

as a potential target therapy. CD33^{low} blasts are associated to a more mature AML and its high expression is related to adverse landscapes, highlighting the importance of CD33 evaluation. Standard care for AML enrolls MRD monitoring and failure to achieve an MRD-negative, CR or detected MRD during or after therapy is associated with relapse or poor outcomes. Still, MRD analysis must embrace a set of standards in order to prevent post-analytic errors. Here we present a case report of AML with absent CD33 maintained from diagnosis to MRD follow-ups, mimicking MRD positivity. This data emphasizes the importance of caution in MFC analysis and correlation of diagnostic and follow-ups results interpretation, as well as constant training of the team.

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ACUTE UNDIFFERENTIATED LEUKEMIA WITH BCR-ABL TRANSLOCATION: CASE REPORT AND LITERATURE REVIEW

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The 2016 WHO classification defines acute undifferentiated leukemia (AUL) as a rare type of acute leukemia (AL) without defining markers for either lymphoid or myeloid lineage commitment. Literature suggests AUL does not harbour the genetic mutations commonly seen in lymphoblastic leukemia or acute myeloid leukemia (AML) including FLT3, WT1, rearrangements of KMT2A or BCR-ABL1 and it highly expresses genes associated with poor prognosis in AML (BAALC, ERG, and MN1); therefore, AUL is often associated with dismal outcome (Arber et al. 2016). This case report depicts an unusual genetic presentation of an AUL with BCR-ABL fusion gene. A 54-year-old female patient presented to our hospital in July 2021 with fatigue and adynamia one month before. She was also found with deep vein thrombosis in the left leg and segmental pulmonary thromboembolism. Laboratory exams showed Hb 9,7 g/dL, WBC 75.77×10⁹/L (77% blasts), platelets 175×10⁹/L. Coagulogram and fibrinogen were normal. Bone marrow (BM) aspiration was hypercellular, showing 86% of agranular blasts. Flow cytometry (FC) from BM blasts exhibited expression of CD34, CD45, HLA-DR, CD33, CD38, and partial expression of CD10 and CD123. The blasts were negative for MPO, CD79a, CD19, CD7, CD3, CD13, C117, CD16, CD11b, CD64, CD15, CD14, CD300e, CD61, NG2, CD71, CD4, CD36, CD22, CD42b. Conventional cytogenetics revealed a Ph+ disease (46,XX,t(9;22)(q34;q11.2)[7]/46, idem, add(4)(q35)[7]/46,XX [6]). Real-time quantitative PCR showed positive BCR-ABL with p190 transcripts. FLT3 and NPM1 were found negative. Diagnosis of AUL Ph+ was made. AML chemotherapy was started with 3+7 standard doses regimen plus imatinib 600 mg/day. On day 14th BM evaluation found 89% of blasts with the same immunophenotype as in the diagnosis. The patient presented neutropenic colitis with the need to delay

rescue therapy. We searched articles in the Pubmed database using the following strategy: “Bcr-Abl Tyrosine Kinase”AND “undifferentiated leukemia”, “Philadelphia chromosome”AND “undifferentiated leukemia”between 1976 and 2021. We found two case reports that described patients with AUL and positive BCR-ABL gene. In 1983, Smadja, N et al. reported a patient with AL considered at diagnosis to be undifferentiated. Neither the diagnosis cytogenetic nor FC data was provided by the authors, and the AL classification was based only on morphological characteristics. After 17 months, the patient developed Ph+ chronic myelogenous leukemia with a complex translocation (2; 9; 22). Hattori, M et al., in 1995, described a patient that following treatment to non-Hodgkin lymphoma developed myelodysplastic syndrome (MDS) which progressed to AUL, both found with the presence of chimeric BCR-ABL transcripts. The FC showed blasts that expressed CD7 and HLA-DR, and were negative for lineage markers: CD14,CD33,CD2,CD3,CD4,CD5,CD8,CD10,CD19,CD20 and CD41. Qasrawi, A. et al., in 2019 and 2020 performed two studies analyzing data from 24 and 1444 cases of AUL, respectively. The most common pathogenic mutations were PHF6, SRSF2, RUNX1, ASXL1, and BCOR. No BCR-ABL mutation was described. We believe that with the advent of target therapies, the prognosis of AUL can improve. The patient described has the BCR-ABL gene as a potential target to a tyrosine kinase inhibitor. Considering this is not a usual finding, we hope this report can further help treatment decisions.

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ARHGAP6 EXPRESSION IS ASSOCIATED WITH MOLECULAR RISK AND IMPACTS CLINICAL OUTCOMES IN ACUTE MYELOID LEUKEMIA

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Objectives: Despite the advances in the large-scale generation of genomic data from acute myeloid leukemia (AML) patients, much effort is still needed to convert these data into biological functions, which may pave avenues for the discovery of new prognostic markers and therapeutic targets. In a previous study, four cytoskeleton-related genes (ARHGAP6, EZR, MAP3K11 and PAK1) were identified as a prognostic factor in AML. In the present study, we detail the prognostic impact, as well as its functional genomic associations, of the ARHGAP6 gene in AML patients. **Material and methods:** Clinical and genomic data were obtained from the TCGA AML cohort (n=173). The clinical impact (overall survival [OS] and disease-free survival [DFS]) for ARHGAP6 expression was investigated by the proportional hazards Cox regression model. Functional genomic analyzes were performed by gene set