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Original Article

Pathology Section

Clinical and Haematological Characteristics of Chronic Myeloid Leukaemia in Adults and Paediatric Patients: A Cross-sectional Study from a Tertiary Care Centre in Bengaluru, India

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ABSTRACT

Introduction: Chronic Myeloid Leukaemia (CML) is the most common myeloproliferative neoplasm and is characterised by presence of Philadelphia chromosome which results from a reciprocal balanced translocation between the chromosome 9 and 22(t9;22) resulting in the formation of Breakpoint Cluster Region - Abelson gene (BCR-ABL) fusion gene. Worldwide, the incidence of Chronic Myeloid Leukaemia (CML) is 1-2 per 100,000 population with a male predominance. The incidence increases with age. The median age of onset in middle-income groups is low. The age incidence is low in Asian countries. The present study was conducted to study the incidence and clinico-haematological profile of CML in South Indian population.

Aim: To evaluate the haematological parameters of CML and to classify the phases of CML.

Materials and Methods: The present 10-year descriptive cross-sectional study from January 2014 to December 2023 was done at the Department of Transfusion Medicine & Immunohaematology in a tertiary care hospital in Bengaluru, Karnataka, India. Data were collected from the laboratory registers and Laboratory information system. Demographic data of patients across all age groups (1 to 100 years), along with findings from peripheral smear, bone marrow and molecular investigations were collected. Flow cytometry was done in cases with Blastic Phase (BP). Data was entered using Microsoft Excel and expressed as percentages and frequencies.

Results: A total of 170 cases diagnosed with CML were included in the present study, with 155 adults and 15 children. Among the adult cases, male female ratio was 1.7:1. Highest incidence was observed in 31-40 age group (45.8%) with a male predominance (64.7%). The majority of adult patients presented in Chronic Phase (CP) (89.6%; 139/155), while 5.1% (8/155) were in High-Risk Chronic Phase (HRCP) and 5.1% (8/155) in BP. Among the 8 blast-phase cases, six were classified as myeloid blast crisis with MPO, CD 34, CD 13 and CD 117 positivity and two were B lymphoid blast crisis which are positive for CD 34, CD10, and CD 19. Of the 15 paediatric cases (0-18 years), five cases were below 10 years, and 10 cases were between 10 to 18 years. The male: female ratio in the paediatric age group was 1:1.5. Two children were in BP. Immunophenotyping confirmed lymphoid blast crisis

Conclusion: The current study demonstrates that the age of presentation of CML in Indian population is one to two decades earlier than that reported in Western population although the aetiology remains unclear. A mild male predominance was observed. CML was also encountered in paediatric age group, with two cases progressing to BP. Myeloid blast crisis constituted the majority of BP.

Keywords: Blastic phase, Chronic phase, High-risk chronic phase, Philadelphia chromosome

INTRODUCTION

The CML is a myeloproliferative neoplasm, arising from the abnormal pluripotent stem cell and characterised by presence of the Philadelphia chromosome with BCR-ABL1 fusion gene resulting from t(9;22) translocation. This leads to formation of a shortened chromosome 22 which harbours the BCR-ABL oncogene. Although this translocation was initially thought to result from radiation exposure, most patients with CML have no history of such exposure. So, experts believe that that Philadelphia chromosome can be formed due to spontaneous mutation. And it was found that Philadelphia chromosome is found in few normal healthy people. The BCR-ABL gene produces an oncoprotein of 210 kDa (p210) which plays a central role in the pathogenesis of CML by activating tyrosine kinase pathway, which in turn leads to downstream stimulation of the signal transduction of JAK/STAT, PI3K/AKT, MYC, and RAS/MEK, which are involved in cell growth, survival and inhibition of apoptosis. Therefore, haematopoietic stem cell with BCR-ABL 1 translocation have a proliferative advantage due to increased growth signalling factors and reduced apoptotic control leading to leukemic transformation. In minority of patients (5-10%) with CML, Philadelphia chromosome is not detected by conventional cytogenetics but 25-30% of these patients have BCR-ABL1 gene rearrangement, identified by molecular studies [1]. The diagnosis of CML is by both morphological and by molecular methods. Diagnosis of CML is very important because of the targeted therapy. The first line treatment in CML is tyrosine kinase inhibitors, except in pregnancy. Tyrosine kinase inhibitors gave a better survival rate than the other treatments. Imatinib was the first tyrosine kinase inhibitor used to treat CML and was found to be more effective when used early in CP [2]. Due to the evolving mutation in ABL1 point mutations the drug resistance is becoming more. Recently, STAMP-(specifically targeting the ABL1 myristoyl pocket) inhibitors have been introduced [3]. Patients with CML can be cured after targeted therapy. The median age of diagnosis of CML in western countries is 60 years, but in Asian and African countries, it occurs one to two decades earlier. The disease burden varies from one country to the other due to the availability of early screening, diagnosis and treatment. More than 95% of the patients

are diagnosed with CP, and some cases are diagnosed, when they do a complete blood count in routine medical check-up. World Health Organisation (WHO) classified CML into three phases, namely CP, Accelerated Phase (AP) and BP. A new modification in WHO (2022) after tyrosine kinase inhibitor era, CML is classified as CP, HRCP and BP. If the CP is left untreated, they progress to AP/CP at high risk and BP. Fatigue, weight loss, anaemia and splenomegaly are the common clinical presentation. CML has an annual incidence of 1-2 cases/100,000 population [4 ,5], CML is extremely rare in children accounting for 2-3% of all leukaemias in children and <0.1/100,000 population [6,7]. In children lymphoid blast crisis is more than the myeloid blast crisis, in contrast, in adult myeloid blast crisis is more common. The prognosis of CML depends on age, additional chromosomal abnormalities, transcript type of BCR-ABL1, and the tyrosine kinase inhibitors. Young adults and adolescent have a worse prognosis than the older people. Additional molecular abnormalities like chromosome 7q deletion affects the prognosis, the second-generation tyrosine kinase inhibitors have a low rate of drug resistance the phase of the disease [2].

The present study was undertaken to study the haematological parameters of CML and to classify CML in CP, AP, Blast phase (BP) and to study the clinic-haematological profile of Paediatric CML. Since CML in paediatric age group is less common, this study is done to identify the incidence and presentation CML in paediatrics.

MATERIALS AND METHODS

The present descriptive10-year study comprising of a 9-year retrospective analysis and a 1-year prospective study was conducted in the Department of Transfusion Medicine & Immunohaematology at a tertiary care hospital in Bengaluru, Karnataka, India, from January 2014 to December 2023. Ethical approval was obtained from the Institutional Ethics Committee (IECno.22/2023). Data were collected from the laboratory registers and Laboratory information system.

Inclusion and exclusion criteria: Peripheral blood smears, bone marrow aspirate with confirmed molecular diagnosis of chronic myeloid leukaemia were included in the present study. Reference smears and slides, as well as cases with only peripheral smear or bone marrows without molecular confirmation were excluded.

Study Procedure

Demographic and clinical data including, age, sex, complete blood count with peripheral smear, bone marrow aspirate, bone marrow biopsy results, molecular testing for BCR-ABL were collected. Flowcytometry data from cases in the BPs of CML data were also included.

Morphology: Morphological evaluation was done in peripheral blood and bone marrow aspirate stained with Leishman stain. A differential count of blast, other precursors, and mature cells was performed in peripheral smear and bone marrow aspirate.

Cytochemistry: In the cases of CML in BP, the peripheral smear and bone marrow aspirate smears were stained for Sudan Black B (SBB) and PAS stain. Bone marrow biopsy sections stained by Haematoxylin and Eosin stain (H&E) and marrow fibrosis were assessed using reticulin stain.

Flow cytometry: The flow cytometry Ethylene Diamine Tetra Acetic (EDTA) Acid samples were processed within 24 hours of collection. Flow cytometry was done in bone marrow aspirate samples in 10 cases using BD FACS Canto™ II instrument. A minimum of 1,000,000 events were acquired. Cells were gated based on low-side scatter and dim CD45 expression. A comprehensive panel of monoclonal antibodies were used to assess the lineage differentiation, including myeloid, monocytic, megakaryocytic, B, and T lymphoid lineages. The precursor markers-Cluster of Differentiation 34 (CD34), Terminal deoxynucleotidyl transferase (TdT) & Human Leukocyte Antigen - DR isotype (HLA-DR), Myeloid markers-Myeloperoxidase (MPO), CD13, CD33, CD117, CD14 and CD64, B Lymphoid markers CD19, CD10, CD20, CD22 and T Lymphoid markers Cytoplasmic and surface CD3, CD5 &CD7 were used. Flow cytometry was specifically done in BP of CML to differentiate between myeloid or lymphoid crisis.

Molecular biology: Bone marrow aspirate samples were sent to the molecular biology laboratory for BCR-ABL translocation.

STATISTICAL ANALYSIS

Data were entered using Microsoft excel and expressed as percentages and frequencies.

RESULTS

A total of 170 cases diagnosed as CML were included in the study. Of these, 155 were adults and 15 were children. Among the adults, the male female ratio was 1.7:1. The highest incidence was seen in 31-40 age group accounting for 71 (45.8%) with a male predominance 46 (64.7%) as shown in [Table/Fig-1]. Haemoglobin levels ranged from 43 g/L to 170 g/L with a mean of 93 g/L. Total count ranged from 4.7to700×10^9/L with a mean of 185×10^9/L. Platelet count ranged from 30 to 1200×10^9/L with a mean of 318×10^9/L as tabulated in [Table/Fig-2]. The clinical features of adult CML are depicted in [Table/Fig-3]. Among adults' cases, 89.6% (139/155) of cases were in CP, 5.1% (8/155) in HRCP/AP and 5.1% (8/155) were in the BP. The details are shown in [Table/Fig-4]. Of the blastic cases,

Age (years)	Male	Female	n (%)
19-20	2	1	3 (1.9)
21-30	19	9	28 (18)
31-40	46	25	71 (45.8)
41-50	15	8	23 (14.8)
51-60	8	6	14 (9)
>60	8	8	16 (10.3)
Total	98	57	

[Table/Fig-1]: Demographic data of adult CML (n=155).

Haematological parameters	Range	Mean
Haemoglobin	43-170 g/L	93 g/L
Total WBC count	4.7-700x10^9/L	185x10^9/L
Platelet count	30-1200x10 ^{9}/L	318x10^9/L

[Table/Fig-2]: Laboratory parameters of adult CML.
WBC-White blood cells

Variables		n (%)
Symptoms	Tiredness	127 (81)
	Body pain	118 (76)
	Fever	59 (38)
	Abdominal pain	137 (88)
	Neck pain	39 (25)
Signs	Pallor	79 (50.9)
	Icterus	54 (34.8)
	Lymphadenopathy	17 (10.9)
	Splenomegaly	128 (82.5)
	Hepatomegaly	22 (14.2)

[Table/Fig-3]: Clinical features of adult CML.

Phases	Male	Female	n (%)
Chronic Phase (CP)	87	52	139 (89.6)
Accelerated Phase (AP) /High Risk Chronic Phase (HRCP)	4	4	8 (5.1)
Blastic phase	7	1	8 (5.1)
Total	98	57	155

[Table/Fig-4]: Phases of adult CML.

six were myeloid blast crisis showing positivity for MPO, CD34, CD13 & CD117, while two were of B lymphoid blast crisis showing CD34, CD19 & CD10 positivity [Table/Fig-5,6]. The paediatric group included 15 cases (0-18 years), comprising 5 cases less than 10 years, and 10 cases aged 10 to 18 years. The male: female ratio is 1:1.5. One six-year-old child presented in HRCP (AP) (WHO 2022) and progressed to blastic phase (B lymphoblastic crisis) in one year. The demographic data, age distribution, lab parameters, phases of paediatric CML clinical features are depicted in [Table/Fig-6-10]. Immunophenotyping confirmed B lymphoid blast crisis. The immunophenotyping of the BP is shown in [Table/Fig-10] along with adult CML BP cases. BCR-ABL translocation was positive in all cases. The morphology of CML in peripheral smear, bone marrow aspirate and biopsy are shown in [Table/Fig-11].

Among the 15 cases in paediatric age group, 7 (46.6%) are on regular follow-up and continue to attend check-ups after transitioning from paediatric-haemato-oncology to adult haemato-oncology services. Out of them 2 (13%) resumed follow-up after two years from the time of diagnosis, while 3 (20%) were lost to follow-up within one month of diagnosis. All 7 patients currently under follow-up are in both haematological and molecular remission. The remaining 3 cases did not return for follow-up.

Among the total, including both adult (8) and paediatric patients (2), 10 cases were in the BP. Of these, 6 cases were classified as myeloid blast crisis and showed positivity for MPO, CD34, CD13, and CD117, whereas 4 cases were classified as B-lymphoid blast crisis with positivity for CD34, CD10, and CD19. Photomicrographic images of CML are depicted in [Table/Fig-12]. The flow cytometry histograms of the BPs are depicted in [Table/Fig-11,13].

Age (years)	Male	Female	Myeloid blast crisis	Lymphoid blast crisis
19-30				
31-40	1			1
41-50	2	1	2	1
51-60	4		4	
Total	7	1	6	2

[Table/Fig-5]: Blastic Phase (BP) of adult CML.

DISCUSSION

Chronic myeloid leukaemia is one of the most common haematological malignancies with worldwide incidence rate ranging from 1 to 2 cases per 100,000 per year. CML constitutes about 15 to 25% of all haematological malignancies, characterised by the presence of Philadelphia chromosome and BCR-ABL1 gene [4,6,8]. About 50% of the patients have no symptoms and are co-incidentally diagnosed when they are doing a routine blood check-up [9]. In the Western countries, the median age at diagnosis is usually in sixth and seventh decades, but in Asian and African countries, it is one to two decades earlier. Tyrosine kinase inhibitors treatment protocols have changed the course of fatal leukaemia to remain in CP for many years. The burden of CML in each country or state depends on the availability of tests and investigations for early diagnosis and treatment [3,4].

Case	Age (years)	Sex	Blast %	Type of blast crisis	Immunophenotyping Positive markers	Denova blastic phase/progressed to blastic phase
1	43	M	30	Myeloid	CD34, MPO, CD13, CD33, CD117	CP to BP in 1 year
2	49	M	66	B-Lymphoblast	CD34, CD19, CD10, CD20, CD79a	CP to BP in 1 year
3	58	M	24	Myeloid	CD34, MPO, CD13, CD33, CD117	Denova blasticphase
4	51	M	35	Myeloid	CD34, MPO, CD13, CD33, CD117	CP to BP in 2 years
5	49	F	55	Myeloid	CD34, MPO, CD13, CD33, CD117	CP to BP in 2 years
6	6	F	74	B-Lymphoblast	CD34, CD19,CD10,CD20, CD79a, tdt	From high-risk CP to BP in 1 year
7	38	M	67	B-Lymphoblast	CD34, CD19, CD10,CD20, CD79a, tdt	Denova BP CSF positive for blast
8	58	M	22	Myeloid	CD34, MPO, CD13, CD33, CD117	CP to BP in 1 year
9	14	F	27	B-Lymphoblast	CD34, CD19, CD10, CD20, CD79a, tdt, HLA-DR	CP to BP in 1 year
10	52	M	45	Myeloid	CD34, MPO, CD13, CD33, CD117	CP to high-risk in 4 years and to BP in 2 years

[Table/Fig-6]: Data of Blastic Phase (BP) of CML (both in adults and children).

Variables	Number	Mean	Median
Age (years)			
1-10	5	7.4	7
11-18	10	15	16
Gender	**Male**	**Female**	**M:F**
1-10	2	3	1:1.5
11-18	4	6	1:1.5

[Table/Fig-7]: Demographic data of Paediatric CML.

Variables		n (%)
Symptoms	Tiredness	2 (13.3)
	Body pain	3 (20)
	Fever	3 (20)
	Abdominal pain	6 (40)
	Neck pain	2 (13.3)
Signs	Pallor	8 (53.3)
	Icterus	4 (26.6)
	Lymphadenopathy	1 (6.6)
	Splenomegaly	7 (46.6)

[Table/Fig-8]: Clinical features of paediatric CML.

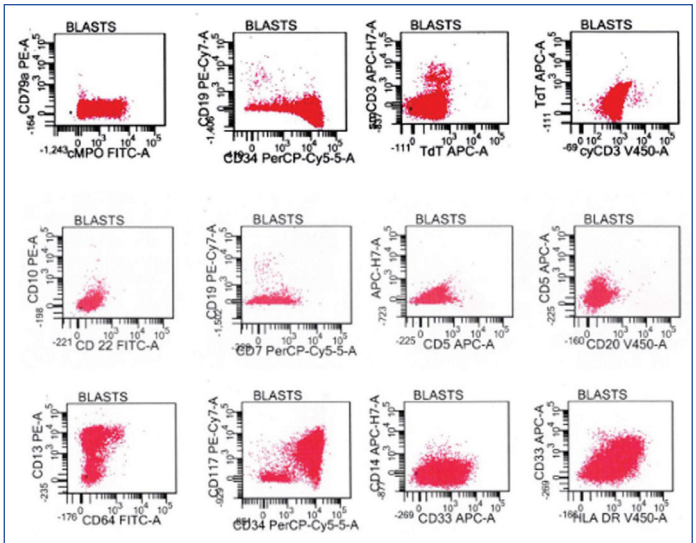
Haematological Parameters	Range	Mean	Median
Haemoglobin	62 to 115 g/L	79 g/L	75 g/L
Total WBC count	12.6 to 746×10^9/L	312.3×10^9/L	327.57×10^9/L
Blast	0-12%	2.8%	2%
Eosinophils	1-11%	4.9%	5%
Basophils	0-10%	4.4%	4%
Platelet count	30 to 1179×10^9/L	420.8×10^9/L	336×10^9/L

[Table/Fig-9]: Haematological profile of paediatric CML.
WBC -White blood cells

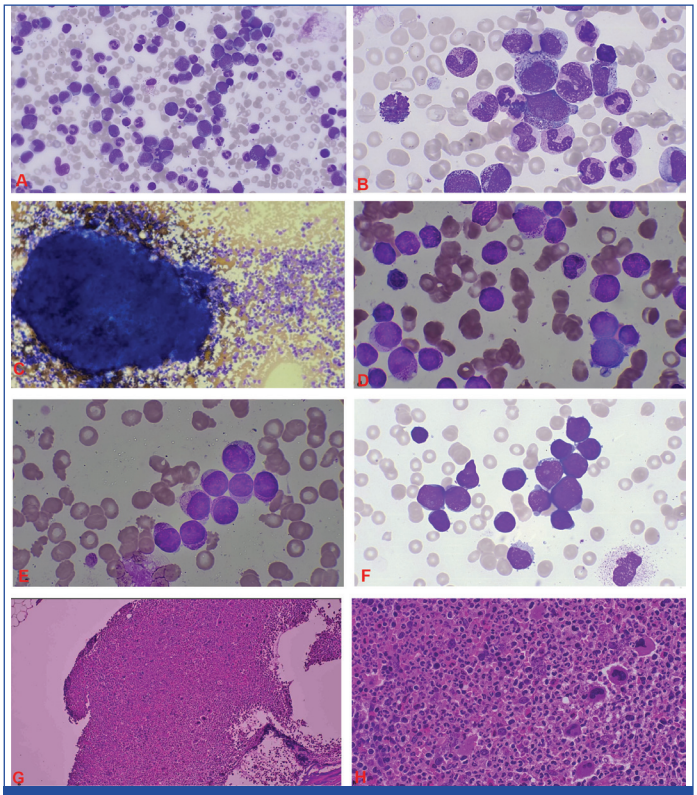
Phases	Male	Female
Chronic Phase (CP)	6	7
Accelerated Phase (AP)/High-Risk Chronic Phase (HRCP)		
Blastic phase		2

[Table/Fig-10]: Phases of paediatric CML.

In the current study, a total of 170 CML cases were identified based on peripheral smear findings, bone marrow examination and molecular study results. Of these 170 cases 155 were adults. The predominant age group affected in adult category was between 31-



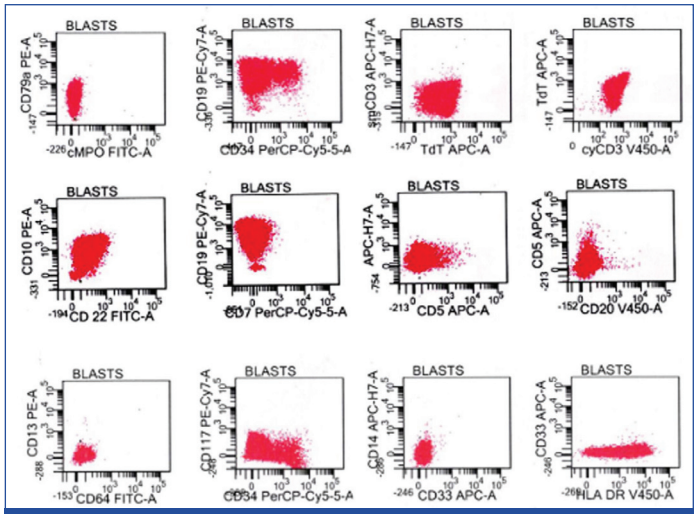
[Table/Fig-11]: Flowcytometric analysis of CML in myeloid blast crisis showing:
Precursor marker-CD 34, HLA-DR positive;
Myeloid marker MPO, CD 13, CD 117-Positive;
Negative for B Lymphoid and T Lymphoid markers.



[Table/Fig-12]: Photomicrograph images of CML.
Peripheral smear of CML 40x showing marked leukocytosis with eosinophils & basophils;
Peripheral smear of CML 100x showing marked leukocytosis with eosinophils & basophils;
Hypercellular bone marrow aspirate fragment 10x;
Bone marrow aspirate showing accelerated/High-Risk Chronic Phase (HRCP) CML 100X;
Bone marrow aspirate showing myeloid Blastic Phase (BP) CML 100X;
Bone marrow aspirate showing lymphoid blastic phase CML 100X;
Hypercellular bone marrow biopsy Bone marrow biopsy 10x;
Bone marrow biopsy showing myeloid hyperplasia 40x.

40 years 71 (45.8%), followed by 21-30 years 28 (18%). Male to female ratio is 1.7:1. The present study infers the incidence of CML in Indian patients is two decades earlier than the western population. This result is supported by studies performed in different parts of India from Punjab, Bihar, Madhya Pradesh, Uttar Pradesh and from Morracco, Nigeria, and Pakistan [9-15].

As per the 2022 WHO classification of haematolymphoid tumours, the natural history of CML has been modified with the introduction targeted tyrosinekinase inhibitors. The designation of AP has been found less useful, and it is termed as HRCP. In the170 cases of CML in the current study, 155 cases were adults. Of these patients, 139 (89.6%) cases were in CP, 5.1% (8/155) in AP/HRCP and 5.1%



[Table/Fig-13]: Flowcytometric analysis of CML in B Lymphoid blast crisis showing:
Precursor marker -CD34, TdT, HLA-DR -Positive;
B lymphoid markers CD19, CD 10-Positive -CD79a dim positive;
Negative for myeloid and T lymphoid markers.

(8/155) in BP. A total of 8 cases of CML in BP was seen. Six patients had myeloid blast crisis and two had B cell lymphoblastic crisis. This is comparable with observations of Kumar S et al., (CP 83%, HRCP 12% and BP 5%), Wang AH et al., (CP 86.5%, HRCP 6%, BP 7.5%) [12,16].

In the present study, the level of haemoglobin varied between 43-170 g/L, with a mean value of 93 g/L. Leukocyte count ranged from 4.70-700×10^9/L with a mean value of 185×10^9/L, and platelet ranged from 30-1200×10^9/L with a mean value of 318×10^9/L. These values of the parameters matched well with values obtained by Sinha R et al., Kumar S et al., and Shittu AO et al., [10,12,14]. Abdominal pain was the predominant symptom experienced by 137 (88%) patients, followed by fatigue 127 (81%) and body pain 118 (76%). Splenomegaly was the most common sign affecting 128 (82.5%) followed by pallor 79 (50.9%). The clinical presentation is consistent with studies by Sinha Ret al., Meena A et al., and Algahtani FH and Alqahtany FS [10,11,17].

The occurrence of CML is rare in children representing 1-2% of all the childhood leukaemia cases [18]. In the present study, 15 of the 170 cases were in paediatric age group. There was a predominance of females with the male: female ratio of 1:1.5, with a slight female predominance. The median age of presentation was 14 years. The commonest complaint was abdominal pain in 6 (40%) of the cases. Splenomegaly was seen in 7 (46.7%) cases. Anaemia was evident in 14 out of 15 (93%) cases. In the present study, among the 15 cases, 13 (86.6%) showed markedly increased total leukocyte count varying between 126-740×10^9/L, while only two patients revealed relatively lower leukocyte count of 12.6×10^9/L and 32×10^9/L. The study done by Bourusly M et al., on paediatric CML showed the median age of presentation was 8.4 years, with with equal sex distribution (6 males, 6 females), and all the 12 cases presented with splenomegaly. These children presented with markedly elevated leukocyte counts and a median of 358 x 10^9/L was found [7]. Similar findings were found in Kesana S et al., study, where haemoglobin <10 g/dL was found in 64% of patients [18]. A study by Chandra D et al., observed in their study total WBC count was >10×10^9 /L [19].

Majority of the cases 13(86.6%) presented in CP. One six-year-old female child first presented with accelerated and later within one year progressed to blasticphase. One more child was diagnosed with CP and transformed to BP. Both children were found to have B lymphoblastic crisis. A similar study by Kesana S et al., showed 88.4% children presented with CML-CP and CML-AP 6.3% and CML-BP 5.3% [18]. A similar study done by Chandra D et al., showed the median age at diagnosis was 16 years and 84% were diagnosed with CML-CP [19]. A study done by Suttorp M et al., 93% of cases presented with CML-CP, 1% in AP and three in BP [20].

In total among the 10 (5.9%) of BP, six patients had myeloid blast crisis and four had B cell lymphoblastic crisis. The prognosis of myeloid blast crisis depends on the presence of other chromosomal abnormalities [21]. The current study showed 10 cases in BP in which six cases were initially diagnosed in CP, two in CPHR which progressed to BP. Two cases presented de novo in BP. This is correlating with a study done by Jain P et al., which showed 80% of the blast crisis patients were diagnosed in CP and it took a median of 41 months to progress to BP. The rest of the BP was presented denovo [22].

Limitation(s)

Complete follow-up of patients could not be achieved due to financial constraints and other logistical challenges. In the present study, CML was classified into CP, AP, and BP. However, following the WHO 2022 classification, CML has been redefined into CP, HRCP, and BP.

CONCLUSION(S)

The age of presentation in the Indian population is reported to be one to two decades earlier than in Western populations, though the underlying aetiology remains unknown. A mild male predominance is observed. The disease has also been reported in children, and in two cases it has progressed to the BP.

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