

restriction with CD38/CD138 positivity and absent marrow disease distinguishes EMP from ocular adnexal MALT lymphoma and IgG4-related disease. (iii) Therapeutic strategy—organ-sparing RT at 40–45 Gy achieves excellent control in EMP; here, 40 Gy sterilized the lesion and eliminated Bence–Jones proteinuria, implying the conjunctival clone was the source of the paraprotein. (iv) Surveillance—despite remission, EMP carries a risk of progression to multiple myeloma; our young patient remains on structured follow-up (periodic CBC, renal function, calcium, SPEP/IFE, serum free light chains, and symptom-directed imaging). **Conclusion:** This CD56-negative conjunctival EMP in a young adult underscores that meticulous staging can confirm true localization, enabling conservative surgery plus moderate-dose orbital RT to deliver durable biochemical and clinical remission. The rapid clearance of Bence–Jones lambda after RT highlights the curative potential of localized therapy while reinforcing the need for vigilant long-term monitoring.

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PP 24

TRANSFORMATION OF CHRONIC MYELOMONOCYTIC LEUKEMIA (CMML) INTO MYELOID SARCOMA: A RARE CASE WITH CERVICAL LYMPH NODE INVOLVEMENT

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Objective: Introduction: Chronic myelomonocytic leukemia (CMML) is a clonal hematologic malignancy with features of both myelodysplastic and myeloproliferative neoplasms [1]. Transformation into acute myeloid leukemia (AML) occurs in 15–20% of cases, while extramedullary presentation as myeloid sarcoma is exceedingly rare and associated with aggressive disease and poor prognosis [2,7]. **Case report: Case Presentation** A 64-year-old male diagnosed with CMML in 2024 was treated with azacitidine, achieving hematologic response after four cycles. Following the tenth cycle, he developed a cervical mass with compressive symptoms. Excisional biopsy confirmed myeloid sarcoma involving the cervical lymph node. Concurrent bone marrow analysis revealed 100% cellularity with grade 2/4 reticulin fibrosis, monocytic proliferation, and 15–16% blasts, consistent with CMML-2. Immunohistochemistry showed CD33+ and MPO+ staining, negative for CD34, CD117, and TdT. Systemic chemotherapy was planned, but the patient deteriorated rapidly with pneumosepsis and died. **Conclusion:** Discussion Extramedullary transformation of CMML into myeloid sarcoma is a rare clinical event, with limited cases reported [3]. Diagnosis can be challenging due to morphologic overlap with lymphoma, underscoring the necessity of immunophenotypic confirmation [6]. Therapeutic options remain limited, ranging from AML-type induction regimens to hypomethylating agents combined with venetoclax, and allogeneic stem cell transplantation for eligible patients [4,5,8]. However, outcomes remain poor, with median survival after extramedullary

progression of ~6 months [1,7]. **Conclusion** This case illustrates the rare transformation of CMML-2 into myeloid sarcoma with cervical lymph node involvement, highlighting diagnostic complexity, limited treatment options, and rapid disease progression. Early biopsy of new masses and bone marrow reassessment are crucial for timely diagnosis, while novel therapeutic strategies are urgently needed to improve outcomes.

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PP 25

Familial HFE Hemochromatosis in Two Siblings: Clinical Course and Response to Deferasirox

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Introduction: Hereditary hemochromatosis (HH) is an autosomal recessive disorder characterized by excessive intestinal absorption of iron and progressive iron overload. Clinical features may include hepatomegaly, cirrhosis, diabetes, cardiomyopathy, hypogonadism, and arthropathy. Diagnosis is based on transferrin saturation, serum ferritin, and confirmation by genetic testing. We present two siblings with homozygous HFE gene mutation who showed marked hyperferritinemia and a favorable response to oral deferasirox therapy. **Methods (Case Presentation):** A 40-year-old male presented to the hematology outpatient clinic on January 17, 2025, with elevated ferritin levels. His 48-year-old brother had been diagnosed with primary hereditary hemochromatosis in 2011. The elder sibling was treated with oral desferrioxamine (1 × 3 tablets daily) for several years but discontinued therapy in 2020 and remained untreated thereafter. Genetic analysis demonstrated that both siblings carried the HFE c.187C>G (p.His63Asp) homozygous mutation. At presentation, the proband's serum ferritin level was 1845 ng/mL, while his brother's level exceeded 1600 ng/mL. Both patients were started on oral deferasirox at a dose of 20 mg/kg/day (1 × 6 tablets). Regular laboratory follow-up was conducted every 4–6 weeks. **Results:** After initiation of deferasirox, both siblings demonstrated significant biochemical improvement. • The proband's ferritin decreased from 1845 ng/mL to 1090 ng/mL within three months. • The elder sibling's ferritin declined from >1600 ng/mL to 945 ng/mL in the same period. Both patients tolerated the medication well, without major adverse events. No hepatic decompensation, cardiac dysfunction, or endocrine complications were observed during follow-up. **Discussion:** This familial case illustrates several important points. First, family history and genetic testing remain critical tools in early recognition of hereditary hemochromatosis. The diagnosis in the younger sibling was established promptly because of the known history in his elder brother. Second, although phlebotomy remains the standard of care in HH, oral iron chelators such as deferasirox may be

effective alternatives, particularly in patients where phlebotomy is less feasible. In both siblings, ferritin levels declined substantially with deferasirox monotherapy. Third, interruption of treatment, as seen in the elder sibling, allows ferritin to rise again, underlining the importance of sustained long-term management. **Conclusion:** We report two siblings with homozygous HFE-related hereditary hemochromatosis and significant hyperferritinemia. Both responded favorably to deferasirox therapy with substantial reductions in ferritin levels. These findings emphasize the value of family screening, genetic testing, and consistent treatment in the management of hereditary hemochromatosis.

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Adult Hematology Abstract Categories

Lymphoma

PP 26

From CD5-Negative Indolent B-Cell LPD to Therapy-Related CLL/SLL with Unmutated IGHV After Breast Cancer: Rationale for BTK-Inhibitor–Based First-Line Therapy

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Introduction: Therapy-related chronic lymphocytic leukemia/small lymphocytic lymphoma (t-CLL/SLL) is uncommon compared with therapy-related AML/MDS. We report a breast-cancer survivor who evolved from a CD5-negative low-grade B-cell lymphoproliferative disorder (LPD) to classical CLL/SLL with **unmutated IGHV**, underscoring why targeted BTK inhibition may supersede chemo-immunotherapy in this setting. **Methods:** Single-patient case review of prospectively collected data. We extracted longitudinal clinical, imaging (PET-CT), bone-marrow histology, multiparameter flow cytometry, and cytogenetics (FISH, IGHV mutation testing). Treatment decisions were individualized by a multidisciplinary team. **Results:** A 1959-born woman had invasive ductal breast carcinoma (2009) treated with adriamycin–cyclophosphamide, weekly paclitaxel, and radiotherapy, achieving long-term remission. In 2018 bone marrow was normal; in 2019 splenectomy for progressive splenomegaly revealed florid follicular hyperplasia. Between 2020–2022, bone-marrow biopsies showed a **low-grade B-cell LPD** (CD20⁺, CD5[–]/CD23[–]/CD10[–]), managed with **rituximab–bendamustine (8 cycles)**, yielding metabolic complete remission. In 2025 she re-presented with profound fatigue and anemia. Labs showed marked lymphocytosis (WBC $46 \times 10^9/L$), hemoglobin severely reduced, and PET-CT consistent with medullary disease. Bone marrow showed **40–50% intertrabecular lymphoid infiltration**. **Flow cytometry now demonstrated classical CLL/SLL** (CD19⁺, CD20⁺, CD5⁺, CD23⁺, κ -restriction). Molecular work-up: **IGHV unmutated**; FISH: **monoallelic del(13q); del(17p)/del(11q) negative**. Given prior anthracycline exposure/radiation and the high-risk biology conferred by unmutated IGHV despite

isolated 13q deletion, the tumor board selected **acalabrutinib plus rituximab** rather than re-exposure to chemo-immunotherapy. Transfusion support and infection prophylaxis accompanied therapy planning. **Discussion:** This case is notable for: (i) **Therapy-related CLL/SLL** emerging years after breast-cancer treatment—an under-recognized survivorship risk; (ii) **Phenotypic evolution** from an initially **CD5-negative** indolent B-cell LPD to **typical CD5⁺/CD23⁺ CLL/SLL**, highlighting clonal drift and the need for repeat immunophenotyping at relapse; (iii) **Risk adjudication** where **unmutated IGHV** outweighs the generally favorable isolated **13q deletion**, steering first-line choice away from bendamustine-rituximab toward **BTK-inhibitor–based therapy**; and (iv) pragmatic considerations in a previously anthracycline-exposed patient, favoring targeted agents for efficacy and tolerability. Educationally, the case adds to scarce real-world documentation of t-CLL, illustrates immunophenotypic switch over time, and provides a clear management rationale aligned with modern risk biology. **Conclusion:** In this therapy-related CLL/SLL with **unmutated IGHV** and prior breast-cancer treatment, **acalabrutinib + rituximab** was selected as the preferred front-line strategy over chemo-immunotherapy. The case emphasizes the importance of serial phenotyping and genomics to detect evolution and to personalize therapy in cancer survivorship.

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PP 27

ISOLATED SPLENIC RELAPSE IN CLASSICAL HODGKIN LYMPHOMA: A CHALLENGING CASE REQUIRING NOVEL THERAPEUTIC APPROACHES

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Case Report: A 63-year-old male initially presented in 2024 with splenomegaly and was diagnosed with classical Hodgkin lymphoma following tru-cut biopsy. Immunohistochemistry confirmed CD30⁺, PAX5⁺, and MUM1⁺ Reed-Sternberg cells with negative CD3, CD20, and LCA expression. Initial staging revealed isolated splenic involvement without mediastinal or peripheral lymph node involvement. The patient received standard ABVD chemotherapy (adriamycin, bleomycin, vinblastine, dacarbazine) with concurrent rituximab therapy. Treatment resulted in partial response with persistent residual splenic lesions despite completing the planned regimen. In June 2025, surveillance PET-CT demonstrated disease progression with a 5-cm splenic mass showing intense metabolic activity (SUVmax: 17.2) without involvement of other anatomical sites. Bone marrow biopsy revealed 50% cellularity with normal hematopoiesis, reticulin score 0/4, negative CD30, and sparse PAX5 positivity, confirming absence of bone marrow involvement. Repeat splenic tru-cut biopsy confirmed relapsed classical Hodgkin lymphoma with characteristic immunophenotype: CD30⁺, PAX5⁺, MUM1⁺, GATA3⁺ with negative CD3, CD20, and LCA, consistent with the