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MYOCARDIAL BRIDGING PHENOMENON AND MYOCARDIAL ISCHEMIA

POJAVA MIOKARDNOG PREMOŠĆAVANJA I SRČANA ISHEMIJA

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Apstrakt

Miokardno premošćavanje je urođeno stanje u kome segment koronarne arterije i/ili njene glavne grane jednim delom prolazi intramiokardno pre svojeg intramiokardnog završetka. Incidenca miokardijalnih mostova na autopsijskom materijalu kreće se od 4.7% do 86%, dok se pomoću koronarne angiografije otkrije između 0.5% i 40%. Moguće je da su ove razlike posledica drugačije metodologije ispitivanja i/ili veličine grupa ispitanika. Pojavu miokardnog premošćavanja kliničari obično smatraju kao ograničenu na jedan krvni sud, najčešće levu prednju descendentnu arteriju, koja je asimptomatska. Međutim, njihovo prisustvo bilo je pokazano kod pacijenata sa atipičnim bolom u grudima ili sa miokardnom ishemijom, infarktom miokarda, poremećajem sprovodnog sistema srca ili iznenadnom srčanom smrti. Iako brojne, studije o miokardnom premošćavanju još nisu objasnile tačan mehanizam kojim miokardni mostovi ponekad postaju simptomatski. Kod klinički simptomatskih pacijenata pokušane su mnoge terapijaske mogućnosti, obično lekovima i ređe hirurškim tretmanom. U novije vreme prikazano je da koronarni stent služi kao efektivna interventna mogućnost koja popravlja simptome kod određenih pacijenata refrakternih na uobičajenu medikamentoznu terapiju. Hirurški tretman sa disekcijom miokardnog mosta (myotomia) ili sa minimalnim invazivnim koronarnim arterijskim premošćavanjem (bypass) mora biti ograničen na pacijente sa teškim simptomima koji perzistiraju uprkos terapiji lekovima. Međutim, najbolji tretman miokardnih mostova ostaje i dalje kontroverzan.

Ključne reči/Key words

Miokardno premošćavanje, miokardna ishemija, infarkt miokarda, poremećaj sprovodnog sistema srca, iznenadna srčana smrt.

Myocardial bridging, myocardial ischemia, myocardial infarction, impairment of the conductive system of the heart, sudden cardiac death.

INTRODUCTION

Myocardial bridging (MBing) is a congenital condition in which a segment of a coronary artery (CA) or/and some of its major branches has a temporary intramyocardial course before its intramyocardial termination. It has been often considered by clinicians as ”usually confined to a single vessel, typically left anterior descending artery” and being asymptomatic. On the other hand, their presence has been reported in patients experiencing atypical chest pain or suffering from myocardial ischemia, myocardial infarction, impairment of the conductive system of the heart, and sudden cardiac death (1, 2, 3, 4, 5, 6, 7, 8).

Myocardial Bridges - Early Descriptions and Terminology.

The phenomenon of MBing was briefly mentioned by Reyman (9) and much later by Tandler (10). The overbridged vessels were described as ”submersing” by Tandler (10), ”mural” by Geiringer (11), ”intramural” by Edwards et al. (12), ”intraparietal” by Morales et al (3), and recently ”tunneled” by Bonvini et al (13). Other authors focused on the muscular fibers crossing over those segments of the coronary vessels and described them as myocardial bridges (MBs) (14, 15, 16). Furthermore, some distinguish ”myocardial bridges” from ”myocardial loops” based on the myocardial fibers location, and their thickness; They, also, described the presence of myocardial bridges over superficial cardiac veins (15, 17).

Myocardial Bridges - The Privilege of the Human Heart?

Due to the fact that the coronary artery pattern is not unique in high primates, being predominantly subepicardial in man, gorilla and gibbon, it was not a surprise when we found MBs in *Cercopithecus and Macaca* (18, 19, 20). Furthermore, myocardial bridges were described in dogs (21, 22, 17, 23), in pigs (24, 25), sheep and goat (14, 17), as well as, in camel (26) and other mammals.

Myocardial Bridges - Incidence

Myocardial bridges gained the interest of scientific community once their role in genesis of atherosclerosis was suggested (11). Since then, the wide range of incidence obtained on autopsy has been reported - from 4.7% (27) and 5.4% (12), over 34% (28), 39% (29), 36-48% (30), 50% (31), 55% (32), 58% (33), up to 78% (34), and even 86% (17). From our experience, we suggest the reported differences are due to the sample size and study design (prospective studies versus retrospective ones). Thus, two independent studies conducted by the same person, but differently designed reported different incidence of myocardial bridges in the same population (29, 35). While in the prospective studies, dissection has been performed with special attention on myocardial bridging phenomenon, in the retrospective one autopsies were routine, and detected myocardial bridges were only collateral findings. It is easy to guess that they were detected only over the LAD.

Consequently, the first description of transitory squeezing of a coronary artery branches, i.e. LAD, on coronary angiography producing the so called ”milking effect” was done by Portsmann and Iwig’s in 1960 (36). That systolic compression was followed by complete or partial release in diastole. Later on, Amplatz and Anderson (37) stressed out the differences between milking effect caused by myocardial bridge and fixed atherosclerotic lesion. The prevalence of MB angiographically reported by numerous studies varies between 0.5% and 40%. (38, 39, 40, 41, 42, 43, 44, 45, 46).

The phenomenon has been most commonly detected in the middle segment of the LAD (47), and being over a single vessel. Multiple MBs, affecting both LCA and RCA branches, are a rarely reported finding on coronary angiography (48).

The large variation in reported incidence of MB phenomenon on coronary angiography could be the result of differences in classification and/or methods of evaluation. It is difficult to demonstrate MB by conventional angiography when the systolic compression is relatively small or is partially occluded. In those cases it has been reported that vasodilatating drugs such as nitroglycerin are helpful to augment vessel wall squeezing (39). However, with new techniques as quantitative coronary angiography (QCA) and intravascular ultrasound (IVUS), it has been proved that the intracoronary administration of nitroglycerin improves the sensitivity for the angiographic detection of MBs up to 50%, although the exact mechanism is not quite well understood (49).

Myocardial Bridges - Role in the Genesis of Atherosclerosis.

Some authors considered MB as an anatomical variation (12, 50, 30), while Geiringer (11) even suggested that MB protects the overbridged segment of the coronary vessel from developing atherosclerosis. The latter was due to his light microscopy investigations which showed that *tunica intima* was thinner in the overbridged part of the vessel where its hyperplasia was evident in epicardial ones. In those cases the tunneled portion was rarely affected by atherosclerosis as if the myocardial bridge would protect the wall against the disease. Such finding was first denied by Edwards et al (12) who did not note any difference in the incidence of atherosclerosis in intra and extramural portions of the affected vessel. On the other hand, the absence of atheroma or its low incidence in the overbridged part of the artery was again pointed out in several publications (33, 51, 3, 52, 53, 54, 55). The ”prevention” effect of atherosclerosis in the overbridged segment might result from its better lymph drainage through myocardial compression (56). In any event, the absence of atherosclerosis in the overbridged segment of the vessel has been reported in clinical studies as well (49, 57, 58).

We, like some others, deeply doubt the suggested protective role of MBs in the evolution of atherosclerosis in overbridged segment of the coronary vessel (59, 60, 61, 62). This being fueled by a consistent finding, in all our series, and that is a significantly high incidence of MBs found on autopsy of patients with prior heart condition where the changes were present both in the segment under the MB and proximal to it (Fig. 1) (35, 63). The latter can be explained by increased axial wall stress just proximal to the MB due to the compression on the overbridged segment (64). Focal degenerative changes were present in the neighboring myocardium too. The similar changes were found in *Cercopithecus aethiops and Macaca fascicularis* (*Cynomolgus*) hearts (19, 65).

Furthermore, light microscopy studies identified no structural change in the myocardium overbridging the artery (50, 66). That suggests only mechanical action of MB on the overbridged segment and coronary circulation in the corresponding area.

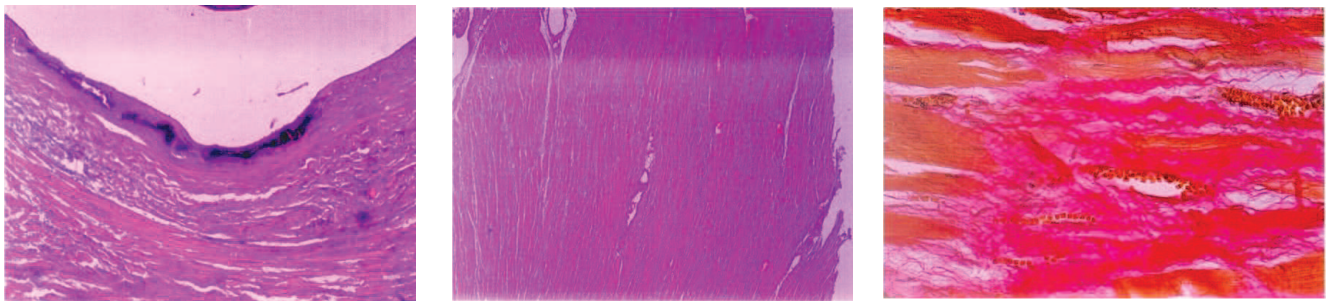

Figure 1. (A) The wall of the left anterior descending coronary artery under the myocardial bridge with calcification, ulceration and hemorrhage within the atheromatose plaque (H.E., 600x), while (B and C) myocardium shows signs of cardiomyofibrosis (H.E., 200x and van Gieson, 800x). Reprinted from Fig. 2 of reference 35 with permission from CIBID - School of Medicine University of Belgrade.

The overbridged segment of the coronary vessel should not be considered apart from its proximal one. Thus, the atherosclerotic plaques just proximal to the overbridged segment were reported by both groups - supporters and opponents of the protective theory against atherosclerosis.

Finally, in 1995 Ge, Erbel et al (54) by IVUS, *in vivo*, clearly visualized an eccentric plaque with calcium deposit in the LAD segment proximal to the bridge. As the pressure in the segment proximal to the myocardial bridging was higher than aortic pressure, it was easy to come to the conclusion that the disturbance of the blood flow and high wall stress proximal to myocardial bridging was a main contributor to the development of atherosclerosis in the segment just proximal to the bridge. The findings were supported by others as well (64).

In any event, one should not consider only the overbridged segment of the coronary vessel, i.e. apart from its proximal and distal segments. Even if we accept that MBs protect the underlying segment of the vessel from atherosclerosis by squeezing, it interferes with normal hemodynamic. Recently, IVUS analysis showed for the first time, that the vessel area in the myocardial bridge segment was significantly smaller than that in the adjacent reference segments proximal and distal to the myocardial bridge throughout the cardiac cycle in vast majority of the myocardial bridges examined (67).

Clinically Symptomatic Myocardial Bridges.

Myocardial bridging has been considered a benign condition for a long period of time. However, since early eighties on many occasions, they have been associated with myocardial ischemia (1, 2, 38, 68, 52, 69). With rapidly advancing technology and possibilities of *in vivo* visualization of MBs, those associations became more frequent and extended to acute coronary syndromes as well (70, 71, 61, 72, 73, 58). Furthermore, the disturbances of the conductive system of the heart as a consequence of myocardial ischemia produced by MBs has been described as well. Thus, patients with MB may experience cardiac arrhythmias (4, 59, 74). Finally, the first involvement of MBs with sudden death was suggested in eighties by Morales et al (3). They described the cases of sudden death of three healthy individuals during strenuous exercise. The death was attributed to muscular bridges over the proximal left anterior descending artery and focal scarring of the interventricular septum. That was the beginning of case reports on presence of MBs in young persons who experienced sudden death during exercise. In all those cases, autopsy would reveal only the presence of MB on otherwise normal coronary arteries. Therefore, the MBs would be accused for the developing ischemia but the exact mechanism of the ischemia has not been known yet (60, 75, 76, 77).

Reviewing the literature it appears more likely that myocardial bridges over the coronary arteries are as frequent as their absence (78). Although mostly clinically silent, under certain circumstances, like stress and exertions effort, may provoke chest discomfort, or atypical chest pain accompanied with sweating and weakness, or arrhythmia, in other words - become clinically loud. These patients' physical exams can be unremarkable, and have normal chest radiographs. Depending on the extent of ischemia, the electrocardiographs can be unremarkable as well or show both T-wave inversion or ST elevations in the corresponding leads. Blood tests can reveal elevated serum levels of troponin and creatine kinase-MB isoenzyme. In cases with normal resting ECG, stress testing can induce nonspecific signs of ischemia, or disturbances of the conductive system of the heart.

Conventional coronary angiography (36) is still the gold standard imaging technique but it has its limitations. Atherosclerotic lesions proximal, or rarely, within the area of bridging which remained undetected by conventional coronary angiography, became detectable by intracoronary ultrasound (IVUS), as well as small size or thin MBs (45, 79). Furthermore, nowadays we are positive that MBs cause not only systolic compression of the tunneled segment of the coronary artery but also accelerated flow velocity at early diastole (finger-tip phenomenon), no or reduced systolic antegrade flow with decreased diastolic/systolic velocity ratio, and a retrograde flow in the proximal segment, which is provoked and enhanced by nitroglycerin injection (48). These functional data help a lot in understanding the development of ischemia in certain MBs holders. Finally, there is a promising non-invasive alternative for the diagnosis of MBs - coronary computed tomography angiography (CCTA) using multidetector computed tomography (MDCT) scanner. It enormously improved the visualization of MBs and thus their *in vivo* diagnosed incidence increased from 0.5% up to 26%. In addition, it provides data on MB's length, thickness and direction of fibers (6, 58, 13).

For clinically symptomatic patients, many therapeutic approaches have been attempted, but the optimal treatment of MB still remains controversial (47, 80, 67). Management of MBs is usually medical and rarely surgical. Available medications include b-blockers and calcium channel blockers. The inotropic negative properties of these drugs might explain the decreased bridge-induced systolic coronary compression (58).

Recently, coronary stenting was reported to serve as an effective interventional approach to improve symptoms in selected patients refractory to standard medical therapy (81, 82, 83, 84, 85). Unfortunately, a high risk of coronary perforation during the angioplasty procedure was reported (86, 84). In addition, it failed to relieve sever angina in others and was associated with high clinical restenosis (85, 87, 7, 8), raising the question should it be considered as a method for revascularization in symptomatic patients.

Surgical treatment by dissection of the overlying myocardium (myotomy) or with minimally invasive coronary artery bypass grafts should be limited to patients with severe symptoms (angina, recurrent myocardial infarction) that persist despite medical treatment. Also, it should be considered in symptomatic patients with myocardial bridging and an atherosclerotic lesion within the bridged segment (88, 58).

In conclusion we can say that in spite of the fact that numerous studies, both from basic medical fields and clinical, as well as clinical case reports enriched our knowledge on myocardial bridging phenomenon, they have not yet explained the exact mechanism by which myocardial bridges, occasionally, become symptomatic. Although the best therapeutic approach will be eventually found in the future, the collected data undoubtedly stressed out the importance of myocardial bridging phenomenon in developing ischemia particularly in people with otherwise normal coronary arteries bearing a low risk for coronary atherosclerosis.

Abstract

Myocardial bridging (MBing) is a congenital condition in which a segment of a coronary artery or/and some of its major branches has a temporary intramyocardial course before its intramyocardial termination. The wide range of myocardial bridge (MB) incidence obtained on autopsy has been reported - from 4.7% up to 86%, while in coronary angiography reports vary between 0.5% and 40%. Perhaps, the reported differences are due to the different study design or/and sample size. Myocardial bridging phenomenon has been often considered by clinicians as usually confined to a single vessel, typically left anterior descending artery, and being asymptomatic. On the other hand, their presence has been reported in patients experiencing atypical chest pain or suffering from myocardial ischemia, myocardial infarction, impairment of the conductive system of the heart, and sudden cardiac death. Although numerous, studies on MBing phenomenon have not yet explained the exact mechanism by which myocardial bridges, occasionally, become symptomatic. For clinically symptomatic patients, many therapeutic approaches have been attempted. Management of MBs is usually medical and rarely surgical. Recently, coronary stenting was reported to serve as an effective interventional approach to improve symptoms in selected patients refractory to standard medical therapy. Surgical treatment by dissection of the overlying myocardium (myotomy) or with minimally invasive coronary artery bypass grafts should be limited to patients with severe symptoms that persist despite medical treatment. However, the optimal treatment of MB still remains controversial.

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