



Figure 2. Lung fibrosis in NSG mice 8 weeks after transplantation
The lung from NSG mouse bearing PBMCs (A) or CD34+ cells (B) 8 weeks after transplantation was stained with Masson's trichrome. Fibrosis is shown in blue.

inflammatory cells, collagen deposition and expansion of airways in the lung despite showing no sign of acute illness or weight loss. Masson's trichrome revealed increased fibrosis in the lung (**Figure 2**), but not in other organs.

Conclusion: We have shown that in the NSG mouse, a combination of Cy/TBI with a low number of G-CSF mobilized human PBMCs causes chronic lung inflammation and fibrosis that can serve as an important pre-clinic model of lung cGVHD.

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GRAFT-Versus-Host Disease Clinical Profile and Duration of Immunosuppression Among Patients WHO Received Cord Blood STEM CELL Transplant: A Single Center Experience

Vaneuza Araujo Moreira Funke¹, Diogo Kloppel², Andresa Melo¹, Lisandro Ribeiro³, Carmem Bonfim², Elenaide Coutinho Nunes Sr.⁴, Caroline Sola⁵, Daniela C. Setubal⁶, Samir Nabhan², Michel Michels Oliveira², Ricardo Pasquini⁷, Mariester Malvezzi². ¹Hematology, Federal University of Parana, Curitiba, Brazil; ²Federal University of Parana, Curitiba, Brazil; ³Bone Marrow Transplantation Service, Federal University of Parana, Curitiba, Brazil; ⁴Nossa Senhora das Graças Hospital, Curitiba, Brazil; ⁵Stem Cell Transplantation, Hospital De Clinicas Da Ufpr, Curitiba, Brazil; ⁶BMT, Federal University of Parana, Curitiba, Brazil; ⁷Internal Medicine, Federal University of Parana, Curitiba, Brazil

Introduction: Transplants from cord blood stem cells is known to have lower incidence of graft-versus-host disease (GVHD). However, in patients who undergo cord blood transplant (CBT) and develop GVHD, its features are not well studied.

Objectives: Determine clinical features of GVHD and duration of therapy in patients who received CBT.

Patients and Methods: From 1993 to 2013, 196 patients received CBT were retrospectively analysed and divided into two categories. Group 1: 64 patients who developed GVHD. Group 2: 132 patients without this complication. Acute GVHD was graded according to Glucksberg criteria and Chronic Graft versus Host Disease was graded by NIH consensus criteria. Statistical analysis: Kaplan Meier (survival) and Fisher test (comparison of categorical variables). P level of significance was <0.05.

Results: Thirty three percent of patients developed GVHD (40 males and 24 females). Median age was 6 years old (1-31). 61 patients received CBT from a mismatched donor. Thirty (48%) were transplanted for malignancies. Five transplants were from a related and 59 from an unrelated

donor. Conditioning: Reduced intensity (RIC) in 6 cases and myeloablative in 58. Engraftment was complete in 48 cases (75%). Median survival in group 1 was 1832d (27-7283) versus 201d in group 2 (1-6242). Twenty nine patients have died. Forty one patients developed acute GVHD (aGVHD), 6 patients classic chronic GVHD (cGVHD) and 17 had an overlap syndrome. Grade II-IV aGVHD was seen in 49 cases (84.4%). Among cGVHD patients 9 (39.2%) were mild, 6 (26%) moderate and 8 (34.8%) severe. Median time for the onset of aGVHD was 23d (7-227) and cGVHD was 176d (64-659). The main sites of aGVHD were skin: 55(86%), gut: 22 (34%) and liver:14 (21%). Among cGVHD patients, 14 had skin (21%), liver:12 (18%), mouth:9 (14%), gut and lung (BO): 6(9%) each. Median time of cyclosporine therapy was 923d (7-3365). Steroids were used for a median time of 290d (8-4303). GVHD was less common in patients with a full match donor ($p=0,001$), those who used thymoglobuline ($p<0,0001$) and methotrexate (MTX) ($p=0,0133$). In contrast, GVHD rates were higher in patients who had an early ($p=0,0111$) and complete ($p<0,0001$) engraftment and had bacterial ($p=0,0133$) or viral ($p=0,0086$) infections during the pre-engraftment time. Survival rates were higher in patients who developed GVHD ($p=0,0256$), had a myeloablative conditioning regimen ($p=0,048$), children <14 yo ($p=0,0002$), patients who used cyclosporine for at least one year ($p<0,0001$) and full chimerism ($p<0,0001$).

Conclusions: We conclude GVHD can be frequent and even serious in CBT recipients. Risk factors included early and complete engraftment, mismatched donor, viral or bacterial infection during the pre-engraftment period, use of RIC and lack of MTX. Risk factors for survival were absence of GVHD, RIC, older age, and lack of full engraftment.

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Vitamin D Deficiency Predicts Acute Cutaneous Graft-Versus-Host Disease in Reduced-Intensity Allogeneic Hematopoietic Stem Cell Transplantation

Alex Ganetsky¹, Lee P. Richman², Noelle V. Frey³, Robert H. Vonderheide², David L. Porter³, Ran Reshef³. ¹Hospital of the University of Pennsylvania, Philadelphia, PA; ²Abramson Family Cancer Research Institute, Perelman School of Medicine, University of Pennsylvania, Philadelphia, PA; ³Blood and Marrow Transplantation Program, Abramson Cancer Center, Perelman School of Medicine at the University of Pennsylvania, Philadelphia, PA

Background: Acute graft-versus-host disease (GVHD) remains a leading cause of morbidity and mortality in allogeneic hematopoietic stem cell transplant (HSCT) recipients.