

to collect while no patient (0%) without previous LEN exposure failed to collect, including 3 pts who received melphalan ($p = 0.105$). Total number of CD34+ cells collected after 2 apheresis sessions for group B = 8.13×10^6 /kg CD34+ cells and group A = 3.34×10^6 /kg CD34+ cells ($p = 0.06$).

Conclusion: Chemomobilization with CTX + filgrastim yields robust PBSC collections irrespective of antecedent lenalidomide. There was a trend towards lesser PBSC collection in LEN-treated pts. Due to retrospective design and limited number of pts, further research is needed to elucidate the effect of chemomobilization on PBSC collection in LEN-treated pts.

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ADDITION OF RITUXIMAB TO EITHER BEAC OR BEAM IN THE PREPARATIVE REGIMEN PRIOR TO AUTOLOGOUS STEM CELL TRANSPLANTATION IN PATIENTS WITH RELAPSED B-CELL NON-HODGKIN LYMPHOMA DOES NOT ADD A SURVIVAL BENEFIT: A SINGLE CENTER EXPERIENCE

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High-dose chemotherapy with carmustine, etoposide, cytosine arabinoside, and melphalan (BEAM) or cyclophosphamide (BEAC) followed by autologous hematopoietic stem cell transplantation (ASCT) represents a standard therapy for many patients with relapsed non-Hodgkin lymphoma (NHL). Whether or not the addition of rituximab to either preparative regimen (R-BEAM or R-BEAC) provides a benefit when given prior to ASCT is unclear.

In an attempt to compare the efficacy, outcome, and toxicity of BEAM (with or without rituximab) ($n = 36$) and BEAC (with or without rituximab) ($n = 21$) in patients with NHL undergoing ASCT, we retrospectively evaluated our center's experience between January 2007 and April 2010.

Out of 57 patients with relapsed NHL (median age 56 y; range 25-72 y), 13 patients received BEAM ($n = 6$) or BEAC ($n = 7$) and 44 patients received R-BEAM ($n = 29$) or R-BEAC ($n = 15$) prior to ASCT. All patients received rituximab containing salvage chemotherapy prior to transplantation.

The probability of disease-free survival (DFS) (80% in BEAM/BEAC versus 70% in R-BEAM/R-BEAC group; $p = 0.69$) and overall survival (OS) (68% in BEAM/BEAC versus 80% in R-BEAM/R-BEAC group; $p = 0.31$) were comparable in both the groups at 2 years. The probability of DFS (100% in BEAC versus 70% in R-BEAC group; $p = 0.55$; and 71% in BEAM and 69% in R-BEAM group; $p = 0.47$) and OS (100% in BEAC versus 92% in R-BEAC group; $p = 0.54$; and 60% in BEAM and 80% in R-BEAM group; $p = 0.22$) were also comparable at 2 years.

There was no difference in engraftment kinetics, grade III-IV toxicities, and average length of stay among the different groups.

In conclusion, the addition of rituximab to either preparative regimen did not add any benefit to our patients undergoing ASCT for B-cell NHL. It is possible that the universal use of rituximab with prior salvage therapies might have abrogated the benefit of R-BEAM/R-BEAC over BEAM/BEAC in this study. Whether or not the addition of rituximab to the preparative regimens prior to ASCT in patients with NHL truly adds any benefit needs to be studied in a larger prospective trial.

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APREPITANT: TREATMENT IN HAEMATOPOIETIC STEM CELL TRANSPLANTATION (HSCT) PATIENTS REFRACTORY TO FIRST AND SECOND LINE ANTIEMETICS

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Chemotherapy induced nausea and vomiting (CINV) is a distressing side effect of the transplant procedure. Evidence based guidelines exist to direct antiemetic prescribing for the prevention of CINV. There is minimal research providing guidance in the management patients with delayed emesis despite appropriate preventative strategies for CINV, particularly after HSCT. Aprepitant is a neurokinin-1 antagonist, which in combination with 5HT₃ antagonists and dexamethasone is effective in

preventing acute and delayed emesis after highly emetogenic chemotherapy. Aprepitant has been utilised in HSCT for prevention of CINV.

Aim: To assess the efficacy of aprepitant for the treatment of refractory emesis following HSCT.

Methods: Patients transplanted between Sept 2008 to Aug 2009, experiencing more than one emetic episode or persistent nausea score $> 5/10$ for more than 24 hours after review of antiemetic therapy were included. Aprepitant was administered in addition to current antiemetic therapy, as either 115mg IV daily or 125mg po stat and then 80 mg daily. The number of vomiting episodes, dry retches, nausea score on a ten point scale and breakthrough antiemetic use was recorded at baseline and daily until aprepitant ceased. A complete response was defined as no emetic episodes, nausea score of 0/10 and no breakthrough antiemetic use. Major response was defined as no emetic episodes with reduction of nausea score from baseline of at least 50% and partial response as no emetic episodes but reduction in nausea score of $< 50\%$.

Results: During the audit period 14 patients were prescribed aprepitant to treat emesis. A complete response was seen in 4/14 (28%), major response in 2/14 (14%) and partial response in 4/14 (28%) with an overall response rate of 10/14 (71%). Responses were sustained.

Conclusion: The addition of aprepitant can prevent emesis and reduce nausea score in HSCT patients refractory to first and second line antiemetic therapy.

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WHAT GOES UP MUST COME DOWN: VORICONAZOLE AND CYCLOSPORIN IN A PAEDIATRIC CORD AND MARROW TRANSPLANT UNIT

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Voriconazole's addition to the antifungal armamentarium has been revolutionary. Its use in invasive fungal infection is of particular utility in haematopoietic stem cell transplant (HSCT). Voriconazole inhibits hepatic enzymes placing HSCT patients at risk of drug interactions and toxicity from calcineurin inhibitors. Limited safety and efficacy data exist for pediatric HSCT warranting investigation.

Objective: To investigate the safety and efficacy of voriconazole in a pediatric HSCT unit 2008-10.

Method: A retrospective chart review of 43 consecutive allogeneic transplants identified patients who received voriconazole from conditioning until Day 100.

Results: 39 out of 43 (90.7%) patients are alive with a median follow up of 17 months (1-32). There were no deaths from fungal infection. 42 of 43 patients received fungal prophylaxis. 35 patients were classified as low risk of infection and received fluconazole. 7 patients were high risk due to pre transplant infection and received voriconazole. 1 patient on voriconazole developed mucormycosis and was effectively treated with posaconazole. 2 patients had suspected fungal disease and had treatment with another agent. 4 patients continued on prophylaxis with no demonstration of fungal disease. 13 of 43 (30.2%) fluconazole prophylaxis patients changed to voriconazole treatment for suspected fungal infection with symptom resolution in all patients.

Voriconazole was well tolerated. 1 patient withdrew due to toxicity (confusion, rash). Hepatic dysfunction (GGT 2x normal) was significantly higher in the voriconazole group ($p < 0.01$). There was no difference in transaminitis across the two groups.

On voriconazole initiation, cyclosporin dose was modified on a risk adjusted basis. Cyclosporin level, creatinine, nephrotoxic agents and GVHD were used to inform decision. There was no difference in cyclosporin levels or renal impairment (2 x baseline CR urea) in the voriconazole group compared to those who received other agents.

Conclusion: Safety and efficacy data are limited for new antifungals when used with standard medications in pediatric HSCT. This small series demonstrated voriconazole has acceptable toxicity and can be prescribed without increasing cyclosporin toxicity. Currently, a dosing algorithm is under evaluation to determine cyclosporin dose

adjustment without compromising efficacy. Drug interaction complexity with new agents and lack of pediatric data highlights a need for collaboration between HSCT units.

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RETROSPECTIVE ANALYSIS OF WEEKLY INTRAVENOUS IMMUNOGLOBULIN PROPHYLAXIS VERSUS INTRAVENOUS IMMUNOGLOBULIN BY IGG LEVEL MONITORING IN HEMATOPOIETIC STEM CELL TRANSPLANT RECIPIENTS

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Background: Patients undergoing hematopoietic stem cell transplant (HSCT) may have a higher incidence of infections due to secondary hypogammaglobulinemia. A recent meta-analysis evaluated intravenous immune globulin (IVIG) in a prophylactic setting and concluded routine use had no benefit in survival or infection prevention in HSCT. The study suggested monitoring of IgG levels and replacing with IVIG in high risk patients.

Methods: All allogeneic HSCT patients who received prophylactic IVIG 0.2 gm/kg IV once weekly (n = 115) from admission to day +90 were compared to patients who received IVIG based on low IgG levels (n = 114) between 1/09 and 8/09. The IgG levels were drawn upon admission, day +30, day +60, and day +90. Utilization of IVIG, incidence of veno-occlusive disease (VOD), graft versus host disease (GVHD), and documented infections were recorded during the first 100 days after transplant.

Results: The weekly control group (n = 115) had a median age of 49 compared to 54 years in the IVIG by level group (n = 114). No significant difference in type of transplant, except a higher number of matched un-related donors (MUD) in the by level group (62 vs 41, p = 0.01). There were no significant differences in occurrence of GVHD (55 vs 50), VOD (2 vs 0), or in infections such as RSV (3 vs 1), VZV (1 vs 0), HSV (3 vs 8), Adenovirus (2 vs 1), polyoma/BK virus (18 vs 26), bacterial infection (49 vs 38), or fungal infection (12 vs 7) in weekly versus by IgG levels respectively. A higher incidence of para-influenza occurred in the weekly group (9 vs 0, p = 0.003) correlating with flu season. IVIG cost in the weekly control group totaled AWP \$924,408 vs \$252,547, with overall savings of \$671,816 in the IVIG by level group.

Conclusion: With no difference in major complications, rate of infection, and a significant savings in IVIG use, a change in institutional practice was implemented. IgG levels are now monitored monthly and replacement is done based on low IgG level.

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RETROSPECTIVE ANALYSIS OF THE INCIDENCE OF SYMPTOMATIC VEIN THROMBOEMBOLISM AMONG PATIENTS WITH HEMATOLOGIC MALIGNANCY

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A growing body of evidence shows similar or increased venous thromboembolism (VTE) risk in specific hematologic malignancies, but few recommendations for VTE prophylaxis in hematologic malignancy exist. This study will investigate the incidence and trends of VTE among hematologic malignancy patients to identify patients at higher risk of VTE.

Methods: Clinical data was collected retrospectively from 200 admissions to Methodist University Hospital from December 2008 to October 2009 with primary or secondary diagnoses of hematologic malignancy. The primary objective is the incidence of symptomatic VTE. Secondary objectives include rate of appropriate VTE prophylaxis, risk of VTE in patients receiving VTE prophylaxis, subgroup analyses to determine populations at higher risk for VTE, length of stay with occurrence of symptomatic VTE, incidence of VTE surrounding implementation of hospital-wide VTE prophylaxis protocol, and all-cause mortality versus possible VTE-associated deaths.

Results: A total of 144 patients were represented in 200 admissions included in this study. The most common diagnoses included were non-Hodgkin's lymphoma (NHL, 24.3%), multiple myeloma (MM, 24.3%), and acute myelocytic leukemia (AML, 18.1%), and a majority of patients were African American. In April 2009, an automated screening tool for VTE prophylaxis was implemented that both assesses VTE risk and bleeding risk and offers three elective therapy options for VTE prophylaxis. Admissions were stratified according to the implementation date (100 before, 100 after protocol). Twelve VTE events were observed across all 200 admissions (6%). Nine VTE occurred prior to the protocol and 3 occurred after the prophylaxis protocol, not statistically significant. NHL (13%), chronic myelocytic leukemia (CML, 12.5%), and Hodgkin's lymphoma (HL, 8.3%) had the highest rates of VTE. VTE prophylaxis use increased by 160% after implementation of the VTE prophylaxis protocol, (p = 0.003). Use of VTE prophylaxis was not associated with any decreased risk of VTE or length of stay.

Conclusion: Incidence of VTE among the local malignant hematology patient population is similar to previously reported data, with highest VTE rates found in those patients with NHL, CML, and HL. The apparent lack of efficacy of VTE prophylaxis in this study may reflect either inaccurate current definitions of adequate VTE prophylaxis or the practice of holding anticoagulants in the setting of thrombocytopenia.

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VANCOMYCIN-RESISTANT ENTEROCOCCUS FAECIUM SURVEILLANCE AND INFECTION IN AUTOLOGOUS AND ALLOGENEIC TRANSPLANTATION

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Introduction: Colonization with Vancomycin-resistant Enterococcus Faecium (VRE) places patients at increased risk for VRE infection. Variable rates of colonization and infection have been reported for hematopoietic stem cell transplantation recipients. Herein we review VRE colonization rates amongst 882 autologous and allogeneic HSCT recipients and assess for risk factors associated with the development of VRE infection.

Methods: Medical records for 822 consecutive HSCT recipient inpatient admissions between 8/2004 to 8/2008 were reviewed for data. Pharmacy records were utilized to obtain data for medication. All patients were screened for VRE upon admission and weekly thereafter. All patients received acyclovir, azole antifungals and ciprofloxacin as prophylaxis. Positive VRE BAL cultures were excluded from infection analysis. Chi-square analysis was used for statistical analysis. This study was approved by NMH IRB.

Results: For 822 HSCT recipients reviewed (207 allogeneic and 615 autologous), new colonization developed in 136 patients (17%), while 41 patients were VRE colonized prior to admission. Overall 28 patients (3.4%) developed VRE infection (urine = 11, blood = 17, multiple = 3). No correlation was observed between age, sex, or administration of a neutropenic diet and VRE colonization or infection. Allogeneic transplant (p = .013), especially melphalan based RIST was associated with increased VRE infection (p = .011). Co-infection with *Strept Viridans* occurred more frequently in VRE infected patients (p = .0001). Prior VRE colonization occurred in 15/28 patients (54%) who developed VRE infection compared to 13/651 who did not (p = .0001). Median time to infection was 10 days. A correlation between C.Diff infection and VRE colonization was identified (p = .0014). Almost all patients received wide spectrum *B-Lactam* antibiotics and vancomycin, which may explain the lack of correlation between these antibiotics and the development of VRE, however prior exposure to caspofungin was observed more frequently in patients with VRE infection (p = .0001). All treated patients received linezolid. Time to engraft was the same for auto and all patients, with or without VRE. Crude in-patient overall mortality was significantly higher in patients with VRE infections (p = .0002).

Conclusion: VRE colonization and infection increase morbidity and mortality post HSCT. Heightened awareness and correction