



SERIAL MEASUREMENT OF TUMOR NECROSIS FACTOR-A, INTERLEUKIN-6 AND VASCULAR ENDOTHELIAL GROWTH FACTOR AS A FUNCTION OF CACHEXIA IN PATIENTS WITH CHRONIC HEART FAILURE: RESULTS FROM THE PROBNP OUTPATIENT TAILORED CHRONIC HEART FAILURE (PROTECT) STUDY

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Background: Cachexia is associated with poor outcomes in patients with heart failure (HF), but little information is available on the pathophysiology behind the phenomenon. While inflammation is thought to play an important role in HF-related cachexia, it is unclear whether information may be gained from measurement of inflammatory biomarkers such as tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6), or vascular endothelial growth factor (VEGF) in this setting.

Methods: TNF- α , IL-6, and VEGF as well as weight and body-mass index (BMI) were measured in 115 subjects with chronic stable HF at each encounter over 10 months. Cachexia was defined as weight loss \geq 5% from baseline or BMI < 20kg/m2.

Results: Baseline values of TNF- α , IL-6 or VEGF were not significantly different in patients with or without cachexia. However, patients with cachexia had increasing TNF- α values across visits (median change=1.6 pg/mL [-1.1, 2.8 pg/mL]) while those without cachexia had slightly decreasing values over time (-0.2 pg/mL [-1.75, 1.4 pg/mL], p=0.03). A change in IL-6 or VEGF over time was not significantly different in patients with or without cachexia. There was a significant inverse relationship between baseline TNF- α values and weight (baseline weight p=-0.207, p=0.03; final weight p=-0.218, p=0.03). A trend towards a significant correlation between time spent with a TNF- α concentration below the median and final weight (p=0.187, p=0.07) was also seen. TNF- α and IL-6 predicted time to first cardiovascular event in unadjusted Cox regression models (p=0.05 and 0.04 respectively), but in adjusted models were not predictive of time to first cardiovascular event (p>0.05 for unadjusted or adjusted analysis).

Conclusion: In patients with chronic stable HF, a rise in TNF- α concentration is linked to incident cachexia. Further studies to understand the pathophysiologic role and therapeutic significance of TNF- α in HF-related cachexia are warranted.