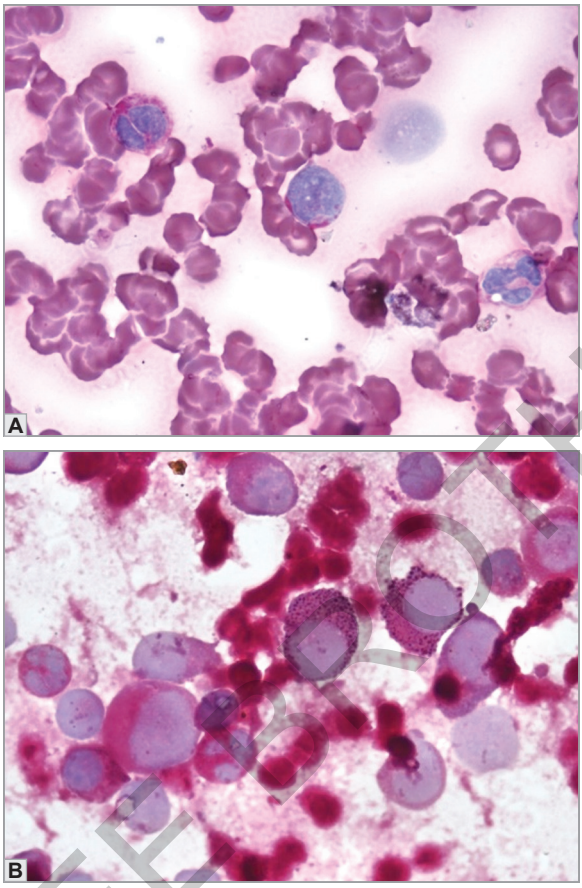

FIGS. 7A AND B: (A) Periodic acid–Schiff (PAS) stain showing block positivity in B-cell acute lymphoblastic leukemia (B-ALL). (B) PAS stain showing coarse globular positivity in erythroblasts in acute myeloid leukemia (AML)-M6.

Acute myeloid leukemia without recurrent cytogenetics and with differentiation markers are classified **(Table 1)** into eight subtypes with cytochemistry and FC. Some AMLs may be MPO negative, where role of monocytic or megakaryoblastic antigens (CD41, CD61) becomes important for lineage assignment. The cases that do not fulfil the criteria of B- or T-ALLs or AML subtypes are of other rare AL variants such as MPAL (when two lineages are found to be present simultaneously) or blastic plasmacytoid dendritic cell neoplasm (BPDCN; blasts are positive for CD4, CD56, HLA-DR, and CD123) or AULs (when it is difficult to put the blasts in any category). **Flowchart 1** concisely describes the flow cytometric antibody combinations useful for clinching the diagnosis of different subtypes of ALs. Expression of some antigens such as CD13, CD33, and CD25 positivity on B-lymphoblasts points toward possibility of Philadelphia-positive B-ALL.

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TABLE 1: Diagnostic criteria for AML types defined by differentiation (WHO, 2022 guidelines).

Type	Diagnostic criteria
AML with minimal differentiation	• Blasts are negative (<3%) for MPO by cytochemistry • Expression of two or more myeloid-associated antigens, such as CD13, CD33, and CD117
AML without maturation	• ≥3% blasts positive for MPO (by immunophenotyping or cytochemistry) • Maturing cells of the granulocytic lineage constitute <10% of the nucleated bone marrow cells • Expression of two or more myeloid-associated antigens, such as MPO, CD13, CD33, and CD117
AML with maturation	• ≥3% blasts positive for MPO (by immunophenotyping or cytochemistry) by cytochemistry • Maturing cells of the granulocytic lineage constitute ≥10% of the nucleated bone marrow cells • Monocyte lineage cells constitute <20% of bone marrow cells • Expression of two or more myeloid-associated antigens, such as MPO, CD13, CD33, and CD117
Acute basophilic leukemia	• Blasts and immature/mature basophils with metachromasia on toluidine blue staining • Blasts are negative for cytochemical MPO and NSE • No expression of strong CD117 equivalent (to exclude mast cell leukemia)
Acute myelomonocytic leukemia	• ≥20% monocytes and their precursors • ≥20% maturing granulocytic cells • ≥3% of blasts positive for MPO (by immunophenotyping or cytochemistry)
Acute monocytic leukemia	• ≥80% monocytes and/or their precursors (monoblasts and/or promonocytes) • <20% maturing granulocytic cells • Blasts and promonocytes expressing at least two monocytic markers including CD11c, CD14, CD36, and CD64, or NSE positivity on cytochemistry
Acute erythroid leukemia	• ≥30% immature erythroid cells (proerythroblasts) • Bone marrow with erythroid predominance, usually ≥80% of cellularity
Acute mega-karyoblastic leukemia	Blasts express at least one or more of the platelet glycoproteins: CD41, CD61, or CD42b

Note: Shared diagnostic criteria include:
- ≥20% blasts in bone marrow and/or blood (except for acute erythroid leukemia).
- Criteria for AML types with defined genetic alterations are not met.
- Criteria for mixed-phenotype acute leukemia are not met (relevant for AML with minimal differentiation).
- Not fulfilling diagnostic criteria for myeloid neoplasm post cytotoxic therapy.
- No prior history of myeloproliferative neoplasm.

(AML: acute myeloid leukemia; MPO: myeloperoxidase; NSE: nonspecific esterase; WHO: World Health Organization)

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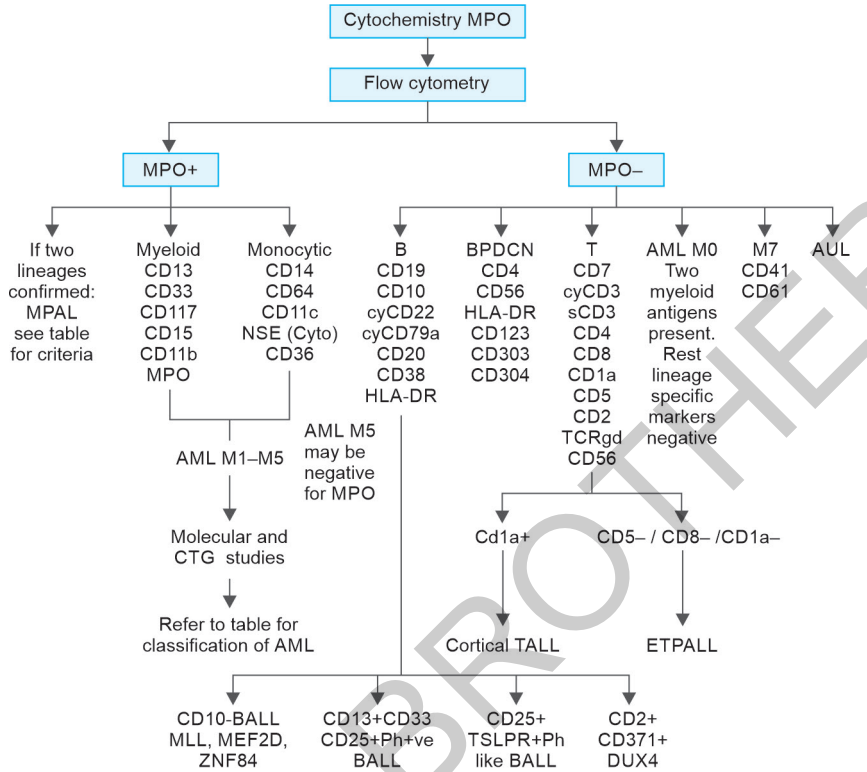

Note: Refer to Table 1 for diagnostic criteria of mixed phenotype AL (MPAL) and Table 2 and Boxes 2 and 3 for AL classification terminology.

FLOWCHART 1: Approach to a case of acute leukemia (AL) diagnosis by flow cytometry.

(AML: acute myeloid leukemia; BPDCN: blastic plasmacytoid dendritic cell neoplasm; CTG: cytogenetics; ETP-ALL: early T precursor acute lymphoblastic leukemia; MPO: myeloperoxidase)

Mixed phenotype acute leukemia is a heterogeneous category that comprises ALs with discrete admixed populations of myeloid and lymphoid blasts ("bilineal") or coexpression of lymphoid and myeloid markers in a single blast population ("biphenotypic"). FC is the preferred method for establishing the diagnosis, especially when a diagnosis of MPAL requires demonstrating coexpression of lymphoid and myeloid differentiation antigens on the same cell. The hallmark of MPAL is that the combination of antigens expressed on the blast population or populations does not allow classification into a single lineage. **Table 2** gives the flow cytometric along with cytochemistry-based characteristics of MPAL. A case could be a mixture of myeloid B, or myeloid T or mixed B- and T-ALL. MPAL group however excludes cases that can be classified in another category, such as cases with the recurrent genetic abnormalities t(8;21), inv(1 6), or *PML-RARA* fusion, cases of leukemia with *FGFR1* rearrangements, cases of CML in blast crisis, AML with myelodysplasia-related changes, and therapy-related AML.

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TABLE 2: Diagnostic criteria of mixed phenotype acute leukemia (MPAL).

	Criterion
B lineage	
CD19 strong	One or more also strongly expressed: CD10, CD22, or CD79a
or,	
CD19 weak	Two or more also strongly expressed: CD10, CD22, or CD79a
T lineage	
CD3 (cytoplasmic or surface)	Intensity in part exceeds 50% of mature T cells level by flow cytometry or, Immunocytochemistry positive with non-zeta chain reagent
Myeloid lineage	
Myeloperoxidase	Intensity in part exceeds 50% of mature neutrophil level
or,	
Monocytic differentiation	Two or more expressed: Nonspecific esterase, CD11c, CD14, CD64 or lysozyme

Acute undifferentiated leukemia expresses no markers considered to be specific for either lymphoid or myeloid lineage. Before categorizing a leukemia as undifferentiated, it is necessary to perform immunophenotyping with a comprehensive panel of monoclonal antibodies in order to exclude leukemias of unusual lineages, such as those derived from plasmacytoid dendritic cell precursors, NK cell precursors, basophils, or even nonhematopoietic tumors. These leukemias typically express no more than one membrane marker for any given lineage. By definition, they lack the T-cell and myeloid markers cCD3 and MPO and do not express B-cell markers such as cCD22, cCD79a, or strong CD19.

Acute leukemias of myeloid, B-/T-cell origin (i.e., AML, B-ALL, T-ALL) with the presence of recurrent chromosomal rearrangements as per the WHO, 2022, are given in **Boxes 2 and 3**. Role of cytogenetic and molecular studies are very important to diagnose these cases.

Cytogenetic studies should be performed on all cases of suspected AL as karyotype analysis currently provides the best overall assessment of chromosomal abnormalities and the deviation from the normal copy number of a given chromosome (aneuploidy).

Fluorescence in situ hybridization (FISH) studies are helpful to detect cryptic abnormalities that may be missed by karyotype analysis such as ETV6:RUNX1 fusion in B-ALL. Genetic mutations (e.g., *NPM1, FLT3, CEBPA, TET-2, c-KIT, DNMT3A, IDH, RUNX1, AXSL1, WT1, Ras* gene mutations) are detected by *polymerase-chain reaction (PCR)-based assays or next generation sequencing (NGS)*, and they help in classification of AMLs and also serve as important prognostic markers and potential therapeutic targets in AML.

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BOX 2 **Revised World Health Organization, 2022 classification of precursor B/T neoplasms.**

Precursor B cell neoplasms:

- B-cell lymphoblastic leukemias/lymphomas NOS
- B-cell lymphoblastic leukemias/lymphomas with high hyperploidy
- B-cell lymphoblastic leukemias/lymphomas with hypoploidy
- B-cell lymphoblastic leukemias/lymphomas with iAMP21
- B-cell lymphoblastic leukemias/lymphomas with BCR-ABL1 fusion
- B-cell lymphoblastic leukemias/lymphomas with BCR-ABL1-like features
- B-cell lymphoblastic leukemias/lymphomas with KMT2A rearrangement
- B-cell lymphoblastic leukemias/lymphomas with ETV6::RUNX1 fusion
- B-cell lymphoblastic leukemias/lymphomas with ETV6::RUNX1 like features
- B-cell lymphoblastic leukemias/lymphomas with TCF3::PBX1 fusion
- B-cell lymphoblastic leukemias/lymphomas with IGH:IL3 fusion
- B-cell lymphoblastic leukemias/lymphomas with TCF3::HLX fusion
- B-cell lymphoblastic leukemias/lymphomas with other defined genetic abnormalities

Precursor T cell neoplasms:

- T-lymphoblastic leukemia/lymphoma NOS
- Early-T precursor lymphoblastic leukemia/lymphoma

(NOS: not otherwise specified)

BOX 3 **Revised World Health Organization, 2022 classification of acute myeloid leukemia.**

Acute myeloid leukemia with defining genetic abnormalities:

- Acute promyelocytic leukemia with PML::RARA fusion
- Acute myeloid leukemia with RUNX1::RUNX1T1 fusion
- Acute myeloid leukemia with CBFB::MYH11 fusion
- Acute myeloid leukemia with DEK::NUP214 fusion
- Acute myeloid leukemia with RBM15::MRTFA fusion
- Acute myeloid leukemia with BCR::ABL1 fusion
- Acute myeloid leukemia with KMT2A rearrangement
- Acute myeloid leukemia with MECOM rearrangement
- Acute myeloid leukemia with NUP98 rearrangement
- Acute myeloid leukemia with NPM1 mutation
- Acute myeloid leukemia with CEBPA mutation
- Acute myeloid leukemia, myelodysplasia-related
- Acute myeloid leukemia, defined by differentiation

Acute myeloid leukemia with minimal differentiation:

- Acute myeloid leukemia without maturation
- Acute myeloid leukemia with maturation
- Acute basophilic leukemia
- Acute myelomonocytic leukemia
- Acute monocytic leukemia
- Acute erythroid leukemia
- Acute megakaryoblastic leukemia

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Philadelphia chromosome-positive ALL (Ph+ ALL) accounts for approximately one-fourth of adult ALL cases. It typically presents with an aggressive clinical course, responds poorly to standard chemotherapy, and carries a high risk for relapse. The landscape of Ph+ ALL therapy has changed favorably since the development of tyrosine kinase inhibitors (TKIs). This entity is easily diagnosed by *reverse transcriptase-based PCR (RT-PCR)* assay.[5]

Philadelphia chromosome-like ALL, also referred to as *BCR-ABL1*–like ALL, is another high-risk subset with a gene expression profile that shares significant overlap with that of Ph+ ALL and is suggestive of activated kinase signaling. Although Ph+ ALL is defined by *BCR-ABL1* fusion, Ph-like ALL cases contain a variety of genomic alterations that activate kinase and cytokine receptor signaling that can be demonstrated by *targeted sequencing*.[6]

Recurring cytogenetic abnormalities are also found in T-ALL, but are not included as diagnostic subcategories in the WHO classification. These translocations most often involve T-cell receptor genes at 7q32 (*TRB*) and 14q11 (*TRA*, *TRD*) and *TLX1*, *TLX3*, and *TAL1*. Prognostic significance has not been determined for many of these genetic changes, although *TLX1* translocations appear to confer a good prognosis. Mutations are also common in T-ALL; NOTCH*1 mutations* found in 50–60% of adult and pediatric cases. Mutations of *FBXW7*, a gene involved in NOTCH signaling, occurs in 10–20% of the cases. These mutations appear to have some prognostic significance in children, at least predicting a favorable treatment response with protocols that include cranial irradiation, but appear to be of less significance in adult patients.[7,8]

Risk Stratification in Acute Leukemias

Risk stratification in ALs is critical for making treatment decisions, such as choice of consolidation chemotherapy, timing of hematopoietic stem cell transplantation (HSCT) or eligibility for clinical trials as well as predicting risk of relapse/refractory disease.

In ALLs, persistence of flow cytometric MRD remains one of the most important criteria for categorizing a case into high risk,[9-12] apart from other prognostic factors such as adverse cytogenetics, steroid refractoriness, age, leukocyte counts, central nervous system (CNS) status, etc., as listed in **Box 4** and **Table 3**.

BOX 4 **Children's Oncology Group (COG) trial-based high-risk features in B-cell acute lymphoblastic leukemia (B-ALL).**

- Age <1 or >10 years
- Initial white blood cell counts >50,000/μL
- Central nervous system involvement
- Testicular involvement
- Unfavorable cytogenetics [hypodiploidy, t(9;22), Ph-like, 11q23, iAMP21]
- Suboptimal induction response (induction failure or positive minimum residual disease)

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TABLE 3: Risk group definitions in T-lymphoblastic leukemia.

Risk	COG T-ALL risk group definitions
Standard risk	M1 day 29 marrow; day 29 MRD* <0.01%; CNS 1; no testicular disease; no steroid pretreatment
Intermediate risk	M1 or M2 day 29 marrow; day 29 MRD ≥0.01%; end-of-consolidation MRD <0.1%; any CNS and testicular disease status; any steroid pretreatment status
Very high risk	M3 day 29 marrow or end-of-consolidation MRD ≥0.1%

*M1 marrow, <5% blasts; M2 marrow, 5–25% blasts; M3 marrow, >25% blasts by morphology; CNS1, <5 WBC/μL and no blasts in the CSF.

(CNS: central nervous system; COG: Children's Oncology Group; CSF: cerebrospinal fluid; MRD: minimal residual disease; T-ALL: T-cell acute lymphoblastic leukemia)

TABLE 4: 2022 European LeukemiaNet (ELN) risk classification of acute myeloid leukemia (AML) by genetics at initial diagnosis.

Risk category	Genetic abnormality
Favorable	• t(8;21) (q22; q22.1)/RUNX1:RUNX1T1 • Inv(16) (p13.1q22) or t(16;16)(p13.1;q22)/CBFB::MYH11 • Mutated NPM1 without FLT3-ITD • bZIP in-frame mutated CEBPA
Intermediate	• Mutated NPM1 with FLT3-ITD • Wild-type NPM1 with FLT3-ITD • t(9;11) (p21.3; q23.3)/MLLT3:KMT2A • Cytogenetic and/or molecular abnormalities not classified as favorable or adverse
Adverse	• t(6;9) (p23; q34.1)/DEK: NUP214 • t(v;11q23.3)/KMT2A-rearranged • t(9;22) (q34.1; q11.2)/BCR: ABL1 • t(8;16) (p11; p13)/KAT6A: CREBBP • Inv(3) (q21.3q26.2) or t(3;3) (q21.3;q26.2)/GATA2, MECOM (EVI1) • t(3q26.2; v)/MECOM (EVI1)-rearranged • −5 or del(5q); −7; −17/abn(17p) • Complex karyotype, monosomal karyotype • Mutated ASXL1, BCOR, EZH2, RUNX1, SF3B1, SRSF2, STAG2, U2AF1, or ZRSR2 • Mutated TP53

In AML, risk stratification relies heavily on cytogenetic and molecular genetics as shown in **Table 4**. Presence of recurrent cytogenetic abnormalities such as t(8;21), t(15;17), Inv16, and NPM1+/FLT3-ITD-ve are considered as favorable risk, whereas presence of AML-MR associated mutations such as EZH2, STAG2, SRSF2, SF3B1, BCOR, and ASXL1 are indicative of poor prognosis and hence counseled for allogenic stem cell transplantation (allo-HCT).[13]

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Salient Features

- Diagnosis, risk stratification, selection of therapy, interim/post-therapy evaluation by MRD/PET-CT-based investigations, and subsequent modification of the therapeutic procedure have transformed the overall outcome of hematological malignancies.
- Molecular investigations are a requirement at all stages: Diagnosis, risk stratification, interim/MRD evaluation, etc.
- Matched unrelated donor/Haplo Allo BMT has enhanced the possibility of Allo BMT in most of the patients as a curative option.
- This first joint venture ICH-ICP e-Monogram, highlighting the above points, is primarily meant for postgraduates.

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