Incidence and Predictors of Pericardial Effusion After Permanent Heart Rhythm Device Implantation
– Prospective Evaluation of 968 Consecutive Patients –

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Background: Pericardial effusion (PE) may complicate permanent heart rhythm device (HRD: pacemaker, implantable cardioverter-defibrillator, and cardiac resynchronization therapy) placement. Incidence of and risk factors for this complication have never been prospectively evaluated.

Methods and Results: The subjects of this prospective observational study were 968 consecutive patients undergoing HRD implantation or upgrade, and underwent echocardiographic evaluation before and 24 h after the operation. PE was documented in 98 patients (10%), 14 (1.5%) of whom progressed to cardiac tamponade requiring pericardiocentesis (n=12; 86%) or surgical treatment (n=2; 14%). In 70% (10/14) of those patients a bloody effusion suggested cardiac perforation of an implanted lead; acute pericarditis was observed in the remaining 30% (4/14). At multivariate analysis, female gender (hazard ratio [HR], 2.7; 95% confidence interval [CI]: 1.4–3.5, P=0.01) was predictive in the case of any post-procedural PE, whereas intake of antiplatelet medication (HR, 3.1; 95% CI: 2.1–3.8, P=0.01) was predictive for cardiac tamponade. Previous cardiac surgery (HR, 0.70; 95% CI: 0.50–0.92, P=0.02) was a protective factor in any PE and cardiac tamponade. None of the 84 patients with small or moderate PE required pericardial drainage. After 3.1±0.5 months, a PE was no longer observed at echocardiography in 71% of those patients.

Conclusions: PE is frequently seen after HRD implantation, but rarely requires any therapy. Female gender and antiplatelet therapy are risk factors, whereas previous cardiac surgery was a protective factor. (Circ J 2013; 77: 975–981)

Key Words: Implantable cardioverter-defibrillator; Pacemaker; Pericardial effusion; Resynchronization therapy; Tamponade

Heart rhythm device (HRD: pacemaker [PM], implantable cardioverter-defibrillator [ICD], and cardiac resynchronization therapy [CRT]) implantation is associated with post-procedural complications in 0.3–7% of patients. In retrospective series, up to 2% of implants are followed by pericardial effusion (PE) and tamponade. Recent analyses have shown that PE and/or tamponade increased the hospital costs by US$8,249, prolonged the hospitalization by 1.9 days, and led to higher mortality rates. Nevertheless, this issue has never been prospectively evaluated. Therefore, the aim of the present study was to prospectively evaluate, in a large cohort of patients, both the incidence and clinical predictors of PE and tamponade following HRD implantation.

Subjects
Between June 2007 and May 2010, all consecutive patients undergoing HRD implantation or upgrade (where ≥1 additional lead(s) had been placed) at Zentralklinik Bad Berka, Germany, were included in this prospective observational study. Presence of pre-procedural PE, planned lead extraction and age <18 years were considered exclusion criteria.

The following risk factors, potentially related to periprocedural PE and tamponade, were assessed: (1) patient related factors: age, gender, evidence of elevated right ventricular (RV) systolic blood pressure [SBP], body mass index (BMI), history of prior myocardial infarction, previous cardiac surgery, hyperlipidemia, diabetes, pulmonary hypertension (mean pul...
monary artery pressure ≥25 mmHg), renal failure (glomerular filtration rate <60 ml/min), and hypertension; (2) procedure-related factors: total procedural time, use of a temporary PM lead, number and location of leads implanted, type of leads (active vs. passive fixation, although there were only active fixation leads in the atrium), site of lead placement (right atrial [RA] appendage vs. other locations, eg, RA lateral wall/RA septum; RV apex vs. other locations, e.g. RV septum/RV outflow tract), devices implanted (ICD/CRT-D vs. PM/CRT-P), and implantation performed by a cardiologist in training; (3) laboratory on admission: platelet count, international normalized ratio, and activated partial thromboplastin time; and (4) current medications: use of platelet aggregation inhibitors (aspirin and/or thienopyridines), warfarin, or chronic oral steroids.

Operative Techniques and Standard of Care
Four different operators performed all procedures. Each implanting physician had an experience of ≥200 procedures. A cardiology fellow was involved in 24/968 (2.5%) of the procedures under direct supervision of an attending cardiologist, although the level of involvement could not be accurately defined in all cases.

After prophylactic antibiotics and local anesthesia, a pectoral incision under the clavicle was made and the subclavian pocket was created. Venous access was achieved by puncture of the subclavian vein (1 puncture per lead), and leads were implanted under fluoroscopic guidance. An active fixation mechanism was chosen for all atrial leads and for ventricular leads in case of severe tricuspid regurgitation, pulmonary hypertension, or implantation of an ICD. All left ventricular leads were inserted via the coronary sinus. Hemostasis was appropriately obtained both with electrocautery and non-treated cotton pledges. After the procedure, bed rest was suggested while a pressure dressing was applied for 24 h.

Echocardiography and Follow-up
Two-dimensional (2-D) echocardiography was performed using commercially available equipment within 24 h after HRD implantation. Location and distribution of the PE were assessed. A semi-quantitative approach for effusion sizing was used as proposed by Roeland and Erbel. Small effusion, ≤10 mm in systole and diastole; moderate effusion, 11–20 mm in diastole; and large effusion, >20 mm in diastole.

M-mode, 2-D echocardiography, and trans-tricuspid/trans-mitral Doppler flow patterns were used to exclude the presence of tamponade. RA compression during late diastole, RV collapse during early diastole, abnormal tricuspid and/or mitral inflow pattern, dilated inferior vena cava with lack of inspiratory collapse, and swinging heart were all considered echocardiographic characteristics of tamponade.

All patients with diagnosis of post-implant PE underwent at least a second echocardiogram before hospital discharge. Outpatient visits were arranged at 4 weeks and 3 months for echocardiographic and device follow-up, respectively. Telephone interviews with patients, their families, and their primary physician were conducted as necessary.

Pericardiocentesis
Cardiac tamponade was diagnosed when the following classical clinical criteria were met: hypotension (SBP <90 mmHg), pulsus paradoxus (decline of >12 mmHg or ≥9% in inspiratory SBP), and increased jugular venous pressure. Patients without the classical clinical criteria but with echocardiographic evidence of hemodynamic relevant PE (as defined here) also underwent pericardiocentesis. We performed X-ray-guided pericardiocentesis in the catheterization laboratory via the subxiphoid approach. A 7-Fr pigtail catheter was used for drainage of the pericardial fluid. This catheter was removed when the reaccumulation of intrapericardial fluid was <50 ml in 24 h.

Effusions were arbitrarily categorized as (1) hemorrhagic due to perforation (hemoglobin >10 g/dl); (2) exudative/reactive due to pericarditis (hemoglobin <2 g/dl); and (3) intermediate (hemoglobin 2–10 g/dl). Pericardial fluid was aspirated for the measurement of hemoglobin, glucose, total protein, cholesterol, and lactate dehydrogenase; for cytology; and for aerobic and anaerobic bacterial cultures. Bloody effusions did not undergo further analysis of the fluid. Pericardial biopsy was not performed because this technique is not established at Zentralklinik Bad Berka.

Statistical Analysis
All patients with postoperative PE comprised the study group, whereas the control group included all patients who did not develop this complication. Comparisons of continuous variables were made with the appropriate 2-sample test; Student’s t-test in cases of normal distribution of variables, and Kruskal-Wallis test otherwise. Averaged data are reported as mean ± SD or median (interquartile range; 25th–75th percentiles) where appropriate. The Fisher exact test for univariate analysis was used to identify risk factors for PE. Multiple logistic regression analysis was performed to identify variables independently associated with PE following HRD implantation. Gender, BMI, previous cardiac surgery, pulmonary hypertension, use of aspirin/thienopyridines, and implantations performed by cardiology fellows were the predictors included in the model. P ≤ 0.05 was considered statistically significant. Statistical analysis was performed using the SAS System for Windows version 9.2 (SAS Institute, Cary, NC, USA).

Results
During the study period, 995 patients underwent HRD implantation at Zentralklinik Bad Berka. Of them 7 (0.7%) had PE prior to the implantation and were excluded, and another 21 patients (2.1%) did not have successful implantation. Finally, 968 HRD were successfully implanted and underwent analysis. Ninety-eight (10%) of the 968 patients developed a PE postoperatively.

Of the 372 implanted PMs (38.4%), 113 (30.4%) were single chamber, 227 (61%) were dual chamber and 32 (8.6%) were CRT devices. Of the 596 ICDs (61.6%), 357 (59.9%) were single chamber, 227 (45.5%) were dual chamber, and 212 (35.6%) were CRT-D. Overall, 649 patients (69%) had a new lead implanted, while in 319 (31%) a previous device was upgraded and at least 1 new lead was added. Demographic and procedural patient characteristics are listed in Tables 1, 2, respectively.

Small/Moderate PE
A small (≤10 mm) or moderate (11–20 mm in diastole) PE was observed in 80/968 (8.3%) and 4/968 (0.4%), respectively. Only 5/84 (6%) complained of chest pain, whereas most patients (79/84, 94%) with either small or moderate PE were completely asymptomatic. All patients with small/moderate PE were managed conservatively and anti-inflammatory therapy was given only to the 5 symptomatic patients. In none of those patients were the leads either repositioned or removed. All patients with small/moderate PE underwent a second echocardiographic evaluation before discharge: none of the patients
Another 2/14 patients (14%) required resuscitation and underwent emergency percutaneous pericardiocentesis 4.5 h and 2 h after HRD implant, respectively. After initial successful pericardiocentesis (bloody fluid in both cases), 1 patient died due to multiorgan dysfunction syndrome, and the second patient died due to hypoxic–ischemic brain damage after resuscitation.

In the remaining 10/14 patients (71%) fluoroscopy-guided percutaneous pericardiocentesis was sufficient to reach and maintain normal hemodynamic parameters. The effusion was bloody (hemoglobin >10 g/dl) in 8 patients (57%) and exudative (hemoglobin <2 g/dl) in the remaining 4 (29%). There were no effusions with intermediate levels of hemoglobin (2–10 g/dl). A significant larger amount of fluid (575 \pm 50 ml vs. 340 \pm 100 ml; P=0.05) was drained when an exudate was found. We evaluated pericardial fluid obtained from all 4 patients who experienced an increase of the PE.

### Table 1. Potential Clinical Risk Factors for Postoperative PE

<table>
<thead>
<tr>
<th>Age (years) [range]</th>
<th>Group 1: Patients without PE (n=870)</th>
<th>Group 2: Patients with any PE (n=98)</th>
<th>Group 3: Patients with tamponade (n=14)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>68.8±10.8 [41–88]</td>
<td>70.0 [IQR, 64–81]</td>
<td>73.0 [IQR, 62–80]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>304 (35)</td>
<td>48 (49)</td>
<td>8 (57)</td>
<td>0.008+</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>27.2±4.5</td>
<td>25.9±3.9</td>
<td>26.9±4.4</td>
<td>0.01</td>
</tr>
<tr>
<td>Prior cardiac surgery</td>
<td>232 (26.7)</td>
<td>14 (14)</td>
<td>0 (0)</td>
<td>0.015+</td>
</tr>
<tr>
<td>PH</td>
<td>190 (21.8)</td>
<td>20 (20)</td>
<td>8 (57)</td>
<td>0.8</td>
</tr>
<tr>
<td>Renal failure</td>
<td>183 (21)</td>
<td>19 (19)</td>
<td>3 (21)</td>
<td>0.5</td>
</tr>
<tr>
<td>Platelets (×10^9/µl)</td>
<td>233±80</td>
<td>218±69</td>
<td>253±89</td>
<td>0.074</td>
</tr>
</tbody>
</table>

Data given as mean ± SD or n (%). *Significant at multivariate analysis.

### Table 2. Potential Procedural Risk Factors for Postoperative PE

<table>
<thead>
<tr>
<th>Operation time (min)</th>
<th>Group 1: Patients without PE (n=870)</th>
<th>Group 2: Patients with any PE (n=98)</th>
<th>Group 3: Patients with tamponade (n=14)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>51.1±30.8</td>
<td>51±26.2</td>
<td>46±19</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temporary pacing lead</td>
<td>70 (8.1)</td>
<td>6 (6)</td>
<td>2 (14)</td>
<td>0.7</td>
</tr>
</tbody>
</table>

Data given as mean ± SD or n (%). PE, pericardial effusion; RA, right atrium; RV, right ventricle; RVOT, right ventricular outflow tract.

Large PE

Large PEs (>20 mm in diastole) with tamponade pattern were observed in 14/968 patients (1.5%). All patients with echocardiographic evidence of tamponade developed arterial hypotension. Patients with large PEs had a significant increase in the postoperative pacing threshold (0.35±0.65 mV vs. -0.08±0.33 mV; P<0.002). The lead parameters sensing and impedance remained similar to the values obtained at the time of implant. In Table 3 clinical characteristics and indications for HRD implantation of the patients with cardiac tamponade are listed.

Two (14%) of the 14 patients with tamponade underwent emergency surgery due to overt lead-induced perforation of the right ventricle. In both cases (1) the diagnosis was reached with transthoracic echocardiography; (2) percutaneous pericardiocentesis did not suffice to control the bleeding; and (3) lead perforation of the myocardium was confirmed at cardiac surgery.

Another 2/14 patients (14%) required resuscitation and underwent emergency percutaneous pericardiocentesis 4.5 h and 2 h after HRD implant, respectively. After initial successful pericardiocentesis (bloody fluid in both cases), 1 patient died due to multiorgan dysfunction syndrome, and the second patient died due to hypoxic–ischemic brain damage after resuscitation.

In the remaining 10/14 patients (71%) fluoroscopy-guided percutaneous pericardiocentesis was sufficient to reach and maintain normal hemodynamic parameters. The effusion was bloody (hemoglobin >10 g/dl) in 8 patients (57%) and exudative (hemoglobin <2 g/dl) in the remaining 4 (29%). There were no effusions with intermediate levels of hemoglobin (2–10 g/dl). A significant larger amount of fluid (575±50 ml vs. 340±100 ml; P=0.05) was drained when an exudate was found. We evaluated pericardial fluid obtained from all 4 pa-
patients with exudative effusion. Protein concentration ranged from 3.5 to 4.1 g/L (normal range, 1.7–3.5 g/L). Cultures for bacteria were negative, no malignant cells were found.

Moreover, in all 4 patients with exudative PE a pericardial-peritoneal window was created. This delayed surgical treatment was needed due to recurrent effusion with tamponade physiology. At surgical exploration, no evidence of lead-induced perforation was found and 3/4 patients were described as having fibrinous pericarditis.

An atrial helical screw-in lead was implanted in 12 of the 14 patients (86%) with tamponade physiology. The atrial lead was placed at the RA appendage and at the lateral wall of the right atrium in 10 and 2 patients, respectively. There was no significant difference, however, with respect to the incidence of PE depending of lead position or mode of lead fixation (Table 2).

### Univariate Analysis

At univariate analysis, the predictors for any types of PE were: female gender (49% vs. 35%; P=0.0001), and implantation performed by a cardiology fellow in training (10% vs. 1.6%; P=0.01). Previous cardiac surgery (14% vs. 26.7%; P=0.015), and higher BMI (26.9 ± 3.9 vs. 27.2 ± 4.5; P=0.01) were protective factors. At univariate analysis, predictors of PE causing tamponade were: pulmonary hypertension (57% vs. 21.8%; P=0.005), current medication with aspirin/thienopyridines (57% vs. 9.6%; P<0.001), and implantation performed by a cardiology fellow in training (14% vs. 1.6%; P=0.03). Again previous cardiac surgery (0% vs. 26.7%; P=0.03) was a protective factor.

### Multivariate Analysis

At multivariate analysis for all types of PE female gender (hazard ratio [HR], 2.7; 95% confidence interval [CI]: 1.41–3.50, P=0.01) independently predicted PE, whereas previous cardiac surgery (HR, 0.70; 95% CI: 0.50–0.92, P=0.02) was a protective factor. Multivariate analysis including only PE causing tamponade demonstrated that current medication with aspirin/thienopyridines (HR, 3.1; 95% CI: 2.1–3.8, P=0.01) independently predicted PE, whereas previous cardiac surgery (HR, 0.72; 95% CI: 0.51–0.89, P=0.02) was again a protective factor.

| Table 3. Cardiac Tamponade After HRD Implantation: Clinical Patient Characteristics |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Age (years) | Sex | HRD    | Indication     | EE (%) | Mediation | Heart Disease | Duration of Implantation (min) | Time to PE (h) | Clinical Presentation | Therapy | Fluid Volume (ml) | Intraoperative Findings | Outcome |
| 75           | F   | DDD-PM | AVB III°       | 65     | DAPT      | IHD           | 50                                         | 4             | Hypertension       | D          | 300 (b)           |                      | Favorable          |
| 68           | M   | VVI—DDD-PM | SA block     | 47     | OAC       | DCM           | 61                                         | 3.5            | Hypertension       | D          | 420 (b)           |                      | Favorable          |
| 73           | F   | DDD-PM | BND            | 65     | None      | None          | 53                                         | 24             | Dyspnea, echo     | D+OP       | 350 (s)           | Pericarditis          | Favorable          |
| 66           | F   | VVI-ICD→CRT-D | CHF       | 32     | DAPT      | IHD           | 69                                         | 4.5            | Cardiac shock     | D          | 100 (b)           |                      | Death              |
| 80           | M   | DDD-PM | AVB III°       | 50     | DAPT      | IHD           | 47                                         | 20             | Tamponade on echo | D+OP       | 550 (b)           |                      | Favorable          |
| 75           | F   | DDD-PM | AVB III°       | 65     | None      | None          | 63                                         | 5              | Hypertension       | D+OP       | 800 (s)           | Pericarditis          | Favorable          |
| 48           | M   | VVI-ICD | PP            | 30     | DAPT      | IHD           | 35                                         | 1.5            | Hemorrhage         | D+OP       | 0 intrapericardial, 3,000 intrapericardial, 3,000 intrapleural (b) | Lead perforation through an RV diverticulum and perforation into the left pleura | Favorable          |
| 73           | F   | DDD-PM | BND            | 65     | OAC       | None          | 53                                         | 24             | Dyspnea, echo     | D+OP       | 450 (b)           | Pericarditis          | Favorable          |
| 68           | M   | VVI—DDD-PM | Sinus bradycardia | 48     | OAC       | DCM           | 61                                         | 3.5            | Hypertension       | D          | 420 (b)           |                      | Favorable          |
| 75           | F   | DDD-PM | AVB III°       | 65     | DAPT      | IHD           | 50                                         | 4              | Hypertension       | D          | 300 (b)           |                      | Favorable          |
| 79           | F   | DDD-PM | AVB III°       | 65     | None      | None          | 63                                         | 5              | Hypertension       | D+OP       | 700 (s)           | Pericarditis          | Favorable          |
| 66           | F   | CRT-D  | CHF            | 32     | DAPT      | IHD           | 69                                         | 2              | Cardiac shock     | D+OP       | 120 (b)           |                      | Death              |
| 80           | M   | DDD-PM | AVB III°       | 50     | DAPT      | IHD           | 47                                         | 19             | Tamponade on echo | D          | 550 (b)           |                      | Favorable          |
| 52           | M   | VVI-ICD | PP            | 30     | DAPT      | IHD           | 35                                         | 1.5            | Hypertension       | D+OP       | 280 (b)           | Lead perforation     | Favorable          |

AVB, atrioventricular block; b, bloody; BND, binodal disease; CHF, congestive heart failure; CRT, cardiac resynchronization therapy; D, drainage; DAPT, dual anti-platelet therapy; DCM, dilated cardiomyopathy; DDD, dual chamber; EF, ejection fraction; HRD, heart rhythm device; ICD, implantable cardioverter-defibrillator; IHD, ischemic heart disease; OAC, oral anticoagulation; OP, operation; PE, pericardial effusion; PM, pacemaker; PP, primary prevention; RV, right ventricle; s, serous; SA, sinoatrial; SND, sinus node disease; VVI, ventricular single chamber.
Incidence and Predictors of Pericardial Effusion

Follow-up
The mean duration of follow-up was 3.14±0.5 months (cumulative, 273 months). None of the 6 patients undergoing emergency or delayed surgery had recurrence of pericardial tamponade or PE.

Out of the 6 patients with successful pericardiocentesis 1 patient was lost to follow-up and 1 patient was alive but not eligible for repeat echocardiography. The remaining 4 patients had no evidence of recurrent PE on follow-up echocardiography.

Of the 84 patients with moderate or small PEs following HRD implantation, 7 (8%) were lost to follow-up, and 77 (92%) underwent further echocardiography. In 55 of them (71%), complete resolution of the PE could be demonstrated (Figure).

Discussion
This prospective observational study suggests that PE following permanent HRD implantation is much more frequent than previously reported. The only independent predictors of PE in the present series were female gender and antiplatelet therapy. Previous cardiac surgery appears to be protective against postoperative PE.

Incidence of Postoperative PE
Permanent HRD implantation is a common procedure and clinical predictors of PE following implantation have been addressed in several retrospective studies. The incidence of symptomatic PE after permanent device implantation has been reported to be 0.3–2%. All studies conducted on this topic provide limited information about the real incidence of PE following HRD implantation, because only symptomatic (chest pain) or hypotensive patients have been included. We report an incidence of symptomatic (0.5%) or hemodynamically relevant (1.4%) PE of 1.9%, which compares well with previously published data. Nevertheless, an additional 79 (8.1%) asymptomatic patients were found to have PE at post-procedural echocardiography. Specific treatment was not required for any of them.

The utilization of ventricular and especially atrial screw-in leads is believed to be associated with a significantly increased risk of post-procedural PE, and 38–86% of the present patients received at least 1 active fixation lead. It is conceivable that the thin atrial wall is more prone to perforation than the thicker ventricular wall. The screw mechanism also has a higher risk of protruding through the walls and irritates the pericardium or causes low-grade perforation resulting in hemorrhagic effusion or pericarditis. There was no significant difference, however, in the present series with respect to the incidence of PE depending of lead position or mode of lead fixation (Table 2).

Pericarditis vs. True Lead Perforation
Pericarditis following PM implantation was first reported in 1975, and to date only a few reports have been published in the literature. Greene et al reported that pericarditis was observed in 6 of 123 (4.9%) consecutive patients undergoing PM implant, whereas Levy et al reported a lower incidence of 8 of 395 (2%).

A delayed immune-mediated pericarditis with prolonged latent period from cardiac injury to the clinical onset of symptoms, as happens in the classical Dressler’s syndrome (which appears a few weeks after the index procedure), might be
implicated in some cases. Nevertheless, this seems to be an unlikely explanation in the present series given the early development of the PE.

Cardiac perforation is a well-established complication of PM lead implantation and it is diagnosed when (1) the tip of a passive fixation lead or (2) the screw of an active fixation lead reaches the pericardial cavity. The fixation screw may cause pericardial abrasion with consecutive intrapericardial bleeding. Torsion on the visceral pericardium, and subsequent perforation with intermittent oozing of blood into the pericardial space, has also been postulated as a possible mechanism.

Perforation of the myocardium was found on computed tomography in 15 of 100 consecutive asymptomatic patients who had cardiac lead placement several years previously. In addition, 1 report of 111 post-mortem examinations of patients implanted with a PM demonstrated an RA leads perforation rate of 27%.

In the present series, the incidence of perforation (hemorrhagic effusion, hemoglobin >10 g/dl; 10/14, 71%) was higher than the incidence of pericarditis (reactive/exudative effusion, hemoglobin <2 g/dl; 4/14, 29%), but hemorrhagic effusion indicating overt lead perforation remains a speculative statement because echocardiography, the only imaging modality used, lacks sufficient sensibility and specificity to properly diagnose lead induced cardiac perforation.

All patients with acute pericarditis experienced recurrent effusion with tamponade physiology, and ultimately needed surgical treatment with the creation of a pericardial-peritoneal window. In these patients, no evidence of cardiac perforation was found and in almost all patients (3/4) a fibrinous pericarditis was described. The mechanism responsible for acute effusive pericarditis following permanent HRD implantation is unclear and only 10 cases have been reported in the literature since 1975. The underlying mechanisms may involve damage of the mesothelial pericardial cells and accumulation of small amounts of blood in the pericardial space, causing a hypersensitivity reaction to these cardiac antigens. Post-cardiac injury syndrome, however, has also been described in children following orthotopic cardiac transplantation. Because these children were immunosuppressed, it has been suggested this syndrome is not always an autoimmune process. Alternatively, traumatic inflammation extending from the lead screw and traversing through the myocardium to the pericardium is also possible without frank perforation of the screw tip or lead body.

Preprocedural Clinical Variables and Risk of PE

Female gender was associated with a higher incidence of any PE. Female gender correlates in population-based studies with reduced extent of ventricular hypertrophy, and lower RV pressure. This might result in thinner RV walls, which, in turn, could increase the risk of ventricular perforation. Interestingly, however, female adult patients have been shown to be also at higher risk for development of early postoperative PE after open heart surgery. While the explanation remains unclear, female sex may indeed be an independent risk factor.

The majority of patients developing tamponade physiology following HRD implantation were taking some form of antiplatelet medication. Aspirin and thienopyridines are often prescribed for primary or secondary prevention of cardiovascular events such as myocardial infarction or stroke. A recent study demonstrated that the likelihood of developing bleeding complications was doubled in patients undergoing PM or ICD implantation receiving aspirin alone, and quadrupled in those receiving dual antiplatelet therapy.

Prior cardiac surgery in the present series was associated with a lower incidence of post-implant PE. After heart surgery intrapericardial adhesions are frequent and this might be playing a protective role, reducing the likelihood of intrapericardial fluid accumulation and growth. This relationship was also described in a recent large series of patients undergoing ICD implantation.

Clinical Implications

We report an incidence of symptomatic (0.5%) or hemodynamically relevant (1.4%) PE of 1.9%, which compares well with previous published data. Systematic echocardiographic evaluation identified an additional 8.1% (79/968) with either small or moderate PE. None of those patients required specific therapy and most PEs (71%) spontaneously resolved in 3.1±0.5 months. This suggests that echocardiographic evaluation limited to symptomatic or hemodynamically unstable patients would be clinically appropriate.

In addition, previous cardiac surgery seems to grant some protection, whereas particular care should be taken when female patients or patients receiving antiplatelet therapy undergo HRD implantation. Careful supervision should always be given when operators at the beginning of their learning curve participate in HRD implantation.

Moreover, an atrial active fixation lead (either at the RA appendage or at the lateral wall of the right atrium) was frequently used (86%) in patients who subsequently developed large PE with tamponade physiology. This suggests caution when screw-in leads are used in the right atrium, where the thickness of the myocardium is limited.

Study Limitations

The aim of the study was to prospectively evaluate the incidence and clinical characteristics of PE acutely following HRD implantation. No specific effort was made to identify PE occurring late after device placement. Therefore, patients without PE within 24 h were not scheduled for long-term echocardiographic or clinical follow-up. For this reason the true incidence of PE might be even higher than that stated in this paper.

Acknowledgment

None of the authors have any conflict of interest to declare.

References

Incidence and Predictors of Pericardial Effusion


