P99 GENETIC MAPPING FOR THYROTOXIC PERIODIC PARALYSIS

LAU KS, CHAN VNY, KUNG AWC.
Department of Medicine, The University of Hong Kong, Queen Mary Hospital, Hong Kong, PRC.

TPP is a complication of thyrotoxicosis affecting 25% of thyrotoxic southern Chinese male but not female patients. Paralysis due to hypokalemia typically occurs after a high carbohydrate meal. This episodic paralysis will remit with the control of thyrotoxicosis but may recur with relapse of the disease. Shifting of potassium into intracellular space results in hypokalemia and paralysis. Increased Na, K-ATPase activity has been observed in TPP patients when compared to thyrotoxic Graves' disease (GD) patients and normal controls. To study the association of Na, K-ATPase genotype with TPP, we evaluated TPP patients (n = 100) and compared them to male GD patients without TPP (n = 60) and normal male subjects (n = 70). A set of polymorphic microsatellite markers with a genetic distance < 10 cM was used to study the allelic distribution. Our results showed that there was no significant difference between the 3 groups in the allelic distribution for all markers associated with the Na, K-ATPase $\alpha 1$ (1p13), $\alpha 2$ (1q21-23), $\alpha 3$ (19q13.2) and $\beta 1$ (1q22-25), $\beta 2$ (17p13.1), $\beta 3$ (3q22-23) genes. There was also no association of CACNL1A3 (dihydropyridine sensitive calcium channel receptor gene, located at chromosome 1q32) with TPP. Mutation of CACNL1A3 gene is linked to Familial Hypokalemia Periodic Paralysis in Caucasians. On screening the X-chromosome, one of the alleles (size 289) of the marker DXS1214 in the region of Xp11.3-21 was observed to be more frequent in TPP subjects when compared to GD patients and normal subjects ($p < 0.05$), suggesting that this locus may be linked to TPP.

P100 The Function of Fas, Fasl, Bcl-2 in the Pathogenesis of Autoimmune Thyroid Disease

Chen Shenren, Zheng Zhichao, Deng Lijuan
Department of Endocrinology, 2nd Affiliated Hospital of Shantou University Medical College Shantou, 515041, China

Objective To investigate the expression and function of apoptosis-related protein: Fas, FasL, and Bcl-2 in the pathogenesis of autoimmune thyroiditis. **Methods** Immunohistochemical staining was performed on 20 thyroid follicular adenoma, 20 Hashimoto's thyroiditis(HT), and 20 graves' disease(GD). **Results** All the cases expressed fas, mainly on the cell surface and cytoplasm. FasL was found in all except one of the thyroid adenoma(TA). Thyroid follicles in HT samples exhibited strong staining for Fas ,in contrast to GD and TA that exhibited moderate Fas. Immunostaining for FasL was high in thyroid follicles but weak in infiltrating lymphocytes(ILC) of HT, moderate in all of GD, and minimal or no in TA. While Bcl-2 was only expressed 15 in HT,14 in GD, and 12 in TA. In HT, ILC stained strongly for Bcl-2 and weakly for FasL, in contrast to follicular cells stained weakly for Bcl-2 and strongly for FasL.In GD, Bcl-2 was moderate to strong similarly in follicular cells and ILC. And, it was weak to moderate in TA. **Conclusion** The expression of Fas , FasL ,Bcl-2 in Hashimoto's thyroiditis and graves' disease was nearly similar . FasL and Fas strong expression and Bcl-2 weak expression on the follicles in HT may induce apoptosis. These results provided further proof of the function of Fas and its ligand and Bcl-2 in the pathogenesis of autoimmune thyroid disease. The lymphocytes do not seem to be directly engaged in the process with their own FasL, but they may provide some cytokines that , in turn , up-regulates Fas and/or FasL leading to apoptosis.