

## Poster Abstracts

### Adult Hematology Abstract Categories

#### Acute Leukemias

PP 01

#### ACUTE MYELOID LEUKEMIA PRESENTING AS ACUTE PANCREATITIS WITH MULTISYSTEM LEUKEMIC INFILTRATION: A CASE REPORT OF PANCREATIC, BILIARY TRACT AND PULMONARY INVOLVEMENT

Yusuf Hekimoğlu<sup>1,\*</sup>, Ethem Ozkaya<sup>1</sup>,  
Vehbi Demircan<sup>1</sup>, Abdullah Karakuş<sup>1</sup>,  
Orhan Ayyıldız<sup>1</sup>

<sup>1</sup>Dicle University

**Objective:** Acute myeloid leukemia (AML) classically presents with symptoms related to anemia, infections, or bleeding. However, atypical presentations involving abdominal pain, acute pancreatitis, and biliary ducts infiltration are rare but have been documented. These unusual manifestations can complicate the diagnosis and delay recognition of AML. Here, we present a case of a young female patient diagnosed with AML, who initially presented with acute pancreatitis and subsequent findings suggestive of biliary tract infiltration and possible pulmonary involvement. **Case Report:** A 22-year-old female with no notable medical history presented to a local healthcare facility with abdominal pain and diarrhea. She was diagnosed with acute pancreatitis based on clinical evaluation. Abdominal ultrasonography revealed a mass at the head of the pancreas, along with dilatation of both intrahepatic and extrahepatic bile ducts. The patient was referred to Dicle University Educational Hospital for further investigations, including endoscopic retrograde cholangiopancreatography (ERCP). Upon admission to the general internal medicine clinic, the pancreatic mass and biliary duct dilation were confirmed, and further laboratory investigations showed an elevated white blood cell count as 24.900/mm<sup>3</sup>. A peripheral blood smear demonstrated abnormal white cells, raising suspicion of a hematologic disorder. A bone marrow biopsy was subsequently performed, confirming the diagnosis of AML. Magnetic resonance cholangiopancreatography

(MRCP) was conducted to further assess the pancreatic and biliary tract lesions, revealing findings consistent with extramedullary hematologic infiltration. The patient was started on the 7+3 chemotherapy regimen (cytarabine 200 mg/m<sup>2</sup> for 7 days and an idarubicin 12 mg/m<sup>2</sup> for 3 days). Following treatment, her abdominal pain and distension improved, and laboratory abnormalities normalized, but She died because of neutropenic sepsis during 35th day of treatment. **Conclusion:** AML can exhibit extramedullary involvement of any organ, though pancreatic, biliary tract, and hepatic enzyme abnormalities are rare and occurs in approximately 8–10% of cases. A study from one center indicated that the most common sites of extramedullary AML involvement are the skin (65%), the central nervous system (23%), and the pleura (7%) . Multi-organ involvement has been reported in around 9% of cases, but pancreatic and biliary duct infiltration is extremely rare, accounting for only 1% of cases. In our case, the patient exhibited involvement of the pancreas, biliary tracts, spleen, and lungs, a situation that is exceedingly rare, with no similar cases found in the existing literature.

**Keywords:** Extramedullary, Leukemia, Myelogenous, Acute pancreatitis.

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PP 02

#### AN AML CASE PRESENTING WITH EXTRAMEDULLARY MYELOID SARCOMA

Songül Beskisiz Dönen<sup>1,\*</sup>, Vehbi Demircan<sup>1</sup>,  
Abdullah Karakuş<sup>1</sup>, Mehmet Orhan Ayyıldız<sup>1</sup>

<sup>1</sup>Dicle University Faculty of Medicine, Department of Hematology

**Objective:** This case highlights an atypical presentation of myeloid sarcoma in a patient with acute myeloid leukemia (AML), focusing on diagnostic challenges, treatment decisions, and outcomes. The case emphasizes extramedullary involvement and therapeutic approaches for patients with

poor performance status. **Case Report:** A 68-year-old woman presented with neck swelling. Ultrasound and CT imaging revealed multiple enlarged cervical lymph nodes, with the largest measuring 30 × 25 mm in the right submandibular region. A tru-cut biopsy confirmed myeloid sarcoma infiltration. Upon admission, she was not cytopenic, but peripheral blood smear revealed blasts. Bone marrow biopsy confirmed AML, and diffuse chloroma foci were noted on her face. Due to poor performance status, the 5+1 chemotherapy regimen (5 days cytarabine, 1 day anthracycline) was initiated. After achieving remission in bone marrow, HDAC (high-dose cytarabine, 1500 mg/day) was administered as consolidation therapy. Severe cytopenias during HDAC led to a switch to azacitidine (Vidaza, 75 mg/m<sup>2</sup>) and venetoclax. Allogeneic stem cell transplantation (AlloSCT) was recommended, but the patient declined. **Conclusion:** This case illustrates the diagnostic challenges of myeloid sarcoma in rare locations like the neck, compounded by diffuse chloroma. For patients with poor performance status, low-intensity regimens such as azacitidine and venetoclax are viable alternatives to intensive chemotherapy. AlloSCT remains the preferred treatment for high-risk AML, but in this case, azacitidine and venetoclax provided an alternative therapeutic pathway.



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PP 03

#### A CASE OF ACUTE LYMPHOBLASTIC LEUKEMIA PRESENTING WITH HYPEREOSINOPHILIA

Bengü Macit<sup>1,\*</sup>, Arzu Akyay<sup>1</sup>, Yurday Öncül<sup>1</sup>

<sup>1</sup>Inonu University Turgut Ozal Medical Center

**Case Report:** Hypereosinophilia (HE) is eosinophil count >500/μL. The association of HE with acute lymphoblastic leukemia (ALL) is extremely rare, with an incidence of less than 1%. HE may precede the common symptoms and signs of ALL by several months or weeks. In some cases, the symptoms may be due to eosinophilic organ or system infiltration, and these findings may be different from the classical ALL symptoms, thus delaying the diagnosis. Here, we report a male patient who presented with HE and was diagnosed as PreB-ALL. A 9-year-old boy patient was admitted to Inonu University Turgut Özal Medical Center with complaints of testicular pain and swelling. The patient's hemogram showed HE, but there was no leukocytosis or cytopenia. No atypical cell was observed

in peripheral smear. On scrotal ultrasonography (USG), the left epididymal head had a mildly heterogeneous appearance and the patient was treated for epididymitis with suspicion of epididymitis. Approximately one week later, the patient presented with fever. The patient's peripheral smear showed 36% blasts, 38% eosinophils, 2% monocytes, 6% lymphocytes, 10% segments and 8% bands. Bone marrow aspiration was performed for the diagnosis of acute leukemia and PreB-ALL was diagnosed. Control testicular USG was evaluated as testicular involvement of leukemia. During follow-up, the patient had nausea, vomiting, dizziness, decreased visual field, nuchal rigidity, and outward gaze limitation. Magnetic resonance (MR) venography revealed thrombosis in the inferior sagittal sinus and anticoagulant therapy was initiated. The patient with central nervous system symptoms was considered to have leukemic involvement and his treatment was adjusted. ALL is a condition that can cause HE. The prognosis is poor in ALL patients presenting with HE. HE may occur before the classical ALL symptoms therefore the diagnosis of ALL should also be considered in patients presenting with HE.

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PP 04

#### EVALUATION OF DRUG EFFECTIVENESS AND CONTROLLED RELEASE PROFILES OF CLAY MINERALS LOADED WITH ANTI-CARCINOGENIC AGENT AS A DRUG DELIVERY SYSTEM ON LEUKEMIA

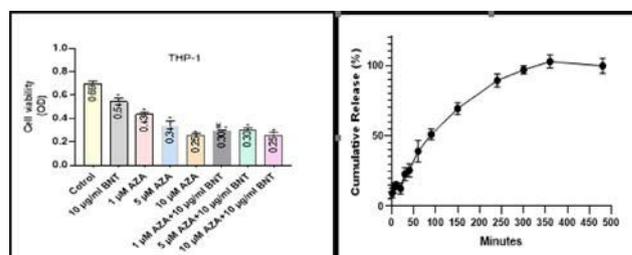
Mustafa Duran<sup>1,\*</sup>, Elif Kağa<sup>2</sup>

<sup>1</sup> Afyonkarahisar Health Science University, Faculty of Medicine, Department of Internal Medicine Hematology

<sup>2</sup> Afyonkarahisar Health Science University, Faculty of Medicine Pathology

**Objective:** This study aimed to evaluate the slow release and internalization of azacitidine-bentonite combination in THP-1 and K562 cell cultures in acute myeloid leukemia morphology. **Methodology:** The morphology of bentonite clay was assessed using two Scanning Electron Microscopes. The bentonite-azacitidine combination was assessed in THP-1 and K562 cell cultures via in vitro cell proliferation tests, proliferation with CCK-8, and drug release tests with dialysis membranes. Additionally, apoptosis and internalization were determined using the Annexin V-FITC Kit and fluorescence methods, respectively. **Results:** Our findings showed that azacitidine achieved complete and controlled release within 8 hours. Bentonite displayed significant antiproliferative effects at concentrations of 10, 25, 50, and 100  $\mu\text{g/ml}$  in both cell lines. The combination of azacitidine and bentonite exhibited a synergistic effect in inhibiting cell proliferation, with significant decreases in cell viability in the 1  $\mu\text{M}$  azacitidine + 10  $\mu\text{g/ml}$  bentonite, 5  $\mu\text{M}$  azacitidine + 10  $\mu\text{g/ml}$  bentonite, and 10  $\mu\text{M}$  azacitidine + 10  $\mu\text{g/ml}$  bentonite groups compared to the controls. The drug release profile of the

bentonite-azacitidine combination demonstrated slow release, with 50% released in the first two hours and approximately 90% released in the fourth hour, with prolonged release exceeding eight hours, potentially reducing side effects and increasing efficacy in target cells. **Conclusion:** In conclusion, bentonite NPs exhibited significant potential as drug carriers for azacitidine in the treatment of leukemia and offered benefits such as improved solubility, bioavailability, controlled release, protection from harsh environments, and cost-effectiveness.



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PP 05

#### LIVER INVOLVEMENT IN ACUTE MYELOID LEUKEMIA: A CASE REPORT

Nida Akgül<sup>1,\*</sup>, Ali Doğan<sup>1</sup>, Cihan Ural<sup>1</sup>, Ramazan İpek<sup>1</sup>

<sup>1</sup> Van Yuzuncu Yil University Faculty of Medicine, Department of Hematology

**Objective:** Although rare, extramedullary involvement can be observed in patients with acute myeloid leukemia (AML). These extramedullary involvements are also known as myeloid sarcoma, granulocytic sarcoma, or chloroma. The most common sites of involvement are soft tissues, bone, periosteum, and lymph nodes. Patients with extramedullary involvement may exhibit a more aggressive clinical course. In this case report, we evaluated an AML patient with liver involvement at the time of diagnosis. **Case Report:** A 66-year-old female patient presented to our hospital with complaints of fatigue, bruising on the skin, and yellowing of the eyes for about a month. Physical examination revealed icterus in the sclera, and widespread ecchymoses on the arms and abdomen. Laboratory findings showed a hemoglobin level of 7.8 g/dL, a leukocyte count of  $4.4 \times 10^9/\text{L}$ , a neutrophil count of  $1.1 \times 10^9/\text{L}$ , a platelet count of  $30 \times 10^9/\text{L}$ , CRP at 29 mg/L, and direct bilirubin at 5.8 mg/dL. Peripheral blood smear revealed notable myeloblasts and auer rods. Bone marrow aspiration smear showed over 20% myeloblasts, supporting the diagnosis of acute myeloid leukemia. Flow cytometry analysis was evaluated as consistent with AML. Abdominal ultrasonography revealed the liver was 19.5 cm and the spleen was 16 cm in size. The patient underwent 7+3 remission induction

chemotherapy. After chemotherapy, bilirubin levels returned to normal, and the patient was diagnosed with liver involvement of AML. **Conclusion:** The clinical presentation of extramedullary involvement varies depending on the affected organ and region. A definitive diagnosis is made through biopsy. In patients with AML, as in our case, a biopsy may not always be feasible due to the risk of bleeding. Therefore, in cases where hepatomegaly, abnormalities in liver function tests, and elevated bilirubin levels cannot be explained by other diseases, liver involvement should be considered.

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#### PP 06

### IS ALL-TRANS RETINOIC ACID EFFECTIVE IN PULMONARY HAEMORRHAGE IN PATIENTS WITH ACUTE PROMYELOBLASTIC LEUKAEMIA?

Süleyman Atay<sup>1,\*</sup>, Ganiye Begül Yağcı<sup>2</sup>

<sup>1</sup> Health Science University, Adana City Training and Research Hospital

<sup>2</sup> Adana City Training and Research Hospital

**Objective:** Acute myeloid leukaemia (AML) develops from myeloid precursor cells in the bone marrow. Acute promyelocytic leukaemia (APL) is an aggressive subtype (5-10%) of AML with the t(15;17) translocation. It is sensitive to all-trans retinoic acid (ATRA). The aim of this article is to emphasise the efficacy of ATRA treatment in pulmonary haemorrhage associated with APL and to contribute to the literature. **Case Report:** A 14-year-old girl presented with malaise, pallor and bruises since 1 month. On examination, she was pale and had bruises on the trunk and extremities, but no organomegaly. Investigations revealed pancytopenia and atypical mononuclear cells in peripheral blood smear. Bone marrow aspirate showed promyeloblasts (80%) and AML-M3 surface markers were positive in flow cytometry. Cytogenetic analysis revealed t(15;17) translocation. AML-BFM 2012 chemotherapy protocol was initiated. During induction chemotherapy, the patient developed dyspnoea and pulmonary haemorrhage. The child was transferred to intensive care unit and ATRA was added to the chemotherapy at a dose of 25 mg/m<sup>2</sup>/day. Coagulation tests improved 2 days after ATRA treatment and clinical findings improved 4 days later. On the 9th day of intensive care unit admission, the patient was transferred to inpatient ward and there was no bleeding during follow-up. **Discussion:** Haemorrhagic complications are frequent in APL patients and are one of the main causes of early death (5-9%) (1,2). Increased plasmin production (60-fold) due to excessive annexin-II receptor expression in promyeloblasts has been shown to cause fibrinolysis. It is thought that patients develop increased hyperfibrinolysis rather than consumption coagulopathy (2). ATRA is highly effective in bleeding control (3). **Conclusion:** Patients should be monitored with coagulation tests at regular intervals due to the high risk of bleeding. Undesirable haemorrhagic conditions may develop

before and during treatment. ATRA can provide effective control in treatment.

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#### Adult Hematology Abstract Categories

##### Chronic Leukemias

##### PP 07

### MULTI-TYROSINE KINASE INHIBITOR-ASSOCIATED APLASTIC ANEMIA AND A BRIEF LITERATURE REVIEW

Veysel Erol<sup>1,\*</sup>, Zeki Guzel<sup>1</sup>, Mustafa Gokoglu<sup>1</sup>

<sup>1</sup> Kahramanmaraş Necip Fazıl City Hospital

**Objective:** Chronic myeloid leukemia (CML) is a malignancy classified under the group of chronic myeloproliferative neoplasms. It is characterized by uncontrolled leukocytosis, bleeding, thrombosis, recurrent infections, and hepatosplenomegaly. With the introduction of imatinib in 2001, followed by the second- and third-generation tyrosine kinase inhibitors (TKIs), a new era in the treatment of CML began, as overall survival rates have since reached levels comparable to normal life expectancy. In this article, we review the first case of aplastic anemia that developed after bosutinib treatment, along with other cases of aplastic anemia reported in the literature following the use of TKIs. **Case Report:** A 57-year-old female was referred for leukocytosis identified during evaluation for fatigue, weakness, and early satiety. Initial lab results showed a WBC of  $384 \times 10^9/L$ , NEU of  $246 \times 10^9/L$ , Hb of 7.2 g/dL, and Plt of  $281 \times 10^9/L$ . Abdominal ultrasound revealed splenomegaly (23 cm), and peripheral blood smear suggested chronic myeloid leukemia (CML), leading to BCR-ABL transcript testing. Hydroxyurea was initiated while awaiting results. Two weeks later, the BCR-ABL transcript level was 49%, and imatinib 400 mg/day was started on December 15, 2022. The February 2023 earthquake disrupted the patient's imatinib use for three months. Upon return in May 2023, labs showed a WBC of  $17 \times 10^9/L$ , NEU of  $14 \times 10^9/L$ , Hb of 14.1 g/dL, Plt of  $424 \times 10^9/L$ , and BCR-ABL remained at 49%. Imatinib was resumed. In August 2023, BCR-ABL decreased to 41%. However, in October 2023, pancytopenia emerged, leading to imatinib discontinuation (WBC:  $2.98 \times 10^9/L$ , NEU:  $0.5 \times 10^9/L$ , Hb: 4.1 g/dL, Plt:  $2 \times 10^9/L$ ). ABL mutation analysis showed no resistance mutations (Hemogram values at diagnosis and after treatment are shown in Figure 1). After two weeks without medication and no improvement in pancytopenia, bone marrow biopsy confirmed aplastic anemia (Figure 2A). Following ten weeks of recovery, normocellular marrow was observed (Figure 2B), and dasatinib 50 mg/day was started on February 1, 2024, later increased to 100 mg/day. Due to worsening cytopenias in late February, dasatinib was reduced and eventually discontinued in March. Bosutinib 500 mg/day was initiated in May, with BCR-ABL at 27.9%. As cytopenias progressed, bosutinib was reduced to 300 mg/day. Despite a BCR-ABL decrease to 8.63% in August, cytopenias persisted, and bosutinib was further

reduced 100 mg/day. The patient is currently being evaluated for allogeneic stem cell transplantation. **Methodology:** Until the 2000s, chronic myeloid leukemia (CML) was a fatal malignancy within the group of chronic myeloproliferative neoplasms. However, a significant breakthrough occurred following the approval of imatinib mesylate, the first tyrosine kinase inhibitor (TKI) for CML treatment, by the FDA in 2001 and the EMA in 2003. This was followed by the development of second-generation (dasatinib, nilotinib, bosutinib) and third-generation (ponatinib) TKIs, which greatly improved disease outcomes and significantly reduced TKI resistance. Common side effects of TKIs include pleural/pericardial effusion, pretibial edema, hyperglycemia, hyperlipidemia, liver dysfunction, diarrhea, and thrombosis, most of which can be managed by temporarily discontinuing the drug or reducing the dosage. Transient myelosuppression is also a frequently observed side effect of TKI therapy. However, prolonged aplastic anemia (AA) is a rare adverse effect secondary to TKIs. To date, cases of bone marrow aplasia associated with imatinib, dasatinib, and nilotinib have been reported in the literature, and the approaches to managing these cases are summarized in Table 1. Common management strategies include discontinuation of the drug, observation without medication, switching to another TKI, or performing allogeneic stem cell transplantation. Additional treatments such as cyclosporine, antithymocyte globulin (ATG), and filgrastim have also been employed. Unfortunately, despite various interventions, some patients have succumbed to septic mortality associated with prolonged neutropenia and intracranial hemorrhage linked to thrombocytopenia. The small number of cases makes it challenging to establish a standardized treatment approach. The mechanism of TKI-induced aplastic anemia (AA) remains unclear, but four potential pathophysiologies are considered: 1) acellularity in the hematopoietic system due to bone marrow infiltration by the CML clone [19], 2) blastic evolution during treatment [13], 3) suppression of hematopoietic stem cell proliferation through inhibition of kinases like c-kit, PDGFR, and SRY [8], and 4) toxic drug levels due to genetic polymorphisms in drug metabolism [20] **Results:** Studies have shown that higher doses of TKIs are associated with increased rates of myelosuppression [21]. Since routine monitoring of drug levels is not available in many healthcare facilities, using lower-than-standard TKI doses may be a viable alternative in cases of prolonged, severe cytopenias. In our patient, the progressive decline in BCR-ABL transcript levels suggests that the cause of AA is more likely due to nonspecific suppression of hematopoietic stem cells by TKIs rather than blastic evolution. Due to the unavailability of drug level testing at our center, we could not rule out bone marrow suppression related to drug toxicity. However, our patient tolerated bosutinib better, with the BCR-ABL transcript level dropping below 10% for the first time, distinguishing bosutinib from other TKIs. The milder cytopenic

profile observed may be related to bosutinib's weaker inhibition of PDGFR and c-kit [22]. **Conclusion:** In cases of aplastic anemia following TKI therapy, various case-based treatment approaches exist, but no standardized method has been widely accepted. During the TKI era, allogeneic stem cell transplantation remains a necessary option for CML patients with AA. Asciminib, with its distinct mechanism of action, could be considered a treatment option in such cases, though no data currently exist in the literature. While the patient's BCR-ABL transcript level after bosutinib 100 mg/day is eagerly awaited, lower-dose bosutinib may present a viable alternative for this patient group.

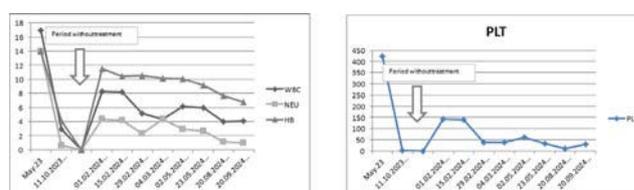


Figure 1: Hemogram values at the time of diagnosis and after treatment: WBC:  $\times 10^9/L$ ; NEU: %; HB: g/dL; PLT:  $\times 10^9/L$ .

Figure 1

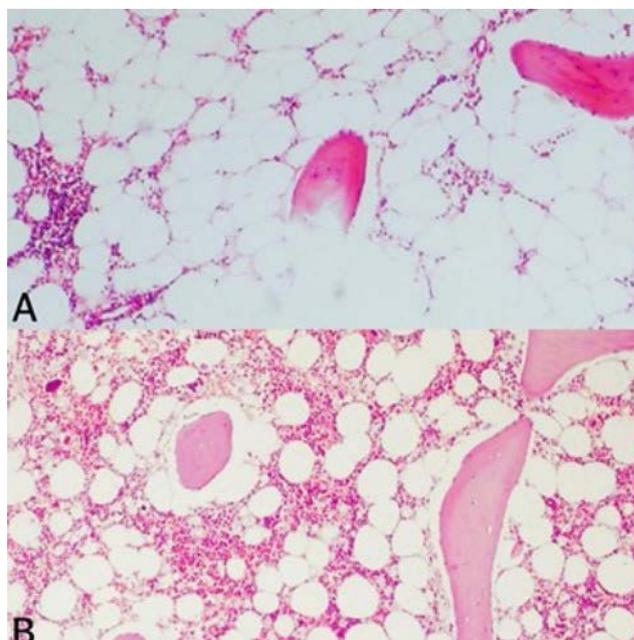


Figure 2: A) Bone marrow biopsy demonstrating loss of cellularity following imatinib-associated pancytopenia. B) Bone marrow biopsy showing increased cellularity performed 10 weeks after discontinuation of imatinib.

Table 1: Cases of aplastic anemia secondary to tyrosine kinase inhibitors reported in the literature to date

	Age/sex	Hematological parameters at starting treatment with TKI or diagnosis	BCR-ABL FISH levels	Type of TKI	Aplaziye kadar geçen tedavi süresi	Hematological parameters at aplasia	Duration of treatment before aplasia <sup>2</sup>	Management of aplasia	Clinical situation when recovery from aplasia	References
Patient 1	46/m	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	128 days	Hb: 5-6 gr/dL, NEU 400*10 <sup>9</sup> /L, Plt: 10-20000*10 <sup>9</sup> /L	222 days and ongoing	cessation of drug	optimal response	Chng WJ et al. (2)
Patient 2	72/m	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	3 years	WBC: 0.2 × 10 <sup>9</sup> L, Hb: N/A, Plt: N/A	1 month	cessation of drug, sisklosporin +ATG ve GCSF treatments were given	major molecular response	Hernández-Boluda JC et al (3)
Patient 3	47/f	WBC: 123*10 <sup>9</sup> /L (tanı anı)	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	3 years	WBC: 0.2 × 10 <sup>9</sup> L, Hb: 4,6 g/dl, Plt: 34*10 <sup>9</sup> /L	1 month	IVIg and prednisolon treatments were given	recovery from aplasia, but BCR-ABL FISH: +41,4 postivei	LeMarbre G et al (4)
Patient 4	46/f	Hb: 10,2 gr/dL, WBC: 73*10 <sup>9</sup> /L, Plt: 533*10 <sup>9</sup> /L (tanı anı, TKI başlangıç değeri belirtilmemiş)	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	53 days	WBC: 0,2*10 <sup>9</sup> /L, Hb: 5 gr/dl, Plt: 17*10 <sup>9</sup> /L	2 weeks, then died	cessation of drug, GCSF was given	died	Lokeshwar N et al (5)
Patient 5	51/m	WBC:56*10 <sup>9</sup> /L, Hb: N/A, plt: N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	19 months	WBC: 1900*10 <sup>9</sup> /L, Hb: 7,3 gr/dl, Plt: 42*10 <sup>9</sup> /L	35 days, then died	cessation of drug, GCSF was given	died	Khan KA (6)
Patient 6	54/f	WBC:130*10 <sup>9</sup> /L, Hb: 10,6 g/dl, Plt:212*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	6 months	WBC: 2.2*10 <sup>9</sup> /dl, Hb: 5,4 g/dl, Plt: 32*10 <sup>9</sup> /L	N/A	cessation of drug	BCR-ABL FISH: Positive	Srinivas U et al (7)
Patient 7	38/f	WBC: 122*10 <sup>9</sup> /L, Hb: 7,2 g/dl, Plt: 100*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	6 months	WBC: 1.9*10 <sup>9</sup> /L, Hb: 5,3 g/dl, Plt: 17*10 <sup>9</sup> /L	N/A	cessation of drug	BCR-ABL FISH: Positive	Srinivas U et al (7)
Patient 8	28/m	WBC: 135*10 <sup>9</sup> /L, Hb: 6,4 g/dl, Plt: 222*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	6 months	WBC: 1.58*10 <sup>9</sup> /L, Hb: 4,8 g/dl, Plt: 12*10 <sup>9</sup> /L	N/A	cessation of drug	BCR-ABL FISH: Positive	Srinivas U et al (7)
Patient 9	15/m	WBC: 157*10 <sup>9</sup> /L, Hb: 6,6 g/dl, Plt: 268*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	3 months	WBC: 1.3*10 <sup>9</sup> /L, Hb: 5,5 g/dl, Plt: 23*10 <sup>9</sup> /L	N/A	cessation of drug	BCR-ABL FISH: Positive	Srinivas U et al (7)
Patient 10	50/m	WBC: 123*10 <sup>9</sup> /dL, Hb: 8,6 g/dl, Plt: 168*10 <sup>9</sup> /L PB-blasts:32%	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	3 months	WBC: 2.5*10 <sup>9</sup> /L, Hb: 6,8 g/dl, Plt: 40*10 <sup>9</sup> /L	N/A	cessation of drug	BCR-ABL FISH: Positive	Srinivas U et al (7)
Patient 11	61/f	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	3,5 months	N/A	N/A	patient expired due to intracranial haemorrhage	N/A	Madabhavi I (8)
Patient 12	65/f	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	4 months	N/A	10 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 13	63/f	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	2 months	N/A	14 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 14	70/f	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	1,5 months	N/A	9 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 15	74/m	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	6 months	N/A	N/A	patient was succumbing due to septicemia	N/A	Madabhavi I (8)
Patient 16	68/f	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	6 months	N/A	5 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 17	76/f	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	4 months	N/A	7 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 18	34/m	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	2,5 months	N/A	15 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 19	42/m	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	2 months	N/A	12 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 20	55/f	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	3 months	N/A	11 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 21	32/m	N/A	BCR-ABL FISH: + Transcript levels: N/A	Imatinib	2,5 months	N/A	11 months	cessation of drug, hydroxiurea was given	BCR-ABL FISH: Positive	Madabhavi I (8)
Patient 22	49/m	WBC: 130*10 <sup>9</sup> /L, Hb: 10,4 gr/dl, Plt: 781*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript levels: N/A	Nilotinib (switched from 7 months dasatinib treatment)	19 months	WBC: 2,4*10 <sup>9</sup> /L, Hb: 9,8 gr/dl, Plt: 16*10 <sup>9</sup> /L	2 months	cessation of drug, 2 months later started nilotinib again due to BCR-ABL FISH postivity	FISH for BCR-ABL negative during aplasia, positive after 2 months later	Prodduturi P (9)

(continued)

	Age/sex	Hematological parameters at starting treatment with TKI or diagnosis	BCR-ABL FISH levels	Type of TKI	Aplaziye kadar geçen tedavi süresi	Hematological parameters at aplasia	Duration of treatment before aplasia2	Management of aplasia	Clinical situation when recovery from aplasia	References
Patient 23	26/m	N/A	BCR-ABL FISH: + Transcript lev- els: N/A	dasatinib 100 mg	5 months	WBC: 2,2*10 <sup>9</sup> /L, Hb: 7,2 gr/dl, Plt: 35*10 <sup>9</sup> /L	3 months	cessation of drug, 3 months later started dasati- nib 20 mg/d >>pancytope- nia>>imatinib 100 mg/d >>imatinib 600 mg/d>>pancy- topenia>>nilo- tinib 2*400 mg>>unde- tectable BCR- ABL>>ASCT	BCR-ABL FISH positive, Triz- omy 8+	Feld J (10)
Patient 24	53/m	WBC: 575*10 <sup>6</sup> /L, Hb: 7,6 gr/dl, Plt: 380*10 <sup>6</sup> /L	BCR-ABL FISH: + Transcript level: %85+	dasatinib 100 mg	5 months	N/A	4 months	cessation of drug, cyclosporine +eltrombopag +GCSF >>4 months later imatinib restarted again	BCR-ABL FISH: Positive	LEWALLE P (11)
Patient 25	59/f	WBC:22*10 <sup>9</sup> /L, Hb:13,1 g/dl, Plt:951*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript level: %100	imatinib	2 months	WBC: N/A, Hb: 8 gr/dl, Plt: <10*10 <sup>9</sup> /L	2 months	maintenance with imatinib after infusion of peripheral blood stem cell collected at diagnosis	BCR-ABL FISH: Positive	LEWALLE P (11)
Patient 26	34/f	WBC:135*10 <sup>9</sup> /L, Hb: 10,6 g/dl, Plt: 326*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript level: %100	imatinib	3 years	WBC: 3,6*10 <sup>6</sup> /L, Hb: 8 gr/dl, Plt: 45*10 <sup>9</sup> /L	9 monts, then died	cessation of drug, cyclosporine was given	major molecular response	KASSAR O (12)
Patient 27	64/m	WBC: 323*10 <sup>9</sup> /L, Hb:N/A, Plt: 428*10 <sup>9</sup> /L	BCR-ABL FISH: + Transcript level: %81	imatinib	8 months	WBC: 15*10 <sup>9</sup> /L, Hb: 10 gr/dl, Plt: <10*10 <sup>9</sup> /L	3 months	cessation of drug, dasatinib star- ted>>aplasia after 15 months lat- er>>dose reduction to 50 mg/d>>high frequency of BCR-ABL as %70+>>omace- taxine started>>4 months later ASCT has done	BCR-ABL FISH: Positive	Ramdial JL (13)
Patient 28	50/m	WBC:26*10 <sup>9</sup> /L, Hb: N/A, Plt: 1042*10 <sup>9</sup> /L	BCR-ABL FISH:+ Transcript level: N/A	imatinib switched to dasatinib due to sub- optimal response then niloti- nib due to intolerance	2 months	N/A	19 months, then died	cessation of drug, eltrombopag started	BCR-ABL FISH: Positive	Ramdial JL (13)
Patient 29	58/m	WBC: 233*10 <sup>9</sup> /L, Hgb: N/A, Plt: N/A	BCR-ABL FISH:+ Transcript level: %97	dasatinib 150 mg	6 months	WBC: 1,2*10 <sup>9</sup> /L, Hb: 2,5 gr/dl, Plt: 7*10 <sup>9</sup> /L	3 months	cessation of drug, ponatinib started and aplasia occurred again then ASCT has done	BCR-ABL FISH: Positive	Kamijo K (14)
Patient 30	63/f	WBC: 380*10 <sup>9</sup> /L, Hb: 3,9 g/dl, Plt: 436*10 <sup>9</sup> /L	BCR-ABL FISH:+ Transcript level: N/A	imatinib	4 months	WBC:0,4*10 <sup>9</sup> /L, Hb: 3,1 gr/dl, Plt: 21*10 <sup>9</sup> /L	6 months	cessation of drug, followed with- out treatment	N/A	Dogra R (15)
Patient 31	46/m	N/A	BCR-ABL FISH:+ Transcript level: N/A	imatinib	8 weeks	WBC: 1,4*10 <sup>9</sup> /L, Hb: 6,4 gr/dl, Plt: 6*10 <sup>9</sup> /L	9 months, then died	cessation of drug, ATG, cyclo- sporine, steroid and GCSF started	partial response	Mabed M (16)
Patient 32	77/m	WBC: 12,3*10 <sup>9</sup> /L, Hb: 12,6 g/dl, Plt: 563*10 <sup>9</sup> /L	BCR-ABL FISH:+ Transcript level: N/A	imatinib switched to nilotinib due to intolerance	2 months	WBC: 0,3*10 <sup>9</sup> /L, Hb: 4,7 gr/dl, Plt: 3*10 <sup>9</sup> /L	4 months	cessation of drug	major molecular response	Song M (17)
Patient 33	73/f	WBC: 8,4*10 <sup>9</sup> /L, Hb:12 g/dl, Plt: 19*10 <sup>9</sup> /L	BCR-ABL FISH:+ Transcript level: N/A	imatinib	17 days	N/A	N/A	N/A	N/A	Sumi M (18)
Patient 34	53/f	WBC: 56*10 <sup>9</sup> /L, Hb:N/A, Plt: 650*10 <sup>9</sup> /L	BCR-ABL FISH:+ Transcript level: %96	nilotinib	2,5 months	WBC: 0,9*10 <sup>9</sup> /L, Hb: 10 gr/dl, Plt: 9*10 <sup>9</sup> /L	5 months	cessation of drug, romiplostim started then dasatinib 50 mg/d started	Optimal response	Estephan F (19)

## PP 08

**CASE PRESENTATION: TREATMENT AND FOLLOW-UP EXPERIENCE FROM MYELODYSPLASTIC SYNDROME (MDS) REAB II TO CHRONIC MYELOMONOCYTIC LEUKEMIA (CMML)**

Harika Shundo <sup>1,\*</sup>, Tuba Öztoprak <sup>1</sup>

<sup>1</sup> Bezmialem Foundation University Hospital

**Objective:** The purpose of this case presentation is to discuss the clinical course, pathological findings, and treatment process of a patient diagnosed with MDS REAB II. It examines the transformation to CMML under the treatment of Venetoclax + Azacitidine. Challenges encountered during the follow-up process of CMML are mentioned. By addressing the continuation of MDS REAB II treatment in CMML, it is aimed that the findings obtained from this case contribute to the diagnostic and treatment processes for similar patients. **Case Report:** A 79-year-old male patient was found to have anemia, thrombocytopenia, and leukocytosis in the hemogram. Atypical cells were observed in the peripheral smear. The patient had lost 8 kg in the last 3 months and experienced night sweats. His medical history includes prostate cancer and heart diseases. Abdominal tomography revealed hepatomegaly and splenomegaly. At presentation: WBC -  $16.12 \times 10^3/\mu\text{L}$ ; absolute monocyte count (MONO) -  $3.61 \times 10^3/\mu\text{L}$ ; Hemoglobin (HGB) - 9.1 g/dL; Hematocrit (HTC) - 29.5%; Mean corpuscular volume (MCV) - 94.9 fL; Platelet (PLT) -  $129 \times 10^3/\mu\text{L}$ ; Creatinine - 1.27 mg/dL; Lactate Dehydrogenase (LDH) - 340 U/L; eGFR - 67 mL/min/1.73 m<sup>2</sup>; Albumin - 3.5 g/dL; Total Protein - 7.9 g/dL; Ferritin - 171.42  $\mu\text{g/L}$ ; Folate - 16.9  $\mu\text{g/L}$ ; B12 - 353 ng/L. Anti-HBc IgG: (+), HBsAg: (-), Anti-HCV: (-), Anti-HIV: (-). On 09/02/2024, the bone marrow pathology result showed an increase in blasts, leading to the diagnosis of MDS REAB-II. Flow cytometry revealed an 11.1% blast rate in the bone marrow. Treatment with Venetoclax and Azacitidine was initiated for MDS REAB-II. After 4 cycles, follow-up results showed: WBC -  $16.38 \times 10^3/\mu\text{L}$ ; MONO -  $3.54 \times 10^3/\mu\text{L}$ ; HGB - 11.6 g/dL; HTC - 35.7%; MCV - 90.4 fL; PLT -  $110 \times 10^3/\mu\text{L}$ ; Creatinine - 1.27 mg/dL; LDH - 257 U/L. Due to ongoing bicytopenia and for treatment response evaluation, a biopsy performed on 24/07/2024 revealed findings consistent with CMML without an increase in blasts. An off-label application was made for the continuation of current treatment. The effectiveness of the combination of Venetoclax and Azacitidine in the treatment of CMML is also being investigated, with the goal of monitoring the patient with the current treatment. **Methodology:** On December 6, 2023, the patient underwent surgery after preoperative severe anemia and thrombocytopenia, requiring blood transfusions. After discharge, the patient was referred to the hematology outpatient clinic. A bone marrow biopsy was planned due to atypical cells observed in the peripheral smear related to bicytopenia, which was sent for pathology and flow cytometry studies. **Results:** Abdominal tomography revealed: liver size increased to 165 mm, with millimetric parenchymal calcifications observed in the liver dome; spleen size increased to 141 mm. On 22/01/2024, pathology results showed MDS REAB II with increased blasts. Flow cytometry indicated a blast rate of 11.1% (CD13/CD117/CD34). Such findings are typically observed

in cases favoring "MDS." After 4 cycles of Venetoclax and Azacitidine treatment, a repeat biopsy on 24/07/2024 showed results indicating RAEB-II type MDS. The bone marrow was sampled regarding blast percentage. It is unclear if the patient has been treated recently. Peripheral blood reports indicate relative (30.7%) and absolute (2.97 k/ $\mu\text{L}$ ) monocytosis, normochromic normocytic anemia, and thrombocytopenia. With absolute (2.97 k/ $\mu\text{L}$ ) and relative (30%) monocytosis present; the hypercellular bone marrow (%60) represents slight maturation anomalies compatible with CMML-I, and no blast increase was detected. **Conclusion:** The challenges in diagnosing and treating CMML arise from the coexistence of dysplasia and myeloproliferative features. According to World Health Organization criteria, the diagnostic criteria have been met considering the patient's condition. For the first time in this case, a transition from MDS REAB II to CMML has been observed under this treatment. Azacitidine and Decitabine, approved for the treatment of MDS, have also been approved for CMML patients. Furthermore, more advanced studies are underway regarding the effectiveness of Azacitidine and Decitabine in CMML treatment. The effectiveness of the combination of Venetoclax and Azacitidine is also being investigated, with the goal of monitoring the patient with the current treatment.

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## PP 09

**PERITONEAL MESOTHELIOMA AS A COMALIGNANCY IN A PATIENT WITH CLL/SLL: CASE REPORT**

Satı Betül Beydilli <sup>1,\*</sup>, Ennur Ramadan <sup>2</sup>, Güven Çetin <sup>3</sup>, Mehmet Aydın <sup>4</sup>

<sup>1</sup> Bezmialem Foundation University Hospital, Faculty of Medicine, Department of Internal Medicine

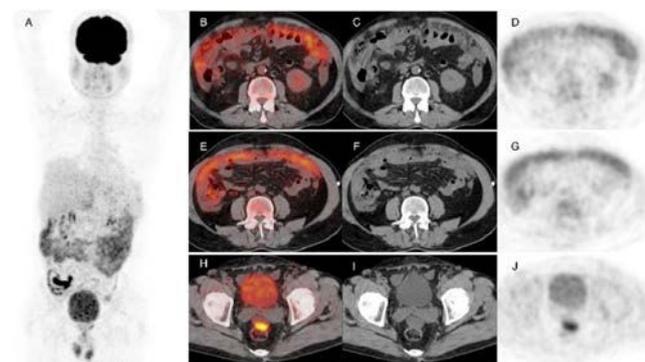
<sup>2</sup> Bezmialem Foundation University Hospital, Faculty of Medicine

<sup>3</sup> Bezmialem Foundation University Hospital, Faculty of Medicine, Department of Hematology

<sup>4</sup> Bezmialem Foundation University Hospital, Faculty of Medicine, Department of Nuclear Medicine

**Objective:** Malignant mesothelioma (MM) is an aggressive tumor typically arising from the pleura, with malignant peritoneal mesothelioma (MPM) accounting for 10-15% of cases. The occurrence of MPM alongside hematologic malignancies is rare. Here, we present a case of peritoneal mesothelioma developing synchronously with CLL/SLL. **Case Report:** A 68-year-old male was referred to our clinic in August 2023 with lymphocytosis, reporting weight loss and night sweats. His medical history included diabetes, hyperlipidemia, and hypertension, and a family history of stomach cancer. The patient had quit smoking 30 years ago and had a history of chronic alcohol use. There was no known asbestos exposure despite his occupation as a construction worker. Physical examination was normal. Routine laboratory tests and flow cytometry were conducted.

Imaging via thoracic and abdominal USG and PET/CT identified multiple lymphadenopathies and omental thickening indicative of peritoneal infiltration (Image-1). The patient was diagnosed with RAI Stage 3 CLL/SLL. In addition to hematological follow-up, the patient was referred to oncology and general surgery. He chose to continue his hematological follow-up in our clinic while receiving oncological and surgical follow-up at an external center. He is treated for CLL with ibrutinib and cisplatin-pemetrexed-altuzan for mesothelioma. **Discussion:** There is limited knowledge about the epidemiology and treatment of malignant peritoneal mesothelioma due to its rarity. In studies of mesothelioma associated with hematological malignancies, patients published predominantly have pleural mesothelioma. **Conclusion:** As a result, mesothelioma should be considered as a differential diagnosis in hematological cancer patients with abdominal masses, and further investigation needs to be conducted.



**Image 1:** Full Body PET scan (A), Axial PET/CT images showing omental thickening and peritoneal involvement (B, E, H), Corresponding axial CT images (C, F, I), PET images highlighting FDG uptake (D, G, J) Bone marrow and omentum biopsies were performed. The bone marrow biopsy confirmed CLL/SLL.

**Table 1:** Omentum biopsy revealed low-grade malignant epithelial mesothelioma

Immunohistochemistry	Case
Calretinin	Positive
BAP1	Negative
P16 (CDKN2A / 9p21)	Homozygous positive

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#### Adult Hematology Abstract Categories

##### Chronic Myeloproliferative Diseases

PP 10

##### HAIR REPIGMENTATION IN AN OLDER PATIENT TREATED WITH ASCIMINIB

Burcu Altındağ Avcı <sup>1,\*</sup>, Burhan Turgut <sup>2</sup>

<sup>1</sup> Tekirdağ City Hospital

<sup>2</sup> Tekirdağ Namık Kemal University

**Objective:** Asciminib may be a promising treatment option for intolerance of tyrosine kinase inhibitors (TKIs). It is a first-in-class inhibitor with a more selective mechanism of action different from the ATP-competitive inhibition that occurs with TKIs. Adverse effects (AEs) related to the inhibition of non-BCR::ABL1 kinases have been expected to be greatly diminished. According to the literature, fifty-five percent of patients experienced some AEs: mostly mild (grades 1–2), with 18% being grade 3–4. The most frequent AEs were fatigue (18%), skin rash (18%), thrombocytopenia (17%), and anemia (12%). The most frequent grade 3–4 AEs were thrombopenia (3.9%) and fatigue (3%). Other AEs were pneumonitis and hypoglycemia reported post-marketing. **Case Report:** A 61-year-old man was diagnosed with chronic myeloid leukemia (CML) and started on 80 mg asciminib. After 20 weeks of treatment, he experienced an unexpected change in hair color from gray to dark brown, without using hair dye or supplements. The same color change was also present in his mustache and beard. No other side effects were observed. **Management and outcome:** It was decided to monitor the patient with no action taken as he feels pleasant with this unexpected side effect of asciminib. CML remained in deep molecular remission. The dark brown hair color persisted over time. **Discussion/Conclusion:** Hair hyperpigmentation likely occurred through melanocyte activation via asciminib. Severe side effects may require dosage adjustments, while milder effects can be monitored closely. The newly observed hair color restoration in this case highlights potential dual (therapeutic and aesthetic) applications of this class of agents.

<https://doi.org/10.1016/j.htct.2024.11.038>

PP 11

##### VULVAR AND VAGINAL GRAFT VERSUS HOST DISEASE IN A PATIENT WITH CHRONIC PHASE CHRONIC MYELOID LEUKEMIA AFTER ALLOGENEIC STEM CELL TRANSPLANTATION

Esra Pirinççi <sup>1,\*</sup>, M. Orhan Ayyıldız <sup>1</sup>,  
Abdullah Karakuş <sup>1</sup>, Reyhan Gündüz <sup>1</sup>

<sup>1</sup> Dicle University, Faculty of Medicine, Department of Hematology

**Objective:** Graft versus Host Disease (GVHD) is one of the serious complications of allogeneic stem cell transplantation used in the treatment of many hematological malignancies. Skin, liver, and eyes are frequently affected areas. In addition to frequently affected areas, genital region involvement can also be seen. Allogeneic stem cell transplantation is one of the definitive treatments for hematological malignancies seen in the young age group. And its use for therapeutic purposes in young patients is increasing day by day. Vulvovaginal GVHD is a disease type that concerns female patients of reproductive age. In this case report, we wanted to include in the literature a case that underwent allogeneic stem cell transplantation after CML diagnosis and TKI resistance and then developed vulvovaginal GVHD. In vaginal disease involvement; in addition to many genitourinary complaints, many negativities in sexual life and deterioration in quality of

life are experienced. The chronic GVHD patient we treated is currently continuing her treatment response follow-ups. Our aim in presenting this case to the literature is to emphasize that GVHD should be included in the differential diagnosis in female patients with hematological disease and vaginal involvement. **Case Report:** 42 years old female patient was diagnosed as chronic phase chronic myeloid leukemia in 2015. She was treated with imatinib 400 mg/day. After 6 months molecular response not obtained and treatment changed to dasatinib 100 mg/day, but after 3 months of dasatinib treatment molecular and hematologic progression occurred and treatment changed to nilotinib and bone marrow transplantation planned. After 4 months the patient transplanted successfully with HLA matched sibling stem cell donor. Tyrosine Kinase inhibitory used till 1 years after transplantation, Bcr/abl was negative after transplantation and until now. At 2 months of transplantation acute GvHD occurred and healed without any serious complication, but after 10 months symptoms and signs of chronic GvHD developed. Dry skin, itching, dark hyperpigmentation occurred in generalized of the body especially in the upper extremities and ocular GvHD was the main symptoms of the patient. She was used siklosporin and steroids for prophylaxis and treatment of GvHD, also she use ursodeoxycholic acid for liver protection. Chronic GvHD sustained more than 2 years especially ocular findings (drying, itching and scarring of conjunctiva and eyelid). After 5 years of transplantation she told to our nurse some symptoms such as vulvodinia, pain during sexual intercourse and decreased sexual function. She has problems with her husband for this reason. She applied on october 2023 for gynecological examination, there were findings consistent with vulvodinia, but there was no genital atrophy. We prescribed 2% amitriptyline + 2% baclofen cream two times a day for the treatment of vulvodinia **Methodology:** When she came for a check-up 1 month later of local treatment, she stated that she was better in terms of sexual function but could not urinate completely. Bacteriuria, pyuria and hematuria was observed in urinalysis. Since there was not much residual urine in the pelvic ultrasonography. We treat her for urinary tract infection. Since the patient's genital atrophy was not evident, we did not prescribe vaginal estrogen during both examinations. If she came for a checkup, we was planning to re-evaluate and treat her if necessary. Hematopoietic stem cell transplantation (HSCT) is a treatment method for malignant and benign hematological diseases as well as in the treatment of some non-hematological disorders such as autoimmune diseases (1). Graft-versus-host disease (GVHD) is an immunity related disease which affects 30-70% of patients after hematopoietic stem cell transplantation (alloHSCT) and is a significant contributor of morbidity and non relapse mortality (NRM) is the reason (2). Chronic GVHD is a mucosal disease of the mouth, eyes, genitals, intestines, and lungs. It includes inflammation and fibrosis of membranes. There are some evidences which indicates clinical symptoms and pathogenesis of GVHD is similar to various autoimmune disorders such as Scleroderma, Sjögren's syndrome and lichen planus. (3,4). Female genital GVHD was first described by Corson et al.

By observing Sclerosing vaginitis and structure problems in 5 women in 1982 (5). Nowadays, it is an underdiagnosed condition and affects the quality of life which occurs in one quarter of long-term surviving women after allogeneic stem cell transplantation (6). The rates of genital GVHD vary widely, with rates ranging from 24.9-69% (7). **Results:** The wide variation in the incidence of genital GVHD is due to a variety of abnormalities, including the time at which incidence is calculated, the systematic and time-dependent gynecological evaluations, and the diagnostic criteria used (findings of examination with or without symptoms, etc.) (8). The main risk factor for the development of chronic genital GVHD issuing of peripheral blood as a source of progenitor cells; It represents a risk of three times higher than that obtained from bone marrow cells (9-11). The presence of GvHD in another organ is also considered one of the risk factors (12). While one study found that 79% of patients with VVGvHD were treated for GvHD in a different organ, another study reported that almost all patients with VVGvHD had active chronic GvHD in the skin, mouth, and eyes (13-14). Our patient was receiving treatment for skin and liver involvement caused by chronic GVHD. It is supported by various studies that it develops after an average of 10.2 months after transplantation (6). In our patient, this condition was detected approximately 5 years after allogeneic transplantation. Clinic may be asymptomatic; The main signs and symptoms are vulvar tenderness to palpation of openings of the mucosa, erosion of the mucosa, cracks, leukokeratosis, labial or clitoral fusion, fibrous vaginal ring, vaginal shortening, vaginal adhesions and complete vaginal stenosis. Other symptoms include dryness, burning, itching, pain to touch, dysuria, dyspareunia and resulting sexual dysfunction takes place (5). **Conclusion:** She has vulvodinia, pain during sexual intercourse and decreased sexual function. Although symptoms are similar to primary ovarian insufficiency which occurs after allogeneic stem cell transplantation, synechia and adhesive bands are not encountered in primary ovarian failure. In addition, studies have shown that hormone replacement therapy is used for the prophylaxis of this condition does not effects development rate of vulvo vaginal GVHD (11). The National Institutes of Health (NIH) Consensus Development Project proposed guidelines for screening, diagnosing, and preventing genital GVHD in HSCT survivors. Treatment goals for Female genital GVHD include symptom relief, disease control and prevention of further damage (7). In its treatment various patient-specific treatment modalities are advocated such as topical estrogens, topical steroids, topical immunosuppressive agents (such as cyclosporine, tacrolimus), vaginal dilators and surgically (9,16). Diagnosis and treatment of post-transplant genital GVHD requires a systematic approach and collaboration between bone marrow transplant physicians and coordinators and gynecologists. A systematic approach is required, requiring close cooperation between gynecologists. Incidence and severity of genital GVHD in women should be included in GVHD intervention studies.

<https://doi.org/10.1016/j.htct.2024.11.039>

PP 12

### EFFECT OF FREQUENT GENERIC IMATINIB SWITCHING ON TREATMENT RESPONSE IN PATIENTS WITH CHRONIC MYELOID LEUKEMIA

Murat Çınarsoy<sup>1,\*</sup>, Buğra Sağlam<sup>2</sup><sup>1</sup> Şanlıurfa Mehmet Akif İnan Training and Research Hospital, Clinic of Hematology<sup>2</sup> Medicalpoint Gaziantep Hospital, Clinic of Hematology, Stem Cell Transplant Unit

**Objective:** The aim of this study was to evaluate the effect of switching one or more generic imatinibs during treatment on outcomes in patients with chronic phase chronic myeloid leukemia. **Case Report:** Chronic myeloid leukemia (CML) is a myeloproliferative neoplasm associated with the Philadelphia chromosome t(9;22)(q34;q11) and the BCR::ABL1 fusion gene, which produces a constitutively active BCR::ABL1 tyrosine kinase. CML accounts for approximately 15 to 20% of adult leukemia. It has an annual incidence of 1 to 2 cases per 100,000, with a slight male predominance. The median age at presentation is approximately 50 years, and the prevalence of CML is steadily increasing in the Western world because of the dramatic effect of ABL1 kinase inhibitors on survival. Imatinib was the first commercially available TKI to approved by the U.S Food and Drug Administration (FDA) and the European Medicines Administration (EMA) for the initial treatment of CML. The high cost of new cancer drugs, including those developed for CML, is a major concern for healthcare payers, especially in countries with limited resources. Reimbursement policies around the world therefore encourage the use of generics to reduce prices. The European LeukemiaNet 2020 recommendation for the use of generic imatinib is as follows: "As long as a generic medicine meets the national standards of the country concerned in terms of quality of production, bioavailability and efficacy, it is an acceptable alternative to the branded product. It is recommended that the patients continue to use the same generic brand whenever possible to avoid potential side effects due to changes in drug structure, bioavailability and excipients." In the NCCN guidelines, the recommendation for generic drugs is as follows: "Innovator and generic drugs approved by regulatory authorities on the basis of pharmacokinetic equivalence can be used interchangeably" and "In countries where more than one generic drug is available, switching from one generic drug to another is not recommended". **Methodology:** We retrospectively analyzed data from patients diagnosed with CML-CP treated with imatinib from 2010 - 2024. Patients with chronic phase chronic myeloid leukemia who were over 18 years of age and who started treatment with original or generic imatinib and switched to generic imatinib at any time during treatment were included. Patients who were diagnosed before the age of 18, patients whose treatment was interrupted during pregnancy, patients who did not use generic imatinib or patients who used only one brand of generic imatinib permanently were excluded from the study. The characteristics of the patients and the follow-up periods were collected retrospectively from the patients' electronic files. The efficacy of treatment was evaluated via standard hematological and molecular assessments to

determine the rates of complete hematological response (CHR), molecular response (MR), and treatment failure, which was defined as a bcr-abl level of 1 % or higher on two occasions with an interval of one month. **Results:** A total of 46 patients, 26 (56.5%) male and 20 (43.5%) female (male/female ratio, 1.3), were included in the study. The median age was 45 years (range, 20-77 years). Forty-one (89.1%) of the patients were under 65 years of age, and 5 (10.9%) were over 65 years of age. The starting dose of imatinib was 400 mg/d in all patients. Treatment was started with Gleevec in 11 patients and generic imatinib in 35 patients. All patients were switched to two or more generic imatinibs during treatment. During the treatment process, 12 patients 2, 13 patients 3, 12 patients 4, 7 patients 5 and 1 patient 6 used different types of generic imatinib. Loss of response occurred in 8 of 46 (17.3%) patients. The earliest loss of response occurred at month 6, and the latest loss occurred at year 9. One patient lost response at month 6, 1 patient at year 1, 2 patients at year 2, 1 patient at year 3, 2 patients at year 7, and 1 patient at year 9. All patients who experienced a loss of response responded to second-generation tyrosine kinase inhibitors, and none developed an accelerated or blastic phase. No dose increases or switches back to the original product in patients with loss of response. No patients had their dose changed or discontinued due to adverse events. When evaluated according to age, sex and number of generic imatinib switches, none of these variables were found to have any effect on response loss. **Conclusion:** Following the introduction of generic imatinib, several studies have shown that there is no loss of efficacy in patients who are switched from Glivec to generic imatinib. Although the ELN and NCCN CML guidelines do not discourage the use of generic imatinib, switching between generic imatinibs is not recommended. A review of both guidelines and the literature revealed no information on the development of adverse outcomes related to treatment response in patients switching between generic imatinibs. In the abovementioned retrospective studies, data on the responses obtained from patients receiving more than one type of generic imatinib were not shared. In our study, the response loss in patients who received more than one generic imatinib was 17.3%, which is comparable to the response losses observed in other studies of patients who received the original imatinib or generic imatinib. The findings of our study indicate that switching between generic imatinibs does not have a detrimental effect on treatment response.

<https://doi.org/10.1016/j.htct.2024.11.040>

PP 13

### ONE CASE OF CHRONIC MYELOID LEUKEMIA IN PEDIATRIC GROUP

Nihal Boz<sup>1,\*</sup><sup>1</sup> Adana City Hospital

**Objective:** Chronic myeloid leukemia (CML) is a myeloproliferative syndrome caused by monoclonal myeloid proliferation with the passage of immature granular elements into the peripheral blood. It is a rare disease in children and adolescents,

accounting for 2-3% of all leukemias in the pediatric population under the age of 15. (1) It is defined by the presence of a translocation (9;22), a cytogenetic abnormality associated with the disease. We report one of these rare cases because of its unusual frequency. **Case Report:** Fourteen year male child came to the pediatric hematology policlinic complaints of abdominal distension, bone pain and weakness. Clinical examination revealed mucocutaneous pallor and hepatosplenomegaly. The complete blood count received on the day of admission showed hyperleukocytosis at 178000/ $\mu$ L, normocytic normochromic anemia at 10,8 g/dl and thrombocytosis at 281000/ $\mu$ L. When the blood smear was examined, it was seen that there were myelocytes, metamyelocytes and promyelocytes, neutrophils and 4% myeloid-appearing blasts. Subsequent bone marrow aspiration showed hyperplasia of the neutrophilic granulocytic lineage at all stages of maturation, with promyelocyte, hyper granular myelocyte, metamyelocyte. (Figure 1) Cytogenetic analysis of the bone marrow as part of the etiological work-up confirmed the presence of the Philadelphia chromosome. Molecular testing for the BCR-ABL1 fusion transcript by RT-PCR on EDTA whole blood detected 64% (IS). The patient was admitted to the pediatric hematology service and started on hydroxyurea treatment. After the genetic diagnosis was confirmed, he was treated with Imatinib, a first-generation tyrosine kinase inhibitor (TKI). In the molecular evaluation performed at the 3-month follow-up, BCR-ABL1 fusion transcript was detected as 5% (IS) by RT-PCR. **Discussion:** Chronic myeloid leukemia (CML) is a rare hematological malignancy in the pediatric population. For treatment, our patient benefited from specific Imatinib therapy. According to the literature, Imatinib is the first-line drug.

<https://doi.org/10.1016/j.htct.2024.11.041>

## Adult Hematology Abstract Categories

### Coagulation Diseases

PP 14

#### PAGET SCHROETTER SYNDROME AND HOMOZYGOUS FACTOR V LEIDEN MUTATION: A CASE PRESENTATION

Damla Cagla Patır<sup>1,\*</sup>, Nigar Abdullayeva<sup>1</sup>, Dogus Berk Kuzucu<sup>2</sup>, Mahmut Tobu<sup>1</sup>

<sup>1</sup> Ege University Faculty of Medicine, Department of Hematology

<sup>2</sup> Ege University Faculty of Medicine, Department of Internal Medicine

**Case Report:** Thrombosis in the deep veins of the upper extremity accounts for only 5% of symptomatic cases but constitutes approximately 50% of hospital-acquired thromboses. The vast majority of upper extremity thromboses, result from the presence of permanent venous catheter. Unprovoked cases are often secondary to "effort" thrombosis. Here, we present a case of Paget-Schroetter syndrome combined with a homozygous mutation of factor V Leiden. A 19-year-old female patient presented with pain and swelling in her right arm. The report of the right arm venous Doppler ultrasound indicated the presence of thrombus within the lumen at the

proximal and distal segments of the basilic vein at the fossa cubiti level. The patient was found to have a homozygous mutation of factor V Leiden, and it was learned that she had been undergoing intense training and was engaged in water polo for the last two months. She had no history of medication use or chronic illnesses, nor any previous history of thrombosis. The patient was started on low molecular weight heparin for three months. A control Doppler ultrasound showed that the existing thrombus had resolved. It was recommended that the patient continue on her current anticoagulation with a new generation oral anticoagulant for one year. During this period, the patient, who ceased sports activities, did not develop any new thrombosis. The combination of young age, intense physical activity, especially in sports that utilize the upper extremities, and risk factors such as the factor V Leiden mutation strengthens the diagnosis. In the pathophysiology of this syndrome, vascular microtrauma and exercise, muscle hypertrophy and thrombophilias contribute to the condition. Low molecular weight heparin and new generation oral anticoagulants are effective in preventing thrombosis formation and in inhibiting the growth of existing thrombus. Thrombolytic therapy may be considered in cases of large thromboses or severe symptoms.

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PP 15

#### DESENSITIZATION TO RIVAROXABAN IN A PATIENT WHO EXPERIENCED ANAPHYLACTOID SHOCK AFTER ANTICOAGULANT USE: CASE REPORT

Damla Cagla Patır<sup>1,\*</sup>, Nigar Abdullayeva<sup>1</sup>, Züleyha Galata<sup>2</sup>, Umitcan Ates<sup>2</sup>, Kutay Kırdok<sup>2</sup>, Tugba Mermer<sup>3</sup>, Sükriye Miray Bozgul<sup>4</sup>, Reyhan Gumusburun<sup>2</sup>, Elif Ertuna<sup>5</sup>, Aytül Zerrin Sin<sup>2</sup>, Mahmut Tobu<sup>1</sup>

<sup>1</sup> Ege University Faculty of Medicine, Department of Hematology

<sup>2</sup> Ege University Faculty of Medicine, Department of Allergy and Immunology

<sup>3</sup> Ege University Faculty of Medicine, Department of Internal Medicine

<sup>4</sup> Ege University Faculty of Medicine, Department of Intensive Care

<sup>5</sup> Ege University Faculty of Pharmacy

**Case Report:** Over the last two decades, new anticoagulants have been developed to prevent and manage thromboembolic diseases, including direct-acting anticoagulants like rivaroxaban, which is used for venous thromboembolism prevention, stroke prevention in atrial fibrillation, and ischemic heart disease. Here, we present the experience of a case with a history of multiple thromboses and an anaphylactoid reaction to anticoagulants, who was able to continue prophylaxis without allergic reactions after rivaroxaban desensitization. A 42-year-old female patient visited the hematology outpatient clinic to obtain a prescription for a new anticoagulant due to a supply issue with her current medication, fondaparinux..

Her medical history included thrombosis in both upper and lower extremities ten years earlier, along with heterozygous mutations for factor V Leiden and MTHFR, necessitating life-long anticoagulant therapy. She had previously experienced anaphylactic shock from enoxaparin, warfarin, tinzaparin, and rivaroxaban, which led her to use fondaparinux without issues. When faced with a supply problem prescribed apixaban, she suffered anaphylactic shock thirty minutes after administration, requiring epinephrine treatment. Following this, the allergy and immunology department recommended a desensitization protocol for rivaroxaban, crucial for her ongoing anticoagulation. After a one-day desensitization, she successfully continued treatment with 20 mg of rivaroxaban without any allergic reactions during follow-up visits. Desensitization is a technique that allows patients with drug hypersensitivity reactions to safely maintain drug therapy by creating temporary tolerance, especially for IgE-mediated reactions. It works by inhibiting mast cell activation and reducing the release of inflammatory mediators, often resulting in decreased skin sensitivity and potentially negative skin test results after the procedure. In this case, the patient had a grade 3 early-type drug allergy, and while literature on desensitization for new-generation oral anticoagulants is scarce, the successful desensitization to rivaroxaban suggests that it may be an effective option for similar patients in the future.

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PP 16

#### INTERVENTIONAL PROCEDURE IN HEMOPHILIA A PATIENT WITH EXTENDED HALF-LIFE FACTOR THERAPY- CIRCUMCISION- CASE REPORT

Ferda Can<sup>1,\*</sup>, Gaye Kalacı<sup>1</sup>,  
Ozge Kösemehmetoğlu<sup>1</sup>, Davut Kamacı<sup>1</sup>,  
Sema Akıncı<sup>1</sup>, Sule Mine Bakanay Öztürk<sup>1</sup>,  
İmdat Dilek<sup>1</sup>, Tekin Güney<sup>1</sup>

<sup>1</sup> Ankara Bilkent Şehir Hastanesi

**Case Report:** Hemophilia A is a hereditary bleeding disorder due to factor VIII deficiency. With the advances in the treatment of hemophilia in recent years, the average life expectancy of patients has reached the healthy population. Along with prolonged life, additional diseases and intervention requirements are developing in this patient group. Due to the developments, management of patients going under interventions are more clear and easier. In this case, a patient who underwent an intervention with extended half-life factor therapy was presented. Forty-three-year-old male patient with severe hemophilia A was evaluated on request for circumcision surgery while using prophylactically extended half-life factor therapy 2 × 1000 Units / week. Tranexamic acid was started one day preoperatively to the patient whose basal factor level was below 1% and whose inhibitory level was negative. Body weight of the patient was 63 kg. Extended half-life factor VIII preparation (efmorogtocog alfa) loading dose of 3000 units was administered before half an hour of the procedure. aPtt was detected for 30 seconds and factor

VIII level was 55% 30 minutes after loading dose. The patient was given appropriate sedative treatment to prevent pre-operative erection. The operation was carried out without any problems. 1500 Units 12 hours after the loading dose, and 24 hours after this dose was performed. The patient was discharged without complications without bleeding. Factor therapy was continued with prophylaxis dosing. Tranexamic acid was continued for 7 days. No complications were observed. Interventional procedures of hemophilia patients can be performed without complications with a multidisciplinary approach under appropriate dose and scheme factor therapy. In the case, an interventional procedure was made by giving an extended half-life factor to a severe hemophilia patient who could not have a circumcision operation for many years due to previous hesitations of both patient and surgeons. ,

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#### Adult Hematology Abstract Categories

##### Lymphoma

PP 17

#### RARE CASE! SECOND PRIMARY MALIGNANCY IN LANGERHANS CELL HISTIOCYTOSIS, A JAK2+ CASE

Engin Yola<sup>1,\*</sup>, Aslı Odabaşı Giden<sup>2</sup>,  
Caner Çulha<sup>1</sup>, Düzgün Özatlı<sup>3</sup>

<sup>1</sup> Department of Internal Medicine, Faculty of Medicine, Ondokuz Mayıs University,

<sup>2</sup> Department of Hematology, Ordu State Hospital

<sup>3</sup> Department of HEMATOLOGY, Faculty of Medicine, Ondokuz Mayıs University

**Objective:** Langerhans cell histiocytosis (LCH) is a rare inflammatory myeloid neoplasm characterised by the infiltration of CD1a+CD207+ myeloid dendritic cells and immune cells, thus described as an inflammatory myeloid neoplasm that clonally expands. LCH is a histiocytic neoplasm affecting both paediatric and adult populations, with an estimated incidence of 3 to 5 cases per million children and 1 to 2 cases per million adults. LCH can involve all organ systems, with symptoms ranging from single organ disease to multi-system disease. While it can appear in any organ system, LCH has a particular affinity for bones, skin, lungs, and the pituitary gland. In 2016, LCH was reclassified from a reactive disorder to an inflammatory myeloid neoplasm following the identification of the recurrent BRAF V600E mutation in half of the cases and the observation of clonality. Recently, additional BRAF mutations that activate the MAP kinase pathway have been demonstrated, shedding more light on the pathogenesis of LCH. Several studies have suggested a high prevalence of second primary malignancies, including haematological and solid organ neoplasms, in LCH patients. **Case Report:** A 58-year-old male patient, with a known history of hypertension and hypothyroidism, presented to a medical facility in Germany in 2011 with skin lesions on the chest and neck swelling. Following lymph node and skin punch biopsies from the sternum, the patient was diagnosed with LCH, with imaging

revealing involvement in the frontal bone of the skull, neck, spleen, axillary, liver, lungs, and skin. The patient was treated with steroids. In 2014, while on holiday in Istanbul, the patient was given 6 cycles of vinblastine in addition to steroids. Steroid treatment was completed over 5 years, followed by regular follow-up. In 2021, the patient presented to Ordu State Hospital with fatigue and skin rashes resembling LCH lesions. Investigations revealed thrombocytosis, erythrocytosis, and leukocytosis. Bone marrow biopsy was reported as normal, and a punch biopsy of the skin lesions showed no evidence supporting LCH. Cytogenetic tests, however, revealed a JAK2+ mutation, which had not been detected in previous tests. The patient was started on hydroxyurea, and imaging showed a 5 cm mass in the spleen, for which splenectomy was recommended, though the patient declined and sought further consultation. Our cytogenetic studies confirmed BCRABL polymerase chain reaction (PCR), PML/RARA, and AML/MDS panel negativity, with JAK2+ positivity. Erythropoietin levels were 6 mU/ml (normal range: 3.7-31.5), LDH was 218 u/l, sedimentation rate was 60 mm/hour, platelet count was 517,000/ $\mu$ l, and white blood cell (WBC) count was 13,000/ $\mu$ l. Physical examination revealed remnants of old skin lesions (Figure 1), and there were no palpable lymph nodes or masses. Imaging showed a significant mass in the spleen and involvement in the frontal bone, liver, lungs, stomach, and neck lymph nodes, similar to previous findings. During follow-up, the patient occasionally reported pain in both legs, and Doppler studies revealed widespread thrombosis, which the patient stated had been occurring for the past 1.5 years but was disregarded. Subcutaneous anticoagulants and anti-stasis treatment (Daflon 1000) were initiated, later transitioning to oral anticoagulants. Follow-up showed improvement in symptoms under hydroxyurea and anticoagulant therapy, but recurring thrombotic events were noted during subsequent check-ups while on oral anticoagulants.

Figure 1: Skin findings and biopsy scar marks on the neck, sternum, and abdominal areas of the patient. **Conclusion:** Discussion Several case reports and smaller case series have observed that malignant diseases may occur before, concurrently with, or after LCH, with a frequency higher than by chance alone. Edelbroek, J. R., and colleagues linked the emergence of second malignancies in LCH to prior treatments with chemotherapeutic agents such as etoposide or vinblastine, with the second malignancies being identified as leukaemia and myelodysplastic syndrome (MDS). Another study by Goyal, Gaurav, and colleagues followed 1,392 LCH cases, showing that Hodgkin and non-Hodgkin lymphomas developed in children during follow-up, while adults developed MDS in early follow-up and had an increased risk of developing B-cell acute lymphoblastic leukaemia (B-ALL) after about five years. In children, the leading cause of death was infections, while in adults, it was second primary malignancies. In our literature review, we did not encounter any JAK2+ cases or studies following LCH, making the JAK2+ positivity observed in our LCH patient a potentially unique case. We did find that JAK2+ positivity has been observed in the follow-up of non-Langerhans cell histiocytosis. Given that LCH is rare and second primary malignancies are even more uncommon, identifying such cases remains challenging, and further clinical studies are clearly needed.

**Keywords:** Langerhans Cell Histiocytosis, JAK2+.



Undefined





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#### PP 18

##### A CASE OF DIFFUSE LARGE B CELL LYMPHOMA PRESENTING AS OSTEOSARCOMA

Mine Ezgi Payaşlı<sup>1,\*</sup>, Müzeyyen Aslı Ergözoğlu<sup>1</sup>, Berksoy Şahin<sup>1</sup>

<sup>1</sup>Çukurova University

**Case Report:** Diffuse large B cell lymphoma (DLBCL) is the most common histologic subtype of non-Hodgkin lymphoma (NHL) accounting for approximately 25 percent of NHL cases. Additionally, Diffuse Large B Cell Lymphoma is the most common lymphoma. In the United States and England, the incidence of DLBCL is approximately 7 cases per 100,000 persons per year. In Europe as a whole, the incidence is approximately 4.92 cases per 100,000 persons per year. Like most other NHLs, there is a male predominance with approximately 55 percent of cases occurring in men. Incidence increases with age; the median age at presentation is 64 years for patients as a whole. IB, 45 years male patient. MRI scan taken in 2022 after a complaint of pain in right knee revealed a malignant tumoral lesion (osteosarcoma?) that caused intramuscular invasion in a segment of approximately 20 cm in the 1/2 distal femur and caused extensive cortical destruction in the distal. A biopsy was taken from the distal right femur. He was diagnosed with non-Hodgkin lymphoma and diffuse large B-cell lymphoma.

Bcl-2, Bcl-6 and c-myc were found to be negative. After 4 cycles of R-CHOP protocol, PET-CT revealed minimal progression in the left clavicle and the IPI score was high. The patient's R-CHOP treatment was completed for 6 cycles with 2 cycles of intrathecal MTX. Afterwards, 2 cycles of maintenance rituximab were given. The patient, who subsequently went into remission, was followed up. This case shows us that NHL cases may present in a location such as primary bone tumor. The possibility of lymphoma should be considered in patients with atypical localization.

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#### PP 19

##### EFFICACY OF GLOFITAMAB IN PRIMARY REFRACTORY LYMPHOMA: A CASE REPORT

Muzaffer Keklik<sup>1,\*</sup>, Kemal Fidan<sup>1</sup>, Ali Unal<sup>1</sup>

<sup>1</sup>Erciyes University

**Objective:** Diffuse large B-cell lymphoma (DLBCL) constitutes 30% of non-Hodgkin lymphomas and is often curable with frontline chemoimmunotherapy. However, in some patients, remission cannot be achieved, and this situation necessitates the application of second, third or even fourth-line salvage therapies. The limited treatment options for relapsed or refractory (r/r) DLBCL underscore an unmet clinical need, which urges the development of new therapies for this patients. Glofitamab is a humanized IgG1 bispecific monoclonal antibody binds to CD20 on malignant B lymphocytes and to CD3 on cytotoxic T cells with promise for treating r/r DLBCL. Here we present a primary refractory DLBCL patient to whom we applied glofitamab treatment as the 5th line. **Case Report:** A 28-year-old male patient was diagnosed with stage IV germinal center DLBCL biopsy of sacral mass. The patient received dose-adjusted etoposide, prednisone, vincristine, cyclophosphamide, doxorubicin, and rituximab (EPOCH-R) as first-line treatment. However, progression was detected by 18F-Fluoro-2-deoxy-D-glucose (FDG) positron emission tomography (PET) with computed tomography (CT). Then, rituximab plus ifosfamide, carboplatin, etoposide (R-ICE), ifosfamide gemcitabine vinorelbine prednisolone (IGEV), salvage radiotherapy (RT), rituximab plus bendamustine (R-B) therapies were given, respectively. Since no response was obtained to all these treatments, glofitamab was started as the 5th line therapy. After the twelve cycles of glofitamab therapy, the patient achieved complete remission (CR). Four months post-treatment, he was still alive. **Discussion:** Glofitamab is approved as a third-line treatment for r/r DLBCL, inducing a CR in nearly 40% of patients in this situation. According to literature, CR can be maintained for years after completion of glofitamab treatment. Data from a follow-up in a cohort of patients who were treated with glofitamab showed a median duration of complete response of 34 months. Our case post-treatment fourth months was still alive. This case indicates that glofitamab is quite effective primary refractory DLBCL.

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PP 20

### A CASE OF MARGINAL ZONE LYMPHOMA PRESENTING WITH DIPLOPIA

Tansu Koparmal<sup>1,\*</sup>, Caner Çulha<sup>1</sup>,  
Aslı Odabaşı Giden<sup>2</sup>, Engin Yola<sup>1</sup>,  
Özgür Meletli<sup>3</sup>, Düzgün Özatlı<sup>4</sup>

<sup>1</sup> Ondokuz Mayıs University, Faculty of Medicine,  
Department of Internal Medicine

<sup>2</sup> Ordu State Hospital, Clinic of Hematology

<sup>3</sup> Samsun Training and Research Hospital, Clinic of  
Hematologyeducation

<sup>4</sup> Ondokuz Mayıs University, Faculty of Medicine,  
Department of Hematology

**Objective:** Marginal zone lymphoma (MZL) is characterized by the proliferation of B cells in post-germinal centers located in mucosa-associated lymphoid tissue (MALT), lymph nodes, and the spleen. MZL typically presents with an indolent clinical course. The average age at diagnosis is 60, with a slight female predominance, and it accounts for 5-17% of non-Hodgkin lymphomas (NHL). MZL is categorized into three subtypes based on the site of involvement: extranodal, splenic, and nodal MZL. Although these subtypes share many morphological and immunophenotypic characteristics as well as a slow clinical course, they can differ in terms of frequency, pathogenesis, clinical presentation, and treatment approach. The most common subtype is extranodal MZL, while nodal MZL is the least common. **Case Report:** A 51-year-old female patient presented to the clinic with a complaint of diplopia that had lasted for the past week. Physical examination revealed limited lateral gaze and anisocoria in the right eye, with other systemic examinations were normal. There were no B symptoms. Complete blood count, biochemical tests, serum electrolytes, and coagulation tests were within normal limits. Contrast-enhanced orbital MRI showed a lesion in the right intraorbital intraconal area, adjacent to the lateral aspect of the optic nerve and the medial aspect of the lateral rectus muscle. The lesion extended from the retroocular area to the orbital apex, obliterating intra-orbital fat planes. It measured 35 × 13 mm in the axial plane, was hypointense on T2-weighted imaging and T1-weighted imaging, and showed homogeneous diffusion restriction on diffusion-weighted imaging. Post-contrast series revealed intense homogeneous enhancement of the soft tissue. The lesion measured 27 × 17 mm in the coronal plane. The findings were primarily suggestive of lymphoma involvement. PET-CT scan identified a hypermetabolic soft tissue lesion in the right intraorbital-retrobulbar area, continuous from the lateral aspect of the lateral rectus muscle to the lateral orbit, consistent with lymphoma. No extraocular nodal or visceral hypermetabolic foci were detected. Orbital biopsy results confirmed marginal zone lymphoma. Although radiotherapy could have been considered as a treatment option for localized involvement, the decision was made to administer 6 cycles of RB (Rituximab and Bendamustine) chemotherapy to the patient in order to avoid complications associated with radiotherapy due to the lesion's location in the orbital region. Follow-up PET-CT after 6 cycles of RB showed complete metabolic response with total regression of the hypermetabolic soft tissue lesion in the right retroocular area. The patient is currently in remission.

This case is discussed due to the rare occurrence of ocular involvement in marginal zone lymphoma.

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### Adult Hematology Abstract Categories

Myeloma

PP 21

### CASE REPORT: PLASMA CELL LEUKEMIA IN A PATIENT WITH CHRONIC LYMPHOCYTIC LEUKEMIA

Beyza Oluk<sup>1,\*</sup>, Hüseyin Çiftlik<sup>2</sup>,  
İlknur Kozanoğlu<sup>3</sup>, Fatih Kula<sup>4</sup>

<sup>1</sup> Kocaeli City Hospital, Department of Hematology

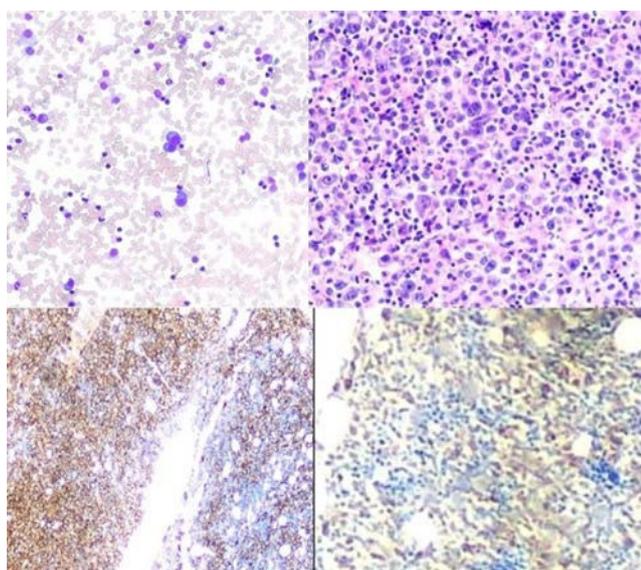
<sup>2</sup> Kocaeli City Hospital, Internal

<sup>3</sup> Acibadem Labmed Clinic Laboratory, Department of  
Hematology

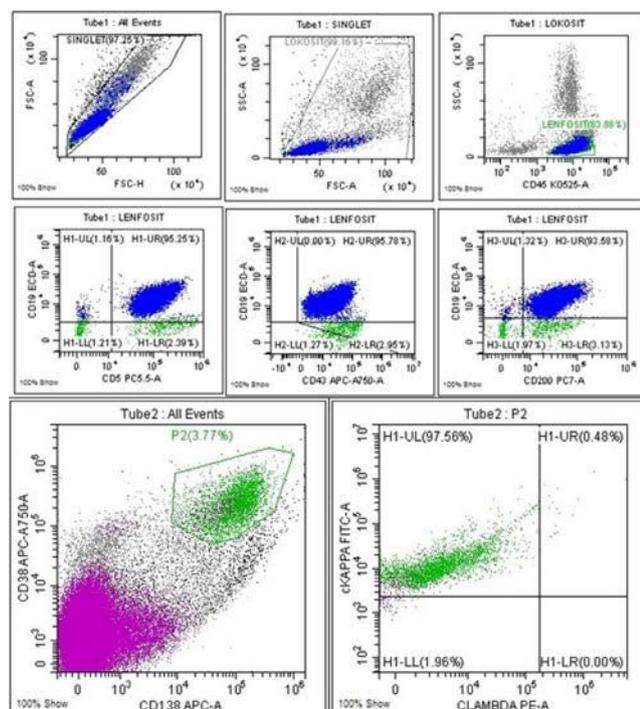
<sup>4</sup> Kocaeli City Hospital, Department of Pathology

**Objective:** Plasma cell leukemia (PCL) is a rare and highly aggressive plasma cell neoplasm that develops in 0.5% to 4% of patients with multiple myeloma (MM). In the diagnostic criteria updated in 2021, the circulating plasma cell rate, which is 20%, is defined as 5% or more. Plasma cell neoplasms originate from post-germinal center B cells and share many biological features with other B-cell lymphoproliferative diseases. Rarely, it can occur simultaneously with some indolent B-cell lymphomas, which may provide insight into common disease-initiating events and genetic changes. In this article, we present a case of primary plasma cell leukemia that presented with acute tumor lysis syndrome in a patient initially diagnosed with chronic lymphocytic leukemia. **Case Report:** A 74-year-old male with RAI Stage 1 Chronic Lymphocytic Leukemia (CLL), previously managed without therapy for the past 3 years, presented with fever, weakness, and elevated white blood cell counts over the past month. Initial laboratory tests revealed anemia (Hb 9.3 g/dL), elevated WBC ( $52 \times 10^3/\mu\text{L}$ ), renal impairment (creatinine 2.5 mg/dL), elevated uric acid (12 mg/dL), and elevated LDH levels. The patient was diagnosed with tumor lysis syndrome and began treatment with intravenous hydration and allopurinole. Peripheral blood smear showed an increase in mature lymphocytes, smudge cells, and plasma cells. Serum protein electrophoresis detected 0.5 g/dL of M-protein, and immunofixation identified a monoclonal IgG kappa band. Bone marrow aspiration revealed two morphologically distinct populations of lymphocytes and plasma cells. Flow cytometry demonstrated a B cell population positive for CD5 and CD19 with kappa light chain restriction, and an increased number of clonal plasma cells (CD38+ CD138+ CD19+ CD45+) with kappa light chain dominance. Bone marrow biopsy confirmed the presence of 85% plasma cells positive for CD138, with kappa monoclonality. FISH analysis was negative for p53 deletion and t(11;14) translocation. Despite initiating anti-myeloma therapy, the patient's condition rapidly deteriorated. The patient was ultimately diagnosed with Stage 1 CLL complicated by plasma cell leukemia but succumbed to respiratory failure. **Conclusion:** Plasma cell leukemia is a disease characterized by abnormal, aggressive plasma cells, while CLL involves malignant mature B-

cell lymphocytes. Although it is extremely rare for both conditions to occur simultaneously, it is important for clinicians to carefully evaluate patients, as both cell types originate from the same multipotent stem cells. Multiparametric flow cytometry of bone marrow samples can aid in the accurate and timely diagnosis of such cases. Key questions have arisen regarding whether B-cell CLL and multiple myeloma originate from a single clone or from two distinct clones appearing simultaneously. Previous studies have utilized various techniques, such as FISH or immunoglobulin gene rearrangement analysis, to explore this issue.



a-b Bone marrow aspirate and biopsy showing two morphologically distinct populations of lymphocytes & plasma cells, many immature. c Immunohistochemical stain on bone biopsy showing plasma cell positive for CD130. d Plasma cells positive for kappa light chains



<https://doi.org/10.1016/j.htct.2024.11.049>

PP 22

## LONG-ACTING ZOLEDRONIC ACID: ONCE-YEARLY ADMINISTRATION AND EFFICACY EVALUATION IN MYELOMA BONE DISEASE

Murat Çınarsoy<sup>1,\*</sup>

<sup>1</sup> Şanlıurfa Mehmet Akif İnan Training and Research Hospital, Clinic of Hematology

**Objective:** The objective of this study was to investigate the preventive effect of long-acting zoledronic acid on the development of new vertebral fractures in multiple myeloma patients with osteoporosis and/or vertebral fractures. **Case Report:** It is observed that osteolytic lesions in multiple myeloma patients lead to skeletal-related events (SRE), which result in a deterioration in quality of life and a shortened life span. It is estimated that up to 80% of all myeloma patients will experience a skeletal-related event. Although surgical and radiotherapy treatments may be required in specific cases, the most effective approach to avoid recurrence of SREs is to implement preventative measures. The current guidelines for the treatment of myeloma recommend the initiation of bisphosphonate therapy for all patients who meet one of the following criteria: 1. those with osteolytic bone disease, 2. those without bone disease but with symptoms, 3. those with osteoporosis. It is recommended that zoledronate be administered on a monthly basis for a minimum of 12 months. In terms of the length of treatment, it is indicated that the treatment interval can be extended to once every three months or discontinued in patients who have achieved a VGPR or above in response to myeloma treatment. Zoledronate is available in two different forms as 4 mg and 5 mg. Once-yearly administration of the 5 mg form is indicated for patients with osteoporosis and long-term steroid use. However, there is currently no data supporting the use of the 5 mg form in patients with myeloma. **Methodology:** The Zoledronate 5 mg formulation was administered parenterally, in a 250 cc isotonic solution for a period of 30 minutes, in patients who fulfilled the requisite study criteria. Patients were monitored for any fracture symptoms and side effects related to the administration of zoledronate at each visit to our clinic for myeloma treatment. In cases where a suspected fracture was identified, an MRI assessment was scheduled to be conducted on the relevant area. MRI scan of the spine and pelvis was conducted to assess the effectiveness of the zoledronate treatment at the six-month mark. **Results:** The results of the evaluation at six months were available for 16 of the 18 patients. Two patients were excluded from the study due to non-attendance at scheduled control visits and a decision to cease myeloma treatment. All 16 patients underwent a vertebral and pelvic MRI evaluation at the six-month mark. Bone fracture symptoms and biochemical values were assessed at each treatment visit. During the follow-up period, none of the patients reported any symptoms suggestive of new bone fractures. There were no instances of hypocalcaemia, renal dysfunction or albuminuria due to zoledronic acid administration. However, one patient did develop jaw osteonecrosis as a result of dental intervention in the fourth month of zoledronic acid administration. At the six-month MRI examination, none

of the patients had developed new fractures. **Conclusion:** 1. The 5 mg formulation of zoledronate has been proven to prevent the development of new vertebral fractures or the recurrence of fractures in all myeloma patients, regardless of whether they have a fracture or osteoporosis. 2. In addition to its efficacy, this application eliminates the shortcomings associated with the aforementioned treatment regimen. With a single administration at the time of diagnosis, compliance is greatly enhanced. 3. From a financial perspective, this has a notable impact on the cost of treatment. In Turkey, the lowest monthly price for a 4 mg dose of zoledronate is 884 TL. If the treatment is administered monthly for 12 months, the total cost is 10,608 TL. The cost of a box of denosumab is 4788 TL, with a total treatment cost of 57,456 TL if applied once a month for 12 months. The cost of a box of zoledronate 5 mg is 898 TL, reflecting the annual application frequency. In accordance with the recommendations set forth by the IMWG guideline, the treatment cost of the zoledronate 5 mg formulation is 11 times less expensive than that of the zoledronate 4 mg formulation and 63 times less expensive than that of denosumab, based on a one-year application period. 4. It is recommended that all myeloma patients, with or without osteolytic bone disease, be evaluated for osteoporosis. There is no clear recommendation in this direction in the guidelines. 5. If we add secondary osteoporosis, glucocorticoid use and previous fracture to the FRAX score, we see that all patients are at very high risk of major osteoporotic fracture and hip fracture. This shows that we need to raise awareness in this area.

<https://doi.org/10.1016/j.htct.2024.11.050>

## Adult Hematology Abstract Categories

### Stem Cell Transplant

PP 23

#### PRESENTATION OF 4 CASES OF AUTOLOGOUS HEMATOPOIETIC STEM CELL TRANSPLANTATION AFTER HIGH-DOSE CHEMOTHERAPY WITH REFRACTORY SOLID TUMOR DIAGNOSIS

Muhammed Murati<sup>1,\*</sup>, Yakup Ünsal<sup>1</sup>, Güler Delibalta<sup>1</sup>, Serdar Bedii Omay<sup>1</sup>

<sup>1</sup>Emsey Hospital

**Case Report:** Hematopoietic stem cell transplantation (HSCT) is a treatment method that can provide cure for most hematological malignant diseases. In addition to hematological malignancy, HCT is also used as a treatment method in benign hematological diseases, solid tumors, and autoimmune diseases. Autologous hematopoietic stem cell transplantation (AHCT) is the most common procedure performed in solid tumors. Transplantation is performed first as high-dose chemotherapy (HDC) and then as OHCT. In our transplant center between 2021 and 2023, we evaluated data of high-dose chemotherapy (HDC) and OHCT. Our first case is a 43-year-old female patient who received multiple treatments

with the diagnosis of refractory primary peritoneal adenocarcinoma. Our second case is a 22-year-old male neuroblastoma patient who was first diagnosed with a retroperitoneal mass. Our third case is a 27-year-old male patient diagnosed with refractory Ewing Sarcoma. Our fourth patient is a 29-year-old male, who was diagnosed with refractory testicular cancer and to whom we performed a transplant.

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PP 24

#### DOES BMI/BSA AFFECT STEM CELL MOBILISATION?: SINGLE CENTRE EXPERIENCE

Seda Yılmaz<sup>1,\*</sup>, Ayşe Günay<sup>2</sup>, Salih Cırık<sup>1</sup>, Abdulkadir Baştürk<sup>1</sup>

<sup>1</sup>Konya City Hospital, Clinic of Hematology

<sup>2</sup>Konya City Hospital, Clinic Pharmacy Unit

**Objective:** Haematopoietic stem cell transplantation is accepted as an important treatment strategy in the treatment of many haematological diseases including acute leukaemia, lymphoma, multiple myeloma as well as sickle cell anaemia and beta thalassaemia major. BMI is an important factor affecting the donor's response to mobilisation and thus haematopoietic progenitor cell yield. This effect is thought to be due to the relatively high dose of filgrastim administered to donors with higher BMI or to the presence of unknown intrinsic factors affecting mobilisation related to the amount of adipose tissue in each donor. In studies examining the relationship between obesity and CD34, negative effects of BMI on the number of progenitor cells have been shown. **Methodology:** A total of 41 patients, including 32 patients and 9 healthy donors, who underwent stem cell mobilisation for bone marrow transplantation in the therapeutic apheresis unit of Konya City Hospital between 10/2023 and 8/2024 were included in our study. The effects of disease diagnosis, age, number and content of chemotherapy, radiotherapy history, body surface area (BSA), body mass index (BMI), chronic habits such as smoking and alcohol, comorbidity and vitamin D level on stem cell mobilisation were investigated. **Results:** In our study, data of 9 healthy donors, 21 multiple myeloma and 11 lymphoma patients were analysed. Median age was 61 (18-72) years, 46.3% (19) were female and 53.7% (22) were male. There was a history of radiotherapy in 9.8% of the patients. While 46.3% of the patients were mobilised with cyclophosphamide+filgrastim, 41.5% with filgrastim, 4.9% with other chemotherapeutic agents+ filgrastim, 4.9% with filgrastim+plerixafor, 2.4% of the patients had stem cell collection by harvest procedure. On day 1 of stem cell mobilisation, there was no difference between those who collected sufficient CD34 positive stem cells and those who failed in terms of gender, height, weight, BMI, BSA, chronic habits, presence of comorbidities, vitamin D level and number of chemotherapy received. There was no statistically significant correlation between the total amount of CD34 positive stem cells and gender, height, weight, BMI, BSA, chronic habits, presence of comorbidities, vitamin D level and number of received

chemotherapy. A negative, strong and statistically significant correlation was found between the number of CD34 positive stem cells and BMI in multiple myeloma patients ( $\rho$ : -0.705  $p$ <0.001). **Conclusion:** Hematopoietic stem cell transplantation used in the treatment of many haematological disorders has become the gold standard treatment. Therefore, the factors affecting the success of transplantation have been the subject of research, and the effects of factors such as BMI, vitamin D, and gender have been investigated. In a cohort of 149 volunteers participating in a weight loss programme, the absolute number of CD34 positive progenitor cells and VEGF receptor-2, CD133 and CD117 positive cell subtypes decreased in relation with increasing BMI and waist circumference. Weight loss caused an increase in CD34 and CD117/CD34 cell counts. In our study, it was shown that high BMI in multiple myeloma patients caused lower CD34 levels in the cell collection process. We believe that it would be useful to perform this analysis with a larger patient population.

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#### Adult Hematology Abstract Categories

##### Transfusion Medicine and Apheresis

PP 25

#### EVALUATION OF IRON ACCUMULATION DURING CHILDHOOD CANCER TREATMENT

Şule Çalışkan Kamış<sup>1,\*</sup>, Metin Çil<sup>1</sup>, Begül Yağcı<sup>1</sup>, Barbaros Şahin Karagün<sup>1</sup>

<sup>1</sup> Adana City Training and Research Hospital

**Objective:** Iron overload is a major concern in pediatric oncology, particularly with frequent blood transfusions. Although serum ferritin levels are commonly used as a marker, cardiac and hepatic T2\* MRI is the gold standard for accurate assessment. This study aimed to evaluate the relationship between serum ferritin levels and T2\* MRI values in pediatric cancer patients, focusing on cases with ferritin levels exceeding 1000 mcg/L. **Methodology:** This prospective study included pediatric patients aged 10-25 diagnosed with malignancies at Adana City Training and Research Hospital from June 2023 to December 2024. Ferritin and C-reactive protein (CRP) levels were measured during non-infectious periods. Elevated ferritin was confirmed if CRP was also raised. Data on transfusions and ferritin levels were collected at 3, 6, and 12 months post-diagnosis. Patients with ferritin levels above 1000 mcg/L underwent cardiac and hepatic T2\* MRI to assess the need for iron chelation therapy. **Results:** A total of 28 patients (median age: 14 years) were analyzed, with 12 females and 16 males. The median ferritin level at diagnosis was 32.5 mcg/L. Significant associations were found between transfusion frequency and ferritin levels over 1000 mcg/L within 3 months ( $p=0.029$ ) and annually ( $p=0.001$ ). Three patients had ferritin levels above 1000 mcg/L: two with acute lymphoblastic leukemia (ALL) and one with non-Hodgkin lymphoma (NHL). One patient died, another received a bone marrow transplant, and the third had normal cardiac but moderate hepatic iron levels. In one case, ferritin dropped below 1000 mcg/L without

chelation by 12 months. Elevated ferritin in the transplant patient was likely related to the procedure. **Conclusion:** Iron overload is a significant challenge in pediatric cancer, particularly during transplants. Early monitoring and timely chelation can help manage this risk. Future research should focus on optimizing iron management strategies in this vulnerable population.

<https://doi.org/10.1016/j.htct.2024.11.053>

#### Adult Hematology Abstract Categories

##### Other Diseases

PP 26

#### IMMUNE THROMBOCYTOPENIA WITH EPSTEIN-BARR VIRUS-ASSOCIATED INFLAMMATORY PSEUDOTUMOR OF THE SPLEEN

Ulviyya Hasanzade<sup>1,\*</sup>, Metban Mastanzade<sup>1</sup>, Kürşat Rahmi Serin<sup>2</sup>, Gorkem Uzunyolcu<sup>2</sup>, Mehmet SemihÇakır<sup>3</sup>, Ali Yılmaz Altay<sup>4</sup>, Gulcin Yeğen<sup>4</sup>, Sevgi Kalayoglu Beşışık<sup>1</sup>

<sup>1</sup> Istanbul University, Istanbul Faculty of Medicine, Department of Internal Medicine, Division of Hematology

<sup>2</sup> Istanbul University, Istanbul Faculty of Medicine, Department of General Surgery

<sup>3</sup> Istanbul University, Istanbul Faculty of Medicine, Department of Radiology

<sup>4</sup> Istanbul University, Istanbul Faculty of Medicine, Department of Medical Pathology

**Objective:** Introduction: Inflammatory pseudotumors (IPTs) are rare and may occur in various anatomic sites. Splenic IPTs are extremely rare, often associated with Epstein–Barr virus (EBV) and have a low-malignant potential with recurrences. The tumor showed a mixed inflammatory infiltrate with spindle cells focally composed of follicular dendritic cell (FDC) proliferations. It can mimic hematopoietic diseases as mostly with solitary mass lesion, but can also be discovered incidentally. **Case Report:** A 64-year-old male patient, admitted to the general surgery department with complaints of hematochezia. He had severe thrombocytopenia ( $2. \times 10^9/L$ ) with mild increased leukocyte count ( $12.270 \times 10^9/L$ ). Endoscopic evaluation of gastrointestinal did not reveal any significant abnormality. Abdominal tomography showed a splenic mass lesion sized of  $40 \times 37$ mm. On MRI the lesion was mildly hypointense on T2-weighted images, not visible on T1-weighted images, and demonstrated progressive peripheral contrast enhancement in dynamic post-contrast series. Bone marrow biopsy showed no hematopoietic disease. A diagnostic splenectomy was decided. Prednisone (1.0 mg/kg/day) was started with a possible diagnosis as immune thrombocytopenia which resulted a significant response and the patients was vaccinated according to the splenectomy vaccination guideline. With a platelet count of  $450. \times 10^9/L$  he underwent splenectomy. Spleen specimen showed a nodular lesion.

Histologic evaluation revealed polytypic lymphoplasmacytic infiltration with focal spindle-shaped cells which were found to be EBER positive. EBV-associated IPT was diagnosed. The patient had no post-operative complaints, and one month after surgery, the platelet count was  $386,000 \times 10^9/\text{ml}$  with no recurrence of thrombocytopenia. Serum EBV-DNA results remained negative before and after diagnosis. **Discussion:** The IPTs of the spleen can develop either via proliferation of **myofibroblasts** or FDC that may be infected by EBV. They may be discovered by investigation of another disorder similar to our case as ITP, leukemoid reaction or hypercalcemia. Total resection of the tumor results in general improvement.

<https://doi.org/10.1016/j.htct.2024.11.054>

#### PP 27

##### CHOROID PLEXUS CARCINOMA AND CHOROID PLEXUS PAPILLOMA; RARE CASES

Şule Çalışkan Kamış<sup>1,\*</sup>, Begül Yağcı<sup>1</sup>,  
Barbaros Şahin Karagün<sup>1</sup>

<sup>1</sup>Adana City Training and Research Hospital

**Case Report:** Choroid plexus carcinoma (CPC) is a rare and aggressive intracranial neoplasm, constituting 1–4% of all brain tumors and approximately 40% of choroid plexus tumors. Classified as a WHO Grade III malignancy, CPC is characterized by a poor prognosis, with reported 5-year survival rates around 40%. In contrast, choroid plexus papilloma (CPP), classified as a WHO Grade I tumor, is a benign and slow-growing lesion originating from the epithelial cells of the choroid plexus. This report presents four cases of choroid plexus tumors: two diagnosed as choroid plexus carcinoma (WHO Grade III) and two as choroid plexus papillomas (WHO Grade I). The CPP cases were managed with observation and followed up without active treatment. Among the CPC cases, a 3-year-old patient received initial radiotherapy followed by chemotherapy based on the CPT-SIOP-2000 protocol. A 7-month-old patient with CPC was treated with chemotherapy (CPT-SIOP-2000 protocol), while radiotherapy was deferred due to her age of less than 3 years. Multidisciplinary treatment strategies for CPC include maximal surgical resection followed by chemotherapy and radiotherapy. The CPT-SIOP-2000 study has demonstrated that the Carboplatin/Etoposide/Vincristine (CarbEV) chemotherapy protocol is effective in treating CPC.

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#### PP 28

##### CLINICAL AND GENETIC FEATURES IN CONGENITAL GLYCOSATION DEFECTS PRESENTING WITH HEREDITARY HEMOLYTIC ANEMIA AND PROLONGED JAUNDICE

Hüseyin Avni Solgun<sup>1,\*</sup>, Mustafa Özyay<sup>1</sup>

<sup>1</sup>Gaziantep City Hospital

**Objective:** Congenital glycosylation disorders (CGD) are a large group of genetic diseases that occur due to a decrease or increase in the glycosylation of glycoconjugates. Congenital glycosylation disorders; They can be grouped under 4 groups: protein N-glycosylation, protein O-glycosylation, combined N- and O-glycosylation and lipid glycosylation disorders. Congenital glycosylation disorders are divided into 2 main groups: Type I and II (CGB-1 and GB-2). In this article, we would like to present a cases of CGB with an atypical presentation, presenting clinical findings with hemolytic anemia and prolonged jaundice, and diagnosed by clinical exon panel genetic study, since it is very rare in the literature. **Case Report:** Our first patient, H1, was a 6-month-old male infant who received erythrocyte transfusion at an external center at the age of 14 days due to jaundice and anemia during the neonatal period (when HB: 5 g/dl), and then applied to the pediatric hematology clinic of our hospital with the same complaints at the age of 43 days. As a result of molecular tests, he was diagnosed with CGD type 2. Our other patient, H2, is a 10-year-old male, our third patient, H3, is a 13-year-old male, and our last patient, H4, is a 17-year-old male; These 3 patients were siblings. All three of them were hospitalized at an external center with jaundice and anemia during the neonatal period, but after diagnostic genetic tests, H4 was diagnosed after 3 years of age, but the other siblings were diagnosed after 6 months of age due to the oldest sibling's history. C.657c>A homozygous mutation was detected in the GSS gene in these siblings. **Methodology:** The diagnostic difficulties and treatment options of 4 patients (H1, H2, H3, H4), who received inpatient treatment with anemia and jaundice in the pediatric hematology clinic between 2022 and 2024 and were ultimately diagnosed with CGD, were obtained from the hospital information processing system and presented because they are very rare in the literature. **Results:** Our first patient, H1, was a 6-month-old male infant who received erythrocyte transfusion at an external center at the age of 14 days due to jaundice and anemia during the neonatal period (when HB: 5 g/dl), and then applied to the pediatric hematology clinic of our hospital with the same complaints at the age of 43 days. As a result of molecular tests, he was diagnosed with CGD type 2. Our other patient, H2, is a 10-year-old male, our third patient, H3, is a 13-year-old male, and our last patient, H4, is a 17-year-old male; These 3 patients were siblings. All three of them were hospitalized at an external center with jaundice and anemia during the neonatal period, but after diagnostic genetic tests, H4 was diagnosed after 3 years of age, but the other siblings were diagnosed after 6 months of age due to the oldest sibling's history. C.657c>A homozygous mutation was detected in the GSS gene in these siblings. **Conclusion:** Although prolonged jaundice and anemia are quite common, we wanted to emphasize with this very unique study that metabolic diseases may be among the differential diagnoses that are very rare in the literature. CGD has been diagnosed in only 40 cases in the last 30 years; Diagnostic evaluation with genetic consultation is very important for diagnosis. Literature data on rare diseases will be strengthened with new studies.

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## PP 29

## A RARE CASE OF PANCREATOBLASTOMA IN A PEDIATRIC PATIENT

Şule Çalışkan Kamış<sup>1</sup>, Defne Ay Tuncel<sup>1</sup><sup>1</sup> Adana City Training and Research Hospital

**Case Report:** Pancreatoblastoma (PB) is a rare malignant neoplasm. PB is frequently detected in children under 10 years of age. Symptoms are nonspecific. When diagnosed, most tumors are enlarged (> 5 cm). Abdominal pain is often the first complaint (44%). Alpha-fetoprotein (AFP) levels are high. Provides long-term survival with surgical resection. It has been reported that the prognosis is poor if metastases are detected. Here we present a seven-year-old female PB case. She applied with the complaint of abdominal pain. On physical examination, a mass was palpated in the epigastric region. The changed laboratory findings were an increased serum AFP level of 171.1 micrograms/L (normal range 0-9 micrograms/L). Abdominal computed tomography (CT) examination revealed a solitary mass of approximately 6 × 4 cm in the tail of the pancreas. Multiple mass lesions were observed in the liver. These lesions were evaluated as compatible with metastasis. She was diagnosed with PB histopathologically after Tru-cut biopsy. Pathologically increased Fluorodeoxyglucose (FDG) uptake (SUVmax: 9.99) was observed in the mass lesion around the right upper quadrant gastric corpus in F-18-Fluorodeoxyglucose Positron Emission Tomography/Computed Tomography (<sup>18</sup>F-FDG PET/CT). Malignant hypermetabolic metastatic multiple hypodense mass lesions (SUVmax:7.7) were seen in the liver. OPEC chemotherapy was given. In the evaluation performed after 5 cycles of chemotherapy, a decrease in FDG uptake <sup>18</sup>F-FDG PET/CT was detected. The patient was evaluated as responsive to treatment. This case report may contribute to the literature with its rarity and treatment approach.

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## PP 30

## RPL5 NOVEL MUTATION IN A PATIENT WITH DIAMOND BLACKFAN ANEMIA

Metin Çil\*

Adana City Training and Research Hospital

**Case Report:** Diamond-Blackfan anemia (DBA) is a rare inherited disorder characterized by macrocytic anemia, congenital malformations, and growth retardation, typically presenting in the first year of life. RPL5 encodes a component of the 60S ribosomal subunit, and mutations in this gene are associated with DBA, which is usually inherited in an autosomal dominant. Our case is presented after identifying a novel mutation that lacks the typical phenotypic features associated with the condition. A 17-month-old female patient was sent to our hospital because she was pale and had anemia. During the physical examination, the patient exhibited growth and developmental retardation (height in the 3rd to 10th

percentile, weight in the 3rd percentile) and a pointed nose; however, no organomegaly or congenital malformations were detected. Laboratory results showed a hemoglobin level of 4.9 g/dL, an MCV of 90 fL, and a corrected reticulocyte count of 0.8%. HbF level in hemoglobin electrophoresis was 3.5%. Bone marrow examination revealed severe hypoplasia in the erythroid series. Genetic examination using next-generation sequencing detected a novel mutation in the RPL5 gene c. 10G>C (p. Val4Leu) (Heterozygous). Although RPL5 mutations show more severe phenotypic features in DBA, the new mutation detected in our patient caused anemia and developmental and growth retardation without congenital malformation. This genetic change has not been previously reported in the literature as a novel mutation. However, according to the American College of Medical Genetics and Genomics (ACMG) criteria, this variant has been classified as a "variant of uncertain significance". Given that no additional mutations were identified in the whole exome sequencing (WES) analysis conducted on our patient, the hematological and bone marrow findings were consistent with a diagnosis of DBA. **Methodology:** A blood transfusion was administered to the patient, and steroid treatment was started. Our patient responded to steroid treatment during follow-up. WES analysis was also requested for our patient's mother, father, and sibling. Based on the results, donor screening for bone marrow transplantation will be initiated. Once the results are available, the phenotype-genotype relationship can be interpreted more accurately.

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## PP 31

## HYPEREOSINOPHILIC SYNDROME

Tuba Öztoprak<sup>1</sup>, Harika Shundo<sup>1</sup><sup>1</sup> Bezmialem Foundation University Hospital

**Case Report:** A 58-year-old female patient was referred to the hematology clinic in July for examination after leukocytosis was detected in her tests. She has a known history of CKD, Type 2 DM, HT, hyperlipidemia. The patient's general condition is good, and she had a complaint of numbness in her hands. First 1.5 months ago, numbness in her right hand up to the wrist began, especially severe in the first 3 fingers. The same complaints started in her left hand 1 month ago. Physical examination findings were within normal limits. On sensory examination, there was hypoesthesia in the first 3 fingers of both hands, especially on the right. In the blood tests performed at the time of admission, leukocytes were 34.440 $\mu$ L, neutrophils 8.840 $\mu$ L, eosinophils 20.540 $\mu$ L, absolute lymphocyte count 3.890 $\mu$ L, monocytes 1.080  $\mu$ L, hemoglobin 12 g/dL, platelets 348.000  $\mu$ L, creatinine 1.07 mg/dL, CRP: 56.6 mg/dL, sedimentation - 10 mm/h were measured. The patient has had borderline eosinophilia (1510 $\mu$ L) since 2022. Flow cytometry was performed on peripheral blood. 11% lymphoid series and 89% myeloid series cells were seen. No abnormality was observed in the lymphoid series. A slight regression in maturation was seen in myeloid series cells and

an increase in eosinophilic series cells. Blast ratio was detected as negative. ECHO findings were normal. No pathology was observed in the lung. Diagnostic bone marrow biopsy was performed. EMG revealed sensorimotor demyelination with block at the wrist level in the right median and neuropathy with secondary axonal damage. It was evaluated as CTS. After the biopsy, corticosteroid treatment was started. On the 2nd day of treatment, the patient's eosinophil count was 350  $\mu$ L. She was discharged with oral steroid treatment and discharged with oral steroid. In the control eosinophils decreased to 2160  $\mu$ L. In the pathology report of biopsy, hypereosinophilic syndrome was considered. No diagnostic findings were detected in favor of neoplastic/clonal eosinophil expansion.

<https://doi.org/10.1016/j.htct.2024.11.059>

PP 32

#### A RARE CAUSE OF THROMBOCYTOPENIA: MALARIA

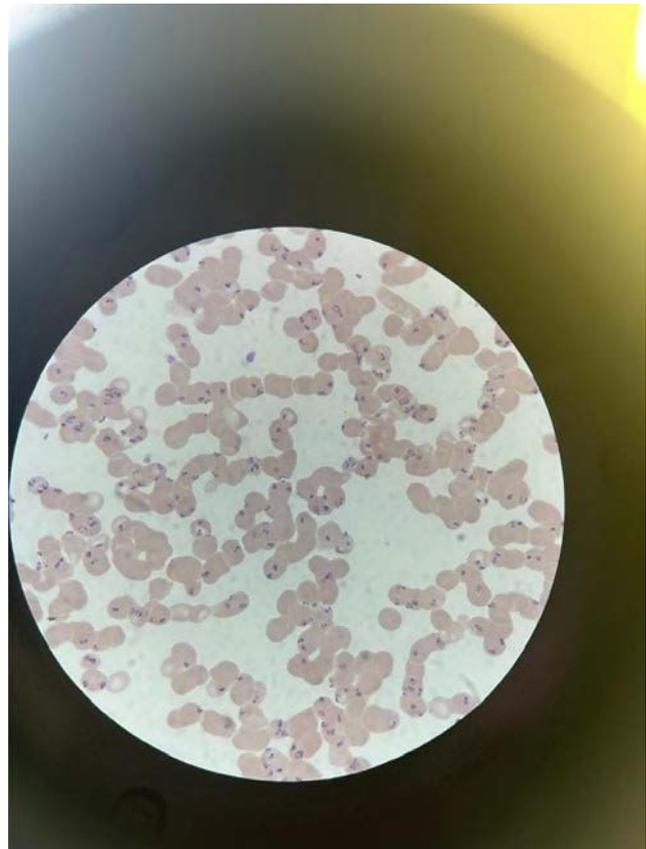
Aslı Odabaşı<sup>1\*</sup>, Düzgün Özatlı<sup>2</sup>

<sup>1</sup>Ordu State Hospital, Department of Hematology

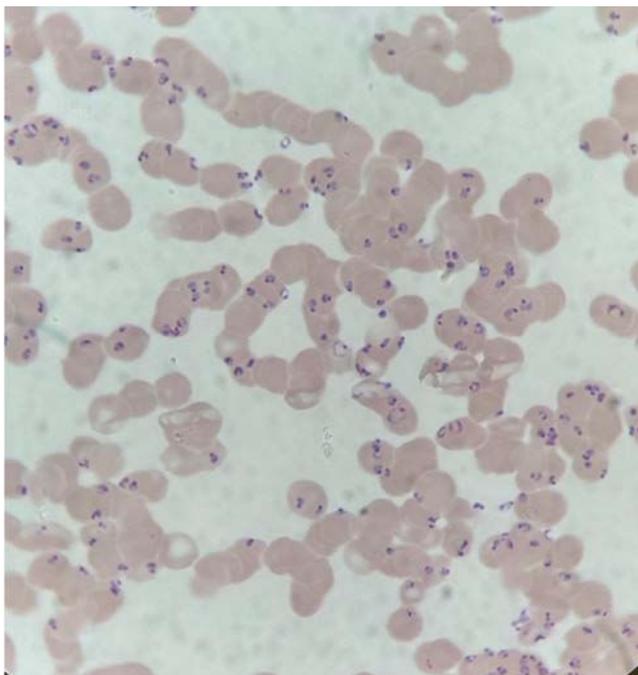
<sup>2</sup>Samsun Ondokuz Mayıs University, Faculty of Medicine, Department of Hematology,

**Objective:** Malaria is a potentially fatal condition caused by parasites that are spread to humans through the bites of infected female Anopheles mosquitoes, according to the World Health Organization (WHO). Two parasite species, *Plasmodium falciparum* and *Plasmodium vivax*, are the most significant threats globally, both known to be infectious to humans. Hematological changes are the most frequent consequences of malaria and have a significant impact on the pathophysiology of the disease. Changes in platelet parameters are considered a hallmark of malaria infection. Often, these changes in malaria infection may be a result of higher levels of parasitemia. Thrombocytopenia is frequently observed in malaria infection. This report presents a case of malaria as a rare cause in a patient investigated for thrombocytopenia. **Case Report:** A 34-year-old male patient with no known medical history presented to the emergency department with complaints of fever, chills, and rigors. Upon admission, his lab results showed wbc: 3,2 thousand/ul, hgb:13.2 gr/dl, neutrophils: 2400, plt:12 thousand/ul, CRP: 232 mg/dl, creatinine: 0.9 mg/dl, AST: 100 u/l, total bilirubin: 2.7 mg/dl, ALT: 66 u/l. The patient was a sailor and had recently returned from the Ecuador Gine region 15 days ago. He had also stayed in Ghana for 40 days prior to that. The patient had taken prophylactic medication for malaria once. Physical examination revealed abdominal tenderness and fever. Peripheral blood smear evaluation revealed widespread ring forms (Figure 1). Following consultation with microbiology, the patient was diagnosed with malaria. The health authority was notified, artemether+lumefantrine medication was procured and the patient was referred to the tertiary care facility. It was later learned that the patient started IV treatment for

malaria, but his condition deteriorated, he was intubated and subsequently expired. **Discussion:** Malaria remains a global public health concern considering the number of cases and death rate worldwide. Changes in platelet parameters are considered a hallmark of malaria infection, and often these changes in malaria infection may be a result of higher levels of parasitemia. Studies have shown that the median platelet count was significantly decreased in adult patients with malaria compared to apparently healthy individuals. Thrombocytopenia is one of the most frequent complications of malaria infection, though it is not a criterion for severe malaria, and it is commonly observed in both *Plasmodium vivax* and *Plasmodium falciparum* malaria. Previous studies have shown a correlation between parasite density and the severity of malaria infection complications. There is uncertainty regarding the degree of platelet parameter changes that occur during malaria infection and the underlying biological mechanisms associated with parasitemia levels. The speculated mechanisms leading to thrombocytopenia include coagulation disturbances, splenomegaly, bone marrow alterations, antibody-mediated platelet destruction, oxidative stress, and the role of platelets as cofactors in triggering severe malaria. There is no clear recommendation for the adequate management of these hematological complications. It is essential to consider thrombocytopenia and changes in platelet parameters in malaria patients. This report also highlights the need for further research on the subject.



Undefined



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PP 33

#### A CASE OF THROMBOTIC THROMBOCYTOPENIC PURPURA RELATED TO MALIGNANCY AND CHEMOTHERAPY

Ebru Kavak Yavuz<sup>1,\*</sup>, Songül Beskisiz Dönen<sup>1</sup>, Etem Özkaya<sup>1</sup>, Esra Pirinççi<sup>1</sup>, Abdullah Karakuş<sup>1</sup>, Orhan Ayyıldız<sup>1</sup>

<sup>1</sup> Dicle University

**Objective:** Thrombotic thrombocytopenic purpura (TTP) is a life-threatening multisystem disease. TTP progresses with Microangiopathic hemolytic anemia (MAHA), fever, thrombocytopenia, neurological symptoms, and renal failure. Due to microangiopathic hemolytic anemia, schistocytes are seen in the peripheral blood smear, resulting in thrombocytopenia. Damage to the brain and kidneys occurs due to microvascular thrombosis, and this is how symptoms appear. In pathogenesis, it is caused by the deficiency of ADAMTS 13 (a disintegrin and metalloproteinase with thrombospondin type 1 motif, member 13), which breaks down the von Willebrand factor (VWF) found in the endothelium into multimers, or the development of antibodies against it. Due to ADAMTS 13 deficiency or decrease in its activity, VWF cannot be separated into small pieces and is arranged in large pieces in the endothelium, causing widespread intravascular thrombosis. Many factors can be

considered as triggers for the development of TTP, such as pregnancy, malignancy, medications, and autoimmune diseases. **Case Report:** A 59-year-old female patient was admitted due to thrombocytopenia, epileptic seizure, hematemesis and decreased consciousness while being followed up due to cholangiocellular carcinoma. Due to malignancy, 6 cycles of gemcitabine and carboplatin treatment were applied. The last cure was 6 months ago. In followers, WBC  $3.24 \times 10^3/\mu\text{L}$  ( $3.7\text{-}10 \times 10^3/\mu\text{L}$ ), Hbg  $8\text{g/dL}$  ( $12.9\text{-}14.2 \text{g/dL}$ ), MCV  $84 \text{fL}$  ( $81\text{-}96\text{fL}$ ), platelet  $27 \times 10^3/\mu\text{L}$  ( $155\text{-}356 \times 10^3/\mu\text{L}$ ), total bilirubin  $13 \text{mg/dL}$  ( $0.3\text{-}1.2 \text{mg/dL}$ ), indirect bilirubin  $5.36 \text{mg/dL}$  ( $0\text{-}1.5 \text{mg/dL}$ ), LDH  $408 \text{U/L}$  ( $0\text{-}247 \text{U/L}$ ), creatinine  $1.59 \text{mg/dL}$  ( $0.51\text{-}0.95 \text{mg/dL}$ ), protein  $1+$  in full criterion examination, INR  $1.36$ , PT  $15.4 \text{sec}$  ( $10\text{-}15 \text{sec}$ ), APTT  $22.2 \text{sec}$  ( $21\text{-}29 \text{sec}$ ) fibrinogen was  $1.46 \text{g/L}$  ( $1.8\text{-}3.5 \text{g/L}$ ), 3-5 schistocytes were seen in each area in the peripheral smear. Plasmapheresis treatment was started with the preliminary diagnosis of TTP and steroid  $80 \text{mg}$  was given. ADAMTS 13 tests were requested. ADAMTS 13 level is  $3.78\%$  ( $40\%\text{-}130\%$ ) low and ADAMTS 13 inhibitor  $> 80 \text{U/mL}$  ( $<12\text{U/mL}$  negative,  $12\text{-}15 \text{U/mL}$  borderline  $>15\text{U/mL}$  positive), ADAMTS 13 antigen  $<0.011\text{U/mL}$  ( $0.19\text{-}0.81 \text{IU/mL}$ ) was seen. As the patient's thrombocytopenia continued, plasmapheresis was started to be performed twice a day after a week. With this treatment, weekly treatment of medicinal rituximab, which could not be treated with platelets, was arranged. However, the patient did not respond to treatment and died. **Conclusion:** In cancer associated TTP, endothelial cells are damaged due to abnormal angiogenesis and tumor cell invasion, and vWF multimers in the endothelial wall are exposed. In addition, ADAMTS 13 activity decreases due to antibodies formed against ADAMTS 13. Some chemotherapeutics such as mitomycin c, gemcitabine can cause TTP. When a diagnosis of TTP is considered, plasma exchange should be started immediately. In addition to plasma exchange, steroids are given in the treatment and if there is no response, other immunosuppressive treatments are added. Our patient with high ADAMTS 13 inhibitors is a condition that is thought to contribute to the mortality of TTP. In a study, it was observed that low ADAMTS 13 activity, as well as high ADAMTS 13 inhibitor and low ADAMTS antigen, caused an increase in mortality.

<https://doi.org/10.1016/j.htct.2024.11.061>

PP 34

#### A RARE DISEASE ASSOCIATED WITH IGA4, CHARACTERIZED BY SYSTEMIC AMYLOIDOSIS AND LYMPHOPLASMACYTIC CELL DOMINANCE: A CASE PRESENTATION

Şerife Emre Ünsal<sup>1,\*</sup>, Mihriban Yıldırım<sup>1</sup>, Hacı Ahmet Aslaner<sup>1</sup>, Neslihan Mandacı Şanlı<sup>1</sup>, Gülşah Akyol<sup>1</sup>, Muzaffer Keklik<sup>1</sup>, Özlem Canöz<sup>2</sup>, Olgun Konaş<sup>2</sup>, Ali Ünal<sup>1</sup>

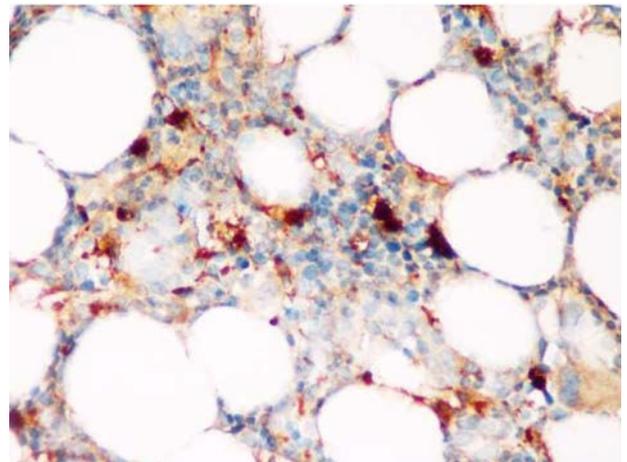
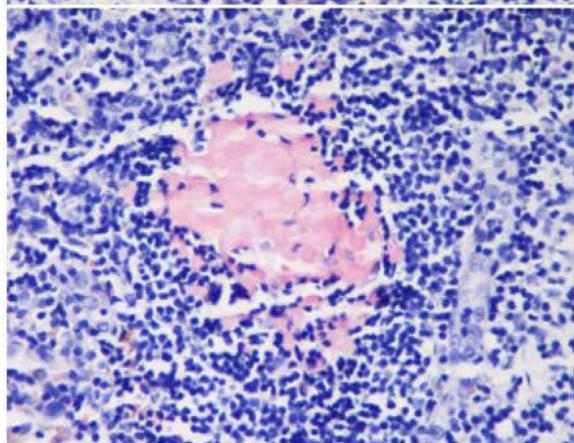
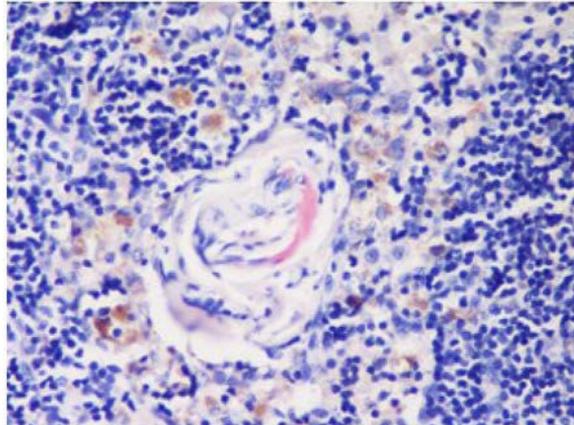
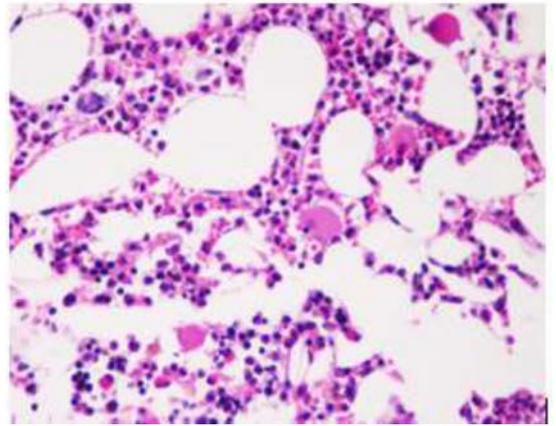
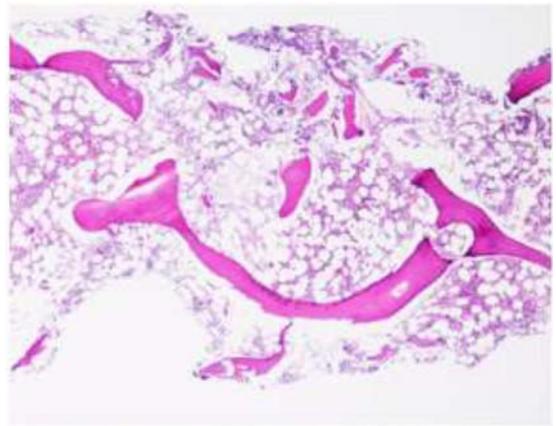
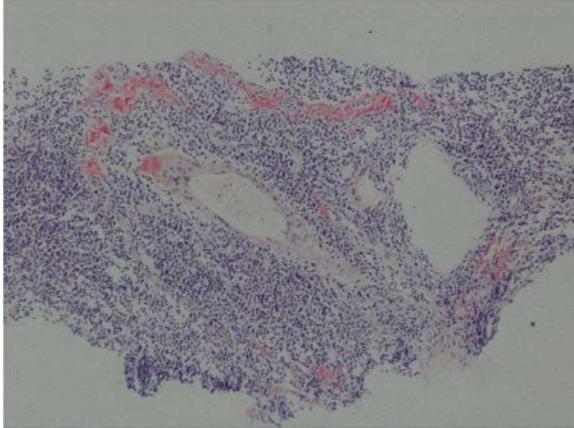
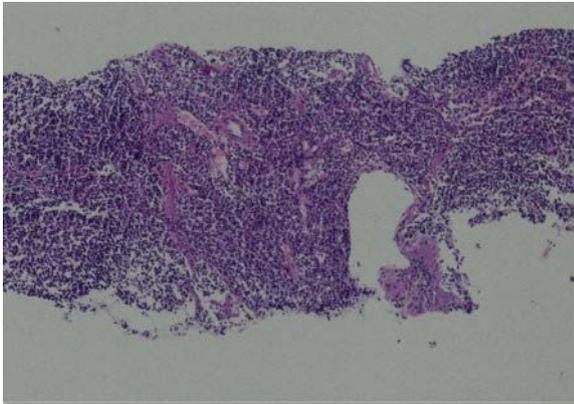
<sup>1</sup>Erciyes University Faculty of Medicine,  
Department of Hematology

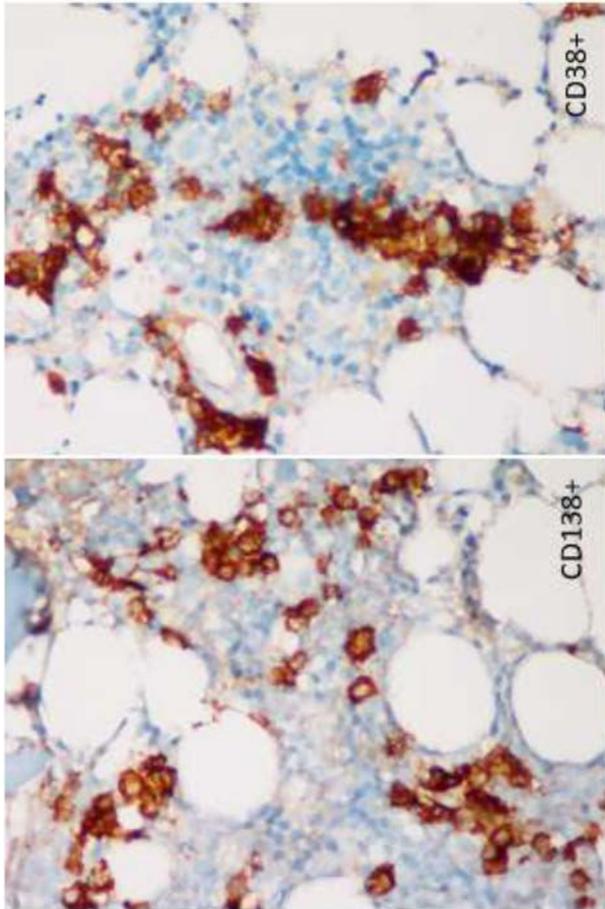
<sup>2</sup>Erciyes University Faculty of Medicine,  
Department of Pathology

**Objective:** Immunoglobulin G4 (IgG4)-related disease has been identified in the last 10-15 years, though it was previously known in the literature under different names as an autoimmune disorder. The spectrum of the disease is quite broad. It can present with involvement of a single organ or multiple organs simultaneously, including autoimmune pancreatitis, Mikulicz syndrome, Küttner tumor (chronic sclerosing sialadenitis), sclerosing cholangitis, and retroperitoneal fibrosis. It most commonly occurs in males over the age of 50. In this case presentation, we will discuss a patient who presented with systemic amyloidosis and was diagnosed with IgG4-related disease. **Case Report:** A 36-year-old male patient presented to the hospital with complaints of abdominal pain and constipation. He was evaluated through detailed anamnesis and physical examination. The patient was found to have iron deficiency anemia and elevated acute phase reactants. An abdominal ultrasound revealed a mass in the epigastric region, leading to admission to the gastroenterology department. A CT scan of the abdomen showed a 62 × 55 mm lesion in the epigastric region. A trucut biopsy was performed, which was reported as amyloidosis. The biopsy revealed an increase in plasma cells. A PET-CT scan identified hypermetabolic lymph nodes in the celiac trunk region. A biopsy taken from these nodes was also reported as amyloidosis, with no evidence of monoclonality. Results showed positivity for CD138, Kappa, Lambda, Congo red, and IgG4, with negativity for HHV8. Serum IgG level was 3256 mg/dL, albumin was 3.59 g/dL, total protein was 8.59 g/dL, sedimentation rate was 65 mm/h, and elevated levels of free kappa and lambda light chains were detected. The patient developed renal failure and hyperkalemia. A renal biopsy showed positive staining for AA amyloid, and a bone marrow biopsy was subsequently performed. The PET-CT scan did not reveal plasmacytoma or osteolytic lesions. The bone marrow biopsy showed 7-8% staining with CD38 and CD138. Positive staining was noted for AA amyloid, IgG, and IgG4, particularly in plasma cells. An initial diagnosis of lymphoplasmacytic lymphoma was considered, and excisional biopsies of lymph nodes were planned. The excisional biopsy of the left axillary lymph node was reported as amyloidosis, leading to a referral to the rheumatology department to investigate secondary causes of amyloidosis. IgG subclasses were tested, revealing an IgG4 level of 700 mg/dL. The patient was started on corticosteroid therapy at a dose of 1 mg/kg. **Conclusion:** IgG4-related disease is a fibro-inflammatory condition that can affect any organ simultaneously or at different times. It is a systemic disease that can involve all organs and often presents with organomegaly, mimicking malignancy. The immunopathogenesis of the disease is not yet fully understood. The most critical step in diagnosis is the histopathological evaluation of the

affected organ. Histopathological features distinguishing the disease include dense lymphoplasmacytic infiltrates with predominance of IgG4-positive plasma cells, storiform fibrosis, and obliterative phlebitis. There are no specific diagnostic tests for IgG4-related disease, making differential diagnosis very important. The first comprehensive diagnostic criteria for IgG4-related disease were established in 2011, and new classification criteria were introduced in 2019. A serum IgG4 level of  $\geq 135$  mg/dL is significant for diagnosis. The primary treatment for IgG4-related disease is corticosteroids, which typically respond well to therapy. Most patients show a response to treatment within 4 weeks. With therapy, patients often experience a reduction in symptoms, a decrease in the size of masses in affected organs, improvement in organ function, and a general decline in serum IgG4 levels over several weeks. After the initial response, the dose should be gradually reduced by 5 mg every 2 weeks to maintain remission, ideally for a duration of 3-6 months at the lowest effective dose. However, relapses can occur, and in cases of resistant or recurrent disease, additional treatments such as rituximab and other immunosuppressive agents may be required. These include azathioprine (2 mg/kg/day), mycophenolate mofetil (1-1.5 g/day), and cyclophosphamide (50-100 mg/day). Biological agents such as infliximab, tocilizumab, calcineurin inhibitors, and bortezomib may be used for refractory cases. Studies evaluating the effectiveness of monoclonal agents like abatacept, inebilizumab, and elotuzumab in the treatment of IgG4-related disease are also available. Early diagnosis and appropriate treatment are crucial for controlling the disease and preventing complications.







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#### PP-35

##### B-LINEAGE PROGENITORS AND CD38-POSITIVE B CELLS ARE ASSOCIATED WITH SURVIVAL RATES IN BREAST CANCER PATIENTS

Svetlana Chulkova<sup>1,2,\*</sup>

<sup>1</sup>FSBU “N.N. Blokhin National medical research center of oncology” of the Russian Ministry of Health, Moscow; Kashyrskoe sh.24, Moscow, 115478, Russia

<sup>2</sup>Pirogov N.I. Russian National Research Medical University of the Russian Ministry of Health; 1, Ostrovitianova st., Moscow, 117997, Russia

**Objective:** The immune system plays an increasingly important role in the development of targeted strategies for breast cancer. According to mRNA sequencing data from The Cancer Genome Atlas (TCGA) high expression B cell signatures has beneficial effects on survival rates in many tumors. Bone marrow (BM) is poorly understood from the point of view of the prognostic role of hematopoietic cells and subpopulations of lymphocytes in patients with breast cancer (BC). **Methodology:** . Study was carried out in 107 BC patients. The immunological and morphological methods were applied.

Multiparameter flow cytometry with antibodies to B-cell populations was used (CD19, CD20, CD5, CD38, CD10, CD45, HLA-DR, CD27), FACSCANTO II. Studies of BM lymphocyte subpopulations were carried out in the gate of CD45++ cells. The duration of the follow-up period after surgery was 8 years. **Results:** The total percentage of B cells in BM was significantly associated with the prognosis of BC. B-1 cells were associated with progression-free and disease-free survival. Disease progression was observed at low levels of B1 cells. In cases more than 10% B-lymphocytes in the BM of BC patients overall survival (OS) rates were more favorable ( $p=0.01$ ). Especially for BC with a high Ki-67. Disease progression was observed in 1/3 of BC patients with low levels of B1 cells. CD38 expression on B cells was a prognostically favorable factor: the role is realized during 5–10 years of follow-up after surgery. Level CD38+ B cells more than 10% correlated with high OS,  $p=0.02$ . The presence of CD10+CD19+ B-lineage precursors was associated with a more favorable prognosis (OS, the threshold level 12%,  $p=0.04$ ). The prognostic role of the CD10 antigen was realized when patients were observed for more than 5 years. **Conclusion:** . Total relative number of (more than 10 %) of BM CD19+ cells were significantly related to OS in BC. B-cell precursors and CD38+ B cells were associated with favorable prognosis. Prognostic role of B-lineage precursors and CD38-positive cells was in the periods of 5–10 years after surgery.

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#### PP 36

##### SUCCESSFUL CHEMOTHERAPY ADMINISTRATION DESPITE HYPERSPLENISM AND PANCYTOPENIA: A CASE OF METASTATIC RECTAL ADENOCARCINOMA

Adil Uğur Kaan Güngör<sup>1,\*</sup>,  
Abdurrahman Aykut<sup>2</sup>, Berksoy Sahin<sup>2</sup>,  
Hatice Asoğlu Rüzgar<sup>2</sup>

<sup>1</sup>Çukurova University, Faculty of Medicine,  
Department of Internal Medicine

<sup>2</sup>Çukurova University, Faculty of Medicine,  
Department of Medical Oncology

**Introduction:** Cytopenias in oncology patients present a significant barrier to the administration of chemotherapy. Hypersplenism is one of the leading causes of cytopenia. In this case report, we aim to present a patient diagnosed with metastatic rectal adenocarcinoma, who developed hypersplenism due to liver metastasis and was successfully treated with chemotherapy despite the cytopenias. **Case Report:** In September 2023, a 42-year-old female patient was diagnosed with rectal adenocarcinoma with liver metastasis. Genetic analysis revealed K-Ras, N-Ras, and BRAF mutant/wild type, MSI stable, and Her2 negative. The patient received 3 cycles of FOLFIRINOX chemotherapy. During follow-up, her hemogram results were as follows: hemoglobin: 8.6 g/dL, platelets:  $26 \times 10^3/\mu\text{L}$ , leukocytes:  $0.81 \times 10^3/\mu\text{L}$ , and neutrophils:  $0.37 \times 10^3/\mu\text{L}$ . PET-CT evaluation showed regression in the metastatic lesions and newly developed splenomegaly

(spleen size: 18 cm). The tumor board assessed the resectability of liver metastases, but surgery was not considered due to the anticipated insufficient remnant liver function, and local ablative therapy was administered. Arterial and venous portal ultrasonography performed to investigate the etiology of the splenomegaly showed normal findings, and no focal lesion was detected in the spleen. No infectious pathology was identified as a cause of the splenomegaly. The cytopenia was attributed to hypersplenism secondary to liver metastasis of rectal cancer. The patient was subsequently treated with 3 additional cycles of FOLFIRINOX and 11 cycles of FOLFOX combined with Bevacizumab. Granulocyte colony-stimulating factor was not administered during the treatment process. The patient remains under oncological follow-up, and chemotherapy treatment is ongoing. **Conclusion:** Splenomegaly and hypersplenism are important causes of pancytopenia. Our clinical experience demonstrated that chemotherapy did not exacerbate cytopenias in a patient with metastatic rectal adenocarcinoma who developed hypersplenism and pancytopenia. We have shown that with close monitoring and supportive care, chemotherapy can be safely administered in patients with pancytopenia due to hypersplenism.

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PP 37

#### COEXISTENCE OF BREAST CANCER AND MANTLE CELL LYMPHOMA

Bengü Sezer<sup>1,\*</sup>, Esra Asarkaya<sup>2</sup>, Tolga Köseci<sup>2</sup>

<sup>1</sup> Cukurova University, Faculty of Medicine, Department of Internal Medicine

<sup>2</sup> Cukurova University, Faculty of Medicine, Department of Medical Oncology

**Introduction:** Patients cured of any cancer have an increased risk of developing a new primary malignancy compared to the general population. However, synchronous presentation of two tumours is a very rare condition. Here we aim to review the treatment approach of a case of synchronous mantle cell lymphoma and invasive ductal carcinoma of the breast. **Case Report:** A 64-year-old woman presented with a right breast mass. Physical examination revealed a 3cm diameter mass lesion in the right breast and lymphadenopathy in the right axilla. Her past medical history was unremarkable except hypertension. In her family history, there was a history of breast cancer in her niece. Breast ultrasonography revealed 3 centimetres (cm) of malignant breast and multiple lymph nodes with thick cortex in bilateral axillae with indistinguishable fatty hilus. Tru-cut biopsy was performed for the mass in the breast and bilateral axilla lymph nodes. The breast biopsy was compatible with invasive ductal carcinoma with ER 90%, PR 10%, her2 negative and Ki67 proliferation index 10%. Bilateral axilla lymph node biopsy was reported as mantle cell lymphoma and immunohistochemically CD20: Positive, CD5: Positive, Cyclin D1: Positive, CD23: Negative, Lef1: Negative, Keratin: Negative, Ki67 proliferation index 25-30%. PET-CT revealed a mass in the right breast, lymph nodes with

pathological appearance in the axillae, various lymph node stations in the abdomen and inguinal areas, and diffuse involvement suggestive of lymphoma infiltration in the right lung. Bone marrow aspiration/biopsy revealed mantle cell lymphoma involvement. The patient was discussed in the multidisciplinary tumour council and right axillary lymph node dissection was performed for staging. 5 lymph nodes showed ductal carcinoma metastasis and the rest of the lymph nodes showed mantle cell lymphoma involvement. Stage IV MHL and hormone positive IDC (T2N2) were detected and R-CHOP treatment was applied. PET-CT performed after three cycles of treatment showed complete response. The patient was discussed again in the multidisciplinary tumour council and surgical treatment for the breast was planned after completing 6 cycles of R-CHOP treatment. After treatment, the patient underwent modified radical mastectomy and the pathological stage was T3N3. After adjuvant RT, endocrine therapy was started and the patient is being followed in remission. **Conclusion:** Coexistence of breast cancer and mantle cell lymphoma is a rare condition. In the few cases reported in the literature, treatment planning was made by considering the stage and treatment priority of both diseases. We planned to prioritise the treatment of lymphoma because our patient had stage 4 mantle cell lymphoma.

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PP 38

#### PRIMARY CONJUNCTIVAL LYMPHOMA, 2 CASES

Günay Süleymanlı<sup>1,\*</sup>,  
Yasemin Aydınalp Camadan<sup>2</sup>, Tuğba Toyran<sup>3</sup>,  
Berksoy Şahin<sup>2</sup>

<sup>1</sup> Cukurova University, Faculty of Medicine, Department of Internal Medicine

<sup>2</sup> Cukurova University, Faculty of Medicine, Department of Medical Oncology

<sup>3</sup> Cukurova University, Faculty of Medicine, Department of Pathology

**Introduction:** Extranodal marginal zone lymphoma (EMZL) is the most common subtype of conjunctival lymphoma. Management of conjunctival lymphoma consists of radiotherapy, surgery, chemotherapy, antibiotics and targeted therapies (Anti-CD 20) based on case series and retrospective studies. Appropriate treatment should be chosen based on the type of lymphoma, extent of spread, and patient-specific factors. We present two patients with localized disease diagnosed with primary conjunctival EZML by biopsy, for whom we planned different treatment plans. **Case Reports: Case 1:** A 64-year-old female patient presented with a pink-red mass on the lateral conjunctiva of her right eye. (Fig. 1A) Conjunctival biopsy was reported as Non-Hodgkin lymphoma, EMZL.(CD 20(+) and Ki-67 3-4%) No extraocular involvement on PET/CT. Orbital MRI showed a 2.5 cm soft tissue lesion surrounding the right globe laterally and posteriorly. The patient started rituximab and

bendamustine treatment, and the lesion in the right orbit was not observed in the current follow-up imaging after 3 cycles of treatment. (Fig. 1B) The patient continued with rituximab and bendamustine treatment. **Case 2:** When the 52-year-old female patient first appeared two years ago, a conjunctival biopsy revealed that she had EMZL. Radiotherapy was recommended for her localized disease, but she declined it. She received eight cycles of rituximab treatment and was monitored in remission. One year later, salmon-colored lesions were found in the inner corner of both eyes. EZML was also found in the new biopsy. There was no ocular involvement. The patient received 6 cycles of rituximab bendamustine and maintenance rituximab for recurrent and bilateral lesions. We are currently monitoring the patient and the disease is in complete remission. **Discussion:** Lymphoma is one of the most frequently occurring malignant tumors of the conjunctiva. In patients with lesions that like a "salmon patch" and unexplained chronic follicular conjunctivitis, lymphoma should be suspected.



(Fig. 1A) (Fig. 1B)

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#### PP 39

##### A RARE CASE: NODAL FOLLICULAR T HELPER CELL LYMPHOMA, ANGIOIMMUNOBLASTIC TYPE

Halil İbrahim Yüksel<sup>1,\*</sup>, Hatice Asoğlu Rüzgar<sup>2</sup>, Mehmet Mutlu Kızı<sup>2</sup>, Berksoy Şahin<sup>2</sup>

<sup>1</sup> Cukurova University, Faculty of Medicine, Department of Internal Medicine

<sup>2</sup> Cukurova University, Faculty of Medicine, Department of Medical Oncology

**Objective:** Angioimmunoblastic T-cell lymphoma (AITL) is the second most common subtype of mature T-cell lymphoma (MTCL). It is caused by monoclonal proliferation of T-follicular helper (TFH) cells. Although advances have been made in its biological knowledge, its treatment is still an unmet medical need. We would like to present a case of Nodal-TFH; AITL that we followed in our clinic. **Case Report:** A 67-year-old male patient presented with cough. Thorax CT revealed left supraclavicular-mediastinal multiple lymphadenopathy with pleural effusion. Supraclavicular LN excision was reported as NHL; nodal follicular T helper cell lymphoma, angioimmunoblastic type. Immunohistochemical CD3, PD-1 and CXC13 were positive, CD4, CD8 and CD10 were sparse, CD21 and 23

were positive in increased dendritic cells, CD20, CD30, EBER and IDH-1 were negative. PET-CT revealed Stage 4BS (multiple LNs with FDG uptake in head-neck, thorax-mediastinum and abdominopelvic FDG uptake, increased FDG uptake in bone marrow-spleen; B symptom: positive). Subcutaneous (sc) Azacitidine + intravenous CHOP (cyclophosphamide, doxorubicin, vincristine, prednisone) was started. The 1st course of azacitidine was administered at 75 mg/m<sup>2</sup> for 7 days 1 week before CHOP treatment and the following courses were administered at 75 mg/m<sup>2</sup> for 14 days 2 weeks before CHOP treatment. After 4 cycles of Azacitidine+CHOP, PET-CT regressed and 2 more cycles of treatment were administered. During the follow-up, the patient's general condition deteriorated and he went into septic shock. **Discussion:** AITL-containing T-follicular helper; nodal PTCL is characterized by recurrent mutations affecting epigenetic regulators. The association of abnormal DNA methylation with lymphomagenesis provides rationale for the administration of hypomethylating agents. The epigenetic modifier azacitidine, which inhibits DNA methyltransferase, has demonstrated clinical activity alone or in combination in relapsed/refractory PTCL. In a phase-2 clinical trial of 20 patients who experienced oral azacitidine + CHOP as initial treatment for PTCL, CR was 76.5%, 1-year PFS 61.1%, 1-year OS 88.9%. In our case, we added the hypomethylating agent azacitidine to the CHOP protocol and aimed to evaluate the efficacy of this combination in the initial treatment of CD30 negative PTCL.

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#### PP 40

##### SINGLE-CENTER EXPERIENCE IN DIFFUSE LARGE B-CELL LYMPHOMA: PROGNOSTIC VALUE OF DEMOGRAPHIC AND MOLECULAR CHARACTERISTICS

Şehmus Tan<sup>1,\*</sup>, Mehmet Mutlu Kızı<sup>2</sup>, Yasemin Aydınalp Camadan<sup>2</sup>, Ertuğrul Bayram<sup>2</sup>, Berksoy Şahin<sup>2</sup>

<sup>1</sup> Cukurova University, Faculty of Medicine, Department of Medical Oncology

<sup>2</sup> Cukurova University, Faculty of Medicine, Department of Internal Medicine

**Introduction:** Diffuse large B-cell lymphoma (DLBCL) is a heterogeneous hematological malignancy, accounting for approximately 30% of all lymphomas, and is associated with diverse clinical outcomes. The onset of DLBCL typically occurs in the sixth decade of life, with a higher incidence in males. The morphological, clinical, and biological diversity of DLBCL underscores the presence of multiple subtypes, each exhibiting distinct behavior. **Objective:** The objective of this study is to assess the demographic characteristics and clinical outcomes of DLBCL patients, as well as to evaluate the prevalence and prognostic significance of MYC and BCL2 co-expression on survival. **Methodology:** A retrospective study was performed on 51 patients with a confirmed diagnosis of DLBCL. We conducted an analysis of the demographic data

and molecular characteristics of patients diagnosed with diffuse large B-cell lymphoma who underwent R-CHOP therapy and were monitored between 2016 and 2022. The MYC and BCL-2 expression levels in the patients were analyzed using immunohistochemical methods, while their genetic rearrangements were assessed by fluorescence in situ hybridization (FISH) at Çukurova University Faculty of Medicine Hospital. **Results:** The median age at diagnosis was approximately 55 years, with a predominance of female patients. The cervical region was the most frequent nodal site of the primary tumor, whereas the stomach represented the most common extranodal site. The majority of patients were diagnosed at Stage III. MYC/BCL2 protein co-expression was identified in approximately 27% of DLBCL cases and was significantly associated with poorer overall survival and progression-free survival compared to cases lacking co-expression. MYC/BCL2 double-hit cases were detected in approximately 2.5% of the total cases. **Conclusion:** MYC and BCL2 co-expression is a significant prognostic marker, correlating with worse survival. Early identification of MYC/BCL2 co-expression could guide personalized treatment strategies for high-risk patients.

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PP 41

#### A RARE CASE REPORT OF ADRENAL GLAND DIFFUSE LARGE B-CELL LYMPHOMA PRESENTING WITH PITUITARY INSUFFICIENCY FINDINGS

Ümmü Gülsüm Uslu<sup>1,\*</sup>, Şuheda ATAŞ İPEK<sup>2</sup>,  
Berksoy ŞAHİN<sup>2</sup>

<sup>1</sup> Çukurova University, Faculty of Medicine,  
Department Of Internal Medicine

<sup>2</sup> Çukurova University, Faculty of Medicine,  
Department Of Medical Oncology

**Objective:** The adrenal glands do not contain lymphoid tissue, and primary adrenal lymphoma (PAL) is extremely rare, accounting for less than 1% of all non-Hodgkin lymphomas and 3% of primary extranodal lymphomas [1, 2]. PAL is primarily bilateral. Approximately 250 cases have been described in the literature to date, with most published articles on PAL being case series with only a limited number of patients. **Case Report:** 74-year-old male patient with known type 2 dm diagnosis, the patient was admitted to our hospital emergency department with complaints of nausea, fatigue, and drowsiness and was followed up in the endocrinology department. laboratory parameters revealed tsh: 0.02 t4: 0.58 Acth: 32.3 Cortisol 7.05 Na: 124 mmol/l K: 4.6 mmol/l. the patient was first given corticosteroids and then levothyroxine replacements in endocrine follow-ups. contrast-enhanced pituitary and brain mris revealed a suspected microadenoma in the left posterior adenohypophysis and suspicious inflammation findings in both optic nerve sheaths. pet ct showed a lesion measuring 41 × 31 mm (suvmax: 19.8) in the right adrenal gland and approximately 40 × 35 mm (suvmax: 21.07) in

the left adrenal gland. low-level increased fdg uptake was observed in the th4 vertebra, l4 vertebra and left femur proximal diaphyseal region. the patient underwent a right adrenal gland biopsy and it was found to be non-hodgkin lymphoma, diffuse large b cell lymphoma (germinal center phenotype) cd 20 +, cd10 +, bcl 6 +, bcl 2+, cmc: 50% +. mild lymphocytosis was observed in the bone marrow aspiration biopsy. DA-R-EPOCH treatment was applied to the patient, who was conscious, oriented, co-operated, general condition and good oral intake under corticosteroid and levothyroxine treatment in the follow-up performance and was externed as no complications were observed. **Discussion:** PAL is extremely rare, primary adrenal DLBCL (PA-DLBCL) is of a non-germinal center B cell (nonGCB) phenotype. PAL usually has no excretory endocrine function and the symptoms are due to the pressure effect of the mass, whereas adrenal insufficiency usually exists. The most common manifestations were B symptoms, which include unexplained fever, weight loss, night sweats (68%), vague abdominal pain (42%), and fatigue (36%), some of which were present in the current patient. There is no correlation between tumor size and adrenal insufficiency. Generally, obvious clinical manifestations of adrenal insufficiency tend to appear when > 90% of the adrenal gland is damaged. It can improve with the destruction of the lymphomatous tissue at the end of the chemotherapy.

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PP 42

#### EXTRANODAL NON-HODGKIN'S LYMPHOMA OF THE ORAL CAVITY: A CASE REPORT

Zeliha Yıldız Kandemir<sup>1,\*</sup>,  
Mustafa Serhan Erayman<sup>1</sup>, Berksoy Şahin<sup>1</sup>

<sup>1</sup> Çukurova University, Faculty of Medicine Training  
and Research Hospital

**Objective:** Lymphomas are indeed complex malignancies with diverse clinical and pathological characteristics. Non-Hodgkin's lymphoma (NHL) is particularly notable for its varying presentations, with a significant number of cases manifesting as lymphadenopathy. The extranodal involvement in about one-third of NHL cases highlights the importance of recognizing atypical presentations. In this case, we present a 59-year-old male patient with non-Hodgkin lymphoma in the right buccal mucosa. **Case Report:** A 59-year-old male patient with a history of allergic asthma and gastroesophageal reflux disease presented to our clinic with swelling in the right maxillary region lasting more than one year. The patient did not have any B symptoms. A biopsy of the right buccal mucosa revealed extranodal marginal zone non-Hodgkin lymphoma. Immunohistochemistry showed: CD20 (+), CD43(+), CD38 positive in plasma cells, diffuse BCL2(+), suboptimal BCL6(+), and a proliferation index of 5% reported with Ki67. An MRI of the orbit demonstrated a mass lesion extending from the right maxillary region into the temporal fossa, with partial external protrusion from the right cheek. After intravenous contrast administration, diffuse

enhancement was observed in the right lateral wall of the sphenoid sinus, which was in close proximity to the right cavernous sinus and caused contrast retention at these levels, extending into the subcutaneous adipose tissue of the right temporal region. The right globe appeared exophthalmic. Simultaneous laboratory parameters were normal, with a beta-2 microglobulin level of 1.65 mg/L and LDH of 180 U/L. An F-18 PET-CT scan showed irregular soft tissue densities in the right maxillary region exhibiting hypermetabolism (primary disease). Several lymph nodes in the right cervical chain showed relative hypermetabolism (possible metastasis). The treatment plan was decided upon in consultation with the ear, nose, and throat and neurosurgery departments regarding potential involvement of the central nervous system. **Discussion:** Non-Hodgkin's lymphomas comprise a varied group of malignancies that primarily affect lymph nodes. Extranodal NHL represents approximately 20-30% of all reported cases. Among the extranodal sites, the head and neck region is the second most frequently involved area, after the gastrointestinal tract. Intraoral non-Hodgkin lymphoma accounts for only 0.1% to 5% of all cases. In summary, our case emphasizes the importance of considering lymphomas in the differential diagnosis of rare malignant lesions in the oral cavity. It is believed that prompt referral for histopathological and immunohistochemical examinations can facilitate early diagnosis and appropriate treatment.

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#### PP 43

##### EXTREME NORMOBLASTOSIS IN A THALASSAEMIA INTERMEDIA PATIENT POST-SPLENECTOMY: THE ROLE OF FLOW CYTOMETRY IN DIAGNOSIS AND MANAGEMENT

İdil Yürekli<sup>1,\*</sup>, Gülçin Dağlıoğlu<sup>2</sup>,  
Naciye Nur Tozluclu<sup>3</sup>, Birol Güvenç<sup>4</sup>

<sup>1</sup> Cukurova University, Faculty of Medicine,  
Department of Anatomy

<sup>2</sup> Cukurova University, Balcalı Hospital Central  
Laboratory, Department of Biochemistry

<sup>3</sup> Cukurova University Medical Faculty Hospital,  
Department of Internal Medicine

<sup>4</sup> Cukurova University Medical Faculty Hospital,  
Department of Internal Medicine, Division of  
Hematology

**Background:** Thalassaemia intermedia is characterized by inefficient red blood cell production (erythropoiesis) and has a wide range of clinical symptoms. Splenectomy, often performed to manage complications, can lead to significant long-term changes in blood cell composition. This case illustrates a striking example of extreme normoblastosis in a patient two decades after a splenectomy. The case also underscores the critical role of flow cytometry in diagnosing blood disorders and differentiating abnormal findings from potential malignancies. **Case Report:** A 45-year-old woman with thalassaemia intermedia, who had her spleen removed at age 25, presented

with severe anaemia, iron overload, and an unusually high normoblast count ranging from 50,000 to 100,000 cells/ $\mu$ L, as seen in a routine complete blood count (CBC). The CBC mistakenly identified the normoblasts as white blood cells, raising concern for possible blood cancer. Closer analysis of the CBC sub-parameters revealed an increased nucleated red blood cell (NRBC) ratio. Further investigation through bone marrow biopsy and flow cytometry was undertaken to rule out malignancy and better understand the extreme normoblastosis. **Methodology:** The diagnostic process involved multiple stages of flow cytometric analysis. First, a chronic lymphocytic leukaemia (CLL) panel was employed, followed by an acute leukaemia panel. Finally, a specialized flow cytometry panel targeting markers such as CD45, CD71, CD41, CD235a, CD19, CD10, CD13, HLA DR, CD36, CD38, and CD117 was used. The gating strategy focused on differentiating erythroid precursor cells based on their size, granularity, and marker expression. **Results:** Flow cytometry identified a significantly elevated population of normoblasts, with these cells displaying low CD45 expression and reduced side scatter. They tested weakly positive for CD71, strongly positive for CD36, and negative for CD235a, confirming their identity as erythroid precursors. Around 70% of the nucleated cells consisted of these normoblasts, representing various stages of erythroid maturation. The absence of lymphoid markers (CD19, CD10, CD5) ruled out lymphoid malignancies, while the exclusion of myeloid malignancies was confirmed through negative results for markers such as CD13, CD33, CD34, CD117, and HLA DR. **Discussion:** This case highlights the occurrence of extreme normoblastosis in a post-splenectomy patient and the challenges in managing such cases. It demonstrated that flow cytometry is essential for accurately identifying erythroid precursors, preventing a misdiagnosis of malignancy based solely on CBC results. The findings underscore the value of flow cytometry in evaluating complex haematological conditions, especially in patients with thalassaemia intermedia after splenectomy. Additionally, the strategic order of tests in the flow cytometry lab, along with collaboration between laboratory and clinical teams, was key to achieving a correct diagnosis. This case reinforces the need for a tailored flow cytometric testing algorithm for complex cases.

**Keywords:** Thalassaemia intermedia, Normoblastosis, Splenectomy, Flow cytometry, Haematological malignancies.

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#### PP 44

##### BLINATUMOMAB BRIDGING THERAPY FOR EFFECTIVE MANAGEMENT OF MRD IN PRO-B ALL WITH CNS INVOLVEMENT: A CASE REPORT OF POST TRANSPLANT PATIENT AT 23 MONTHS AFTER ALLOGENIC HEMATOPOIETIC CELL TRANSPLANTATION

Ceren Dehri Bahşi<sup>1,\*</sup>, Birol Güvenç<sup>2</sup>

<sup>1</sup> Cukurova University, Medical Faculty Hospital,  
Department of Internal Medicine

<sup>2</sup> Cukurova University, Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Objective:** Pro-B ALL is an unusual and highly malignant form of ALL often presenting with CNS involvement. The involvement of the CNS makes the central objective of these treatments that is attaining and maintaining remission more challenging. This is a report of Pro-B ALL of a 52-year old female who had a CNS involvement and received blinatumomab both as bridge to allo -HSCT and post transplantation consolidation for MRD positivity. Case Report This 52 year old female is presented with Pro-B ALL. Standard chemotherapy was complicated by intracranial extension of the disease. The patient was positive for the Philadelphia chromosome with BCR-ABL (9;22) translocation hence dasatinib was added. Intrathecal therapy of blinatumomab was used as well due to infiltration of cytokines in the central nervous system. Following several sessions of treatment, complete remission including of central nervous system was achieved. After all the patient was to receive matched allo-HSCT post which clinical stabilization was ascertained. However bone marrow aspiration, biopsy and flow cytometry showed that there was persistence of MRD. However the patient had blinatumomab as targeted therapy. **Discussion:** This case illustrates the effective use of blinatumomab in managing Pro-B ALL with CNS involvement, particularly in the post-transplant setting. CNS involvement complicates treatment due to the blood-brain barrier, requiring targeted intrathecal therapy alongside systemic chemotherapy. Blinatumomab played a crucial role as a bridging therapy to allo-HSCT and in addressing MRD post-transplant, significantly reducing the risk of relapse. This case demonstrates that blinatumomab can effectively target MRD, even in patients with CNS involvement, contributing to better disease control and outcomes.

**Keywords:** Acute Lymphoblastic Leukemia Pro-B, Central Nervous System Involvement, Blinatumomab, Allogeneic Stem Cell Transplantation, Minimal Residual Disease.

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PP 45

**CARCINOID SYNDROME PRESENTING AS AN ELEVATED 5-HIAA IN A PATIENT EVALUATED FOR AN ELEVATED WBC COUNT: BEWARE OF THE POSSIBLE DIAGNOSTIC DIFFICULTY.**

Bengisu Ece Duman<sup>1,\*</sup>, Bengü Sezer<sup>1</sup>, Birol Güvenç<sup>2</sup>

<sup>1</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine,

<sup>2</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** Carcinoid syndrome is an extremely rare paraneoplastic disorder associated with serotonin-secreting neuroendocrine tumors, which classically present with flushing,

weight loss, hypertension, and gastrointestinal complaints. In fact, symptoms are often nonspecific, and the presentation could promote confusion with hematologic or inflammatory diseases. Early diagnosis is of great importance in allowing proper therapy to avoid delays. **Case Report:** A 45-year-old female was referred to the hematology clinic owing to high WBC count (21,000/ $\mu$ L), associated with fatigue, flushing, and unintentional weight loss of 10 kg over the past 3-4 months. Her history included hypothyroidism on thyroxin and asthma—both on symptomatic medications. Gastroenterology work-ups, including endoscopy, showed mild antral gastritis and a hiatal hernia but no evidence of malignancy. Thus, the imaging studies demonstrated a low-density nodule measuring 1 cm in size on the right adrenal gland, hence the suspicion of a neuroendocrine tumor. Excess serotonin production was confirmed by demonstrating a 24-hour urinary 5-HIAA level of 18.7 mg/day, with a reference range being 2-9 mg/day, compatible with carcinoid syndrome. Confirmatory Ga-68 DOTA-TATE PET-CT revealed moderate increased somatostatin receptor expression in the adrenal lesion. No anemia or other hematologic disorders were observed, despite the initial suspicion of one. **Discussion:** This case highlights carcinoid syndrome as a potential cause of systemic symptoms such as flushing, weight loss, and leukocytosis, even in cases referred for suspected hematologic conditions. Confirmation was based on the elevated level of 5-HIAA and advanced imaging with Ga-68 DOTA-TATE PET-CT. This report emphasizes the need for interdisciplinary collaboration between hematology, endocrinology and oncology for managing complex systemic cases. Early diagnosis of carcinoid syndrome ensures appropriate care, prevents misdiagnosis, and improves outcomes.

**Keywords:** Carcinoid Syndrome, Neuroendocrine Tumor, 5-HIAA, Flushing, Leukocytosis.

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PP 46

**MYCOSIS FUNGOIDES PROGRESSING TO PERIPHERAL T-CELL LYMPHOMA AND THE POTENTIAL ROLE OF ROMIDEPSIN THERAPY**

Bengisu Ece Duman<sup>1,\*</sup>, Ceren Deveci<sup>1</sup>, Birol Güvenç<sup>2</sup>

<sup>1</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine

<sup>2</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** PTCL-NOS is an uncommon and highly aggressive kind of non-Hodgkin lymphoma. Transformation of MF, a cutaneous T-cell lymphoma, into systemic PTCL is infrequent and poses serious challenges both diagnostically and therapeutically. This report describes the challenges in diagnosis and therapy of a transformation case from MF to PTCL which responded to romidepsin. **Case Report:** A 58-year-old male presented to the OPD in the year 2022 with complaints

of chronic itching. Skin biopsy diagnosis was lichen planus. Further skin biopsies done in the year 2023 established mycosis fungoides with patch-stage disease. Thereafter, the disease evolved to involve lymph nodes within a year. Excisional biopsies of these lymph nodes showed dermatopathic lymphadenopathy, which later was transformed into T-cell lymphoid neoplasia indicating transformation into PTCL-NOS. Immunohistochemical analysis showed positivity for CD3+, CD4+, CD7+, GATA3+, and Ki-67 expression. CD30 was negative. In spite of first-line therapies administered, such as photopheresis, methotrexate, and PUVA, the disease further progressed, as indicated in the PET-CT scan with increased metabolic activity in multiple lymph nodes and cutaneous thickening. The patient was initiated with romidepsin—a histone deacetylase inhibitor—on salvage therapy for PTCL. The current follow-up represents clinical stability, with no development of new lesions or disease progression. **Discussion:** The case serves to underline the complex evolution as seen from mycosis fungoides to systemic PTCL and challenges in the management of refractory disease. Use of romidepsin underlines the potential of epigenetic therapies in the treatment of advanced T-cell lymphomas, especially in relapsed or refractory states. The patient's journey underlines the importance of early diagnosis, a multidisciplinary approach, and adaptive treatment strategy in the management of these aggressive lymphomas.

**Keywords:** Peripheral T-Cell Lymphoma, Mycosis Fungoides, Romidepsin, Epigenetic Therapy, PET-CT.

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#### INNOVATIVE MANAGEMENT OF REFRACTORY CLASSICAL HODGKIN LYMPHOMA WITH ATYPICAL HEPATIC PRESENTATION: A CASE STUDY

Candaş Mumcu<sup>1\*</sup>, Birol Güvenç<sup>2</sup>

<sup>1</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine

<sup>2</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** Classical Hodgkin Lymphoma (cHL) typically manifests through swollen lymph nodes, yet unusual cases do arise with atypical presentations. This report focuses on the management of a challenging case of refractory cHL, where the disease initially presented in the liver. The case underscores the effectiveness of a customized, multimodal treatment strategy. **Case Report:** A 59-year-old man was diagnosed with stage 4A cHL after a liver biopsy confirmed the disease. His initial PET-CT scans showed extensive involvement, with spread to cervical lymph nodes, nasopharyngeal and oropharyngeal regions, as well as diffuse splenic activity. The patient underwent six cycles of ABVD chemotherapy, but follow-up PET-CT scans revealed disease progression,

confirming primary refractory status. Subsequently, he was given salvage therapy with BV-DHAP, followed by high-dose chemotherapy and an autologous stem cell transplant (ASCT). Post-ASCT PET-CT scans demonstrated a significant metabolic response, with near-complete resolution of previous lesions, though splenomegaly persisted. Currently, the patient is undergoing maintenance therapy with brentuximab vedotin and has completed seven cycles successfully. **Discussion:** This case illustrates key challenges in the treatment of refractory cHL, particularly with atypical liver involvement, stressing the importance of considering lymphoma in cases of unexplained liver lesions. When the standard ABVD regimen failed, prompt initiation of aggressive salvage therapy was crucial in halting disease progression. The role of serial PET-CT imaging was pivotal in monitoring treatment effectiveness and guiding further clinical decisions. The tailored combination of salvage chemotherapy, ASCT, and maintenance with brentuximab vedotin showcases the evolving strategies in handling refractory cHL. Despite a significant overall response, the persistence of splenomegaly post-ASCT highlights the need for vigilant follow-up. This case emphasizes the potential for successful remission in refractory cHL through personalized, comprehensive treatment approaches, while also recognizing the need for continued exploration of emerging therapies.

**Keywords:** Classical Hodgkin Lymphoma, Refractory cHL, Hepatic Involvement, Autologous Stem Cell Transplant, Personalized Treatment.

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#### MULTIPLE MYELOMA IN A PATIENT WITH SJÖGREN'S SYNDROME: A CASE REPORT OF DIAGNOSTIC AND THERAPEUTIC CHALLENGES

Feride Aslanca<sup>1\*</sup>, Zeliha Yıldız Kandemir<sup>1</sup>, Naciye Nur Tozluklu<sup>1</sup>, Birol Güvenç<sup>2</sup>

<sup>1</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine

<sup>2</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** MM is a hematologic malignancy characterized by the proliferation of a clone of plasma cells that ultimately causes organ damage and the production of abnormal proteins. Sjögren's syndrome rarely coexisted with MM, a chronic autoimmune disease affecting exocrine glands and presenting very unique diagnostic and management challenges to the physician. This case illustrates the therapeutic journey of a patient with coexisting MM and Sjögren's syndrome and points out the importance of care provided in a multidisciplinary fashion. **Case Report:** A 64-year-old female patient with a history of hypertension for 14 years and prostheses of both hips was referred to the rheumatology department with

dry eyes and joint pains. The presence of anti-SSA antibodies and diminished results of the Schirmer test supported the diagnosis of Sjögren's syndrome; thus, hydroxychloroquine and prescription of artificial tears were started. Symptomatic treatment was begun because the development of albumin and total protein inversion suggested plasma cell dyscrasia. Further work-up for immunofixation electrophoresis and bone marrow biopsy confirmed IgG lambda-positive MM. She was subsequently treated with VRD (bortezomib, lenalidomide, and dexamethasone), followed by an autologous BMT in May 2024. Post-transplant maintenance was given with lenalidomide. She also developed sensory neuropathy, which was managed with pregabalin, with no recurrence of MM on follow-up. **Discussion:** The case epitomizes the complex diagnostic interplay between MM and Sjögren's syndrome. Symptoms of fatigue and protein abnormalities can easily be attributed to an autoimmune condition, with a delayed diagnosis of MM. Multidisciplinary collaboration has been critical for management of comorbidities and assurance of timely diagnosis. The patient responded well to BMT and maintenance therapy, proving personalized care. Furthermore, long-term treatment shows the necessity of monitoring drug-induced neuropathy. This case report adds to the growing awareness of rare concomitant autoimmune disorders and hematologic malignancies, with a reminder for vigilance in complex presentations and the delivery of adaptive multidisciplinary care.

**Keywords:** Multiple Myeloma, Sjögren's Syndrome, Bone Marrow Transplantation, Lenalidomide, Neuropath.

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**PRIMARY PALATAL ALK-NEGATIVE ANAPLASTIC LARGE CELL LYMPHOMA: RARITY TREATED SUCCESSFULLY WITH BRENTUXIMAB VEDOTIN**

Müjgan Çözeli<sup>1,\*</sup>, Elif Canbolat Hirfanoğlu<sup>1</sup>, Birol Güvenç<sup>2</sup>

<sup>1</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine

<sup>2</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** ALCL is an extremely rare T-cell non-Hodgkin lymphoma subtype made up of CD30-positive tumor cells, which are very aggressive. Though it most frequently involves lymph nodes and skin, less frequently, it affects other organs as well. Primary oral involvement, particularly of the palate, is highly uncommon. The paper reports a peculiar case of localized primary ALK-negative ALCL of the palate in a 73-year-old female patient treated successfully with brentuximab vedotin, pointing to the importance of identifying atypical presentations. **Case Report:** A 73-year-old female with a history of presenting a painless ulcer on her palate, which did

not heal with local treatments for two months, presented to the otolaryngology clinic and underwent an incisional biopsy. Histopathological findings showed large atypical lymphoid cells with prominent nucleoli, consistent with ALCL. Immunohistochemical staining was positive for CD30 and negative for ALK; in addition, Epstein-Barr virus testing returned negative. PET-CT showed localized uptake of FDG in the palate, SUVmax 8.5, with no significant lymphadenopathy and no systemic involvement. Bone marrow biopsy showed normal hematopoiesis with no evidence of infiltration. The patient was diagnosed with primary breast ALK-negative ALCL and started on brentuximab vedotin. The patient went into complete remission after three cycles of therapy with no residual disease evident on follow-up imaging. **Discussion:** This case illustrates the need to consider ALCL in the differential diagnosis of atypical sites, such as the palate, when lesions fail to respond to conventional therapy. Early biopsy and a wide panel of immunohistochemical tests are crucial for accurate diagnosis. Due to the high recurrence rates as well as poor prognosis associated with ALK-negative ALCL, highly active targeted therapies include brentuximab vedotin. The complete remission attained in this patient underlines the promise of personalized therapies in dealing with rare malignancies. Awareness of such atypical presentations may help in early diagnoses and improve patient outcomes. This case further stresses that management of lymphoma with such unusual presentations may be effectively accomplished using an interdisciplinary approach.

**Keywords:** anaplastic large cell lymphoma, ALK-negative, CD30, brentuximab vedotin, palatal lymphoma.

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**PRIMARY EXTRAMEDULLARY PLASMACYTOMA OF THE LYMPH NODES**

Ali Turunç<sup>1,\*</sup>, Birol Güvenç<sup>1</sup>

<sup>1</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** Extramedullary plasmacytomas are rare malignant neoplasms that can arise in various organs; however, lymph node involvement is uncommon. The cervical lymph nodes are most frequently affected. We present the case of a 68-year-old female diagnosed with a primary extramedullary plasmacytoma involving multiple lymph nodes, primarily in the cervical region. **Case Report:** A 68-year-old female patient presented with a one-month history of progressive enlargement and painful swelling of the right subclavicular and cervical areas. Imaging revealed pathological lymphadenopathy, and excisional biopsy was performed from the right cervical level 5 lymph node. Histopathological analysis confirmed the diagnosis of a plasmacytoma. A subsequent bone marrow biopsy revealed normocellular marrow without any evidence of infiltration. Positron emission tomography-CT staging

demonstrated further lymph node involvement in the right cervical, subclavicular, supraclavicular, axillary, and mediastinal regions. **Discussion:** This case was classified as a primary extramedullary plasmacytoma of the lymph nodes, given the absence of multiple myeloma markers in the bone marrow and immunoelectrophoretic studies. Lymph node plasmacytomas are exceedingly rare, comprising approximately 2% of all extramedullary plasmacytomas. Clinically, these patients often present with localized masses and minimal systemic symptoms. While recurrence is possible, primary lymph node plasmacytomas rarely progress to multiple myeloma and are associated with a more favorable prognosis than other solitary extramedullary plasmacytomas. The distinct clinical behavior of these lesions suggests that they may represent a unique subset of plasmacytomas with a lower risk of transformation into multiple myeloma. Most patients respond well to surgical excision, with minimal risk of recurrence or progression, even in the absence of adjuvant therapy. Although some patients develop osseous plasmacytomas, none have progressed to multiple myeloma in reported series.

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#### MANAGEMENT OF CHEMOTHERAPY-RESISTANT GASTRIC DIFFUSE LARGE B-CELL LYMPHOMA: A CASE REPORT

Hüseyin Derya Dinçyürek<sup>1,\*</sup>, Müjgan Çözeli<sup>2</sup>, Rashad Abdullayev<sup>3</sup>, Emircan Kiracı<sup>4</sup>, Naciye Nur Tozluklu<sup>2</sup>, Burak Demir<sup>2</sup>, Birol Güvenç<sup>5</sup>

<sup>1</sup> Mersin City Hospital, Division of Hematology

<sup>2</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine

<sup>3</sup> Cukurova University Medical Faculty Hospital, Department of Medical Genetics

<sup>4</sup> Cukurova University, Faculty of Medicine

<sup>5</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** Diffuse large B-cell lymphoma (DLBCL) is the most common subtype of non-Hodgkin lymphoma, often affecting extranodal sites like the stomach. While R-CHOP chemotherapy is the standard treatment, some patients fail to respond, requiring alternative approaches. In this report, we describe a case of gastric DLBCL in a 68-year-old man who became resistant to R-CHOP but achieved remission with R-DHAP. **Case Report:** A 68-year-old man came to the hospital with symptoms of persistent indigestion. After undergoing an endoscopic biopsy in October 2020, he was diagnosed with high-grade gastric diffuse large B-cell lymphoma. A PET-CT scan revealed a large mass in his stomach. He started R-CHOP chemotherapy, completing eight cycles. However, after five cycles, imaging showed remaining disease in the stomach, along with new lesions in the left lung. Despite ongoing treatment, a biopsy after the sixth cycle confirmed that the lymphoma was still active. The situation worsened—his disease

had become resistant to R-CHOP. In response, his treatment shifted to R-DHAP chemotherapy. After just two cycles, an endoscopic biopsy revealed no active lymphoma, and only signs of chronic atrophic gastritis remained. PET-CT scans over the following months showed no recurrence of lymphoma. However, in March 2023, a PET-CT showed some hypermetabolic lymph nodes in the cervical region, but these had regressed significantly compared to previous scans. As of October 2024, the patient continues to be closely monitored and remains asymptomatic. **Discussion:** This case highlights the challenges faced when dealing with chemotherapy-resistant DLBCL. It emphasizes the need to pivot quickly to alternative therapies, like R-DHAP, when first-line treatments fail. The successful response in this patient demonstrates that adjusting treatment strategies can make a significant difference in outcomes. Additionally, it shows the importance of long-term follow-up, especially with extranodal lymphomas, where the risk of relapse is ongoing.

**Keywords:** Diffuse large B-cell lymphoma, R-CHOP, Chemotherapy resistance, R-DHAP, Gastric lymphoma.

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#### SYSTEMIC NODULAR SCLEROSING HODGKIN LYMPHOMA WITH UNUSUAL HEPATIC AND GASTRIC INVOLVEMENT: A CASE REPORT

Hüseyin Derya Dinçyürek<sup>1,\*</sup>, Müjgan Çözeli<sup>2</sup>, Naciye Nur Tozluklu<sup>3</sup>, Bulut Sat<sup>3</sup>, Birol Güvenç<sup>3</sup>

<sup>1</sup> Mersin City Hospital, Division of Hematology, Mersin, Turkey

<sup>2</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine

<sup>3</sup> Cukurova University Medical Faculty Hospital, Department of Internal Medicine, Division of Hematology

**Introduction:** Hodgkin lymphoma (HL) is typically known for presenting as enlarged lymph nodes, but occasionally, it takes an unexpected turn, spreading to less common locations. In this report, we dive into a rare case of nodular sclerosing Hodgkin lymphoma, where the disease had aggressively spread, invading the liver and stomach—locations rarely associated with HL. **Case Report:** A 40-year-old woman came to the clinic with persistent back pain, trouble walking, and noticeable weight loss. At first, these symptoms seemed to point to a spinal issue, prompting an L4 kyphoplasty. However, things quickly worsened, and her condition began to deteriorate. A PET-CT scan soon revealed troubling results—multiple areas of hypermetabolic activity across her lymph nodes and bones, which were now lighting up with disease. A biopsy of the inguinal lymph node confirmed the diagnosis: classical Hodgkin lymphoma, nodular sclerosing type. Treatment started with Brentuximab vedotin paired with the AVD regimen (Adriamycin, Vinblastine, and Dacarbazine), but complications arose. During therapy, she developed a painful perianal abscess,

which needed surgical drainage. Yet the disease kept advancing. New imaging showed a more aggressive spread: multiple hypermetabolic lesions were found in the liver, and another was detected in the gastric fundus. Despite a clear endoscopy, which didn't show any visible abnormalities in the stomach, a liver biopsy confirmed what the team feared—Hodgkin lymphoma had infiltrated her liver. Her treatment continues with careful monitoring as the medical team adapts to these complications. **Discussion:** This case paints a picture of the diagnostic and treatment challenges that arise when Hodgkin lymphoma doesn't follow the expected path. Instead of typical lymphadenopathy, the disease made itself known through musculoskeletal pain and neurological issues, creating a complex clinical puzzle. The rare involvement of the liver and stomach emphasizes just how unpredictable the systemic spread of this disease can be. While hepatic involvement in HL is unusual, it's critical to confirm this through biopsy, as it can easily be mistaken for other liver-related conditions. Gastric involvement, though rare, must be kept in mind when dealing with extensive disease spread. Advanced imaging, particularly PET-CT, played a pivotal role in uncovering these unexpected sites of involvement, guiding the treatment plan. This case is a testament to the importance of recognizing atypical presentations of Hodgkin lymphoma and the need for flexible, evolving treatment strategies. The use of Brentuximab vedotin in combination with AVD has shown promise, especially in complicated cases like this one, where the disease has spread far beyond the usual lymphatic system. Understanding HL's ability to infiltrate uncommon sites like the liver and stomach is essential for improving patient outcomes. This case reminds us of the disease's unpredictable nature and the need for vigilance in detecting and managing its spread to rare locations.

**Keywords:** Nodular Sclerosing Hodgkin Lymphoma, Hepatic Infiltration, Gastric Involvement, Systemic Spread, Brentuximab Vedotin Treatment.

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#### SYSTEMIC AMYLOIDOSIS PRESENTING WITH LYMPHADENOPATHY: A DIAGNOSTIC OVERLAP WITH MULTIPLE MYELOMA AND POSSIBLE CARDIAC INVOLVEMENT

Naciye Nur Tozluclu<sup>1,\*</sup>, İdil Yürekli<sup>2</sup>, Şule Menziletoğlu Yıldız<sup>3</sup>, Birol Güvenç<sup>4</sup>

<sup>1</sup> Cukurova University, Faculty of Medicine, Department of Internal Medicine

<sup>2</sup> Cukurova University, Faculty of Medicine, Department of Anatomy

<sup>3</sup> Cukurova University, Faculty of Medicine, The Blood Center of Balcali Hospital,

<sup>4</sup> Cukurova University, Faculty of Medicine, Department of Internal Medicine, Division of Hematology

**Introduction:** Systemic amyloidosis is a condition where amyloid proteins accumulate in organs and tissues, causing multisystem dysfunction. Its presentation often overlaps with other conditions like lymphoproliferative disorders and multiple myeloma (MM). Lymphadenopathy is rare in amyloidosis but can complicate the clinical picture, mimicking more common hematological diseases. We present a case of systemic amyloidosis in a patient initially suspected of having lymphoma, complicated by underlying multiple myeloma and probable cardiac amyloidosis. **Case Report:** A 63-year-old male with a history of heart failure and chronic kidney disease presented with frequent hospital admissions due to dyspnea. Axillary lymphadenopathy prompted referral to hematology. PET-CT revealed widespread FDG-avid lymphadenopathy, suggesting lymphoma. Biopsy showed plasma cell infiltration (10-11%) with kappa light chain monotypic plasma cells and amyloid deposits, indicating systemic amyloidosis. Concurrent imaging revealed pleural effusions, calcified lymphadenopathies, and findings consistent with granulomatous disease. Further hematological evaluation suggested underlying plasma cell dyscrasia, likely multiple myeloma. The patient's history of heart failure raised the suspicion of cardiac amyloidosis, a common complication in systemic amyloidosis, warranting cardiology evaluation and planned cardiac MRI. **Discussion:** This case underscores the diagnostic challenge posed by systemic amyloidosis, especially when lymphadenopathy is present, leading to initial misdiagnosis as lymphoma. Amyloidosis-related lymphadenopathy is uncommon but should be considered, especially when plasma cell dyscrasias like multiple myeloma are involved. The concurrent diagnosis of multiple myeloma further complicates the clinical course, necessitating a tailored therapeutic approach. Cardiac amyloidosis is a serious complication often seen in patients with systemic amyloidosis, especially AL-type, where amyloid deposits infiltrate the myocardium, leading to restrictive cardiomyopathy. In this case, the patient's long-standing heart failure and arrhythmia raised the likelihood of cardiac involvement. Early detection is crucial, as cardiac amyloidosis is associated with a poor prognosis. The integration of advanced cardiac imaging, such as MRI, is essential in confirming the diagnosis and guiding treatment. This case illustrates the importance of considering systemic amyloidosis in patients with unexplained lymphadenopathy and highlights the need for multidisciplinary management, particularly when cardiac involvement is suspected.

**Keywords:** Amyloidosis, Lymphadenopathy, Multiple Myeloma, Cardiac Amyloidosis, Plasma Cell Dyscrasia.

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