Dr. dr. Agus Susanto Kosasih, SpPK, MARS

Birth Place and Date: Jakarta, Feb 1st 1961
Current Position: Medical Staff of Clinical Pathology Department
Dharmais National Cancer Hospital, Jakarta, Indonesia

Education
2015 Graduated as Doctoral in Medicine, Medical Faculty University of Indonesia, Jakarta
2001 Graduated as MARS (Magister of Hospital Administration), University of Indonesia, Jakarta
1991 Graduated as Clinical Pathologist, Medical Faculty University of Indonesia, Jakarta
1987 Crash Program doctor type C hospital laboratory, Medical Faculty University of Indonesia, Jakarta
1985 Graduated as Medical Doctor, Medical Faculty University of Indonesia, Jakarta

Professional Experience
1992 - present Consultant of Clinical Pathology Laboratory in Metropolitan Medical Centre Hospital, Jakarta, Indonesia
2002 – present Responsible for Clinical Pathology Laboratory of Hermina Hospital, Jatinegara, Jakarta, Indonesia
2011 – present Consultant of Clinical Pathology Laboratory Siloam Semanggi (MRCCC) Jakarta, Indonesia
Molecular diagnosis in Acute Leukemia
Benchmarking Dharmais Cancer Hospital

DR. dr. Agus S. Kosasih, SpPK, MARS
INTRODUCTION

- Leukemia accounted for some 352,000 new cases (2.5% of all new cancer cases) and for 265,000 deaths (3.2% of all deaths).

- Mortality rates range between 1.3 per 100,000 and 6.3 per 100,000 in males, and 1.1 and 3.8 in females.

- Indonesia, Dharmais National Cancer Centre 2007
  - Leukemia is no 8 from all cancer
  - DNCC data in 2005-2007:
    - 300 new cases of acute Leukemia,
    - 151 (±50%) cases are adult
  - Cancer Registry 2008-2010:
    - 108 cases of acute leukemia (56 with ALL & 52 AML)
Estimated New Cases (%) of Leukemia, Lymphoma, and Myeloma, 2016

- Myeloma: 18% (30,330 cases)
- Lymphoma: 47% (81,080 cases)
- Leukemia: 35% (60,140 cases)

Total cases: 171,550

Figure 1. Source: Cancer Facts & Figures, 2016. American Cancer Society; 2016.


Leukemia

- **AML**
  Occurs in both adults and children

- **ALL**
  Most common type of leukemia in children

- **CML**
  Mainly affects adults

- **CLL**
  Most often in people over age 55
ALL

Lymphoid progenitor

CLL

B-lymphocytes

Lymphoma

germinal center

T-lymphocytes

MM

Plasma cells

stem cell

AML

Myeloid progenitor

Mieloproliferative Disorders

Neutrophils

Eosinophils

Basophils

Monocytes

Platelets

Red cells
Acute Leukemia

- Stem cell disorder characterized by malignant neoplastic proliferation and accumulation of immature and nonfunctional hematopoietic cells in the BM

- Characterized by two major features
  - Ability to proliferate continuously
    - Due to mutations affecting growth factors
    - Transcription errors
  - Arrested development of normal cells
  - Lacks apoptosis
Diagnosis

- Anamnesis
- Physical examination
- Laboratory Findings:
  - Morphology
  - Cytochemistry
  - Immunophenotyping
  - Cytogenetic
  - Molecular Biology
Diagnosis

- In establishing a diagnosis of Acute Leukemia, it is critical to obtain an accurate **blast count** performed on at least 500 nucleated bone marrow cells and at least 200 peripheral blood leukocytes.

- The starting point for diagnosis of leukemia is morphologic examination to document the presence of at least 20% **blasts** in bone marrow or in blood.

- In rare cases, the blast count is **below 20%**, but **cytogenetic abnormalities** are present that by convention warrant a diagnosis of AML.
Leukemia: Classification

- FAB Classification:
  - Blasts must comprise at least 30% of nucleated cells in bone marrow or blood to establish a diagnosis of AML.
  - Morphology, Cytochemistry, Immunophenotyping

- WHO Classification:
  - Newer version
  - Blasts must comprise at least 20% of nucleated cells in bone marrow or blood to establish a diagnosis of AML.
  - Morphology, Immunophenotyping, Cytogenetic and molecular biology.
Lab Features

- Normochromic and normocytic anemia
- Thrombocytopenia
- Platelet morphology and function can be abnormal
- Leukocyte count can be increased, decreased or normal
- Immature leukocyte precursors seen
- Bone marrow hypercellular
- Maturation abnormalities in all cell lines
- Uric acid increased
WHO Classification of Tumors of Haematopoietic and Lymphoid Tissues

Basic principle: Classification for all neoplasms based on:

- Morphology and biologic features
- Cytogenetic
- Immunophenotype
- Molecular Biology
DIAGNOSIS OF HAEMATOLOGICAL MALIGNANCIES

Clinical symptoms and signs

Laboratory findings

Morphology + cytochemistry

Cytogenetics

Immunophenotyping

Molecular biology/FISH
### AML: WHO Classification

<table>
<thead>
<tr>
<th>Types</th>
<th>Genetic abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>AML with recurrent genetic abnormalities</td>
<td>AML with t(8;21)(q22;q22); RUNX1-RUNX1T1</td>
</tr>
<tr>
<td></td>
<td>AML with inv(16)(p13.1;q22) or t(16;16)(p13.1;q22); CBFB-MYH11</td>
</tr>
<tr>
<td></td>
<td>APL with PML-RARA</td>
</tr>
<tr>
<td></td>
<td>AML with t(9;11)(p21.3;q23.3); MLLT3-KMT2A</td>
</tr>
<tr>
<td></td>
<td>ML with t(6;9)(p23;q34.1); DEK-NUP214</td>
</tr>
<tr>
<td></td>
<td>AML with inv(3)(q21.3;q26.2) or t(3;3)(q21.3;q26.2); GATA2, MECOM</td>
</tr>
<tr>
<td></td>
<td>AML (megakaryoblastic) with t(1;22)(p13.3;q13.3); RBM15-MKL1</td>
</tr>
<tr>
<td></td>
<td>AML with BCR-ABL1 (provisional entity)</td>
</tr>
<tr>
<td></td>
<td>AML with mutated NPM1</td>
</tr>
<tr>
<td></td>
<td>AML with biallelic mutations of CEBPA</td>
</tr>
<tr>
<td></td>
<td>AML with mutated RUNX1 (provisional entity)</td>
</tr>
<tr>
<td>AML with myelodysplasia-related changes</td>
<td>AML with minimal differentiation</td>
</tr>
<tr>
<td>Therapy-related myeloid neoplasms</td>
<td>AML without maturation</td>
</tr>
<tr>
<td></td>
<td>AML with maturation</td>
</tr>
<tr>
<td></td>
<td>Acute myelomonocytic leukemia</td>
</tr>
<tr>
<td></td>
<td>Acute monoblastic/monocytic leukemia</td>
</tr>
<tr>
<td></td>
<td>Acute erythroid leukemia</td>
</tr>
<tr>
<td></td>
<td>Pure erythroid leukemia</td>
</tr>
<tr>
<td></td>
<td>Acute megakaryoblastic leukemia</td>
</tr>
<tr>
<td></td>
<td>Acute basophilic leukemia</td>
</tr>
<tr>
<td></td>
<td>Acute panmyelosis with myelofibrosis</td>
</tr>
<tr>
<td>Myeloid sarcoma</td>
<td>Transient abnormal myelopoiesis</td>
</tr>
<tr>
<td>Myeloid proliferations related to Down syndrome</td>
<td>ML associated with Down syndrome</td>
</tr>
</tbody>
</table>

**Abbreviations:** AML, acute myeloid leukemia; APL, acute promyelocytic leukemia; ML, myeloid leukemia; WHO, World Health Organization.
## AML: FAB Classification

<table>
<thead>
<tr>
<th>Subclass</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>M0</td>
<td>Acute non-differentiated leukemia – immature blast with minimal differentiated</td>
</tr>
<tr>
<td>M1</td>
<td>Acute myeloblastic leukemia without maturation</td>
</tr>
<tr>
<td>M2</td>
<td>Acute myeloblastic leukemia with granulocytic maturation</td>
</tr>
<tr>
<td>M3</td>
<td>Promyelocytic or acute promyelocytic leukemia</td>
</tr>
<tr>
<td>M4</td>
<td>Acute myelomonocytic leukemia</td>
</tr>
<tr>
<td>M4Eo</td>
<td>Variant → increase in abnormal marrow eosinophils</td>
</tr>
</tbody>
</table>
| M5       | M5a → acute monocytic leukemia without maturation  
|          | M5b → acute monocytic leukemia with partial maturation |
| M6       | Acute erythromyelosis |
| M7       | Acute megakaryoblastic |
# ALL : FAB Classification

<table>
<thead>
<tr>
<th>Cytological</th>
<th>L1</th>
<th>L2</th>
<th>L3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cell size</td>
<td>Small cells &gt;&gt;, homogenous</td>
<td>Large, heterogenous</td>
<td>Large, homogenous</td>
</tr>
<tr>
<td>Cytoplasm</td>
<td>Scanty</td>
<td>Variable</td>
<td>Moderately abundant</td>
</tr>
<tr>
<td>Nucleoli</td>
<td>Small</td>
<td>One or more, often large</td>
<td>One or more, prominent</td>
</tr>
<tr>
<td>Nuclear size</td>
<td>Homogenous</td>
<td>Variable, heterogenous</td>
<td>Stippled, homogenous</td>
</tr>
<tr>
<td>Nuclear shape</td>
<td>Regular</td>
<td>Irregular clefts</td>
<td>Regular</td>
</tr>
<tr>
<td>Cytoplasm basophilian</td>
<td>Variable</td>
<td>Variable</td>
<td>Intensely basophilic</td>
</tr>
<tr>
<td>Cytoplasm vacuolation</td>
<td>Variable</td>
<td>Variable</td>
<td>Prominent</td>
</tr>
</tbody>
</table>
## Table 17.1 Classification of acute lymphoblastic leukaemia (ALL) according to the World Health Organization (WHO) classification (modified).

**Precursor lymphoid neoplasms**

- **B lymphoblastic leukaemia/lymphoma**
- **B lymphoblastic leukaemia/lymphoma, NOS**
- **B lymphoblastic leukaemia/lymphoma with recurrent genetic abnormalities**
  - B lymphoblastic leukaemia/lymphoma with t(9; 22)(q34; q11.2); *BCR-ABL1*
  - B lymphoblastic leukaemia/lymphoma with t(v; 11q23); *MLL* rearranged
- **B lymphoblastic leukaemia/lymphoma with t(12; 21)(p13; q22); *TEL-AML1 (ETV6-RUNX1)*
- **B lymphoblastic leukaemia/lymphoma with hyperdiploidy**
- **B lymphoblastic leukaemia/lymphoma with hypodiploidy (hypodiploid ALL)**

- **T lymphoblastic leukaemia/lymphoma**

NOS, not otherwise specified.
Acute Leukaemia

AML

ALL
Morphology
The weakness of cytochemistry method:

→ Can’t identify M0 and M7

→ Can’t distinguish ALL-B and ALL-T
Immunophenotyping analysis of the reactivity of leukemic cells with monoclonal antibodies has proved useful and nowadays essential in the diagnosis of acute leukemia.

There is currently a large panel of monoclonal antibodies (over 1000) available which detect different molecules (over 70) on normal hemopoietic and leukemia cells of various lineages.

These monoclonal antibodies are grouped according to the molecule or antigen that they recognize under a cluster designation or differentiation (CD).
Immunophenotyping: Indications

Immunophenotyping analysis should be systematically performed in all cases:

1. Acute leukemia AML VS ALL
2. Blast transformation of CML
3. Blast transformation of other myeloproliferative disorders and myeloid dysplasia.
4. Mixed Lineage Acute Leukemia (MPAL)

Minimal residual disease (MRD) for ALL.
Flowcytometry Principle
Since $FSC \approx$ size and $SSC \approx$ internal structure, a correlated measurement between them can allow for differentiation of cell types in a heterogeneous cell population.
FLOWCYTOMETRY OF IMMUNOPHENOTYPING

FACSCantoII

Software Diva
Figure 1.5  Gating strategy. Part of the sample from the bone marrow aspirate or blood (tube) is smeared on the microscope glass slide for morphologic correlation, while the rest is incubated with antibodies, lysed, fixed and submitted for flow cytometry analysis; see text for details.
Goal: To identify the leukemic population CD45 vs. SSC

The abnormal sample contains a prominent population of blast cells (red) with low SSC and less expression of CD45 than normal lymphocytes (green).
Phenotyping in Acute Leukemia

- CD34
- TdT
- CD2
- cCD3
- CD7
- T-ALL
- CD22
- cCD22
- B-ALL
- CD10
- CD19
- cCD79a
- CD33
- CD13
- CD117
- Stem Cells
- cMPO

Immunophenotyping marker

Markers of Immaturity – TdT, CD34

Lineage Specific markers
Myeloid  - cMPO
B cell    - cCD22/cCD79a
T cell    - cCD3

Lineage Associated markers
Myeloid   - Common CD13, CD33, CD117
           - Other CD11b, CD15
Monocytic - CD13, CD33, CD64, CD68, CD117, CD11b, CD14, CD4, cLysozyme
Erythroid - CD36, CD71, CD105, CD235a (Glycophorphin A), Hb
Megakaryocytic - CD36, CD41, CD42, CD61 and CD62
B cell    - CD19, CD22, CD20, cCD79a, CD10, cIgM, slg
T cell    - Common CD1a, CD2, CD5, CD7, CD10
           - Other CD4, CD8, CD3,
NK cell   - CD16, CD56, CD57, CD94, KIR
PDC       - CD123, CD4, CD56, CD68, CD33, CD43, BDCA,
           - Other on PB subset CD2, CD5, CD7
Immunophenotyping marker

**Lineage Infidelity markers**
(Leukemia associated immunophenotype; LAIP)
Lymphoid markers in AML - CD7, CD56, CD2, CD5 and CD19.
Myeloid markers in ALL – CD13, CD33, CD117, CD15

**Other Markers useful for MRD detection**
Associated with AML – CD38, CD45, CD68, HLADR
Associated with ALL – CD9, CD24, CD25, CD52, CD58, CD81, CD123
Lymphoid differentiation
Myeloid differentiation
Phenotypic changes in the monocytic differentiation pathway

<table>
<thead>
<tr>
<th>MONOBLAST</th>
<th>PROMONOCYTE</th>
<th>MONOCYTE</th>
</tr>
</thead>
<tbody>
<tr>
<td>HLA-DR</td>
<td>CD14</td>
<td>CD14</td>
</tr>
<tr>
<td>CD34</td>
<td>CD45</td>
<td>CD45</td>
</tr>
<tr>
<td>CD117</td>
<td>CD11c</td>
<td>CD11c</td>
</tr>
<tr>
<td>CD64</td>
<td>CD36</td>
<td>CD36</td>
</tr>
<tr>
<td></td>
<td>CD11b</td>
<td>CD11b</td>
</tr>
</tbody>
</table>
Leukemia Phenotyping

A

CD 33 (+) / POS
CD 34 (+) / POS
HLA DR (+) / POS
CD 117 (+) / POS
CD 13 (+) / POS
KESAN: MIELOID LINEAGE

B

CD 33 (+) / POS
CD 34 (+) / POS
HLA DR (+) / POS
CD 117 (+) / POS
CD 13 (+) / POS
CD 19 (+) / POS
KESAN: MIELOID LINEAGE WITH ABBERANT EXP CD 19
Mixed Phenotype Acute Leukemia (MPAL)

WHO 2008 kriteria untuk diagnosis MPAL

Mieloid : MPO pos or monocytic differentiation
   two pos from : NSE, CD11c, CD14, CD64 atau lysozyme

T lineage : cCD3 pos or surface CD3 pos

B lineage : - CD19 strong pos, with one of
   CD79a, CD10, cCD22 pos or
   - CD19 dim pos with two of
     CD79a, CD10, cCD22 pos
   - CD19 negatif with
     CD79a, CD10, cCD22 pos (rare)

Undifferentiated : cCD3, MPO, cCD22 or cCD79a neg and
   CD19 ekspresion neg or weak
Cytogenetics

- Conventional cytogenetics using classical karyotyping of chromosomes remains the most comprehensive method for assessing chromosome abnormalities, especially:
  1. Numeric → Polyploid aneuploid.
  2. Structural → translocation, deletion, inversion

- Techniques:
  1. Fluorescence in situ hybridization (FISH)
  2. Multicolor FISH
  3. Spectral karyotyping (SKY)
Cytogenetics and Prognostic

- t(15;17)$^{PML/RARa}$ is characteristic for acute promyelocytic leukemia (APL), a unique variant of acute myeloid leukemia (AML) treated with ATRA and arsenic dioxide.

- t(8;21) or inversion (16) comprises the favorable risk group of AML, whereas a complex karyotype in AML predicts a poor prognosis.

- t(9;22) $^{BCR/ABL}$ is typical for chronic myeloid leukemia (CML) in more than 95% cases, where it is associated with a poor prognosis $\rightarrow$ development of Imatinib.
Cytogenetics

AML with 5q- and t12;22)
Cytogenetics: FISH

AML with t(8;21)

AML with inversion(16)
Molecular

- PCR (Polymerase Chain Reaction) → targets a segment of DNA (or RNA in reverse-transcriptase PCR) and produces multiple copies (usually between $10^7$ and $10^{11}$) of a DNA region of interest.

- RT-PCR (Reversed Transcriptase PCR) → It is widely used in the diagnosis of genetic diseases, since it makes it easier to detect the presence of specific aberrations by directly detecting the product (mRNA), e.g. fusion transcripts encoded by the translocations.
Mutation in AML

Class I mutations
- FLT3-ITD
- FLT3-TKD
- KIT
- RAS
- PTPN11
- JAK2

Class II mutations
- PML-RARA
- RUNX1-RUNX1T1
- CBFB-MYH11
- MLL fusions
- CEBPA
- NPM1?

Proliferation and/or survival advantage; not affecting differentiation

AML

Impaired haematopoietic differentiation and subsequent apoptosis
# AML: Prognostic Gene Alterations

<table>
<thead>
<tr>
<th>Gene Alteration</th>
<th>Gene Location</th>
<th>Prognostic</th>
</tr>
</thead>
<tbody>
<tr>
<td>FLT3-ITD</td>
<td>13q12</td>
<td>Adverse</td>
</tr>
<tr>
<td>WT1 mutation</td>
<td>11p13</td>
<td>Adverse</td>
</tr>
<tr>
<td>NPM1 mutation</td>
<td>5q35</td>
<td>Favorable</td>
</tr>
<tr>
<td>CEBPA mutation</td>
<td>19q13.1</td>
<td>Favorable</td>
</tr>
<tr>
<td>BAALC overexpression</td>
<td>8q22.3</td>
<td>Adverse</td>
</tr>
<tr>
<td>ERG overexpression</td>
<td>21q22.3</td>
<td>Adverse</td>
</tr>
<tr>
<td>EVI1 expression</td>
<td>3q26.2</td>
<td>Adverse</td>
</tr>
<tr>
<td>MN1 overexpression</td>
<td>22q21.1</td>
<td>Adverse</td>
</tr>
<tr>
<td>FLT3-TKD</td>
<td>13q12</td>
<td>Adverse</td>
</tr>
<tr>
<td>MLL-PTD</td>
<td>11q23</td>
<td>Neutral</td>
</tr>
</tbody>
</table>
## Table 2. Prognostic-risk group based on cytogenetic and molecular profile

<table>
<thead>
<tr>
<th>Prognostic-risk group</th>
<th>Cytogenetic profile alone</th>
<th>Cytogenetic profile and molecular abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Favorable</strong></td>
<td>t(8;21)(q22;q22)</td>
<td>t(8;21)(q22;q22) with no c-KIT mutation</td>
</tr>
<tr>
<td></td>
<td>inv(16)(p13;1q22)</td>
<td>inv(16)(p13;1q22)</td>
</tr>
<tr>
<td></td>
<td>t(15;17)(q22;q12)</td>
<td>t(15;17)(q22;q12)</td>
</tr>
<tr>
<td></td>
<td>Mutated NPM1 without FLT3-ITD (CN-AML)</td>
<td>Mutated biallelic CEBPA (CN-AML)</td>
</tr>
<tr>
<td></td>
<td>t(8;21)(q22;q22) with mutated c-KIT</td>
<td>t(8;21)(q22;q22) with mutated c-KIT</td>
</tr>
<tr>
<td></td>
<td>CN-AML other than those included in the favorable or adverse prognostic group</td>
<td>CN-AML other than those included in the favorable or adverse prognostic group</td>
</tr>
<tr>
<td></td>
<td>t(9;11)(p22;q23)</td>
<td>t(9;11)(p22;q23)</td>
</tr>
<tr>
<td><strong>Intermediate</strong></td>
<td>CN-AML</td>
<td>Cytogenetic abnormalities not included in the favorable or adverse prognostic risk groups</td>
</tr>
<tr>
<td></td>
<td>t(9;11)(p22;q23)</td>
<td>TP53 mutation, regardless of cytogenetic profile</td>
</tr>
<tr>
<td></td>
<td>Cytogenetic abnormalities not included in the favorable or adverse prognostic risk groups</td>
<td>CN with FLT3-ITD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CN with DNMT3A</td>
</tr>
<tr>
<td></td>
<td></td>
<td>CN with KMT2A-PTD</td>
</tr>
<tr>
<td></td>
<td></td>
<td>inv(3)(q21q26.2)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>t(6;9)(p23;q34)</td>
</tr>
<tr>
<td></td>
<td>11q abnormalities other than t(9;11)</td>
<td>11q abnormalities other than t(9;11)</td>
</tr>
<tr>
<td></td>
<td>– 5 or del(5q)</td>
<td>– 5 or del(5q)</td>
</tr>
<tr>
<td></td>
<td>– 7</td>
<td>– 7</td>
</tr>
<tr>
<td></td>
<td>Complex karyotype</td>
<td>Complex karyotype</td>
</tr>
<tr>
<td><strong>Adverse</strong></td>
<td>inv(3)(q21q26.2)</td>
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<td>t(6;9)(p23;q34)</td>
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<td></td>
<td>– 5 or del(5q)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>– 7</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Complex karyotype</td>
<td></td>
</tr>
</tbody>
</table>

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**Abbreviations:** AML, acute myeloid leukemia; ITD, internal tandem duplications.

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Algorithmic of acute leukemia

Presentation of fatigue, malaise, fever, weight loss, bleeding/bruising, symptoms related to anemia, thrombocytopenia, leukocytosis or leukopenia

- CBC analysis/blood smear
  - Blasts present
    - Flow cytometry
      - Bone marrow morphology
        - Acute biphenotypic leukemia
          - Myeloid & lymphoid markers+
            - CD4+/CD56+
          - Blastic NK-cell leukemia
        - Precursor T-ALL
          - Cytogenetics/FISH
      - Precursor B-ALL
        - Cytogenetics/FISH
        - DNA ploidy
      - AML
        - Cytogenetics/FISH
  - Abnormal karyotype
    - APL (AML-M3)
      - t(15;17)
      - t(5;17)
      - t(11;17)
    - AML M4eo
      - t(16;16) inv(16)
    - AML with RUNX1/ETO
      - t(8;21)
    - c-KIT
    - AML with BCR/ABL
      - t(9;22)
      - other abnormalities
      - Molecular tests (PCR)
        - CEBHA
        - MLL
        - FLT3
        - NPN1
        - WT-1
      - AML with BCR/ABL
        - CD19+/CD56+
Distribution of Acute Leukemia in Children 2011 – 2015

- T-LINEAGE: 1078 (61%)
- B-LINEAGE: 543 (30%)
- MIELOID LINEAGE: 154 (9%)
Distribution of Acute Leukemia in Adult 2011 – 2015

- **T-Lineage**: 88 (5%)
- **B-Lineage**: 510 (31%)
- **Mieloid Lineage**: 1063 (64%)
Aberrant Exp Acute Leukemia 2011 - 2015

LEUKEMIA AKUT, 2514, 74%
MIXED, 16, 0%
ABBERANT, 885, 26%
## SUMMARY

### Molecular Diagnosis of Acute Leukemia

<table>
<thead>
<tr>
<th>Cell morphology</th>
<th>Changes in cellular and nuclear size</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cell/nucleus proportion</td>
</tr>
<tr>
<td></td>
<td>Chromatin structure</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Surface markers</th>
<th>CD’s</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>Used for diagnosis/DD/MRD</td>
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<tr>
<td></td>
<td>Classification</td>
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<td>Prognosis</td>
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<th>prognosis</th>
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</table>
Thank You

2nd Jakarta International Society for Advancement of Cytometry (ISAC) meeting, October 11-13, 2016.
CYTO ASIA 2017
SINGAPORE

WORKSHOP: 22nd - 24th October 2017
CONFERENCE: 25th - 27th October 2017

A joint meeting of the International Society for Advancement of Cytometry (ISAC), the Australasian Cytometry Society (ACS) and the Singaporean Society for Immunology (SgSI)

Venue: Grand Copthorne Waterfront Hotel - Singapore

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