

Right atrial size and function to predict left atrial voltage defined fibrosis in patients with long-standing persistent atrial fibrillation

Przewidywanie obecności zwłóknienia lewego przedsionka oparte na pomiarze amplitudy elektrogramów przedsionkowych na podstawie oceny wymiarów i funkcji prawego przedsionka u pacjentów z długo trwającym przetrwałym migotaniem przedsionków

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Abstract

Introduction. Right atrial (RA) size and function are not well described in long-standing persistent AF (LSPAF) patients, nor their value as a predictor for the left atrial (LA) voltage-defined fibrosis.

Methods. An evaluation was made as to whether echocardiography determined RA length, planimetric area, volume, emptying fraction, stroke volume, expansion index and calculated derivatives among the LSPSAF population predict LA low voltage areas (LVA) acquired with high-density and high-resolution bipolar voltage mapping using the CARTO®3 system.

Results. 142 patients aged 63 (58–67) years old, 117 males, were enrolled in this study. LVA were detected in 54% of the patients. Severe global LVA burden was present in 15% of the patients, whereas 30% of the patients presented a disseminated pattern of remodelling. It was shown that: (1) the majority of the study population (76%) presented enlarged RA, however, RA volumes were larger than LA volumes in the minority of cases; (2) RA enlargement had a positive correlation with the presence of mild-to-moderate tricuspid regurgitation, left ventricular hypertrophy, LA enlargement, LA area and volume; (3), none of the RA indices was associated with the prediction of absolute LVA or advanced LA fibrotic remodelling, although patients with severe LVA burden presented longer RA length and a larger area than compared to patients with less advanced remodelling pattern.

Conclusions. It was found that RA enlargement and decreased RA function, common in LSPSAF patients, do not translate to the presence of voltage-derived LA fibrotic remodelling.

Key words: atrial fibrillation, long-standing persistent atrial fibrillation, fibrosis, low voltage areas, right atrium

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Introduction

Low voltage areas (LVA) in the left atrium (LA) are well documented in electrophysiology studies and reported as an arrhythmogenic substrate operating in atrial fibrillation (AF) and suggested as fibrotic areas [1]. To date, there has been limited research evaluating the predictors of LVA among long-standing persistent AF (LSPAF) patients. Recently, it was established that LA size and function do not correlate with LVA burden in this population [2]. The relationship between the LVA documented in the LA and right atrial (RA) size and function remains unknown. Moreover, in contrast to the LA, RA structure and function are not as well described in patients with LSPAF. This study aimed to determine RA parameters among the LSPAF population and hypothesised that echocardiography-derived RA indices may be helpful to detect LA LVA acquired with high-density and high-resolution bipolar voltage mapping in addition to LA parameters.

Methods

Study population

Consecutive LSPAF patients referred to the centre for RF point-by-point catheter ablation were prospectively enrolled. The patients with a history of AF ablation procedure, any cardiac surgery, severe valvular disease, previous myocarditis or pericarditis, known or suspected pulmonary hypertension, atrial septal defect and those who were not able to maintain sinus rhythm during LA mapping were excluded. All patients underwent an electrophysiological study and ablation under the approval of a local institutional committee on human research after providing written informed consent.

Echocardiography examination

All patients had well-controlled heart rates and underwent transthoracic and transoesophageal echocardiography before the ablation using a Vivid E9 ultrasound system (GE Vingmed Ultrasound AS). The following variables were prospectively measured during AF as an average of five cardiac cycles and indexed to the body surface area: LA anteroposterior diameter, RA and LA maximum and minimum length (long axis) and planimetric area and volume. All measurements were performed by a single experienced physician using well-established criteria [3]. RA and LA contractile and reservoir functions were analysed by calculating the following indices and expressed as a percentage where appropriate: (1) emptying fraction (EF), by dividing the difference between the maximum and minimum volume by maximum volume; (2) stroke volume (SV), as the difference between the maximum and minimum volume;

(3) expansion index (EI), by dividing the difference between the maximum and minimum volume by minimum volume. The right-to-left atrial ratio of all determined parameters was then calculated and expressed as a percentage. Moreover, a biatrial volume index expressed as the sum of the right and left atrial indexed maximum and minimum volume and biatrial area index expressed as the sum of the right and left atrial indexed maximum and minimum planimetric area were calculated. Enlargement of the LA was defined with a maximum indexed LA volume $> 34 \text{ mL/m}^2$ whereas enlarged RA with a maximum indexed RA volume $> 25 \text{ mL/m}^2$ in men and $> 21 \text{ mL/m}^2$ in women. Left ventricular hypertrophy (LVH) was defined based on indexed ventricular mass calculated by system software $> 115 \text{ g/m}^2$ in men and $> 95 \text{ g/m}^2$ in women [3]. Mitral (MR) and tricuspid (TR) regurgitation were assessed according to guidelines [4].

Detection of LVA

The mapping protocol is described in detail elsewhere [2]. Briefly, following pulmonary vein isolation (PVI) and cardioversion, all patients underwent high density-high resolution LA bipolar voltage mapping (2959 [2212–3143] points per map) during coronary sinus pacing using the CARTO®3 system (Biosense-Webster, BW) with a Pentaray catheter (BW) acquired with a CONFIDENSE™ module (BW). To ensure detailed mapping, the distance filling threshold was set at 5 mm, the tissue proximity filter was always enabled and only mapping sites that were within 5 mm from the acquired shell contributed to a voltage map. Further discrete mapping using a SmartTouch catheter (BW), which always covered less than 10% of the total LA surface area (TSA), at sites presenting inadequate Pentaray-tissue contact was performed if necessary. Electrograms were only accepted if a contact force was $\geq 6 \text{ g}$. EGM amplitude $\geq 0.5 \text{ mV}$ was defined as normal and $< 0.5 \text{ mV}$ as diseased tissue. All points presenting low voltage were visually inspected and those incorrectly annotated were deleted from the map. An extension of all the areas showing low voltage potentials at least 5 mm away from the ablation lesion set was measured with custom CARTO®3 system software. The global LVA burden was calculated as a sum of the LVA and then expressed as a percentage of the TSA. The section of the PV inside the ablation encirclement, LAA and an area adjacent to the fossa ovalis were excluded from the TSA calculations. The extent of global LVA burden $> 20\%$ of the TSA was arbitrarily considered as severe based on an observation that all detected LVA can be easily ablated if it occupies less than 20% of the TSA [2]. The body of LA was segmented into 5 areas: septum, anterior, posterior, inferior and lateral wall. If LVA were identified within 3 out of 5 LA segments it was considered a disseminated pattern of voltage-defined remodelling (Figure 1).

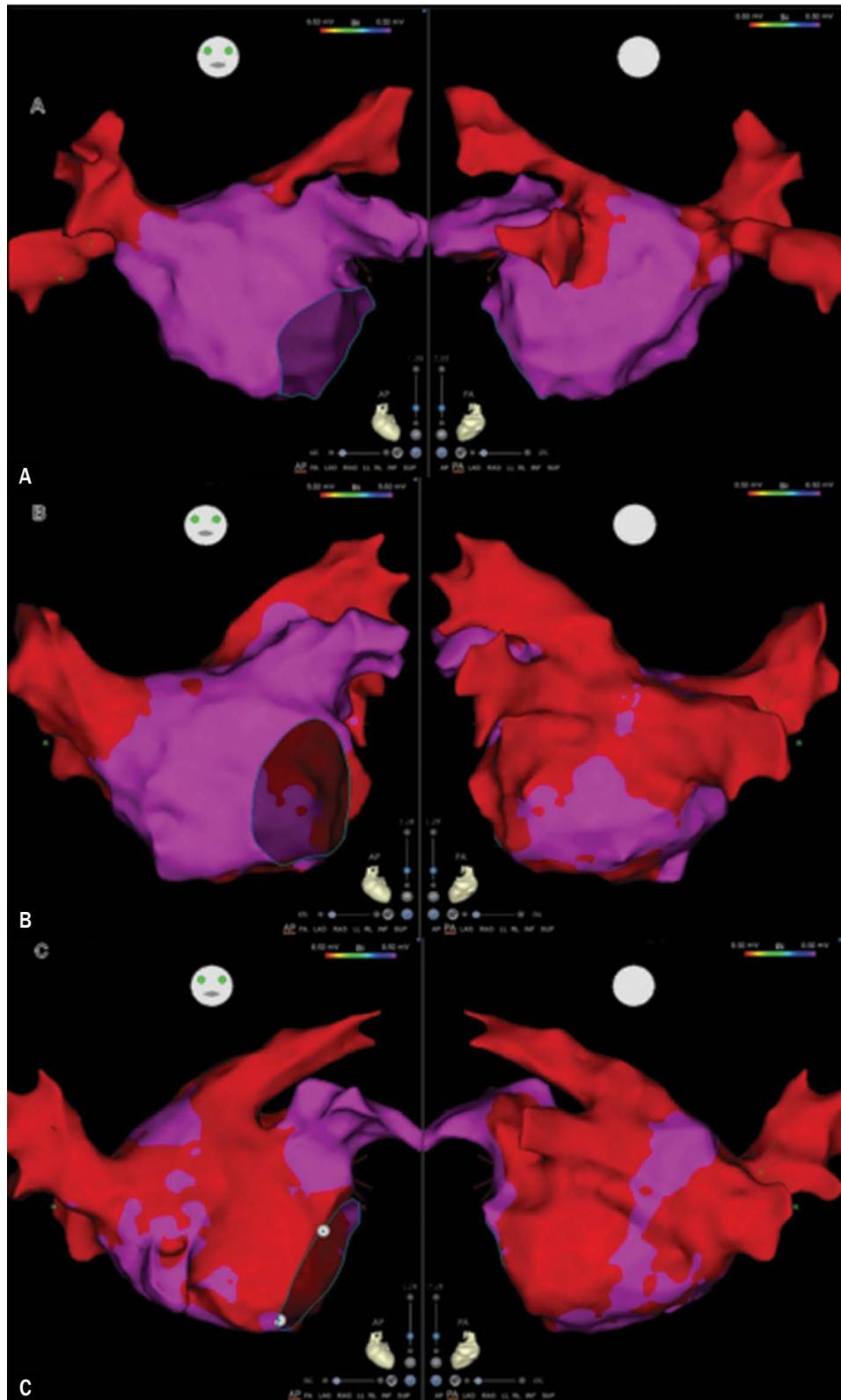


Figure 1. Left atrial (LA) distribution of low voltage areas (LVA) and examples of voltage maps of patients without LVA (A), with a disseminated pattern of voltage-defined remodelling and not severe LVA burden (B) and severe LVA burden (C). All maps show anteroposterior and posteroanterior views of CARTO@3 LA shells acquired following pulmonary veins isolation and restoration of sinus rhythm with cardioversion. Color-coding is defined as follows: <math>< 0.5\text{ mV}</math> = diseased atrial tissue (red), > 0.5 mV = healthy atrial myocardium (pink). In the patient presented at panel B LVA were limited to posterior, inferior and lateral LA segments (3 out of 5) whereas in the patient presented at panel C LVA involved all LA segments

Statistical analysis

Continuous data are presented as median and IQR, and the categorical variables as values and percentages. Comparisons between groups were performed with the Mann-Whitney U-test, or the χ^2 test, as appropriate. Multivariate stepwise forward logistic regression analyses were performed to isolate factors associated with the existence of LVA. Only variables with significant p-values on univariate analysis were included in the multivariate model. The correlation between variables was assessed using a Spearman rank test. Statistical significance was accepted at p-value < 0.05. The analysis was performed using Statistica software version 13.3 (StatSoft).

Results

One hundred and forty two patients aged 63 (58–67) years old, 117 males (82%), were enrolled in this study. The median continuous AF duration was 24 (12–36) months, ranging from 12–204 months and the median LA anteroposterior diameter was 47 (45–50) mm, ranging from 31–63 mm. Further detailed data on patient characteristics and echocardiographic findings are presented in Table 1.

LVA were detected in 54% (77/142) of the patients. Severe global LVA burden was present in 15% (21/142; 27% of the patients with LVA), whereas 30% of the patients (42/142; 55% of the patients with LVA) presented a disseminated pattern of remodelling. Twenty five percent of the patients (36/142) had documented LVA on the septum, 31% (44/142) on the anterior wall, 42% (59/142) on the posterior wall, 24% (34/142) on the inferior wall, and 10% (20/142) on the lateral wall.

No differences were found among the RA dataset in patients presenting LVA and disseminated patterns of LVA remodelling. However, patients with severe LVA burden showed significantly longer RA maximum and minimum length, larger maximum and minimum RA indexed area and biatrial maximum and minimum area (Table 2).

Using a univariate logistic regression technique it was found that none of the RA indices was associated with the prediction of the LVA, severe LVA or a disseminated LVA pattern.

Further analysis revealed that RA maximum volume had a positive correlation with the presence of mild-to-moderate TR ($r = 0.24$; $p = 0.0009$) and Congestive heart failure or left ventricular dysfunction, Hypertension, Age ≥ 75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74, Sex category (female) ($\text{CHA}_2\text{DS}_2\text{-VASc}$) score ≥ 4 ($r = 0.22$; $p = 0.01$). The presence of RA enlargement positively correlated with TR ($r = 0.23$; $p = 0.02$) and LVH ($r = 0.2$; $p = 0.02$). Moreover, both RA maximum volume and the presence of RA enlargement positively correlated with the presence of LA enlargement ($r = 0.2$; $p = 0.04$), maximum LA volume ($r = 0.44$; $p < 0.0001$) and area ($r = 0.43$;

Table 1. Baseline characteristics and echocardiographic parameters among the overall population

BMI, kg/m ²	30 (27.5–32)
Hypertension, n (%)	115 (81)
Mild to moderate mitral regurgitation, n (%)	120 (85)
Mild to moderate tricuspid regurgitation, n (%)	89 (63)
Left ventricular hypertrophy, n (%)	67 (47)
Chronic coronary syndrome, n (%)	31 (22)
Heart failure, n (%)	35 (25)
eGFR < 60 mL/min/1.73 m ² , n (%)	15 (11)
Diabetes, n (%)	30 (21)
Hyperthyroidism, n (%)	15 (11)
$\text{CHA}_2\text{DS}_2\text{-VASc} \leq 1$, n (%)	49 (35)
$\text{CHA}_2\text{DS}_2\text{-VASc} \geq 2$, n (%)	83 (58)
$\text{CHA}_2\text{DS}_2\text{-VASc} \geq 3$, n (%)	48 (34)
$\text{CHA}_2\text{DS}_2\text{-VASc} \geq 4$, n (%)	18 (13)
Left ventricular ejection fraction (%)	60 (55–65)
Enlarged LA, n (%)	120 (86)
Enlarged RA, n (%)	106 (76)
RA maximum length (mm/m ²)	31 (28–32)
RA maximum area (cm ² /m ²)	11 (10–13)
RA maximum volume (mL/m ²)	39 (30–44)
RA emptying fraction (%)	13 (7–20)
RA stroke volume (mL)	10 (5–18)
RA expansion index (%)	15 (8–25)
Biatrial maximum volume (mL/m ²)	86 (70–103)
Biatrial maximum area (cm ² /m ²)	25 (22–28)
% RA/LA maximum length	74 (61–93)
% RA/LA maximum area	95 (88–98)
% RA/LA maximum volume	85(76–95)
% RA/LA emptying fraction	64 (27–114)
% RA/LA stroke volume	61 (24–111)
% RA/LA expansion index	55 (21–117)
% RA/LA maximum volume > 100%, n	24 (17)
% RA/LA maximum volume < 50%, n	16 (11)
% RA/LA maximum area > 100%, n	17 (12)

BMI – body mass index; $\text{CHA}_2\text{DS}_2\text{-VASc}$ – Congestive heart failure or left ventricular dysfunction, Hypertension, Age ≥ 75 (doubled), Diabetes, Stroke (doubled), Vascular disease, Age 65–74, Sex category (female); eGFR – estimated glomerular filtration rate; LA – left atrial; RA, right atrial; % RA/LA – right to left atrial ratio expressed in percentages

$p < 0.0001$). Right-to-left maximum volume ratio > 100% had a positive correlation with TR ($r = 0.23$; $p = 0.016$), LVH ($r = 0.2$; $p = 0.02$), and an inverse correlation with female sex ($r = -0.2$; $p = 0.035$). Right-to-left maximum area ratio > 100% had an inverse correlation with female sex ($r = -0.2$; $p = 0.03$). Biatrial maximum volume had a positive correlation with age ($r = 0.23$; $p = 0.026$) and LVH ($r = 0.37$; $p < 0.0001$), whereas biatrial maximum area had

Table 2. Echocardiographic RA parameters and its derivatives among different LA LVA patterns

	LVA (+) n = 77	LVA (-) n = 65	p-value	Severe LVA (+) n = 21	Severe LVA (-) n = 121	p-value	Disseminated LVA pattern (+) n = 42	Disseminated LVA pattern (-) n = 100	p-value
Enlarged RA	62 (81%)	44 (68%)	0.5	19 (90)	87 (72)	0.5	35 (83)	71 (71)	0.4
RA max. length	30 (28–32)	31 (27–33)	0.78	31 (30–35)	30 (27–32)	0.04	31 (28–33)	30 (27–32)	0.7
RA min. length	29 (26–31)	28 (25–31)	0.22	29 (29–32)	28 (25–31)	0.03	29 (27–31)	28 (25–31)	0.3
RA max. area	12 (10–13)	11 (10–12)	0.3	12 (11–14)	11 (10–12)	0.04	12 (10–14)	11 (10–12)	0.3
RA min. area	11 (9–12)	10 (9–11)	0.16	12 (10–12)	10 (9–12)	0.02	11 (9–12)	10 (9–12)	0.2
RA max. volume	40 (30–48)	37 (31–41)	0.4	42 (32–52)	37 (30–43)	0.09	40 (29–50)	37 (31–41)	0.4
RA min. volume	34 (23–41)	30 (25–35)	0.23	35 (29–42)	30 (23–39)	0.08	34 (24–43)	30 (24–39)	0.3
RA emptying fraction	13 (7–18)	14 (8–25)	0.71	15 (7–20)	13 (8–20)	0.9	13 (7–20)	13 (8–21)	0.9
RA stroke volume	10 (6–17)	10 (4–20)	0.99	11 (4–22)	10 (5–18)	0.6	10 (5–20)	9 (5–8)	0.7
RA expansion index	15 (8–22)	16 (8–32)	0.71	17 (8–25)	15 (8–25)	0.8	14 (7–25)	15 (8–26)	0.9
Biatrial max. volume	94 (71–104)	82 (70–96)	0.21	100 (80–107)	84 (70–102)	0.08	94 (71–106)	84 (69–102)	0.3
Biatrial min. volume	75 (61–90)	69 (55–81)	0.25	85 (68–96)	70 (55–86)	0.09	78 (61–90)	70 (55–88)	0.3
Biatrial max. area	26 (22–28)	24 (22–27)	0.22	28 (24–29)	24 (21–28)	0.03	26 (22–28)	25 (21–28)	0.3
Biatrial min. area	24 (19–26)	22 (19–25)	0.16	26 (22–27)	22 (19–26)	0.04	24 (20–28)	22 (19–26)	0.3
% RA/LA max. length	73 (62–95)	77 (61–94)	0.76	69 (60–104)	74 (61–94)	0.9	73 (66–91)	74 (61–96)	0.8
% RA/LA min. length	82 (74–95)	86 (74–95)	0.83	82 (73–96)	83 (74–95)	0.9	82 (75–94)	84 (73–95)	0.9
% RA/LA max. area	94 (87–98)	95 (91–99)	0.41	94 (88–97)	95 (88–99)	0.8	94 (87–97)	95 (89–98)	0.5
% RA/LA min. area	93 (87–100)	93 (88–98)	0.64	93 (87–97)	93 (88–99)	0.7	93 (87–100)	93 (87–98)	0.8
% RA/LA max. volume	84 (74–100)	86 (76–95)	0.93	83 (72–98)	86 (76–95)	0.9	85 (74–94)	86 (76–95)	0.8
% RA/LA min. volume	76 (60–94)	75 (66–91)	0.9	72 (58–91)	76 (64–93)	0.5	78 (59–93)	75 (65–95)	0.7
% RA/LA emptying fraction	63 (28–114)	65 (25–114)	0.92	78 (24–113)	61 (28–114)	0.7	77 (27–126)	61 (25–112)	0.5
% RA/LA stroke volume	60 (23–100)	75 (25–115)	0.66	56 (17–100)	61 (25–115)	0.5	56 (22–131)	61 (24–100)	0.8
% RA/LA expansion index	55 (23–114)	55 (18–118)	0.93	69 (18–114)	54 (23–118)	0.8	69 (23–135)	54 (21–113)	0.5
% RA/LA max. volume > 100%	12 (16)	12 (18)	0.5	5 (24)	19 (16)	0.3	6 (14)	18 (18)	0.4
% RA/LA max. volume < 50%	9 (12)	7 (11)	0.6	3 (14)	13 (11)	0.4	5 (12)	11 (11)	0.5
% RA/LA max. area > 100%	9 (12)	8 (12)	0.9	3 (14)	14 (12)	0.5	5 (12)	12 (12)	0.5

LA – left atrial; LVA – low voltage areas; RA – right atrial; % RA/LA – right to left atrial ratio expressed in percentages

a correlation with age ($r = 0.28$; $p = 0.002$), LVH ($r = 0.37$; $p < 0.0001$), CHA₂DS₂-VASc score ≥ 4 ($r = 0.24$; $p = 0.01$) and inversely correlated with body mass index ($r = -0.34$;

$p < 0.0001$). No correlations between RA features and AF duration along with concomitant diseases other than those included in the CHA₂DS₂-VASc score were found.

Discussion

LA remodelling is a common response to AF, manifesting as increased size and reduced function, deteriorating with increasing AF burden [5]. RA structure and function were not thoroughly investigated in patients with LSPSAF as compared to LA. Moreover, data on the association between RA parameters and LVA burden seem to be lacking. Therefore, this study aimed to describe the echocardiography-determined RA dataset among LSPSAF patients and identify if any of these could predict the existence of voltage-derived LA fibrotic remodelling.

It was shown that: 1) the majority of the study population presented enlarged RA, however, RA volumes were larger than LA volumes in the minority of cases; 2) RA enlargement had a positive correlation with the presence of mild-to-moderate TR, LVH, LA enlargement, LA area and volume; 3) none of the RA indices was associated with the prediction of the absolute LVA or advanced LA fibrotic remodelling, although patients with severe LVA burden presented longer RA length and a larger area comparing to patients with a less advanced remodelling pattern.

Many studies reported that voltage-defined LA remodelling increases with LA size [6–8], however, not among LSPSAF patients [2]. It was suggested that the presence of functional LA enlargement due to AF persistence exclusively, seems not to translate into atrial fibrosis. Three patterns of increased LA size in the LSPSAF population were proposed: 1) functional and at least partially reversible as a consequence of AF itself; 2) secondary to other causes e.g. valvular regurgitation (primary or functional due to annular dilatation caused by AF); 3) a combination of both [2].

In the present study cohort, 76% of patients presented with enlarged RA. In most cases (83%) RA indexed volumes were lower than LA volumes, in 11% it represented half of the LA volume. As RA maximum volume and the presence of RA enlargement correlated with the presence of LA enlargement, maximum LA volume and its area may suggest that increased RA size was the consequence of increased LA size in the context of AF persistence. It was previously reported that both LA and RA enlargement can develop as a consequence of AF in patients who had no evidence of significant structural or functional cardiac abnormalities other than AF [9]. This could explain why there was no association between RA size expressed in various parameters and LVA in the authors' dataset. However, the presence of mild-to-moderate TR and LVH that correlated with RA enlargement and CHA₂DS₂-VASc score ≥ 4 that correlated with RA maximum volume could suggest that increased RA size was, at least in some cases, secondary to other scenarios than AF persistence. It is worth noting that cases where RA-indexed volumes were higher than LA volumes also correlated with TR and LVH. RA enlargement is a common finding in patients with pulmonary arterial hypertension and

atrial septal defect, though unlikely in this study group, and was found to be associated with the increased prevalence of AF [10]. Hypertensive LVH, very common in this cohort, is the known causative factor of diastolic dysfunction and has been shown to affect right-sided cardiac morphology and haemodynamics [11]. Therefore, it seems that increased RA size among LSPSAF patients is multifactorial.

The role of RA systolic (EF, SV) and diastolic (EI) function in the pathophysiology of AF persistence is yet to be analysed systematically. Moreover, due to the absence of established cut-off values [3], it is hard to assess the RA function pattern. Accepting magnetic resonance-derived normative data [12] all patients presented severely decreased RA function. Larger RA volume index and lower RA EF are a predictor of the recurrence of AF after direct current cardioversion and were superior to LA parameters in one study [13]. In the present study, RA functional parameters were not associated with LVA. A clear interpretation of these findings remains unclear.

Conclusions

It was found that RA enlargement and decreased RA function, common in LSPSAF patients, do not translate to the presence of voltage-derived LA fibrotic remodelling.

Study limitations

1) Whilst LSPSAF is considered more like a LA disease, to what extent the RA contributes to the role in the development and maintenance of AF remains unclear. Whether RA voltage-defined fibrosis, not assessed in this study, could improve risk stratification in LSPSAF patients should be analysed in future research investigations. 2) The accuracy of LA voltage mapping might have been influenced by several factors, such as mapping following PVI or using voltage cut-off values < 0.5 mV. The authors cannot further exclude the fact that the overall LVA burden might have been altered due to the exclusion of patients not able to maintain sinus rhythm, presenting LAA thrombus or if another method of LVA detection had been applied. 3) Women were underrepresented in the study.

Conflict of interest

RMK has received fellowship support from BW. RMK, MW and JK have received educational support, travel grants, and compensation for proctoring services from BW. AW declares no conflict of interest.

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Streszczenie

Wstęp. W populacji pacjentów z długo trwającym przetrwałym migotaniem przedsionków (LSPAF) wymiary i funkcja prawego przedsionka (RA) oraz ich znaczenie w przewidywaniu obecności włóknienia lewego przedsionka (LA) opartego na pomiarze amplitudy elektrogramów przedsionkowych nie zostały dokładnie zbadane.

Materiał i metody. W populacji pacjentów z LSPAF oceniono, czy uzyskane podczas badania echokardiograficznego długość, pole powierzchni, objętość, frakcja opróżniania, objętość wyrzutowa, wskaźnik rozszerzania RA oraz ich pochodne przewidują obecność obszarów niskoamplitudowych LA (LVA) uzyskanych podczas mapingu napięciowego wysokiej rozdzielczości i gęstości z użyciem systemu CARTO®3.

Wyniki. Do badania włączono 142 chorych w wieku 63 (58–67) lat. LVA stwierdzono u 54% pacjentów. Znacznie zaawansowany remodeling LA manifestujący się dużymi obszarami LVA stwierdzono u 15% chorych, natomiast rozlane obszary LVA u 30%. Zaobserwowano, że: 1) Większość pacjentów (76%) prezentowała powiększenie RA, jakkolwiek w niewielu przypadkach objętość RA była większa niż objętość LA; 2) Powiększenie RA dodatkowo korelowało z obecnością małej do umiarkowanej niedomykalności trójdzielnej, przerostem lewej komory, powiększeniem LA, powierzchnią i objętością LA; 3) Żaden z parametrów RA nie przewidywał obecności LVA i zaawansowanego stopnia włóknienia LA, chociaż u pacjentów z zaawansowanym stopniem remodelingu stwierdzono większą długość i powierzchnię RA w porównaniu z pacjentami z mniej zaawansowanym stopniem remodelingu LA.

Wnioski. Powiększenie i zaburzona funkcja RA jest częsta wśród pacjentów z LSPAF, ale nie pozwala przewidzieć obecności włóknienia LA.

Słowa kluczowe: migotanie przedsionków, długo trwające przetrwałe migotanie przedsionków, włóknienie, obszary niskoamplitudowe, prawy przedsionek

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