Risk Factors for Mortality in Patients With Cardiac Device-Related Infection

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**Background**—Because of the increased use of pacemakers and implantable cardioverter defibrillators, infection has become a complication with significant morbidity and mortality. Data on risk factors for mortality in patients with cardiac-device-related infection are limited. We evaluated the prognostic significance of key clinical and echocardiographic variables in a large retrospective population of patients with cardiac-device related infection.

**Methods and Results**—Two hundred ten patients with cardiac-device related infection were identified at the University of Michigan between 1995 and 2006. Data were abstracted on key clinical and echocardiographic variables, treatment strategy, and 6-month outcomes. We used multivariable Cox proportional hazards models to examine clinical and echocardiographic variables that were associated with 6-month mortality. Mean age for our study population was 63±17 years, and 72 (44%) were women. All-cause 6-month mortality was 18% (n=37). Independent variables associated with death were systemic embolization (hazard ratio 7.11; 95% CI 2.74 to 18.48), moderate or severe tricuspid regurgitation (hazard ratio 4.24; 95% CI 1.84 to 9.75), abnormal right ventricular function (hazard ratio 3.59; 95% CI 1.57 to 8.24), and abnormal renal function (hazard ratio 2.98; 95% CI 1.17 to 7.59). Size and mobility of cardiac device vegetations were not independently associated with mortality.

**Conclusions**—We identified several clinical and echocardiographic variables that identify patients with cardiac-device related infection who are at high-risk for mortality and may benefit from more aggressive evaluation. ([Circ Arrhythmia Electrophysiol. 2009;2:129-134.])

**Key Words:** infection • risk factors • mortality • outcome assessment

Implantation of electrophysiological cardiac devices such as pacemakers and implantable cardioverter defibrillators (ICDs) has become a widely available and routine procedure in cardiovascular medicine. Numerous trials have shown that pacemakers,1 biventricular pacemakers,2–3 and ICDs4–7 decrease mortality and improve quality of life. A growing list of indications, in conjunction with an aging population, projects that even a greater number of patients will acquire such devices in the future.

**Clinical Perspective** see p 134

One of the most feared complications of device placement is infection, which can be associated with substantial morbidity and mortality. Infection rates for these devices reportedly vary from 0.7% to 7.0%8–12 with a resultant 3.1-fold increase in the number of associated hospitalizations in recent years.13 Mortality rates attributable to infection have ranged from 2.6% to 3.3%,14–15 Although recent reports have identified several clinical characteristics associated with developing cardiac device-related infection (CDI), there are limited data on outcomes after treatment. The purpose of the present study was to determine risk factors associated with mortality in a large study population of patients with CDI.

**Methods**

A retrospective chart review was conducted after identifying all hospitalized patients with a discharge diagnosis of pacemaker or ICD infection (International Classification of Diseases-9th Revision-Clinical Modification [ICD-9-CM] code 996.61) at the University of Michigan between 1995 and 2006. To ensure our search for these patients was as exhaustive as possible, we also screened the medical records of any patients with device explantation (ICD-9-CM codes 37.77, 37.79, 37.89, or 37.99) and a discharge diagnosis of sepsis (ICD-9-CM code 038 or 785.59), bacteremia (ICD-9-CM code 790.7), endocarditis (ICD-9-CM codes 421.0, 421.9, or 424.90), cellulitis (ICD-9-CM code 682.9), or fever (ICD-9-CM code 780.6) to identify additional cases. A total of 1163 patients were identified through this database search with 210 noted as having CDI. The Institutional Review Board of the University of Michigan approved this study.

Demographic, clinical, and echocardiographic data were collected from the University of Michigan electronic medical record. The demographic information consisted of age, gender, and length of hospital stay. Clinical variables of interest included evidence of local infection at the generator site, bacteremia, microbiological information, presence of diabetes, coronary artery disease, coronary artery bypass surgery, hypertension, presence of HIV, steroid use, presence of central line, renal dysfunction, pulmonary embolism, and systemic embolization. Information on the type, age, and location of device was also collected. Echocardiographic variables included presence and description of vegetations, left and right ventricular (RV) function, valvular endocarditis, valvular regurgitation, and estimation of pulmonary artery pressure via RV systolic pressure.
Definitions
CDI has been previously defined by other groups.12,16 The Duke criteria17 for the diagnosis of endocarditis were applied to systemic infections related to cardiac devices. Clinical evidence of pacemaker/ICD infection included local signs of inflammation at the generator site such as erythema, warmth, fluctuance, wound dehiscence, erosion, tenderness, or purulent drainage. Device related endocarditis was clinically confirmed if valvular or lead vegetations were detected by echocardiography or if the Duke criteria for infective endocarditis were met.18 The diagnosis of systemic embolization including cerebral, renal, or spinal embolic infarction was based on high clinical suspicion as well as data derived from diagnostic procedures.

Echocardiography
An experienced echocardiographer independently reviewed all the transthoracic and transesophageal echocardiography studies without knowledge of the patient’s clinical history or subsequent clinical course. Vegetation was defined as a fixed or oscillating echodensity adherent to a lead, valve leaflet, or other cardiac structure with independent motion. The lesion had to be visible in multiple views. The measurement of the vegetation was obtained in different planes and the maximal length was used. In the presence of multiple vegetations, the largest diameter was included in the analysis. The mobility of the vegetation was evaluated using a 4-point scale adopted from valvular endocarditis analysis: absent=fixed vegetation with no detectable independent motion; low=mobilization with a fixed base but with a mobile free edge; moderate=pedunculated vegetation that remains within the same chamber throughout the cardiac cycle; severe=prolapsing vegetation that crosses the coaptation plane of the leaflets during the cardiac cycle.19 Severity of valvular regurgitation was assessed using the standard guideline recommendations from the American Society of Echocardiography.20

End Points
The primary end point was death from all causes at 6 months. Death status was ascertained in all patients by querying medical records and the Social Security Death Index. Cause of mortality was determined by review of medical record or through the National Death Index.21,22 For each patient submitted to the National Death Index a match was made based on name, Social Security number, date of birth, first and last names, and sex. A match was generated if the patient resided in the United States at the time of death and the Social Security死亡 index determined the cause of death. Such a match was considered a true match.

A secondary end point of pulmonary embolism confirmed by diagnostic imaging during hospitalization was also used.

Statistical Analysis
Continuous variables were expressed as mean±SD, and categorical variables were expressed as frequencies. Six-month survival was estimated by Kaplan–Meier method using size of lead vegetations as a potential predictor of death. Clinical and echocardiographic variables were tested as independent predictors of 6-month all-cause mortality using Cox proportional hazards analysis after controlling for age, sex, and treatment method (surgical removal versus percutaneous removal versus medical management). Variables significantly associated with 6-month mortality were included as candidate predictors in a multivariable analysis using Cox proportional hazards model with stepwise forward regression (entry criterion $P\leq0.10$).

Table 1. Unadjusted Hazard Ratios for 6-Month Mortality Associated With Age, Sex, and Treatment Group

<table>
<thead>
<tr>
<th>Total (n=210)</th>
<th>Survived (n=173)</th>
<th>Died (n=37)</th>
<th>HR</th>
<th>95% CI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>63±17</td>
<td>61±17</td>
<td>71±11</td>
<td>1.04</td>
<td>1.01–1.06</td>
</tr>
<tr>
<td>Male gender</td>
<td>138 (66)</td>
<td>112 (65)</td>
<td>26 (70)</td>
<td>1.56</td>
<td>2.91–3.45</td>
</tr>
<tr>
<td>Treatment option</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical management</td>
<td>23 (11)</td>
<td>15 (9)</td>
<td>8 (22)</td>
<td>1.00</td>
<td>. . . (reference)</td>
</tr>
<tr>
<td>Percutaneous device removal</td>
<td>170 (81)</td>
<td>142 (82)</td>
<td>28 (76)</td>
<td>0.44</td>
<td>0.20–0.96</td>
</tr>
<tr>
<td>Cardiac surgical removal</td>
<td>17 (8)</td>
<td>16 (9)</td>
<td>1 (3)</td>
<td>0.15</td>
<td>0.02–1.19</td>
</tr>
</tbody>
</table>

Values are n (%) or mean±SD.

Outcomes
Six-month all-cause mortality in our study was 18% (n=37). Of this group, 17 patients had in-hospital mortality with major causes of death including sepsis (76%) and cardiac arrest (26%). With regards to all-cause 6-month mortality, 73% (n=27) were secondary to cardiovascular causes. Failure to thrive or hospice care accounted for 14% (n=5) of deaths. Noncardiovascular causes of mortality accounted for the remaining 14% (n=5).

Univariable Predictors of 6-Month Mortality

Table 1 shows unadjusted hazard ratios for 6-month mortality using the clinical characteristics of age, sex, and treatment method.
Table 2. Adjusted Hazard Ratios for 6-Month Mortality Associated With Patient Characteristics and Controlling for Age, Sex, and Treatment Group

<table>
<thead>
<tr>
<th>Type of device</th>
<th>Total (n=210)</th>
<th>Survived (n=173)</th>
<th>Died (n=37)</th>
<th>HR</th>
<th>95% CI</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pacemaker</td>
<td>126 (60)</td>
<td>103 (60)</td>
<td>23 (62)</td>
<td>1.00</td>
<td>(reference)</td>
<td></td>
</tr>
<tr>
<td>ICD</td>
<td>69 (33)</td>
<td>59 (34)</td>
<td>10 (27)</td>
<td>0.93</td>
<td>0.47–2.41</td>
<td>0.88</td>
</tr>
<tr>
<td>Biventricular PM/ICD</td>
<td>15 (7)</td>
<td>10 (6)</td>
<td>5 (14)</td>
<td>1.53</td>
<td>0.56–5.44</td>
<td>0.47</td>
</tr>
<tr>
<td>Previous device upgrade/replacement</td>
<td>38 (18)</td>
<td>34 (20)</td>
<td>4 (11)</td>
<td>1.37</td>
<td>0.48–3.94</td>
<td>0.56</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical variables</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Diabetes</td>
<td>57 (27)</td>
<td>45 (26)</td>
<td>12 (32)</td>
<td>1.59</td>
<td>0.78–3.22</td>
<td>0.20</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>110 (52)</td>
<td>83 (48)</td>
<td>27 (73)</td>
<td>2.12</td>
<td>1.10–5.00</td>
<td>0.07</td>
</tr>
<tr>
<td>CABG</td>
<td>51 (24)</td>
<td>42 (24)</td>
<td>9 (24)</td>
<td>0.81</td>
<td>0.37–1.74</td>
<td>0.58</td>
</tr>
<tr>
<td>Hypertension</td>
<td>137 (65)</td>
<td>106 (61)</td>
<td>31 (84)</td>
<td>2.20</td>
<td>0.90–5.36</td>
<td>0.08</td>
</tr>
<tr>
<td>Abnormal creatinine &gt;1.5 mg/dl</td>
<td>99 (47)</td>
<td>70 (40)</td>
<td>29 (78)</td>
<td>3.99</td>
<td>1.71–9.30</td>
<td>≤0.01</td>
</tr>
<tr>
<td>Positive blood culture</td>
<td>108 (51)</td>
<td>77 (45)</td>
<td>31 (84)</td>
<td>6.05</td>
<td>2.30–15.97</td>
<td>≤0.01</td>
</tr>
<tr>
<td>Positive device culture</td>
<td>147 (70)</td>
<td>120 (69)</td>
<td>27 (73)</td>
<td>1.52</td>
<td>0.62–3.75</td>
<td>0.37</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Echocardiographic variables</th>
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<tbody>
<tr>
<td>Lead vegetation visualized</td>
<td>48 (23)</td>
<td>37 (21)</td>
<td>11 (30)</td>
<td>1.47</td>
<td>0.69–3.15</td>
<td>0.32</td>
</tr>
<tr>
<td>Lead vegetation size</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent/not obtained</td>
<td>168 (80)</td>
<td>139 (81)</td>
<td>29 (78)</td>
<td>1.00</td>
<td>. . .</td>
<td>(reference)</td>
</tr>
<tr>
<td>1–10 mm</td>
<td>10 (5)</td>
<td>9 (5)</td>
<td>1 (3)</td>
<td>0.84</td>
<td>0.11–6.21</td>
<td>0.86</td>
</tr>
<tr>
<td>11–20 mm</td>
<td>19 (9)</td>
<td>13 (8)</td>
<td>6 (16)</td>
<td>2.05</td>
<td>0.81–5.23</td>
<td>0.13</td>
</tr>
<tr>
<td>≥21 mm</td>
<td>13 (6)</td>
<td>11 (6)</td>
<td>2 (5)</td>
<td>1.03</td>
<td>0.23–4.73</td>
<td>0.97</td>
</tr>
<tr>
<td>Lead vegetation mobility</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Absent/not obtained</td>
<td>169 (80)</td>
<td>139 (80)</td>
<td>30 (81)</td>
<td>1.00</td>
<td>. . .</td>
<td>(reference)</td>
</tr>
<tr>
<td>Low</td>
<td>16 (8)</td>
<td>12 (7)</td>
<td>4 (11)</td>
<td>1.65</td>
<td>0.56—4.83</td>
<td>0.36</td>
</tr>
<tr>
<td>Moderate</td>
<td>13 (6)</td>
<td>12 (7)</td>
<td>1 (3)</td>
<td>0.37</td>
<td>0.05–2.74</td>
<td>0.33</td>
</tr>
<tr>
<td>Severe</td>
<td>12 (6)</td>
<td>9 (5)</td>
<td>3 (8)</td>
<td>2.04</td>
<td>0.54–7.67</td>
<td>0.29</td>
</tr>
<tr>
<td>Left ventricular dysfunction</td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>93 (44)</td>
<td>79 (46)</td>
<td>14 (38)</td>
<td>1.00</td>
<td>. . .</td>
<td>(reference)</td>
</tr>
<tr>
<td>Mild</td>
<td>14 (7)</td>
<td>13 (8)</td>
<td>1 (3)</td>
<td>0.43</td>
<td>0.06–3.33</td>
<td>0.42</td>
</tr>
<tr>
<td>Moderate</td>
<td>22 (10)</td>
<td>17 (10)</td>
<td>5 (14)</td>
<td>1.33</td>
<td>0.47–3.77</td>
<td>0.59</td>
</tr>
<tr>
<td>Severe</td>
<td>42 (20)</td>
<td>30 (17)</td>
<td>12 (32)</td>
<td>1.68</td>
<td>0.75–3.74</td>
<td>0.21</td>
</tr>
<tr>
<td>Abnormal right ventricular function</td>
<td>35 (17)</td>
<td>22 (13)</td>
<td>13 (35)</td>
<td>4.22</td>
<td>1.91–9.29</td>
<td>≤0.01</td>
</tr>
<tr>
<td>Moderate to severe tricuspid regurgitation</td>
<td>20 (10)</td>
<td>9 (5)</td>
<td>11 (30)</td>
<td>5.93</td>
<td>2.73–12.89</td>
<td>≤0.01</td>
</tr>
<tr>
<td>Right ventricular systolic pressure (RVSP)</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>RVSP not obtained</td>
<td>69 (33)</td>
<td>60 (35)</td>
<td>9 (24)</td>
<td>1.00</td>
<td>. . .</td>
<td>(reference)</td>
</tr>
<tr>
<td>10–39 mm Hg</td>
<td>52 (25)</td>
<td>46 (27)</td>
<td>6 (16)</td>
<td>0.74</td>
<td>0.26–2.09</td>
<td>0.57</td>
</tr>
<tr>
<td>40–59 mm Hg</td>
<td>35 (17)</td>
<td>23 (13)</td>
<td>12 (32)</td>
<td>2.19</td>
<td>0.88–5.44</td>
<td>0.09</td>
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<tr>
<td>≥60 mm Hg</td>
<td>15 (7)</td>
<td>9 (5)</td>
<td>6 (16)</td>
<td>4.40</td>
<td>1.48–13.02</td>
<td>0.01</td>
</tr>
<tr>
<td>Aortic stenosis</td>
<td>7 (3)</td>
<td>5 (3)</td>
<td>2 (5)</td>
<td>0.86</td>
<td>0.45–1.63</td>
<td>0.63</td>
</tr>
<tr>
<td>Aortic insufficiency</td>
<td>9 (4)</td>
<td>7 (4)</td>
<td>2 (5)</td>
<td>1.07</td>
<td>0.56–2.02</td>
<td>0.85</td>
</tr>
<tr>
<td>Moderate to severe mitral regurgitation</td>
<td>31 (15)</td>
<td>19 (11)</td>
<td>12 (32)</td>
<td>1.61</td>
<td>1.12–2.31</td>
<td>≤0.01</td>
</tr>
<tr>
<td>Prosthetic valve</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aortic</td>
<td>9 (4)</td>
<td>7 (4)</td>
<td>2 (5)</td>
<td>1.21</td>
<td>0.12–7.02</td>
<td>0.81</td>
</tr>
<tr>
<td>Mitral</td>
<td>6 (3)</td>
<td>5 (3)</td>
<td>1 (3)</td>
<td>1.08</td>
<td>0.09–13.36</td>
<td>0.94</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical events</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary embolism</td>
<td>9 (4)</td>
<td>5 (3)</td>
<td>4 (11)</td>
<td>3.76</td>
<td>1.25–11.30</td>
<td>0.02</td>
</tr>
<tr>
<td>Systemic embolization</td>
<td>23 (11)</td>
<td>10 (6)</td>
<td>13 (35)</td>
<td>9.02</td>
<td>3.95–20.57</td>
<td>≤0.01</td>
</tr>
</tbody>
</table>

PM indicates pacemaker; CABG, coronary artery bypass graft; RVSP, right ventricular systolic pressure.
Values are n (%) or mean±SD.
Multivariable Predictors of Six-Month Mortality and Pulmonary Embolism

From univariate analysis, the factors associated with an increased 6-month mortality include age (P≤0.01), presence of positive blood culture (P≤0.01), abnormal renal function (P≤0.01), moderate or severe tricuspid regurgitation (P≤0.01), abnormal RV function (P≤0.01), an RV systolic pressure ≥60 mm Hg (P=0.01), moderate to severe mitral regurgitation (P≤0.01), pulmonary embolism (P=0.02), and systemic embolization (P≤0.01; Table 2). Including these factors in a multivariable analysis that also adjusted for age, gender, and treatment group, we found systemic embolization (P≤0.01), moderate or severe tricuspid regurgitation (P≤0.01), abnormal RV function (P≤0.01), and abnormal renal function (P=0.02) to remain independently associated with worse 6-month survival (Table 3). Tricuspid regurgitation remained an independent predictor even when abnormal RV function was included in the model (P≤0.01). The proportionality assumption was verified for all variables in the multivariate model by testing for interaction with time.

In our cohort of 210 patients, 9 (4%) had documentation of pulmonary embolism. Neither lead vegetation size (P=0.89) nor mobility (P=0.31) was found to correlate with an increased risk for pulmonary embolism (Table 4).

**Discussion**

With expanding indications for cardiac device implantation, many physicians are witnessing a rapid increase in the incidence of infection rates. In agreement with the study recently published by Sohail et al,16 the majority of patients with CDI in our cohort presented with evidence of infection limited only to the generator site. It must be reemphasized that such a benign clinical presentation can lead to an outcome of substantial morbidity and mortality.

| Table 3. Independent Variables Associated With 6-Month Mortality* |
|-------------------------|------------------|------------------|
|                        | HR               | 95% CI           | P Value |
| Systemic embolization   | 7.11             | 2.74–18.48       | <0.01   |
| Moderate or severe tricuspid regurgitation | 4.24       | 1.84–9.75       | <0.01   |
| Abnormal right ventricular function | 3.59       | 1.57–8.24       | <0.01   |
| Abnormal renal function (Cr >1.5 mg/dl) | 2.98   | 1.17–7.59   | 0.02  |

*All final models controlled for age, sex, and treatment group as well as other variables listed in the table.

| Table 4. Predictors of Pulmonary Embolism (Logistic Regression Analysis)* |
|-------------------------|------------------|------------------|
|                        | Pulmonary Embolism (n=9) | No Pulmonary Embolism (n=201) | OR       | 95% CI          | P Value |
| Lead vegetation size   |                      |                      |         |                |         |
| 0–10 mm                | 6 (67)             | 172 (86)            | 1.00    | ... (reference) |         |
| >10 mm                 | 3 (33)             | 29 (14)             | 0.88    | 0.14–5.42       | 0.89    |
| Lead vegetation mobility |                      |                      |         |                |         |
| Absent to low          | 6 (67)             | 179 (89)            | 1.00    | ... (reference) |         |
| Moderate to severe     | 3 (33)             | 22 (11)             | 2.38    | 0.45–12.48      | 0.31    |

*All final models controlled for age, sex, and treatment group as well as other variables listed in the table.
The findings of this retrospective analysis, which spans an 11-year period, highlight the mortality accompanying CDI (18% at 6 months) and potential risk factors associated with increased mortality in patients with infected cardiac devices. Importantly, readily available clinical and echocardiographic predictors do exist and allow clinicians to stratify patients who are at highest risk for death and may play a role in determining treatment strategies.

Several clinical characteristics have previously been identified as predictors for initially developing CDI. The most comprehensive study was recently published by Klug et al. This study identified febrile illness before implantation, use of temporary pacing before implantation, and early reintervention as factors associated with a higher probability of infection. Other studies have shown that long-term corticosteroid use and the presence of more than 2 pacing leads were predictors of a higher risk of infectivity. Finally Bloom et al established that diabetes mellitus, renal disease, and congestive heart failure were also associated with the occurrence of device infection. None of these studies examined the correlation of clinical risk factors and outcome after treatment for CDI.

Accordingly, to the best of our knowledge, this study is the first to evaluate the association of clinical and echocardiographic risk factors with subsequent mortality after treatment. We have shown that independent predictors of mortality in patients with infected cardiac devices include systemic embolization, moderate to severe tricuspid regurgitation, right ventricle dysfunction, and abnormal creatinine. Abnormal renal function appears to be a risk factor for initial device infection as well as a predictor for mortality.

Echocardiographic findings of right ventricular dysfunction, moderate to severe tricuspid regurgitation, and increased RV systolic pressure can be associated with elevated pulmonary artery pressures and right-sided failure secondary to pulmonary embolism. In our cohort of patients, only a small number had clinically significant pulmonary embolism when confirmed by appropriate testing (4%). However, a study performed by Klug and others showed that the majority of patients with pacemaker infection and concurrent pulmonary embolism displayed no clinical symptoms. The results of our study also raise the concern whether a larger number of patients may have had underlying silent pulmonary embolism leading to RV dysfunction and significant tricuspid regurgitation, which were independent factors associated with increased 6-month mortality. Consequently, one may question whether we should be more aggressively pursuing this diagnosis in the CDI population.

Moreover, our results show that evidence of right-sided failure and pulmonary embolism correlate with poor survival. When reviewing the literature regarding right-sided endocarditis and subsequent risk for mortality, Hecht et al as well as other studies have shown that size of valvular vegetations is associated with significant 1-year mortality. In contrast, size and mobility of lead vegetations in our CDI population are not independent predictors of 6-month survival (Figure) or pulmonary embolism (Table 4). Previous data on pacemaker infections confirm that there is no statistical difference in lead vegetation size between patients with and without pulmonary embolism. One hypothesis may be that mortality and morbidity attributable to increased embolic risk is associated with factors such as antiphospholipid antibodies, coagulation parameters, and endothelial cell activation, rather than the echocardiographic characteristics of the lead vegetations themselves.

Finally, because of small sample size in the surgical and medical management groups, we were unable to confirm whether a particular method of treatment has an effect on mortality. In addition, examining this impact can be challenging given the high level of selection bias in determining treatment options.

This study has the following limitations. First, it is an observational and retrospective analysis. Our ability to adjust for all the critical variables associated with mortality is limited by the documentation available in the medical records that were abstracted. Second, our study was limited to a single large tertiary-care center with an extensive referral base for electrophysiological services. Similar to other studies, our CDI population was predominately composed of pacemakers instead of ICDs and cardiac resynchronization devices which currently account for the majority of device implantations. Our findings therefore may not be generalizable to other settings or institutions. Finally, data from transesophageal echocardiography were available in only 63% of patients where the study was determined to be clinically indicated. Although this may have underestimated the true frequency of some echocardiographic findings (such as vegetations), we had surface echocardiograms available in the great majority of patients.

In summary, we found 4 parameters that were independent predictors of mortality in patients with CDI: systemic embolization, moderate to severe tricuspid regurgitation, abnormal right ventricular function, and abnormal renal function. These findings will require confirmation in additional future studies, but suggest that patients with CDI who are at high-risk for death after treatment may be identified and managed with more aggressive evaluation.

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Disclosures
None.

References
Because of the increased use of pacemakers and implantable cardioverter defibrillators in medical practice, cardiac device infection (CDI) has become a complication with significant mortality. Although recent studies have identified clinical characteristics for developing CDI, risk factors for mortality have yet to be determined. The purpose of the present study was to establish risk factors associated with mortality in a large study population of 210 patients with CDI. In our cohort, 6-month all-cause mortality was 18% with major causes of death including sepsis and cardiac arrest. After adjusting for age, sex, and treatment strategy, independent variables associated with death were systemic embolization, moderate or severe tricuspid regurgitation, abnormal right ventricular function, and abnormal renal function. Interestingly, size and mobility of device vegetation were not clinical predictors of increased mortality. The presence of these clinical risk factors target patients with CDI who are at high-risk for mortality and may benefit from more aggressive evaluation.
Risk Factors for Mortality in Patients With Cardiac Device-Related Infection
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