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/μL). 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

epidermolysis bullosa. **Case presentation:** A 20-year-old female with Norwegian scabies and epidermolysis bullosa was admitted due to fatigue and worsening skin lesions. Laboratory findings included severe thrombocytosis (PLT: 947,000 μL), microcytic anemia (Hb: 8.3 g/dL, MCV: 69.6 fL), elevated inflammatory markers (CRP: 118 mg/L, sedimentation rate: 63 mm/h), and positive direct Coombs test. Imaging revealed multiple mildly enlarged lymph nodes (axillary, inguinal, iliac) and hepatosplenomegaly, but bone marrow biopsy showed normocellular marrow with increased megakaryocytes. Molecular testing for JAK2, CALR, MPL, and BCR-ABL mutations was negative, ruling out Essential Thrombocythemia (ET) and Chronic Myeloid Leukemia (CML). Since the patient's thrombocytosis was determined to be secondary to chronic inflammation, she was treated with Hydroxyurea (Hydrea) 2×500 mg/day and aspirin, leading to a gradual decrease in platelet counts, confirming a reactive process rather than a primary hematologic disorder. Concurrent corticosteroid therapy for epidermolysis bullosa resulted in significant improvement in dermatologic symptoms and inflammatory markers. Given the severity of epidermolysis bullosa, therapeutic apheresis was performed as part of supportive treatment, contributing to clinical stabilization and symptom relief. **Conclusion:** This case underscores the importance of differentiating secondary thrombocytosis from primary myeloproliferative disorders and highlights therapeutic apheresis as a supportive intervention in severe epidermolysis bullosa. It emphasizes the role of multidisciplinary management, where targeting the underlying dermatologic inflammation can help control hematologic abnormalities. In complex inflammatory disorders, therapeutic apheresis may serve as an adjunct therapy, improving patient outcomes.

Keywords: Chronic Inflammation, Epidermolysis Bullosa, Norwegian Scabies, Secondary Thrombocytosis, Therapeutic Apheresis.

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PP 36_Case report

TRANSFORMATION OF FOLLICULAR LYMPHOMA INTO DIFFUSE LARGE B-CELL LYMPHOMA AFTER A DECADE OF REMISSION: A CASE REPORT

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Introduction: Follicular Lymphoma (FL) is the second most common subtype of Non-Hodgkin Lymphoma (NHL) and is generally indolent. However, a significant proportion of patients experience histologic transformation to Diffuse Large B-Cell Lymphoma (DLBCL), which leads to a more aggressive clinical course and worsened prognosis. Transformation typically occurs within the first few years of diagnosis, but this case presents a rare instance of transformation after a decade of complete remission, emphasizing the importance of long-term monitoring. **Case presentation:** A 78-year-old male was diagnosed with FL in 2014 following excisional biopsy of a left supraclavicular lymph node. The patient underwent six cycles of R-CHOP chemotherapy, achieving complete remission and remained asymptomatic for 10-years. In 2024, he presented with a rapidly enlarging anterior chest wall mass. A contrast-enhanced CT scan revealed a 46×76 cm pleural-based tumor invading the sternum and pectoral muscle. A tru-cut biopsy confirmed Diffuse Large B-Cell Lymphoma (DLBCL) with CD20 positivity. Notably, there were no systemic B symptoms (fever, weight loss, night sweats), but the rapid extranodal tumor growth raised suspicion for transformation. Given the patient's age and disease aggressiveness, rituximab plus ibrutinib therapy was initiated instead of intensive chemotherapy. The patient's response is being closely monitored. **Conclusion:** This case underscores the importance of long-term surveillance in FL patients, as transformation to DLBCL can occur even after a decade of remission. The presence of a rapidly growing, painless mass should raise suspicion for transformation, particularly in the absence of B symptoms. Extranodal involvement is a critical prognostic factor and often necessitates targeted therapeutic approaches. The use of rituximab and ibrutinib in this elderly patient represents a modern, less intensive treatment option for transformed FL, reflecting evolving lymphoma management strategies.

Keywords: Diffuse Large B-Cell Lymphoma, Follicular Lymphoma, Ibrutinib, Lymphoma Transformation, Rituximab.

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