Abstract: P131

Effect of human epicardial adipose tissue on calcium-overload-induced atrial fibrillation in human cardiac muscles

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Background: Atrial fibrillation (AF) is the most common type of irregular heart rhythm or arrhythmia. Excessive epicardial adipose tissue (EAT), located between the epicardium and visceral pericardium, is regarded as an emerging factor for developing AF. EAT might promote AF through secreting paracrine factors including adipokines and metabolites, which potentially can result in calcium overload leading to AF. However, the functional interaction between EAT and myocardium to enhance AF, especially in humans, is unknown.

Purpose: This study aimed to determine whether human EAT promotes calcium-overload-induced AF in human cardiac muscles.

Methods: After informed consent, human right atrial appendage muscles (trabeculae), and a piece of human EAT (weight = 49 ± 10 mg), were obtained from open chest surgery patients. In the trabeculae, the susceptibility for developing spontaneous contractions (automaticity), as measure for AF, was tested using a 1-minute rest period following a 1-minute 1 Hz (60 bpm) stimulation period at normal (1 mM), and at high (3 mM) levels of external calcium. Hereafter, the superfusate of the patients’ own EAT, from an adjacent bath, was recirculated over the muscle for 30 minutes, and the automaticity protocol repeated. The EAT-muscle superfusate was analysed with enzyme linked immunosorbent essay (ELISA) for the release of resistin, an EAT-derived factor associated with increased risk of developing AF.

Results: High calcium levels increased the proportion of human muscles that developed spontaneous contractions (1mM: 10\% vs. 3mM: 30\%, p=0.02, Chi-square test, n=10). Moreover, recirculation of the human EAT superfusate over the trabeculae increased the proportion of muscles that developed spontaneous contractions at low, but not at high calcium (1mM: 30\%, p=0.04 vs. no-EAT; 3mM: 40±\%, p=0.1 vs. no-EAT; Chi-square test, n=10). Resistin was detected in the human EAT superfusate recirculated over the muscles (23 ± 12 pg/ml per mg wt EAT, n=10).

Conclusion: Human EAT increased the susceptibility of the human cardiac muscle for spontaneous contractions, which might be due to release of resistin from the EAT, however this appears to be calcium independent. This is the first study to show a functional interaction between human EAT and human myocardium to modulate the susceptibility for AF, potentially through release of EAT-derived factors.