a significant control of CINV in pts receiving moderately/highly emetogenic chemotherapy. We prospectively evaluated the efficacy of a single or double i.v. dose PALO in pts undergoing HDT and ASCT.
Methods: A total of 60 pts (M/F = 32/28), median age 45 yrs (r16−64), with diagnosis of lymphoma (29), myeloma (24), sarcoma (5), acute leukemia (1), breast cancer (1) were accrued. The first cohort (30 pts) received a single iv PALO dose (0.25 mg) plus 8 mg of dexamethasone (DMS) 30′ before starting of HDT while in the second cohort (30 pts) the first dose was followed by a further PALO (0.25 mg)/DMS (8 mg) injection 48h after HDT. The distribution of conditioning regimens (high-dose melphalan = 28, BEAM = 25, MitoMel = 6, ThioEpiCTX = 1) was comparable between the two cohorts. Acute (24h) and delayed (120h) CINV episodes were rated by the visual analogic scale (MASCC/MAT) while CINV impact on daily activities was self-assessed by pts (at 120h from starting of HDT), through the Functional Living Index-Emesis (FLIE) tool.
Results: No significant differences between the two groups (single vs double PALO) emerged as to acute CINV evaluation (MAT) since 98% of pts achieved a complete response (CR = no emesis, no need for rescue therapy) with only 17 pts (28%) experiencing moderate nausea (median intensity = 5, r 1−10). Double-dose PALO displayed a trend for a better control of delayed nausea which occurred in 53% vs 77% of pts ($p = 0.0581$). In addition, double PALO dosing had a highly significant impact on nausea-related modifications of daily activities. FLIE nausea score was of a median value of 55.26 (r47.5−58.9) in pts receiving two doses of PALO vs 40.92 (r35−45.2) for pts treated with the single PALO dosing ($p = 0.0009$).
Conclusion: Our results indicate that double dose PALO achieves an optimal control of acute/delayed CINV and significantly reduces the detrimental impact of nausea on daily activities in patients undergoing HDT. The impressive activity of PALO in the ASCT setting might be possibly improved by combination with NK1 receptor antagonists.

9250 POSTER

Secondary malignancies after stems cell transplantation – a single centre experience

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Background: It is considered that patients (pts) submitted to Stem Cell Transplantation (SCT) are at increased risk of develop secondary primary malignancies which may be related to several factors, such as primary malignant disease, radiation/chemotherapy treatment or graft-versus-host disease. Authors aim to report a single centre experience on this issue.
Material and Methods: Retrospective analysis of pts who developed secondary malignancies (SM), selected from all stem cell transplanted patient, from June 1989 to December 2008, in the Portuguese Oncology Institute, Porto. Statistical analysis was performed using SPSS v16.0.
Results: A total of 1026 patients underwent 1190 SCT during this period - 599 autologous (AutoSCT) and 591 allogenic (AlloSCT). SM after SCT were identified in 28 pts (2.7%) – 16 in autoSCT and 12 in alloSCT. 78.6% of pts were male and median age was 38 years (range: 3−66). Conditioning plan did not predict total body irradiation or any other radiation strategy. Most common primary malignancy was hodgkin lymphoma (7 cases), followed by non-hodgkin lymphoma and acute myeloid leukaemia (6 cases each). Secondary malignancies, according to their frequency, were: oral cavity/tong, colon, sarcoma, acute myeloid leukaemia, and myelo-displasic syndrome (3 cases each); thyroid, urinary tract and cervix (2 cases each); breast, esophagus, PNET, skin (basocelular carcinoma), renal, gastric and hepatocelular cancer (1 case each). Four pts developed a third malignancy (sarcoma, esophagus, thyroid and non-hodgkin lymphoma). SM was responsible for 12 (42.9%) of the 16 deaths observed in this set of patients. Ten years overall survival after SM diagnose was 41.7% (31.2% in AutoSTC and 60% in AlloSCT). Median time from STC to SM was 51.5 months (range: 1−177). Hodgkin lymphoma seems to be related with secondary myelo-displasic syndrome ($p = 0.055$). No statistical significant independent prognostic factor was found.
Conclusions: A low incidence of SM was found, particularly in AlloSCT pts. It is important to conduct larger studies in order to determinate possible risk factors and better manage these events.

9251 POSTER

Hepatitis B virus (HBV) reactivation in patients with resolved prior HBV according to HBV profiles in the era of immunochemotherapy

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Background: The epidemiology and factors affecting HBV reactivation in patients with resolved prior HBV (HBsAg -ve; anti-HBc +ve) has not been defined. We aim to analyse the HBV profiles of patients with resolved prior HBV and correlate them with the risk of reactivation, particularly in those receiving immunochemotherapy.
Materials and Methods: 374 patients from 2005−09 were included. HBsAg, anti-HBc, anti-HBs and HBV DNA were tested. HBV reactivation was defined as the reappearance of HBsAg with an increase in HBV DNA levels when compared with baseline.
Results: 374 had both HBsAg and anti-HBc tested, 124 (33.2%) had resolved prior HBV. The proportion of resolved HBV increases with age, rising from 12.5% in patients 10−20 yrs to 58.5% in 70−80 yrs, ($p < 0.01$). 95 were tested for anti-HBs, which was positive in 67 (67.4%). 87 were tested for HBV DNA, which was detectable in 5 [(5.7%), 2 were anti-HBs positive, 2 were negative and 1 was not tested]. Overall, 103 patients with prior HBV received systemic treatment and 3 reactivated (2.9%). Of note, 1 reactivated despite having high anti-HBs titer (90mIU/ml). Among patients receiving Rituximab-based treatment and no anti-viral prophylaxis, the reactivation rate was 3.1% (2/65). Of note, 4 patients received maintenance-Rituximab and 1 (25%) reactivated. Of the 5 patients with detectable HBV DNA, 1 reactivated (20%).
Conclusions:
1. In 3 lymphoma patients in an endemic area have prior HBV, although the prevalence appears decreasing in younger patients, possibly due to an active immunization program;
2. 6% of our patients were viraemic despite being HBsAg negative, suggesting that a polyclonal EIA (enzyme immunoassay) for HBsAg may have a role in detecting HBV mutants, compared to the current monoclonal EIA;
3. The rate of reactivation in patients with resolved HBV is relatively low, even among patients treated immunotherapy without antiviral prophylaxis. This coupled with the high prevalence of resolved HBV suggest that routine prophylaxis may not be feasible or indicated in all;
4. Nonetheless, our data caution that patients receiving maintenance-Rituximab or having detectable HBV DNA may be at increased risk of reactivation and the role of prophylaxis further evaluated;
5. Anti-HBs appears to have a limited immune-protective role as 2 patients were viraemic despite having high anti-HBs titers (values). Further, 1 patient without viraemia reactivated despite having high anti-HBs titer;
6. These results are novel.

9252 POSTER

Influence of bevacizumab on platelet function in vivo

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Background: Haemorrhage, as well as arterial and venous thromboembolism are well known side effects associated with bevacizumab. The pathophysiological mechanisms involved are poorly understood. We hypothesized from preclinical data, that platelet function may be a possible site of interaction of vascular endothelial growth factor (VEGF) and bevacizumab. We aimed at testing this hypothesis in a clinical observational study.
Material and Methods: Platelet adhesive and aggregatory functions were tested with a platelet function analyzer (PFA-100) inducing platelet activation with either epinephrine (EPI) or adenosinediphoshpate (ADP). Soluble P-selectin (sP-sel) as a marker of platelet activation was measured using a commercially available immunoassay-kit. In 22 patients with advanced metastatic disease treated with cytostatic chemotherapy and bevacizumab, PFA-100 closure times (CTs) and sP-sel plasma levels were assessed before and immediately after first treatment with bevacizumab and after 6 weeks of treatment.
Results: Mean PFA-100 CTs (with 95% confidence interval (CI)) before, after first bevacizumab application and after 6 weeks were 112.6 sec (99.8−125.4), 110.9 sec (98.5−123.3) and 110.8 sec (94.0−127.7) respectively with EPI ($p > 0.05$). CTs for ADP were 79.7 sec (71.9−86.7), 82.1 sec (72.7−90.8) and 81.0 sec (72.8−89.2), respectively ($p > 0.05$).