up to 42.6% for tumors >5 cm. The mean age of the first 424 MAGE-A3-positive patients randomized was 63 years. Gender: 24.1% females. 84.1% of the patients had received (bi-)lobectomy/sleeve lobectomy and 15.7% pneumonectomy. Radical mediastinal lymphadenectomy had been performed in 57%. Pathological stage: 52.4% stage IB, 34.2% stage II and 13.0% stage IIIA. Histopathological type: 50.7% squamous cell carcinoma and 33.3% adenocarcinoma. Adjuvant platinum-based chemotherapy was given to 46.8% of patients (25.7% Stage IB, 68.1% Stage II, 78.8% Stage IIIA).
Conclusions: The expression rate reported here confirms previously reported expression rate from a Phase II trial of MAGE-A3 in resected NSCLC[1] and from smaller cohorts in comparable stages of NSCLC[2,3]. Although the MAGE-A3 expression appears constant throughout disease stages, it differs according to the histological type, with a more frequent expression in squamous cell tumors, and also according to the tumor size, with increased expression in large tumors. Radical mediastinal lymphadenectomy is performed in more than half of the patients and about half of patients randomized do receive adjuvant chemotherapy.

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9022 POSTER

The nicotinic acetylcholine receptor (nAChR) subunit α3 (CHRNA3) polymorphism in advanced non-small-cell lung cancer (NSCLC) patients (p) with EGFR mutations treated with erlotinib

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Background: Polymorphisms in the 15q region, containing the genes for α3 and α5 subunits of heteromeric nAChRs, are associated with a predisposition to lung cancer even in a smoking-independent manner. Activation of the nAChR pathway leads to upregulation of the CREB (CMP response element-binding) protein, which transactivates the EGFR and induces the release of EGF and VEGF. CREB overexpression decreases survival in never-smokers with NSCLC. We hypothesized that CHRNA3 would influence outcome in NSCLC p with EGFR mutations treated with erlotinib.
Material and Methods: Stage IV NSCLC p with EGFR mutations were prospectively treated with erlotinib. Genomic DNA was derived from tumor tissue obtained by laser capture microdissection. Deletions in exon 19 (del 19) were determined by length analysis after PCR amplification with a FAM-labelled primer in an ABI Prism 3130 DNA Analyzer. Exon 21 point mutations L858R were detected with a TaqMan assay. DNA was extracted from lymphocytes and CHRNA3 (rs1051730) polymorphism was genotyped with the TaqMan allele discrimination assay.
Results: 185 NSCLC p with EGFR mutations were treated with erlotinib. Median age, 68; 136 females; 182 Caucasians, 3 Asians; 123 never-smokers, 44 ex-smokers, 10 current smokers; 145 adenocarcinomas, 21 BAC, 19 LCC; 179 stage IV, 6 stage IIIB with malignant pleural effusion; 99 first-line erlotinib, 86 second-line; 110 del 19, 75 L858R. 62 p had the homozygous (CC) CHRNA3 genotype; 94 were heterozygous (CT); 29 were homozygous for the variant (TT). No differences in p characteristics were found according to the CHRNA3 genotype. Overall response: CR, 19 p (11.6%); PR, 97 p (59.1%); ORR, 116 p (70.7%); SD, 33 p (20.1%); PD, 15 p (9.1%); 21 p had no measurable disease according to RECIST criteria. No differences in response were observed according to the CHRNA3 genotype. Median follow-up, 14 months (m) (range, 1−42 m). Median time to progression (TTP), 14 m (95% CI, 10.9−17.1). Median survival (MS), 28 m (95% CI, 24.9−31.1). Hazard ratios for shorter TTP were 3.7 for male gender (P = 0.05), 4.65 for PS 2 (P = 0.19), 0.18 for CT genotype (P = 0.03), 0.21 for CC genotype (P = 0.07), and 2.31 for L858R (P = 0.23).
Conclusions: p with EGFR mutations have an impressive response rate and TTP when treated with erlotinib. However, a subgroup of p with the variant CHRNA3 TT genotype have a significant risk of relapse, perhaps due to overexpression of EGF and VEGF through hyperactivation of the nAChR pathway. Assessment of the CHRNA3 polymorphism can help identify these patients and provide a useful guide for additional therapeutic decisions.

9023 POSTER

Relationship between SNPs of glucose transporter related genes and 18F-deoxyglucose (FDG) uptake of PET-CT in non-small cell lung cancers (NSCLCs)

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Backgrounds: Lung cancer cell growth is an energy-related process using glucose metabolism. This uptake is mediated by glucose transporters (Gluts). Previous study revealed that increased expression of Gluts are related with increased uptake of FDG and clinically related with poor outcome. But there were no reports about the relationship between genetic phenotypes of Gluts and increased FDG uptake in NSCLCs. In this study, the authors checked the SNPs of Gluts and other related genes and its relationship with the uptake of FDG in NSCLCs.
Materials and Methods: From October, 2005 to February 2008, 122 blood samples were collected from NSCLCs patients. Male to female ratio was 2: 1 and mean age was 64.5 year. Of the 122 patients, adenocarcinomas were 75, squamous cell carcinomas were 47. SNPs of *Glut1, VEGFA, APEX2, HIF1A* were checked by using SNaPShot assay (ABI PRISM ANaPShot Multiplex kit, CA, USA) and analysed using Genemapper software. Also PET-CT were checked and SUVmax value were taken and compared with the SNPs of the each gene.
Results: In squamous cell lung cancers, the level of SUVmax was higher in GLUT1 TT type than in AA+AT type (11.6 8.9. Also in squamous cell lung cancers, GlUT1 type showed shorter survival time than AA+AT type.
Conclusions: Non-small cell lung cancers, especially in squamous cell type which have GLUT1 recessive type (TT) showed increased level of SUVmax and shortened survival time indicates that genotypic types of glucose transporters have clinical implication regarding to prognosis.

9024 POSTER

LCK-positive tumor-infiltrating lymphocytes is associated with a better prognosis in stage I non-small cell lung cancer patients

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Objective: The correlation between tumor-infiltrating immune cells and the prognosis of lung cancer is controversial. For this reason, we have invistigated the expression in the tumor infiltrate of a T-cell activation marker, the "lymphocyte-specific protein tyrosin kinase" (LCK), to assess if it could be associated with a better prognostic outcome in early stage NSCLC patients.
Methods: This retrospective study included 25 patients undergoing lobectomy with standard hilo-mediastinal lymphadenectomy for pathological stage I NSCLC between 7−2003 and 6−2005. The presence of LCK was detected in the tumor infiltrate by immunohistochemistry on the specimen of all patients. No patient received adjuvant therapy.
Results: Resection was radical (R0) in all the patients. There was no post-operative mortality. Median follow-up time was 48 months (range 40−60). Twelve patients had a recurrence within 40 months from the operation while 13 patients had no recurrence. The presence of LCK in the tumor-infiltrate was found in 3 of 12 patients (25%) showing recurrence and in 9 of 13 patients (69%) without recurrence (Fisher's exact text p = 0.01). Relapse-free survival (RFS) and overall survival (OS) (Kaplan-Meier analysis) resulted significantly longer in the LCK-positive group (median-RFS: not reached Vs 25 months, Log-rank p < 0.001; median-OS: not reached Vs 30 months, Log-rank p = 0.02). The distribution of patients according to T-stage was similar between the LCK-positive group (6 T1, 6 T2) and the LCK-negative group (6 T1, 7 T2).
Conclusion: LCK-positive tumor-infiltrate is clearly associated with a longer RFS and OS and a lower relapse rate in patients with radically resected stage I NSCLC.