Myocardial bridging: what have we learned in the past and will new diagnostic modalities provide new insights?

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Abstract The clinical significance of myocardial bridging has been a subject of discussion and controversy since the introduction of coronary arteriography (CAG) in the early 1960s. More recently computed tomography coronary angiography (CTCA) has made it possible to visualise the overlying muscular bands and appears to have a higher sensitivity for detecting myocardial bridging than CAG. Combining CTCA with invasive techniques such as CAG should make it possible to improve our understanding of the pathophysiology of myocardial bridging and to provide answers to hitherto unresolved questions. This paper critically reviews the outcomes of previous studies and defines remaining questions that should be answered to optimise the management of the presumably fast growing number of patients in whom a diagnosis of myocardial bridging has been made.

Keywords Myocardial bridging · Computed tomography angiography · Coronary arteriography

According to the original definitions the term ‘myocardial bridging’ is used for an anatomic variation in which a band of cardiac muscle overlies a segment of an epicardial coronary artery while the artery involved is referred to as being ‘tunnelled’. Initially the diagnosis could only be made at autopsy but soon after coronary arteriography (CAG) had become an established clinical diagnostic tool systolic compression of a coronary artery, usually the left anterior descending artery

(LAD), was occasionally observed and correctly interpreted as evidence of myocardial bridging [1, 2]. Since then the clinical significance of myocardial bridging has been a subject of discussion and controversy. In the last decade it was shown that myocardial bridging can be very well depicted by computed tomography coronary angiography (CTCA) [3, 4] which has rekindled the interest in this anomaly. We expect that in the near future increasing numbers of patients with myocardial bridging will be detected by CTCA which may significantly enhance our understanding of the pathology and clinical consequences. At the same time, to ensure that these patients receive optimal treatment it is imperative that we stay aware of what previous studies have taught us. It is the purpose of this paper to critically review the outcomes of previous studies and to define remaining questions of clinical relevance that hopefully can be resolved with newer diagnostic modalities.

Diagnosis

Although myocardial bridging has been held responsible for a variety of symptoms in individual cases, ranging from atypical chest pain to sudden death, thus far clinical studies have been unable to identify specific diagnostic features that allow a diagnosis of myocardial bridging without visualisation of the coronary arteries [5–11]. The diagnosis is usually established by chance in patients who are examined by CAG or CTCA for various reasons but not because they are suspected of having myocardial bridging. It should be noted that there are essential differences between the diagnostic information provided by each of the two visualisation methods. CAG does not show the muscular bands overlying the artery but demonstrates primarily the effect on the artery, that is: systolic compression of the artery with narrowing of the lumen and diastolic relaxation, also called a ‘milking effect’. Provided the recording speed is not too low (preferably ≥ 25 frames.s⁻¹) the CAG allows an accurate assessment of the

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arterial lumen dimensions throughout the cardiac cycle. The CAG may also show a sudden deviation towards the septum and backwards in the course of the left anterior descending artery (LAD), which is suggestive of a partial intraseptal course and has been called a ‘step down-step up’ phenomenon [12]. In contrast, CTCA clearly demonstrates the intramural course and the overlying muscular bands but provides no direct information about the dynamic effect on the vessel involved. Attempts have been made to estimate the degree of systolic narrowing by reconstruction of images at different phases of the cardiac cycle [7, 13]. Currently, to minimise the radiation exposure during CTCA acquisition, prospective gating techniques are used where only the diastolic phase is imaged. To capture both the diastolic and the systolic phase, the entire heart cycle must be imaged which would result in a substantial increase in radiation dose. In addition CTCA has lower temporal resolution than CAG, and often requires administration of a β -blocker to reduce the heart rate which may reduce or abolish the arterial compression.

CTCA depicts the beginning and end of the tunnelled segment and the length and depth of the bridging [5, 13–16] which is often best demonstrated by curved multiplanar reconstruction (Fig. 1). This has led to a classification of myocardial bridging as either complete or incomplete. In complete myocardial bridging the artery is completely covered with myocardium whereas in incomplete myocardial bridging the artery is covered by a thin layer of connective tissue and fatty tissue [13, 17]. A similar division was used by Kim et al. who classified bridging of the left anterior descending artery (LAD) as either partial encasement defined as the LAD being within the interventricular gorge and in direct contact with left ventricular myocardium or full encasement defined as the LAD being surrounded by the myocardium and with (measurable (>0.7 mm) or not measurable) overlying muscle [16] (Fig. 1). Another often used division relates to the depth of the artery in the muscular wall, usually the interventricular septum. Bridging is categorised as deep or superficial using cut-off values of 1 mm [14] or 2 mm [6, 17] for the thickness of the overlying myocardium. In addition superficial bridging may be subdivided into cases with full or partial encasement [14]. From the functional standpoint these classifications may be useful but one may argue that, according to the original anatomical descriptions, incomplete bridging or partial encasement may not satisfy the criteria for myocardial bridging. Nonetheless, these CTCA classifications have merit and are currently being used in many centres.

Prevalence

The prevalence of myocardial bridging varies considerably among *in vivo* studies irrespective of the diagnostic method used while a large variation also exists among autopsy studies.

An overview of the prevalence in CTCA studies was recently published by Wirianta et al. in this Journal [11] showing that in most studies the prevalence lies between 15 % and 50 %. Overall the prevalence in CTCA studies is in the same order as in autopsy studies [18, 19].

In CAG studies the prevalence is mostly between 0.5 % and 5 % [18]. An exception is a study in a Chinese population that reported a prevalence of 16.1 %, which was attributed by the investigators to the routine use of intracoronary nitroglycerine that could have elicited or enhanced evidence of myocardial bridging [20].

The lower prevalence of myocardial bridging in CAG studies compared with CTCA studies [19] may in part be explained by the differences between the diagnostic methods, particularly the fact that bridging as demonstrated by CTCA may not always cause systolic compression of the artery involved and therefore may escape detection by CAG. This hypothesis is corroborated by findings at autopsy and a few small clinical studies comparing the results of CTCA and CAG in patients who had undergone both examinations. At autopsy Ferreira et al. found that deep bridges may twist the artery and thus reduce its lumen and compromise flow whereas this characteristic is not present in superficial bridges [21]. Kim et al. reported the results of CAG in 174 patients in whom myocardial bridging of the LAD had been demonstrated by CTCA [16]. The CAG showed systolic compression of the LAD in 97.5 % of the cases with full encasement but in only 2.5 % of the cases with partial encasement. Likewise, Leschka et al. found that only 12 of 26 patients with myocardial bridging detected by CTCA also had evidence of myocardial bridging by CAG [12]. Furthermore, mild degrees of myocardial bridging may not always be recognised by routine interpretation of CAGs [22]. In this context it is significant that Kramer et al. found myocardial bridging of the LAD in an unusually high proportion (12 %) of patients when they carefully reviewed 658 ‘normal’ CAGs specifically for this anomaly [23].

In CTCA as well as in CAG studies the LAD is the artery most frequently involved. Bridging of other arteries or side branches is rarely diagnosed *in vivo* and if found it is usually in conjunction with myocardial bridging of the LAD (Fig. 1). Bridging of other arteries is more often observed at autopsy. Loukas et al. found myocardial bridging in 69 of 200 autopsy specimens (34.5 %) with a total of 81 bridges but in only 35 cases the LAD was involved [24]. Similar findings had been reported earlier by Poláček and Králové who in addition described muscular loops formed by atrial myocardium which attached the artery involved to atrial myocardium [25].

Hypertrophic cardiomyopathy (HCM) is relatively frequently associated with myocardial bridging. The angiographic prevalence of myocardial bridging in children and adults with HCM is in the order of 15 % to 40 % [26–28].

Fig. 1 This patient has two myocardial bridges, namely of the LAD and a large first diagonal branch (DB), as is demonstrated in the 3-D reconstruction in panel **a**. Panel **b** shows a multiplanar reconstruction of the LAD which demonstrates that a portion of the LAD is completely surrounded by left ventricular myocardium. Lines **c**, **d**, and **e** indicate the levels of the axial views that are shown in panels **c**, **d** and **e**, respectively. The axial views in panels **c** and **d** confirm the

myocardial bridging of the LAD (marked by *arrows*). This type of bridging has been termed ‘complete myocardial bridging’ [13, 17] or ‘full encasement’ [16]. Panel **e** shows the free epicardial course of the distal LAD. Axial views also confirm the myocardial bridging of the diagonal branch. Of note, the typical ‘step-up’ phenomenon (*red arrow* in panel **b**) that was originally described for CAG recordings is often more conspicuous in multiplanar reconstructions

An association of myocardial bridging with a left dominant coronary system has also been described [24] but is not as strong as the association with HCM.

Pathophysiology

When myocardial bridgings were first identified by CAG most investigators thought that this anomaly could have no clinical consequences because the compression of the artery only occurs during systole when the coronary perfusion is minimal. A study by Kramer et al. supported this concept; the investigators measured the maximal severity of the systolic narrowing and determined by frame count the duration of significant narrowing in relation to the cardiac cycle and concluded that significant narrowing extending beyond the systolic period did not occur in any of the cases [23]. However, at about the same time, Bourassa et al. reported their findings in 88 patients with myocardial bridging (out of 8501 patients undergoing CAG) and demonstrated that the compression may extend into early diastole, especially at high heart rates induced by atrial pacing [29]. In some cases

they also found evidence of ischaemia assessed by lactate metabolism but many patients had concomitant obstructive coronary atherosclerosis which makes it impossible to ascertain the contribution of the bridges to myocardial ischaemia.

Interesting observations, obtained by coronary intravascular ultrasound (IVUS) and Doppler studies in 62 patients with myocardial bridging at CAG, were reported by Ge et al. [30, 31]. In all patients IVUS demonstrated a systolic eccentric or concentric compression with delayed relaxation in diastole and a specific echolucent half-moon phenomenon over the tunnelled segment that exists throughout the cardiac cycle. Coronary flow velocity recordings in the tunnelled segment showed a characteristic pattern with a prominent peak in early diastole followed by a plateau at mid to late diastole whereas the flow velocity pattern proximal to the bridge was normal (smaller early diastolic peak with gradual descent) apart from some retrograde flow during systole after administration of nitroglycerin. The high peak-plateau pattern that has been called a ‘finger-tip pattern’ (Fig. 2) indicates that the tunnelled segment is still narrowed in early diastole as was proven by Schwartz et al.

Fig. 2 Diagram of flow velocity in the tunnelled segment demonstrating a ‘finger-tip’ pattern. The high flow velocity in early diastole (the finger) indicates that at that moment in the cardiac cycle the tunnelled segment is still narrowed [30, 32]. The coronary flow velocity is minimal during systole

who compared flow patterns with serial changes of lumen diameter assessed by quantitative CAG [32]. The findings of Ge et al. may, like the results of most other studies, be influenced by coexisting obstructive atherosclerotic lesions. In addition, when the lumen dimensions change during the cardiac cycle, flow velocity patterns cannot directly be translated into changes in flow volume; therefore the finger-tip pattern may indicate that the tunnelled segment is still narrowed in early diastole but it does not prove that the flow volume is significantly diminished. Nonetheless, the conclusion seems warranted that in some cases myocardial bridging impedes diastolic coronary flow and may cause or aggravate myocardial ischaemia. Probably a better assessment of ischaemia resulting from myocardial bridging may be obtained by other diagnostic modalities such as isotope stress testing but these methods may yield inconclusive results if bridging is associated with obstructive CAD.

Remarkably, comparing CAG and CTCA, Kim et al. observed that in all cases the length of the dynamic compression was longer than the tunnelled segment as demonstrated by CTCA [16], a phenomenon that merits further study.

Several studies using CAG, CTCA and IVUS have demonstrated that myocardial bridging of the LAD is often associated with atherosclerotic lesions proximal to the bridge whereas the tunnelled segment and the distal portion are conspicuously free from atherosclerosis [9, 15, 31, 33, 34] (Fig. 3). Factors that may play a role in causing the atherosclerotic lesions include flow disturbances (such as retrograde flow during systole), pressure disturbances that may cause endothelial injury, high wall stress proximal to the bridge and tensile stress; conversely development of atherosclerosis may be prevented in the tunnelled and distal segments by inhibition of coronary artery wall motion of the tunnelled segment and lower pressures [5, 19, 31, 34, 35].

Evaluation of patients

In many studies patients with myocardial bridging as sole cardiac and coronary abnormality (isolated myocardial bridging) are not clearly distinguished from patients with concomitant coronary artery disease or other cardiac abnormalities such as valvular disease, yet the two groups may require a different approach.

Patients with isolated myocardial bridging

Usually these patients have undergone CTCA or CAG for evaluation of chest pain or other symptoms that suggest the possibility of coronary artery disease (CAD) and the question has to be answered if the myocardial bridge is just a coincidental finding or if it may explain the patient's symptoms and could be a risk factor for future cardiac events. This often requires a detailed analysis of the bridging. Given the supplementary character of the information obtainable by CAG and CTCA respectively, combining these diagnostic modalities appears to be the most promising diagnostic approach. Combining CTCA with invasive investigations entails an extra burden for the patients; however, the clinical consequences appear to justify this approach in selected cases. CAG should include quantitative analysis of lumen dimensions throughout the cardiac cycle, especially during early diastole, and may be extended with intracoronary IVUS and Doppler flow measurements, and assessment of coronary flow reserve (CFR) or fractional flow reserve (FFR) although a decrease of CFR may have various causes, including microvascular disease. In addition several tests, including intracoronary nitroglycerin [20, 31] and intravenous dobutamine [36], have been used to elicit or aggravate systolic compression of the artery involved. Hazenberg et al. described the results of multiple invasive tests in a series of 12 patients with myocardial bridging and demonstrated that extensive testing is feasible and provides more insight into the mechanisms underlying myocardial bridging [36] but the clinical consequences in individual patients have yet to be determined. When analysing the CTCA results particular attention should be given to the length and depth of the bridge, the overlying tissue, and the proximity to right and left ventricular myocardium [15] (Figs. 1 and 4). In some cases isotope stress testing may be indicated.

Patients with concomitant coronary artery disease (CAD) or other cardiac disorders

In the presence of obstructive CAD or microvascular disease additional investigations such as determination of FFR or stress testing may reflect the flow-limiting effect of these conditions rather than the effect of myocardial bridging. Furthermore, if an obstructive lesion is present in the proximal

Fig. 3 CAG of myocardial bridging of the LAD in diastole and systole in right anterior oblique projection. During systole the segment between the *white arrows* is almost occluded. This angiogram also shows a fixed atherosclerotic lesion in the proximal LAD (proximal to the first

septal branch, marked by a *black arrow*) whereas the distal LAD looks normal, which is quite typical for this anomaly. In addition, slight atherosclerotic changes are present in the distal circumflex branch. (Figure adapted from reference [47], page 44, with permission)

LAD this may cause a pressure drop in the distal LAD and thus aggravate the systolic lumen reduction caused by the bridge. In these cases it may only be possible to estimate the influence of the bridging on the basis of the length, depth, and proximity to left ventricular myocardium of the tunnelling as can be assessed by CTCA [15].

Prognosis

Only a few studies on the prognostic implications of myocardial bridging are available. Kramer et al. found that patients with isolated myocardial bridging during a 5-year follow-up period had the same survival as angiographically similar patients without bridging while none of the survivors sustained an acute myocardial infarction [23]. Juillière et al. found in an 11-year follow-up study of patients with isolated myocardial

bridging that there were no cardiac deaths or acute myocardial infarctions [37]. More recently similar results were reported by Çiçek et al. who observed no major cardiac events or need for revascularisation in a 4-year follow-up of 118 patients with isolated myocardial bridging [38]. The outcomes of these studies are reassuring but do not prove that myocardial bridging is an innocent anomaly in all cases. First, the number of patients included in the follow-up studies is relatively small and the follow-up periods are limited. In the second place no data are available about the additional risk of myocardial bridging in the presence of obstructive CAD, such as the relatively common atherosclerotic lesions proximal to bridging of the LAD. In the third place myocardial bridging has been implicated as a potential cause of sudden death. Desseigne et al. analysed 19 cases with myocardial bridging out of a series of 930 medicolegal autopsy studies [39]. In 11 cases additional potentially lethal conditions and in 7 cases minor associated

Fig. 4 Bridging of the middle portion of the LAD seen in 3-D (panel **a**) and curved multiplanar reconstruction (panel **b**). The trabeculated myocardium indicates that the LAD runs on the right ventricular side of the interventricular septum. This variant probably has less haemodynamic consequences than bridging by left ventricular myocardium, as depicted in Fig. 1, because the compression pressure generated by right ventricular contractions is relatively low

cardiac abnormalities were found. The authors conclude that it cannot be excluded that myocardial bridging had been responsible for sudden death in some cases. At autopsy, Morales et al. found myocardial lesions that were indicative of ischaemia in 22 of 39 hearts with myocardial bridging; all of these had a deep intramural LAD and 13 of these persons had died suddenly [40]. Eckart et al. reviewed 126 cases of non-traumatic sudden deaths in military recruits and found in 2 cases myocardial bridging but no other cardiac abnormalities [41]. Corrado et al. found isolated myocardial bridging in 2 of 49 athletes who had died suddenly [42]. Maron et al. reported that a tunnelled LAD had been the cause of death in 3 % of competitive young athletes who had died suddenly [43]. These data suggest that myocardial bridging may be a cause of sudden death but given the very low incidence of sudden unexpected death in young persons, including athletes [44], and the small proportion of these cases in which myocardial bridging may have been responsible versus the high prevalence of myocardial bridging in CTCA and autopsy studies the conclusion is justified that in patients with myocardial bridging the probability of sudden death is extremely low.

In view of the association between HCM and myocardial bridging it has been suggested that in patients with HCM the presence of bridging may be an additional risk factor for sudden death or life-threatening arrhythmias. This hypothesis was corroborated by a study of Yetman et al. who saw angiographic evidence of myocardial bridging in 10 of 36 children with HCM and found that, compared with patients without bridging, the patients with bridging had more abnormalities at exercise testing, a greater incidence of chest pain, cardiac arrest, and ventricular tachycardia [45]. However, there was a highly significant difference between the two groups concerning the age at which the diagnosis of HCM was made which is a serious limitation of the study. A convincing study was published by Mohiddin et al. [27] who found bridging in 23 of 57 children with HCM. The bridging involved the LAD in 57 % of the affected vessels and compression of septal LAD branches (also known as ‘septal squeeze’ [46]) was present in 65 % of the children with bridging. The authors conclude that bridging in children with HCM is related to the severity of HCM and probably does not result in myocardial ischaemia and does not cause arrhythmias or sudden death. Similar results in adults were reported by Soraja et al. who found myocardial bridging in 15 % of 425 adult patients with HCM and observed no increased risk of death, including sudden death, in the patients with myocardial bridging [28]. These studies indicate that myocardial bridging is not a significant risk factor in patients with HCM.

It may be important that in the prognostic studies that are referred to in this paragraph the diagnosis of myocardial bridging was made by CAG. Thus far no similar prognostic studies using CTCA to diagnose myocardial bridging are

available; however, since particularly the lighter (superficial) forms of myocardial bridging detected by CTCA may be missed with CAG [3, 16] we suspect that overall the prognosis of patients with myocardial bridging detected by CTCA is at least as favourable as reported in CAG studies.

Therapy

In view of the overall benign character of isolated myocardial bridging, therapeutic intervention in asymptomatic patients is currently not warranted. Patients with symptoms that may be attributable to myocardial bridging may require therapy, particularly if there is objective evidence of myocardial ischaemia.

Current therapeutic options have been reviewed by several investigators [5, 10, 19] and were recently discussed by us [47]. Should therapy be indicated then medical management is the first choice. This should primarily include beta-blockers because beta-blockers cause reduction of the compression by the muscular band and slowing of the heart rate with prolongation of the diastolic period. If beta-blockers are not tolerated, calcium channel blockers, particularly calcium channel blockers with negative chronotropic effect, may be an alternative. There is some controversy over the administration of nitrates since these may relieve symptoms but may also increase compression of the tunnelled segment and are therefore considered contraindicated by some investigators.

In view of the frequently found atherosclerotic changes proximal to the tunnelled segment, administration of anti-atherosclerotic drugs such as anti-platelet drugs and statins may be considered as a preventive measure.

If the results of medical management are insufficient, stent implantation or surgery are therapeutic options. Theoretically one may wonder whether stent implantation is a valid option because it is conceivable that the pressure from outside by the contracting myocardial bridges may cause compression of the vessel wall against the stent and thus cause vessel wall damage and subsequently in-stent stenosis or stent fracture. These potential problems were already indicated by Stables et al. who were the first to publish a case report of stent implantation [48]. In spite of these concerns acceptable results of stent implantations have been reported [49]. However, in 2008 Kunamneni et al. reported their experiences in a series of 12 patients with a mean follow-up of 15 months and found that the patients with stents experienced more adverse events than patients who received solely medical therapy [50]. The investigators concluded that coronary stent placement for medically refractory symptomatic bridge should be avoided. Furthermore, in 2011 four cases of stent fracture were reported by Srinivasan et al. [51].

Surgical treatment of myocardial bridges is another option. Left internal mammary artery (LIMA) anastomosis to the

LAD has been advocated; however, this can only be expected to be successful if the myocardial bridges are located solely in the proximal LAD. Recently satisfactory results of bypass surgery using the LIMA were reported by Sun et al. in 13 patients with isolated myocardial bridging [52].

Good results of myotomy were already reported more than 30 years ago [53] but, although surgical techniques have improved since then, myotomy may still present problems if the bridge is long and deep [52].

It should be emphasised that the interventions described above in principle relate to patients with symptomatic myocardial bridging without coronary or valvular disease. If a patient requires coronary or cardiac surgery for other reasons it merits consideration to also perform myectomy of long or deep bridges. The presence of bridging may also tip the balance in favour of surgery if surgery as well as percutaneous intervention is a realistic option.

Conclusions

There can be little doubt that in the majority of cases isolated myocardial bridging is a benign anomaly that, however, may cause symptoms and in some cases may be a risk factor for sudden death, albeit that the probability of sudden death is extremely low. The challenges we are facing are to determine in symptomatic patients if the symptoms are caused by myocardial bridging and to identify patients who are at increased risk for complications. We expect that an optimal use of new non-invasive diagnostic modalities in combination with invasive methods will fundamentally enhance our understanding of this intriguing anomaly and will eventually resolve remaining questions. However, if we do not use new techniques intelligently or disregard lessons learned in the past then myocardial bridging may well remain subject of debate and controversy for another half century. For the time being we must be cautious not to do more harm than good by aggressively treating patients who may require no treatment.

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