Acute pericardial effusion following atrial fibrillation ablation: Characteristics and relationship with arrhythmia recurrences

Épanchement péricardique aiguë postablation de fibrillation atriale : caractéristiques et relation avec les récurrences d’arythmie

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KEYWORDS
Pericardial effusion; Atrial fibrillation ablation; Early arrhythmia recurrences

Summary
Background. — Pericardial effusion (PE) can occur during or after atrial fibrillation (AF) ablation, and may induce atrial arrhythmia.
Aim. — To characterize the impact of PE on arrhythmia recurrences following AF ablation.
Methods. — Patients referred for a first radiofrequency AF ablation were studied prospectively. Transthoracic echocardiography was performed before and 24 h after the procedure. If PE was present, transthoracic echocardiography was repeated at 1 month to evaluate PE evolution. Early arrhythmia recurrences (EARs) were defined as any arrhythmia documented within 1 month of the procedure.
Results. — PE was diagnosed in 18/81 patients (22%); and was present in significantly more patients with persistent versus paroxysmal AF (14/40 [35%] vs 4/41 [10%]; \( P = 0.008 \)). PEs were mild (mean 6 ± 3 mm), mainly asymptomatic (89%), and none required pericardiocentesis. Early and late arrhythmia recurrences were present in 25/81 (31%) and 29/81 (36%), respectively. The incidence of PE was significantly higher among patients with EARs versus those without (12/25 [48%] vs 6/56 [11%]; \( P = 0.0004 \)). By multivariable analysis, PE and duration in AF were the two significant factors for the risk of early arrhythmia recurrences.

Abbreviations: AF, atrial fibrillation; EAR, early arrhythmia recurrence; ECG, electrocardiogram; IQR, interquartile range; LA, left atrium; LVEF, left ventricular ejection fraction; PE, pericardial effusion; TTE, transthoracic echocardiography.

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Background

Catheter ablation of AF is an established therapy for patients with both paroxysmal and persistent AF [1,2]. However, numerous complications have been associated with this procedure, including PE [3]. It has recently been reported that PE is common (10—14%) following AF ablation, but is mainly mild and asymptomatic [4,5].

EARs are common during the first month following AF ablation [6,7]. However, the significance of these EARs in predicting the long-term success of the procedure is controversial [8,9]. Consistent with this, the consensus document on catheter and surgical ablation of AF has suggested a uniform 'blanking period' of 3 months [10]. The pathophysiology of these early arrhythmias is also unclear. Transient autonomic neural modifications or acute tissue inflammation have been suggested as possible mechanisms [11,12].

We have studied the incidence and characteristics of PE following paroxysmal and persistent AF ablation. As PE is known to be a common cause of atrial arrhythmias [13], we also evaluated the relationship between early and late arrhythmia recurrences and the occurrence of acute PE following AF ablation.

Methods

Study population

We prospectively studied patients referred to our centre for a first radiofrequency AF ablation between September 2008 and January 2010. Patients undergoing paroxysmal AF ablation with cryotherapy were excluded in order to homogenize our population, as radiofrequency and cryotherapy create different tissue lesions that can influence arrhythmia recurrences after ablation, and also because persistent AF ablation could not be performed with cryotherapy. Patients with PE before AF ablation were also excluded. The study was approved by an institutional review committee, and all subjects gave written informed consent.

Echocardiography data

Comprehensive echocardiography examination including transoesophageal echocardiography before AF ablation to exclude left appendage thrombus and standard TTE before and 24 h after AF ablation procedure were performed for each patient, with a particular focus on the presence of
a new PE. Left ventricular function was assessed by the Simpson biplane method and LA diameter was measured on the parasternal long-axis view. PE was assessed from standard TTE using parasternal long-axis, short-axis and sub-costal views, and was quantified during diastolic timing using M mode on the posterior wall. To differentiate PE from epicardial fat, echocardiography data before and after AF were compared. The diagnosis of epicardial fat tissue was retained if the echo-free space was located anteriorly, tended to move in concert with the heart, and remained stable compared to echocardiography data before AF ablation.

Electrophysiological study

All patients received effective anticoagulation therapy (target international normalized ratio 2–3) for at least 1 month before AF ablation. All antiarrhythmic drugs, with the exception of amiodarone, were discontinued five half-lives before the procedure. Oral anticoagulation was stopped 2 days before the procedure, and low-molecular-weight heparin was started. The electrophysiological study was performed under conscious sedation using remifentanil.

The following catheters were introduced via the right femoral vein for electrophysiological study: (i) a steerable quadripolar or decapolar catheter (Xtrem; Ela Medical, Le Plessis-Robinson, France) was positioned within the coronary sinus, positioned at 4–5 o’clock along the mitral annulus in a left anterior oblique projection; (ii) a circumferential mapping catheter (Lasso; Biosense Webster, Diamond Bar, CA, USA) was introduced following transseptal access and stabilized using a long sheath (SL0 sheath; St Jude Medical, Sylmar, CA, USA) continuously perfused with heparinized saline solution; and (iii) a 3.5 mm externally irrigated tip ablation catheter (Celsius Thermocool; Biosense Webster) used for mapping and ablation.

Transseptal puncture was performed with the guidance of intracardiac echocardiography used transoesophageally [14]. Following transseptal access, a single bolus of 100 IU/kg body weight of heparin was administrated and repeated if activated clotting time was < 300 ms.

Monitoring with a surface electrocardiogram (ECG) and bipolar endocardial electrogram was carried out continuously and recorded on a computer-based digital amplifier/recorder system for off-line analysis (Bard Electrophysiology, Lowell, MA, USA). Intracardiac electrograms were filtered from 30–500 Hz and measured at a sweep speed of 100 mm/s.

Ablation of paroxysmal and persistent atrial fibrillation

Radiofrequency energy was delivered at up to 35 W using an irrigation rate of ≥20 mL/min (0.9% saline via Cool Flow; Biosense-Webster) to achieve the desired power delivery. Temperature was limited to 45 °C.

For paroxysmal AF ablation, the procedure was considered successful when all the pulmonary veins were isolated electrically. If a patient experienced AF during the procedure, AF ablation was continued until termination of AF was achieved as described for persistent AF ablation.

Ablation of persistent or long-lasting persistent AF was performed sequentially as described previously [15], and involved pulmonary vein isolation, electrogram-based ablation, and linear ablation, with termination of AF via catheter ablation being the endpoint (stepwise approach). If the patient converted into atrial tachycardia, this arrhythmia was mapped and ablated until sinus rhythm was restored. Finally, if patient was still in AF after 5 h of procedure, an external cardioversion was performed.

Follow-up

Patients were followed at 1 month and then every 3 months with systematic 12-lead ECG and 24-hour Holter monitoring in our centre for 1 year. They also underwent routine follow-up by their referring cardiologist. From 1 year after the last procedure, they were followed every 6 months by their referring cardiologist.

All patients had antiarrhythmic therapies, including flecainide and beta-blockers for 1 month (for paroxysmal AF) and amiodarone for 3 months (for persistent or long-lasting persistent AF). If arrhythmia recurred after stopping these drugs, antiarrhythmic therapies were restarted. Further ablation was performed if arrhythmias were still present. Oral anticoagulation treatment was restarted 1 day following the procedure, and was continued for at least 3 months.

Patients with PE following AF ablation systematically had another TTE to study PE evolution 1 month after the index procedure. Patients with significant pericarditis pain associated with PE had non-steroidal anti-inflammatory drugs for 1 month.

Definition of early and late arrhythmias

Recurrences were defined as documented atrial arrhythmias after the index AF ablation that lasted for ≥3 minutes [16]. The arrhythmia recurrences were characterized as follows: AF defined as chaotic and uncoordinated activity of the atrium [10] and atrial tachycardia defined as a monomorphic atrial activity on 12-lead surface ECG. EARs were defined as any arrhythmia documented within 1 month of the procedure.

Statistical analysis

Continuous variables are expressed as mean ± SD, or median ± interquartile range (IQR) when variables appeared to be non-normally distributed. Statistical significance was assessed using the unpaired Student’s t test or Mann-Whitney test, if necessary. Categorical variables, expressed as numbers or percentages, were analysed with the Chi² test or Fisher’s exact test. Univariable factors presenting P < 0.1 were analysed using a logistic regression method for multivariable analysis (performed with StatView Software). All tests were two-tailed and P < 0.05 was considered statistically significant.

Results

Study population

A total of 98 consecutive patients were referred for a first AF ablation from September 2008 to January 2010. Among
Table 1 Characteristics of all patients, and of those with paroxysmal or persistent atrial fibrillation (AF).

|                          | All patients (n = 81) | Paroxysmal AF (n = 41) | Persistent AF (n = 40) | p  
|--------------------------|-----------------------|------------------------|------------------------|---
| Male                     | 57 (70)               | 28 (68)                | 29 (72)                | 0.67  
| Age (years)              | 56 ± 11               | 56 ± 12                | 56 ± 11                | 0.99  
| Hypertension             | 34 (42)               | 18 (44)                | 16 (40)                | 0.72  
| Diabetes                 | 8 (10)                | 4 (10)                 | 4 (10)                 | 0.97  
| Hypercholesterolaemia    | 27 (33)               | 12 (29)                | 15 (37)                | 0.43  
| History of AF (months)   | 64 ± 66               | 66 ± 79                | 61 ± 49                | 0.76  
| Structural heart disease | 31 (38)               | 12 (29)                | 19 (48)                | 0.09  
| Ischaemic cardiomyopathy | 8 (10)                | 2 (5)                  | 6 (15)                 | 0.13  
| LVEF (%)                 | 55 ± 10               | 58 ± 7                 | 52 ± 12                | 0.01  
| LA diameter (mm)         | 43 ± 5                | 42 ± 6                 | 44 ± 5                 | 0.31  
| Beta-blockers            | 42 (52)               | 18 (44)                | 24 (60)                | 0.14  
| Amiodarone               | 30 (37)               | 13 (32)                | 17 (42)                | 0.32  
| Oral anticoagulation     | 75 (93)               | 37 (90)                | 38 (95)                | 0.41  
| Antiplatelet therapy     | 19 (23)               | 14 (34)                | 5 (12)                 | 0.02  
| Radiofrequency time (min)| 70 ± 36               | 48 ± 26                | 92 ± 30                | < 0.0001  
| Fluoroscopy time (min)   | 75 ± 25               | 62 ± 21                | 88 ± 22                | 0.007  

Data are mean ± standard deviation or number (%). AF: atrial fibrillation; LA: left atrium; LVEF: left ventricular ejection fraction.

these, 17 underwent cryoablation for paroxysmal AF and were therefore excluded from our analysis. No patients had PE on TTE before AF ablation. Therefore, 81 patients were included in the analysis. For each patient, only the first procedure was analysed in this study. The median follow-up for our population was 8 (range 6–23) months. Four patients were lost during our long-term follow-up.

Table 1 describes the characteristics of the total study population, as well as in patients with paroxysmal AF (n = 41) or persistent AF (n = 40; including 15 with long-lasting persistent AF).

Patients with paroxysmal versus persistent AF ablation had significantly higher left ventricular ejection fraction (LVEF) (58 ± 7% vs 52 ± 12%; P = 0.01); a higher rate of antiplatelet therapy (34% vs 12%; P = 0.02); and shorter radiofrequency time (48 ± 26 vs 92 ± 30 min; P < 0.0001).

Characteristics of pericardial effusions

Following AF ablation, 18 patients (22%) exhibited PE, detected by TTE. The characteristics of the PEs in these patients are shown in Table 2. Only two of the 18 patients (11%) complained of pericarditis pain associated with PE, and they were treated with non-steroidal anti-inflammatory drugs. None of these patients had tamponade or required surgical evacuation. In all of these patients, PE was self-limiting, as none of the patients had PE on TTE performed 1 month after the index procedure.

Characteristics of patients with or without pericardial effusion

As shown in Table 3, patients with PE had a significantly longer duration in AF before ablation (P = 0.002) and they experienced a longer duration of radiofrequency during the index procedure (P = 0.002). Interestingly, as shown in Fig. 1, the rate of PE was significantly higher following AF ablation in patients with persistent AF versus paroxysmal AF (35% vs 10%; P = 0.008).
Table 3  Characteristics of patients with or without pericardial effusion.

<table>
<thead>
<tr>
<th></th>
<th>Patients with PE (n=18)</th>
<th>Patients without PE (n=63)</th>
<th>p (^a)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>10 (56)</td>
<td>47 (75)</td>
<td>0.14</td>
</tr>
<tr>
<td>Age (years)</td>
<td>60 ± 12</td>
<td>56 ± 16</td>
<td>0.78</td>
</tr>
<tr>
<td>Hypertension</td>
<td>7 (39)</td>
<td>27 (43)</td>
<td>0.79</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1 (6)</td>
<td>7 (11)</td>
<td>0.68</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>9 (50)</td>
<td>18 (29)</td>
<td>0.10</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>14 (78)</td>
<td>26 (41)</td>
<td>0.008</td>
</tr>
<tr>
<td>Duration in AF (months)</td>
<td>7 ± 26</td>
<td>1 ± 6</td>
<td>0.002</td>
</tr>
<tr>
<td>Structural heart disease</td>
<td>7 (38)</td>
<td>24 (38)</td>
<td>0.99</td>
</tr>
<tr>
<td>Ischaemic cardiomyopathy</td>
<td>0</td>
<td>8 (13)</td>
<td>0.19</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>60 ± 10</td>
<td>60 ± 5</td>
<td>0.90</td>
</tr>
<tr>
<td>LA diameter (mm)</td>
<td>43 ± 5</td>
<td>44 ± 7</td>
<td>0.84</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>10 (56)</td>
<td>32 (51)</td>
<td>0.79</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>8 (44)</td>
<td>22 (35)</td>
<td>0.58</td>
</tr>
<tr>
<td>Oral anticoagulation</td>
<td>17 (94)</td>
<td>58 (92)</td>
<td>0.99</td>
</tr>
<tr>
<td>Antiplatelet therapy</td>
<td>2 (11)</td>
<td>17 (27)</td>
<td>0.22</td>
</tr>
<tr>
<td>Radiofrequency time (min)</td>
<td>93 ± 27</td>
<td>54 ± 50</td>
<td>0.002</td>
</tr>
</tbody>
</table>

Data are median ± interquartile range or number (%). AF: atrial fibrillation; LA: left atrium; LVEF: left ventricular ejection fraction; PE: pericardial effusion.

\(^a\) Mann-Whitney test and Fisher’s exact test were used to compare continuous variables and nominal variables, respectively.

Characteristics of patients with or without arrhythmia recurrences

In our population, 25 patients (31%) experienced EAR and 29 patients (36%) had late arrhythmia recurrences. Among patients experiencing EAR, 14/25 (56%) had AF, and 11/25 (44%) had atrial tachycardia. The characteristics of patients with or without EAR are described in Table 4. Most baseline characteristics were not significantly different between these two groups. However, patients with EAR had a significantly longer duration in AF before AF ablation compared to those without EAR (P < 0.0001). Additionally, patients with EAR had a higher prevalence of persistent AF (76% vs 38%; P = 0.002). Moreover, radiofrequency time was significantly longer in patients with versus without EAR (P = 0.0009).

As shown in Fig. 2A, the rate of PE was significantly higher among patients with EAR compared to those without (48% vs 11%; P = 0.0004). However, the rate of PE was similar in patients with or without late arrhythmia recurrences (Fig. 2B).

Multivariable analysis

Multivariable analysis showed that occurrence of PE and duration in AF were independent predictors of EAR (Table 5).

Redo ablations

Only 15 patients underwent a redo AF ablation. Among these, three (20%) developed a PE. Interestingly, these three patients also experienced PE after their first AF ablation procedure.

Figure 2.  Comparison of PE incidence occurring during the 24h after AF ablation in patients with or without early (A) or late (B) arrhythmia recurrences. AF: atrial fibrillation; PE: pericardial effusion.
Pericardial effusion and recurrences after atrial fibrillation ablation

Discussion

In our study, PE following AF ablation occurred in 22% of patients, and was more common following persistent than paroxysmal AF ablation (35% vs 10%). We also found that PE was independently associated with the occurrence of EAR after AF ablation, but was not related to late arrhythmia recurrences. Fortunately, the PEs in our study were mild, mainly asymptomatic, and always resolved within 1 month of the index procedure.

However, some patients can suffer major PE after AF ablation, resulting in cardiac tamponade that usually requires pericardiocentesis. Tamponade is considered to be one of the potentially life-threatening complications associated with AF ablation. Usually, cardiac tamponade happens during AF ablation and is associated with traumatic transseptal puncture. In a large, prospective study, Bertaglia et al. reported that 0.6% of 1011 patients had tamponade during AF ablation that was successfully treated with pericardiocentesis [3]. A similar rate (0.8%) was reported by Pappone et al. among 251 patients undergoing AF ablation [17]. Likewise, Chierchia et al. reported that 1.5% of 133 patients undergoing AF ablation with either radiofrequency or cryoablation had cardiac tamponade [4]; and Chen et al. found that tamponade occurred in 0.6% of 156 patients following AF ablation [9].

Contrary to acute tamponade, minor PEs are mainly asymptomatic and do not require pericardiocentesis. In the studies by Bertaglia et al. and Pappone et al., the rate of mild PE was 0.8% [3,17]. However, neither of these studies specifically looked for PE, and as such would not have recorded mild, asymptomatic PEs. In two studies that did specifically look for PE, as we did in our study, the rate of minor PE was much higher. In the study by Chierchia et al., 12% of patients had a minor PE, and 0.8%, a moderate PE [4]. Consistent with this, Chen et al. found mild PE in 10% of 156 patients following AF ablation [9]. In our study, 22% of patients had PE following AF ablation, which is somewhat higher than previously described. The discrepancy between the PE rate in our study and those in the former studies could be due to the fact that our rate of persistent AF ablation (49%) was much higher than in the previous studies (0% [4] and 27% [5]). We also found that PE occurred more frequently after persistent AF ablation, possibly due to longer radiofrequency time.

The pathophysiology of mild PEs following AF ablation is unknown. However, histopathological evidence of myocardial damage, including important local inflammatory responses following radiofrequency ablation, has been demonstrated in animal models [18] and human hearts [19]. Therefore, one possible mechanism could be a pericarditis due to local tissue inflammation secondary to radiofrequency. Moreover, AF ablation is associated with systemic inflammation activation [20,21] that could also explain the occurrence of pericarditis following AF ablation. Also, atrium trauma secondary to extensive biatrial radiofrequency ablation or catheter manipulation by itself could cause mild pericardial haemorrhage. As none of our patients had pericardiocentesis, we could not analyse the characteristics of the effusion in our population.

Table 4 Characteristics of patients with or without early arrhythmia recurrence (EARs).

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Patients with EAR (n=25)</th>
<th>Patients without EAR (n=56)</th>
<th>p a</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male sex</td>
<td>16 (64)</td>
<td>41 (73)</td>
<td>0.44</td>
</tr>
<tr>
<td>Age (years)</td>
<td>60±12</td>
<td>57±17</td>
<td>0.37</td>
</tr>
<tr>
<td>Hypertension</td>
<td>11 (44)</td>
<td>23 (41)</td>
<td>0.81</td>
</tr>
<tr>
<td>Diabetes</td>
<td>3 (12)</td>
<td>5 (9)</td>
<td>0.70</td>
</tr>
<tr>
<td>Hypercholesterolaemia</td>
<td>11 (44)</td>
<td>16 (29)</td>
<td>0.21</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>76</td>
<td>3</td>
<td>0.002</td>
</tr>
<tr>
<td>Duration in AF (months)</td>
<td>13±33</td>
<td>1±4</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Structural heart disease</td>
<td>6 (24)</td>
<td>25 (45)</td>
<td>0.09</td>
</tr>
<tr>
<td>Ischaemic cardiomyopathy</td>
<td>2 (8)</td>
<td>6 (11)</td>
<td>0.99</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>60±4</td>
<td>60±6</td>
<td>0.79</td>
</tr>
<tr>
<td>LA diameter (mm)</td>
<td>43±5</td>
<td>45±8</td>
<td>0.86</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>15 (60)</td>
<td>27 (48)</td>
<td>0.35</td>
</tr>
<tr>
<td>Amiodarone</td>
<td>7 (28)</td>
<td>23 (41)</td>
<td>0.32</td>
</tr>
<tr>
<td>Oral anticoagulation</td>
<td>24 (95)</td>
<td>51 (91)</td>
<td>0.66</td>
</tr>
<tr>
<td>Antiplatelet therapy</td>
<td>4 (16)</td>
<td>15 (27)</td>
<td>0.40</td>
</tr>
<tr>
<td>Radiofrequency time (min)</td>
<td>98±46</td>
<td>53±45</td>
<td>0.0009</td>
</tr>
</tbody>
</table>

Data are median±interquartile range or number (%). AF: atrial fibrillation; LA: left atrium; LVEF: left ventricular ejection fraction; PE: pericardial effusion.

Table 5 Multivariable analysis of predictors of early arrhythmia recurrence (EAR).

<table>
<thead>
<tr>
<th>Predictor</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Duration of AF</td>
<td>1.045</td>
<td>1.001± 0.085</td>
<td>0.04</td>
</tr>
<tr>
<td>Persistent AF</td>
<td>0.864</td>
<td>0.182± 4.053</td>
<td>0.84</td>
</tr>
<tr>
<td>Pericardial effusion</td>
<td>3.466</td>
<td>1.001± 14.22</td>
<td>0.04</td>
</tr>
<tr>
<td>Radiofrequency time</td>
<td>1.024</td>
<td>0.999± 1.042</td>
<td>0.06</td>
</tr>
</tbody>
</table>

AF: atrial fibrillation; CI: confidence interval.
The clinical significance of mild PEs on arrhythmia recurrences following AF ablation has not, to the best of our knowledge, been specifically evaluated in previous studies. The definition of early recurrences following AF ablation varies in the literature depending on the timing and the type of arrhythmia documented after the first AF ablation procedure. Oral et al. defined EAR as a sustained episode of AF within 15 days of the procedure [8]. However, Lee et al. defined EAR as a sustained episode of AF within 1 month of the procedure [9], while O’Donnell et al. defined EAR as a recurrence of AF within 3 months of the ablation procedure [22]. Since the optimal time limit to define EAR has not been specifically established, in this study, arrhythmia recurrences were considered to be early if they occurred within 1 month of the index procedure, as has been used in other studies [9,23]. This is thought to correspond with an unstable state due to the initial inflammatory process following the ablation [18,23].

In our study, the EAR rate was 31%. This is consistent with previous studies that have reported that 13—50% of patients have EAR following paroxysmal or persistent AF ablation [8,9,24]. However, the clinical significance of EAR is controversial [8,9,24]. Many investigators consider that EAR should be treated by antiarrhythmic drugs until a stable atrial tissue state is present [8,25]. Consistent with this, the recent consensus document on catheter and surgical ablation of AF has suggested a uniform ‘blanking period’ of 3 months [10].

The pathophysiology of EAR after AF ablation has not been elucidated. However, multiple hypotheses have been suggested, such as LA oedema following radiofrequency tissue lesion that could lead to alterations in conduction velocity [26—28]. Moreover, AF ablation can modify the autonomic nervous system, leading to an increased risk of atrial arrhythmias [11,12].

Additionally, some patients with EAR will not experience late recurrences. Therefore, some investigators have suggested that a delayed beneficial effect of radiofrequency ablation, secondary to scar consolidation, may explain this phenomenon [29—31]. In our study, 48% of patients with EAR experienced PE immediately after AF ablation. We also found that PE was independently associated with EAR. We therefore hypothesize that mild PE could be a significant marker of pericardial irritation leading to EAR.

Most of our patients with PE were asymptomatic; and the two patients who developed pericarditis-like chest pain were treated with non-steroidal anti-inflammatory drugs. However, due to the small number of patients, we could not analyse the effect of non-steroidal anti-inflammatory drugs in this population on mid- or long-term clinical outcome after AF ablation. Recently, Koyama et al. showed that corticosteroids administered 3 days after AF ablation could decrease the occurrence of EAR [32]. Although the main mechanism suspected by the authors was a decrease in atrial tissue proarrhythmogenic inflammation, another explanation could be a direct effect of corticosteroids on PE.

Finally, we could hypothesize that cardioversion in patients experiencing acute PE and EAR following AF ablation may be postponed until PE resolution. However, larger studies are required to confirm this interesting hypothesis.

Limitations
This was a single-centre study, and our results need confirmation in a larger population of patients. In our study, the overall clinical success may have been overestimated due to some undiagnosed episodes of asymptomatic arrhythmia. Moreover, we did not assess systemic inflammatory activation, such as C-reactive protein measurement, to correlate these markers with PE occurrence. Finally, the effect of drugs on PEs and the impact on future arrhythmias were not specifically studied.

Conclusions
PE following AF ablation with radiofrequency is frequent, particularly following persistent AF ablation. In our study population, PE was mild, mainly asymptomatic, and independently associated with EARs.

Disclosure of interest
The authors declare that they have no conflicts of interest concerning this article.

References


