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RESEARCH ARTICLES

Varian Transkrip Bcr-Abl1 pada Pasien Leukemia Mieloid Kronis: Hubungan dengan Parameter Hematologi Rutin

Bcr-Abl1 Transcript Variants in Chronic Myeloid Leukemia Patients: Relationship with Routine Hematological Parameters

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Abstract

Chronic myeloid leukemia (CML) was a myeloproliferative neoplasm characterized by the Philadelphia chromosome resulting from the BCR-ABL1 translocation. The most common BCR-ABL1 transcript variants were b2a2 and b3a2, which encoded the p210 protein with increased tyrosine kinase activity. This study was an analytical observational study with a cross-sectional design using retrospective data obtained from medical records. The study analyzed the relationship between BCR-ABL1 transcript variants and routine hematological parameters (hemoglobin, leukocyte count, and platelet count) among CML patients at Adam Malik General Hospital from 2020 to 2024 using a total sampling method. A total of 38 patients met the inclusion criteria, of whom 71,1% had the b3a2 variant and 28,9% had the b2a2 variant. Males predominated (60,5%), with a mean age of 28,37 ± 16,23 years. Leukocyte counts were significantly lower in patients with the b3a2 variant than in those with the b2a2 variant (p = 0,045), whereas hemoglobin and platelet levels showed no significant differences. A statistically significant relationship was found between leukocyte count and BCR-ABL1 transcript variants, while no significant relationship was observed between hemoglobin or platelet levels and transcript variants. The study highlighted the potential association between BCR-ABL1 transcript variants and leukocyte count in CML patients.

Keywords: *bcr-abl1 variants, chronic myeloid leukemia, hematological profile*

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Abstrak

Leukemia Mieloid Kronis (LMK) adalah neoplasma mieloproliferatif yang ditandai dengan kromosom Philadelphia akibat translokasi BCR-ABL1. Varian transkrip BCR-ABL1 yang paling umum adalah b2a2 dan b3a2, keduanya mengkode protein p210 dengan aktivitas tirosin kinase yang meningkat. Penelitian ini merupakan penelitian observasional analitik dengan desain potong lintang menggunakan data retrospektif yang diperoleh dari rekam medis, bertujuan untuk menganalisis hubungan antara varian transkrip BCR-ABL1 dan parameter hematologi rutin (hemoglobin, leukosit, trombosit) pada pasien LMK di Rumah Sakit Adam Malik dari tahun 2020 hingga 2024, dengan menggunakan metode total sampling. Dari 38 pasien yang memenuhi kriteria inklusi, 71,1% memiliki varian b3a2, dan 28,9% memiliki varian b2a2. Mayoritas pasien adalah laki-laki (60,5%), dengan usia rata-rata 28,37 ± 16,23 tahun. Jumlah leukosit pada varian b3a2 secara signifikan lebih rendah dibandingkan b2a2 *(p = 0,045)*, sementara kadar hemoglobin dan trombosit tidak menunjukkan perbedaan yang signifikan. Perbedaan yang signifikan secara statistik ditemukan antara kadar leukosit dan varian transkrip BCR-ABL1, namun tidak ditemukan hubungan yang signifikan antara kadar hemoglobin atau trombosit dengan varian transkrip BCR-ABL1. Temuan ini menyoroti potensi asosiasi antara varian transkrip BCR-ABL1 dan jumlah leukosit pada pasien LMK.

Kata Kunci: *varian bcr-abl1, leukemia mieloid kronis, profil hematologi*

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Highlight:

- The b3a2 variant is the most common BCR-ABL1 transcript found in Chronic Myeloid Leukemia (CML) patients, accounting for 71,1% of the study sample.
- There is a significant association between transcript variants and white blood cell levels (p-value = 0,045), where patients with the b2a2 variant exhibit higher leukocyte counts than those with the b3a2 variant.
- No statistically significant relationship was found between the BCR-ABL1 transcript variants and other hematological parameters, specifically hemoglobin levels and platelet counts.

INTRODUCTION

Chronic myeloid leukemia (CML) is a type of myeloproliferative neoplasm characterized by the presence of the Philadelphia chromosome, which results from a genetic alteration in a single hematopoietic stem cell. This mutation leads to uncontrolled proliferation and progressive accumulation of leukemic cells**.** Clinically, CML is classically divided into three phases chronic phase, accelerated phase, and blast crisis which reflect disease progression and biological aggressiveness rather than a simple time-based course (Provan, 2018).

CML accounts for approximately 15% of all adult leukemia cases, with a global incidence rate estimated at 0,87 per 100,000 individuals annually (Rajabto et al., 2022; Shah et al., 2024). The diagnosis median age is around 67 years, where the cases can occur among the age groups with higher prevalence among the males (Foucar, 2023;

Siegel et al., 2023).

The preliminary clinical assessment involves a thorough history, physical examination, and complete blood count. Diagnosis confirmed through cytogenetic and molecular techniques including reverse transcriptase polymerase chain reaction (RT-PCR) that showed the BCR-ABL1 fusion gene transcript (Rinaldi and Winston, 2023; Shah et al., 2024).

The ABL1 gene commonly breaks at exon a2, meanwhile the breakpoint on the BCR gene happen in different regions, there are: the major BCR (M-BCR), minor BCR (m-BCR), or micro BCR. The most frequently observed BCR-ABL1 transcript variants in CML are b2a2 (e13a2) and b3a2 (e14a2) from splicing exon 13 or 14 of the BCR gene to exon 2 of ABL1. These variants encode a 210-kDa fusion protein as p210 and found in > 95% of CML cases (Amin and Ahmad, 2021; Khazaal et al., 2019). Several studies demonstrated significant differences in hematologic profiles associated with these BCR-ABL1 transcript variants. Some studies show that b2a2 related to higher white blood cells, than b3a2 related to more platelets (Achkar et al., 2016; Vasconcelos et al., 2017). Meanwhile, other studies show opposite trends or no meaningful differences among variants (Laabidi et al., 2023; Morelos et al., 2024).

Evidence related to BCR-ABL1 transcript variants to age or sex limited and inconsistent. Several studies hint at differences among age groups or sexes, while the findings are inconclusive and reported less often than blood-related connection. The possible population of specific effects and more research is needed. This study to explore the BCR-ABL1 transcript variants related to routine hematological parameters (hemoglobin concentration, leukocyte count, and platelet count) and to describe the distribution among demographic characteristics in CML patients treated at Adam Malik General Hospital.

METHODS

The retrospective cross-sectional observational design employed to evaluate the BCR-ABL1 transcript variants and routine hematological parameters in patients' related to chronic myeloid leukemia (CML) at Adam Malik General Hospital. The study population included all patients diagnosed with CML in January 2020 and December 2024. The cases were collected in >5-year period and the variables were analyzed at a single time point per patient related to the baseline data obtained at initial diagnosis that fulfill cross-sectional design. A total sampling method applied including all patients who fulfil inclusion criteria.

The participants included patients with chronic-phase CML in 2020 WHO criteria confirmed positive for BCR-ABL1 and with complete baseline blood data. Hemoglobin, white blood cell, and platelet counts collected from routine lab tests at diagnosis before the treatment. Patients in blast phase or incomplete records were excluded. The study approved by Health Research Ethic Committee of Universitas Sumatera Utara (Ref: 1259/KEPK/USU/2024), and authorized by Adam Malik General Hospital (Ref: DP.04.03/D.XXVIII/8829/2024). Statistical analysis through SPSS and the data normality used Shapiro-Wilk test. Normally distributed variables analyzed using an independent t-test, while non-normally distributed data analyzed using the Mann-Whitney U test. A two-tailed *p-value* < *0,05* was considered statistically significant.

RESULTS AND DISCUSSIONS

There were 152 patients diagnosed with CML and screened for eligibility, while 38 of them fulfill inclusion and exclusion criteria. The demographic BCR-ABL1 profile and distribution transcript variants showed.

Table 1. Characteristics demographics and BCR-ABL1 variant distribution (n=38)

Characteristics Demographics	**n**
Type Gender, n (%)	
Male	23(60,5)
Female	15(39,5)
Age, year	
≥ 18 year	26(68,4)
< 18 years	12(31,6)
Average ± SD	28,37 ± 16,23
Median (Min – Max)	29,5(3–61)
Variants Transcript BCR- ABL1	**n = 38**
b2a2	11(28,9)
b3a2	27(71,1)

There 23 were male (60,5%) and 15 were female (39,5%) from 38 patients with male-to-female ratio of 1,53:1. The majority (68,4%) were adults aged 18 years or older, while 31,6% were children and adolescents <8 years old. The mean participants age was 28,37 ± 16,23 years that ranging from 3 to 61 years. Among the patients, the b3a2 variant detected in 27 patients (71,1%) and b2a2 variant detected in 11 patients (28,9%). None of the patients exhibited co-expression of both transcript types.

Table 2. Descriptive statistics of hematological parameters

Diagnosis	**n= 38**	***p****
Hemoglobin, g/dL		
Median (Min–Max)	8,3 (5,1)–13,7)	*0,008*
leukocyte count, thousand/µL		
Average ± SD	429,23 ± 236,3	*0,324*
Platelets, thousand/µL		
Median (Min–Max)	442,5 (76–2473)	*<0,001*

**Shapiro-Wilk*

The median hemoglobin level was 8,3 g/dL with 5,1 to 13,7 g/dL values**.** According to the Shapiro-Wilk test, hemoglobin levels were not normally distributed *(p = 0,008)*. The mean leukocyte 429,23 ± 236,3 × 10^3/µL. The Shapiro–Wilk test showed leukocyte counts followed a normal distribution (p = 0.324). Platelet counts showed a median 442,5×10^3/µL value 76 to 2473×10^3/µL range. The Shapiro–Wilk test demonstrated that platelet counts were not normally distributed *(p < 0.001)*. These findings show that different statistical approaches required for subsequent analyses related to data distribution.

Table 3. Hematological Parameters by Sex

Parameter	Type Sex		*p-value*
	Male	Female	
Hemoglobin, g/dL	8,37±1,93	8,29±1,95	0,903 [a]
leukocyte count, thousand/µL	407,36±221,2	462,75±261,09	0,488 [a]
Platelets	554 (76-2470)	343(113-1160)	0,026 [b]

Note: [a] Independent t-test, [b] Mann-Whitney, significant if the p-value < 0,05

The comparation of male and female patients, no statistically significant differences showed in hemoglobin *(p-value = 0,903)* or leukocyte counts *(p-value = 0,488)*. However, there was a significant difference in platelet counts with males having higher median levels (554 × 10^3/µL) than females (343 × 10^3/µL) with a *0,026 p-value* (Mann-Whitney test).

Comparison among transcript types showed leukocyte count: Patients with the b2a2 variant significantly higher leukocyte count (mean: 548.93 ± 289.06 × 10^3/µL) than b3a2 (mean: 402,26 ± 196,96×10^3/µL), *p-value = 0,045*. Hemoglobin and Platelets: No significant differences in hemoglobin or platelet levels among b2a2 and b3a2 groups *(p-value = 0,499* and *p-value = 0,115*, respectively).

Table 4. Hematological Parameters by Transcript Variant

Parameter	Variants Transcript BCR- ABL1		*p-value*
	b2a2	b3a2	
Hemoglobin	8,41±1,63	8,1 (5,1)–13,7)	0,499[b]
Leukocytes	548,93±289,06	402,26±196,96	0,045[a]
Trombosit	388,45±210,17	539 (76–2470)	0,115[b]

Note: [a] Independent t-test, [b] Mann-Whitney, significant if the p-value < 0,05

There are 38 patients with 60,5% were male and 39,5% were female with 1,53:1. The male predominance related to previous studies like Khazaals in Iraq with 58% males and 42% females (1,38:1) and Paramita et al. (2020) at Dr. Sardjito Hospital with male domination than female, there are 63,8% are male and 36,2% are female. The characteristic presented descriptively that sex was not a primary interest variable in the study

The higher CML prevalence in males hypothesized related with biological factors. Males may possess a greater hematopoietic stem cells (HSCs) number and increased spatial proximity among BCR and ABL loci, that potentially elevate chromosomal breaks risk and fusion events. Meanwhile, females may benefit from protective mechanisms like X chromosome inactivation, Y chromosome absence, and hormonal influences. Estrogen modulates hematopoietic proliferation, reduce oxidative stress, and enhance immune surveillance, while lowering BCR-ABL1 formation risk (Radivoyevitch et al., 2013; Zhao et al., 2025). Sex-related hormonal differences affect the hematopoietic lineages like megakaryopoiesis. Androgens showed to promote megakaryocyte activity and platelet production, whereas estrogen may exert differential modulatory effects on hematopoiesis. In CML context, thrombocytosis shows myeloid proliferation development and sex-related variations in disease biology affect to differences in hematological profiles. Further studies are needed to clarify the focus on mechanisms (Dupuis et al., 2019; Warren and Grossmann, 2022)

Data from the American Cancer Society (ACS) report the median age at diagnosis for CML in Western populations is 64 years with most cases in individuals aged >65

(ACS, 2024).

However, in several Asian countries, including China, India, the Philippines, and others showed the median age at diagnosis tends to be youngerfrom 36 to 55 years (Fentie et al. 2019; Rajabto et al., 2022). The patients were considerably younger with 28,37 ± 16,23 years mean age and 29,5 years median age . This age distribution is lower than that reported in most Asian populations and explained through pediatric and adolescent patients, referral patterns at the study center, and the limited sample size. Age was provided epidemiological context, but was not included in the primary analytical study objectives.

Regarding BCR-ABL1 transcript variants, where study confirmed b3a2 variant predominance in 71,1% of cases, than 28,9% for the b2a2 variant. These studies related to reports from several countries like India, Korea, Japan, Germany, and Brazil with b3a2 is commonly observed (Morelos et al., 2024; Vasconcelos et al., 2017). However, in Pakistan, United States, Argentina, and Sudan with the b2a2 appears to be more prevalent. These discrepancies may stem from differing methodologies, sample sizes, or underlying genetic diversity among populations.

Regarding hematological profiles, the median hemoglobin level was 8.33 g/dL, supporting prior literature that CML frequently presents with anemia. The result from suppressed erythropoiesis caused by excessive granulocytic cells proliferation, a hallmark of CML pathogenesis (Kaushansky et al., 2021).

There are no significant relation was observed among transcript variants and hemoglobin levels, where suggest that anemia in CML affected by multifactorial mechanisms beyond BCR-ABL1 transcript type**.**

The average leukocyte count was 400,67 ± 236,3×10³/µL related to the CML well-established pathophysiology, where deregulated leukocyte count proliferation affect to significant leukocytosis. The BCR-ABL fusion gene encodes a constitutively active tyrosine kinase, which continuously triggers intracellular signal, promote cell growth, block apoptosis, connect hematopoietic differentiation, and reduce cellular adhesion to the bone marrow niche that drive CML development (Cortes et al., 2021; Provan, 2018; Vasconcelos et al., 2017).

The most notable finding was the statistically significant difference in leukocyte counts among transcript variants with the b2a2 variant related to higher leukocyte levels compared to b3a2 *(p = 0,045)*. The study suggest that BCR-ABL1 transcript variants differentially influence leukocyte proliferation pathways**.** The study related to baseline hematological data obtained at a single time point. In line with Vasconcelos et al showed b2a2 connected to elevated leukocyte counts (Vasconcelos et al., 2017).

Meanwhile, inconsistent with the global research. Khazaal et al. (2019) showed significantly higher leukocyte counts in patients with the b3a2 variant *(p = 0,031)*. Kagita et al. showed leukocyte counts development in b3a2 cases, though the difference was not statistically significant *(p = 0,757)*. Morelos et al. (2024) showed higher leukocyte levels in b3a2 without significance *(p = 0,46)*.

These conflicting findings affected by variations in disease phase at diagnosis, ethnic background, sample size, laboratory methodologies, or treatment status in assessment. The inconsistencies underline the complex genotype–phenotype relationship in CML and indication of transcript type not determine leukocyte proliferation.

The median count in platelets was 442,5×10³/µL that range widely among patients. The elevated platelet levels observed in several cases are increased megakaryopoiesis that influenced by signaling pathways activated by the BCR-ABL1

fusion (Cortes et al., 2021; Provan, 2018).

There is no significant relation among transcript variants and platelet counts that suggest platelet production modulated by additional clinical and biological factors like disease stage, inflammatory status, and bone marrow compensation mechanisms.

The study contributes to the evidence through a significant relation among b2a2 transcript variant and leukocyte counts in this population, while underlining heterogeneity of hematologic manifestations in CML. Further large-scale and multicenter studies are warranted to clarify the biological mechanisms underlying transcript-specific effects on hematopoietic lineages.

The limitations of study, where total sampling in 2020–2024 period, the sample size was relatively small (38 patients) that develop selection bias risk and reduce the statistical study contribution. In addition, data were collected from a single tertiary referral hospital that limiting the generalizability of the findings to broader populations. The retrospective cross-sectional design precludes causal assessment relationships and changes in hematological parameters over time.

CONCLUSIONS

The study demonstrated a statistically significant relation BCR-ABL1 transcript variants and leukocyte counts in chronic myeloid leukemia patients. Specifically, individuals with the b2a2 variant exhibited higher leukocyte levels than those with the b3a2 variant. However, no meaningful differences were observed in hemoglobin or platelet counts across the transcript groups.

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REFERENCES

Achkar, W., Moassass, F., Youssef, N., Wafa, A., 2016. Correlation of P210 BCR-ABL Transcript Variants with Clinical, Parameters and Disease Outcome in 45 Chronic Myeloid Leukemia Patients. Journal of Balkan Union of Oncology 21(2), 444–449. https://pubmed.ncbi.nlm.nih.gov/27273956/

(ACS) American Cancer Society., 2024. Key Statistics for Chronic Myeloid Leukemia (CML) [WWW Document]. Https://Www.Cancer.Org/Cancer/Types/Chronic-Myeloid-Leukemia/About/Statistics.Html. [Accessed July 2025].

Amin, H., Ahmed, S., 2021. Characteristics of BCR–ABL Gene Variants in Patients of Chronic Myeloid Leukemia. Open Medicine 16(1), 904-912. https://doi.org/10.1515/med-2021-0309

Cortes, J., Pavlovsky, C., Saußele, S., 2021. Chronic Myeloid Leukaemia. Lancet

20(392), 1914-1926. https://www.thelancet.com/journals/lancet/article/PIIS0140-6736(21)01204-6/abstract

Dupuis, M., Severin, S., Esclassan, E.N., Arnal, J.F., Payrastre, B., Valéra, M., 2019. Effects of Estrogens on Platelets and Megakaryocytes. International Journal of Molecular Sciences 20(12), 1-10. https://doi.org/10.3390/ijms20123111

Fentie, A.M., Tadesse, E., Engidawork, A., Gebremedhin, A., 2019. Prevalence and Determinants of Nonadherence to Imatinib in The First 3-Months Treatment Among Newly Diagnosed Ethiopian's with Chronic Myeloid Leukemia. Plos One14(3), 1–12. https://doi.org/10.1371/journal.pone.0213557

Foucar, K., 2023. Diagnostic Pathology: Blood and Bone Marrow. Elsevier, Philadelphia.

Kaushansky, K., Prchal, J.T., Lichtman, M.A., Levi, M., Linch, D.C., 2021. Williams Hematology. Mcgraw-Hill, New York.

Khazaal, M., F. Hamdan, F.B., Mayah, Q.S.A., 2019. Association of BCR/ABL Transcript Variants with Different Blood Parameters and Demographic Features in Iraqi Chronic Myeloid Leukemia Patients. Molecular Genetics and Genomic Medicine 7(8), 1-9. https://doi.org/10.1002/mgg3.809

Laabidi, B., Slama, N., Ouahchi, I., Boufirkha, W., Laatiri, M.A., 2023. Chronic-Phase Chronic Myeloid Leukemia: Incidence of BCR/ABL Transcript and Its Correlation with Presenting Features, Response to Treatment, and Survival. Leukimia Research Reports 20, 1-12. https://doi.org/10.1016/j.lrr.2023.100373

Morelos, P.R., Yebra, A.L.G., Garcia, A.H., Velaszquez, F.A.R., Rosario, L.J.B., Yebra, B.G., 2024. Distribution of BCR ABL1 Transcripts in The Different Clinical Phases of Chronic Myeloid Leukemia: Effect on Hematological Parameters and Patient Survival. Genes 15(5), 1-10. Doi:10.3390/Genes15050567

Paramita, D.K., Hutajulu, S.H., Syifarahmah, A., Sholika, T.A., Fatmawati, S., Aning, S., Sulistyawati, D., Wahyuni, S., Hariadi, K.W.T., Kurnianda, J., 2020. BCR-ABL Gene Transcript Types of Patients with Chronic Myelogenous Leukemia in Yogyakarta, Indonesia. Asian Pacific Journal of Cancer Prevention 21(6), 1545-1550. Doi: 10.31557/APJCP.2020.21.6.1545

Provan, D., 2018. ABC of Clinical Haemotology. John Wiley & Sons, Hoboken.

Radivoyevitch, T., Jankovic, G.M., Tiu, R.V., Sauntharajah, Y., Jackson, R.C., Hlatky, L.R., Gale, R.P., Sachs, R.K., 2013. Sex Differences in The Incidence of Chronic Myeloid Leukemia. Radiation and Environmental Biophys 53, 55-63. Doi: 10.1007/S00411-013-0507-4

Rajabto, W., Rexodiputro, A.H., Rinaldi, I., Tadjoedin, H., Priantono, D., Angkasa, Y.H., 2022. Chronic Myeloid Leukemia (CML) at National Referral Hospital in Indonesia. Siriraj Medical Journal 74(8), 530-536. https://he02.tci-thaijo.org/index.php/sirirajmedj/article/view/258796

Rinaldi, I., Winston, K., 2023. Chronic Myeloid Leukemia, from Pathophysiology to Treatment-Free Remission: A Narrative Literature Review. Journal of Blood Medicine 6(14), 261-277. Doi: 10.2147/JBM.S382090

Shah, N.P., Bhatia, R., Altman, J.K., Amaya, M., Begna, K.H., Berman, E., Chan, O., Clements, J., Collins, R.H., Curtin, P.T., DeAngelo, D.J., Drazer, M., Maness, L., Metheny, L., Mohan, S., Moore, J.O., Oehler, V., Pratz, K., Pusic, I., Rose, M.G., Shomali, W., Smith, B.G., Styler, M., Talpaz, M., Tanaka, T.N., Tantravahi, S., Thompson, J., Tsai, S., Vaughn, J., Welborn, J., Yang, D.T., Sundar, H., Gregory, K., 2024. Chronic Myeloid Leukemia, Version 2.2024, NCCN Clinical Practice in Oncology. Journal of the National Compherensive Cancer Network 22(1), 43-69.

https://pubmed.ncbi.nlm.nih.gov/38394770/

Siegel, R.L., Miller, K.D., Wagle, N.S., Jemal, A., 2023. Cancer Statistics, 2023. A Cancer Journal for Clinical 73(1),17–48. https://doi.org/10.3322/caac.21763

Vasconcelos, A.P., Azevedo, I.F., Melo, F., Neves, W.B., Azevedo, A., Melo, R., 2017. BCR- ABL1 Transcript Types Showed Distinct Laboratory Characteristics in Patients with Chronic. Genetic and Molecular Research 16(2), 1-9. https://doi.org/10.4238/gmr16029541

Warren, A.M., Grossmann, M., 2022. Haematological Actions of Androgens. Best Practice and Research Clinical Endocrinology & Metabolism 36(5), 1-11. https://doi.org/10.1016/j.beem.2022.101653

Zhao, J., Yu, Y., Liu, C., Liu, R., Sun, M., Zhuang, J., Sun, C., Wu, Q., 2025. Elucidating The Role of Estrogen Effects in Leukemia: Insights from Single-Cell RNA Sequencing and Mendelian Randomization. Journal of Cancer 16(3), 888-897. Doi: 10.7150/Jca.100610