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Conference Abstract A24

Chronic Myeloid Leukemia with Concomitant Immune Thrombocytopenia: A Rare Case Highlighting the Importance of Bone Marrow Analysis

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Introduction

Chronic myeloid leukemia (CML) is a myeloproliferative disease characterized by the presence of the BCR-ABL fusion gene. Immune thrombocytopenia (ITP) is an autoimmune disorder leading to platelet destruction and an increased risk of bleeding. The combination of CML and ITP is extremely rare and presents diagnostic and therapeutic challenges, particularly in differentiating thrombocytopenia induced by tyrosine kinase inhibitors (TKIs) from immune-mediated mechanisms.

Objective

The aim of this clinical observation is to demonstrate a rare combination of chronic myeloid leukemia and immune thrombocytopenia, and to emphasize the key role of bone marrow histological analysis in differentiating thrombocytopenia caused by TKI therapy from autoimmune mechanisms, which is crucial for selecting the correct treatment strategy.

Methods and Case Description

A case of a 41-year-old female patient diagnosed with CML in 2016, receiving tyrosine kinase inhibitor (TKI) therapy, is presented. Persistent thrombocytopenia was initially interpreted as hematologic toxicity; however, the lack of recovery after treatment modification and a positive response to glucocorticosteroids (GCS) led to suspicion of concomitant ITP.

Molecular testing confirmed the presence of the BCR-ABL translocation (t(9;22)).

Bone marrow histology (2024) confirmed the diagnosis of ITP, revealing the following findings:

-Normocellular bone marrow (~60% cellularity) with focal moderate hypercellularity

-Adequate megakaryopoiesis with small clusters (6–8 cells), but 10–15% of megakaryocytes showed dysplastic changes (monolobulated, hyperlobulated, and anucleated forms)

-Scattered hemorrhagic foci and rare lymphoid cells in the stroma

-Absence of fibrosis and dysplasia

The combination of persistent thrombocytopenia, bone marrow findings, and response to GCS confirmed the diagnosis of ITP, allowing distinction from isolated TKI-induced thrombocytopenia.

Treatment strategy: Prednisolone (1 mg/kg/day) led to restoration of platelet count (from 10×10^9/L to 131×10^9/L), enabling continuation of nilotinib therapy.

Results

In 2023, a major molecular response (MMR) was achieved on nilotinib therapy.

In 2024, the diagnosis of ITP was confirmed by comprehensive clinical, laboratory, and histological evaluation.

This case underscores the pivotal role of bone marrow histology in differentiating TKI-induced hematologic toxicity from immune thrombocytopenia, which is critical for correct treatment strategy selection.

Conclusion

This case demonstrates the necessity of a multidisciplinary approach in managing patients with thrombocytopenia in the setting of CML. Accurate diagnosis of immune thrombocytopenia using bone marrow analysis is essential to prevent unjustified dose reductions or discontinuation of TKIs, which may negatively impact CML control.

Glucocorticosteroids remain an important therapeutic option for treating ITP in patients with CML.