Haghi et al. also think that RV involvement in TTC seems to be associated with a more severe impairment in LV systolic function, which could affect the precise evaluation of prevalence of RV involvement in TTC and further the prehension of pathophysiological mechanisms of TTC. RV WMA condition in patients with TTC and normal LV function needs to be studied.

References

Ze-Zhou Song
Department of Ultrasound
The First Affiliated Hospital
College of Medicine
Zhejiang University
#79 Qingchun Road
Hangzhou 310003
Zhejiang Province
PR China
Fax: +86 571 8723 6628
E-mail address: zezhou_song@126.com

Jing Ma
Internal Medicine Department
Hangzhou 3rd Hospital
Hangzhou
PR China

Dariusch Haghi
I. Medical Department
University Hospital Mannheim
Mannheim
Germany
Tel: +49 621 3832612
Fax: +49 621 3832172
E-mail address: dariush.haghi@med.ma.uni-heidelberg.de

Theano Papavassiliu
I. Medical Department
University Hospital Mannheim
Mannheim
Germany

Udo Sechtem
Department of Cardiology
Robert-Bosch-Krankenhaus
Stuttgart

doi:10.1093/eurheartj/ehl572

Online publish-ahead-of-print 5 March 2007

Right ventricular involvement in Takotsubo cardiomyopathy: reply

We thank Ze-Zhou Song and Jing Ma for their interest in our work. We are unaware of any data showing that hypertension, hypercholesterolaemia, diabetes, Graves’ disease, or paroxysmal atrial fibrillation can cause regional right ventricular wall motion abnormalities, although some of these conditions may affect global parameters of ventricular function. However, pulmonary hypertension which can be encountered in chronic obstructive pulmonary disease may cause regional right ventricular dysfunction. In fact, we have observed reversible akinesis of the right ventricular apex indistinguishable from right ventricular dysfunction of Takotsubo cardiomyopathy in a case of pulmonary embolism. As exacerbation of obstructive pulmonary disease was the triggering event in two of our patients, we cannot exclude that acute pulmonary hypertension has contributed to the observed right ventricular wall motion abnormalities in those two patients.

We did not specifically look for changes of treatment regimens upon follow-up cardiovascular magnetic resonance imaging. These data were only available for those three patients who had their follow-up exam within 10 days of admission. In these three patients, there was no change of treatment with regard to their co-morbidities.

The idea that left ventricular dysfunction in itself may cause right ventricular dysfunction is intriguing. In fact, this issue is currently under investigation at our institutions and results will shortly be available.

Reference

Dario Schimpf
E-mail address: darioschimpf@usa.net

Udo Sechtem
Department of Cardiology
Robert-Bosch-Krankenhaus
Stuttgart

doi:10.1093/eurheartj/ehm006

Online publish-ahead-of-print 21 March 2007

New risk factors of heart failure?

We appreciate very much Siirilä-Waris et al. for their excellent work on risk factors of heart failure. Progression of heart failure may be due to an initial cardiac injury, or mutation of the genetic programme, in association with activation of neurohormones and proinflammatory cytokines, resulting into immune activation, which worsens heart failure. Therefore, it may be proposed that any factor which can block neuroendocrine activation would be protective, whereas other factors that can enhance neurohormonal activity would be the risk factors of heart failure. Decreased heart rate variability, increased blood pressure variability, dyslipidemia, increased IL-6, IL-1, TNF-alpha, C-reactive protein, and adhesion molecules are other important determinants of mortality in patients of heart failure. Presence of coronary artery disease is also an important risk factor of mortality, which becomes worse if there is coexisting cardiac cachexia or obesity among these patients.

Apart from above risk factors, nutritional factors such as increased consumption of proinflammatory foods; refined starchy and sugar, trans fatty acids, w-6 fatty acids, and saturated fat may enhance proinflammatory cytokines. Therefore eating proinflammatory foods could be an important cause of increased mortality in heart failure, because these patients have a pre-existing proinflammatory milieu. These foods may produce oxidative stress, free fatty acids, and proinflammatory substances, which result in endothelial dysfunction. Glucose ingestion in normal subjects is associated with increased superoxide generation in leukocytes and mononuclear cells, as well as with raised amount and activity of nuclear factor-kB (NF-kB), a transcriptional factor regulating the activity of at least 125 genes, most of which are pro-inflammatory. Increased consumption of refined carbohydrates also causes an increase in two other proinflammatory transcription factor, activating protein-1 (AP-1), and Egr-1, the first regulating the transcription of matrix metallo-proteinases and the second modulating the transcription of tissue factor and plasminogen activator inhibitor-1. These adverse factors related to diet may worsen the prognosis in heart failure.

A mixed meal from a fast-food chain has also been shown to induce activation of NF-kB associated with the generation of reactive oxygen species (ROS) by mononuclear cells. Superoxide anion appears to be an activator of at least two major proinflammatory transcription factor, NF-kB and AP-1. These observations are consistent with previous findings, demonstrating that after oral or intravenous glucose challenges, in both normal subjects and patients with type 2 diabetes mellitus, there is an increased generation of ROS and raised circulating levels of proinflammatory cytokines, such as TNF-α, IL-6, and IL-18. In apparently healthy subjects, a single high-fat meal produces endothelial activation, as evidenced by increased concentrations of the adhesion molecules VCAM-1 (vascular cell adhesion molecule-1) and ICAM-1 (intercellular adhesion molecule-1), in association with raised plasma concentrations of IL-6 and TNF-α. A high-fat meal may increase the circulating levels of...
IL-18, a proinflammatory cytokine supposed to be involved in plaque destabilization associated with the simultaneous decrease of circulating adiponectin, an adipocyte-derived protein with insulin sensitizing, anti-inflammatory, and antiatherogenic properties. Consumption of a high-fat meal together with vegetable foods rich in natural antioxidants largely prevent the negative effects on endothelial function.\(^3\)\(^4\) In particular, endothelial dysfunction acutely triggered by the consumption of a high-fat meal rich in saturated fatty acids is reduced by the simultaneous consumption of a vegetable serving including pepper (100 g), tomatoes (100 g), and carrots (200 g). It seems that these foods are slowly digested and absorbed without causing any significant increase in free radical stress and free fatty acids, which is a characteristic of Columbus foods (www.Columbus-concept.com) and therefore such foods may improve the prognosis in heart failure. Cytokines are known to worsen the neurons which worsen in presence of deficiency of ω-3 fatty acids, responsible for the survival of neurons. Omega-3 fatty acids can regulate leptin gene expression and the concentrations of anandamides in the brain, which in turn binds to endogenous cannabinoid receptors and regulate food intake. It is possible that in the clinical trials in heart failure, using agents to inhibit TNF-alpha activity showed disappointing results, because proinflammatory effect of diet was not considered. It seems that anticytokine therapy in heart failure would work only in patients with proven proinflammatory status with due consideration to diet. Coenzyme Q10 and ω-3 fatty acids can regulate leptin gene expression and deficiency of ω-3 fatty acids, responsible for the survival of neurons. Omega-3 fatty acids can regulate leptin gene expression and the concentrations of anandamides in the brain, which in turn binds to endogenous cannabinoid receptors and regulate food intake. It is possible that in the clinical trials in heart failure, using agents to inhibit TNF-alpha activity showed disappointing results, because proinflammatory effect of diet was not considered. It seems that anticytokine therapy in heart failure would work only in patients with proven proinflammatory status with due consideration to diet. Coenzyme Q10 and ω-3 fatty acids can regulate leptin gene expression and deficiency of ω-3 fatty acids, responsible for the survival of neurons. Omega-3 fatty acids can regulate leptin gene expression and deficiency of ω-3 fatty acids, responsible for the survival of neurons. Omega-3 fatty acids can regulate leptin gene expression and deficiency of ω-3 fatty acids, responsible for the survival of neurons. Omega-3 fatty acids can regulate leptin gene expression and deficiency of ω-3 fatty acids, responsible for the survival of neurons.