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


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.\(^{2,3}\) 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 w-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 antioxidant therapy in heart failure would work only in patients with proven proinflammatory status with due consideration to diet. Coenzymes Q10 and w-3 fatty acids or even anti-inflammatory foods and statins, because of their pleitropic effects, may prove beneficial in this setting. We hope that future studies will bring further insight to the mechanisms by which inflammation affects prognosis in patients with heart failure and thus help creating new therapeutic options.


**References**


**References**


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