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ROLE OF PTEN ON COLORECTAL CANCER PROGNOSIS

Vargas Caroline T, Arruda Polliane, Natividade Leonardo F and Montemor Netto Mario R

State University of Ponta Grossa, Brazil

Background: Colorectal cancer (CRC) is the third most frequently diagnosed malignancy in men and the second most frequently diagnosed cancer type in women worldwide. Colorectal carcinogenesis phenomena may be studied through the evaluation of signaling pathways and protein expression. PTEN (Phosphatase and tensin homolog deletion on chromosome 10) is expressed and inhibits the PI3K action suppressing apoptosis therefore acting as a negative regulator of PI3K-ATK-mTOR pathway and processes related do cell proliferation, survival and growth playing a role of silencing other growth factors in the ATK cascade and regulating tumor cellular activity.

Methods: Clinical and histopathological data were retrospectively collected from 2010 to 2015. Patients presenting familial adematous polyposis (FAP) and hereditary nonpolyposis colorectal cancer (HNPCC) were excluded. The formalin-fixed paraffin-embedded blocks were collected in a medical pathology lab in Brazil and new samples were obtained using tissue micro array technique, containing a biopsy from superficial tissue (ST) and deep tissue (DT). Immunohistochemistry performed using PTEN monoclonal antibody (Novocastra Buffalo, Grove, IL, USA), followed by a measurement of percentage of colored surface area. Student's T Test was used and considered significant values of $p<0.05$. The number of patients differs in the analysis groups due to incomplete medical records.

Results: A total of 211 patients were added, 110(52.1%) women and 101(47.9%) men. Data involving metastasis were available for 188 patients of which 128 (68.1%) did not presented. Presence of metastasis is correlated with the percentage of PTEN-DT expression ($p=0.047$) and a ROC curve was established with a cutoff equal to 22.6. Expression of PTEN-ST and PTEN-DT and its correlation to death outcome also presented positive ($p=0.0.29/p=0.037$ respectively) and ROC curves were performed. Tumor grading, colon region didn't have positive correlations.

Discussion: Analysis revealed that patients who did not present metastasis or had 2-year survival are correlated with higher PTEN immunoreactivity. It shows the down-regulation/inhibition of the PI3K-ATK-mTOR pathway and the protective role of PTEN in carcinogenesis. Smaller areas of PTEN activity are related to death outcome as revealed by ROC curves and cutoff points. Both PTEN-ST and PTEN-DT had significant correlations with death outcome, suggesting a global lack of activity. These cutoffs from ROC curves were able to establish a value of PTEN immunoactivity with statistical sensitivity and can be reproduced for future studies as an outcome predictor.

Conclusion: The loss of PTEN expression might be related to advanced carcinogenesis, tumor malignancy, metastasis and poor prognosis in CRC which should be measured by further studies

caroline.saad@hotmail.com