Table 1.

	Age	Nadir wbc	Nadir plt	Days to wbc>1	Days to CD34>10, median (quartiles)	Days between wbc>1 to 34>10, median (quartiles)
Good (n=370)	58	0.3	68	11	12 (11-13)	0 (0-1)
Poor (n=26)	64	0.2	30	12	13 (13-16)	2 (2-3.5)
P value	0.003	0.07	0.02	0.03	0.01	0.0005
Optimal (n=340)	58	0.3	67	11	12 (11-13)	0 (0-1)
Suboptimal (n=30)	65	0.3	83	12	14 (11.75-15)	1.5 (0.75-2)
P Value	>0.05	0.3	0.5	0.1	0.0001	0.0001
Optimal <3apheresis (n=307)	58	0.3	67	11	11 (11-13)	0 (0-0)
Optimal >3 apheresis (n=33)	59	0.4	80	12	14 (12-16.75)	2 (0-3.75)
P Value	>0.05	0.2	0.9	0.2	0.0001	0.0001

Good: CD34+cells>2x106/kg, Poor: CD 34+ cells <2x106/kg, Optimal: CD34+cells >5x106/kg, Suboptimal: CD34+cells: 2-5x106/kg

factors for outcome CY-mob. If a higher CD34+ cell count is targeted for transplantation (> $5x10^6$/kg), the delay in the days to CD34+ cells > 10 in peripheral blood can be considered to be a reason for modifying the mobilization regimen, like adding plerixafor. If the CD34 count is still < 10 one day after the day WBC > 1.0, addition of plerixafor to the mobilization regimen should be strongly considered.

134

AUTOLOGOUS STEM CELL TRANSPLANTATION IN MULTIPLE MYELOMA IN THE ERA OF NOVEL AGENTS: IMPACT OF DEPTH OF RESPONSE TO INDUCTION THERAPY. A SINGLE INSTITUTION EXPERIENCE

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Background: High-dose chemotherapy and autologous stem cell transplantation (ASCT) in newly diagnosed multiple myeloma is the standard of care for eligible patients. Prior to ASCT, induction therapy is administered for 1 to 4 months before stem cell collection. Eligible patients undergo ASCT regardless of the response to induction therapy because it has been shown that a good response is not essential in order to obtain survival benefit from ASCT. A recent study that evaluated the novel agents, thalidomide and lenalidomide demonstrated that failure to achieve a partial response after induction therapy predicts a poorer outcome after ASCT.

Methods: Fifty patients (60% male; median age 59 years) with multiple myeloma who underwent high-dose chemotherapy and ASCT after induction therapy with thalidomide, lenalidomide or bortezomib were retrospectively studied. The patients were divided into two groups according to their response after induction therapy: those with CR or VGPR; and those with PR or less than PR. Statistical analyses were performed.

Results: The median follow up was 33.4 (2.9-71.2) months. The M-protein at diagnosis was 3.4 ± 1.7 g/dl and the stage (Durie-Salmon) of the multiple myeloma was IIIa in 33 (66%) and IIIb in 9 (18%) patients. Beta-2 microglobulin was 4.9 ± 4.7 g/ml and 40% of patients had unfavorable cytogenetics defined as 13q- by cytogenetics, t (4,14), t(14,16), 17p- and complex karyotype. Favorable cytogenetics included normal, t(11,14) or 13q- by FISH. The induction therapy was thalidomide in 23 (46%), lenalidomide in 9 (18%), bortezomib in 8 (16%) and a combination in 10 (20%). The patients who achieved CR or VGPR after induction therapy were 23 (46%). These responses were associated with longer time to progression (HR = 0.42; p = 0.055) after ASTC. The progression-free survival (PFS) estimates for CR or VGPR vs PR or < PR were 48.6 months and 21.5 months respectively (p = 0.013). The presence of favorable cytogenetics was associated with a longer time to progression (HR = 0.345; p = 0.0174). A higher beta-2 microglobulin was associated with earlier time to progression (HR = 1.073; p = 0.0272).

Conclusion: Achieving CR or VGPR with the novel agents prior to ASCT predicts a better PFS after transplantation. This suggests that extending induction therapy to achieve CR or VGPR prior to ASCT might be beneficial. Larger studies are needed to confim this finding.

135

CARDIOMYOPATHY FOLLOWING HIGH DOSE MELPHALAN CONDITIONING PRIOR TO AUTOLOGOUS PERIPHERAL BLOOD STEM CELL TRANSPLANTATION FOR MULTIPLE MYELOMA AND PRIMARY AMYLOIDOSIS

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Background: High dose melphalan (HDM) is a commonly utilized conditioning regimen prior to autologous peripheral blood stem cell transplantation (PBSCT) for multiple myeloma and primary amyloidosis. Although cardiotoxicity from this regimen has not been described, there are multiple anecdotal cases of transient cardiomyopathy following HDM in the Mayo Clinic transplant experience. Thus, we reviewed our experience to determine the frequency of cardiomyopathy in this setting.

Methods: From our database of all patients undergoing autologous PBSCT for myeloma or amyloidosis from 1989-2009, we identified those with echocardiograms performed before and within 4 months of PBSCT, and reviewed these for evidence of cardiomyopathy, defined as a decrease in left ventricular ejection fraction (LVEF) of ≥ 10% to a value of ≤ 50%. Charts of patients meeting these criteria were reviewed to determine conditioning regimen, pre-existing cardiac risk factors and other potential causes of cardiomyopathy to determine the association between the cardiomyopathy and HDM.

Results: Of 1476 patients (1050 myeloma; 426 amyloidosis) who received HDM as a component of conditioning prior to PBSCT, 407 had echocardiography before and within 4 months after PBSCT, and 40 (2.7%) met criteria for cardiomyopathy. When accounting for other risk factors, HDM was felt to be a probable cause of cardiomyopathy in 16 patients, a possible cause in 18, and an unlikely cause in 6. Cardiomyopathy was noted more frequently in patients undergoing PBSCT for amyloidosis (23/426, 5.4%) than myeloma (17/1050, 1.6%). All 23 patients with amyloidosis received HDM alone as conditioning (dose range 140-200 mg/m^2). Fifteen of the 17 myeloma patients received HDM alone as conditioning (dose range 140-200 mg/m^2), with two patients receiving HDM in concert with total body irradiation and ibritumomab, respectively.

The mean pre-PBSCT LVEF in this cohort was 59% (range 42-70%), which decreased to a mean of 38% (range 15-50%) following PBSCT. Diagnosis of cardiomyopathy was made within 30 days of PBSCT in 65% (26/40) of patients. Follow up echocardiography was performed in 26 patients, with 15 showing improvement in LVEF to ≥ 50%.

Conclusion: To our knowledge, this is the first series to report cardiomyopathy following HDM conditioning. Further work should focus on identifying pre-transplant variables which may predispose patients to cardiotoxicity following conditioning with HDM.