Original Article

Morphology of myocardial bridging of right coronary artery: delineation using coronary computed tomography angiography

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Abstract: Objectives: To reveal the morphological features and follow-up outcome of right coronary artery myocardial bridging (RCA-MB) using coronary computed tomography angiography (CCTA). Methods: 37 consecutive patients with RCA-MB referred for CCTA were obtained. All subjects were divided into incomplete MB and complete MB groups according to the continuity of the myocardium over the tunneled segment of RCA. The length, Thickness, stenosis rate was evaluated. An individual follow-up study was executed to the subjects without coronary artery disease (CAD). Follow-up data were obtained from telephone contact and all available medical records in hospital. Results: Total 41 RCA-MB were detected (complete type, 63.4%). 60.9% (25/41) occurred in mid segment of RCA. 73% (30/41) occurred in right atrium and 68.3% (28/41) coexisted with MB in other segment. The mean length, thickness and stenosis rate were 11.7 ± 7.5 mm, 1.8 ± 0.3 mm and 29 ± 10.8% respectively. 21 patients finished the follow-up study with 2 positive readmitting subjects. There was no significant difference in clinical symptoms and objective signs of ischemia between controls and patients with RCA-MB. Conclusions: Although it did not increase prevalence of myocardial ischemia statistically, RCA-MB still could induce clinical symptoms by chance and should be paying attention in daily reconstructing workflow.

Keywords: Coronary anomalies, coronary CT angiography, right coronary artery, myocardial bridges, follow up

Introduction

MB is a congenital anomaly in which one or more segments of a major coronary artery have an intra-myocardial course. As such bridging is often observed in “non-symptoms” patients, it is very clear that not all arteries bridged by myocardial segments induce clinical symptoms. It suggests that the feature may simply be an anatomical variant. Studies on the potential clinical significance of MB have been presented [1-3]. However, it is mainly related to MB located in LAD. Only limited cases of right coronary artery myocardial bridge have been reported [4-10]. However, the blood supply shortage provoked by RCA-MB may also lead to fatal consequence by chance. We have studied 37 unusual cases of myocardial bridging with right coronary artery distribution and try to present the morphological features and follow-up outcome about RCA-MB to better understand the relationship between the anatomical variation and clinical significance in a relatively large patient cohort.

Methods and materials

A total of 37 consecutive patients (30 men and 7 women) were examined with dual source CT between January 2007 and June 2011. All subjects were referred to our department for CCTA with atypical chest pain, follow-up after coronary stents or/and coronal artery bypass grafting, inconclusive stress test results, or annual physical examination. 18493 cases were selected (all the subjects had CCTA). And the exclusion criteria for CCTA were: the presence of severe arrhythmia, atrial fibrillation, heart
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Table 1. Patient characteristics (n = 37)

<table>
<thead>
<tr>
<th>Demographics</th>
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<tbody>
<tr>
<td>Age, yrs</td>
<td>57 ± 10.9</td>
</tr>
<tr>
<td>Male</td>
<td>30 (81.1)</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>68 ± 6</td>
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<td>BMI, kg/m²</td>
<td>26.4 ± 2.2</td>
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<table>
<thead>
<tr>
<th>Clinical symptoms and heart surgery history</th>
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<tbody>
<tr>
<td>Angina</td>
<td>6 (16)</td>
</tr>
<tr>
<td>Palpitation</td>
<td>21 (57)</td>
</tr>
<tr>
<td>Stents, RCA/total</td>
<td>1/6 in 4 patients (11)</td>
</tr>
<tr>
<td>CABG</td>
<td>2 (5)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CAD and CAD risk factors</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>CAD</td>
<td>7 (19)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>11 (30)</td>
</tr>
<tr>
<td>Dyslipidemia</td>
<td>10 (27)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>15 (41)</td>
</tr>
<tr>
<td>Tobacco use</td>
<td>15 (14)</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Medications</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Antihypertensive medications</td>
<td>10 (27)</td>
</tr>
<tr>
<td>Beta-blocker</td>
<td>12 (32)</td>
</tr>
<tr>
<td>Calcium-channel blocker</td>
<td>14 (38)</td>
</tr>
<tr>
<td>Statin</td>
<td>8 (22)</td>
</tr>
<tr>
<td>Aspirin</td>
<td>8 (22)</td>
</tr>
</tbody>
</table>

Values are mean ± SD or n (%). BMI: body mass index; RCA: right coronary artery; CABG: coronary artery bypass grafting; CAD: coronary artery disease.

rate greater than 80 beats per minute (bpm) despite therapy by β-Blocker, renal failure, asthma, severe lung disease, hyperthyroidism and a history of allergic reaction to iodine-containing contrast material. The mean age was 56.2 ± 9.5 years (age range, 43-77 years). The characteristics of all subjects are depicted in terms of demographics, clinical symptoms and heart surgery history, CAD and CAD risk factors, medications (Table 1). Written informed consent was obtained from all subjects and our institutional review board has approved the study.

Image acquisition

All subjects were in sinus rhythm. Patients received an intravenous β-Blocker infusion 2-5 minutes before the scan (atenolol 5-10 mg intravenously based on body mass) with heart rate > 80 bpm. The variation of HR was from 44 to 78 bpm (average, 68 ± 6 bpm) with or without premedication. All CT examinations were performed by dual source CT (SOMATOM Definition, Siemens Healthcare, Forchheim, Germany). The retrospective ECG-gated scan mode was applied to all the subjects. The scan was performed from the tracheal bifurcation to the diaphragm in a cranio-caudal direction with the following parameters: collimation, 32×0.6 mm; slice acquisition, 64×0.6 mm with the z-flying focal spot technique; gantry rotation time, 330 ms; tube voltage 120 kV or 100 kV adapted to BMI; pitch, 0.2-0.4 adapted to the heart rate; effective mAs 350, and ECG based dose modulation during 40%-80% of the cardiac cycle. ECG modulation of the tube current was all activated in this study. A bolus of iodinated contrast material (Ultravist 370; Schering, Berlin, Germany) was IV injected via an 18-gauge catheter placed in the antecubital vein followed by a bolus of 30-40 mL of saline. The amount of the contrast agent was decided according to the patient’s body weight, heart rate and scan time. The total amount of the contrast agent was 62.3 ± 11.2 mL (range 50-80 mL) and the contrast injection speed was 4.8 ± 0.7 mL/s. A bolus tracking technique was applied to synchronize the data acquisition with the arrival of contrast agent in the coronary arteries. The region of interest (ROI) was placed within the ascending aorta and the scan was triggered when the CT density in ROI was 120 Hounsfield units higher than the baseline CT density. The patients were instructed to maintain an inspiratory breath-hold during which the CT data and ECG trace were acquired. All raw data were reconstructed with the following parameters: section thickness, 0.75 mm; increment, 0.4 mm; medium-to-smooth convolution kernel, B26f (stent, B46f); spatial resolution, 0.6-0.7 mm (in plane) and 0.5 mm (through plane). To detect the level of compressed lumen, 10 data sets were reconstructed in every 5% steps from 30% to 80% of the R-R interval. The tube voltage was selected as a function of body mass index (BMI): BMI < 20 kg/m², 80 kV; BMI20-30 kg/m², 100 kV; BMI > 30 kg/m², 120 kV [1]. CT dose index volume (CTDIcon, mGy) and dose-length product (DLP, mGy·cm) were recorded as evaluation of scanning radiation dose.
Effective doses (ED, mSv) were generated from the DLPs using published conversion coefficients (0.014) [2].

Image reconstruction

The retrospective ECG-gating method was used to reconstruct image. Data phases of the R-R cycle were acquired by using “minidose” software. The best systole phase and the best diastole phase were automatically chosen to measure. The image data sets were imported into a separate workstation (Syngo, version: 2008-2010, Siemens Medical Solutions, Erlangen, Germany) and analyzed using curved multiplanar reconstruction (MPR) in various planes and thin-slab maximum-intensity-projection (MIP) reconstructions in addition to the axial images. Coronary artery findings were reviewed in consensus by two experienced investigators (with > 5 and > 6 years of cardiac CT experience, respectively) by using a modified 17-segment model of the coronary artery tree from the American Heart Association (AHA) reporting system [3]. RCA was divided into 6 segments including proximal, the first bend, mid, the second bend, distal and posterior descending artery (PDA) when evaluating distributions of RCA-MB. Readers were blinded to the patient’s clinical history and symptom. If a consensus could not be reached, a third expert reader determined the final diagnosis (with > 10 years of cardiac CT experience).

Data analysis

In all cases, the diagnosis of myocardial bridge was achieved on the basis of the cross-sectional, thin-slab MIP and MPR images and the axial source images. Myocardial bridge was defined as an epicardial segment of a coronary artery that courses through the myocardium. In our study, RCA-MB was classified as complete and incomplete bridges with respect to continuity of the myocardium over the tunneled segment of coronary artery. The complete type was defined as the tunneled segment totally buried into the myocardium and not protruded out of the external surface of epicardium. The incomplete type was defined as the tunneled segment partially buried into the myocardium and at least more than half the cross-sectional lumen area covered. The fact needed to be point out that most of RCA-MB does not bury quite deeply into the myocardium because the right atrium and ventricular were almost thin wall. The Classification principle made for MB of LAD does not apply to the RCA-MB quite well. The RCA-MB length, MB thickness, MB muscle index (MB thickness multiplied by MB length) and MB luminal stenosis ([target narrowest diameter/(proximal diameter of MB + distal diameter of MB)/2] of RCA myocardial bridges in systole were measured. The length of MB was defined as the distance of the covering myocardial tissue from the entrance to the exit of the tunneled artery, which was measured in both MPR and axial images. The thickness of MB was defined as the thickness of the deepest part from the surface of the covering myocardial tissue to the tunneled artery, which was also measured in an axial image. Multifocal coronary artery MB was classified as Parallel multifocal MB and Series multifocal MB. Parallel multifocal MB was defined as myocardial bridging involving other coronary branch besides RCA; Series multifocal MB was defined as myocardial bridging involving two segments of RCA successively. Coronary artery disease was defined as coronary wall atheromatous change (calcified, noncalcified and mixed plaque) with significant stenosis greater than 50% reduction of the lumen diameter. The coronary CTA findings were classified as the following: normal, no atheromatous changes or luminal narrowing; mild disease, atheromatous changes without luminal narrowing; moderate disease, atheromatous changes with insignificant stenosis; and severe disease, atheromatous changes with significant stenosis [4]. The number and severity of lesions in segments in LAD, RCA, posterior descending artery (PDA), left circumflex artery (LCX), and obtuse marginal artery (OM) were measured.

Follow up study

Thirty-one consecutive patients who underwent diagnostic CCTA in the same period for suspected coronary ischemia but did not demonstrate CAD or MB or other cardiac diseases assigned into the control group. Follow-up data were obtained from telephone contact directly and all available medical records in our hospital were reviewed. The inclusion criterion was that subject underwent CCTA without CAD. The patients with other disease causing chest pain were excluded. The primary endpoint was readmission during the follow-up period. Readmission comprised: recurrent chest pain, cardiac death, myocardial infarction, target vessel
Table 2. RCA-MB morphology

<table>
<thead>
<tr>
<th>Segment of RCA</th>
<th>MB location</th>
<th>Number (n = 41)</th>
<th>MB type</th>
<th>Length (mm)</th>
<th>Thickness (mm)</th>
<th>Muscle Index</th>
<th>Lumen narrowest percent in systole</th>
<th>Lumen narrowest percent in diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proximal</td>
<td>Posterior-superior wall of RV</td>
<td>2 (4.9)</td>
<td>complete</td>
<td>11.6 ± 0.4</td>
<td>0.9 ± 0.21</td>
<td>0.09 ± 0.02</td>
<td>24%, TSTBO*</td>
<td>TSTBO</td>
</tr>
<tr>
<td>The first bend</td>
<td>Anterior-superior wall of RA</td>
<td>3 (7.3)</td>
<td>incomplete</td>
<td>2.6 ± 2.1</td>
<td>N/A</td>
<td>N/A</td>
<td>TSTBO</td>
<td>TSTBO</td>
</tr>
<tr>
<td>Mid</td>
<td>Posterior-superior or posterior-lateral wall of RV</td>
<td>25 (60.9)</td>
<td>complete</td>
<td>13.4 ± 1.8</td>
<td>0.96 ± 0.15</td>
<td>0.07 ± 0.01</td>
<td>40%, 25%, 25%, TSTBO, TSTBO, 20%</td>
<td>25%, TSTBO</td>
</tr>
<tr>
<td>Mid</td>
<td>Anterior wall of RA</td>
<td>3 (7.3)</td>
<td>complete</td>
<td>8.7 ± 2.4</td>
<td>TSTBO</td>
<td>N/A</td>
<td>TSTBO</td>
<td>TSTBO</td>
</tr>
<tr>
<td>Mid</td>
<td>Anterior wall of RA</td>
<td>16 (39)</td>
<td>incomplete</td>
<td>9.6 ± 3.0</td>
<td>N/A</td>
<td>N/A</td>
<td>TSTBO</td>
<td>TSTBO</td>
</tr>
<tr>
<td>The second bend</td>
<td>Posterior-inferior wall of RA</td>
<td>3 (7.3)</td>
<td>complete</td>
<td>19.6 ± 5.1</td>
<td>0.81 ± 0.04</td>
<td>0.03 ± 0.01</td>
<td>54%, 25%, TSTBO</td>
<td>28%, TSTBO</td>
</tr>
<tr>
<td>Distal</td>
<td>Posterior-inferior wall of RA</td>
<td>2 (4.9)</td>
<td>complete</td>
<td>10.7 ± 3.3</td>
<td>TSTBO</td>
<td>N/A</td>
<td>TSTBO</td>
<td>TSTBO</td>
</tr>
<tr>
<td>Distal</td>
<td>Posterior-inferior wall of RA</td>
<td>3 (7.3)</td>
<td>incomplete</td>
<td>9.3 ± 4.0</td>
<td>N/A</td>
<td>N/A</td>
<td>TSTBO</td>
<td>TSTBO</td>
</tr>
<tr>
<td>PDA</td>
<td>Posterior interventricular septum</td>
<td>3 (7.3)</td>
<td>complete</td>
<td>16.9 ± 8.7</td>
<td>2.33 ± 0.21</td>
<td>0.15 ± 0.08</td>
<td>25%, 25%, TSTBO</td>
<td>TSTBO</td>
</tr>
</tbody>
</table>

Age, length, thickness and muscle index are presented as mean ± SD; MB narrowest Lumen diameter is presented as level (%); all other data are presented as number (%). *TSTBO: too slight to be observed. RV: right ventricle; RA: right atrium.
revascularization, non-cardiac death, or life-threatening arrhythmia. Readmitted patients were all undergone ECG and treadmill exercise ECG Stress test (TEEST) in our hospital to diagnose myocardial ischemia.

Evaluation of recurrent hospitalizations due to symptoms

All patients were evaluated for: (1) clinical symptoms defined as typical angina (chest pain during stress with relief at rest), atypical angina (non-exertional chest pain), non-specific (palpitations, fatigue), or no symptoms; (2) objective signs of myocardial ischemia as assessed by TEEST [5].

Statistical analysis

Statistical analysis was performed using commercially version SPSS 13.0 for Windows (SPSS, Chicago, IL, USA). Continuous variables are expressed as mean ± standard deviation (SD). Two sample t tests were employed for comparisons of MB parameters between the groups. When MB parameters did not abnormally distribute statistically, a Mann-Whitney’s U test was employed for comparisons. The univariable relationships between the patients’ demographic and pathological characteristics and the groups defined as above were analyzed with X² or Fisher’s exact test when the predictor was categorical and with Wilcoxon rank sum test when the predictor was quantitative. Two-tailed P < 0.05 was considered statistically significant.

Results

Patient characteristics, prevalence, and radiation dose

41 right coronary artery MBs were detected in 37 patients (Mean age, 57 ± 10.9) (30 male, 7 female). The prevalence of RCA-MB was 0.2% from CT imaging. 26/41 (63.4%) bridged segments in 19 patients were complete and the rest were incomplete. The radiation dose was as follows: CTDIvol, 28.1 ± 1.0 mGy; DLP, 482.5 ± 171.2 mGy*cm; ED, 6.8 ± 2.4 mSv.

Morphology of RCA-MB by CT imaging (Table 2)

The RCA-MB in our collection spanned an enormous distribution in segments of RCA from proximal to distal (Figure 1). It occurred mostly in mid segment of RCA (25/41, 60.9%), and were most common with incomplete type in anterior wall of right atrium (16/37, 43%). 19 (46.3%) cases were complete type, and 11 (57.9%) of 19 were located in right ventricle wall. The length of the bridges varied from 1.2 to 38.4 mm (mean 11.7 ± 7.5 mm) and the length of MB occurred in the second bend segment of RCA was 19.6 ± 5.1 mm compared with the length in other segment of RCA (P < 0.05). The thickness of bridging myocardium (27/41, 65.9%) over the involved segment was too thin to be measured exactly. Only 14 (34.1%) cases located in posterior-superior wall of right ventricle, posterior-lateral wall of right ventricle and interventricular septum were deep enough to be measured. The mean thickness of bridging myocardium was 1.8 ± 0.3 mm. The thickest bridging myocardium occurred in posterior interventricular septum with mean thickness of 2.33 ± 0.21 mm. The maximum muscle index was observed in the same segment (0.15 ± 0.08). The lumen stenosis during systole varied from 20 to 54 percent (mean 29 ± 10.8%). The
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lumen narrowing during diastole was not observed in our study.

Multifocal coronary artery myocardial bridging involving the right coronary, left anterior descending arteries, left circumflex coronary artery, obtuse marginal branch

Totally 28 (68.3%) RCA-MBs were multifocal origin. There were 24 (58.5%) parallel multifocal RCA-MBs including 21 (51.2%) combined with LAD (Figure 3), 1 (2.4%) combined with LCX, 1 (2.4%) jointly combined with LAD and OM, 1 (2.4%) combined with LAD and LCX concurrently. There were 4 (9.8%) series multifocal MB occurred in RCA, all of 3 (7.3%) located in mid segment of RCA, and 1 (2.4%) involved both in mid segment and the second bend of RCA (Figure 2).

Comparison between RCA-MB type groups

There were 19 (46.3%) complete RCA-MBs and 22 (53.7%) incomplete RCA-MBs involved in our study. Sex, age, heart rate (HR), BMI, number, difference of two set of patients have no statistics significance (P > 0.05). The length was 18.5 ± 9.4 mm in the complete MB group compared with 6.3 ± 3.4 in the incomplete MB group (P < 0.001) (Table 3).

Prevalence of lumen narrowing of RCA-MB

During systole, non-narrowed coronary were observed in 34 cases (82.9%). There were only 7 cases (13.5%) of complete type MB observed with varied degrees of lumen narrowing (Figure 3). MB in incomplete group has not accompanied with lumen narrowed (P < 0.001). In the complete type group, 5 cases (31.8%) had less than 25% stenosis, and 1 case had 40% stenosis. Significant stenosis (≥ 50%) was observed in one case. The cases which were observed with stenosis were all located in the right ventricle wall.

Atheromatous change proximal to RCA-MB (Table 3)

We examined plaque characteristics of proximal to MB in both complete type and incomplete type. For 5 cases with plaque of proximal to MB in complete type group, 4 cases (80%) had non-calcified plaque, 1 case (20%) had patchy calcification, 1 case (20%) had spotty calcification and 1 cases (20%) had severe mixed and calcified plaque. 3 (60%) mild disease were leaded by non-calcified plaque and spotty calcified plaque. 1 (20%) moderate disease arose from patchy calcification and non-calcified plaque. 1 (20%) severe disease was induced by severe mixed and calcified plaque. In incomplete type group, total 5 cases with

Figure 2. A RCA-MB located in mid segment of RCA (arrow, A) coexisted with a MB of LAD (arrow head, A). A patient has two RCA-MB involved both in mid segment and the second bend of RCA (arrow head, B).

Figure 3. A complete type MB located in the mid of RCA observed with about 40% of lumen narrowing in systole at right, and no obvious narrowing in diastole at left.
plaque of proximal to MB were observed, 3 cases (60%) had non-calcified plaque, 1 case (20%) had patchy calcification, 2 cases (20%) had spotty calcification and 1 case (20%) had severe mixed plaque. 1 (20%) was after stent placement. 3 (60%) mild disease were leaded by non-calcified plaque and spotty calcified plaque. 1 (20%) moderate disease arose from patchy calcification and non-calcified plaque. 1 (20%) severe disease were induced by severe mixed plaque, and a drug-eluting stent replaced in mid segment proximal to MB. There was no statistical significant between 2 group of proximal plaque burden to RCA-MB (P > 0.05).

**Clinical follow-up of RCA-MB**

Thirteen patients with complete type and eleven patients with incomplete type of RCA-MB were involved into the follow-up study. Two patients with complete RCA-MB were lost in the 21th month and the 17th month respectively for changing contact information by themselves (Table 4). One patient with incomplete RCA-MB refused to join the follow-up. Total 21 patients completed our examination. The mean period was 26.5 ± 7.2 months. 2 (9.5%) patients were readmitted with different clinical symptoms. 1 (4.8%) cases of recurrent chest pain, proven by 1 (4.8%) arrhythmia. Both of them were complete RCA-MB There was no major adverse cardiac event or a need for any revascularization in the follow-up period. The multifocal origin cases (1/21, 4.8%) were ruled out to avoid the analysis influenced by coexisting MB occurred in another branch (LAD or OM). All of these cases showed positive results on TEEST defined as typical angina. One case was located in second bend of RCA (length, 18.5 mm; thickness, 1.8 mm), showed lumen narrowing in systole (54%) (Figure 4). The other one was located in mid segment of RCA (length, 9.7 mm) with 25% lumen narrowing. There were 3 cases were readmitted with different clinical symptoms in control group (1 for arrhythmia, 2 for chest pain). There was no significant difference in clinical symptoms and objective signs of ischemia between controls and patients with MB.

**Discussion**

Myocardial bridging is identified as a simple variant of normal anatomy of coronary arteries which commonly defined as a segment of a major epicardial coronary artery running intramurally through the myocardium. It was also
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called ‘tunneled artery’. The myocardial bridging is associated with the intracoronary hemodynamic alteration during systole and diastole, which is called ‘milking effect’ determined by the severity and the location of the bridging within the coronary artery from coronary angiography (CAG). It was first mentioned by Reyman in 1737 and first described in public by Crainician in 1922 [6]. Portmann and Iwig first reported it angiographically in 1960 [7].

Previous study mainly has focused on myocardial bridging located in LAD, fewer cases about right coronary artery myocardial bridge have been reported. To our knowledge, this is the first original study to present MB occurred in RCA with larger cohort of patients. The main findings of our study are: (1) a prevalence of RCA-MB of 0.2% depicted by using CCTA less than Postmortem results; (2) RCA-MB commonly locates in the mid segment of the RCA; (3) most of RCA-MB occurred in right atrium wall, and most present incomplete type in anterior wall of right atrium; (4) RCA-MB occurs in second bend segment of RCA is usually much longer than that in other segment and the thickest bridging myocardium generally occurs in posterior interventricular septum; (5) More than 50% of RCA-MBs are multifocal origin and commonly combined with LAD [8, 9]; (6) There was no statistical difference between incomplete group and complete group when evaluating proximal plaque burden to RCA-MB [10]; (7) RCA-MB is not completely harmless and recurrent hospitalization is observed occasionally. The reason of readmission is the recurrence of chest pain in patients with RCA-MB. During the follow-up, there were no significant difference in clinical symptoms and objective signs of ischemia between controls and patients with MB.

Prevalence of RCA-MB

The probability of myocardial bridge detection varied depending on the examination methods. The prevalence of tunneled coronary arteries identified at autopsy is 5.4-85.7%, which is significantly different from that determined from angiography (0.5-12%) [11]. The information on MB distributions was gotten mostly from the autopsy studies. In 1985, Risse and Weiler observed 1056 hearts in total and found that the right coronary arteries involved in 5.7% [12]. Polacek demonstrated that the relative frequency of myocardial bridging exclusively involving the right coronary artery was 36% [13]. In our study, the prevalence of RCA-MB was only 0.2% far less than the results mentioned above. This may due to the limited spatial resolution of CCTA to detect all the RCA-MBs, especially for the incomplete type. In addition, RCA has greater mobility than LAD. Motion artifact makes diagnosis more difficult.

Morphology features and clinical relevance

Many literatures have mentioned that the location, length and depth of MB related to clinical symptoms [7]. In this study, we try to depict the distributions and morphological features of RCA-MB including the length and thickness. The MB occurred most in mid segment of RCA (25/41, 60.9%), and the length of MB between vary segments of RCA is significantly different. The MBs occurred in the second bend were much longer and all involved in right atrium. From the follow-up outcome, we found that the MBs occurred in the second bend of RCA with long course beneath the myocardium could.
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combine with higher frequency of clinical symptoms, and was proven by TEEST. It is not too conforms to the common sense. Generally, the deeper the bridges are, the greater the compressive forces. However, the cases mostly occurred in atrium. We assume that the reason is the second bend with the maximal range of mobility compared with other segment enlarging MB's fulcrum effect [14]. This effect is due to the compression produced by the heart contraction at the fulcrum lumen. This effect could increase the risk of myocardial ischemia.

The wall of right ventricle is much thinner than that of left ventricle because it pumps the blood at a lower pressure. Thus most of MB located in RCA isn’t deep buried. In the previous study, the thickness of the right ventricle, right atrium and posterior interventricular septum is approximately 5 mm, 2 mm and 8.3 in the end-systole respectively [15]. The thickness of myocardial bridging occurred in right ventricle is much deeper than that in right atrium. Therefore, the deeper the bridges, the greater the compressive forces. As a result, the phenomenon of lumen narrowing is only observed on MBs in ventricle in systole. However, there is no severe systolic narrowing (more than 50% stenosis) of the tunneled segment seen in our study. It is probably because all the subjects in this study are not in a stress condition.

The clinical significance of MB is controversial [16-21]. It is clear that not all intramural arteries produce symptoms from the reported incidences of MB confirmed from autopsy studies. However, several authors reported RCA-MB cases with positive clinical event. Riezzo and his colleagues reported a sudden death patient with RCA-MB [6]. There was no evidence of previous health problem and no positive clinical history available. The culprit MB was superficial with 5 cm extension of the mid right descending coronary artery. Chen [22] reported a patient with retrosternal chest pain which was not relieved by sublingual nitroglycerin tablets. The culprit MB is approximately 4 cm occurred in the middle segment of second bend. Nguyen [14] report a patient’s ST-elevation myocardial infarction arising from myocardial bridging located in the mid segment of right coronary artery with severe spasm, thrombolytic treatment was given. These reported cases had the same morphological characteristics as the ones from our study, it is the larger course beneath the myocardium and it is from the same origin site (mid segment of RCA).

Most RCA-MBs in this study are multifocal origins and commonly coexist with LAD. It indicated that RCA-MB occurs with the combination of other MBs, especially LAD-MB. Further study is needed to clarify whether the multifocal origins would increase the risk of myocardial ischemia.

There are only a few subjects with plaque burden of proximal in this study. Limited information is available. At this stage, the result we got from study is that there was no statistical different between complete type group and incomplete type group.

Limitations

Sample of RCA-MB is too small to evaluate the relationship between length, thickness of the bridge and severity of plaque burden in the right coronary artery proximal to the bridge statistically, and the relationship between frequency of readmission and morphological features of RCA-MB. We are not able to answer the question whether multifocal MBs could increase the risk of provoking myocardial ischemia.

RCA-MB is not completely harmless and recurrent hospitalization is observed occasionally. The clinical relevance from the combined LAD-MB cannot be ruled out.

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Disclosure of conflict of interest

None.

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References


