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Myocardial Bridges in the ERA of Non-Invasive Angiography

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1. Introduction

Cardiovascular (CV) and cerebrovascular (CBV) diseases are the leading causes of mortality in developed countries. In these countries CV risk factors like smoke, obesity, sedentary life, dyslipidemias, hypertension and diabetes are well recognized and efforts have been efficaciously undertaken so that CV and CBV mortality in the second half of the last century has been significantly reduced. Cardiovascular and CBV diseases are also the leading cause of mortality in the developing world where in the last century we witnessed a rapid epidemiological as well as nutritional transition related mainly to increased urbanization and market globalization. Now the majority of CV and CBV mortality occurs in low and middle-income countries [1].

Indeed the major responsible of CV as well as CBV diseases is the vascular atherosclerotic process. In particular the atherosclerotic calcified and non calcified plaques that cause coronary artery vessel lumen reduction are worldwide the leading cause of myocardial ischemia, which can lead to asymptomatic myocardial dysfunction, life threatening arrhythmias, angina, myocardial infarction and sudden death.

Besides the atherosclerotic coronary artery diseases there are also other non atherosclerotic coronary artery vessel lumen reductions, although their prevalence is less common. The non atherosclerotic coronary artery diseases are related to prolonged coronary artery spasm, hypertrophic cardiomyopathy, vasculitis and congenital coronary anomalies.

Congenital coronary artery anomalies are a heterogeneous group of diseases. In the majority of cases congenital coronary artery anomalies lack clinical significance and are merely epiphenomena found accidentally during necropsies, while performing invasive or non invasive coronarography or during surgical interventions. However in some cases they may be

responsible for chest discomfort, malignant arrhythmias, fatal or non fatal acute myocardial infarction, ventricular septum rupture, myocardial stunning, paroxysmal atrio-ventricular block, syncope and sudden death. In particular 19% of sudden deaths in young athletes are due to coronary artery anomalies [2].

In this chapter we will focus our attention on describing the second most common type of coronary congenital anomaly: the myocardial bridges (MBs). We will discuss the nature of MBs and how to diagnose them with particular attention to the use of cardiac computed tomography (CCT).

2. Classifications

In describing coronary anomalies Angelini et al. proposed that a condition should be considered "normal when it is observed in > 1% of an unselected population; normal variant, an alternative, relatively unusual, morphological feature seen in > 1% of the same population; and anomaly, a morphological feature seen in < 1% of that population". These Authors performed their study using cine-angiograms [3]. The procedure used to define a normal from an abnormal coronary may be a bias. In fact coronary angiography is performed in symptomatic patients while necropsies are usually done for medico-legal purposes especially for violent non hospital based deaths whereas necropsy for hospital based deaths is decreasing. This bias explains why coronary anomalies of origination and course are rare during autopsy (0.17% of the cases) while their incidence is higher in the population of patients referred for coronary angiography (0.6-1.3%).

Clinically coronary anomalies are evaluated with the same diagnostic tests used to study the atherosclerotic coronary artery diseases: electrocardiogram, exercise stress test, trans-thoracic and trans-esophageal echocardiography, stress echocardiography, stress single photon emission computed tomography, myocardial perfusion imaging, magnetic resonance, fractional flow reserve, electron beam computed tomography, invasive coronary angiography (ICA) and non-invasive coronary angiography with CCT.

Myocardial bridges in humans are inborn coronary anomalies of intrinsic coronary arterial anatomy with an intramural course. Although it was Reyman in 1737 and then Black in 1805 who first described, as a curiosity during necropsy, the presence of a MB overlaying the left anterior descending coronary artery (LAD), the first detailed postmortem analysis of this anomaly was reported by Geiringer in 1951 [4].

In fact in humans coronary arteries and their main branches have an epicardial course running over the cardiac musculature. In the presence of a MB a portion of one coronary artery or more dips into and underneath the heart muscle to come back out again in the majority of the cases. This condition is also known also as "intramuscular coronary artery", "tunnelled artery", "myocardial loop", "mural coronary artery", "intramural coronary artery", "myocardial bridging" or "coronary artery over bridging".

The real incidence of this entity is unknown and varies according to the procedure used to study it. Myocardial bridges are rare in patients referred for cardiac surgery (0.2-0.3%) or ICA (0.4-4.9%) while they are very frequent during autopsy (5.4-85.7%) [5]. Such disparate autopsy prevalence rates may result from the selection and preparation of the hearts as well as variations in definitions of MBs and probably also to ethnicity [6]. On average MBs are present in about one third of adults [7]. Thus, MB should not be defined as a congenital coronary anomaly, but rather as a normal variant [3]. According to some Authors superficial MBs may not be exclusively congenital in origin, but may result from adulthood disease processes that partially cover the artery with fibro-fatty connective tissue [7].

There are also myocardial loops that are thinner and derive from atrial myocardium, surround the vessel three quarters of the circumference, and return to atrial myocardium. Occasionally, a bridge may involve also a coronary vein. Both, myocardial loops and venous bridges appear to have no clinical relevance [7].

The wide variation in frequency of MBs indicates that many MBs do not produce symptoms. Subjects may become symptomatic after the third decade of life unless MBs are associated with precipitating factors (i.e. high heart rate, myocardial contractility state, hypertrophic cardiomyopathy, decreased peripheral vascular resistance). Myocardial ischemia due to MB could be attributed to a combination of the following factors: increased heart rate compromising the diastolic filling, exercise-induced spasm, and systolic kinking which may cause endothelium damage with platelet activation and thrombus formation [4, 7].

A milestone work in studying MBs is that of Ferreira *et al*. These Authors found a MB in 50 of the 90 hearts studied (55.6%) mainly on LAD. They distinguished MBs into two types. In the superficial type (75% of cases) the myocardial fibres cross the artery transversely towards the apex of the heart at an acute angle or perpendicularly. In the deep type (25% of cases) the myocardial fibres arise from the right ventricular apical trabeculae, surround the LAD with a muscle bundle that crosses the artery transversely, obliquely, or helically before terminating in the interventricular septum. In the deep variant, no direct contact occurs between the MB and the adventitial wall of the tunnelled artery. In addition, adipose, neural, and loose connective tissues are interposed between the MB and the artery. The Authors speculated that the vessel may be more distorted and compressed in the deep type of MB [8].

Some superficial MBs may be not completely covered by myocardial fibres, but by a thin layer of connective tissue, nerves and fatty tissue [7]. Obviously in these cases the systolic compression is light and may not be appreciated during angiographic studies.

Recently the use of multi-detector computed tomography (MDCT) made it possible to visualize in vivo the MBs. Konen *et al*. were moreover able to describe three types of MBs. Beside the superficial type (29% of cases) and deep type (41% of cases) described by Ferreira *et al*. the Authors characterized a third one called "right ventricular type" (29% of cases. In this type the descending coronary artery "disappears" and is visible only in the axial images where it has a course near the right ventricular wall. This type of MB seems to be more potentially pathologic and more difficult to treat surgically [5].

Myocardial bridges can be classified also depending on the thickness, the length and the number (one or more) of MBs. Obviously MBs are also classified according to the coronary artery and the segment of the coronary artery involved. The majority of MBs are in the mid portion of LAD. However MBs have been found also over the proximal and distal parts of the LAD, the diagonal and marginal branches and over the posterior interventricular branch of the LAD. Bridging of the circumflex or the right coronary artery or one of their branches is not so common [9]. In the presence of two parallel LADs one of them frequently takes an intramural course [7].

Although autopsy studies did not demonstrate any difference in the frequency of MBs by age or sex [8, 10] angiographic studies indicate that males have a higher incidence and longer MBs probably owing to a higher musculature of the body in respect to females [9, 11].

A fairly large percentage of subjects with MBs may have concomitant atherosclerotic, muscular, or valvular heart diseases, which may independently affect the clinical outcome as well as the treatment strategy [4]. Typically, the MB patients are 5 to 10 years younger than those with symptomatic coronary disease. Typical angina is present in 55% to 70% of the cases, and atypical angina is often reported in association with rest angina. The co-presence of MBs with atherosclerotic coronary artery disease should be taken into account when it is not possible to detect a culprit lesion in symptomatic patients. Although MBs have excellent prognosis even in patients with ≥ 50% systolic compression, early diagnosis and treatment are important due to their possible complications [5].

Nowadays there is a debate concerning the evaluation of asymptomatic young athletes who have a low probability to have an atherosclerotic coronary artery disease. Some of these athletes however during or just after physical exertion or in circumstances non-associated with sports, during routine daily activities, while sedentary or even asleep may have an unexpected death. In an autopsy-based registry comprising 1866 young athletes (19 ± 6 years) the cause of sudden death was in 56% of the cases due to CV disease. Of these the cause of sudden death was attributable to hypertrophic cardiomyopathy in 36% of the cases and to coronary artery anomalies in 19% of the cases (119 cases of coronary artery anomalies of wrong sinus origin and 24 cases of MBs) [2].

3. Anatomic properties of myocardial bridges

In a necropsy study Morales et al. found that hearts with MBs but with no evidence of other cardiac abnormalities had gross or microscopic alterations (or both), such as interstitial fibrosis, replacement fibrosis, contraction-band necrosis, or increased vascular density, in areas of the myocardium supplied by the bridged LAD. According to the Authors, the histologic heterogeneity of these findings, with closely interspersed patches of normal myocardium, is related to the attenuation of blood flow due to the intramural course of the vessel. These blood flow alteration may induce chronic and/or acute transient myocardial ischemia. The myocardial ischemia may be responsible of life threatening

arrhythmias, such as ventricular fibrillation. In fact many of the analyzed hearts with MBs were from subjects who died of sudden death [12].

Recently several histopathologic studies clearly demonstrated that while the arterial intima beneath the MB is significantly spared from atherosclerotic changes, the segments proximal to the MB are not interested by this atherosclerosis suppression. By scanning electron microscopy the endothelial cells in the tunnelled segment had a helical, spindle-shaped orientation along the course of the segment as a sign of laminar flow and high shear. In the segments proximal to the MB the endothelium was flat, polygonal, and polymorph, indicating low shear. Low shear stress facilitates adhesion and aggregation of platelets followed by subsequent thrombosis and is associated with a release of endothelial vasoactive agents such as endothelin-1, nitric oxide synthase and angiotensin-converting enzyme which favour mass transfer of lipids into subentothelial space [13, 14]. Higher shear stress on the other side, results in lower levels of these vasoactive agents and in a suppression of lipid infiltration into subentothelial space. It has also been found that the intima beneath the bridged segment always consisted of contractile-type smooth muscle cells, while the segment proximal to the MB had synthetic-type smooth muscle cells. These types of cells usually proliferate and produce collagen fibrils and elastic fibres in the intima as atherosclerosis progresses. Moreover in the proximal segments to the MB the flow is turbulent accentuated by the retrograde blood flow caused by the "squeezing" of the MB during systole and a "sucking effect" of the proximal segment during the early phases of diastole. The increase of local wall tension and stretch in the segment proximal to MB may induce endothelial injury and plaque fissuring with subsequent thrombus formation. All these complex hemodynamic alterations may explain the atherosclerotic plaque formation, mainly eccentric, at the entrance of the tunnelled segment [4, 7, 9, 10, 14]. However, although the endothelium of MB is spared from atherosclerotic lesions its function seems to be significantly impaired as estimated by the vasoactive response to achetylcholine and increased vasoconstriction [15]. These data suggest that MB itself may have a dysfunctional endothelium, a strong atherogenic factor that can cause myocardial ischemia, chest pain, life threatening arrhythmias, and sudden cardiac death [5].

4. Angiographic findings

The current gold standard technique for diagnosing MBs is coronary angiography. Portman and Iwing in 1960 were the first to report the radiological appearance of transient stenosis in a segment of the LAD during systole in a 19 year old patient. The typical angiographic finding of a MB is a systolic narrowing of an epicardial artery, known also as a "milking effect" phenomenon induced by systolic compression of the tunnelled segment. Another angiographic finding is the presence of the "step down-step up" appearance, namely, a significant tortuosity of the segment beneath MB at the entrance (step-down) and the exit (step-up) sites [4, 10] (Fig 1).
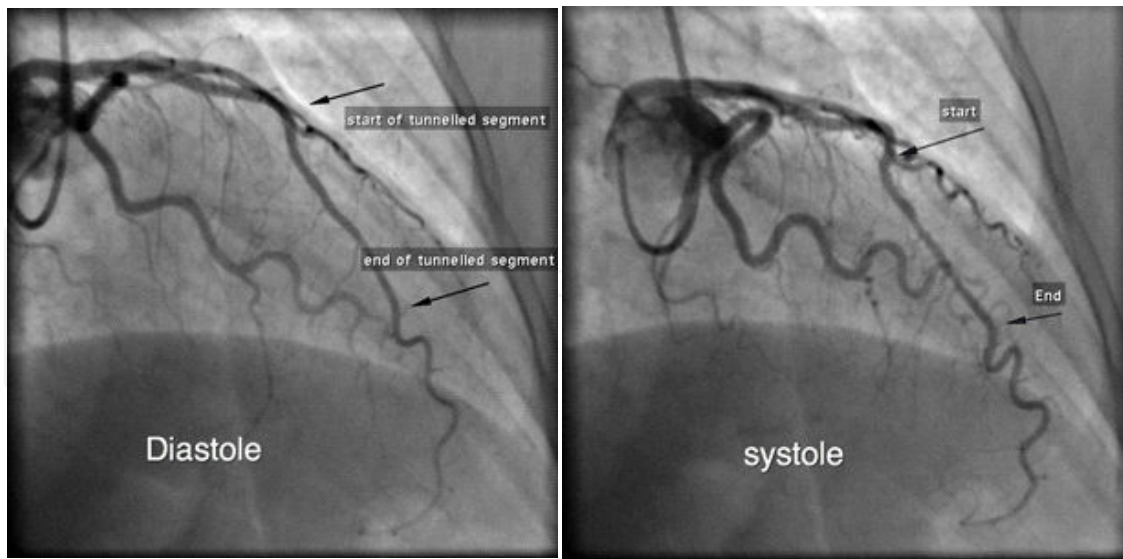
Figure 1. Myocardial bridging on conventional coronary angiography in diastole and systole. Compression at the middle of the left descending coronary artery occurs during systole with a clear step-down and step us phenomenon. Arrows indicate the beginning and the end of the tunnelled segment. The left descending coronary artery and the circumflex artery are free of atherosclerotic lesions.

The systolic compression is usually eccentric rather than concentric [11]. However, also the diastole is compromised. In fact measurements in patients with MB have shown a persistent diastolic diameter reduction enduring mid diastole. In a series of 42 patients a mean maximum systolic diameter reduction of 71% was found with a persistent reduction of 35% during mid diastole, while 12% of patients showed a reduction of more than 50% in mid diastole [16]. Almost the same results were found by Bourassa et al. in a frame-by-frame analysis of cine-angiograms during a complete cardiac cycle. The Authors were able to demonstrate that 17 of 20 patients (85%) with a ≥ 75% milking effect of the LAD had an extension of the obstruction into diastole, which averaged 136 ms or 26% (range 4% to 50%) of diastole [4]. In borderline cases intracoronary nitroglycerine administration may uncover the systolic coronary compression. The milking effect is evaluated as grade I when the narrowing is less than 50%, grade II when it is between 50 and 75%, and grade III when it is greater than 75% [4].

The frequency of MBs reported in angiographic studies varies from 0.5 to 33%. This wide variation at angiography may in part be attributable to technologic advances in cine-angiography; to the orientation of the coronary artery and myocardial fibres; to the state of myocardial contractility; to the fact that small and thin bridges cause little compression badly detectable during angiography specially with no previous percentage of systolic narrowing specified for the designation of MB; if the study was retrospectively reviewed for the specific purpose of assessing the frequency of MBs; to sample size and finally to different population selection and probably also to ethnicity. In patients with MBs chest pain is the common reason for angiography. At angiography the mid portion of the LAD is the most frequently affected vessel.

A limit of ICA is that it estimates coronary artery diameter as a percent by comparing it with the adjacent segment, which arbitrarily is considered normal. This visual procedure to esti‐ mate lesions has a high degree of intra and inter-observer variability. These limits have been reduced by improving the software (quantitative coronary angiography) and hardware (flat panel digital detectors) of angiographs.

5. Intravascular ultrasound, intracoronary Doppler and intracoronary pressure

The performance of ICA increased with the introduction of important tools such as intravas‐ cular coronary ultrasound (IVUS), that for the first time visualized, in vivo, both vessel lu‐ men and walls, intracoronary Doppler-ultrasound and intracoronary pressure-wire. These tools increased our understanding of the morphological and functional features of MBs.

Although its anatomy and physiology are not fully understood the "half moon phenom‐ enon" is a characteristic and highly specific IVUS observation of MBs as it is only found in the tunnelled segments, but not in the proximal or distal segments of the vessel or in other arteries. The "half moon phenomenon" appears as an echolucent area surrounding the bridge segment. In the presence of a "half-moon phenomenon" the milking effect can be induced by intracoronary provocation tests, such as intracoronary nitroglycerin injec‐ tion, even if the bridge was previously angiographically undetectable [11]. Ultrasound pullback studies confirmed the histological findings of absence of atherosclerosis within the tunnelled segments, whereas there was a plaque in the segment proximal to the MB in about 90% of subjects. None of these proximal atherosclerotic lesions detected by IVUS has been seen on angiography confirming the known superiority of IVUS on an‐ giography in detecting atherosclerotic plaques [11].

In presence of MB the pullback of the intracoronary Doppler (0.0014 inch wire) reveals a characteristic flow pattern: "fingertip phenomenon" or "spike-and-dome pattern" which is present in most of the patients with MBs. This flow pattern described by Ge et al. [17] can be observed within and just proximal to the tunnelled segment and consists in a sharp accelera‐ tion of flow in early diastole followed by immediate marked deceleration and a mid to late diastolic pressure plateau. The Authors explain this flow pattern as an increase in the pres‐ sure gradient in the early diastole as a result of reduced distal coronary resistance while there is a delay in the relaxation of the myocardial fibres. The subsequent sharp deceleration in the coronary flow velocity results from the relaxation of the myocardial fibres and an in‐ crease in the vascular lumen. After the release of the compression, the lumen of the bridge segment remains unchanged in the second half of diastole and this corresponds to the pla‐ teau of the flow pattern at this phase. In deep myocardial bridges, rapid diastolic forward flow may be preceded by end-systolic flow inversion as a result of systolic squeezing of the bridge segment. In the subjects where the "fingertip phenomenon" is not present (13% of cases) this may be related to the fact that the bridging segment was not so severe to induce the hemodynamic disorders that lead to the "fingertip phenomenon" formation [17]. The

consequence of these phenomena is that in the segment proximal to the MB the pressure can become even higher than that in the aorta. At the entrance of the MB the high wall stress and disturbance in blood flow promote atherosclerosis [17]. Finally in subjects with MBs the coronary flow reserves, defined as the ratio of mean flow velocity achieved at peak hyperemia obtained after intracoronary injection of papaverine or adenosine to mean resting flow velocity, is frequently reduced (2.0-2.6), values below 3.0, which is regarded as the lower normal limit [4].

6. Cardiac computed tomography

The introduction of multidetector row systems in the field of cardiac computed tomography (CCT) has made imaging of the heart and in particular of epicardial coronary arteries feasible. In the last two decades CCT has been used to study different group of subjects becoming in some cases the new "gold standard technique" instead of invasive coronary angiography (ICA), because of it's ability to visualize correctly coronary arteries and most interestingly to obtain this information non-invasively [18, 19, 20].

In particular CCT is widely used to study coronary artery anomalies. In fact ICA has some limits as it provides a few 2D view images of the coronary arteries and sometimes it fails to clearly visualize the relationship between the coronary vessels and the surrounding structures. With ICA it is not always easy to selectively engage the anomalous coronary vessel, which may lead to the erroneous assumption that the coronary vessel is occluded. In addition with this traditional 2D technique is more difficult to understand the course of the coronary vessels within the heart and discern the anterior versus the posterior direction of the anomalous vessels. On the other side CCT provides an unlimited number of 2D reformatted images as well as 3D images of the single vessel making it possible to have a 3D depiction of the whole heart [19].

The CCT information is very useful to the surgeon as it helps him to plan the surgery by seeing the exact course of the vessel and its relationship within the heart and with the other intra-thoracic organs and chest wall [19]. In addition, in case of extensive and deep MB there may be a technical challenge during coronary arterial bypass. The intramuscular coronary artery may be difficult to localize and may require the use of intraoperative echocardiographic Doppler to explore the coronary artery to avoid, for example, accidental opening of the right ventricle during dissection of intramuscular LAD. It has also been suggested that a preoperative diagnosis of MBs on CCT may help in planning the surgery strategy allowing a key information for selecting the standard midsternotomy with or without cardiopulmonary bypass (coronary artery bypass graft or off-pump coronary bypass graft, respectively) or a minimally invasive approach through the small left anterior thoracotomy [5].

In the recent American Appropriate Use Criteria Task Force for CCT, the use of CCT in the "assessment of anomalies of coronary arterial and other thoracic arteriovenous vessels" was pointed to be most appropriate (i.e. the test is acceptable and considered a reasonable ap-

proach to study the disease and its expected incremental information, combined with clinical judgment exceeds the expected negative consequences by a sufficiently wide margin) with a score of 9 out of 9 [20].

Cardiac computed tomography has however some important limits that must be considered. Invasive coronary angiography is still superior over CCT because it has, for the moment, a higher spatial (<0.16 mm vs approximately 0.4 mm of CCT) as well as temporal resolution (33 msec. vs 140 to 200 msec. of the recent cardiac computed systems or 83 msec. of the dual source system). Another limit of CCT present also with the currently available 64 channel systems is related to patient's heart rate which must be rhythmic and around or less than 60-64 beats per minute. Patients with atrial fibrillation or with a heart rate that can not be reduced to a rate of 60-64 beats per minute, for the moment, are not eligible to undergo this kind of examination. The introduction of new tools like the "ECG-tube current modulation" and the "step and shoot" procedures and the 128, 256, 320 and 640 channel or dual source scanners offers the possibility to study also patients with higher heart rates and with atrial fibrillation, making it possible to image the entire heart not only, as it is now, in a single breath hold, but in a single heartbeat [19]. Moreover less than or equal to 5% of patients have a un-valuable CCT scans due to motion artefacts, because the patient cannot follow breathing commands, involuntary motion of the diaphragm or because the patient is over-weigh or has respiratory problems.

Particular attention must be also given to the dose of radiation delivered to patients. In the commonly used CCT systems the amount of radiation, expressed as units of millisieverts (equivalent to millijoules per kilogram of tissue), absorbed by patients during the test is 2-4 folds that of ICA [19]. However the introduction of improvements in CCT technologies decreased significantly the radiation dose to equal almost that of traditional coronary angiography [21]. Finally it is worth noting that both ICA and CCT use non-ionic contrast medium to visualize coronary artery lumen. For this reason particular attention must be given in allergic patients and in patients with a pre-existing renal impairment [19].

While studying MBs it is also important to consider that CCT analysis are mainly performed with images reconstructed during diastole (70-80% of the cardiac cycle) when there is the maximal vasodilatation and minimal motion artifacts. Conversely maximal lumen narrowing of MB is during the systolic phase (30-40% of cardiac cycle) where usually there are more motion artifacts. To better evaluate patient's MB it is therefore important to analyze the whole cardiac cycle, but good quality CCT images in both the diastolic and systolic phases are obtained only with the more recent CCT machines.

7. Myocardial bridges and cardiac computed tomography

For the final interpretation of MBs conventional post-processing tools are used, namely: cross-sectional imaging, multiplanar reconstructions (MPR), curved MPR (cMPR), maximum intensity projections (MIP) and three-dimension volume rendering (Fig 2).

Figure 2. Myocardial bridging at 64 multi-detector computed tomography. Volume rendering image of the heart (A). 3D image of the coronary tree (B). Multiplanar reconstructed image of the left descending coronary artery. The middle segment of the vessel is tunnelled by overlying myocardium (C). It is clearly evident the step up phenomenon.

The high temporal resolution obtained with the most recent scanners or dual source scanners enable the visualization of the vessel lumen during most of the cardiac cycle, and thus permit the observation of the milking effect in the 4-dimensional reconstruction [22]. Cardiac computed tomography helped to better evaluate the anatomical properties of MB. Several Authors using CCT confirmed what was already know from necropsies, CCA and IVUS studies that the tunnelled segments are spared from atherosclerotic changes [23]. However Zeina et al. found that the thickness and length of the bridge correlated with the presence of stenosis in the LAD proximal to the MB suggesting that the MB may predispose to the development of atherosclerosis in the coronary artery segment proximal to the bridge and that MB should be considered an anatomic risk factor in the evaluation of coronary artery disease patients [23]. Also Takamura et al. demon-

strated that, in patients with culprit lesions in the LAD segment proximal to MB, the length and thickness of MBs were significantly greater, and the distance from the orifice of the left coronary artery to the entrance of MB was significantly shorter than those in patients with no culprit lesion in the LAD segment proximal to MB [24]. These results are similar to those of the autopsy studies that demonstrated that the anatomical properties of MB muscle were closely associated with a shift of coronary intimal lesion more proximally, an effect that may increase the risk of myocardial infarction [14].

Since the introduction of CCT in the last decade of last century many papers have been published showing the feasibility of CCT in evaluating patients with MB (Fig 3).

In particular many papers compared CCT to ICA. Recently a significant correlation was found between the within-MB diameters obtained with CCT and ICA during the systolic (1.3±0.3 mm vs. 1.2±0.5 mm: r= 0.394, P=0.028) and diastolic phases (1.4±0.4 mm vs. 1.6±0.6 mm: r= 0.524, P=0.001) [25]. However CCT is superior to ICA in diagnosing the presence of MB. Kim et al. found that while dynamic compression was present in 13.3% of the subjects (40/300) who underwent ICA, CCT revealed that 58% of the subjects (178/300) had myocardial bridging (partial encasement in 57 and full encasement in 117 subjects) [26]. Leschka et al. found that MB was revealed with CCT and ICA respectively in 26 and 12 of the 100 subjects studied [27].

When comparing CCT with IVUS, the sensitivity of detecting MB by CCT was found to be 93%, specificity 100%, positive predictive value and negative predictive value 100% and 91% respectively. A significant correlation was also observed between lumen diameters derived from CCT and IVUS (systolic phase: r=0.87, P<0.05; diastolic phase: r=0.92, P<0.05). Although minimal and maximal diameters of MB during systolic and diastolic phases derived from CCT were significantly smaller than those from IVUS (2.4±0.4 mm vs 2.6±0.5 mm, P<0.05) and (2.9±0.3 mm vs 3.3±0.3 mm, P<0.05) the narrowing percent derived from the two methods was similar ((21.4±10.9% vs 17.4±7.6%, P>0.05). The Authors however note that CCT offers a safer, more comfortable and cost-effective examinations (in China the prices of IVUS and MSCT are 1500 US $ and 95 US $ respectively [28].

Usually in the CCT studies where MB is evaluated the coronary arteries are classified according to the American Heart Association classification system: right coronary artery: 1, proximal; 2, mid; 3, distal; 4a, posterior descending; 4b, posterolateral; left main coronary artery: 5, LAD; 6, proximal; 7, mid; 8, distal; 9, first diagonal; 10, second diagonal; circumflex coronary artery: 11, proximal; 12, first marginal; 13, mid; 14, second marginal; 15, distal.

All studied performed the evaluation of MB mainly in the diastolic phase while a few studies performed it in the systolic phase due to technical problems related to the increased motion of the heart due to myocardial contraction in the systolic phase and to the limited temporal resolution of routinely available scanners [5, 22, 27, 29].

In the literature there is not a consensus in the definition of MB. Usually MB is defined as the existence of tissues exhibiting soft tissue density covering a part of the vessel, which had the same contrast enhancement as myocardial tissue [24].
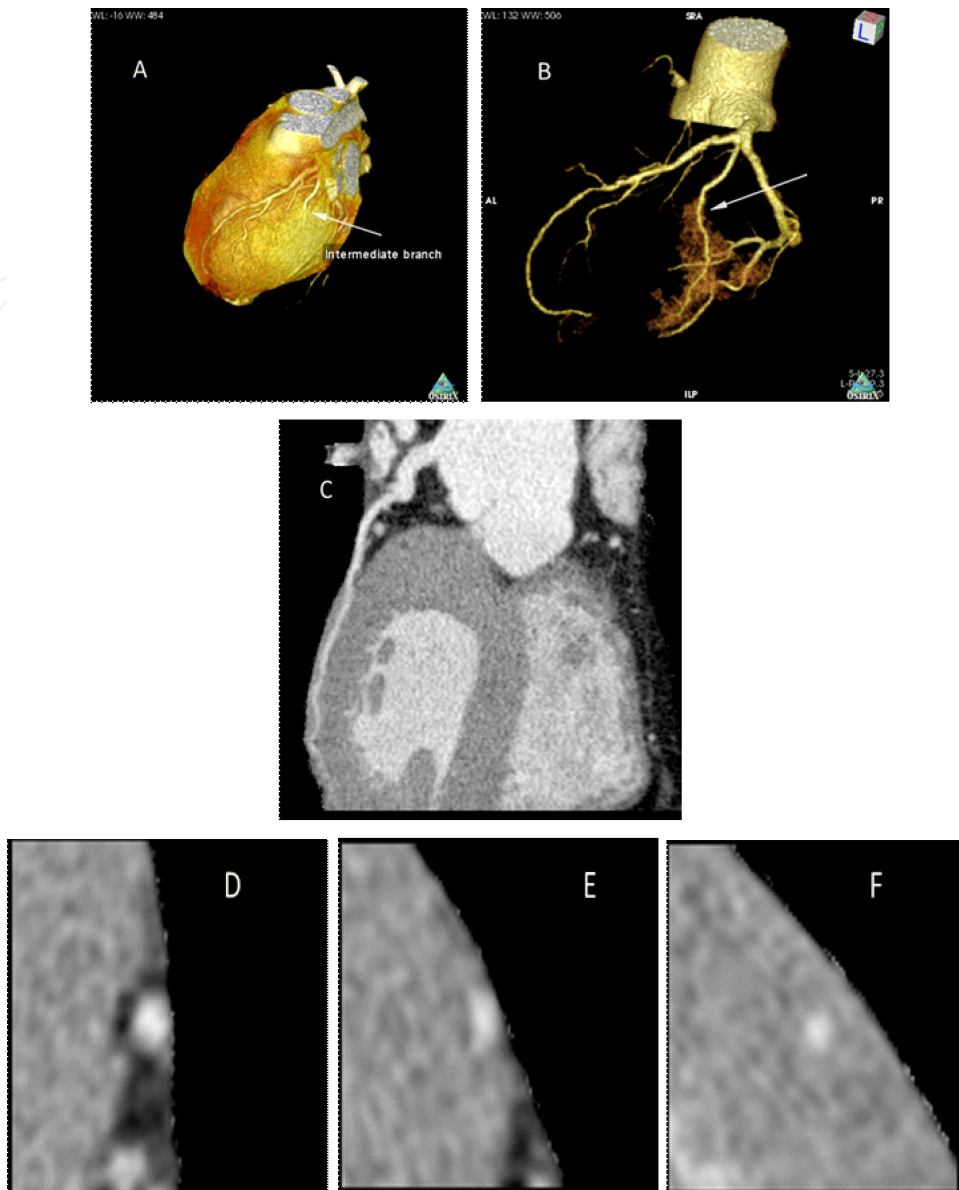
Figure 3. Myocardial bridging at 64 multi-detector computed tomography. Volume rendering image of the heart (A). 3D image of the coronary tree (B). Multiplanar reconstructed image of intermediate branch which is tunnelled by overlying myocardium (C). While in D the vessel has an epicardial course in E and especially F the vessel is completely encompassed by the myocardium.

The length of MB is usually defined as the distance of the covering myocardial tissue from the entrance to the exit of the tunnelled artery, which is measured by curved MPR images (i.e. parallel to the course of the vessel) [24].

There are several definitions to describe the depth of MB. In the majority of the papers the depth is defined as the thickness of the deepest part from the surface of the covering myocardial tissue to the tunnelled artery, which is measured in an axial image (i.e. perpendicular to the course of the vessel) (Fig. 3) [24]. Myocardial bridges were divided into two types: superficial and deep. In the superficial type a myocardial band overlies the vessel with no de-

viation of the vessel into the myocardium. In the deep type the vessel dips as a U-shaped curve into the myocardium [30]. Another classification divided MB in complete or incomplete. The complete types of MB were those where it was possible to demonstrate the continuity of myocardium over the tunnelled segment [31].

Another definition of superficial and deep MB was given by Jodecy et al. These Authors defined the MB as "deep" when the vessel was surrounded entirely by myocardium in depth of a more than 2 mm, whereas it was defined as "superficial" when the vessel appeared either not entirely surrounded (but with a minimum of 75% of the circumference), or entirely surrounded by myocardium in less than 2 mm depth [29].

Author [reference]	type of MDCT	number of pts (% of MB)	% of MB in LAD	length mean (range)	depth mean (range)
Kawaka et al. [34]	16 slices	148 (26)	91	20±8.6 (10.5-50.2)	1.8±0.7 (1.1-3.7)
Kantraci et al. [35]	16 slices	626 (3.5)	100	17 (6-22)	2.5 (1.2-3.3)
Ko et al. [36]	16 slices	401 (5.7)	91	15.7 (5-27)	3.2 (1.0-7.0)
Canyigit et al. [31]	16 slices	280 (38.5)	81.6	15.8 (4-50.9)	1.7 (1-6.4)
Chen et al. [37]	16 slices	276 (8.7)	76.7	24.6±11.8 (5.2-50.6)	3.7±1.9 (0.5-9.1)
Takamura et al. [24]	16 slices	228 (18.8)	100	20.0 (2.4-54.7)	1.7 (0.4-9.7)
Zeina et al. [23]	16/64 slices	300 (15.8)	87.5	19.5±5.7 (8-30)	2±0.6 (1-3.1)
Konen et al. [5]	40/64 slices	118 (30.5)	72	23±9 (13-50)	(0.1-6.2)
Lubarsky et al. [38]	64 slices	245 (44)	100	28.7±16.5	NA
Johansen et al. [32]	64 slices	152 (32)	69.4	NA	NA
Kim PJ et al. [26]	64 slices	300 (58)	100	29.1±15.5	1.4±1.0
Leschka et al. [27]	64 slices	100 (26)	98	24.3±10 (8-53)	2.6±0.8 (1.4-4.8)
Koşar et al. [39]	64 slices	700 (37)	NA	NA	NA
Kim SY et al. [30]	64 slices	607 (6.4)	84.2	16.3±6.3 (6.9-30)	1.8±0.8 (0.5-3.9)
Jeong et al. [25]	64 slices	120 (25)	47.4	20.5±6.8 (8-35)	2.3±1.2 (0.8-6.6)
Jodocy et al. [29]	64 slices	221 (23)	91	14.9±6.5 (2.5-43.8)	2.6±1.6 (0.5-9.4)
La Grutta et al. [40]	64 slices	254 (29)	93	NA	NA
Wrianta et al. [33]	64 slices	934 (16.3)	94	NA	NA
Jacobs et al. [41]	64 slices-DSCT	506 (10.4)	96	23.4 (4.1-53.9)	2.6 (1-7.8)
Lu et al. [22]	DSCT	53 (39.6)	57	23.2±9.5	3.5±1.0
Hwang et al. [42]	DSCT	1275 (42)	100	21.0±11.6	3.0±1.4

MDCT: multidetector computed tomography; pts: patients; MB: myocardial bridges; LAD left anterior descending coronary artery; NA : non available; DSCT: dual source computer tomography.

Table 1. Cardiac computed tomography papers where myocardial bridges were evaluated

Arterial segments located in a deep gorge but covered only by a thin layer of muscle or fibrous-fatty tissue were also considered by some Authors as MB because they also may be compressed during systole by the surrounding muscle [5]. According to other Authors the presence of myocardial bridging was defined as myocardium completely encompassing a section of coronary artery in at least one transverse image [32]. For Wirianta et al. MB was defined when at least half of the coronary artery was imbedded within the myocardium with a normal epicardial course of the proximal and distal portion [33].

The prevalence of MB according to CCT studies increased progressively with the introduction of more modern scanners approaching values found in autopsy studies, which should be considered the ultimate gold standard method, rather than the results obtained in the ICA studies. This wide variation may be related to different reasons: differences between temporal and spatial resolution parameters of the scanners; different post processing techniques; different inclusion or exclusion of borderline cases; retrospective observation of arteries with the specific purpose to analyze MB; different population selection (i.e presence of symptomatic or asymptomatic patients, patients with hypertrophic cardiomyopathy); probably also to ethnicity (Tab 1).

8. Conclusion

Myocardial bridges are normal variants of intrinsic coronary arterial anatomy with an intramural course that till 20 years ago were visualized during necropsies, surgery or conventional coronary angiography. Invasive coronary angiography alone or with the use of important tools such as intravascular coronary ultrasound, intracoronary Doppler-ultrasound and intracoronary pressure-wire, is still considered the gold standard technique to study in vivo MBs. The introduction in the cardiac arena of CCT, that with very good accuracy investigates coronary arteries, gave us a complementary and sometimes an alternative test to ICA and more interestingly provides this information non-invasively. In particular settings such as that of a coronary artery with MB, CCT seems to be even superior to ICA and to have results similar to autopsy which is the real gold standard technique to evaluate MBs. However to better understand the real usefulness of CCT in this particular field, further multi-centric interdisciplinary studies must be performed, to link the morphological with the clinical information especially in those patients who have MB and normal coronary arteries or coronary arteries with no culprit atherosclerotic lesions, but who may be at risk for cardiovascular morbidity or mortality.

Acknowledgements

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