Coronary artery compression from epicardial leads: More common than we think

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BACKGROUND A child with an epicardial pacemaker presented with sudden death at our institution, secondary to coronary artery compression. This case prompted enhanced surveillance of all patients with epicardial pacing or defibrillation systems.

OBJECTIVE The purpose of this study was to determine the incidence of coronary artery compression and the diagnostic yield of catheter angiography (CA) and computed tomography (CT).

METHODS All patients with epicardial leads who underwent CA or cine CT were retrospectively reviewed. A patient with postmortem diagnosis of coronary compression was also included.

RESULTS Coronary compression was noted in 8 of 145 patients (5.5%) with epicardial leads. Median age at diagnosis was 11.4 years (range 3.0–29.6 years). Six patients had compression noted by CA, 6 by CT, and 1 by postmortem analysis. Chest radiography had sensitivity and specificity of 57% and 96%, respectively; CT 100% and 93%, respectively; and CA 86% and 100%, respectively. There was no difference in age or weight at the time of lead implant between those who did and those who did not have compression (age: median 1.3 vs 2.4 years; P = .36; weight: 9.5 vs 11.8 kg; P = .46). Among those with coronary compression, 6 of 8 (75%) had symptoms (1 sudden death, 3 chest pain [2 with associated troponin leak], 2 unexplained fatigue). Seven patients had surgical repositioning of their lead.

CONCLUSION We found a higher incidence of coronary artery compression by epicardial leads (5.5%) than previously reported in the literature. Chest radiography can serve as a good surveillance tool, with cine CT scans considered in those with concerning radiographs or with symptoms. Confirmatory CA can be performed before surgical intervention.

KEYWORDS Congenital heart disease; Coronary artery; Defibrillator; Epicardial lead; Pacemaker; Pediatrics; Sudden death

Introduction

Pediatric and congenital cardiac patients in need of a pacemaker or defibrillator may require the placement of epicardial leads due to small body size or anatomic limitations. Complications from epicardial leads are well documented, but cardiac strangulation resulting in myocardial and/or coronary artery compression is thought to be rare. However, its true incidence may be higher because of a lack of either awareness or reporting in the literature. More stringent screening was instituted at our center after a child presented with sudden death secondary to cardiac strangulation from an epicardial pacing system.

The purpose of this study was to determine the incidence of coronary artery compression from epicardial leads in patients who had coronary imaging performed by catheter angiography or computed tomography (CT) after placement of an epicardial pacemaker or defibrillator. The utility of chest radiography (CXR), catheter angiography, and CT in the diagnosis of coronary artery compression was also evaluated.

Methods

This study was approved by the institutional review board at Boston Children’s Hospital. All patients with epicardial leads were retrospectively reviewed and included in the study if they had undergone catheter angiography (January 2000–June 2017) or ECG-gated cine CT (January 2013–June 2017) after epicardial lead implantation. Coronary imaging in these patients was performed as part of routine care for their underlying heart disease or because of symptoms or CXR concerning for coronary artery compression. One patient with an ungated CT and 1 patient diagnosed with coronary artery compression on autopsy were also included in the cohort.

Patient characteristics were recorded, including the presence of structural heart disease, indication for pacing, type of pacing or defibrillation system placed, and demographics at the time of implant, catheter angiography, and/or CT.
CXR (posteroanterior [PA] and lateral) performed before catheter angiography or CT was reviewed, evaluating the epicardial lead position in relation to the cardiac silhouette. Patients with the “classic pattern” of cardiac strangulation, as previously defined by Carreras et al\(^1\) as a heart-shaped orientation of the atrial and ventricular leads within the cardiac silhouette on PA film, were noted.

Patients with suspicious chest X-ray films and those with symptoms suspicious for coronary artery compression, such as chest pain, fatigue, or ventricular dysfunction, underwent a CT scan. Coronary artery compression was suspected if the coronary artery passed under an epicardial lead, with indentation of the coronary or surrounding myocardium by the lead. All patients with suspicious CT images underwent catheter angiography to confirm the diagnosis. Coronary artery compression at angiography was defined as thinning or loss of contrast within the coronary as it passed under the epicardial lead.

### Statistical analysis

Results are summarized as median (range) or number (percent). Subjects with and those without coronary artery compression were compared using the Fisher exact test for categorical variables and the unpaired t test or Wilcoxon rank-sum test for continuous variables.

### Results

Over the study period, a total of 145 patients met our inclusion criteria. Patient demographics and clinical characteristics are summarized in Table 1. Median age of the cohort at the time of their most recent coronary imaging was 8.7 years (0.3–48.6 years). Coronary artery compression was noted in 8 patients (5.5%). The diagnosis of coronary artery compression was made by catheter angiography in 6 patients, CT in 6, and post-mortem analysis in 1. Comparing patients with and without coronary artery compression, there was no difference in age or weight at the time of lead implant (age: median 1.3 vs 2.4 years; \(P = .36\); weight: 9.5 vs 11.8 kg; \(P = .46\)).

The majority of patients had structural heart disease (137 [94.5%]); the remainder had congenital complete heart block in 2, long QT syndrome in 2, idiopathic ventricular tachycardia in 1, and cardiac tumor in 1. Patients predominantly were ventricular paced (including single-chamber, dual-chamber, and biventricular pacemakers) (81%), reflecting the principal indication for epicardial leads in this cohort was high-grade heart block (70.3%).

### Coronary artery compression

Characteristics of the patients with coronary artery compression are summarized in Table 2. One patient (Patient 8) presented with sudden death, 3 with chest pain (2 of whom had concurrent troponin leaks), 2 with unexplained fatigue, and 2 had no symptoms. One had moderate systemic ventricular dysfunction on transthoracic echocardiography at the time of coronary compression diagnosis but no regional wall-motion abnormalities.

Five of the leads causing compression were coursing posteriorly behind the ventricle, 2 of which were abandoned with no functional pacemaker attached. Three leads (2 atrial, 1 ventricular) were more anterior in appearance but still caused coronary artery compression. The coronary most likely to be compressed was the left anterior descending in 5, followed by the circumflex artery in 3 and the right coronary in 1. One patient had compression of both the circumflex and left anterior descending by an abandoned posterior ventricular lead.

### Epicardial leads

Patients had 1–6 epicardial leads present. There was no difference in the number of epicardial leads between those who did (median 2 leads, range 1–3) and those who did not (median 2 leads, range 1–6) have compression (\(P = .24\)). The majority of the leads (n = 257 [77%]) were bipolar sew-on leads (Medtronic 4968, Minneapolis, MN). Twenty-four leads (7%) were unipolar sew-on leads (Medtronic 4965), and 39 (11%) were unipolar screw-in leads (Medtronic 5071, Boston Scientific 4315, Boston Scientific 4316, Malborough, MA). Six patients had unipolar leads adapted together into a bipolar configuration; none of these patients had coronary compression. Nine leads (3%) were implantable cardioverter–defibrillator coils (Medtronic 6996SQ, Medtronic 6931, Boston Scientific 0180, Boston Scientific 0185), and the remainder were transvenous pacing leads placed in epicardial locations (Medtronic 4023, Medtronic 4068, Boston Scientific 4269). Lead characteristics did not correlate with coronary compression (Table 3).
<table>
<thead>
<tr>
<th>Pt no.</th>
<th>Age at lead implant (yrs)</th>
<th>Weight at lead implant (kg)</th>
<th>Age at diagnosis of coronary artery compression (yrs)</th>
<th>Structural heart disease (Y/N)</th>
<th>Rhythm disorder</th>
<th>Symptoms</th>
<th>Systemic ventricular function (echo)</th>
<th>Suspected coronary compression</th>
<th>Lead causing compression (model no.)</th>
<th>Coronary compressed</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5.4</td>
<td>16.9</td>
<td>6.1</td>
<td>Y</td>
<td>High-grade block</td>
<td>Chest pain + troponin leak</td>
<td>Normal</td>
<td>+/- + -</td>
<td>Atrial (4968-25 cm)</td>
<td>Right coronary</td>
<td>Surgery</td>
</tr>
<tr>
<td>2</td>
<td>0.4</td>
<td>7.1</td>
<td>4.7</td>
<td>Y</td>
<td>Complete heart block</td>
<td>Fatigue</td>
<td>Normal</td>
<td>- +</td>
<td>Posterior ventricular (4968-25 cm)</td>
<td>Left anterior descending</td>
<td>Surgery</td>
</tr>
<tr>
<td>3</td>
<td>4.9</td>
<td>15.5</td>
<td>12.9</td>
<td>Y</td>
<td>Sinus node dysfunction</td>
<td>Fatigue</td>
<td>Moderate dysfunction</td>
<td>+ + +</td>
<td>Atrial (4968-35 cm)</td>
<td>Circumflex</td>
<td>Surgery</td>
</tr>
<tr>
<td>4</td>
<td>1.7</td>
<td>11.5</td>
<td>9.9</td>
<td>N</td>
<td>Ventricular tachycardia</td>
<td>Chest pain</td>
<td>Normal</td>
<td>+ + +</td>
<td>Posterior coil (0180-59 cm)</td>
<td>Obtuse marginal</td>
<td>Surgery</td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>3.4</td>
<td>29.6</td>
<td>Y</td>
<td>Complete heart block</td>
<td>None</td>
<td>Normal</td>
<td>+ + +</td>
<td>Abandoned posterior ventricular (4315)</td>
<td>Left anterior descending, circumflex</td>
<td>Surgery</td>
</tr>
<tr>
<td>6</td>
<td>0.9</td>
<td>7.5</td>
<td>3.0</td>
<td>Y</td>
<td>Complete heart block</td>
<td>None</td>
<td>Normal</td>
<td>- + +</td>
<td>Anterior ventricular (4968-25 cm)</td>
<td>Left anterior descending</td>
<td>Surgery</td>
</tr>
<tr>
<td>7</td>
<td>10.8</td>
<td>25.0</td>
<td>20.3</td>
<td>N</td>
<td>LQTS</td>
<td>Chest pain + troponin leak</td>
<td>Normal</td>
<td>+ + +</td>
<td>Posterior coil (6931-52 cm)</td>
<td>Diagonal branch off LAD</td>
<td>Surgery</td>
</tr>
<tr>
<td>8</td>
<td>0.01</td>
<td>3.5</td>
<td>13.0</td>
<td>N</td>
<td>Complete heart block</td>
<td>Sudden death</td>
<td>Postmortem diagnosis</td>
<td>Abandoned posterior ventricular (4965)</td>
<td>Abandoned posterior ventricular (4965)</td>
<td>Left anterior descending</td>
<td>N/A</td>
</tr>
</tbody>
</table>

CT = computed tomography; CXR = chest radiography; Echo = echocardiography; LQTS = long QT syndrome.
Method of diagnosis

Figure 1 summarizes the testing and results for each patient, and Table 4 lists the diagnostic yield of each modality. Chest X-ray films were available for review for 140 patients. The time from earliest lead implant to coronary artery imaging by catheter angiography or CT was 4.1 years (0.02–34.1 years). Catheter angiography was performed in 126 patients, CT in 37, and both in 19. In the patients who underwent catheter angiography, the coronaries were visualized using aortography in 60; the remaining 67 patients underwent selective coronary angiography.

Median age at CXR was 9.0 years (0.4–48.6 years). Coronary artery compression was suspected on CXR in 9 patients (6.4%) and was equivocal in another 13 patients (9.0%). Of the 9 patients who had suspicious CXR, 4 (40%) were confirmed to have coronary compression (Figures 2A–B, 3A–3B, and 4A–4B). Among the 13 patients with equivocal CXRs, 1 (7.7%) had coronary compression. Among the remaining 119 CXRs with no concerning findings, 2 patients (1.7%) were subsequently noted to have coronary compression.

Two patients (patients 4 and 5) had the “classic pattern” on CXR for their epicardial lead positions, resulting in coronary compression (Figures 2A–2B and 3A–3B). However, in the remaining patients with coronary artery compression, the classic pattern was not present on CXR. These patients either had no epicardial leads that seemed to be in a concerning location on CXR or had films that were suspicious for compression but in a “nonclassic” pattern (Figures 4A–4B), specifically: 1. On the lateral projection, the offending epicardial lead made a bend around the heart, heading posteriorly; and 2. On the PA projection, the bend was noted within the cardiac silhouette. Patient 6 had a unique pattern of coronary compression with loops of an anterior ventricular lead compressing the left anterior descending coronary artery (Supplementary Figure 1A–1B).

Cardiac CT was performed in 37 patients with suspicious CXRs and/or symptoms (Figures 2C and 4C, and Supplementary Figure 1C). Coronary compression was suspected in 9 patients, and the majority of them underwent catheter angiography to confirm the findings (Figures 2D, 3D, and 4D, and Supplementary Figure S1D). Coronary compression was confirmed in 6 patients by catheter angiography and/or surgical inspection, 2 were negative by catheter angiography, and 1 is awaiting catheterization. The latter patient has 18-year-old abandoned atrial and ventricular leads.

Table 3  Lead characteristics

<table>
<thead>
<tr>
<th>Presence of a bipolar lead</th>
<th>Coronary compression (n = 8)</th>
<th>No coronary compression (n = 137)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>6</td>
<td>123</td>
<td>.21</td>
</tr>
<tr>
<td>No</td>
<td>2</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Presence of a unipolar lead</td>
<td>Yes</td>
<td>2</td>
<td>37</td>
</tr>
<tr>
<td>No</td>
<td>6</td>
<td>100</td>
<td></td>
</tr>
<tr>
<td>Presence of a sew-on lead</td>
<td>Yes</td>
<td>7</td>
<td>127</td>
</tr>
<tr>
<td>No</td>
<td>1</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>Presence of a screw-in lead</td>
<td>Yes</td>
<td>1</td>
<td>24</td>
</tr>
<tr>
<td>No</td>
<td>7</td>
<td>113</td>
<td></td>
</tr>
<tr>
<td>Presence of an ICD coil</td>
<td>Yes</td>
<td>2</td>
<td>7</td>
</tr>
<tr>
<td>No</td>
<td>6</td>
<td>130</td>
<td></td>
</tr>
<tr>
<td>Presence of a lead ≤25 cm</td>
<td>Yes</td>
<td>5</td>
<td>71</td>
</tr>
<tr>
<td>long (n = 118)</td>
<td>No</td>
<td>1</td>
<td>41</td>
</tr>
</tbody>
</table>

ICD = implantable cardioverter–defibrillator.
and recurrent chest pain but no troponin leak or dysfunction (patient 11; Figure 1). Two patients had equivocal findings on CT: 1 (patient 12; Figure 1) has an abandoned anterior ventricular lead with a suspicious CT but no symptoms or dysfunction and a negative single-photon emission computed tomography scan, and the other (patient 13; Figure 1) has a 14-year-old atrial and ventricular leads and moderate systemic right ventricular dysfunction and will be undergoing catheterization in the near future.

Of the 126 patients who underwent catheter angiography, coronary compression was noted in 6, all with selective angiography. All 6 patients were sent to the operating room (patients 2–7; Figure 1). Only 1 patient was later determined to have a false-negative finding on catheter angiography (patient 1; Figure 1). Dynamic compression was noted on cine CT and later confirmed in the operating room, with the proximal right coronary compressed by an anteriorly placed atrial lead.

### Catheter angiography vs CT

Nineteen patients underwent both catheter angiography and cardiac CT. Sixteen patients had concordant findings: 11 with negative catheter angiography and CT, and 4 with positive catheter angiography and CT. Four patients (patients 1, 4, 9, and 10; Figure 1) had discordant findings on catheter angiography and CT, all with positive CTs and negative angiography. Two of these patients were sent to the operating room, 1 for concurrent troponin leaks (patient 1) and the other (patient 4) primarily for aortic valve surgery. Coronary compression was confirmed by visual inspection in both. The epicardial leads were noted to run over the coronaries with dense adhesions onto the arteries, presumably the cause of intermittent compression during diastole, especially during exercise. The epicardial leads were removed and adhesions were freed. Since the operation, patient 1, with 1.1 years of follow-up, has experienced no further episodes of chest pain or troponin leak. Patient 4 is doing well after aortic valve surgery and lead repositioning.

The remaining 2 patients with discordant catheter angiography and CT findings were ultimately determined to have no coronary compression. Patient 10 underwent aortic valve surgery and a posterior ventricular pacing lead was repositioned because it was compressing the ventricular myocardium during diastole, noted on both CT scan and visual inspection in the operating room. Patient 9 was noted on catheter angiography to have a posterior coil that was abutting the posterior descending coronary artery but not compressing it. This patient is being monitored with plans for repeat imaging in 3 years.

### Outcomes

Seven patients with coronary artery compression underwent surgical removal or repositioning of the epicardial lead. Median follow-up since surgical intervention was 0.5 years (0.3–1.6 years). Removal or repositioning of the leads was challenging in some patients, with the leads significantly adhered and/or calcified to the epicardial surface of the heart. This also obscured epicardial landmarks and made visualization of the compressed coronary arteries challenging, requiring careful dissection and removal. There were no complications from surgery. The patients with chest pain have experienced no further episodes since their surgery. Only patient 6 has undergone repeat imaging thus far, and notably the left anterior descending and circumflex coronaries are still somewhat distorted from residual fibrous sheaths but fill distally. A perfusion scan is planned for the future.

### Discussion

Although coronary artery compression from epicardial leads is thought to be a rare phenomenon, we found a 5.5% incidence in our patient cohort. Based on our data, none of the 3 tests—CXR, cine CT, and catheterization—are perfect tools for assessment of possible compression, but each offers some advantages and disadvantages and should be considered as part of an overall strategy for assessment of these patients.

Previous reports of cardiac strangulation from epicardial leads have been limited to isolated case reports, dating back to the 1980s. The only case series reported in the literature was published in 2015, by Carreras et al. They reviewed their 20-year experience with epicardial leads. Two of 86 patients (2.3%) had cardiac strangulation, diagnosed by patient symptoms and CXR. One patient in their series was thought to have strangulation based on CXR but was ruled out after CT imaging noted no circumferential looping of the leads. The higher incidence of coronary artery compression noted in our study can be attributed to an increased awareness of this phenomenon, improved CT image quality with newer CT technology, and the understanding that coronary artery compression can occur without “strangulation” of the heart, in that it is not necessary for the leads to be looping around the heart (ie, the “classic pattern” on CXR or circumferential looping on CT).

In our study, CXR with high specificity and negative predictive values may serve as a good screening tool in patients with a low suspicion for coronary artery compression. Our current practice is to screen all asymptomatic patients with...
periodic CXRs to identify those with potential compression and who may require greater vigilance or further testing. However, given the poor sensitivity of CXR for detecting coronary compression, patients with suspicious or equivocal films, concerning symptoms (chest pain, unexplained fatigue, or syncope), or ventricular dysfunction should undergo cine CT or catheter angiography to assess for this potentially serious complication.

Cine CTs, in addition to being noninvasive, can now generate excellent images at low radiation doses (typically 1.5–2.5 mSv in our experience). These high-quality moving images have aided in our identification of dynamic coronary artery compression, as it may be seen only at specific times in the cardiac cycle. Sedation may be required for these studies but usually is minimal, except in the youngest patients. Streak artifact from the epicardial leads themselves may also make it challenging to assess the coronary arteries; however, our ability to determine which lesions are higher risk is growing and evolving, as we learn more about this complication. Therefore, we currently recommend confirmatory catheter angiography in all patients with a positive CT as well as those with an equivocal CT who have concerning symptoms or ventricular dysfunction. Catheter angiography has the disadvantage of being an invasive procedure that may require higher radiation doses to achieve projectional images without the visualization of surrounding cardiac structures, but it is not affected by streak artifact. With careful visualization of the coronaries in multiple planes guided by the cine CT data, catheter angiography can provide confirmation of the CT findings, allowing for better determination of the severity of lesions, especially if intervention is being considered. As experience with cine CT increases, the need for catheter angiography may diminish. We may also find that periodic CXR screening is unnecessary in patients who are no longer growing. Instead, single cine CT or catheter angiography can be performed once if patients are not symptomatic, after they have reached their full growth potential.

As the pendulum potentially swings toward more aggressive screening, we are likely to identify more patients with more subtle degrees of compression or compression in the absence of any obvious clinical sequelae in whom the best course of action would be unclear. The decision to intervene with the epicardial leads may be more straightforward in patients who have the potential for significant somatic growth and worsening compression over time and/or will be undergoing concurrent cardiac surgery. However, for adult patients who are finished growing, patients with no clinical manifestations of coronary ischemia, and those who do not require other cardiac surgical interventions, there may be a role for functional imaging such as positron emission tomography.

Figure 2  Patient 4. Chest X-ray films in the posteroanterior (A) and lateral (B) projections showing the classic pattern of cardiac strangulation from epicardial leads, as the ICD lead courses leftward and posterior around the heart. C: Computed tomography shows the ICD lead constricting the left ventricle and obtuse marginal branch of the circumflex artery. D: Catheter angiography shows loss of contrast within the obtuse marginal branch as it courses below the ICD lead. ICD = implantable cardioverter-defibrillator.
or single-photon emission computed tomography scan. We are starting to systemically assess myocardial perfusion in patients with known or suspected compression to help understand the functional significance of the lesion, especially in those without clinical manifestations, to determine whether these modalities may be helpful either to follow patients after lead removal or repositioning or for risk stratification. This may be of particular importance in patients with long-standing leads that have calcified and are more adherent to the heart and coronaries and, therefore, are difficult to remove fully. Follow-up imaging studies with CT or catheter angiography will also be needed to determine how well the coronary artery compression has been relieved by surgical repositioning or removal of the leads.

As we learn more about the best diagnostic and management strategies for this potentially fatal complication of epicardial systems, strategies to avoid coronary artery compression from epicardial leads need to be evaluated. The practice of placing leads “prophylactically” at the time of cardiac surgery should be reconsidered, especially if the leads are to be placed in young patients and/or along the posterior epicardium. If leads are to be placed, surgical strategies to avoid coronary or myocardial impingement should be considered, including the avoidance of multiple loops of lead overlying each other, and the placement of excess lead material within the pleural space or secured along the diaphragm to prevent lead shifting and future entanglement. For patients who already have epicardial leads, preoperative evaluations before subsequent cardiac surgery or generator changes should include thorough assessment of the coronary arteries by cine CT or catheter angiography. Patients who are identified as high risk for coronary compression would benefit from lead repositioning or removal at the time of surgery.

**Study limitations**

The findings of this study should be interpreted in light of its limitations. This was a retrospective study evaluating patients with epicardial leads who have subsequently undergone coronary imaging. This leads to an inherent selection bias that is reflected in the large number of study patients having structural heart disease. Patients with congenital heart block and primary cardiomyopathies or channelopathies are underrepresented because they do not require regular catheterizations.
or CT scans unlike patients with structural heart disease. The true incidence of coronary and myocardial compression may be underestimated and may only be truly elucidated after all patients with epicardial pacing or defibrillation systems are fully screened. Functional imaging to better assess myocardial perfusion and follow-up imaging of the coronary arteries for patients who underwent surgery for their epicardial leads is not available at this time. Although the majority of our patients had surgical revision of their epicardial leads, further study is required to determine whether all epicardial leads causing coronary artery compression will require removal, as the diagnosis and significance of compression need to be balanced against the difficulty and risks of an operation. Finally, as demonstrated by the follow-up imaging in patient 6, removal of the leads may not be sufficient if the fibrous sheath is not fully removed at the same time. Therefore, effective surgical strategies and associated outcomes need to be evaluated further.

**Conclusion**

The incidence of coronary artery compression from epicardial leads was high in our cohort of patients. We hope to increase awareness of this important, possibly preventable, and potentially fatal complication. Based on our experience, we present a strategy for evaluation of these patients, which includes screening by periodic CXR, with cine CT and/or catheter angiography recommended for those with concerning CXR, symptoms, or ventricular dysfunction. As we learn more about this complication, this strategy can be refined and modified to optimize outcomes and minimize unnecessary testing and radiation exposure.
Appendix
Supplementary data
Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2018.06.038.

References