lymphoma is challenging due to its nonspecific symptoms, such as chronic abdominal pain, weight loss, and anemia, which can mimic benign gastrointestinal disorders. This case highlights a patient with persistent GI symptoms who was ultimately diagnosed with DLBCL, underscoring the importance of considering lymphoma in cases of unexplained GI complaints and treatment-resistant anemia. A 45-year-old female presented with eight months of persistent epigastric pain, bloating, and indigestion. Despite undergoing multiple endoscopic and colonoscopic evaluations, no active pathology was identified. Due to persistent symptoms and treatment-resistant anemia, a bone marrow biopsy was performed, which was reported as normocellular. Over the next two months, she experienced unintentional weight loss of 25 kg raising suspicion for an underlying malignancy. FDG-PET/CT was performed, revealing diffuse thickening of the bowel wall in the left abdomen and periumbilical region, increased metabolic activity in mesenteric lymph nodes, mild bone marrow uptake, and abnormal activity in the anal canal. Given the concern for a lymphoproliferative disorder, the patient underwent diagnostic laparoscopy followed by excisional mesenteric biopsy, which confirmed Diffuse Large B-Cell Lymphoma (DLBCL) of non-germinal center B-cell phenotype. This case emphasizes the importance of recognizing lymphoma as part of the differential diagnosis in chronic gastrointestinal complaints, particularly when associated with unexplained anemia and significant weight loss despite normal endoscopic findings. It also underscores the critical role of PET/CT in identifying occult lymphoma and the necessity of excisional biopsy for definitive diagnosis in cases where conventional diagnostic methods fail to reveal a cause. Early recognition and diagnosis of GI-DLBCL are crucial for timely treatment and improved patient outcomes.

Keywords: Anemia, Diffuse Large B-Cell Lymphoma, Gastrointestinal Lymphoma, PET-CT, Weight Loss.

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PP 32_Case report

ACUTE MYELOID LEUKEMIA PRESENTING AS ISOLATED MYELOID SARCOMA: A CASE REPORT

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Myeloid Sarcoma (MS), also known as granulocytic sarcoma or chloroma, is a rare extramedullary tumor consisting of immature myeloid cells. It can occur as an isolated entity, concurrently with Acute Myeloid Leukemia (AML), or as a relapse manifestation. In cases where myeloid sarcoma presents without prior hematologic malignancy and with normal peripheral blood counts, diagnosis can be significantly delayed, leading to disease progression. Recognizing MS as a potential early sign of AML is crucial to initiating timely treatment. A 48-year-old female with a history of hypertension and a prior L1 vertebral compression fracture in 2016 presented with new-onset lumbar pain in 2024. Lumbar MRI revealed a paraspinal soft tissue lesion at the T12–L1 level, prompting further investigation. The patient's hematologic parameters were within normal limits, with a white blood cell count of 8290 μL, hemoglobin of 13 g/dL, and platelet count of 400,000 μL. The lesion was surgically excised, and histopathological examination confirmed myeloid sarcoma. Following this diagnosis, hematology consultation was requested, and bone marrow aspiration and biopsy were performed. Although the blast percentage was only 7%–8%, flow cytometry findings were consistent with AML. PET-CT revealed hypermetabolic activity in the paravertebral region with a maximum SUV of 10.94 and abnormal uptake in both humeri and femurs, suggesting possible bone marrow involvement. The patient was diagnosed with AML and started on 7+3 induction chemotherapy with cytarabine and daunorubicin, along with radiotherapy for local disease control. This case highlights the diagnostic challenge of isolated myeloid sarcoma in the absence of peripheral blood abnormalities and emphasizes the importance of early hematologic evaluation. PET-CT played a crucial role in detecting subclinical bone marrow involvement, guiding treatment decisions. Recognizing myeloid sarcoma as a potential precursor to AML is essential for timely diagnosis and intervention, as early systemic chemotherapy can prevent disease progression and improve patient outcomes.

Keywords: 7+3 Chemotherapy, Acute Myeloid Leukemia, Extramedullary Leukemia, Myeloid Sarcoma, Soft Tissue Involvement.

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PP 33_Case report

COLD AGGLUTININ DISEASE IN A PATIENT WITH WALDENSTRÖM'S MACROGLOBULINEMIA: A DIAGNOSTIC AND THERAPEUTIC CHALLENGE

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Introduction: Cold Agglutinin Disease (CAD) is a form of Autoimmune Hemolytic Anemia (AIHA) caused by IgM antibodies binding to erythrocytes at low temperatures, leading to complement-mediated hemolysis. CAD can be primary (idiopathic) or secondary, often associated with lymphoproliferative disorders, infections, or autoimmune diseases. Waldenström's

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