European Journal of Clinical and Experimental Medicine

ONLINE FIRST

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Article type: Original Article

Received: 3 June 2025

Accepted: 28 September 2025

Published online: 22 November 2025

eISSN: 2544-1361

Eur J Clin Exp Med

doi:10.15584/ejcem.2026.1.8

Molecular analysis of BCR-ABL1 fusion transcripts in acute leukemia patients in Southern Odisha, India

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ABSTRACT

Introduction and aim. The occurrence of BCR-ABL1 fusion transcript in acute-leukemia cases are limited and is characterized with various subtypes. This study was based on investigation of the BCR-ABL1 fusion transcript in acute-leukemia cases in a tertiary care hospital.

Material and methods. This cross-sectional study was carried out in suspected cases of acute leukemia. Cases were analyzed for complete blood counts, peripheral smear, bone marrow and flow-cytometry for the diagnosis of acute-leukemia. Additionally, diagnosed cases were investigated for BCR-ABL1 fusion transcript by PCR amplification using reverse transcriptase enzyme followed by documentation.

Results. Approximately 55 acute leukemia cases included, diagnosed by flow cytometric analysis. Analysis of the BCR-ABL1 fusion-transcript in 55 cases showed its occurrence in only 2 cases, where one case with the B-ALL was characterized with e1a2 subtype and a other case with AML was characterized with the e13a2 subtype.

Conclusion. The BCR-ABL1 fusion transcript in acute-leukemia cases was found in 3.64% (2/55) of cases. The diagnosis of BCR-ABL1 fusion transcript in acute-leukemia cases is important for its poor prognosis, as well as calls for the use of ABL tyrosine kinase inhibitors. The diagnosis of BCR-ABL1 fusion-transcript

in acute leukemia cases in a tertiary health care facility will be helpful for better treatment of patients in the study area.

Keywords. acute leukemia, acute lymphoblastic leukemia, acute myeloid leukemia, BCR-ABL1fusion transcript, polymerase chain reaction

Introduction

According to the Global Cancer Project (GLOBOCAN, 2022) report, leukemia ranks 13th as the most diagnosed cancer and 10th most common cause of cancer death globally, accounting for 487,294 new cases and 305,405 cancer deaths.1 According to the fourth edition of the WHO classification of tumors of hematopoietic and lymphoid tissues, leukemia can be broadly classified into myeloid or lymphoid lineages.[2] In India, the incidence of chronic myeloid leukemia (CML) was 0.8–2.2 among 100,000 males and 0.6-1.6 in 100,000 female subjects respectively; while acute leukemia (AL) including acute lymphoblastic leukemia (ALL) and acute myeloid leukemia (AML) are 35% and 15% respectively.[3,4]

There are several subtypes of leukemia based on its gene alteration and prognosis, including BCR-ABL1 fusion transcript. The BCR-ABL1 transcript is generated by balanced reciprocal translocation between chromosomes 9 and 22 to generate a short chromosome called the Philadelphia (Ph) chromosome {t(9;22)(q34.1;q11.2)}. The breakpoint cluster region (Bcr), composed of 23 exons and breakpoint 9 is present between 1a,1b and a2, whereas the breakpoint location on the BCR gene occurs between b1-b5 exons, e1-e2 and e19-e20, called major breakpoint cluster region (M-bcr) with molecular weight p210 (e13a2or e14a2), minor (m-bcr) with molecular weight p190 (e1a2) and micro (μ-bcr) p230 (e19a2) respectively. Furthermore, atypical BCR breakpoints outside the cluster regions generated by gene alteration to generate proteins such as e8a2, e15a2, e13a3, e14a3, and e6a2, where clinical significance was being investigated for the same.[5,6]

Although Ph chromosomes have been used as diagnostic markers in CML cases, there occurrence in AL cases is rare and limited to case series only. According to the WHO classification 2016, AML with BCR-ABL1 is rare and termed as subtype 2 contributing to approximately less than 1% of new cases of AML.[7-9] There are many reports on the presence of BCR-ABL1 fusion transcripts in ALL cases in India,10-14, while there is a paucity of report on AML, so this study was designed to determine the frequency of BCR-ABL1 fusion transcripts in cases diagnosed with AL in a tertiary health care setup in the state of Odisha, India.

Aim

This study was carried out to analyze the BCR-ABL1 fusion transcripts in cases of AL diagnosed in a tertiary health care setting in the state of Odisha, India.

Material and methods

This was a cross-sectional study carried out in cases suspected with AL, attending the outpatient Department of Medicine and the Department of Pediatrics in KCG. Medical College and Hospital, Berhampur, Odisha, from April 2022 to March 2024. After obtaining the proper consent of the patients (in the case of minors, consent has been obtained from either parents or guardians), 3 ml of venous blood or bone marrow samples were collected from the subjects in EDTA vials for various investigations including examination of peripheral smear (PS)/bone marrow (BM), complete blood counts (CBC), flow cytometer analysis and BCR-ABL1 fusion transcript. The complete blood count was performed using a 5-part differential analyzer (XN-550, Sysmex Europe SE, Hamburg, Germany). The flow cytometer analysis for the diagnosis of AL was performed using 6 colors BD FACS Canto^{TM}-II flow cytometer (BD Biosciences, 2350 Qume Drive, San Jose, CA, USA) using BD FACSDiva^{TM} Software v8.0.2 with a predefined panel of antibodies (BD Lifesciences) within 24 hours after sample collection (supplementary Table1).

AL cases of all age groups and both genders were recruited. Taking into account the occurrence of the BCR-ABL1 fusion transcript in AL cases of around 7%,[15] population size of 100 (50 AL cases per year in the study center) with 95% confidence level and absolute error of 5%, the minimum number of samples required for the study (sample size) was 51 (sample size was calculated using OpenEpi, open source calculator, SSPropor, Version 3). Along with the AL cases, some known CML cases attending this hospital during the study period were included for standardization of the molecular investigation of BCR-ABL1 fusion transcript. This study was approved by the Institutional Ethics Committee (IEC) of M.K.C.G Medical College, Berhampur, Odisha, India. (No. 1381/Chairman-IEC, MKCG Medical College, Brahmapur-4).

All confirmed cases of AL were subjected for analysis of BCR-ABL1 fusion transcript. Briefly, RNA was isolated from fresh blood samples using an RNA isolation kit (HiPureA Blood RNA Purification Kit, HiMedia Laboratories Pvt. Ltd.) according to the manufacturer's instructions and cDNA was prepared using the SuperScript TM VILOTM cDNA synthesizer kit (Invitrogen by Thermo Fisher Scientific Inc.) by taking 4 µL of 5X VILO^{TM} Reaction Mix, 2µL 10X Superscript TM enzyme mix, 2.5 µg of isolated RNA and DEPC treated water to make up the volume to20 µL. The incubation period for the preparation of the cDNA was at 25°C for 10 minutes, 42°C for 60 minutes, 85°C for 5 minutes followed by 4°C.

The BCR-ABL1 fusion transcript was analyzed by PCR using the primers described by Aazad et al. for the regions e1a2, e13a2, and e14a2.[16] In detail, a single PCR reaction included 20ng of the cDNA, 25 mM $MgCl_2$, 10 mM of dNTP mix, primer of 10 picomoles each and 5 units of Taq DNA polymerase with a final volume of 25µL. The reaction condition for the amplification was carried out with denaturation at 94°C for 35 seconds, annealing at 61°C for 30 seconds, and extension at 72°C for 30 seconds for 30 cycles followed by electrophoresis and documentation. The primers and amplicon size of various BCR-ABL1 fusion transcripts are depicted in Table 1. Amplified products were resolved in 2% agarose gel with a 100-bp DNA ladder as a marker (Figure 1).

Data collection and statistical analysis

All patient information, including socio-demographic and laboratory data, was collected in a case format. The generated data were entered in an excel sheet for further analysis. Descriptive data was presented in numbers and percentages. The recruited cases were categorized into different groups according to the types of leukemia (CML, B-ALL, T-ALL and AML) and types of BCR-ABL1 fusion transcripts (normal, e1a2, e13a2 (b2a2) and e14a2 (b3a2).

Results

The study was carried out in 69 cases of leukemia, including 55 suspected cases of AL and 14 cases of CML. The 55 suspected cases were confirmed to be AL by flow cytometry analysis, including 30 (54.5%) males and 25 (45.5%) female cases. The mean age of the patients was 26.82±18.94 years (age range of 1 year to 65 years) and 27 (49.1%) being in pediatric population. The age and gender distribution of patients is depicted in Figure 2.

The flow cytometer analysis of 55 AL cases resulted in 25 ALL cases (14 cases with B-ALL and 11 cases with T-ALL) and the remaining 30 cases were diagnosed with AML. The molecular analysis of the the subtypes of BCR-ABL1 fusion transcript in both CML and AL patients showed the presence of the BCR-ABL1 fusion transcript in all CML cases and only 2 (3.64%) cases of AL (1 case each with AML and B-ALL). The AML case had a BCR-ABL1 fusion transcript with the e13a2 subtype while the B-ALL case had a BCR-ABL1 fusion transcript with the e1a2 subtype. The detailed analysis of the BCR-ABL1 fusion transcript subtypes in the study cases is depicted in Table 2.

The hematological presentation of both cases, AML with BCR-ABL1 fusion transcript with the e13a2 subtype (case 1) and B-ALL with BCR-ABL1 fusion transcript with e1a2 subtype (case 2), are described in details.

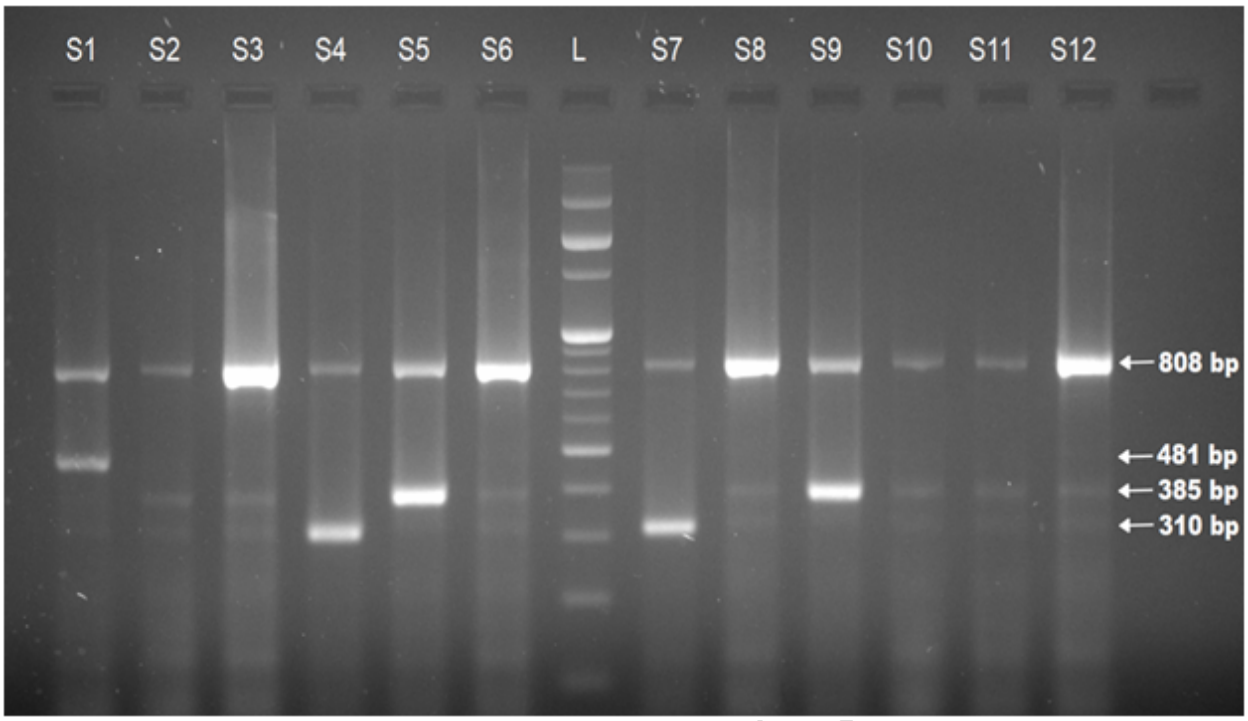


Fig. 1. Agarose gel electrophoresis (2%) for the BCR-ABL1 fusion transcript (Lane S2, S3, S6, S8, S10, S11 and S12 with 808 bp: Normal; Lane S1 with 481 bp: e1a2; Lane S4 and S7 with 310 bp: e13a2; Lane S5 and S9 with 385 bp: e14a2; L: 100bp DNA ladder)

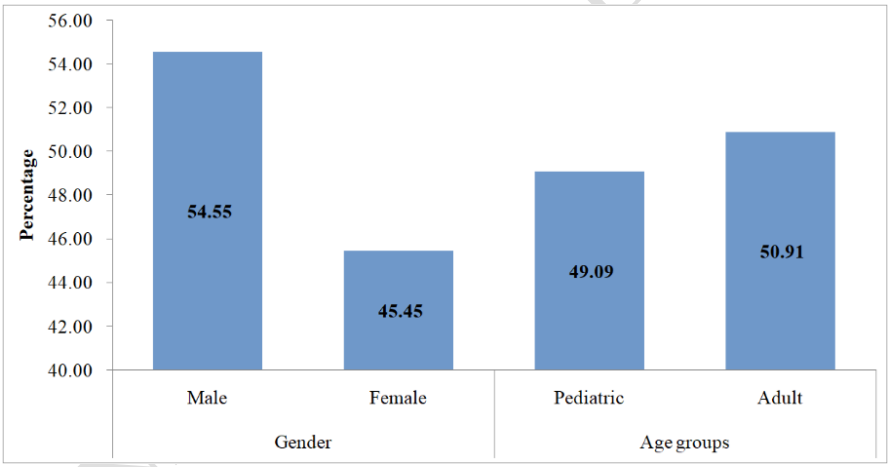
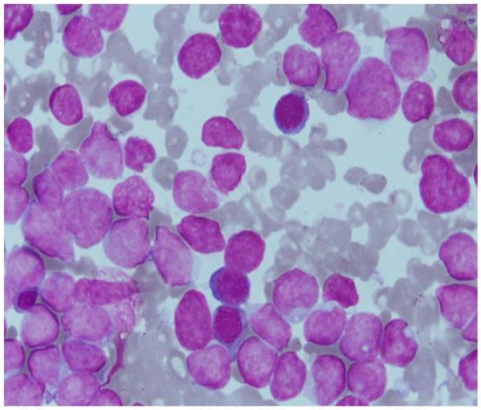
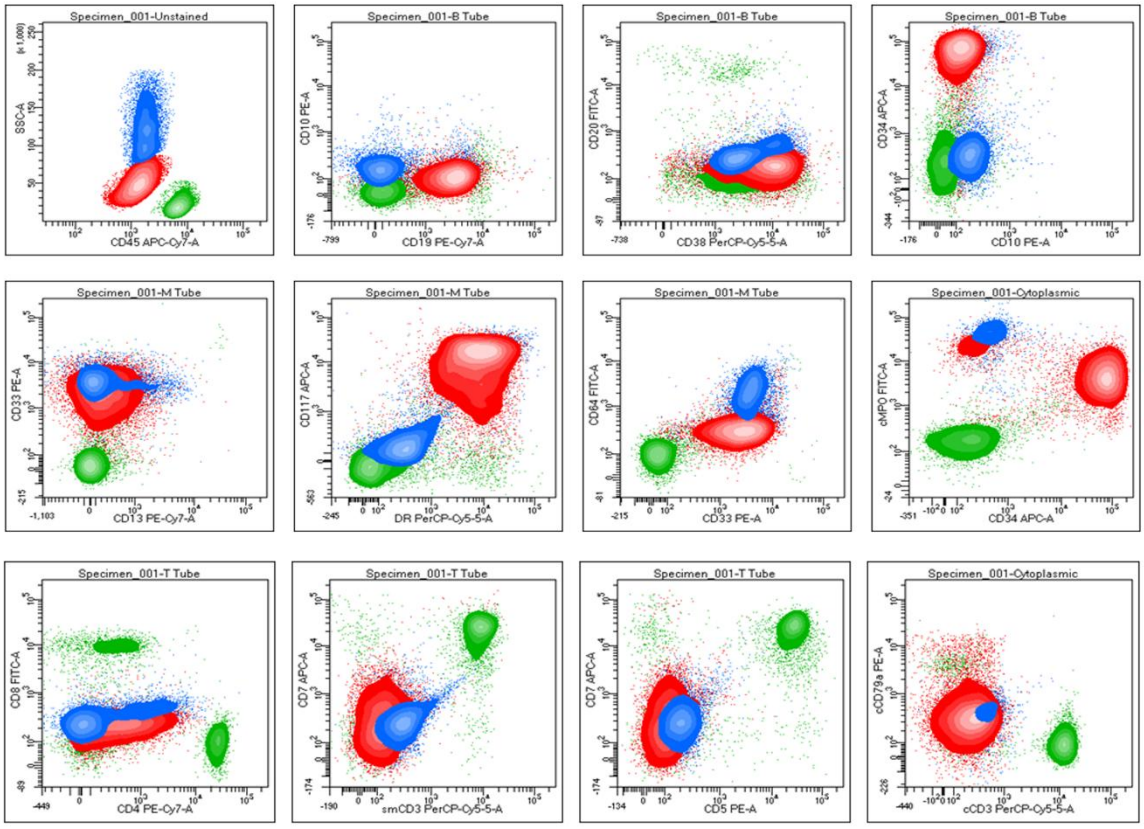


Fig. 2. Age and sex distribution of patients with AL (n=55)

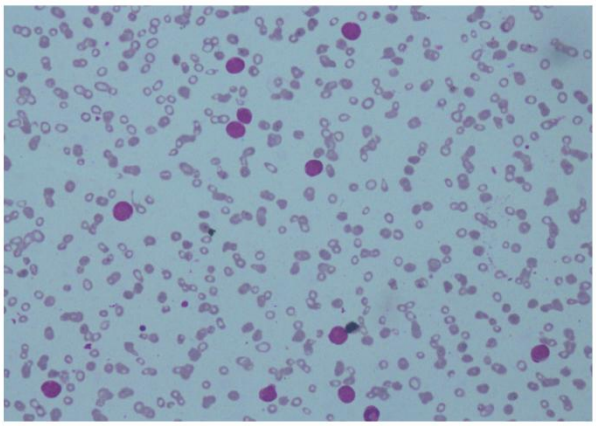


Leishman stain (100x10) of Bone Marrow

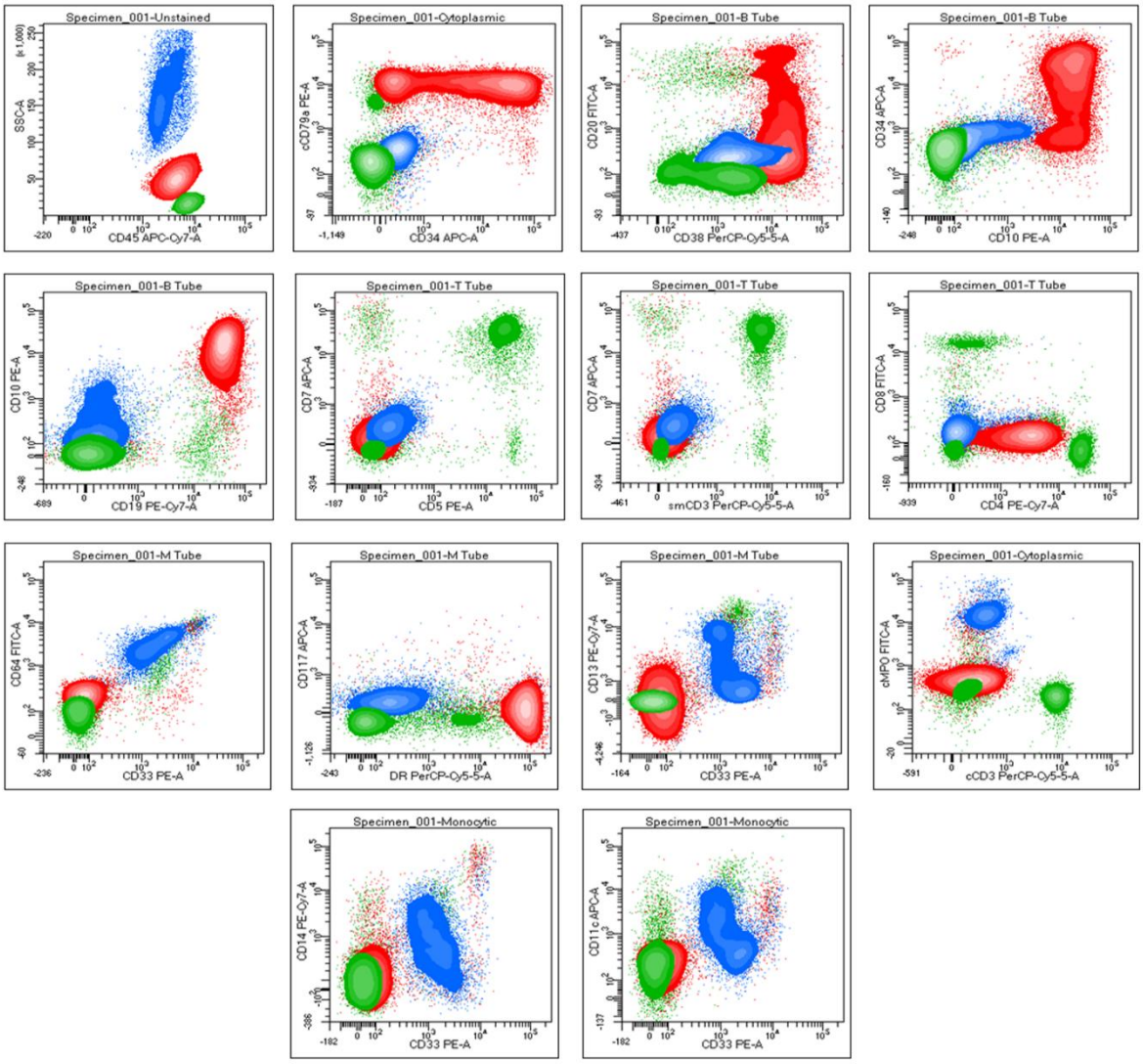


Flow cytometry analysis

Fig. 3. Flow cytometric analysis of AML case with BCR-ABL1 fusion transcript



Leishman stain (40x10) of Perpheral Blood



Flow cytometry analysis

Fig. 4. Flow cytometric analysis of ALL case with BCR-ABL1 fusion transcript

Case 1. AML case with BCR-ABL1 fusion transcript (e13a2)

An 11-year-old boy was admitted with fever, weakness, and a history of weight loss for 15 days without a relevant medical history. Physical examination revealed pallor and the absence of hepatomegaly as well as splenomegaly. Furthermore, biochemical examination it was observed, ESR of 170 mm in 1st hour and CBC bicytopenia (hemoglobin level of 4.7 g/dL, white blood cell count of 10.43×10^9/L and platelet count of 27×10^9/L). The peripheral smear showed blast cells and bone marrow aspiration revealed hypercellular marrow with 40% blast. The blast cells were medium to large in size having round to oval nuclei and some had irregular nuclear borders with fine chromatin, some blast cells showed 1-2 inconspicuous nucleoli and scanty granular basophilic cytoplasm. The cytochemistry of the blast cells showed positivity for the MPO stain and negative for the PAS stain. Thus, PS, bone-marrow study and cytochemistry, the case was diagnosed as AML. Therefore, the sub-classification of leukemia subclassification was performed by flowcytometry and the blast cells were positive for CD19, CD38, CD34, CD33, CD117, HLA-DR, CD4, CD7 and MPO; and negative for CD10, CD20, CD79a, smCD3, cyCD3, CD5, CD8, CD13 and CD64. Therefore, the final diagnosis based on flow cytometry was compatible with AML with differentiation and aberrant expression of CD4, CD7 and CD19 (Figure 3). The bone marrow was not done and subsequently the molecular analysis of BCR-ABL1 fusion transcript revealed the presence of Major subtype (M-bcr) BCR-ABL1 fusion transcript.

Case 2. B-ALL case with BCR-ABL1 fusion transcript (e1a2)

A 19-year-old male patient complained of fever and weakness without any relevant history since 45 days. On physical examination, it was revealed pallor and absence of hepatomegaly and splenomegaly were revealed. Additionally, CBC showed bicytopenia (hemoglobin level of 5.5g/dL, white blood cell count of $8.97\text{x }10^9$/L, and platelet count of $14\text{x}10^9$/L) and bone marrow aspiration revealed hypercellular marrow with variable-size leukemic blast of 45%. The blasts were small to medium in size, had round to oval nuclei with coarse chromatin, and had little granular basophilic cytoplasm. The cytochemistry of the blasts is blocked positive (course positive) with PAS stain and negative for MPO stain. Therefore, the diagnosis based on morphology was a case of ALL. The flow-cytometric analysis showed, that the blasts population was positive for CD79a, CD34, CD38, CD10, CD19, CD4, CD13, and HLA-DR, and negative for CD20, CD5, CD7, CD8, smCD3, cyCD3, CD33, CD64, CD117, MPO, CD14, and CD11c. Therefore, the diagnosis based on flow cytometry was B-ALL with aberrant expression of CD13 and CD4 (Figure 4). The bone marrow cytogenetic study was not analyzed. Analysis of the BCR-ABL1 fusion transcript revealed the presence of a minor subtype (m-bcr) e1a2 (p190) BCR- ABL1 fusion transcript.

Table 1. List of primers used for BCR-ABL1 fusion transcript analysis

Transcript	Primers	Primer sequences (5'-------3')	Amplicon size (base pairs)
Normal BCR	B2B+C5e	ACAGAATTCCGCTGACCATCAATAAG ATAGGATCCTTTGCAACCGGGTCTGAA	808
e1a2	BCR-C+CA3	ACCGCATGTTCCGGGACAAAAG TGTTGACTGGCGTGATGTAGTTGCTTGG	481
e13a2 (b2a2)	B2B+CA3	ACAGAATTCCGCTGACCATCAATAAG TGTTGACTGGCGTGATGTAGTTGCTTGG	310
e14a2 (b3a2)	B2B+CA3	ACAGAATTCCGCTGACCATCAATAAG TGTTGACTGGCGTGATGTAGTTGCTTGG	385

Table 2. BCR-ABL1 fusion transcript in different cases of leukemia (n=69) *

Type of leukemia	Subtypes of the BCR-ABL1 fusion transcript subtypes		
	e13a2	e14a2	e1a2
CML (n=14)	5	8	1
AML (n=30)	1	0	0
B-ALL (n=14)	0	0	1
T-ALL (n=11)	0	0	0

* chronic myeloid leukemia – CML, acute myeloid leukemia – AML, B-ALL – B-acute lymphoblastic leukemia, T-ALL – T-acute lymphoblastic leukemia

Discussion

During the study period, a total of 55 patients diagnosed with AL by flow cytometry were included along with 14 patients with CML for the molecular characterization of the BCR-ABL1 fusion transcript. Although the main objective of this study was to observe the presence of the BCR-ABL1 fusion transcript only in cases only, the CML cases were included for standardization of molecular analysis of BCR-ABL1 fusion transcript in the study center. Of 55 cases with AL, 30 (54.5%), 14 (25.5%) and 11 (20.0%) cases were diagnosed with AML, B-ALL and T-ALL respectively.

Among the 14 cases with CML, the BCR-ABL1 fusion transcript has been characterized in all cases. Most (8 cases) of cases were diagnosed as e14a2, followed by 5 cases with e13a2 type and one single case with e1a2 type. All CML cases analyzed in the present study were characterized by a typical type of BCR-ABL1 fusion transcript, which is consistence with many previous reports. Along with the typical types, many atypical types like e13a3 and e14a3 have also been reported from many previous studies.[17-22]

The BCR-ABL1 fusion transcript was found to be positive in only 2 cases of 55 cases of AL. One case with AML (out of 30 cases) was of type e13a2 type and another case with B-ALL (out of 14 cases) presented with e1a2 type. Although the BCR-ABL1 fusion transcript is the hallmark for CML, cases with ALL or AML have also been reported with the BCR-ABL1 fusion transcript. It has also been reported that around 2-3% of children and around 20-30% of adults diagnosed with ALL may present with this translocation.[23] The presence of AML de novo with BCR-ABL1 fusion transcript is rare with an incidence rate of 0.5-3%.[8,24,25] Both cases of AML and B-ALL cases with BCR-ABL1 fusion transcript in this present study presented with a typical type of translocation; while the occurrence of many atypical types has also been reported in many studies.[15]

Both the cases with the AML and B-ALL with BCR-ABL1 fusion transcript in this study showed aberrant expression of various CD markers. The AML case with the BCR-ABL1 fusion transcript had aberrant expression of CD4, CD7, and CD19 whereas the B-ALL case with translocation had aberrant expression of CD4 and CD13. There is no consistency in the expression of various CD markers in both AML and ALL with respect to the BCR-ABL1 fusion transcript. However, most studies have reported with aberrant expression of CD7 in cases of AML cases with BCR-ABL1 fusion transcript,[7,26,27] and CD13 in ALL cases with the BCR-ABL1 fusion transcript.[28-30] Aberrant expression of CD markers in AL cases with BCR-ABL1 fusion transcript does not have any clinically significant influence on treatment outcome; but there is a recommendation for the analysis of other genetic associations for better clinical outcomes.[31]

The diagnosis of the BCR-ABL1 fusion transcript in AL is important due to its poor prognosis under conventional therapy in patients with AL, which in turn identified them as high-risk groups. Furthermore, these patients may have a better prognosis in treatment regimens with ABL tyrosine kinase inhibitors such as imatinib or dasatinib.[32-35]Acute leukemia with BCR-ABL1 fusion transcript has been included in the updated WHO (2016) as a provisional entity.[36] AML with the BCR-ABL1 fusion transcript is stratified and allocated as an intermediate risk group according to the European LeukemiaNet risk stratification and a poor risk group according to the National Comprehensive Cancer Network.[37,38] Thus, it is a need for molecular characterization of the BCR-ABL1 fusion transcript in all newly diagnosed cases with AL for better treatment and survival of the patients.

There are few limitations in this study. First, in this study only 3 typical types of BCR-ABL1 fusion transcript have been considered for the analysis by using conventional PCR methods; the occurrence of other rare or atypical types of BCR-ABL1 fusion transcript in our study population cannot be ignored as we have not analyzed in this study. Second, as a diagnostic center, there is a paucity of information on clinical outcomes as well as treatment regimens, or the follow-up data of the patients.

In conclusion, this is a pilot study carried out to initiate the BCR-ABL1 fusion transcript in flow cytometric confirmed cases with AL in a tertiary health care center in southern Odisha, India. With a low prevalence (2 out of 55 cases) of BCR-ABL1 fusion transcript in overall AL cases in the study population, warn against

the analysis of other rare translocation types for better treatment, risk stratification and clinical outcomes of patients.

Acknowledgements

The authors acknowledge laboratory technicians, Mrs. M. Radha Rani Dora and Mr. Himanshu Sekhar Sahu, for processing blood samples during the investigation. The authors also acknowledge Mr. Satyanarayana Mohanty, Data Entry Operator, MRU, for data processing.

Declarations

Funding

This study (Research proposal ID: MKCGMC/MRU/034/2021) was funded by Department of Health Research, Ministry of Health and Family Welfare, Government of India for the establishment of Multi-Disciplinary Research Unit (MRU) at Maharaja Krishna Chandra Gajapati (M.K.C.G.) Medical College, Berhampur, Odisha, India

Author contributions

Conceptualization, M.K.P. and S.K.B.; Methodology, S.S., S.B., C.P. and P.P; Validation, P.P. and S.K.B.; Formal Analysis, S.S. and P.P.; Investigation, S.S., S.B., C.P. and P.P; Resources, S.S. and P.P.; Data Curation, P.P.; Writing – Original Draft Preparation, S.S., A.N., and P.P.; Writing – Review & Editing, A.N., P.P., M.K.P. and S.K.B.; Supervision, M.K.P. and S.K.B.; Project Administration, S.K.B.; Funding Acquisition, S.K.B.

Conflicts of interest

The authors declare no conflicts of interest.

Data availability

The data will be available on request to corresponding author.

Ethics approval

This study was approved by the Institutional Ethical Committee (IEC) of M.K.C.G Medical College, Berhampur, Odisha, India. (No. 1381/Chairman-IEC, M.K.C.G. Medical College, Brahmapur-4).

Reference

1. GLOBOCAN, 2022 https://gco.iarc.who.int/media/globocan/factsheets/populations/900-world-fact-sheet.pdf. Accessed September 3, 2024.

2. Du M, Chen W, Liu K, et al. The Global Burden of Leukemia and Its Attributable Factors in 204 Countries and Territories: Findings from the Global Burden of Disease 2019 Study and Projections to 2030. *J Oncol*. 2022;2022:1612702. doi:10.1155/2022/1612702
3. Kumar L. Chronic myelogenous leukaemia (CML): an update. *Natl Med J India*. 2006;19(5):255-263.
4. Ahirwar R, Nigam RK, Parmar D. A study of leukemias Profile in central India. *Tropical Journal of Pathology and Microbiology*. 2018;4(2):181-187. doi:10.17511/jopm.2018.i02.12
5. Carter BZ, Mak DH, Cortes J, Andreeff M. The elusive chronic myeloid leukemia stem cell: does it matter and how do we eliminate it? *Semin Hematol*. 2010;47(4):362-370. doi:10.1053/j.seminhematol.2010.06.006
6. Yao J, Douer D, Wang L, Arcila ME, Nafa K, Chiu A. A case of acute myeloid leukemia with e6a2 BCR-ABL fusion transcript acquired after progressing from chronic myelomonocytic leukemia. *Leuk Res Rep*. 2017;7:17-19. doi:10.1016/j.lrr.2017.01.003
7. Sheets JW, Eulitt P, He R, et al. Philadelphia Chromosome-positive Acute Myeloid Leukemia With e1a3 BCR-ABL1 Fusion Transcript. *Hemasphere.* 2020;4(6):e484. doi:10.1097/HS9.0000000000000484
8. Soupir CP, Vergilio JA, Dal Cin P, et al. Philadelphia chromosome-positive acute myeloid leukemia: a rare aggressive leukemia with clinicopathologic features distinct from chronic myeloid leukemia in myeloid blast crisis. *Am J Clin Pathol*. 2007;127(4):642-650. doi:10.1309/B4NVER1AJJ84CTUU
9. Loknatha D, Rao SA, Lokesh KN, Babu KG, Suresh Babu MC, Lakshmaiah KC. A case series of patients with de novo Philadelphia-positive acute myeloid leukemia at a tertiary care center in South India - clinical profile, outcomes, and review of literature. *Journal of Applied Hematology* 2016;7(2):66-69. doi:10.4103/1658-5127.186326
10. Mondal BC, Bandyopadhyaya A, Majumdar S, et al. Molecular profiling of chronic myeloid leukemia in eastern India. *Am J Hematol* 2006;81:845-849. doi:10.1002/ajh.20682
11. Hasan SK, Sazawal S, Kumar B, et al. Childhood CML in India: b2a2 transcript is more common than b3a2. *Cancer Genet Cytogenet*. 2006;169(1):76-77. doi:10.1016/j.cancergencyto.2006.01.015
12. Polampalli S, Choughule A, Negi N, et al. Analysis and comparison of clinicohematological parameters and molecular and cytogenetic response of two Bcr/Abl fusion transcripts. *Genet Mol R*es. 2008;7(4):1138-1149. doi:10.4238/vol7-4gmr485.
13. Sharma P, Kumar L, Mohanty S, Kochupillai V. Response to Imatinib mesylate in chronic myeloid leukemia patients with variant BCR-ABL fusion transcripts. *Ann Hematol*. 2010;89(3):241-247. doi:10.1007/s00277-009-0822-7

14. Anand MS, Varma N, Varma S, Rana KS, Malhotra P. Cytogenetic & molecular analyses in adult chronic myelogenous leukaemia patients in north India. *Indian J Med Res*. 2012;135(1):42-48. doi:10.4103/0971-5916.93423
15. Gupta DG, Varma N, Naseem S, et al. Characterization of immunophenotypic aberrancies with respect to common fusion transcripts in B-Cell precursor acute lymphoblastic leukemia: A report of 986 Indian patients. *Turk J Haematol*. 2022;39:1-12 doi:10.4274/tjh.galenos.2021.2021.0326
16. Azad NA, Shah ZA, Pandith AA, et al. Prognostic Implication of BCR-ABL Fusion Transcript Variants in Chronic Myeloid Leukemia (CML) Treated with Imatinib. A First of Its Kind Study on CML Patients of Kashmir. *Asian Pac J Cancer Prev*. 2018;19(6):1479-1485. doi:10.22034/APJCP.2018.19.6.1479
17. Li Y, Zhang Y, Meng X, et al. Chronic myeloid leukemia with two rare fusion gene transcripts of atypical BCR::ABL: A case report and literature review. *Medicine (Baltimore).* 2024;103(3):e36728. doi:10.1097/MD.0000000000036728.
18. Massimino M, Stella S, Tirrò E, et al. Rapid decline of Philadelphia-positive metaphases after nilotinib treatment in a CML patient expressing a rare e14a3 BCR-ABL1 fusion transcript: A case report. *Oncol Lett*. 2019;18(3):2648-2653. doi:10.3892/ol.2019.10558.
19. Sazawal S, Chhikara S, Singh K, et al. Distribution of common BCR-ABL fusion transcripts and their impact on treatment response in Imatinib treated CML patients: A study from India. *Indian J Pathol Microbiol*. 2019;62(2):256-260. doi:10.4103/IJPM.IJPM_726_17.
20. Chisti MM, Sanders DS. Chronic Myeloid Leukemia with b3a3 (e14a3) Fusion: A Rare BCR/ABL Rearrangement Presenting with Thrombocytosis - Does MTHFR Polymorphism Matter. *Case Rep Oncol*. 2018;11(2):485-492. doi:10.1159/000490697
21. Duan MH, Li H, Cai H. A rare e13a3 (b2a3) BCR-ABL1 fusion transcript with normal karyotype in chronic myeloid leukemia: The challenges in diagnosis and monitoring minimal residual disease (MRD). *Leuk Res.* 2017;59:8-11. doi:10.1016/j.leukres.2017.05.009
22. Hu LH, Pu LF, Yang DD, et al. How to detect the rare BCR-ABL (e14a3) transcript: A case report and literature review. *Oncol Lett.* 2017;14(5):5619-5623. doi:10.3892/ol.2017.6847
23. Chissoe SL, Bodenteich A, Wang YF, et al. Sequence and analysis of the human ABL gene, the BCR gene, and regions involved in the Philadelphia chromosomal translocation. *Genomics.* 1995;27(1):67-82. doi:10.1006/geno.1995.1008.
24. Takeuchi A, Kondo T, Tasaka T, et al. Successful treatment with ABL tyrosine kinase inhibitor for patients with acute myeloid leukemia with BCR-ABL1. *Leuk Res Rep*. 2022;17:100309. doi:10.1016/j.lrr.2020.100233

25. Low SK, Nanua S, Patel M, Renteria AS. AML with BCR-ABL1 Fusion treated with Imatinib, a Hypomethylating Agent and Venetoclax. *Leuk Res Rep*. 2022;17:100333. doi:10.1016/j.lrr.2022.100333
26. Piedimonte M, Ottone T, Alfonso V, et al. A rare BCR-ABL1 transcript in Philadelphia-positive acute myeloid leukemia: case report and literature review. *BMC Cancer*. 2019;19(1):50. doi:10.1186/s12885-019-5265-5
27. da Silva FB, Machado-Neto JA, Koury LCA, et al. Case Report Acute myeloid leukemia with e1a2 BCR-ABL1 fusion gene: two cases with peculiar molecular and clinical presentations. *Rev Bras Hematol Hemoter*. 2017;39(4):379-384. doi:10.1016/j.bjhh.2017.07.001
28. Shin SY, Cho JH, Kim HJ, Jang JH, Lee ST, Kim SH. Two cases of acute lymphoblastic leukemia with an e1a3 BCR-ABL1 fusion transcript. *Ann Lab Med*. 2015;35(1):159-161. Erratum in: *Ann Lab Med*. 2015;35(4):477. doi:10.3343/alm.2015.35.1.159
29. Deshpande PA, Srivastava VM, Mani S, et al. Atypical BCR-ABL1 fusion transcripts in adult B-acute lymphoblastic leukemia, including a novel fusion transcript-e8a1. *Leuk Lymphoma*. 2016;57(10):2481-2484. doi:10.3109/10428194.2016.1151512.
30. Chen Z. The e1a3 BCR-ABL1 fusion transcript in Philadelphia chromosome-positive acute lymphoblastic leukaemia: a case report. *Hematology.* 2023;28(1):2186040. doi:10.1080/16078454.2023.2186040
31. Jaso J, Thomas DA, Cunningham K, et al. Prognostic significance of immunophenotypic and karyotypic features of Philadelphia positive B-lymphoblastic leukemia in the era of tyrosine kinase inhibitors. *Cancer*. 2011;117(17):4009-4017. doi:10.1002/cncr.25978
32. Gleissner B, Gökbuget N, Bartram CR, et al. German Multicenter Trials of Adult Acute Lymphoblastic Leukemia Study Group. Leading prognostic relevance of the BCR-ABL translocation in adult acute B-lineage lymphoblastic leukemia: a prospective study of the German Multicenter Trial Group and confirmed polymerase chain reaction analysis. *Blood.* 2002;99(5):1536-1543. doi:10.1182/blood.v99.5.1536
33. Talpaz M, Shah NP, Kantarjian H, et al. Dasatinib in imatinib-resistant Philadelphia chromosome-positive leukemias. *N Engl J Med*. 2006;354(24):2531-2541. doi:10.1056/NEJMoa055229
34. Pfeifer H, Cazzaniga G, van der Velden VHJ, et al. Standardisation and consensus guidelines for minimal residual disease assessment in Philadelphia-positive acute lymphoblastic leukemia (Ph + ALL) by real-time quantitative reverse transcriptase PCR of e1a2 BCR-ABL1. *Leukemia.* 2019;33(8):1910-1922. doi:10.1038/s41375-019-0413-0. Erratum in: *Leukemia*. 2020;34(7):1970.
35. Leoni V, Biondi A. Tyrosine kinase inhibitors in BCR-ABL positive acute lymphoblastic leukemia. *Haematologica.* 2015;100(3):295-299. doi:10.3324/haematol.2015.124016

36. Arber DA, Orazi A, Hasserjian R, et al. The 2016 revision to the World Health Organization classification of myeloid neoplasms and acute leukemia. *Blood.* 2016;127(20):2391-2405. doi:10.1182/blood-2016-03-643544
37. Döhner H, Wei AH, Appelbaum FR, et al. Diagnosis and management of AML in adults: 2022 recommendations from an international expert panel on behalf of the ELN. *Blood*. 2022;140(12):1345-1377. doi:10.1182/blood.2022016867
38. Pollyea DA, Altman JK, Assi R, et al. Acute Myeloid Leukemia, Version 3.2023, NCCN Clinical Practice Guidelines in Oncology. *J Natl Compr Canc Netw*. 2023;21(5):503-513. doi:10.6004/jnccn.2023.0025