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Case Report

MYOCARDIAL BRIDGE STENTING COMPLICATED BY CORONARY ARTERY PERFORATION AND MIDLAD-RIGHT VENTRICLE FISTULA FORMATION IN NSTEMI PATIENT

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ABSTRACT – We report a case of a patient who presented with non ST-elevation myocardial infarction and coronary angiography showing a long myocardial bridge (MB) of the left anterior descending artery (LAD) with a fixed significant stenosis at the entry point of the MB. The lesion was treated with a semi-compliant balloon and drug-eluted stent (DES). After the stent placement, a large arterial perforation with fistula to right ventricle was observed. There was no hemodynamic instability due to the fistula progression during the procedure and the perforation point was successfully closed with a stent-graft. Additionally, we provide a short review of diagnostic and therapeutic approach to MB stenting and reports of cases with coronary artery perforation after stenting of MB.

Key words: *coronary intervention, bridging, stent, perforation*

Case report

We present a case of a 62-year-old male patient who presented at our emergency department with acute myocardial infarction without ST segment elevation. Before the index event, the patient suffered from arterial hypertension and hyperlipidemia and was treated with bisoprolol, perindopril/amlodipine, atorvastatin for more than 5 years.

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Initial electrocardiogram was innocent and preprocedural echocardiography showed no wall motion disturbances.

Retrospectively, the patient reported intermittent mild chest pain during the previous year, with a significant progression in pain intensity during the admission day.

Coronary angiography was performed and a 15-mm-long myocardial bridge (MB) of the left anterior descending artery (LAD) was described with a fixed 80% stenosis at the entry point of the MB, which caused a dynamic 90% stenosis during systolic contraction (Figure 1). The remaining coronary arteries were free of significant atherosclerosis. The patient was already taking the optimal medical therapy (beta

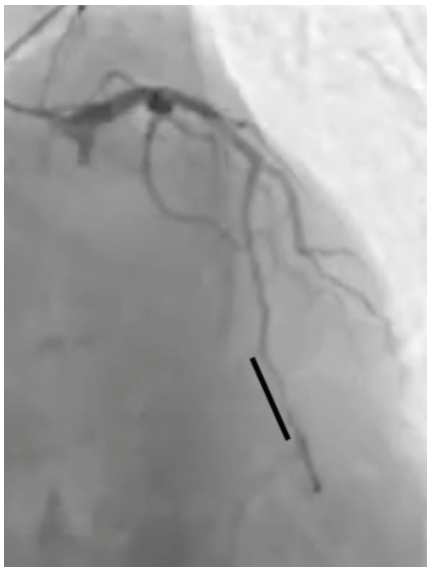

Figure 1. Myocardial bridge in the midLAD segment (black line)

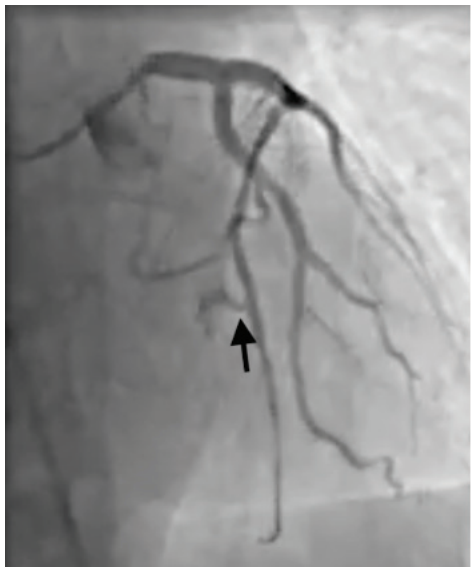

Figure 2. Coronary perforation with LAD-RV fistula formation (arrow)

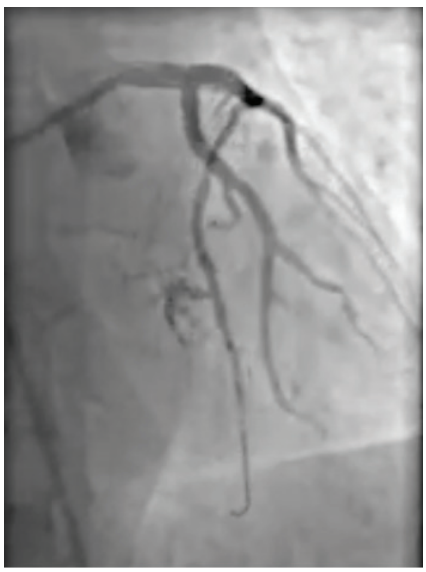

Figure 3. Fistula diameter progression with a massive contrast leak into RV

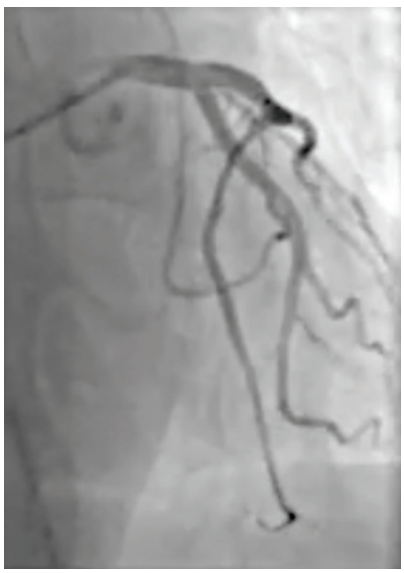

Figure 4. Complete fistula closure after stent-graft implantation

blockers + calcium channel blockers) for a longer period of time and presented with acute infarction so percutaneous intervention was chosen as a treatment option. Predilatation was performed with a semi-compliant balloon (2.5 mm) and a drug-eluted stent (DES) was implanted (Orsiro 2.75/26 mm inflated at 10 atm). Soon after the stent placement, a large arterial perforation with fistula to right ventricle was observed (Figure 2). Due to the left-right shunt, the patient remained asymptomatic but progression of the contrast extravasation during the next few minutes was noted (Figure 3) and due to the inability to rule-out the stent fracture, stent-graft was implanted (Biotronic PK Papyrus 2.5/20 mm) with a

complete resolution of the contrast extravasation and a TIMI 3 flow in the LAD distal to the MB (Figure 4). During the remaining hospital stay, the patient was free of symptoms with a stabile haemoglobin value. Dual antiplatelet therapy was reintroduced the evening after the procedure. The patient was doing well at follow up visits, without any ischemic symptoms or exercise intolerance.

Discussion

Myocardial bridging is defined as a coronary artery that tunnels through the myocardium under the overlying muscle bridge. It is considered the most common congenital coronary anomaly and almost exclusively affects the LAD artery, especially the middle part. It is found in up to 40% of patients when using coronary angiography, but it seems like the real prevalence is much higher, according to autopsy studies, which found MB in almost 90% of the population. There is a high prevalence (14%) in adults with hypertrophic cardiomyopathy. MB is characterized by the systolic myocardial muscle compression of the underlying artery. The length of MB is usually within 10 and 30 mm (1–4).

MB is usually an incidental finding and is generally considered as a benign anomaly, but there is rising evidence that MB can cause symptoms such as exercise intolerance, myocardial ischemia, ventricular and supraventricular arrhythmias, atrioventricular block, syncope and sudden death (5–10).

There are several independent factors that can explain the causal link between MB and ischemic symptoms in patients without significant atherosclerotic coronary disease. Qian et al. showed that the vessel area in the myocardial bridge segment was smaller than the adjacent reference segments proximal and distal to the MB throughout the entire cardiac cycle (5.48 mm^2; 4.52 mm^2, 9.40 mm^2, 7.22 mm^2; vessel area in the MB during diastole, systole and the segments proximal and distal to the MB, respectively). (11) Angiographic and intravascular ultrasound (IVUS) studies showed that the vessel compression is not limited to systole but also persisted during diastole, limiting the coronary perfusion and reducing blood flow (12,13). The bridged part of the vessel usually remains free of atherosclerosis, probably due to high shear stress underneath the tunnelled segment induced by the systolic compression. This phenomenon leads to alignment of endothelial cells in the direction of blood flow, therefore being more resilient to the diffusion of the atherogenic factors (1). However, a recently published IVUS study showed that a majority of patients with MB had an atherosclerotic plaque located proximally to the MB (usually not detectable with „classic“ coronary angiography), which can be associated with the turbulent and complex blood flow due to the systolic flow reversal and the increased intraluminal pressure in the segment proximal to the MB. (14). All the mentioned findings can be exacerbated with left ventricular hypertrophy development (by increasing the compression and reducing the coronary microvascular reserve and aggravating the supply-demand mismatch imposed by the MB), concomitant vasospasm/microvascular dysfunction/endothelial dysfunction (commonly confirmed in patients with MB) and progression of the plaque proximal to the MB (15). In addition, ischemia can be provoked by tachycardia and increased contractility during stress or exercise. (16)

Diagnosis of hemodynamic significance of MB in patients with no obstructive coronary artery disease poses a clinical challenge. Non-invasive techniques are limited. Development of multiple slices computed tomography (MSCT) techniques allowed the use of MSCT for the physiologic coronary flow assessment but has not been used in daily clinical practice yet. Stress single photon emission computed tomography (SPECT) can detect reversible myocardial perfusion defects in patients with myocardial bridging and relate the amount of ischemia to the degree of systolic arterial narrowing. Stress echocardiography can detect a typical finding for MB (focal septal buckling with apical sparing and with no typical regional hypokinesis) and is a promising method but not sufficiently validated (14,17,18).

Coronary angiography remains the most commonly used method for detecting MB. A significant „milking effect“ is present when there is >70% reduction in luminal diameter during systole and persistent >35% luminal reduction during mid-to-late diastole and can be exacerbated my intracoronary application of nitro-glycerine by vasodilating the non-bridged coronary segment (15,19). Our patient had a 90% dynamic stenosis and no other significant atherosclerosis, so it was rational to link the MB as a cause of the myocardial infarction.

When coronary angiography remains unclear, IVUS is shown to be superior in detecting MB. The

typical finding consists of the „half-moon" sign – an echo-lucent area presents only between the bridged artery and the surrounding tissue. IVUS can be used to detect the plaque proximal to the MB (19).

Coronary flow evaluation has been studied and can be used as a valuable tool to assess hemodynamic significance of MB.

MB flow during intracoronary Doppler measurement shows a typical „fingertip" sign, characterized by a rapid early diastolic acceleration and rapid mid-diastolic deceleration, followed by a diastolic plateau velocity. When dobutamine infusion is used during measurement, the Doppler flow velocity increases significantly in all vessel segments (proximal and within the MB) but the greatest increase is noted within the MB. A significant increase of the MB's flow velocity combined with a reproduction of the patient's symptoms would suggest a clinically significant myocardial bridge (14,20).

Fractional flow reserve (FFR) has been studied for detecting a hemodynamically significant MB. A patient with an FFR <0,75 probably has MB-related ischemia. Mean FFR with adenosine was shown to be inconclusive in the mentioned settings because bridge is a dynamic rather than a fixed stenosis. The hemodynamic change generated by MB occurs during diastole (contrary to the fixed stenosis, where the pressure drop occurs in systole and diastole, respectively) and is caused by a delayed luminal diameter recovery due to the arterial compression from the MB (21). So, dobutamine rather than adenosine is most likely to produce the dynamic compression of the MB (due to the chronotropic and inotropic effect of dobutamine) and the diastolic FFR rather than mean FFR is preferred for hemodynamic testing. It has also been noted that the systolic compression of the MB produces an abrupt rise of pressure, which can surpass the aortic pressure, causing a retrograde systolic flow. This phenomenon can affect the mean FFR and mislead the results. Pargaonkar et al. recently showed that MB muscle index (MMI), which can be calculated as MB length x halo („half-moon" sigh in mm), using IVUS is highly predictive of a diastolic FFR ≤ 0,76 (14).

Instantaneous wave-free radio (iFR) has been shown to be superior to the mean FFR in detecting hemodynamically significant MBs but further evaluation is needed (22). Other coronary abnormality, such as endothelial dysfunction, is frequently present in patients with MB. Therefore, a comprehensive assessment during coronary angiography, including dobutamine, acetylcholine and nitroglycerin infusion, IVUS and diastolic FFR, is needed for a full evaluation of the patient with ischemic symptoms and MB but without obstructive coronary disease (23).

Treatment of patients with MB consists of pharmacologic therapy, percutaneous coronary interventions and surgical procedures.

Pharmacologic therapy is a cornerstone for patients with a symptomatic MB. Beta-blockers are the mainstay of therapy. They improve symptoms by reducing heart rate, increasing diastolic coronary pressure and decreasing contractility. Calcium blockers are also used in combination with beta-blockers due to their vasodilatory effect that may be beneficial in patients with concomitant vasospasm. Nitroglycerin is paradoxically contraindicated in patients with MB. It can worsen the symptoms by increasing the systolic compression of the bridged segment and dilating the segments around the bridge, leading to an increase in the retrograde flow in the proximal segment (19,24).

In patients with an objective signs of ischemia and altered MB hemodynamics who remain symptomatic despite medication, one of the invasive procedures may be considered.

The most commonly used surgical techniques are coronary artery bypass grafting (CABG) and myotomy. Surgical myotomy is performed in patients with a superficially located MB. Given the concern of graft failure due to the competitive flow, CABG seems reasonable in patients with a long (>25 mm) and deep (>5 mm) MB.(25).

Percutaneous coronary intervention (PCI) for MB was introduced in 1995 but still remains controversial. In-stent restenosis was reported to be found in 75% of patients treated for symptomatic MB with bare-metal stents and in around 20% when drug-eluting stent were used (26). External compression of the stent combined with an increased shear stress stimulate neointimal proliferation. It is known that need for re-intervention is much higher if the initial stenting included the bridging segment and not just the segment before the MB (27). Stenting in patients with MB should be considered for treating the stenosis proximal to the MB and to reverse the dynamic obstruction within the MB. There are no randomized trials to prove the effectiveness of scaffolding the bridged segment with stents, but real

data suggest that if stent with adequate dimension is used, stenting the MB is successful in achieving an adequate diastolic and systolic flow and sustaining stress over time, but always keeping in mind the higher rate or stent restenosis, thrombosis and fracture when MB is stented. According to available data, if the interventional strategy is preferred, it is rational to carefully evaluate and select the balloon and DES size using IVUS (28). Winter et al. recommended CABG as a treatment strategy following restenosis at the index lesion (29).

Stenting the MB is also associated with a high risk of coronary perforation. Tunnelled segments in myocardial bridging tend to be deficient in smooth muscle density, resulting in higher possibility of disruption during high-pressure balloon inflation (30,31). As mentioned before, the vessel area in the MB is smaller than in the segment proximal and distal to the MB, so selecting the appropriate stent and balloon size in not important only to avoid underexpansion (and further stent thrombosis) but also to avoid overexpansion and help reduce the risk of coronary perforation (11). The superficial and deep types of MB can be found. The deep type is characterized as the LAD deviating toward the right ventricle (RV) and deeply situated on the interventricular septum. In contrast, the superficial type is described when the LAD is running on the interventricular septum. It has been speculated that perforation of the deep type tends more often to result in cavity spilling and the right ventricle fistula formation while perforation of the superficial type may more likely lead to pericardial effusion and tamponade (32). Few case reports have been published regarding coronary perforation after stenting MB. Most of them resulted in pericardial effusion and tamponade, needing pericardiocentesis to be performed (28,33,34). Only few cases reported a RV fistula formation. Following the standard clinical practice for managing perforation in non-bridged arteries, almost all reported cases were successfully resolved using a prolonged balloon infusion (5,34) or stent-graft implantation. Only two cases reported emergency CABG performed after unsuccessful percutaneous treatment. It is interesting that almost all rupture into pericardium were symptomatic with at least some degree of hemodynamic repercussion (32). Contrary rupture into the RV led to a wide spectrum of clinical scenarios. Tomasevic et al. reported a small RV fistula found on a repeated angiography the day after stenting MB, left untreated due to TIMI 3 flow in the main artery and asymptomatic course of the patient. On a planned repeated angiography 8 days later, the fistula showed progression and was treated with a cover stent. During the entire hospital stay the patient had no symptoms of cardiac congestion or ischemia. (35) Becher et. al reported a case of a massive rupture of LAD into the RV, successfully treated with a cover stent but due to persistent symptoms of the right ventricle failure, repeated coronary angiography was performed showing persisting shunting with the site of extravasation being under the implanted covered stent (confirmed with IVUS) (32). The most conservative approach was used in a case published by Hering et al. Due to the lack of symptoms and hemodynamic stability of the patient, a "wait-and-watch" strategy was chosen, eventually showing a fistula closure after 3 months (36). According to shown data, it is important to mention that RV fistula formation can be clinically dramatic but also (or even more often) silent and slow in progression. A clinician should keep in mind a possibility of coronary-to-RV fistula formation when the patient after MB stenting starts suffering from RV failure. According to case-by-case data using IVUS while stenting, MB is a recommended and rational solution but there is a gap in the evidence on how to perform IVUS measurements in patients with MB - should the largest diameter of the MB itself during diastole be taken as the reference size for the balloon or stent selection or should the reference diameter be the vessel part outside of the MB? Furthermore, should the size of the vessel proximal to the vessel be preferred as a reference measure (thereby risking overexpansion in the bridge or distally to it) or should it be aligned with the part of the vessel distal to the bridge, with stent diameter optimization in the proximal segment? Should we insist that the dimensions of the MB after stenting reach the dimensions of the proximal/distal part of the vessel? As long as clearly defined IVUS parameters are not known, it is our opinion that stenting MB in elective patients (especially if there is not clear evidence of the MB-related ischemia) should be avoided as much as possible as the consequences can be catastrophic. Although the use of drug-coated balloons (DCB) may be a rational option, if we decide to intervene in the almost always present atherosclerotic plaque proximal to MB (if we believe that it, and not the dynamic stenosis is re-

sponsible for ischemia), this method is questionable because it is not clear whether stenting *per se* increases the risk of perforation or whether perforation is due to the sum of the forces delivered to the vessel by stenting plus balloon dilatation. If the latter is true and if severe atherosclerosis is not present, perhaps a direct stent implantation, without predilataion, could be a safer approach, in order to reduce the total force directed toward the vessel wall. We can speculate if maybe more flexible self-expandable stents could be a better option in the above-mentioned scenarios. The stent self-deploys gently and atraumatically without the need for a deployment balloon or aggressive post-dilatation. Due to their elastic properties and short stent struts, the stent conforms to the shape of the vessel, allowing for a more anatomical fit and expands until it reaches the vessel wall but also can continue to expand the post-procedure if the vessel undergoes positive remodelling, which could be a less traumatic option for the dynamic stenosis in the MB. The device is used in vessels with calibre difference along the treated segment, or an expected significant change in diameter in the future (37–39). It is important to take into consideration that artery rupture can be a consequence of stent fracture (but seen less frequently in self-expandable stents), so an IVUS-guided treatment and postprocedural control is considered when hemodynamics is not deteriorated (40). Treatment with stent-grafts (or cover-grafts), although being successful in closing the ruptured site, can lead to subacute thrombosis, with an incidence much higher than after conventional coronary stents are being used (10.3% vs 3.4%, respectively) (34). Therefore, dual antiplatelet therapy longer than 1 year should be considered.

Conclusion

Patients with MB can experience MB-related ischemic symptoms and therefore the initial treatment approach should be medical, with beta-blockers and calcium channel blockers and aggressive risk-factors modification. Patients who remain symptomatic despite medical therapy should be considered for surgical or interventional treatment. PCI should be performed in patients with symptoms, usually after performing the invasive hemodynamic assessment of the bridged segment. IVUS should be used to choose the appropriate balloon and stent size. The most common late complication of MB stenting is in-stent restenosis but due to the MB's thin intima, smaller vessel diameter of the tunnelled segment, overinflating of the balloon and oversizing of the stent, stenting MB can lead to arterial perforation, which can be treated successfully with covered stents.

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Sažetak

STENTIRANJE FUNKCIONALNE STENOZE KOMPLICIRANO PERFORACIJOM KORONARNE ARTERIJE I FORMIRANJEM FISTULE PREMA DESNOM VENTRIKULU U BOLESNIKA S INFAKRTOM MIOKARDA BEZ ST ELEVACIJE

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U ovom prikazu predstavljen je slučaj bolesnika koji je hospitaliziran u našoj ustanovi zbog infarkta miokarda bez elevacije ST segmenta, a na koronarnoj angiografiji je opisano značajno dinamičko suženje ("myocardial bridge") lijeve silazne koronarne arterije s fiksnom stenozom ulaznog dijela. Lezija je tretirana balonskom predilatacijom i stentom obloženim s lijekom, a netom nakon otpuštanja stenta zamijećena je značajna vaskularna fistula između koronarne arterije i desnog ventrikula. Zbog susljedne progresije fistule tijekom sljedećih nekoliko snimanja, fistula je uspješno zatvorena koristeći stent-graft. Također, ukratko su opisane trenutno poznate činjenice oko dijagnostike i terapije dinamičkih suženja koronarnih arterija, kao i do sada opisani slučajevi perforacije koronarnih arterija nakon pokušaja intervencije na dinamičkim suženjima koronarnih arterija.

Ključne riječi: *koronarna intervencija, dinamičko suženje, stent, perforacija*