(group 2); 69 had lung omo/controlateral metastases (group 3, one study) and 20 pts had other sites of disease (pancreas, n = 2; gastrointestinal system, other, n = 5; soft tissue, n = 5; lymph-nodes, n = 1; skin, n = 3; bone, n = 3; kidney, n = 1) (group 4). The average value of median and 5-year survival was of 19.4 months (n = 277, range 11−26.4) and 23.4% (n = 270, range 6.6−70%) for group 1; of 22 months (n = 170, range 12−66) and 26.15% (n = 62, range 23.3−29%) for group 2; 5-year survival of group 3 was 33.4% (n = 69). In group 4 (isolated case reports) MS largely ranged from 2−81 months.
Conclusions: A tailored attempt at curative resection might be a reasonable approach to NSCLC pts with a SSM, especially considering the current availability of novel and refined diagnostic and surgical techniques.

9045 POSTER

Serum metastin is not involved in metastatic potential of non-small cell lung cancer (NSCLC)

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Background: Metastin, the product of the KISS-1 gene, seems to represent a strong suppressant of metastasis for some types of cancer. The aim of this study is to explore whether metastin could be used as diagnostic and prognostic marker in Non-Small Lung Cancer (NSCLC) patients. The serum metastin levels in metastatic and in locally advanced disease were also studied.
Methods: Fasting serum levels of metastin were determined in 96 NSCLC patients (76 with metastatic disease and 21 with locally advanced disease) and 49 healthy volunteers using commercial available ELISA. Metastin serum levels were determined at diagnosis, at the end of first line chemotherapy and at the time of disease progression for those who responded to treatment. Epidemiological, anthropometrical and laboratory data were assessed for patients as well as for healthy volunteers.
Results: Serum metastin levels presented no differences between NSCLC patients and healthy volunteers (1.2±1.0 vs. 1.2±0.4, P = 0.973) as well as between patients with metastatic disease and patients with locally advanced disease (1.2±0.6 vs. 1.2±1.0, P = 0.714). No statistically significant difference in metastin serum levels from the baseline was observed at the end of chemotherapy or at the time of relapse. Multivariate analysis also showed that serum levels of metastin could not be used as predictive factors for Overall Survival or Time to Progression.
Conclusions: There was a lack of direct involvement of metastin in the metastatic potential and prognosis of NSCLC.

9046 POSTER

A phase II retrospective trial of Platinum/Gemcitabine (P/G)-based in first line treatment of advanced NSCLC with genetic polymorphisms analysis

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Background: Selecting patients according to key genetic characteristics may help to tailor chemotherapy and optimize the treatment in NSCLC. Genetic variations in drugs metabolism may affect the clinical response, toxicity and prognosis of NSCLC pts treated with P/G-based therapy.
Material and Methods: We evaluated 8 single nucleotide polymorphisms (SNPs) of 6 genes (P53 Arg72Pro (G/C); XRCC3 Thr241Met (C/T); XPD Lys751Gln (A/C); ERCC1 Asn118Asn (C/T); CDA Lys27Gln (A/C), Ala70Thr (G/A), Thr145Thr (C/T) and RRM1 C524T) involved in P/G-based metabolism in a homogeneous population of pts with advanced NSCLC treated with this regime. Genomic DNA was extracted from whole blood samples of pts using the QIAamp DNA estraction kit and automatically purified by Biorobot EZ1 (Qiagen). Polymorphisms were detected with TaqMan-probe based assays using the ABI PRISM 7300 instrument equipped with the Sequence Detection System version 2.0 software (Applied Biosystems, Foster City, CA). Association between SNPs and response, toxicity, progression free survival and overall survival was estimated using logistic regression model, the Kaplan-Meier method, the long-rank test and the Cox proportional hazard model.
Results: We performed a retrospective analysis in 192 chemotherapy-naive pts (median age 63 years), including M/F: 74/26%; stage IIIb/IV: 24/76%; Adeno/Squa/other Ca: 42/27/31%; ECOG PS: 0−1/2−3: 94/6%. Overall response rate was 32.3%, stable disease 25% and disease progression 42.7%. The CDA Thr145Thr T/T genotype significantly correlated with poorer response (partial response in 23.1% of pts versus 42.3% and 34.6% in C/T and C/C genotypes, respectively; p = 0.03). The ERCC1 T/T genotype was significantly associated with hematological toxicity (G1−4) (p = 0.05) compared to ERCC1 C/C and C/T allele. The CDA Thr145Thr C/T genotype was significantly associated with non-hematological toxicity (G3−4) (p = 0.02) compared to CDA Thr145Thr C/C and T/T allele. The median overall survival (OS) time and progression free survival (PFS) were 12.7 and 4.7 months, respectively. None of the analyzed polymorphisms was related to PFS or OS.
Conclusions: These data suggest that genetic polymorphisms in the ERCC1 and CDA genes may modulate the toxicity and response to P/G-based therapy in pts with advanced NSCLC.

9047 POSTER

Intrapleural IL-2 immunotherapy of patients with malignant effusion

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Background: Combined intrapleural immunotherapy by IL-2 and lymphokine-activated killer cells of malignant pleural effusion (MPE) showed high effectiveness in patients with some cancer types. The study aimed to assess clinical effectiveness of intrapleural monotherapy by recombinant IL-2 (Proleukin, the Netherlands) for MPE treatment.
Materials and Methods: 25 patients (pts) with metastatic pleural effusion were enrolled in the pilot study: lung cancer 3, breast cancer 17, renal carcinoma 2, ovarian cancer 3, including 4 male and 21 female pts aged 42−73 years. Previously all pts received appropriate complex therapy, namely, surgery combined with chemo-, hormone-, immuno- or radiotherapy. Pleural cavity was drained under anesthesia to provide liquid evacuation every other day and IL-2 infusion for intrapleural immunotherapy. The course included 14-day consequent infusions of Proleukin in the dose of 1m IU in 20 ml of physiological solution. Before and at the end of therapy, effusion was tested cytologically and controlled by x-ray of the chest. X-ray examination was also performed 1, 6, 12 months after the end of treatment.
Results: Prior to the therapy pts had 1000 – 2 400 ml of pleural effusion. Treatment effectiveness was estimated on weeks 4 or 5 after the end of IL-2 infusion. Over this period 11 pts reached complete effect, i.e. no effusion accumulated. Significant reduction of intrapleural effusion was registered in 7 pts, 3 pts had stabilization of the process and 4 pts did not have any effective decrease of effusion accumulation. Recurrences were registered in 5 pts within 2−18 months after the end of treatment. Total efficacy of IL-2 intrapleural immunotherapy of MPE after 4−5 weeks following the last infusion was 84%.
Cytological examination showed that before treatment all MPE samples contained tumor cells and in the last evacuated sample there were no tumor cells in most patients (n = 21) and only 4 patients still had a small number of tumor cells in MPE.
Conclusion: Intrapleural IL-2 immunotherapy of 25 patients with MPE showed high effectiveness (84%), including complete effect −44%, partial effect −28%, stabilization of the process −12% confirmed by cytological and x-ray examination. Considering its good tolerability and high efficacy, intrapleural IL-2 immunotherapy may be recommended for MPE treatment after necessary clinical trials.

9048 POSTER

Aprepitant for the prevention of chemotherapy-induced nausea and vomiting associated with moderately emetogenic (MEC) or highly emetogenic (HEC) chemotherapies in patients with lung cancer

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Background: Aprepitant was shown previously to be effective for prevention of chemotherapy-induced nausea and vomiting (CINV) associated with HEC in patients with solid tumors (including lung cancer) and MEC in breast cancer patients. A recent study (NCT00337727, PN130) assessed aprepitant in patients with a variety of tumor types receiving a broad range of MEC regimens. A post-hoc subgroup analysis of patients with lung cancer was performed and results are reported along with previous CINV data for lung cancer patients receiving HEC.
Materials and Methods: This randomized, gender-stratified, double-blind trial enrolled patients with confirmed malignancies, naïve to HEC or MEC agents, who received at least one MEC agent. Patients