European Society of Pathology

25th European Congress of Pathology

Pathology – A gate to the future

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Final Programme

www.esp-congress.org

in collaboration with the Portuguese Society of Pathology/Portuguese Division of the International Academy of Pathology

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Brief Description of the ESP

The European Society of Pathology (ESP) was established in 1963 in Brussels and is a leading force in European pathology and, as such, is pivotal to the future of the specialty in Europe and beyond. As a scientific society, the ESP has, as its primary aim, the promotion of high quality diagnostic practice, applied and translational research and under- and postgraduate education in the field of human pathology. This is achieved through its congresses, its journal Virchows Archiv and various other activities. The ESP interacts with national pathology societies in Europe and has developed links with other EU bodies in close collaboration with UEMS (Union Européenne des Médecins Spécialistes) and EAPCP (European Association of Pathology Chairs and Programme Directors). The European School of Pathology under the auspices of the ESP and with the support of 19 Working Groups representing different fields of pathology is involved in the organisation of courses to meet the needs of young pathologists. The ESP also provides an infrastructure for quality assurance projects in diagnostic molecular pathology, such as the ESP QA KRAS project for testing of colorectal cancer. The aim of the ESP is that its new permanent headquarters in Brussels (capable of holding meetings for up to forty people) will become the "hub" of European pathology.

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Conclusion: We intend to work on a larger specimen size to find out if there is any statistically significant correlation with breast cancer prognostic factors.

PS-17-011

Association between BMI-1 and homologous recombination markers in human breast cancer

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Objective: B-cell-specific Moloney murine leukemia virus integration site 1 (Bmi-1) is a Polycomb group protein that is able to induce telomerase activity, enabling the immortalization of epithelial cells. Immortalized cells are more susceptible to double-strand breaks (DSB), which are subsequently repaired by homologous recombination (HR). BRCA1 is among the HR regulatory genes involved in the response to DNA damage associated with the RAD51 protein, which accumulates in DNA damage foci after signaling H2AX, another important marker of DNA damage. Topoisomerase IIIB (topoIIIB) removes HR intermediates before chromosomal segregation, preventing damage to cellular DNA structure. Our objective is to evaluate the relationship between BMI-1 and these regulatory proteins of homologous recombination in human breast carcinomas.
Method: We analyzed protein expression by immunohistochemistry in 239 cases of primary breast tumors. For gene expression, we performed Real-Time PCR reactions in MCF-7 cell line.
Results: BMI-1 immunohistochemistry overexpression was related to p53 (p=0,003), BRCA-1 (p=0,003), H2AX (p=0,024) and TopIIIB (p<0,001). Real-Time PCR assay showed that BMI positive cells have high expression of H2AX and P53.
Conclusion: Our results point to a relationship between BMI-1 and homologous recombination markers, suggesting that the presence of BMI-1 is an important event in breast cancer homologous recombination.

PS-17-012

PIK3CA and EGFR mutation in triple negative breast carcinoma: Comparison with hormone receptors positive and HER2 positive breast carcinomas

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Objective: We aim to investigate PIK3CA and EGFR mutation in triple negative breast carcinomas and compare the results with hormone receptor positive and HER2 overexpressed breast carcinomas.
Method: Genomic DNA was isolated from formaline fixed parafin embedded tumor samples. 38 with Triple negative breast cancer (TNBC), 10 patients with hormone receptors positive, HER2-negative tumors, 10 patients with hormone receptors positive, HER2-positive tumors, 10 patients with hormone receptors negative, HER2-positive tumors. EGFR gene (Exon 18, 19, 20, 21) and PIK3CA gene (Codon 540–546 and Codon 1,042–1,049) were analyzed by pyrosequencing method (Qiagen).
Results: No mutations in Exon 18, 19, 20 and 21 of EGFR gene were detected in all groups. PIK3CA mutations were detected in 9 of TNBC (2: E542K, 1: E54K, 5: H1042R and 1: M1043I mutation), 3 of hormone receptors positive, HER2-positive tumors (1: E545K, 2: H1047R mutation), 5 of hormone receptors positive, HER2-negative tumors (2: E545K, 1: H1047L, 1: H1047R and 1 had both E542K and M1043I mutations). In HER2-positive tumors, no mutations were detected in PIK3CA gene.
Conclusion: PIK3 CA mutation is mostly occured in hormone positive breast cancers; only 23.7 % of triple negative tumors have PIK3CA mutation and as no EGFR mutation has benn detected; EGFR amplification seen in TNBC might occur by other mechanisms.

PS-17-013

Clinicopathological features and prognosis of pregnancy associated breast cancer: A matched case control study

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Objective: Pregnancy Associated Breast Cancer (PABC) manifests during pregnancy or within a year following delivery. We sought to investigate differences in management, outcome, histopathology and immunohistochemistry (IHC) characteristics of PABC and control cases.
Method: PABC and matched control patients were selected from breast cancer cases of women ≤45 years, diagnosed in our institution between 1998 and 2012. Histopathology of invasive and associated in situ lesions, ER, PgR, HER2, Ki67, p53 expression data, IHC-based -subtype, clinical, management and outcome information were analysed.
Results: Thirty-one women had PABC. Clinical, management data, histopathology of disease at presentation was not significantly different, but Nottingham Prognostic Index (NPI) assessed the PABC group as of poor prognosis, while controls as of intermediate prognosis. The associated in situ lesion was mainly high grade Extensive Intraductal Carcinoma component (EIC) in PABC. Triple negative and LumBprol tumors predominated in PABC. Post-partum patients' both disease-free and overall survival, while pregnant patients' disease-free survival was inferior to that of controls. PABC patients with LumBprol and Triple negative tumors had worse prognosis. On multivariate analysis inferior prognosis of PABC was associated with pregnancy.
Conclusion: Our study has demonstrated difference in tumor biology and inferior outcome of PABC compared to controls.

PS-17-015

Immunohistochemical expression of some markers of invasivity in breast cancer

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Objective: Breast carcinoma (BCa) is the most common neoplasia in women that is caused by progressive accumulation of genetic and epigenetic abnormalities. During the tumorigenesis many tumour suppressor genes are inactivated with following decreasing of relevant protein expression and function. This study is focused on immunohistochemical evaluation of proteins in normal and tumor breast tissues that are responsible for self-sufficiency in growth signals and inhibition of cell invasion and metastases forming.
Method: The total of 5 normal breast tissue samples and 20 biopsy specimens with metastatic duct and lobular BCa were evaluated by light microscope semiquantitatively for SOCS1, CDH1, TIMP3 and ADAM23. These proteins are responsible for invasivity and metastasis regulation of tumor cells.
Results: In normal breast tissue we found intensive expression of these proteins. In invasive breast carcinoma we found a decreased expression of evaluated markers.
Conclusion: Next study will investigate the role of epigenetic inactivation of relevant genes in correlation with protein expression changes in BCa. Causal DNA methylation profiles in promoters of evaluated genes could be utilized for identification of new biomarker for invasivity and metastatic potential of breast cancers. This study was supported by the grant APVV-0076-10.

PS-17-016

Rectal metastases originating from occult lobular breast carcinoma

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Objective: We present a case of a patient complaining of hemorrhoid bleeding that underwent colonoscopy. The clinician took biopsies from a