Coming close and then pulling away

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This editorial refers to 'Late incomplete apposition after drug-eluting stent implantation: incidence and potential for adverse clinical outcomes' by D.A. Siqueira et al., on page 1304

Interventional cardiologists have focused great interest and resources on vascular healing. The obvious reasons for this interest relate to what happens after vascular segments have been manipulated—expanded, stretched, cracked, or otherwise broken—during percutaneous intervention. The earliest energies focused on what happened after balloon dilatation alone. There were four possibilities: (i) a significant dissection developed which disrupted flow and resulted in acute or threatened closure, (ii) the artery was initially stretched but then recoiled within the first 24 h, (iii) the artery healed after the initial injury but then developed neointimal hyperplasia which often resulted in significant stenosis or ischaemia which required treatment, and (iv) the artery healed and did not develop significant neointimal hyperplasia but instead became inert and resistant to subsequent instability.

With the introduction of stents, the situation became more complex because a foreign body was placed and became part of the enduring landscape. This development either eliminated or greatly decreased the above-mentioned issues of acute or threatened closure and recoil. However, the longer term issues remained. With the subsequent introduction of drug-eluting stents (DESs), additional issues were introduced because of the presence of a bioactive drug and a polymer delivery vehicle, both of which could affect the healing process.

Siqueira et al.1 studied the specific process of late incomplete apposition after the implantation of DESs. This interesting and potentially very important phenomenon was defined as 'separation of at least one stent from the vessel wall in a segment without a side branch where the immediate post-implantation IVUS revealed complete apposition of stent struts'. In their series of 195 patients (175 with sirolimus-eluting stents (SESs) and 20 with paclitaxel-eluting stents (PESs)), they identified 10 cases of late incomplete stent apposition (ISA). This late ISA was the result of the marked increase in vessel volume from baseline to follow-up (416–514 mm³) without a change in plaque volume. This can be further defined as regional positive remodelling.

This finding of late ISA is not new. Ako et al.2 in the multi-centre SIRIUS trial using the same definition identified late ISA in 8.7% (n=7) of SES patients but no patient with a bare metal stent (BMS). In a smaller study of 24 patients with SES and 10 with BMSs, Degertekin et al.3 found late ISA in one SES patient (4%) but not in any BMS patient. In other series, late ISA has been seen with both DESs as well as BMSs. Tanabe et al.4 found in the TAXUS trial of 469 patients, late ISA in 5.4% of BMS, 8.0% of slow release, and 9.5% of moderate release TAXUS stents. This phenomenon may be seen more frequently in selected patient cohorts such as diabetics. Jimenez-Quevedo et al.5 found late ISA in 14.5% of 75 patients treated with SES in the DIABETES trial.

One could summarize this IVUS descriptive information as (i) indicating that vascular healing after stent placement may include an increase in vessel volume (positive remodelling) resulting in late ISA, (ii) that this may occur with both BMS and the current DES, (iii) the frequency is low but it may be more frequent with DES although the number of patients studied is small, and (iv) specific patient subsets, e.g. diabetics may be found to have increased remodelling.

The next obvious question is the clinical relevance of these findings, and here, there is uncertainty because of the small numbers of patients studied with late ISA and the very infrequent clinical event that might be related to it in a cause and effect way—namely stent thrombosis. Given these uncertainties, it is not surprising that there may be conflicting results.

Siqueira et al.1 found that one patient with SES and one patient with PES with late ISA had late stent thrombosis. This is in contrast to some other studies6–8 including (i) TAXUS II, in which no stent thrombosis occurred in 23 patients with late ISA,4 (ii) Kimura et al.,7 in which no stent thrombosis was found in 61 ISA sites in 46 SESs in 31 patients, (iii) Ako et al.,7 in which no negative clinical event was found in 19 patients with late ISA with either BMSs or SESs, and (iv) Degertekin et al.,6 in which no adverse event was found at late follow-up in 13 patients with late ISA.

What then can we conclude about this interesting group of patients and the relationship of this interesting physiological response to arterial injury and healing to subsequent adverse events? The amount of data is very limited, but the preponderance of data would indicate no clear-cut relationship. However, perhaps Siqueira et al.1 have the final word of this work in progress: ‘the relationship...
between late acquired ISA and long term adverse outcomes requires further analysis’.

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References


Clinical vignette

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Primary mural endocarditis

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A 57-year-old male was admitted with a 2-week history of fever, rigours, and confusion and a single episode of retrosternal chest pain. He had recently undergone a circumcision. Clinical examination revealed a temperature of 38.3°C and a balanitis. Urinalysis revealed haematuria and proteinuria. He had acute renal failure (urea 24.5 mmol/L, creatinine 341 μmol/L) and an elevated C-reactive protein (356 mg/L). Twelve-lead electrocardiogram demonstrated anterolateral T-wave inversion and troponin T was elevated (0.176 ng/mL), suggesting an acute coronary syndrome. Ultrasound of renal tracts was normal. Group B haemolytic Streptococcus was isolated from both blood cultures and from swabs of his balanitis. Ultrasound of renal tracts was normal. Group B haemolytic Streptococcus was isolated from both blood cultures and from swabs of his balanitis. Trans-thoracic and trans-oesophageal echocardiography demonstrated normal left-ventricular systolic function and an echo dense irregular mass at the apex of the left ventricle (Panel A) but no valvular abnormality. Computed tomography (CT) of brain revealed an infarct adjacent to the right caudate nucleus (Panel B). A radio-labelled isotope white cell scan demonstrated focal uptake at the apex of the left ventricle (Panel C). A diagnosis of mural endocarditis with coronary and cerebral embolization was made. Despite appropriate antibiotic therapy, a left ventricular apical myomectomy was required because of his continuing clinical deterioration. Surgical excision and pathological examination confirmed this rare occurrence.

Mural endocarditis in the absence of pre-disposing factors is extremely rare. This condition is usually fatal; however, our patient was fortunate to survive and make a full recovery.

Panel A. Echocardiogram demonstrating apical vegetation. LV, left ventricle; RV, right ventricle.

Panel B. CT scan of brain demonstrating an infarct adjacent to the right caudate nucleus.

Panel C. Radioisotope-labelled white cell scan with focal uptake at the apex of the left ventricle.