Suppression of plasma free fatty acids reduces myocardial lipid content and systolic function in type 2 diabetes

P. Wolf[a], Y. Winhofer[a,*], M. Krssak[a,b], S. Smajis[a], J. Harreiter[a], L. Kosi-Trebotic[a], C. Fürnsinn[a], C.-H. Anderwald[a,c,d], S. Baumgartner-Parzer[a], S. Trattnig[b], A. Luger[a], M. Krebs[a]

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Highlights

- Circulating free fatty acids acutely impact myocardial lipids and heart function.
- Adipose tissue lipolysis is prerequisite to maintain systolic heart function.
- Acute changes in cardiac lipids and function are similar between diabetics and controls during free fatty acids suppression.
- Myocardial lipid stores might serve as a readily available source of energy.

Abstract

Background and Aim

Type 2 diabetes (T2DM) is closely associated with the development of heart failure, which might be related with impaired substrate metabolism and accumulation of myocardial lipids (MYCL). The aim of this study was to investigate the impact of an acute pharmacological inhibition of adipose tissue lipolysis leading to reduced availability of circulating FFA on MYCL and heart function in T2DM.

Methods and Results

8 patients with T2DM (Age: 56 ± 11; BMI: 28 ± 3.5 kg/m^2; HbA1c: 7.29 ± 0.88%) were investigated on two study days in random order. Following administration of Acipimox or Placebo MYCL and heart function were measured by ^{1}H-magnetic-resonance-spectroscopy and tomography at baseline, at 2 and at 6 h. Acipimox reduced circulating FFA by −69% ($p < 0.001$), MYCL by −39 ± 41% ($p < 0.001$) as well as systolic heart function (Ejection Fraction (EF): −13 ± 8%, $p = 0.025$; Cardiac Index: −16 ± 15%, $p = 0.063$ compared to baseline). Changes in plasma FFA concentrations strongly correlated with changes in MYCL ($r = 0.707$; $p = 0.002$) and EF ($r = 0.651$; $p = 0.006$). Diastolic heart function remained unchanged.

Conclusions

Our results indicate, that inhibition of adipose tissue lipolysis is associated with a rapid depletion of MYCL-stores and reduced systolic heart function in T2DM. These changes were comparable to those previously found in insulin sensitive controls. MYCL thus likely serve as a readily available energy source to cope with short-time changes in FFA availability.

Keywords:
Ectopic lipids, Metabolic inflexibility, Cardiac steatosis, Diabetic cardiomyopathy

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