0%; MUD, Grade I-II: 20%, Grade III-IV: 20%. CIN of chronic GVHD was: MSD, limited: 11%, extensive: 0%; MUD, limited: 0%, extensive: 0%. Acute GVHD was treated to resolution with steroid and ongoing CSA therapy, and all patients weaned from immunosuppression by 1 year. There was no significant difference in regimen-related morbidity or mortality between MSD and MUD HCT using this risk-adapted modification of conditioning for MUD HCT. Though in a limited number of patients, our results suggest that immunomodulatory TLI/CTX/rATG conditioning is a promising option for MUD HCT in SAA patients. We propose to examine this in a prospective clinical trial using CTX/rATG for MSD HCT versus TLI/CTX/rATG for MUD HCT within 3 months of diagnosis for pediatric and young adult patients with idiopathic SAA.

79

ALLOGENEIC HEMATOPOIETIC STEM CELL TRANSPLANT IN SEVERE THALASSEMIA PATIENTS IN A SINGLE INSTITUION WITH RELATED AND UNRELATED DONORS AND MYELOABLATIVE AND REDUCED INTENSITY REGIMENS

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Recently published reports indicate that the outcome of unrelated donor hematopoietic stem cell transplantation (HSCT) in patients with leukemia is comparable with that of transplantation from identical family donors. We would like to prove that the outcomes of related and unrelated transplantation are favorably comparable in children with severe thalassemia. We reviewed transplant outcome in 87 consecutive children with severe thalassemia who underwent allogeneic HSCT the related-donor stem cells (n = 57) or unrelated-donor stem cells (n = 30) between September 1992 and July 2010. Analysis of engraftment, frequency of procedure related complications, and thalassemia-free survival revealed no advantage from use of related-donor stem cells. The thalassemia-free survival estimate for recipients of related-donor stem cells was 86% compared with 75% in the unrelated-donor stem cells group (p = 0.09). The present study provides and evidence to support the view that it is therefore quite reasonable to consider unrelated HSCT as an acceptable therapeutic approach in severe thalassemia, at least for patients who are not fully compliant with conventional treatment and do not yet show irreversible severe complications of iron overload. Moreover, we also have proved that our new reduced intensity conditioning regimen approach can overcome the past poor result of HSCT in class 3 lucarelli thalassemia (age $\geq$ 10 years) patients (n = 16). Both of the thalassemia free survival and overall survival rates in this group are 88%. Finally, we have studied the cost utility analysis between HSCT and hypertransfusion with iron chelation therapy in thalassemia patients. We can prove that matched related donor transplantation in thalassemia patients appears to be a cost effective and affordable treatment for thalassemia patients younger than 10 years of age.

80

CLINICAL OUTCOMES OF CHILDREN REQUIRING INTENSIVE CARE FOLLOWING HEMATOPOIETIC STEM CELL TRANSPLANTATION

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Clinical outcomes for children admitted to the pediatric intensive care unit (PICU) are improving with advances in supportive care strategies. We have reviewed outcomes of children admitted to PICU after hematopoetic stem cell transplantation (HSCT) to determine the likely outcome for this challenging group of patients. A retrospective review of our PICU and HSCT databases was performed from July 2004 through June 2009. Over this period, 136 HSCT recipients accounted for 260 PICU admissions. Eighty seven percent of patients (118/136) had allogeneic HSCT and 13% (17/136) autologous HSCT. Patients admitted to PICU were at increased risk of subsequent PICU admission, with an average of 2 PICU admissions per patient. Sixty six percent of transplants (90/136) were myeloablative, 32% (43/136) were reduced intensity and 2% (2/136) were without conditioning regimen (SCID patients). Eighty six percent of all allogeneic HSCT (102/118) were from unrelated donors. Eighty two percent of admitted patients (213/260) survived to PICU discharge. Forty three percent of patients are currently surviving with a median follow up of 2.9 years (1.1-6.1 yrs). The median PRISM II score for this cohort was 8 (0-47) days, and median length of stay was 4 days (1-172 days). Thirty nine percent of admitted (100/260) required intubation and mechanical ventilation and 58% survived to be discharged from the PICU. Forty two percent of admitted (109/260) required inotropes or vasopressor support and 66% of these cases survived to PICU discharge. Fourteen percent of admitted (36/260) required renal replacement therapy and 50% survived to PICU discharge. The LOS, PRISM II score, and ventilator days were all significantly lower for patients who survived to PICU discharge compared to patients who died during their PICU admission [LOS (median 3 vs. 13 days, $p < 0.001$), PRISM II score (median 7 vs. 13, $p < 0.001$), ventilator days (median 5 vs. 12 days, $p < 0.002$)]. In summary, we report 82% survival to PICU discharge, with overall 43% survival at a median follow-up of nearly 3 years for all HSCT patients admitted to our PICU. Our data suggest improved survival outcomes for this high risk patient population compared to historical values.

81

BLOOD AND NOT URINE BK VIRAL LOAD PREDICTS OUTCOME IN CHILDREN WITH HEMORRHAGIC CYSTITIS AND VIREMIA AFTER STEM CELL TRANSPLANTATION

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BK virus is a significant cause of hemorrhagic cystitis after stem cell transplantation (SCT), however, its role in nephropathy post-SCT is not well studied. We retrospectively evaluated 314 consecutive SCT patients treated at our institution since 2007, when BK PCR first became clinically available. BK studies were performed for hematuria or cystitis, not as routine viral screening. Thirty five SCT patients (11%) had documented hemorrhagic cystitis. Urine for BK virus was tested in 33/35 patients and was positive in 30 (91%). Average maximum urine viral load was ~8 billion copies/ml, and 73% of patients had a maximum urine viral load > 1 billion copies/ml. Sixty percent (21/35) of patients with hemorrhagic cystitis had plasma BK virus tested, and all of them had viremia (n = 21, 100%). Patients with a maximum plasma viral load of > 10,000 copies/ml (high viremia; n = 10) had worse renal outcome and greater mortality attributed to BK virus compared to patients with $\leq$ 10,000 copies/ml (low viremia; n = 11) (table). Specifically, patients with high viremia had significantly higher creatinine elevation from pre-SCT baseline. Sixty percent of patients with high plasma viral load required renal replacement therapy, and half of them developed end stage renal disease despite BK therapy. Three patients with high viremia had renal biopsies diagnostic for polyoma virus-associated nephropathy. Hemorrhagic cystitis was also more severe in the high viremia group, with half of patients requiring surgical interventions and bladder irrigations for obstructive uropathy. Conversely, the majority of patients with low viremia had transient elevations in creatinine and hemorrhagic cystitis that resolved over time. In summary, BK viremia > 10,000 copies/ml was associated with significant nephropathy and severe hemorrhagic cystitis following SCT in our patient cohort. Although majority of SCT patients with hemorrhagic cystitis had very high urine viral load, only patients with high viremia developed nephropathy, end stage renal disease and died from this infection. Therefore, the degree of viremia, but not viruria, may identify patients at risk who would benefit from early or aggressive therapy. Future studies should determine optimal monitoring and treatment protocols for these patients.