Morphological features in myocardial bridging

Dan Dermengiu1*, Iason Vovolis2, Sorin Hostiuc3, George CristianCurcă4, Mugurel ConstantinRusu5, Lăcrămioara Luca3

Abstract: Myocardial bridging is a common coronary anomaly characterized by the presence of a muscle bridge above an epicardial artery. Although its involvement in the development of severe cardiovascular pathologies is disputed there are many proofs that it may possibly be associated, in particular circumstances, with sudden cardiac death, the development of malignant arrhythmias, atherosclerosis, myocardial infarction, hibernation or stunning, etc. The development of cardiovascular complications is mainly dependent upon the presence of a significant hemodynamic obstruction. In order for a myocardial bridging to determine such an obstruction it must present some specific morphological characteristics which we will present in this article.

Key words: Myocardial bridging, Sudden cardiac death, Cardiovascular pathologies

Although myocardial bridging is a common coronary anomaly, with a prevalence in general population averaging 30% (with limits from below 1% [1-3] obtained in angiography series to 86% [4] obtained in anatomical studies) its cardiovascular consequences are still disputed. Various studies revealed an association between myocardial bridging and sudden cardiac death [5-17], myocardial infarction [18-31], arrhythmias [27, 32-33], myocardial ischemia [10, 15, 32, 34-48], myocardial stunning [20, 32, 37, 49-50], myocardial hibernation [32, 37], etc. The incidence seems higher in patients with hypertrophic cardiomyopathy (CMH); children with CMH and MB seem to have an increased risk of increased QTc dispersion, myocardial infarction and sudden cardiac death [17]; in adults, although in association with HCM MB is proportionally more frequent, the cardiovascular consequences are uncertain. Although empirically men seemed more predispose to MB than women, clinical studies couldn’t find significant differences; by analyzing however seven studies together and cross tabulating their results a significant difference was found (Chi square value of 7.44, in the 95 confidence interval with a p value of 0.006, see Table 1).

Myocardial bridging position

The most frequent location of MB is on the left anterior descending artery (anterior interventricular artery – AIVA). Anatomic studies depicted a medium incidence of AIVA MB ranging around 58% (data obtained by computing the results from [4, 36, 52-54, 56-63] (Figure 1) whilst angiographic studies revealed an incidence of over 98% (data obtained by computing the results obtained from [1, 17, 60, 64-73]. Data obtained from MSCT varies - 16-section MSCT has results closer to angiography while 64-section MSCT is closer to anatomical studies. Myocardial fibers covering AIVA originate from the pulmonary infundibulum and cross the artery on a perpendicular
course. The medium width of the myocardial bridge is around 2-3 mm[4, 55, 74-75], higher in particular subgroups (hypertrophic cardiomyopathy, cases of sudden cardiac death, etc.); medium length is about 10-30 mm[15, 74], and usually the bridging is located in the middle third of the AIVA, more than half the cases analyzed by [1, 17, 55-56, 58, 60-61, 69, 71, 73, 76] being located here (see Figure 2).

Myocardial bridging on the right coronary (Figure 3) and left circumflex artery (Figure 4) are only rarely described in angiographic studies; anatomical studies found them in about 15% of MB cases (RCA) and 6% (LCx). These results may underestimate the true incidence of these bridges, the main reason being related to their width, much smaller than the ones on AIVA or its branches, making them harder to identify. Usually these bridges are superficial, with minimum hemodynamic impact, the origin of the fibers being atrial myocardium.

Other branches, like the diagonal, the obtuse marginal, etc., account for 19% of all MB cases. Often branches located on the free margin of the left ventricle plunge into the myocardium, take a direction parallel with myocardial fibers and never resurface [74].

Myocardial bridging width

Depending on its width MB was usually classified as superficial (Figure 5) and deep (Figure 6) [51]; Fereirra described these two variants on the AIVA only – a MB is superficial if AIVA runs within the interventricular groove and is crossed perpendicularly or at an acute angle by the bridged fibers and deep if the AIVA was deviated towards the right ventricle, is deeply situated within the interventricular septum and is crossed helically, obliquely or transversally by longitudinal fibers originating from the right ventricle and inserting into the interventricular septum [51].

Mohlenkamp updated this classification by adding incomplete myocardial bridging as a third type, defined by the presence of a tunneled artery covered partially by myocardial fibers and partially by fatty, fibrous or nervous tissue; this type can cause systolic compression and determine myocardial ischemia [77] and is somehow similar to the myocardial loops described by von Polacek [4].

Other authors relied more on MB width to differentiate superficial from deep MB – Jodocy for example gave as a cut-off value for deep MB a width of two millimeters [78]; although these two definitions of superficial/deep MN are similar they are not identical as sometimes a deep Fereirra MB can have less than two millimeters and vice versa. Konen has united Ferreira and Jodocy variants and classified MB in superficial, deep and right ventricular types [55], whilst other described a fourth type, intracavitary or subendocardial coronary artery (Figure 7) [9, 79-83].

First description of an intracavitary coronary artery was made by McAlpine in 1975 who described both intracavitary AIVA (into the right ventricle) and RCA (into the right atrium) [84]. First case of subendocardial LCx/LCx branch was described by Dermengiu et al. who identified a subendocardial sinus node artery originating from the LCx [9]. Oschner found the incidence for intracavitary RCA to be 0.09% and for intracavitary AIVA 0.2% [79].

The intracavitary LAD usually enters the right ventricle close to its origin, in an acute angle and reemerges after a long intramyocardial and subendocardial loop [79, 81]. RCA enters the right atrial myocardium in an acute angle, makes a subendocardial or intracavitary loop of about 1.5-3

<table>
<thead>
<tr>
<th>Study</th>
<th>Men</th>
<th>Women</th>
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<tr>
<td>Fereirra[51]</td>
<td>T</td>
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<tr>
<td>Ishi[56]</td>
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<td>165</td>
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<td>Total</td>
<td>732</td>
<td>343</td>
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Chi Square (Yates) 7.44 P=0.0063

Table 1. Sex ratio in various studies: T – total number of cases; P – positive cases
centimeters and then resurfaces [83-84]. A study realized on 100 cases found 29 intramyocardial branches from the RCA, suggesting a much higher occurrence of this variant than was believed previously [83].

As possible explanations are cited the thickness of the atrial myocardium and a higher origin for the coronary artery [83].

For the current clinical use, Jodocy variant seems more useful and easier to determine; also, it can be used in other MB locations. A deep MB is usually considered to have the potential to determine significant hemodynamic effects and be associated with sudden cardiac death. Morales for example separated his MB cases in two subgroups – with and without myocardial fibrosis – in the group with myocardial fibrosis medium MB width was 3.8 +/- 1.8 mm and SCD was present in 13 out of 19 cases whilst in the groups without myocardial fibrosis medium MB width was 1.9 +/- 0.9 mm. and there was no SCD case [47, 85].

**Myocardial bridging length**

The length of a typical MB is usually within the 10 to 30 mm range, only rarely exceeding 40 mm (Figure 3). A longer MB seems to be associated with more significant hemodynamic effects, and more severe clinical symptoms. For example Feldman [86] found, by increasing the length of a coronary obstruction and maintaining constant the resting and reactive hyperemic flow, the pressure gradient between aorta and the distal coronary artery to be increased; therefore a smaller dynamic stenosis can be associated with increased hemodynamic effects during at rest and reactive hyperemia if the length of the stenotic segment is increased. Other authors however couldn’t find a correlation between MB length and the severity of cardiac symptoms[69]. Typically longer bridges thicker and situated more closer to the origin [51, 87].

**Myocardial Bridging angle**

The angle between the direction of myocardial fibers and that of the tunneled coronary artery depends upon the direction of the coronary artery and the distribution of the bridged myocardial fibers.

According to Baptista [57] average angle values (α) were: for AIVA 81.5 degrees (range 40°- 90°), for AIVA branches 40° (range 1°-90°), PIVA (posterior interventricular artery) 60° (range 40°- 80°), left marginal 10° (range 1°-20°), and ramus diagonalis 43° (range 30°-60°).

During systolic contraction the most important hemodynamic effect is obtained during the perpendicular shortening over the tunneled artery. If the bridging is perpendicular on the tunneled artery the effect is dependent upon the difference between diastole and systolic length of the myocardial fibers. If the bridge is not perpendicular on the tunneled artery the effect is dependent upon
the difference between diastole and systolic length of the myocardial fibers multiplied with the sinus of the angle made by the direction of the artery and the bridge ($\Delta l \sin \alpha$) (Figure 8). Even though a correlation between the angle and clinical symptoms/morphological signs wasn’t yet researched, the fact that the highest angle is associated with the location where MB is correlated with the most severe signs/symptoms (AIVA) might suggest a possible correlation. Specific studies are needed to clarify this problem.

**Coronary intima**

Intima of the tunneled artery has different characteristics before, below and after the bridge. Before the bridge the medium width of the intima is about 406.6 µm while under the bridge is about 66 µm. Endothelial cells have a helical orientation (associated with a laminar blood flow and a high endothelial shear stress under the bridge and polymorph, flat or polygonal shapes before the bridge (shapes associated with low endothelial shear stress); the latter are associated with increased mitotic and apoptotic activity, increased MMP-2 and MMP-9 synthesis, increased gap junctions, etc., all pro-atherosclerotic factors. Intimal hyperplasia and atherosclerosis are the landmarks of a hemodynamically significant myocardial bridging in the proximal area whilst in the distal part coronary hypoplasia and decreased atherosclerotic disease are the most common.

**Atherosclerosis and Myocardial Bridging**

The presence of a myocardial bridge was shown to suppress atherosclerosis below the obstruction site [56, 87-89], irrespective of serum cholesterol and blood pressure. The main cause seems to be the development of a dynamic obstruction which proximally decreases endothelial shear stress and subsequently favors various metabolic pathways involved in atherogenesis [90], although other factors like increased proximal wall stress or coronary geometry can be involved as well [91].

Under and below the bridge on the other side atherosclerosis occurs rarely, due to a decreased tensile stress, increased endothelial shear stress and decreased wall motion during the cardiac cycle [92]. There are a few studies dealing specifically with the association between atherosclerosis and myocardial bridging; for example La Grutta found proximal intima to be without atherosclerosis in 33%, with positive remodelling in 27%, with stenosis less than 50% in 20% and with stenosis more than 50% in 20% of cases; distally atherosclerosis was absent in 84%, positive remodelling in 11%, stenosis less than 50% in 3% and stenosis more than 50% in 2% [93].

Hsu, by inducing atherosclerosis on rabbit models (whose coronaries are entirely intramyocardial) found that, even if severe atherosclerotic lesions were developed on the aorta and other major vessels, the intramyocardial parts of the major coronary arteries were free of it [74]. Ishikawa found that atherosclerosis was more pronounced proximally, has a predilection for extension towards the coronary ostium, augments the natural history of MB, and is a predisposing factor for myocardial infarction [87].

Atherosclerosis under/below the bridge normally occurs in two situations: the presence of a highly atherogenic state (e.g. congenital or acquired defects of cholesterol or plasma lipoproteins) or a superficial/incomplete bridge without significant hemodynamic consequences. For example, Ishi found the extent of athero-sclerotic lesions on the right coronary to be greater compared to the left anterior
descendent in cases of myocardial bridging (RCA bridges are usually more superficial and with less hemodynamic impact); in the general population atherosclerotic lesions on the AIVA are more frequent than the ones found on the RCA [56].

Fig. 4. Deep LCx myocardial bridging

Fig. 5. Superficial myocardial bridging on the anterior interventricular artery

Fig. 6. Deep myocardial bridging on the anterior interventricular artery: PT – pulmonary trunk, CxA – circumflex artery, LCA – left coronary artery

Fig. 7. Subendocardial sinus node artery, originating from the left circumflex artery

**Myocardial fibers**

There seem to be significant differences within the myocardial structure between samples taken from the bridging, areas vascularized by tunneled coronaries, and the rest of the myocardium. The nuclei from bridged myocardial fibers are always smaller than the ones corresponding to myocardial fibers from other places, suggesting that the mechanical response of these fibers is significantly distinct, probably due to a heterogeneous stretching overload determined by the particular circum-
stanes of the bridge – an isolated band of cardiomyocytes without circumferential contact with other myocardial fibers leads to a reduced response to mechanical overload [54].

By comparing the characteristics of the myocardium vascularized by a tunneled artery versus myocardium vascularized by a non-tunneled artery interstitial fibrosis, interstitial edema and hypoxia are more severe in cases with bridging (personal, unpublished data); these results are similar to the ones obtained by other authors: Brodsky for example, comparing hearts with and without MB found a significantly increased interstitial fibrosis in samples obtained from the anterior wall of the left ventricle as compared with equivalent samples from cases without MB[94]. A quantitative computerized analysis found interstitial fibrosis to be 33% more severe in patients with myocardial bridging (significant, p=0.0006) [94].

Theoretically, for the same systolic myocardial fiber shortening (Δl) the greatest effect is obtained when the fibers are perpendicular on the tunneled artery; if the fibers are not perpendicular the effect is equal to Δlsinα.

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**References**


