2-9). Acute GvHD (grade I, at day +47 and day +97 [patient who had prior allogeneic transplant with chronic GvHD]) was observed in 2 patients while mild chronic GvHD in 7 (\leq grade II, one due to donor lymphocyte infusion). With a median follow-up of 13.5 months (9.5-20), 12 patients were alive and 9 remained in CR. No transplant related mortality was seen but 2 AML patients had early relaysed (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.

116

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

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G-CSF or GM-CSF can hasten myeloid engraftment post MA AlloSCT. GM-CSF is an earlier and broader acting CSF, which also induces more TH1. DC1 immune subsets preferentially compared to G-CSF. The objective of this study is to evaluate efficacy/safety of sequential administration of GM-CSF followed by G-CSF in children s/p MA AlloSCT. From 1/01-6/05 we enrolled 34 children: mean age 7.2 yrs; wt 32.3 kg; M:F = 19:15; poor:average risk = 13:21, TBI vs non-TBI based conditioning = 12:22; MFD vs umbilical cord blood (UCB) = 12:22. Indications for AlloSCT included malignant (MDS, ALL, AML, APL, NHL [n = 22]) and non-malignant (SAA, HLH, SCD, B-thal [n = 12]) disorders. GM-CSF (250 µg/m² IV QD) was initiated on day 0 post stem cell infusion. GM-CSF was switched to G-CSF (10 μ g/kg IV QD) when WBC \geq 300/ mm³ × 2 days. G-CSF was continued until ANC \geq 2500/mm³ × 2 days, then tapered to maintain ANC \geq 1000/mm³. GVHD prophylaxis: tacrolimus (target 5-20 ng/mL) and mycophenolate as previously described (Osunkwo/Cairo, BBMT 2004). All pts were prophylaxed against PCP, HSV, CMV, and fungal infections. There were no statistically significant differences in baseline characteristics between MFD vs UCB groups. Mean CD34⁺ cell dose/kg = 36.3×10^5 and TNC dose/kg = $39.3 \times$ 10^7 . Median time to WBC $\geq 300/\text{mm}^3 = 15.5 \text{ d}$ (10 vs 19.5 d MFD vs UCB). Median time of switch from GM-CSF (WBC \geq 300/mm³) to G-CSF and myeloid engraftment (ANC \geq 500/ $mm^3 \times 2$ d) was 0.5 d (1.5 vs 3.5 d MFD vs UCB). Median time to myeloid and platelet engraftment (untransfused count \geq 20000/mm³ × 7 d) were 17 d (13 vs 23.5 d MFD vs UCB) and 28 d (19 vs 33.5 d MFD vs UCB). Kaplan-Meier probability of 1 year OS was 67.2% (CI: 50.3-84.1). Adverse events attributable to G-CSF (n = 1) or GM-CSF (n = 3) were: bone pain, pleural and pericardial effusions. Of these, 3/4 pts received concomitant oprelvekin (IL-11). Infections occurring prior to day +60 included 21 (62%) pts with CVL bacteremia (coagulase (-) staphylococci [n = 9], gram (-) bacilli [n = 10]) and 2 (6%) pts with invasive fungal infections. This pilot study demonstrated safety and efficacy of sequential GM-CSF/G-CSF administration in pediatric AlloSCT recipients. Myeloid recovery after WBC $\geq 300/\text{mm}^3$ on GM-CSF only took 1-3 days after switch to G-CSF. Platelet engraftment following UCBT appears more rapid with sequential GM-CSF/G-CSF compared to historical controls. Sequential GM-CSF/G-CSF may be more cost-effective than G-CSF alone (cost-saving of \$1402/patient based on 2004 Red Book AWP).

ALLOGENEIC STEM CELL TRANSPLANTATION (SCT) FOR THE TREAT-MENT OF 15 PATIENTS WITH PAROXYSMAL NOCTURNAL HEMOGLO-BINURIA (PNH) IN BRAZIL

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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 v). Gender: 6F/9M. Time from diagnosis to SCT ranged from 2 to 133 months (M: 29.5 mo). Previous blood transfusions: 25 UI (range: 8-200 UI). Total nucleated cell infused: $1.72-4.56 \times 10^8$ /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: 125-5998 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 patents we did not observe significant transplant related complications.

118

ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANTATION FOR NON-HODGKIN'S LYMPHOMA IN QUEEN MARY HOSPITAL—A SINGLE CENTRE EXPERIENCE

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Objectives: Non-Hodgkin's lymphoma (NHL) is one of the major indications for haematopoietic stem cell transplantation (HSCT). For chemo-sensitive relapsed cases, autologous HSCT is often the treatment of choice. Relapsed cases after autologous HSCT as well as those with extensive disease generally have poor prognosis and allogeneic HSCT becomes the only option for a cure. We summarized our experience in Queen Mary Hospital regarding the outcome of allogeneic HSCT for this disease. Methods: Hospital records of allogeneic HSCT for underlying NHL in the past fourteen years were retrieved. Clinical data were summarized according to the pre-HSCT characteristics, disease relapse and transplant-related mortality. Event-free (defined as either disease relapse or death after HSCT) and overall survival were evaluated by Kaplan-Meier analysis. Results: Since 1991, a total of 48 NHL patients (M:F 25:23) have undergone allogeneic HSCT. The median age was 42 (range: 23-63 years). Histological diagnoses were obtained from 40 patients and they comprised both B-cell (Burkitt or Burkitt-like = 5, diffuse large B-cells = 19, follicular = 3, maltoma = 1, Mantle cell = 3) or \tilde{T} -cell lymphoma (angioimmunoblastic T-cell = 3, T-lymphoblastic = 4, peripheral T-cell = 2). Allogeneic HSCT was performed mostly as salvage therapy for