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A Case of B-Lineage Acute Lymphoblastic Leukemia with Blast Cell Using the White Precursor Cell (WPC)

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Abstract

Background: Acute Lymphoblastic Leukemia (ALL) is a blood cancer driven by clonal proliferation of immature lymphoid precursors in the bone marrow and peripheral blood. One key cytogenetic feature is the Philadelphia chromosome (Ph), formed through t(9; 22)(q34;q11.2), which generates the BCR-ABL1 fusion gene. The p210 isoform is mainly associated with Chronic Myeloid Leukemia (CML) and occurs in more than 90% of cases. **Objective:** This case report describes a patient with BCR-ABL1 p210-positive B-lineage acute lymphoblastic leukemia (B-ALL) who initially presented with clinical features suggestive of blast-phase chronic myeloid leukemia (CML). The case highlights the potential role of the White Precursor Cell (WPC) channel of the Sysmex XN-series analyzer in supporting blast lineage characterization during the diagnostic evaluation. **Presentation:** A 31-year-old female presented to the hospital with complaints of dizziness and nausea. The dizziness had been present for one month prior to hospital admission. A diagnosis of blast-phase Chronic Myeloid Leukemia had been established two weeks earlier, based on detection of the BCR-ABL1 (p210) fusion gene. Additional diagnostic workup, including peripheral blood evaluation, bone marrow aspiration, and immunophenotyping, indicated B-cell Acute Lymphoblastic Leukemia (B-ALL). **Result:** In this patient, the WPC scattergram demonstrated a blast population with large cell size and high RNA content, consistent with a lymphoid lineage, which was subsequently confirmed by bone marrow examination and immunophenotyping. **Conclusion:** This case highlights the potential utility of the WPC parameter as an adjunct to the diagnostic evaluation of acute leukemia, particularly when differentiating lymphoid from myeloid blast populations before confirmatory testing.

Keywords: B-Acute Lymphoblastic Leukemia (B-ALL), Blast cell, Sysmex XN, White Precursor Cell (WPC)

Case Report

INTRODUCTION

Acute Lymphoblastic Leukemia (ALL) is a cancer of the hematopoietic system marked by abnormal clonal growth of early-stage lymphoid cells in both the bone marrow and bloodstream. (Rahmat et al., 2022; Suwandari et al., 2023). Although notable progress has been achieved in hematologic cancer management, leukemia continues to impose a considerable burden on global health systems. According to estimates from 2020, leukemia was among the most common causes of cancer morbidity and mortality, ranking 15th in incidence and 11th in cancer-related deaths worldwide. (Du et al., 2022; Hu et al., 2024). Recent epidemiological data indicate that the global incidence of Acute Lymphoblastic Leukemia cases increased modestly from 98,149 in 1990 to 103,727 in 2021, with prevalence also rising from 237,056 to 386,813 (Fan et al., 2025).

The lifetime risk of developing Acute Lymphoblastic Leukemia (ALL) is estimated to be approximately 1 in 1,000 individuals. Although the overall incidence of ALL is lower compared to other types of cancer, the incidence is highest among children under five years of age. This disease is the most common form of leukemia in children, with most cases occurring in pediatric and young adult populations, whereas the highest mortality rates are observed among adults (Ciesielska et al., 2024).

The disease course and aggressiveness of Acute Lymphoblastic Leukemia (ALL) are strongly influenced by underlying genetic and molecular abnormalities involved in the leukemogenesis process. One of cytogenetic abnormality is the Philadelphia chromosome (Ph), resulting from the translocation t(9;22)(q34;q11.2), which gives rise to the BCR-ABL1 fusion gene. In ALL, its presence is associated with a more aggressive phenotype and worse prognosis. BCR-ABL1 exists in multiple isoforms (p190, p210, and p230), with p190 being the most common in ALL. The p210 variant is predominantly linked to CML and is detected in over 90% of cases, although clinical differences between p190 and p210 remain unclear. (Hamad, 2022; Pepeler et al., 2026). The p230 isoform can be found in cases of Chronic Neutrophilic Leukemia (CNL) (Altaf et al., 2021).

Here, we report a rare case of de novo Philadelphia-positive B-lineage acute lymphoblastic leukemia (B-ALL) with a p210 BCR-ABL1 b3a2 transcript that initially mimicked CML in blast crisis, highlighting the diagnostic challenge posed by the shared BCR-ABL1 abnormality and the potential supportive value of the White Precursor Cell (WPC) channel in the initial characterization of blast lineage.

CASE PRESENTATION

A 31-year-old female presented to the hospital with complaints of dizziness and nausea. The dizziness had been present for one month prior to hospital admission. There was no family history of malignancy. A diagnosis of Chronic Myeloid Leukemia in blast crisis had been established two weeks prior, supported by laboratory findings indicating a positive BCR-ABL1 fusion transcript corresponding to the p210 isoform (b3a2, major breakpoint), detected using a qualitative reverse-transcription multiplex polymerase chain reaction (RT-multiplex PCR) assay. A quantitative BCR-ABL1 transcript level expressed as a percentage on the International Scale (%IS) was not available.

On physical examination, the patient appeared moderately ill, with *compos mentis*, with Eye 4, Verbal 5, Motoric 6. The patient's vital parameters were recorded as blood pressure about 135/90 mmHg, heart rate 94 beats per minute, respiratory rate 20 breaths per minute, and temperature was 36.6°C. Head examination revealed pale conjunctiva consistent with anemia. Cervical examination demonstrated lymphadenopathy. Cardiopulmonary examination was within normal limits. Abdominal examination revealed no palpable hepatomegaly or splenomegaly. Examination of the extremities showed no edema.

Complete blood count on January 11th, 2026, when the patient was admitted to the hospital, the following results were obtained: hemoglobin 6.2 gr/dL, MCV 67.6 μ m³, MCH 23.3 pg, MCHC 34.6 g/dL, Erythrocytes: 2.65 $10^6/\mu$ L, RDW-CV 19.2%, Hematocrit 17.9%, Leukocytes 159,050/cmm, platelets 14,000/cmm, reticulocytes 0.5%.

The patient received a transfusion of three units of blood, after which laboratory examinations were performed. Complete blood count on January 12th, 2026, when the patient was admitted to the hospital, the following results were obtained: hemoglobin 8.5 gr/dL, MCV 74.4 μm^3 , MCH 26.1 pg, MCHC 35.0 g/dl, Erythrocytes: $3.26 \times 10^6/\mu\text{l}$, RDW-CV 19.6%, Hematocrit 24.3%, Leukocytes 135,490/cmm, platelets 33,000/cmm, reticulocytes 0.8%, differential count = Eosinophil/Basophil/Band/Segment/Lymphocyte/Monocyte = 0/0/2/20/45/6; blast cell: 27%

The patient received a transfusion of two blood units again, after which laboratory examinations were performed. Complete blood count on January 14th, 2026, when the patient was admitted to the hospital, the following results were obtained: hemoglobin 10.2 gr/dL, MCV 73.5 μm^3 , MCH 24.8 pg, MCHC 33.6 g/dl, Erythrocytes: $4.13 \times 10^6/\mu\text{l}$, RDW-CV 19.7%, Hematocrit 30.4%, Leukocytes 48,400/cmm, platelets 10,000/cmm, reticulocytes 0.7%, differential count = Eosinophil/Basophil/Band/Segment/Lymphocyte/Monocyte = 0/0/3/41/45/5; blast cell: 30%

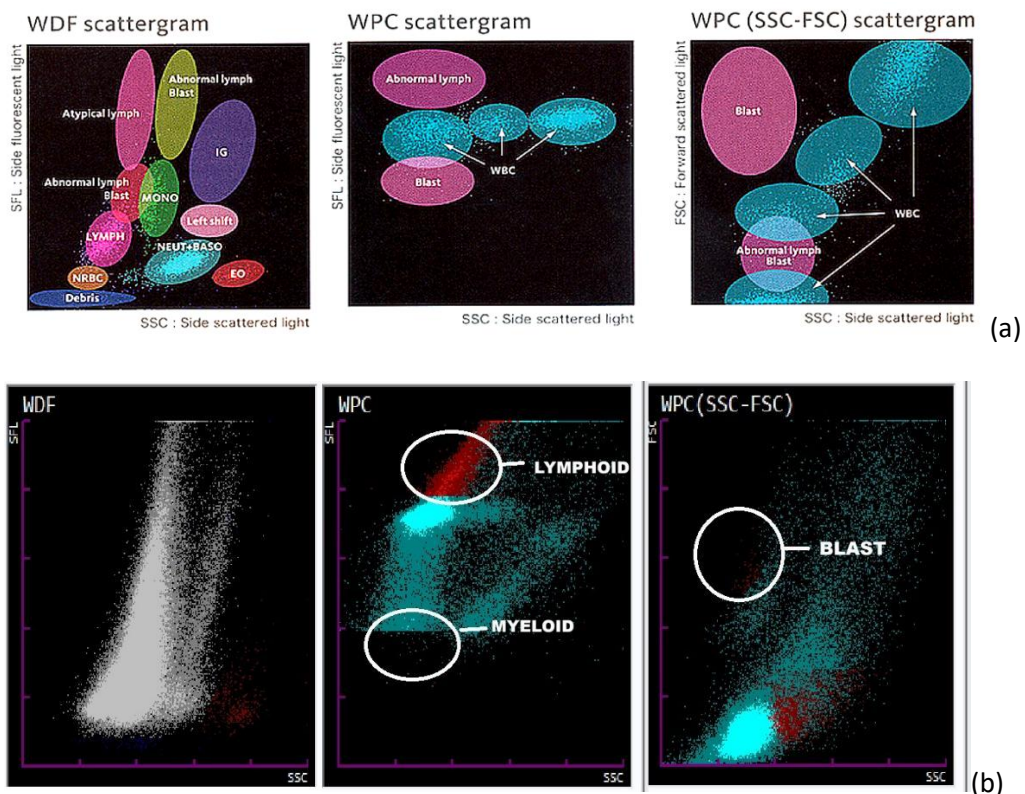


Figure 1. (a) Sysmex XN-series flagging algorithm. If no abnormal leukocytes are detected, no WPC flag is generated (adapted from Werner et al.). (b) WPC scattergram of the patient on January 12, 2026, demonstrating a red-colored blast cell population located in the high side fluorescence light (SFL) region, indicating high nucleic acid content consistent with lymphoid blasts. No myeloid blast population was observed in this patient.

Bone marrow aspiration performed on January 15, 2026, showed hypercellular marrow with a myeloid-to-erythroid (M) ratio of 4:1. Erythropoiesis was decreased, whereas granulopoiesis was increased. The bone marrow aspirate was predominantly composed of blast cells (79%) with lymphoblastoid morphology. The blasts were relatively small and morphologically homogeneous, with scant-to-moderate agranular cytoplasm, relatively high nuclear-to-cytoplasmic ratios, finely dispersed to slightly clumped chromatin, and inconspicuous nucleoli. Thrombopoiesis was decreased. Overall, the bone marrow findings were consistent with acute lymphoblastic leukemia (ALL), FAB subtype L1. Cytochemical staining, including myeloperoxidase (MPO) and periodic acid–Schiff (PAS), was not performed. The diagnosis was established based on bone marrow morphology, flow cytometric immunophenotyping, and molecular detection of the BCR-ABL1 fusion transcript.

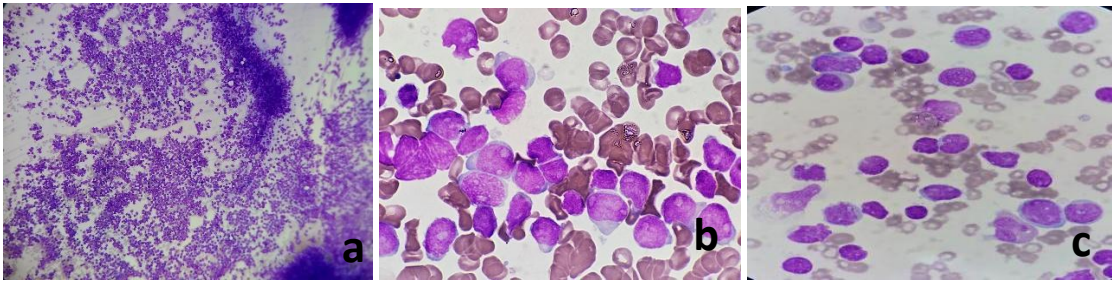
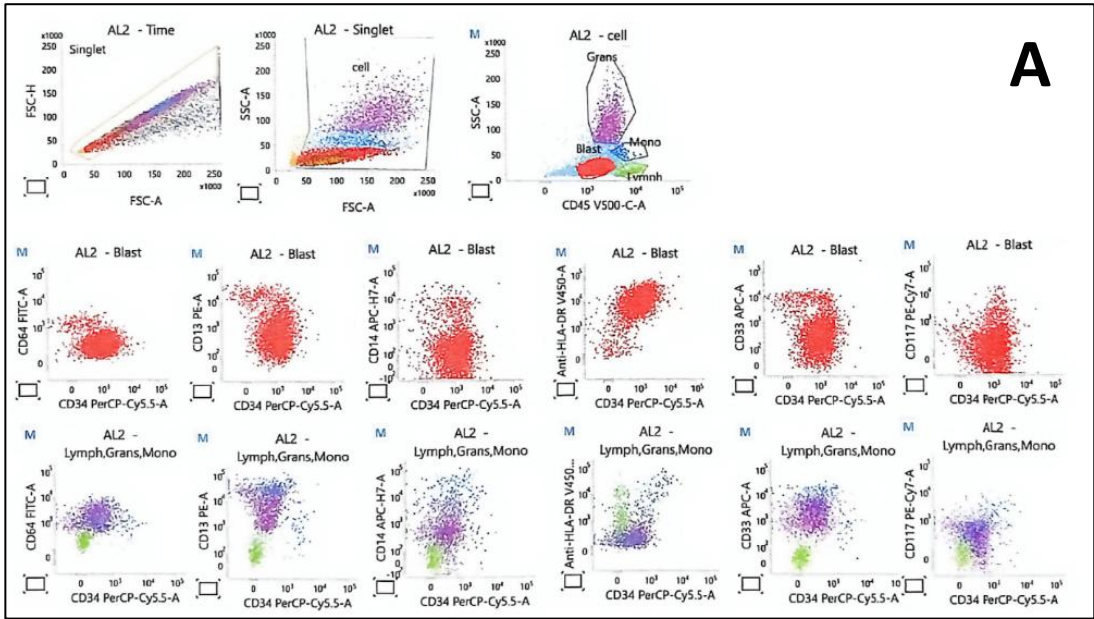


Figure 2. Cellularity was hypercellular (a), with the bone marrow predominantly composed of blast cells demonstrating homogeneous lymphoblastoid morphology. (b, c)

Immunophenotyping was performed on the bone marrow specimen on January 15th, 2026, and demonstrated that the blast population expressed CD19, CD10, CD34, and HLA-DR, confirming B-lineage acute lymphoblastic leukemia (Figure 3). The blasts were negative for T-cell markers, including CD3, CD4, and CD8.

Flow cytometric immunophenotyping was performed on the bone marrow specimen on January 15th, 2026. The blast population expressed CD19, CD10, CD34, and HLA-DR, confirming B-lineage acute lymphoblastic leukemia. The blasts were negative for T-cell markers, including CD3, CD4, and CD8. Aberrant expression of the myeloid-associated antigens CD13 and CD33 was also observed. Flow cytometric immunophenotyping was performed on the bone marrow specimen on January 15th, 2026. The blast population expressed CD19, CD10, CD34, and HLA-DR, confirming B-lineage acute lymphoblastic leukemia. The blasts were negative for T-cell markers, including CD3, CD4, and CD8. Aberrant expression of the myeloid-associated antigens CD13 and CD33 was also observed.



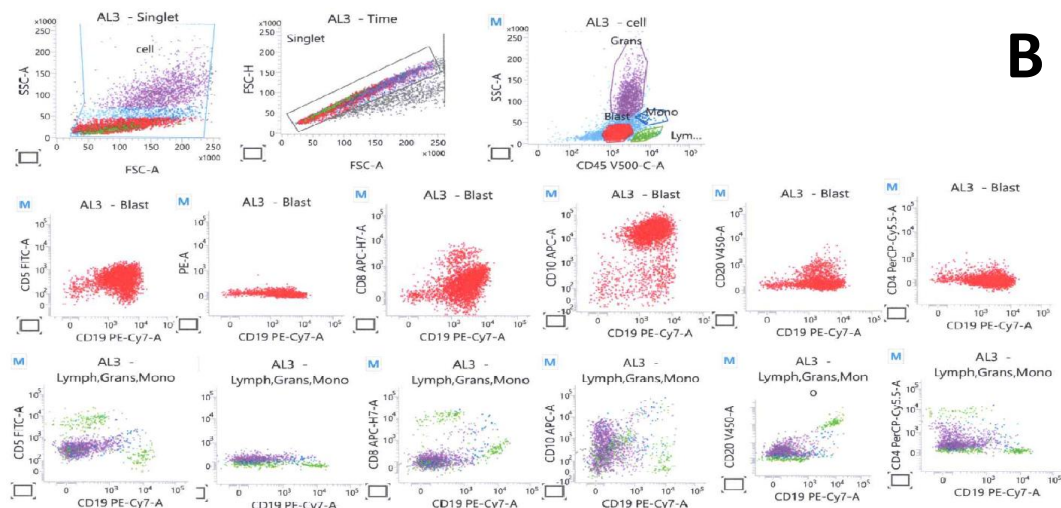


Figure 3. Flow cytometric immunophenotyping. (A) Representative plots showing expression of CD34, HLA-DR, CD13, CD33, and CD117, indicating aberrant expression of myeloid-associated antigens. (B) Representative plots demonstrating positivity for CD19 and CD10 and negativity for T-cell markers (CD3, CD4, and CD8), confirming B-lineage acute lymphoblastic leukemia.

The patient was started on a 3-day Acute Lymphoblastic Leukemia (ALL) treatment protocol, which included cyclophosphamide 300 mg/m² administered intravenously every 12 hours, followed by vincristine 2 mg IV on day 4, doxorubicin 50 mg/m² IV on day 4, and dexamethasone 40 mg/day given orally or intravenously on days 1–4. The patient also received additional therapy with the tyrosine kinase inhibitor Imatinib at a dose of 600 mg per day.

After the patient received chemotherapy, laboratory evaluations were performed. A complete blood count on January 19th, 2026, revealed the following results: hemoglobin 10.8 g/dL, MCV 75.2 fL, MCH 25.5 pg, MCHC 34.3 g/dL, erythrocytes 4.25 × 10⁶/μL, RDW-CV 20.5%, hematocrit 31.2%, leukocytes decreased into 18,100/mm³, platelets 50,000/mm³, and reticulocytes 0.8%. The differential count of blast cells decreased to 8%. The patient was subsequently referred to a tertiary referral hospital for definitive management. No additional follow-up data or treatment outcomes were available because the patient continued to receive care at the referral center.

DISCUSSION

Leukemia remains an important cause of cancer-related morbidity and mortality worldwide. Adult acute lymphoblastic leukemia (ALL) accounts for approximately 20% of acute leukemia cases and requires prompt diagnosis because of its aggressive clinical course (Jerez et al., 2024).

A hallmark genetic feature of chronic myeloid leukemia (CML), the Philadelphia chromosome (Ph+), is present in more than 90% of cases, but is only identified in about 20–30% of acute lymphoblastic leukemia (ALL) (Azambuja et al., 2024; Budiyo et al., 2024). This abnormality produces the p210 BCR-ABL1 fusion gene through a translocation involving chromosomes 9 and 22, specifically between the ABL1 and BCR genes. In ALL, the p190 isoform predominates, whereas p210 is comparatively rare. Consequently, Ph+ ALL represents a less common subtype of the disease. However, when present, it is associated with prognosis (Raharjo et al., 2025; Haider et al., 2022; Shah et al., 2024).

Cases of acute lymphoblastic leukemia (ALL) with Philadelphia chromosome positivity should be differentiated from chronic myeloid leukemia (CML) in blast crisis. The distinction can be made based on several aspects: (1) clinical history—ALL typically presents with an acute onset, with symptoms developing within less than 3 months, whereas CML in blast crisis is usually preceded by a chronic phase with symptoms lasting more than 3 months; (2) physical examination—splenomegaly is more commonly observed in CML blast crisis compared to ALL; and (3) laboratory findings—complete blood

count may reveal increased granulocytic precursors, including basophilia and eosinophilia, in CML (Momen et al., 2022; Shah et al., 2024).

In blast crisis phase Chronic Myeloid Leukemia (CML), the characteristic findings of eosinophilia and basophilia may be absent despite their well-recognized association with the disease, particularly during the chronic and accelerated stages. As CML progresses to blast transformation, the hematologic profile often shifts toward an overwhelming expansion of blast cells, which can obscure or diminish previously evident increases in eosinophils and basophils. Consequently, the diagnosis of blast crisis relies primarily on the presence of $\geq 20\%$ blasts in the peripheral blood or bone marrow, evidence of extramedullary blast proliferation, and confirmation of the BCR-ABL1 fusion gene, rather than on differential leukocyte abnormalities alone. Patients may present with marked leukocytosis, normal or reduced white blood cell counts, and frequently develop thrombocytopenia, while eosinophilia and basophilia may persist, decrease, or be entirely absent. Therefore, the lack of these features should not preclude a diagnosis of CML blast crisis, especially in individuals with a documented history of CML, detection of the Philadelphia chromosome or BCR-ABL1 rearrangement, and morphologic or immunophenotypic findings consistent with blastic transformation. Moreover, distinguishing *de novo* blast crisis phase CML, in which the disease is first recognized at the blast stage, from BCR-ABL1 positive Acute Myeloid Leukemia (AML) remains clinically important, as both entities can exhibit highly similar clinical, morphologic, and immunophenotypic characteristics, and the presence or absence of eosinophilia and basophilia is often of limited diagnostic value in differentiating between them (Pamuk & Ehrlich, 2024; Senapati et al., 2023; Wang et al., 2021).

In the present case, several findings favored a diagnosis of *de novo* Philadelphia chromosome-positive B-lineage acute lymphoblastic leukemia (Ph-positive B-ALL) rather than chronic myeloid leukemia (CML) in lymphoid blast crisis. The patient presented with a relatively short clinical history of approximately one month and had no documented history of chronic-phase CML. Physical examination revealed no splenomegaly, and the peripheral blood examination showed no eosinophilia or basophilia. The peripheral blood differential showed 27% blasts on January 12, 2026, increasing to 30% on January 14, 2026. Bone marrow aspiration performed on January 15, 2026, demonstrated 79% blasts with lymphoblastoid morphology. Flow cytometric immunophenotyping showed expression of HLA-DR, CD34, CD10, and CD19, confirming B-lineage acute lymphoblastic leukemia. Although the detection of the BCR:ABL1 p210 fusion transcript initially raised suspicion for CML in lymphoid blast crisis, the absence of a prior CML history, together with the clinical, hematologic, morphologic, and immunophenotypic findings, supported a diagnosis of *de novo* Ph-positive B-ALL. Following initiation of chemotherapy, the peripheral blood blast percentage decreased to 8%.

The Sysmex XN-series hematology analyzer includes a White Precursor Cell (WPC) channel, which is designed to assist in differentiating neoplastic cells from atypical lymphocytes using the Lysercell WPC reagent. The reagent lyses erythrocytes and platelets while permeabilizing leukocyte membranes, allowing fluorescent dyes to bind intracellular nucleic acids. Consequently, leukocyte populations can be characterized according to their fluorescence intensity. In one study by Ratnasari et al. (2020), the WPC channel demonstrated a sensitivity of 93.75% and a specificity of 94.74% for blast lineage assessment. However, these findings should be interpreted cautiously because they were derived from a single study. In the present case, the WPC channel provided supportive information for blast lineage characterization, whereas the definitive diagnosis was established by bone marrow examination and immunophenotyping.

Peripheral blood examination in this case revealed an increased population of blast cells of lymphoblast morphology, without evidence of eosinophilia or basophilia. Immunophenotyping of the peripheral blood demonstrated positivity for HLA-DR, CD34, CD10, and CD19, consistent with B-lineage acute lymphoblastic leukemia (B-ALL). In this patient, analysis using the Sysmex XN-series hematology analyzer assisted in blast cell to differentiate between myeloid and lymphoid lineages. In the present case, distinction between blast cells and atypical lymphocytes was facilitated by evaluation of the WPC scattergram, particularly in the forward scatter light (FSC) region, which reflects larger cell size (blast population). Differentiation of blast lineage can be further evaluated in the side fluorescence light (SFL) region of the WPC scattergram (Ningombam et al., 2021; Werner et al., 2016). The White Precursor

Cell (WPC) channel enables preliminary differentiation between myeloid and lymphoid blast populations. Blast cells are initially identified on the Forward Scatter (FSC) channel, where they are in the upper region due to their larger cell size (Figure 1b). After the blast population has been identified, lineage differentiation can be assessed using the Side Fluorescence Light (SFL) and Side Scatter (SSC) parameters. Lymphoid blasts are characterized by high RNA content, resulting in increased SFL intensity, together with low cellular complexity, reflected by low SSC signals. In contrast, myeloid blasts exhibit lower RNA content, corresponding to lower SFL intensity, while maintaining relatively low SSC signals because of their low cellular complexity.

In the present case, the blasts also expressed CD13 and CD33, representing aberrant expression of myeloid-associated antigens in B-lineage acute lymphoblastic leukemia (B-ALL). Aberrant expression of myeloid antigens is a well-recognized immunophenotypic feature of B-ALL, particularly in Philadelphia chromosome-positive (BCR-ABL1-positive) cases, and does not exclude the diagnosis of B-lineage ALL when lineage-defining B-cell markers are present. Although these markers may initially raise suspicion for myeloid leukemia, the final diagnosis should be established by integrating morphology, immunophenotyping, cytogenetic, and molecular findings (Liao et al., 2023).

The prognostic significance of aberrant CD13 and CD33 expressions remains controversial. A large real-world study involving 1,005 adult patients with B-ALL demonstrated that CD13/CD33 expression was associated with a higher frequency of BCR: ABL1 rearrangement and measurable residual disease (MRD) positivity; however, it did not adversely affect complete remission, relapse-free survival, or overall survival. Furthermore, patients receiving tyrosine kinase inhibitor (TKI)-based therapy showed favorable long-term outcomes despite CD13/CD33 expression. Therefore, in the current era of TKI-based treatment, aberrant expression of CD13 and CD33 appears to have greater value as a diagnostic and disease-monitoring marker than as an independent adverse prognostic factor (Liao et al., 2023).

The BCR-ABL1 translocation with p210 isoform expression is identified in approximately 30% of adult patients with Philadelphia chromosome-positive acute lymphoblastic leukemia (Ph+ ALL), although the p190 isoform is more commonly observed in this subtype. In a study conducted by Ting Shi et al. (2022), it was reported that differences between BCR-ABL p190 and p210 in Ph+ ALL patients indicate that the p210 variant tends to exhibit clinical characteristics such as higher white blood cell counts, platelet counts, and BCR-ABL1 transcript levels. At the molecular level, p210 more frequently harbors mutations associated with chemotherapy resistance and poor prognosis, including T315I and E255K/V. In terms of therapeutic response, both isoforms demonstrate comparable complete remission rates; however, p190 is associated with a more rapid molecular response and a higher likelihood of achieving minimal residual disease (MRD)-negative status compared to p210. From a prognostic perspective, p210 is associated with inferior overall survival and serves as an independent risk factor for poorer outcomes, although interventions such as allogeneic hematopoietic stem cell transplantation may improve prognosis (Bogdanov et al., 2025; Mohamed et al., 2024). Furthermore, these isoforms differ biologically in their signaling pathways, kinase activity, and gene expression profiles, reflecting distinct cellular origins, with p210 arising from more primitive hematopoietic stem cells and p190 originating from more differentiated B-cell lymphoid progenitors (Shi et al., 2022).

The appropriate therapy in acute lymphoblastic leukemia (ALL) is essential, and assessment of BCR-ABL1 mutation status is required to determine the presence of the chromosomal translocation between chromosomes 9 and 22. In cases where BCR-ABL1 is detected in ALL, additional treatment with tyrosine kinase inhibitors such as Imatinib, Dasatinib, or Nilotinib may be administered (Outersterp et al., 2024; Shah et al., 2024). In this patient, the presence of a BCR-ABL1 mutation with the p210 isoform prompted the addition of imatinib therapy, which was associated with a reduction in leukocyte count and peripheral blood blast cells.

CONCLUSION

A case of BCR-ABL1 p210-positive B-lineage acute lymphoblastic leukemia initially presenting with features suggestive of blast-phase chronic myeloid leukemia has been reported. In this patient, the White Precursor Cell (WPC) channel provided supportive information for blast lineage characterization

before confirmation by bone marrow examination and immunophenotyping. This case highlights the potential complementary role of the WPC parameter in the diagnostic evaluation of suspected acute leukemia, particularly in settings where access to advanced diagnostic techniques may be limited.

ETHICS STATEMENT

Ethical approval for this case report was obtained from the Ethics Committee of Wijaya Kusuma University (Approval No. 62/SLE/FK/UWKS/2026). Written informed consent was obtained from the patient for the publication of this case report and any accompanying images.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

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Not applicable

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