

Macroglobulinemia (WM), a rare B-cell malignancy characterized by IgM overproduction, is an uncommon but important cause of secondary CAD. This case highlights the diagnostic and therapeutic challenges of CAD in a patient with relapsed WM. **Case presentation:** A 66-year-old female was diagnosed with Waldenström's Macroglobulinemia (WM) in 2012 based on a bone marrow biopsy. She initially received R-CHOP chemotherapy, achieving remission in 2015, followed by Autologous Stem Cell Transplantation (ASCT) in October 2015. After relapse in 2016, she was treated with bortezomib-rituximab followed by bortezomib monotherapy between 2016 and 2018. In October 2023, she started ibrutinib therapy for disease control. During routine blood tests in October 2023, hematologic discrepancies were noted: Hemoglobin (Hb): 7 g/L, Hematocrit (Hct): 13%, which corrected to Hb: 10.5 g/L and Hct: 31.2% after warming the sample to 37°C, raising suspicion for Cold Agglutinin Disease (CAD). Direct Coombs test was positive (1/16 IgM titer), confirming the diagnosis. Given the underlying lymphoproliferative disorder, the patient was started on rituximab therapy for CAD management while continuing ibrutinib for WM. **Conclusion:** This case underscores the importance of considering CAD in patients with hematologic malignancies presenting with unexplained anemia and hemoglobin/hematocrit discrepancies. It highlights the necessity of warming blood samples in suspected cases, preventing misinterpretation of CBC results. Additionally, it demonstrates the crucial role of rituximab in managing CAD secondary to WM by targeting IgM-producing B-cells. Early recognition and treatment of secondary CAD in lymphoproliferative disorders can prevent complications and improve patient outcomes.

**Keywords:** Autoimmune Hemolytic Anemia, Cold Agglutinin Disease, Hematologic Discrepancy, Rituximab, Waldenström's Macroglobulinemia.

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#### PP 34\_Case report

##### REVERSAL OF ACCELERATED PHASE CML WITH HIGH BLAST COUNT FOLLOWING 5+2 CHEMOTHERAPY AND DASATINIB: A CASE REPORT

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**Introduction:** Chronic Myeloid Leukemia (CML) typically progresses through chronic, accelerated, and blast phases, with most patients responding well to Tyrosine Kinase Inhibitors (TKIs) in the chronic phase. However, patients with Accelerated-Phase (AP) CML who develop high blast counts and TKI resistance are often considered at risk for transformation into Blast-Phase (BP) CML or secondary AML, requiring intensive chemotherapy or stem cell transplantation. This case

highlights a patient with AP-CML who achieved full hematologic and molecular remission after receiving 5+2 chemotherapy and dasatinib, despite a high blast count and a prolonged TKI-free period before treatment initiation. **Case presentation:** A 44-year-old male was diagnosed with CML (February 2022) and initially treated with imatinib, followed by dasatinib, bosutinib, and nilotinib due to persistent BCR-ABL positivity and extreme thrombocytosis (> 1 million/ $\mu$ L). By October 2024, disease transformation was suspected due to BCR-ABL levels rising to 85% and bone marrow biopsy showing 17% blasts. Notably, the patient had discontinued dasatinib at least three months before hospitalization, further contributing to disease progression. Given the high blast count and persistent thrombocytosis, 5+2 induction chemotherapy (cytarabine + idarubicin) was administered, followed by a reassessment bone marrow biopsy in December 2024, which was inconclusive. Post-chemotherapy, the patient refused further AML-directed treatment and instead resumed dasatinib therapy. Over the following six months, the patient's hematologic parameters normalized, and repeat bone marrow biopsy confirmed complete remission, demonstrating a remarkable reversal from the accelerated phase. **Conclusion:** This case illustrates the potential for AP-CML with a high blast count to revert to the chronic phase following 5+2 chemotherapy and re-initiation of TKI therapy. It also underscores the risks associated with TKI discontinuation in advanced CML and suggests that targeted therapy with TKIs can remain effective even after transient chemotherapy-induced cytoreduction. This highlights the importance of individualized treatment approaches in advanced CML and the potential for avoiding AML-directed therapies in select cases.

**Keywords:** 5+2 Chemotherapy, Accelerated Phase, Chronic Myeloid Leukemia, Disease Reversion, Tyrosine Kinase Inhibitor.

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#### PP 35\_Case report

##### THERAPEUTIC APHERESIS FOR EPIDERMOLYSIS BULLOSA AND SECONDARY THROMBOCYTOSIS IN NORWEGIAN SCABIES: A CASE REPORT

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**Introduction:** Secondary thrombocytosis is a well-recognized response to chronic inflammation, infections, and systemic disorders, but its association with dermatologic diseases such as Norwegian scabies and epidermolysis bullosa is rare. In severe cases of epidermolysis bullosa, therapeutic apheresis may be used as part of supportive care. This case highlights a young patient with extreme thrombocytosis managed with myelosuppressive therapy and therapeutic apheresis for