Case Report

Correlation between T-Wave Alternans and Cardiac Volume Status via Intrathoracic Impedance Measurements

Jose' Dizon,1, 2 Kathleen Hickey,1 and Hasan Garan 1

1 Division of Cardiology, Department of Medicine and Columbia University Medical Center, Columbia University, New York, NY 10032, USA
2 222 Westchester Avenue, White Plains, NY 10604, USA

Correspondence should be addressed to Jose' Dizon, jmd11@columbia.edu

Received 29 May 2012; Accepted 25 July 2012

Academic Editors: A. J. Mansur and K. Nikus

Copyright © 2012 Jose’ Dizon et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

1. Introduction

The presence of microvolt T-wave alternans has been shown to correlate with a higher risk of sudden cardiac death in patients with structural heart disease [1]. The mechanism of T-wave alternans is unclear, but may be related to altered intracellular calcium handling, also commonly observed in congestive heart failure [2, 3]. Altered calcium cycling and mechanical stretch in heart failure have been implicated in the development of repolarization abnormalities and arrhythmogenesis [4, 5]. Certain models of implantable defibrillators have the capability of monitoring cardiac fluid status via intrathoracic impedance measurements. We sought to determine if a correlation exists between T-wave alternans and cardiac volume status, as monitored by intrathoracic impedance (Optivol, Medtronic, Inc., Minneapolis, MN, USA). Patients with implanted biventricular defibrillator systems for dilated cardiomyopathy, Class III congestive heart failure, and left bundle branch block had T-wave alternans testing via the spectral method (Cambridge Heart, Inc.) by atrial pacing up to 110 bpm during routine defibrillator followup (or atrial-biventricular pacing in cases of atrioventricular block). Intrathoracic impedance measurements were performed monthly by home monitoring of the defibrillator system. When impedance measurements suggested fluid overload, the patients returned to the clinic for T-wave alternans testing.

2. Case Report

The current example is a 54-year-old man with nonischemic dilated cardiomyopathy and left bundle branch block. He had well-compensated heart failure following biventricular defibrillator insertion in 2007. His intrathoracic impedance...
measurements and T-wave alternans tests were normal at baseline (Figure 1) until March 3, 2011, when he had a heart failure exacerbation. Vital signs included blood pressure of 100/60, pulse 88, and respiratory rate 20/minute. Weight was 175 pounds, and physical exam revealed clear lungs but peripheral edema and ascites. Brain natriuretic peptide measurement (1854 pg/mL) was consistent with fluid overload, and intrathoracic impedance measurements dropped with resultant increased fluid index (Figures 2(a) and 2(b)). T-wave alternans was markedly positive with sustained alternans present at an onset heart rate of less than 105 bpm (Figures 2(c) and 2(d)). The patient was given daily doses of 80 mg intravenous furosemide until he reached his baseline weight of 168 pounds, with restoration of normal clinical status. Three weeks later, intrathoracic impedance measurements and T-wave alternans tests returned to normal (Figure 3).

3. Discussion

The implantable defibrillator has become standard therapy for the prevention of sudden death in patients with severe structural heart disease. The questionable cost effectiveness of routine implantation for primary prevention has resulted in the search for better methods of risk stratification. One such modality is microvolt T-wave alternans, a test that can
Figure 2: Data during heart failure exacerbation. Plots arranged as in Figure 1. The fluid index (a) is markedly elevated as intrathoracic impedance values drop (b). Sustained T-wave alternans is present as evidenced by the dark shading on the plots c and d (arrows).

detect beat-to-beat variations in the T-wave that may correspond to spatial dispersion of refractoriness or alternations in repolarization that underlie a substrate that can support lethal arrhythmias [6]. The utility of T-wave alternans for risk stratification has been previously investigated [1, 7].

The mechanism of T-wave alternans is unclear, but data point to abnormalities in intracellular calcium handling [2, 3, 8]. Changes in calcium handling have been shown to underlie heart failure, and conditions that lead to stretch in the heart such as volume overload are associated with altered calcium metabolism [9], arrhythmias [10], and sudden death [11]. A relationship between volume overload or stretch and T-wave alternans magnitude or susceptibility has been demonstrated in experimental models [10, 12, 13]. Thus a molecular connection in the form of altered calcium metabolism exists between heart failure, arrhythmias, and T-wave alternans.

However, a correlation between T-wave alternans and cardiac volume status in the ambulatory clinical setting has not been demonstrated.

Intrathoracic impedance measurements have been shown to correlate with cardiac fluid status and predict heart failure hospitalizations [14, 15]. Using this technology in an implantable defibrillator, we were able to detect fluid overload in a patient with cardiomyopathy, which was confirmed by physical exam and brain natriuretic peptide measurement. The dramatic increase in T-wave alternans during acute heart failure and disappearance after treatment suggest T-wave alternans status, and by extension, propensity to lethal arrhythmias, may be influenced by cardiac volume status. Such a link may help to explain the association between acute heart failure syndromes, arrhythmias, and sudden death.
Figure 3: Data after patient treated for fluid overload with diuretics. The intrathoracic impedance measurements and fluid index return to baseline (a and b). No sustained alternans is present on a T-wave alternans test conducted 3 weeks after diuresis (c and d).

Acknowledgment

The authors would like to appreciate Medtronic, Inc., Minneapolis, MN, for supporting this work.

References


