LONGTERM NATURAL HISTORY OF RADIOFREQUENCY INTRAVASCULAR ULTRASOUND IDENTIFIED CORONARY PLAQUES

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Figure 1 VH-IVUS thin-capped fibroatheroma (VHTCFA).
**Introduction**  Prior studies have shown that virtual-histology intravascular ultrasound (VH-IVUS) identified thin-capped fibroatheroma (VHTCFA) (figure 1) and plaque burden (PB) >70% are associated with major adverse cardiovascular events (MACE). This study examined non-culprit lesion features that predict MACE in long-term follow up and culprit lesion features responsible for myocardial infarction (MI).

**Methods**  170 patients with stable angina (n=100) or MI (n=70) underwent three-vessel VH-IVUS prior to percutaneous coronary intervention (PCI). Patients were followed for MACE which consisted of death, MI, cerebrovascular event, hospitalisation due to unstable angina, or unplanned revascularisation. Non-culprit lesion features were tested for association with future MACE, and culprit lesion features were assessed for initial presentation with MI, using univariate and multivariate analysis.

**Results**  30 372 mm of VH-IVUS were analysed and 1096 plaques classified. 45 MACE occurred in 30 patients over a median follow up of 1115 (968–1537) days. These included 3 deaths, 6 MIs, 3 cerebrovascular events, 15 hospitalisations due to unstable angina, 3 unplanned coronary bypass operations and 15 unplanned PCI.

By univariate analysis, non-culprit VHTCFA (HR=7.57, p=0.014), MLA<4 mm² (HR=3.61, p=0.028) and PB>70% (HR=7.77, p<0.001) were associated with future non-restenotic MACE on long-term follow up (table 1). By multivariate analysis PB>70% (HR=7.77, p<0.001) remained independently associated with MACE.

On univariate analysis, multiple culprit lesion features were associated with initial presentation with MI (table 2), including total and calcified VHTCFA, remodelling index, PB>70%, MLA<4 mm², plaque rupture and thrombus. By multivariate analysis, PB>70% (OR=6.32, p<0.001), thrombus (OR=9.03, p<0.001) and MLA<4 mm² (OR=3.01, p=0.02) were independently associated with MI.

Interestingly, culprit lesion calcified VHTCFA were associated with initial MI (OR=2.59 (1.61–4.16), p<0.001), whereas non-calcified VHTCFA were more likely to be associated with future MACE HR=4.01 (0.87–18.68), p=0.077.

**Conclusion**  Despite the dynamic nature of coronary plaques, non-culprit VHTCFA, MLA<4 mm² and PB>70% were associated with future MACE on long-term follow up, with PB>70% being independently associated. These same features in culprit lesions (amongst others) were associated with MI presentation, emphasizing their biological importance. Interestingly, non-calcified VHTCFA are more likely to be associated with future MACE, whereas it is the calcified variant that is associated with MI.
presentation. This may represent a phenotypic transformation in the VHTCFA from non-calcified to calcified which could reflect multiple healed plaque rupture events (figure 2). This theory requires further investigation.

Table 1 Non-culprit lesion features associated with future MACE
VHThCFA (VH-IVUS thick-capped fibroatheroma), VHTCFA (VH-IVUS thin-capped fibroatheroma), MLA (minimum luminal area), NC (necrotic core), HR, CI

Table 2 Culprit lesion features associated with MI presentation
OR

Note that this fibroatheroma has two distinct layers (arrows) of necrotic core (red) and dense calcium (white), perhaps representing two temporally distinct plaque rupture events that have now healed.