Influence of Steroid Therapy on the Incidence of Pericarditis and Atrial Fibrillation After Percutaneous Epicardial Mapping and Ablation for Ventricular Tachycardia

Katia Dyrda, MD, MSc*; Sebastiaan R.D. Piers, MD*; Carine F. van Huls van Taxis, MD; Martin J. Schalij, MD, PhD; Katja Zeppenfeld, MD, PhD

Background—This study evaluates the influence of 3 therapeutic approaches on the incidence of pericarditis and atrial fibrillation (AF) after percutaneous epicardial mapping and ablation for ventricular tachycardia.

Methods and Results—Eighty-five consecutive procedures (2006–2011) were retrospectively reviewed. After the first 17 procedures (20.0%), no steroids were administered. For the subsequent 30 procedures (35.3%), systemic steroids were administered intravenously or orally, whereas the last 38 procedures (44.7%) were followed by intrapericardial steroid injection. Compared with no steroids, the incidence of pericarditic chest pain was significantly reduced by intrapericardial steroids (58.8% versus 21.1%; P=0.006) but not by intravenous or oral steroids (58.8% versus 43.4%; P=0.31). There was no significant difference in the incidence of pericarditic ECG with steroids (36.8%, 30.0%, and 41.2% for intrapericardial, intravenous or oral, and none, respectively). There was a nonsignificant reduced incidence of chest pain with ECG changes with steroids (13.2%, 10.0%, and 29.4% for intrapericardial, intravenous or oral, and none, respectively). Radiofrequency applications (65.9% of procedures) did not affect the incidence of pericarditic ECG changes, pericarditic chest pain, or pericarditis (all P>0.05). In 7 (8.3%) patients with no prior history of AF, AF was documented a median 36 hours after procedure. Patients with pericarditic ECG tended to be at greater risk of AF (16.7 versus 3.6%; P=0.091).

Conclusions—There is a high incidence of pericarditic chest pain and ECG changes after epicardial ventricular tachycardia mapping and ablation. Pericarditic chest pain is significantly decreased by intrapericardial steroids. Procedure-related AF is relatively frequent and tends to occur more commonly with pericarditic ECG changes. (Circ Arrhythm Electrophysiol. 2014;7:671-676.)

Key Words: pericarditis, tachycardia, ventricular

Clinical Perspective on p 676

The occurrence of new-onset atrial fibrillation (AF) after epicardial VT ablation was recently reported to be 19.5% within 7 days. In that study, all patients who developed AF had symptoms of pericarditis. Of note, episodes of AF after epicardial VT ablation may be accompanied by the risk for thromboembolic complications because oral anticoagulation is relatively contraindicated as a result of the risk for pericardial bleeding.

This study was thus designed to evaluate the influence of 3 therapeutic approaches on the incidence of pericarditis and AF after percutaneous epicardial mapping and ablation for VT.
Methods
A total of 85 consecutive procedures in which epicardial mapping or mapping and radiofrequency ablation was performed for VT between 2006 and 2011 were retrospectively reviewed. All patients were treated according to our standard clinical protocol and provided informed consent.

Access to the Pericardial Space
Before the procedure, oral anticoagulation was discontinued. On the day of the procedure, all forms of heparin were withheld. Local anesthesia with conscious sedation was the standard approach for procedural analgesia. Femoral venous and arterial access was obtained, and a quadripolar catheter was positioned at the right ventricular apex. The pericardial space was then entered via a transthoracic percutaneous subxyphoid puncture with a 17-gauge Tuohy needle. Under fluoroscopic guidance, contrast media was injected as the needle was advanced to the pericardium. Once access into the pericardial space was obtained, a guidewire was passed through the needle, over which a 6F introducer sheath was advanced. A 6F pigtail catheter was then introduced into the epicardial space to protect the sheath, and the guidewire was removed. Intravenous heparin was then administered to allow for safe endocardial mapping and ablation.

Mapping and Ablation Procedure
Before epicardial mapping, detailed endocardial mapping was performed to determine the epicardial area of interest to restrict epicardial mapping and minimize potentially painful manipulations. A 3.5 mm irrigated-tip catheter (NaviStar ThermoCool, Biosense Webster Inc, Diamond Bar, CA) was then inserted after replacing the 6F by an 11 cm 8F introducer sheath (Cordis) and was maneuvered within the pericardial space for cardiac mapping and ablation. A 63 cm 8.5F sheath (SLO St-Jude Medical) and subsequently a steerable 71 cm 8.5F sheath (Agilis NxT, medium curl, St-Jude Medical) were used in later procedures. The radiofrequency ablation generator used was the StocKert generator (Biosense-Webster Inc). Epicardial applications were delivered ≤50 W, flow rate 20 mL/min with a temperature limit of 45°C, and delivered for ≤60 seconds each. Fluid was removed after a maximum of 5 radiofrequency applications corresponding to an estimated amount of 100 mL.

Administration of Anti-Inflammatory Agents
The therapeutic approach evolved over time from no steroid therapy to systemic steroids to intrapericardial steroids without overlap between the groups. At the end of the first 17 procedures (20.0%), no steroids were administered. For the subsequent 30 procedures (35.3%), systemic steroids were administered intravenously or orally at a dose of 1 mg per kg per day for 3 consecutive days. The last 38 (44.7%) procedures were followed by complete fluid removal and intrapericardial steroid injection, consisting of triamcinolone acetate (2 mg/kg), which was injected in the pericardial space via a pigtail catheter and left in situ by capping the pigtail. The pigtail was not placed under vacuum. Nonsteroidal anti-inflammatory drugs (NSAIDS), in the form of diclofenac 50 mg every 8 hours, were offered on an as-needed basis to all patients who did not have contraindications.

Postablation Care and Evaluation
Standard postoperative monitoring was performed in all patients, consisting of the first 24 hours in the coronary care unit and at least another 48 hours on the ward. ECGs were obtained immediately after the procedure and subsequently every 12 hours until discharge. Transthoracic echocardiography was performed before pigtail removal and again 12 and 24 hours after pigtail removal. Anticoagulation, where indicated, was restarted after removal of the pigtail catheter.

In patients who developed AF, an early cardioversion at <24 hours of AF duration was favored to avoid the need for anticoagulation. Otherwise, for persistent AF, anticoagulation was initiated 224 hours after pigtail removal.

Pericarditis Definition
Pericarditis was defined as typical pericarditic chest pain (pleuritic, improved with sitting upright) with acute pericarditic ECG changes (new widespread ST elevation or PR depression).

Statistical Analysis
Statistical analyses were performed with SPSS version 20 (IBM, Somers, New York, USA). The continuous variables were expressed as number (percentage), mean±SD, or median (interquartile range) where appropriate; χ² tests, Fisher exact tests, Student t tests, and Mann–Whitney U tests were performed where applicable. To analyze the effect of steroid treatment on pericarditic chest pain, pericarditic ECG changes, and pericarditis, overall χ² tests were performed to compare the 3 treatment groups. Then, 2 one-by-one comparisons were performed, applying the Bonferroni correction for multiple comparisons so that P<0.025 was considered to be statistically significant. For all other tests, P<0.05 was considered to be statistically significant.

Results
Eighty-five epicardial procedures were reviewed, which were performed in 76 patients. Seven patients required a second epicardial procedure (4 of 7 within 2 weeks after the first procedure), and of those 7 patients, 2 underwent a third epicardial procedure 67 and 84 days after the second procedure. The patient characteristics are presented in Table 1. Only 8 procedures (9.4%) were performed under general anesthesia. Epicardial mapping alone was performed in 29 (34.1%), whereas 56 (65.9%) underwent mapping and ablation. A nonsteerable long sheath (SRO or SL0) was used in 22 cases (25.9%), whereas an Agilis sheath was used in 42 cases (49.4%). In the mapping only group, epicardial ablation was not performed because of anatomic limitations (coronary arteries or thick fat layer) or because no epicardial ablation target site for VT could be identified.

In the first 17 procedures (20.0%), no steroids were administered. In the subsequent 30 procedures (35.3%), systemic steroids were administered intravenously or orally, whereas in the last 38 procedures (44.7%), intrapericardial steroids were given. On the ward, 62% of patients received NSAIDS for a variety of complaints including chest pain, pain at the pigtail insertion site but also headaches, back pain, and others. The distribution of NSAIDS was the following: 4 of 17 patients (24%) received NSAIDS who had received no steroids, 28 of 30 patients (93%) received NSAIDS who had received systemic steroids, and 21 of 38 patients (55%) received NSAIDS who had received intrapericardial steroids (P<0.001).

Pericarditis occurred after 15.3% of procedures. Pericarditic ECG changes alone, or typical pericarditic chest pain alone, were diagnosed in 20.0% and 21.2%, respectively. There was no age difference between the patients who developed pericarditis and those who did not, with mean ages of 57.6±17.3 versus 58.9±15.1 years, respectively (P=0.474). Clinical chest pain developed within 12 hours of the procedure while the ECG changes occurred within 20 hours at the latest.

The results of the influence of steroid therapy on the incidence of pericarditis are presented in Figure 1. The incidence of pericarditic chest pain was significantly reduced if patients received intrapericardial steroids (21.1%) compared with no steroids (58.8%; P=0.006). Systemic steroids administration did not significantly reduce the incidence of pericarditic chest pain.
pain (43.4% versus 58.8% for patients receiving intravenous or oral steroids or no steroids, respectively, \(P=0.31\)). There was no significant difference in the incidence of pericarditic ECG with steroid therapy (36.8%, 30.0%, and 41.2% for intrapericardial, intravenous or oral, or none, respectively). There was a nonsignificant reduced incidence of chest pain with ECG changes with steroids (13.2%, 10.0%, and 29.4% for intrapericardial, intravenous or oral, or none, respectively). Results were similar when only first procedures were analyzed.

There was no statistical difference between mapping alone and mapping with ablation in the incidence of pericarditic chest pain (44.8% versus 32.1%; \(P=0.249\)), pericarditic ECG (34.5% versus 35.7%; \(P=0.910\)), or both chest pain and ECG changes (24.1% versus 10.7%; \(P=0.103\)) as demonstrated in Figure 2.

The details relating to procedure duration are displayed in Table 2 including procedure duration from time of sheath insertion to sheath removal (median, 4 hours:01 minutes; interquartile range [IQR], 3 hours:20 minutes–4 hours:40 minutes), procedural duration from pericardial puncture to pigtail placement (median, 3 hours:08 minutes; IQR, 2 hours:15 minutes–4 hours:02 minutes), and procedure duration from start of epicardial mapping to pigtail placement (median, 2 hours:00 minutes; IQR, 1 hour:14 minutes–2 hours:40 minutes). There was no difference comparing the above 3 duration times between patients with versus without chest pain, with versus without pericarditic ECG changes, and with versus without both chest pain and pericarditic ECG changes. A pigtail catheter was left in the pericardial space ≤3 hours in 8% of patients, whereas 44% patients had the pigtail for ≥24 hours. Patients with a pigtail for ≥24 hours more often had chest pain (51% versus 25%; \(P=0.012\)) but less often ECG changes (19% versus 48%, \(P=0.006\)) as shown in Table 3.

### Table 1. Patient Characteristics

<table>
<thead>
<tr>
<th></th>
<th>All Patients (n=85)</th>
<th>No Steroids (n=17)</th>
<th>Systemic Steroids (n=30)</th>
<th>Intrapericardial Steroids (n=38)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>58±17</td>
<td>56±21</td>
<td>58±17</td>
<td>59±15</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>67 (79%)</td>
<td>16 (94.1%)</td>
<td>22 (73.3%)</td>
<td>29 (76.3%)</td>
</tr>
<tr>
<td>LVEF, %</td>
<td>42±13</td>
<td>46±13</td>
<td>40±15</td>
<td>41±12</td>
</tr>
<tr>
<td>VT related to</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ischemic CMP, n (%)</td>
<td>13 (15.3%)</td>
<td>1 (5.9%)</td>
<td>6 (20.0%)</td>
<td>6 (15.8%)</td>
</tr>
<tr>
<td>DCM, n (%)</td>
<td>32 (37.6%)</td>
<td>8 (47.1%)</td>
<td>12 (40.0%)</td>
<td>12 (31.6%)</td>
</tr>
<tr>
<td>Other structural heart disease, n (%)</td>
<td>26 (30.6%)</td>
<td>5 (29.4%)</td>
<td>8 (26.6%)</td>
<td>13 (34.2%)</td>
</tr>
<tr>
<td>Idiopathic VT, n (%)</td>
<td>14 (16.5%)</td>
<td>3 (17.6%)</td>
<td>4 (13.3%)</td>
<td>7 (18.4%)</td>
</tr>
<tr>
<td>β-Blocker therapy, n (%)</td>
<td>45 (52.9%)</td>
<td>8 (47.1%)</td>
<td>18 (60.0%)</td>
<td>19 (50.0%)</td>
</tr>
<tr>
<td>Amiodarone, n (%)</td>
<td>32 (37.6%)</td>
<td>6 (35.3%)</td>
<td>13 (43.3%)</td>
<td>13 (34.2%)</td>
</tr>
<tr>
<td>Other AAD, n (%)</td>
<td>38 (44.7%)</td>
<td>5 (29.4%)</td>
<td>15 (50.0%)</td>
<td>18 (47.4%)</td>
</tr>
</tbody>
</table>

AAD indicates antiarrhythmic drug; CMP, cardiomyopathy; DCM, dilated cardiomyopathy; LVEF, left ventricular ejection fraction; and VT, ventricular tachycardia.

---

**Figure 1.** Impact of steroid therapy on the incidence of pericarditis.

**Figure 2.** Influence of mapping and ablation on the incidence of pericarditis.
### Table 2. Procedural Length

<table>
<thead>
<tr>
<th>Procedure Duration</th>
<th>All Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Procedure duration from sheath placement to sheath removal</td>
<td>4 h:01 min (3 h:20 min–4 h:40 min)</td>
</tr>
<tr>
<td>Duration from pericardial puncture to pigtail placement</td>
<td>3 h:08 min (2 h:15 min–4 h:02 min)</td>
</tr>
<tr>
<td>Duration from start epicardial mapping to pigtail placement</td>
<td>2 h:00 min (1 h:14 min–2 h:40 min)</td>
</tr>
<tr>
<td>Fluoroscopy time</td>
<td>47 min (32–56 min)</td>
</tr>
</tbody>
</table>

Variables are expressed as median (interquartile range).

A total of 13 patients developed a postprocedural pericardial effusion or tamponade with a mean effusion of 12±7 mm by transthoracic echocardiography, observed 18±14 hours after the procedure. The effusion was noted at first echo in 8 of 13 patients, at second echo in 4 patients (20, 20, 20, and 19 hours after procedure), and at third echo in 1 patient (50 hours after procedure but <5 min).

In 7 (8.3%) patients with no prior history of AF, procedure-related AF was documented (paroxysmal in 4 and persistent in 3, requiring cardioversion). The median time to new AF was 36 hours (IQR, 19–60 hours). For the persistent cases, 2 underwent rapid cardioversion while the last was anticoagulated for 6 weeks before cardioversion. Patients who developed new-onset AF were older than the patients who did not, with ages of 64.7±7.6 versus 57.2±17.3 years (P=0.016). Patients with a pericarditic ECG tended to be at greater risk of developing AF (16.7% versus 3.6%; P=0.091). There were no complications associated with the episodes of AF. One patient had a recurrence of paroxysmal AF 18 months after resolution of the pericarditis.

The median length of stay in hospital postprocedure was 5 days (IQR, 3–8 days). There was no statistically significant increase in the hospital stay duration because of the incidence of pericarditic chest pain (median 5 days, IQR 3–9 days versus median 5 days, IQR 3–7 days, P=0.75), pericarditic ECG (median 5 days, IQR 3–8 days versus median 4 days, IQR 3–8 days, P=0.98), or of pericarditis (median 5 days, IQR 3–9 days versus median 5 days, IQR 3–8 days, P=0.88).

In 7 patients, repeat epicardial access was required for a total of 9 procedures. In only 1 case, epicardial access to the area of interest could not be reobtained. The patient thus underwent surgical dissection of adhesions and cryoablation. This patient had received oral steroids after his first 2 procedures. Of the 6 patients who underwent unimpeded repeat procedures, 1 had received no steroid, 3 had received oral steroids while 2 had received intrapericardial steroids after the first procedure.

Figure 3 demonstrates pericardial adhesions as photographed in the operating room. Interestingly, although an inflammatory process clearly took place, this patient never reported chest pain, and the ECG never demonstrated ECG changes typical of pericarditis.

### Complications

In total, 5 patients experienced a bleeding complication that can be further described as follows: 1 case (1.2%) of groin hematoma and 4 cases (4.7%) of moderate to severe pericardial bleeding (>80mL) occurring acutely in 2 and delayed in 2. For the acute bleeding cases, one occurred after epicardial puncture (without evidence of right ventricular puncture) and resolved within 20 minutes, whereas the second was because of right ventricular puncture. In the first of the delayed bleeding cases, an echo performed because of clinical deterioration with shortness of breath demonstrated tamponade 13 hours after procedure in a patient who had accidentally been administered low-molecular weight heparin. The second case of delayed bleeding cases occurred in a patient on heparin and dual antiplatelet therapy because of recent coronary artery stenting and was noted 4.75 hours after procedure on an echo performed as a result of low blood pressure and poor diuresis. The bleeding persisted for the following 48 hours. Both were managed with percutaneous drainage. There were no procedure-related deaths.

There were no adverse consequences from the use of intrapericardial triamcinolone more specifically, no infection or myocardial perforation. The patients who underwent redo procedures did so, either as a planned staged approach because of extensive disease and lengthy first ablation procedure or after VT recurrence more than a month after the first ablation procedure.

### Discussion

The prophylactic instillation of 2 mg/kg of intrapericardial corticosteroids has been demonstrated to prevent inflammatory adhesions effectively in a porcine model of postprocedural pericarditis after epicardial mapping and ablation. To date, the impact of intrapericardial steroid administration after epicardial mapping and ablation has not been studied in humans. In the present study, it is demonstrated for the first time that the use of intrapericardial steroids portends an important clinical benefit by significantly reducing the incidence of pericarditic chest pain. This is a valuable
clinical gain because it is associated with increased patient satisfaction. The clinical benefits of the use of intrapericardial steroids may stem from the mode of delivery, the pharmacokinetics, metabolism of the drug, and the duration of exposure to the drug. Triamcinolone is an intermediate acting, liver metabolized glucocorticoid. Triamcinolone acetate is the more potent type of triamcinolone, being ≈8× as effective as prednisone. The drug thus delivered directly and left in the pericardial space can be absorbed locally. In addition, in this work the drug was left in situ rather than being simply flushed through the epicardial space.

Intrapericardial instillation of steroids has also been used in other clinical settings including recurrent idiopathic pericarditis and uremic pericarditis, where studies have consistently demonstrated safety. In the present series, a therapeutic dose of intrapericardial triamcinolone 2 mg/kg was thus chosen. Importantly, there were no adverse consequences from the use of intrapericardial triamcinolone more specifically, no infection or myocardial perforation.

Two recently published studies have reported a widely varying incidence of chest pain after mapping and ablation in the epicardium.10,11 The first reported chest pain in almost all patients,10 whereas the second reported a 21% incidence with an infusion of steroids routinely.11 The pathogenesis of the pain in some cases was felt to be related not only to the epicardial and another 1 of 6 participating centers using systemic steroids routinely and changes (data not shown). Because this is a retrospective study, it is difficult to ascertain from the charts whether patients requested NSAIDs only after the onset of chest pain. The use of NSAIDs however may have had an influence on clinical variables such as pericarditic ECG changes even if the NSAIDs were started after the onset of chest pain. The dose of steroids was unchanged over time and as a result the most effective dose of steroids for the prevention of pericarditic chest pain could not be determined. Although the number of procedures in this study was relatively large, some subgroup analyses were still limited by the sample size. In addition, given that the introduction of the Agilis sheath was contemporaneous to the introduction of intrapericardial steroids, the relative impact of each variable on the incidence of pericarditis cannot be further determined. However, because the steerable Agilis sheath is assumed to be more traumatic than a non-steerable sheath, the impact of the intrapericardial steroid on the reduction of clinical pericarditic chest pain may be even underestimated.

Conclusions

There is a high incidence of pericarditic chest pain and of pericarditic ECG after percutaneous epicardial access for VT mapping and ablation. Complaints of pericarditic chest pain are significantly decreased by the administration of intrapericardial steroids but not intravenous or oral steroids. Procedure-related AF is relatively frequent and tends to occur more commonly in patients with pericarditic ECG changes.

Disclosures

The Department of Cardiology at the Leiden University Medical Centre receives unrestricted research and fellowship grants from Biotronik, Boston Scientific, GE Healthcare, Medtronic, and St. Jude Medical. Dr Zeppenfeld receives consulting fees from St. Jude Medical. The other authors report no conflicts.

References


**CLINICAL PERSPECTIVE**

Catheter ablation for ventricular tachycardia is increasingly used and may require a percutaneous, epicardial approach. Complications of an epicardial approach include, among others, pericarditis and atrial fibrillation. This study was thus designed to evaluate the influence of steroid therapy, delivered systemically or intrapericardially versus the absence of steroids, on the incidence of pericarditis and atrial fibrillation after percutaneous epicardial mapping and ablation for ventricular tachycardia. The results demonstrate a high incidence of pericarditic chest pain and ECG changes after epicardial ventricular tachycardia mapping and ablation. Pericarditic chest pain is significantly decreased by the use of intrapericardial steroids. Procedure-related atrial fibrillation is relatively frequent and tends to occur more commonly with pericarditic ECG changes. There were no adverse consequences from the use of intrapericardial triamcinolone. The administration of intrapericardial triamcinolone does thus provide clinical benefits, specifically greater patient comfort, and may be considered for routine use after epicardial ventricular tachycardia ablation procedures.
Influence of Steroid Therapy on the Incidence of Pericarditis and Atrial Fibrillation After Percutaneous Epicardial Mapping and Ablation for Ventricular Tachycardia
Katia Dyrda, Sebastiaan R.D. Piers, Carine F. van Huls van Taxis, Martin J. Schalij and Katja Zeppenfeld

_Circ Arrhythm Electrophysiol_. 2014;7:671-676; originally published online June 26, 2014; doi: 10.1161/CIRCEP.113.001148
_Circulation: Arrhythmia and Electrophysiology_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2014 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-3149. Online ISSN: 1941-3084

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circep.ahajournals.org/content/7/4/671

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in _Circulation: Arrhythmia and Electrophysiology_ can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to _Circulation: Arrhythmia and Electrophysiology_ is online at:
http://circep.ahajournals.org/subscriptions/