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Therapeutic Response Monitoring in Chronic-Phase Chronic Myeloid Leukemia Treated with Tyrosine Kinase Inhibitors: A Narrative Review

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ABSTRACT

Background: *Therapeutic response monitoring is essential in chronic-phase chronic myeloid leukemia (CML) treated with tyrosine kinase inhibitors (TKIs), as it guides the evaluation of treatment efficacy, detection of inadequate response, and individualized treatment decisions.*
Methods: *This narrative review summarizes the current approaches to response monitoring in chronic phase CML treated with TKIs, focusing on hematologic, cytogenetic, and molecular assessments. Particular attention is given to BCR::ABL1 monitoring, response milestones, mutation-guided treatment decisions, and implementation challenges in resource-limited settings, including Indonesia.*
Results: *Hematologic response is an early and accessible indicator of treatment activity, whereas cytogenetic assessment is useful for evaluating Philadelphia chromosome-positive cells and detecting additional chromosomal abnormalities. Molecular monitoring using quantitative BCR::ABL1 transcript measurement on the International Scale is the most sensitive method for assessing residual disease, defining treatment milestones, identifying warning or unfavorable responses, and selecting patients who may be considered for treatment-free remission (TFR). However, access to standardized molecular testing remains limited in many low- and middle-income countries because of costs, geographic disparities, laboratory infrastructure constraints, and health-system barriers. Practical strategies include prioritizing molecular testing at key milestones, strengthening referral laboratory networks, using automated platforms where feasible, supporting patient adherence, and developing clinical registries.*

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Conclusion: *Integrated resource-adapted response monitoring is crucial for optimizing TKI therapy in chronic-phase CML and reducing disparities in long-term outcomes.*

Keywords: *Leukemia myelogenous; chronic; BCR-ABL positive; tyrosine kinase inhibitors; treatment outcome; resource-limited settings*

INTRODUCTION

Chronic myeloid leukemia (CML) is classified as a myeloproliferative neoplasm, characterized by the presence of the Philadelphia (Ph) chromosome. This chromosome arises due to the reciprocal translocation t(9;22)(q34;q11), leading to the formation of the BCR::ABL1 fusion gene. The resulting BCR::ABL1 oncoprotein has constitutive tyrosine kinase activity that promotes uncontrolled myeloid proliferation, inhibits apoptosis, and contributes to genomic instability.[1] Most patients in developed countries are diagnosed during the chronic phase (CP), an indolent stage characterized by the expansion of mature granulocytes and a substantially better prognosis than accelerated phase or blast phase disease.[2]

The management of chronic-phase CML has been revolutionized by the advent of tyrosine kinase inhibitors (TKIs). Imatinib established targeted BCR::ABL1 inhibition as the therapeutic standard, and subsequent generations of TKIs have expanded treatment options by improving potency, overcoming selected resistance mechanisms, and enabling deeper molecular responses in some patients.[1] Consequently, many patients with chronic-phase CML can achieve long-term disease control, provided that treatment response is monitored appropriately and therapy is adjusted when necessary.

Therapeutic response monitoring is essential because clinical improvement alone is insufficient to determine the depth and durability of response. Monitoring in CML is generally categorized into hematologic, cytogenetic, and molecular assessments, each providing different levels of sensitivity and clinical information.[3] Normalization of blood counts and the resolution of clinical symptoms characterize a complete hematologic response, while a complete cytogenetic response is marked by the absence of detectable Ph-positive metaphases. Molecular monitoring using quantitative real-time PCR for BCR::ABL1 transcripts provides the greatest sensitivity and allows clinicians to assess residual disease, evaluate response milestones, detect loss of response, and identify patients who may be considered for treatment-free remission.[2,4]

Although international guidelines provide clear recommendations, the availability of standardized molecular monitoring is still inconsistent in various healthcare environments. In low- and middle-income countries (LMICs), including Indonesia, routine BCR::ABL1 testing is often constrained by cost, limited laboratory infrastructure, geographic barriers, and inconsistent availability outside major referral centers.[5,6] These limitations may delay the recognition of warning or unfavorable responses and reduce opportunities for timely treatment optimization. In the Indonesian context, disparities between national referral centers and regional facilities further contribute to variations in monitoring frequency and therapeutic decision-making.[5]

This narrative review summarizes current approaches to therapeutic response monitoring in patients with chronic-phase CML treated with TKIs, spanning the continuum from newly diagnosed patients to those requiring mutation-guided TKI switching and management of treatment resistance or intolerance. It discusses the clinical roles of hematologic, cytogenetic, and molecular assessments, with an emphasis on response milestones and their implications for clinical decision-making. This review emphasizes the difficulties encountered when applying guideline-based monitoring in settings with limited resources, especially in Indonesia. It suggests practical approaches to enhance access, refine treatment choices, and minimize disparities in long-term results.

METHODS

A narrative literature review was conducted using PubMed, Google Scholar, and relevant clinical practice guidelines, including recommendations from the National Comprehensive Cancer Network (NCCN) and the European LeukemiaNet (ELN). The databases were last searched on March 31, 2026. Search terms included combinations of "chronic myeloid leukemia," "tyrosine kinase inhibitor," "BCR::ABL1 monitoring," "molecular response," "treatment-free remission," "resource-limited settings," and "Indonesia."

Articles were included if they were peer-reviewed clinical guidelines, randomized controlled trials, cohort studies, or review articles addressing therapeutic response monitoring in chronic-phase CML, published within the last decade; older landmark studies were retained when historically or clinically significant. Articles were excluded if they were case reports, conference abstracts without peer-reviewed full text, non–English-language publications, or studies not directly addressing CML response monitoring or TKI therapy.

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LITERATURE REVIEW

Chronic-Phase CML and the Evolution of TKI Therapy

According to the World Health Organization (WHO), chronic-phase CML is identified by having less than 10% blasts in the peripheral blood and bone marrow, lacking features of the accelerated phase, and containing the BCR::ABL1 fusion gene. The pathophysiology of CP-CML is centered on the constitutively active BCR::ABL1 kinase, which drives the expansion of the myeloid compartment. The therapeutic evolution of CML began with the first-generation tyrosine kinase inhibitor (1G-TKI), imatinib, which selectively inhibits the ATP-binding site of the BCR::ABL1 protein.[1]

Subsequently, second-generation TKIs (2G-TKIs), including nilotinib, dasatinib, and bosutinib, were developed to provide higher potency and overcome some forms of imatinib resistance.[1,7-9] Dasatinib and nilotinib were initially approved for second-line use but were later moved to the frontline setting based on their ability to induce faster and deeper molecular responses than imatinib.[4] Bosutinib has also been integrated into the frontline therapeutic armamentarium. Third-generation TKIs, most notably ponatinib, were designed to target the T315I gatekeeper mutation, which is resistant to all 1G and 2G agents.[1]

Recently, asciminib has emerged as a novel therapeutic option. Unlike traditional TKIs that are ATP-competitive, asciminib acts as a STAMP (specifically targeting the ABL myristoyl pocket) inhibitor.[10] This allosteric mechanism provides a different resistance profile and potentially fewer off-target side effects compared to traditional TKIs.[11]

Table 1. Generations of tyrosine kinase inhibitors used in chronic-phase chronic myeloid leukemia and their clinical characteristics[1,10]

TKI class/generation	Representative agents	Common clinical setting	Key characteristics
First-generation TKI	Imatinib	Frontline therapy	Historical standard of care; established long-term safety profile; widely available generic formulations
Second-generation TKIs	Dasatinib, nilotinib, bosutinib	Frontline or later-line therapy	Greater potency than imatinib and faster molecular responses; drug-specific adverse event profiles, including pleural effusion with dasatinib, vascular or metabolic events with nilotinib, and gastrointestinal or hepatic toxicity with bosutinib

TKI class/generation	Representative agents	Common clinical setting	Key characteristics
Third-generation TKI	Ponatinib	Resistant disease, particularly T315I mutation or multiple TKI failure	Active against BCR::ABL1 T315I mutation; requires careful cardiovascular risk assessment and monitoring
STAMP inhibitor	Asciminib	Later-line therapy; emerging frontline option where approved or available	Allosteric BCR::ABL1 inhibition through myristoyl pocket binding; favourable tolerability profile and higher molecular response rates reported in recent frontline comparative trials

BCR::ABL1, breakpoint cluster region-Abelson 1 fusion gene; STAMP, specifically targeting the ABL myristoyl pocket; T315I, threonine-to-isoleucine substitution at position 315 of BCR::ABL1; TKI, tyrosine kinase inhibitor.

Framework of Therapeutic Response Monitoring

The overarching goal of response monitoring is to ensure that the patient is reaching predefined molecular and cytogenetic milestones that correlate with a high probability of survival and a low risk of progression. Monitoring serves to guide clinicians in determining when to continue a current TKI, when to evaluate for non-compliance, and when to switch to an alternative agent.[4]

Responses are sequential: hematologic remission occurs first (usually within weeks), followed by cytogenetic remission (months), and finally molecular remission (months to years). The initial response, particularly at the 3-month mark, is critical; patients who achieve an early molecular response (EMR; BCR::ABL1 ≤10% IS) have significantly better long-term survival and deep response rates compared to those who do not.[4] Monitoring also facilitates the identification of selected patients who may be eligible for treatment-free remission, although long-term disease control, tolerability, and prevention of progression remain the primary therapeutic priorities for many patients.[12]

Hematologic response

Hematologic monitoring is the initial and most basic level of response assessment. It requires a complete blood count (CBC) with differential and physical examination, focusing on signs of splenomegaly and hepatomegaly.[4] A complete hematologic response (CHR) is defined by the return of peripheral blood counts to normal levels. The criteria include a white blood cell count of less than 10×10^9/L and a platelet count of fewer than 450×10^9/L. Moreover, the peripheral blood differential must not reveal any immature granulocytes, including blasts, promyelocytes, or myelocytes. Additional

criteria include basophils <5% in peripheral blood and the absence of palpable splenomegaly on physical examination.[4,12]

The primary advantage of hematologic monitoring is its widespread availability and low cost, which is particularly relevant in the early stages of treatment in LMICs. However, hematologic response is not sensitive enough to detect low levels of residual disease or to provide prognostic information regarding long-term molecular stability. Although almost all patients achieve CHR within the first three months of TKI therapy, this milestone does not guarantee that the Ph+ clone has been sufficiently suppressed.[4]

Cytogenetic response

Cytogenetic monitoring focuses on the reduction of Ph+ cells in the bone marrow. Although molecular monitoring has largely superseded in high-resource settings, cytogenetics remains essential for detecting clonal evolution.[4] G-banded karyotyping of at least 20 bone marrow metaphases is the standard method for cytogenetic assessment, which enables the estimation of the proportion of Philadelphia chromosome-positive (Ph+) cells. The classification of cytogenetic response (CyR) is based on the proportion of Ph+ metaphases: no response is indicated by more than 95%, minimal response ranges from 66% to 95%, minor response is between 36% and 65%, partial cytogenetic response (PCyR) falls within 1% to 35%, and complete cytogenetic response (CCyR) is defined as 0%.

A major cytogenetic response (MCyR) includes both CCyR and PCyR (0%–35% Ph+). CCyR is a major milestone that is historically correlated with a drastic reduction in the risk of progression.[3] In the current monitoring algorithm, CCyR is generally expected by 6–12 months.[4] Fluorescence in situ hybridization (FISH) on peripheral blood or bone marrow is an alternative when metaphase analysis is not feasible; however, it cannot detect additional chromosomal abnormalities (ACAs).[2]

Molecular response

Quantitative real-time PCR (qPCR) serves as the "gold standard" in the management of CML, enabling the identification of residual disease with a sensitivity reaching 4–5 log reductions from the baseline.[2] Reporting is standardized using the International Scale (IS), where a value of 100% IS represents the standardized baseline in untreated patients. Responses are calculated as the ratio of BCR::ABL1 transcripts to a control gene (usually ABL1).[2]

Molecular response milestones are central to therapeutic response monitoring in chronic-phase CML. Early molecular response (EMR) is generally defined as a BCR::ABL1 transcript level of ≤10%

on the International Scale (IS) at 3 and 6 months, and failure to achieve this milestone is associated with an increased risk of poor clinical outcomes.[4] Major molecular response (MMR), also referred to as MR3, is defined as BCR::ABL1 ≤0.1% IS; achieving MMR by 12 months is considered an optimal response and is associated with durable long-term disease control. Deep molecular response (DMR) includes MR4, defined as BCR::ABL1 ≤0.01% IS, and MR4.5, defined as BCR::ABL1 ≤0.0032% IS. Sustained DMR, commonly defined as MR4 or deeper for at least 2 years, is an important prerequisite for considering treatment-free remission (TFR).[2,13] Standardization across laboratories remains a significant challenge. In LMICs, the requirement for frequent testing (every 3 months) is often not met owing to logistical and financial constraints.[14] The cost of reagents and the need for specialized molecular biology expertise further restrict access.

An integrative approach ensures that clinicians use the most appropriate tool at each stage of therapy. Hematologic monitoring provides the first signal of drug activity. Cytogenetics acts as a "bridge," confirming the elimination of Ph+ cells and screening for ACAs that may suggest early transformation or resistance. Molecular monitoring then provides the long-term precision needed to manage therapy, especially when aiming for TFR.[2] The current consensus emphasizes that once CCyR (or ≤1% IS) is achieved, monitoring should primarily focus on qPCR. Bone marrow assessments are then reserved for cases of therapeutic failure or when the patient develops unexplained cytopenias, which may indicate clonal evolution in Ph-negative cells.[4]

Response Milestones and Clinical Decision-Making

Therapeutic decisions are based on the achievement of specific milestones. The ELN 2025 definitions are summarized in Table 2.

Table 2. Treatment response milestones for tyrosine kinase inhibitor therapy in chronic-phase chronic myeloid leukemia[15]

Time point	Favorable	Warning	Unfavorable
Baseline	N/A	High-risk ACA; high-risk ELTS score	N/A
3 months	BCR::ABL1^{IS} ≤10%	BCR::ABL1^{IS} >10%	BCR::ABL1^{IS} >10% if confirmed within 1–3 months
6 months	BCR::ABL1^{IS} ≤1%	BCR::ABL1^{IS} >1%–10%	BCR::ABL1^{IS} >10%

Time point	Favorable	Warning	Unfavorable
12 months	BCR::ABL1^{IS} ≤0.1%	BCR::ABL1^{IS} >0.1%–1%	BCR::ABL1^{IS} >1%
At any time	BCR::ABL1^{IS} ≤0.1%	BCR::ABL1^{IS} >0.1%–1% or loss of MMR	Loss of previous response; resistant BCR::ABL1 mutations; high-risk ACA

ACA, additional chromosomal abnormalities in Philadelphia chromosome-positive cells; BCR::ABL1^{IS}, BCR::ABL1 transcript level reported on the International Scale; ELTS, EUTOS long-term survival score; MMR, major molecular response; N/A, not applicable.

In patients with a favorable response, continuation of the current TKI with routine molecular monitoring is generally appropriate. A warning response should prompt closer follow-up and a comprehensive reassessment of potentially modifiable factors, including adherence, drug–drug interactions, dose interruptions or reductions, treatment intolerance, and the kinetics of BCR::ABL1 decline. In contrast, an unfavorable response should be confirmed and interpreted in the context of previous molecular results and patient-related factors; when persistent or accompanied by rising BCR::ABL1 levels without an identifiable explanation, cytogenetic evaluation, BCR::ABL1 kinase domain mutation analysis, and a switch to a more appropriate TKI are preferred.[15]

Mutation-Guided TKI Selection and Management of Intolerance

BCR::ABL1 kinase domain mutation analysis should be considered when an unfavorable response is confirmed, when BCR::ABL1 transcript levels increase without an identifiable explanation, or when resistance is suspected. Current recommendations favor mutation-guided TKI selection rather than an automatic switch based on a single molecular result. For example, the T315I mutation supports the use of ponatinib or asciminib, whereas V299L may favor nilotinib, ponatinib, or asciminib, and F317L/V/I/C may favor nilotinib, bosutinib, ponatinib, or asciminib. When available, validated cDNA-based next-generation sequencing is preferred for BCR::ABL1 mutation screening, although Sanger sequencing remains acceptable when next-generation sequencing is not accessible. In cases of intolerance, treatment modification should be individualized according to the adverse-event profile, comorbidities, prior TKI exposure, and response status; dose adjustment or switching to an alternative TKI with a more suitable toxicity profile may be considered.[1]

Monitoring Challenges in Resource-Limited Settings

In low- and middle-income countries (LMICs), the implementation of the recommended standard of care for response monitoring in chronic-phase CML remains challenging because of financial, geographic, infrastructural, and health system barriers. Molecular monitoring using polymerase chain reaction (PCR) is costly, and one estimate across 60 LMICs suggested that the funding gap for PCR monitoring exceeds US$29 million, with cartridge procurement accounting for approximately 86% of the total cost.[6] Geographic disparities further limit access, as most molecular laboratories are concentrated in metropolitan areas, while patients from remote Indonesian islands may face substantial travel expenses and logistical delays in transporting blood samples to referral centers. These challenges are compounded by limited laboratory infrastructure, a shortage of certified molecular facilities, and insufficient trained personnel to perform and interpret complex molecular assays. Although imatinib and nilotinib are covered under Indonesia's national health insurance system, access to molecular monitoring at the recommended frequency is not always guaranteed.[5]

A Resource-Adapted Monitoring Strategy

A resource-adapted monitoring strategy is needed based on current response milestones and the documented barriers to molecular monitoring in LMICs. In settings where routine 3-monthly BCR::ABL1 monitoring cannot be consistently implemented, a tiered monitoring model may provide a pragmatic compromise between guideline-based care and local resource constraints. At a minimum, regular clinical assessment and complete blood count monitoring should be maintained, while molecular testing should be prioritized at clinically important decision points, particularly at 3, 6, and 12 months after TKI initiation and whenever a loss of response is suspected.[2,15] This approach is particularly relevant in LMICs, where molecular monitoring is often limited by cost, geographic barriers, laboratory capacity, and health system constraints.[5,6,14] Patients with warning or unfavorable responses should be referred, whenever feasible, to centers with access to standardized BCR::ABL1 testing, cytogenetic evaluation, and BCR::ABL1 kinase domain mutation analysis to guide further treatment decisions.[15]

Practical Strategies for Monitoring in Low- and Middle-Income Countries

Improving therapeutic response monitoring in resource-limited settings requires an integrated approach that combines prioritization of testing, laboratory strengthening, patient-centered interventions, and health system planning. In settings where frequent molecular monitoring is

constrained by cost or infrastructure, BCR::ABL1 testing may be prioritized at clinically meaningful milestones, including 3, 6, and 12 months after treatment initiation. The implementation of automated cartridge-based assays, such as GeneXpert BCR::ABL1 Ultra, may improve assay standardization and reduce the technical complexity associated with conventional quantitative PCR.[6] Furthermore, referral laboratory networks and collaborative diagnostic access programs may help extend molecular testing to underserved areas.[6] These laboratory-based strategies should be accompanied by patient education and adherence interventions, particularly in case where socioeconomic factors affect long-term treatment persistence.[16] At the policy level, hospital-based and national registries may provide essential real-world data to identify service gaps and inform resource allocation.

Future Directions

Future directions in CML monitoring are increasingly focused on improving assay sensitivity, accessibility, and integration into long-term care. Digital PCR (dPCR) has emerged as a promising technology because it enables absolute quantification and may offer higher sensitivity than conventional quantitative PCR, particularly for detecting deep molecular response (DMR).[17] This approach may be useful in refining treatment-free remission (TFR) eligibility, although proposed predictive thresholds, such as 0.0023% on the International Scale, still require broader validation before routine clinical adoption. Beyond laboratory innovation, telemedicine may support long-term CML follow-up, particularly in geographically dispersed countries such as Indonesia, where remote consultation can reduce the travel burden for patients who primarily require discussion of laboratory results and treatment adherence. Artificial intelligence (AI)-based tools remain investigational and should be viewed as supportive technologies rather than replacements for validated laboratory and clinical assessments.[18] In parallel, continued efforts to standardize DMR reporting, including MR4 and MR4.5, remain essential to ensure consistent interpretation of molecular results and safe consideration of TFR.[2]

CONCLUSION

In conclusion, therapeutic response monitoring in chronic-phase CML relies on an integrated framework of hematologic, cytogenetic, and molecular assessments that together guide treatment continuation, intensification, or switching across the course of therapy. Quantitative BCR::ABL1 monitoring on the International Scale remains central to defining treatment milestones and identifying candidates for treatment-free remission, while mutation-guided TKI selection supports management of

resistant or intolerant disease. In resource-limited settings, including Indonesia, cost, infrastructure, and geographic barriers continue to constrain standardized molecular testing. A resource-adapted strategy that prioritizes testing at key milestones, strengthens referral laboratory networks, and supports patient adherence is therefore essential to optimize TKI therapy and reduce disparities in long-term outcomes for patients with chronic-phase CML.

Conflicts of Interest

The authors declare that they have no competing interests.

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