2-9). Acute GvHD (grade I, at day +47 and day +97 [patient who had prior autologous transplant with chronic GvHD]) was observed in 2 patients while mild chronic GvHD in 7 (<grade II, one due to donor lymphocyte infusion). With a median follow-up of 13.5 months (9.3-20), 12 patients were alive and 9 remained in CR. No transplant related mortality was seen but 2 AML patients had early relapsed (day +59, and +130). Conclusion: The NMT regimen with triple GvHD prophylaxis appears to be safe with no transplant related mortality. It may be an effective treatment for hematological malignancies in Asian patients with high risk features. Longer follow-up and additional patients are required to confirm the efficacy.

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**SEQUENTIAL ADMINISTRATION OF SARGRAMOSTIM (GM-CSF) AND FILGRASTIM (G-CSF) IN PEDIATRIC ALLOGENEIC STEM CELL TRANSPLANT (AlloSCT) RECIPIENTS UNDERGOING MYELOABLATIVE (MA) CONDITIONING: COST-EFFECTIVE AND MORE RAPID PLATELET RECOVERY IN UCB RECIPIENTS**


PNH is a clonal disorder caused by acquired mutations of the PIG-A gene. Many pts achieve long term survival if adverse factors are not present. SCT can be curative for young pts with severe pancytopenia, massive hemolysis, thrombotic complications or leukemic transformation. SCT complications are frequent reported due to an increased risk of graft failure and transplant related mortality. In this study we retrospectively analyze 15 pts with PNH submitted to SCT in our BMT center. Period: 03/88-02/05. Indication for SCT: severe pancytopenia: 12 pts, thrombosis: 2 pts and hemolysis: 1 pt. Age: 14-42 y (M: 29 y). Gender: 6F/9M. Time from diagnosis to SCT ranged from 2 to 133 months (M: 29.5 mo). Previous blood transfusions: 25 U1 (range: 8-200 U1). Total nucleated cell infused: 1.72±4.56 × 10^9/kg (median: 2.7). Donor type: HLA identical siblings: 14 pts, unrelated identical donor: 1 pt. Stem cell source: bone marrow: 14 pts; peripheral blood: 1 pt. Preparatory regimen: busulfan (BU) 12 mg/kg + cyclophosphamide (CY) 120 mg/kg: 11 pts (1 pt received BU 16 mg/kg), BU 8 mg/kg + fludarabine 125 mg/m²: 2 pts; CY 120 mg/kg + TBI + ATG: 1 pt and CY 200 mg/kg: 1 pt. GVHD prophylaxis: methotrexate + cyclosporine: 12 pts; others: 3 pts. Eleven pts are alive without evidence of PNH with a median follow-up of 1691 days (range: 125-3998 days). Estimated 5 year survival: 73%. Two pts died before day +28 and were not evaluable for engraftment, 13 pts engrafted and the median time to reach ANC >500/μl was 19 days (range: 14-25). Mucositis grade III-IV occurred in 7 pts. Two patients developed grade III-IV acute GVHD. One pt had progressive extensive chronic GVHD and another one had de novo extensive C-GVHD. No pt developed veno-occlusive disease. Four pts (median age of 36.5 yr) died on day +10, +11, +71 and +330 after SCT. Causes of death included infection (2 pts) and GVHD (2 pts). We conclude that pts with PNH and life threatening complications can achieve long term survival after SCT when HLA identical donors are used. In this group of patients we did not observe significant transplant related complications.

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**ALLOGENEIC STEM CELL TRANSPLANTATION (SCT) FOR THE TREATMENT OF 15 PATIENTS WITH PAROXYSMAL NOCTURNAL HEMOGLOBINURIA (PNH) IN BRAZIL**


PNH is a clonal disorder caused by acquired mutations of the PIG-A gene. Many pts achieve long term survival if adverse factors are not present. SCT can be curative for young pts with severe pancytopenia, massive hemolysis, thrombotic complications or leukemic transformation. SCT complications are frequent reported due to an increased risk of graft failure and transplant related mortality. In this study we retrospectively analyze 15 pts with PNH submitted to SCT in our BMT center. Period: 03/88-02/05. Indication for SCT: severe pancytopenia: 12 pts, thrombosis: 2 pts and hemolysis: 1 pt. Age: 14-42 y (M: 29 y). Gender: 6F/9M. Time from diagnosis to SCT ranged from 2 to 133 months (M: 29.5 mo). Previous blood transfusions: 25 U1 (range: 8-200 U1). Total nucleated cell infused: 1.72±4.56 × 10^9/kg (median: 2.7). Donor type: HLA identical siblings: 14 pts, unrelated identical donor: 1 pt. Stem cell source: bone marrow: 14 pts; peripheral blood: 1 pt. Preparatory regimen: busulfan (BU) 12 mg/kg + cyclophosphamide (CY) 120 mg/kg: 11 pts (1 pt received BU 16 mg/kg), BU 8 mg/kg + fludarabine 125 mg/m²: 2 pts; CY 120 mg/kg + TBI + ATG: 1 pt and CY 200 mg/kg: 1 pt. GVHD prophylaxis: methotrexate + cyclosporine: 12 pts; others: 3 pts. Eleven pts are alive without evidence of PNH with a median follow-up of 1691 days (range: 125-3998 days). Estimated 5 year survival: 73%. Two pts died before day +28 and were not evaluable for engraftment, 13 pts engrafted and the median time to reach ANC >500/μl was 19 days (range: 14-25). Mucositis grade III-IV occurred in 7 pts. Two patients developed grade III-IV acute GVHD. One pt had progressive extensive chronic GVHD and another one had de novo extensive C-GVHD. No pt developed veno-occlusive disease. Four pts (median age of 36.5 yr) died on day +10, +11, +71 and +330 after SCT. Causes of death included infection (2 pts) and GVHD (2 pts). We conclude that pts with PNH and life threatening complications can achieve long term survival after SCT when HLA identical donors are used. In this group of patients we did not observe significant transplant related complications.
Chemosensitive relapses after chemotherapy and autologous HSCT. Sources of HSCT included HLA-matched sibling (n = 41), matched unrelated donors (n = 6) and parents (n = 1). Forty-five patients received myeloablative and 3 received reduced intensity conditioning. The median EFS and OS were 38 and 40 months and at 160 months, the EFS and OS were 47% and 50%. When patients with diffuse large B-cell lymphoma were separately analyzed, the EFS and OS at 40 months were about 52% and none of the patients had disease relapse or mortality thereafter. Our results compared favorably with the NMDP Quality Standards subcommittee. The conclusion: We demonstrated that allogeneic HSCT may offer a cure for high risk NHL patients. Our results compared favorably with those reported in the literature.

119 RECIPIENT OUTCOMES DATA COLLECTION AND REPORTING FOR CORD BLOOD BANK FOLLOW-UP AND QUALITY ASSURANCE PURPOSES: THE NATIONAL MARRON DONOR PROGRAM® EXPERIENCE

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Cord blood banks (CBB) are required to monitor clinical outcomes of cord blood unit (CBU) transplants performed by transplant centers (TC). Obtaining accurate and timely follow-up data on clinical outcomes from the TC can be very difficult. The National Marrow Donor Program (NMDP), by virtue of their comprehensive activities with member network centers, collects outcomes data on recipients transplanted with either cord blood or adult donor cells. NMDP member transplant centers are required to submit data at defined intervals as part of their participation agreement. A system is in place to monitor compliance that includes a due process procedure for non-compliant centers. Recipients sign an IRB-approved consent form for data submission to the NMDP. All submitted data are verified through system checks at time of data entry. Identified errors are subject to a formal error correction process. Transplant centers are audited on a four year cycle to assure the accuracy and integrity of the data provided. At any given time, >90% of NMDP centers are compliant with forms submission requirements. The data elements include information on the results of the thaw, infusion related reactions, preparative regimens, neutrophil and platelet engraftment, GVHD, relapse, survival and death. Member cord blood banks receive a quarterly report from the NMDP which includes comprehensive data on the individual recipients for which their CBUs were used. The data include recipient demographics, infused cell dose, degree of HLA match, engraftment, GVHD, relapse, survival, and cause of death. While outcomes data are reported quarterly to member banks, thaw data are reported and reviewed on a continuous basis. TNC recoveries that are low (<60%) or high (>100%) are reviewed by NMDP staff to detect problems with CBU potency that might be linked to a certain bank, shipping procedure or thawing protocol at a TC. Future directions include trending analysis reports and linked to a certain bank, shipping procedure or thawing protocol at NMDP staff to detect problems with CBU potency that might be linked to a certain bank, shipping procedure or thawing protocol at a TC.

120 ADOPITIVE IMMUNOTHERAPY WITH TUMOR-DERIVED DONOR LYMPHOCYTES AFTER ALLOGENIC HEMATOPOIETIC STEM CELL TRANSPLANTATION

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Treatment of refractory or recurrent malignancy with donor lymphocyte infusion (DLI) after allogeneic hematopoietic stem cell transplantation (alloHSCT) is limited in efficacy, and graft-vs-tumor (GVT) is often accompanied by graft-versus-host disease (GVHD). After alloHSCT, lymphocytes infiltrating residual tumors are likely of donor origin. Compared with DLI, they may provide enhanced antigen specificity and maintain tumor-specific homing, thus generate better GVT with less GVHD. We are testing this hypothesis through administration of ex-activated tumor-derived lymphocytes (TDL) after alloHSCT. Clinical evaluation of TDL therapy was initiated with a 51-year-old woman for metastatic breast cancer whose disease progressed after matched-sibling alloHSCT and subsequent conventional therapy plus DLI. Metastases were surgically removed two weeks after administration of unmanipulated DLI. Lymphocytes were isolated from 9.4 cm of tumor and expanded for 14 days with anti-CD3/CD28-coated magnetic beads (3:1 bead-to-total nucleated cell ratio) and media containing IL-2 (100 or 1000 IU/mL). The cell products from the two culture conditions were similar. The process yielded 42.5 × 10⁶ cells, 33% expressing CD3, and generated 14.7 × 10⁹ TDL, 85% expressing CD3 (3.1-log T cell expansion). There was no tumor contamination of the T cell product by immunohistochemistry. Chimerism analysis revealed the TDL to be of donor origin. Flow cytometry showed an increase in the CD4/CD8 ratio from 1.3 to 1.9 after expansion. 76% of CD8 and 31% of CD4 cells secreted IFN-γ, and none secreted IL-2, IL-4 or IL-10. 76% of CD8 and 57% of CD4 cells expressed CXCR3. Three infusions of TDL were given in a dose-escalating manner (5, 25 and 100 × 10⁶ CD3⁺ cells/kg). Two additional infusions were given in conjunction with low-dose IL-2, the second of which was preceded by one cycle of paclitaxel and trastuzumab cytoreductive therapy. No infusion-related or delayed toxicities were observed. At the patient had no evidence of GVHD, even after the highest dose of 10⁸ allogeneic T cells. Evaluation of the remaining thoracic lesion demonstrated progressive disease after the first two DLI infusions, transient disease stability after the third and fourth infusions, and at present, the patient has stable disease one month after the fifth infusion. This is the first clinical report of the application of TDL and represents a novel approach for adoptive immunotherapy in the setting of alloHSCT.