

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

Esra Nur Saygeçitli\*, Hüseyin Koçak,  
Ali Turunç, Birol Güvenç

Çukurova Üniversitesi, Türkiye

**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

İdil Yürekli<sup>1,\*</sup>, Birol Güvenç<sup>2</sup>

<sup>1</sup> Çukurova University, Abdi Sutcu Health Services  
Vocational School, Adana, Türkiye

<sup>2</sup> Çukurova University, Dept. of Hematology,  
Balcalı Adana, Türkiye

**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 defer-  
asirox 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 defer-  
asirox 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 defer-  
asirox, 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 defer-  
asirox may be