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Editorial Comment

Is no-no-reflow following PCI in AMI due to distal embolization of plaque and thrombus?

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Of 3400 patients enrolled in the Horizons-AMI (acute myocardial infarction) trial that assessed the use of bivalirudin versus heparin plus routine GP IIb/IIIa inhibition, 402 patients had intravascular ultrasound (IVUS) imaging poststenting, and of these, 43 patients had IVUS imaging before any intervention as well as post stenting. The accompanying paper [\[1\]](#) describes the observations in this final select group of patients. The authors conclude:

1. In addition to the artery lumen enlarging from the stent, there was a decrease in lesion segment plaque volume. The decrease in plaque volume was greater than the shift of plaque longitudinally from the lesion to the proximal and distal references.
2. The decrease in lesion plaque volume was greater in patients with angiographic thrombus compared to those with no angiographic thrombus.
3. The post-intervention CK-MB correlated with the reduction in plaque volume.

This article represents a large amount of meticulous work, measurements and analysis, not only on this subset of patients but also for the larger IVUS cohort and for the multicenter Horizons-AMI trial. The authors should be congratulated because it is difficult to obtain these data.

Of the 43 patients who had an acute MI and had IVUS imaging prestenting and poststenting, 20 had thrombus identified by angiography and 23 did not. None of the patients with thrombus received thrombectomy. There was more loss of plaque volume in pts with thrombus versus those without thrombus. five patients with thrombus had no-reflow but none of the patients without thrombus had no-reflow. It is a bit difficult to draw emphatic conclusions with such small numbers of patients, but it certainly is consistent with the hypothesis that percutaneous coronary intervention (PCI) with stenting breaks up thrombus which then heads downstream and causes no-reflow. The periprocedural increase in CK-MB correlated with the reduction in plaque volume which also is suggestive that PCI can release material downstream and cause myocardial necrosis, especially if thrombus is present.

So why are there such ambiguous results with thrombectomy in AMI? The authors conclude: “The current findings may also explain why thrombus aspiration or distal protection is not universally beneficial since not all patients with AMI have thrombus,” as documented in this study with IVUS. Perhaps the identification of thrombus would be more sensitive with OCT imaging, but I believe that IVUS is accurate for quantifying a large intraluminal mass of thrombus.

This article raises some interesting questions: how reliable is the measurement of plaque volume post PCI? If there is a difference in pre versus post PCI measurements, is it all explained by downstream embolization? Perhaps. It would be nice to have an in vitro study that measured the downstream effluent material and showed that this corresponds with the delta of plaque + thrombus burden. Speaking in my role as a mentor of young physicians, this would be a nice project for some aspiring interventional cardiology fellow.

What about the accuracy of IVUS for identifying thrombus? Dr. Alibelli-Chemarin, Jacques Puel, and coworkers in Toulouse tried to correlate IVUS imaging of thrombus with the histology of plaque removed by atherectomy in patients with AMI [2]. The difficulty with that study was the sampling error of atherectomy and the fact that the large bore device may have macerated the thrombus or sent it downstream.

What about the accuracy of the particular IVUS device used: the Volcano synthetic aperture machine imaging at 20 MHz? This machine is significantly less accurate than the mechanical rotating devices, even from the same company. The only direct comparison was performed in the early days of IVUS and the amount of information that is not visualized on the synthetic aperture devices is amazing. You don't know what you are missing unless you use both IVUS catheters (mechanically rotating and synthetic aperture) in the same artery (in vivo or in vitro). It always amazed me that the FDA approved the use of the synthetic aperture device. I have never believed that it has sufficient image resolution to be of adequate diagnostic quality for clinical use. The fact that this technology is then used as the basis for computerized interpretation of plaque composition (so called Virtual Histology) is just another manifestation of the wisdom of the venerable statement: “garbage in, garbage out.” However, that is a topic for another editorial.

Conflict of interest: Nothing to report.

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