Neoatherosclerosis as reason for stent failures beyond 5 years after drug-eluting stent implantation

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A 69-year-old male (case 1) was admitted due to acute non-ST-segment elevation myocardial infarction (NSTEMI). Eight years earlier, he had previously undergone treatment with a sirolimus-eluting stent (SES). Four years after stent implantation, a follow-up angiography was obtained showing a patent stent without obstructive in-stent restenosis (Panel A). Angiograms obtained at the time of NSTEMI (Panel B) disclosed subtotal occlusion in the middle of the SES (arrowheads). Optical coherence tomography revealed a signal intense luminal layer with an underlying, highly attenuating, diffusely demarcated area, suggestive for an instent fibroatheroma (Panel D) with a minimal cap thickness of 80 μm. Accordingly, ischaemia was caused by the high degree of stenosis (Panel E). Similarly, a 59-year-old male (case 2) was admitted due to STEMI. Nine years before, he had received a paclitaxel-eluting stent (PES). Five years after stent implantation, a follow-up angiography revealed a patent stent (Panel F). Angiograms obtained at the time of STEMI (Panel G) disclosed total occlusion in the proximal of PES (arrowheads). Optical coherence tomography showed a rupture of thin cap fibroatheroma within the stented segment (Panel I). The thin cap fibroatheroma caused a severe stenosis with superimposed thrombus (Panel J).

Neoatherosclerosis has been recently described as particular disease entity being responsible for very late stent failures. These two cases illustrate that the presence of a favourable long angiographic result years after DES implantation does not exclude a future neoatherosclerosis-related event (restenosis or stent thrombosis). Large observational and long-term intracoronary imaging studies are required to fully elucidate the dynamics and clinical relevance of neoatherosclerosis.

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