Virchows Arch (2011) 459 (Suppl 1):S1–S329
DOI 10.1007/s00428-011-1113-y

ABSTRACTS

Plenary Oral Free Paper Sessions

Thursday, 1 September 2011, 11.00–12.00, Hall 5bc

PL-01 Plenary Oral Free Paper Session

001

Investigation of the correlation between up-regulated ERG expression and PTEN loss with capsular penetration in prostate cancer

R. Dittamore*, R. Nagle, L. Brosch, E. Abril, C. Cortez, A. Algotar, W. Meek, K. McDaniel, E. Dexter, B. Dalkin, G. Pestano
*Roche Diagnostics, Tucson, USA

Objective: Previous studies have demonstrated the existence of ETS gene rearrangement in prostate cancers. TMPRSS2–ERG gene fusion is the most common rearrangement and the overexpression of the ERG oncogene occurred in 50% of localized and metastatic prostate cancers. Studies have identified the relationship between ERG overexpression and PTEN deletions as a critical gateway to predicting poor outcome. We hypothesized that ERG overexpression and PTEN deletions status may correlate with capsular penetration.
Method: From a cohort of 210 fully embedded prostatectomy patients, we selected cases that had capsular penetration, pT3a with a contralateral organ confined lesion. PTEN and ERG expression were compared between the penetrating lesion (CP) and the contralateral organ confined lesion (non-CP).
Results: See image

Results:

Groups	CP (N, %)	Non-CP (N, %)	Total	Odds Ratio	p-value
Pos PTEN & Neg ERG	13 (31.7)	18 (43.9)	31	Ref	
Pos PTEN & PosErg	17 (41.5)	18 (43.9)	35	1.4	0.51
Neg PTEN & Pos ERG	6 (14.6)	1 (2.44)	7	8.3	0.06
Neg PTEN & Neg ERG	5 (12.2)	4 (9.76)	9	1.7	0.47
Total	41	41			

Conclusion: Our analysis of this cohort showed an OR of 8.3, indicating that capsular penetration is 8.3 times more likely in lesions demonstrating negative PTEN expression and positive ERG over-expression. This profile was generated after comparison with lesions demonstrating a normal expression profile for PTEN and ERG (Pos PTEN and Neg ERG). We conclude that having an expression profile of PTEN loss and ERG over-expression appears to be a risk factor for capsular penetration in prostate cancer. Additional studies should be conducted in larger, prospective and retrospective biopsy studies to validate these findings.

002

EGFR gene copy number assessment from areas with highest EGFR expression predicts response to anti-EGFR therapy in colorectal cancer

A. Aalgars*, M. Lintunen, R. Ristamäki, J. Sundström
*Dept. of Oncology, Turku University Hospital, Finland

Objective: Only 40–70% of metastatic colorectal cancers (mCRC) with wild-type KRAS oncogene respond to anti-epidermal growth factor receptor (anti-EGFR) antibody treatment. EGFR amplification has been suggested as an additional marker to predict the response. However, improved methods for bringing the EGFR analysis into routine laboratory are needed.
Method: The material consisted of 80 patients with mCRC, 54 of them receiving anti-EGFR therapy. EGFR gene copy number (GCN) was analysed by automated silver in situ hybridisation (SISH). Immunohistochemical EGFR protein analysis was used to guide the SISH assessment.
Results: Clinical benefit was seen in 73% of high-EGFR (≥ 4.0) GCN patients, in comparison to 59% of KRAS WT patients. Only 20% of low-EGFR GCN patients responded to therapy. A high EGFR GCN number associated with longer progression-free survival ($p<0.0001$) and overall survival ($p=0.004$). Together with KRAS analysis, EGFR GCN identified the responsive patients to anti-EGFR therapy more accurately than either test alone. The clinical benefit rate of KRAS WT/high GCN tumours was 82%.
Conclusion: Our results demonstrate that automated EGFR SISH, in combination with KRAS mutation analysis, can be a useful and easily applicable technique in routine diagnostic practise for selecting patients for anti-EGFR therapy.