P044 Clinical impact of ERG and PTEN alterations in men underwent radical prostatectomy

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Introduction & Objectives: Several molecular pathways and oncogenes are involved in prostate cancer (PCa) progression. PTEN, a tumor suppressor gene located on chromosome 10q23.3, acts as a regulator of PI3-K-Akt molecular pathway. ERG oncogene located in chromosome 21q22.2 is involved in PCa by a fusion protein with transmembrane protease, serine 2 (TMPRSS2), a protein encoded by TMPRSS2 gene. The aim of this study is to evaluate the clinical impact of PTEN loss and ERG rearrangement in terms of oncologic results in patients diagnosed with localized PCa who
underwent radical prostatectomy.

Materials & Methods: Data were collected in a prospective way from a total of 74 patients who underwent open radical retropubic prostatectomy for localized PCa. Immunohistochemical study was performed in paraffin-embedded formalin-fixed tissue samples. For anti- ERG antibody, the primary antibody was obtained from DAKO (FLEX Monoclonal Rabbit Rabbit Anti-Human ERG, Clone EP111, Ready-to-Use). For anti-PTEN antibody, the primary antibody was obtained from DAKO (Monoclonal Mouse Anti-Human PTEN (Concenctrate) Clone 6H2.1). ERG was considered positive if at least 20% of the evaluated cells (neoplastic or with HGPIN) were stained at least with medium intensity. A tissue sample was considered to have PTEN protein loss if the intensity of cytoplasmic and nuclear staining was mild or entirely negative across >10% of tumor cells, compared with surrounding benign glands and stroma. Regarding statistical methodology, continuous variables were expressed using medians, minimum-maximum values and interquartile range, while categorical variables using numbers and proportions. Fisher's exact test or Chi square test and Kruskal-Wallis test were used to analyze data.

Results: In terms of correlation of PTEN status with ISUP grade, homogenous loss was associated with higher clinical ISUP grade (p=0.018) while the medians of intact- homogeneous groups differed significantly for median ISUP pathology grade (p=0.022). On the other hand, no statistical significant association was present regarding the presence of ERG rearrangement with either ISUPc or ISUPp. After a median follow up of 34 months, 24 patients developed biochemical recurrence. No statistical significant correlation of ERG status with biochemical recurrence was noted. Both homogenous and heterogenous loss were associated with biochemical recurrence development in a statistical significant way. As far as it concerns the combination of PTEN loss with ERG rearrangement presence, a trend in higher ISUPc and ISUPp as well as biochemical recurrence development was detected, although in a non statistical significant way.

Conclusions: Homogenous and heterogenous PTEN loss was associated with biochemical recurrence. No association of ERG and biochemical recurrence was noted. The combination of PTEN loss and ERG rearrangement presented a trend for higher ISUPc and ISUPp as well as biochemical recurrence but not in a statistical significant way.