Chapter 7

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

Sebastiaan RD Piers, MD, Katia Dyrda, MD, MSc, Carine F. van Huls van Taxis, MD, Martin J Schalij, MD, PhD, Katja Zeppenfeld, MD, PhD

Sebastiaan R.D. Piers and Katia Dyrda share first authorship on this manuscript

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ABSTRACT

Background

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

Methods and Results

Eighty-five consecutive procedures (2006 to 2011) were retrospectively reviewed. After the first 17 procedures (20.0%), no steroids were administered. For the subsequent 30 (35.3%), systemic steroids were administered intravenously or orally (IV/PO) while the last 38 (44.7%) were followed by intrapericardial steroid injection.

Compared to no steroids, the incidence of pericarditic chest pain was significantly reduced by intrapericardial steroids (58.8% vs. 21.1%, p=0.006), but not by IV/PO steroids (58.8% vs. 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, IV/PO and none, respectively). There was a non-significant reduced incidence of chest pain with ECG changes with steroids (13.2%, 10.0%, and 29.4% for intrapericardial, IV/PO 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 post procedure. Patients with pericarditic ECG tended to be at greater risk of AF (16.7 vs. 3.6%, p=0.091)

Conclusion

There is a high incidence of pericarditic chest pain and ECG changes following epicardial VT 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.

INTRODUCTION

Catheter ablation of ventricular tachycardia (VT) is increasingly employed for both idiopathic and scar-related etiologies.[1] In particular, in patients with nonischemic cardiomyopathy, the substrate for VTs is often located intramurally or subepicardially and may require a percutaneous, transpericardial approach. In virtually all patients who undergo an epicardial procedure, pericarditis occurs to a variable extent as a result of local inflammation.[4] This can manifest clinically from mild pericarditis to hemorrhagic pericardial effusion and, rarely, cardiac tamponade.[4] If symptoms occur, patients may be treated with oral anti-inflammatory nonsteroidal and corticosteroid agents. Data on the pharmacokinetic profile of intrapericardially delivered agents has proven favorable suggesting that targeted local concentrations can be reached with minimal systemic concentrations (therefore reducing the potential risk of systemic side effects).[4] Intrapericardial corticosteroids and Hyaluronic Acid have been successfully used to treat recurrent noninfectious pericarditis[5-8] and to prevent adhesions after open-chest cardiac surgery.[9, 10] The intrapericardial instillation of 2 mg/kg of triamcinolone acetate after epicardial radiofrequency ablation was demonstrated to successfully prevent the occurrence of postprocedure pericarditis and adhesions in porcine animal.[4] Prevention of adhesions may be important given the potential need to re-obtain access to the pericardial space due to recurrence of arrhythmia.

The occurrence of new onset atrial fibrillation (AF) after epicardial VT ablation was recently reported to be 19.5% within 7 days.[13] 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, as oral anticoagulation is relatively contraindicated due to 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 following 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

Prior to 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 (RV) 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

Prior to epicardial mapping, detailed endocardial mapping was performed to determine the epicardial area of interest in order to restrict epicardial mapping and minimize potentially painful manipulations. A 3.5mm irrigated-tip catheter (NaviStar ThermoCool, Biosense Webster Inc, Diamond Bar, CA, USA) was then inserted after replacing the 6F by an 11cm 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) was used in later procedures. The radiofrequency (RF) ablation generator used was the Stockert generator (Biosense-Webster Inc). Epicardial applications were delivered up to 50W, flow rate 20ml/min with a temperature limit of 45°C, and delivered for up to 60 seconds each. Fluid was removed after a maximum of 5 RF applications corresponding to an estimated amount of 100cc.

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 (35.3%), systemic steroids were administered intravenously or orally (IV/PO) at a dose of 1mg 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 (2mg/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. Non-steroidal 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 post-operative 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. Electrocardiograms (ECGs) were obtained immediately following the procedure and subsequently every 12 hours until discharge. Trans-thoracic echocardiography was performed prior to 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 in order to avoid the need for anticoagulation. Otherwise, for persistent AF, anticoagulation was initiated ≥24 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. Chi-square tests, Fisher's exact tests, Student's 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 chi-square tests were performed to compare the three treatment groups. Then, two one-by-one comparisons were performed, applying the Bonferroni correction for multiple comparisons so that a p-value of $p < 0.025$ was considered to be statistically significant. For all other tests, a p-value of $p < 0.05$ 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%) while 56 (65.9%) underwent mapping and ablation. A non-steerable long sheath (SR0 or SL0) was used in 22 cases (25.9%) while an Agilis sheath was used in 42 cases (49.4%). In the mapping only group,

Table 1. Patient characteristics

	All patients (n=85)	No Steroids (n=17)	Systemic Steroids (n=30)	Intra-Pericardial Steroids (n=38)
Age (years)	58 ±17	56 ±21	58 ±17	59 ±15
Male, n (%)	67 (79%)	16 (94,1%)	22 (73,3%)	29 (76,3%)
LVEF(%)	42 ±13	46 ±13	40 ±15	41 ±12
VT related to				
Ischemic CMP, n (%)	13 (15,3%)	1 (5,9%)	6 (20,0%)	6 (15,8%)
DCM, n (%)	32 (37,6%)	8 (47,1%)	12 (40,0%)	12 (31,6%)
Other structural heart disease, n (%)	26 (30,6%)	5 (29,4%)	8 (26,6%)	13 (34,2%)
Idiopathic VT, n (%)	14 (16,5%)	3 (17,6%)	4 (13,3%)	7 (18,4%)
Beta-blocker therapy, n (%)	45 (52,9%)	8 (47,1%)	18 (60,0%)	19 (50,0%)
Amiodarone, n (%)	32 (37,6%)	6 (35,3%)	13 (43,3%)	13 (34,2%)
Other AAD, n (%)	38 (44,7%)	5 (29,4%)	15 (50,0%)	18 (47,4%)

ADD: anti-arrhythmic drug; CMP: cardiomyopathy; DCM: dilated cardiomyopathy; LVEF: left ventricular ejection fraction; VT: ventricular tachycardia

epicardial ablation was not performed due to anatomical 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 (35.3%), systemic steroids were administered IV or PO while in the last 38 (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 vs. 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 to no steroids (58.8%) (p=0.006). Systemic steroids administration did not significantly reduce the incidence of pericarditic chest pain (43.4% vs. 58.8% for patients receiving IV/PO 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, IV/

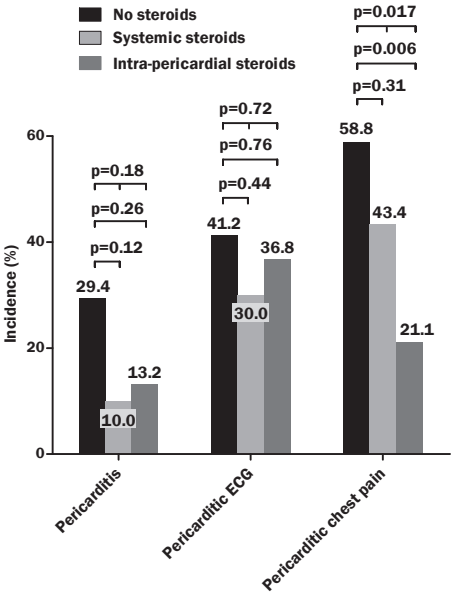

Figure 1. Impact of steroid therapy on the incidence of pericarditis

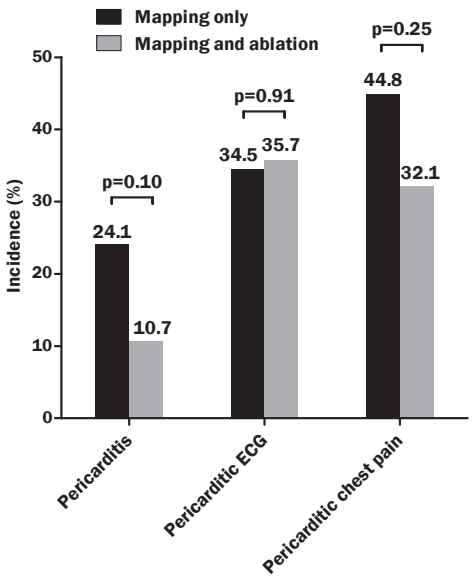

Figure 2. Influence of mapping and ablation on the incidence of pericarditis

PO or none, respectively). There was a non-significant reduced incidence of chest pain with ECG changes with steroids (13.2%, 10.0%, and 29.4% for intrapericardial, IV/PO 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 vs. 32.1%, p=0.249), pericarditic ECG (34.5 vs 35.7%, p=0.910), or both chest pain and ECG changes (24.1 vs. 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 4h:01min, IQR 3h:20min – 4h:40min), procedural duration from pericardial puncture to pigtail placement (median 3h:08min, IQR 2h:15min – 4h:02min) and procedure duration from start of epicardial mapping to pigtail placement (median 2h:00min, IQR 1h:14min – 2h:40min).

Table 2. Procedural length

	All patients
Procedure duration from sheath placement to sheath removal	4h:01min (3h:20min – 4h:40min)
Duration from pericardial puncture to pigtail placement	3h:08min (2h:15min – 4h:02min)
Duration from start epicardial mapping to pigtail placement	2h:00min (1h:14min – 2h:40min)
Fluoroscopy time	47 min (32 – 56 min)

Variables are expressed as median (interquartile range).

There was no difference comparing the above three duration times between patients with vs. without chest pain, with vs. without pericarditic ECG changes, and with vs. without both chest pain and pericarditic ECG changes.

A pigtail catheter was left in the pericardial space less than 3 hours in 8% of patients while 44% patients had the pigtail for greater or equal to 24 hours. Patients with a pigtail for ≥24 hours more often had chest pain (51% vs. 25%, p=0.012), but less often ECG changes (19% vs. 48%, p=0.006) as shown in Table 3.

Table 3. Influence of the presence of a pigtail catheter on the incidence of pericarditis

	Pigtail ≥24 hours	Pigtail < 24 hours	p
Chest pain	19/37 (51%)	12/48 (25%)	0.012
ECG changes	7/37 (19%)	23/48 (48%)	0.006
Chest pain and ECG changes	4/37 (11%)	9/48 (19%)	0.31

A total of 13 patients developed a post-procedural 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 < 5mm).

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 prior to cardioversion. Patients who developed new onset AF were older than the patients who did not, with ages of 64.7±7.6 vs. 57.2±17.3 years (p=0.016). Patients with a pericarditic ECG tended to be at greater risk of developing AF (16.7 vs. 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 post-procedure was 5 days (IQR 3 – 8 days). There was no statistically significant increase in the hospital stay duration due to the incidence of pericarditic chest pain (median 5 days, IQR 3 – 9 days vs. median 5 days, IQR 3 – 7 days, p = 0.75), pericarditic ECG (median 5 days, IQR 3 – 8 days vs. median 4 days, IQR 3 – 8 days, p = 0.98) or of pericarditis (median 5 days, IQR 3 – 9 days vs. 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 one case, epicardial access to the area of interest could not be re-obtained. 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, one 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.

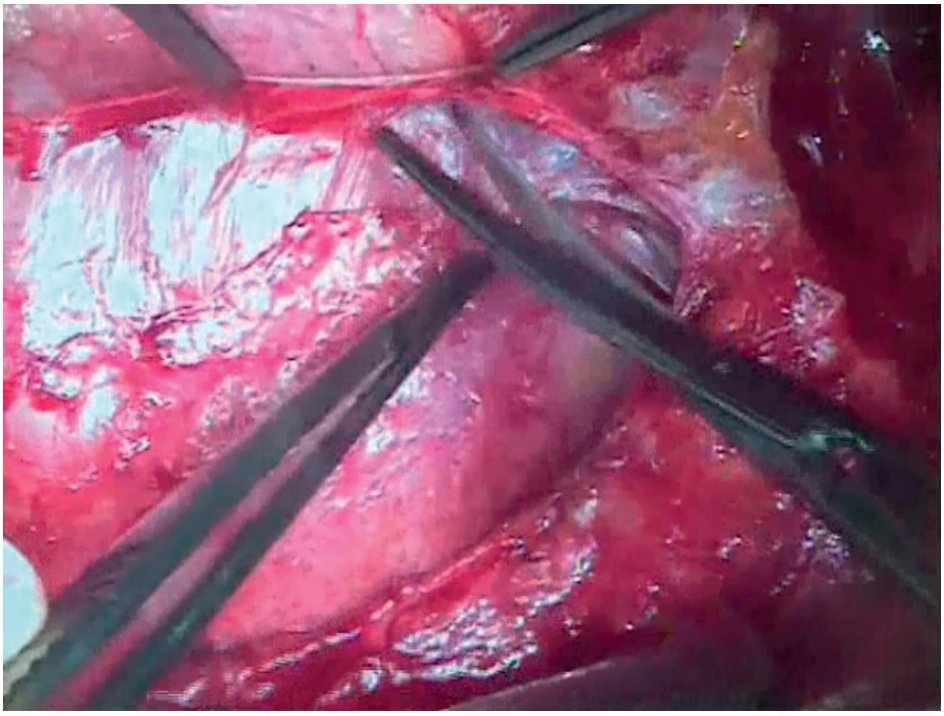

Figure 3. Dissection of epicardial adhesions during surgery

Complications

In total, 5 patients experienced a bleeding complication which can be further described as follows: 1 case (1.2%) of groin hematoma and 4 cases (4.7%) of moderate to severe pericardial bleeding (>80cc) occurring acutely in 2 and delayed in 2. For the acute bleeding cases, one occurred following epicardial puncture (without evidence of RV puncture) and resolved within 20 minutes while the second was due to RV puncture. In the first of the delayed bleeding cases, an echo performed due to clinical deterioration with shortness of breath demonstrated tamponade 13 hours post 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 due to recent coronary artery stenting and was noted 4.75 hours post procedure on an echo performed due to 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 IP triamcinolone more specifically, no infection or myocardial perforation. The patients who underwent redo procedures did so, either as a planned staged approach due to extensive disease and lengthy first ablation procedure or, following 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 effectively prevent inflammatory adhesions in a porcine model of post procedural pericarditis after epicardial mapping and ablation.[4] 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 as 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 and 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 about eight times 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 2mg/kg was thus chosen. Importantly, there were no adverse consequences from the use of IP triamcinolone more specifically, no infection or myocardial perforation.

Two recently published studies have reported a widely varying incidence of chest pain after mapping and/or ablation in the epicardium.[11,12] The first reported chest pain in almost all patients,[11] whereas the second reported a 21% incidence with 1 out of 6 participating centers using systemic steroids routinely and another 1 out of 6 participating centers using intrapericardial infusion of steroids routinely[12]. The etiology of the pain in some cases was felt to be related, not only to the epicardial mapping and ablation, but also to the friction of the pigtail left in the pericardium for continuous drainage.[12]

In our series, we report an incidence of chest pain of 36.5% (21.2% chest pain only and 15.3% chest pain with ECG changes). Some of the difference in the reported numbers may arise from the difficulty in accurately assessing the quality of the pain retrospectively. In addition, similarly to our approach, the centers included in the above series used a conscious sedation approach, where the analgesic drugs and dosages administered varied and thus provided a varying degree of post-procedural pain control. Also, the type of ablation catheter and sheaths varied from center to center. Furthermore, the use of NSAIDs was not uniform, where in one series it ranged from being left to the discretion of the physician to being prescribed almost routinely for a week[11]. Nevertheless, in the series where steroids were used,[12] including but not only intrapericardial steroids, a lower incidence of chest pain was reported.

With respect to AF, the incidence of 8.3% reported in the present series sits within the range previously quoted in the literature. Already in 1976, the incidence of AF in a cohort of 100 patients with pericarditis was reported to be 5%.[14] An incidence of 19.5% was reported for new onset AF within 7 days of procedure in a recent series of 41 patients undergoing epicardial VT ablation.[13] In that series, patients developing AF were significantly younger than those without AF whereas our study demonstrated the opposite. They also described lack of amiodarone therapy as a risk factor for procedurally related new onset AF. Interestingly, in the data presented here, none of the patients who developed new onset AF were receiving amiodarone therapy for VT nor did they receive it post-procedurally for AF prevention. On a more concordant note, in their series, all patients with new onset AF had clinical symptoms of pericarditis while in the present data there was a trend towards AF occurring more commonly in patients with pericarditic ECG changes. In addition, 85.7% of the patients with new onset AF in the present series had both mapping and ablation performed. The diverging results between the 2 studies can be in large part explained by the small number of patients developing procedure-related AF in the present series.

Limitations

No randomization was performed, however, the therapeutic approaches in the current study were applied non-selectively in consecutive patients. The findings are therefore highly suggestive for an effect of intrapericardial steroids on pericarditic chest pain after epicardial mapping and/or ablation. As 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 contemporary to the introduction of intrapericardial steroids, the relative impact of each variable on the incidence of pericarditis cannot be further determined. However, as 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.

CONCLUSION

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

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.

REFERENCE LIST

1. Sacher F, Tedrow UB, Field ME, Raymond J-M, Koplan BA, Epstein LM, Stevenson WG. Ventricular tachycardia ablation evolution of patients and procedures over 8 years. *Circ Arrhythm Electrophysiol*. 2008;1(3):153-161.
2. Cesario DA, Vaseghi M, Boyle NG, Fishbein MC, Valderrabano M, Narasimhan C, Wiener I, Shivkumar K. Value of high-density endocardial and epicardial mapping for catheter ablation of hemodynamically unstable ventricular tachycardia. *Heart Rhythm*. 2006;3(1):1-10.
3. Roberts-Thomson KC, Seiler J, Steven D, Inada K, Michaud GF, John RM, Koplan BA, Epstein LM, Stevenson WG, Tedrow UB. Percutaneous access of the epicardial space for mapping ventricular and supraventricular arrhythmias in patients with and without prior cardiac surgery. *J Cardiovasc Electrophysiol*. 2010;21(4):406-411.
4. D'Avila A, Neuzil P, Thiagalingam A, Gutierrez P, Aleong R, Ruskin JN, Reddy VY. Experimental efficacy of pericardial instillation of anti-inflammatory agents during percutaneous epicardial catheter ablation to prevent postprocedure pericarditis. *J Cardiovasc Electrophysiol*. 2007;18(11): 1178-1183.
5. Buselmeier TJ, Simmons RL, Najarian JS, Kjellstrand CM. Intrapericardial steroids in uremic pericarditis. *Lancet*. 1977;2(8032): 301-302.
6. Grubb SR, Cantley LK, Jones DL, Carter WH. Iatrogenic cushings-syndrome after intra-pericardial corticosteroid-therapy. *Ann Intern Med*. 1981;95(6):706-707.
7. Maisch B, Ristic AD, Pankuweit S. Intrapericardial treatment of autoreactive pericardial effusion with triamcinolone - the way to avoid side effects of systemic corticosteroid therapy. *Eur Heart J*. 2002;23(19):1503-1508.
8. Medani CR, Ringel RE. Intrapericardial triamcinolone hexacetonide in the treatment of intractable uremic pericarditis in a child. *Pediatr Nephrol*. 1988;2(1):32-33.
9. Mitchell JD, Lee R, Neya K, Vlahakes GJ. Reduction in experimental pericardial adhesions using a hyaluronic-acid bioabsorbable membrane. *Eur J Cardiothorac Surg*. 1994; 8(3):149-152.
10. Seeger JM, Kaelin LD, Staples EM, Yaacobi Y, Bailey JC, Normann S, Burns JW, Goldberg EP. Prevention of postoperative pericardial adhesions using tissue-protective solutions. *J Surg Res*. 1997;68(1):63-66.
11. Sacher F, Roberts-Thomson K, Maury P, Tedrow U, Nault I, Steven D, Hocini M, Koplan B, Leroux L, Derval N, Seiler J, Wright MJ, Epstein L, Haissaguerre M, Jais P, Stevenson WG. Epicardial ventricular tachycardia ablation a multicenter safety study. *J Am Coll Cardiol*. 2010;55(21):2366-2372.
12. Della Bella P, Brugada J, Zeppenfeld K, Merino J, Neuzil P, Maury P, Maccabelli G, Vergara P, Baratto F, Berruezo A, Wijnmaalen AP. Epicardial ablation for ventricular tachycardia: a European multicenter study. *Circ Arrhythm Electrophysiol*. 2011;4(5): 653-659.
13. Mahapatra S LD, Bhamidipati CM, McDaniel G, Kamath S, Bunch TJ, Ailawadi G. Incidence, risk factors, and consequences of new-onset atrial fibrillation following epicardial ablation for ventricular tachycardia. *Europace*. 2011;13(4):548-554.
14. Spodick D. Arrhythmias during acute pericarditis. A prospective study of 100 consecutive cases. *JAMA*. 1976;235(1):39-41