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Review

Repeat revascularization: Percutaneous coronary intervention after coronary artery bypass graft surgery

Roberto Scarsini *, Carlo Zivelonghi, Gabriele Pesarini, Corrado Vassanelli, Flavio L. Ribichini

University of Verona, Department of Medicine, Section of Cardiology, Italy

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ABSTRACT

Repeat myocardial revascularization procedures are markedly different from de novo interventions, with increased procedural risk and technical-demanding complexity.
However the number of patients previously treated with coronary artery bypass graft (CABG) that need a repeat revascularization due to graft failure is increasing consistently. Late graft failure, usually caused by saphenous vein grafts (SVG) attrition, is certainly not uncommon. However PCI on degenerated SVG presents higher complication rate and worse clinical outcome compared with native arteries interventions.
In acute graft failure setting, PCI represents a valuable option to treat postoperative myocardial infarction.

1. Introduction

The number of coronary procedures on patients who underwent prior myocardial revascularization increases over time as a consequence of the larger cumulative number of patients treated with CABG surgery or PCI and the longer life expectancy [1]. Repeat myocardial revascularization procedures, whether surgical or interventional, are markedly different from de novo interventions, with increased procedural risk and technical-demanding complexity. Indeed, patients who need repeat revascularization present typically a high-risk profile. They are older, with many comorbidities, extensive vascular disease, complex coronary anatomy and degenerate grafts. They are more likely to present concurrent cardiovascular risk factors and diffusely atherosclerotic and calcified vessels, features that lead to suboptimal stent expansion, defective graft anastomosis and fewer amenable options for re-intervention [2,3].

It has been calculated that 14% to 17% of patients who underwent coronary revascularization in the past 10 years had a history of CABG surgery [4,5]. The likelihood of undergoing more than one coronary intervention has been increased dramatically in the last 30 years due to the ageing of the population and the possibility of disease progression or surgical graft failure [6]. Moreover the age at the moment of the first intervention is predictive of the need of repeat revascularizations, with the younger patients being more likely to undergo repeat procedures [7–9].

Therefore it will be more and more common to deal with this kind of high risk and complex patients in the catheterization laboratory in the next years.

2. PCI in graft failure

Graft failure may occur either early after CABG or after several months or years after surgical revascularization (late graft failure). The type of lesion responsible for graft failure depends on the time of onset of ischemic symptoms.

Ischemia in early postoperative setting (acute graft failure) is mostly due to surgical problems as incomplete revascularization or vascular injury [10]. Late graft failure is usually caused by disease progression, graft degeneration and atherosclerosis. The longer the time from CABG surgery is, the higher is the possibility to deal with complex atherosclerotic plaques with high risk of embolization, especially for saphenous vein grafts (SVGs) [11].

2.1. Acute graft failure

Perioperative myocardial infarction (PMI) is one of the most serious complications after CABG since it is associated with increased perioperative morbidity and mortality as well as poor long-term outcome [12]. The reported incidence of PMI varied considerably, from 3% to 30%,

Abbreviations: BMS, bare metal stent; CABG, coronary artery bypass graft; cTn, cardiac troponin; CTO, chronic total occlusion; DES, drug-eluting stent; EPD, embolic protection device; FFR, fractional flow reserve; IMA, internal mammary artery; IVUS, intra-vascular ultrasound; LAD, left anterior descending; LCx, left circumflex artery; MI, myocardial infarction; MRI, magnetic resonance imaging; OCT, optical coherence tomography; PCI, percutaneous coronary intervention; PMI, perioperative myocardial infarction; RCA, right coronary artery; SVG, saphenous vein graft; TIMI, thrombolysis in myocardial infarction.

* Corresponding author at: Department of Medicine, Division of Cardiology, University of Verona, Piazzale A. Stefani 1, 37100 Verona, Italy. Tel.: +39 0 458 122 320; fax: +39 0 458 027 307.
E-mail address: scarsini.roberto@gmail.com (R. Scarsini).

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because of different diagnostic criteria and variable patient populations [13,14]. In the contemporary large cohort of the PREVENT IV trial, PMI was relatively common in patients undergoing CABG surgery, occurring in approximately 10% of cases. In this trial, PMI was associated with a higher complication rate, longer duration of mechanical ventilation, longer intensive care unit and hospital length of stay. Moreover, patients with PMI had worse outcomes at 2 years compared with those without PMI [15].

Early graft failure after CABG may occur in 8–30% of cases [16–18], depending on the accuracy of the diagnostic method applied to investigate this complication. Systematic perioperative angiography showed defects in 8% of saphenous vein grafts (SVGs) and 7% of left internal mammary artery (IMA) grafts [19], but only a minority of these patients become clinically symptomatic. The rate of early graft occlusion varies from 3% to 12% for vein grafts, 3% to 4% for radial arteries and 1% to 2.5% for internal mammary arteries [20,21].

In most of symptomatic patients after CABG (up to 75%), the main cause of chest pain is ischemia due to early graft failure, while other causes including pericarditis and coronary spasm can be diagnosed in a smaller percentage of patients. [22] (Figs. 1 and 2).

The early identification of patients with graft failure allows adequate early re-intervention strategy such as percutaneous coronary intervention or reoperation with surgical graft revision.

However, early diagnosis of perioperative myocardial ischemia is challenging, since from 5% to 30% of patients present elevation of biomarkers of myonecrosis and/or ischemic electrocardiographic changes after CABG surgery [23].

ECG signs of ischemia, biomarkers modifications, wall motion abnormalities at echocardiography and arrhythmias may raise the suspicion of acute graft failure.

When the diagnosis of graft failure is suspected it should be rapidly assessed by coronary angiography, in particular if this causes hemodynamic impairment [24].

Causes leading to myocardial damage after CABG surgery are classified as graft-related or non-graft-related injuries. Perioperative myocardial ischemia is caused most of the time by the newly implanted graft (graft related injuries), although in a relatively high percentage of case (13–42%) coronary angiography does not reveal any graft failure or new native artery occlusion [22,25,26]. In these cases ischemia is ascribed to air-embolism or microcirculatory damage or inappropriate myocardial protection (non graft-related injuries) [27].

Graft-related ischemia is caused by problems related to the anatomy and the function of the graft such as stenosis in the sites of anastomosis, kinking, overstretching or tension of the grafts, significant spasm, early graft thrombosis, or competitive flow with native coronary vessels [24,28].

Cardiac troponin (cTn) levels have been proposed as a valuable tool in identifying patients who developed graft-related ischemia and consequently most likely to benefit from early invasive strategy. Particularly cTn levels were significantly increased in patients with graft-related PMI compared with patients with non-graft-related PMI. However, cardiac biomarkers for myocardial damage did not separate between graft-related and non-graft related PMI until 12 h after CABG, reaching accurate discrimination capability only 24 h after CABG [12].

Early graft failure, occurring within 30 days from surgery, is primarily triggered by graft thrombosis derived from technical failure in graft manipulation and implantation and consequent endothelial damage and dysfunction. After the implantation of a graft a local inflammatory response can be activated by cytokines and other inflammatory agents, resulting in an endothelial dysfunction, platelets adhesion can lead to thrombosis and early graft occlusion.

Other acute graft failure mechanisms included conduit related problems such as small diameter and pre-existing vein pathology.

It is recommended, when feasible, to confirm acute graft failure by coronary angiography before returning to operating room in order to avoid unnecessary reoperation since a limited but significant percentage of CABG patients presenting with myocardial ischemia signs and symptoms showed no evidence of graft failure at coronary angiography [19].

In early postoperative graft failure, emergency PCI aims to limit the extent of myocardial infarction [29]. Comparison data between redo-CABG and PCI in the contest of PMI are limited but reported PCI angiographical and clinical outcomes resulted equivalent or better compared with redo-CABG [30,22,26,31].

Nevertheless, rescue PCI for acute graft failure is associated with significant higher morbidity and mortality compared with PCI in other contests. In acute postoperative setting, the risk of perforation is high especially at the new anastomosis site. In one series rescue PCI was associated with an overall mortality rate of 21% (15% in-hospital and 6% during the follow-up period) [25].

2.2. Late graft failure

Late graft failure (>6 months after CABG surgery) is usually caused by saphenous vein graft attrition or by the progression of the atherosclerotic process in the native arteries [32].

Saphenous vein grafts (SVGs) are routinely used in CABG surgery as additional conduits to arterial grafts. However SVGs typically present accelerated atherosclerosis resulting in high rate of stenosis or occlusion of the graft.

About 10% of SVGs are occluded early postoperatively, 20% at 1 year and 50% at 10 years of follow-up. Moreover 70% of SVGs are diffusely diseased at 10 years [21].

The prognostic implications of chronic vein graft occlusion have not been completely defined. Vein graft occlusion contributes to higher morbidity and mortality when is the culprit lesion of new MI or is responsible for reoccurrence of angina [33]. In a substudy of the PREVENT-IV trial, the composite end point of repeat revascularization, death and MI occurred more frequently in patients with vein graft failure but the difference was driven by higher rates of revascularization but not by death or MI [34].

The remodeling process is typical of vein grafts. In fact, vein grafts need to compensate the shear stress developed by pulsatile flow which comes from the arterial system and it consists in neointimal formation from vascular smooth muscle cells. The cytokines and local mediators involved in this process help to develop a highly atherogenic substrate on which atherosclerosis develops [35].

It has been shown that the remodeling process of neointimal formation and reendothelialization in the early postoperative period is a critical determinant of vessel patency [36].

Remodeling of the newly implanted graft, with neointimal formation, is followed by a process of increased wall stiffness during the first 6 months [37].

Hyperplasia of neo smooth muscle cells involves mostly the anastomosis site, leading to a progressive stenosis of the graft lumen and anastomosis. Commonly after the first year from surgery, atherosclerosis rather than remodeling is the cause of late graft failure [38].

SVGs attrition is characterized by accelerated forms of atherosclerosis, with friable plaques at high risk for embolization and thrombosis as previously described in detail by our group [11]. A process of “arterialization” with intimal fibrous thickening, medial hypertrophy and lipid deposition has been described by IVUS studies [39], where SVGs usually show an echolucent zone around the vessel that mimics the arterial external elastic membrane. Intracoronary OCT is able to demonstrate SVGs attrition at very early stage with the evidence of wall thickening [40].

Arterial graft patency at long term after CABG surgery is significantly higher compared with SVG (Table 1).

Arterial grafts, contrary to SVGs, rarely undergo attrition from accelerated form of atherosclerosis and they usually require intervention as a