Vascular Inflammation in Plaque Rupture vs. Plaque Erosion: PCAT Attenuation

TCTAP 2022

Ik-Kyung Jang, MD, PhD
Allan and Gill Gray Professor of Medicine
Harvard Medical School

HARVARD MEDICAL SCHOOL
TEACHING HOSPITAL

MASSACHUSETTS GENERAL HOSPITAL
CORRIGAN MINEHAN HEART CENTER

Disclosure

- Allan and Gill Gray Professorship
- Allan Gray Fellowship Funds
- Abbott Fellowship Grant

Background

- Vascular inflammation is a critical factor not only in atherogenesis but also in triggering ACS.

  Ross R. N Engl J Med 1999;340:115-26.

- Systemic inflammatory markers lack specificity for coronary vascular inflammation.

- Recently, a novel non-invasive marker of vascular inflammation measured by peri-coronary adipose tissue (PCAT) attenuation using CCTA has been developed.

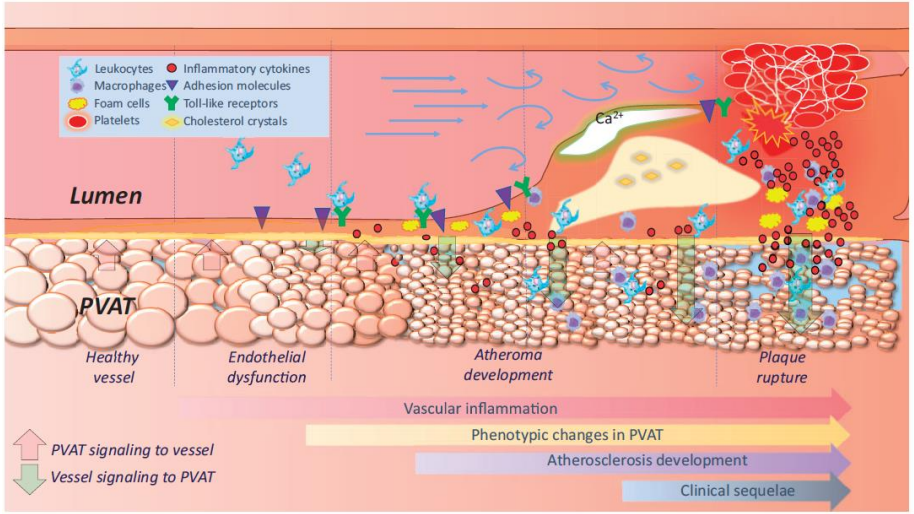
  Goeller M and Dey D. et al. JAMA Cardiol 2018;3:858-863.

- A previous study reported that high PCAT attenuation (high vascular inflammation) is associated with increased cardiac mortality.

  Oikonomou et al. Lancet 2018;392:929-939.

The role of inflammation and perivascular adipose tissue in atherosclerosis

- Peri-coronary adipose tissue secretes pro-inflammatory cytokines and other bioactive mediators which diffuse into the adjacent vascular wall, promoting atherogenesis in a paracrine manner. However, reverse signaling from vessels to the surrounding fat also takes place.
- Inflammatory molecules (TNF-α, IL-6) released from the inflamed arterial wall, diffuse into the perivascular space inducing lipolysis and suppressing adipogenesis.
- This response reduces adipocyte size and creates gradient of lipophilic phase, resulting in peri-coronary adipose tissue (PCAT) attenuation.



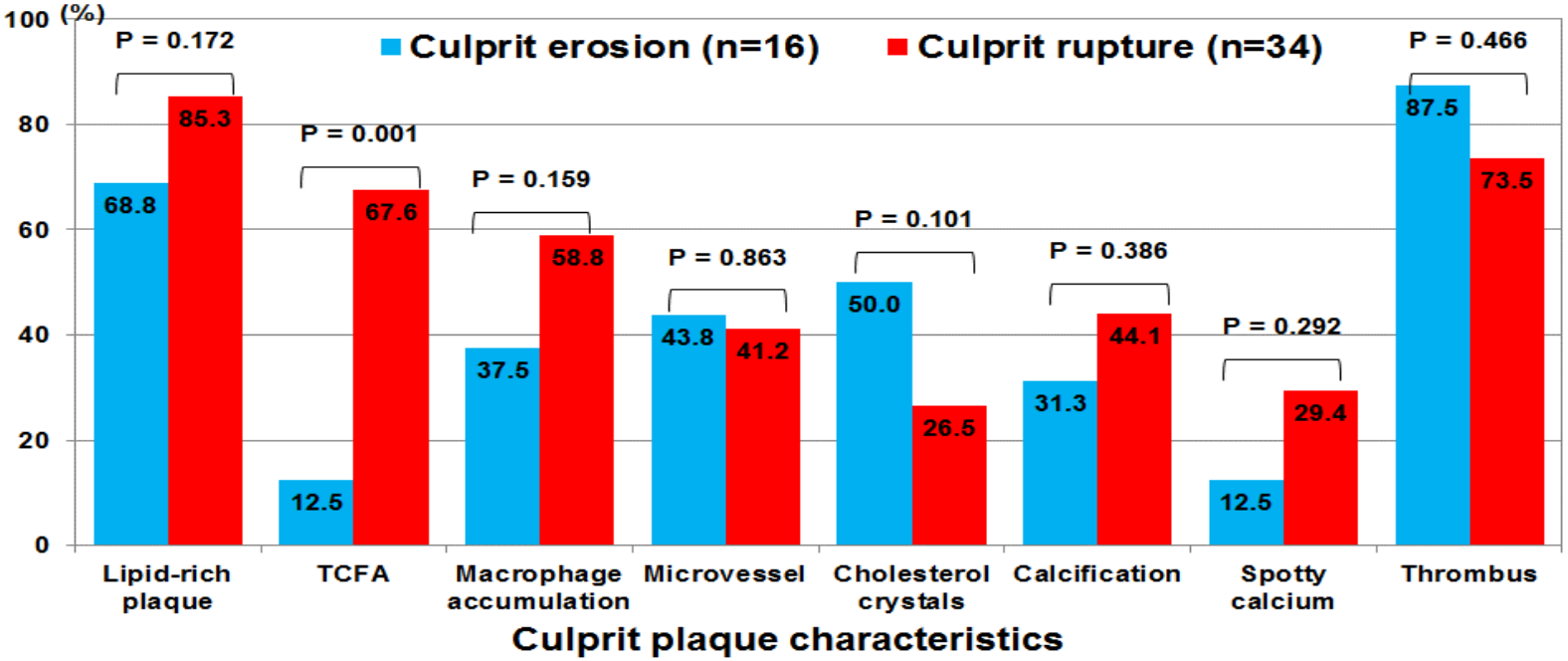
Antoniades et al. *Eur Heart J*. 2020

Hypothesis

The level of vascular inflammation is higher in “plaque rupture” than in “plaque erosion”.

Culprit plaque characteristics : Plaque erosion vs. Plaque rupture

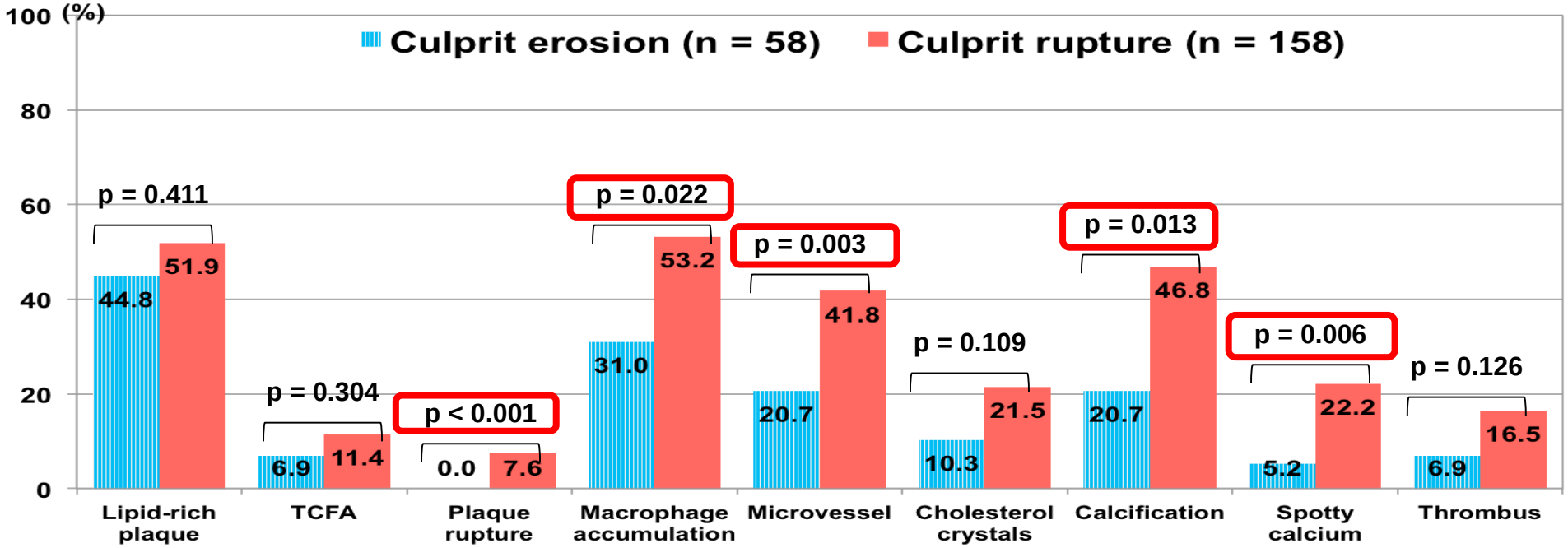
MASSACHUSETTS GENERAL HOSPITAL
CORRIGAN MINEHAN HEART CENTER



Sugiyama T. JAMA Cardiol 2018

Non-culprit plaque characteristics : Plaque erosion vs. Plaque rupture

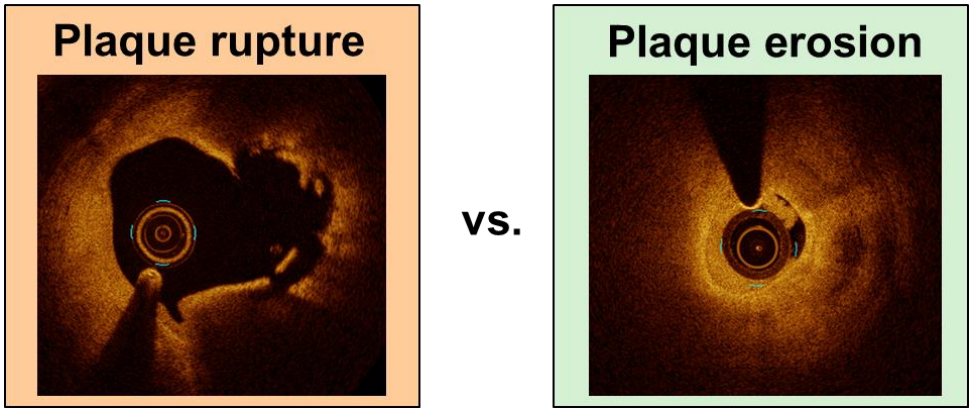
MASSACHUSETTS GENERAL HOSPITAL
CORRIGAN MINEHAN HEART CENTER



Sugiyama T. JAMA Cardiol 2018

Background and Aim

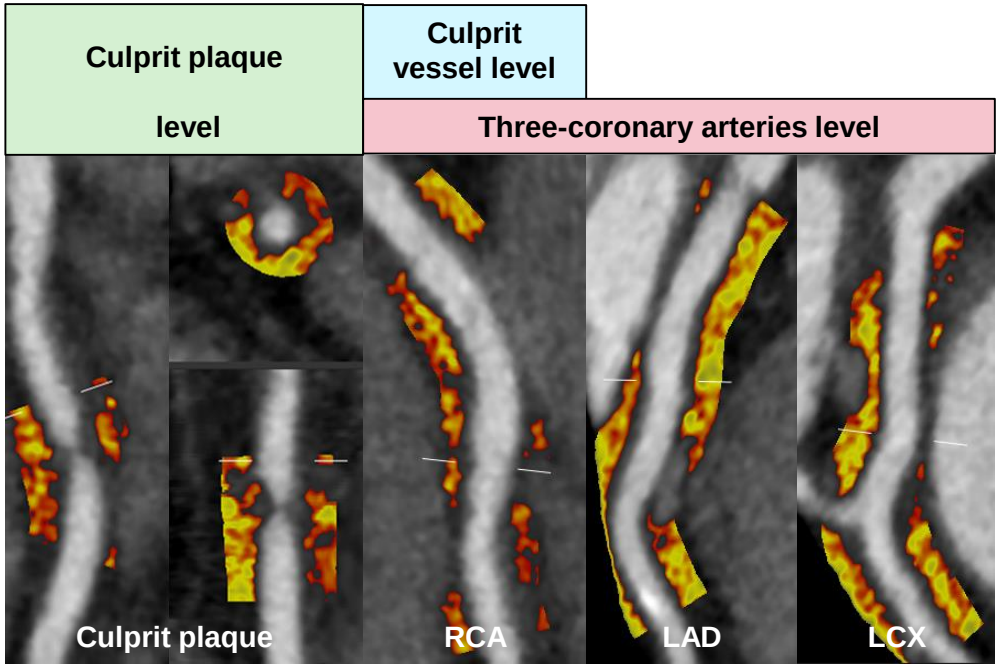
- However, the previous study was phenotyping of coronary plaques and lacks biological information.
- The aim of the current study was to compare the level of vascular inflammation measured by PCAT attenuation between patients with plaque rupture versus plaque erosion.



Methods

- 198 patients with NSTE-ACS, who underwent pre-intervention CCTA and OCT (NCT04523194).
- PCAT attenuation was measured by semi-automated software using Autoplaque version 2.5 (Cedars-Sinai Medical Center, California, USA).
- Culprit lesion pathology was identified by OCT: 107 plaque rupture and 91 plaque erosion.

Methods

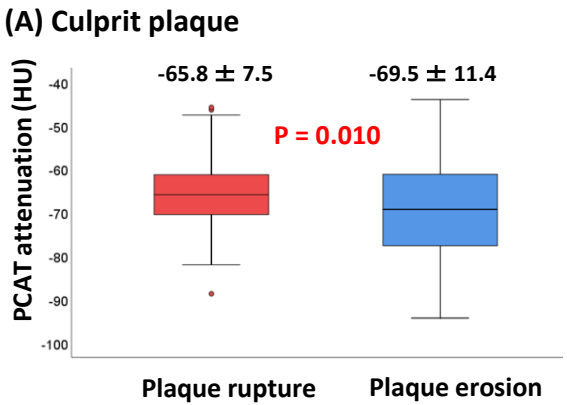


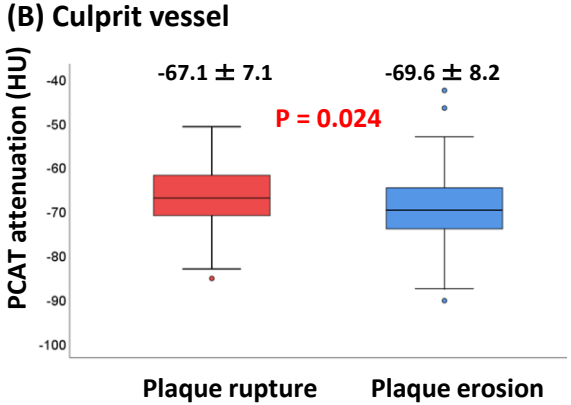
PCAT attenuation measurement in a patient who had culprit plaque in the RCA.

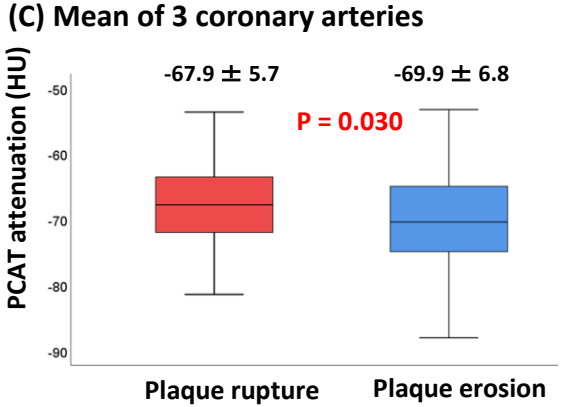
The level of vascular inflammation was measured at 3 levels:

- Culprit plaque
- Culprit vessel
- All 3 coronary arteries

Results

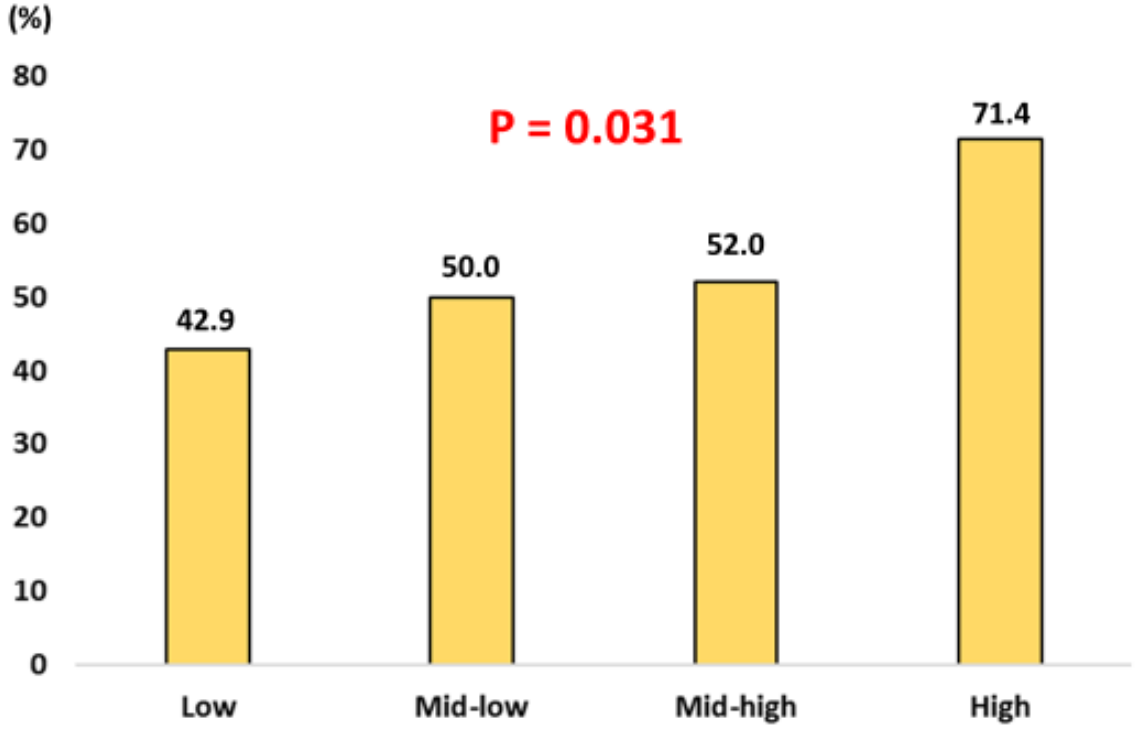






Nakajima A. JACC Img 2021

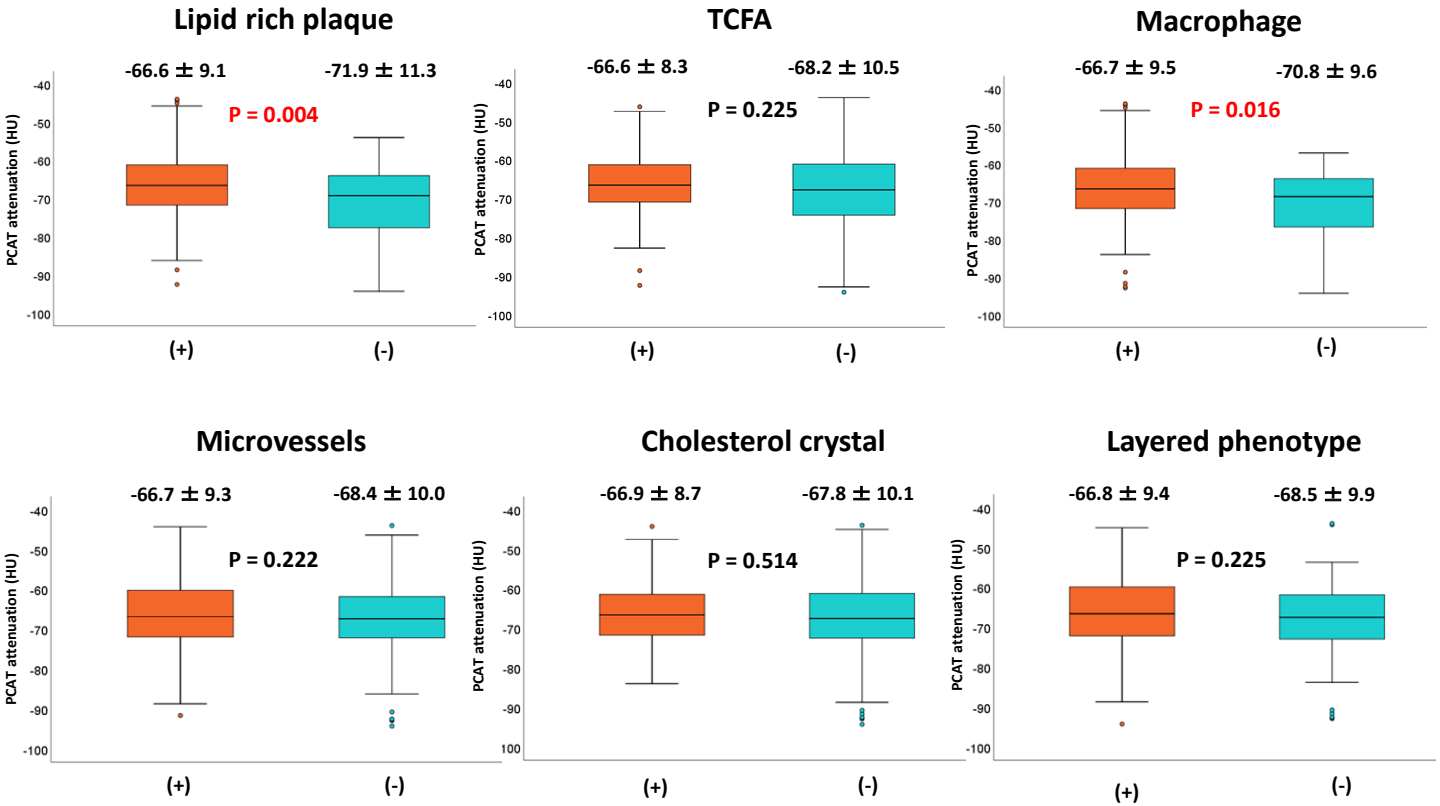
Results
(PCAT attenuation level and Prevalence of Plaque rupture)



Quartile based on culprit vessel PCAT attenuation

Results
(Culprit plaque features of vulnerability and PCAT attenuation)

MASSACHUSETTS GENERAL HOSPITAL
CORRIGAN MINEHAN HEART CENTER



Results
(Univariable and multivariable analysis)

MASSACHUSETTS GENERAL HOSPITAL
CORRIGAN MINEHAN HEART CENTER

	Univariable		Multivariable	
	Regression coefficient b [95%CI]	P value	Regression coefficient b [95%CI]	P value
Culprit Plaque PCAT attenuation				
Plaque rupture (vs. Plaque erosion)	**3.647 [0.917, 6.376]**	**0.009**	**2.902 [0.319, 5.484]**	**0.028**
Age, y	-0.003 [-0.126, 0.120]	0.961		
Male (vs. Female)	5.960 [1.629, 10.291]	0.007	5.968 [1.999, 9.937]	0.003
NSTEMI (vs. unstable angina)	4.051 [0.504, 7.598]	0.025	3.710 [0.548, 6.871]	0.021
HT	2.548 [-0.329, 5.425]	0.083	1.848 [-0.789, 4.485]	0.170
DL	-1.242 [-3.930, 1.446]	0.365		
DM	0.860 [-1.881, 3.601]	0.538		
Current Smoker	1.480 [-1.193, 4.153]	0.278		
ASA	0.419 [-2.736, 3.573]	0.795		
Statin	-2.048 [-5.116, 1.019]	0.191		
Culprit Vessel PCAT attenuation				
Plaque rupture (vs. Plaque erosion)	**2.475 [0.327, 4.623]**	**0.024**	**2.429 [0.389, 4.470]**	**0.020**
Age, y	-0.011 [-0.095, 0.074]	0.806		
Male (vs. Female)	6.039 [3.012, 9.066]	< 0.001	6.009 [3.071, 8.946]	< 0.001
NSTEMI (vs. unstable angina)	2.078 [-0.631, 4.788]	0.133		
HT	1.600 [-0.708, 3.907]	0.174		
DL	-1.489 [-3.626, 0.649]	0.172		
DM	1.148 [-1.097, 3.393]	0.316		
Current Smoker	1.338 [-0.811, 3.488]	0.222		
ASA	-0.408 [-3.125, 2.309]	0.768		
Statin	-0.995 [-3.458, 1.467]	0.428		

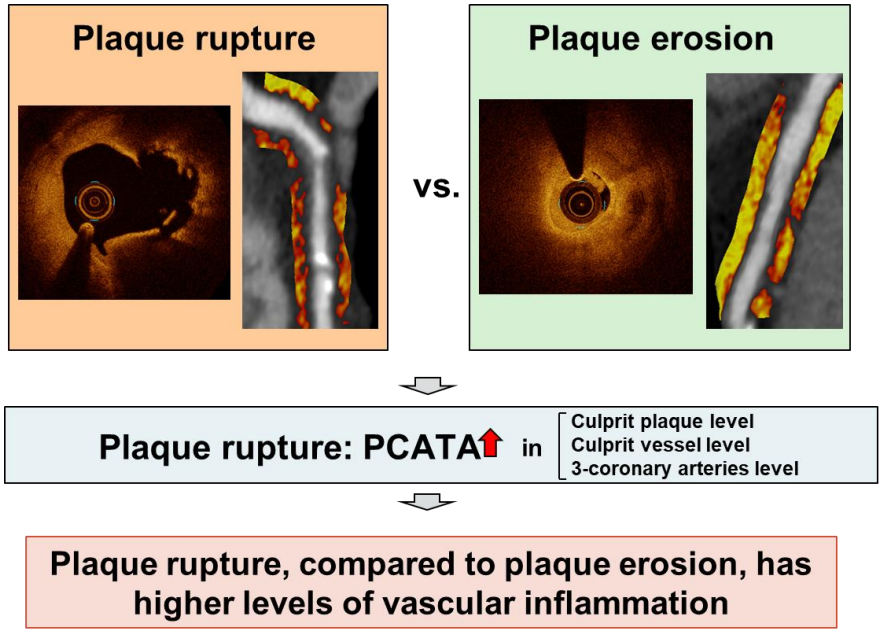
	Univariable		Multivariable	
	Regression coefficient b [95%CI]	P value	Regression coefficient b [95%CI]	P value
3-coronary arteries PCAT attenuation				
Plaque rupture (vs. Plaque erosion)	**1.978 [0.211, 3.745]**	**0.028**	**1.730 [0.136, 3.325]**	**0.033**
Age, y	0.002 [-0.071, 0.075]	0.953		
Male (vs. Female)	5.742 [3.235, 8.249]	< 0.001	5.531 [3.271, 7.791]	< 0.001
NSTEMI (vs. unstable angina)	0.842 [-1.337, 3.022]	0.449		
HT	2.122 [0.215, 4.009]	0.029	1.527 [-0.153, 3.208]	0.075
DL	-1.175 [-2.924, 0.574]	0.188		
DM	1.609 [-0.138, 3.357]	0.071	1.386 [-0.287, 3.059]	0.104
Current Smoker	1.031 [-0.728, 2.790]	0.251		
ASA	-0.357 [-2.574, 1.861]	0.753		
Statin	-0.110 [-2.104, 1.884]	0.914		

Plaque rupture was significantly associated with higher PCAT attenuation values at all 3 levels: culprit plaque, culprit vessel, and 3-coronary arteries.

Conclusions

MASSACHUSETTS GENERAL HOSPITAL
CORRIGAN MINEHAN HEART CENTER

PCAT attenuation was higher in plaque rupture than in plaque erosion at the culprit plaque, culprit vessel, and all 3 coronary arteries. The results indicate pan-coronary inflammation plays a more important role in plaque rupture than in plaque erosion.



Thank you

MASSACHUSETTS GENERAL HOSPITAL
CORRIGAN MINEHAN HEART CENTER

1811

ijang@mgh.harvard.edu

2025