Implantation of nitinol self-expanding carotid stent to release circumflex artery compression caused by mitral valve prosthesis

Milosz Jaguszewski*†, Dariusz Ciecwierz, Marcin Fijalkowski, and Andrzej Rynkiewicz

First Department of Cardiology, Medical University of Gdansk, 80-952 Gdansk, Poland
*Corresponding author. Tel: +48 606877500, Fax: +48 414425554401, Email: jamilosz@gmail.com or milosz.jaguszewski@usz.ch
†Present address: Department of Cardiology, University Hospital Zurich, 8091 Zurich, Switzerland

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A 31-year-old female was admitted to the Cardiosurgery Department, Medical University of Gdansk, with symptoms of congestive heart failure. The patient underwent aortic valve replacement (AVR) with homograft due to severe aortic insufficiency after inflammatory process 12 years previously. Echocardiography revealed severe aortic homograft regurgitation (AR). Left ventricle (LV) was enlarged and LV ejection fraction (LVEF) was 50%. Aortic valve replacement was postponed due to active endocarditis. Two weeks later, control transoesophageal echocardiography showed severe AR and new vegetations on mitral valve leaflets with torrential regurgitation, increased systolic right ventricle pressure to 55 mmHg, and decreased LVEF to 30%. Two weeks after changing the antibiotic therapy, patient underwent AVR (Medtronic 21 mechanical prosthesis) and mitral valve replacement (Medtronic 29 prosthesis). Improvement of LVEF and normal function of mitral and aortic prosthesis were observed. On fifth postoperative day, recurrent ventricular fibrillation occurred. The ECG demonstrated significant ST-segment depression in I, II, III, aVL, aVF, and V5-V6 leads. Coronary angiography was immediately performed and revealed significant systolic occlusion of the left circumflex artery (LCX) around the mitral mechanical prosthesis ring (Panels A and B). The possibility of a suture mishap of the LCX was suspected. Finally, compression of circumflex coronary artery by the mitral prosthesis ring was recognized as a result of systolic–diastolic movement of the prosthesis ring. Percutaneous stenting of the target lesion instead of reoperation with bypass grafting of the LCX was performed. The Acculink self-expanding carotid stent (5.0/40 mm) was implanted (Panels C–E). For post-dilatation, Maveric balloon 4.0/20 mm was used with pressure of 12 atmospheres, resulting in the residual lesion of 10% and TIMI 3 grade flow with normal myocardial blush. Control transthoracic echocardiography showed inferior wall akinesia, anterior intraventricular septum hypokinesia, and normal function of both mechanical prostheses 1 month after discharge. Control angiography 6 months after patients discharge demonstrated no in-stent restenosis (Panels F and G) with no new myocardial perfusion defect in exercise scintigraphy. Patient remained angina free with NYHA II and no severe arrhythmias were recorded during 12 months follow-up.

Majority of patients with accidental LCX compression were treated with immediate saphenous vein bypass grafting. The nitinol self-expanding stent is characterized by its high-tech longitudinal flexibility and compression resistance. Even after compression, it has the ability to regain its original diameter, which determined our decision to use it in this case and avoid reoperation. However, the long-term outcomes and the safety profile of nitinol self-expanding stent implantation to coronary arteries have not been well documented so far.

Panels A and B. Total occlusion of the proximal left circumflex artery in systole (A) and partial compression release during diastole (B) (LAO 30° caudal 30°).
Panels C–E. Self-expanding carotid stent implantation to left circumflex artery (C), angiogram after stent implantation in systole (D) and diastole (E) (LAO 30° caudal 30°).
Panels F and G. Control coronary angiography after 6 months—left circumflex artery without significant compression by the mitral valve prosthetic ring in systole (F) and diastole (G) (LAO 30° caudal 30°).