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Patients with Chronic Myeloid Leukemia Diagnosed with Predominant Thrombocytosis without Marked Leukocytosis: Case Series

Belirgin Lökositoz Olmaksizin Baskin Trombositozla Tani Alan Kronik Miyeloid Lösemili Hastalar: Vaka Serisi

Fatih Yaman[1], Filiz Yavaşoğlu[2], Neslihan Andıç[2], Eren Gündüz[2], Hava Üsküdar Teke[2]

[1]Tokat Public Hospital, Department of Hematology, Tokat, Türkiye
[2]Eskişehir Osmangazi Faculty of Medicine, Department of Hematology, Eskişehir, Türkiye

ORCID ID of the authors
FY. 0000-0002-0494-571X
FY. 0000-0002-4017-4668
NA. 0000-0003-0510-4733
EG. 0000-0001-7455-2949
HUT. 0000-0002-4434-4580

Correspondence / Sorumlu yazar:
Fatih YAMAN

Tokat Public Hospital, Department of Hematology, Tokat, Türkiye

e-mail: ymnfth87@gmail.com

Abstract: Chronic myeloid leukemia (CML) is a clonal hematopoietic pluripotent stem cell disease characterized by excessive and uncontrolled proliferation of myeloid lineage cells. Platelet count is increased in more than half of patients and the appearance of platelets is variable. When patients present with predominant thrombocytosis without marked leukocytosis, they should be tested for the Philadelphia chromosome or (breakpoint cluster region- Abelson) BCR-ABL to distinguish cases of CML. In this study, the data of 215 patients diagnosed with CML between 2010 and 2023 were retrospectively evaluated. The study enrolled patients aged ≥18 years with leukocyte count <40 x109/L and platelet count >500 x109/L at the time of diagnosis. While investigating the etiology of predominant thrombocytosis and no significant leukocytosis, 13 patients diagnosed with CML were identified. The proportion of these patients among all patients with CML was 6%. This study showed that CML should be considered in the differential diagnosis of patients with predominant thrombocytosis without marked leukocytosis. In these patients, the Ph chromosome should definitely be checked before ET is diagnosed. Making a correct diagnosis in this patient group is important in order to start tyrosine kinase inhibitor treatment before it progresses to accelerated or blastic phases.
Keywords: Chronic Myeloid Leukemia, Myeloproliferative Disorders, Essential Thrombocythemia, Thrombosis, Tyrosine Kinase İnhibitor

Özet: Kronik miyeloid lösemi (KML), miyeloid seri hücrelerinin aşırı ve kontrolsüz çoğalmasıyla karakterize klonal hematopoietik pluripotent bir kök hücre hastalığıdır. Hastaların yarısından fazlasında trombosit sayısı artmıştır ve trombositlerin görünümü değişkendir. Hastalarda belirgin lökositoz olmaksızın baskın trombositoz mevcutsa, KML vakalarını ayırt etmek için Philadelphia kromozomu veya (breakpoint cluster region -Abelson) BCR-ABL testi yapılmalıdır. Bu çalışmada 2010-2023 yılları arasında KML tanısı almış 215 hastanın verileri retrospektif olarak değerlendirildi. Çalışmaya tanı anında lökosit sayısı <40 x109/L ve trombosit sayısı >500 x109/L olan ≥18 yaş hastalar dahil edildi. Belirgin lökositoz olmadan baskın trombositozla tetkik edilirken KML tanısı alan 13 hasta belirlendi. Bu hastaların tüm KML hastaları içindeki oranı %6 bulundu. Bu çalışma, belirgin lökositoz olmaksızın baskın trombositozu olan hastaların ayırıcı tanısında KML'nin düşünülmesi gerektiğini göstermektedir. Bu hastalarda ET tanısı konulmadan önce mutlaka Ph kromozomuna bakılmalıdır. Bu hasta grubunda doğru tanı koymak, hastalığın akselere veya blastik faza ilerlemeden tirozin kinaz inhibitörü tedavisine başlanması açısından önemlidir.
Anahtar Kelimeler: Kronik Miyeloid Lösemi, Miyeloproliferatif Hastalık, Esansiyel Trombositoz, Tromboz, Triozin Kinaz İnhibitörü

Ethics Committee Approval: The study was approved by Eskişehir Osmangazi University Noninterventional Clinical Research Ethical Committee (Decision no: 37, Date: 21.02.2023).

Informed Consent: The authors declared that it was not considered necessary to get consent from the patients because the study was a retrospective data analysis.

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1. Introduction

Chronic myeloid leukemia (CML) is a clonal hematopoietic pluripotent stem cell disease characterized by excessive and uncontrolled proliferation of myeloid lineage cells(1). It is among the myeloproliferative disorders (MPDs), which are clonal disorders of myeloid origin, including essential thrombocythemia, polycythemia vera and myelofibrosis(2). The Philadelphia (Ph) chromosome, an abnormal chromosome 22 resulting from a reciprocal translocation [t(9;22)q34;q11)] leading to the fusion of the Abelson (ABL) protooncogene on chromosome 9 and the breakpoint cluster region (BCR) gene on chromosome 22, is detected in approximately 95% of CML cases(3). The resulting product of this chimeric gene is a protein with tyrosine kinase activity and a molecular weight of 210 kDa (p210) and is responsible for the development of the leukemic phenotype in CML. Depending on the cleavage sites in the BCR and ABL genes, different BCR-ABL fusion transcripts are formed. The most well-known are the p190, p210, and p230 fusion transcripts.

The annual incidence for CML is 1-2/100,000 and accounts for 15-20% of adult leukemias. It is more common in men than women (Male/Female=3/2) and can develop at any age, although patients are often diagnosed in their 50s and 60s(4).

The clinical course of the disease has 3 stages. These three stages are the chronic stage, which represents the majority of patients at the time of diagnosis, the accelerated stage, which can be observed when the disease is left untreated and allowed to run its natural course or when there is no response to treatment, and the blastic stage, which indicates disease progression. The diagnosis is usually made by leukocytosis detected during routine tests and 20-40% of these patients are asymptomatic at the time of diagnosis. The most common symptoms are weakness due to anemia, decreased exercise capacity, abdominal swelling due to splenomegaly, pain, and rapid satiety(5).

In CML, leukocytosis is the typical finding at the time of diagnosis and there is a picture of granulocytosis in which all stages of granulocytic series maturation from blasts to fragmented neutrophils can be observed. The basophil count is always elevated and can be detected in the early stages of the disease even before the leukocyte count increases. Platelet count is increased in more than half of patients and the appearance of platelets is variable(6).

When patients present with isolated thrombocytosis, they should be tested for the Philadelphia chromosome or BCR-ABL to distinguish cases of CML(7). Literature includes a few case reports of patients with CML presenting with isolated thrombocytosis at the time of diagnosis(8,9). This study reviews the clinical and laboratory findings, treatment and prognosis of patients with CML diagnosed with isolated thrombocytosis without significant leukocytosis.

2. Materials and Methods

In this study, the data of 215 patients diagnosed with CML between 2010 and 2023 in Eskişehir Osmangazi University, Department of Internal Medicine, Division of Hematology were retrospectively evaluated. Demographic and clinical properties of the patients were retrieved from patient files and hospital information record system. The study enrolled patients aged ≥18 years with leukocyte count <35 $x10^9$/L and platelet count >500 $x10^9$/L at the time of diagnosis. Overall survival (OS) was defined as the time from the date of initial diagnosis to the date of last visit or death.

Approval for the study was obtained from the Eskişehir Osmangazi University Non-Interventional Ethics Committee with the number 21.02.2023-37.

Statistical Analysis

The study evaluated the patients' age at diagnosis, bone marrow pathology, hemogram results, lactate dehydrogenase, cytogenetic analysis and polymerase chain reaction (PCR) results from bone marrow aspiration at diagnosis, Janus Kinase-2 (JAK-2), calreticulin (CALR), myeloproliferative leukemia (MPL), comorbid diseases, SOKAL, HASFORD, ELTS scores, history of thrombosis and bleeding, PCR results at 3 months, 6 months and 12 months, treatments and clinical responses.

Results were analyzed using SPSS (Statistical Package for Social Sciences) for Windows 24 program. For descriptive statistics, numerical data were expressed as median (min-max) and categorical data were expressed as number and percentage.

3. Results

After exclusion, 13 patients who were diagnosed with CML while being examined for isolated thrombocytosis without significant leukocytosis were detected. The proportion of these patients

among all patients with CML was 6%. The mean age at diagnosis was 53.5 (26-79) years. Six (46%) patients were over 60 years of age at diagnosis. Of the patients, 84% (n=11) were female. Female to male ratio was 11:2.

One patient (7%) was examined for hematuria and fever on admission. Twelve patients (93%) were asymptomatic. Three patients (23%) had mild splenomegaly, while 77% had none. No thrombosis was detected on admission. The mean platelet count was 1068x10^9/L (582-2392 x10^9/L). Four patients (30%) had a platelet count above 1000 x10^9/L. Six patients (46%) had anemia. The lowest hemoglobin value was 10.3 g/dL. The mean leukocyte count was 20.4 x10^9/L (8.0-31.2 x10^9/L). Eight patients (61%) had elevated lactate dehydrogenase (LDH) levels at the time of diagnosis. Table-1 shows the clinical and demographic properties and laboratory results of the patients.

All patients (100%) were positive for BCR-ABL translocation and all had chronic phase CML at the time of diagnosis. Eight patients (61%) were tested for JAK-2 mutation and all of them were negative. No patient was tested for CALR and MPL gene mutations. Cytogenetic analysis was performed on 11 patients at diagnosis, but it was observed that metaphase plates could not be obtained in 3 of these cases. Cytogenetics study showed Ph chromosome t(9,22) in 7 patients (53%). In one patient, the cytogenetic results were normal. Of the 13 patients tested for t(9,22) translocation by fluorescence in situ hybridization (FISH), 12 (92.3%) were positive.

The mean t(9,22) positivity rate by FISH in bone marrow was 80.63% (22.44-99.5%). All patients were tested for p210 by PCR. p210 results were positive in 7 patients. Results of 6 patients could not be reached. One patient had normal FISH and normal cytogenetics. This patient was diagnosed with CML due to p210 positivity.

Bone marrow biopsy results were available for 11 patients (Table-1). The risk score results are shown in Table-1 and no significant correlation was shown Sokal and Hasford risk scores and OS (p>0.05). However, low ELTS score was associated with increased OS (p=0.017) (Figure-1).

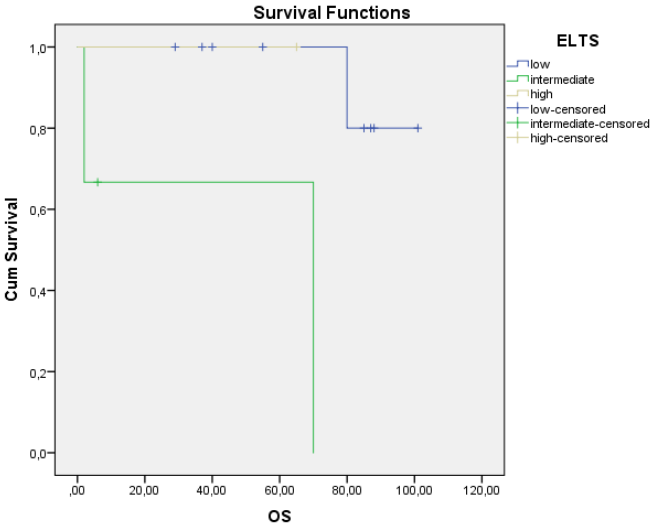

Figure-1. Low ELTS score was associated with increased OS (p=0.017).

Until the results of genetic tests are available five patients (38%) received hydroxyurea treatment. One patient was initially misdiagnosed with ET and briefly received anagrelide prior to confirmation of CML. Upon diagnosis of CML, all patients were started on imatinib. Three patients (23%) did not respond to imatinib treatment at the end of 12 months. Two of them were switched to nilotinib and one patient was switched to dasatinib. Bosutinib was started in one of the patients on nilotinib because of loss of response at 5 years of nilotinib treatment. At the 2nd year of dasatinib treatment, the patient developed plerual effusion and was switched to nilotinib. There were no other patients with complications. No patient developed accelerated or blastic phase CML during follow-up. None of the patients underwent allogeneic stem cell transplantation.

The mean follow-up period was 57.3 months (2-101 months). Ten patients (76%) were alive and followed for chronic phase CML. Three patients (23%) died during the chronic phase. One patient died due to COVID pneumonia and one patient died due to urinary tract infection. The cause of death of the third patient was not available. All deceased patients were >70 years old (72-79 years).

Table-1: Clinical and laboratory characteristics of patients

Patient#	Age	Gender	Symptom	SM	PLT ($x10^9$/L)	Hgb (g/dL)	WBC ($x10^9$/L)	NEU# ($x10^9$/L)	LYMP# ($x10^9$/L)	EOS# ($x10^9$/L)	BAS# ($x10^9$/L)	Diagnostic cytogenetics	Diagnostic Ph	Diagnostic p210 (%)
1	72	E	A	None	1418	13.8	14.40	10.2	3	0.4	0.1	46,XY,t(9;22)(q34;q11)[16]	22.44%	NA
2	77	K	A	Yes	734	12.7	18.22	8.88	5.31	0.6	2.52	46,XX,t(9;22)(q34;q11)[15]	93.89%	16.529
3	45	K	A	None	2392	10.9	12.8	9.7	2.1	0.4	0.1	46,XX,t(9;22)(q34;q11)[20]	55%	NA
4	29	K	Hematuria, fever	Yes	1669	11.1	27.23	19.47	5.84	0.46	0.99	No plaque	99.50%	100
5	66	E	A	None	824	16.8	27.6	18.6	3.1	1.2	4.2	46,XY,t(9;22)(q34;q11)[11]	99.50%	195.17
6	26	K	A	None	582	11.9	24.37	19.01	3.79	0.07	0.43	NA	99%	NA
7	63	K	A	Yes	892	10.3	24.58	20.6	1.8	0.1	0.7	46,XX,t(9;22)(q34;q11)[5]	94.33%	NA
8	39	K	A	None	733	11.2	31.2	27.6	1.5	1.7	0.2	46,XX,t(9;22)(q34;q11)[20]	95.67%	200
9	75	K	A	None	615	13.6	21.7	16.9	3	0.3	0.3	NA	41.8%	NA
10	45	K	A	None	772	12.9	8	4.5	2.2	0.3	0.4	46,XX,t(9;22)(q34;q11)[13]	85.87%	51.41
11	79	K	A	None	1477	11.1	15.1	12.9	1.2	0.7	0.8	No plaque	99.50%	17.64
12	43	K	A	None	895	12.2	26.8	20.7	3.6	0.6	1.3	46,XX[7]	Negative	NA
13	37	K	A	None	888	13.5	13.9	9.69	2.87	0.24	0.16	No plaque	99.50%	100

Table-1: Clinical and laboratory characteristics of patients (continued)

Patient#	Month 3 p210 (%)	Month 6 p210 (%)	Month 12 p210 (%)	Bone Marrow	SOKAL	HASFORD	ELTS	Treatment	Complication	Follow up (months)	Result
1	NA	NA	NA	Normocellular, megakaryostic hyperplasia, small and dysplastic megakaryocytes	1.7 H	782.24 I	1.27 L	imatinib/dasatinib/nilotinib	pleural effusion with dasatinib	80	R/D
2	NA	NA	NA	Hypercellular, myeloid and megakaryocytic hyperplasia, M/E:10/1, small megakaryocytes, stage 1 reticulin fibrosis	1.1 I	1048.79 I	1.68 I	imatinib	None	6	R/A
3	0.158	0.36	0.953	megakaryocytic hyperplasia, small megakaryocytes, stage 1 reticulin fibrosis	5.3 H	1223.63 I	0.49 L	HU/anagralide/imatinib/nilotinib/bosutinib	None	101	R/A
4	100	100	23,54 *	Hypercellular, myeloid and megakaryocytic hyperplasia, stage 1 reticulin fibrosis	1.8 H	1453.71 I	0.50 L	HU/imatinib	None	37	R/A
5	57.12	3.066	0.01	Hypercellular, megakaryocytic hyperplasia, small megakaryocytes, stage 2 reticulin fibrosis	1.1 I	978.46 I	2.74 H	HU/imatinib	None	65	R/A
6	NA	NA	NA	NA	0.5 L	12.39 L	0.58 L	imatinib	None	29	R/A
7	36.03	40.54	56.54	Hypercellular, granulocytic and megakaryocytic hyperplasia, stage 1 reticulin fibrosis	1.2 I	762.99 L	1.18 L	HU/imatinib/nilotinib	None	88	R/A
8	4.53	2.283	0.34	Hypercellular, granulocytic and megakaryocytic hyperplasia, small megakaryocytes	0.8 I	302.89 L	0.75 L	imatinib	None	87	R/A
9	NA	NA	NA	NA	1.0 I	728.55 L	1.57 İ	imatinib	None	70	R/D
10	0.06	Negative	Negative	Hypercellular, granulocytic and megakaryocytic hyperplasia, small megakaryocytes, pleomorphic megakaryocytes	0.7 L	348.45 L	0.69 L	imatinib	None	55	R/A
11	NA	NA	NA	Hypercellular, granulocytic and megakaryocytic hyperplasia, small megakaryocytes, pleomorphic megakaryocytes	2.0 H	899.41 I	1.57 I	HU/imatinib	None	2	D**
12	6.780	30.93	0.10	Granulocytic hyperplasia	0.8 I	298.89 L	0.63 L	imatinib	None	85	R/A
13	Negative	Negative	Negative	Suboptimal	0.7 L	70.21 L	0.56 L	imatinib	None	40	R/A

** The fourth patient did not receive imatinib treatment regularly and remained BCR-ABL translocation positive until year 1. During follow-up, the patient turned negative after starting to take medication regularly.*

***Patient died of urosepsis at 2 months of treatment before response could be evaluated.*

PLT: Platelet count, Hgb: Hemoglobin, WBC: Leukocyte count, NEU#: Neutrophil count, LYMP#: Lymphocyte count, EOS#: Eosinophil count, BAS#: Basophil count, Ph: Philadelphia chromosome, A: Asymptomatic, NA: Not available, SM: Splenomegaly, H: High, I: Intermediate, L: Low, HU: Hydroxyurea, R/A: In

4. Discussion

Leukocytosis is a typical finding in patients with CML and they are usually diagnosed with leukocyte counts higher than $100x10^9$/L(11). Thrombocytosis is a common finding at the time of diagnosis of CML, but rarely exceeds 1000×10^9/L(5). In CML, the bone marrow is hypercellular and the myeloid to erythroid cell ratio is increased in favor of the myeloid series. Reticulin fibrosis may be seen in the bone marrow(12). Megakaryocytes are typically hypolobulated nuclei and smaller than normal(13).

In this study, the proportion of patients diagnosed with predominant thrombocytosis was 6%, the mean platelet count was $1068x10^9$/L ($582\text{-}2392x10^9$/L). No thrombotic events were observed. In this study, similar to the literature, megakaryocytes were small in size in seven patients (63%) and hypolobulated in five (45%) in addition, pleomorphic megakaryocytes were detected in two (18%) patients. Reticulin fibrosis was observed in five patients (45%). Two patients (18%) showed signs of dysplasia.

In the review by Findakly and Arslan, the mean age was 40.5 years and the mean platelet count was $1923x10^9$/L ($584\text{-}8688\ x10^9$/L). 50% of patients were asymptomatic. Women accounted for 65%. Splenomegaly was found in 13.3% of patients(8). Megakaryocytes in bone marrow were 71.4% small, 21.4% pleomorphic and 7.1% dysplastic. It is known that CML diagnosed with isolated thrombocytosis is more common in young people and in women(10). In this study, splenomegaly was more common in women and the rate of splenomegaly was similar to the literature. However, in contrast to the literature, the proportion of elderly patients was higher in this study. The mean age was 53.5 years (26-79) and 46% of patients were older than 60 years at diagnosis. The proportion of asymptomatic patients in our study was also high (93%).

Hydroxyurea may be used in the treatment of these patients with very high platelet values ($>1000x10^9$/L). Platelet apheresis can be performed in patients showing significant symptoms of leukostasis. Imatinib, nilotinib, dasatinib may be preferred as first-line treatment agents(14). Hydroxyurea treatment was given to five patients in this study. Imatinib was started in all patients after Ph chromosome was detected positive. In patients unresponsive to imatinib treatment, nilotinib and dasatinib were used. All the patients in the study were followed up for chronic phase CML. No patient progressed to accelerated phase or blastic phase.

Among the risk scores, the ELTS score was found to be low in 69% of the patients at the time of diagnosis. Hasford risk score was low in 53% and Sokal risk score was low in 23% of patients. Risk scores were not calculated in other studies. In this study, ELTS score was found to be associated with OS.

In patients with predominant thrombocytosis without marked leukocytosis, the Ph chromosome should definitely be checked before ET is diagnosed. One case from the literature(15) and one of our cases was negative for Ph chromosome by FISH and the patient was diagnosed with BCR-ABL fusion gene. Atypical transcripts and RNA sample of poor quality can also cause false negative results(16). Therefore, patients with isolated thrombocytosis should be assessed by bone marrow biopsy to avoid missing the diagnosis of CML. In addition, Ph chromosome along with JAK-2 should be requested in all patients presenting with thrombocytosis before CALR and MPL studies. p190 and p230 should definitely be checked in patients with negative p210 results.

5. Conclusion

This study showed that CML should be definitely considered in the differential diagnosis of patients with predominant thrombocytosis without marked leukocytosis. The diagnosis should include a bone marrow biopsy and genetic testing for the BCR-ABL fusion gene. In patients with small hypolobulated megakaryocytes and basophilia, CML rather than ET with large hyperlobulated megakaryocytes should be considered in the prediagnosis.

CML is a disease in which a average OS similar to the general population can be achieved with a daily, single, oral medication. Although it may present like ET at the time of diagnosis, they may be misdiagnosed if Ph is not requested. Patients from being mistakenly diagnosed and treated Ph-negative MPD which could result highly progressive clinical scenarios such as accelerated and blastic phases. In these patients, thrombotic events and bleeding disorders may occur. In this patient group, correct diagnosis and rapid initiation of tyrosine kinase inhibitor therapy may help prevent problems that may lead to serious morbidity and mortality.

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