HEMATOLOGY, TRANSFUSION AND CELL THERAPY

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Poster Abstracts

PP 01

CD180 EXPRESSİON ON ACUTE MYELOİD LEUKEMİA BLASTS

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Objective: CD180 is a Toll-like receptor expressed primarily in B-cell groups and has been identified as a potential therapeutic target in diseases such as B-cell non-Hodgkin lymphoma. However, the expression profile of CD180 and its clinical significance in patients with Acute Myeloid Leukemia (AML) remain largely uncharacterized. While the only existing study in the literature has reported high CD180 expression in a subset of AML samples, these findings have not been validated by other studies. Therefore, the objective of this study is to determine the presence and level of CD180 expression on leukemic blasts at the time of diagnosis in a cohort of AML patients. **Methodology:** Between November 15, 2024 and December 31, 2024, five patients diagnosed with Acute Myeloid Leukemia at the Ege University Immunology Laboratory were included in this study.Informed consent was obtained from all patients. Peripheral blood or bone marrow samples were collected at the time of diagnosis. Flow cytometry was used to determine the percentage of leukemic blasts and to evaluate the expression of CD180 on these cells. Demographic, clinical, and molecular data obtained from patient records were used for patient follow-up analyses, Türkiye. **Results:** In this study, data from a total of five AML patients, including four newly diagnosed and one with refractory disease, were evaluated. The median age of the cohort was 65 years (range: 20–66), and the patients' blast percentages ranged from 50% to 95%. Initial laboratory findings included a White Blood Cell (WBC) count ranging from 1.45 to 87.92 $\times$ 10^9/L, a platelet count from 25 to 206 $\times$ $10^3/\mu$L, and a hemoglobin (Hb) value from 6.8 to 12.2 g/dL. Flow cytometry analysis revealed that very low-density CD180 expression was present on leukemic blast cells in all five patients examined. According to molecular and cytogenetic data, three patients (ASXL1 mutation and BCR::ABL1 fusion gene) were included in the ELN Adverse Risk Group, while the remaining two patients were included in the ELN Intermediate Risk Group. Based on clinical follow-up results, one patient in the adverse risk group was deceased after 2 months, and another was deceased after 27 months. All patients in the intermediate risk group were alive at the end of an 8-month follow-up period. **Conclusion:** In conclusion, our findings demonstrate the low-density of CD180 expression on leukemic blasts in all five AML patients examined. This observation contradicts the findings of the only study in the literature. Therefore, further studies involving larger patient groups are needed to accurately determine the presence of CD180 on AML blasts.

https://doi.org/10.1016/j.htct.2025.106135

PP 02

IMPAIRED COAGULATION AT DIAGNOSIS AND INDUCTION PHASE OF ACUTE LYMPHOBLASTIC LEUKEMIA

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Objective: Disseminated intravascular coagulation (DIC) has been reported in 8-25% of acute lymphoblastic leukemia (ALL). Coagulopathy may accompany leukemia at diagnosis and during the induction phase and negatively impact prognosis. However, recognizing coagulopathy during this period can be challenging due to the accompanying bone marrow failure. Furthermore, distinguishing between asparaginase-associated hypofibrinogenemia and disseminated intravascular coagulation is challenging in clinical practice. It is important to determine which patients are at clinical risk and how they should be managed. **Methodology:** Fifty patients with

ALL followed at our center, diagnosed between 16. August.2019 and 17.June.2025 were retrospectively evaluated. The relationship between the patients' coagulation parameters and clinical data at diagnosis and during the induction period was investigated. **Results:** The median age at diagnosis was 39 (18-79), and the majority of the patients were male (31/19). Nine of the patients had T-ALL, 17 had Ph-positive B-ALL, and 24 had Ph-negative B-ALL. The median follow-up duration was 15.3 (0.2-71.9) months. At the time of diagnosis, mild hypofibrinogenemia (<200 mg/dL) was detected in 8 (17%) and severe hypofibrinogenemia (<100 mg/dL) was detected in 2 (4%) patients. During the induction phase, mild hypofibrinogenemia was detected in 36 (72%) and severe hypofibrinogenemia was detected in 11 (22%) patients. No statistically significant association was found between mild or severe hypofibrinogenemia at diagnosis and induction phase with age, gender, and ALL subtype. Fibrinogen level at diagnosis was lower in patients who developed mild hypofibrinogenemia at induction phase compared to those who did not (median 278 vs. 453) (p=0.004). In patients who received an asparaginase-containing induction regimen, both mild hypofibrinogenemia (92.9% vs. 63.9%) and severe hypofibrinogenemia (42.9% vs. 13.9%) were observed more frequently at induction phase (p=0.039 and p=0.036, respectively). In patients with mild hypofibrinogenemia at induction, the requirement for cryoprecipitate or fresh frozen plasma (FFP) was higher than in patients with normal fibrinogen levels (55.6% vs. 21.4%, p=0.030). D-dimer levels at diagnosis were higher in Ph-positive B-ALL than in Ph-negative B-ALL (median 15 vs. 4.3; p=0.030). D-dimer levels at induction phase were also higher in patients requiring cryoprecipitate or FFP (median 14.6 vs. 7.1; p=0.07). Early mortality (in the first 30 days) was 1 (2%), and was not associated with bleeding or thrombosis. No statistically significant association was found between age, gender, disease subtype, fibrinogen and D-dimer levels at diagnosis and induction phase, asparaginase use, or cryoprecipitate or FFP requirement and overall survival. **Conclusion:** In this study, we demonstrated that hypofibrinogenemia, while observed at diagnosis of ALL, is particularly prevalent during the induction phase. Hypofibrinogenemia at induction phase is determined by the fibrinogen levels at diagnosis and the use of asparaginase-containing regimens. Following, consumption of the blood products containing coagulation factors determined by the hypofibrinogenemia at induction phase. Although coagulopathy increased the frequency of blood product use, it was observed that it did not negatively impact patient survival. Clinical guidelines should be reviewed for newly diagnosed ALL patients with and without asparaginase use and updated based on large-scale studies.

https://doi.org/10.1016/j.htct.2025.106136

PP 03

RAM-like Acute Myeloid Leukemia in an Elderly Patient: A Rare Phenotypic Variant

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Introduction: Acute myeloid leukemia (AML) is a heterogeneous hematologic malignancy with diverse morphologic and immunophenotypic subtypes. The RAM (rapidly maturing) phenotype is a rare and poorly characterized variant, initially described in pediatric acute leukemia, but has also been identified in older adults (Wells et al., 2018). It is typically defined by CD45^dim, CD34−, CD117+, CD33++, and aberrant CD7 expression, with aggressive clinical behavior and poor prognosis (Nguyen et al., 2021). Here, we present a case of elderly-onset AML with RAM-like immunophenotypic features. **Methods:** A 81-year-old female presented with pancytopenia and recurrent subdural hemorrhages. Flow cytometry revealed CD45^dim, CD34−, CD117+, CD33++, and aberrant CD7+ blasts, consistent with RAM-like AML. Cytogenetic analysis showed no recurrent AML-defining translocations by FISH. Comprehensive molecular testing, including FLT3, NPM1, and CEBPA, was negative. Clinical frailty assessment demonstrated a high CIRS score, limiting intensive treatment options. **Results:** Bone marrow examination confirmed AML with RAM-like immunophenotype. Given the patient's age, comorbidities, and recurrent intracranial hemorrhages, intensive induction chemotherapy was contraindicated. Supportive care and hypomethylating agent-based therapy were considered but deferred due to poor functional status and ongoing hemorrhagic risk. The patient remained under best supportive care, including transfusions and infection prophylaxis. Prognosis was explained to the family as extremely poor, consistent with published literature (Al-Kershi et al., 2023). **Discussion:** RAM-like AML represents a high-risk immunophenotypic subset, characterized by treatment resistance and inferior outcomes (Wells et al., 2018). Most reported cases occur in children; however, adult and elderly cases are being increasingly recognized (Nguyen et al., 2021). This case highlights the diagnostic challenge and limited therapeutic options in elderly patients, particularly when performance status and comorbidities preclude intensive therapy. Early recognition through flow cytometry is essential for risk stratification and counseling. **Conclusion:** We report an elderly female with AML exhibiting RAM-like phenotype, an aggressive and rare immunophenotypic variant. Awareness of this entity is important for hematologists, as it informs prognosis and guides therapeutic decision-making, even when curative approaches are not feasible.

https://doi.org/10.1016/j.htct.2025.106137

PP 04

Incidentally Detected Precursor B-Cell Acute Lymphoblastic Leukemia in a Patient Monitored for Myocarditis: A Case Report

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Introduction: Hematologic abnormalities in young adults are frequently attributed to infections or reactive processes, yet concurrent cytopenias and lymphocytosis may herald

malignant conditions. Acute lymphoblastic leukemia (ALL) is uncommon in adults but should be considered in the presence of persistent unexplained hematologic abnormalities (Inaba et al., 2021). Here, we present a 29-year-old male patient initially hospitalized with myocarditis, in whom incidental hematologic findings prompted further investigation and ultimately led to the diagnosis of precursor B-cell acute lymphoblastic leukemia (B-ALL), Türkiye. **Methods:** The patient, with comorbid obesity, hyperlipidemia, prediabetes, and coronary artery disease, was admitted to the coronary intensive care unit due to myocarditis. Laboratory evaluation revealed neutropenia, lymphocytosis, anemia, and severe thrombocytopenia. Hematology consultation was obtained, and systematic infectious and metabolic workup was performed, including TORCH, hepatitis panel, HIV, brucella, and syphilis, all of which were negative. Nutritional deficiencies were excluded. Bone marrow aspiration and biopsy were conducted to clarify the unexplained cytopenias. **Results:** Peripheral smear showed marked lymphocytosis. Bone marrow evaluation demonstrated precursor B-cell blasts consistent with B-ALL. The patient had a prior history of episodic polycythemia treated with phlebotomy at an external center, but no prior evaluation for myeloproliferative neoplasm was documented. Physical examination was remarkable for obesity and cervical lymphadenopathy. Despite the confirmed diagnosis of B-ALL, the patient declined further therapy and left the clinic against medical advice. **Discussion:** This case underscores the diagnostic challenge posed by overlapping cardiac and hematologic findings. While myocarditis can present with systemic manifestations that mimic hematologic disorders, persistent cytopenias with lymphocytosis should prompt early hematology evaluation (Terwilliger & Abdul-Hay, 2017). Adult B-ALL often carries a poor prognosis compared to pediatric cases, and early initiation of therapy is critical to improving outcomes (Kantarjian et al., 2017). Moreover, this case highlights the importance of considering hematologic malignancy in young adults with incidental laboratory abnormalities, even in the context of alternative explanations such as infection or cardiac disease. Systematic diagnostic workup, including bone marrow biopsy, remains the gold standard for definitive diagnosis (Hunger & Mullighan, 2015). **Conclusion:** We report a young adult male followed for myocarditis in whom incidental hematologic abnormalities revealed underlying precursor B-cell ALL. This case emphasizes the necessity of maintaining a broad differential diagnosis in young adults with unexplained cytopenias and lymphocytosis, and of not delaying bone marrow evaluation. Prompt recognition is essential for timely treatment initiation and improved patient outcomes.

https://doi.org/10.1016/j.htct.2025.106138

Adult Hematology Abstract Categories

Chronic Leukemias

PP 05

A CASE OF CHRONIC LYMPHOCYTIC LEUKEMIA WITH CENTRAL NERVOUS SYSTEM INVOLVEMENT

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Although chronic lymphocytic leukemia (CLL) is very common in adults, complications associated with CLL involvement of the nervous system are very rare (1). The case report describes a CLL patient with leptomeningeal and orbital involvement. There is no standard treatment protocol for this pattern of involvement. Our patient received maintenance therapy with ibrutinib after chemoimmunotherapy, and her neurological symptoms completely resolved. CASE: A 48-year-old female housewife was diagnosed with CLL in 2018. The 17p deletion-negative patient is being followed without treatment. She has no known history of the disease. The patient presented with complaints of decreased vision, severe headache, and double vision. Her symptoms had been present for approximately two weeks. Laboratory tests revealed WBC: 156 10e3/uL, HBG: 9.2 g/dL, platelets: 188 10e3/uL, lymphocytes: 128.41 10e3/uL, creatinine: 1.07 mg/dL, urea: 48 mg/dL, LDH: 534 U/L, sodium: 134 mmol/L, potassium: 3.38 mmol/L, and sedimentation rate: 38 mm/h. Imaging revealed a liver of 180 cm and a spleen of 180 cm. Additionally, multiple lymphadenomegaly was detected in the axillary, inguinal and neck regions.An ophthalmology consultation was performed for the patient's complaints of headache, decreased vision, and diplopia. The evaluation revealed bilateral grade 3 papilledema. Detailed cranial imaging revealed no pathology during the neurological evaluation. Cerebrospinal fluid (CSF) sampling was performed. Results for neuromyelitis optica and other neurological disorders were negative. Results for meningitis were also negative. Direct microscopic examination of the CSF revealed widespread lymphocytosis consistent with CLL. The patient's headache and visual symptoms were interpreted as CLL neurological involvement. A course of R-FC was administered. A follow-up fundus examination after the course revealed resolution of the patient's grade 3 bilateral papilledema, and her headache complaints significantly decreased. Ibrutinib was initiated as maintenance therapy and the patient was discharged for routine follow-up visits. Conclusion: DISCUSSIONOur patient

presented with neurological symptoms resulting from intraorbital and leptomeningeal disease. Leptometrial disease as the initial manifestation of CLL is extremely rare (2). A large-scale CLL autopsy study reported brain and leptomeningeal involvement in 20% and 8% of cases, respectively. This study demonstrated that CNS involvement in CLL patients is underdiagnosed. Another study revealed orbital involvement in 14 of 97 autopsies (14%) of CLL patients (3). None of the studies demonstrated a correlation between leptomeningeal spread and CLL stage or duration. Standard risk factors for CNS involvement in CLL have not been systematically investigated (4).Clinical manifestations of CNS involvement in CLL are heterogeneous and include headache, cranial nerve palsies, cerebellar findings, visual problems, and motor or sensory deficits. Imaging studies do not provide sufficient evidence of CNS involvement in CLL. The diagnosis is usually confirmed by lumbar puncture. In the present case, the CSF sample showed widespread lymphocytes. In this case, a CSF sample contaminated with peripheral blood leukocytes as a result of a traumatic lumbar puncture is unlikely, as no erythrocytes and no myeloid cells were observed in the sample. The optimal treatment for CLL patients with CNS involvement is unclear. Most such patients receive treatment that includes intrathecal chemotherapy, either with or without radiotherapy or systemic chemotherapy. The most commonly used intrathecal chemotherapy agents are methotrexate, cytarabine, and corticosteroids, used alone or in combination. Our patient is currently a high-risk patient and responded well and rapidly to chemoimmunotherapy. In general, the prognosis for patients with CLL with neurological involvement is poor. Systemic chemoimmunotherapy is the most effective treatment for rapid symptom resolution in this patient group.

https://doi.org/10.1016/j.htct.2025.106139

PP 06

A CASE OF HAIRY CELL LEUKEMIA ASSOCIATED WITH CD10 EXPRESSION: THE SIGNIFICANCE OF AN ATYPICAL IMMUNOPHENOTYPIC PROFILE

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Case report: Hairy cell leukemia (HCL) represents a distinct subtype of mature B-cell lymphoproliferative disorders, predominantly affecting older individuals, with a median age of onset around 55 years. The disease exhibits a marked male predominance, with a male-to-female ratio of approximately 5:1. The spleen and bone marrow are the primary sites of involvement, and the majority of patients present with splenomegaly and pancytopenia at the time of diagnosis. Flow cytometric immunophenotyping (FCI) is an indispensable tool for the definitive diagnosis of HCL. The disease exhibits a characteristic immunophenotypic profile, defined by the absence of markers such as CD5, CD10, and CD23, and the presence of high-level or aberrantly bright expression of CD20, CD22, CD11c, and CD25. Furthermore, HCL cells are typically positive for CD103 and CD123. The current case report presents an atypical case of HCL with unexpected CD10 expression. A 36-year-old male with no comorbidities presented with a 1.5-month history of fatigue and exertional dyspnea, as well as B-symptoms. Physical examination revealed a palpable spleen in the left upper quadrant, with a dull percussion note over Traube's space, supporting the presence of splenomegaly. Initial complete blood count showed pancytopenia: a white blood cell count of 6210/μL (neutrophils: 190/μL; lymphocytes: 3120/μL; monocytes: 2880/μL), a hemoglobin level of 9.1 g/dL, and a platelet count of 56,000/μL. Further laboratory investigations for the etiology of anemia showed an iron level of 67 μg/dL, a transferrin saturation of 25%, a folate level of 9.4 ng/mL, and a vitamin B12 level of 270 pg/mL. A contrast-enhanced whole-body computed tomography (CT) scan revealed no pathological lymphadenopathy, but the spleen was measured at 140 $\times$ 60 mm. Peripheral blood smear analysis suggested the presence of atypical lymphoid cells with abundant cytoplasm. For a definitive diagnosis, a bone marrow biopsy was performed, and flow cytometry was conducted. Flow cytometry on the bone marrow samples demonstrated an increased percentage of B-lineage lymphocytes. These cells were positive for CD19, lambda light chain, CD20, CD22, FMC7, CD79a, CD27, CD11c, CD25, CD103, and CD10. HCL is a distinct lymphoproliferative disorder with an established immunophenotype essential for diagnosis. Our case, however, demonstrates atypical immunophenotypic features, posing a diagnostic challenge due to unexpected CD10 expression. While this occurrence is rare, a previous study identified aberrant CD10 expression in approximately 10% of HCL cases, suggesting such instances are not isolated. Although CD10 is a hallmark of other B-cell malignancies, such as follicular and Burkitt lymphomas, its presence should not automatically exclude HCL. In our case, the diagnosis was confirmed by the co-expression of the entire classic HCL marker panel. This highlights the crucial role of a comprehensive immunophenotyping panel, rather than reliance on a single marker, especially when faced with suboptimal morphological features or limited cell numbers. This case emphasizes the importance of expanding the differential diagnosis for CD10(+) B-cell lymphomas to include HCL. In conclusion, our case serves as a valuable reminder that the immunophenotypic profile of HCL can be more diverse than typically understood. Further research into the clinical and prognostic implications of this rare CD10 expression is warranted.

https://doi.org/10.1016/j.htct.2025.106140

Adult Hematology Abstract Categories

Chronic Myeloproliferative Diseases

PP 07

BCR-ABL1 MINOR (P190, E1A2) POSITIVE CHRONIC MYELOID LEUKEMIA: A RARE CASE REPORT

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INTRODUCTION: Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm characterized by the BCR-ABL1 fusion gene and accounts for approximately 15–20% of all leukemias. While the majority of cases harbor the p210 (major) transcript, the p190 (minor, e1a2) transcript is exceedingly rare, representing only about 1–2% of CML cases. This subtype may exhibit distinct hematologic features compared to p210-positive cases, particularly peripheral monocytosis and marked splenomegaly. In the literature, responses to tyrosine kinase inhibitor (TKI) therapy in p190-positive CML have been reported to be variable, and long-term outcomes are described only in limited case reports. Therefore, presenting the clinical and laboratory features of this uncommon subtype is of particular importance. **CASE PRESENTATION:** A 23-year-old female patient presented with complaints of fatigue and dyspeptic symptoms. Complete blood count revealed WBC: $70 \times 10^3/\mu L$, neutrophils: $58.5 \times 10^3/\mu L$, monocytes: $7.38 \times 10^3/\mu L$, hemoglobin: 10.4 g/dL, and platelets: $871 \times 10^3/\mu L$. Abdominal ultrasonography demonstrated splenomegaly with a longitudinal diameter of 175 mm. Peripheral blood smear and bone marrow aspiration-biopsy findings were consistent with chronic myeloid leukemia, with blasts reported as <5%, and the overall evaluation was described as a "myeloproliferative neoplasm." Molecular testing showed negative results for the major BCR-ABL1 transcript, whereas the minor BCR-ABL1 (e1a2) transcript was detected at 3.4%. The patient was started on first-line therapy with imatinib. At the third month of treatment, BCR-ABL1 (minor) was 10.51%, although hematologic parameters had improved. With continuation of imatinib, the sixth-month evaluation showed a decrease in BCR-ABL1 (minor) to 1.56%, with a normalized blood count (WBC: $5.31 \times 10^3/\mu L$, Hb: 10.6 g/dL, platelets: $226 \times 10^3/\mu L$). The patient's clinical symptoms had resolved, and she remains on imatinib therapy with ongoing follow-up. **DISCUSSION AND CONCLUSION:** While the p210 (major) transcript is the most frequently detected form in chronic myeloid leukemia (CML), the p190 (minor, e1a2) transcript is exceedingly rare, occurring in only about 1–2% of cases. In the literature, this subtype has been associated with peripheral monocytosis and marked splenomegaly, and responses to tyrosine kinase inhibitors (TKIs) have been reported as variable. In some patients, imatinib therapy may not achieve sufficient molecular response, whereas deeper responses have been described with second-generation TKIs. In our patient, early hematologic response was achieved with imatinib, and by the sixth month a marked molecular reduction was observed. Through this case, we aim to highlight the clinical and laboratory characteristics of p190-positive CML and to emphasize the importance of close molecular monitoring and careful evaluation of treatment response in this rare subtype.

https://doi.org/10.1016/j.htct.2025.106141

Adult Hematology Abstract Categories

Coagulation Disorders

PP 08

AN UNUSUAL DIAGNOSIS IN A TODDLER PRESENTING WITH MASSIVE GASTROINTESTINAL BLEEDING: A CASE OF ANGIODYSPLASIA AND TYPE 3 VON WILLEBRAND DISEASE

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Case report: Von Willebrand disease (VWD), caused by a deficiency or dysfunction of the von Willebrand protein (VWF), presents with a wide range of clinical manifestations. VWF is known to play a role in both platelet adhesion and angiogenesis. Consequently, defective angiogenesis can lead to angiodysplasia, particularly in the gastrointestinal system, occurring in 2-5% of VWD cases, typically in adults. Herein, we present what is, to our knowledge, the youngest reported case of a patient diagnosed with VWD following a presentation of gastrointestinal bleeding secondary to angiodysplasia. A 2-year and 10-month-old female patient was admitted to our hospital for melena and hematemesis. Her medical history was unremarkable, with no reported fever, diarrhea, or use of anti-inflammatory medications. There was no consanguinity between the parents, and no known family history of bleeding diathesis, Türkiye. Upon physical examination, the patient was lethargic, weak, and pale. A cardiac murmur was noted. Several 0.5 cm ecchymoses were present on her legs, though petechiae were absent. Initial laboratory tests revealed severe anemia (hemoglobin 3.5 g/dL). Her platelet count was within the normal range, as was her INR (0.96; normal range: 0.8-1.2). However, a prolonged aPTT (46.4 s; normal range: 20-34 s) and a bleeding time greater than 5 minutes were noted. An erythrocyte transfusion was immediately administered. Treatment with somatostatin and tranexamic acid was initiated. Despite this, the patient experienced three

more episodes of bright red bleeding and required two additional erythrocyte transfusions. An upper endoscopy was performed, revealing no esophageal varices. A 2 × 3 cm angiodysplastic lesion was observed in the gastric corpus and was cauterized with an argon laser. A scintigraphy scan confirmed increased activity in the same area. Following the procedure, a fresh frozen plasma transfusion was administered and propranolol treatment was started. With the bleeding controlled, and given the concurrent angiodysplasia, a detailed work-up for coagulopathy was performed. The patient's von Willebrand factor activity was below 5%, the von Willebrand factor antigen was below 3%, and Factor VIII was less than 3.5%. Platelet aggregation tests were normal. Genetic analysis of the VWF gene identified a novel homozygous mutation, c.7176T>G p.(Tyr2392Ter), and a heterozygous mutation, c.817C>G p.(Ar273Gly). Considering the clinical presentation, laboratory findings, and genetic analysis in a patient with no parental bleeding history, a diagnosis of Type 3 von Willebrand disease was established. Gastrointestinal bleeding due to angiodysplasia in VWD is a well-known complication that typically arises in adults. The only previously reported pediatric case was by Aggoune et al., who described a 14-year-old with Type 3 VWD and duodenal angiodysplasia who required surgical resection for recurrent bleeding. In contrast, our patient's initial bleeding episode was successfully managed with a combination of argon laser cauterization and medical therapy. This case highlights the importance of considering VWD in pediatric patients who present with severe gastrointestinal bleeding, especially when routine coagulation tests show a prolonged aPTT and bleeding time in the absence of risk factors for esophageal varices. It also serves as a crucial reminder that angiodysplasia is a known complication of VWD that can present even in the youngest of patients

https://doi.org/10.1016/j.htct.2025.106142

PP 09

Marrow-Dominant Marginal Zone Lymphoma with Plasmacytic Differentiation in a Frail and old patient: Immunophenotypic Pitfalls and Rituximab-Only Strategy

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Introduction: Marginal zone lymphoma (MZL) with plasmacytic differentiation can mimic other B-cell entities—particularly CLL/SLL and lymphoplasmacytic lymphoma (LPL)—and often presents in the elderly with cytopenias rather than bulky nodal disease. Correct classification is critical, as comorbidity and frailty frequently constrain treatment intensity. We report a bone marrow–dominant, plasmacytoid MZL in an 84-year-old woman successfully managed with rituximab monotherapy. **Methods:** We conducted a single-patient, retrospective case review of prospectively collected clinical, laboratory, pathology, and imaging data. Diagnostic workflow integrated complete blood count, immunoglobulin quantification, bone marrow histology with immunohistochemistry (IHC), multiparameter flow cytometry, and FDG-PET/CT. Treatment selection followed a frailty-adapted decision process. **Results:** An 84-year-old woman presented with progressive fatigue and dyspnea on exertion. Baseline labs showed leukocytosis with marked lymphocytosis and macrocytic pancytopenia (WBC 22.2 × 10^9/L; absolute lymphocytes 18.4 × 10^9/L; Hb 8.1 g/dL; MCV 105 fL; platelets 42 × 10^9/L). Immunoglobulins were not suggestive of LPL/WM (IgM 0.73 g/L; IgG 13.6 g/L; IgA 1.7 g/L). FDG-PET/CT revealed mediastinal/abdominal lymphadenopathy, splenomegaly, and a sternal cortical irregularity suspicious for osseous involvement. Bone marrow biopsy was hypercellular (∼95%) with ∼90% interstitial/patchy small-to-intermediate B-cell infiltration; reticulin fibrosis 0/4; Congo red negative. IHC supported a non-CLL, non-mantle phenotype: CD20 strong positive; CD5, CD23, CD10, cyclin D1, annexin A1, TRAP all negative. Flow cytometry demonstrated a clonal mature B-cell population (CD19+, CD20+, CD38+, cCD79a+) without CLL-type markers (CD5/CD23 negative). Plasmacytic differentiation was present, yet serum IgM remained normal, arguing against LPL/WM. Overall, findings established marginal zone lymphoma with plasmacytic differentiation, stage IV-A (marrow ± splenic/possible bone involvement). Given advanced age, cytopenias, and frailty, cytotoxic chemo-immunotherapy was deferred. The patient received rituximab monotherapy with antiviral prophylaxis and supportive care (transfusion as needed). Treatment was well tolerated; early follow-up showed clinical improvement with rising hemoglobin and platelet counts and reduction in lymphocytosis. **Discussion:** This case highlights three practice points. First, plasmacytic differentiation in MZL can masquerade as CLL or LPL/WM; a disciplined panel—CD5/CD23/cyclin D1 negativity with strong CD20 and compatible flow cytometry—prevents misclassification. Second, serological context matters: normal IgM helped exclude LPL/WM despite plasmacytoid histology. Third, in the very elderly/frail, rituximab monotherapy is a rational, lower-toxicity strategy that can reverse cytopenias and improve function when marrow disease predominates. The possible osseous signal on imaging further underlines the heterogeneity of MZL dissemination. Educationally, the case underscores integrating morphology, IHC, flow, and serology to secure diagnosis and tailor therapy beyond one-size-fits-all chemo-immunotherapy. **Conclusion:** Bone marrow-dominant MZL with plasmacytic differentiation presents diagnostic challenges but can be accurately classified through integrated morphology, IHC, flow cytometry, and serology. In very elderly or frail patients, rituximab monotherapy represents a rational and effective treatment strategy, offering hematologic recovery and functional benefit while minimizing toxicity.

https://doi.org/10.1016/j.htct.2025.106143

PP 10

CD5-positive Grade 3A Follicular Lymphoma Following Resected Cutaneous Squamous Cell Carcinoma: A Case Report

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Introduction: Follicular lymphoma (FL) is a germinal-center B-cell neoplasm that typically expresses CD10/BCL6 and lacks CD5. CD5-positive FL is uncommon and may mimic mantle cell lymphoma (MCL), creating critical diagnostic and therapeutic implications. We report an older male with a history of resected cutaneous squamous cell carcinoma (SCC) who presented with a new inguinal lymphadenopathy ultimately diagnosed as FL grade 3A, despite an atypical CD5-positive flow phenotype. **Methods:** This single-patient case report summarizes clinical data, ^18F-FDG PET/CT findings, flow cytometry, histopathology, and management. PET/CT was performed for staging. Lymph node excision provided tissue for histology and immunohistochemistry (IHC). Peripheral blood flow cytometry used a chronic lymphocytic leukemia (CLL) panel. Bone-marrow aspirate/biopsy were attempted for staging. **Results:** A 70-year-old man with previously excised cutaneous SCC (disease-free) was evaluated for new left inguinal lymphadenopathy. PET/CT demonstrated a metabolically active left inguinal node (~17 × 15 mm, SUVmax 12.18) with no other pathologic uptake in the neck, chest, liver, spleen, or adrenals. A posteromedial femoral hypodense nodule (~20 × 15 mm) and a subcutaneous scapular lesion (~26 × 20 mm) showed no increased FDG uptake. Excisional biopsy of the inguinal node revealed non-Hodgkin lymphoma, classic follicular lymphoma, grade 3A (WHO 2016). IHC showed CD20+, BCL6+, BCL2+, CD10+, CD21 positivity in follicular dendritic cells, CD3−, and Ki-67 ~25%. Peripheral blood flow cytometry demonstrated B-cell markers with CD5 high (~71%), CD23 low/negative (~16%), CD10 low (~4%), CD43 (~78%), and mild kappa predominance; findings raised concern for MCL. However, nodal histomorphology with CD10/BCL6 positivity supported FL. Bone-marrow aspirate was suboptimal (particle-poor), and iron score could not be assessed; marrow staging biopsy was planned. Given grade 3A FL and PET-positive nodal disease, the multidisciplinary tumor board recommended R-CHOP chemoimmunotherapy. Additional work-up (cyclin D1/SOX11 IHC and/or t(11;14) FISH) was advised to definitively exclude MCL due to CD5 expression. **Discussion:** This case highlights two challenges: (1) Dual malignancy in the same patient (prior SCC, now FL) and (2) immunophenotypic discordance between flow cytometry and histology. CD5-positive FL is rare and easily misclassified as MCL; mislabeling could alter therapy (e.g., bendamustine-rituximab vs R-CHOP and consideration of BTK inhibitors in MCL). When flow suggests MCL but node histology/IHC favors FL, tissue-based cyclin D1/SOX11 and t(11;14) are decisive. Suboptimal marrow underscores the need for core biopsy to complete staging. The absence of systemic FDG-avid disease supports localized nodal involvement at presentation. **Conclusion:** An older male with previously cured cutaneous SCC developed CD5-positive FL grade 3A presenting as isolated FDG-avid inguinal lymphadenopathy. Despite CD5 expression on flow cytometry, nodal morphology and germinal-center IHC secured an FL diagnosis, and R-CHOP was initiated. This case emphasizes rigorous correlation of flow cytometry with histopathology and the importance of cyclin D1/SOX11/t(11;14) testing when CD5 positivity creates ambiguity.

https://doi.org/10.1016/j.htct.2025.106144

PP 11

A CASE REPORT OF PRIMER REFRACTORY DIFFUSE LARGE B-CELL LYMPHOMA

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INTRODUCTION: Diffuse large B-cell lymphoma (DLBCL) is the most common histological subtype of non-Hodgkin lymphoma (NHL) and accounts for approximately one-quarter of NHL cases. Patients typically present with enlarged lymph nodes in the neck or abdomen. DLBCL's first-line immunochemotherapy such as R-CHOP (rituximab, cyclophosphamide, doxorubicin, Oncovin and prednisone) curing approximately two-thirds of patients. The prognosis is poor for DLBCL patients who receive first-line chemoimmunotherapy but develop early relapse or refractoriness. Treatment options for refractory patients include salvage chemoimmunotherapy, monoclonal antibodies, CAR-T or autologous stem cell transplantation. We will present a case of primary refractory DLBCL. **CASE:** A 57-year-old male patient with no chronic illness presented to the internal medicine outpatient clinic complaining of abdominal pain and was referred to us due to the detection of conglomerate lymphadenopathy (LAP) in the abdomen on imaging. Positron Emission Tomography (PET-CT) revealed multiple LAP's within the abdomen, the largest measuring 80 mm in diameter and with an SUV(max) value of 21.8. A tru-cut biopsy was performed from the large intra-abdominal LAP. The results were DLBCL with Bcl-2 (+), Bcl-6 (+), and Ki-67 85-90%. Myc could not be tested for technical reasons. No infiltration was detected in the bone marrow biopsy. The patient received 3 cycles of R-CHOP chemotherapy protocol, and a PET-CT scan was performed for interim evaluation. The PET-CT scan showed persistent conglomerate LAP's with an SUV(max) value of 27.02 and a maximum diameter of 58 mm. The patient, considered refractory, received two cycles of R-DHAP (Rituximab-Dexamethasone, Cytarabine Cisplatin) chemotherapy protocol and a PET-CT scan was performed for response evaluation. The PET-CT scan showed multiple LAPs, the largest of which was 83 mm in diameter and had an SUV(max) of 31.45. A tru-cut biopsy was performed again from the largest intra-abdominal lymph node for confirmation of the diagnosis. Pathology was similar to the previous biopsy and c-myc was weak (+) (10-15%). The patient received two cycles of the R-GemOX (rituximab, gemcitabine, oxaliplatin) protocol. Only abdominal CT was

scanned and no reduction in the mass was observed. Glofitamab therapy was initiated with off-label consent. The first and second cycle was completed. The patient did not develop cytokine release syndrome or neuropathy. A PET-CT scan was scheduled for 3 weeks later for response evaluation. The PET-CT scan showed that the intra-abdominal mass had regressed to 8 mm and the SUV(max) value to 3.44. The patient, who responded to glofitamab treatment, was offered autologous stem cell transplantation or CAR-T (Chimeric Antigen Receptor T-cell) therapy options. The patient requested to be referred to a center where CAR-T therapy could be performed. The patient is currently awaiting CAR-T therapy. **CONCLUSION:** In eligible DLBCL patients, salvage chemoimmunotherapy and/or monoclonal antibodies can be used as bridge therapies to OKIT or CAR-T therapies. Glofitamab is a bispecific antibody targeting CD20 and CD3, approved for r/r DLBCL patients after at least two prior lines of therapy. In a study, Glofitamab demonstrated a 46% ORR (27% CR; 19% PR) and manageable safety in heavily pretreated r/r DLBCL patients.

https://doi.org/10.1016/j.htct.2025.106145

PP 12

A CASE OF REFRACTORY MULTIPLE MYELOMA WITH HYPOGLOSSAL NERVE INVOLVEMENT

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Multiple myeloma (MM) is a clonal stem cell disease originating from plasma cells. The development of MM neurological findings is mostly caused by hyperviscosity, hypercalcemia, amyloidosis, vertebral bone involvement and spinal cord compression due to fractures or nerve compression, neuropathy due to paraproteinemia. Brain involvement is very rare. It may present as cerebral lesion, parenchymal disease or leptomeningeal involvement. A case of isolated hypoglossal nerve involvement under MM treatment at the age of 52 will be presented. Cranial nerve involvement in multiple myeloma patients can occur without the development of plasmacytoma at the skull base, but rather through infiltration of the nerve sheath with plasma cells. Combination therapies must be administered to the patient. Isolated cranial nerve involvement is a very rare complication of multiple myeloma. INTRODUCTION: Multiple myelom is clonal stem cell disease originating from plasma cells. These cells produce monoclonal immune globulins, most commonly Immunoglobulin G (IgG) or Immunoglobulin A (IgA). The disease often leads to a variety of symptoms, including anemia, bone pain, increased incidence of fracture, hypercalcemia, renal failure and increased susceptibility to infections(1).Neurological complications in MM most commonly occur due to spinal cord compression from bone lesions, paraprotein associated neuropathy, hypercalcemia, hyperviscosity or amyloidosis(2). Central nervosus system (CNS) involvement may manifest as either a solitary cerebral lesion, intra-parenchymal infiltration, or diffuse leptomeningeal disease such as CNS myelomatosis(3). The average survival after CNS involvement is 3 months (1,3). **Clinical Presentation:**A 46-year-old male patient presented with complaints of pain in the left shoulder and chest that started on January 0, 2019, in addition to weight loss, weakness in the legs, and difficulty in walking. Laboratory Findings:In the examinations performed, wbc: 8.24 10e3/uL hbg: 9.23 gr/dl, plt: 118 10e3/uL, pnl: 5.65 10e3/uL, urea: 117 mg/dl, creatinine: 3.85 mg/dl, albumin: 2.79 gr/dl, globulin: 3.81 gr/dl, corrected calcium: 9.16 mg/dl, beta2 microglobulin: 0.44 mg/dl, serum free lambda light chain: 1190 mg/l increased and serum free kappa light chain: 5.25 mg/l, 24-hour urine immunofixation electrophoresis revealed free lambda light chain: 5.65 mg/l, Chain band was detected. In the MR imaging, there were nodular lesions in the iliac bone and sacrum, immunoglobulin values were IgA: 25 mg/dl, IgG: 293 mg/dl, IgM: 68 mg/dl, 80% plasma cells in the bone marrow. Treatment: The patient was diagnosed with multiple myeloma and started on bortezomib, cyclophosphamide, dexamethasone (VCD) chemotherapy. After 4 cycles of VCD and radiotherapy (RT), an increase in light chains was observed in the control evaluation, and a bortezomib, lenalidomide and dexamethasone (VRD) course was started. The patient, who underwent autologous BMT in December 2019, was followed up under lenalidomide cordexa maintenance treatment, and a relapse was detected in the control evaluations in June 2022. Ixazomib, lenalidomide and dexamethasone (IRD) treatment was started, and daratumumab VCD treatment was switched to due to lack of response. Outcome: The patient, who was followed up under daratumumab VCD treatment, developed complaints of decreased hearing and numbness in the jaw. In addition to the atrophy in the left half of the tongue and the complaint of shifting to the left when the tongue was taken out of the mouth, speech and swallowing were impaired. (Figure 1)Neurology consultation and detailed brain imaging showed increased thickness in the right maxillary sinus. In the PET-CT imaging, although there was no pathological involvement in the head, neck and mediastinal structures, widespread lytic lesions were seen especially in various vertebrae, femur and tibia, and it was evaluated as progressive disease. No plasma cells or other pathology was detected in intrathecal sampling. Peripheral smears were examined daily with suspicion of plasma cell leukemia, but plasma cells were not detected. No signs of neurological diseases such as cranial hemorrhage or embolism were found that would cause this clinic. Conclucion and Results: The patient was evaluated in the council with neurology and it was evaluated that there was isolated myeloma involvement of nervus hypoglossus and the treatment was arranged as dartumumab, pomalidomide and dexamethasone. Brain RT was performed. After 2 cycles of chemotherapy and radiotherapy, the patient's tongue numbness, speech and swallowing disorders improved. The complaint of left shift when the tongue came out of the mouth regressed(Figure 2). The patient's isolated nervus hypoglossus involvement improved with treatment, and his follow-ups are continuing. In conclusion, cranial nerve involvement in multiple myeloma patients can occur without the development of plasmacytoma at the skull base, but rather through infiltration of the nerve sheath with plasma cells. Combination therapies must be administered to the patient. Conclusion: Brain involvement may very rarely develop in 1% of patients with multiple

myeloma (4). Parenchymal involvement may be in the form of mass compression or leptomeningeal involvement due to plasmacytomas (5). The most common cranial nerve involvements in the literature are the oculomotor nerve, abducens nerve and hypoglossal nerve (2). Involvement of these cranial nerves is most commonly due to plasmacytomas originating from the skull base and sinuses (6). When other cases of multiple myeloma with hypoglossal nerve involvement were scanned in the literature, it was seen that there was plasmacytoma or leptomeningeal involvement (7,8). In our case, it is a very rare condition in terms of the absence of a mass lesion, plasmacytoma and leptomeningeal involvement in the brain. When the case was re-examined after the literature scan, it was seen that there was a prominent paranasal sinus wall thickening on the right side that developed during the period when the patient's complaints began, without any mass lesion or leptomeningeal involvement. However, in a case report, a patient with a soft tissue mass in the right paranasal sinus had significant plasmacytomas at the skull base, and clinically, there was oculomotor, facial and hypoglossus nerve involvement (9). This situation suggests that involvement in the paranasal region may require differentiation from classical infection conditions in patients at risk and close monitoring in terms of intracranial events. Plasmacytomas of the skull base occur as an extension of plasmacytoma originating from the clivus, petrous part of the temporal bone or from the submucosa of the sinonasal and nasopharyngeal (extramedullary plasmacytoma) region. Extramedullary plasmacytomas are most commonly seen in the nasal and paranasal sinuses, nasopharynx, tonsils and larynx (10). Due to the rarity of extramedullary plasmacytomas, data on treatment and prognosis are limited. However, studies have shown the effectiveness of radiotherapy. There are publications showing that 30-50 gy radiation most effectively reduces tumor sizes caused by multiple myeloma (11)..In our case, radiotherapy and appropriate chemotherapy were initiated. Cranial nerve involvement is very rare in cases of relapsed refractory disease, and radiotherapy and combined chemotherapy are among the treatment options.

https://doi.org/10.1016/j.htct.2025.106146

Adult Hematology Abstract Categories

Platelet Diseases

PP 13

A RARE DIAGNOSIS IN ADULTS: HEREDITARY THROMBOTIC THROMBOCYTOPENIC PURPURA

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Introduction: Thrombotic thrombocytopenic purpura (TTP) is a thrombotic microangiopathy (TMA) resulting from severely reduced activity of ADAMTS-13 (a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13), a metalloproteinase responsible for cleaving von Willebrand factor (vWF). It is characterized by disseminated platelet-rich microvascular thrombi leading to organ ischemia, neurological abnormalities, renal dysfunction, thrombocytopenia, and microangiopathic hemolytic anemia (MAHA). Hereditary TTP (hTTP; also referred to as congenital TTP [cTTP] or Upshaw–Schulman syndrome) arises from pathogenic variants in the *ADAMTS-13* gene and follows an autosomal recessive inheritance pattern. Although extremely rare, it can be life-threatening. Patients with hTTP require special attention during certain life stages such as the neonatal period and pregnancy . In contrast to immune-mediated TTP (iTTP), which typically presents with a dramatic and acute onset, hTTP may manifest with a more insidious clinical picture including lethargy, impaired concentration, abdominal pain, and headache . Severe renal failure, although uncommon in iTTP, may occur in hTTP patients due to lifelong ADAMTS-13 deficiency, which can cause progressive accumulation of thrombi within the renal vasculature Hematologic examination may reveal pallor, purpura, and jaundice as signs of hemolysis, while laboratory findings typically show thrombocytopenia, unconjugated hyperbilirubinemia, elevated LDH levels, and decreased haptoglobin . Peripheral blood smear is often diagnostic, demonstrating schistocytes, nucleated red blood cells, and polychromatic red cells, consistent with intravascular hemolysis . Here, we describe the case of an 18-year-old patient with congenital TTP who was initially misdiagnosed with myelodysplastic syndrome (MDS) at an early age and received intermittent transfusions due to cytopenias. The aim of this case report is to raise clinical awareness regarding this rare and potentially fatal subtype of TTP, which can be rapidly and effectively treated if recognized early. In addition, it underscores the importance of reassessing patients at each presentation, even when a pre-existing diagnosis is available, and highlights the critical diagnostic value of peripheral blood smear, as the presence of schistocytes is pathognomonic for this condition. **Case report:** We present the case of an 18-year-old female patient, with no family history of hematologic disorders, who has been followed since childhood for anemia and thrombocytopenia. At the age of 18, she was diagnosed with congenital thrombotic thrombocytopenic purpura (TTP) and treatment was initiated. Her hematologic evaluation began in 2009, at the age of three, due to anemia and thrombocytopenia, during which she received platelet and red blood cell transfusions. Following a bone marrow examination, she was diagnosed with myelodysplastic syndrome (MDS). In 2018, she was also diagnosed with chronic kidney disease secondary to vesicoureteral reflux by the pediatric nephrology department, Türkiye. In 2024, at the age of 18, she presented to the emergency department with complaints of fatigue and dizziness. Laboratory tests revealed: WBC 5.36×10^9/L, Hgb 8 g/dL, Plt 8×10^9/L, INR 0.98, fibrinogen 211 mg/dL, creatinine 5.8 mg/dL, AST 21 U/L, ALT 15 U/L, uric acid 8.6 mg/dL, LDH 622 U/L, total bilirubin 2.5 mg/dL, direct bilirubin 0.35 mg/dL, and both direct and indirect Coombs tests were negative. The patient was admitted to the hematology clinic for further evaluation, Türkiye. Physical examination was notable only for mild pallor; no lymphadenopathy or organomegaly was detected. Peripheral

blood smear performed in our clinic demonstrated approximately 7–8% schistocytes, polychromatic erythrocytes, and thrombocytopenia, consistent primarily with thrombotic microangiopathy Review of the patient's previous laboratory records revealed episodes of anemia and thrombocytopenia, accompanied by elevated LDH, indirect bilirubin, and reticulocyte counts during these periods. ADAMTS-13 antigen, activity, and inhibitor levels were subsequently evaluated. The patient received red blood cell transfusions and 3 units of fresh frozen plasma. Laboratory results showed ADAMTS-13 activity of 23.61% (reference range: 40–130%), ADAMTS-13 antigen 0.06 IU/mL (reference range: 0.19–0.81), and ADAMTS-13 inhibitor 3.36 U/mL, with the inhibitor interpreted as negative (<12 U/mL), borderline (12–15 U/mL), or positive (>15 U/mL). Given the severely reduced ADAMTS-13 antigen and negative inhibitor, the patient was diagnosed with congenital TTP and initiated on biweekly therapeutic plasma infusion (10 mL/kg). Two weeks later, follow-up blood tests showed a platelet count of 219×10^9/L, and peripheral smear findings had completely normalized (Figure 3). At the subsequent follow-up, ADAMTS-13 tests were repeated, revealing activity <0.20%, antigen <0.01 IU/mL, and inhibitor 2.96 U/mL, consistent once again with congenital TTP. The patient continues to be followed and managed in our clinic. **DISCUSSION:** The aim of this case report is to raise awareness among clinicians about this rare syndrome, which, if accurately diagnosed, can be effectively treated, whereas misdiagnosis may lead to fatal outcomes. In neonates and children, clinicians may suspect congenital TTP in the presence of jaundice, hemolytic anemia, and thrombocytopenia. This rare syndrome was first described in 1960 by Schulman in an eight-year-old girl who experienced recurrent thrombocytopenia episodes responsive to plasma infusions [7]. In 1978, Upshaw reported a similar case in a 29-year-old patient with recurrent thrombocytopenia attacks associated with microangiopathic hemolytic anemia (MAHA), also responsive to plasma infusions, documenting multiple MAHA episodes often triggered by acute infections or stressors such as pregnancy or surgery [8].Classically, TTP is characterized by a pentad of fever, microangiopathic hemolytic anemia, thrombocytopenia, and variable renal and neurological dysfunction (observed in 20–30% of patients).However, this full presentation is often absent inmost patients [9,10].The current standard treatment for congenital TTP involves prophylactic or on-demand infusions of fresh frozen plasma (FFP) or plasma-derived factor VIII–vWF concentrates containing ADAMTS-13 for replacement therapy [11,12]. Until recently, no drug had been specifically approved for routine prophylaxis in patients with congenital TTP. Recombinant ADAMTS-13 received FDA approval in November 2023 for prophylactic or on-demand ADAMTS-13 replacement therapy in both adults and children with congenital TTP [13].Scully et al. [14] recently reported a phase 3 study comparing recombinant ADAMTS-13 with standard therapy for prophylaxis in congenital TTP patients. The study demonstrated that recombinant ADAMTS-13 is an effective prophylactic treatment approach for these patients. No safety concerns were reported, and no neutralizing antibodies against ADAMTS-13 were detected. Our patient had previously been diagnosed with MDS by apediatric hematology clinic and monitored with intermittent blood transfusions.MDS in children (≤18 years) is exceedingly rare, with an incidence of 1–4 cases per million [15].Therefore, we approached the initial diagnosis cautiously. During an MAHA episode, detailed investigations led to the diagnosis of congenital TTP. The patient responded well to fresh frozen plasma therapy. Currently, she continues biweekly FFP infusions at 10 mL/kg in our center, has not experienced further microangiopathic episodes, and remains under close follow-up.

https://doi.org/10.1016/j.htct.2025.106147

PP 14

A CASE OF X-LINKED THROMBOCYTOPENIA CONFUSED WITH IMMUNE THROMBOCYTOPENIA

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Wiskott-Aldrich Syndrome (WAS) is an X-linked disorder characterized by severe thrombocytopenia, eczema, humoral and cellular immunodeficiency, and an increased susceptibility to lymphoid malignancies. The milder form is X-linked thrombocytopenia (XLT), characterized by persistent thrombocytopenia with minimal or no signs of eczema or immunodeficiency. An eight-year-old male patient was admitted to the hospital at six months of age with complaints of coughing and wheezing. Bone marrow aspiration was performed after thrombocytopenia was detected in a complete blood count, and the bone marrow was interpreted as technically hypocellular. Immun thrombocytopenia was suspected, and follow-up was recommended. Since he had no bleeding, he did not return to our clinic. When the patient was scheduled circumcision at the age of seven, his platelet count was 30,000/mm3, so he was referred to our clinic from anesthesia. In his medical history, he had severe eczema as a baby, which later improved, and he has no history of bleeding. In his family history, his uncles also had low platelet counts, and three of his uncles underwent splenectomy for this reason, after which their platelet counts returned to normal. On physical examination, the patient had no signs of dermatitis, petechiae, purpura, or ecchymosis. The liver and spleen were palpable at 2 cm. Laboratory tests: Hgb: 12.9 g/dl, Htc: 36.7%, white blood cells: 12,360/mm3, platelets: 30,000/mm3, MPV: 9.1 fl (normal), and platelet size appeared normal in the peripheral smear. Sedimentation was normal, and immunoglobulin values were normal for age. Based on these findings, a preliminary diagnosis of XLT was considered, and WAS genetics were sent. In the WAS gene, a c.223G>A (p.V75M) hemizygous mutation was detected. The patient was diagnosed with XLT based on clinical findings and this mutation in the WAS gene. Eltrombopag treatment was initiated but was ineffective, so a splenectomy was performed.Subsequently, the platelet count reached 323,000/mm3. No decrease was observed during follow-up. This situation can cause confusion with immune thrombocytopenia (ITP) and delay the diagnosis of XLT. Since XLT patients present with a milder clinical picture than

classic WAS, treatment selection should be based on the patient's clinical presentation. Studies have shown that IVIG or antibiotic prophylaxis has no effect on the frequency or severity of infections in these patients. Since our patient did not have a history of frequent or severe infections, we did not initiate these treatments. Studies have shown that the incidence of bleeding decreases significantly after splenectomy, but the incidence of infection increases. Our patient's platelet counts returned to normal after splenectomy. To reduce the frequency of infections, we administered encapsulated bacterial vaccines and started penicillin prophylaxis for our patient. Due to the permanent morbidity in XLT, hematopoietic stem cell transplantation (HSCT) is the definitive treatment method. However, considering the side effects of HSCT, this decision should be made according to the patient's clinical condition.

https://doi.org/10.1016/j.htct.2025.106148

Adult Hematology Abstract Categories

Stem Cell Transplantation

PP 15

COMPARISON OF FEBRILE NEUTROPENIA IN PATIENTS UNDERGOING AUTOLOGOUS STEM CELL TRANSPLANTATION WITH BEAM AND HIGH-DOSE MELPHALAN CONDITIONING REGIMENS

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Objective: Objective: Conditioning regimens used before autolog stem cell transplantation (ASCT) have a direct impact on post-transplant complications and infectious morbidity. The BEAM regimen (carmustine, etoposide, cytarabine, and melphalan) is frequently preferred for patients with Hodgkin and non-Hodgkin lymphomas, while high-dose melphalan is commonly used in multiple myeloma. This study aims to compare the incidence of febrile neutropenia (FN) in patients undergoing ASCT with either the BEAM or high-dose melphalan conditioning regimen. **Methods:** In this study, febrile neutropenic patients who underwent autologous stem cell transplantation between 2010 and 2023 at the Hematology Department of Bursa Uludağ University Faculty of Medicine were analyzed. We evaluated the patients' demographic and clinicopathological data, duration of FN episodes, depth of neutropenia, and length of hospital stay. Additionally, the causative pathogens of FN and FN-related mortality were also analyzed, Türkiye. **Result:** A total of 164 patients were included in this study. Seventy-three of the patients were female and 91 were male. There were 131 multiple myeloma, 23 Non-Hodgkin lymphoma, and 10 Hodgkin lymphoma. One-hundred thirty one (%80) received high-dose melphalan, 33 (%20) received BEAM. The median dose of CD34+ cells was similar in both groups (p=0,938). The duration of FN episode and length of hospital stay were significantly longer in the BEAM arm (p=0,001 and p=0,001). Invasive pulmonary aspergillosis was significantly more common in the BEAM arm (p=0,013). Of the bacteria isolated in culture, 29% (n=48) were gram-positive and 9% (n=14) were gram-negative. The most frequently isolated gram-positive bacteria were Staphylococcus epidermidis (n=29) and Staphylococcus aureus (n=7), while gram-negative bacteria were Klebsiella pneumoniae (n=5) and Pseudomonas aeruginosa (n=4). CRP and Pitt score were similar in bot groups (p=0,152 vs p=0,247). No significant difference in FN-related mortality was seen between the two arms (p=0,802), Türkiye. **Conclusion:** Conclusion: The BEAM regimen significantly increased the risk of invasive pulmonary aspergillosis, length of hospital stay, and duration of febrile neutropenia. These results suggest that, particularly in lymphoma patients, the risks of FN should be taken into account when selecting the BEAM regimen, Türkiye.

https://doi.org/10.1016/j.htct.2025.106149

PP 16

HEMATOLOGICAL APPROACHES IN AUTOIMMUNE ENCEPHALITIS: OFATUMUMAB EXPERIENCE

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Objective: Autoimmune encephalitis (AE) is a group of encephalitides caused by immune-mediated inflammatory disorders of the brain. While B cell–mediated autoimmunity is observed in many patients, some subtypes also involve T cell–mediated mechanisms. AE-related antibodies are classified into three groups: paraneoplastic antibodies, synaptic antibodies, and antibodies of uncertain significance. Paraneoplastic antibodies are frequently associated with systemic tumors and show poor responsiveness to immunotherapy. Synaptic antibodies, on the other hand, display variable associations with systemic tumors but are generally more responsive to immunotherapy. The diagnosis of AE is based on clinical features, radiological findings (such as abnormalities on T2 and FLAIR brain MRI), slow-wave activity in the temporal lobe, cerebrospinal fluid (CSF) pleocytosis, and the exclusion of alternative causes. Although antibody detection remains one of the best diagnostic tools, many cases may still be seronegative. Common paraneoplastic antibodies include anti-Hu, anti-Yo, anti-CV2, anti-Ma2, anti-Ri, anti-amphiphysin, ZIC4, and GAD65. Major synaptic autoantibodies include anti-NMDA, anti-AMPA, anti-GABA-B receptor, anti-CASPR,

and anti-LG1. Antibody-positive AE represents a distinct subgroup of encephalopathies characterized by autoimmune responses against various antigens in the brain parenchyma[1,2]. Due to clinical, imaging, and laboratory similarities with infectious and other autoimmune encephalitides, AE remains a diagnostic challenge. Patients typically present with subacute memory and cognitive decline over days to weeks. Encephalopathic syndromes may include behavioral changes, psychosis, seizures, and coma, reflecting a broad neuropsychiatric spectrum[3]. In addition to supportive and antiepileptic therapies, early initiation of immunosuppressive treatment is essential. First-line immunosuppressive therapies in AE include corticosteroids, intravenous immunoglobulin, and plasma exchange[4,5]. For patients unresponsive to first-line treatments, second-line therapies include anti-CD20 monoclonal antibodies (rituximab and ofatumumab), mycophenolate mofetil, cyclophosphamide, and azathioprine. In refractory cases, third-line therapies such as daratumumab, bortezomib, obinutuzumab, tocilizumab, anakinra, tofacitinib, and intrathecal methotrexate may be considered[5]. Several studies have demonstrated that the use of ofatumumab, a second-generation CD20 monoclonal antibody, results in reduced antibody titers and significant clinical improvement in AE[6,7]. **Case report:** A 22-year-old female patient was admitted to an ICU in Kosovo in June 2025 with impaired consciousness and was intubated. Her Glasgow Coma Scale (GCS) score was 3. She was transferred to our hospital's intensive care unit on June 19, 2025, with a presumptive diagnosis of autoimmune encephalitis. A brain MRI performed externally on June 16 showed symmetric signal abnormalities in the bilateral basal ganglia. A lumbar puncture was performed upon admission, and CSF was sent for paraneoplastic and autoimmune antibody panels. Immunosuppressive therapy with 1000 mg methylprednisolone was initiated. For focal motor seizure control, levetiracetam (3000 mg/day) and valproic acid (2000 mg/day) were added. Plasma exchange (1/1) was started every other day beginning on June 19. Tracheal aspirate cultures grew carbapenem-resistant Acinetobacter baumannii. The patient was started on colistin, to which the isolate was sensitive. Despite therapy, focal motor seizures persisted. A brain MRI performed on June 26 showed widespread encephalitic signal changes in the left fronto-temporal and parieto-occipital regions, as well as in the right temporal region. CSF analysis revealed negative results for both paraneoplastic and autoimmune antibody panels. Given the lack of response to 1000 mg methylprednisolone and seven sessions of plasma exchange, the patient was considered to have autoimmune encephalitis refractory to first-line therapies. Daily IVIG (20 g/day for 5 days, total 100 g) was initiated, followed by second-line therapy with ofatumumab on June 27, 2025. The treatment regimen consisted of 20 mg subcutaneous injections at weeks 0, 1, and 2, followed by monthly subcutaneous injections starting from week 4. Mycophenolate mofetil (2000 mg/day) was added to the regimen. For refractory seizures, lacosamide (2 × 200 mg) was started. Clobazam was added but proved ineffective. High-dose topiramate (800 mg/day) was initiated, which achieved substantial seizure control. Antiepileptic dosages were subsequently optimized. Weekly brain MRI scans demonstrated partial regression of prior lesions. The patient developed anemia following plasma exchange, for which erythrocyte transfusions were administered. The patient was extubated on July 14, 2025, and was able to follow simple motor commands such as eye opening and closing. On July 18, she was transferred from the ICU to a general ward. Due to weight loss, a high-protein diet was initiated. A follow-up brain MRI after extubation showed partial regression and signal changes in prior lesions. Immunosuppressive therapies (ofatumumab and mycophenolate mofetil 2000 mg/day) were continued. PET-CT revealed no evidence of malignancy. Viral serologies were unremarkable. At follow-up on August 11, 2025, neurological examination revealed normal conjugate gaze, full muscle strength (5/5), normal tandem gait, mild action tremor in both hands, negative parkinsonian signs, normal deep tendon reflexes, and absence of ataxia or pathological reflexes. Speech was mildly dysphonic with occasional brief hesitations during reading. Overall, her condition was stable, and she was discharged, Türkiye. **Conclusion:** DISCUSSIONAutoimmune encephalitis is an inflammatory brain disorder that may mimic infectious and other etiologies in terms of clinical, radiological, and serological findings, making diagnosis challenging. Since diagnosis is based on exclusion, immunosuppressive therapy must be initiated promptly. First-line therapies, including corticosteroids, intravenous immunoglobulin, and plasma exchange, are generally effective. In resistant cases, second-line therapy, especially anti-CD20 monoclonal antibodies such as ofatumumab, should be considered. Studies have demonstrated favorable responses with ofatumumab in patients with AE refractory to first-line therapies[8,9]. In our case, ofatumumab led to significant clinical improvement in a patient with severe, treatment-resistant AE. In conclusion, ofatumumab represents a promising treatment option for AE patients who fail to respond to first-line therapies such as corticosteroids, intravenous immunoglobulin, and plasma exchange.

https://doi.org/10.1016/j.htct.2025.106150

Adult Hematology Abstract Categories

Akut Lösemiler

PP 17

Long-term Success of Prophylactic Intrathecal Therapy in AML Patients with CNS Involvement: Two Cases with Extended Remission Following Stem Cell Transplantation

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Case 1: A 25-year-old female (born 1993) presented in 2018 with fatigue, anemia, and pancytopenia. Bone marrow biopsy revealed high blast count with flow cytometry showing CD33 (94%), CD117 (73%), MPO (98%) positivity, and limited CD34 (3-4%) expression. Cytogenetic analysis was negative for t(8;21), inv(16), t(15;17), and BCR-ABL, classifying the case as cytogenetically normal intermediate-risk AML. Brain MRI revealed

meningeal involvement at diagnosis. The patient received standard 7+3 induction (cytarabine + idarubicin) achieving complete hematologic remission. Due to absence of suitable donor, autologous stem cell transplantation was performed. Despite achieving complete remission and remaining asymptomatic, prophylactic intrathecal methotrexate and cytarabine was initiated every 6 months for CNS protection. Serial CSF examinations from 2019-2024 showed no blast cells. Bone marrow biopsies consistently demonstrated hypocellular marrow without blasts, with negative CD34 and CD117. The patient has maintained complete remission for 7 years without neurological symptoms or complications. Case 2: A 46-year-old male (born 1973) presented in 2019 with anemia, thrombocytopenia, and fatigue. Bone marrow analysis confirmed AML with flow cytometry showing CD33 (99%), MPO (98%), high HLA-DR expression, but negative CD34 and CD117, consistent with aggressive AML phenotype. CSF examination at diagnosis confirmed CNS involvement. After achieving complete remission with standard 7+3 induction (cytarabine + daunorubicin), the patient underwent allogeneic stem cell transplantation. Similar to Case 1, prophylactic intrathecal methotrexate and cytarabine was administered every 6 months despite clinical remission. Follow-up from 2020-2023 showed consistently negative CSF examinations and stable bone marrow remission with <5% blasts. The patient has maintained complete remission for 6 years without transplant complications or neurological sequelae. Discussion: These cases demonstrate several important clinical principles in managing AML with CNS involvement. First, both patients achieved sustained remission despite CNS involvement at diagnosis, traditionally associated with poor prognosis. The combination of intensive systemic therapy, stem cell transplantation, and prolonged prophylactic intrathecal therapy appears crucial for success. The extended prophylactic intrathecal therapy regimen (6-7 years) far exceeds standard recommendations but proved remarkably safe and effective. The 6-monthly interval appears optimal, providing adequate CNS protection while minimizing procedure-related risks and patient burden compared to more frequent administration. The contrasting transplant approaches (autologous vs. allogeneic) achieved similar outcomes, suggesting that the prophylactic intrathecal strategy may be more important than transplant type for CNS disease control. Both patients demonstrated excellent tolerance to repeated lumbar punctures without cumulative neurotoxicity. The absence of CNS relapse in both cases over 6-7 years strongly supports the efficacy of this prophylactic approach. Traditional concerns about prolonged intrathecal therapy causing neurotoxicity were not observed, possibly due to the extended interval between treatments. Conclusion: Extended prophylactic intrathecal therapy administered every 6 months following stem cell transplantation represents a safe and highly effective strategy for preventing CNS relapse in AML patients with initial CNS involvement. These cases challenge conventional limitations on prophylactic therapy duration and support consideration of extended prophylaxis in high-risk patients. The excellent long-term outcomes without significant complications suggest this approach should be considered for similar cases, potentially improving survival in this traditionally poor-prognosis population.

https://doi.org/10.1016/j.htct.2025.106151

PP 18

MYELOID SARCOMA PRESENTING IN THE RETROMOLAR TRIGONE WITHOUT MARROW INVOLVEMENT

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Introduction: Myeloid sarcoma (MS), also known as granulocytic sarcoma or chloroma, is a rare extramedullary tumor composed of myeloblasts. It may occur de novo, concurrently with acute myeloid leukemia (AML), or as a relapse of previously treated AML. Oral cavity involvement is rare, and isolated presentations without bone marrow disease pose significant diagnostic challenges . MS is biologically considered equivalent to AML and should be treated accordingly, even in the absence of systemic disease . **Case Presentation:** A 51-year-old woman presented with left facial swelling and dysphagia. MRI revealed a large mass in the left retromolar trigone extending to the skull base and infratemporal region with associated mandibular bone destruction. Incisional biopsy showed sheets of immature myeloid cells. Immunohistochemistry was positive for CD117, CD34, myeloperoxidase (MPO), and CD99, with a Ki-67 proliferation index of ~40%, confirming myeloid sarcoma. PET-CT revealed a hypermetabolic mass (SUVmax 7.27) and ipsilateral cervical lymphadenopathy but no systemic FDG-avid disease. Bone marrow biopsy showed no leukemic infiltration The patient was treated for acute myeloid leukemia and was started on a 7+3 chemotherapy protocol. The patient is being monitored during the post-treatment cytopenic period. **Conclusion:** ConclusionThis case highlights the diagnostic complexity of isolated myeloid sarcoma in an unusual location. Comprehensive immunophenotypic analysis is essential for diagnosis. Although marrow was uninvolved, the patient was initiated on AML-type induction chemotherapy due to the high risk of progression . Early systemic treatment, rather than localized therapy alone, is critical to avoid transformation into overt leukemia . Systemic chemotherapy using AML-like regimens should be commenced early, even in nonleukemic disease. Surgery and/or radiotherapy may be indicated for symptomatic lesions or tumors causing local organ dysfunction or obstruction. Allogeneic hematopoietic stem cell transplantation has demonstrated promising results, particularly in patients who achieved complete remission with AML-induction protocols, and recent advances in genetic profiling may enable the development of novel targeted therapies . Clinicians should maintain a high index of suspicion for MS in atypical head and neck masses.

https://doi.org/10.1016/j.htct.2025.106152

PP 19

Oral and Maxillary Mucormycosis in a Patient with Acute Myeloid Leukemia: A Rare Case Report

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Introduction: Mucormycosis is an opportunistic fungal infection with rapid progression and high mortality, typically occurring in patients with hematologic malignancies, diabetes mellitus, organ transplantation, or prolonged immunosuppression. Clinically, the most common forms are rhinocerebral, pulmonary, cutaneous, and gastrointestinal involvement. Rhino-cerebral mucormycosis often originates in the paranasal sinuses and may extend to the orbit and brain. Oral mucormycosis is less common and usually presents with maxillary bone necrosis and palatal perforation. Early diagnosis and appropriate antifungal therapy are critical for improving prognosis. In this report, we present a case of newly diagnosed acute myeloid leukemia (AML) who developed rhino-orbito-cerebral mucormycosis involving the maxilla following chemotherapy. **Case Report:** A 52-year-old male patient was admitted to the hematology outpatient clinic with complaints of epistaxis and fatigue. Laboratory evaluation revealed pancytopenia, and peripheral smear, bone marrow aspiration, and flow cytometry confirmed the diagnosis of acute myeloid leukemia (AML). The patient received induction chemotherapy with daunorubicin (60 mg/m^2 for 3 days) and cytarabine (100 mg/m^2 for 7 days). In the second week of treatment, the patient developed pain in the left maxillary region and was referred to the Faculty of Dentistry, Inönü University. Oral and radiological examination (Figure 1)revealed a fixed dental bridge with good marginal adaptation.The prosthetic device was removed, and no pathology was observed in the teeth or surrounding mucosa, Türkiye.

https://doi.org/10.1016/j.htct.2025.106153

PP 20

A CASE OF COVID-19 PNEUMONIA DEVELOPING DURING ALLOGENEIC STEM CELL TRANSPLANTATION IN A PATIENT WITH ACUTE MYELOID LEUKEMIA

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Objective: Allogeneic hematopoietic stem cell transplantation (HSCT) is a curative therapeutic modality for patients with acute myeloid leukemia (AML)[1]. Patients undergoing HSCT are highly susceptible to SARS-CoV-2 infection due to profound immunosuppression, delayed immune reconstitution, and graft-versus-host disease (GVHD) prophylaxis[2],[4]. Covid-19 infection in HSCT recipients, particularly during the early post-transplant period, has been associated with high morbidity and mortality[5]. Studies have demonstrated increased mortality in patients diagnosed with Covid-19 during HSCT, especially in the early phase[6]. Herein, we present a case of Covid-19 pneumonia that developed during allogeneic HSCT with myeloablative conditioning in a patient with high-risk AML. **Case report:** Our patient was a 33-year-old female diagnosed with AML in March 2025. Following diagnosis, she received induction therapy with the 3+7 regimen (Idarubicin + Cytarabine). As remission was not achieved, she was administered FLAG-Mito as reinduction therapy. Despite two cycles of induction, no response was obtained, and the patient was considered refractory AML; thus, allogeneic transplantation was planned. During the pre-transplant period, she received two cycles of azacitidine + venetoclax. On July 21, 2025, myeloablative conditioning (Fludarabine + Treosulfan) was initiated. During conditioning, her caregiver developed upper respiratory tract infection symptoms and tested positive for Covid-19. Since the patient was asymptomatic, the transplantation procedure was continued. On July 28, 2025, she underwent allogeneic transplantation from her HLA-matched sibling donor. GVHD prophylaxis consisted of CsA + MTX, and voriconazole was given for antifungal prophylaxis. On day +3 post-transplant, the patient developed fever (38°C) and was treated as febrile neutropenia with broad-spectrum antibiotics (Meropenem). SARS-CoV-2 PCR testing was positive. Initially, she presented with mild symptoms, but one week after positivity, chest imaging revealed diffuse pulmonary infiltrates consistent with Covid-19 pneumonia (Figure-1). Despite broad-spectrum antibiotics, she required high-dose corticosteroids and a single dose of tocilizumab (400 mg) for cytokine release syndrome. Oxygen support was initiated. Ten days after pneumonia diagnosis, respiratory distress worsened, and she was admitted to the intensive care unit. On day +18, CPAP was initiated. Neutrophil engraftment was achieved on day +19. However, despite non-invasive respiratory support, progressive respiratory failure necessitated intubation. Shortly after intubation, the patient developed cardiac arrest and, despite CPR, she passed away. **DISCUSSION:** Patients undergoing allogeneic HSCT develop profound immunosuppression, predisposing them to opportunistic infections with high mortality. COVİD-19, which caused a global pandemic in 2020, has also emerged as a life-threatening infection in HSCT recipients[5]. Even after neutrophil engraftment, severe viral pneumonia-related mortality has been reported in transplant patients diagnosed with Covid-19[7]. When SARS-CoV-2 infection occurs in the very early post-transplant period, such as the first week, rapid clinical deterioration may occur due to insufficient immune response and cytokine dysregulation[8]. Multicenter studies have demonstrated that early post-HSCT Covid-19 infection is associated with high non-relapse mortality (NRM)[5],[9]. In our case, Covid-19 symptoms began in the early post-transplant phase and rapidly progressed to pneumonia. Despite neutrophil engraftment, the pulmonary disease worsened. This

finding is consistent with results reported during the pandemic period. Although immunosuppressive and cytokine-blocking therapies (steroids, tocilizumab) were administered, they failed to prevent mortality. **Conclusion:** Patients undergoing allogeneic HSCT and their caregivers should undergo comprehensive pre-transplant infectious disease screening. In addition, early initiation of cytokine-targeted therapies may play a role in reducing mortality in this high-risk patient population.

https://doi.org/10.1016/j.htct.2025.106154

PP 21

Autologous Transplant Alone Achieving Decade-Long Remission in AML: A Single-Center Two-Patient Case Report

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Introduction: Allogeneic HSCT is the conventional curative strategy in fit AML patients lacking favorable biology. Autologous HSCT (auto-HSCT) is rarely curative and is typically considered consolidation in selected complete remissions (CR). We report two adults with de novo AML who achieved and maintained extraordinarily long complete remissions after auto-HSCT as the sole transplant approach—highlighting potential patient-selection signals and the under-recognized curative potential of auto-HSCT in carefully chosen cases. **Methods:** This single-center, retrospective two-patient case report summarizes baseline features, induction/consolidation regimens, transplant details, and long-term outcomes from clinic records. Both patients received standard 7+3 induction, high-dose cytarabine (HiDAC) consolidation, and auto-HSCT due to the absence of suitable allogeneic donors. Follow-up included serial clinical assessment, complete blood counts, and routine biochemistry. **Results:** Case A (FM, male): Diagnosed 2017–2018 with AML; marrow blasts >20%. Immunophenotype at diagnosis included MPO positivity with CD34 −/CD117− profile. He achieved CR after 7+3 and completed four cycles of HiDAC. In 2008, he underwent auto-HSCT (G-CSF–mobilized peripheral blood). Engraftment was uneventful. Over serial evaluations he has remained in continuous first remission without relapse, secondary malignancy, or organ dysfunction. Current remission duration: 7+ years (2018→2025). Case B (MA,male): Diagnosed in 2010 with AML (marrow blasts ∼40%). After CR with 7+3 and HiDAC consolidation, lack of a matched donor prompted auto-HSCT the same year using mobilized peripheral blood stem cells. Early and late post-transplant courses were uncomplicated. He remains in continuous first remission with stable counts and no major late toxicities. Current remission duration: ∼15 years (2010→2025). **Discussion:** These two cases share three notable features. First, both achieved rapid CR to anthracycline–cytarabine induction and completed HiDAC—conditions associated with deeper molecular remissions and lower relapse risk. Second, in the absence of a donor, auto-HSCT alone consolidated remission and, in these patients, appears functionally curative across one and a half decades. Third, neither patient developed clinically significant late toxicities or second cancers during long follow-up. Although contemporary risk genomics were unavailable, the CD34-negative/MPO-positive phenotype in Case A and the brisk chemo-sensitivity in both suggest favorable disease biology. These observations reinforce that, for rigorously selected AML patients in high-quality CR—particularly when allografting is not feasible—auto-HSCT may deliver durable disease control approaching cure. The report is limited by the small sample size and absence of uniform molecular profiling; nonetheless, the remission lengths (≥15 and ≥17 years) are exceptional and educational. **Conclusion:** Two adults with AML achieved very long, ongoing first remissions (≈15 and >17 years) after autologous HSCT following 7+3 and HiDAC, without allogeneic rescue. In carefully selected CR patients lacking donors, auto-HSCT can be a valid, potentially curative strategy that merits consideration within individualized treatment algorithms.

https://doi.org/10.1016/j.htct.2025.106155

Adult Hematology Abstract Categories

Chronic Leukemias

PP 22

Richter Transformation with Spontaneous Splenic Hematoma: A Life-threatening Complication in Chronic Lymphocytic Leukemia

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Introduction: Richter transformation occurs when chronic lymphocytic leukemia transforms into aggressive lymphoma, most commonly diffuse large B-cell lymphoma (DLBCL), in approximately 5-10% of CLL patients. While typically presenting as rapidly enlarging lymph nodes, extranodal involvement can occur. Splenic transformation is uncommon, and spontaneous splenic hemorrhage represents an extremely rare, life-threatening complication requiring immediate recognition and intervention. **Case Report:** A 71-year-old female with established CLL presented with progressive abdominal pain, fatigue, and anorexia. She had previously received CLL-directed therapy with initial lymph node regression during follow-up. Physical examination revealed poor general condition, left upper quadrant tenderness with fullness, and minimal peripheral edema. No palpable lymphadenopathy was detected. Laboratory evaluation demonstrated cytopenias: hemoglobin 9.1 g/dL, leukocytosis $13.6 \times 10^3/\mu$L (lymphocyte-predominant), and thrombocytopenia $83 \times 10^3/\mu$L.

Coagulopathy was evident with prolonged PT (17.2 seconds) and elevated INR (1.46). Additional findings included suppressed TSH (0.07 mIU/L) suggesting hyperthyroidism and elevated ferritin (402 ng/mL). PET-CT performed on May 22, 2025, showed regression of previously enlarged cervical, axillary, iliac, and inguinal lymph nodes, indicating prior treatment response. However, a large hypermetabolic splenic lesion measuring 103 × 60 × 61 mm with SUVmax 32.82 was identified, with evidence of lateral capsular invasion. No bone marrow or hepatic FDG uptake was observed. Subsequent CT imaging on July 24, 2025, revealed alarming findings: intraparenchymal and subcapsular splenic hematoma with perihepatic, perisplenic, and pelvic free fluid consistent with hemoperitoneum. Additional incidental findings included a 17 mm right thyroid nodule and minimal left pleural effusion. Splenic tru-cut biopsy performed on July 17, 2025, confirmed diffuse large B-cell lymphoma with germinal center phenotype. Immunohistochemistry showed CD20(+), Bcl-2(+), Bcl-6 (+), MUM-1(+) with high proliferation index (Ki-67: 80%) and elevated c-Myc expression (60%). CD10 and CD5 were negative. The clinical constellation of findings confirmed Richter transformation with splenic DLBCL complicated by spontaneous splenic hemorrhage and hemoperitoneum, representing a medical emergency. **Discussion:** This case demonstrates an exceptionally rare presentation of Richter transformation. While most Richter transformations present with rapidly enlarging lymph nodes, isolated splenic involvement is uncommon. The extremely high SUVmax (32.82) indicated aggressive disease with high metabolic activity, consistent with high-grade DLBCL. The development of spontaneous splenic hematoma likely resulted from tumor infiltration weakening the splenic capsule and parenchyma, combined with underlying thrombocytopenia and coagulopathy. The resulting hemoperitoneum represents a life-threatening complication requiring urgent intervention. The coagulopathy and cytopenias observed may reflect both disease progression and splenic sequestration. The concurrent thyroid abnormalities warrant investigation for secondary malignancies or treatment-related complications. Management challenges include balancing the need for immediate treatment of aggressive lymphoma against the risk of exacerbating bleeding complications. Careful coordination between hematology, surgery, and radiology teams is essential for optimal outcomes. **Conclusion:** Richter transformation can present with rare but life-threatening complications including spontaneous splenic hemorrhage. High clinical suspicion, urgent imaging, and multidisciplinary management are crucial for patients with CLL developing new abdominal symptoms. This case underscores the importance of recognizing atypical presentations of Richter transformation to ensure prompt diagnosis and appropriate intervention.

https://doi.org/10.1016/j.htct.2025.106156

Adult Hematology Abstract Categories

Chronic Myeloproliferative Diseases

PP 23

CD56-Negative Conjunctival Solitary Extramedullary Plasmacytoma with Bence–Jones Lambda: Organ-Sparing Therapy and Durable Remission in a Young Adult

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Introduction: Solitary extramedullary plasmacytoma (EMP) accounts for a small fraction of plasma-cell neoplasms and rarely involves the conjunctiva. Distinguishing localized EMP from ocular adnexal lymphomas and reactive plasmacytosis is crucial, as management and prognosis differ substantially. We report a CD56-negative, lambda-restricted conjunctival EMP in a 27-year-old male with baseline Bence–Jones proteinuria, successfully treated with organ-sparing surgery plus orbital radiotherapy (RT) and maintained in remission at one year. **Methods:** This single-patient case review used prospectively recorded clinical data. Diagnostic workflow comprised ophthalmologic examination, complete blood count and chemistry, serum protein electrophoresis (SPEP) with immunofixation, 24-hour urine immunofixation, bone marrow aspirate/biopsy with immunohistochemistry (IHC), whole-body PET/CT, and brain/orbital MRI as indicated. Response was assessed clinically, biochemically (paraprotein clearance), and radiologically. **Results:** A painless, 1.5-cm vascular conjunctival mass was excised. Histology showed dense plasmacytic infiltration. IHC: CD38+, CD138+, lambda light-chain restriction, CD56−; B- and T-cell markers were non-diagnostic for lymphoma. SPEP showed no serum M-spike, while urine immunofixation revealed monoclonal lambda (Bence–Jones) positivity. Bone marrow morphology and flow cytometry demonstrated normal hematopoiesis without clonal plasma cells. PET/CT showed avid uptake confined to the conjunctival lesion; minor uptakes in stomach/sacrum lacked structural correlates. CRAB criteria were absent. Definitive local therapy consisted of adjuvant orbital RT (40 Gy in 20 fractions) after complete excision. Treatment was well tolerated. At 3 months, urine monoclonal lambda resolved; at 12 months, there was no local recurrence or new systemic disease clinically or on surveillance imaging/labs. **Discussion:** Key learning points include: (i) Localization and phenotype—conjunctival EMP is exceptional; CD56 negativity, while not universal, may be more frequent in extramedullary disease and can correlate with reduced bone tropism, supporting a truly localized process. (ii) Diagnostic clarity—lambda

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.

https://doi.org/10.1016/j.htct.2025.106157

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:** DiscussionExtramedullary 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].ConclusionThis 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.

https://doi.org/10.1016/j.htct.2025.106158

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.

https://doi.org/10.1016/j.htct.2025.106159

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 46×10^9/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.

https://doi.org/10.1016/j.htct.2025.106160

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

original diagnosis. Cardiac evaluation revealed preserved ejection fraction (65%) with mild left ventricular diastolic relaxation abnormality, indicating reasonable cardiac reserve but potential limitations for intensive chemotherapy regimens. Given the patient's age (63 years), cardiac status, and previous treatment exposure, he was deemed unsuitable for conventional high-dose salvage chemotherapy followed by ASCT. The isolated nature of splenic relapse and excellent performance status made him an ideal candidate for novel targeted approaches. Treatment planning focused on brentuximab vedotin-based combination therapy, specifically BV plus bendamustine, given the CD30+ phenotype and the patient's clinical profile. Alternative regimens including BV plus ICE or BV plus nivolumab were considered as backup options. The treatment strategy included 2-4 cycles of BV-based therapy with interim PET-CT response assessment. Achievement of PET-negative status would prompt consideration of ASCT consolidation if the patient's performance status improved, or continuation with BV maintenance or immunotherapy with nivolumab if transplant remained contraindicated. **Discussion:** This case illustrates several important aspects of relapsed Hodgkin lymphoma management. Isolated splenic relapse represents an uncommon pattern that may result from inadequate initial therapy or inherent disease biology. The patient's age and cardiac comorbidities precluded standard intensive salvage approaches, highlighting the need for effective, well-tolerated alternatives. Brentuximab vedotin, an anti-CD30 antibody-drug conjugate, has demonstrated significant efficacy in relapsed/refractory Hodgkin lymphoma, with response rates exceeding 70% in various combination regimens. The choice of BV plus bendamustine reflects a balance between efficacy and tolerability, particularly suitable for older patients. The isolated splenic presentation also raises consideration of surgical management. Splenectomy could be considered if systemic therapy fails, though the preference remains for systemic approaches given potential for occult disease. **Conclusion:** Isolated splenic relapse in classical Hodgkin lymphoma requires individualized treatment approaches, particularly in elderly patients. Brentuximab vedotin-based combinations offer effective alternatives to intensive chemotherapy, demonstrating the evolving landscape of lymphoma therapy toward more targeted, personalized treatment strategies.

https://doi.org/10.1016/j.htct.2025.106161

PP 28

SEQUENTIAL DEVELOPMENT OF DIFFUSE LARGE B-CELL LYMPHOMA FOLLOWING SUCCESSFUL HAIRY CELL LEUKEMIA TREATMENT: A CASE REPORT WITH COMPLETE REMISSION

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Case Report: A 53-year-old male from Samandağ presented in early 2024 with progressive anemia, fatigue, and splenomegaly. Laboratory evaluation revealed pancytopenia with atypical lymphoid cells on peripheral smear and mildly elevated LDH. Physical examination confirmed palpable splenomegaly without lymphadenopathy. Bone marrow biopsy performed on August 5, 2024, demonstrated classic hairy cell leukemia with characteristic immunophenotype: CD20+, CD103+, CD25+, Annexin A1+, TRAP+ with negative CD3, CD5, CD23, and CD34. Flow cytometry confirmed 8-10% clonal B-cell population with CD103+, CD25+, CD11c+ expression and aberrant kappa/lambda ratio, establishing HCL diagnosis. Treatment was initiated with rituximab plus cladribine combination therapy along with G-CSF support and prophylactic antifungal therapy. Post-treatment evaluation on September 17, 2024, demonstrated exceptional response with complete disappearance of all HCL-specific phenotypic markers (0% residual disease) and minimal CD20+ cells (1.8%) reflecting rituximab effect. Bone marrow biopsy confirmed morphologic remission. Imaging showed dramatic spleen size reduction from 22 cm to 14 cm with regression of retroperitoneal lymphadenopathy. Eight months later, in April-May 2025, the patient developed B-symptoms including persistent fever, weight loss, and dyspnea. HRCT and PET-CT revealed concerning new findings: left lower lobe pulmonary lesion with intense metabolic activity (SUVmax: 34.07), mediastinal involvement (SUVmax: 16.13), and new abdominal lymphadenopathy (SUVmax: 7-10). Lung biopsy performed on June 16, 2025, revealed diffuse large B-cell lymphoma with non-germinal center phenotype: CD20+, PAX5+, Bcl-6+, MUM1+ with extensive Bcl-2 expression (95%) and low c-Myc expression (10%), confirming systemic DLBCL diagnosis. Standard R-CHOP chemotherapy (six cycles) was administered from July through November 2025. The patient tolerated treatment well with only mild neutropenia as significant toxicity. Post-treatment PET-CT demonstrated complete metabolic remission with disappearance of all metabolically active lesions, achieving Deauville score $\leq$2. At current follow-up, the patient remains in complete remission from both malignancies with excellent performance status and no evidence of disease recurrence. **Discussion:** This case represents a rare scenario of sequential B-cell malignancies with successful treatment outcomes for both conditions. The eight-month interval between HCL remission and DLBCL development, combined with distinct immunophenotypes, suggests either treatment-related secondary malignancy or activation of a dormant malignant clone rather than clonal evolution. The non-germinal center DLBCL phenotype with high Bcl-2 expression indicates aggressive biology requiring prompt intervention. The excellent response to standard R-CHOP therapy demonstrates that DLBCL following HCL treatment responds comparably to de novo DLBCL, supporting conventional treatment approaches. This case emphasizes the critical importance of long-term surveillance in HCL patients, as secondary malignancies can develop despite achieving complete remission. The development of new constitutional symptoms or imaging abnormalities warrants thorough evaluation for secondary malignancies. **Conclusion:** Sequential development of DLBCL following successful HCL treatment represents a rare but treatable clinical scenario. Standard DLBCL therapy remains highly effective in this setting, achieving complete remission comparable to de novo cases. This case underscores the

importance of continued surveillance in HCL survivors and demonstrates excellent outcomes with appropriate treatment of secondary lymphomas.

https://doi.org/10.1016/j.htct.2025.106162

PP 29

High FDG Uptake in Low-Grade Follicular Lymphoma: A Clinico-Radiologic Discordance Case

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Case Report: A 53-year-old female presented with a 2-month history of progressive, painless left axillary mass without B-symptoms (fever, night sweats, weight loss). Medical history was unremarkable without chronic diseases, previous malignancy, or family history of cancer. Physical examination revealed good general condition with stable vital signs. A 3-cm, rubbery, mobile lymph node was palpated in the left axilla without other palpable lymphadenopathy. Abdominal examination demonstrated mild hepatomegaly (2 cm below costal margin) without splenomegaly. Laboratory evaluation showed normal complete blood count (Hb: 12.5 g/dL, WBC: 6.3×10^9/L, PLT: 220×10^9/L) with mildly elevated LDH (270 U/L). Renal and hepatic function tests were normal, and viral serologies were negative. PET-CT imaging revealed significant findings: left axillary lymphadenopathy (30×22 mm) with SUVmax 9.12, mediastinal involvement in para-aortic and aortopulmonary regions (SUVmax: 5.89), bilateral apical pulmonary nodules (7.5 mm) with low FDG uptake, and a hypodense hepatic lesion (15×12 mm) with mild FDG uptake. No splenic involvement or bone metastases were detected. Excisional biopsy of the left axillary lymph node confirmed follicular lymphoma, grade 1-2 according to WHO 2016 criteria. Immunohistochemistry demonstrated CD20(+), CD23(+), with negative CD5 and cyclin D1, consistent with follicular lymphoma. Critically, Ki-67 proliferation index was only 10%, indicating low proliferative activity. Bone marrow examination showed normal hematopoiesis with reticulin grade 0/4, negative amyloid staining, and no evidence of lymphomatous infiltration. Based on Lugano criteria, the patient was staged as advanced disease (stage IIIA-IIIB) due to mediastinal involvement and hepatomegaly. Discussion: This case presents a striking clinico-radiologic discordance between low-grade histological features and high metabolic activity. The SUVmax of 9.12 is unusually high for grade 1-2 follicular lymphoma, typically associated with more aggressive histologies or transformed lymphomas. Several mechanisms may explain this phenomenon. First, inflammatory microenvironment within lymph nodes can increase FDG uptake independent of tumor grade. Second, early transformation to diffuse large B-cell lymphoma may be focal and missed on single biopsy sampling. Third, some low-grade lymphomas may exhibit metabolically active behavior without histological transformation. The management approach requires careful consideration. While current guidelines recommend "watch and wait" for asymptomatic, low tumor burden indolent FL, the high metabolic activity and advanced stage disease create uncertainty. Options include close surveillance with repeat biopsy if progression occurs, rituximab monotherapy, or combination therapy with R-CHOP or R-bendamustine for bulky/symptomatic disease. The hepatomegaly and mediastinal involvement, combined with high SUVmax, may favor earlier intervention despite the indolent histology and absence of B-symptoms. Conclusion: High FDG uptake in low-grade follicular lymphoma represents a rare clinico-radiologic discordance that challenges standard management algorithms. This case emphasizes the importance of integrating clinical, histological, and radiological findings in lymphoma management and suggests the need for individualized treatment approaches when conventional parameters conflict. Close monitoring with consideration for earlier intervention may be warranted in such cases.

https://doi.org/10.1016/j.htct.2025.106163

PP 30

Indolent Follicular Lymphoma with "Hot" PET: A Clinic–Radiologic Mismatch That Challenges Early Treatment vs Watchful Waiting

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Introduction: Follicular lymphoma (FL) grade 1–2 typically behaves indolently and is often managed with watchful waiting when tumor burden is low. However, moderately high FDG uptake on PET-CT may suggest biological heterogeneity or incipient transformation despite low-grade histology, creating a management dilemma. We report a patient with biopsy-proven FL grade 1–2 and unexpectedly "hot" PET signals, illustrating decision points between immediate therapy and surveillance. **Methods:** Single-patient case report. We reviewed clinical data, laboratory tests, excisional lymph-node histology with immunohistochemistry (IHC), bone marrow (BM) evaluation, and whole-body PET-CT at diagnosis. Management decisions were based on symptoms, tumor burden, and longitudinal imaging. **Results:** A 53-year-old woman presented with a painless, mobile left axillary mass detected 2 months earlier. She denied fever, drenching night sweats, or weight loss. Physical exam revealed a ~3 cm left axillary node; no hepatosplenomegaly or other palpable lymphadenopathy. PET-CT demonstrated a **30 × 22 mm** left axillary node with **SUVmax 9.12**, additional mediastinal paraaortic/aortopulmonary nodes (**SUVmax 5.89**), and tiny bilateral apical lung nodules with low uptake. The liver contained a **15 × 12 mm** hypodense lesion with faint FDG avidity and mild hepatomegaly; spleen and adrenals were normal; bone involvement was absent. Excisional node biopsy showed **classical FL, grade 1–2**. IHC: **CD20+, CD23+, CD5−, Cyclin D1−; Ki-67 ≈10%**. BM aspirate/biopsy exhibited normal hematopoiesis with **no lymphoma infiltration** (reticulin 0/4; amyloid negative). Baseline blood counts and biochemistry were within

reference limits except for a mildly elevated LDH. Composite staging favored **advanced-stage (IIIA–IIIB) FL** owing to mediastinal involvement and hepatomegaly, yet **clinical tumor burden was low**: solitary bulky node absent, no B symptoms, preserved counts, and no organ compromise. Given the discrepancy—**indolent histology with relatively high axillary SUV**—management options were discussed. Because transformation was not proven (low Ki-67, no high-grade features on biopsy, and no PET focus >10 with structural suspicion elsewhere), we selected **watchful waiting** with close clinical and PET/CT surveillance, reserving therapy for symptomatic progression, GELF high-tumor-burden criteria, rising SUVs or node growth, or any histologic evidence of transformation (repeat biopsy triggered by interval changes). Single-agent rituximab or R-based chemoimmunotherapy would be considered if progression occurs. **Discussion:** This case highlights a **clinic–radiologic mismatch**: low-grade FL with **SUVmax ~9** in the index node. While high SUVs in FL can raise concern for transformation, histology and low proliferation argued against immediate cytotoxic therapy. In asymptomatic, low-burden FL, **watch-and-wait remains appropriate**, provided that surveillance is disciplined and **re-biopsy is performed for PET-dominant changes** or clinical progression. Educationally, the case underscores the limits of relying on SUV alone, the centrality of tissue confirmation, and the value of individualized triggers for treatment versus observation. **Conclusion:** In FL grade 1–2 with "hot" PET but low clinical burden, **structured watchful waiting with planned re-biopsy on interval change** can safely balance overtreatment risks against the need to detect transformation early.

https://doi.org/10.1016/j.htct.2025.106164

PP 31

Mantle Cell Lymphoma Presenting with Gastrointestinal Bleeding in an Elderly Patient: A Case of Stage IV Disease Treated with Rituximab Monotherapy

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Case Report: An 85-year-old male presented with progressive fatigue and melena over several weeks. His medical history was notable for advanced age with overall frailty but no significant comorbidities. Physical examination revealed poor general condition with pallor and mild dehydration. No palpable lymphadenopathy, hepatomegaly, or splenomegaly was detected on initial examination. Laboratory evaluation demonstrated severe anemia (hemoglobin 8.1 g/dL, hematocrit 27%) with significant leukocytosis (20.9×10^9/L) and marked monocytosis (46%). Platelet count remained normal (181×10^9/L). Additional findings included hypoalbuminemia (28.5 g/L), elevated LDH (218 U/L), and moderate renal impairment (creatinine 1.23 mg/dL, eGFR 53 mL/min). Endoscopic evaluation revealed erosive pangastritis with antral and duodenal ulcers. Colonoscopy identified a 3.5-4 cm ulcerative, polypoid mass in the cecum with additional rectal involvement prompting biopsy. Histopathological examination of gastrointestinal biopsies confirmed mantle cell lymphoma with characteristic immunophenotype: CD20(+), Cyclin D1(+), SOX11(+), BCL2(+), and CD43(+) with negative CD3, CD5, and CD23. The Ki-67 proliferation index was 20%, indicating moderate proliferative activity. PET-CT staging revealed extensive disease with widespread lymphadenopathy involving cervical, axillary, mediastinal, retroperitoneal, and pelvic regions. Gastrointestinal involvement showed intense FDG uptake (SUVmax 12.1) in cecum and rectum. Diffuse hepatic and splenic involvement was present along with diffuse bone marrow uptake, establishing stage IV disease. Given the patient's advanced age (85 years), frailty, history of gastrointestinal ulceration, and moderate renal impairment, intensive chemotherapy regimens were deemed inappropriate. Treatment was initiated with rituximab monotherapy (626 mg every 28 days) with antiemetic prophylaxis. BTK inhibitor therapy was considered but deferred due to high bleeding risk given active gastrointestinal ulceration. Supportive care included proton pump inhibitor therapy and red blood cell transfusions as needed. The patient demonstrated good tolerance to rituximab therapy with early symptomatic improvement and stabilization of hematological parameters. **Discussion:** This case illustrates several important aspects of MCL management in elderly patients. The presentation with gastrointestinal bleeding and extensive disease is typical for MCL, which frequently involves the GI tract at diagnosis. The moderate Ki-67 proliferation index (20%) suggested less aggressive biology, supporting a less intensive treatment approach. The decision to use rituximab monotherapy reflects the growing recognition that treatment intensity must be individualized based on patient fitness and comorbidities. While intensive regimens like hyperCVAD or Nordic protocols achieve superior outcomes in younger patients, they carry prohibitive toxicity in frail elderly populations. Rituximab monotherapy has shown activity in MCL with response rates of 40-60% and manageable toxicity profiles, making it suitable for elderly, frail patients. The early tolerance and symptomatic improvement observed support this approach. **Conclusion:** MCL management in elderly, frail patients requires individualized treatment decisions balancing disease control with quality of life. Rituximab monotherapy represents a reasonable option for patients unsuitable for intensive chemotherapy, providing disease control with acceptable toxicity. This case demonstrates the feasibility of this approach in carefully selected patients.

https://doi.org/10.1016/j.htct.2025.106165

Adult Hematology Abstract Categories

Myeloma

PP 32

Secondary Primary Malignancy in Multiple Myeloma: Prostate Adenocarcinoma Following Long-term Lenalidomide Maintenance Therapy

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Introduction: Multiple myeloma patients have an increased risk of developing secondary primary malignancies, with reported incidence ranging from 3-20% depending on treatment regimens and follow-up duration. Lenalidomide maintenance therapy following autologous stem cell transplantation significantly improves progression-free survival but carries potential long-term risks including secondary malignancies. While hematologic secondary malignancies are well-documented, solid tumor development during lenalidomide maintenance is less frequently reported but increasingly recognized. Case Report: A 73-year-old male initially presented in 2016 with fatigue, bone pain, and normocytic anemia. Laboratory evaluation revealed IgG-kappa multiple myeloma with positive serum M-protein, elevated free light chain kappa/lambda ratio, and 40% plasma cell infiltration on bone marrow biopsy. Imaging demonstrated extensive osteolytic lesions without renal involvement. Family history was negative for malignancy, and the patient had no smoking history or significant comorbidities. Initial treatment consisted of bortezomib, lenalidomide, and dexamethasone (VRD) induction therapy from 2016-2017. Following excellent response, the patient underwent high-dose melphalan conditioning and autologous stem cell transplantation in 2017 without complications. Lenalidomide maintenance therapy (10 mg daily) was initiated in 2018, with regular hematology follow-up demonstrating sustained remission through 2023. During routine surveillance in 2024, elevated PSA (8.4 ng/mL) was detected, prompting urological evaluation. Prostate biopsy performed on August 20, 2024, revealed adenocarcinoma in two locations: right apex showing Gleason 6 (3+3), Grade Group 1 with 20% tumor involvement, and right basal region with Gleason 6 (3+3), Grade Group 1 with 5% tumor involvement. Remaining biopsy cores showed benign prostate tissue. Immunohistochemistry with high molecular weight keratin confirmed the diagnosis. Computed tomography on January 27, 2025, demonstrated prostatomegaly (64×51 mm), right renal pelvic dilatation ($\sim$3 cm) with 4 mm right ureteral stone, 4 mm left renal cyst, and multiple enlarged periaortic and peripancreatic lymph nodes, raising concern for advanced prostate cancer or possible myeloma progression. The patient continued to show no evidence of myeloma progression with maintained remission status throughout this period. However, the constellation of prostatic enlargement and lymphadenopathy suggested either advanced prostate cancer or concurrent disease processes requiring careful differentiation. Discussion: This case illustrates several important clinical considerations in long-term multiple myeloma survivorship. The development of prostate adenocarcinoma following 6 years of lenalidomide maintenance raises questions about treatment-related secondary malignancy risk. While lenalidomide-associated secondary malignancies typically manifest as hematologic disorders, solid tumors including prostate cancer have been reported with increasing recognition. The clinical challenge lies in distinguishing between prostate cancer progression and myeloma relapse, particularly given the lymphadenopathy observed on imaging. The patient's sustained myeloma remission suggests the lymph node enlargement may represent prostate cancer dissemination rather than plasma cell dyscrasia. The low-grade nature of the prostate adenocarcinoma (Gleason 6) typically indicates indolent disease, but the substantial prostatic enlargement and lymphadenopathy suggest more advanced local disease requiring comprehensive staging and multidisciplinary treatment planning. Conclusion: This case demonstrates the importance of comprehensive long-term surveillance for secondary primary malignancies in multiple myeloma patients receiving lenalidomide maintenance therapy. The development of solid tumors, particularly prostate cancer, warrants systematic screening and multidisciplinary management to optimize outcomes while maintaining myeloma disease control.

https://doi.org/10.1016/j.htct.2025.106166

PP 33

AL Amyloidosis Presenting with Cardiac Involvement in a 43-Year-Old Woman with Oligosecretory Multiple Myeloma

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Objective: Introduction: AL amyloidosis results from deposition of misfolded immunoglobulin light chains in various organs, with cardiac involvement occurring in approximately 60-70% of cases. Cardiac amyloidosis typically presents with heart failure symptoms and distinctive echocardiographic features including increased wall thickness, "sparkling" myocardium appearance, and restrictive physiology. While commonly associated with multiple myeloma, oligosecretory variants can pose diagnostic challenges due to minimal or absent monoclonal protein secretion in serum. **Case Report:** A 43-year-old female presented with progressive palpitations, dyspnea, fatigue, and peripheral edema. Initial evaluation by cardiology revealed significant cardiac abnormalities prompting comprehensive investigation. Echocardiography demonstrated characteristic findings highly suggestive of cardiac amyloidosis: concentric left ventricular hypertrophy with "sparkling" myocardium appearance, restrictive diastolic

pattern, biatrial enlargement, moderate tricuspid regurgitation, and mild pulmonary hypertension. The constellation of findings was inconsistent with hypertensive heart disease, raising suspicion for infiltrative cardiomyopathy. Given the typical echocardiographic appearance, the patient was referred to hematology for amyloidosis evaluation. Laboratory assessment revealed elevated inflammatory markers (CRP: 59-74 mg/L) but notably, serum protein electrophoresis showed no distinct M-band. However, serum immunofixation was positive only for lambda light chains with negative IgA, IgG, and IgM, suggesting an oligosecretory plasma cell disorder. Bone marrow biopsy revealed 40% plasma cell infiltration with immunophenotype showing CD38(+), CD56(+), and CD19 (-) with lambda light chain restriction. Critically, Congo red staining was positive, confirming amyloid deposition and establishing the diagnosis of AL amyloidosis. Cytogenetic analysis by FISH was negative for high-risk abnormalities including p53 deletion, RB1 deletion, t(11;14), and t(4;14). Additional imaging revealed multisystem involvement: chest CT showed ground-glass opacities in lower lobes with reactive mediastinal lymphadenopathy, while abdominal ultrasound demonstrated grade 1 hepatosteatosis, minimal splenomegaly, and mild ascites, consistent with systemic amyloid deposition. The patient's medical history was notable for appendiceal mucinous neoplasm in 2022, raising questions about potential relationships between these conditions. Clinical presentation included progressive heart failure symptoms with peripheral edema, confirming cardiac involvement as the primary manifestation. **Discussion:** This case illustrates several important clinical aspects of AL amyloidosis. The presentation in a 43-year-old patient is relatively uncommon, as AL amyloidosis typically affects older adults with median age around 65 years. The cardiac-predominant presentation with characteristic echocardiographic findings enabled early recognition and appropriate referral. The oligosecretory nature of the underlying plasma cell dyscrasia initially complicated diagnosis, as conventional serum protein studies were unrevealing. This emphasizes the importance of comprehensive light chain analysis in suspected cases, as oligosecretory variants can account for up to 15% of cases. The "sparkling" myocardium appearance on echocardiography, while not pathognomonic, represents a classic finding in cardiac amyloidosis resulting from increased acoustic reflectance of amyloid-infiltrated myocardium. Combined with restrictive physiology and biatrial enlargement, these findings strongly suggest amyloid cardiomyopathy. The multisystem involvement demonstrated by imaging studies indicates advanced disease requiring prompt treatment initiation. Cardiac amyloidosis carries poor prognosis without treatment, with median survival often less than one year in symptomatic patients.

https://doi.org/10.1016/j.htct.2025.106167

PP 34

High-Risk IgA-κ Myeloma with Sacral Mass in a 31-Year-Old: Deep Response to Daratumumab −Lenalidomide−Dexamethasone plus Local RT without ASCT

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Introduction: Multiple myeloma (MM) in young adults is uncommon, and high-risk cytogenetics complicate standard pathways. We report a 31-year-old woman with IgA-κ MM, large sacral involvement, and adverse genetics, achieving a deep remission with daratumumab−lenalidomide−dexamethasone (DRd) plus focal radiotherapy (RT), electing to defer autologous transplant. **Methods:** Single-patient case review from prospectively maintained records. Data included presenting features, MRI/PET-CT, serum/urine monoclonal studies, bone-marrow histology/flow, and plasma-cell FISH. Treatment, response, and tolerability were documented. **Results:** A previously healthy 31-year-old presented with severe nocturnal lumbosacral pain and right-sciatic radiation. MRI revealed a left-lateral sacral mass (77 × 56 mm) with contrast enhancement; PET-CT demonstrated focal hypermetabolic lytic lesions in sacrum, L1, pubis, and scapula (SUVmax 5.4−5.9), with no visceral/extramedullary organ disease. Serum studies showed an IgA-κ M-component with elevated free light-chain ratio; β2-microglobulin was 4.2 mg/L (ISS stage II). Bone-marrow biopsy displayed intertrabecular plasma-cell infiltration; immunophenotype CD38+, CD56+, κ-restricted, CD19−; reticulin 0−1/4; amyloid negative. Plasma-cell FISH identified t(14;20)(IGH−MAFB) in ~35% of cells, indicating high-risk disease. She commenced DRd and received concurrent local RT to the sacrum (fractionated) for rapid pain control. Treatment was well tolerated, without renal or calcium derangements. Clinically, pain resolved; biochemically, the M-component cleared; radiologically, bone foci regressed with disappearance of pathologic uptake on interval imaging. Bone-marrow reassessment showed marked reduction of clonal plasma cells, consistent with deep response. Given age, recovery, and patient preference, autologous transplant was performed; she continued maintenance (daratumumab ± lenalidomide) with sustained remission on follow-up. **Discussion:** This case underscores four practice points. (1) Aggressive osseous disease at young age can herald high-risk biology; early, integrated MRI/PET staging captures true burden and guides focal RT for symptom control while systemic therapy acts on disseminated marrow disease. (2) Immunophenotype and marrow context (CD38 +/CD56+, κ-restriction; low reticulin) affirmed clonal

plasmacytosis consistent with MM rather than solitary plasmacytoma or IgG4-related processes. (3) Cytogenetic risk—notably t(14;20)—supports intensified monoclonal-antibody –based induction (DRd) and vigilant surveillance, as this lesion associates with inferior outcomes on IMiD/PI-only backbones. (4) In select young patients achieving deep remission, deferring ASCT after robust daratumumab-based induction and consolidative RT can be reasonable when aligned with patient values and close monitoring—especially if toxicity, fertility considerations, or personal preference weigh heavily. **Conclusion:** Young-onset, high-risk IgA-κ MM with a large sacral mass achieved a durable, deep remission on DRd plus focal RT, permitting ASCT deferral with maintenance therapy and sustained disease control. Pairing comprehensive imaging with cytogenetic risk and early antibody-based induction may optimize outcomes in comparable high-risk, bone-predominant presentations.

https://doi.org/10.1016/j.htct.2025.106168

PP 35

FAMILIAL MULTIPLE MYELOMA: SIBLING CASES WITH DISTINCT CLINICAL MANIFESTATIONS

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Introduction: Multiple myeloma (MM) is a malignant plasma cell disorder that typically occurs sporadically. Familial clustering is rare, with only a limited number of cases reported worldwide. Such familial presentations suggest a possible hereditary predisposition or shared environmental risk factors contributing to disease development [1,2]. Here, we present two siblings with distinct plasma cell neoplasms: one with recurrent extramedullary plasmacytoma and the other with multiple myeloma. **Case Presentation:** The first case was a 69-year-old woman who underwent surgery in 2017 for a proximal femoral mass, diagnosed as plasmacytoma. In 2024, she presented with a cervical swelling; excisional biopsy of a right level-5 lymph node again revealed plasmacytoma. Bone marrow biopsies performed at that time did not show features of multiple myeloma. Her brother, one year older, was diagnosed with multiple myeloma in June 2025. PET-CT revealed lytic lesions in the axial skeleton, and systemic therapy was initiated. **Discussion:** Familial occurrence of plasma cell neoplasms is exceedingly uncommon. Reported cases often involve either multiple relatives with MM or, less frequently, different manifestations of plasma cell disorders within the same family [3,4]. The present siblings illustrate divergent clinical phenotypes: persistent extramedullary plasmacytoma without myeloma progression in the sister, versus classical MM with lytic bone disease in the brother. This highlights the potential role of shared genetic background with variable penetrance and expression. Genetic susceptibility loci, immune dysregulation, and epigenetic mechanisms have all been proposed as contributors to familial myeloma [5]. Recognizing such familial patterns may have implications for surveillance strategies in high-risk relatives. **Conclusion:** We report a rare familial clustering of plasma cell neoplasms in siblings, underlining the importance of considering hereditary predisposition in plasma cell disorders. Further genetic and epidemiological studies are warranted to elucidate the underlying mechanisms.

https://doi.org/10.1016/j.htct.2025.106169

PP 36

ACQUIRED PYRUVATE KINASE DEFICIENCY FOLLOWED BY MYELODYSPLASTIC SYNDROME: A CASE REPORT

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Introduction: Pyruvate kinase (PK) deficiency is an autosomal recessive red blood cell (RBC) enzymopathy leading to chronic hemolysis. It is the second most common RBC enzymopathy and the most frequent cause of chronic hemolytic anemia due to an enzyme defect. PK enzymes consist of various isoforms encoded by PKLR and PKM genes, which catalyze the conversion of phosphoenolpyruvate (PEP) to pyruvate and ATP in the final step of glycolysis. Clinically significant PK deficiency is associated with PKLR mutations. Acquired PK deficiency is extremely rare, and its molecular basis remains unclear. Some cases have been associated with AML. Here we present a rare case of acquired PK deficiency followed by myelodysplastic syndrome (MDS). **Case Presentation:** A 70-year-old male presented with fatigue, weakness, and jaundice. Laboratory findings were as follows: WBC: 7.0×10^9/L, Hemoglobin: 7.9 g/dL, MCV: 101 fL, Platelets: 601×10^9/L, Total bilirubin: 1.6 mg/dL (indirect: 1.0 mg/dL), LDH: 280 U/L. Other biochemical parameters were within normal limits. Hemoglobin electrophoresis was normal. Direct and indirect Coombs tests were negative. Haptoglobin was 14 mg/dL (low). Erythrocyte PK activity was reduced at 3.16 U/g Hb (reference: 4.4–5.9). G6PD activity and osmotic fragility were normal. The patient had no prior anemia history. Genetic analysis for PKLR mutations was negative, supporting an acquired form. During follow-up, bilirubin increased to 8.6 mg/dL, LDH rose to 800 U/L, and hemoglobin decreased to 6.0 g/dL. The patient was taking gliclazide for diabetes mellitus, which was discontinued due to suspicion of hemolysis induction. Bilirubin subsequently decreased. Bone marrow biopsy showed dysplastic erythroid changes without blast increase, consistent with MDS. The patient initially required two RBC transfusions weekly, but after gliclazide withdrawal, the requirement decreased to one unit every two weeks. Genetic testing for MDS is ongoing. **Discussion & Conclusion:** Acquired PK deficiency is extremely rare. In this case, a 70-year-old patient developed PK deficiency followed by a diagnosis of MDS. While congenital hemolytic anemias usually present in younger patients, clinicians should be aware that acquired cases may appear later in life. Careful evaluation of medications and bone marrow disorders is essential in elderly patients with unexplained hemolysis.

https://doi.org/10.1016/j.htct.2025.106170

PP 37

A CASE OF THALASSEMIA DIAGNOSED WITH AUTOIMMUNE HEMOLYTIC ANEMIA

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A 37-year-old female patient with a diagnosis of thalassemia major was admitted to the emergency department with complaints of fatigue, nausea, vomiting, and abdominal pain. Laboratory tests revealed elevated liver enzymes and pancytopenia, prompting her hospitalization. It was noted that the patient had not received chelation therapy for the past three months and had a history of irregular use of chelating agents. Her laboratory values were as follows: WBC: 2,460/mm^3, Neutrophils: 700/mm^3, Hemoglobin: 5.2 g/dL, MCV: 62.8 fL, Platelets: 15,000/mm^3. Due to her symptomatic presentation, the patient received cross-matched erythrocyte and platelet suspensions for transfusion. CRP was 0.8 mg/dL; coagulation and renal function tests were within normal limits. The patient had indirect hyperbilirubinemia, LDH: 984 U/L, vitamin B12: 467 pg/mL, folate: 9.53 pg/mL, and ferritin: 956 ng/mL. Both direct and indirect Coombs tests were initially negative. Tests for hepatitis markers, EBV, TORCH, and HIV were also negative. Parvovirus evaluation could not be performed. Peripheral blood smear revealed schistocytes, fragmented erythrocytes, and target cells, thrombocytopenia but no atypical cells. The patient underwent abdominal ultrasonography, which showed hepatosplenomegaly, with the spleen measuring 19 cm. Chest X-ray revealed pleural effusion, and thoracic and abdominal CT scans were planned. Thoracic CT revealed mass-like lesions in the vertebral area with unclear distinction, areas of pneumonic consolidation, and pleural effusion. Intravenous cephalosporin therapy was initiated for presumed pneumonia. ANA and anti-dsDNA tests were sent and returned negative. A PET-CT scan was planned. As the patient's cytopenias persisted despite ongoing transfusion needs, a bone marrow biopsy was performed. Bone marrow aspiration revealed increased cellularity and erythropoiesis without any abnormal findings. PET-CT demonstrated vertebral involvement attributed to extramedullary hematopoiesis; no malignant uptake was detected. Methylprednisolone was initiated at 1 mg/kg. Although platelet levels increased, anemia persisted. Repeated Coombs tests later returned strongly positive (+3) for both direct and indirect Coombs. Direct Coombs was positive for both IgG and C3. The patient had an elevated LDH (1200 U/L) and decreased haptoglobin levels. Due to steroid-refractory autoimmune hemolytic anemia, Rituximab 375 mg/week was administered for four doses, and the steroid dosage was tapered off. After two months, lab results showed WBC: 5,150/mm^3, Hemoglobin: 9.2 g/dL, Platelets: 168,000/mm^3. With a now negative direct Coombs test and a post-transfusion ferritin level of 2,322 ng/mL, chelation therapy was reinitiated. The patient, diagnosed with infection-related autoimmune hemolytic anemia, continues to receive monthly transfusions of cross-matched erythrocyte suspensions, Türkiye. In this patient with thalassemia major who developed infection-associated autoimmune hemolytic anemia, rituximab was initiated due to steroid resistance and a favorable response was achieved. Conclusion: This patient, who developed infection-associated autoimmune hemolytic anemia and was reinitiated chelation therapy, continues to receive monthly transfusions of cross-matched erythrocyte suspensions

https://doi.org/10.1016/j.htct.2025.106171

PP 38

COLD AGGLUTININ DISEASE ASSOCIATED WITH COVID-19 INFECTION IN A PEDIATRIC PATIENT: A RARE CASE PRESENTING WITH SEVERE HEMOLYTIC ANEMIA AND LOBAR PNEUMONIA

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Objective: Cold agglutinin disease (CAD) is a form of autoimmune hemolytic anemia caused by antibodies—typically immunoglobulin M (IgM), and less frequently IgA or IgG—that target antigens on the surface of erythrocytes. Although the etiology may involve infections or immunologic disorders, most cases are idiopathic. The clinical picture results from hemolysis triggered by antibodies that become active at cold temperatures, leading to degenerative changes in the erythrocyte membrane and autoagglutination. This causes a drop in erythrocyte count and hematocrit, while MCV, MCH, and MCHC values appear markedly elevated. Peripheral blood smears often reveal erythrocyte agglutination. Here in, we present a case of cold agglutinin disease secondary to COVID-19 infection. **Case Presentation:** A 14-year-old previously healthy girl was initially treated with amoxicillin-clavulanate for upper respiratory tract infection symptoms, including fever and cough. Her symptoms worsened, and she tested positive for COVID-19 at an outside hospital. She was diagnosed with lobar pneumonia, and significant anemia noted during follow-up prompted her referral to our institution, Türkiye. Upon admission to our pediatric intensive care unit, three consecutive hemogram samples were clotted and could not be analyzed. Venous blood gas revealed hemoglobin (Hb) of 4.2 g/dL. Biochemical analyses showed LDH: 724 U/L (range 110-295 U/L), total bilirubin: 1.85 mg/dL (range 0.3-1.2 mg/dL), direct bilirubin: 0.29 mg/dL (range 0-0.2 mg/dL), and haptoglobin: 0.38 g/L (range 0.35-2.5 g/L). Direct Coombs test was negative. Peripheral smear demonstrated erythrocyte agglutination clusters. Blood samples were delivered to the laboratory in warm water immediately after collection to prevent in vitro agglutination. Repeat tests showed Hb: 8.2 g/dL, MCV: 100 fL, and a markedly elevated MCHC of 683 g/dL. Quantitative cold agglutinin testing could not be performed due to technical limitations at our center. In addition to pneumonia treatment, the patient was started on methylprednisolone at 2 mg/kg/day for presumed cold agglutinin disease. She

was discharged on day 10 of treatment and her steroid therapy was tapered and discontinued by day 21. At follow-up on day 21, the patient's hemoglobin had increased to 13.9 g/dL, and no erythrocyte agglutination was observed on peripheral smear. **Conclusion:** This case highlights a rare pediatric presentation of cold agglutinin disease associated with COVID-19 infection, complicated by severe hemolysis and lobar pneumonia. Early recognition and a multidisciplinary approach including corticosteroids and supportive care played a critical role in the patient's favorable outcome.

https://doi.org/10.1016/j.htct.2025.106172

PP 39

CASE REPORT: WIDESPREAD BONE INVOLVEMENT AFTER ALLOGENEIC TRANSPLANTATION IN A PATIENT WITH BIPHENOTYPIC ACUTE LEUKEMIA

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Objective: Biphenotypic acute leukemia (BAL) is a rare hematologic malignancy characterized by blasts expressing both myeloid and lymphoid markers, and is generally associated with poor prognosis. The advancement of cytochemical and immunophenotypic diagnostic techniques has improved recognition of such rare leukemias, which account for approximately 5% of adult acute leukemias. Despite recent developments, challenges remain in the diagnosis and treatment of BAL. The European Group for the Immunological Characterization of Leukemias (EGIL) and the World Health Organization (WHO) scoring systems, primarily based on flow cytometry, are widely used for diagnosis. Due to disease heterogeneity, there is no standardized chemotherapy for BAL; however, because of the high relapse risk, allo-HSCT is recommended as soon as remission is achieved. Following allo-HSCT, extramedullary relapse occurs in 3–12% of acute leukemia patients. In this study, we present a case of BAL with isolated widespread bone involvement occurring after allo-HSCT. **Case report:** A 33-year-old male patient was diagnosed with B/Myeloid biphenotypic acute leukemia in January 2024. Flow cytometric evaluation showed aberrant myeloid markers, while cytogenetic analysis did not reveal FLT3-ITD, t(8;21), t(9;22), or inv(16) mutations. He received induction therapy with 3+7 Idarubicin & Cytarabine, which failed to achieve remission. FLAG-Mito reinduction therapy was administered, but bone marrow evaluation still showed 8% blasts, and the patient was considered refractory. On March 24, 2024, he underwent allo-HSCT from an HLA-matched sibling donor after a myeloablative conditioning regimen with Fludarabine and Treosulfan. Post-transplant chimerism was 96%, and remission was achieved. In December 2024, the patient presented with left knee pain. Imaging revealed a bone lesion in the proximal left tibia, and biopsy confirmed BAL relapse. Bone marrow biopsy was normal. PET-CT revealed widespread skeletal involvement, including bilateral humeri, right clavicle, right scapula, sternum, L2 vertebra, left sixth rib, sacrum, pelvic bones, right femur, and proximal bilateral tibiae. Due to severe pain, palliative radiotherapy (2000 cGy to the left tibia and 800 cGy to the left sixth rib) was administered. As there was no bone marrow involvement, the patient was started on Decitabine (20 mg/m^2/day for 5 days) combined with Venetoclax (200 mg for 14 days per cycle, reduced due to concomitant posaconazole use). After four cycles, PET-CT demonstrated complete remission. Donor lymphocyte infusions (DLI) were administered in four doses (2.42×10^7/kg total). The patient remains in remission with mild chronic GVHD (grade 1–2). **Discussion:** Biphenotypic acute leukemia is a rare subtype of acute leukemia, most commonly presenting with a B/Myeloid phenotype. High-dose chemotherapy protocols derived from ALL or AML regimens are generally used, and allo-HSCT is recommended for patients achieving remission. Extramedullary relapse after allo-HSCT has been reported with variable incidence, most often accompanied by bone marrow relapse. Isolated extramedullary relapse without marrow involvement is rare. A European multicenter study reported isolated extramedullary relapse in 0.65% of cases after allo-HSCT, while another study of 287 patients identified such relapse in approximately 4%, most frequently in the CNS, skin, bone, pelvis, and breast. In our case, the patient relapsed nine months after allo-HSCT with widespread isolated bone involvement. Treatment with hypomethylating agent Decitabine combined with Venetoclax achieved remission, and subsequent DLI helped maintain disease control. There is limited literature regarding isolated bone relapse in BAL after allo-HSCT, highlighting the uniqueness of this case. **Conclusion:** Biphenotypic acute leukemia is a rare disease with poor prognosis and no standardized therapy. Treatment approaches usually involve high-dose chemotherapy regimens for ALL or AML followed by allo-HSCT. Although extramedullary relapse after allo-HSCT is known, isolated widespread bone involvement is extremely rare. Our case demonstrates successful treatment with Decitabine and Venetoclax, followed by donor lymphocyte infusions.

https://doi.org/10.1016/j.htct.2025.106173

PP 40

"Kappa Light-Chain Multiple Myeloma Without Serum M-Spike: A Diagnostic and Therapeutic Challenge in an Elderly Patient"

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Introduction: Light-chain multiple myeloma (LCMM) accounts for a subset of myeloma cases characterized by the absence of an M-protein spike on serum protein electrophoresis. This diagnostic challenge often delays recognition and treatment. We present the case of a 75-year-old woman with kappa-dominant LCMM, where conventional marrow and serum

studies were inconclusive, yet clinical and imaging findings confirmed active disease. **Methods:** A comprehensive diagnostic evaluation was performed, including hematology and biochemistry profiles, serum protein electrophoresis, serum and urine immunofixation, serum free light chain (sFLC) quantification, bone marrow aspiration and biopsy with immunohistochemistry, and 18F-FDG PET-CT imaging. **Results:** Gülüşen Kellesibüyük, a 75-year-old female, presented with fatigue, anemia, and back pain. Laboratory evaluation revealed hemoglobin of 9.7 g/dL, elevated inflammatory markers, and preserved renal and calcium levels. Serum protein electrophoresis demonstrated no monoclonal spike. Immunofixation of urine identified monoclonal kappa light chains. sFLC testing showed markedly increased kappa levels (121–270 mg/L) with a pathological κ/λ ratio between 3.9 and 4.2. Bone marrow aspirates revealed only 2–3% plasma cells with polytypic staining, and biopsies were normocellular without evidence of clonal infiltration. Despite these inconclusive marrow results, PET-CT demonstrated a metabolically active lytic lesion in the L4 vertebra (SUVmax 11.4) and multiple punctate cranial lytic lesions. The combination of anemia, abnormal light chain ratio, and PET-CT–confirmed bone lesions established the diagnosis of active LCMM. **Discussion:** This case emphasizes the diagnostic complexity of LCMM, where reliance solely on serum electrophoresis or marrow histology may be misleading. The absence of an M spike, coupled with non-diagnostic marrow sampling, initially obscured the diagnosis. However, integration of sFLC analysis, urine immunofixation, and advanced imaging confirmed the presence of active myeloma. Elderly, transplant-ineligible patients such as this one benefit from modern therapeutic approaches that combine efficacy with tolerability. Triplet regimens including daratumumab with lenalidomide and dexamethasone or reduced-intensity bortezomib-based protocols are recommended as first-line options. For patients with limited access to hospital care, oral regimens may be considered, though efficacy is comparatively lower, Türkiye. **Conclusion:** The case of demonstrates that light-chain multiple myeloma can be present despite normal serum electrophoresis and non-clonal marrow findings. Comprehensive evaluation with free light chain assays, urine studies, and PET-CT is essential to avoid underdiagnosis. This case highlights the importance of applying full diagnostic criteria to detect atypical myeloma presentations early, ensuring timely initiation of therapy and improved patient outcomes.

https://doi.org/10.1016/j.htct.2025.106174

PP 41

Early-Stage Nodular Lymphocyte-Predominant Hodgkin Lymphoma (NLPHL) in a Young Woman: A Rare Subtype Managed Without Chemotherapy

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Introduction: Nodular lymphocyte-predominant Hodgkin lymphoma (NLPHL) is a rare subtype of Hodgkin lymphoma, comprising approximately 5–7% of cases. Unlike classical HL, NLPHL is characterized by CD20-positive "popcorn" cells (LP cells), lacks Epstein-Barr virus association, and tends to follow an indolent course. Accurate diagnosis is critical, as the therapeutic approach differs substantially. We report an early-stage NLPHL case in a young woman managed successfully without chemotherapy, emphasizing the value of histopathological precision and risk-adapted therapy. **Methods:** A 33-year-old woman presented with a painless cervical swelling. Physical examination revealed enlarged left cervical and supraclavicular lymph nodes. She had no B symptoms such as fever, night sweats, or weight loss. Blood counts and biochemistry were within normal limits. An excisional biopsy of a lymph node was performed, followed by immunohistochemistry and whole-body 18F-FDG PET-CT for staging. Bone marrow aspiration and biopsy were also conducted to rule out marrow involvement. **Results:** Histopathological examination demonstrated nodular architecture containing scattered lymphocyte-predominant (LP) cells. Immunophenotyping revealed strong CD20 and Pax5 expression, with negativity for CD3 and CD15. CD21 staining highlighted an expanded follicular dendritic cell meshwork, confirming the diagnosis of NLPHL. PET-CT showed FDG-avid lymph nodes localized to the left cervical and supraclavicular regions, with a maximum SUV of 27.9. No pathological uptake was seen in the mediastinum, abdomen, bones, or spleen. Bone marrow biopsy was normocellular without evidence of infiltration. The disease was staged as Stage IA (non-bulky), CD20-positive NLPHL. The patient was treated with rituximab monotherapy (375 mg/m^2 weekly for 4 doses), followed by involved-field radiotherapy (30 Gy) to the involved nodal regions. Given her age and reproductive status, fertility preservation was discussed before initiating treatment. The plan aimed to minimize long-term toxicity while maintaining curative potential. **Discussion:** This case illustrates several important themes. First, accurate histological subtyping allowed for a deviation from standard chemotherapy-based HL protocols. Second, the use of rituximab and radiotherapy alone is an emerging and evidence-supported strategy for early-stage NLPHL, particularly in CD20-positive, non-bulky cases. Third, the patient's demographic—young and female—makes chemotherapy-free management especially attractive given concerns about fertility and late effects. Finally, the case has strong educational value, highlighting the need to distinguish NLPHL from classical HL and indolent B-cell lymphomas, both histologically and metabolically. **Conclusion:** This case demonstrates how a rare Hodgkin lymphoma subtype can be successfully managed with a chemotherapy-free, targeted approach. It reinforces the importance of accurate subtyping and risk-adapted treatment in delivering personalized care, especially in young patients where fertility and quality of life are key considerations

https://doi.org/10.1016/j.htct.2025.106175

PP 42

GLYCEMIC CONTROL, RENAL FUNCTION, AND HEMATOLOGICAL PARAMETERS: A RETROSPECTIVE REAL-WORLD ANALYSIS

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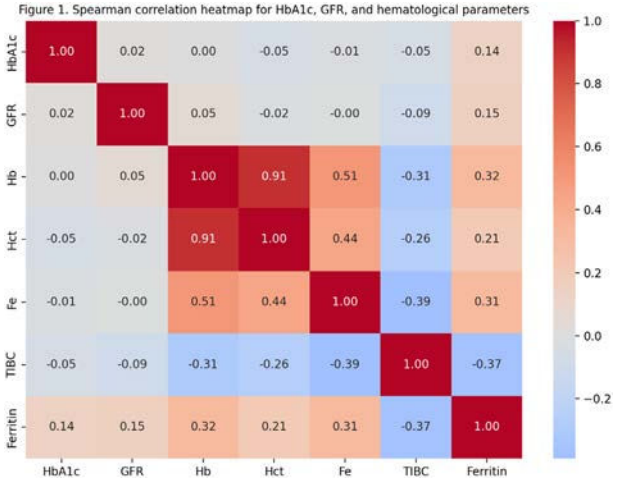
Introduction: Diabetes mellitus exerts detrimental effects that extend beyond glycemic imbalance, frequently involving hematological and renal systems. The coexistence of anemia and declining renal function in diabetes substantially increases cardiovascular risk [1]. HbA1c, as a marker of long-term glycemic control, may have significant implications for hematological indices and iron metabolism. Recent evidence suggests that alterations in iron handling and ferritin levels may be linked to both glycemic status and renal function [2,3]. The present retrospective study investigated the associations between HbA1c, estimated glomerular filtration rate (GFR), and hematological parameters in a real-world patient cohort. **Methods:** Data from 205 adult patients were retrospectively retrieved from the Internal Medicine Clinic of Düziçi State Hospital. The collected variables included hemoglobin (Hb), hematocrit (Hct), serum iron (Fe), total iron-binding capacity (TIBC), ferritin, HbA1c, and estimated GFR. Patients were categorized into two groups according to glycemic status: HbA1c <7% and HbA1c ≥7%. Spearman's correlation analysis was applied to determine associations among variables. Group comparisons were performed between the two HbA1c subgroups using appropriate statistical tests depending on data distribution. Ethical approval was obtained from the Adana City Training and Research Hospital Scientific Research Ethics Committee (Decision No: 508, Date: 08.05.2025). **Results:** No significant association was observed between HbA1c and hemoglobin or hematocrit. A borderline positive correlation was identified between HbA1c and ferritin ($r=0.14$, $p\approx0.05$). GFR demonstrated a weak but significant correlation with ferritin ($r=0.15$, $p<0.05$). Hemoglobin and hematocrit showed strong positive associations with serum iron, whereas TIBC was inversely correlated with ferritin. When comparing patients by glycemic status, those with HbA1c ≥7% exhibited slightly lower hemoglobin, hematocrit, and GFR values, alongside modestly higher ferritin levels compared with patients with HbA1c <7%. These findings are summarized in Table 1. A correlation heatmap integrating all variables is presented in Figure 1, where strong positive associations are observed between Hb, Hct, and serum iron, while TIBC and ferritin demonstrate an inverse relationship. Borderline positive associations of ferritin with both HbA1c and GFR are also highlighted. **Discussion and Conclusion:** This retrospective analysis of 205 patients demonstrated that glycemic control, as assessed by HbA1c, does not directly predict hemoglobin or hematocrit levels. However, a borderline positive correlation with ferritin was observed, suggesting a potential link between glycemic status and iron metabolism. GFR also exhibited a weak positive correlation with ferritin, consistent with previous reports that renal dysfunction alters iron homeostasis and contributes to anemia in diabetes [4,5]. Expected associations among hematological indices were confirmed, such as strong correlations of hemoglobin and hematocrit with serum iron, and the inverse relationship between TIBC and ferritin. These findings reinforce the integrated role of iron regulation in the pathophysiology of diabetes. Overall, the results suggest that while HbA1c may not serve as a strong predictor of anemia itself, it may indirectly influence iron metabolism, potentially through inflammatory or renal mechanisms. Clinical management of diabetes should therefore extend beyond strict glycemic control, incorporating comprehensive evaluation of renal function and iron status.

Keywords: Diabetes mellitus, HbA1c, Anemia, Ferritin, Iron metabolism, Glomerular filtration rate, Hematological parameters.

Table 1. Comparison of hematologic and renal indices between glycemic control groups (HbA1c <7 vs ≥7).

Parameter	HbA1c <7 (mean ± SD, median)	HbA1c ≥7 (mean ± SD, median)
Hemoglobin (g/dL)	14.4 ± 1.4 (14.7)	14.2 ± 1.5 (14.0)
Hematocrit (%)	43.5 ± 3.7 (43.0)	42.6 ± 4.3 (42.1)
Serum Iron (μg/dL)	79.8 ± 37.6 (71.0)	77.5 ± 29.1 (74.0)
TIBC (μg/dL)	286 ± 74 (297.0)	279 ± 51 (280.5)
Ferritin (ng/mL)	59.0 ± 56.8 (42.6)	66.1 ± 51.0 (46.0)
GFR (mL/min/1.73m^2)	99.5 ± 13.0 (101.0)	97.2 ± 13.6 (99.0)

Figure 1. Spearman correlation heatmap for HbA1c, GFR, and hematological parameters (Hb, Hct, Fe, TIBC, Ferritin).



https://doi.org/10.1016/j.htct.2025.106176