

Table 1 (continued)

	Patient 1	Patient 2	Patient 3
Gum hypertrophy	-	+	-
Peripheral Blast %	%77,2	%89,8	90,9
CD 34	%9,8	negative	%1,4
CD 19	negative	negative	negative
CD13	%25	% 8	% 30,6
CD33	%99,8	%99,9	% 95
HLA-DR	negative	negative	negative
FLT-3 ITD	positive	positive	positive
t(15;17)PML/RARA	negative	negative	negative

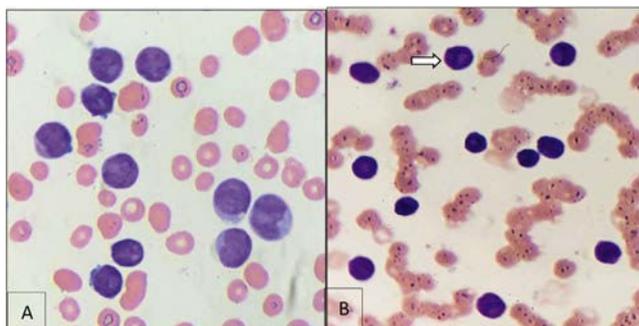


Figure 1: A) Blast Cells B) "Cup-like" Nucleus Morphology

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#### OP 20

##### A CASE OF ACUTE CORONARY SYNDROME DEVELOPING AFTER GRANULOCYTE COLONY STIMULATING FACTOR (G-CSF) ADMINISTRATION

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**Objective:** Common side effects after use of granulocyte colony-stimulating factor (G-CSF) include bone and muscle pain, headache, fever, and inflammation at the injection site. Less common side effects that cause serious morbidity and mortality in the early period include stroke, myocardial infarction (0.1%), and clinical conditions resulting from thrombosis, such as deep vein thrombosis. **Case Report:** A 60-year-old, Stage 3A, Multiple Myeloma (MM) patient with a very good partial response was planned stem cell mobilization with G-CSF. The patient, who had no abnormalities in blood values and a normal cardiological examination approximately 1 month ago, was administered 10 micrograms/kg/day G-CSF and approximately 6 hours after the first dose, the patient developed severe chest pain radiating to the left arm. The patient's ECG evaluation showed sinusoidal rhythm but tachycardia (115/beat/one minute). WBC:  $29.7 \times 10^3/\mu\text{L}$ , d dimer:  $3510 \mu\text{g/L}$  and troponin: positivity. Angiography was performed on the patient because troponin values were increasing. A decrease in blood flow was detected in the LAD and right coronary artery branches. The patient's complaints

improved with medical antiaggregant, anticoagulant and vasodilator treatment and he was discharged. **Discussion:** G-CSF acts as a regulator of myeloid progenitors and acts by promoting cell proliferation, differentiation, and maturation. The G-CSF receptor is found on hematopoietic stem cells, granulocytes, monocytes, and lymphocytes and also expressed on non-hematopoietic cardiovascular, neuronal, endothelial, and placental cells. There are rare reports that G-CSF and hematopoietic stem cells play a role in the development of atherosclerosis. Halter et al. reported in their study on 388 donors that 4 of the donors developed cardiac arrest within 30 days after stem cell mobilization, and 3 of these were due to myocardial infarction. This case is important in terms of emphasizing that we should follow the patient's clinical and biochemical evaluations very closely and be careful during the mobilization process.

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#### OP 21

##### A HEMATOLOGICAL CHAMELEON: THE TRANSITION FROM MDS TO NON-SECRETORY MULTIPLE MYELOMA AND BACK – UNRAVELING DIAGNOSTIC COMPLEXITIES AND ADAPTING THERAPEUTIC PATHWAYS

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**Objective:** The interplay between myelodysplastic syndrome (MDS) and non-secretory multiple myeloma (MM) can confound even seasoned hematologists, particularly when these conditions shift over time. This case presents a remarkable instance of a patient transitioning from MDS to non-secretory MM and then reverting back to MDS, underscoring the need for meticulous monitoring and adaptable treatment strategies when dealing with complex hematological landscapes. **Case Presentation:** An 82-year-old patient initially sought care for severe anemia, leading to a diagnosis of MDS based on bone marrow findings. At this point, no signs of MM were present. However, later investigations—specifically urine immunofixation—suggested the emergence of non-secretory MM, which was confirmed through a second bone marrow biopsy. The patient began treatment with Velcade, Revlimid, and Dexamethasone (VRD), showing marked improvement in anemia. Yet, given the patient's age and frailty, hematopoietic stem cell transplantation (HSCT) was not considered viable. As treatment progressed, the regimen evolved to ixazomib, lenalidomide, and dexamethasone, achieving remission for several years. Despite this stability, a resurgence of anemia signaled a reversion to MDS. A fresh treatment strategy was introduced, combining azacitidine, low-dose lenalidomide, and erythropoietin, aimed at maintaining functionality and quality of life without aggressive interventions. **Discussion:**

This case encapsulates the volatile nature of hematologic disorders, illustrating how diseases like MDS and non-secretory MM can morph and evolve. It emphasizes the importance of adaptive management, especially in elderly patients, where rigid treatment paradigms may fall short. The use of lenalidomide throughout the patient's journey reflects its dual utility in both plasma cell and myeloid disorders, while also sparking questions about whether prolonged exposure could influence secondary disease development. **Conclusion:** The patient's journey through MDS, MM, and back again underscores the critical need for dynamic reassessment, vigilance, and personalized care. This case exemplifies the blurred boundaries between plasma cell dyscrasias and myeloid neoplasms, raising thought-provoking questions about disease progression and therapeutic strategies. In navigating these complexities, clinicians are reminded of the importance of flexible, patient-centered approaches in managing intricate hematological disorders.

**Keywords:** Myelodysplastic Syndrome, Non-Secretory Multiple Myeloma, Disease Evolution, Adaptive Treatment, Elderly Patient Care.

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#### OP 22

##### BRAIN-INVOLVED MULTIPLE MYELOMA: STABILITY ACHIEVED WITH BENDAMUSTINE-POMALIDOMIDE, RADIOTHERAPY AND DARATUMUMAB-BASED THERAPY

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**Objective:** Multiple myeloma is a plasma cell malignancy that mainly affects the bones and skeletal system. The involvement of the brain as a site is very rare; it usually takes place via calvarial lesions with intracranial extension and is considered resistant to treatment. This report presents the case of a patient presenting with refractory MM and discusses in detail the efficiency of bendamustine-pomalidomide therapy and daratumumab-based maintenance after ASCT. **Case Report:** A 62-year-old female was diagnosed with kappa-positive MM in 2015, when plasma cell infiltration in the bone marrow was 20%. The patient underwent chemotherapy followed by ASCT in 2016. This patient attained remission after the transplant. Three years later, she presented with brain involvement, and MRI confirmed lesions of the parietal calvarium along with soft tissue expansion into the brain. The patient received radiotherapy to the affected area of the brain and initiated bendamustine-pomalidomide therapy; indeed, remarkable improvements were made in lesions of the brain and skeleton. Following that response, daratumumab, lenalidomide,

and dexamethasone maintenance therapy was initiated to ensure ongoing disease control. Currently, the patient is clinically stable, with no evidence of further progression on follow-up imaging. **Discussion:** This case underlines the rarity of brain involvement in MM, as well as the role of ASCT as part of first-line treatment. The late appearance of extramedullary brain involvement three years post-transplantation truly epitomizes the whim of MM. Bendamustine-pomalidomide therapy was effective for refractory disease management, whereas daratumumab-based maintenance has helped maintain stability.

**Keywords:** Multiple Myeloma, Brain Involvement, Extramedullary Disease, Bendamustine, Autologous Transplantation.

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#### OP 23

##### SUCCESSFUL LONG-TERM REMISSION IN AGGRESSIVE YOUNG-ONSET CHRONIC LYMPHOCYTIC LEUKEMIA WITH IBRUTINIB AND VENETOCLAX: A DECADE-LONG CASE STUDY

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Chronic Lymphocytic Leukemia (CLL) typically affects older individuals and is frequently associated with genetic mutations that help predict its progression. However, this report presents a rare case of aggressive CLL in a young woman with no unfavorable genetic markers, who achieved lasting remission following the use of dual targeted therapy, after standard treatments repeatedly failed. A 40-year-old woman was diagnosed with CLL during a routine blood examination. Despite lacking any high-risk genetic indicators, her disease advanced swiftly over the next ten years. Initial treatment with CVP (cyclophosphamide, vincristine, prednisone) provided only a brief partial remission. A subsequent course of R-CHOP (rituximab, cyclophosphamide, doxorubicin, vincristine, prednisone) also led to relapse within a few months. Ibrutinib was introduced as a single-agent treatment but failed to control the disease. The patient's condition continued to deteriorate, with recurring lymph node enlargement and rising lymphocyte counts. Three years ago, venetoclax was added to her treatment alongside ibrutinib. This combination therapy produced an extraordinary result—complete remission was achieved, blood counts normalized, lymphadenopathy disappeared, and bone marrow tests showed no trace of residual disease.

**Keywords:** Chronic Lymphocytic Leukemia, Early-Onset CLL, Resistant CLL, Venetoclax, Ibrutinib.

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