Conclusion: These results suggest that cell viability loss promoted by 2-AG and AEA was associated with ER-stress since both PERK and IRE1 arms of UPR are activated. Prolonged ER-stress, contributes to the expression of pro-apoptotic proteins, such as CHOP.

These findings shed light to the impact of endocannabinoids induced-ER stress which may negatively affect trophoblast cell turnover and pregnancy outcomes.

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PS163

Analysis of imaging characteristics, incidence, and prognosis of brain metastases from thyroid cancer

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Aim: The main objectives of this study were to evaluate the incidence, imaging characteristics, and prognosis of parenchymal brain metastases originating in thyroid cancer.

Introduction: While thyroid cancer is a relatively common type of cancer, it is usually highly curable.[1] Brain metastases from thyroid cancer are rare and their imaging appearance has not been well defined.[2]

Methods: Review of case records of thyroid cancer patients within the IPO Porto data base from 2005 to 2015 was conducted in order to identify the patients with thyroid cancer and evidence of brain metastases.

Results: We identified 3175 patients with thyroid cancer, with only five having evidence of brain metastases (two from papillary thyroid cancer, two from follicular thyroid cancer and one from poorly differentiated thyroid cancer). At the time of brain metastases detection, 100% of the patients had concurrent lymph node metastases, 80% lung metastases and 60% osseous metastases. Of those brain metastases, 60% were multifocal and 40% presented as partially cystic/necrotic. Of the two cases in which the patients died, the median overall survival after brain metastasis detection was less than one year.

Conclusion: Brain metastasis from thyroid cancer remains a rare phenomenon that most frequently occurs in the setting of widely disseminated lymph node disease. The imaging appearance is highly variable and the prognosis is poor.

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PS166

The association of GSTP1 genotype with the risk and survival in ccRCC patients with advanced tumor stage

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Aim: The aim of this study was to evaluate specific role of glutathione S-transferase P1 (GSTP1) gene variants as determinants of ccRCC risk in patients with advanced tumor stage (pT3 and pT4). Furthermore, we evaluated the effect of GSTP1 gene variants on postoperative prognosis in these patients.

Introduction: Renal cell carcinoma (RCC) accounts for up to 90% of malignant kidney tumors with clear renal cell carcinoma (ccRCC) being the most frequent and the most aggressive subtype of sporadic RCC in adults. Unfortunately, most RCCs are asymptomatic in early stages, whereas symptomatic RCC correlates with aggressive histology and advanced disease. Aside from known risk factors for RCC, evidence suggest that the development of RCC can be partially explained by genetic variations among the populations. Highly polymorphic cytosolic glutathione S-transferases are known to be involved in both the development and the progression of renal cell carcinoma.

Methods: GSTP1 genotype was determined in 99 ccRCC patients and 326 matched-controls by qPRC method, using TaqMan® SNP Genotyping Assay. The risk for disease was computed by odds ratios (OR) and 95% confidence intervals (CI) using logistic regression analysis Furthermore, overall survival was analyzed as well by Kaplan-Meier method and Cox proportional hazard regression model.

Results: GSTP1-variant genotype was associated with 5-fold increased risk for ccRCC in comparison with GSTP1-wild type genotype ($p < 0.001$). Moreover, survival analysis clearly indicated shorter overall survival in ccRCC patients with GSTP1-variant genotype, however without reaching statistical significance ($p = 0.166$). Additionally, ccRCC patients with GSTP1-variant genotype had a 7-fold higher hazard ratio ($p = 0.177$), compared to the carriers of GSTP1-wild type genotype.

Conclusion: GSTP1-variant genotype contributed independently towards the risk of ccRCC in our patients. Moreover, GSTP1-variant genotype is associated with poor postoperative prognosis in ccRCC.

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