LAML - Acute Myeloid Leukemia

Subtype	Biology & Expression	Genomic Alterations	Clinical Features
FLT3-ITD–enriched	• Enriched for proliferation, MYC targets, and stem-like expression • Low differentiation gene expression	• **FLT3-ITD** mutations (internal tandem duplication) • Often co-occurs with **NPM1** or **DNMT3A** mutations	• Poor prognosis (especially with high FLT3-ITD allelic ratio) • Targetable with FLT3 inhibitors
CEBPA-mutant	• High granulocytic differentiation genes • Favorable-risk gene expression profile	• **Biallelic CEBPA** mutations	• Favorable prognosis • Younger patients • Good response to standard chemotherapy
NPM1-mutant/normal karyotype	• Intermediate differentiation • Variable stem/progenitor-like signatures	• **NPM1** mutations • Often with **DNMT3A**, **IDH1/2**, or **FLT3-ITD** co-mutations	• Intermediate to poor prognosis depending on co-mutations • Common in cytogenetically normal AML
TP53/complex karyotype	• Deregulated cell cycle, high chromosomal instability	• **TP53** mutations • Complex structural chromosomal abnormalities	• Very poor prognosis • Elderly patients • Chemotherapy resistant
IDH1/2-mutant	• Epigenetically dysregulated, DNA hypermethylation signature	• **IDH1** or **IDH2** mutations	• Variable prognosis • Targetable with IDH inhibitors
RUNX1-mutant	• Impaired hematopoietic differentiation	• **RUNX1** mutations	• Poor prognosis • Often therapy-related AML or secondary AML