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LONGTERM NATURAL HISTORY OF RADIOFREQUENCY INTRAVASCULAR ULTRASOUND IDENTIFIED CORONARY PLAQUES

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doi:10.1136/heartjnl-2013-304019.53

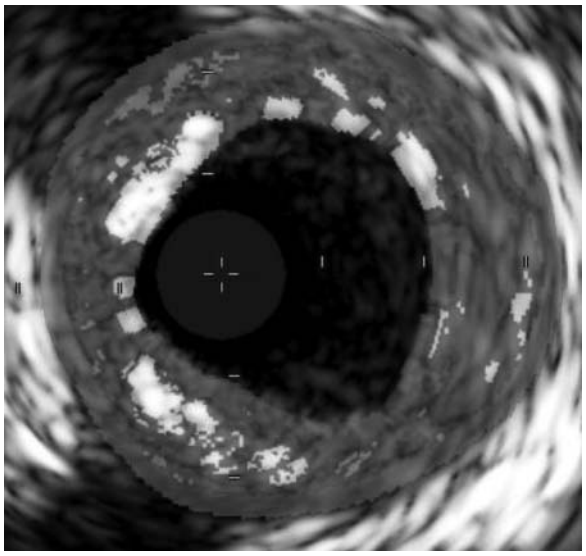

Figure 1 VH-IVUS thin-capped fibroatheroma (VHTCFA).

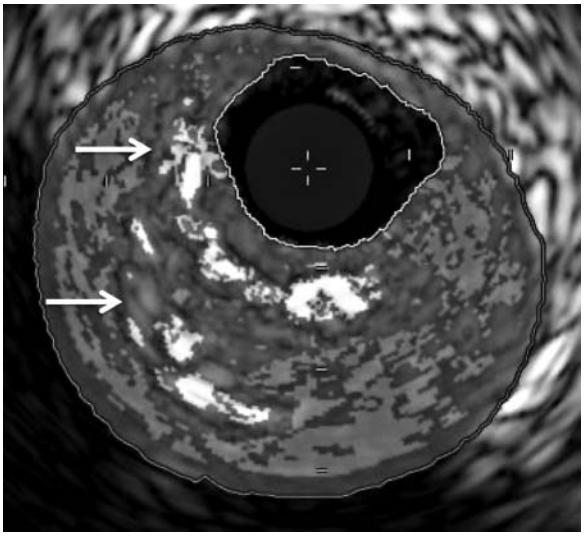

Figure 2 VH-IVUS thick-capped fibroatheroma (VHThCFA).

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 with 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=7.37, p=0.014), MLA<4 mm^2 (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 mm^2, plaque rupture and thrombus. By multivariate analysis, PB>70% (OR (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 mm^2 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, emphasising 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

Table 1

Plaque characteristics	Univariate analysis HR (95% CI)	Univariate analysis p value	Multivariate analysis HR (95% CI)	Multivariate analysis p value
VHThCFA	1.17 (0.23 to 6.24)	0.86		
Total VHTFCA	6.37 (1.45 to 27.94)	0.014	2.18 (0.41 to 11.71)	0.36
Non-calcified VHTCFA	4.01 (0.87 to 18.68)	0.077	2.91 (0.57 to 15.00)	0.20
Calcified VHTCFA	1.51 (0.40 to 5.75)	0.55		
Remodelling index	26.82 (0.36 to 1975)	0.13		
MLA<4 mm^2	3.61 (1.15 to 11.32)	0.028	1.23 (0.24 to 6.22)	0.80
Plaque burden>70%	7.77 (2.06 to 29.28)	0.002	7.77 (2.06 to 29.28)	0.002
Plaque volume (mm^3)	1.00 (1.00 to 1.00)	0.34		
Necrotic core volume (mm^3)	1.00 (0.99 to 1.01)	0.57		
NC percentage	1.01 (0.93 to 1.10)	0.79		

Table 2

Plaque characteristics	Univariate analysis OR (95% CI)	Univariate analysis p value	Multivariate analysis OR (95% CI)	Multivariate analysis p value
VHThCFA	0.84 (0.44 to 1.60)	0.59		
Total VHTFCA	3.05 (1.78 to 5.23)	<0.001		
Non-calcified VHTCFA	1.18 (0.66 to 2.10)	0.58		
Calcified VHTCFA	2.59 (1.61 to 4.16)	<0.001		
Remodelling index	28.05 (5.53 to 142.25)	<0.001		
MLA<4 mm2	8.86 (5.18 to 15.14)	<0.001	3.01 (1.52 to 5.96)	0.002
Plaque burden>70%	15.41 (8.80 to 27.01)	<0.001	6.32 (3.23 to 12.37)	<0.001
Plaque volume (mm^3)	1.004 (1.002 to 1.005)	<0.001		
Thrombus	20.29 (5.58 to 73.76)	<0.001	9.03 (1.60 to 50.98)	0.013
Ruptured plaque	6.77 (2.09 to 21.92)	0.001		

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.