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REVIEW ARTICLE

MYELOGENOUS LEUKEMIA: DIAGNOSIS, THERAPEUTIC APPROACH AND FUTURE PERSPECTIVES: A NARRATIVE REVIEW

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Abstract

Acute myeloid leukaemia (AML) and Chronic myeloid leukaemia (CML) present unique and significant difficulties in the field of blood cancers. They originate from the harmful transformation of myeloid cells and are linked to distinct pathophysiological characteristics that require specialized diagnostic and treatment methods. The diagnosis of CML primarily relies on the detection of the BCR-ABL1 fusion gene, which represents a key feature of the disorder. This genetic marker not only validates the diagnosis but also guides the treatment approach. Nonetheless, AML is characterized by its genetic and clinical diversity, making diagnosis challenging and necessitating a more personalized treatment strategy. Progress in molecular diagnostics has uncovered particular mutations that can be addressed by new therapies, enabling more individualized treatment plans. Studies in both AML and CML seek to determine the shortcomings of current treatments. The main focus of research in CML involves creating next-generation tyrosine kinase inhibitors (TKIs) and methods to eliminate minimal residual disease (MRD), whereas in AML, combining targeted therapies, immunotherapies, and progress in molecular diagnostics offers potential for improved patient results. Trustworthy data was gathered from Tailor and Francis, PubMed, Springer, Nature, Google Scholar, MDPI, BMC, and several other relevant sources. This review explores the molecular mechanisms, diagnostic methods, potential treatment options, and future outlook of myelogenous leukaemia.

Keywords: Diagnosis, future perspectives, myelogenous leukaemia, therapeutic approach.

INTRODUCTION

Myelogenous leukaemia, or myeloid leukaemia, is a form of blood cancer impacting peripheral blood and bone marrow, marked by the swift proliferation of atypical white blood cells from the myeloid lineage. This type of leukaemia is categorized as acute myeloid leukaemia (AML) and chronic myeloid leukaemia (CML). Acute myeloid leukaemia (AML) is a cancer that originates in the bone marrow and impacts blood cells[1]. It is characterized by the swift rise of abnormal white blood cells, which gather in the bone marrow and hinder the creation of healthy blood cells. Individuals with AML might show signs like tiredness, an increased susceptibility to infections, easy bruising or bleeding, and difficulty breathing. Timely intervention is vital for AML and can include chemotherapy, targeted therapies, radiation, and sometimes stem cell transplants. Although it is more prevalent among older individuals, it can arise at any age. AML includes a variety of disorders, defined by the clonal expansion of immature myeloid cells in the peripheral blood and bone marrow, and is frequently linked to bone marrow failure[2]. In 2012, AML recorded a global total of 351,965 cases, yielding an age-standardized rate of 4.7 per 100,000 people. The 5-year prevalence was recorded at 1.5%, exhibiting a male-to-female ratio of roughly 1:4. Moreover, the occurrence of AML appears to be rising in developed nations[3].

Chronic myeloid leukaemia (CML), known as chronic myelogenous leukaemia, is a disorder of hematopoietic stem cells similar to other forms of leukaemia. CML is defined by a unique genetic translocation (9;22) (q34; q11), leading to the merger of the BCR and ABL1

genes, thus producing the detrimental BCR-ABL1 oncogene.

This genetic alteration triggers multiple subsequent effects. The main effect of this combined oncogene is the continuous activation of the tyrosine kinase pathway, granting mutated hematopoietic stem cells a growth benefit compared to their normal counterparts, ultimately resulting in the slow replacement of the standard HSC population (Table 1)[4,5].

Table 1: **Diagnostic modalities and their clinical significance in myelogenous leukemia.**

Diagnostic Modality	Key Features	Clinical Significance
Complete Blood Count (CBC)	Leukocytosis or leukopenia, anemia, thrombocytopenia	Initial screening; identifies cytopenias and abnormal leukocyte counts
Peripheral Blood Smear	Presence of blasts, dysplastic myeloid cells, basophilia (CML)	Helps differentiate AML from CML; detects circulating abnormal cells
Bone Marrow Examination	Blast percentage, cellularity, morphologic dysplasia	Gold standard for diagnosis; determines disease burden and lineage involvement
Flow Cytometry (Immunophenotyping)	Detection of myeloid markers (CD13, CD33, CD117) and aberrant antigen expression	Confirms myeloid lineage, distinguishes AML subtypes, and monitors minimal residual disease
Cytogenetic Analysis (Karyotyping)	Chromosomal abnormalities (t(8;21), inv(16), t(15;17), Philadelphia chromosome)	Prognostic stratification; guides therapy selection, especially HSCT decisions
Fluorescence In Situ Hybridization (FISH)	Detects BCR-ABL1 fusion, other cryptic translocations	Rapid confirmation of key genetic alterations, particularly in CML
Polymerase Chain Reaction (PCR)	Quantifies BCR-ABL1 transcript or specific mutations	Sensitive detection of minimal residual disease and treatment response monitoring
Next-Generation Sequencing (NGS)	Mutational profiling (FLT3, NPM1, IDH1/2, TP53)	Risk stratification, targeted therapy selection, and prognostication
Imaging (Optional)	CT/MRI for organ infiltration	Assesses extramedullary disease in AML or blast crisis in CML

Pathophysiology of AML

Acute myeloid leukaemia (AML) is characterized by its complexity, marked by the irregular proliferation and development of myeloid precursor cells in the bone marrow[3]. AML is a clonal disorder that stems from a modified hematopoietic stem cell (HSC), leading to a clonal proliferation that can develop in otherwise healthy people and frequently appears before leukemia onset. With advancing age, repeated mutations associated with AML can build up in hematopoietic progenitor cells, even in those who are healthy[4,5]. This buildup can result in clonal expansion without advancing to leukaemia, a condition referred to as clonal haematopoiesis of indeterminate potential (CHIP). Mutated cells acquire a growth benefit by multiplying while still preserving normal blood cell generation. The likelihood of advancing from clonal haematopoiesis to AML is estimated to be between 0.5% and 1% each year. Significant differences between age-associated clonal hematopoiesis and pre-leukemic states encompass the number of mutations per sample, an elevated variant allele frequency, and mutations in particular genes like DNMT3A, TET2, SRSF2, and ASXL1[6].

Pathophysiology of CML

The existence of the BCR-ABL1 fusion gene in hematopoietic stem cells has been shown to be enough to trigger chronic myeloid leukaemia (CML). As a result, the pathophysiology of CML is intimately linked to the BCR-ABL1 fusion gene. Around 90% to 95% of CML patients show a reciprocal translocation between chromosomes 9 and 22, referred to as (9;22) (q34; q11.2). This translocation results in a truncated chromosome 22, known as the Philadelphia chromosome, which houses the BCR-ABL1 oncogene[7]. It is essential to point out that BCR-ABL1 can also be detected in other types of leukaemia, including acute lymphoblastic leukaemia (ALL) and acute myeloid leukaemia (AML). The importance of BCR-ABL1 in ALL and AML is still being explored[8]. The process leading to the creation of the Philadelphia chromosome and the duration from its formation to the appearance of clinical signs of chronic myeloid leukaemia (CML) is still unclear. At first, it was thought that the Philadelphia chromosome was a result of radiation exposure from the atomic bombings in Hiroshima and Nagasaki, especially impacting CML patients in Japan. In fact, in vitro studies have shown that exposure to elevated radiation levels in myeloid cell lines can result in the expression of the BCR-ABL1 fusion gene. Nonetheless, most people do not encounter long-term radiation exposure. Certain specialists suggest that the Philadelphia chromosome might arise from random spontaneous translocation. This theory is backed by the finding that the BCR-ABL1 gene exists in a minor proportion of healthy individuals, indicating that not every occurrence of the BCR-ABL1 fusion results in the onset of CML[9].

Epidemiology of AML

Acute myeloid leukaemia (AML) constitutes about 25% of all leukaemia cases in adults in the Western world, making it the most common form of leukaemia in this age demographic. In a study, which included 82 patients across all age groups, the male-to-female ratio was 1.2:1. The exact frequency of gender distribution has also been reported in different international and local studies, e.g. 1.5:1. 1.2:1 and 1.7:1[10]. The epidemiology of Acute Myeloid leukaemia (AML) in Nigeria presents considerable challenges. Many clinicians report unfavourable outcomes, with over 67% indicating that AML patients have a poor or very

poor prognosis. The survival rate is alarmingly low, with less than 31% of patients surviving induction therapy and even fewer managing to survive after induction. Contributing factors to this grim scenario include late diagnosis, limited diagnostic resources, and inadequate supportive care. Additionally, the availability of essential blood components necessary for AML treatment is restricted in many regions of Nigeria[11].

Epidemiology of CML

Chronic myeloid leukaemia (CML) in Nigeria, similar to numerous low- to middle-income nations, poses significant epidemiological difficulties. CML is a disorder of hematopoietic stem cells, with its incidence increasing due to improved diagnostic methods; nonetheless, there is a scarcity of data concerning Nigeria[12]. Worldwide, CML represents about 15-20% of adult leukemia cases. The occurrence of chronic myeloid leukaemia (CML) generally falls between 1 and 2 cases for every 100,000 individuals each year. In Africa, especially in Nigeria, the occurrence is thought to be less; however, accurate data is rare due to underreporting and restricted healthcare access. In Nigeria, elements like economic status, healthcare facilities, and access to treatments such as tyrosine kinase inhibitors (TKIs) greatly affect disease outcomes. Access to TKIs, which have greatly enhanced the outlook for CML globally, is frequently limited, resulting in increased mortality and morbidity rates in the area. Research shows that low- to middle-income nations, such as Nigeria, have a higher percentage of CML-related fatalities among younger demographics than high-income countries. This gap highlights the critical requirement to improve diagnostic and treatment services for better management of CML in Nigeria. The overall impact of CML in Nigeria highlights the urgent need for enhanced healthcare strategies, involving improved access to treatments, advanced diagnostic tools, and thorough epidemiological research to properly evaluate the disease's effects[13].

Diagnosis of AML

Diagnostic methods for AML include blood tests, bone marrow examinations, immunopheno typing, cyto-genetic analysis, and advanced molecular genetic testing to identify mutations critical for treatment[14].

1. **Morphology and cytochemistry:** The first step in diagnosing acute myeloid leukaemia (AML) typically includes analysing blood and bone marrow samples under a microscope to detect the presence of myeloblasts, which are immature white blood cells. Cytochemical stains like myeloperoxidase and Sudan Black B assist in distinguishing AML from other forms of leukaemia.
2. **Immunophenotyping:** This approach employs flow cytometry to identify cell markers, helping to discern particular subtypes of AML by detecting antigens such as CD13, CD33, and CD34 located on the surface of leukaemia cells.
3. **Cytogenetic analysis:** Chromosomal abnormal-ities frequently occur in acute myeloid leukaemia (AML) and play a crucial role in determining prognosis. Techniques like karyotyping and fluorescence in situ hybridisation (FISH) are employed to detect chromosomal translocations, inversions, and deletions. Significant abnormalities include t (8;21), inv (16), and t (15;17).
4. **Molecular genetic testing:** Genetic mutations in specific genes are frequently associated with Acute Myeloid Leukaemia (AML). Techniques like polymerase chain reaction (PCR) and next-generation sequencing (NGS) effectively detect mutations in genes such as FLT3, NPM1, and CEBPA. These genetic alterations can influence treatment strategies and patient outcomes. Molecular genetic testing, especially NGS, is the most accurate diagnosis of AML. NGS can detect minimal residual disease (MRD) and identify mutations in genes like FLT3, NPM1, and IDH1/2, essential for determining diagnosis, prognosis, and treatment options[15].

Diagnosis of CML

1. Complete Blood Count (CBC) and Peripheral Blood Smear: A complete blood count (CBC) is generally the first test conducted when chronic myeloid leukaemia (CML) is suspected. This test commonly shows an increased white blood cell count, particularly with neutrophils and basophils. Additionally, a peripheral blood smear may display immature granulocytes, a characteristic indicator of CML[16].

2. Bone marrow biopsy and aspiration: Bone marrow biopsy and aspiration are essential procedures for confirming the diagnosis by allowing for the examination of bone marrow architecture and cell morphology. Typically, these tests reveal hypercellularity with an increase in myeloid precursors[17]. This methodology is vital for determining whether the chronic myeloid leukaemia (CML) phase is chronic, accelerated, or in the blast phase.

3. Cytogenetic testing (Karyotyping): Cytogenetic analysis is a fundamental diagnostic technique for identifying the Philadelphia chromosome (Ph) due to the translocation between chromosomes 9 and 22. This translocation gives rise to the BCR-ABL1 fusion gene, characteristic of chronic myeloid leukaemia (CML)[18,19].

4. Fluorescence in Situ Hybridization (FISH): FISH directly detects the BCR-ABL1 fusion gene in blood or bone marrow cells. It is more sensitive than karyotyping and can identify even low levels of the Philadelphia chromosome[20].

5. Polymerase Chain Reaction (PCR) testing: PCR is the most sensitive technique for detecting the BCR-ABL1 fusion gene. Real-time quantitative PCR (qPCR) allows for measuring BCR-ABL1 transcript levels in the blood, which is crucial for monitoring therapeutic response[21]. This method can also identify minimal residual disease, aiding prognosis and treatment modifications.

6. Next-Generation Sequencing (NGS): Next-generation sequencing (NGS) has emerged as a significant tool for the molecular characterisation of chronic myeloid leukaemia (CML). It can detect additional mutations within the BCR-ABL1 gene that

may contribute to resistance against tyrosine kinase inhibitors (TKIs), thereby impacting treatment decisions[22]. NGS provides comprehensive genomic profiling and can identify co-morbid mutations that may influence disease progression.

7. Flow cytometry: Flow cytometry is utilised in instances of CML that advance to the blast phase. It assists in determining the lineage of the blast cells (myeloid or lymphoid), which is crucial for guiding treatment decisions[23]. Flow cytometry is particularly valuable in distinguishing CML from other myeloproliferative disorders.

Therapeutic approach of AML

Acute Myeloid Leukaemia (AML) is a fast-growing blood cancer that requires prompt intervention. In recent years, the treatment options for AML have evolved considerably, blending conventional methods with new and innovative strategies to improve patient outcomes. Below are some important therapeutic approaches for AML:

1. Chemotherapy: Conventional chemotherapy remains the cornerstone of acute myeloid leukaemia (AML) treatment. The most frequently used regimen consists of a combination of cytarabine and an anthracycline, usually daunorubicin or idarubicin, and is commonly known as the "7+3" protocol[1]. The goal of chemotherapy is to attain remission by removing leukemic cells from the bone marrow, followed by consolidation therapy to minimise the chances of recurrence.

2. Targeted therapy: The development of targeted therapies has revolutionised the treatment of AML, particularly in patients with specific genetic mutations:

a. **FLT3 inhibitors:** About 30% of individuals with acute myeloid leukaemia (AML) have mutations in the FLT3 gene. The FLT3 inhibitors midostaurin and gilteritinibhave been approved for treating AML with FLT3 mutations. These drugs specifically target and inhibit the activity of the mutant FLT3 kinase, leading to a decrease in the growth of leukemic cells.

b. **IDH inhibitors:** Isocitrate dehydrogenase (IDH) mutations are found in approximately 20% of acute myeloid leukaemia (AML) cases. Ivosidenib and enasidenib specifically target IDH1 and IDH2 mutations, encouraging leukemic cells' differentiation and programmed cell death[24].

3. Hypomethylating agents: Hypomethylating agents, such as azacitidine and decitabine, are mainly used in older patients or are not candidates for intensive chemotherapy. These drugs work by reactivating tumour suppressor genes and promoting apoptosis in leukemic cells. They are frequently administered with venetoclax, a BCL-2 inhibitor, to enhance treatment efficacy[13].

4. Immunotherapy: Immunotherapy for AML is an emerging field. The main strategies include:

a. **Monoclonal antibodies:** Gemtuzumabozogamicin is a targeted antibody-drug conjugate for treating CD33-positive acute myeloid leukaemia (AML). It delivers a cytotoxic payload directly to leukemic cells, sparing normal cells[25].

b. **Chimeric Antigen Receptor (CAR) T-cell therapy:** Although still in the preliminary stages of development, CAR T-cell therapy that targets antigens such as CD33 and CD123 is currently under investigation as a potential treatment for relapsed or refractory acute myeloid leukaemia (AML)[25].

5. Stem cell transplantation: Allogeneic hematopoietic stem cell transplantation (HSCT) is a potential curative treatment for acute myeloid leukaemia (AML), particularly suitable for younger or high-risk disease variants. The process involves substituting the patient's diseased bone marrow with healthy stem cells from a compatible donor[27].

6. Venetoclax-based therapy: Venetoclax, a drug that inhibits BCL-2, marks noteworthy progress in treating acute myeloid leukaemia (AML), particularly for older patients or those not candidates for aggressive chemotherapy. When combined with hypomethylating agents or low-dose cytarabine, venetoclax has shown encouraging results in improving overall survival and remission rates[26].

7. Maintenance therapy: Continuing treatment with targeted therapies like midostaurin (for patients with FLT3-mutated AML) or hypomethylating agents (for older individuals) can help reduce the chance of recurrence following the initial treatment. These therapies aim to prolong remission and improve long-term survival[16].

Therapeutic approach for CML

The approaches for managing Chronic Myeloid Leukaemia (CML) have significantly improved with the emergence of targeted therapies. Here are the main strategies:

Tyrosine Kinase Inhibitors (TKIs)

First-Line Therapy: TKIs targeting the BCR-ABL protein are the standard treatment for CML.

Imatinib (Gleevec): The first TKI approved for CML and still widely used.

Dasatinib (Sprycel): A second-generation TKI with a different side effect profile and potency.

Nilotinib (Tasigna): Another second-generation TKI, often used for patients resistant or intolerant to imatinib.

Bosutinib (Bosulif): Approved for patients resistant to other TKIs.

Third-line therapy: Ponatinib (Iclusig) is used for patients with the T315I mutation or those resistant to other TKIs[27,28].

1. Stem Cell Transplantation (SCT):

This treatment option is considered for patients unresponsive to TKIs or in an advanced phase of the disease. Although it is now less frequently utilised because of the effectiveness of TKIs, it still represents a possible cure for CML[29].

2. Interferon-alpha

Originally used before TKIs were introduced as standard treatment; it is now primarily reserved for rare situations, like pregnant patients for whom TKIs are not recommended[30].

3. Monitoring and treatment adjustments

Regularly monitoring BCR-ABL transcript levels using qPCR to assess response to treatment. TKI doses may

be modified depending on side effects and patient response. Patients can switch to a different TKI if there is resistance or intolerance[31].

4. Treatment-Free Remission (TFR)

Criteria: Patients who achieve a deep and sustained molecular response (MR4 or MR4.5) for at least 2 years may consider discontinuing TKIs under close medical supervision.

Monitoring: Intensive monitoring post-discontinuation to detect any relapse early.

Managing side effects: Addressing side effects of TKIs such as fatigue, oedema, and gastrointestinal issues.

Addressing comorbidities: Ensuring comprehensive care, including cardiovascular health, is important, given the potential side effects of some TKIs on heart health[32].

5. Experimental therapies and clinical trials

Research continually explores new TKIs and their combinations with other treatments to enhance results and tackle resistance. Innovative strategies are being investigated to leverage the immune system against CML through immunotherapy and vaccines. These methods are adapted to suit each patient's specific needs, considering the stage of the disease, the mutation profile, and how they responded to the initial treatment[7].

Future Perspective of AML and CML

1. Personalized medicine and targeted therapies: The application of precision medicine in acute myeloid leukaemia (AML) is expected to continue its expansion. A growing number of targeted therapies are being developed through comprehensive genomic profiling focusing on specific molecular alterations, including FLT3, IDH1/IDH2, and TP53 mutations[24]. In the future, identifying additional genetic mutations will allow for increasingly tailored therapeutic regimens aimed at maximising efficacy while minimising toxicity. With advancements in genomic profiling, the future of chronic myeloid leukaemia (CML) treatment will likely embrace more personalised approaches. Precision medicine strategies will concentrate on customising therapy based on individual genetic and molecular characteristics.

2. Immunotherapy advancements and vaccine development in AML and CML: Immunotherapy represents a promising area of research in acute myeloid leukaemia (AML), with various innovative strategies under exploration. These approaches include chimeric antigen receptor (CAR) T-cell therapy and bispecific antibodies targeting AML-specific antigens such as CD33 and CD123. Ongoing research is focused on improving the effectiveness of these treatments while addressing the immune evasion mechanisms that arise in AML[17]. Furthermore, creating vaccines against the BCR-ABL1 fusion protein and other leukaemia-specific antigens may offer an immune-oriented method for eradicating residual leukemic cells and preventing relapse in chronic myeloid leukaemia (CML). Early-stage clinical trials examine the potential of immune checkpoint inhibitors and leukaemia vaccines to improve treatment-free remission (TFR) rates and reduce the likelihood of relapse[21].

3. Next-Generation Sequencing (NGS) for risk stratification: Next-generation sequencing (NGS) will increasingly be vital in the diagnosis and outcome prediction for acute myeloid leukaemia (AML) and chronic myeloid leukaemia (CML). NGS-based assays facilitate the identification of co-occurring mutations and clonal evolution, allowing for more accurate patient stratification into risk categories and the determination of the most suitable therapies[33].

4. Stem cell transplantation and maintenance therapies: Allogeneic hematopoietic stem cell transplantation (HSCT) continues to be a potentially curative option for acute myeloid leukaemia (AML) and chronic myeloid leukaemia (CML), particularly for high-risk patients. Future efforts are aimed at enhancing transplant outcomes through improved conditioning regimens, minimising graft-versus-host disease (GVHD), and investigating post-transplant maintenance therapies, such as FLT3 inhibitors and hypomethylating agents, to prevent relapse[17,34].

5. Epigenetic therapies: Epigenetic changes, such as DNA methylation and histone modifications, are pivotal in the pathogenesis of acute myeloid leukaemia (AML). Research on hypomethylating agents and various epigenetic therapies is gaining momentum, aiming to enhance response rates and response durability in newly diagnosed and relapsed/refractory AML patients[17].

6. Venetoclax combinations: Venetoclax, a BCL-2 inhibitor, has demonstrated considerable potential in treating older patients and those ineligible for intensive chemotherapy. Ongoing research is aimed at optimising combinations of venetoclax with other agents, including hypomethylating agents, FLT3 inhibitors, and IDH inhibitors, to improve response rates and prolong survival in both frontline and relapsed scenarios[35].

7. Minimal Residual Disease (MRD) monitoring: MRD monitoring is anticipated to become a standard practice in the management of AML. Utilising highly sensitive assays such as PCR and flow cytometry, clinicians will be equipped to detect minimal levels of disease and intervene sooner in cases of potential relapse. Future considerations involve the development of MRD-guided adjustments to therapy aimed at prolonging remission and enhancing survival rates[16].

CONCLUSIONS

CML and AML pose unique but notable obstacles in the field of blood cancers. Both conditions result from the cancerous conversion of myeloid cells and are distinguished by distinct pathophysiological characteristics that require specialized diagnostic and treatment strategies. The diagnosis of CML mainly depends on detecting the BCR-ABL1 fusion gene, which is a defining feature of the condition. This genetic marker verifies the diagnosis and guides the treatment plan. Conversely, AML is characterized by its genetic and clinical variability, making diagnosis challenging and necessitating a more personalized treatment strategy. Conventional treatment usually includes cytotoxic chemotherapy, frequently succeeded

by allogeneic stem cell transplantation for patients who qualify.

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AUTHOR'S CONTRIBUTION

Obeagu EI: conceived the idea, writing the manuscript. **Kwaifa IK:** editing, critical review. **Abubakar MK:** literature survey. **Rabiu U:** editing. **Ayodeji OA:** literature survey. **Obeagu EI:** formal analysis, data processing. Final manuscript is checked and approved by both authors.

DATA AVAILABILITY

Data will be made available on request.

CONFLICT OF INTEREST

None to declare.

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