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Myocardial Bridging- Depiction by Conventional CAG/ CT Angiogram and Clinical Profile

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Abstract

Background: Myocardial bridging is a congenital condition in which a segment of an epicardial artery has an intramural course within the myocardium. The aim of the present study was to evaluate the prevalence, length, depth, and location of myocardial bridging of the coronary arteries using conventional/ 128-multi detector computed tomography coronary angiography and their clinical profile.**Material and Methods:**350 consecutive symptomatic patients underwent conventional / CT angiogram of coronary arteries over a 3 month period. Patients who had myocardial bridging were included in this study. Vessels involved, length & depth of bridging were studied. Other parameters like comorbidities, association with CAD were also studied.**Results:**Myocardial bridges were found in 30 (8.57%) of 350 patients. Among patients with myocardial bridging, males were 22 (73.3%), females were 8(26.6%). Mean age among these patients was 57.1 + 9.01 years. 5 patients (16.6%) had past history of CAD. All patients had myocardial bridging involving a single artery. Left anterior descending artery was involved in all (100%) of the patients. Mid LAD was most common location involving 23 (76.6%) patients, distal LAD in 5 (16.6%), proximal LAD in 2 (6.6%) patients. In CT CAG, Mean length of myocardial bridge was 14.53+3.04mm and mean depth was 3.04+0.69mm. whereas in conventional CAG, mean length of bridging was 17.9+3.4mm (p value<0.0001).**Conclusion:**We found that prevalence of myocardial bridging was 8.57%. Both conventional angiogram & CT angiogram are reliable in diagnosis of myocardial bridging. But, CT angiogram is noninvasive and can accurately locate the the site of myocardial bridging, and measure its thickness, course, and length.

Keywords: Myocardial bridge, CT angio, CAD.

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Introduction

The coronary arteries are normally located on the epicardial surfaces of the heart, surrounded by epicardial fat. Myocardial bridging, first described by Villa et al., is a congenital condition in which a segment of an epicardial artery has an intramural course.[1] The myocardial tissue covering the artery is termed a myocardial bridge, and the artery itself is called a tunneled segment. It is typically confined to a single vessel, usually the mid left anterior descending (LAD) artery. The reported frequency at autopsy ranges from 5% to 86%.[2–11] Myocardial bridging is usually asymptomatic and is traditionally considered to be a normal variant or benign coronary anomaly. However, clinical interest has been triggered by the observation that myocardial bridging may be a contributory factor in the development of myocardial ischemia, angina pectoris, myocardial infarction, arrhythmias, and even sudden death.[12–20] Those studies suggest that coronary compression by the myocardial bridge at systole could lead to a reduction in blood flow and subsequent ischemia. Furthermore, myocardial bridging

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may pose a technical challenge during coronary bypass surgery, because intraoperative echocardiography may be necessary to expose the intramuscular coronary artery.[5–7,21,22]

The diagnosis of MB usually is made with conventional coronary angiography. Conventional coronary angiography indirectly indicates the anomaly by demonstrating systolic compression of the tunneled segment and a localized change in direction of the vessel course towards the ventricle. Although conventional angiography is recognized as the reference standard examination for the diagnosis of myocardial bridging, its detection rate is <5%, which is significantly lower than that of autopsy studies.[2,3] This difference has been suggested to be caused by superficial MB not constricting the tunneled segment during systole to an extent that allows the indirect identification of MB with conventional coronary angiography. Detection has improved with the application of newer, more sensitive imaging techniques, such has intravascular ultrasound (IVUS), intracoronary Doppler (ICD) ultrasound, and multidetector computed tomography (MDCT).[2,3,8,9]

The aim of the present study was to evaluate the prevalence of myocardial bridging using conventional coronary angiogram/CT angiogram and the clinical profile of patients with myocardial bridging.

Materials and Methods

It was an observational analysis that was performed from march 2019 to May 2019 in cardiology department, Nizams institute of medical sciences, Hyderabad. Patients who underwent conventional / CT angiogram of coronary arteries were enrolled and patients who had myocardial bridging in either of angiograms- 30 patients, were included in the study. Patients included 22 males, 8 females. Age ranged from 40 to 76 years (mean: 57.1years). all patients were symptomatic at the time of presentation- either chronic stable angina or unstable angina.

Detailed clinical & medical data (history of Diabetes, Hypertension, past history of CAD) were also recorded. hypertension was defined as current use of antihypertensive medications or known but untreated hypertension (blood pressure >140/90 mm Hg). Diabetes was defined as currently receiving insulin or oral hypoglycemic agents.

Treadmill ECG-stress test

The treadmill exercise ECG-stress test was performed and interpreted according to the AHA guidelines.[22] The test was regarded as “positive” for anterior myocardial ischemia, if STsegment depression of greater than 0.1mV was found in at least one or more anterior leads V2–V5.[22]

Three hours before the CCTA examination, oral beta blockers (metoprolol 50 mg or propranolol 20 mg) were given to all patients who were not already taking them to maintain heart rates at <70 beats/minutes and thereby improve image resolution. To facilitate adequate breath-hold, the patients were connected to an oxygen mask and asked to hyperventilate. All examinations were performed with 128-slice MDCT (GE Healthcare, Naarden, The Netherlands) with dedicated cardiac reconstruction software and electrocardiography (ECG) triggering. Patients were scanned in the supine position. Nonionic contrast agent (Ultravist 370; Schering AG, Berlin, Germany), 80–120 mL, was injected into the antecubital vein at a rate of 4–6 mL/second with the use of a single-head injector (Envision CT; Medrad, Indianola, PA) without saline flush. SmartPrep software (General Electric Medical Systems, Milwaukee, WI) was used to calculate the time to peak arterial contrast enhancement.

CT images were analyzed for the direct signs length, depth, and degree of systolic compression, while conventional angiograms were analyzed for the indirect signs step down–step up phenomenon, milking effect, and systolic compression of the tunneled segment.

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The scan volume was determined from the tracheal bifurcation to the diaphragmatic surface of the heart. Scan parameters were as follows: 140 KV, 400 mA, 0.4 second rotation speed, 64 * 0.625 mm detector array. Single-slice collimation was 1 mm with 50% overlap. Pitch, which depended on the heart rate, averaged 0.3. Images were reconstructed in different phases of the cardiac cycle with the use of a retrospective ECG-gated algorithm with 1-mm thick sections and a 0.4-mm interval. All images were transferred to a workstation with dedicated cardiac reconstruction software (GE Healthcare) for review in a blinded manner by 2 experienced CT radiologists, by consensus. The initial retrospective ECG-gated reconstruction was generated with the reconstruction window, starting at the end-diastolic phase (i.e, 70% of the R-peak interval). When the data were insufficient because of motion artifacts, additional reconstruction data were obtained in increments and decrements of 10%. All segments >2 mm in diameter belonging to the left main, LAD, left circumflex, and right coronary arteries were included, according to the modified classification of the American College of Cardiology/American Heart Association. The images were evaluated by axial scans, curved multiplanar reformations through the lumen of the coronary vessels, and 3-dimensional volume rendered visualization.

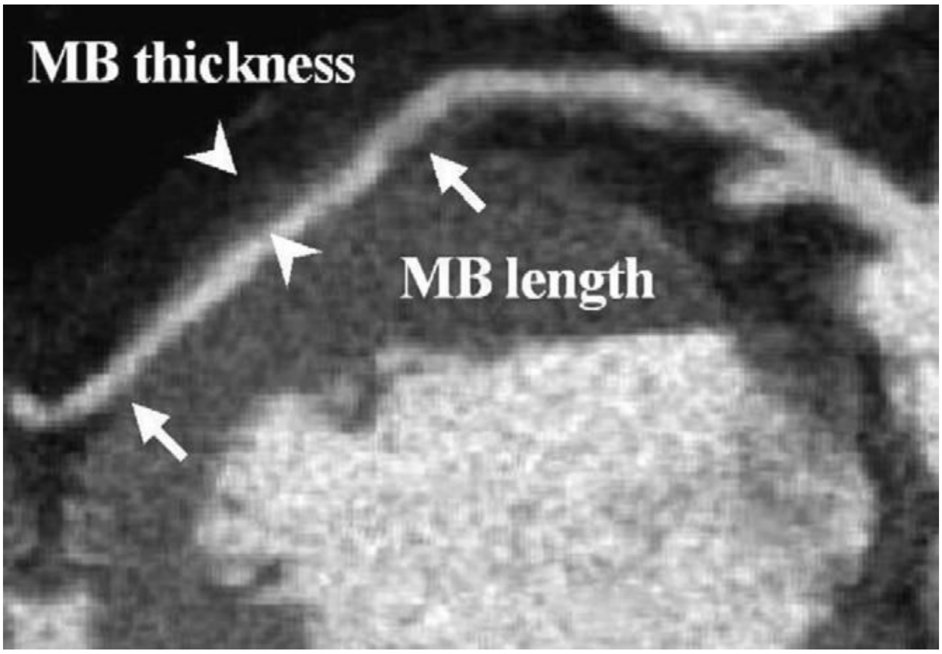

Figure 1: Measurements of MB on coronary MSCT. The thickness of the MB is evaluated as the widest part from the myocardial surface surrounded by epicardial adipose tissue to the contrast enhancement of the tunneled artery (arrowhead to arrowhead) on the CPR image. The length of the MB is also evaluated as the distance from the entrance to the exit of MB (arrow to arrow).

Conventional coronary angiography was performed by one cardiologist (F.R.E., with 13 years of experience in performing conventional coronary angiography) according to standard techniques, and multiple views were stored on a compact disc. Briefly, conventional coronary angiography was performed with a transradial or transfemoral approach, and at least two different projections for each coronary artery were obtained after intracoronary application of iodinated contrast material. The angiograms were evaluated in consensus by two cardiologists (P.K. and F.R.E., with 4 and 13 years of experience in interventional cardiology, respectively) who were both blinded to the clinical history and the results of CT coronary angiography. Analysis of conventional coronary angiographic data was performed in two steps: First, all angiograms were reviewed by both readers, and each vessel segment was

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visually analyzed for the presence of MBon the basis of the following indirect signs: systolic diameter narrowing; milking effect, defined as diameter narrowing limited to a restricted vessel segment with extraction of contrast agent not explainable by normal coronary artery flow; and/or the step down–step up phenomenon, defined as a localized change in direction of the vessel course toward the ventricle (1,8). Second, if MB was considered to be present, the grade of systolic diameter narrowing was determined—similarly to how the CT measurements were determined—by comparing the luminal diameter within the bridged segments in end systole and end diastole with automated quantitative coronary angiography software [Figure 1b]. The coronary artery tree was subdivided according to the same guidelines (21) as for CT analysis.

Myocardial bridges were identified by 2 conditions: (1) "step-down/step-up" appearance and (2) density of the soft tissue covering the coronary artery of the same contrast enhancement threshold as the myocardial tissue, thickness >1 mm. The cutoff in tissue thickness for distinguishing the myocardium from the coronary arterial wall or atherosclerotic plaque was set at >1 mm. The presence of coronary wall lesions in the myocardial bridge and the arterial segment proximal next to it were evaluated.

Results

Myocardial bridges were found in 30 subjects (8.57%), with a higher prevalence in men (n=22; 89.2%). All subjects (100%) had 1 bridge. All 30 bridges (100%) were located over the anterior interventricular branch of the left coronary artery. Mid LAD was most common location involving 23(76.6%) patients, distal LAD in 5(16.6%), proximal LAD in 2(6.6%) patients. The myocardial bridges ranged in length from 10 to 23mm, with a mean of 14.53 mm. Thickness ranged from 1.3 to 4.5 mm, with a mean of 3.04 mm.

Discussion

The current imaging standard of reference for the diagnosis of myocardial bridging is conventional coronary angiography, which shows the milking effect and step-down/ step-up phenomenon induced by systolic compression of the tunneled segment.

The incidence of myocardial bridging in our study was 8.57%, compared with <5% in angiography 6,7 and 15%– 85% in autopsy studies.[3,6]

Kawawa et al., who reported a 15.8% prevalence of myocardial bridging in 146 Japanese patients with various coronary heart diseases.[12] At the same time, according to pathology studies, myocardial bridging is present in approximately one-third of adults, and Konen et al,[8] reported a

30.5% prevalence of myocardial bridging in 118 patients with various heart diseases. However, 29.4% of their 34 myocardial bridges were of the superficial septal type.[10]

Myocardial bridging is confined mostly to the LAD artery and is usually located between the proximal and middle third of the vessel. In our series as well, myocardial bridges were located over the LAD of the left coronary artery in all patients (100%). No bridging was detected in the right coronary artery. The length of the tunneled artery ranged from 10 to 23 mm (mean: 14.53mm). Bridge thickness ranged from 1.3 to 4.5 mm (mean, 3.04 mm). These results are in agreement with other CCTA studies in the literature.[3,8,9]

Arterial segments may also be located in a deep interventricular gorge, where the surface is not fully covered by myocardial Wbers but by a thin layer of connective tissue, nerves, and fatty tissue. This kind of bridging is defined as incomplete and may also show compression during systole in some cases.[13,21,29] However, incomplete bridging may be more difficult to diagnose as they may not show systolic compression and thin fibrous bridges may be invisible.[35]

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Asymptomatic patients with myocardial bridging do not need therapy. In symptomatic patients, the primary step is medical treatment[3], which includes beta-blockers, calcium channel blockers, and anti-platelet agents.[2,9] Nitrates generally should be avoided because they increase systolic compression and may lead to worsening of the clinical symptoms.[2]When patients are unresponsive to medical treatment, stent implantation, surgical myotomy, or coronary bypass grafting can be performed.[9]

CCTA offers several potential advantages over conventional angiography: it is noninvasive; it shows the coronary artery lumen, wall, and surrounding myocardium; and it provides data on the length, depth, and precise location of the myocardial bridge and the presence or absence of atherosclerosis. In addition, it is easy to perform and requires only single breath-hold and a short scan.

The present study has several limitations. (1) The results may be biased by the retrospective design and the inclusion of self-referred patients. (2) CCTA is inapplicable in patients with arrhythmia and patients who cannot hold their breath, although, in our series, only 4 subjects were excluded for these reasons. (3) We were unable to compare our results with other methods, and there is no external reference standard to confirm the findings of CT. Nevertheless, CCTA has been validated for use in coronary artery imaging, so it is unlikely that our findings represent artifacts. (4) In the present study we could not determine clinical significance of the observed myocardial bridges.

Conclusion

We found that prevalence of myocardial bridging was 8.57%. Both conventional angiogram & CT angiogram are reliable in diagnosis of myocardial bridging. But, CT angiogram is noninvasive and can accurately locate the the site of myocardial bridging, and measure its thickness, course, and length.

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