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Clinical presentations and ten-year follow-up of chronic myeloid leukemia patients receiving imatinib in Iraqi Kurdistan

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Abstract:

BACKGROUND: Chronic myeloid leukemia (CML) is driven by the BCR–ABL fusion gene, a constitutively active tyrosine kinase that can be effectively targeted with tyrosine kinase inhibitors (TKIs), particularly imatinib. Long-term real-world data on the effectiveness and safety of imatinib in low-resource settings remain limited.

OBJECTIVES: The aims was to evaluate the ten-year clinical outcomes, treatment responses, and tolerability of imatinib in CML patients in Iraqi Kurdistan.

MATERIALS AND METHODS: A retrospective cross-sectional study involved 160 patients with CML using recorded hospital data from three different hematology centers in Iraqi Kurdistan, from 2014 to 2024. Collected data included patients' sociodemographic, clinical presentation, blood parameters, blood film, bone marrow examination, response to therapy, and survival rate. Then, they were analyzed and interpreted.

RESULTS: Most patients were females (51.25%), from Sulaimaniyah city (55%), aged 40–49 years old (27%), diagnosed between 2020 and 2024 (43%), and presented with fatigue (50%). Blood tests indicated low hemoglobin (<12 mg/dL) with extremely high leukocytes (44%), but normal platelets (66%). The blood film and bone marrow biopsy were abnormal in most patients. Furthermore, P210 expression was higher than P230 (98% vs. 2.0%). All patients received first-line treatment (only imatinib), 49 patients received a second line (mostly nilotinib), and only 7 patients received a third line. Using imatinib for 1–3 years results in resistance development in (26%) of patients and leads to discontinuation of the therapy. Despite several side effects of imatinib (especially anemia), the survival rate was very high (87%).

CONCLUSION: Imatinib remains a highly effective frontline therapy for CML in resource-limited settings, demonstrating durable responses and favorable long-term survival despite moderate rates of resistance and side effects.

Keywords:

Chronic myeloid leukemia, imatinib, Kurdistan Iraq, long-term follow-up, survival

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Introduction

Chronic myeloid leukemia (CML) is an uncommon myeloproliferative malignancy of the bone marrow considered as an expansion of a clone of hematopoietic cells, including an increased leukocyte in the blood. It has a tendency to progress more slowly than severe form.[1] It typically affects middle-aged adults and rarely children; however, it can affect any age. CML often does not cause symptoms and might be detected incidentally during a blood test. Advances in treatment have improved the prognosis of people with CML as people can achieve remission and live for many years after diagnosis.[2]

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Breakpoint cluster region (BCR) is an active Abelson (ABL) tyrosine kinase that is expressed by the Philadelphia chromosome number 22. It is made upon the t(9;22) reciprocal translocation that fused the BCR gene with the ABL. Based on the translocation breakpoint in BCR, different BCR–ABL protein isoforms are articulated that cover exons 2–11 of the ABL1 gene but vary in the distance of their BCR components. The most common BCR–ABL isoforms are P210, P190, and P230, in which the former is the most common in CML.[3,4]

The introduction of tyrosine kinase inhibitors (TKIs) raised a debate on CML management.[5] However, imatinib (Gleevec) is a particular inhibitor of the BCR–ABL tyrosine kinase and is effective in CML. Imatinib binds to the BCR–ABL kinase domain by averting the handover of a phosphate to tyrosine on the protein and the following initiation of phosphorylated protein. Thus, the transmission of signals to the nucleus is clogged and results in leukemic cell death.[6]

The Food and Drug Administration accepted imatinib as the first-line management for CML in December 2002 following an international randomized study that showed imatinib (400 mg once a day) was more active with fewer side effects than interferon-alpha plus cytarabine in patients with newly diagnosed CML. Consequently, imatinib enhanced the prognosis of CML.[7] Hence, development to progressive phases and secondary malignancies are the most common causes of death during imatinib. In addition, resistance development was the main reason for stopping treatment.[5]

Morphologic, biologic, immunologic, cytogenetic, and molecular genetic characterizations of CML are needed to establish the correct diagnosis or to exclude other possible causes of BM failure.[8] Therefore, we aimed to report 10-year follow-up data in patients who received imatinib as a primary treatment.

Materials and Methods

Study design and setting

Over the last 10 years (from January 2014 to July 2024), a total of 533 CML cases were diagnosed in three Kurdistan Hematology Centers (Hiwa Hematology/Oncology Hospital at Sulaimaniyah, Nanakali Blood and Oncology Hospital at Erbil, and Azadi Hematology/Oncology Hospital at Duhok, Iraq). A complete data of only 160 patients were available in the hospital record. Thus, this retrospective, cross-sectional, hospital-based study included 160 CML patients' using recorded hospital data from three Kurdistan Hematology Centers, and the duration of data collection was 4 months, from February to June 2024.

Patients' inclusion criteria

Individuals with CML (chronic accelerated phase) who were >18 years of age and had positive BCR–ABL tyrosine kinase.

Patients' exclusion criteria

Patients with atypical CML, chronic myelomonocytic leukemia, and those with other myeloproliferative disorders.

Study protocol

Patients' sociodemographic data (age, gender, and residency), year of CML diagnosis, clinical presentation, performed blood parameters (hemoglobin, leukocyte count, platelets, and differential cell count), stained blood film and bone-marrow biopsy, lines of treatment, imatinib (crossover) information (treatment duration, side effects, causes of discontinuation, patients' response, and causes of crossover), and survival rate among patients were collected using a standard questionnaire. In addition, the data on a molecular study using polymerase chain reaction (PCR) to determine BCR-ABL gene mutation, type of PCR (quantitative or qualitative), and patients' molecular response were collected. Then, collected data were tabulated, analyzed, interpreted, explained, and compared to the outcomes of other studies.

Ethical approval

Approval was obtained from the Ethical and Scientific Committees of the Kurdistan Higher Council of Medical Specialties, Iraq, with reference number 1871 on November 6, 2023. Permission was also obtained from the hospital's authority and ethical committee to use their saved data for publication. Participants' consent was waived due to the study nature.

Statistical analysis

The Statistical Package for Social Science (SPSS, IBM, Chicago, USA, version 27) was used. The Chi-square test was utilized to find the association between independent and dependent variables. $P \leq 0.05$ was considered statistically significant, $P \leq 0.001$ as highly significant, and $P \leq 0.000$ as very highly significant.

Results

Regarding the sociodemographic data of the studied patients, most of them were females (n = 82, 51.25%) with male:female ratio of $\approx$ 0.95, aged 40–49 years of age (n = 43, 27%) with median age of 48.5 years and age range of 20–80 years. Furthermore, majority of patients were residents of Sulaimaniyah city (n = 88, 55%), diagnosed as CML between 2018 and 2022, (n = 69,

43%), presented with fatigue (n = 80, 50%), followed by abdominal pain (n = 48, 30%) and had normal spleen using ultrasound (n = 49, 31%), as shown in Table 1. Whereas, 21% (n = 33) of patients were asymptomatic and incidentally diagnosed to have CML (chronic phase).

Regarding blood parameters, most patients had low mean hemoglobin (<12 mg/dL) (9.81 ± 1.12), while 13.14 ± 1.17 had mean hemoglobin of > 12 mg/dL. Furthermore, the majority of patients had extremely high leukocyte count (n = 71, 44%) and normal platelets (n = 105, 66%). Leukocytosis and left shift appeared most frequently in the blood films of patients (n = 89, 56%), while only 2 patients had normal blood film (1.0%). Bone marrow study showed that 157 patients had myeloid hyperplasia (98%) and only one patient had myeloid hyperplasia with marrow fibrosis (1.0%), as shown in Table 2.

With respect to differential cell count, only the data of 75 patients were available in the hospital record, in which the majority of them had normal levels of granulocytes, monocytes, and blast cells, as shown in Table 3.

After running of PCR to determine the BCR–ABL fusion gene, it was indicated that the majority of patients had P210 expression (n = 157, 98%), and only 3 patients had P230 expression (2.0%). The first line of treatment (imatinib) was used in all patients (n = 160, 100%), 49 patients used the second line (nilotinib + bosutinib + dasatinib), while only 7 patients underwent the third line of treatment (bosutinib + dasatinib) (14%). In the second line of treatment, most patients used nilotinib (n = 42, 86%). Most patients (n = 70, 44%) received imatinib for 1–3 years, and the molecular response was reported after 12 months of treatment in most patients (n = 33, 21%). Development of resistance was seen in 41 patients (26%) (primary = 94% and secondary = 6.0%) and resulted in discontinuation of the therapy. The main reason for crossover was other than progression in most patients (n = 47, 29%), as shown in Table 4.

Among patients who received imatinib, 37 showed no side effects, while the more common side effect was anemia (n = 46, 29%), followed by fatigue/thrombocytopenia (n = 31, 19% each), then muscle cramp (n = 17, 11%), and many other side effects, as shown in Table 5.

Ten-year overall survival was found in 140 patients (87%) and 20 patients passed away, in which 9 (6.0%) died due to CML and 8 (73%) due to any other causes, as shown in Table 6.

Discussion

Many factors directly and indirectly correlate with incidence, epidemiology, line of treatment, recovery

Table 1: Sociodemographic data of the studied patients

Variables	Frequency (%)	*P*
Gender		
Female	82 (51.25)	0.7518
Male	78 (48.75)	
Residency		
Sulaimaniyah	88 (55)	0.0000***
Erbil	56 (35)	
Duhok	16 (10)	
Age group (years)		
20–29	16 (10)	0.0000***
30–39	23 (14)	
40–49	43 (27)	
50–59	39 (24)	
60–69	22 (14)	
70–79	12 (8.0)	
Above 79	5.0 (3.0)	
Year of diagnosis		
2014–2016	48 (30)	0.0000***
2016–2018	11 (7.0)	
2018–2020	32 (20)	
2020–2024	69 (43)	
Clinical presentation		
Fatigue	80 (50)	0.0000***
Abdominal pain	48 (30)	
Night sweats	35 (22)	
Weight loss	29 (18)	
Poor appetite	21 (13)	
Fever	20 (13)	
Pallor skin	12 (8.0)	
Headache	12 (8.0)	
Early satiety	5.0 (3.0)	
Back pain	5.0 (3.11)	
Bleeding	3.0 (1.86)	
Dyspnea	3.0 (1.86)	
Ecchymosis	3.0 (1.86)	
Itching	2.0 (1.24)	
Joint pain	2.0 (1.24)	
Bone pain	2.0 (1.24)	
Visual disturbance	2.0 (1.0)	
Nasal bleeding	1.0 (0.62)	
Epigastric pain	1.0 (0.62)	
Diarrhea	1.0 (0.62)	
Rectal bleeding, diarrhea, and vomiting	1.0 (0.62)	
Muscular pain	1.0 (0.62)	
Cough	1.0 (0.62)	
Melena	1.0 (0.62)	
Back pain and loin pain	1.0 (0.62)	
Chest pain	1.0 (0.62)	
Splenomegaly using ultrasound		
Normal	49 (31)	0.0013*
Mild (13–15 cm)	35 (22)	
Moderate (16–20 cm)	54 (34)	
Severe (>20 cm)	22 (14)	
Total	160 (100)	

*Significant difference, ***Highly significant difference, using Chi-square test

Table 2: Hematological values of the studied patients

Variables	Frequency (%)
White blood cell (10^6/μL)	
Low (<4500)	6.0 (4.0)
Normal (4500–10,000)	7.0 (4.0)
High (10,000–100,000)	69 (43)
Extremely high (100,000–400,000)	71 (44)
Out of range	7.0 (4.0)
Platelets	
Low (<150,000)	20 (13)
Normal (150,000–450,000)	105 (66)
High (>450,000)	35 (22)
Blood film	
Leukocytosis and left shift	89 (56)
Leukocytosis, left shift, and increased platelets	31 (19)
Neutrophilic leukocytosis	17 (11)
Neutrophilic leukocytosis and eosinophilia	1.0 (1.0)
Leukocytosis and basophilia	8.0 (5.0)
Leukocytosis left shift, eosinophilia, basophilia, and occasional blast	1.0 (1.0)
Leukocytosis, basophilia, and eosinophilia	2.0 (1.0)
Leukocytosis, monocytopenia left shift, and increased platelets	2.0 (1.0)
Platelets reduced with no immature cell	3.0 (2.0)
Neutrophilia and thrombocytopenia	1.0 (1.0)
Normal blood film	2.0 (1.0)
Normochromic normocytic anemia and leukopenia	2.0 (1.0)
Leukopenia and mild left shift	1.0 (1.0)
Bone marrow study	
Myeloid hyperplasia	157 (98)
Diluted marrow	2.0 (1.0)
Myeloid hyperplasia with marrow fibrosis	1.0 (1.0)

Table 3: Differential cell count among studied patients

Differential cell count	Normal range, *n* (%)	Below normal range, *n* (%)	Above normal range, *n* (%)
Neutrophil	51 (68)	3.0 (4.0)	21 (28)
Eosinophil	68 (91)	0	7.0 (9.0)
Basophil	40 (53)	0	35 (47)
Monocytes	71 (95)	0	4.0 (5.0)
Blast cell	49 (65)	0	26 (35)

rate, outcomes, and recurrence rate of cancers, including CML.[7] Thus, for 10 years, we followed patients with CML who received TKI inhibitors as initial therapy and we highlighted the safety and efficacy of therapy and the feasibility of discontinuation therapy.

Hence, in the current study, most patients were females (51.25%) and middle-aged (40–49 years, 27%) with median age of 48.5 years. In this regard, Hochhaus *et al*.[7] mentioned that 61.7% of the CML patients were males and their median age was 50 years, while Cheng *et al*.[9] stated that 51.1% of the CML patients were males and their median ages were 46 years. Furthermore, Nicolini *et al*.[10] showed that 57% of the CML patients were males and their median age was 67 years. These variations might be related to the sample size and completed patients' data in hospital records. In addition, most of our CML patients were from Sulaimaniyah city (55%), followed by Erbil, and then Duhok. This might be related to the population in each city, as Sulaimaniyah has the highest population compared to other cities, while Duhok has the lowest population. The incidence of CML among patients was highest between 2020 and 2024 (43%) which agreed with the global burden of CML worldwide as the CML drastically and gradually increased compared to the previous years.[11] However, Sokal score or other scoring system was not used at presentation because it is a retrospective study and we obtained the data from the hospital record. Hence, no cases of blast crises were reported at presentation.

In this study, most patients had low hemoglobin (9.81 ± 1.12) and leukocytosis (44%) but normal platelets (66%) and abnormal appearance of blood films and bone marrow with no splenomegaly. In this respect, Findakly and Arslan, 2020,[12] in their systematic review mentioned that most studies reported high platelet values, leukocytosis, hemoglobin value above 12 mg/dL, normal bone marrow cellularity, and splenomegaly (13%). Collectively, our data are lined with typical blood/bone marrow findings for patients with CML.

All our patients had positive BCR–ABL translocation and were diagnosed with chronic-phase CML, in which most of them were positive for P210 protein (98%) and the rest were positive for P230. No cases of p190 were recorded. Studies indicated that CML patient with P230 mutation has different clinical presentations and responses to first-line imatinib, while it has a poor response to front-line imatinib with better response to second-line nilotinib/dasatinib.[13] Furthermore, all patients in this study used imatinib as the first line of treatment, 49 used the second line (nilotinib + bosutinib + dasatinib), and 7 used the third line (bosutinib + dasatinib). The main reason for crossover imatinib was other than progression in most patients (29%). In this regard, García-Gutiérrez and Hernández-Boluda *et al*.[14] mentioned that nilotinib + dasatinib are second-generation TKI with superior efficacy and higher potency than imatinib as a first-line treatment for chronic phase CML and less cases with resistance or intolerance to prior TKI therapy were reported.

A complete molecular response means that the PCR test does not find the BCR–ABL gene, while a major molecular response means that the amount of BCR–ABL gene is $\leq 1/1000^{th}$ (or less) in untreated CML patients.[15] In the current study, most patients (44%) received imatinib for 1–3 years and their molecular response was reported after 3 months (6.0%) with the highest value after 12 months of treatments (21%).

Table 4: Molecular study and clinical data of studied patients

Variables	Frequency (%)
Gene expression	
P210	157 (98)
P230	3.0 (2.0)
PCR type	
Quantitative	81 (51)
Qualitative	79 (49)
Line of treatment	
First (only imatinib)	160 (100)
Second (nilotinib + bosutinib + dasatinib)	49 (31)
Third (bosutinib + dasatinib)	7.0 (14)
Second line of treatment	
Nilotinib	42 (86)
Bosutinib	6.0 (12)
Dasatinib	1.0 (2.0)
Third line of treatment	
Bosutinib	6.0 (86)
Dasatinib	1.0 (14)
Exposure duration to imatinib (years)	
3 months to 1 year	31 (19)
1–3	70 (44)
4–7	33 (21)
8–10	26 (16)
Molecular response	
12 months	33 (21)
6 months	32 (20)
18 months	15 (9.0)
3 months	9.0 (6.0
No MMR	59 (37)
24 months	5.0 (3.0)
Not done	2.0 (1.0)
MMR still positive	2.0 (1.0)
Transformed to acute myeloblastic leukemia	1.0 (1.0)
Stopped due to pregnancy	2.0 (1.0)
Reason for discontinuation imatinib	
Resistance	41 (26)
Loss of follow-up	20 (13)
Intolerance	9.0 (6.0)
Reason of crossover	
Other than progression	
Other	37 (23)
No complete hematological response in 6 months	10 (6.0)
Other	
No MMR	25 (16)
Intolerance	5.0 (3.0)
MMR increased	4.0 (3.0)
The drug was stopped by the patient	1.0 (1.0)
Stopped due to pregnancy	2.0 (1.0)
Progression only	
Loss of complete hematological response	7.0 (4.0)
Increase white blood cells	2.0 (1.0)
Loss of cytogenetic response	2.0 (1.0)

MMR=Major molecular response, PCR=Polymerase chain reaction

Table 5: Side effects of imatinib among patients

Variables	Frequency (%)
Anemia	46 (29)
Fatigue	31 (19)
Thrombocytopenia	30 (19)
Muscle cramp	20 (13)
Renal impairment	17 (11)
Elevated LFT	15 (9.0)
Leukopenia	12 (8.0)
Weight gain	8.0 (5.0)
Rash	7.0 (4.0)
Diarrhea	7.0 (4.0)
Nausea	6.0 (4.0)
Periorbital edema	5.0 (3.0)
Constipation	5.0 (3.0)
Epigastric pain	7.0 (4.0)
Bone pain	2.0 (1.0)
Chest tightness	2.0 (1.0)
Heavy menses	1.0 (1.0)
Headache	1.0 (1.0)
Hair loss	1.0 (1.0)
Epigastric pain and weight loss	1.0 (1.0)
Dyspnea	1.0 (1.0)
Bone pain and dyspnea	1.0 (1.0)
Loin pain and frequent urination	1.0 (1.0)
Blurred vision	1.0 (1.0)
Epigastric pain and headache	1.0 (1.0)
Abdominal pain and fluid retention	1.0 (1.0)
Back pain	1.0 (1.0)
Itching	1.0 (1.0)
Leg edema	1.0 (1.0)
Bone marrow failure	2.0 (1.0)
Itching and eczema	1.0 (1.0)
Dyspepsia	1.0 (1.0)
Gastrointestinal discomfort	1.0 (1.0)
Fluid retention	1.0 (1.0)
Vomiting	1.0 (1.0)

LFT=Liver function test

In this respect, Druker *et al*.[16] showed high rates of complete cytogenetic response among those received imatinib (69% by 12 months and 87% by 60 months). Thus, 48.3% of patients completed imatinib treatment, and 82.8% had a complete cytogenetic response.

Furthermore, we found that imatinib produced resistance in 26% of patients and resulted in discontinuation of the therapy. The cessation of imatinib, even in those with unnoticeable BCR–ABL transcripts, leads to recurrence. Although imatinib cannot eliminate the malignant clone, probable mechanisms comprise drug efflux and amplification/mutation of the BCR–ABL gene. It is also possible that imatinib cannot totally impede BCR–ABL kinase activity; low levels of activity would allow cells to survive but not proliferate.[16]

Moreover, in the present study, imatinib therapy showed no side effects in 37 patients; however, anemia was the more common side effect (29%), followed by fatigue/thrombocytopenia (19% each), and then muscle

Table 6: Survival and death rates among studied patients

Variables	Frequency (%)
Due to CML	9.0 (6.0)
Due to any other causes	11 (7.0)
Event free survival	140 (88)

CML=Chronic myeloid leukemia

cramps (11%). These outcomes are agreed with that of Hochhaus *et al*.[7] which stated that severe adverse effects related to imatinib were not common and mostly arose during the 1st year of treatment.

Ten-year overall survival was found in most patients of this study (87%) and 13% died due to various causes, especially loss of follow-up (73%), while 6.0% of death was due to CML. Similarly, Druker *et al*.[16] found that 7.0% developed to accelerated-phase CML or blast crisis, and the overall survival of those who received imatinib as initial therapy was 89% at 60 months, while Hochhaus *et al*.[7] showed that the assessed overall survival rate at 10 years was 83.3% among the patients in the imatinib group. These outcomes might be related to the fact that the surviving patients were obedient to their oncologist in following up and taking therapies or might be related to the severity of cancer, age, and immune status of the patients.

The limitation of the study was not calculating the exact sample size, its retrospective nature, and lacking some other important parameters related to long-term imatinib usage. The strength of the study was multiple center study and had a long duration of 10 years.

Conclusion

Over a 10-year period, chronic myeloid leukemia was most frequently diagnosed between 2020 and 2024, particularly among middle-aged females residing in Sulaimaniyah. Imatinib, used as frontline therapy, demonstrated high long-term efficacy, with an overall survival rate of 87%, aligning with international benchmarks. Although resistance emerged in approximately one-quarter of patients—mostly within the first 3 years—and adverse effects such as anemia and thrombocytopenia were common, the majority of patients achieved durable clinical benefit. These findings underscore imatinib's sustained effectiveness and tolerability in a real-world, resource-limited setting and highlight the importance of ongoing monitoring and access to second-line TKIs for patients who develop resistance or intolerance.

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Conflicts of interest

There are no conflicts of interest.

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