

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

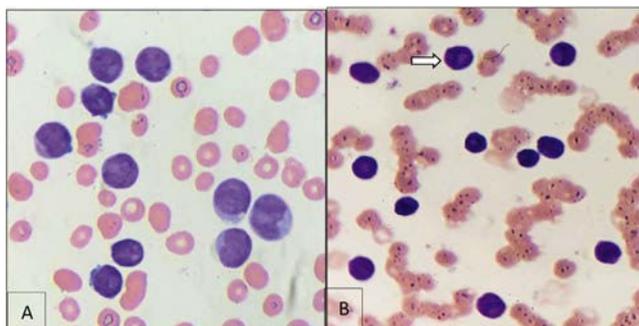


Figure 1: A) Blast Cells B) "Cup-like" Nucleus Morphology

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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:**