

## Relationship between epicardial fat and left atrium fibrosis in patients with atrial fibrillation

D. Nascimento Matos, A. Ferreira, P. Freitas, G. Rodrigues, J. Carmo, M.S. Carvalho, J. Abecasis, P. Carmo, C. Saraiva, D. Cavaco, F. Morgado, M. Mendes, P. Adragao

Hospital Santa Cruz, Cardiology, Carnaxide, Portugal

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**Background:** Epicardial adipose tissue (EAT) has recently been shown to be associated with the presence, severity, and recurrence of atrial fibrillation (AF). Although the pathophysiological mechanisms underlying this association remain to be established, several hypotheses have been put forward, including direct adipocyte infiltration, oxidative stress, and the secretion of adipokines causing inflammation and fibrosis of atrial tissue. We hypothesized that the volume of EAT and the amount of left atrium (LA) fibrosis assessed by non-invasive imaging would be significantly correlated in patients with AF, and that both would predict time to relapse after pulmonary vein isolation (PVI).

**Methods:** Sixty-eight patients with AF being studied for a first PVI procedure underwent both cardiac computerized tomography (CT) and cardiac magnetic resonance (CMR) within less than 48h. EAT was quantified on contrast-enhanced CT images. LA fibrosis was quantified on isotropic 1.5mm 3D delayed enhancement CMR for image intensity ratio values  $>1.20$ . Radiofrequency PVI was performed using an irrigated contact force-sensing ablation catheter, guided by electroanatomical mapping. After PVI, patients were followed for AF recurrence, defined as symptomatic or documented AF after a 3-month blanking period. Pearson's correlation coefficient was used for gauging the correlation between EATLM volume and LA fibrosis. The relationship between these two variables and time to AF recurrence was assessed by Cox regression.

**Results:** Most of the 68 patients (46 men, mean age  $61\pm 12$  years) had paroxysmal AF (71%,  $n=48$ ). The mean body mass index (BMI) was  $28.0\pm 4.0$  kg/m<sup>2</sup>. Patients had a median EATLM volume of  $2.4$  cm<sup>3</sup>/m<sup>2</sup> [interquartile range (IQR)  $1.6$ – $3.2$  cm<sup>3</sup>/m<sup>2</sup>], and a median estimated amount of LA fibrosis of  $8.9$  g (IQR  $5$ – $15$  g), corresponding to  $8\%$  (IQR  $5$ – $11\%$ ) of the total LA wall mass. The correlation between EATLM and LA fibrosis was statistically significant but weak (Pearson's  $R = 0.38$ ,  $P=0.001$ ) – Figure 1. During a median follow-up of 22 months (IQR  $12$ – $31$ ), 31 patients (46%) suffered AF recurrence. Four predictors of relapse were identified in univariate Cox regression: EATLM (HR  $2.19$ , 95% CI  $1.65$ – $2.91$ ,  $P<0.001$ ), LA fibrosis (HR  $1.05$ , 95% CI  $1.01$ – $1.09$ ,  $P=0.033$ ), non-paroxysmal AF (HR  $3.36$ , 95% CI  $1.64$ – $6.87$ ,  $P=0.001$ ), and LA volume (HR  $1.03$ , 95% CI  $1.01$ – $1.06$ ,  $P=0.006$ ). Multivariate analysis yielded two independent predictors of time to AF relapse: EATLM (HR  $2.05$ , 95% CI  $1.51$ – $2.79$ ,  $P<0.001$ ), and non-paroxysmal AF (HR  $2.36$ , 95% CI  $1.08$ – $5.16$ ,  $P=0.031$ ).

**Conclusion:** The weak correlation between EAT and LA suggests that LA fibrosis is not the main mechanism by which EAT and AF are linked. EAT was more strongly associated with AF recurrence than LA fibrosis, which supports the existence of other, more important mediators between EAT and this arrhythmia.

