



ACC.14

TCT@ACC-12 | innovation in intervention

A462

JACC April 1, 2014

Volume 63, Issue 12



## Arrhythmias and Clinical EP

**WHITE BLOOD CELL RESPONSE AFTER CRYOBALLOON ABLATION FOR ATRIAL FIBRILLATION IS MODIFIED BY VIRAL ANTI-INFLAMMATORY PROTEIN TREATMENT**

Poster Contributions

Hall C

Monday, March 31, 2014, 9:45 a.m.-10:30 a.m.

Session Title: Arrhythmias and Clinical EP: New Observations on Pathophysiology of Atrial Fibrillation

Abstract Category: 4. Arrhythmias and Clinical EP: AF/SVT

Presentation Number: 1256-114

Authors: *Mohammad Al-Ani, Zheng Donghang, Adisson Fortunel, Thomas Burkart, William Miles, Alexandra Lucas, Matthew McKillop, Division of Cardiovascular Medicine, University of Florida, Gainesville, FL, USA*

**Background:** Activation of innate immune responses after atrial fibrillation (AF) ablation may lead to collateral atrial myocardial damage, fibrosis, and recurrent arrhythmia. The extent of the inflammatory response to cryoballoon ablation (CB) is not yet well defined. Non-specific inflammation suppression with corticosteroids has had a varied clinical response in post-AF ablation patients. Two highly potent immunomodulatory Myxoma viral proteins, Serp-1 and MT-7, have been isolated. These proteins block cytokine/chemokine and serine protease activation. Both have been studied in humans and animal models. In this study, we examined the systemic inflammatory response to AF ablation and the potential to modify inflammatory cell activation after ablation using these viral proteins.

**Methods:** Fifteen patients were studied after CB (n=10) or CB with radiofrequency ablation (CBRF) (n=5). White blood cells (WBCs) were isolated for analysis and treatment with the anti-inflammatory viral proteins ex-vivo. Serum markers of inflammation and myocardial injury were measured pre and post ablation.

**Results:** There was a statistically significant rise in mean troponin, high-sensitivity C-reactive protein, WBCs, and Fibrinogen by 1.55 ng/L, 17.82 mg/L, 3.21 cells/ $\mu$ L and 38.32 mg/dL respectively ( $P < 0.001$ ) with no difference between CB and CBRF groups. Isolated cell activation was assessed by membrane fluidity analysis using a fluorescent bispyrenylpyrene assay. WBCs isolated from venous blood after CB and exposed to phorbol myristate acetate with either MT-7 or Serp-1 produced a significant decrease in membrane fluidity indicating reduced cell activation (corrected mean lex/lmon for Serp-1 0.086, control 0.981,  $P = 0.001$ ; M-T7 0.026, control 1.032,  $P = 0.005$ ). Both proteins demonstrated equal efficacy.

**Conclusion:** CB and CBRF similarly increase serum markers of inflammation. Ex-vivo treatment of circulating WBCs from these patients with viral anti-inflammatory proteins blocks cell activation. Suppression of the inflammatory chemokine or protease response to AF ablation represents a potential new target to reduce post-ablation inflammation and possibly arrhythmia recurrence.