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Circulating cytokine levels are elevated in symptomatic patients with congestive heart failure (CHF). Whether peripheral abnormalities which are present in the skeletal muscles (SM) and vasculature of symptomatic patients contribute to cytokine production in CHF is unknown. Accordingly, vastus lateralis SM biopsies were obtained in 14 patients with functional class (FC) I to IV CHF (peak aerobic capacity (VO₂) ranging from 6 to 23 ml/kg min⁻¹) and in 6 age-matched normal subjects who served as controls. Mean percent areas and mean fractional area were 55.7 ± 22.2% respectively. The etiology of CHF was coronary artery disease (n = 9) or hypertension (n = 5). All patients were treated with angiotensin converting enzyme inhibitors, diuretics, and loop diuretics. Paraffin sections were evaluated by hematoxylin and eosin stain and immunostained using a histocore monoclonal (KP-1), 10 cm cell marker (C23a) and an B cell marker (L26). Tumor necrosis factor α (TNFα) levels were determined by immunoassay (Quantikine HS R&D Systems). No B cells were detected. Reproductibility of SM biopsy findings was established in 3 patients who underwent 2 serial biopsies. In conclusion, increased numbers of perivascular and interstitial T cells in SM accompany the rise in circulating cytokine levels as the symptoms progress in patients with CHF.

Clinical Cardiology: Exercise Factors by Gender, Age, and Functional Status
Tuesday Afternoon
Exhibit Hall
Abstracts 2908 – 2916

Does the Change in Quantitatively Assessed Coronary Artery Disease After Lipid-Lowering Therapy Relate to the Change in Functional Status of the Patient?


In patients with chronic congestive heart failure (CHF), the plasma levels of soluble cytokine receptors such as soluble tumor necrosis factor receptor-I (sTNF-R-I) and sTNF-R-II, and soluble adhesion molecules such as soluble vascular adhesion molecule-1 (sVCAM-1), and soluble vascular cell adhesion molecule (sVCAM-1) in 83 patients with CHF (all ventricular ejection fraction(EF)<45%, mild CHF; NYHA II, n=40, severe CHF; NYHA III-IV, n=43) by means of enzyme-linked immunosorbent assay.

Furthermore, they were monitored for a follow-up period of more than 1 year. The plasma level of sTNF-R-I increased with the severity of CHF (mild CHF: 1025 ± 74 pg/ml vs. severe CHF: 1264 ± 207 pg/ml, p<0.0001) and the plasma level of sTNF-R-II also increased with the severity of CHF (mild CHF: 3320 ± 250 pg/ml vs. severe CHF: 4834 ± 452 pg/ml, p<0.0025). The plasma levels of sVCAM-1 and sICAM-1 were also increased in relation to the severity of CHF (mild CHF: 234 ± 11 ng/ml vs. severe CHF: 318 ± 22 ng/ml, p=0.001, mild CHF: 798 ± 30 ng/ml vs. severe CHF: 1103 ± 35 ng/ml, p<0.0001, respectively ).

Cor peripheral capillaries was performed to determine the independent significant predictors of EF; plasma levels of sTNF-R-I, sTNF-R-II, sICAM-1, and sVCAM-1, high plasma levels of sTNF-R-I (p=0.013), sVCAM-1 (p=0.04), and EF=p=0.004) were shown to provide independent significant prognostic values in 83 CHF patients. These findings indicate the significant relation between the plasma levels of soluble cytokine receptor and soluble adhesion molecule and the severity and mortality of patients with CHF, suggesting an important role of the immune system activation in the pathophysiology and progression of CHF.