Table 3 (continued)

	Multivariate HR (95% CI)	p
HSCT		<0.001
None	1.00	
Autologous	6.25 (3.40-11.51)	
Chemotherapy		0.203
No	1.00	
Yes	3.63 (0.49-26.44)	

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OP 19

FLT3-ITD POSITIVE ACUTE MYELOİD LEUKEMIA MIMICKING ACUTE PROMYELOCYTIC LEUKEMİA

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Objective: Acute myeloid leukemia (AML) is a heterogeneous disease due to genetic abnormalities and differences in immunophenotypes. The diagnosis of AML requires a careful evaluation of clinical morphology, immunophenotyping, cytogenetics, and molecular analyses. [1] In current practice, flow cytometry-based immunophenotyping provides a rapid and reliable method for diagnosing AML, including acute promyelocytic leukemia (APL). APL is a subtype of AML with distinct morphological, biological, and clinical characteristics. It can be effectively treated with ATRA-based therapy protocols. However, if not treated quickly, it can be fatal due to the risk of disseminated intravascular coagulation (DIC). Therefore, the diagnosis and treatment of APL represent a true medical emergency.[2] The absence of CD34, HLA-DR, and CD11b is a characteristic immunophenotypic feature that often distinguishes APL from other AML subtypes. However, AML subtypes other than APL that lack CD34 and HLA-DR expression have also been reported. APL accounts for 8% to 17% of AML patients. In AML patients without the PML-RARA fusion gene, 12% to 21% of cases have been identified as HLA-DR negative. These HLA-DR negative AML cases are distinct from APL because they do not carry the characteristic PML-RARA fusion.[3] HLA-DR and CD34 negativity is generally observed in AML-M1 and AML-M2 subtypes and is associated with nucleophosmin (NPM1) gene mutations and FMS-like tyrosine kinase-internal tandem duplication (FLT3-ITD) mutations.[4] NPM1 mutations are among the most common genetic abnormalities in AML, occurring in 27-35% of adult AML cases. Although rare, an "APL-like" immunophenotype has been reported in some de novo acute myeloid leukemia (AML) cases with NPM1 gene mutations. These cases show some immunophenotypic similarities to APL, despite being genetically different. AML cases with NPM1 mutations have unique clinical and biological characteristics.[5] In this study, we aimed to highlight the association of FLT3-ITD positivity, as opposed to NPM1, in HLA-DR negative non-APL AML cases in our clinic. **Methodology:** We examined three acute leukemia patients who were referred to our clinic within one month and were initially reported as APL based on flow cytometry analysis. Our focus on these patients stemmed from the fact that a condition with an incidence of 1-2 cases per 1 million people per year was diagnosed consecutively as APL in flow cytometry analysis within a short period. Fluorescent in situ hybridization (FISH) analysis for the 15;17 translocation and polymerase chain reaction (PCR) for FLT3-ITD and FLT3-TKD mutations were performed on the patients' peripheral blood. NPM1 mutations could not be analyzed in these patients. **Results:** Morphological examination of the patients' peripheral blood smears showed prominent nucleoli, Auer rods, and cup-like nuclei. Due to the CD34 and HLA-DR negativity in the flow cytometry analysis, these cases were initially considered APL. However, cytogenetic results revealed a negative t(15;17) translocation in all three patients, excluding APL. Additionally, all three patients tested positive for FLT3-ITD mutations. The peripheral blood white blood cell (WBC) count, blast percentage, and D-dimer levels were significantly elevated at the time of presentation in all patients. (Table 1) **Conclusion:** In cases with APL-like immunophenotypes, these similarities pose diagnostic challenges in daily practice. In this study, the APL-like AML cases exhibited CD34 and HLA-DR negativity and carried FLT3-ITD mutations. These de novo cases were characterized by high WBC counts, blast percentages, and elevated D-dimer levels. NPM1 is one of the most frequently mutated genes in AML, often seen alongside FLT3-ITD. Morphological and immunophenotypic similarities between many AML cases with NPM1 mutations and APL are well-known. In the first case, the blasts resembled the abnormal promyelocytes of APL (Figure 1.A). In the second case, a blast with a cup-like nucleus was observed (Figure 1.B). The "cup-like" nucleus morphology is specifically associated with acute myeloid leukemia (AML) with NPM1 gene mutations. The WBC count was very high in all cases a feature that is unusual for APL, especially the hypergranular variant. High WBC counts and blast cell percentages are typically described in NPM1-mutated AML.[6] Similarly, this could also be considered for FLT3-ITD, based on our findings. However, further studies with more cases are needed to confirm this. All patients demonstrated elevated D-dimer levels, which is more strongly associated with APL.[7] Unlike high D-dimer levels, fibrinogen levels were within acceptable limits. One patient had prominent gum hypertrophy, and frequent gum bleeding was observed during clinical follow-up. In conclusion, for cases with APL-like features but negative PML-RARA results by FISH and/or molecular methods, it is important to consider AML with NPM1 and/or FLT3-ITD mutations.

Table 1 – Clinical-Pathological Parameters of the Patients

	Patient 1	Patient 2	Patient 3
Age	34	35	57
Gender	Female	Female	Female
Hemoglobin(gr/dl)	6,3	9,4	8,8
Plateletes	12,000	75,000	91,000
Leukocytes(WBC)	159,000	120,000	307,000
LDH	841	405	471
Fibrinogen	246	419	341
D-dimer	7499	7853	1020

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

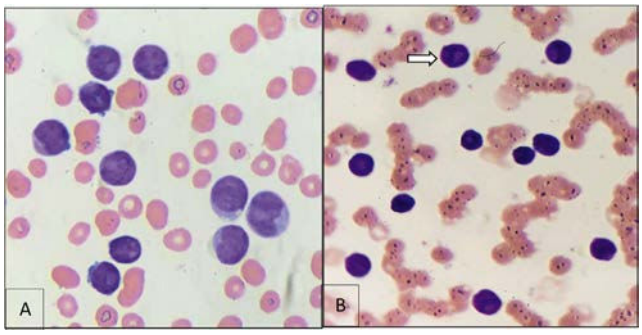

Figure 1: A) Blast Cells B) "Cup-like" Nucleus Morphology

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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 GCSF. The patient, who had no abnormalities in blood values and a normal cardiological examination approximately 1 month ago, was administered 10 micrograms/kg/day GCSF 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: $29.7 \times 103/\mu$L, d dimer: 3510 μg/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 – UNRAVELİNG 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:**