(50%) had renal involvement. The stages of these involvements are summarized in Table 1. In the group treated without Dara, the triplet regimens were VCD (n=30), VRD (n=3), and VMP (n=2). Mortality was significantly lower in the Dara-VCD group. When evaluating responses, progression was only in 1 (9%) patient in the Dara group, whereas in 8 (32%) in the without Dara group. The overall survival was not statistically significant between the two groups (log-rank p=0.394). (Figure 1). Conclusion: We used Dara after Health Authority approval and reimbursement in our country. So, we get the opportunity to compare Dara-added VCD effectiveness to VCD or VRD as a real-life analysis. Dara-VCD resulted in a significantly lower rate of progression and mortality compared to those without Dara. The follow-up duration was shorter for comment on overall survival. Additionally, 10 patients without the Dara group, did receive daratumumab with VCD (n=5), or with other agents (n=5)). With this study, we documented from a real-life experience addition of daratumumab to the VCD regimen in first-line treatment reduces mortality and progression in AL amyloidosis.

Table 1 – Patient Characteristics				
	Dara-VCD (n=17)	Other Triplet Regimens (n=35)	P-value	
Age (mean ± std)	65±6	57±11	0,003	
Gender (F/M)	10 (%58.8)/7 (%41.2)	14 (%40)/21 (%60)	0,202	
Median follow-up (months)	15(1-68)	36 (1-114)	0,108	
ECOG performance score >2	7 (%43.8)	11 (%33.3)	0,273	
Mayo 2012 Staging System			0,954	
Stage 1	6 (%37.5)	10 (%29.4)		
Stage 2	3 (%18.8)	7 (%20.6)		
Stage 3	5 (%31,3)	12 (%35,3)		
Stage 4	2 (%12.5)	5 (%14.7)		
Palladini et al. Staging System			0,166	
Stage 1	7 (%43.8)	18 (%51,4)		
Stage 2	7 (%43.8)	7 (%20)		
Stage 3	2 (%12.5)	10 (%28,6)		
dFLC (mg/L)	106,5 (4-1945)	95 (0-2425)	0,624	
hs Pro BNP (median)	1769 (25-30117)	1184 (17-25113)	0,4	
eGFR (mL/min)	71 (2-106)	85 (9-124)	0,264	
ASCT	3 (%17,6)	12 (%34,3)	0,214	
Mortality	3 (%17,6)	18 (%51,4)	0,02	
Treatment Response (Hematologic)			0,042	
VGPR and above	8 (%72,7)	7 (%28)		
SD	2 (%18,2)	10 (%40)		
Progression	1 (9,1)	8 (%32)		

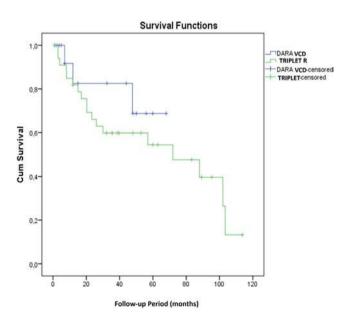


Figure 1: Overall Survival in Dara-VCD and without-Dara Triplet Treatment Groups

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OP 08

FROM MULTIPLE MYELOMA TO ACUTE MYELOID LEUKEMIA

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Objective: Multiple myeloma (MM) and acute myeloid leukemia (AML) are malignant clonal diseases of cells in different lineages. Only 79 cases of the two diseases occurring together have been reported in Pubmed in the last 10 years. However, simultaneous occurrence of MM and AML on presentation in chemotherapy-naive patients is rare, with only a 25 cases reported in the literature so far. (1)This case presents our experience with a patient who was diagnosed with AML shortly after the diagnosis of MM. Case report: A 57-year old male patient was referred to hematology due to pancytopenia. Serum immunofixation confirming monoclonal gammopathy of IgG kappa was detected. ß-2 microglobulin was found as 3,56 mg/dL. All routine tests required for MM diagnosis and follow-up were performed. (table 1) Bone marrow biopsy showed 40% plasma cell infiltration stained with CD38, CD138 and kappa. PET/CT scan revealed lytic lesions in the bones. t(4;14) was found positive in fluorescence in situ hybridization analysis. Bortezomib, lenalidomide, dexamethasone (VRD) regimen was started. After 4 cycles of VRD, the patient was hospitalized due to febrile neutropenia and severe dyspnea. Pulmonary embolism and pneumonic infiltration were not detected in thorax CT. However; bilateral septal edema and pleural effusion up to 3,5 cm were detected. Pleural fluid sampling was performed. No infectious agent was detected in exudative effusion. 3,25% myeloid blasts, 1,99% plasma cells were found by flow cytometry of pleural fluid. (figure 1) A bone marrow biopsy was performed again due to blasts in the pleural fluid results. Biopsy showed 40% myeloid blasts (CD117 and CD34) and 18% plasma cells in the bone marrow parenchyma. (figure 2) We decided to treat the patient with 7+3 induction chemotherapy (Idarubicin and cytarabine) with daratumumab and dexamethasone (Dara-d). After induction and one course of dara-d, bone marrow biopsy was performed again to evaluate response. The patient could not achieve remission and died due to acute respiratory failure. Results Conclusion: Multiple myeloma (MM) and acute myeloid leukemia (AML) may usually develop in the same patient but they are generally seen in MM patients receiving chemotherapy and in due course of treatment AML develops. (2) Presence of AML with MM in a shortly after treatment begins is an extremely rare occurrence. Concurrent diagnosis of these two hematological malignancies yields a poor prognosis. (3)References:(1) Jamal I, Shuchismita S, Choudhary V. Twin Malignancy of Acute Myeloid Leukemia and Multiple Myeloma in a Chemotherapy-Naïve Patient: A Rare Occurrence. J Lab Physicians. 2022 Oct 20;15(2):306-310. doi: 10.1055/ s-0042-1757588. PMID: 37599817; PMCID: PMC10437150.(2) Parapia L, Abbott CR, Masters G, Roberts BE. Simultaneous pre

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Table 1 – Laboratory and bone marrow biopsy findings of the patient at the time of diagnosis

Laboratory tests	Results	Normal Value
Hemoglobin	8,00 g/dL	12,1 - 16,6
Total leukocyte count	$2,51 \times 10^{3}/\mu L$	3,46 - 10,04
Platelets	$76 \times 10^3 / \mu L$	172-380
Creatinine	0,86 mg/dL	0,7 - 1,2
Serum immunoglobulin G	57,82 g/L	7-16
ß-2 microglobulin	3,56 mg/dl	-
Serum kappa free light chain	243	17-37
Serum lambda free light chain	10,8	9-21
Bone marrow plasma cell percentage	%35-40	

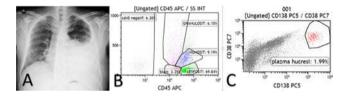


Figure 1: (A) Chest X-ray showed pleural effusion. (B) Pleural fluid was positive for myeloid blasts. (C) Plasma cell persentage of pleural fluid.

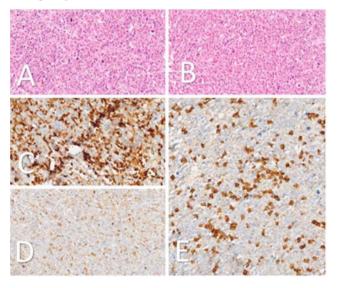


Figure 2: (A) Bone marrow biopsy, H & E stain \times 40. The bone marrow is hypercellular. (B) Bone marrow biopsy, H & E stain \times 40, The number of normal hematopoietic elements is markedly increased, the infiltrate consists of plasma vcells and blasts. (C) Approximately 40% CD34 staining in the bone marrow parenchyma. (D) CD117 staining cells are 18% of the

mrrow. (E) Number of cells stained with CD138, 18% of parenchyma.

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OP 09

CASE REPORT: SIMULTANEOUS OCCURRENCE OF PLASMA AND B CELL MALIGNANCY

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Objective: Multiple myeloma (MM) and chronic lymphocytic leukemia (CLL) are two distinct hematological malignancies thought to arise at different stages of the B cell maturation pathway. Here, we aimed to present our approach to such a patient. Case report: A 59-year-old male patient was examined for hematuria in 2019, lymphocytosis was detected, and flow cytometry (FC) was performed. The result was found to be compatible with CLL. The patient was evaluated as Binet A, Rai 0 at the diagnosis, and was followed without treatment. In the fourth year of treatment-free follow-up, the patient developed severe B symptoms and widespread lymphadenopathies (LAP), splenomegaly (size 15.5 cm) on imaging, and shortened lymphocyte doubling time, so bone marrow aspiration biopsy (BMAB), FC and cytogenetic tests were performed. The patient had a CLL immunophenotype score of 1 in flow cytometry, and cytogenetics showed negative 17p del and TP53 mutations. In BMAB, 80% atypical morphology and immunohistochemical (IHI) examination showed small lymphoid cells with CD19(+), CD20(+), CD23(+), and CD5(+) staining. The patient was evaluated as Binet B, Rai 2, and got 6 cycles of Chemoimmunotherapy (Rituximab, Fludarabine, Cyclophosphamide). After treatment, B's symptoms regressed, and LAP and splenomegaly returned to normal. The patient developed neutropenia requiring granulocyte colony-stimulating factor (G-CSF) during follow-up, therefore, the patient underwent repeat BMAB. In the result, plasma cells with intense kappa positive staining were observed, and in the IHI examination, the CD38 and 138 positivity rates were evaluated as 20%. The patient, who did not have hypercalcemia, renal dysfunction, anemia, bone lesions, and extramedullary involvement, was assessed as MGUS, neutropenia resolved spontaneously during follow-up, and it was decided to follow the patient at three-month intervals without treatment. Conclusion: MM and CLL were rare in the same patient, and there is limited information regarding clinical outcomes and management. The clonal relationship between them is controversial.

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