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JOURNAL FULL TITLE: Journal of Biomedical Research & Environmental Sciences
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JOURNAL OF BIOMEDICAL RESEARCH & ENVIRONMENTAL SCIENCES
ISSN: 2766-2276
SUBJECT AREA(S): BIOMEDICAL SCIENCE | BIOMEDICINE | BIOTECHNOLOGY
DOI: 10.37871

REVIEW ARTICLE

A Comprehensive Review on Acute Myelogeneous Leukemia

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Abstract

Neoplastic myeloid diseases are clonal hematopoietic stem cell disorders characterized by abnormal proliferation, impaired differentiation, and accumulation of myeloid cells, resulting in disrupted normal hematopoiesis. Acute Myelogenous Leukemia (AML) is the most aggressive of these disorders and predominantly affects older adults, although it may occur at any age. Its development is driven by genetic and epigenetic alterations, environmental exposures, previous cytotoxic therapy, and antecedent hematologic diseases. Patients typically present with manifestations of bone marrow failure, including anemia, infections, bleeding, and constitutional symptoms. Diagnosis relies on complete blood count, peripheral blood smear, bone marrow examination, immunophenotyping, cytogenetic analysis, and molecular testing for prognostic mutations. Prognosis depends on age, cytogenetic and molecular risk profiles, treatment response, and measurable residual disease status. Management combines supportive care, intensive or low-intensity chemotherapy, targeted therapies, and allogeneic hematopoietic stem cell transplantation, with individualized treatment strategies improving survival and long-term outcomes.

Introduction

Acute Myelogenous Leukemia (AML) is a rapidly progressive hematological malignancy characterized by the uncontrolled proliferation of immature myeloid precursor cells (myeloblasts) in the bone marrow, peripheral blood, and occasionally other tissues [1-4]. It is the most common acute leukemia in adults, although it can occur at any age [1,2]. The accumulation of leukemic blasts suppressesnormalhematopoiesis,resultinginanemia,neutropenia,

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DOI: 10.37871/jbres2324

Submitted: 26 July 2026

Accepted: 12 August 2026

Published: 14 August 2026

Keywords

- Acute Myeloid Leukemia
- Epidemiology
- Etiology
- Presentations
- Diagnosis
- Prognosis
- Management
- Polymer
- Water reclamation

VOLUME: 7 ISSUE: 8 - AUGUST, 2026

How to cite this article: Ozochukwu OC, Ufelle SA, Ogbuabor AO. A Comprehensive Review on Acute Myelogeneous Leukemia. J Biomed Res Environ Sci. 2026 August 14; 7(8): 6. Doi: 10.37872/jbres2324

and thrombocytopenia, which clinically manifest as fatigue, recurrent infections, fever, and bleeding tendencies [1,2,5]. AML develops through the acquisition of genetic, epigenetic, and chromosomal abnormalities that disrupt normal myeloid cell differentiation and promote uncontrolled proliferation of leukemic clones [2-4,6,7]. Recent advances in cytogenetic and molecular diagnostics have enhanced disease classification, prognostic stratification, and the development of targeted therapies, significantly improving treatment outcomes and survival for many patient populations [1,2,6,7].

Epidemiology

Acute Myelogenous Leukemia (AML) is the most common acute leukemia in adults, accounting for approximately 80% of adult acute leukemia cases [8,9]. Its incidence increases with age, with a median age at diagnosis of approximately 68–70 years [1,8]. AML occurs slightly more frequently in males than females and is more prevalent in high-income countries [8,10]. Globally, the incidence is approximately 3-5 cases per 100,000 persons annually, with survival influenced by age, cytogenetic risk, access to treatment, and healthcare resources [8-10].

Etiology

Acute Myelogenous Leukemia (AML) arises from the interaction of genetic susceptibility and environmental exposures that induce somatic mutations in hematopoietic stem cells [2,6]. Established risk factors include advanced age, previous chemotherapy or radiotherapy, benzene exposure, tobacco smoking, ionizing radiation, inherited disorders such as Down syndrome and Fanconi anemia, and antecedent myelodysplastic syndromes or myeloproliferative neoplasms [2,6]. Recurrent mutations involving FLT3, NPM1, CEBPA, IDH1/2, TP53, and RUNX1 disrupt normal myeloid differentiation, resulting in clonal expansion and leukemogenesis [2,6,7].

Pathogenesis

The disease develops through the sequential acquisition of somatic mutations in hematopoietic stem or progenitor cells, producing uncontrolled proliferation of immature myeloid blasts and impaired differentiation. Mutations involving FLT3, NPM1, IDH1/2, RUNX1, TP53, DNMT3A, and CEBPA disrupt signaling, transcription, epigenetic regulation, and apoptosis, conferring clonal advantage. Leukemic blasts progressively replace normal bone marrow, suppressing erythropoiesis, granulopoiesis, and thrombopoiesis, while additional cooperating mutations promote disease progression, immune evasion, and therapeutic resistance [2,6,7].

Presentations

Patients with Acute Myelogenous Leukemia (AML) commonly present with symptoms of bone marrow failure, including fatigue, pallor, dyspnea, and weakness due to anemia. Neutropenia predisposes to recurrent infections and persistent fever, while thrombocytopenia causes easy bruising, petechiae, mucosal bleeding, and epistaxis. Leukemic infiltration may produce hepatosplenomegaly, lymphadenopathy, bone pain, gingival hypertrophy, or leukemia cutis. Hyperleukocytosis can result in leukostasis with respiratory distress or neurological deficits. Constitutional symptoms such as weight loss, night sweats, and anorexia are also frequent manifestations [1,2,11].

Classification

Acute Myeloid Leukemia (AML) is classified using morphology, immunophenotype, cytogenetics, and molecular abnormalities, with current systems emphasizing genetically

Table 1: The classification and treatment approach are based mainly on the WHO/ICC frameworks and ELN 2022 recommendations.

AML classification	ELN risk group	Important examples	Common treatment options
AML with defining genetic abnormalities	**Favorable**	RUNX1::RUNX1T1, CBFB::MYH11, NPM1-mutated without FLT3-ITD, bZIP CEBPA mutation	Intensive induction chemotherapy, usually cytarabine + anthracycline; consolidation with high-dose cytarabine. Targeted therapy may be added where indicated.
AML with defining genetic abnormalities	**Intermediate**	NPM1 with FLT3-ITD, FLT3-ITD without adverse abnormalities, KMT2A::MLLT3	Intensive chemotherapy ± FLT3 inhibitor (e.g., midostaurin); consolidation based on response and MRD. Allogeneic transplant may be considered.
AML with myelodysplasia-related abnormalities	**Adverse**	Mutations in ASXL1, RUNX1, SRSF2, U2AF1, STAG2, etc.; adverse cytogenetics	Intensive chemotherapy where appropriate; CPX-351 may be considered for therapy-related/AML-MR disease; allogeneic stem-cell transplantation in eligible patients.
AML with adverse genetic abnormalities	**Adverse**	TP53 mutation, MECOM rearrangement, DEK::NUP214, KMT2A rearrangement, BCR::ABL1, complex/ monosomal karyotype	Clinical trials, intensive or lower-intensity therapy according to fitness; azacitidine + venetoclax commonly used in less-fit patients; transplant considered selectively.
Acute promyelocytic leukemia (APL)	**Favorable/intermediate depending on WBC**	PML::RARA	**ATRA + arsenic trioxide (ATO)** for non-high-risk APL; high-risk disease generally receives ATRA + ATO with additional cytoreductive/chemotherapy approaches.
AML, not otherwise specified / other AML	**Intermediate or adverse**	Cases without defining favorable genetic abnormalities	Treatment according to age, fitness, molecular profile, ELN risk, MRD response, and transplant eligibility.

Key point: ELN 2022 divides AML genetically into **favorable, intermediate, and adverse-risk groups**. Importantly, **FLT3-ITD allelic ratio is no longer used** for ELN risk assignment; FLT3-ITD AML is generally classified as intermediate risk unless an adverse-risk abnormality is present.

defined disease. As shown in table 1, the WHO 5th edition (WHO-HAEM5) and International Consensus Classification (ICC) recognize AML with defining genetic abnormalities, including PML::RARA, RUNX1::RUNX1T1, CBFB::MYH11, KMT2A rearrangement, DEK::NUP214, RBM15::MRTFA, MECOM rearrangement, NUP98 rearrangement, NPM1 mutation, and CEBPA mutation [3-5,12]. Both classifications have reduced reliance on morphology and incorporated molecular genetics into diagnosis [4,5,12].

An important difference concerns the blast threshold. WHO-HAEM5 generally does not require a specific blast percentage for most genetically defined AML, whereas ICC generally requires ≥10% blasts for many recurrent genetic abnormalities, with specific exceptions [3,4,12]. The WHO also uses a broader, less hierarchical structure, whereas ICC applies a hierarchical classification. Furthermore, WHO and ICC differ in their definitions of myelodysplasia-related AML, TP53-mutated AML, and certain genetic entities [3-5,12]. Despite these differences, both systems substantially improve biologic and clinically relevant AML classification.

ELN Risk Classification

The 2022 European LeukemiaNet (ELN) risk classification stratifies AML into favorable, intermediate, and adverse-risk groups using cytogenetic and molecular abnormalities to predict treatment response, relapse, and overall survival [2,13]. Favorable-risk AML includes *RUNX1::RUNX1T1*, *CBFB::MYH11*, *NPM1*-mutated AMLwithoutFLT3-ITD,andin-framebZIP*CEBPA*

mutations. Intermediate-risk AML includes NPM1-mutated AML with FLT3-ITD, FLT3-ITD without favorable or adverse abnormalities, and certain KMT2A rearrangements. Adverse-risk AML includes TP53 mutations, myelodysplasia-related gene mutations, complex karyotype, and specific adverse genetic abnormalities [2,13]. The 2022 ELN system provides improved prognostic discrimination compared with earlier classifications [13,14]. Measurable residual disease (MRD) assessment provides additional prognostic information and can refine risk assessment after treatment [15,16].

Diagnosis

The diagnosis of Acute Myeloid Leukemia (AML) integrates clinical findings, peripheral blood and bone marrow morphology, immunophenotyping, cytogenetics, and molecular genetic studies [1,2]. Patients commonly present with manifestations of anemia, neutropenia, and thrombocytopenia, including fatigue, infections, fever, bruising, and bleeding. Initial laboratory investigations include a complete blood count and peripheral blood film, which may demonstrate circulating myeloblasts and other abnormal cells. Bone marrow aspiration and biopsy remain central to diagnosis, allowing assessment of blast morphology and marrow architecture [1,2]. Multiparameter flow cytometry establishes myeloid lineage and characterizes leukemic blasts using markers such as CD13, CD33, CD117, Myeloperoxidase (MPO), and HLA-DR [17].

Cytogenetic analysis, including conventional karyotyping and fluorescence in situ hybridization, identifies recurrent chromosomal abnormalities. Molecular testing detects important mutations and gene fusions, including NPM1, FLT3, CEBPA, TP53, RUNX1::RUNX1T1, CBFB::MYH11, and PML::RARA [1,2,18]. The WHO and ICC classifications emphasize genetically defined AML and permit diagnosis of several AML subtypes without the traditional 20% blast threshold [3,4,19,20]. Cytochemistry, particularly MPO staining, may provide supportive evidence of myeloid differentiation. Together, these investigations establish the diagnosis, define the AML subtype, determine prognosis, and guide treatment selection.

Prognosis

The prognosis of Acute Myeloid Leukemia (AML) is highly variable and depends on patient-, disease-, and treatment-related factors. Important patient factors include age, performance status, comorbidities, and tolerance of intensive therapy. Older patients generally have poorer outcomes because of adverse disease biology and reduced treatment tolerance [1,2]. Cytogenetic and molecular abnormalities are major determinants of prognosis, with the ELN classification categorizing patients into favorable, intermediate, and adverse-risk groups [2,13,14]. Favorable-risk abnormalities are generally associated with better survival, whereas TP53 mutations, complex karyotypes, and myelodysplasia-related abnormalities are associated with poor outcomes [2,13,14]. Measurable Residual Disease (MRD) is another important prognostic marker. Patients achieving MRD negativity following treatment generally have substantially better overall and disease-free survival than those with persistent MRD [16,21,22]. Prognosis is also influenced by achievement and duration of complete remission, relapse status, response to targeted therapy, and successful allogeneic hematopoietic stem-cell transplantation [2,23]. Consequently, modern AML prognostication integrates clinical characteristics, cytogenetic and molecular findings, ELN risk category, treatment response, and MRD status to support individualized treatment decisions [2,16,23].

Treatment

Treatment of Acute Myeloid Leukemia (AML) depends on disease subtype, age,

fitness, molecular abnormalities, ELN risk, and Measurable Residual Disease (MRD). Acute Promyelocytic Leukemia (APL) is treated separately because it is highly sensitive to differentiation therapy. All-Trans Retinoic Acid (ATRA) should be initiated immediately when APL is suspected. ATRA plus arsenic trioxide (ATO) is standard for non-high-risk APL, while high-risk disease requires additional cytoreduction [11,24].

For non-APL AML, fit patients generally receive intensive induction chemotherapy, commonly cytarabine plus an anthracycline, followed by consolidation and, where indicated, allogeneic hematopoietic stem-cell transplantation [2,11,24]. Targeted agents include FLT3 inhibitors for FLT3-mutated AML and IDH inhibitors for IDH1/2-mutated disease [25]. Older or medically unfit patients commonly receive lower-intensity hypomethylating agents combined with venetoclax [26,27]. Maintenance therapy, including oral azacitidine or selected targeted agents, may reduce relapse risk in appropriate patients [2,28]. Relapsed/refractory AML is managed with salvage chemotherapy, molecularly targeted therapy, clinical trials, and transplantation when feasible (Table1) [29]. Supportive care includes red-cell and platelet transfusion, antimicrobial prophylaxis, treatment of febrile neutropenia, tumor-lysis prevention, and management of treatment-related complications [2].

Conclusion

Acute myelogenous leukemia is a heterogeneous hematologic malignancy requiring prompt diagnosis, accurate risk stratification, and individualized treatment. Advances in molecular diagnostics, targeted therapies, and measurable residual disease monitoring have significantly improved patient outcomes. Continued research and multidisciplinary care remain essential for optimizing survival, reducing relapse, and improving quality of life.

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