Quantification of epicardial adipose tissue in patients with major depressive disorder by magnetic resonance imaging

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Target audience: Radiologists and physicists with an interest in quantification of the components of cardiac adipose tissue and identification of risk factors for coronary artery disease.

Introduction: Major depressive disorder (MDD) is associated with an increased risk for the development of coronary artery disease[1]. Epicardial adipose tissue (EAT), which is a metabolically active visceral fat depots, has been implicated in the pathogenesis of coronary artery disease[2]. We therefore hypothesized increased volumes of EAT in patients with MDD compared to healthy controls.

Methods: 50 inpatients with MDD and 25 healthy controls were included into the study. MDD was diagnosed according to the Diagnostic and Statistical Manual of Mental Disorders (DSM IV) and by structured clinical interviews (SCID). MDD patients were divided into two groups: 33 patients with recurrent MDD (≥2 major depressive episodes) and 17 patients with MDD plus dysthymia ("double depression", a chronic subtype of depressive disorder with symptoms ≥2 years). MRI was performed using a 1.5 Tesla scanner (MAGNETOM Avanto, Siemens Healthcare). For quantification of EAT ECG-gated T1-weighted dark blood turbo spin echo sequences were acquired in short- and long-axis views. EAT, paracardial adipose tissue (PAT) and total cardiac adipose tissue (TAT = EAT + PAT) were calculated between the atrioventricular plane and the apex by manual segmentation using QMass 7.1 software (Medis, Leiden, The Netherlands) as shown in Figure 1. Concentrations of fasting glucose, cortisol, insulin, tumor necrosis factor-alfa (TNF**α**), and interleukin-6 were measured, and factors contributing to the metabolic syndrome were assessed according to ATP-III criteria. Differences between groups were determined by univariate analysis of variance with age and weight as covariates. Values are given as mean±SEM after adjustment for age and weight.

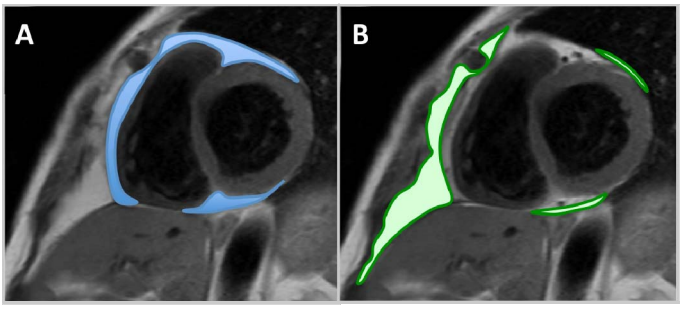

Figure 1: Segmentation of epicardial adipose tissue (EAT; A) and paracardial adipose tissue (PAT; B) from short axis T1 dark blood images.

Results: In all 50 MDD patients, age and weight adjusted volumes of EAT (79±3 ml versus 67±5 ml, *p<0.05), PAT (197±8 ml versus 145±11 ml; ***p<0.001) and TAT (276±11 versus 212±15 ml, **p<0.01) were significantly increased compared to controls (Figure 2). After subdividing the depressed patients into patients with recurrent MDD and "double depression" significant differences between cardiac fat volumes of the subgroups compared to controls were found. Cardiac fat volumes were highest in the subgroup of patients with "double depression" (Figure 2). Corresponding to increased volumes of EAT, TNFα levels were significantly increased in patients with MDD and both subgroups (**p<0.01) and fasting cortisol was higher in MDD patients than in control subjects (*p<0.05). The metabolic syndrome was observed in 8% of controls, 9% of patients with recurrent MDD and 25% of patients with "double depression".

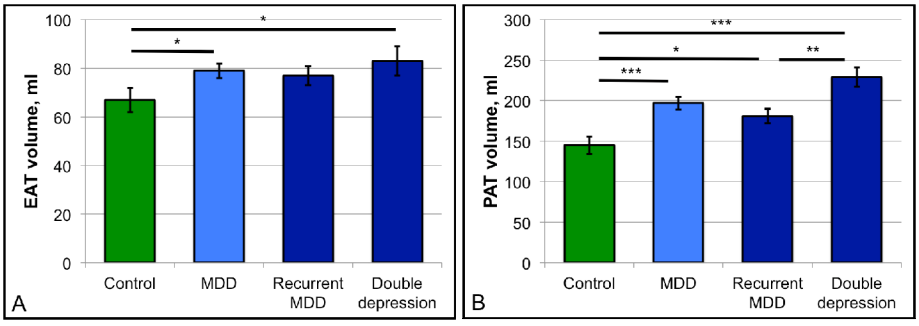

Figure 2: EAT and PAT in patients with depression and controls. Depicted are volumes of EAT (A) and PAT (B) in controls, all depressed patients (MDD) and subgroups of patients with a recurrent MDD and "double depression" (chronic depression). Depicted are mean values and SEM after adjustment for age and weight. Values are standardized to an age of 42.5 years and a weight of 79.3 kg. *p<0.05, **p<0.01, ***p<0.001.

Discussion: EAT and PAT were increased in patients with major depressive disorder and highest in a subgroup of patients with chronic depressive disorder ("double depression"). Therefore, increased EAT may independently contribute to the higher cardiovascular morbidity in depressed patients and particularly in the chronic form of this disease.

References:

[1]Surtees PG, et al. (2008): Depression and ischemic heart disease mortality: evidence from the EPICNorfolk United Kingdom prospective cohort study. Am J Psychiatry. 165:515-523.

[2]Yerramasu A, et al. (2012): Increased volume of epicardial fat is an independent risk factor for accelerated progression of sub-clinical coronary atherosclerosis. Atherosclerosis. 220(1):223-30.