these were enrolled in registered clinical trials (n = 61). KRAS analysis was successful in 125 cases. Main reasons for no KRAS analysis were clinical deterioration (n = 13), inadequate or insufficient tumour material (n = 6) or other/not specified (n = 4).
All patients but one were Caucasian, most were women (n = 91) and the median age was 68 years (range 26−83). Nineteen patients had KRAS mutant tumours, and all mutations were found in exon 12. The median overall survival was 6.6 months. It was independent of KRAS status (wild type 7.0 months and mutant 8.3 months, p = 0.75). Median survival for patients without KRAS analysis was 2.3 months.
In the 109 patients receiving chemotherapy, 16 (14.7%) had tumours with KRAS mutations.
Conclusion: KRAS mutations were found in 14.7% of a cholangiocarcinoma population eligible for chemotherapy. The number differs from surgical cohorts and this must be taken into account when designing studies testing the influence of KRAS status on treatment effect.

6129 POSTER

Analysis of Surfactant on Primary(COLO-320) and Metastatic(COLO-741) Human Colon Cancer Cells Treated With A-Lactalbumin or Sulindac

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Background: Colon cancer is the second most frequent reason in the cancer-related deaths in the world. During cancer therapy, the correct time and correct medicine is crucial for different patients. In addition, the primary and metastatic colon cancer therapy may also be different because of cancer cell behavior. Our aim is investigating the surfactant has efficiency on the medicine during treatment of cancer cells.
Materials and Methods: Colo-320 and Colo-741 lines were used in this study. The cells were cultured in RPMI-1640 media including %10 FCS, %1 L-glutamine and %1 penicillin-streptomycine. The cells were cultured in 24 wells of tissue culture plate. After subculturing of cells, they were cultured 24 hours. After 24 hours of culture, the cells will be treated with either α-lactalbumin or sulindac or α-lactalbumin+surfactants or sulindac +surfactants. After 48 hours of treatment, culture mediums from all groups were collected for cytotoxicity analysis, the cells from all groups were fixed in %4 paraformaldehyde for 30 minutes for histochemical analysis. Cell cytotoxicity were evaluated with ELISA. Cell death was investigated using TUNNEL assay.
Results: The Colo-320 cells were semi-adhesive cells; the Colo-741 cells were attachment cells. After treatment with sulindac of Colo-320 cells, the number of alive cells was less when compared with other groups. It was also observed that the number cells in Colo-741 cells which were treated with only α-lactalbumin or only sulindac groups had less cell amount than the other groups. We are still evaluating the affect of surfactants during treatments of colon cancer.
Conclusions: Our hypothesis suggests that both primary and metastatic cells will be affected when surfactants during treatment. However, the addition of surfactants during treatment protocols may cause differences in drug interactions with cells. In farther researchs. Simultaneously, Surfactants might be used to treat with different medicines of other cancer types. Researchs are still going on about this issue in our faculty.

6130 POSTER

Survivin Expression in Rectal Cancer During Preoperative Radiochemotherapy and Its Impact on Metastasis and Patients' Survival

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Background: Valid molecular markers need to be implemented in clinical trials to fulfill the demand of a risk-adapted and more individualized multimodal therapy of locally advanced primary rectal cancer. In the present study the expression of the inhibitor-of-apoptosis (IAP) protein Survivin was evaluated in pre-treatment biopsies and corresponding post-treatment resection specimens, and was correlated to histo- pathological tumour characteristics and clinical follow-up.
Material and Methods: 116 patients with stage II/III rectal cancer treated with 5-FU/Oxaliplatin based neoadjuvant radiochemotherapy (RCT) at a single university medical centre within the German Rectal Cancer Trials were investigated. Survivin expression in pre-treatment biopsies and surgical resection specimens were determined by immunohistochemistry by two independent institutions and correlated with histopathologic parameters, tumour recurrences, disease-free and cancer-specific overall survival.
Results: In pre-treatment biopsies, a higher Survivin expression correlated with advanced ypT (p = 0.026) and ypUICC (p = 0.05) stage as well as decreased disease- free survival (p = 0.038) after preoperative RCT. High post-treatment Survivin levels were associated with advanced ypT stage (p = 0.03) and residual lymph node metastases (p = 0.04). Moreover, neoadjuvant RCT resulted in a significant down-regulation of Survivin expression (p < 0.0001). A failure of RCT-induced down-regulation was associated with development of distant metastases (p = 0.0056) and cancer-related death (p = 0.026), and was significantly correlated with disease-free (p = 0.011*/0.02**) and cancer- specific survival (p = 0.0017*/0.01**) in uni* and multivariate** analyses.
Conclusions: Survivin expression in rectal cancer displays a marker with prognostic validity. These results underline the usefulness of Survivin to monitor individual response to RCT in rectal cancer, and encourage anti-Survivin strategies in multimodal rectal cancer therapy within future randomised clinical trials.

6131 POSTER

Evaluation of BRAF Mutational Status in Wild Type (WT) KRAS Metastatic Colon-Rectal Cancer (MCRC) Patients (pts) Treated With Cetuximab (C) − a Single Institution Experience

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Background: KRAS mutations in pts with mCRC have since emerged as the major negative predictor of efficacy in pts receiving anti-EGFR therapies such as C. Nevertheless, the occurrence of KRAS mutation only accounts for approximately 35−45% of nonresponsive pts. Mutations in BRAF have been recently shown to impair responsiveness to these agents, with no response observed in BRAF mutated pts.
Materials and Methods: From 11/2008 to 01/2011, 95 mCRC pts were tested for KRAS mutations and 30 pts resulted KRAS WT, all of them were treated with C+ chemotherapy (CHT). We now retrospectively analyze the clinical outcome of 20 pts according to their BRAF mutational status, in order to evaluate if BRAF mutation influence the clinical outcome. fivepts were excluded from the analysis due to the impossibility to evaluate BRAF status for little DNA. Genomic DNA was extracted from formalin-fixed, paraffin embedded (FFPE) tumour samples using a commercial kit (Invisorb Spin Tissue Mini Kit, Invitek, Berlin, Germany). V600E mutation of the BRAF gene was evaluated through an allele specific multiplex PCR (CRC Kit 2, Experteam, Venezia, Italy). Median age was 69 years (42−81), all pts underwent surgery for colon (16/20, 75%) or rectal (5/20) cancer, 8/20 (40%) received adjuvant CHT, with FOLFOX4 (50%) or De Gramont regimen.
Results: All pts received C with Irinotecan16/20, 80%) or FOLFIRI (4/20) as 2nd or 3rd line treatment. Five pts (25%) resulted as BRAF mutated (BRAF+). No response has been observed among BRAF+ pts, whereas PR or SD was obtained in 6/15 (40%) BRAF− ones. Median TTP was significantly longer in BRAF− vs BRAF+ (12 vs 8 months).
Conclusion: Our single institution experience confirms the recent data about the hypothesis that BRAF mutational status could be a predictive factor for response to anti-EGFR therapy in KRAS WT mCRC pts. Further evaluation of the RAS/RAF pathway or the analysis of polymorphism of the EGFR in BRAF− pts who didn't obtained a response to anti-EGFR therapy is needed to better understand the lack of response in KRAS WT pts.

6132 POSTER

Efficacy of Panitumumab Plus FOLFIRI Versus FOLFIRI Alone in Patients With Wild-Type (WT) KRas Metastatic Colorectal Cancer (mCRC) Treated With Prior Oxaliplatin or Bevacizumab Regimens: Results From 20050181

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Background: First-line treatment regimens for mCRC often contain oxaliplatin and the anti-vascular endothelial growth factor antibody,