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MYOCARDIAL DYSFUNCTION AND METABOLIC DERANGEMENT IN TYPE 2 DIABETES: RELATIONSHIP WITH PROCOLLAGEN BIOMARKERS OF MYOCARDIAL FIBROSIS

ACC Poster Contributions
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Session Title: Stress Echocardiography in special populations and CAD
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Background: Myocardial fibrosis is an intrinsic feature of non-ischemic cardiomyopathy in type 2 diabetes (T2DM). Types I and III collagen predominate, although hyperglycemia causes proportionally increased type III collagen synthesis. Procollagen biomarkers have been validated against biopsy studies as markers of myocardial fibrosis. We sought to examine their relationship with metabolic derangement and myocardial dysfunction in T2DM.

Methods: Anthropometric and metabolic data were measured in asymptomatic subjects with T2DM. Myocardial function was examined at rest and post exercise stress on echo with standard parameters and color TDI (systolic tissue velocity [Sm], strain and strain rate). The amino-terminal propeptides of procollagen type I (PINP) and type III (PIIINP) were measured by radio-immuno assay and the carboxy-terminal propeptide of procollagen type I (PICP) by enzyme immunoassay.

Results: In 117 subjects (67 men, 59±9 years), mean PINP was 50.2±34.1 µg/L, PIIINP was 3.3±1.4 µg/L and mean PICP was 271.7±87.9 ng/ml. PIIINP was best associated with features of the metabolic syndrome including: HbA1c (r=0.214, p=0.020), body mass index (r=0.209, p=0.024), waist circumference (r=0.276, p=0.003), hypertriglyceridemia (r=0.306, p=0.001) and fasting glucose (r=0.289, p=0.002). On multivariate analysis, hypertriglyceridaemia (β=0.297, p=0.001) and waist circumference (β=0.247, p=0.006) were independently related to PIIINP. PICP was associated with total cholesterol (r=0.184, p=0.048). PIIINP was poorly associated with myocardial function, whilst PINP and PICP were related to impaired myocardial function at peak stress. PICP correlated with impaired peak strain rate (r=-0.219, p=0.02) and PINP reflected reduced peak strain (r=-0.219, p=0.032). Both demonstrated a truncated change in Sm (PICP: r=-0.192, p=0.039; PINP: r=-0.223, p=0.029).

Conclusion: PIIINP is best associated with the metabolic derangement often noted in T2DM. However, the relationship between PICP & PINP and impaired myocardial function at peak stress suggests differing roles of type 1 and III collagen in the pathophysiology of myocardial fibrosis in diabetic cardiomyopathy.