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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

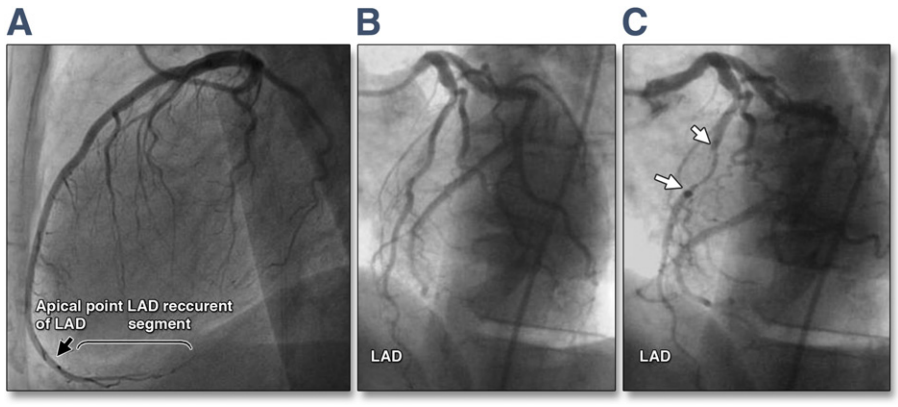

Figure 1. Recurrent Segment of the LAD and Myocardial Bridging by Angiography

Recurrent segment of the left anterior descending coronary artery (LAD) is defined as the part of the vessel between the apical point of the LAD and its visible end of the coronary artery **(A)**. Left lateral projection of the left coronary artery showing a normal lumen of the LAD middle segment in the end-diastolic phase **(B)**, and its dynamic compression in the end-systolic phase **(arrows) (C)**.

before the scan (17). Patients with a systolic blood pressure >100 mm Hg received 5 to 10 mg of isosorbide dinitrate. A 80-ml bolus of nonionic iodinated contrast agent (iomeprol, Iomeron 400, Bracco, Milan, Italy), depending on scan range, was injected at a flow rate of 5 ml/s using an automatic power injector (Stellant, MedRAD, Pittsburgh, Pennsylvania). To optimize intraluminal enhancement of the coronary arteries, the beginning of the scan was synchronized with the passage of the contrast bolus by using the bolus tracking technique with a region of interest in the ascending aorta. The scan started automatically with a 6-s delay after a threshold of 100 HU was reached within the region of interest. Data were retrospectively reconstructed at end-diastolic (65% to 80% of the R-R interval) or end-systolic phase (25% to 40%) when deemed necessary. Images were analyzed by a dedicated workstation (Extend Brilliance TM Workspace, Version 3.0.13200, Philips Medical System, Cleveland, Ohio). All data were analyzed by post-processing tools such as multiplanar reformation, curved multiplanar reformation, maximum intensity projection, and volume-rendering reformation to visualize myocardial bridging. Myocardial bridging was diagnosed when a segment of coronary artery was located within the interventricular groove and was in contact with LV myocardium, completely or incompletely surrounded by the myocardium at axial and multiplanar reformation images. Myocardial bridging was classified as 1 of 2 types according to the extent of vessel encasement by the myocardium: 1) myocardial bridging with partial encasement or 2) myocardial bridging with full encasement (16)

Figure 2. Classification of Myocardial Bridging by CTA

Normal pattern of the left anterior descending coronary artery (LAD), which is embedded in the epicardial fat at some distance from the interventricular gorge **(A)**. Partial encasement myocardial bridging is defined as the LAD being within the interventricular gorge in direct contact with the myocardium and partially surrounded by muscle **(B)**. Full encasement myocardial bridging is defined as the LAD being deeply in the myocardium and completely surrounded by overlying muscle **(C)**. The **arrows** indicate LAD.

Table 1. Baseline Clinical Characteristics of the Study Population on Admission

Age, yrs	66 ± 7
Female	40 (95)
Body mass index, kg/m^2	25.2 ± 3.5
Coronary risk factors	
Hypertension*	32 (76)
Dyslipidemia†	13 (31)
Family history of coronary disease‡	7 (16)
Current smoking	6 (14)
Diabetes mellitus	3 (7)
Atrial fibrillation	4 (9)
Depression/anxiety	13 (31)
History of cancer	10 (24)
Medication on admission	
Aspirin	5 (12)
Warfarin	2 (4)
Angiotensin-converting enzyme inhibitor	14 (33)
Beta-block/calcium channel blocker	4 (9)
Angiotensin II receptor blocker	4 (9)
Diuretic	2 (4)
Digoxin	1 (2)
Oral hypoglycemic agent	2 (4)
Benzodiazepine	11 (6)
Serotonin reuptake inhibitor	6 (14)
Clinical presentation	
Chest pain	38 (90)
Dyspnea	9 (21)
Syncope or pre-syncope	7 (16)
Shock	4 (9)
Palpitations	3 (7)
Stressful event reported	33 (78)
Emotional stressor§	29 (69)
Physical stressor‖	4 (9)
Peak troponin I, μg/l	2.5 (1.9–4.0)

Values are mean ± SD, n (%), or median (interquartile range). *Systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg. †Hypercholesterolemia >220 mg/dl and/or hypertriglyceridemia >150 mg/dl. ‡Previous myocardial infarction, coronary intervention, or stable angina in first-degree relatives. §Relative's death, severe illness, or injury of a family member; stressful hospital stay; distressing argument. ‖Physical exercise, chemotherapy.

(Fig. 2). In each group, the location and length of the tunneled segment were analyzed. The location of the bridge was classified as proximal (before the first septal branch), middle (after the first septal branch), or distal. Images were analyzed by 2 observers (A.L.A. and F. Cademartiri) who were blinded to the clinical information; disagreements were resolved by consensus.

Control group. The prevalence of myocardial bridging in ABS patients was compared with a large cohort of 401 Caucasian subjects without ABS who underwent both CTA and cardiac catheterization for suspected coronary artery disease. They were recruited from a database and randomly selected using a computerized method (SAS software, version 9.1.3, SAS Institute, Cary, North Carolina) with the same mean age, percentage of female patients, and prevalence of hypertension (defined as systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg) as those in the study group. Angiographic and CTA data were blinded during the selection process. The indications for CTA were as follows: typical angina (n = 157); atypical angina (n = 114); nonanginal symptoms (n = 85); and dyspnea (n = 45). Patients with cardiomyopathy, valvular diseases, coronary stents, or bypass grafts were excluded. The CTA dataset of the controls was reviewed by using the same methodology as that for the study population and analyzed independently by 2 observers (A.L.A. and F. Cademartiri) who were blinded to clinical information. Cardiac catheterization data were

Table 2. Electrocardiographic, Laboratory and Hemodynamic Data of the Study Population

Admission electrocardiographic findings	
Sinus rhythm	39 (93)
ST-segment elevation	32 (76)
ST-segment depression	2 (4)
T-wave inversion	8 (19)
Echocardiographic findings	
Acute LVEF, %	41 ± 5
LVED, ml/m^2	65 ± 14
Wall motion score index	2.2 ± 0.3
Septal hypertrophy	24 (57)
Concentric LV hypertrophy	12 (28)
Dynamic LV obstruction	6 (14)
Follow-up LVEF, %	64 ± 4
Coronary angiography and LV angiographic findings	
LVEF, %	44 ± 6
LVEDV, ml/m^2	74 ± 16
Dynamic LV obstruction	6 (14)
LAD recurrent segment	33 (78)
Myocardial bridging	17 (40)
Maximal LAD stenosis	
Visual estimation, %	30 ± 4
QCA, %	32 ± 5
IVUS findings at minimal lumen site of the LAD	
External elastic membrane CSA, mm^2	11 ± 4.8
Lumen CSA, mm^2	7 ± 2.4
Plaque CSA, mm^2	6 ± 2.6
Lumen area stenosis, %	33 ± 3

Values are n (%) or mean ± SD.
CSA = cross-section area; IVUS = intravascular ultrasound; LAD = left anterior descending coronary artery; LV = left ventricular; LVEDV = left ventricular end-diastolic volume; LVEF = left ventricular ejection fraction; QCA = quantitative coronary analysis.

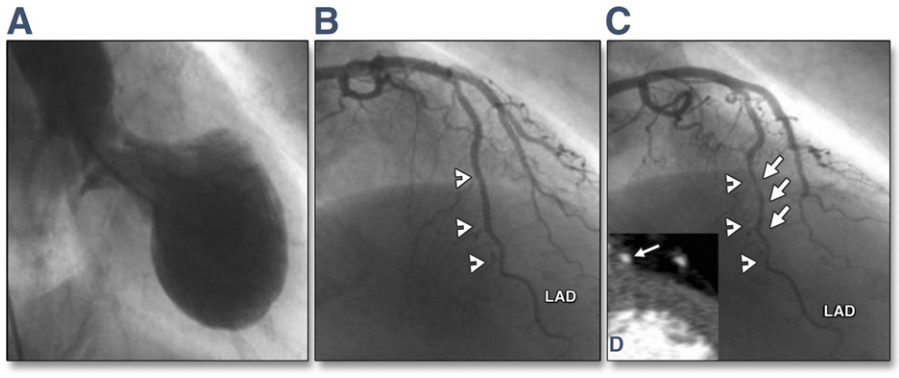

Figure 3. Partial Encasement Myocardial Bridging With Transient Occlusion of Septal Branches

Cardiac catheterization showing apical ballooning **(A)**, normal lumen of the middle segment of the left anterior descending coronary artery (LAD) in the end-diastolic phase **(B)**, and its dynamic compression in the end-systolic phase **(arrowheads)**. Of note, transient occlusion of septal branches is apparent during the end-systolic phase **(arrows and arrowheads) (C)**. **(D)** Computed tomography angiography (short-axis view) showing the presence of myocardial bridging of the LAD with partial encasement myocardial bridging within the interventricular gorge **(arrow)**.

also reviewed by 2 observers (G.T. and M.N.) to evaluate the prevalence of dynamic compression by using the same definition as that for the study population.

Follow-up. All patients were followed during the hospital stay for the occurrence of following adverse events: cardiac death; arrhythmias; heart failure; and cardiogenic shock. At least 1 month after discharge, all patients were readmitted for electrocardiographic (ECG) and echocardiographic evaluation. The adverse events recorded during long-term follow-up included cardiac or noncardiac death, hospitalization for recurrence of ABS, chest pain, and heart failure.

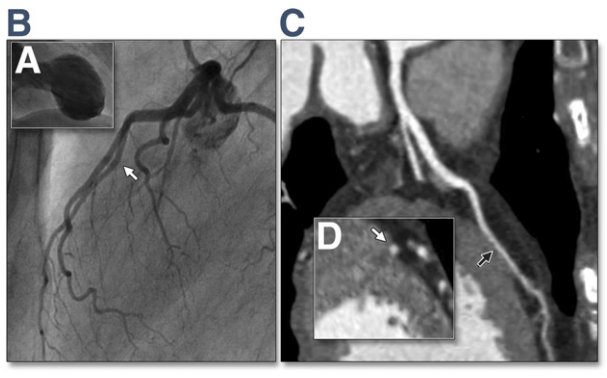

Figure 4. Myocardial Bridging With Partial Encasement by CTA Missed by Angiography

Typical apical ballooning is demonstrated at left ventriculography **(A)**. Coronary angiography showing normal lumen of the LAD during end-systolic phase in the absence of dynamic compression **(arrow) (B)**. Computed tomography angiography (CTA) showing the myocardial bridging **(arrow)** with partial encasement in multiplanar reformats, longitudinal view **(C)** and short-axis view **(arrow) (D).** Of note the typical deviation and straightening of the mid left anterior descending coronary artery (LAD) by CTA longitudinal view.

Statistical analysis. Categorical variables were expressed as the number and percentage of patients. Continuous data were estimated as mean ± SD or as median and interquartile range for non-normally distributed data. Normality was assessed using the Shapiro-Wilk test. Categorical variables were compared by the chi-square test or Fisher exact test, as appropriate. Continuous variables were compared by the Student *t* test or Wilcoxon rank sum test, as appropriate. The chi-square test was used to compare the prevalence of myocardial bridging in ABS patients and controls. The chi-square test or Fisher exact test and Student *t* test or Wilcoxon rank sum test were used to compare categorical variables and continues variables, respectively, in patients with and without myocardial bridging by cardiac catheterization and by CTA. A p value <0.05 was considered significant. Intraobserver and interobserver agreement for the presence of myocardial bridging by CTA were assessed by the kappa test. Data were analyzed with SAS 9.1.3 for Windows (SAS Institute Inc.).

RESULTS

Clinical characteristics. Baseline clinical characteristics of ABS patients are reported in Table 1. The vast majority of the patients presented with chest pain (n = 38; 90%); in 33 patients (78%), a significant stressful event occurring <24 h before presentation could be identified. ECG parameters

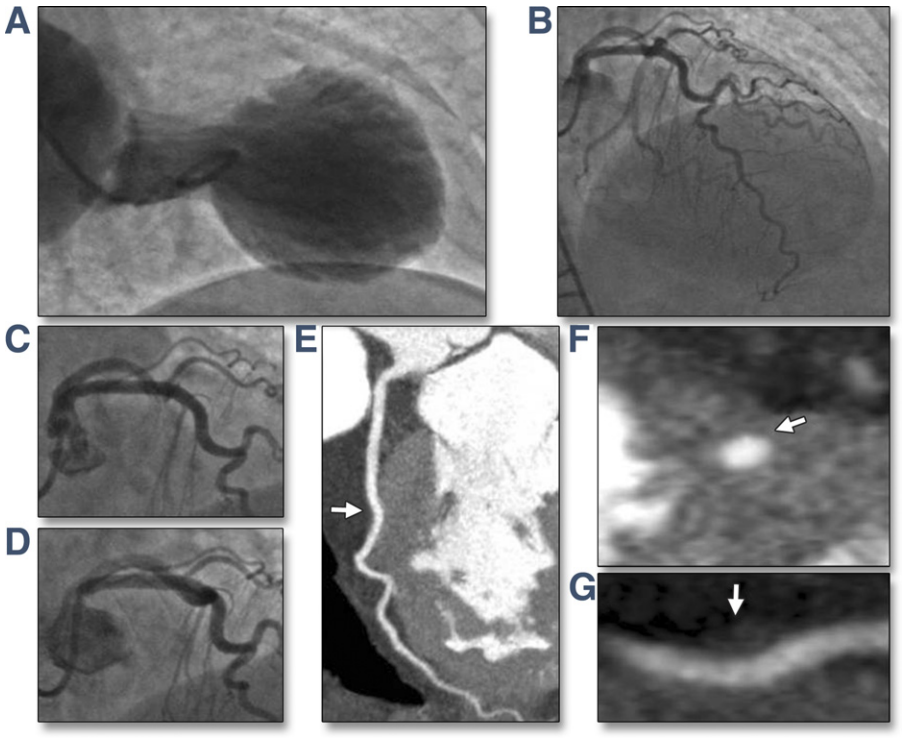

Figure 5. Myocardial Bridging With Full Encasement by CTA Missed by Angiography

Left ventriculography showing apical ballooning **(A)**. Coronary angiography showing normal lumen of the LAD **(B)** during end-diastolic **(C)** and end-systolic **(D)** phases without the milking effect. CTA reveals the presence of myocardial bridging of the middle segment of the LAD with full encasement **(arrows)** during multiplanar reformats, longitudinal **(E and G)** and short-axis view **(F)**. Abbreviations as in Figure 4.

are reported in Table 2. ST-segment elevation was the most common ECG abnormality at the time of presentation (n = 32; 76%). Significant ECG changes were observed during hospitalization including resolution of ST-segment elevation and progressive T-wave inversion with the nadir occurring within 3 days since the admission and concomitant prolongation of the corrected QT interval. Echocardiographic data are summarized in Table 2.

Cardiac catheterization findings. All ABS patients had mild nonobstructive coronary artery disease by coronary angiography and IVUS. Details of angiography and IVUS are reported in Table 2. Neither angiographic nor IVUS examination of the LAD revealed any evidence of unstable intracoronary lesions, plaque rupture, thrombus fragmentation, or features suggestive of distal embolization. A LAD recurrent segment, with a mean length of 26.8 ± 8.4 mm was observed in 33 patients (78%). Myocardial bridging was detected in 17 patients (40%), which was located in the mid segment of the LAD in all cases. Transient systolic occlusion of septal branches arising from the segment with myocardial bridging was observed in 4 patients (9%) (Fig. 3). There was no statistically significant correlation between the presence of myocardial bridging and baseline clinical and instrumental findings.

CTA findings. Of 42 patients with ABS, 32 (76%) showed myocardial bridging by CTA (23 partial encasement and 9 full encasement). Fifteen patients (36%) with no myocardial bridging by coronary angiography showed the presence of myocardial bridging by CTA (14 partial encasement and 1 full encasement) (Figs. 4 and 5). All patients in whom dynamic compression (milking effect) was observed by coronary angiography showed myocardial bridging by CTA, and none of the patients with negative findings for myocardial bridging by CTA revealed dynamic compression. Of the 17 patients exhibiting dynamic compression at coronary angiography, 9 showed a partial encasement and 8 showed a full encasement pattern by CTA (Figs. 6 and 7). All myocardial bridging was located in the mid portion of the LAD with a mean length of 17 ± 9 mm. There was no statistically significant relationship between the presence of myocardial bridging by CTA and other baseline clinical characteristics. Intraobserver and in-

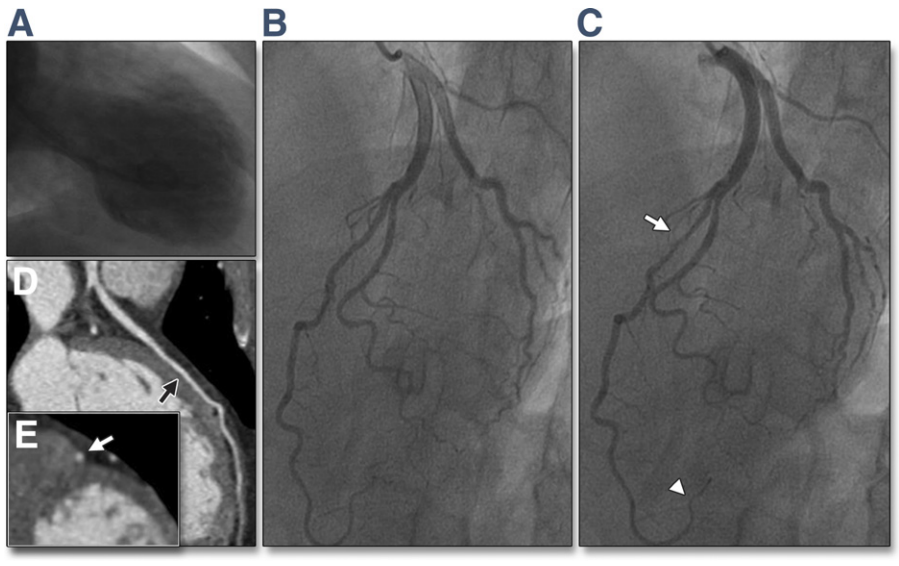

Figure 6. Myocardial Bridging With Partial Encasement and Dynamic Compression

Left ventriculography showing apical ballooning **(A)**. Coronary angiography showing normal lumen of the middle segment of the LAD in the end-diastolic phase **(B)**, and its dynamic compression in the end-systolic phase **(arrow)**; of note is the presence of a recurrent segment of the LAD **(arrowhead) (C)**. CTA confirms the presence of myocardial bridging with partial encasement in both multiplanar reformats and longitudinal **(arrow) (D)** and short-axis **(arrow) (E)** views. Abbreviations as in Figure 4.

terobserver agreement for any encasement were kappa = 1 and kappa = 0.98, respectively.

Follow-up. In-hospital events occurred in 8 patients (19%), such as LV apical thrombi in 5 (12%) and atrial fibrillation in 3 (7%). No life-threatening ventricular arrhythmias, including torsade de pointes and ventricular fibrillation, were recorded during hospitalization. At discharge, 66% patients were treated with aspirin, 71% with a beta-blocker, 24% with a calcium antagonist, 69% with an angiotensin-converting enzyme inhibitor, 39% with a statin, and 12% with an oral anticoagulant. At 1 month of follow-up, all patients showed resolution of ECG abnormalities and LV function recovery by echocardiography. During follow-up (mean 36 ± 5 months), clinically significant events occurred in 3 patients (7%). Of these, an ABS recurrence developed in 2 patients (1 patient showed full encasement myocardial bridging) and 1 had recurrent episodes of chest pain in the absence of ECG and enzymatic changes. These patients were not taking a beta-blocker at the time of hospital readmission.

Control group. There were no significant differences in baseline clinical characteristics between ABS patients and controls (Online Table 1). Of 401 patients, 124 (31%) presented myocardial bridging by CTA: partial encasement (n = 112) and full encasement (n = 12) with a mean length of 16 ± 8 mm; in 32 (8%), dynamic compression (milking effect) was observed by cardiac catheterization (4 with partial and 28 with full encasement). All patients in whom dynamic compression was observed by coronary angiography showed myocardial bridging by CTA. Compared with controls, ABS patients showed a significantly higher prevalence of myocardial bridging in the LAD, either by cardiac catheterization (40% vs. 8%; $p < 0.001$) or by CTA (76% vs. 31%; $p < 0.001$). Moreover, ABS patients showed a significantly higher prevalence of LAD recurrent segment compared with controls (78% vs. 31%; $p < 0.001$). The morphological characteristics of myocardial bridging by conventional coronary angiography and by CTA among patients with ABS and controls are reported in Table 3.

DISCUSSION

The results of our prospective study suggest that myocardial bridging of the LAD is very frequent in ABS patients, as revealed both by coronary angiography and mostly by CTA compared with controls. Myocardial bridging is usually considered a congenital coronary anomaly with no hemodynamic relevance, but it has been associated with different clinical scenarios, such as typical or atypical angina and myocardial infarction (9–13). From the pathophysiological point of view, myocardial bridging–related myocardial ischemia may be attributed to a

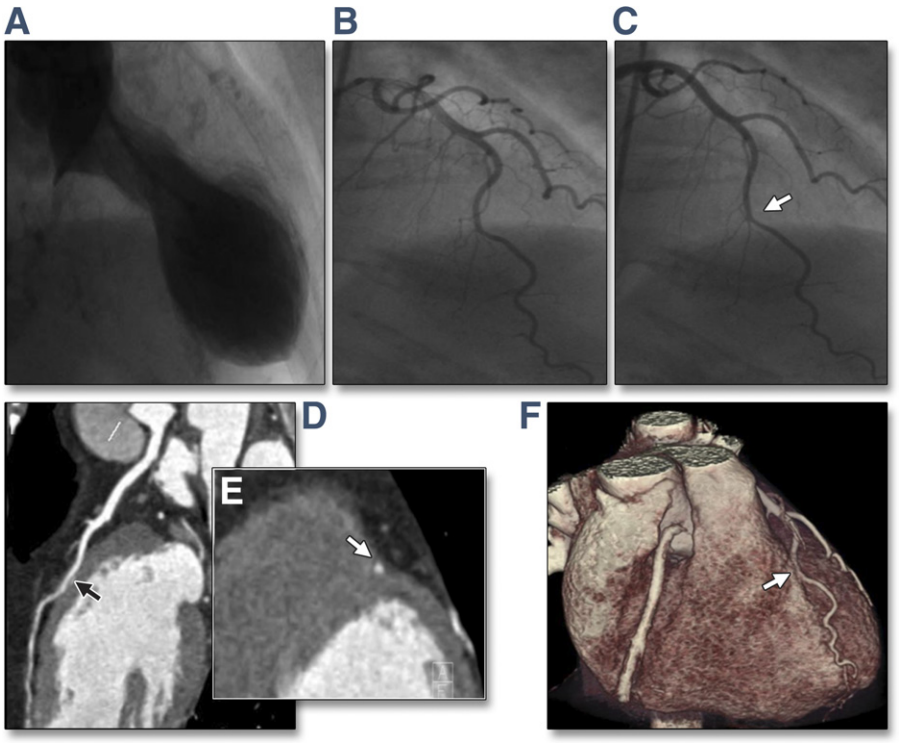

Figure 7. Myocardial Bridging and Minimal Dynamic Compression

Left ventriculography showing apical ballooning **(A)**. Coronary angiography showing a normal lumen of the middle segment of the LAD in the end-diastolic phase **(B)**, and its minimal dynamic compression in the end-systolic phase **(arrow) (C)**. CTA showing the myocardial bridging with partial encasement in multiplanar reformats, longitudinal view **(arrow) (D)**, short-axis view **(arrow) (E),** and in volume-rendering reformation **(arrow) (F)**. Abbreviations as in Figure 4.

combination of different factors: sudden tachycardia (compromising diastolic filling of coronary arteries); increased contractility; coronary spasm and systolic kinking of the coronary arteries (leading to endothelial damage, platelet activation and thrombosis, or mechanical reduction of the blood flow) (9–13).

The true prevalence of myocardial bridging is not fully known because it is usually underdiagnosed by conventional angiography. CTA should be the preferred noninvasive imaging modality because it can visualize not only the coronary lumen but also the vessel walls and the neighboring myocardium. Moreover, myocardial bridging can be identified by CTA even when no significant "milking effect" and/or change in vessel course at conventional coronary angiography are present. Classically, the prevalence of myocardial bridging ranges from 0.8% to 4.9% in angiographic series and 4% to 44% in CTA studies (18–22), depending on the heterogeneity of the population (including sex and age), the type of CTA scanner, and the inclusion/exclusion of superficial myocardial bridging. The greater prevalence of myocardial bridging in ABS patients compared with controls suggests a possible association between myocardial bridging and ABS. Previously, a relationship between myocardial bridging and ABS was limited to single case reports or small case series (6–8). Lemaitre et al. (6) reported an angiographic rate of myocardial bridging at the level of mid LAD of 62.5% (5 of 8 patients with ABS), but this finding was not observed by others (1,2,23). However, these studies were retrospective, heterogeneous, and used only conventional angiography. Although the association may be fortuitous, myocardial bridging might represent a substrate of ABS. Indeed, emotional distress, a characteristic feature of ABS, increases sympathetic drive resulting in tachycardia, decreased diastole, and increased contractility, which aggravate systolic compression and/or kinking of myocardial bridging, particularly if LV hypertrophy is present. The result is the precipitation of symptoms in an otherwise asymptomatic individual with myocardial bridging as well as the onset of ABS.

The involvement of all the apical segments in ABS might be explained by a dynamic compression of a long recurrent distal LAD supplying a signif-

Table 3. Prevalence and Morphological Characteristics of Myocardial Bridging by CTA and Conventional Coronary Angiography Among Patients With ABS and Controls

	ABS Patients (n=42)	Controls (n=401)
CTA		
Patients with myocardial bridging	32 (76)	124 (31)
Location of myocardial bridging		
Proximal LAD	—	14/124 (11)
Middle LAD	32/32(100)	105/124 (85)
Distal LAD	—	5/124 (4)
Type of myocardial bridging		
Partial encasement	23/32 (72)	112/124 (90)
Full encasement	9/32 (28)	12/124 (10)
Length of myocardial bridging, mm	17 ± 9	16 ± 8
Conventional coronary angiography		
Patients with myocardial bridging (milking effect)	17 (40)	32 (8)
Location of myocardial bridging		
Proximal LAD	—	3/32 (9)
Middle LAD	17/17 (100)	28/32 (87)
Distal LAD	—	1/32 (3)

Values are n (%) or mean ± SD.
ABS = apical ballooning syndrome; CTA = computed tomography angiography; LAD = left anterior descending coronary artery.

icant area of the inferior LV segments as suggested by the high proportion (78%) of patients in our series presenting with an LAD recurrent segment along the diaphragmatic surface of the LV as well; this was also demonstrated by others (15). Our findings confirmed previous reports showing that dynamic compression of the vessel may also occur in segments without fully overlying muscle by the entrapment of myocardial bridging within the interventricular gorge (16). In addition, transient occlusion of prominent septal branches arising from or near the involved segment may occur (16).

Although our results indicate that myocardial bridging is a frequent finding in ABS, 24% of patients did not show any myocardial bridging. Several and controversial mechanisms have been proposed, but none of them seem to clarify the overall ABS cases. Thus, ABS might represent a common and unique pattern of LV dysfunction (apical ballooning), resulting from different concurrent pathological processes.

Study limitations. We acknowledge several limitations inherent to this study. First, the lack of stress testing for ischemia or inducibility of ABS, as assessed by cardiac magnetic resonance with dobutamine, may limit the full comprehension of the clinical relevance of myocardial bridging in our cohort of ABS patients. However, noninvasive tests have shown a low diagnostic accuracy in this subset of patients in whom the trigger of the disease is mostly acute distress (24). Second, we used as controls a large cohort of patients who underwent both coronary angiography and CTA because of suspected coronary artery disease. Although control patients presented similar clinical characteristics compared with ABS patients, they cannot be considered fully representative of the general population. Finally, larger studies are needed to confirm and extend the consistency of our data.

CONCLUSIONS

Our results, although not conclusive, are hypothesis generating: myocardial bridging might have a potential role in the pathogenesis of ABS, with promising implications for more focused management strategies, including beta-blockers or calcium antagonists.

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Key Words: apical ballooning syndrome ■ computed tomography angiography ■ coronary angiography ■ myocardial bridging ■ takotsubo cardiomyopathy.

▶APPENDIX

For a supplemental table, please see the online version of this article.