expressão de CD169 e CD106 (2,221±322.4x3,150±321.8, p = 0.0371; 2,186±201.7x2,721±153.5, p = 0.0238, respectivamente); e os não clássicos $CD14^{low}CD16^{+}$ (MC-NC) de CD206, CD163, CD172 e CD106 (181.2±8.5x268.6±13.4, p < 0.0001; 312.3±15.1x368.8±12.1, p = 0.0174; 13,923±2256x22,792±3211, p = 0.0161; 1,234±96x1,498±58.9 p = 0.004, respectivamente). A menor expressão de CD172 nos MC-Cs PV sugere menor sinal inibitório para a eritrofagocitose nessas células. Não houve diferença na expressão de Fpn. Apesar dos MC-Cs e MC-I já terem sido relacionados com a eritropoiese, os MC-NC apresentaram maior número de moléculas diferencialmente expressas, mostrando que também podem estar envolvidos. Isso sugere que os MCs PV são mais propensos a aderir a células eritroides e contribuir para a formação de IEs. A maior eritrofagocitose dos MCs PV circulantes foi acompanhada da maior expressão de marcadores relevantes para a adesão de células eritroides nos MCs de todos os subtipos. Mais estudos são necessários para um melhor entendimento do papel dos MCs na EE e na formação de IEs, fornecendo possíveis alvos para o tratamento da EE crônica presente na PV.

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NATURAL KILLER CELLS PHENOTYPE CORRELATES WITH TYROSINE KINASE INHIBITORS TREATMENT RESPONSE IN CHRONIC MYELOID LEUKEMIA PATIENTS

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Background: Despite well-known Natural Killer (NK) cells antitumor activity, its potential in regulating Chronic Myeloid Leukemia (CML) is not fully stablished. Tyrosine kinase inhibitors (TKI) treatment, besides reducing BCR-ABL1 transcripts, seems to result in immunomodulation that partly restore the immune dysregulation of the disease. Numerical, functional and maturation defects might be associated with CML risk stratification, molecular response profile or even with resistance to treatment with TKI. Therefore, we aimed to immunophenotypically characterize frequency, subtypes and maturation profile of NK cells in CML patients, by Multiparametric Flow Cytometry (MFC), during monitoring of TKI treatment. **Methods:** 11 Peripheral Blood samples from patients with CML (Clinical Hospital of Ribeirão Preto - HCRP-USP) and 10 healthy donors (controls) were analyzed. Clinical, laboratory and ELN2017 risk stratification were assessed. NK cells were defined as CD19-CD3-CD56+ and: CD56brightCD16- (secretory), CD56dimCD16+ (cytotoxic), CD11b-CD27- (tolerant); CD27+CD11b- (immature secretor), CD27+CD11b+ (secretory) and CD11b+CD27- (cytotoxic). Based on molecular response levels assessed by *BCR-ABL1* RT-qPCR, patients were classified in G1 (without loss of molecular response to the 1st line of TKI; n = 3), G2 (loss of molecular response to 1st line of TKI; n = 3) and G3 (loss of molecular response to 2nd generation TKI; n = 5). Mann Whitney or Kruskal-Wallis tests ($p < 0.05$) were applied using SPSS (v.20). **Results:** A higher frequency of lymphocytes in CML patients was observed when compared to controls (p = 0.0242), but no difference was found in the frequency of NK cells. G1 and G2 showed a higher frequency of NK cells than G3 (p = 0,7774). Even though no statistical significance was found, CD27-CD11b- and CD27+CD11b- NK cells showed a higher frequency, while CD27+CD11b+ NK cells showed a lower frequency in CML when compared to controls. Frequency of CD27+CD11b- NK cells was higher in G2 and G3 when compared to G1. G3 showed an immature profile with higher percentages of CD27-CD11b- and CD27+CD11b- NK cells when compared to controls, while the more mature CD27+CD11b+ NK cells showed a lower frequency in CML. Furthermore, a lower frequency of CD56brightCD16- (p = 0.0002) and CD56dimCD16+ (p = 0.0448) NK cells was observed in CML, as wells as the frequency of CD27-CD11b+ cytotoxic NK cells, consistent to what we found in G3 and supporting our hypothesis. **Conclusion:** We observed that patients with loss of molecular response in treatment with 1st and 2nd TKI generation presented an immature non-effective NK cell profile, with deficient cytotoxicity. This scenario might result in impaired tumor surveillance of CML, meaning that phenotypic changes in NK cells may directly affect risk stratification and treatment response in CML. However due to the small number of patients and the lack of a functional assay, it is not possible to confirm that the cytotoxic cell may actually be deficient. Our findings and further functional studies can contribute to a better understanding of the physiopathology, prognosis and monitoring of CML, response to TKI therapy, as well as help to define therapeutic strategies based on immunotherapy mediated by NK cells.

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PERFIL CLÍNICO E TERAPÊUTICO DAS LEUCEMIAS MIELÓIDES CRÔNICAS EM FASE CRÔNICA ASSISTIDAS EM SERVIÇO DE HEMATOLOGIA

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Objetivo: Descrição da experiência dos últimos 8 anos de acompanhamento, diagnóstico e terapêutica de pacientes com leucemia mielóide crônica (LMC) em serviço médico de hematologia & hemoterapia no interior do estado de São Paulo. **Material e métodos:** Estudo descritivo, retrospectivo e observacional realizado a partir de dados obtidos por meio da análise de prontuários eletrônicos de portadores de LMC em fase crônica admitidos e atendidos de janeiro de 2012 (ano de implantação do sistema de prontuários eletrônicos nesta instituição) a dezembro de 2020 em serviço médico