Characteristic changes of systolic and diastolic function in rat models of type 1 versus type 2 diabetes mellitus assessed by speckle-tracking echocardiography

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Left ventricular (LV) dysfunction is a frequent consequence of diabetes mellitus (DM) even in the absence of coronary artery disease. Comparison of animal models of type 1 and type 2 DM may contribute to a deeper pathophysiologic understanding of diabetic cardiomyopathy. Gold standard LV pressure-volume (PV) analysis provides a detailed hemodynamic characterization, however, the non-invasive speckle-tracking echocardiography (STE) may be a powerful method to assess the deterioration of systolic and diastolic function.

Therefore, we aimed to comparatively investigate diabetic cardiomyopathy by PV analysis and STE in rat models of type 1 and type 2 DM.

Rat models of type 1 (8 weeks after DM induction in Sprague-Dawley rats by streptozotocin, n=8) and type 2 DM (inbred Zucker Diabetic Fatty rats at the age of 32 weeks, n=7) and corresponding control animals (n=5 and n=8, respectively) were compared. Echocardiography was performed using a 13MHz linear transducer to obtain LV short-axis recordings for STE analysis (EchoPAC). Beyond global circumferential strain (GCS), peak strain rate values in systole (SrS), isovolumic relaxation (SrIVR) and early diastole (SrE) were measured. LV PV analysis was performed to calculate load-independent contractility indices (i.e. preload-recruitable stroke work [PRSW]), time constant of LV pressure decay (tau), and diastolic stiffness parameters (i.e. slope of end-diastolic PV relationship [EDPVR]).

In type 1 DM, contractility and active relaxation were deteriorated to a greater extent compared to type 2 (relative impairment type 1 vs. type 2 DM; PRSW 46±13 vs. 21±14%; tau: 64±20 vs. 10±7%, both $p<0.01$). In contrast, diastolic stiffness impaired more significantly in type 2 DM (EDPVR: 22±11 vs. 46±17%, $p<0.01$). Correspondingly, STE described more severe systolic dysfunction in type 1 (type 1 DM vs. control; GCS: -13.1±1.8 vs. -16.9±1.3%, SrS: -2.48±0.37 vs. -4.58±0.18 1/s, both $p<0.01$) compared to type 2 DM (type 2 DM vs. control; GCS: -14.2±1.8 vs. -16.0±2.1%, NS; SrS: -2.68±0.35 vs. -3.23±0.57 1/s, $p<0.05$; relative impairment type 1 vs. type 2 DM, SrS: 46±8 vs. 17±11%, $p<0.001$). Among diastolic STE parameters, SrIVR was more decreased in type 1 (relative impairment type 1 vs. type 2 DM; 55±5 vs. 22±16%), however, SrE

referring to diastolic stiffness was more reduced in the type 2 DM model (23±8 vs. 32±7%, both $p<0.05$). In type 1 DM rats, SrS correlated robustly with PRSW ($r=-0.924$, $p<0.001$), SrIVR with tau ($r=-0.729$, $p<0.05$), while in type 2 DM rats SrE was closely related to EDPVR ($r=-0.722$, $p<0.01$).

Diabetic cardiomyopathy is characterized by overt systolic dysfunction and impaired active relaxation in type 1 DM, while increased diastolic stiffness is the leading abnormality in type 2 DM. STE corresponds to PV analysis by unveiling key differences between LV dysfunction caused by type 1 and type 2 DM.