apresentação inicial de LMC como LMA-M6. O diagnóstico preciso impacta diretamente na condução terapêutica e no prognóstico do paciente.

https://doi.org/10.1016/j.htct.2025.104411

ID - 1062

DEBUT OF CHRONIC MYELOID LEUKEMIA WITH EXTREME THROMBOCYTOSIS AND COEXISTENCE WITH JAK2-V617F MUTATION IN RESPONSE TO IMATINIB: CASE REPORT AND REVIEW OF THE LITERATURE

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Introduction: Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm driven by the BCR-ABL1 fusion oncogene. While thrombocytosis is common, extreme thrombocytosis at presentation is atypical. Co-occurrence of BCR-ABL1 and JAK2 mutations in CML appears rare. **Case report:** A 63-year-old male office worker was admitted emergently after routine occupational testing revealed extreme thrombocytosis. He was asymptomatic with no visceromegaly on exam. Bloodwork showed leukocytosis (WBC 45.02 K/μL) with 60% neutrophils, 9% basophils, 2% metamyelocytes/myelocytes, hemoglobin 13.5 g/dL, and platelets 2,545 K/μL. The peripheral smear demonstrated leukocytosis with maturing granulocytes, eosinophilia, <5% blasts, and marked thrombocytosis. Coagulation, hepatic, and renal profiles were normal apart from LDH 335 IU/L. He was admitted for further evaluation, with plans to start hydroxyurea cytoreduction pending confirmatory studies for definitive treatment. Bone marrow biopsy revealed 95% cellularity, markedly increased myeloid/megakaryocytic series, decreased erythroid series, and immunohistochemistry consistent with myeloproliferative neoplasm. Quantitative RT-PCR detected BCR-ABL1 p210 at 57% and JAK2 V617F mutation. Cytogenetics confirmed t(9;22) in all 20 metaphases analyzed. Imaging showed mild splenomegaly without lymphadenopathy/masses. CML in chronic phase was diagnosed. Imatinib 400 mg daily was initiated with excellent response, and JAK2 V617F persistence was confirmed on retesting. **Conclusion:** We present a rare case of chronic myeloid leukemia with extreme thrombocytosis at diagnosis, compounded by the infrequent co-occurrence of BCR-ABL1 and JAK2 V617F mutations. While the patient achieved deep molecular response on imatinib, the precise role this mutational interplay exerts on CML's clinical phenotype and molecular behavior remains incompletely characterized. Further study of such compound mutant cases is warranted to understand how these genetic lesions may modify disease manifestations and outcomes when co-occurring versus their isolated molecular subtypes. This report highlights the importance of comprehensive mutation profiling in myeloproliferative disorders to uncover unexpected findings with potential diagnostic, prognostic, and therapeutic implications.

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https://doi.org/10.1016/j.htct.2025.104412

ID - 1064

DENGUE EM PACIENTES COM LEUCEMIA MIELOIDE CRÔNICA E NEOPLASIAS MIELOPROLIFERATIVAS BCR::ABL1 NEGATIVAS

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Introdução: A dengue é uma infecção viral transmitida aos humanos por mosquitos fêmeas infectados, principalmente Aedes aegypti. No Brasil, até 6 de abril de 2024, houve 2.965.988 casos prováveis de dengue (incidência de 1.460,6/100 mil hab.). Desses, 28.395 (3,93%) foram graves ou com sinais de alerta, e 1.117 pessoas morreram (letalidade de 3,93% nesses casos). Há poucos relatos de dengue em pacientes com leucemia mieloide crônica (LMC) e neoplasias mieloproliferativas (NMP) BCR::ABL1 negativas. **Objetivos:** Avaliar características clínicas e laboratoriais de pacientes com LMC e NMP com dengue e investigar o impacto da infecção no tratamento e nos desfechos da LMC e NMP. **Material e métodos:** Estudo observacional, ambispectivo,