In stent restenosis (ISR) is a heterogeneous phenomenon. The classical paradigm is neointimal proliferation in response to vessel wall injury often in combination with incomplete stent expansion. Drug-eluting stents (DES) have been designed to reduce or eliminate hyperplasia and in fact dramatically reduced restenosis and target lesion revascularization (TLR). The drawback of DES has been an increase in late and very late stent thrombosis, a potentially life-threatening complication considered a consequence of delayed and incomplete strut endothelialization.

The article of Habara et al. challenges this concept by analyzing in-stent restenosis with optical coherence tomography (OCT) in the first generation DES at different time intervals from the implantation. In this study of 86 sirolimus-eluting Cypher and paclitaxel-eluting Taxus stents ISR they described different neointimal patterns and assessed their prevalence at various time intervals from DES implantation. They demonstrated that restenotic tissue changes its characteristics over time. In the first months after implantation a speckled pattern is frequent and they hypothesized this was caused by proteoglycan deposition and delayed arterial healing. Later after implantation neoatherosclerosis develops with a pattern of thin-cap fibroatheroma (TCFA) observed in >10% of 1–3 years old ISR and >30% IRS older than 3 years, some with plaque rupture and thrombus formation. The majority of these patients was identified with elective angiography and since restenosis is rare also in first generation DES they had to perform control angiography in 2666 patients to identify 91 eligible ISR patients.

Stent neoatherosclerosis has been described more than a decade ago but the reports were dismissed as non-clinically relevant anectodical findings till a growing series of OCT and pathology studies suggested that this was a frequent mode of failure of bare metal stent (BMS) and first generation DES. A histopathology studies suggested that this was a frequent mode of failure and very late anectodical findings till a growing series of OCT and pathology studies suggested that this was a frequent mode of failure and as the Authors actually did in this study approach to treatment, as the Authors actually did in this study post-implantation. We always consider ISR a stable process and the first reaction when we find a borderline significant narrowing is to assess functional severity with FFR. Maybe we should change our attitude, image them with OCT and, if we find high-risk patterns, have a more liberal approach to treatment, as the Authors actually did in this study and as we normally do for lesions in saphenous vein grafts, prone to rapid progression and occlusion. Is the phenomenon specific of the first generation DES? The characteristics of the polymers and platforms radically differentiate stent performance and the lower incidence of restenosis, stent thrombosis, and mortality with the second generation vs. the first generation DES is reassuring. Still we may need to wait few years from the introduction of second generation DES in Japan and another careful OCT Japanese follow-up serial study to have this important answer.

References

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**IMAGE FOCUS**

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Diastolic fluttering of the aortic valve: pathognomonic M-mode features of flail cusp

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An 85-year-old woman presented with recent-onset dyspnoea and fatigue, blood pressure of 120/45 mm Hg, diastolic thrill on the left sternal border, and grade V/VI, high-frequency harsh (buzzing) holodiastolic murmur radiating to shoulder, spine, and vertex of the head. Two-dimensional (2D) (Panel A, see Supplementary data online, Video S1), three-dimensional (3D) (Panel B, see Supplementary data online, Video S2), colour-flow Doppler (Panel C, see Supplementary data online, Video S3), and M-mode transthoracic and transoesophageal echocardiographic images revealed severe aortic regurgitation due to an unsupported non-coronary flail cusp with high-frequency diastolic flutter, and diastolic aortic wall flutter responsible for the murmur’s transmission to the upper torso (Panel D). Surgery confirmed the flail cusp. Pathology showed myxoid degeneration with no endocarditis. Flail aortic valve leaflet results in pathognomonic high-frequency diastolic flutter of the torn aortic cusp and occasionally of the aortic walls. This condition is best demonstrated by M-mode echocardiography because of its higher temporal resolution than other imaging modalities including 2D/3D echocardiography, computed tomography, and magnetic resonance imaging.

Supplementary data are available at European Heart Journal – Cardiovascular Imaging online.

Conflict of interest: none declared.

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