Joint meeting

The 7th International Symposium on Neurocardiology

NEUROCARD 2015

The 6th International Symposium on Noninvasive Electrocardiology

SCIENTIFIC PROGRAM & BOOK OF ABSTRACTS

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O6 Effects of glutathione S-transferase T1 and M1 deletions on electrocardiographicand heart rate variabilityparameters in patients with vasovagal syncope

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Background: Glutathione (GSH) conjugating enzymes, glutathione S-transferases (GSTs), are present in different subcellular compartments including cytosol, mitochondria, endoplasmic reticulum, nucleus and plasma membrane. Altered GST expression has been implicated in cardiac and neurological diseases. Vasovagal syncope (VVS) is the most frequent type of syncope affecting about 25% of the population at least once during life. Sympathetic nervous system can control cardiovascular function and its failure could result in syncope. Depletion of the intracellular antioxidant GSH extended to the level of the whole heart could result in heterogeneous reactive oxygen species (ROS) production, and inhibition of oxidative phosphorylation could slow the heart rate and shortening of the action potential duration. Decreased functionality of antioxidant enzymes may pose a greater risk of toxic insult caused by chronic oxidative stress. Two detoxification enzymes GST isoforms Mu1 (GSTM1) and Theta1 (GSTT1) have role in antioxidant defense. Genetic polymorphisms of GST enzymes are gene deletions yielding no transcription or translation of the respective enzymes. The aim of the study was to investigate the association of GTSs polymorphisms with frequency domain and time domain indexes of HRV and BPV in Serbian patients with VVS.
Methods: The 70 patients with VVS of mean age 32.9±12.4 years (mean±SD, 80% females and 20% males) where tested for association of GST T1 and GST M1 deletion genetic variants with frequency domain and time domain parameters of HRV. All patients underwent 24-h holter ambulatory electrocardiographic (ECG) monitoring and analyzed for HRV parameters (Task Force® Monitor) prospectively, before and after therapy. Genotyping was done by PCR. Statistical analysis was performed using SPSS software (SPSS 20.0).
Results and Conclusion: The GSTT1 deletion variant showed no significant association with any of the tested HRV parameters in either of two performed measurements. We have found trend toward association of GSTM1 deletion variant with significantly lower values of maxQTc (p=0.066) and SDNN (p=0.053). After the second measurement, the significant association was found for the Max Qt (p=0.01), Max QTc (0.004) and SDNN (p=0.03). Deletion genetic variant carriers had significantly lower values of tested parameters. These results show possible influence of oxidative stress on HRV, and suggest the need for further genetic association studies on larger patient groups.