CLINICAL PRESENTATION OF THE NON-ATHEROSCLEROTIC CORONARY LESIONS, MORPHOLOGICAL EVALUATION OF THE MYOCARDIAL BRIDGE

PhD thesis

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1. Introduction

Coronary arteries - that could be detected in vivo - mainly run in the epicardial space. If – during ontogenesis - a segment tunnels through the myocardium refer to myocardial bridging (MB). Depending on the imaging method, MB is a frequently detected congenital anomaly. According to a meta-analysis, the prevalence in autopsy reports is 30-55%, evaluated with computed tomography: approximately 22% and with invasive coronary angiography: 5-8%. MB involves mainly (82%) the middle segment of the left anterior descending (LAD) with a mean length of 19-20 mm. If the myocardium is hypertrophic (e.g., due to hypertension or hypertrophic cardiomyopathy) the phenomenon might be more pronounced and described more often. Overall MB has been considered a benign anomaly, although several case reports were published where MB was held responsible for ischemia. MB is a unique non-atherosclerotic coronary lesion with two main aspects. On the one hand it causes dynamic stenosis. Theoretically, most of the coronary flow occurs during diastole. So, at rest, when the heart rate is low and there is sufficient time for the artery to fill (the diastole is long enough), the MB might not provoke symptoms. However, during physical effort or mental stress – when the heart rate increases and the diastolic filling time shortens -ischemic symptoms can occur. Its pathological relevance is more complex: beyond anatomic features like the MB's length, depth, the surrounding

muscle thickness, also other factors could play a role, including concomitant vasospasm, the number of side branches that arise from “tunneled” part, the presence of atherosclerosis, or the micro- and macro-dissections. On the other hand, it is considerable that – according to several studies – the affected part is free from atherosclerosis, but the proximal segment from the bridge is more exposed to it, due to the sheer stress caused by turbulent blood flow. The former issue is explained by the lack of epicardial adipose tissue and vasa vasorum here. These pathophysiological factors play an important role in the pathogenesis of coronary artery disease. Ischemia provocation might help to reveal the role of revealed MB. Proper physical stress or dobutamine stress echocardiography used commonly. In the last two decades we have been able to perform functional evaluations also in the catheterization laboratory.

The prognostic factors and the clinical evaluation of MB has remained limited. Key questions remain unresolved and wait for answers: - How can we model dynamic obstruction? - Can myocardial bridge cause symptoms per se or only with other concomitant factors (triggers)? - If MB is a congenital anomaly, why do patients develop symptoms typically after the age of 40? - What is the role of MB in plaque burden and morphology in the coronary which is concerned by the anomaly? - How can the benign and malignant type be differentiated morphologically and functionally? - If once symptomatic, should it be treated medically or invasively?

Our group aimed to approach these questions.

2. Objectives

2.1. Aims of our study

First, we would investigate the relation of long-term outcome and coronary morphology in a large MB population. We looked for the characteristics of morphological factors detected by angiography, those may be associated with angina pectoris. We aimed to compare the life expectancy among patients with isolated LAD-MB and patients who had LAD-MB and additional significant atherosclerotic disease. We also aimed to study whether these morphological factors influence long-term mortality.

Secondly, in a retrospective case control study we investigated the plaque morphology beneath the myocardial bridge by computer tomography angiography (CTA).

Thirdly, we aimed to present an own case where LAD-MB led to ischemia, myocardial infarction and ventricular septal rupture (Case I.).

Fourthly, in another case report, we show a case where the treatment choice of a symptomatic, therapy refractory LAD-MB was percutaneous coronary intervention (Case II). This case refers as an example that the mode of revascularization may result in a long-term success in the treatment of symptomatic MBs.

3. Methods

3.1. Invasive study to investigate morphological and clinical factors on long-term mortality.

A total of 11,385 diagnostic coronary angiographies were performed at our center between 25 March 2009 and 12 March 2011. Ethical approval (of enrolled patients) was obtained from the Central Ethics Committee of Hungary, with all the participants completing informed consent forms.

We enrolled a total of 203 patients (1.78%) with a clear presence of MBs. We excluded patients with MBs affecting arteries other than the LAD and where any other obvious cause of angina pectoris was presented (n=57). The remaining patients (n=146) were divided into two groups according to accompanying coronary artery disease. In case of 78 patients, no other underlying, epicardial coronary disease was found, except LAD-MB (LAD-MBneg group). At 68 patients significant coronary artery disease was also revealed in addition to the LAD MB (LAD-MBpos group). In the LAD-MBpos group coronary plaque was considered significant if lumen narrowing (>50% in diameter) of significant epicardial coronary arteries (>1.5 mm diameter) was observed, and we provided therapy according to current guidelines.

Quantitative angiography was performed according to our standard clinical practice. All the MBs were measured in lateral view (angulation of the “C” arm left lateral, LAO: 90 degrees, caudal: 0 degrees) in end-systole and end-diastole by an expert interventionist. Four main parameters were measured:

(1) length of the MB, defined as the distance from the most proximal point to the most distal point of the LAD, where the systolic narrowing phenomenon could be observed.

(2) reference diameter of the MB, defined as the diameter of the vessel immediately proximal to the point where the systolic narrowing started; and

(3) minimal diameter of the MB, measured also in end-systole at the point where the thickening was the most prominent. Additionally, from these parameters we calculated.

(4) minimal diameter to reference diameter, the ratio between minimal stenosis and reference diameter in percentage to characterize shortening of bridge for each patient.

In addition to these data, we also recorded height, weight, sex of patients and presence of main cardiovascular risk factors, such as diabetes mellitus, hypertension, and dyslipidemia. We collected information about the patients' mortality by phone visit and we also checked data on survival status (according to the Hungarian National Database) in 1. April 2020.

3.2. Coronary CTA study

In another patient group we quantified plaque volumes proximal to the MB and beneath it in patients with MB and in the equivalent coronary segments in patients without MB. We searched for patients' data who underwent coronary CTA between April of 2016 and May of 2017 May. Patients with established cardiac disease were not selected. The control group also had an excellent image

quality of coronary CTA but without LAD-MB. The controls were matched for the variables listed below: age (±10% range), gender, body mass index (±10% range) and the presence of typical cardiovascular risk factors (hypertension, diabetes mellitus, dyslipidaemia, smoking) and for scan parameters. We used a 256-slice multidetector row computed tomography scanner (Brilliance iCT 256; Philips Healthcare) and analyzed coronary CTA scans using a semi-automated plaque quantification software. After the manual tracing of the vessel centerline, the software reconstructed the straight multiplanar view of the segmented vessel. For the control patients, we extracted two corresponding segments with equal length as their matched pairs' segments. The software calculated the total plaque volume and the plaque composition according to predefined density ranges based on Hounsfield unit (HU) categories: −100 to 30 HU necrotic (low attenuation non-calcified) core, 31 to 130 HU fibrofatty (mid attenuation noncalcified) plaque, 131 to 350 HU fibrous (high attenuation noncalcified) plaque, and > 350 HU calcified plaque component were identified. We separately analyzed the entire segment and a ~ 20-mm-long subsegment immediately proximal to MB.

3.3. Statistics

2020. Statistics GraphPad Prism (version 6, GraphPad Software, San Diego, CA, USA) and SPSS (version 22, SPSS Inc., Chicago, IL, USA) were used for the clinically standard statistical analysis.

Continuous data are expressed as means with standard deviation.

4. **Results**

4.1. Invasive study

Clinical and LAD-MB morphological data are summarized in Table 1. LAD-MBpos patients were characterized by older age and increased presence of type 2 diabetes mellitus. According to our results, LAD-MBneg group was characterized with more severe morphological features. The shortening of MB (minimal diameter to reference diameter) significantly decreased, while the length and reference diameter showed a strong tendency towards increased value in the LAD-MBneg group compared to LAD-MBpos group.

Table 1. The distribution of the data of patients presenting with angina pectoris and with a myocardial bridge detected in the left anterior descendent artery (n = 146). Data is shown as mean (SD).

	Overall population n = 146	**LAD-MB^{neg} n = 78**	**LAD-MB^{pos} n = 68**	**LAD-MB^{neg} vs. LAD-MB^{pos}**
Mean age (years)	**60.6 (12.7)**	**57.6 (12.4)**	**64.5 (11.5)**	***0.001***
Male sex	**94 (64%)**	**50 (64%)**	**43 (64%)**	**0.99**
Hypertension	**105 (72%)**	**57 (73%)**	**48 (72%)**	**0.87**
Type 2 diabetes mellitus	**36 (25%)**	**13 (17%)**	**24 (36%)**	***0.008***
Hyperlipidemia	**77 (53%)**	**37 (47%)**	**40 (60%)**	**0.14**
Body mass index (kg/m^2)	**27.6 (3.8)**	**27.2 (3.4)**	**28.2 (4.3)**	**0.11**
LAD-MB length (mm)	**21.4 (8.2)**	**23.4 (8.3)**	**20.0 (7.7)**	***0.05***
Reference diameter (mm)	**2.18 (0.46)**	**2.23 (0.42)**	**2.09 (0.41)**	**0.06**
Minimal diameter (mm)	**1.10 (0.41)**	**1.02 (0.36)**	**1.11 (0.38)**	**0.39**
Minimal diameter to reference diameter (%)	**49.5 (15.5)**	**54.5 (13.1)**	**46.5 (16.4)**	***0.006***

The average follow-up period of this patient population was 3115±249 days, almost ten years. When we checked survival status, we found that mortality was 16.4% in the overall population. Eight persons died in the LAD-MBneg group; thus, the all-cause mortality rate in this population was 10.3% for this follow-up period that is comparable with healthy population of a similar age. We searched for differences between the two prespecified subgroups: the Kaplan-Meier analysis revealed significant disparity in mortality between LAD-MBneg and LAD-MBpos groups (Figure 2.).

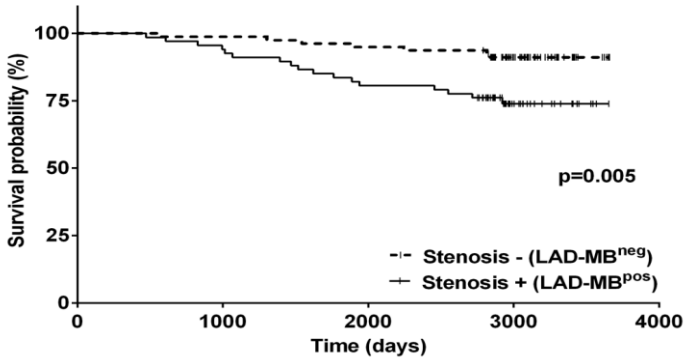

Figure 1. Kaplan–Meier curve of the long-term follow-up comparing LAD-MBpos and LAD-MBneg group.

We also looked after factors that could influence long-term mortality both in overall population and LAD-MBneg group. In the overall population the presence of coronary stenosis, diabetes mellitus and older age were associated with increased mortality, while in the LAD-MBneg group only age influenced survival outcome. To determine the role of morphological parameters, a

multiple variable Cox regression was performed with the morphological parameters and influencing factors from single variable analysis. Our results show that none of the morphological parameters influenced long-term mortality (Table 2).

Table 2. Summary of univariate and multivariate Cox regression analysis of overall survival in overall population and LAD-MBneg group. HR: hazard ratio.

	Overall population n = 146		**LAD-MB^{neg} n = 78**	
	Single variable analysis	**Multiple variable analysis**	**Single variable analysis**	**Multiple variable analysis**
Stenosis	**0.005 HR:3.45**	**0.11 HR:2.14**	**NA**	**NA**
Mean age (years)	**< 0.001 HR:1.08**	**0.001 HR:1.08**	**0.03 HR:1.07**	***0.03*** **HR:1.09**
Male sex	**0.77 HR:0.89**	**NA**	**0.42 HR: 0.56**	**NA**
Hypertension	**0.62 HR:1.26**	**NA**	**0.93 HR:1.07**	**NA**
Type 2 diabetes mellitus	**0.06 HR:2.14**	**0.251 HR:1.62**	**0.73 HR:0.69**	**NA**
Hyperlipidemia	**0.36 HR:0.69**	**NA**	**0.55 HR:0.64**	**NA**
BMI (kg/m^2)	**0.86 HR:1.01**	**NA**	**0.95 HR:1.01**	**NA**
LAD-MB length (mm)	**NA**	**0.83 HR:1.01**	**NA**	**0.15 HR:1.07**
Reference diameter (mm)	**NA**	**0.48 HR:2.73**	**NA**	**0.51 HR:6.30**
Minimal stenosis (mm)	**NA**	**0.77 HR:0.45**	**NA**	**0.50 HR:0.02**
Minimal stenosis to reference diameter (%)	**NA**	**0.70 HR:0.98**	**NA**	**0.59 HR:0.94**

This result was also confirmed by Kaplan-Meier analysis, when median values were used to dichotomize variables of MB morphology (Figure 3.).

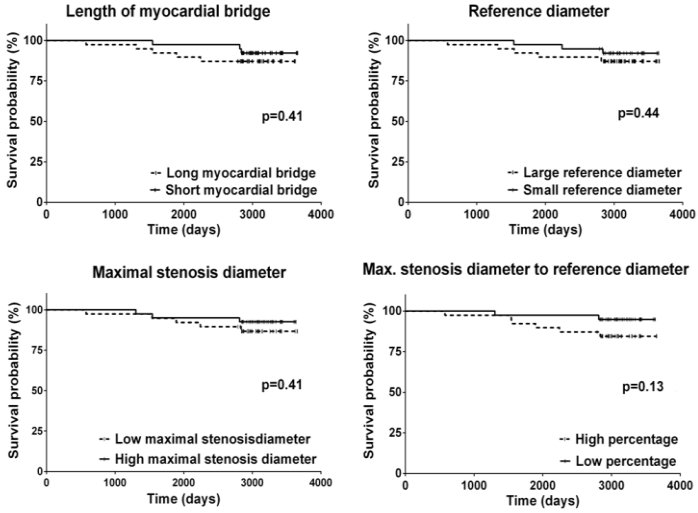

Figure 2. Kaplan–Meier curve of the long-term follow-up comparing morphological features of the myocardial bridge in LAD-MBneg

4.2. Results of the case control, CCTA study. Plaque morphology beneath the MB

There were no significant differences regarding different plaque components in segments proximal to the MB. Fatty plaque and necrotic core volumes were smaller or negligible in coronary segment beneath MB than in controls (0.07 mm3 [IQR: 0.005-0.27 mm3] vs. 12.7 mm3 [IQR: 7.4-24.4 mm3]) and 0.00mm3 [IQR: 0.00-0.04 mm3] vs. 0.06 mm3 [IQR: 0.03-2.8 mm3], respectively ($p<0.001$). (Table 3.)

Table 3. Plaque characteristics of left anterior descending coronary artery segments beneath myocardial bridge in cases and those of corresponding coronary segments in controls (median values with interquartile range [IQR]).

	Patients with myocardial bridge n = 50		**Patients without myocardial bridge (controls) n = 50**		**p**
	Median	**IQR**	**Median**	**IQR**	
Segment length (mm)	**19.6**	**14.1-24.7**	**19.4**	**14.0-24.3**	**0.95**
Vessel volume (mm^3)	**110.9**	**61.5-154.6**	**146.4**	**114.3-203.7**	***<0.001***
Lumen volume (mm^3)	**93.3**	**48.8-128.9**	**112.62**	**94.5-160.2**	***0.002***
Total plaque volume (mm^3)	**16.2**	**12.6-25.8**	**21.1**	**14.0-42.4**	***0.03***
Necrotic core volume (mm^3)	**0.00**	**0.00-0.04**	**0.06**	**0.03-2.8**	***<0.001***
Fatty plaque volume (mm^3)	**0.07**	**0.007-0.27**	**12.7**	**7.4-24.4**	***<0.001***
Fibrosus plaque volume (mm^3)	**12.8**	**5.4-18.3**	**14.3**	**10.0-26.5**	**0.07**
Calcified plaque volume (mm^3)	**1.1**	**0.4-3.2**	**1.1**	**0.2-2.9**	**0.82**

4.3. Case I. Ventricular septal rupture caused by MB, solved by interventional closure device.

An 88-year-old female patient was admitted due to chest pain which lasted for 20 hours. Her ECG showed a presumably new left bundle branch block. Coronary artery stenosis was ruled out with immediate coronarography, however a MB 19.1 mm in length was described on the mid third of the left anterior descending coronary artery. The MB's maximum systolic lumen reduction compared with the diastolic diameter was 82.8%. Laboratory results revealed markedly elevated cardiac necro enzymes. Echocardiography showed a 22×15 mm aneurysm on the distal third of the anterior septum, in which 8-mm wide VSR jet was detected (peak gradient: 100 mmHg). The positive biomarkers and the detected signs led to the diagnosis of a myocardial infarction. To clarify we carried out MRI, which demonstrated colocalization of the late contrast enhancement (myocardial infarction) and the VSR (Figure 4.).

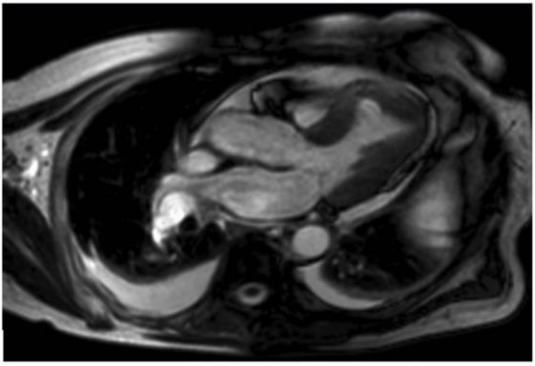

Figure 3. The aneurysm containing the rupture can be seen on the distal septum (MRI).

Taking into consideration the discrepancy between the right and the left ventricular cardiac output Qp/Qs:1.75, we decided to close the rupture. As open-heart surgery was considered high-risk, a transcatheter closure approach was decided upon. The VSD was closed with a 12 mm AMPLATZER muscular ventricular septal defect occluder. The intervention was successful and after the procedure minimal residual blood flow was detected through the device. The patient was discharged and for one year follow-up did not occur any adverse event.

4.4. Case II. Fractional flow reserve (FFR) guided stenting of a myocardial bridge

A 52-year-old man admitted with recurrent chest pain provoked by emotional stress. Laboratory tests and echocardiography revealed no abnormality. An exercise treadmill test demonstrated significant ST segment depressions on ECG at 125 W workload. Beta-blocker was up titrated. Despite the medical therapy, the patient remained symptomatic. Coronary angiography showed MB in the mid LAD artery with significant lumen compression (minimal diameter: 0.3 mm, reference vessel diameter: 2.6 mm, lesion length: 25.4 mm). Atherosclerotic lesions were not detected. FFR measurement proved relevant myocardial ischemia (Pd/Pa=0.69). After FFR measurement, the lesion was stented with a 3.0×38 mm paclitaxel eluting stent. Control FFR (Pd/Pa=0.96) verified improved hemodynamics. The patient remained asymptomatic and at the 18-months control multislice CT angiography excluded the restenosis or any other procedure-related complications.

5. Conclusions

- Isolated, symptomatic LAD-MB (associated with angina pectoris) are longer, with a more expressed systolic caliber variation compared to LAD-MB where other coronary artery disease exists. Those patients who have significant arteriosclerosis besides MB have a worse prognosis.

- The morphological features of LAD-MB do not influence long-term (10 years) survival. This result and our long-term data suggest benign nature of isolated LAD-MB.

- Fatty plaque and necrotic core volumes were smaller or negligible in coronary segment beneath LAD-MB compared to control LAD segments. This finding suggest that MB might hold a protective role against plaque rupture.

- LAD-MB might cause significant ischemia, that can lead to myocardial infarction and its mechanical complications (Case I.).

- Percutaneous coronary intervention and stent implantation might be an option in medical therapy refractory cases of LAD-MB (Case II).

6. Bibliography of the candidate's publications

6.1. Publications related to the dissertation:

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