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Research Article

Navigating the Post-Targeted Therapy Era in Chronic Myeloid Leukemia: An In-Depth Look at the Landscape via Incidence-Base Mortality

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Abstract

Background: Chronic myeloid leukemia (CML) is a leading cause of leukemia in North America and around the world. According to the American Cancer Society, one in every 526 Americans will acquire CML in their lifetime. CML accounts for around 15% of all new instances of leukemia.

We examined the incidence-based mortality rates (IBM rates) of CML by gender, race, rural/Urban residency, age groups and income level in adult populations in the United States from 2000 to 2019 using the SEER.gov database.

Methods: Incidence-based Mortality rates of CML were calculated using IBM SEER Research Data, 17 Registries, Nov 2021 Sub (2000-2019), which covers the largest geographic area and approximately 26.5% of the US population (based on 2020 Census data). SEER*Stat 8.4.1 was used to calculate age-adjusted rates across age groups, races, and genders. We utilized the National Cancer Institute's Join-point program, version 5.0.2, to generate log-linear temporal trends on an annual basis.

Results: IBM rates for CML improved for the studied age groups, income levels, area of residence, race/ethnicity, and sex/gender.

Though there was synonymous improvement across all strata, there were still differences in rates within the groups studied.

Conclusion: Our findings revealed that IBM rates for CML improved across the board, regardless of age group, income level, place of residence, race/ethnicity, or gender. Interestingly, the data strongly suggests a big change in IBM rates around 2002, coinciding well with FDA approval of Targeted therapy in 2001.

Keywords

Chronic Myeloid Leukemia (CML), Metastatic, Incidence-based mortality, Screening, Disparities, Tyrodine Kinase Inhibitors (TKIs), SEER database.

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Introduction

Chronic myeloid leukemia (CML) is mainly defined as a myelogenous neoplasm characterized by the presence of the Philadelphia chromosome (t(9;22)(q34;q11.2)). This balanced genetic translocation creates the BCR-ABL1 fusion oncogene and its associated oncoproteins. This genetic mutation results in the proliferation of myeloid cells, which turns into an acute form of leukemia. CML makes up about 15% of all new cases of leukemia in adults. It occurs in 1-2 out of every 100,000 people. Our knowledge and treatment of hematologic cancers have considerably changed because of CML. Its pathophysiology was described in previous literature as constantly cycling between the chronic phase and blast crisis. However, since the development of targeted therapies, especially tyrosine kinase inhibitors (TKIs), it has become a manageable chronic disease for many patients [1]. The significant difference in the management of CML with the use of TKIs has improved the life expectancy of many patients. For the most part, individuals with CML no longer experience debilitating symptoms and can resume their everyday lives as usual.

Even though the incidence of new cases of CML has remained constant, the number of people with CML has increased. The general increase in the life expectancy of human beings may play a role in the increased prevalence of the condition. The improvements in the management of CML, such as with TKIs, which block the BCR-ABL1 tyrosine kinase enzyme, also help increase prevalence of CML.

Still, this generally positive picture is broken by the inequalities in the frequency, survival, and death rates between different groups. It is still a major concern that some cases of CML progress to the blast phase and that some individuals with the gene known as T315I do not respond to TKIs. The primary objective of the current CML project is to discover effective ways to address these issues, enhance the prognosis for later stages, and increase the number of people who can live their entire lives without requiring any treatment [2].

Since the advent of TKIs, the landscape of treating CML has changed significantly. To understand these changes better, our study aims to provide a clear picture of CML in the post-TKI era. The results will be compared to progress made in other types of cancer, to understand how much progress we have made and what more needs to be done in oncology.

Methods

Databases

For the calculation of incidence-based mortality rates of CML, we extracted data from the Surveillance Research Program (SEER) of the National Cancer Institute, specifically, the Incidence-Based Mortality SEER Research Data, 17 Registries, Nov 2021 Sub (2000-2019), which provides the broadest geographic coverage for incidence-based mortality rates representing approximately 26.5% of the US population based on the 2020 Census. The database classified CML and other cancers' histology and topography using the third version of the International Classifications of Diseases for Oncology (ICD-O-3/WHO 2008) [3].

For this investigation, we used data from patients with CML diagnosis, defined by the same classification. This study did not include distant/metastatic computation because CML is a liquid tumor.

We calculated the annual age-adjusted incidence-based mortality rates (AAIBMR) of CML per 100,000 person-years and stratified it accordingly. Sex (determined at birth) was classified as male or female. The races/ethnicities included in this study were non-Hispanic White (NHW), non-Hispanic Black (NHB), non-Hispanic American Indians/Alaskan Natives (NHI/AN), non-Hispanic Asians/Pacific Islanders (NHAPI), and Hispanics. This information was from the 'race and origin recode' database. Residence types were described as either metropolitan or non-metropolitan. The information was derived from the 'county attributes/rural-urban continuum' database [4]. The age adjustment for cancer incidence rates in SEER was calculated using the 2000 US standard population. Ethical approval was granted by the McLaren Hospital, Flint Institutional Review Board.

Statistical Analysis

Trends in CML AAIBMR by sex (male and female), race/ethnicity, and residence (metropolitan, non-metropolitan) were examined using the joinpoint regression models.

To identify when and how frequently the annual percentage change (APC) changes (number of joinpoints), the joinpoint model employs two fit indices: Akaike information (AIC) and Bayesian information criterion (BIC). On a log scale, APC implies a steady percentage change per year. The APC of two neighboring joinpoints is calculated as:

$$APC=(e-1)\times 100$$

The coefficient (β) represents the estimated average change (slope) determined using linear regression.

$$\log y = \alpha+\beta w$$

where y is the age-adjusted rate of CML, and w is the number of years between two adjacent joinpoints. β represents the fixed influence of time on the age-adjusted incidence of metastatic lung cancer rates. We set no limit on the number of joinpoints.

We used the joinpoint trend parallelism test 22 to examine CML trends by sex (male versus female) and residency (metropolitan versus non-metropolitan).

This study utilized the SEER*Stat software version 8.4.1, and the National Cancer Institute Joinpoint regression program (Joinpoint Regression Program, Version 5.0.2.0) to generate log-linear time trends.

Results

Patient Characteristics

We examined data from 13,429 CML patients; about 60% (n= 8018) were males and 40% (n= 5411) were females. The bulk (85%) came from metropolitan counties, with approximately 15% from non-metropolitan counties. From 2000 to 2019, CML's total incidence-based mortality rate was 2.96 (95% confidence interval 2.91-3.01) per 100,000 person-years for both males and females.

Table 1 is a summary of the study population characteristics.

Table 1: Study population characteristics with CML diagnosis 2000-2019.

	Total CML Case reported in seer Count (%)	IBM rates
Sex, n (%)		
Male	8,018 (60)	4.28
Female	5,411 (40)	2.02
Race/Ethnicity, n (%)		
Non-Hispanic White	10,537 (79)	3.19
Non-Hispanic Black	1,019 (7.6)	2.78
Non-Hispanic American Indian/ Alaska native	63 (0.4)	2.2
Non-Hispanic Asian/Pacific Islander	698 (5)	1.73
Hispanics (All Races)	1,092 (8)	2.3
Residence, n (%)		
Metropolitan	11,394 (85)	2.88
Non-metropolitan	2,020 (15)	3.47
Household Income, n (%)		
<$35,000	243 (5)	1.52
$75,000+	4,232 (95)	1.13
Age groups, n (%)		
55-59	639 (14)	0.65
60-64	904 (19)	1.12
65-69	1,387 (29)	2.18
70-74	1,798 (38)	3.66

IBM stratified by Sex

From 2000 to 2019, men had a higher incidence-based mortality (IBM) of CML than women. The total IBM rate in men was 4.28 (95% CI 4.19 - 4.38) per 100,000 person-years, whereas in women it was 2.02 (95% CI 1.97-2.08) per 100,000 person-years. The IBM rate of CML in men increased at an annual percent change (APC) of 22.8% from 2000 to 2002, then decreased to 2.69% from 2002 to 2019. In contrast, women experienced an initial increase in IBM at an APC of 46.1% from 2000 to 2002, followed by a change in annual percent change of 1.79% from 2002 to 2019.

The average APC (AAPC) for men and women was 4.64% (95% CI 5.38-6.03) and 5.74% (95% CI 4.19-8.62) per 100,000 person-years, respectively.

Figure 1 graphically displays these changes.

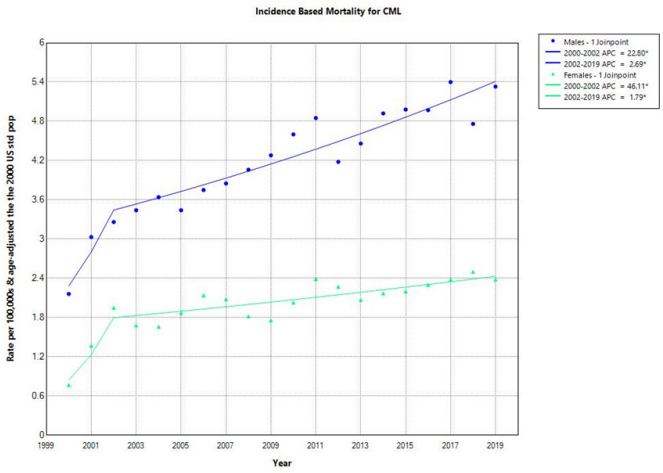

Figure 1: IBM for CML 2000-2019 by Sex/Gender.

The joinpoint parallelism test revealed that the overall patterns in CML IBM between men and women did not reject parallelism (P = 0.07, significance level of 0.05), implying that both men and women experienced similar alterations in IBM for CML.

IBM Trend stratified by Race/Ethnicity

Non-Hispanic White (NHW), non-Hispanic Black (NHB), non-Hispanic American Indians/Alaskan Natives (NHI/AN), non-Hispanic Asians/Pacific Islanders (NHAPI), and Hispanics

NHWs experienced an increase in IBM for CML and APC of 33.6% (95% CI 19.7 - 48.75) from 2000-2002, with an alteration in the rate of change to an APC of 2.83% (95% CI 2.36 - 3.29) between 2002 to 2019. NHB did not experience any joint points throughout the study from 2000-2019 and maintained an APC of 2.18% (95% CI 0.77 - 3.96). NHAPI experienced a joint point in 2011. Their APC was 6.20% (95% CI 2.95 - 29.79) from 2000-2011 and 1.82% (95% CI -14.75 - 1.95) between 2011 and 2019. There was insufficient data to characterize the trend for NHI/AN. Hispanics experienced an increase in IBM for CML with an APC of 12.81% (95% CI 4.93 - 49.32) between 2000-2006 and an APC of 1.10% (95% CI -2.93 - 2.79) between 2006 and 2019.

The trends are graphically depicted in Figure 2.

The AAPC for the IBM for CML was 5.70%, 2.18%, 2.74%, and 4.66% for NHW, NHB, NHAPI, and Hispanic of all races, respectively.

IBM Trend Stratified by Residence

From 2000 to 2019, non-metropolitan residents (NMR) had a higher IBM for CML than their metropolitan counterparts (MR). The total IBM rate in NMR was 3.47 (95% CI 3.32-3.62) per 100,000 person-years, compared to 2.88 (95% CI 2.83-2.93) per 100,000 person-years in the MR. The IBM of CML in the NMR increased at an APC of 35.36% from 2000 to 2002, with a reduction in the rate

of increase to 3.15% from 2002 to 2019. The IBM of CML increased at an APC of 31.42% in MR from 2000 to 2002. However, the rate of increase decreased to 2.35% from 2002 to 2019.

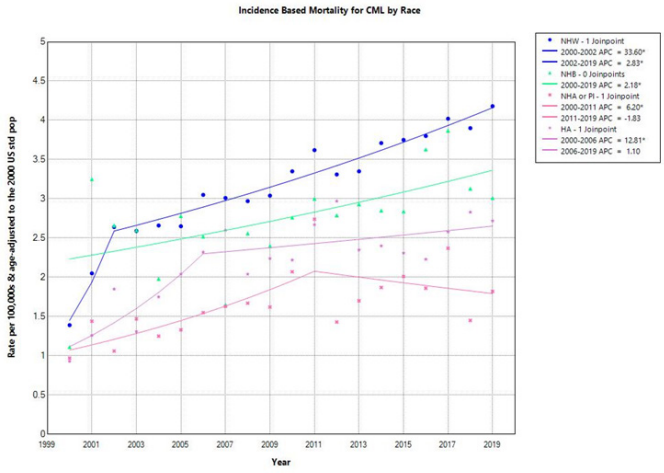

Figure 2: IBM for CML 2000-2019 by Race/ethnicity.

The AAPC for NMR and MR was 6.14% (95% CI 3.16 -9.19) and 5.08% (95% CI 4.03 - 6.25) per 100,000 person-years, respectively.

Figure 3 graphically depicts these changes.

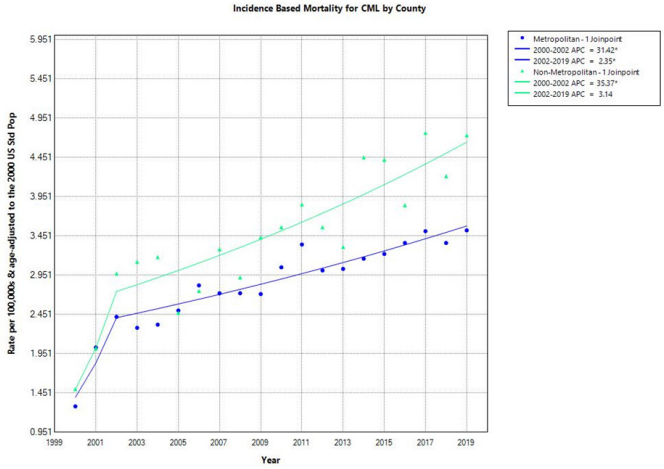

Figure 3: IBM for CML 2000-2019 by Residence.

The joinpoint parallelism test showed that the overall trends in CML IBM between NMR and MR failed to reject parallelism (P= 0.128, significance level of 0.05).

IBM Trend Stratified by Household Income

We looked at household incomes less than (<) $35,000 and households earning $75,000+. From 2000 to 2019, the IBM rate was 1.52 (1.34–1.73) and 1.13 (1.09–1.16), respectively. Households with $75,000+ experienced a joint point in 2002. However, no joint point was appreciated for households with <$35,000.

The IBM of CML in households with incomes <$35,000 had an APC of 4.52%. Meanwhile, households with incomes of $75,000+ had an APC of 34.35% from 2000-2002, and APC changed to 2.21% from 2002 to 2019.

The AAPC for household incomes with <$35,000 was 4.52% (95% CI 2.27-8.02%), and the AAPC for household income earnings $75,000+ was 5.19% (95% CI 4.32-6.21%).

Figure 4 depicts these changes graphically.

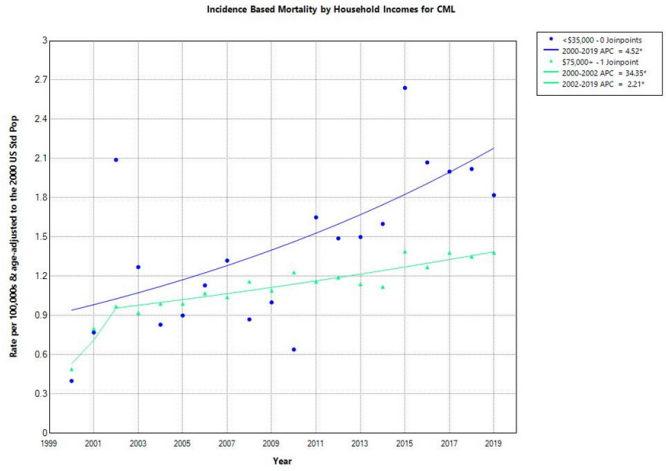

Figure 4: IBM for CML 2000-2019 by Household Income.

A Parallelism test failed to reject parallelism (p=0.14, significance level of 0.05)

IBM Trend Stratified by Age Group

According to the American Cancer Society, the average age of CML diagnosis is about 64-66. We looked at the incidence-based mortality for CML between 55 and 74. Between 2000 and 2019, the IBM rate for age groups 55-59, 60-64, 65-69, and 70-74 were 0.65, 1.12, 2.18, and 3.66, respectively.

IBM significantly decreased over the century since early 2000. The age group 55 to 59 experienced no joint points and had an AAPC of 2.87%. The age group 60 to 64 experienced one jointpoint in 2002, with an AAPC of 6.1%. The age groups 65-69 experienced one joinpoint in 2011 with an AAPC of 2.3%, and ages 70-74 experienced one joinpoint in 2002 with an AAPC of 4.8%.

Figure 5 graphically depicts these changes.

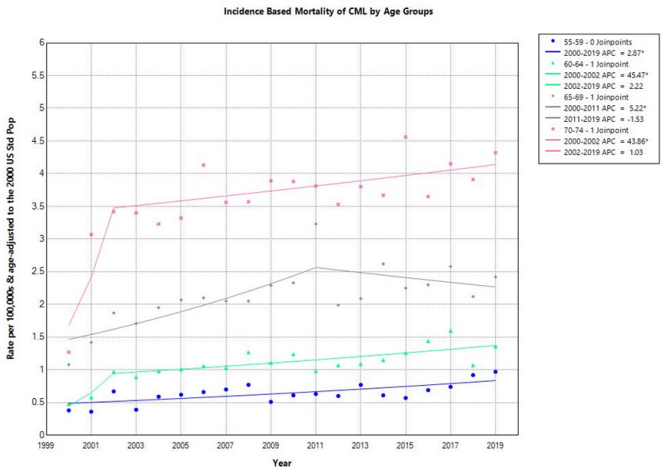

Figure 5: IBM for CML 2000-2019 by Age Group.

Discussion

Interpretation of Study Results

The observed improvement in IBM for CML from 2000-2019 appears to be closely tied to the introduction of TKIs, particularly imatinib, approved for use in 2001 by the Food and Drug Administration. The significant change in IBM around 2002 aligns with the timeline for the introduction of TKIs in the management plan of the disease.

While there seemed to be initial differences in IBM rates when comparing sexes, household income levels, and metropolitan versus non-metropolitan residency, a parallelism test revealed that the groups experienced a similar rate of change in IBM, highlighting that the impact of TKIs on CML mortality is consistent across diverse demographics.

The improvement in IBM rates underscores the effectiveness of TKIs in managing CML, contributing to a positive trend in patient outcomes. The uniformity of this positive change across various demographic groups indicates that the benefits of TKIs extend broadly, irrespective of gender, income, or residence.

Also notable is the fact that people are living longer with CML, evidenced by the increase in population count with increasing age groups.

These findings depict the transformative impact of targeted therapy on disease outcomes. The data suggests that these treatments have played a crucial role in reducing CML-related mortality, offering hope and improved prognosis for individuals across different backgrounds.

Notwithstanding these encouraging results, there is much room for improvement.

Disparities in Incidence and Mortality

Long-term myeloid leukemia and death rates are very different for different groups of people. This information is crucial in selecting appropriate therapies and offsetting healthcare inequality [5].

There is evidence that people over the age of 60 die more often from CML than those who were younger at diagnosis. At an advanced age, individuals may not be able to withstand aggressive treatments like their younger counterparts. Pre-existing ailments and comorbidities are also other factors. Most people are between 60 and 65 years old when receiving a diagnosis of CML. For this age group, receiving care is more complicated. Clinicians are also challenged to individualize management plans owing to pre-existing ailments.

It is more common for men to develop CML than women [6]. The occurrence and outcomes of this disease also vary between men and women. The ratio of men to women with CML is about 1.2-1.7, which suggests that some people may be more susceptible to certain risk factors than others.

CML affects people of different races in different ways. Several factors contribute to this, including genetics, access to healthcare, household income, and response to treatment. The rates of acquiring the disease and dying from it vary across races/ethnicities. To improve healthcare and reduce disparities among the races and ethnic groups, it is essential to know these differences [7].

Individuals from low-income households or those who encounter obstacles in reaching healthcare facilities are at a disadvantage. They more often fail to receive prompt diagnoses and advanced therapies, resulting in poorer prognoses. Disparities in healthcare access can also impede adherence to treatment protocols, ultimately impacting survival rates.

Comparison with Other Cancers

CML is unique because its phenotype and genome are always constant. It is almost always caused by a single mutation called BCR-ABL1. This sets CML apart from other leukemias, blood cancers, and solid tumors, which usually have a wider range of phenotypes and genotypes. This characteristic of CML makes it especially susceptible to the action of TKIs, which does not apply to other cancers with variable associated mutations. The BCR-ABL fusion protein, created when chromosomes 9 and 22 permutate in CML, has Abl tyrosine kinase activity that is "always on". This causes the disease to grow out of control [7]. TKIs, including imatinib, dasatinib, and nilotinib, target this abnormal tyrosine kinase activity and have been proven to change the prognosis of CML for the better effectively.

Historically, resistance has been a major obstacle in the management of cancers. Despite the significant progress made with TKIs, developing resistance to this therapy remains a possibility. Therefore, it is crucial to continue funding research to understand the intricacies of pathophysiology and mechanisms of this condition to find innovative approaches to address it [8].

While TKI treatment for CML has dramatically affected the landscape of the disease, the same therapy cannot be used for other types of cancer. TKI owes much of its success to the fact that the genes in CML are affected in a simple and consistent manner. On the other hand, the fact that TKIs work in CML tells us a lot about how to treat cancer, especially when it comes to targeted therapies. The idea of finding and treating a specific driver mutation, as in CML, has been an example for other pathologies where finding and treating significant genetic changes could lead to similar improvements in treatment success. This demonstrates the significance of dissecting each cancer's distinctive genetic and molecular environment, as it plays a pivotal role in determining the disease's pathogenesis, prognosis, and therapeutic response. As some cancers are more complex, they need more advanced treatment methods. A mix of therapies is often needed to target different mutations and pathways.

The development of resistance to therapy and the progression of the disease are persistent challenges in the treatment of all cancers, including CML, and require ongoing research and innovation to improve patient outcomes [9].

Advances in Treatment

There have been remarkable changes in the management of CML since the approval of tyrosine kinase inhibitors (TKIs). Imatinib, nilotinib, dasatinib, and bosutinib have improved the treatment and prognosis of the disease. These agents target the BCR/ABL1 fusion protein induced by the mutation t(9;22)(q34;q11.2). This targeted method ensures that normal myeloid cells continue proliferating with high differentiation levels in the long-term phase of CML. As a result, most people with CML can now experience long-term remissions and lead normal lives [10].

Even with these improvements, there are still some restrictions in managing the condition. Resistance to TKIs develops in about 5–10% of patients. Changes in the BCR-ABL1 gene, like the T315I "gatekeeper" mutation, are a common culprit of resistance. This mutation makes all TKIs except ponatinib less effective. This is an ongoing area of research. Scientists continue to search for ways to overcome TKI resistance and provide hope for those battling this disease.

In the natural course of the disease, chronic phase CML (CP-CML) turns into blast phase CML (BP-CML). This acute leukemia can only be treated by hematopoietic stem cell transplantation. This change into BP-CML is inevitable without an adequate response to TKI therapy. A minimal number of patients stay in remission after discontinuing TKI treatment. A major area of study is figuring out and raising treatment-free remission (TFR) rates, the percentage of people who can stay in remission without continuing treatment [11]. When first-line therapy does not work or stops working, there are a few options to consider. The choice of therapy is tailored to the types of cancer and illnesses based on symptomatology, disease stage, and BCR-ABL1 mutation status. Second and third generation TKIs, with their respective pharmacological profiles and reaction patterns, may be considered. Allogeneic stem cell transplantation remains a good choice for those with advanced disease or who have failed many TKI treatments. People over 65 who have cytogenetic relapse after failing all TKIs can still have a good chance of a long life in remission if they continue treatment with the best and safest TKIs, sometimes along with a combination of other anti-CML drugs like hydroxyurea, omacetaxine, azacytidine, decitabine, cytarabine, or busulfan [12].

Socioeconomic Factors and Healthcare Access

There are many ways that socioeconomic factors affect patient outcomes, treatment choices, and treatment compliance in CML. A study found that lower income and education, living alone, and an older age are linked to poor survival rates after a CML diagnosis. However, these links were identified in a matched control group, suggesting that socioeconomic factors may be related to differences in overall health, such as comorbidities, at the outset rather than directly affecting survival rates from CML [13]. Another study showed that socioeconomic conditions seemed to impact therapy decision [14].

The effect of personal and household income on treatment decisions is complex. People have varying opinions on money. Understandably, people who have cancer may worry about the impact of their financial situation on treatment outcomes. Regardless, every patient with CML should have access to the best therapy that significantly increases survival rates.

According to patient-reported outcomes (PROs), treatment satisfaction is a crucial factor in the management of CML. Negative experiences or treatment limitations can be detrimental to their health-related quality of life (HRQoL) and adherence to management plans. Therefore, clinicians need to tailor each treatment plan to a patient's individual needs and habits, particularly with TKI. This flexibility can significantly improve HRQoL and treatment adherence [15].

Future directions

We can improve the treatment of CML by advancing therapies and enhancing our understanding of the disease at the molecular level. Innovative first-line treatments, pursuing treatment-free recovery, and managing incurable diseases have been recognized as the three main areas of focus [16].

More and more patients receive a mix of treatments to improve the early response to treatment. Interferons and immune checkpoint inhibitors are used to change the immune system. Venetoclax and pioglitazone target leukemia progenitor cells, while asciminib is used for BCR-ABL1 treatment. By inducing treatment response earlier, these methods aim to make remission last longer and stronger. These innovative practices are backed by logical and pre-clinical data.

The idea of treatment-free remission (TFR) has grown in importance in treating CML. People who have hit a stable deep

molecular response (DMR) and continue to respond should stop taking TKIs. In this way, care for people with CML has shifted from continuous treatment to a more focused approach, and many of them can safely discontinue treatment [17]. Nicotinib is currently the only drug approved for this use. Investigations are still underway to speed up the path to DMR and TFR. Researchers continue seeking better ways to enhance treatment choices, timings, predictive factors, and patient communication.

Finding new tyrosine kinase inhibitors for people with chronic CML, especially those who become immune to TKIs, is still very important. The refractory stage of the disease, often caused by changes that make it resistant, can be devastating. We still need more treatment options [18]. All the current treatment options for CML are evidence of the remarkable progress made in the last few decades. They provide an all-around method that not only improves the first response to treatment but also keeps the disease in remission for a long time and manages it well into its later stages.

Conclusion

This research and analysis study on CML uncovers the significant steps forward we have made in understanding and treating this challenging condition. The advent of TKIs has shifted the prognosis for patients afflicted with CML from an inevitably terminal diagnosis to a treatable chronic condition. However, studies and therapy for CML continue. Researchers are actively working to overcome resistance to TKIs, improve outcomes for advanced disease, and increase drug-free remission rates. CML studies will continue to focus on drug resistance and developing treatments for later stages of the disease. To achieve this, researchers are searching for new TKIs, exploring combination treatments, and using personalized approaches. It is also essential to consider age, gender, race, socioeconomic position, and access to care to ensure equitable healthcare delivery.

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