graphed ECV-15% total body volume. Patients are on a cardiac monitor and observed during the 3-5 hour treatment. ECP is performed 2 days in a row. Initially, we started every other week and now have moved to starting weekly as we have noticed faster improvement with this schedule. As symptoms improve, ECP schedule is tapered. Seven patients have been treated (median age 13 years (9-28), 4 males/3 females). Median duration of ECP: 11 months (1-26). The procedure is well tolerated with mild symptoms of hypovolemia noted in patients <25 kg. Weight at start of ECP was median 48 kg (22.2-79). Patients <25 kg have received about 1 pRBC transfusion/month. All patients had failed standard therapy with cyclosporine and prednisone in addition to a secondor third-line agent. All were on prednisone at start of treatment. Two patients have renal insufficiency. An attempt to lower prednisone dose and discontinue other immunosuppression was made after stabilization of cGVHD. Three patients expired due to cGVHD complications. Of the remaining 4 patients: 1 is too early to evaluate, all 3 others have responded, of which 2 have discontinued steroid therapy. Response by organ and steroid doses are listed below. All patients remain on a tapered schedule of ECP (every 3 or 4 weeks) since ECP discontinuation led to cGVHD flares in 2, which responded with reinstitution of ECP. The patients with scleroderma and oral sensitivity appear to have had significant benefit from ECP. ECP is feasible in pediatric patients with cGVHD if hct and hydration are carefully maintained at adequate levels for lower-weight patients. ECP appears to be efficacious for refractory cGVHD. Prospective studies are needed to define treatment duration and schedule, and benefits of using ECP concurrently with other immunosuppressants. Finally, we plan on demonstrating feasibility in patients <20 kg using a similar algorithm (Table 1).

Table 1. Pediatric Patients with Refractory CGVHD Treated with Extracorporeal Photopheresis

Age (yrs)	Duration (mos)	Symptoms at Diagnosis	Most Recent Symptoms	Intital and Most Recent PDN Dose, mg/ kg/d	Line Type	Outcome	Weight a ECP Star (kg)
12	7	Eyes, mouth, skin > 50%*	Eyes-SD. mouth- CR, Skin- PR	2 ightarrow 0.6	I 0F Double Lumen CVL	Dead	28.6
18	20	Liver	Liver-CR	1.6 ightarrow 0	10 F single lumen CVL	Alive and Well	48
20	П	Mouth, Skin > 75%, Liver	Mouth-PR, skin-PR, Liver-PD	0.5 ightarrow 0.5	9.6 F Vortex Port	Dead	79
П	3	Pulmonary	Pulmonary- PD	I → 0.5	10 F Double Lumen CVL	Dead	26
28	26	Skin > 50% (Scleroder	Skin-PR ma)	l → 0.25	9.6 F Vortex port	Alive and well	60.6
9	14	Skin > 50% (Scleroder	Skin-PR ma)	I → 0	5 F Yuey PIV Placed Before Each ECP	Alive and Well	22.2
13	I	Liver	Too Early to Evaluate	l → 0.67	I 2 F Double Lumen CVL	Alive and Well	61.5

SD = stable disease, CR = complete response, PR = partial response, PD = progressive disease, PDN = prednisone, *%body surface area.

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Poster Session II

Graves' disease (GD) is the most prevalent autoimmune disease in the United States with a peak incidence in the fifth to sixth decades of life and a female to male predominance of $\sim 10:1$. In recipients of allogeneic bone marrow and peripheral blood stem cell transplants, GD recurs in previously affected recipients, occurs in unaffected recipients by adoptive transfer of autoimmune thyroiditis from an affected donor, and occurs less frequently in unaffected recipients transplanted with unaffected donors. GD was diagnosed through laboratory surveillance in 3 male pediatric patients ages 5.5-14.3 years, 30-60 months after UCBT. All 3 affected patients received grafts from female donors and were full donor chimeras. None had graft-versus-host disease (GvHD) at diagnosis with GD. Two were transplanted for metabolic diseases (ALD, PNP deficiency) and 1 for ALL. All were conditioned with busulfan/ATG + cyclophosphamide (n = 2) or melphalan (n \leq 1) and received cyclosporine and methylprednisolone for prophylaxis against GvHD. All were off immunosuppression therapy for >1year at diagnosis of GD. Treatment with readioactive iodide (n = 2) and Propylthiouracil (n = 1) corrected laboratory abnormalities. The 3 boys were part of a cohort of 265 patients transplanted at our center between 9/1993 and 9/2004, surviving for >1 year post UCBT. Within this group, 55% of the patients had female donors and 45% had male donors. Interestingly, the three cases of GD occurred among the 86 male patients who received female-donor UUCBTs. No cases of GD were seen in female recipients. Neither the donors nor mother of donors developed autoimmune diseases. However, given their young ages, absence of disease does not rule out the possibility of development of this disease several decades into the future. The development of GD in 3 boys after UCBT from female donors is interesting. The etiology of Graves' Disease remains incompletely understood but genetic susceptibility contributes to the development of disease. One affected patient and its donor expressed the GD susceptibility allele, HLA-DRB1*0301, and conversely, a second affected patient and its donor expressed the GD protective allele, DRB1*0701. This could indicate the presence of other immunologic tendencies towards autoimmune disease in the cord blood donor or an alloreactive process from donor or low levels of maternal donor cells contaminating the UCB graft. Further studies will be necessary to determine the etiology of GD in these patients.

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RISK FACTORS FOR DEVELOPMENT OF SYMPTOMS AFTER AUTOLO-GOUS TRANSPLANTATION FOR MULTIPLE MYELOMA

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Background: Multiple myeloma (MM) is the most common indication for autografting in the United States. Although safe, autografting can be associated with substantial morbidity due to the toxic side effects of chemotherapy. Strategies aimed at minimizing symptoms post autografting may result in better tolerance. The risk factors for symptom development post autografting for MM have not been well characterized. Purpose: To define pretransplant conditions, which may be predictive of post-transplant symptom burden. Methods: We performed prospective evaluation of symptom burden among 64 myeloma patients undergoing autograft at MDACC as well as retrospective review of pretransplant variables including patient demographics, performance status, albumin, disease status, and Charlson Comorbidity Index (CCI). Univariate analysis was performed to correlate pretransplant variables with post transplant symptom burden as defined by M. D. Anderson Symptom Inventory (MDASI) scores at different time points post transplant. Results: 64 patients were studied from 6/2000 to 5/2003. Symptom burden increased from baseline to day 0 to nadir, with most patients returning to their baseline by day 30 post transplant. Table 1 summarizes the potential impact of pretransplant variables on median MDASI scores at nadir. Patients with the highest MDASI scores at baseline had the highest MDASI scores at nadir in quartile analysis (P = .001). Patients with Charlson score of ≥ 3 , age > 60, $\beta 2$ microglobulin ($\beta 2M$) > 3, albu-