Graft occlusion after coronary artery bypass grafting and stent deformation and in-stent restenosis after succedent stenting in a patient with deep position myocardial bridging

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Abstract

We present a case of deep position myocardial bridging in a patient who had early graft occlusion after coronary artery bypass grafting and had stent deformation and in-stent restenosis after succedent stenting.

Keywords: Myocardial bridging • Coronary artery bypass • Stents • Graft occlusion • Coronary restenosis

INTRODUCTION

Myocardial bridging, an inborn coronary abnormality [1] is defined as a segment of a major epicardial coronary artery, the ‘tunnelled artery’, that goes intramurally through the myocardium beneath the muscle bridge. The estimated frequency that has been reported varies from 1.5 to 16% when assessed by coronary angiography, but in some autopsy series, it is as high as 80% [2]. Myocardial contraction during systole can lead to coronary stenosis. Moreover, the initial systolic compression may cause endothelial dysfunction, and then promote inadequate coronary dilation during diastole in some cases [2]. All of these will induce myocardial ischaemia. Phenotypic expressions of myocardial bridging include asymptomatic, recurrent chest pain, myocardial infarction, life-threatening arrhythmia or sudden cardiac death and so on. Though myocardial bridging is such a well-known coronary abnormality, it still raises clinical concerns due to the lack of definite guidelines for therapy. Therapeutic approaches that have been attempted for myocardial bridging include drugs, such as β-adrenergic receptor blockers and calcium channel blockers, stenting of the tunnelled segment, minimally invasive surgical myotomy and coronary artery bypass grafting. We present a case of myocardial bridging in a patient who had early graft occlusion after coronary artery bypass grafting and had stent deformation and in-stent restenosis after succedent stenting.

CASE REPORT

We report a case of a 34-year old man who presented with intermittent chest distress that had no significant correlation with effort. One year later, his dyspnoea got worse and was accompanied with dizziness and amaurosis. He had hypertension which was well controlled by Telmisartan taken orally. He had no family history of heart disease. The physical examination was unremarkable. Electrocardiogram showed normal. His heart rate was ~65 bpm. On transthoracic echocardiogram, the left ventricular ejection fraction was 59%, with normal systolic function. Coronary angiograph confirmed a myocardial bridging in the middle of his left anterior descending artery, with nearly 90% systolic stenosis. A minimally invasive off-pump coronary artery bypass grafting was performed by a small median sternal incision. Myotomy was tried, but not completed because the bridge was very extensive and deep. The anastomosis of the left internal mammary to the left anterior descending artery was preferred. On the third postoperative day, an acute myocardial infarction located in the partial anterior wall was diagnosed in terms of the significant increase of serum creatine kinase isoenzyme and cardiac troponin-I, and the dynamic electrocardiogram changes. The patient was discharged without any symptoms after drug therapy.

Eight months later, the patient had intermittent chest distress and dizziness again. Coronary angiography and intravascular ultrasound showed that there was a long-segment restenosis near the original myocardial bridging in the left anterior descending artery, and that the graft of the left internal mammary artery was occluded near the anastomotic stoma.

Two stents (Partner Sirolimus-eluting Coronary Stent; Lepu Medical Technology, Beijing, China) were implanted and the stenosis was relieved. His symptoms disappeared. Six months later his symptoms recurred. His further re-examination by coronary angiography showed that the stents were deformed, and that there was an in-stent stenosis close to 90%. Successful percutaneous balloon coronary angioplasty was performed on 23 December 2010 and the stenosis was relieved. There has been no recurrence of his symptoms until now. We continue to follow him up.
DISCUSSION

There is no definite guideline or consensus regarding the therapy of myocardial bridging. Treatment includes drug therapy, stenting, minimally invasive surgical myotomy or coronary artery bypass grafting. The preferred method is drug therapy, mainly including β-adrenergic receptor blockers and calcium channel blockers and so on, which can decrease the tachycardia and increase the diastolic time, with a decrease in contractility and compression of the coronary arteries.

Stenting of the tunnelled segment, minimally invasive surgical myotomy or coronary artery bypass grafting should be tried and performed due to the probability of serious cardiac complications and their poor prognosis when myocardial bridging causes a serious coronary artery stenosis or when drug therapy does not work.

It is very clear that stenting of the tunnelled segment can relieve its stenosis and its relevant symptoms. However, the short-term results of stenting are not satisfactory, not to mention that the long-term results are unclear. The high incidence of in-stent restenosis has been described [2, 3]. Haager et al. [3] reported that of the 11 patients with myocardial bridging undergoing stenting of the tunnelled segment, 5 had shown mild-to-moderate or severe in-stent stenosis by quantitative coronary angiography 7 weeks after the stenting.

The inflammation response caused by the percutaneous coronary intervention may promote the formation of a thrombus and the muscle contraction of myocardial bridging can lead to stent deformation, in-stent stenosis or even coronary artery perforation. In the present case a serious deformation and stenosis of stents had been found 6 months after the stenting. High inflation pressures may be required for optimal stent implantation, but with a higher risk of coronary perforation [2]. Hinan et al. [4] reported a case of the use of a self-expanding intracoronary stent (Radius; Boston Scientific, Boston, MA, USA) for symptomatic myocardial bridging. The results and safety of the self-expanding stent are unclear at present.

Minimally invasive surgical myotomy is the optimal therapy. However, there are some cases with a very extensive and deep myocardial bridging that cannot be myotomied thoroughly due to some serious complications such as an aneurysm or rupture of the heart. Therefore, it is necessary to discover a safe and feasible method of myotomy.

If myotomy could not be done, coronary artery bypass grafting would be performed. Usually left internal mammary artery was preferred as the graft for the myocardial bridging of left anterior descending artery. The biggest problem of coronary artery bypass grafting is the blood flow competition between the graft flow and the native flow of the tunnelled artery, especially in diastole, which lead to thrombosis formation and a bad short-time result. Sabik and Blackstone [5] reported that an important factor affecting early arterial graft patency was native coronary artery blood flow especially when native coronary artery stenosis was not serious. In our case, the patient had a perioperative myocardial infarction and a graft occlusion. There is no consensus as to whether we should ligate the distal artery of myocardial bridging to avoid the blood flow competition when coronary artery bypass grafting is done (Fig. 1).

CONCLUSION

For those myocardial bridging patients with mild tunnelled artery stenosis, drug therapy, mainly β-adrenergic receptor blockers, is preferred. For those who have a serious tunnelled artery stenosis or those whose symptom cannot be relieved by drug, minimally invasive surgical myotomy should be performed. If both do not work, stenting or coronary artery bypass grafting should be attempted. But to get the best patency, we need to find an optimal stent and find evidences as to whether we should ligate the distal artery of myocardial bridging to avoid blood flow competition when coronary artery bypass grafting done.
Conflict of interest: none declared.

REFERENCES