The emerging role of thrombus aspiration in contemporary percutaneous coronary intervention practice

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Coronary atherosclerosis may lead towards thrombogenesis, usually triggered by rupture or erosion of a vulnerable epicardial coronary artery plaque. Most acute coronary syndromes are caused by ruptured atherosclerotic plaques with superimposed thrombus. Following rupture of the fibrous cap covering the atherosclerotic plaque the thrombogenic material in the core of the plaque becomes exposed to the arterial lumen. This causes platelet aggregation and the formation of thrombus. A clinical asymptomatic scenario may follow due to thrombotic sealing of the rupture. In ST-elevation myocardial infarction (STEMI) red thrombus formation often leads to acute vessel occlusion, whereas in non-ST-elevation myocardial infarction (NSTEMI) a non-occluding (mural) platelet-rich thrombus is formed.¹ Intermittent obstruction of flow may be present because the coronary thrombosis consisting of platelet aggregates is unstable and the thrombus waxes and wanes. At this point lesion site constituents may embolise into the microcirculation.² Formation of a fibrin network stabilises the white platelet-rich thrombus as platelet aggregation continues, sometimes leading to occlusion of the epicardial vessel lumen. Persisting alternation of flow, in combination with (partial) obstruction at the lesion site, may result in blood coagulation proximal and/or distal to the plaque rupture that may induce red thrombus formation.

Distal coronary microembolisation of atherosclerotic and/or thrombotic fragments is respon-

sible for a substantial part of clinically observed microvascular obstruction. The embolisation of these fragments may occur spontaneously, as well as due to iatrogenic mechanisms. During balloon angioplasty or stent implantation in percutaneous coronary intervention (PCI) particles of atherosclerotic and/or thrombotic material from the epicardial culprit lesion site embolise into the distal myocardial vessels causing decline of microvascular perfusion. Therefore, removal of atherothrombotic debris has the potential to limit microvascular obstruction as a complication of mechanical reperfusion, as it reduces the burden of debris that may embolise. Several techniques have been developed to remove thrombus from the infarct-related vessel during percutaneous treatment of acute myocardial infarction. The systematic use of thrombus aspiration devices is associated with less distal embolisation, and improved post-procedural myocardial blush grade and ST-segment resolution, two well-validated measures of myocardial reperfusion.³ Moreover, particularly manual thrombus aspiration significantly improves clinical outcome in patients with STEMI undergoing PCI and this effect seems to be adjunctive to that of GP IIb/IIIa inhibitors.⁴ However, thrombus aspiration is unable to limit microvascular obstruction that has already occurred spontaneously before PCI. Intensive multi-drug pharmacological therapy targeting platelets is therefore needed to optimise myocardial perfusion.

In their interesting article Hermens et al.⁵ describe attempted thrombus aspiration in patients with stable and unstable angina pectoris and angiographic evidence of lesion-site thrombus. Manual thrombus aspiration was attempted in 14 patients with a range of clinical presentations. In eight patients TIMI flow grade improved after thrombus aspiration. Also in eight patients visible thrombus was successfully aspirated and TIMI flow grades improved in six of these patients. After PCI, myocardial blush grade was improved in 11 out of 14 patients. Distal embolisation visualised by angiography occurred in one patient.

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Although with a limited number of patients, Hermens et al. provide interesting data on the potential applicability of thrombus aspiration in the prevention of embolisation outside the boundaries of STEMI management. It must be appreciated that the investigators only attempted thrombus aspiration in patients with visible thrombus on coronary angiography; however, thrombus is often not detectable on coronary angiography. In the TAPAS trial thrombus could be obtained in 146 out of 217 patients (67.3%) without visible thrombus on coronary angiography.³ A more widespread use of thrombus aspiration could therefore lead towards better results in the prevention of (micro)embolisation during PCI.

So far, studies with thrombus aspiration data have been focusing on patients with STEMI. It would be of great interest to know what the effect of thrombus aspiration is in patients with other clinical presentations. A success rate of 70% of effective thrombus aspiration in NSTEMI patients has been reported in a pilot study.⁶ A randomised clinical trial is currently being performed comparing thrombus aspiration with conventional PCI in NSTEMI patients.⁷ It would be of great interest to know if thrombus aspiration also limits microvascular ob-

struction within this category of patients, and the findings of Hermens and coworkers suggest an even broader applicability of active removal of atherothrotic debris during PCI. ■

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