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Kasuistika | Case report

A case of dynamic segmental saphenous vein graft compression during diastole

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SOUHRN

Popisujeme případ pacienta, u něhož koronarografické vyšetření pro infarkt myokardu bez elevací úseku ST pět měsíců po provedení aortokoronárního bypassu prokázalo těsnou stenózu v proximálním segmentu štěpu safény (saphenous vein graft, SVG), navíc s kompresí diagonální tepny v diastole. Segment SVG bezprostředně distálně ke stenóze vykazoval obstrukční dynamickou kompresi v diastole. Po úspěšné implantaci stentu skrze proximální stenózu došlo k vymizení dynamické stenózy dále po proudu. Jde o dosud třetí případ komprese segmentu SVG v diastole bez souvislosti s konstrikcí perikardu, jenž byl publikován v anglicky psané literatuře, a o první případ implantace balon-expandibilního stentu v léčbě významné organické stenózy, navíc s kompresí v diastole. V článku se zabýváme patofyziologickými charakteristikami tohoto případu a možnou využitelností současných stentových platforem v klinické praxi pro dostatečné vyztužení podobných dynamických lézí.

Keywords:
Coronary angiography
Percutaneous coronary intervention
Saphenous vein graft disease

ABSTRACT

We present the case of a patient in whom coronary angiography performed for non-ST-segment elevation myocardial infarction five months after coronary artery bypass graft surgery disclosed a tight stenosis with superimposed diastolic compression in the proximal segment of a saphenous vein graft (SVG) to a diagonal artery. Furthermore, the SVG segment immediately distal to the stenosis exhibited obstructive dynamic compression during diastole. Stent implantation across the proximal stenosis was undertaken successfully with a consequent resolution of the dynamic downstream stenosis. This is the third case of diastolic segmental SVG compression not related to pericardial constriction ever reported in the English literature and the first case where implantation of a balloon-expandable stent was performed in an SVG to treat a significant organic stenosis with superimposed diastolic compression. We discuss the pathophysiologic characteristics of this case and the potential clinical utility of the current stent platforms to adequately scaffold such dynamic lesions.

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Introduction

Saphenous vein grafts (SVGs) are the most frequently used conduits for coronary artery bypass graft (CABG) surgery; yet their utility is limited by gradual attrition leading to recurrent angina in 25% of CABG patients at 1 year [1]. Failure of SVGs during the first postoperative month is due to thrombotic occlusion while later SVGs are affected by intimal hyperplasia, a process that unless being extensive does not cause significant stenosis [2,3]. Intimal hyperplasia with superimposed thrombosis is the main mechanism of SVG failure between the first month and the first year after CABG while the main cause of SVG failure beyond the first postoperative year is atherosclerotic obstruction occurring on a foundation of neointimal hyperplasia. Aside from these intraluminal causes of SVG obstruction, extraluminal causes are also possible but have rarely been reported. To the best of our knowledge, fixed compression of an SVG has been described only once previously; it was due to a large pericardial hematoma formed in the immediate post-CABG period and resulted in transmural ischemia in the territory of the grafted left anterior descending (LAD) artery [4]. Dynamic segmental SVG compression has been reported in only four cases thus far [5–8]. One case involved systolic compression of an SVG to the right posterior descending artery from a dilated right atrium causing angina whereas the remaining three cases involved dynamic SVG compression during diastole. Herein, a unique case of diastolic segmental SVG compression not related to constrictive pericarditis is presented.

Case report

A 46-year-old male patient was referred for coronary angiography because of non-ST-segment elevation myocardial infarction. He had a history of arterial hypertension, hyperlipidemia, cigarette smoking and coronary artery disease status postquadruple CABG with a left internal mammary artery (LIMA) graft to the LAD artery and SVGs to the first diagonal (Dg), second obtuse marginal (OM) and right posterior descending arteries five months prior to his admission. Admission electrocardiography showed dynamic ST-segment depression in leads I, aVL and V4 through V6 whereas echocardiography revealed a normal-sized left ventricle (LV) with lateral wall hypokinesia and an ejection fraction of 45%. Coronary and graft angiography showed a subocclusive left main coronary artery stenosis, a patent LIMA with good distal runoff into the mid LAD artery and retrograde flow into the proximal LAD artery and the LCx artery, occlusion of the SVG to a second OM branch and a patent SVG to the posterior descending branch of a proximally occluded right coronary artery. The SVG to the first Dg artery contained a tight proximal stenosis and non-obstructive atheromatosis in the immediately downstream segment; interestingly, these two SVG segments exhibited dynamic compression during diastole with complete obliteration of the lumen proximally and obstructive narrowing of the immediately downstream segment (Fig. 1 and Video 1). We proceeded with stent angioplasty of the proximal SVG lesion. After low pressure predilation with 1.5 mm and 2.0 mm diameter semi-compliant balloons, a 3.0 mm × 24 mm MGuard stent (MGuard™ Coronary Stent System; Inspire-MD Ltd., Tel-Aviv, Israel) was deployed across the lesion with full stent coverage of the SVG ostium at 10 atm. Stent postdilation and "flaring" were performed with a 3.0 mm × 18 mm non-compliant balloon inflated up to 18 atm. No-reflow phenomenon was not observed and final angiography showed good dilation and patency of the SVG with normal distal runoff into the Dg artery and retrograde flow distribution to a large area of the anterior–lateral wall. Interestingly, the diastolic narrowing of the segment distal to the stented lesion was no longer present (Video 2). Postprocedural creatine kinase and creatine kinase-myocardial band isoenzyme levels were normal and the patient was discharged home the next day on

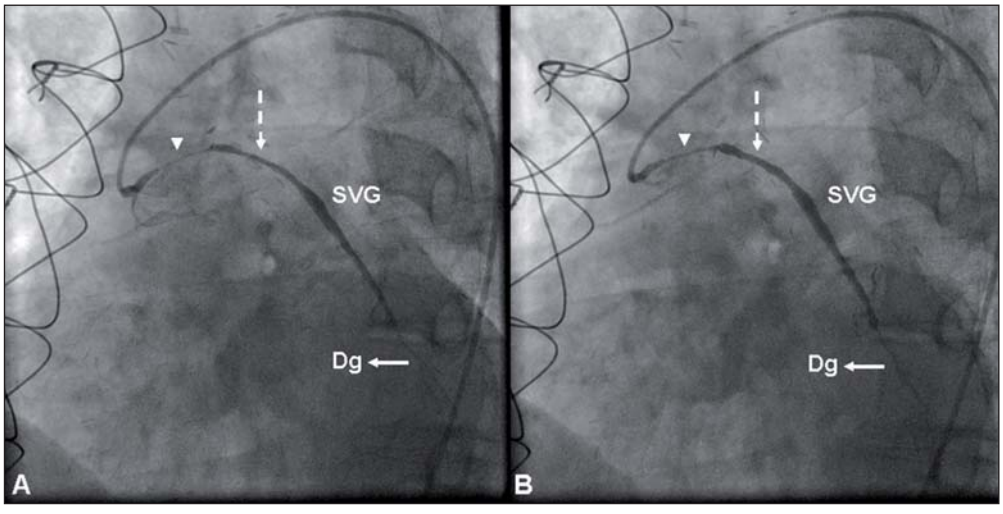

Fig. 1 – Saphenous vein graft angiograms. Left anterior oblique angiographic views of the saphenous vein graft (SVG) to a diagonal artery (Dg) during diastole (A) and systole (B). Note the diastolic lumen compromise of the proximal tightly stenosed segment (arrowhead) and the immediately downstream mildly atheromatous segment (dashed arrow).

dual antiplatelet therapy with aspirin and clopidogrel, an angiotensin-converting enzyme inhibitor, a beta-blocker, and a statin. The patient remained stable for 1-year post stenting with Canadian angina class I and no evidence of a cardiovascular event.

Discussion

Dynamic segmental compression of SVG during diastole is a rare, yet clinically relevant angiographic finding and thus far, only three cases have been reported in the English literature [6–8]. All reported cases involved diastolic segmental compression of the mid or distal shaft of a SVG to the right coronary artery in a setting of severe LV systolic dysfunction (two cases) or constrictive pericarditis (one case). The phasically compressed SVG segment was presumed to be affected by adhesions to the chest wall or nearby structures or the pericardium and diastolic compression was thought to result from cardiac dilation against the resistance offered by the adhesions. Herein, the proximal segment of the SVG to a Dg artery showed a tight stenosis with superimposed dynamic compression during diastole that is a previously unreported angiographic finding. The segment immediately distal to that lesion showed only mild atheroma and an obstructive dynamic stenosis due to diastolic compression. Intimal hyperplasia due to a proliferation of smooth muscle cells and deposition of extracellular matrix is the main disease process affecting SVGs during the first months after implantation [2,3]. Ingrowth of smooth muscle cells into mural, platelet-abundant thrombus, transient SVG wall ischemia during explantation with reperfusion after grafting, and exposure of the SVG to arterial pressures resulting in a marked increase in wall stress are important factors contributing to intimal hyperplasia and fibrosis [2,3]. Our patient presented with a significant stenosis only five months after CABG and there was no angiographically evident thrombus to account for the significant lumen compromise; therefore, the lesion might have been composed predominantly of neointimal tissue [3]. However, our patient had a history of premature coronary artery disease; therefore the bulk of the tight SVG lesion might have been composed predominantly of an atherosclerotic plaque. We presume that the first half of the SVG shaft exhibited fibrotic tethering to the chest wall and/or the aortic wall and adjacent structures that hindered its free movement during LV dilation thereby leading to diastolic narrowing. Pericardial constriction was not shown during echocardiography and nevertheless it could not be the underlying pathology in our case because the portion of the SVG shaft undergoing diastolic narrowing was sufficiently far from the cardiac surface to undergo compression associated with pericardial adhesions.

Given that blood flow in SVGs is predominantly diastolic, dynamic SVG compression during diastole may compromise flow and lead to ischemia. One of the two reported cases of diastolic SVG compression not associated to pericardial constriction, involved angiographically significant diastolic graft narrowing, but scheduled stenting of the affected graft segment was not performed owing to patient's death [8]. There is a single case report describing successful stent implantation across a functionally significant native coronary artery stenosis due to diastolic compression; the patient was implanted with a self-expanding stent which unlike balloon-expandable stents exerts constant outward radial expansive force after deployment thereby opposing the phasically active compressive forces on the vessel wall [9]. Herein, we report the first case of implantation of a balloon-expandable stent in an SVG to treat a significant organic stenosis with superimposed diastolic compression. Because the lesion was bulky, the risk of distal embolization of atherosclerotic debris and slow/no-reflow that is known to be associated with a worse prognosis was judged to be high. In order to prevent these adverse phenomena we selected an MGuard stent (MGuard™ Coronary Stent System; Inspire-MD Ltd., Tel-Aviv, Israel) that is a bare metal stent with a polymeric net covering. Once in place, MGuard seals the atherosclerotic plaque and captures potential embolic debris between the net and the arterial wall [10]. The consequent abolition of diastolic segmental narrowing immediately distal to the stented segment is likely due to normalization of perfusion pressure in the SVG exceeding the collapsing forces produced during LV dilation. Stent implantation for symptomatic myocardial bridging has been shown to neutralize the hemodynamic impact of the muscle bridge and alleviate symptoms of ischemia at the cost of a high rate of in-stent restenosis requiring repeat revascularization at 7 weeks' angiographic follow-up [11]. Persistent external compression potentially inducing neointimal hyperplasia within the bridged segment through increased shear stress and stent fatigue with consequent fracture have been proposed as potential mechanisms of in-stent restenosis in such settings [11,12]. In myocardial bridging, the systolic external compressive forces exerted on the vessel wall are active, in fact, being the high intramural pressure generated during cardiac contraction. In contrast, the forces driving diastolic coronary or graft compression are rather passive and much smaller than the intramural forces in fact, being the resistance to cardiac movement during LV dilation offered by fibrous adhesions affecting the involved vessel segment. Therefore, the persistence of repetitive passive diastolic compressive forces at a vessel segment treated with stent implantation for functionally significant diastolic compression might not be a risk factor for stent fracture or in-stent restenosis.

Conclusion

Diastolic SVG compression is a potential cause of graft failure and should be sought in patients presenting with ischemic symptoms after CABG. In the absence of pericardial constriction, ischemia related to such lesions, might be adequately tackled with the current stent platforms. Obstructive segmental diastolic SVG compression distal to a significant organic stenosis can be abolished after stent implantation across the proximal stenosis likely because the consequent normalization of the perfusion pressure neutralizes the phasically active passive collapsing forces on the conduit wall.

Conflict of interest
No conflict of interest.

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None.

Ethical statement
I declare, on behalf of all authors that the research was conducted according to Declaration of Helsinki.

Informed consent
I declare, that informed consent requirements do not apply to this manuscript.

Appendix A. Supplementary data
Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.crvasa.2015.07.002.

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