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to controls. Therefore, a low EF, an important prognostic determinant, is not (PWV) or even inversely (central PP, Alx) represented by these measures of arterial function. When they are used for risk stratification, knowledge of systolic function is required as well.

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INTENSIVE MEDICAL THERAPY NORMALISES FLOW-MEDIATED VASODILATATION AND INTIMA-MEDIA THICKNESS OF PATIENTS WITH COEXISTING HEART FAILURE AND DIABETES

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Coexistence of heart failure (HF) and type 2-diabetes is associated with high cardiovascular mortality. Intensive medical treatment of HF patients with diabetes may reduce the endothelial dysfunction and the accelerated atherosclerotic process seen in these patients.

To study this, we investigated the endothelial function and the presence of atherosclerosis measured by flow-mediated vasodilatation (FMD) and intima-media thickness (IMT) in intensively treated patients with coexisting HF and diabetes.

Methods: FMD of the brachial artery and IMT of the common carotid arteries were determined in 26 patients with HF and diabetes who were in intensive medical therapy as well as in 19 healthy controls. The two groups were matched according to age and sex. In all subjects left ventricular ejection fraction was measured by two-dimensional echocardiography (LVEF). Biochemical parameters including serum cholesterol, high and low density lipoprotein-cholesterol, triglyceride, glucose, hemoglobin/hemoglobin-A_{1c} (HbA_{1c}), brain natriuretic peptide (BNP) and N-terminal pro-BNP were also assessed.

Results: Mean FMD and IMT did not differ significantly between patients and controls. LVEF was lower in patients compared to controls ($P < 0.001$). The group of patients had a higher mean BNP, NT pro-BNP, triglyceride, HbA_{1c} and glucose in comparison to controls. Cholesterol, HDL-cholesterol and LDL-cholesterol were lower in patients compared to controls.

Conclusion: Intensively treated patients with coexisting HF and diabetes seem to have normal endothelial function as measured by FMD and they have no sign of accelerated atherosclerosis as measured by IMT. This suggests a positive effect of medication on the cardiovascular alterations in this group of patients.

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CAROTID INTIMA-MEDIA THICKNESS IN NON-SMOKING HIV PATIENTS TREATED WITH ANTIRETROVIRAL THERAPY: FOCUS ON LIPID PROFILE

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Introduction: Increased cardio-vascular risk in HIV patients in antiretroviral therapy (ART) may be due to HIV infection, direct effect of ART or dyslipidemia induced by ART. Our aim was to study the relative importance of HIV, ART and dyslipidemia on atherosclerosis as measured by carotid artery intima-media thickness (IMT). To do so, we compared IMT in non-smoking HIV patients with high or low serum cholesterol levels as well as in healthy volunteers.

Methods: HIV patients in ART with normal (≤ 5.5 mmol/L; $n = 13$) or high cholesterol (≥ 6.5 mmol/L; $n = 12$) as well as healthy controls ($n = 14$) were included. All were non-smokers and had never received medication for dyslipidemia or hypertension. IMT was measured by ultrasonography.

Results: IMT in HIV patients with hypercholesterolemia (≤ 5.5 mmol/L), HIV patients with normal cholesterol (≥ 6.5 mmol/L) and controls was 683 ± 119 , 656 ± 99 and 657 ± 99 μm , respectively. IMT was similar in patients receiving and not receiving protease inhibitors (658 ± 117 vs. 687 ± 97 μm). IMT in HIV patients correlated inversely with HDL-cholesterol levels ($r = -0.50$; $p = 0.01$), whereas no correlation was found with total cholesterol or LDL-cholesterol.

Conclusions: In non-smoking HIV patients receiving ART no sign of accelerated atherosclerosis as assessed by IMT were found even if patients were hypercholesterolemic. IMT correlated with HDL- but not LDL-cholesterol further indicating that reduction in cardio-vascular risk in these patients probably mainly should be aimed at reducing traditional risk factors rather than lipid lowering with statins.

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ARTERIAL STIFFNESS AND ITS RELATION TO ENDOTHELIAL AND MICROCIRCULATORY FUNCTIONS IN HEALTHY MALES

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Arterial stiffness is suggested to be a replacement method to measurements of brachial diameter following the release of an ischemic stimulus. We tested this hypothesis by comparing resting large [C1] and small [C2] arterial stiffness (as well as total peripheral resistance [TPR]) with brachial flow mediated dilation (FMD, indicator of endothelial function). Furthermore, the anatomical location that C2 represents is unclear. Could this reflect microcirculatory changes? We tested this hypothesis by comparing C2 with concurrent microcirculatory responses.

29 subjects (aged 18 to 30 years) were investigated. A tonometer recorded the radial blood pressure; pulse waveform analysis (PWA) was used to calculate C1, C2 and TPR. These parameters were correlated with forearm FMD responses. FMD was taken as the % maximal change in brachial arterial lumen diameter following cuff release and measured by B-mode ultrasound. Microcirculatory parameters included finger flux measured by Laser Doppler Fluxmetry [LDF]; pulsatile finger volume measured by photoplethysmography [PPG] and palm skin temperature measured by infrared thermography [Tpalm].

FMD linearly related to C1 ($r = 0.4$, $P = 0.04$), but not C2 ($P > 0.05$). Comparison of arterial stiffness against microcirculatory responses revealed a weak relation between C2 and %PPG ($r = 0.38$, $P = 0.07$), but no relations were found for C1 ($P > 0.05$). For TPR, a linear relation was found with %PPG ($r = 0.50$, $P = 0.01$) and %Tpalm ($r = 0.59$, $P < 0.001$). No other correlations were evident ($P > 0.05$).

Small arterial stiffness (C2) is neither a viable marker for endothelial function nor of microcirculatory responses. The findings show that sonographically assessed brachial FMD is the better method for endothelial function testing.