

bocytopenia. No patient experienced fever or infections or required transfusions. The majority of the patients complained of mild to moderate back pain easily controlled by oral analgesics. **Conclusions.** This study shows that a single fixed dose (6 mg s.c.) of PEGf is safe and effective to mobilize adequate number of CD34+ cells in the majority of myeloma patients, a category usually considered worse mobiliser than patients with other hematological malignancies. In addition, the single administration of PEGf shows better compliance than repetitive doses of filgrastim.

1227**EXANTHEMA AND HERPES ZOSTER INFECTION DURING VELCADE USE INCIDENCE, TREATMENT AND PROFYLAXIS**

L.P. Pour, R.H. Hajek, Z.A. Adam, M.K. Krejci, A.K. Krivanova, Z.L. Zahradova, T.B. Buchler, J. Vorlicek

Faculty hospital Brno, BRNO, Czech Republic

Backgrounds. Bortezomib has been shown to be highly effective in the treatment of relapsed multiple myeloma (MM). Data from clinical trials show that the incidence of herpes zoster during bortezomib therapy is about 13%. Skin rash is quite common toxicity seen in MM patients treated with bortezomib. Where reported, its incidence in clinical trials ranged from 8 to 18%. We reported our results and treatment of this two adverse effects of bortezomib treatment. **Methods.** From December 2004 we treated 48 relapsed MM patients with bortezomib. Patients were treated with standard dosage schedule (intravenous infusions of bortezomib 1.3 mg/m² on days 1, 4, 8, and 11 of a 21-day cycle). **Results.** Our first ten patients treated with bortezomib did not receive varicella-zoster virus (VZV) prophylaxis and herpes zoster developed in three of these patients (33%). Clinical manifestation of herpes zoster was typical, starting with itching and pain and exanthema appearing later. All patients were treated for the 2nd relapse of MM. Herpes zoster developed in the first patient during the 3rd cycle, in second patient during 6th cycle and in third patient during the 8th cycle of bortezomib. Therapy with bortezomib was subsequently interrupted and all three patients received treatment with acyclovir intravenously. Based on experience, we started to use prophylaxis with acyclovir 400 mg per os 3 times daily during bortezomib therapy. We did not note any VZV reactivations in 36 consecutive patient receiving VZV prophylaxis. This group also included five patients who already had VZV reactivation before bortezomib treatment. In 12 of 48 patients (25%), rash developed during the second treatment cycle. The first cycle of bortezomib was well tolerated in all cases. Skin biopsy was done in first three patients, in all cases perivascular lymphoid infiltrates were found. Rash resolved rapidly in all cases after treatment with prednisone 20 mg/day. Two patients were treated with prednisone and cetirizine (10 mg/day). After resolution of rash, prednisone was discontinued, but rash recurred with the next bortezomib infusions despite continued treatment with cetirizine. To prevent recurrence of the rash, it was necessary to administer corticosteroids (10 mg prednisone) prophylactically before every administration of bortezomib. Two patients with bortezomib-associated rash were treated with dexamethasone together with bortezomib from 3rd cycle onwards due to minimal treatment response. In these patients, rash resolved and did not recur. **Conclusions.** VZV reactivation is common and serious consequence of bortezomib therapy. According to our experience, prophylaxis with acyclovir is very effective and should be considered for all patients treated by bortezomib. The minimal sufficient dose of acyclovir for prophylaxis of VZV reactivation remains to be established but in our group of patients dose of 400mg of acyclovir thrice daily was effective in 100% of cases. Rash is common toxicity seen in patients treated with bortezomib. In our cases lesions are infiltrated by lymphocytes seem to be the most typical after bortezomib. According to our clinical experience, corticosteroids are useful for prevention and treatment of bortezomib-associated rash while maintenance treatment with antihistamines alone is not effective.

1228**SELECTED ELDERLY MYELOMA PATIENTS CAN BENEFIT FROM AUTOLOGOUS STEM CELL TRANSPLANTATION AND HAVE A SIMILAR CLINICAL OUTCOME AS YOUNGER PATIENTS: A SINGLE CENTRE STUDY**

L. Ysebrant, N. Meuleman, J. Bennani, I. Ahmad, M.C. Ngirabacu, C. Nguyen van Cau, P. Lewalle, O. Dehenau, D. Bron

Jules Bordet, BRUSSEL, Belgium

High-dose chemotherapy with autologous stem cell transplantation (ASCT) is currently the standard treatment in myeloma patients below

65 years but transplant remains questionable in elderly patients. The aim of this study is to evaluate the feasibility and the efficacy of ASCT in elderly myeloma patients. We retrospectively reviewed the medical files of forty seven patients with stage II or III multiple myeloma treated with high doses melphalan followed by ASCT between 1995-2003. We compared the clinical outcome of patients older than 65 years at the time of the ASCT to younger ones. Progression-free survivals (PFS) and overall-survivals (OS) curves were compared using the Kaplan-Meier model on an intent-to-treat basis. Forty seven symptomatic myeloma patients treated with ASCT were followed: 10 patients were 65 years or more and 37 patients were younger. Median age at the time of ASCT was respectively 67 years (65-71) and 55 years (39-63). There were no significant differences in the distribution of pre-treatment characteristics: β 2 microglobulinemia, chromosome 13 deletion, renal dysfunction, stages (two patients were diagnosed stage II and forty five at stage III), PS and number of comorbidities. Sixty eight percent of the patients had none or single comorbidity. There were no significant differences in median PFS between patients older than 65 years and younger (13 months versus 17 months, $p=0.36$) and in median OS, respectively 57 months versus 59 months ($p=0.32$). A trend to a better complete remission rate was observed in younger patients ($p=0.09$) but good partial remission rate was similar in both groups of patients. Transplant related mortality (TRM) was 0% and serious adverse events (SAE) are similar in both groups (34% vs 36%). Clinical outcome was similar in both groups of patients treated by ASCT. OS, TRM and SAE of elderly patients (65+) were improved compared to previous studies. These differences could be explained by the population of fit elderly patients with few adverse prognosis factors (β 2 microglobuline, chromosome 13 deletion, renal dysfunction) in our study. Selected elderly patients with few comorbidities, can benefit from ASCT and have a similar clinical outcome as younger patients.

1229**PLASMA CELL IMMUNOPHENOTYPE CD56 POSITIVE AS GOOD PROGNOSIS MARKER**

H. Bumbea,¹ A.M. Vladareanu,¹ M. Begu,¹ I. Tuca,¹ V. Motronea Vasilache,¹ D. Casleanu,¹ I. Voican,¹ C. Marinescu,¹ C. Ciufu,¹ A. Petre,¹ V. Popov²

¹Emergency University Hospital, BUCHAREST, Romania; ²Spitalul Judetean, PITESTI, Romania

Background. Malignant plasma cell could have a classical appearance, or it could be atypical at the optical microscopy analysis. This is known to be a B-cell without expression of lineage markers (CD19, CD20), with lack of CD45 expression and typical expression of CD38, CD138, and CD56. There were described aberrant coexpressions of CD10, CD28, c-kit, CD20 in a few cases. **Aims.** We have analyzed our cases of multiple myeloma and plasma cell leukemia to correlate with the immunophenotype and microscopic appearance. **Methods.** We performed optical microscopy and immunophenotyping on peripheral blood cells and bone marrow aspirate, in 67 patients, with median age of 63 years. **Results.** We found on optical microscopy classical plasma cell in 84,3% and 15,7% plasmoblasts. Immunophenotyping by flowcytometry found in 70% of patients the expression of CD38, CD138 and CD56. In 15% we found lack for CD56 and in 20% lack for CD38. We found also that those patients with lack for CD56 had poor prognosis and the lack for CD38 didn't change the prognosis. In a patient with plasma cell leukemia positive expression of CD56 could be considered as good prognosis marker, despite of aberrant lack of CD38 expression on plasmoblasts. Aberrant coexpression of CD20, CD13 or CD33 didn't associate poor outcome. In 5% patients we found plasma cells in peripheral blood associated with poor prognosis and terminal phase of disease. In conclusion, we consider that immunophenotyping in plasma cell leukemia and multiple myeloma is very important, and critical for the quickly diagnosis, too. We can find important prognostic markers, and we consider that lack of expression of CD56 could be the most important, associated with poor prognosis.

1230**MORPHOFUNCTIONAL STATUS OF LIVING PLATELETS AND THROMBOSIS RISK IN PATIENTS WITH CHRONIC RENAL FAILURE AT THE END STAGE OF HEMODIALYSIS**

E. Vlasova, I. Vasilenko, V. Metelin, V. Shabalina, S. Babakova
Rheumatology, MOSCOW, Russian Federation

Backgrounds. Heparin-induced thrombocytopenia and thrombosis is a severe complications in patients on hemodialysis. To appreciate the character of cellular hemostasis disorders in patients with chronic renal fail-