

Table 2 – Mean for Survival

Subtypes	PFS Mean				OS Mean			
	Estimate	Std. Error	95% CI		Estimate	Std. Error	95% CI	
			Lower Bound	Upper Bound			Lower Bound	Upper Bound
HL	63.640	8.071	47.821	79.460	65.546	7.501	50.844	80.248
DLBCL	70.205	9.593	51.402	89.008	94.927	16.105	63.361	126.492
FL	60.899	13.367	34.700	87.098	113.000	8.485	96.369	129.631
MZL	36.200	9.543	17.495	54.905	69.250	19.674	30.689	107.811
MCL	61.075	15.882	29.947	92.203	84.429	15.081	54.870	113.988
T-cell	14.667	3.928	6.969	22.365	33.333	6.760	20.083	46.583
Total	72.452	6.735	59.252	85.652	114.038	7.918	98.519	129.557

HL Hodgkin Lymphoma, DLBCL Diffuse Large B-cell Lymphoma, FL Follicular Lymphoma, MZL Marginal Zone, Lymphoma, MCL Mantle Cell Lymphoma

Table 3 – Cox Regression for PFS and OS

	PFS				OS			
	p	HR	95% CI		p	HR	95% CI	
			Lower	Upper			Lower	Upper
age	.311	.986	.959	1.013	.793	1.004	.974	1.035
group	.290				.423			
HL-DLBCL	.515	1.516	.432	5.317	.365	1.733	.527	5.703
HL-FL	.251	2.059	.600	7.062	.143	.190	.021	1.754
HL-MZL	.357	2.377	.376	15.011	.805	1.263	.199	8.015
HL-MCL	.194	2.523	.624	10.199	.954	.954	.193	4.710
HL-T-cell	.020	7.663	1.385	42.389	.635	1.528	.265	8.805
sex	.743	1.146	.507	2.591	.851	1.096	.419	2.871

HL Hodgkin Lymphoma, DLBCL Diffuse Large B-cell Lymphoma, FL Follicular Lymphoma, MZL Marginal Zone Lymphoma, MCL Mantle Cell Lymphoma, CI Confidence Interval, HR Hazard Ratio

limitation. However, the strength of our study is the ability to compare six lymphoma subtypes within the same study. Further research is needed to focus on larger patient cohorts and incorporate detailed evaluations of risk factors in patients undergoing autologous transplantation.

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OP 12

RESULTS OF UNRELATED ALLOGENEIC STEM CELL TRANSPLANTATION: A SINGLE CENTER EXPERIENCE

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Objective: Evaluation of data from unrelated hematopoietic stem cell transplants performed in our transplant center.

Methodology: At the Private Emsey Hospital Adult Stem Cell Transplantation Unit were evaluated retrospectively between 2016 and 2023 to Allogeneic hematopoietic stem cell transplantations performed on 76 patients with different diagnoses from unrelated donors. **Results:** Data of patients with a mean age of 41.9 years were retrospectively analyzed. All donors were from a Turkish stem cell bank and 51% had HLA 1 allele incompatibility. 28 transplants were performed between different genders. Average follow-up was 17.3 months. Neutrophil engraftment occurred in an average of 18.1 days. Acute GVHD was detected in 26% and chronic GVHD in 41%. 1-year overall survival was 37% and disease-free survival was 32%. **Conclusion:** Non-relative stem cell transplantation is an important option especially in hematological diseases where there is no family donor and allogeneic transplantation is required. It has been observed that non-relative data performed in our clinic are similar to data from other centers.

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OP 13

MANTLE CELL LYMPHOMA PATIENT WITH SKIN GVHD AFTER AUOTOLOGOUS STEM CELL TRANSPLANT

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Objective: Graft-versus-host disease (GVHD) after ASCT is an immunologically developing process. T cells and inflammatory cytokines formed against the recipient's alloantigens are responsible for this. It is less common in autologous SCT than in allogeneic SCT. It has been reported in the literature that there are MM patients who developed autologous GVHD after Autologous SCT. **Case report:** Here, we present a case

of mantle cell lymphoma who developed autologous GVHD after ASCT, not previously reported. Fifty-four-year-old male patient was diagnosed with Stage 4A Mantle cell lymphoma according to the Ann Arbor staging system based on the result of inguinallymph node excisional biopsy performed in July 2021. Three courses of R-CHOP, DHAP treatment was started for the patient who did not respond after R-CHOP treatment. Complete response was achieved after three cycles of R-DHAP therapy, and mobilization was performed with filgrastim and plerixafor protocol. Then, autologous SCT was performed with the BEAM protocol (Karmustine 300mg/m², Etoposide 200mg/m², Cytosine Arabinoside 2 × 100mg/m², Melphalan 140mg/m²). Post-transplant neutrophil engraftment occurred on the +14th day and platelet engraftment on the +32nd day. Piperacillin Tazobactam was started due to fever and sore throat on the 3rd day of ASCT. Teicoplanin was added to the treatment after the fever persisted on the 5th day of ASCT. Piperacillin Tazobactam treatment was discontinued due to acute phase reactant increase and fever on ASCT +7th day and Meropenem was started. Teicoplanin treatment was stopped on the +16th day after transplantation, and Meropenem was stopped on the +17th day. On the +14th day of autologous SCT, complaints of itching and rash started on the patient's body. The biopsy result of the patient who underwent skin biopsy was compatible with grade 1-2 GVHD. The patient was started on high-dose corticosteroid therapy. On the +21st day, the complaint of itching and on the +28th day, the skin findings disappeared. On the +28th day of ASCT, corticosteroid therapy was tapered and discontinued. The patient is still being followed without disease. **Conclusion:** Autologous GVHD is an autoimmune syndrome initiated by autoreactive T cells that recognize major histocompatibility complex (MHC) class II antigens. CD8+ T cells recognize MHC class II determinants. There are three types of autologous GVHD: 1. spontaneous autologous GVHD 2. induced autologous GVHD; using cyclosporine, tacrolimus, interferon- α , interferon- γ and alemtuzumab to induce the effect of GVT; GVHD induced by transfusion of non-irradiated blood products in patients with Hodgkin lymphoma, Non-Hodgkin Lymphoma (NHL), chronic lymphocytic leukemia, and acute myeloid leukemia 3. induced by transfusion of non-irradiated blood products. In the study of Drobyski et al. in 2008, it was reported that autologous GVHD developed in 5 of 386 patients who underwent autologous SCT. Response to steroids was not good. It was the first transplant of 223 multiple myeloma patients. Only 2 patients developed autologous GVHD. Autologous GVHD developed in 3 of 27 patients with a second transplant. Autologous GVHD did not develop in Hodgkin lymphoma, non-Hodgkin lymphoma and AML patients. Therefore, it will be important to consider this complication while planning the second autologous stem cell transplant in these patients. Our case presents skin GVHD developing after autologous SCT. However, since our patient has lymphoma and/or did not take a proteasome inhibitor before, it is important by distinguishing it from other cases. When the literature is examined,

it is thought that the preparation regimens and post-transplant CsA application prepare the ground for autologous GVHD. In addition, it is known that patients with hematological malignancies who are female, given high-dose CD34+ stem cells, bortezomib, lenalidomide, pomalidomide and alemtuzumab used in their pre-transplant treatments are at risk for autologous GVHD.

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OP 14

ACUTE GRAFT-VERSUS-HOST DISEASE (AGVHD) PRESENTING AS STEVENS-JOHNSON SYNDROME (SJS)

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Case report: This case report describes a 34-year-old male with AML, intermediate risk, initially treated with standard 7 +3 induction chemotherapy followed by high-dose cytarabine consolidation. Despite achieving medullary remission, minimal residual disease (MRD) persisted. The patient underwent allogeneic HSCT from an HLA-matched sibling donor. At the 33rd-month post-transplant, the patient, in full donor chimerism, developed a pituitary macroadenoma and hypopituitarism, alongside CNS relapse and medullary remission was confirmed in the bone marrow. Management included cranial radiotherapy and pituitary hormone replacement. Subsequent bone marrow relapse was treated with salvage chemotherapy (high-dose cytarabine and mitoxantrone), achieving medullary remission. Persistent CNS disease necessitated intrathecal triple therapy until cerebrospinal fluid clearance. MRI response was also obtained. A second allogeneic HSCT was performed from the same donor using a myeloablative FLU-TBI conditioning regimen. GVHD prophylaxis consisted of Cyclosporine-A and Methotrexate. On post-transplant day +25, the patient presented with severe cutaneous manifestations with some bullous lesions initially suspected as aGVHD or drug eruptions. The patient was initiated on a high-dose corticosteroid (2 mg/kg prednisolone) as a primary treatment. However, due to an inadequate response, ruxolitinib (10 mg twice-daily) was added to the treatment regimen after the first week. Dermatological evaluation raised suspicion of SJS, leading to IVIG administration. The skin biopsy report indicated the possibility of grade 2 GVHD, although the possibility of a drug reaction could not be excluded. Significant clinical improvement was observed within one week of ruxolitinib initiation. Corticosteroids were tapered over six weeks to physiological replacement doses. Ruxolitinib was continued for 56 days before gradual discontinuation. Cyclosporine was maintained with target trough levels of 100-250 ng/mL and