thrombotic complications had decreased plasma ADAMTS13 activity ($P < .01$) and increased VWF antigen level after pretreatment ($P < .01$) as compared with the non-thrombotic patients; three out of 8 (37.5%) showed more than 60% decrease in plasma ADAMTS13 activity. The level of ADAMTS13 activity dropped in the 49 patients with aGVHD as compared with healthy controls ($P < .01$), but there was no significant difference between patients with and without aGVHD. Twenty-five patients showed decreased plasma ADAMTS13 activities only at the onset of aGVHD occurrence ($P < .01$), in which two of them decreased more than 60% (6%). Logistic regression analysis showed that the ADAMTS13 activity declined by more than 60% was the risk of thrombosis ($P < .01$).

Conclusions: We observed decreased plasma ADAMTS13 activity and increased plasma level of VWF antigen in patients following HSCT after pretreatment, especially in the patients with thrombotic complications. A decrease more than 60% in plasma ADAMTS13 activity is the risk factor of thrombotic complications. Therefore, the plasma ADAMTS13 activity could be an important parameter for the development of vascular disorder, which has a potential role for the early diagnosis of thrombotic complications.

365

Retrospective Survey of Hemostatic Complications in 680 Patients Undergoing Hematopoietic Stem-Cell Transplantation (HSCT)

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Background: Hemostatic disorders are common and potentially fatal complications in patients undergoing hematopoietic stem-cell transplantation (HSCT). Limited data exist on early diagnosis and prevention of these complications. In this study, we retrospectively investigated the outcome and risk factors associated with thrombotic and bleeding complications in HSCT recipients.

Methods: From April 2004 to December 2011, 680 hematologic patients receiving HSCT were enrolled in the study, and their clinical manifestation and laboratory parameters were analyzed for evaluating the outcome of hemostatic complications and related risk factors.

Results: Overal incidence of thrombotic complication, which included 12 veno-occlusive diseases (VOD), 2 transplantation related thrombotic microangiopathy (TA-TMA), 1 pulmonary embolism (PE) and 1 deep vein thrombosis (DVT), was 2.4% (16 cases). The overall mortality after thrombotic events was 68.8% (11 cases) in all HSCT recipients with thrombotic complications. A total of 510 HSCT recipients (72.5%) developed bleeding events, including minor bleeding of 67.1%, moderate bleeding of 28.4%, and severe bleeding of 3.9% of all bleeding patients. By bleeding sites, 218 patients developed hemorrhagic cystitis. Other organs of hemorrhage involved skin or mucosa (46.5%), gastrointestinal tract (21.1%), vagina (9.3%), and respiratory tract (1.3%). By risk factors analysis, CD33 mAb use and preparative regimen containing total body irradiation were significantly associated with the occurrence of thrombotic disorders ($P < .05$). Thrombocytopenia, grade 2-4 acute graft-versus-host disease (aGVHD), allogeneic transplantation and infection were independent risk factors for bleeding complication ($P < .05$). Polyomavirus and grade 2-4 aGVHD were risk factors for hemorrhagic cystitis ($P < .05$). The number of hemorrhagic sites was significantly correlated with bleeding severity ($P < .05$). Neither thrombotic nor bleeding disorders was correlated with age, disease category, gender, transplantation types, routine hemostatic parameters, or biochemical indicators. Survival rate was correlated with the bleeding site and intensity of bleeding disorders ($P < .01$). Respiratory and gastrointestinal bleeding independently increased the mortality of HSCT recipients, while overal cumulative survival was decreased in patients with thrombotic complications. In addition, PAI-1 level in the HSCT recipients with thrombotic complications were significantly higher than other complications ($P < .01$).

Conclusions: Our study suggested that HSCT patients with thrombotic complications experienced high mortality while the HSCT recipients with bleeding disorders had high morbidity. Hence, early diagnosis and therapy of hemostatic complications are crucial to improve the prognosis of HSCT recipients.

366

Predictive Classification of Chronic GVHD Status by Immune Reconstitution Testing

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Background: Many individual and treatment-related factors impact post-allogeneic hematopoietic cell transplant (HCT) immune reconstitution (IR). Our institution measures lymphocyte subsets/activation status with 8-color flow cytometry panels at important milestones (e.g., development of GVHD, day +365 evaluation), allowing for sensitive monitoring of GVHD and post-HCT IR. We sought to identify whether previously unrecognized patterns of CBC data and lymphocyte subsets are associated with GVHD status.

Methods: 288 allogeneic HCT recipients have undergone clinical IR testing at our institution since 2010. Included in this analysis are 43 HCT patients who underwent testing for IR with our updated antibody panel (CD3, CD4, CD8, CD8beta, CD19, CD25, CD27, CD45, CD45RO, CD56, CD69, IgD, and HLA-DR). Patients were classified according to clinical status: no GVHD (9), active acute (3), resolved acute (6), active chronic (18), and treated chronic GVHD (7). Differences in cell populations were determined by Kruskall-Wallis tests. Multivariate pattern recognition techniques of principal components/factor analysis (PCA/FA) and discriminant analysis (DA) were used for exploratory predictive modeling.

Results: Patients with active chronic GVHD had a significant increase in CD4-CD8- T cells ($P = .01$) and trended towards increased in NKT cells ($P = .06$). PCA/FA revealed 3 factors that account for 88% of the variability of the IR data (Factor 1=CD4+, CD8+, and naïve T cells, Factor 2=naïve and switched memory B cells, and Factor 3=absolute lymphocyte counts [ALC] and platelet counts). By applying all variables from the 3 factors in a DA model (Table), the ability to discriminate between active vs. no GVHD approached statistical significance ($P = .06$). Adding CD4-CD8- T cells and NKT cells to the model did not allow for discrimination of active vs. treated chronic GVHD. Lymphocyte activation (e.g., by HLA-DR or CD69 expression) could be identified in a few patients who did not have overt GVHD or infections.