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CASE REPORT

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Coronary myocardial bridge: an innocent bystander?

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Abstract Myocardial bridge (MB) or tunneled coronary artery is an inborn abnormality, which implicates a systolic vessel compression with a persistent mid-late diastolic diameter reduction. Myocardial bridges are often observed during coronary angiography with an incidence of 0.5%–5.5%. The most involved coronary artery is the left anterior descending artery followed by the diagonal branches, the right coronary artery, and the left circumflex. The overall long-term prognosis is generally benign. However, several risk or precipitating factors (e.g., high heart rate, left ventricular hypertrophy, decreased peripheral vascular resistance) may trigger symptoms (most frequently angina). Herein, we describe two cases of symptomatic myocardial bridge, where medical treatment (i.e., inotropic negative drug) and coronary stenting were successfully utilized to treat this pathology. We also focus on the clinical presentation, and the diagnostic and therapeutic modalities to correctly manage this frequently observed congenital coronary abnormality, underlining the fact that in cases of typical angina symptoms without any significant coronary artery disease, MB should be considered as a possible differential diagnosis.

Key words Angina symptoms · Myocardial bridge · Calcium channel antagonist · Coronary stent

Introduction

Myocardial bridge (MB) or tunneled coronary artery is an inborn abnormality, which implicates a systolic vessel compression with a persistent mid-late diastolic diameter reduction.[1] This entity was first described in 1951[2] and angiographically documented in 1960.[3] The first described case series in 1976[4] reported an incidence of 0.5%–5.5% of MB observed during coronary angiography; a percentage which was later confirmed by more recent angiography reports.[5–9] However, contrary to what is observed during coronary angiography, at necropsy the reported incidence of MB could be much higher (up to 55%).[10]

Fifty percent of patients presenting with MB have concomitant coronary artery disease (CAD) in terms of atherosclerotic lumen vessel narrowing,[4,11,12] especially at the proximal edge of the MB probably due to a turbulent flow phenomenon.[2,6,13] More than two thirds of these patients have systolic vessel compression of >50% of the lumen diameter.[4,11] Several risk factors implicated in symptoms development are the high heart rate, the myocardial contractility state, the presence of a left ventricle (LV) hypertrophy, a decreased peripheral vascular resistance, and the length and localization of the MB.[1,14]

Even if only 15% of the normal coronary flow occurs during systole, this percentage is increased during accelerated heart rate, thereby increasing the significance of the MB.[14] In fact, in cases of MB with a significant persistent diastolic diameter reduction, the coronary flow reserve (CFR) may decrease to 2–2.5 (normal range >3).[7]

The length of MB may vary from 4 up to 40 mm and its thickness between 1 and 4 mm.[4,8,10] The most involved coronary artery is the left anterior descending (LAD) artery followed by the diagonal branches, the right coronary artery (RCA), and the left circumflex (LCX).[1,13] Herein, we describe two cases of symptomatic myocardial bridge, where medical treatment and coronary stenting were successfully utilized to treat this pathology.

Case reports

Case 1

A 73-year-old hyperlipidemic patient known since 18 years previously for a myocardial bridge in the LAD was referred to our center for treatment of moderate drug-resistant angina pectoris. The coronary angiography did not show any

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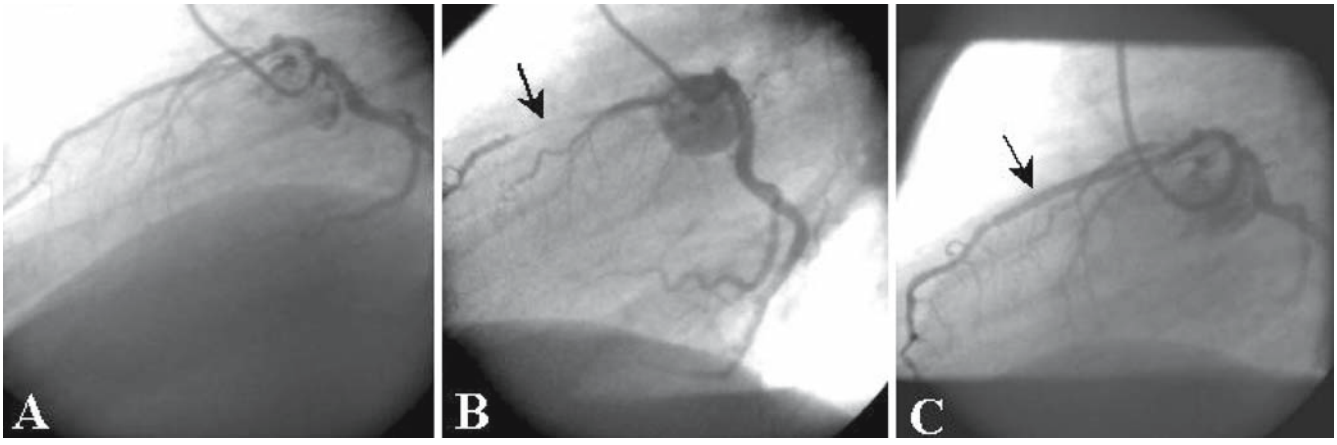

Fig. 1. **A** Lateral view of the left anterior descending artery (LAD) without any significant coronary vessel narrowing. **B** Left anterior descending artery during the isoprenaline i.c. provocative test showing a mid-LAD complete obliteration responsible for the angina symptoms of the patient (*arrow*). **C** Final result after stenting of the LAD, showing no systolic vessel compression at rest and after i.c. isoprenaline injection (*arrow*)

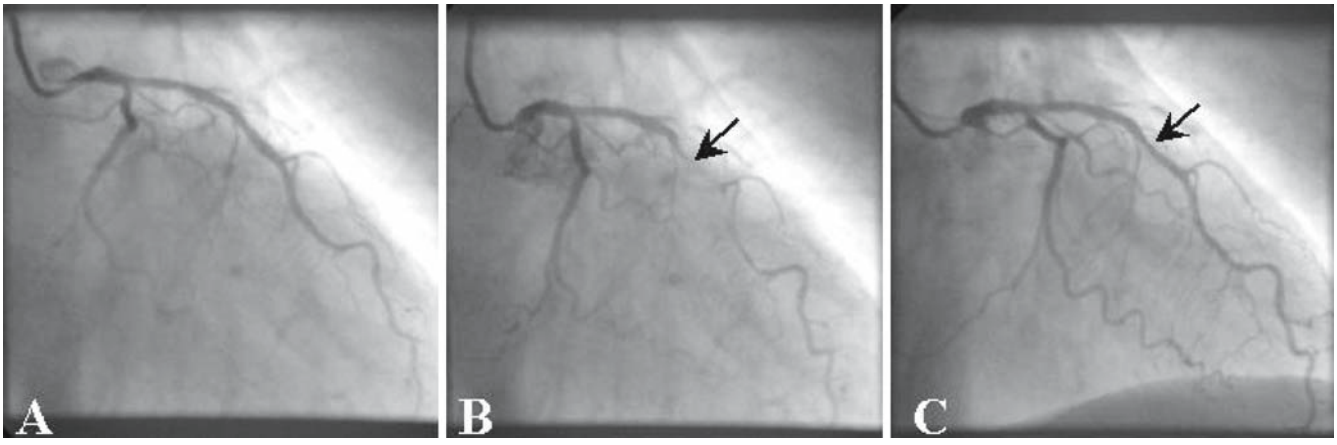

Fig. 2. **A** Left anterior oblique view of the LAD showing no significant coronary artery disease on the LAD. **B** After the i.c. nitrates injection, an important systolic vessel compression appeared on the mid part of the LAD (*arrow*). **C** Disappearance of the LAD vessel compression after i.c. injection of 1 mg verapamil (*arrow*)

significant CAD (Fig. 1A), which could explain the symptoms of the patient and confirm the presence of a mild MB on the mid part of the LAD. In this segment the intracoronary (i.c.) fractional flow reserve (FFR) at rest and after i.c. adenosine injection was normal (0.91). Therefore, we decided to perform an i.c. isoprenaline provocative test aiming to increase heart rate and contractility. At heart rate of 80 beats/min the patient developed angina symptoms with ST depression on the surface ECG, and the angiography revealed a severe compression of the mid-LAD with a pathological FFR (0.71) (Fig. 1B).

Confronted with this clinical and angiographical picture we decided to stent the mid-LAD segment with a Cypher Stent (Cordis, Miami Lakes, FL, USA) (3.0/33 mm at 18 atm) with final abolition of the MB (angiography and FFR normalized) (Fig. 1C) and of his angina symptoms on long-term (i.e., 18 months) follow-up.

Case 2

A 68-year-old hypertensive, hyperlipidemic patient was referred to our center for unstable angina of several weeks' duration. The patient, known for a three-vessel CAD, already percutaneously revascularized a few years before, showed an anterolateral ischemia on exercise ^{201}Thallium myocardial scintigraphy. The coronary angiography showed no significant progression of the known CAD (Fig. 2A). However, probably aggravated by the administration of i.c. nitrates, an important MB on the mid-LAD associated with angina symptoms was observed during the catheterization (Fig. 2B). After the i.c. administration of 1 mg verapamil this MB completely disappeared (Fig. 2C) and the symptoms regressed, therefore we decided to continue an intensive calcium channel antagonist (CCA) treatment (360 mg/day verapamil) with favorable long-term (i.e., 30 months)

outcome (i.e., no symptoms, no recurrent myocardial ischemia at 9 months follow-up scintigraphy).

Discussion

Clinical presentation of MB

Superficial MBs are often asymptomatic and probably only the deep intramural course may provoke cardiac symptoms.[13] A myocardial bridge may damage the coronary endothelium, producing platelet aggregation and vasospasm leading to acute coronary syndromes (ACS).[15] Even if most of the time MBs are incidental findings during angiography, a various spectrum of clinical presentation has already been reported, including ACS, myocardial infarction, sudden cardiac death, ventricular septum rupture, myocardial stunning, and malignant arrythmias.[1,13,16–20]

Men are more involved than women and usually patients presenting with symptomatic MB are 10 years younger than a similar cohort of patients presenting "standard" CAD. Angina pectoris, occurring in up to 70% of the cases, is the cardinal symptom in patients presenting deep intramural MB. These patients may also present pathological functional stress tests especially in the LV anterior wall. Ischemia is mainly due to the systolic vessel compression and the persistent diastolic lumen diameter reduction which, associated with an increased and abnormal coronary flow, triggers symptoms.[6,7]

Diagnosis

Myocardial bridges may be confirmed by quantitative coronary angiography (QCA), by intravascular ultrasound (IVUS), or by i.c. Doppler measurements.[7,9,12]

QCA. If QCA shows a >70% minimal lumen diameter vessel narrowing during systole or a >35% during diastole, significant MB is present.[1]

IVUS. In cases of MB, IVUS analysis shows a typical pattern (half moon phenomenon) with phasic systolic vessel compression and persistent diastolic vessel diameter reduction.[9]

Intracoronary Doppler. By i.c. Doppler, the increased velocity with its typical flow pattern is characteristic of MB (i.e., systolic flow reversal, reduced CFR, diastolic fingertip phenomenon).[1,9] However, in doubtful cases, some i.c. provocative tests may be helpful in posing the diagnosis.[21–24] To further evaluate the clinical relevance of a moderate MB observed during coronary angiography the FFR measurement in this specific region may also be of great interest in the decision making of when and how to treat this coronary milking phenomenon.

Provocative tests. There are several provocative tests that may be performed in the catheterization laboratories in order to better detect clinical relevant MB. To stimulate the heart (inotropism+chronotropism), physical effort during catheterization, or a dobutamine or isoprenaline perfusion are usually utilized according to the local expertise. To test the coronary vasoreactivity and an eventual endothelial dysfunction, a cold test alone or in combination with an acetylcholine or methylergometrine test may also be performed.[24] These pharmacological provocative tests are, however, not complication free and should therefore be utilized only in case of a high clinical suspicion (presence of a MB in the territory of the suffering myocardium) and not routinely in all cases of patients presenting with angina and no significant CAD.[25]

Treatment modalities

The first-line treatment modality of symptomatic MB should be the administration of inotropic negative drugs (i.e., beta-blockers or CCA).[26,27] Nitrates, which increase the degree of the systolic compression, should be avoided,[28,29] and in case of drug-resistant MB, percutaneous (first described in 1995)[8,24,30] or surgical (i.e., myotomy or bypass graft) should be proposed.[31,32] Due to the procedural safety and the reported long-term efficacy of stenting symptomatic MB,[8] this minimally invasive procedure seems to be the first-line treatment in case of drug-resistant MB. However, some caution should be taken, especially if considering the fact that large case series utilizing stents (especially drug-eluting stents) for the treatment of symptomatic MB are still lacking.

Prognosis

The overall long-term prognosis is generally benign,[5] even if some authors are less optimistic regarding a conservative approach of symptomatic MB.[16–18]

Conclusion

Despite myocardial bridges being quite often observed during coronary angiography, they are only rarely clinically relevant. Our two cases illustrate two different clinical presentations of this pathology, underlining two of the possible treatment modalities in case of symptomatic MB.

In case of typical angina symptoms without any significant CAD revealed by angiography but the presence of a mild coronary MB, the possibility of an increased coronary compression during effort as the cause of the angina symptoms should be evoked, especially in concomitance with the above-mentioned risk or precipitating factors. Thus, the eventuality that this mild MB is clinically relevant should be confirmed and actively tested during the cardiac catheterization (i.e., i.c. provocative tests). In fact, the i.c. nitrates injection, usually utilized for coronary vasodilatation, may unmask a symptomatic MB (see Case 2), which in doubtful cases may be further accentuated utilizing several other provocative tests (e.g., i.c. isoprenaline or dobutamine injection) aimed to increase heart rate and inotropism, and thus the coronary milking phenomenon (see Case 1).

Negative inotropic drugs should be the first-line treatment modality of these symptomatic MBs. However, in case of recurrence, despite optimal medical treatment, direct stenting of the compressed coronary segment can be safely attempted, even if more data confirming its long-term efficacy will be necessary before stating that this percutaneously performed procedure should replace surgery.

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