Medicina Universitaria. 2017;xxx(xx):xxx–xxx

MEDICINA UNIVERSITARIA

www.elsevier.es/rmuanl

REVIEW ARTICLE

Lineage switch from acute lymphoblastic leukemia to myeloid leukemia

G.J. Ruiz-Delgado[a,b,c,*], **A.K. Nuñez-Cortez**[a,b,c,d], **J.C. Olivares-Gazca**[a,b,c], **Y.C. Fortiz**[a,b,c,d,e], **A. Ruiz-Argüelles**[b,c,d,e], **G.J. Ruiz-Argüelles**[a,b,c]

[a] *Centro de Hematología y Medicina Interna de Puebla, Clínica Ruiz, Puebla, Mexico*
[b] *Laboratorios Clínicos de Puebla, Clínica Ruiz, Puebla, Mexico*
[c] *Popular Autonomous University of the State of Puebla, Puebla, Mexico*
[d] *Praiseworthy Autonomous University of Puebla, Puebla, Mexico*
[e] *University of the Americas of Puebla, San Andrés Cholula, Mexico*

Received 9 February 2017; accepted 23 February 2017

KEYWORDS
Acute leukemia;
Lymphoblastic leukemia;
Myeloid leukemia;
Lineage switch;
B-cell leukemia

Abstract
Objective and background: Acute lymphoblastic leukemia (ALL) is the most common pediatric malignancy, representing 25% of childhood cancers, with a peak prevalence between 2 and 9 years. Conversions of the leukemic cell lineage throughout the duration of the disease is a rare manifestation, accounting for 6–9% of relapsed cases and being more frequently observed in pediatric patients. We present a case of a patient with a lineage switch from lymphoblastic leukemia to myeloid leukemia.
Case presentation: A 60-year-old male was seen due to pancytopenia, weight loss and weakness. Initial laboratory work-up was performed. Bone and marrow aspirate flow cytometric analysis disclosed pre-B lymphoblastic acute leukemia BCR ABL (−), 46 XY, hyperdiploid, CD20(−), CD 10 (−), CD19 (+), CD33 (−), CD34 (+), CD38 (+), CD79a (+), TdT (+), IgS(−), CD45 (+/−), HLA-DR (+), MLL (−), FLT3 (−), TEL AML (−). He was treated with a pediatric-inspired TOTAL XI schedule. Sixty days afterward, induction blasts appeared in the peripheral blood, but immunophenotyping was not conclusive for MRD+ status. One week later, he presented blasts in the peripheral blood compatible with acute myeloid leukemia. CD7 (+−), CD13 (+), CD14 (−), CD15(−), CD33(+), CD34(+), CD38(+), CD45(+−), CD64(−), CD117(+), HLA-DR heterogenous. BCR-ABL, PML-RAR alfa, and FLT-3 were repeated in peripheral blood when AML developed and was negative. The patient started subcutaneous cytarabine and was alive 90 days after initial diagnosis with active AML leukemia.

* Corresponding author at: Clínica Ruiz, Mártires del 2 de Octubre 3710, Anzures, 72530 Puebla, Pue., Mexico. Tel.: +52 222 243 8100; fax: +52 222 243 8428.
E-mail address: gruiz2@clinicaruiz.com (G.J. Ruiz-Delgado).

http://dx.doi.org/10.1016/j.rmu.2017.02.001

Please cite this article in press as: Ruiz-Delgado GJ, et al. Lineage switch from acute lymphoblastic leukemia to myeloid leukemia. Medicina Universitaria. 2017. http://dx.doi.org/10.1016/j.rmu.2017.02.001

Conclusion There is a small number of reports of lineage conversion in the literature, probably because immunophenotyping is performed at diagnosis without a follow up.

Introduction

Approximately 5000 new cases of acute lymphoblastic leukemia (ALL) are diagnosed each year in the United States, more than half of these in children. ALL represents the most common pediatric malignancy, accounting for at least 25% of childhood cancer. The peak prevalence of ALL is between the ages of 2 and 9 years. There is a slight male predominance, and Caucasians have a twofold increased risk compared to African Americans. Lineage switch accounts for approximately 6–9% of relapsed cases, and is more often observed in childhood patients, for whom an appropriate standard treatment is not available.[1]

We report a case of an adult with lineage switch from acute lymphoblastic leukemia to acute myeloid leukemia (AML).

Case report

A 60-year-old male was seen at our Hematology Center due to pancytopenia, weight loss and weakness. Upon physical examination, he was pale, referred asthenia, bone pain, and hepatosplenomegaly. The rest of the examination showed no other abnormalities. Initial laboratory work was performed. Bone and marrow aspirate flow cytometric analysis disclosed pre-B lymphoblastic acute leukemia BCR ABL (−), 46 XY, hyperdiploid, CD20(−), CD 10 (−), CD19 (+), CD33 (−), CD34 (+), CD38 (+), CD79a (+), TdT (+), IgS(−), CD45 (+/−), HLA−DR (+), MLL (−), FLT3 (−) and TEL AML (−) (Fig. 1). Cerebrospinal fluid cytology showed no blasts. He was treated with a modification of the pediatric-inspired TOTAL XI schedule. During the induction phase, he presented significant myelotoxicity and significant adverse reactions

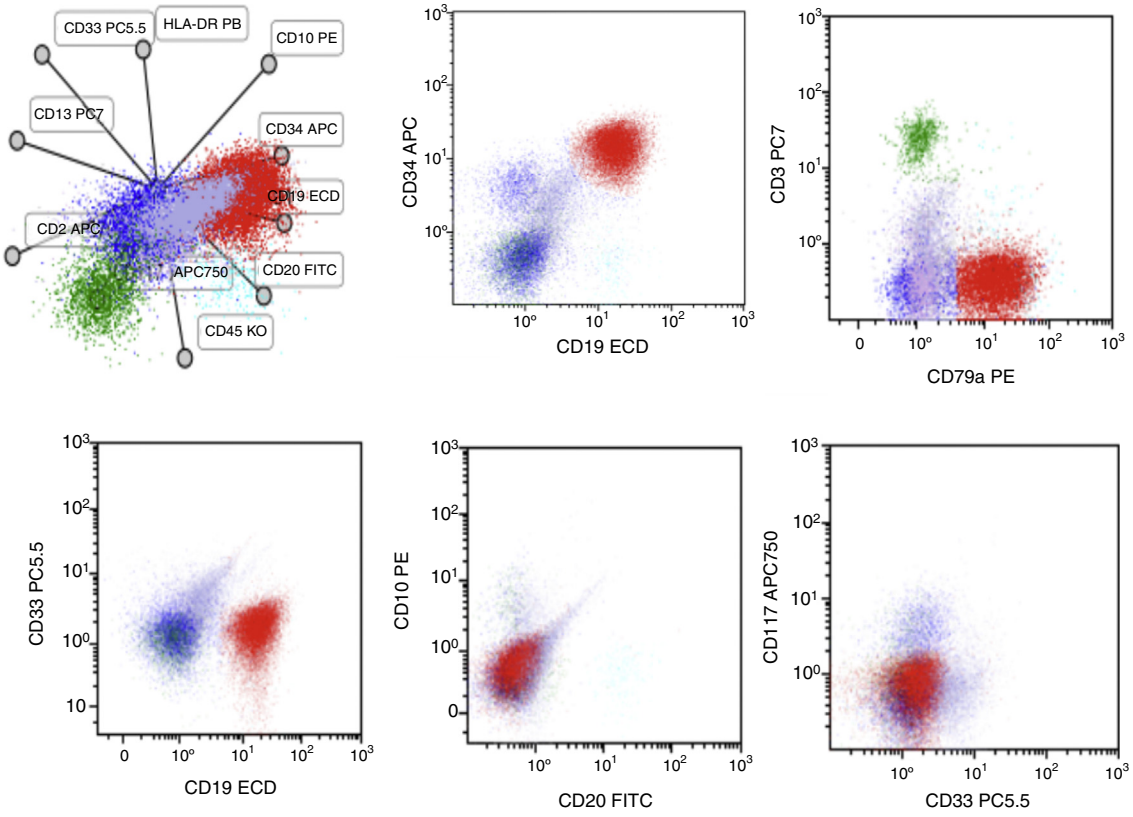

Figure 1 Salient features of the immunophenotype at diagnosis (2016-August-16). Blasts (red) co-express CD19/CD34 and CD79a antigens, whereas they do not express CD10, CD20, CD33 or CD117 antigens. The expressions of other myeolid and lymphoid antigens of the T lineage was not detected.

Please cite this article in press as: Ruiz-Delgado GJ, et al. Lineage switch from acute lymphoblastic leukemia to myeloid leukemia. Medicina Universitaria. 2017. http://dx.doi.org/10.1016/j.rmu.2017.02.001