

LAD Coronary Artery Myocardial Bridging and Apical Ballooning Syndrome

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OBJECTIVES This study sought to evaluate the prevalence and potential role of myocardial bridging in the pathogenesis of apical ballooning syndrome (ABS).

BACKGROUND ABS is characterized by reversible left ventricular dysfunction, frequently precipitated by a stressful event, but the pathogenesis remains still unclear.

METHODS Forty-two consecutive patients (40 female, mean age 66 ± 7 years) with ABS underwent echocardiography, cardiac magnetic resonance, coronary angiography (CA) with intravascular ultrasound, and computed tomography angiography (CTA). Myocardial bridging was diagnosed by CA when a dynamic compression phenomenon was observed in the coronary artery and by CTA when a segment of coronary artery was completely (full encasement) or incompletely (partial encasement) surrounded by the myocardium. The prevalence of myocardial bridging detected by CTA and CA in ABS patients was compared with 401 controls without ABS who underwent both CTA and CA.

RESULTS Myocardial bridging by CTA was observed in 32 ABS patients (76%): 23 with partial encasement and 9 with full encasement. All myocardial bridging was located in the mid segment of the left anterior descending coronary artery (LAD) with a mean length of 17 ± 9 mm. CA revealed myocardial bridging in 17 subjects (40%) (9 with partial encasement and 8 with full encasement by CTA). All subjects in which dynamic compression was observed by CA showed myocardial bridging by CTA, while none of the subjects with negative findings for myocardial bridging by CTA revealed dynamic compression by CA. Compared with controls, ABS patients showed a significant higher prevalence of myocardial bridging in the LAD either by CA (40% vs. 8%; $p < 0.001$) or by CTA (76% vs. 31%; $p < 0.001$).

CONCLUSIONS Our study showed that myocardial bridging of the LAD is a frequent finding in ABS patients as revealed both by CA and, mostly, by CTA, suggesting a role of myocardial bridging as potential substrate in the pathogenesis of ABS. (J Am Coll Cardiol Img 2013;6:32–41) © 2013 by the American College of Cardiology Foundation

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Apical ballooning syndrome (ABS), also known as typical takotsubo syndrome, is characterized by reversible left ventricular (LV) dysfunction that is frequently precipitated by a stressful event (1,2). The etiology of ABS remains unclear, and ABS may be due to catecholamine-mediated myocardial stunning, coronary spasm, coronary emboli with spontaneous fibrinolysis, or microvascular dysfunction (1–5). Although the coronary arteries of ABS patients are described as normal, an association between ABS and myocardial bridge has been recently reported (6–8). In this regard, myocardial bridging is usually considered a normal variant with no hemodynamic relevance, but it has been associated also with relevant clinical complications, such as myocardial ischemia and myocardial infarction (9–13). This study was designed to explore the hypothesis that an underlying myocardial bridging in the left anterior descending coronary artery (LAD) could represent a pathophysiological substrate of ABS.

METHODS

Study population. The study population consisted of 42 (40 female, 95%, mean age 66 ± 7 years) consecutive Caucasian patients referred to the Division of Cardiology of the University of Padova, Padova, Italy, between September 2005 and June 2010, who fulfilled the Mayo Clinic diagnostic criteria for ABS (2). Apical sparing patterns of Takotsubo cardiomyopathy were excluded. Based on our study protocol, all patients underwent electrocardiography, echocardiography, cardiac catheterization with intravascular ultrasound (IVUS), and computed tomography angiography (CTA). The study was approved by the institutional review board, and all patients gave their informed consent.

Electrocardiogram. Recorded measurements included heart rate, ST-segment deviation, T-wave inversion, and corrected QT interval. The ST-segment elevation/depression was defined as a deviation of >1 mm in amplitude measured 80 ms after the J point in ≥ 2 contiguous leads and T-wave inversion as negative T waves >1 mm in amplitude in ≥ 2 contiguous leads.

Echocardiography. Two-dimensional echocardiography was performed in all patients on admission, before discharge, and 1 month later to evaluate the complete normalization of LV systolic function. LV volume, LV ejection fraction, and regional wall motion score index were calculated (14). Images were analyzed by 2 independent observers (M.P.M. and C.S.), blinded to the clinical and

instrumentals data. Disagreements were resolved by consensus.

Cardiac catheterization. Cardiac catheterization was performed in all patients, on admission in 31 patients (74%) and within 72 h after hospital admission in the remaining, using the femoral or radial approach with 6-French catheters for both LV and coronary angiography. Three different views for the right coronary artery and 6 for LAD were used. The degree of LAD stenosis was evaluated visually and by quantitative coronary angiography. To determine whether a single coronary artery could supply the entire akinetic area of the left ventricle, the presence and length of the LAD recurrent segment on left lateral projection were recorded. We defined a LAD recurrent segment as that part of the vessel between the apical point of the LAD and the visible end of the coronary artery, as previously reported in detail (15) (Fig. 1A). Myocardial bridging was diagnosed when an angiographic dynamic compression “milking effect” phenomenon (i.e., systolic compression and complete or partial decompression in diastole) was observed in the coronary artery (9–11) (Figs. 1B and 1C). Invasive provocative tests were not performed during coronary angiography. Transient systolic occlusion of septal branches arising from the compressed segment during systole was also assessed (16). IVUS of the LAD was performed with a 40-MHz catheter (Atlantis SR Pro 2, Boston Scientific, Natick, Massachusetts) and a 30-MHz catheter (Volcano Corp., Rancho Cordova, California) with a monitored pullback of 0.5 mm/s. IVUS data were evaluated to exclude severe coronary artery disease, coronary dissection, or intramural hematoma. All images were independently reviewed by 2 investigators (G.T. and M.N.) blinded to the clinical information. Disagreements were resolved by consensus.

Computed tomography angiography. CTA was performed using a 64-slice computed tomography scanner (Sensation 64, Siemens Medical Solutions, Forchheim, Germany) with the following parameters: slices/collimation, 32/2/0.6 mm; rotation time, 330 ms; effective temporal resolution (with 180° algorithm), 165 ms, 120 kV, 600 to 900 mA; submillimeter isotropic voxel (reconstructed slice thickness of 0.75 mm; reconstruction increment of 0.4 mm), and field of view 140 to 160 mm. Patients with a heart rate >65 beats/min received intravenously 5 to 10 mg of metoprolol tartrate 5 min

ABBREVIATIONS AND ACRONYMS

ABS	= apical ballooning syndrome
CTA	= computed tomography angiography
ECG	= electrocardiographic
IVUS	= intravascular ultrasound
LAD	= left anterior descending coronary artery
LV	= left ventricular

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