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Transient apical wall thickening in patients with stress cardiomyopathy: Prevalence, profile, and impact on clinical course

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ABSTRACT

Background: Transient apical wall thickening (TAWT), mimicking apical hypertrophic cardiomyopathy during recovery from stress cardiomyopathy (SCM), has recently been reported. However, the clinical significance of this phenomenon has not yet been assessed. We aimed to explore the prevalence, profiles, and impact on the clinical course of TAWT in patients with SCM.

Methods: We retrospectively analyzed the SCM registry from January 2009 to December 2013. Of 429 patients with SCM, 124 patients who had typical features of transient apical ballooning were included. We identified patients who showed evidence of TAWT, which became normalized on serial echocardiograms. Clinical characteristics, incidence of cardiac complications (arrhythmia, pulmonary edema, cardiogenic shock, or left ventricular thrombus), and in-hospital mortality were compared between patients with and without TAWT.

Results: Among 124 patients, 17 (14%) patients showed TAWT. During the follow-up period, TAWT was observed 14.6 ± 10.3 days after the initial SCM diagnosis. Patients with TAWT showed a higher prevalence of septic shock as a triggering factor of SCM than those without TAWT (41.2% vs. 19.6%, $p = 0.048$). Furthermore, cardiac complications were more prevalent in patients with TAWT compared to patients without (64.7% vs. 33.6%, $p = 0.03$). Finally, in-hospital mortality was significantly higher in patients with TAWT group during the clinical course of SCM ($p = 0.009$).

Conclusion: TAWT in patients with SCM is not uncommon. Patients with SCM and systemic inflammation with hemodynamic instability might be susceptible to TAWT, which is often associated with cardiac complications. These patients showed worse prognosis compared to those without TAWT during recovery from SCM.

1. Introduction

Stress cardiomyopathy (SCM) is generally characterized by transient systolic dysfunction of the apical and/or mid-segments of the left ventricle (LV) that mimics myocardial infarction [1–3]. SCM is increasingly observed in patients with physical stressors such as procedures, surgeries, or management in intensive care unit [4–6]. In addition, reports are increasing of various acute complications including heart failure, arrhythmias, apical thrombus, and cardiogenic shock [7,8]. The clinical course of SCM shows a spectrum from full recovery to death. Although reports on SCM are increasing, the nature of SCM remains ill-defined. Recently, transient apical wall thickening (TAWT), which mimics apical hypertrophic cardiomyopathy during recovery from SCM, has been reported [9–12]. This phenomenon developed during recovery of SCM patients who showed a marked increase in LV apical wall thickness that subsequently resolved [9–12]. However, current data consist of isolated case reports, and the incidence and clinical impact for these patients have not been adequately assessed. Thus, the aim of this study was to evaluate the prevalence, profiles, and impact of TAWT on the clinical course of SCM.

2. Methods

2.1. Study design and patient selection

A total of 429 patients who were consecutively included in SCM registry at the Severance Hospital (Yonsei University College of Medicine, Seoul, Republic of Korea) from January 2009 to December 2013 were reviewed and retrospectively analyzed. The SCM registry consists of patients who were suspected to have SCM with regional wall motion abnormality (RWMA) that was not compatible with typical coronary

Abbreviations: SCM, stress cardiomyopathy; LV, left ventricle; TAWT, transient apical wall thickening; RWMA, regional wall motion abnormality; EF, ejection fraction; WMSI, wall motion score index; RVSP, right ventricular systolic pressure.

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artery territory in echocardiography and with an antecedent trigger factor, according to previously described criteria: 1) development of typical apical ballooning or inverted apical ballooning on echocardiography; 2) no evidence of obstructive epicardial coronary artery disease, including no history of angina and a normal coronary angiogram or myocardial perfusion scan; 3) the presence of a definite antecedent stressor, identified by the attending physicians; and 4) the absence of preexisting cardiomyopathy, head trauma, intracranial bleeding, pheochromocytoma, or catecholaminergic inotropic medication before echocardiography [7].

The patient selection processes is outlined in Fig. 1. Patients were classified into the typical type of SCM (n = 341) and or the atypical type (n = 88) based on echocardiographic findings. The typical type was defined as having RWMA predominantly involved at the apical portion, with the apical ballooning configuration of the LV in systole. The atypical type was defined as transient LV hypokinesia restricted to the mid-ventricular or basal segments without involvement of the apex. Among 341 patients with typical SCM, 232 patients had undergone serial follow-up echocardiography. Among these 232 patients, 124 patients who showed full recovery of RWMA on follow-up echocardiogram were included in this study.

TAWT was defined using the following criteria: 1) normal diastolic LV maximal apical wall thickness (≤10 mm) at the time of SCM diagnosis, 2) increased LV maximal apical wall thickness ≥13 mm in follow-up echocardiography, and 3) LV maximal apical wall thickness recovery to ≤10 mm in the last follow-up echocardiography. We divided the study population into two groups and compared the clinical and echocardiographic parameters. Group 1 was defined as SCM patients who showed TAWT during the recovery phase (n = 17) and group 2 included SCM patients without TAWT (n = 107). The Institutional Review Board of Yonsei University College of Medicine approved this study, which was conducted in compliance with the Declaration of Helsinki.

2.2. Clinical and laboratory findings

Demographic data and clinical characteristics were recorded including age, sex, initial presentation, past medical history, and trigger factors. ECG at the time of SCM presentation and serial follow-up electrocardiograms were analyzed. Cardiac biomarkers, including creatinine kinase MB and troponin-T, C-reactive protein (CRP), N-terminal prohormone of brain natriuretic peptide (NT-pro BNP), white blood cell count, and hemoglobin, were measured at the time of SCM presentation.

2.3. Echocardiography

Comprehensive transthoracic echocardiography was performed using commercially available equipment. Standard two-dimensional measurements were obtained as recommended by the American Society of Echocardiography [13]. LV ejection fraction (EF) was calculated using the modified Simpson method from the apical four- and two-chamber views [13]. The wall motion score index (WMSI) was derived by dividing the sum of the wall motion scores by the number of 17 visualized segments. For determining the apical wall thickness, the apical wall thickness was measured from the standard apical 4 and 2 chamber views, and we selected the largest values as maximal apical wall thickness. Detection of right ventricular involvement was visually evaluated [14]. For Doppler assessment, mitral inflow velocities were traced, and the following variables were obtained from the apical window: peak velocity of early diastolic mitral inflow (E), late diastolic mitral inflow (A), and deceleration time of the E velocity. Early diastolic mitral annular velocity (e′), late diastolic mitral annular velocity, and systolic mitral annular velocity (S′) were measured from the apical four-chamber view with a 2- to 5-mm sample volume placed at the septal corner of the mitral annulus [15]. Tricuspid and mitral valve regurgitation grade was measured. Right ventricular systolic pressure (RVSP) was calculated from the Doppler-derived velocity of the tricuspid regurgitation jet with the simplified Bernoulli equation and mean right atrial pressure. Recommendations of serial echocardiography for patients who enrolled in our SCM registry are as follows; 1) 7–14 days after the diagnosis, 2) a day before discharge, and 3) 6 months and 1 year after the recovery of RWMA. All echocardiographic images were digitally recorded by experienced echocardiographers and reviewed by two independent experienced cardiologists.

2.4. Clinical follow-up

Clinical follow-up data on SCM patients were collected during hospital admission. Cardiac complications were defined as significant arrhythmia, pulmonary edema, cardiogenic shock, or LV thrombus formation during the follow-up period. Significant arrhythmia was defined as newly

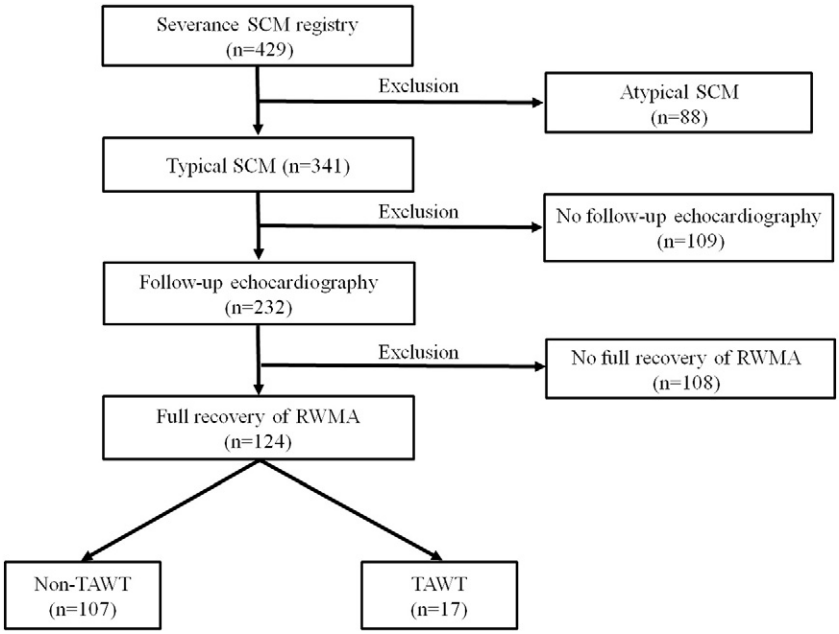

Fig. 1. Flow diagram of the study population. SCM = stress cardiomyopathy; RWMA = regional wall motion abnormality; TAWT = transient apical wall thickening.