STUDIUM PRZYPADKU / CLINICAL VIGNETTE

Recurrent restenosis in saphenous vein graft. What is the next step?

Nawracająca restenoza w żylnym pomoście aortalno-wieńcowym

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The long-term outcome after percutaneous treatment of saphenous vein graft (SVG) is worse than of native coronaries. We present a patient with degenerated SVG, which had been treated with several interventions. Despite this, there was multiple recurrence of SVG-restenosis. A 76-year-old patient after implantation of a cardiac pacemaker, with arterial hypertension, atrial fibrillation, and chronic kidney disease, underwent coronary artery bypass grafting in 1993. Left internal mammary artery to left anterior descending artery, jump-SVG to marginal (Marg) and diagonal branches and SVG to right coronary artery (RCA) were inserted. Control angiography carried out 18 years later revealed occluded native coronary arteries and occluded SVG segment to Marg. Additionally, there was tight stenosis in RCA-SVG (Fig. 1A). Immediate intravascular ultrasound-guided percutaneous coronary intervention (PCI) of RCA-SVG was done with a 2.5×12 mm paclitaxel-eluting stent implantation and postdilatation. Seven months later, tight in-stent restenosis was diagnosed (Fig. 1B). After predilatation, a 2.5 × 18 mm zotarolimus-eluting stent was implanted. Subsequent two in-stent restenoses a few months later were treated twice with 3.0 × 25 mm paclitaxel-eluting balloons (Figs. 1C, D). The last in-stent restensis (Fig. 1E) was treated with a 3.0×15 mm everolimus-eluting stent. There is no convincing proof of better long-term outcomes after SVG-PCI with drug-eluting stents (DES) compared to bare metal stents (BMS). DES significantly reduces the target vessel revascularisation (TVR) in SVG. However, there is no clear benefit on mortality and myocardial infarction. Histological examination of SVG-lesions shows a different pattern of restenosis compared to native coronaries, with enhanced inflammatory and thrombotic response. A Japanese restenosis registry in native coronaries indicated that if the original restenosis developed in a sirolimus-eluting stent (SES), it would more likely occur again after the next in-stent SES implantation. However, the need for TVR for second restenosis was rarer, if the original restenosis developed in BMS (and not in SES) and was subsequently treated with in-BMS SES implantation. Both first and second generation DES failed in our case. There have been no direct comparisons from randomised trials of different DES types for SVG. Chatani et al.

(J Interv Cadiol, 2009; 22: 354-361) suggested that the only independent predictive factor of re-restenosis after SES-in-SES implantation in native coronaries was the reference diameter. In our case, we implanted two second generation DESs into a first generation DES. Additionally, two PCIs with drug-eluting balloons, postponed further metal jacket insertion. After careful lesion preparation, implantation of the last (third) DES with larger diameter than the previous stents was possible. DESs have the advantage over BMSs in terms of SVG-TVR reduction, but in rare cases both DESs of first and second generation and drug-eluting balloon fail to prevent it. If a sixth restenosis were to occur, implantation of bioabsorbable vessel scaffolding or rotablation might be the treatment option.

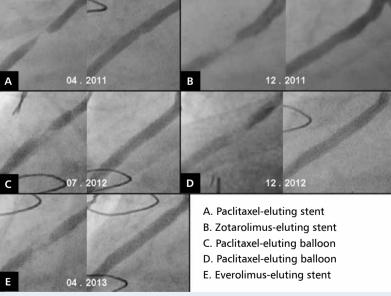


Figure 1. Recurrent restenosis in saphenous vein graft treated with drug-eluting stents (A, B, E) and drug-eluting balloons (C, D)

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