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Left atrial epicardial adipose tissue radiodensity is associated with electrophysiological properties of atrial myocardium in patients with atrial fibrillation

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Abstract

Objective To evaluate whether the epicardial adipose tissue (EAT) phenotype is associated with the electrophysiological properties of adjacent atrial myocardium in patients with atrial fibrillation (AF).

Methods Between January and May 2017, 30 consecutive patients referred for a first AF catheter ablation were prospectively included. For each patient, a pre-procedural computed tomography scan was performed to assess total and left atrial (LA) EAT amount and radiodensity. A detailed point-by-point voltage mapping using an electroanatomic mapping system was realized to assess the presence of LA low-voltage zone (LVZ).

Results Ten patients (33.3%) presented at least one LVZ. Older age (65 ± 7 vs. 58 ± 10 years, $p = 0.05$) was the only clinical parameter associated with LVZ. Despite no greater LA-EAT thickness by CT scan (3.0 [2.6–3.5] mm vs. 2.8 [2.2–3.1] mm, $p = 0.354$), patients with LA-LVZ presented significantly lower LA-EAT radiodensity than patients with no LA-LVZ (-101.8 ± 12.5 HU vs. -90.4 ± 6.3 HU, $p = 0.004$). No difference between total-EAT volume (131 ± 61 cm^3 vs.107 ± 58 cm^3, $p = 0.361$) and total-EAT radiodensity (-106.8 ± 4.3 HU vs. -102.4 ± 6.9 HU, $p = 0.119$) was found.

Conclusion Low LA-EAT radiodensity is associated with the presence of LVZ in patients with medical history of AF.

Key Points
• *Cardiovascular risk factors are associated with low adipose tissue computed tomography attenuation.*
• *Epicardial adipose tissue (EAT) has emerged as an important factor in the pathogenesis of metabolic-related cardiac diseases such as atrial fibrillation.*
• *We showed that low left atrial EAT attenuation is associated with the presence of low-voltage zone, a surrogate for atrial fibrosis, within the adjacent myocardium.*

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Abbreviations
AF   Atrial fibrillation
EAT   Epicardial adipose tissue
LA   Left atrium
LVZ   Low-voltage zone
MS   Metabolic syndrome

Introduction

Atrial fibrillation (AF) is the most common sustained supra-ventricular arrhythmia [1]. A long-term follow-up cohort study involving more than 30,000 middle-aged individuals showed that hypertension, obesity, and diabetes are strongly associated with AF incidence [2].

Although the mechanism linking atrial fibrillation and the metabolic syndrome (MS) is not completely understood, obesity and other components of MS might increase cardiomyocyte exposure to cytokines and other inflammatory mediators that could in turn promote structural and functional atrial remodeling [3].

Adipose tissue (AT) is a metabolically active organ secreting various bioactive molecules directly involved in inflammation. It is well-known that AT amount, composition, and functionality are highly dependent on individual cardiovascular risk factors [4].

Epicardial adipose tissue (EAT) is a specific AT located between the visceral pericardium and the epicardium. EAT is in direct contact with the adjacent myocardium without any barrier able to limit paracrine crosstalk between the two tissues. By using a rat organo-culture model, Hatem et al showed that conditioned medium from human EAT, but not from subcutaneous adipose tissue, induces marked myocardial fibrosis, a common substrate in patients with AF [5].

We hypothesized that EAT amount and quality would be associated with the electrophysiological properties of adjacent atrial myocardium in patients with medical history of AF.

To get insight into this hypothesis, we explored atrial myocardial electrophysiological properties by electroanatomic mapping and EAT quality and quantity by computed tomography scanner (CT scan) in patients referred for AF catheter-ablation procedure.

Methods

Study population

Between January and May 2017, a prospective cohort study including all consecutive patients referred for AF catheter ablation in Lille University Hospital was performed. According to the 2016 ESC Guidelines for the Management of AF [5], patients were eligible to catheter ablation if they had new-onset or under-treated paroxysmal, persistent, or long-standing persistent AF. Exclusion criteria included prior cardiac surgery, prior AF catheter ablation, and severe valvular heart disease. A flow chart showing the study design is presented in the Supplementary Appendix (Annexe 2). The protocol was approved by the local ethics committee and patients gave informed consent.

Cardiac imaging

All patients underwent a pre-procedural computed tomography (CT) scan routinely performed to assess detailed left atrial (LA) and pulmonary vein (PV) anatomy. Images were acquired using a dual-source 128-section multidetector CT system (*Somatom Definition Flash*; *Siemens*) with an ECG-gated dual-source single-energy protocol over the entire heart. The acquisition parameters were automatic tube voltage adjustment (*Care kV*; *Siemens*) from 80 to 120 kVp; automatic tube current modulation (*CARE Dose4D*; *Siemens*) ranging between 150 and 400 mAs; collimation $= 64 \times 2 \times 0.6$ mm; rotation time $= 0.28$ s with 75-ms temporal resolution; and pitch $= 3$. The injection protocol included the administration of 60 ml of a non-ionic low-osmolar 35% iodinated contrast agent (Optiject, *Ioversol*; *Guerbet*) at flow rate of 4 ml/s. The mean dose-length-product (DLP) was 97.0 ± 63.5 mGy cm (range 38–354).

Post processing was performed with a dedicated software (Syngo; Siemens) using the "Volume Perfusion CT Body" module to obtain total-EAT volume and radiodensity. To analyze EAT features (global volume, mean density), all voxels with density between -250 and -50 HU within the pericardial sac were semi-automatically selected in axial slices from the bifurcation of the pulmonary trunk to the diaphragm. EAT volume (cm^3) and mean EAT density (HU) were automatically calculated by the software (Fig. 1).

LA-EAT thickness was measured using digital calipers in a short-axis view as the shortest distance between the mid left atrium (LA) wall and three anatomic landmarks as previously described [6]: the esophagus (LA-ESO), the main pulmonary artery (LA-PA), and the descending thoracic aorta (LA-TA) (Fig. 2).

As previously described, LA-EAT radiodensity was evaluated within five anatomic regions around the left atrium: superior left region (SL), inferior left region (IL), anterior right region (AR), inferior right region (IR) within the right atrioventricular groove, and Marshall tract region (MT) [7]. For each region, several ROIs (number > 4, size ≥ 5 mm^2, circular shape) were manually drawn and mean regional density was calculated. The LA-EAT radiodensity was determined as the average of all regional densities (Fig. 3a, b).

The CT data was evaluated by two senior radiologists specialized in cardiovascular imaging (JB and FP) with 3 and 13 years' experience in CT, respectively. Global EAT density and volume and LA-EAT thickness were evaluated by consensus. LA-EAT

Fig. 1 Contrast-enhanced computed tomography (CT) scan presenting the region of interest (ROI) and pixels attributed to epicardial adipose tissue (pink areas). Cardiac CT scans in 3 perpendicular planes (**a** sagittal, **b** coronal, **c** transverse) presenting the ROI outline (pink line) and pixels attributed to EAT within the ROI (pink areas). **d** Calculations generated by the software for the pixels within the range corresponding to adipose tissue (i.e., from −250 to −50 HU)
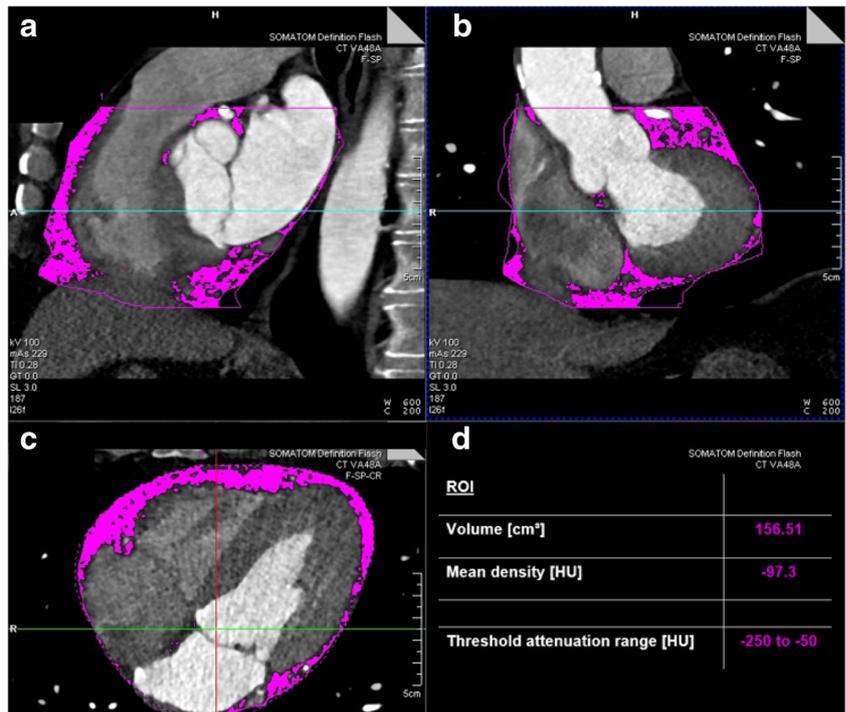
radiodensity was evaluated independently and the junior reader measurements used to calculate intra- and interobserver agreement. The radiologists interpreting the CT results were blinded to the clinical and to the electrophysiological results.

All patients underwent a standard transthoracic echocardiographic examination using a Vivid-E9 ultrasound system (GE Healthcare) a few days prior to catheter ablation. LV ejection fraction (EF) was obtained by modified biplane Simpson's method from four- and two-chamber views (CV). The LA volumes were calculated from the apical 4CV and 2CV of the LA using the biplane discs method. LA volumes index was defined as LA volumes divided by body surface area.

Ablation procedure

All procedures were performed by two experienced operators (more than 10 years AF ablation) under local anesthesia and conscious sedation using intravenous boluses of morphine. Two catheters were advanced from the right femoral vein to the LA through a dual transseptal puncture, a 20-pole circular mapping catheter and a 3.5-mm irrigated ablation catheter. A third, standard quadripolar catheter was used for reference and placed into the coronary sinus.

Electroanatomic mapping

At the beginning of the procedure, left atrium voltage mapping was performed using an electroanatomic mapping (EAM) system (CARTO3, Biosense Webster) and a mapping catheter with a 3.5-mm distal tip and a 2-mm inter-electrode spacing (NaviStar, Thermocool Smartouch, Biosense Webster Inc).

Adequate endocardial contact was confirmed by stable electrograms and increased contact force values of ≥ 10 g. Considering temporal EGM amplitude variability during AF,

Fig. 2 Magnified short-axis view of the periatrial fat pad between the esophagus and mid LA (left figure) and between the mid LA and thoracic aorta (right figure). Orientation cube: A indicates anterior; F, feet; and L, left. LA, left atrium; AO, aorta; ESO, esophagus
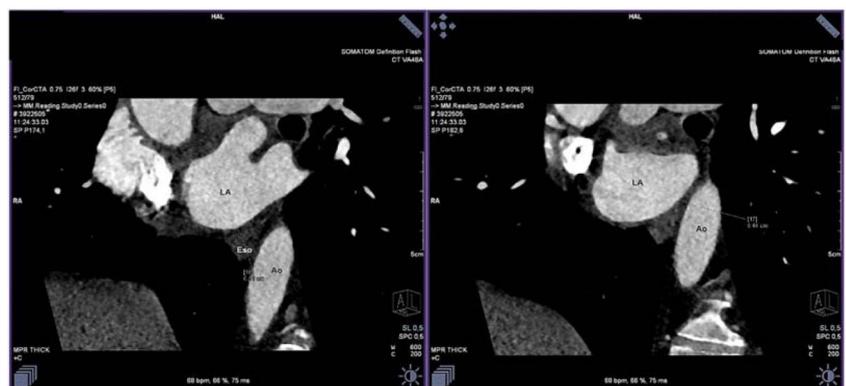
Fig. 3 a Representative measurement of LA-EAT density. After calculations generated by the software for the pixels within the range corresponding to adipose tissue (i.e., from − 250 to − 50 HU, pink area on the ROIs), five regions around the left atrium were analyzed: superior left region (SL), inferior left region (IL), anterior right region (AR), inferior right region (IR), and Marshall tract region (MT). b Region of interests manually drawn within the LA-EAT at the level of superior left (SL) to calculate mean regional EAT radiodensity. Several ROIs were manually draw in the pink areas (adipose tissue predefined by the software) in each region. The same axial level is shown: on the right, adipose tissue and on the left, manually drawn ROIs to calculate mean regional EAT density
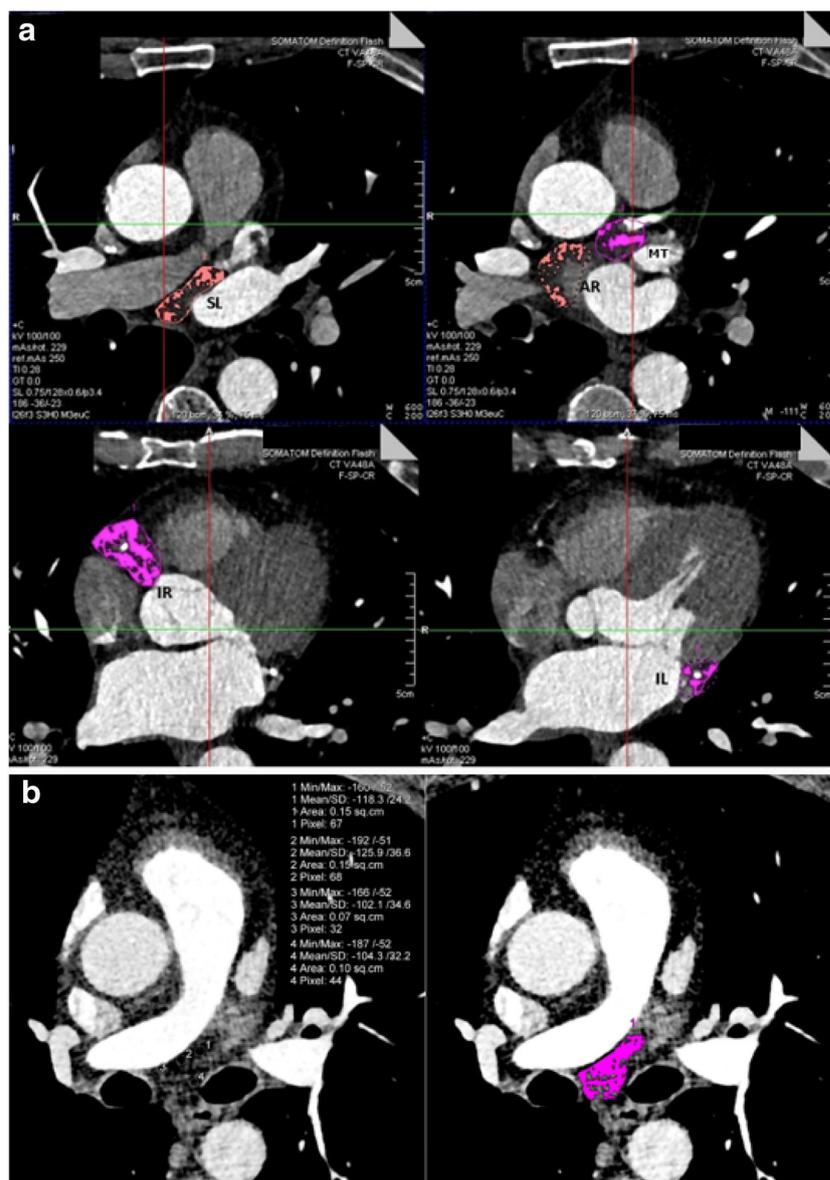
each point was manually selected as the maximum peak-to-peak bipolar voltage amplitude among all atrial EGMs occurring during three consecutive QRS complexes.

The left atrium was divided into nine regions, i.e., septum, superior, posterior, inferior, and lateral walls, and the 4 PVs antrum (Annexe 1). In each predefined region, at least 12 voltage-mapping points were collected. A low-voltage zone (LVZ) was defined by a region with bipolar voltage less than 0.5 mV in patients in SR and less than 0.2 mV in patients in AF [8, 9] at the time of point acquisition. Patients were splitted in two groups according to the presence or the absence of LVZ.

Statistical analysis

Continuous variables were tested for normal distribution using Shapiro–Wilk test. Data with normal distribution was

presented as means ± SD and data without normal distribution was presented as median [Q1–Q3]. Categorical variables were given as number and percentage.

Differences between continuous normally distributed data were tested for statistical significance using the two-tailed independent sample Student's t test. In the case of continuous data without a normal distribution, the Mann–Whitney Wilcoxon test was used. Differences of categorical data were tested for statistical significance using chi-square or Fisher's exact test where necessary.

To calculate the target sample size of our cohort, we hypothesized that the presence of LA-LVZ will be associated with a 10% difference in the value of LA-EAT radiodensity (HU). We calculated that 8 patients per group were needed to get a statistical power of 90% and a probability of a type I error of 0.10 [7]. As 26–42% of patients referred for a first AF

catheter ablation have LA-LVZ [10, 11], a population study of 30 patients was adequate to include at least 8 patients for LVZ group and to test our hypothesis.

Intra- and interobserver agreement for the LA-EAT radiodensity measurements was examined using intraclass correlation coefficients (ICCs), with their 95% CI, and an ICC greater than 0.8 was considered as good.

A p value less than 0.05 was considered significant for all statistical determinations. All analysis was performed using SPSS software version 22.0.

Results

Included patients

Patients' characteristics are summarized in Table 1.

A total of 30 patients were prospectively included, i.e., 18 males (60%) with symptomatic AF [14 (46.6%) paroxysmal AF and 16 (53.4%) with persistent AF, including 2 long-lasting persistent]. The mean CHA2DS2-VASc score was 1.7 ± 1.3. Left atrial volume index was high (42.7 ± 9.7 ml/m^2).

Most of the patients (77%) were overweight or obese with features of metabolic syndrome: 43% were hypertensive, 47% had dyslipidemia, and 13% had diabetes mellitus.

Clinical correlates of atrial low-voltage zones

A total of 3240 points were acquired on EAM of 30 patients (mean of 108 points-per-patients).

The mean bipolar voltage was 1.61 ± 1.32 mV in patients in SR and 0.79 ± 0.67 mV in patients in AF at the time of mapping.

Ten patients (33.3%) presented at least one LVZ (Table 1). These patients were significantly older ($p = 0.050$) than patients with no LVZ but were very similar for other variables. Of note, hypertension and AF pattern trended to be associated with LVZ presence.

In our population, diabetes mellitus and body mass index were not correlated with LVZ.

Clinical correlates of EAT volume and phenotype

EAT amount and density were modulated by clinical parameters. Total-EAT volume was higher in male ($\beta = 0.453$; $p = 0.026$). Body mass index (BMI) exhibits a positive but non-significant correlation with total-EAT volume ($\beta = 0.336$; $p = 0.078$). Conversely, diabetes mellitus was not correlated with EAT amount (Table 2).

Relation between cardiovascular disease risk factors and EAT density on cardiac CT scan revealed that total-EAT density was negatively correlated with age ($\beta = -0.438$; $p = 0.032$) and

CHA2DS2Vasc score ($\beta = -0.458$; $p = 0.025$) and positively correlated with male gender ($\beta = 0.703$; $p < 0.001$).

There was a good intra- (ICC = 0.893; 95% CI 0.787–0.948) and interobserver agreement (ICC = 0.887; 95% CI 0.776–0.945) regarding LA-EAT radiodensity measurements.

LA-EAT density was associated with male gender ($\beta = 0.423$; $p = 0.028$) and AF pattern ($\beta = 0.346$, $p = 0.028$). Patients with long-standing persistent AF had more negative LA-EAT radiodensity (-113 ± 12 HU) than patients with persistent (-94 ± 9 HU) or paroxysmal AF (-93 ± 10 HU) ($p = 0.028$).

Hba1c trended to be negatively correlated with both total ($\beta = -0.326$; $p = 0.160$) and left atrial ($\beta = -0.288$; $p = 0.183$) EAT radiodensity.

Correlation between EAT properties and myocardial phenotype

No difference between total-EAT volume (131 ± 61 cm^3 vs.107 ± 58 cm^3, $p = 0.361$) and total-EAT radiodensity (-106.8 ± 4.3 HU vs. -102.4 ± 6.9 HU, $p = 0.119$) was found regarding the presence or the absence of left atrial LVZ (Table 2).

However, patients with LA-LVZ had significantly lower LA-EAT radiodensity (-101.8 ± 12.5 HU vs. -90.4 ± 6.3 HU, $p = 0.004$), despite no greater LA-EAT thickness (3.0 [2.6–3.5] mm vs. 2.8 [2.2–3.1] mm, $p = 0.354$) (Table 3).

After multivariable adjustment, LA-EAT radiodensity remains independently associated with the presence of LVZ ($p = 0.005$) while age turned as non-significant ($p = 0.059$).

Discussion

Exploring EAT characteristics by CT scan and electrophysiological properties of atrial myocardium by EAM, we found a significant association between the presence of LVZ and EAT radiodensity regarding LA in patients referred for AF catheter ablation.

Determinants of LVZ

In our study, an older age (65 ± 7 vs. 58 ± 10 years) was the only clinical parameter that was associated with LVZ. This is consistent with Huo et al findings that showed that advanced age, but also female gender, larger surface area of LA, and persistent AF are associated with the presence of LVZ [12].

Conversely to experimental studies [13, 14], we did not find any association between other cardiovascular risk factors and electrical left atrial remodeling, possibly because of the small size of our population.

Table 1 Baseline characteristics

	Total population (n = 30)	No LVZ (n = 20)	LVZ (n = 10)	p value
Age (years)	60 ± 10	58 ± 10	65 ± 7	0.050*
Male gender	18 (60)	13 (65)	5 (50)	0.461
BMI (kg/m^2)	27.3 [25.3–30.0]	27.5 [25.0–30.4]	27.3 [26.1–27.9]	0.826
Diabetes mellitus	4 (13.3)	4 (20)	0 (0)	0.272
Dyslipidemia	14 (46.7)	10 (50)	4 (40)	0.709
Hypertension	13 (43.3)	7 (35)	7 (70)	0.122
Metabolic syndrome	10 (33.3)	8 (40)	2 (20)	0.251
Obstructive SAS	10 (33.3)	5 (25)	5 (50)	0.231
Coronary disease	2 (6.7)			
AF pattern				0.117
Paroxysmal	14 (46.6)	10 (50)	4 (40)	
Persistent	14 (46.6)	10 (50)	4 (40)	
Long-standing persistent	2 (6.7)	0 (0)	2 (20)	
CHA2DS2-Vasc score	1.7 ± 1.3	1.7 ± 1.5	2.0 ± 1.1	0.507
MAFSI score	12 [9–16]	10 [8–19]	13 [10–15]	0.685
Time from first diagnosis of AF (months)	57 [32–104]	69 [32–109]	55 [37–88]	0.581
Prior electrical cardioversion(s)	18 (60)	13 (65)	5 (50)	0.461
LVEF (%)	60 ± 7	58 ± 7	64 ± 6	0.052
LA volume index (ml/m^2)	42.7 ± 9.7	41.8 ± 10.5	44.3 ± 8.0	0.523
Prior antiarrhythmic drug(s)	28 (93.3)	19 (95)	9 (90)	1.000
Beta-blocker	13 (43.3)	8 (40)	5 (50)	0.705
ACE inhibitor—ARB	9 (30)	6 (30)	3 (30)	1.000
Diuretic	6 (20)	6 (30)	0 (0)	0.074
Flécainide	9 (30)	7 (35)	2 (20)	0.675
Sotalol	6 (20)	5 (25)	1 (10)	0.633
Amiodarone	7 (23)	3 (15)	4 (40)	0.181
Vitamin K antagonist	1 (3.3)	1 (5)	0 (0)	1.000
Apixaban	12 (40)	9 (45)	3 (30)	0.694
Dabigatran	5 (16.7)	2 (10)	3 (30)	0.300
Rivaroxaban	12 (40)	8 (40)	4 (40)	1.000
Fasting plasma glucose (g/L)	1.04 [0.96–1.15]	1.05 [0.96–1.24]	1.04 [0.97–1.10]	0.738
HbA1c (%)	5.6 [5.4–5.85]	5.65 [5.35–5.98]	5.62 [5.55–5.80]	0.909
LDL cholesterol (g/l)	1.15 ± 0.39	1.22 ± 0.45	1.01 ± 0.16	0.094
C-reactive protein (mg/l)	1.8 [0.9–2.0]	1.8 [1.0–2.0]	0.9 [0.7–3.5]	0.354

ACE angiotensin-converting enzyme, *ARB* angiotensin receptor blocker, *BMI* body mass index, *EAT* epicardial adipose tissue, *HbAc* hemoglobin A1c, *HU* Hounsfield units, *LDC* low-density lipoprotein, *LVEF* left ventricular ejection fraction, *MAFSI* Mayo AF-Specific Symptom Inventory, *SAS* sleep apnea syndrome

* $p \leq 0.05$

Prognostic value of EAT volume

Clinical studies showed a statistical relationship between epi-cardial fat volume and AF. The Framingham Heart Study reported that CT-derived EAT volume was associated with AF risk independently of age, sex, systolic blood pressure, and BMI [15]. Apart from the total-EAT volume, the EAT volume surrounding the LA demonstrated a strong relation-ship with an increased risk of AF, independently from left

atrial area and BMI [6, 16]. In addition to AF occurrence, EAT amount is associated with AF chronicity, symptom bur-den, and recurrence after radiofrequency ablation [17, 18].

As EAT surrounds the coronary arteries, it is presumed to play a key role in the development of coronary artery disease. Lee et al showed that EAT area in low-dose chest computed tomography is an independent factor for coronary atheroscle-rosis in an asymptomatic population considered for lung can-cer screening [19].

Table 2 Determinants of EAT amount and radiodensity

	Total-EAT volume		Total-EAT radiodensity		LA-EAT thickness		LA-EAT radiodensity	
	Beta	p value	Beta	p value	Beta	p value	Beta	p value
Age (years)	0.223	0.296	− 0.438	0.032*	0.207	0.300	− 0.209	0.296
Male gender	0.453	0.026*	0.703	< 0.001*	0.044	0.826	0.423	0.028*
BMI (kg/m²)	0.366	0.078	0.144	0.502	− 0.083	0.680	0.004	0.983
Diabetes mellitus	− 0.313	0.137	− 0.100	0.641	− 0.047	0.816	− 0.117	0.562
HbA1c (%)	0.226	0.338	− 0.326	0.160	0.133	0.546	− 0.288	0.183
Dyslipidemia	− 0.317	0.131	0.108	0.615	− 0.107	0.596	0.091	0.652
LVEF (%)	− 0.283	0.181	− 0.221	0.299	0.041	0.840	− 0.256	0.198
LA volume (cm³)	0.446	0.025*	− 0.156	0.477	− 0.175	0.382	− 0.273	0.178
AF pattern	0.221	0.299	0.062	0.775	− 0.056	0.780	0.346	0.028*
CHA2DS2-VAsc	− 0.230	0.280	− 0.458	0.025*	0.022	0.913	− 0.273	0.168

Beta coefficient in italics if the related *p* value is less than 0.05

AF atrial fibrillation, *BMI* body mass index, *HbA1c* hemoglobin A1c, *LA* left atrium, *LVEF* left ventricular ejection fraction

* $p \leq 0.05$

In addition to cardiovascular diseases, EAT volume seems to be associated with extra-cardiac conditions such as the severity of liver fat content and liver fibrosis in patients with nonalcoholic fatty liver disease [20].

Finally, EAT has been showed to provide incremental prognostic information over and above coronary artery calcium score and traditional risk factors for the prediction of major adverse cardiac events [21].

EAT biological activity and radiodensity

Experimental studies showed that obesity and other cardiovascular risk factors may modulate AT composition and functionality by inducing a shift to a pro-inflammatory state [22, 23].

In addition to adipose amount, CT imaging can provide information about AT quality. Previously described by Rosenquist et al to assess abdominal visceral adipose tissue quality in the Framingham Heart Study CT [24], Franssens et al confirmed that cardiovascular risk factors including age,

obesity, and metabolic syndrome are associated with low EAT CT attenuation, which is associated with adipocyte hypertrophy and lipid-dense adipose tissue and finally related to systemic inflammation [25].

In line, we found that EAT phenotype is modulated by clinical parameters, but our study was not designed to demonstrate the impact of metabolic disorders on EAT radiodensity.

Paracrine crosstalk between EAT and myocardium

Although EAT is believed to induce myocardium fibrosis through paracrine mechanisms [5], specific data relating the expression of inflammatory and profibrotic markers in EAT and its interaction with the myocardium are scarce because of the very limited access of these tissues which are virtually only accessible during open-heart surgery.

Interestingly, our proof-of-concept cohort study showed a significant association between LA-LVZ presence and LA-EAT radiodensity (− 101.8 ± 12.5 HU vs. − 90.4 ± 6.3 HU,

Table 3 Comparison between myocardial phenotype and EAT features

	No LVZ (n = 20)	LVZ (n = 10)	p value
Total-EAT volume (cm³)	107 ± 58	131 ± 61	0.361
Total-EAT radiodensity (HU)	− 102.4 ± 6.9	− 106.8 ± 4.3	0.119
LA-EAT thickness (mm)	2.8 [2.2–3.1]	3.0 [2.6–3.5]	0.354
LA-EAT radiodensity (HU)	− 90.4 ± 6.3	− 101.8 ± 12.5	0.004
Marshall tract region MT	− 87.5 ± 13.8	− 101.9 ± 16.8	0.024
Inferior right region IR	− 98.6 ± 13.7	− 111.7 ± 12.2	0.020
Anterior right region AR	− 78.7 ± 12.8	− 94.3 ± 10.6	0.003
Inferior left region IL	− 97.8 ± 10.2	− 108.7 ± 14.1	0.030
Superior left region SL	− 88.4 ± 6.6	− 98.1 ± 14.8	0.027

$p = 0.004$). No association between total-EAT phenotype and LA-LVZ was found.

Limitations

A limitation of this study is the small number of patients included ($n = 30$). However, this proof-of-concept study aimed to show that LA-EAT radiodensity was associated with LA-LVZ in patients with AF and sample size was calculated based on previous literature in the topic and a stringent type 2 error, i.e., 10%.

The recently published Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation stated that *dense fibrosis* can be defined as atrial sites with voltages amplitude ≤ 0.5 mV [3]. The methods to describe the fibrotic substrate with EAM in patients with AF rhythm during mapping are less consensual [3] even if the relationship of LVZ on electroanatomic mapping between SR and AF has already been investigated. Thereby, Yagishita et al showed a significant linear correlation of LA bipolar voltage between patients in SR and AF ($r = 0.707$, $p < 0.001$) with a lower voltage in AF than in RS for a same point, suggesting a lower cutoff value to define LVZ in patients with AF [9]. According to these data, we adjusted the voltage cutoff value for LVZ in AF patients at 0.2 mV.

The voltage maps using point-by-point mapping are time-consuming and we aimed to create homogeneous voltage maps with an acceptable point distance. It is possible that small low-voltage areas were not included in our voltage maps. However, the aim of the study was not to detect minimal low-voltage areas but to differentiate patients with relevant LVZ from patients with apparently healthy LA tissue. In order to ensure an adequate endocardial contact, peak-to-peak bipolar voltage points were acquired with a contact force ablation catheter and each point was individually validated by an experienced operator.

Conclusion

Low LA-EAT attenuation assessed by CT scan is associated with the presence of left atrial low-voltage zone in patients with medical history of AF.

Further studies are needed to explore the mechanism involved.

Compliance with ethical standards

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