Clinical Research

Peridevice Leak After Left Atrial Appendage Closure: Incidence, Risk Factors, and Clinical Impact

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See editorial by Freixa and Regueiro, pages 370–372 of this issue.

ABSTRACT

Background: Limited studies reported the rate and clinical impact of peridevice leaks (PDL) after percutaneous left atrial appendage closure (LAAC).

Methods: All consecutive patients with a nonvalvular atrial fibrillation admitted for LAAC between November 2011 and October 2016 were prospectively enrolled. The follow-up included clinical, transesophageal echocardiography, and/or cardiac computed tomography angiogram (CCTA). PDL was defined by the presence of contrast within the left atrial appendage on CCTA, and Major Adverse Cardiac Event (MACE) included stroke, device-related thrombosis, and cardiovascular death.

Results: Overall, 77 patients (mean CHA2DS2-VASc score = 4.4 ± 1.5 and mean HAS-BLED = 3.4 ± 1.1) were implanted using Amplatzer Cardiac Plug (n = 24), Amulet (n = 37), or Watchman devices (n = 16). Indications were stroke recurrence despite adequate oral anticoagulation (OAC, n = 6) or contraindication to long-term OAC (n = 71). From 3-month to 12-month CCTA follow-up, the PDL rate decreased from 68.5% to 56.7% (P = 0.02), without any difference between the various devices. Patients with PDL were more often in permanent atrial fibrillation, and had a larger landing zone diameter, a

RÉSUMÉ

Introduction : Peu d’études ont fait état des taux et des répercussions cliniques des fuites periprothétiques (FPP) après la fermeture de l’appendice auriculaire gauche (AAG) par voie percutanée.

Méthodes : Nous avons inscrit de manière prospective tous les patients consécutifs atteints de fibrillation auriculaire non valvulaire admis pour la fermeture de l’AAG par voie percutanée entre novembre 2011 et octobre 2016. Le suivi comportait une échocardiographie transösophagienne clinique ou une angiographie cardiaque par tomodensitométrie (TDM), ou les deux. La FPP était définie par la présence du produit de contraste dans l’AAG à l’angiographie cardiaque par TDM. Les événements cardiaques indésirables majeurs (ECIM) étaient les suivants : l’accident vasculaire cérébral (AVC), la thrombose de l’endoprothèse et le décès d’origine cardiovasculaire.

Résultats : Dans l’ensemble, 77 patients (score moyen au CHA2DS2-VASc, soit l’insuffisance cardiaque congestive, l’hypertension, l’âge [≥ 75 ans], le diabète, l’AVC/l’accident ischémique transitoire, la maladie vasculaire, l’âge [de 65 à 74 ans], le sexe [féminin] = 4.4 ± 1.5 et score moyen au HAS-BLED = 3.4 ± 1.1) ont subi l’implantation d’un dispositif Amplatzer Cardiac Plug (n = 24), d’un dispositif Amplatzer Amulet.

Atrial fibrillation (AF) is responsible for 15% to 20% of ischemic strokes, and the overall risk of stroke in patients with nonvalvular AF is as high as 5% per year.1 Long-term oral anticoagulation (OAC) is recommended by current guidelines to prevent thromboembolic events in patient with AF and elevated Congestive Heart Failure, Hypertension, Age (≥ 75 years), Diabetes, Stroke/Transient Ischemic Attack, Vascular Disease, Age (65-74 years), Sex (Female) (CHA2DS2-VASc) score. However, approximately 30% of patients with AF who require long-term OAC are not correctly treated because of contraindications, intolerance, high bleeding risk, or patient refusal.2,3 Autopsy and surgical studies including patients with nonvalvular AF demonstrated that 90% of thrombi originate from the left atrial appendage (LAA).4 Therefore, surgical techniques and device implantations to exclude the LAA from the systemic circulation have been explored since 1949 as a

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lower ratio of device compression, and a more frequent off-axis position of the device. A device compression ratio < 10% was the only parameter associated with PDL occurrence. During follow-up (median 236 days) the MACE rate was 9.1%, with no statistically significant difference between patients with vs without PDL (12% vs 4.3%, P = 0.3).

Conclusions: The PDL rate detected by CCTA after LAAC was high, especially in cases with a low device compression ratio (< 10%), but decreased over time. The incidence of MACE was quantitatively greater with PDL, but the difference was not statistically significant. Larger studies are needed to determine the clinical importance of PDL.

Methods

Study population

We prospectively included all patients admitted in our institute (Henri Mondor Hospital, Créteil, France) for percutaneous LAAC. All patients had nonvalvular AF associated with a contraindication to long-term OAC or a recurrence of thromboembolic event despite adequate OAC. Exclusion criteria were procedure failure (n = 3) and the absence of follow-up in our hospital (n = 13). All patients underwent an LAA analysis during the month before the procedure using transesophageal echocardiography (TEE) associated with cardiac computed tomography angiogram (CCTA) when possible (ie, clearance > 20 mL/min/1.73 m²). LAA analysis included LAA shape (“chicken-wing” or not), the maximal diameter of the LAA landing zone, and exclusion of thrombosis. The local ethic committee approved the protocol and patients gave informed consent.

LAAC procedure

Percutaneous LAAC was performed by only 2 operators (ET and JT) and under general anaesthesia with TEE guidance. Transseptal punctures were performed inferoposteriorly at the fossa ovalis with an SL1 (St Jude Medical, Burlington, MA) transseptal sheath and the BRK-1TM XS needle (St Jude Medical). After crossing the atrial septum, a guidewire was positioned into the left upper pulmonary vein for the positioning of the delivery sheath into the LA and an intravenous bolus of heparin was given to maintain the activated clotting time > 250 seconds. Then, LA pressure was measured and a volume loading of saline solution was performed to achieve a mean pressure of 10 mm Hg. A 5F marker pigtail was advanced into the LAA, and an angiogram was performed to find the best orientation on fluoroscopy. Then, the delivery sheath was advanced into the LAA. The 2 available devices were used at the discretion of the operators (E.T. and J.T.) in accordance with the constructor’s guidelines. The choice of the device size was defined during the procedure and based on the largest landing zone diameter measured by TEE oversized from 10% to 20%. For the Amplatzer Cardiac Plug (ACP) and the Amulet devices (St Jude Medical), the widest LAA diameter measured at a depth of 10 to 15 mm behind the LAA ostium defined the landing zone. For the Watchman device (Boston Scientific, Quincy, MA), the widest anatomic orifice from circumflex artery (inferiorly) to a point positioned 1 to 2 cm inside behind the tip of the pulmonary vein ridge (superiorly) defined the landing zone. Before device release, we strove to achieve the recommended position, anchor, size, and seal criteria for the Watchman device, and the 5 signs of correct deployment for ACP/Amulet devices: (i) tire-shaped lobe, (ii) separation of the lobe from the disc, (iii) concavity of the disc, (iv) axis of the lobe should be perpendicular to the neck axis at the landing zone, and (v) width of the lobe is ≥ 2/3 within the circumflex artery.
After the procedure, patients were admitted 24 to 48 hours and had a transthoracic evaluation the next day.

**Antithrombotic regimen after the procedure**

Antithrombotic regimen after LAAC was defined by a multidisciplinary team and consisted in a single or a dual antiplatelet therapy (DAPT, low-dose aspirin plus clopidogrel) for 1 to 6 months in patients with contraindication to long-term OAC, and OAC associated or not with aspirin in patients with a recurrence of stroke despite adequate OAC. Discontinuation of the antithrombotic treatment in patients contraindicated for OAC was systematically discussed by the team, but low dose of aspirin was generally continued in patients with a history of coronary artery disease. The presence of a PDL did not influence the therapeutic management.

**Follow-up after the procedure**

Patients were followed clinically and using CCTA and/or TEE at 3, 6, and 12 months, and annually after the procedure. Clinical data included the occurrence of Major Adverse Cardiac Event (MACE) including stroke, device-related thrombosis, and cardiovascular death. Acquisition and analysis protocols with PDL definitions using CCTA and TEE during the follow-up are detailed in the Supplemental Appendix S1. Both CCTA and TEE were used to (1) detect device embolization, (2) detect and size PDL defined by the presence of contrast into the LAA beyond the device on CCTA (Fig. 1A) or color Doppler flow around the device by TEE, (3) detect device-related thrombosis, (4) determine the off-axis position of the device defined by the nonperpendicular apposition of the ACP/Amulet lobe against the wall of the LAA, and (5) measure the device compression ratio (Fig. 1B).

**Objectives of the study**

The objectives of the study were to (1) assess the incidence of PDL and their evolution, (2) identify the mechanism and factors associated with PDL, and (3) evaluate the association between PDL and MACE.

**Statistical analysis**

Descriptive statistics were used to describe the baseline characteristics of patients. Continuous variables were summarized as mean ± standard deviation, or median and interquartile range. Categorical variables were summarized as frequency and percentage. Categorical variables were compared using a χ² test or Fisher’s exact test, and continuous variables using the Student t-test or the Mann-Whitney U test. Univariate analysis was used to identify factors associated with the presence of PDL. The cutoff value for the device compression was defined by the median value. The intra- and interobserver variability was assessed blindly by 2 readers (A.N. and J.T.) in 20 random CCTA. Multivariate linear regression analysis was performed to identify variables independently associated with the presence of PDL. Variables with a P value < 0.05 on bivariate analysis were entered into the multivariable analysis. A P value < 0.05 was considered statistically significant. Statistical analyses were performed with the JMP software (SAS Institute Inc, Cary, NC).

**Results**

**Study population**

Overall, 93 patients were admitted in our institute (Henri Mondor Hospital) for an LAAC procedure between November 2011 and October 2016. Among them, 3 patients were excluded because of procedure failure and 13 patients were excluded because of the absence of follow-up at Henri Mondor Hospital. Thus, the technical success rate was 96.8%. Baseline and procedural characteristics of the excluded patients were similar to the final study population. Finally, 77 patients (mean age 75.3 ± 8.3 years, 64% male) were enrolled in the study and their clinical characteristics are shown in Supplemental Table S1. Forty-two percent of patients were in sinus rhythm before the procedure. In a vast majority of patients (92%), LAAC was performed because of the presence of a contraindication to long-term OAC, whereas only 8% of patients had LAAC for a recurrence of a thromboembolic event despite adequate OAC. The reasons for OAC contraindication were mainly intracranial and gastrointestinal.

**Figure 1.** Cardiac computed tomography angiogram image illustrated the peridevice leak definition (A, red arrow indicates the contrast beyond the device) and the device compression ratio evaluation (B).
bleedings (Supplemental Table S2). The majority of patients had both a high thromboembolic and bleeding risk with a CHA₂DS₂-VASc score of ≥ 4 and a HAS-BLED score of ≥ 3 in 76.7% and 85.8% of cases, respectively (Fig. 2).

LAAC procedure and in-hospital complications

Imaging analysis before the procedure identified 22 patients (28%) with an LAA “chicken-wing” anatomy and found a mean diameter of the landing zone of 21 ± 4 mm (13-28 mm). LAAC was performed using a St Jude Medical device in 79% of patients (48% Amulet and 31% ACP). During the procedure, only 5 patients required a device exchange for another size, whereas 94% (n = 72/77) of patients were implanted at the first attempt. The main procedural data are shown in Table 1. Before hospital discharge, no death, stroke, or pericardial effusion was reported. The complications after the procedure were mainly related to vascular access according to the Valve Academic Research Consortium (1 major and 4 minor gastrointestinal bleedings). Bleeding according to the Bleeding Academic Research Consortium (1 major and 6 minor), and to the Watchman device (2 major and 4 minor gastrointestinal bleedings).

Follow-up and antithrombotic therapy management after LAAC

Clinical and imaging follow-up were available in 77 (100%), 48 (62%), and 30 (39%) patients at 3, 6, and 12 months. The median follow-up was 236 days (160-404 days).

At hospital discharge, most of patients (48.1%) were treated by a DAPT, whereas 2.6% of patients had no antithrombotic therapy (Fig. 3). At 3 months of follow-up, the majority of patients (51.3%) were treated by aspirin or clopidogrel alone, whereas a DAPT was continued in only 13.2% of patients. At 12 months, a single and a DAPT were continued in 60.7% and 10.2% of patients, respectively. Interestingly, the proportion of patients without antithrombotic therapy increased from 17% at 3 months to 25% at 12 months.

Incidence and mechanism of PDL

CCTA follow-up was available in 95% (n = 73/77), 62% (n = 48/77), and 40% (n = 30/77) of patients at 3, 6, and 12 months, respectively. In addition, 27 patients had a complete CCTA follow-up at 3, 6, and 12 months. Between 3-month and 12-month CCTA follow-up (Table 2), the PDL rate decreased (P = 0.02) from 68.5% (n = 50/73) to 56.7% (n = 17/30). The same results were observed in the 27 patients with a complete imaging follow-up: the PDL rate decreased from 74.1% at 3 months to 59.3% at 12 months (P < 0.05). No difference in the PDL rate was observed between the 2 types of devices at 3 months: 68% (40/59) with the ACP and Amulet devices, and 71% (10/14) with the Watchman device (P = 0.8). Regarding the echocardiography follow-up, 41.5% (n = 32/77) of patients underwent a TEE examination at 3 months, showing a PDL in 9.4% of cases. Interestingly, 28 patients had both CCTA and TEE examinations at the 3-month follow-up. Among these patients, only 2 PDL were observed using TEE (7.1%), whereas PDL was observed in 18 patients (64.3%) using CCTA.

The PDL mechanisms were identified on CCTA in 98% (n = 49/50) of patients. The 3 observed mechanisms (Fig. 4) were: (1) absence of device sealing in 5 patients, (2) presence of a gap between the device and the LAA wall in 14 patients, and (3) an off-axis position of the device (only for the St Jude Medical devices) in 30 patients. The distribution of PDL mechanisms according to the type of device is detailed in Figure 5. In patients with a peridevice gap, the mean size of the defect was 2.8 ± 1.3 mm but all were < 5 mm. Interestingly, in patients with a complete imaging follow-up, PDL related to an absence of device sealing was the only mechanism that completely disappeared at 6 and 12 months.

Parameters associated with PDL occurrence

Using univariate analysis, patients with PDL on CCTA after LAAC were more often in permanent AF, with a larger

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**Table 1. Details of the LAAC procedures**

<table>
<thead>
<tr>
<th>Procedures course</th>
<th></th>
</tr>
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<tbody>
<tr>
<td>Procedure duration, min</td>
<td>55 (range, 40-80)</td>
</tr>
<tr>
<td>Fluoroscopy time, min</td>
<td>12 (range, 9-16)</td>
</tr>
<tr>
<td>Amount of iodine contrast, ml.</td>
<td>90 (range, 60-125)</td>
</tr>
<tr>
<td>Irradiation dose, mGy cm²</td>
<td>22.440 (range, 14.705-33.420)</td>
</tr>
</tbody>
</table>

**Devices used**

<table>
<thead>
<tr>
<th>Amulet, No. (%)</th>
<th>37 (48%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACP, No. (%)</td>
<td>24 (31%)</td>
</tr>
<tr>
<td>Watchman, No. (%)</td>
<td>16 (21%)</td>
</tr>
</tbody>
</table>

**Size of most implanted devices**

<table>
<thead>
<tr>
<th>Amulet 22 mm, No. (%)</th>
<th>12 (32)</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACP 22 mm, No. (%)</td>
<td>7 (29)</td>
</tr>
<tr>
<td>Watchman 30 mm, No. (%)</td>
<td>5 (31)</td>
</tr>
</tbody>
</table>

ACP, Amplatzer Cardiac Plug; LAAC, left atrial appendage closure.

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**Figure 2.** Distribution of the study population according to the (A) CHA₂DS₂-VASc and (B) HAS-BLED scores. CHA₂DS₂-VASc, Congestive Heart Failure, Hypertension, Age ≥ 75 years, Diabetes, Stroke/Transient Ischemic Attack, Vascular Disease, Age 65-74 years, Sex [Female]; HAS-BLED, Hypertension, Abnormal Renal/Hepatic Function, Stroke, Bleeding, Labile INRs, Elderly ≥ 65 Years, Drugs or Alcohol Use.
maximal diameter of the landing zone measured on CCTA before the procedure, a lower ratio of device compression, and a more frequent off-axis position of the device. Interestingly, the “chicken-wing” anatomy was not associated with the presence of PDL (Table 3). According to the median value of the device compression ratio (10%), we found that patients with a device compression ratio < 10% had a higher rate of PDL (85% ± 0.4%) than patients with > 10% of device compression ratio (50% ± 0.5%, P = 0.001). Using multivariate analysis, only a device compression ratio < 10% was independently associated with the presence of PDL (odds ratio 5.5, 95% confidence interval [1.3-23.1]). Interobserver and intraobserver variability for device compression ratio measurement was 1% (r = 0.99 [0.95-1.03]) and 0.8% (r = 0.99 [0.73-0.80]), respectively.

Association between PDL and MACE

During follow-up, MACE occurrence was observed in 9.1% of all patients including 2 strokes (2.6%), 3 device-related thrombosis (4.1%), and 2 cardiovascular deaths (heart failure, 2.6%). The incidence of MACE was quantitatively greater with PDL, but the difference was not statistically significant (12% in patients with PDL vs 4.3% in patients without PDL, P = 0.3). The same results were observed in patients with a TEE follow-up (13% in patients with PDL vs 2.2% in patients without PDL, P = 0.9). Details of MACE between patients with and without PDL are listed in Table 4. The 3 patients with device-related thrombosis (1 ACP, 1 Amulet, and 1 Watchman) were asymptomatic, and the diagnosis was made by the systematic CCTA control at 3 months. Among patients with device-related thrombosis, 2 were treated by aspirin alone and 1 by OAC. Among patients who experienced a stroke, the first was treated by aspirin alone and the second by a DAPT. Interestingly, no adverse event was observed in patients with no antithrombotic therapy.

Discussion

In this prospective study, we found that (1) PDL after LAAC is very common and decreased over time, (2) a low device compression ratio (< 10%) is associated with PDL, and (3) there was no association between PDL and MACE.

Table 2. Prevalence of peridevice leak and device-related thrombosis during the cardiac computed tomography angiogram follow-up

<table>
<thead>
<tr>
<th></th>
<th>3 month</th>
<th>6 month</th>
<th>12 month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>73</td>
<td>48</td>
<td>30</td>
</tr>
<tr>
<td>Passage of contrast into the LAA, No. (%)</td>
<td>50 (68.5%)</td>
<td>30 (62.5%)</td>
<td>17 (56.7%)</td>
</tr>
<tr>
<td>Device-related thrombosis, No. (%)</td>
<td>3 (4.1%)</td>
<td>2 (4.2%)</td>
<td>2 (6.9%)</td>
</tr>
</tbody>
</table>

LAA, left atrial appendage.
from 68.5% to 56.7%. Importantly, more than a half of the patients had persistent PDL 12 months after the procedure.

Mechanisms and risk factor: how to prevent PDL occurrence?

As demonstrated by Saw et al., we also identified 3 mechanisms causing PDL after LAAC. The first mechanism was very frequent (62% of our patients with an Amulet or ACP device) but specific of the St Jude Medical devices: the off-axis position. The absence of perpendicularity between the LAA landing zone and the lobe of the St Jude Medical device is responsible for a misalignment of the sealing system of the device into the LAA. The second mechanism was the presence of a gap related to a lack of expansion of the device against the LAA wall at the landing zone. The last mechanism was the absence of device sealing related to an incomplete endothelialization. Interestingly, we identified 5 patients with this mechanism by CCTA at the 3-month follow-up, which implies that the device endothelialization is longer than 45 days contrary to what is expected or reported in animal studies. The first 2 mechanisms were responsible for persistent PDL throughout the follow-up, whereas the incomplete device sealing disappeared at 6 months. Thus, the control by CCTA should be performed at least 3 months after the LAAC procedure to detect PDL related to persistent mechanisms.

Before and during the LAAC procedure, cardiac imaging allows us to exclude LAA thrombosis, to analyse LAA anatomy, and to determine the LAA landing zone position and size to anticipate the correct device choice and deployment. In this setting, we identified the device compression ratio as the major parameter associated with the presence of PDL during follow-up with a threshold of 10%. This value of 10% is consistent with previous publications and should be a goal during the procedure before device releasing to avoid large PDL. However, the challenge is to obtain a device compression ratio over 10% while avoiding an off-axis position of the

![Figure 4. Cardiac computed tomography angiogram examples of the 3 peridevice leak mechanisms: absence of device sealing (A with the Amulet device and B with the Watchman device; red arrow indicates the contrast beyond the device), peridevice gap (C and D with the Watchman device; red arrow indicates the gap), and off-axis position of the Amulet device (E and F; red line indicates the main axis of the left atrial appendage and the theoretical correct device position is represented in blue).]
device, which is difficult in some anatomies. As proposed by Ciobotaru et al.,21,22 the use of 3-dimensional printing models could be useful in patients with a complex LAA anatomy for optimizing the procedure.

Relation between PDL and MACE

The objective of LAAC is to prevent thromboembolic complications in patients with nonvalvular AF in patients with a contraindication to long-term OAC. Previous prospective studies and registries, and our results confirm the LAAC efficacy. Thus, during the follow-up, we observed only 2 strokes (2.6%), whereas the predicted annual risk according to the CHA2DS2-VASc score was between 4% and 6.7%. Several authors apprehend the association between PDL and thromboembolic complications. Persistence of an incomplete LAA occlusion is responsible for a residual flow between the left atrium and the LAA, which may promote thrombosis due to blood turbulence and stagnation. However, the present study and others15,18,19 did not find significant association between PDL after LAAC and MACE, including thromboembolic events. Thus, the mere presence of a small PDL (< 5 mm) does not seem to justify a systematic imaging follow-up.

Study limitations

First, the population study was small but patients were included and followed on a regular and prospective basis. Second, few patients had both TEE and CCTA for the comparison of imaging diagnostic performance, but it was not the aim of the study. Third, the device compression ratio was measured on the 3-month CCTA, whereas this parameter should be assessed during the procedure. However, this ratio could be assessed using TEE before device release. Finally, the rate of MACE in our population was low. This is good news for the efficiency of the LAAC procedure, but it can be a limit to the interpretation of the results. The MACE rate in patients with PDL was slightly higher, but not significantly, than in patients without PDL. The small size of the population studied can explain this lack of power, and larger studies are needed to determine the clinical significance of PDL. It is also important to note that no patient had severe PDL as defined

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Presence of PDL (n = 50)</th>
<th>Absence of PDL (n = 23)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Permanent atrial fibrillation, No. (%)</td>
<td></td>
<td></td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Maximal diameter of LAA landing zone, mm</td>
<td></td>
<td></td>
<td>0.04</td>
</tr>
<tr>
<td>“Chicken-wing” shape of LAA, No. (%)</td>
<td></td>
<td></td>
<td>1</td>
</tr>
<tr>
<td>Device size, mm</td>
<td></td>
<td></td>
<td>0.09</td>
</tr>
<tr>
<td>Device compression ratio, %</td>
<td></td>
<td></td>
<td>0.01</td>
</tr>
<tr>
<td>Device compression ratio &lt; 10%, No. (%)</td>
<td></td>
<td></td>
<td>0.001</td>
</tr>
<tr>
<td>Off-axis position of the device, No. (%)</td>
<td></td>
<td></td>
<td>0.03</td>
</tr>
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</table>

LAA, left atrial appendage; LAAC, LAA closure; PDL, peridevice leak.

Table 4. MACE description between patients with and without PDL

<table>
<thead>
<tr>
<th></th>
<th>Patients with PDL (n = 50)</th>
<th>Patients without PDL (n = 23)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke, No. (%)</td>
<td>2 (4)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Thrombosis, No. (%)</td>
<td>2 (4)</td>
<td>1 (4.3)</td>
</tr>
<tr>
<td>Cardiovascular death, No. (%)</td>
<td>2 (4)</td>
<td>0 (0)</td>
</tr>
</tbody>
</table>

MACE, Major Adverse Cardiac Event; PDL, peridevice leak.
in the literature (> 5 mm). Thus, these results should be interpreted with caution in such patients.

**Conclusions**

The PDL rate detected by CCTA after LAAC is high, especially in cases with a low device compression ratio (< 10%), but decreased over time. The incidence of MACE was quantitatively greater with PDL, but the difference was not statistically significant. Larger studies are needed to determine the clinical importance of PDL.

**Disclosures**

The authors declare that they have no relevant conflicts of interest to disclose.

**References**


**Supplementary Material**

To access the supplementary material accompanying this article, visit the online version of the Canadian Journal of Cardiology at www.onlinejcc.ca and at https://doi.org/10.1016/j.cjca.2018.12.022.