firmando o diagnóstico de LLA de células B. Iniciou tratamento de indução com vincristina 1 mg e dexametasona 20 mg semanal, durante quatro semanas de acordo com o protocolo PETHEMA. Apresentou boa resposta ao tratamento iniciando manutenção com purinetol 50 mg/noite e dexametasona 20 mg/semana. Em 06/23, seguia estável em tratamento, quando apresentou quadro infeccioso, retornando ao serviço com febre e quadro séptico que levou ao falecimento após duas semanas. **Discussão:** A patogênese da transformação da MF em LLA ainda não é bem esclarecida, mas a literatura sugere que as mutações encontradas em pacientes com fibrose medular acometem tanto a linhagem mieloide quanto a linfoide, principalmente no desenvolvimento das células B, o que permitiria a transformação em LMA ou LLA. Como a MF apresenta um fenótipo mieloproliferativo a evolução para LMA é mais frequente. Visto a raridade da apresentação linfoide associada a MF, os poucos estudos encontrados levantam a hipótese de que subgrupos distintos da LLA podem estimular uma reação fibrótica na medula óssea, tornando a MF tanto um diagnóstico diferencial quanto uma doença concomitante, uma vez que a fibrose medular pode mascarar o excesso de blastos característicos da LLA. No caso relatado, a fibrose foi diagnosticada em 2019 e muito tempo depois evoluiu para LLA, o que afasta o diagnostico inicial de LLA com fibrose. **Conclusão:** O presente relato de caso alerta para a possibilidade de evolução da MFP para LLA.

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IMMUNOPHENOTYPIC PROFILING REVEALS ALTERED EXPRESSION OF PLATELET ACTIVATION MARKERS IN PATIENTS WITH MYELOPROLIFERATIVE NEOPLASMS

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Introduction and aims: Myeloproliferative neoplasms (MPN) are hematological diseases associated with the Janus Kinase 2 (JAK2), calreticulin (CALR), and thrombopoetin receptor (MPL) genetic driver mutations. These mutations leads to a constitutive activation of the JAK-STAT pathway and results in the clonal proliferation and accumulation of myeloid cells in bone marrow and peripheral blood. Along with genetic alterations, inflammation is a key feature of MPN's pathogenesis, especially in polycythemia vera (PV), essential thrombocythemia (ET), and primary myelofibrosis (MF). The oncoinflammation *status* seems to contribute to immune cell activation, pro-coagulant state, and increased risk for thromboembolic events in MPN patients. In this context, potential alterations in platelets'immunophenotype may contribute to oncoinflammation and the occurrence of thromboembolic events in MPN patients. Therefore, the present study characterized the immunophenotype of peripheral blood platelets from patients with PV, ET, and MF and healthy subjects. **Subjects and methods:** Peripheral blood from patients with PV (n = 19; 15 JAK2+, four with undetermined mutational status), ET (n = 10; seven JAK2+, one CALR+, two triple negative), MF (n = 14; 10 JAK2+, two CALR+, two MPL+), and controls (CTRL, n = 17) were collected using the 3.2% sodium citrate buffer. The platelet-rich plasma was obtained after centrifugation and platelets were immunophenotyped using the BD LSRFortessa™ flow cytometer to quantify the percent of labeled cells (%) and the median fluorescence intensity (MFI) for CD41a, CD62P, CD36, CD38, CD63, and CD154 (CD40L) markers. These markers are involved in platelet activation and pro-inflammatory *status*. Platelets'immunophenotype was evaluated under three conditions: without stimulation, after stimulation with calcium ionophore A23187 at 0.5 μM for 15 minutes and thrombin at 50 U/mL for 15 minutes. **Results:** At basal level, PV, ET, and MF patients'presented higher frequency of CD62P+, CD36+, and CD63+ platelets than the controls. The frequency of CD63+ platelets were higher in PV and ET patients than in MF patients. The frequency of CD38+ platelets was increased in MF patients, and the frequency of CD154+ platelets was higher in PV patients than in MF patients and controls. The stimuli with calcium and thrombin increased the frequency of CD154+ platelets in controls and in PV, ET, and MF patients and the frequency of CD62P+ platelets in PV and MF patients. In the controls and in PV patients, calcium and thrombin stimuli increased the MFI of the CD62P, CD63, and CD154 markers. In ET patients, calcium stimuli increased the MFI of CD63 and CD154 markers, and in MF patients, calcium increased the MFI of CD154. In PV patients, thrombin stimuli increased the MFI of CD62P and CD154 markers. In MF patients, thrombin increased the MFI of CD62P, CD36, and CD154 markers. Thrombin stimuli did not altered the expression of any markers in ET patients'platelets. **Conclusion:** Platelets from MPN patients present alterations in the expression of activation markers at basal level and under stimuli with calcium and thrombin. These immunophenotypic alterations may contribute to oncoinflammation and to the pro-thrombotic status in PV, ET, and MF patients. **Funding:** Coordination of Superior Level Staff Improvement (CAPES), #88887.688764/2022/00.

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