# Acute myeloid leukemia: A detailed overview.

**Riley Morgan***

Department of Transfusion Medicine, Massachusetts Institute of Technology, United States

## Introduction

Acute Myeloid Leukemia (AML) is a malignant disorder of the hematopoietic system characterized by the rapid proliferation of abnormal myeloid precursor cells in the bone marrow and peripheral blood. This condition leads to the replacement of normal hematopoietic cells, resulting in anemia, infection, and bleeding tendencies [1].

AML is the most common acute leukemia in adults, with increasing incidence in the elderly. This article explores the pathophysiology, risk factors, clinical presentation, diagnostic criteria, and treatment options for AML [2].

AML arises from genetic and epigenetic alterations in hematopoietic stem cells or early progenitor cells, leading to uncontrolled proliferation and accumulation of immature myeloid cells, known as blasts. These blasts interfere with normal hematopoiesis by crowding out healthy cells in the bone marrow and by secreting inhibitory cytokines [3].

Several risk factors have been identified for AML: Age: The incidence of AML increases with age, with a median age at diagnosis of around 68 years. Genetic Disorders: Conditions such as Down syndrome, Fanconi anemia, and Bloom syndrome predispose individuals to AML [4].

Previous Hematologic Disorders: Myelodysplastic syndromes (MDS) and chronic myeloproliferative disorders can evolve into AML. Exposure to Carcinogens: Long-term exposure to benzene, ionizing radiation, and chemotherapy agents (alkylating agents, topoisomerase II inhibitors) increases the risk of AML. Smoking: Cigarette smoking has been associated with an increased risk of AML [5].

Patients with AML often present with symptoms related to bone marrow failure and organ infiltration by leukemic cells. Common symptoms and signs include: Anemia: Fatigue, pallor, and dyspnea due to reduced red blood cell production. Infection: Fever and infections caused by neutropenia (low neutrophil count) [6].

Bleeding: Easy bruising, petechiae, and hemorrhages due to thrombocytopenia (low platelet count). Bone Pain: Due to marrow expansion. Organomegaly: Hepatosplenomegaly and lymphadenopathy in some cases [7].

The diagnosis of AML is based on a combination of clinical evaluation, laboratory tests, and bone marrow examination: Complete Blood Count (CBC): Often shows anemia, neutropenia, and thrombocytopenia with circulating blasts [8].

Bone Marrow Biopsy: Essential for diagnosis, typically showing hypercellularity with ≥20% blasts.

Cytogenetic Analysis: Identifies chromosomal abnormalities such as t(8;21), inv(16), and t(15;17). Molecular Testing: Detects gene mutations like FLT3, NPM1, and CEBPA, which have prognostic and therapeutic implications [9].

Consolidation Therapy: Post-remission therapy to eliminate residual disease, which may include additional chemotherapy or hematopoietic stem cell transplantation (HSCT). Targeted Therapy: Agents such as FLT3 inhibitors (midostaurin), IDH1/2 inhibitors (ivosidenib, enasidenib), and BCL-2 inhibitors (venetoclax) are used for specific genetic mutations. Supportive Care: Includes blood product transfusions, antimicrobial prophylaxis, and management of treatment-related complications [10].

## Conclusion

Acute Myeloid Leukemia is a complex and aggressive hematologic malignancy with significant morbidity and mortality. Advances in genetic and molecular understanding have improved the diagnosis, risk stratification, and treatment of AML. Early detection and tailored therapies are crucial for improving patient outcomes. Ongoing research and clinical trials continue to explore novel therapeutic strategies to enhance survival and quality of life for AML patients.

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***Correspondence to:** Riley Morgan, Department of Transfusion Medicine, Massachusetts Institute of Technology, United States, E-mail: Morgan67@mit.edu

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