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## Delayed Cardiac Tamponade After Radiofrequency Catheter Ablation of Atrial Fibrillation A Worldwide Report

**To the Editor:** Catheter ablation is increasingly being offered for the curative treatment of atrial fibrillation (AF). Generally, the procedure is safe and effective (1), but the intense intraprocedural and post-procedural anticoagulation regimen used, together with extensive catheter manipulation and application of radiofrequency energy, may expose patients to an excessive risk for bleeding. Although intraprocedural tamponade has been reported (1,2), little is known about the incidence of delayed cardiac tamponade (DCT). We investigated the incidence, modes of presentation, and outcomes of DCT in a large population of patients undergoing this procedure.

An anonymous volunteer reporting method was used to identify cases that occurred between January 1995 and December 2006 at 102 centers contributing to our worldwide surveys on the efficacy and safety of AF ablation (1,2). We defined DCT as hypotension or cardiogenic shock requiring pericardial drainage or causing death due to documented pericardial effusion occurring at least 1 h post-procedurally but attributable to the ablation procedure. Center experience, mapping and ablation technique and catheter used, type of AF treated, intraprocedural and post-procedural anticoagulation strategy, and the occurrence of tamponade during the procedure were compared at centers with and without cases of DCT to determine potential risk factors. This survey was approved by the institutional review board at Policlinico San Donato.

Patient and center characteristics were described by location indexes if continuous or by frequencies and percentages if noncontinuous. Contingency tables were created to calculate the influence on the occurrence of DCT of center characteristics with regard to experience, mapping and ablation strategy and catheter used, type of AF, and anticoagulation strategy during and after the procedure. Differences were calculated using chi-square tests of independence. If any of the expected cell counts were lower than 5, the Fisher's exact test was applied using the algorithm proposed by Mehta and Patel where the contingency table dimensions were greater than  $2 \times 2$ . All the center-related variables that were associated ( $p < 0.10$ ) with the presence of at least 1 DCT event were subsequently tested in a multivariate model. Only the variable with the highest statistical significance was tested in the multivariate model.

Forty-five cases of DCT after 27,921 procedures performed in 21,478 patients were reported. Patients presented with DCT a median of 12 days (range: 0.2 to 45 days) after the ablation procedures. Four patients presented during their initial hospital stays, whereas 41 patients were discharged after their ablation procedures and presented subsequently to their primary care physicians, emergency departments, or cardiologists with warning symptoms. Echocardiography proved diagnostic in all such cases.

Most patients presented with nonspecific symptoms, including constant thoracic pain, neck or back pain, pain during breathing, dyspnea, dizziness, nausea, fever, peripheral or global edema, impending sense of doom or death, nausea, fever or general malaise. One patient had cardiac arrest while swimming and was found to have cardiac tamponade after resuscitation. Another patient died suddenly and was found to have a large pericardial effusion during the in-hospital resuscitation attempt. In 6 patients, hypotension developed without warning. Two patients (5%) died, 1 from cardiac arrest 14 days after the procedure and 1 at 30 days after a procedure complicated by cerebral thromboembolism that had occurred 24 h after ablation. The mode of presentation of hypotension varied, with 39 patients exhibiting gradual progression to cardiac tamponade and 6 patients experiencing severe symptoms within minutes.

Therapy included pericardiocentesis in 36 patients and also required pleural evacuation in another patient. A pericardial surgical window was needed in 1 patient and open-chest surgery in 7 patients. In 36 patients, the hemorrhagic content of the pericardial fluid was reported at visual inspection, whereas serous effusion was the only finding in the remaining 9 patients.

When tested in the multivariate model (Table 1), the following factors were independent predictors of at least 1 DCT event: large volume of patients treated (odds ratio: 5.03), use of irrigation catheter (odds ratio: 2.77), and treatment of paroxysmal AF only (odds ratio: 3.97).

DCT occurred in 0.2% of patients in our series. Although warning symptoms were present in most patients, hypotension and shock were the presentation in 13% of patients. Warning symptoms were variable, including thoracic, neck and back pain, and dyspnea and sometimes nonspecific symptoms such as dizziness, nausea, fever, peripheral or global edema, impending sense of doom or death, or general malaise. Eighty-four percent of patients sought care from their primary care physicians, emergency departments, or general cardiologists rather than from the interventional electrophysiologist. Evolution to cardiac tamponade mostly occurred over hours or days, allowing access to medical aid, but hemodynamic deterioration within minutes from the onset of symptoms was observed in 13% of patients.

Retrospective series based on anonymous identification of cases and retrospective medical record review have limitations. Although anonymous identification of cases facilitates reporting of data on poor outcomes, variability in prospective monitoring by many physicians and of many procedures prevents accurate assessment of the incidence of events, their presentation, and outcomes. These limitations are further expanded by the late occurrence of DCT in relationship to the date of AF ablation.

**Table 1** Multivariate Analysis of the Risk for Experiencing at Least 1 Event

Factor	Regression Coefficient	Relative Risk (95% Confidence Interval)	p Value
Number of procedures >299 (n = 36)	1.61	5.03 (1.85–13.64)	0.002
Ablation catheter irrigation (n = 57)	1.02	2.77 (1.00–7.86)	0.050
Type of atrial fibrillation ablated			
Paroxysmal (n = 28)	1.38	3.97 (1.42–11.14)	0.009
Constant	–2.62		

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**Letters to the Editor**

## Late Gadolinium Enhancement in Left Ventricular Dysfunction After Trastuzumab

We read with great interest the report by Fallah-Rad et al. (1) regarding echocardiography, biomarkers, and cardiac magnetic resonance (CMR) in patients with trastuzumab-induced heart failure. Cardiotoxicity after chemotherapy remains a clinical challenge. There is great interest in early detection of myocardial damage before left ventricular (LV) function deteriorates, and CMR is a good candidate to deliver this information. CMR is known to differentiate transient and permanent myocardial injury

in various systemic and inflammatory diseases using specific techniques (2). We would like to focus on the late gadolinium enhancement (LGE) CMR findings.

The authors state in their discussion that “delayed enhancement of the lateral wall of the LV within the mid-myocardium portion [represents] a common feature in breast-cancer patients with trastuzumab-induced cardiomyopathy.” This is an interesting finding with a potentially high clinical impact. However, from our point of view, more evidence is required to support this general statement. Their current paper is the third publication by the group regarding this subject with the same single CMR image (once as a magnitude image, twice as a phase-sensitive reconstruction) depicting an intramural lateral lesion (1,3,4). This LGE lesion in itself is unspecific and may or may not be related to