Abstracts of Invited Lectures
Poster Abstracts

Falk Workshop

INFLAMMATION & CANCER

Hamburg
January 26 – 27, 2012

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Association of chronic systemic inflammation, liver cirrhosis, cancerogenesis with T894G polymorphism of endothelial nitric oxide synthase gene (eNOS) and vascular injury

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Introduction: Whilst the role of proangiogenic and proinflammatory factors is well established in cancerogenesis, the association of T894G polymorphism of endothelial nitric oxide synthase (eNOS) gene with cytokines, cardiovascular injury in patients with hepatic carcinoma is unclear.

Methods: Study group included 50 patients with liver malignancies due to non-viral cirrhosis (20 female, 24 male, mean age 63.2 ± 8.7); control group included 10 healthy volunteers. All patients had vascular injury in a form of concomitant Arterial Hypertension (AH) and Chronic Heart Failure (CHF). IL-4, TNF-α, TGF-β1, pro-Atrial Natriuretic Peptide (proANP) plasma concentrations were defined by IEA; eNOS (T894G) gene polymorphism was assessed with PCR.

Results: Difference in genotypes distribution between groups was not significant. Presence of T-allele in patients with liver cirrhosis was associated with reliable increase of AST activity (27.4%, $p < 0.05$), urea concentration (33.3%, $p < 0.05$), creatinine (22.2%, $p < 0.05$) compared to GG-carriers. In T-allele carriers concentration of proANP was higher (89.2%, $p < 0.001$), as well as diameter of left atrium was larger (13.6%, $p < 0.01$), than in patients with GG-genotype. IL-4, TNF-α and TGF-β1 didn't differ reliably between genotypes, but TNF-α and proANP level were significantly higher in research patients than in control group ($p < 0.001$). In research group males T-allele also combined with the increased LVMI (by 12.2%, $p < 0.05$) compared to GG-genotype patients.

Discussion/Conclusion: eNOS is connected with hepatic circulation via changes of endothelial function, inflammation and metabolic syndrome and reflects widespread vascular damage as proved by cardiovascular changes. We hypothesized that oxidation, systemic inflammatory reaction and unregulated cellular proliferation depends on eNOS gene expression. T-allele associates with cytolysis, indirect fibrotic liver changes, cardiovascular failure progression (proANP) and may be a risk factor of liver cancer.