



A Teaching Affiliate
of Harvard Medical School

Panvascular Plaque Vulnerability in ACS

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Background

- Patients with ACS have a higher incidence of recurrent ischemic events.
- Pan-vascular plaque instability may be the underlying mechanism.
- No *in vivo* study has been reported on non-culprit plaque morphology in patients with ACS.



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Aim

To investigate the plaque characteristics of non-culprit lesions in ACS, and compare them with those in non-ACS patients using 3-vessel OCT imaging.



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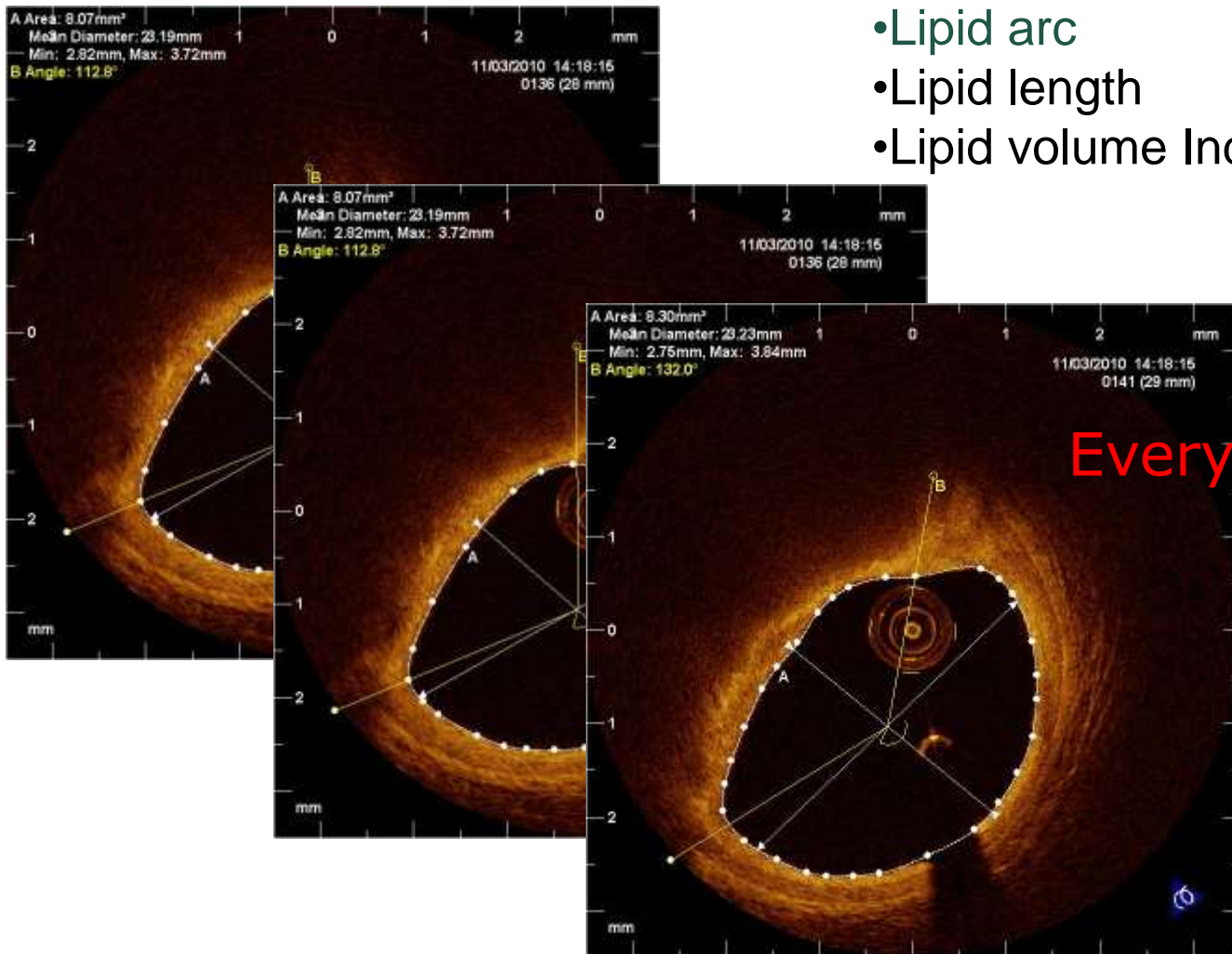
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Study Population

- A total of 108 patients were identified, who underwent 3 vessel OCT imaging from the MGH OCT Registry database.
 - Four patients were excluded due to poor image quality.
 - 104 patients (96.3%) were included in the final analysis.
- Non-culprit plaques with more than 30% diameter stenosis by OCT were included in our study.



Measurement



- Lipid arc
- Lipid length
- Lipid volume Index

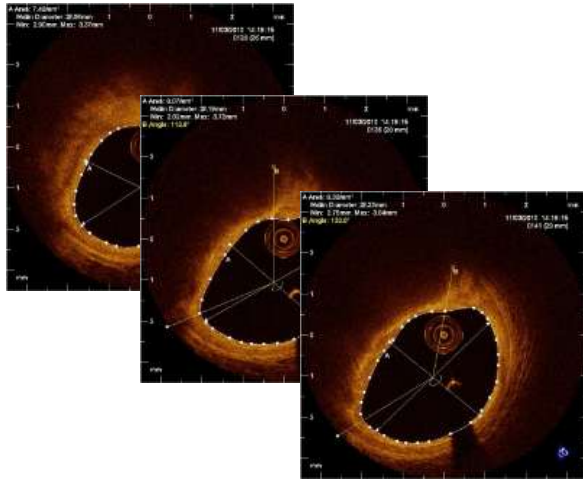
Every 1 mm interval



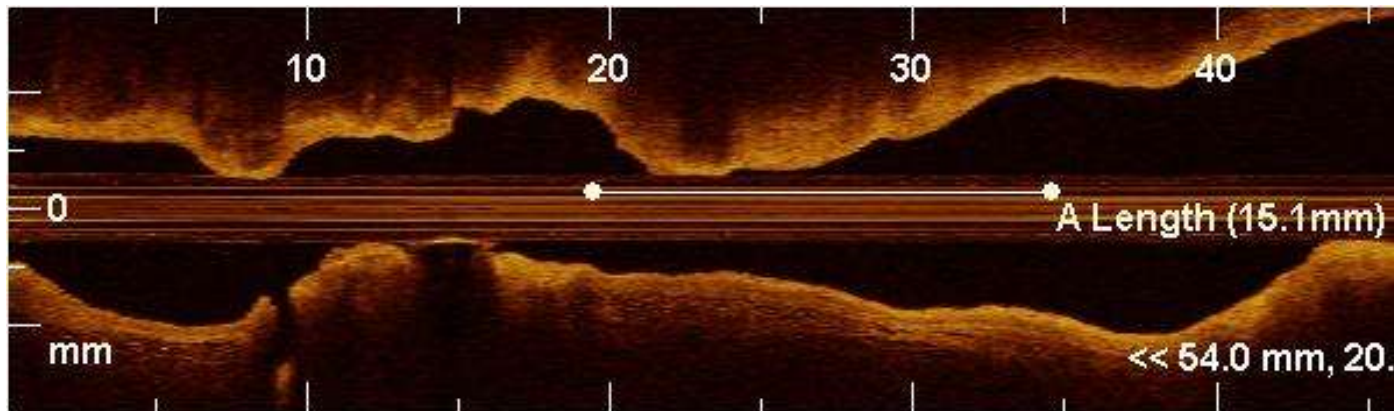
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Measurement

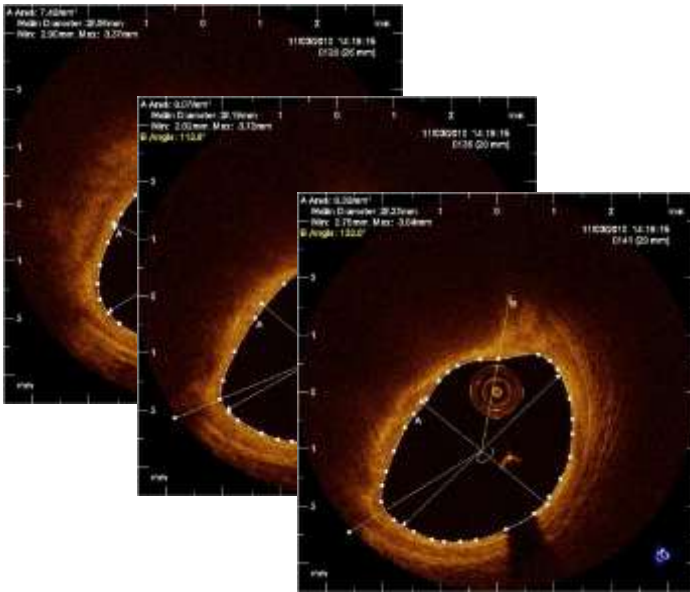


- Lipid arc
- Lipid length
- Lipid volume Index

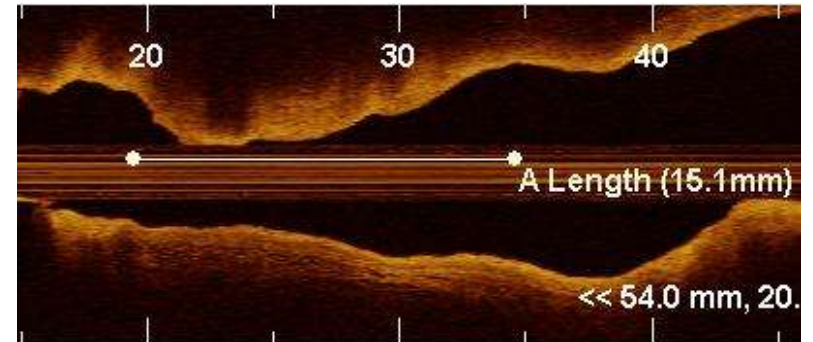


Measurement

- Lipid arc
- Lipid length (longitudinal view)
- Lipid Index



×



$$\text{Lipid Index} = \text{Mean lipid arc} \times \text{length}$$



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Results

Baseline Patients Characteristics

	ACS (n=17)	Non-ACS (n=87)	p value
Age	58.8 ± 14.1	59.1 ± 9.6	0.931
Male	14 (82.4%)	60 (69.0%)	0.383
Hypertension	10 (58.8%)	55 (63.2%)	0.787
Hyperlipiemia	14 (82.4%)	71 (81.6%)	0.999
Smoking	10 (58.8%)	47 (54.0%)	0.794
Diabetes Mellitus	6 (35.3%)	30 (34.5%)	0.999
Prior myocardial infarction	5 (29.4%)	32 (36.8%)	0.783
Peripheral artery disease	1 (5.9%)	3 (3.4%)	0.516
Chronic kidney disease	2 (11.8%)	8 (9.2%)	0.666



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Plaque-based comparison (n=248)

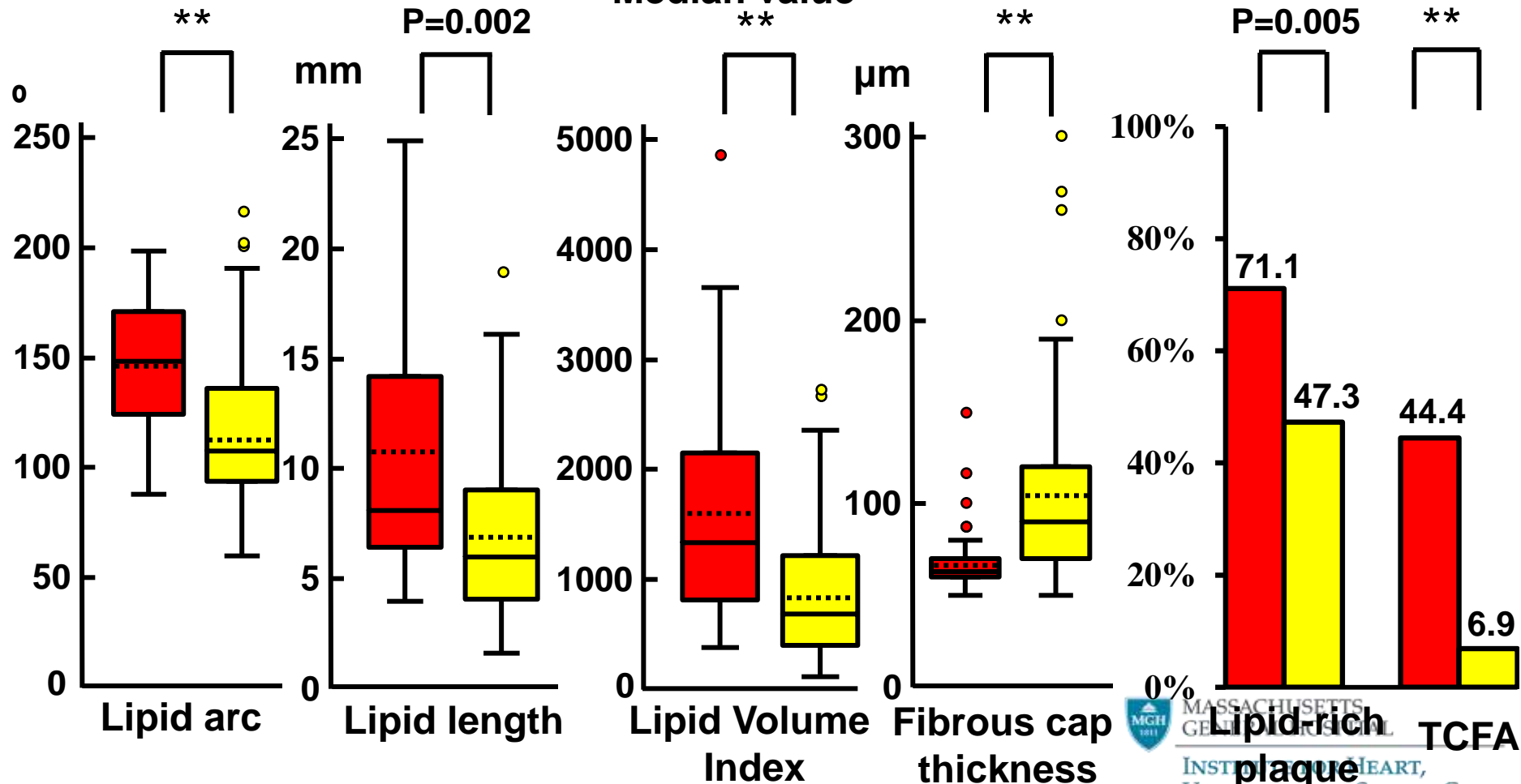
■ ACS plaques

■ Non-ACS plaques

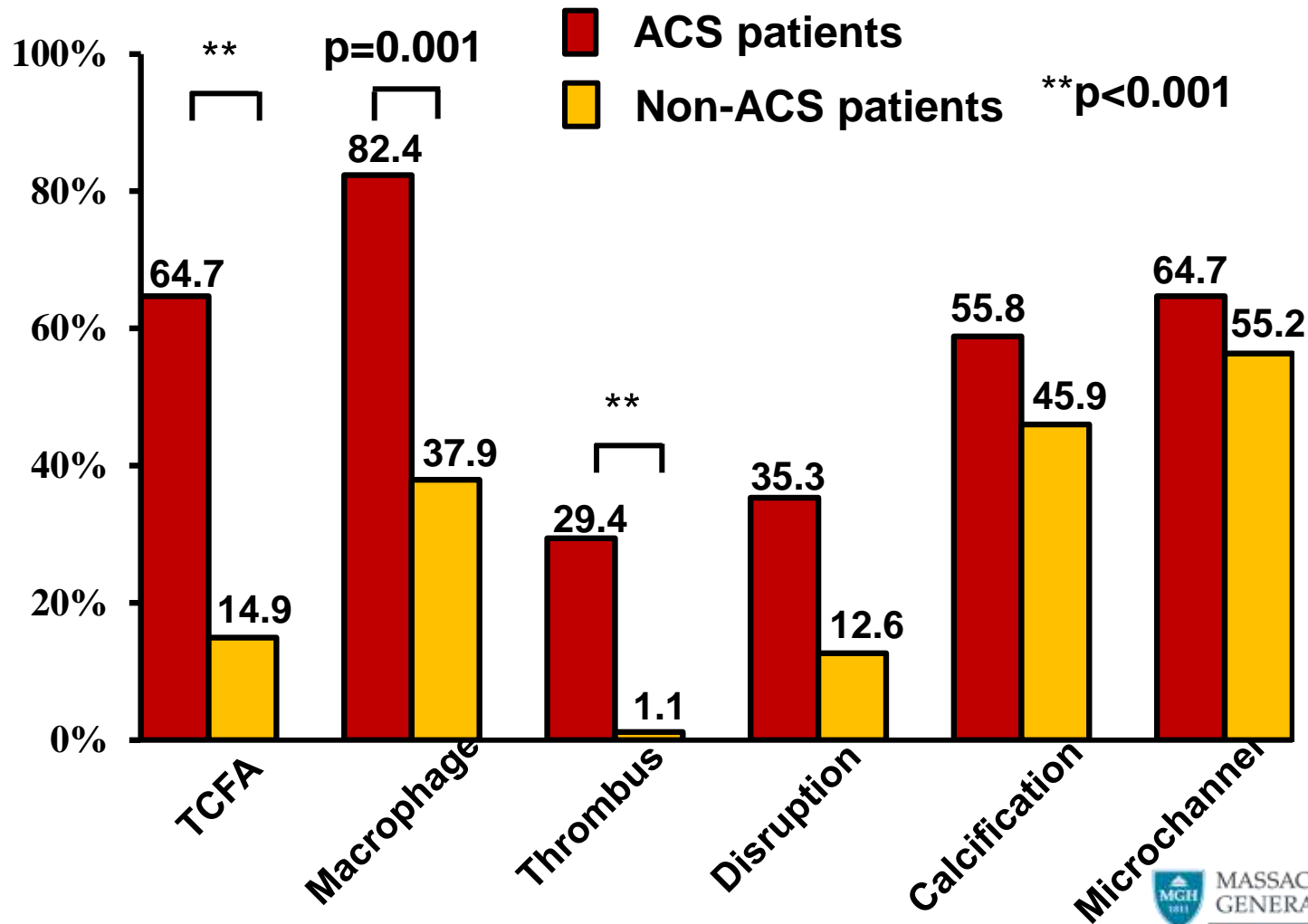
..... Mean value

— Median value

**p<0.001



Patient-based comparison (n=104)



Summary

- Compared to non-ACS plaques, ACS plaques in the non-culprit lesion had a larger lipid volume index and a thinner fibrous cap. TCFA, macrophage, and thrombus in the non-culprit plaques were more frequent in ACS patients.



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Comparison of Non-Culprit Plaque Characteristics between ACS Patients with Ruptured and Non-ruptures Plaque at the culprit Site

: A 3-vessel OCT Study



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Hypothesis and Aims

- We hypothesized that ACS patients with different underlying mechanisms may have different plaque characteristics of non-culprit lesions.
- We sought to compare non-culprit plaque characteristics between patients with and without plaque rupture at the culprit site.

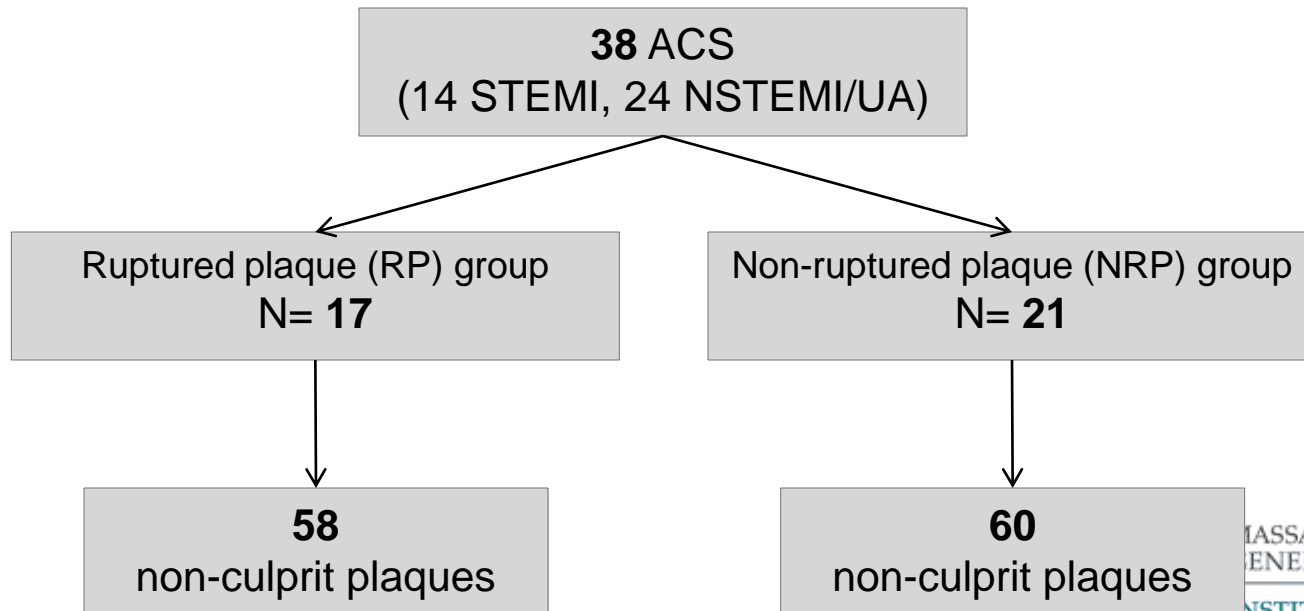


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Methods

- **38** ACS patients with 3-vessel OCT imaging and culprit plaque imaged before PCI were identified from the MGH OCT Registry.
- Patients were divided in two groups according to the culprit plaque morphology: **ruptured plaque group** (RP) and **non-ruptured plaque group** (NRP). Characteristics of non-culprit plaques were compared between the two groups.



Patients characteristics

	RP group (n=17)	NRP group (n=21)	p value
Age, yrs	60.8±13.4	59.8±12.1	0.808
Male, n (%)	16 (94.1)	17 (81.0)	0.233
STEMI, n (%)	9 (52.9)	5 (23.8)	0.064
Family history, n (%)	1 (5.9)	3 (14.3)	0.401
Hypertension, n (%)	12 (70.6)	15 (71.4)	0.955
Hyperlipidemia, n (%)	14 (82.4)	16 (76.2)	0.643
Diabetes mellitus, n (%)	4 (23.5)	5 (23.8)	0.984
Current smoking, n (%)	6 (35.3)	6 (28.6)	0.658
Prior MI, n (%)	2 (11.8)	4 (19.0)	0.540
Creatinine, mg/dL	0.9±0.1	1.2±1.2	0.358
LDL-C, mg/dL	117.5±34.3	94.4±33.0	0.051
HDL-C, mg/dL	42.3±11.4	42.6±12.3	0.940
Aspirin, n (%)	7 (41.2)	7 (33.3)	0.618
Clopidogrel, n (%)	4 (23.5)	5 (23.8)	0.984
β-blockers, n (%)	5 (29.4)	3 (14.3)	0.255
Statins (%)	6 (35.3)	6 (28.6)	0.658
ACE-I/ARBs, n (%)	4 (23.5)	7 (33.3)	0.508

Angiographic characteristics

	Culprit plaques			Non-culprit plaques		
	RP group (n=17)	NRP group (n=21)	p value	RP group (n=58)	NRP group (n=60)	p value
Location			0.582			0.311
RCA	8 (47.1)	7 (33.3)		20 (34.5)	27 (45.0)	
LAD	4 (23.5)	8 (38.1)		24 (41.4)	17 (28.3)	
LCX	5 (29.4)	6 (28.6)		14 (24.1)	16 (26.7)	
QCA analysis						
MLD, mm	0.85±0.62	0.91±0.37	0.727	1.91±0.60	1.83±0.48	0.411
DS, %	72.7±18.5	68.4±11.2	0.386	35.0±11.7	33.5±10.2	0.458
RD, mm	3.04±0.71	2.91±0.56	0.559	2.92±0.69	2.74±0.54	0.117
Lesion length, mm	12.8±5.1	9.7±3.1	0.025*	8.5±3.1	7.4±3.2	0.063



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Patient-based analysis

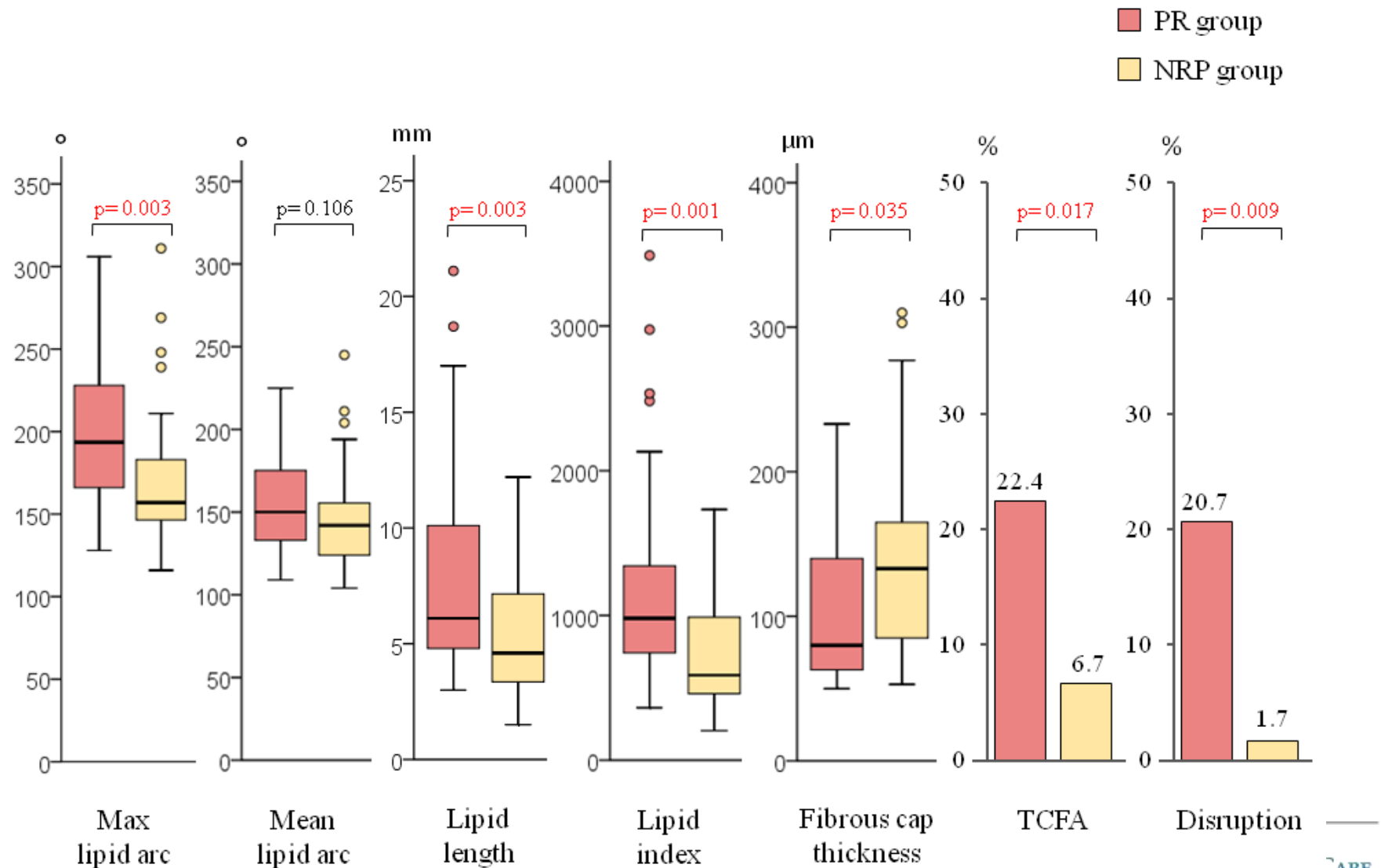
	Patients			Number of plaques/patient		
	RP group (n=17)	NRP group (n=21)	p value	RP group (n=17)	NRP group (n=21)	p value
Plaques	17 (100.0)	19 (90.5)	0.191	3.4±1.5	2.9±1.9	0.339
Lipid-rich plaques	16 (94.1)	15 (71.4)	0.073	2.5±1.3	1.6±1.5	0.079
TCFA	9 (52.9)	4 (19.0)	0.029	0.8±0.9	0.2±0.4	0.013
Disruption	6 (35.3)	1 (4.8)	0.016	0.7±1.1	0.1±0.2	0.011
Thrombus	4 (23.5)	1 (4.8)	0.089	0.4±0.9	0.1±0.2	0.072
Calcifications	12 (70.6)	12 (57.1)	0.393	1.7±1.4	1.3±1.4	0.419
Macrophages	13 (76.5)	9 (42.9)	0.037	1.7±1.3	0.9±1.2	0.057
Microchannels	9 (52.9)	8 (38.1)	0.360	1.2±1.4	0.6±0.9	0.108
Cholesterol Crystals	9 (52.9)	6 (28.6)	0.126	0.5±0.5	0.4±0.7	0.488



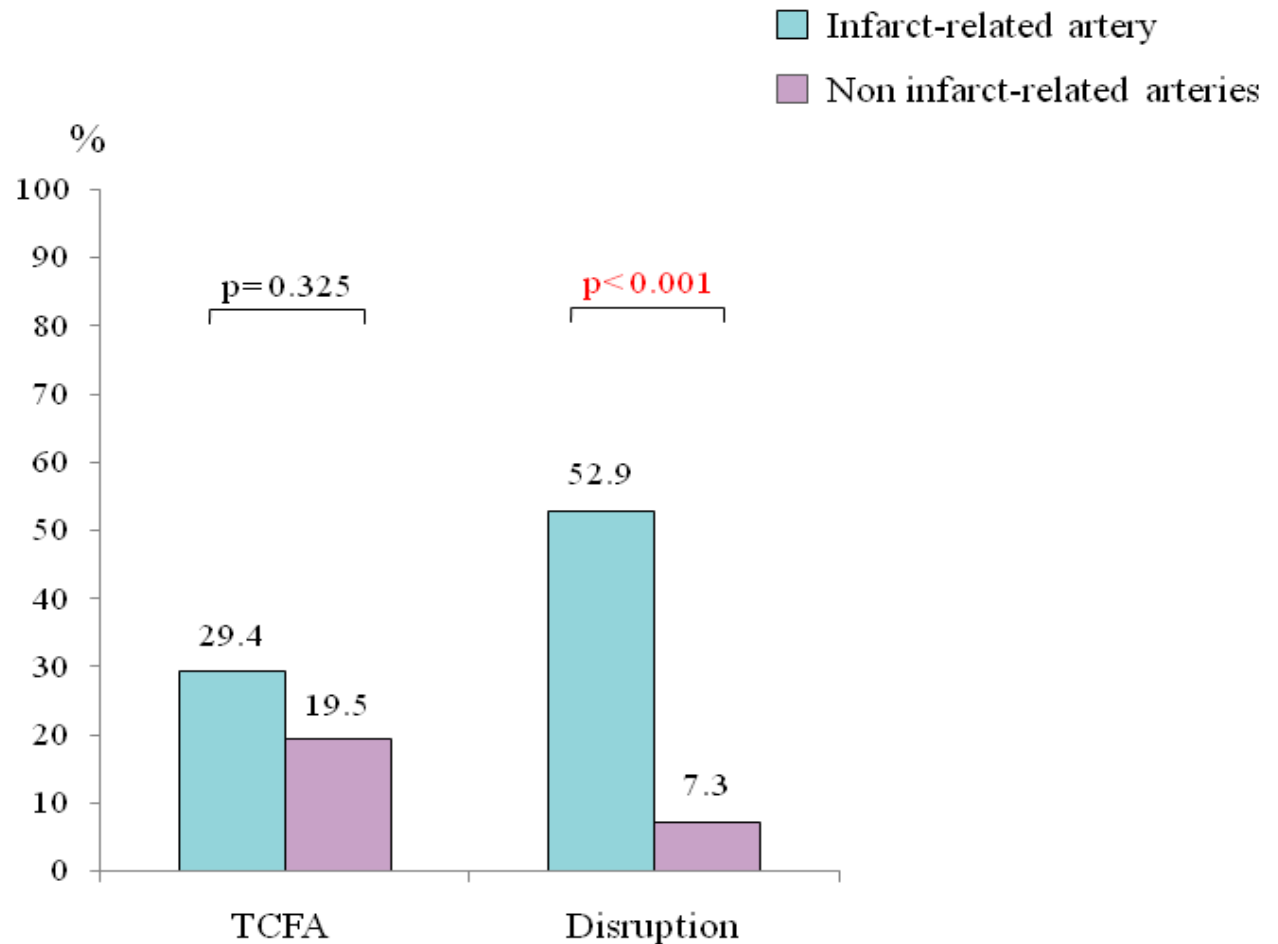
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Plaque-based analysis



Infarct-related vs. Non infarct-related arteries



Summary

- Patients with ACS caused by plaque rupture have non-culprit plaques with greater lipid volume, and higher incidence of TCFA and disruption.
- These results support the concept that ACS is not a focal “accident”, but rather a pancoronary process of instability.



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Conclusions

- The non-culprit plaques in patients with ACS have more vulnerable plaque characteristics compared to those with non-ACS.
- ACS patients with ruptured culprit lesion, compared to with non-ruptured culprit lesion, have more vulnerable features in non-culprit plaques.
- These findings support the concept that plaque vulnerability is a pan-vascular phenomenon in ACS.



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Thank You



MGH history book to comm

AS PART OF the MGH's bicentennial celebrations, a commemorative book of MGH history will be published in 2011. "Something in the Ether, A Bicentennial History" was written by author and publisher Webster Bull. Much of the content was the result of countless hours of research of historical records and archival material. The book is available at the MGH General Store and select booksellers.

(Continued on page 2)



THE MGH AND HARVARD MEDICAL SCHOOL CIRCA 1853:
In the 1800s, the Charles River extended to the hospital, allowing patients and supplies to be dropped off at the MGH wharf.



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