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BLASTIC TRANSFORMATION IN MEXICAN POPULATION WITH CHRONIC MYELOMONOCYTIC LEUKEMIA

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Background: Chronic myelomonocytic leukemia (CMML) is the most aggressive of chronic leukemias, with a short overall survival and a high transformation rate to acute leukemia. We investigated factors related to blastic transformation in Mexican population treated in a tertiary referral center.
Methods: Records of patients diagnosed with CMML between 2000-2015 were reviewed; patients with incomplete data were excluded. IBM SPSS Statics 21.0 software was used to perform statistical analysis.
Findings: 54 patients were included, with a median age of 71 years and an overall survival of 16 months. The rate of blastic transformation found was 33% (18 patients), with a time to progression of 9 (0-87) months. Comparing patients who didn't underwent blastic transformation to those who did, those who progressed to acute leukemia tend to be younger (58 vs. 71 years, p=0.001), had a higher peripheral blood blast count (≥2% vs. 0%, p= 0·003), and where more likely to have immature myeloid precursors circulating in peripheral blood (94% vs. 64%, p=0.02). In multivariate analysis, age continued to be a statistically significant factor to progression (HR 0.97, 95% IC = 0·929-0·987).
Interpretation: Mexican patients with CMML that progressed to overt acute leukemia were considerably younger, with a higher tumor burden and short overall survival. In this population, it is important to consider a more aggressive treatment at diagnosis, focusing in high dose chemotherapy and hematopoietic stem cell transplantation in a short term.
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INTRODUCTION

Chronic myelomonocytic leukemia (CMML) is a clonal, hematopoietic stem cell malignant disorder, characterized by overlapping features of both myeloprolifetative neoplasm and myelodisplasic syndrome (MDS). According to the 2016 WHO Classification the main characteristics are (1) persistent peripheral blood (PB) monocytosis (> 1 X 10^9/L) that account for more than 10% of the white cell blood differential count, (2) absence of Philadelphia chromosome, (3) absence of PDGFRA (Platelet derived growing factor receptor-α) or PDGFRB (Platelet derived growing factor receptor-β) gene rearrangements (4) less than 20% blast and promonocytes in PB and bone marrow (BM), and (5) dysplasia involving one or more myeloid lineages. According to the newest WHO revision, this disease is further sub-classified into CMML-0 (<2% PB blast and