

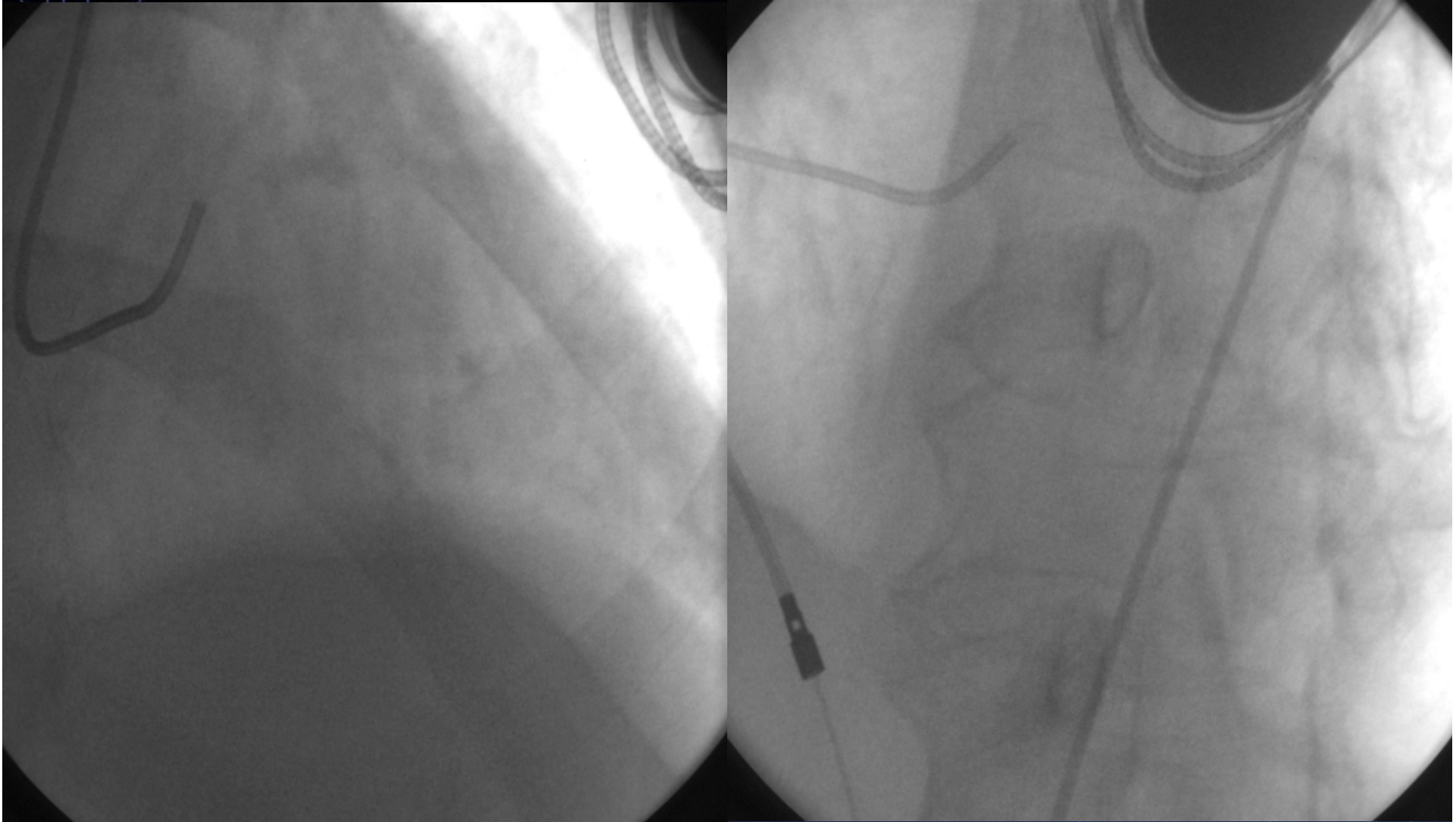


Relation Between Baseline Plaque Characteristics and Post-PCI Outcome

Young Joon Hong, MD, PhD

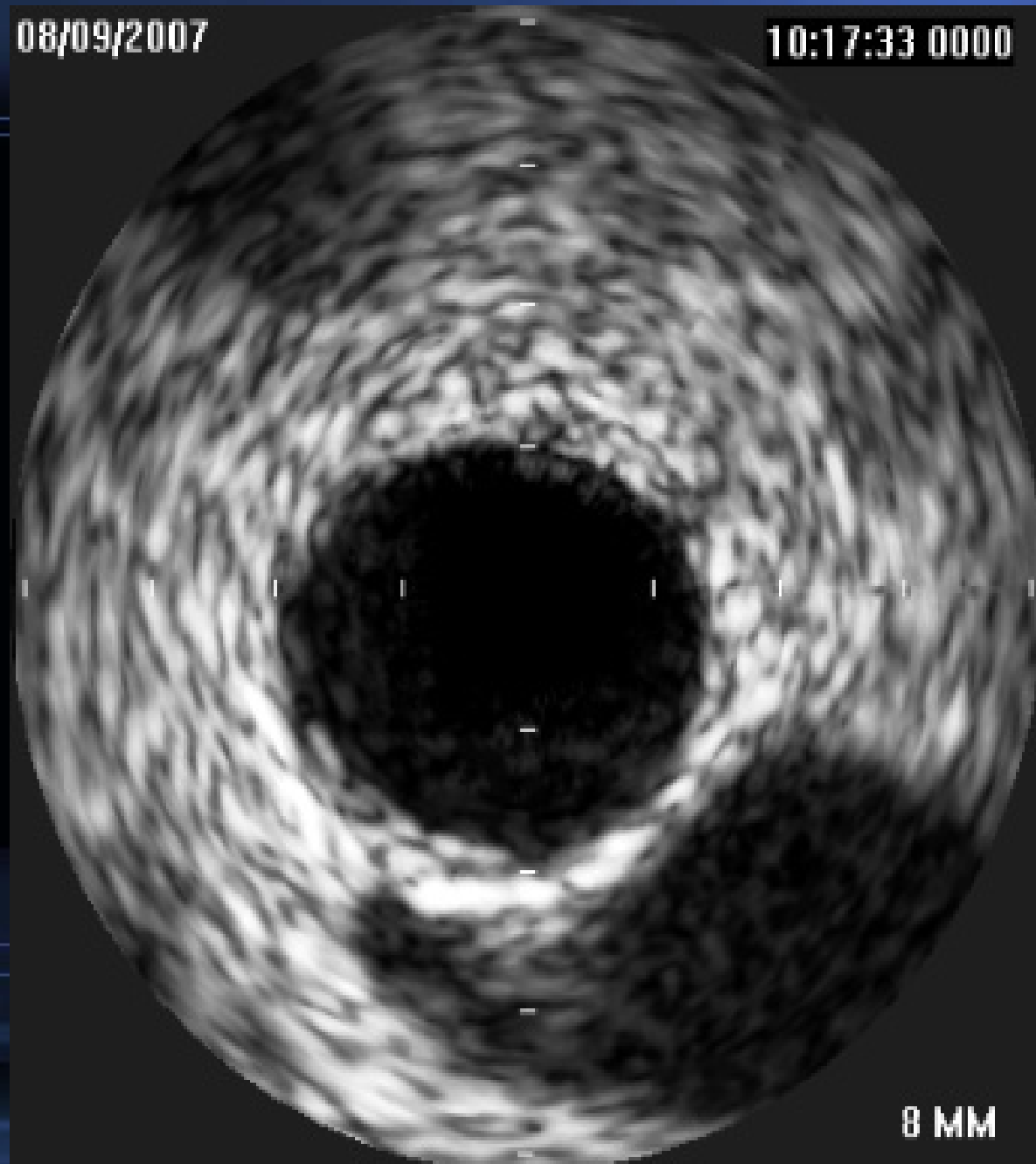
**Department of Cardiology,
Heart Center of Chonnam National University Hospital,
Gwangju, Korea**

74/M NSTEMI, HT, DM, ESRD, s/p PPM (VDD)

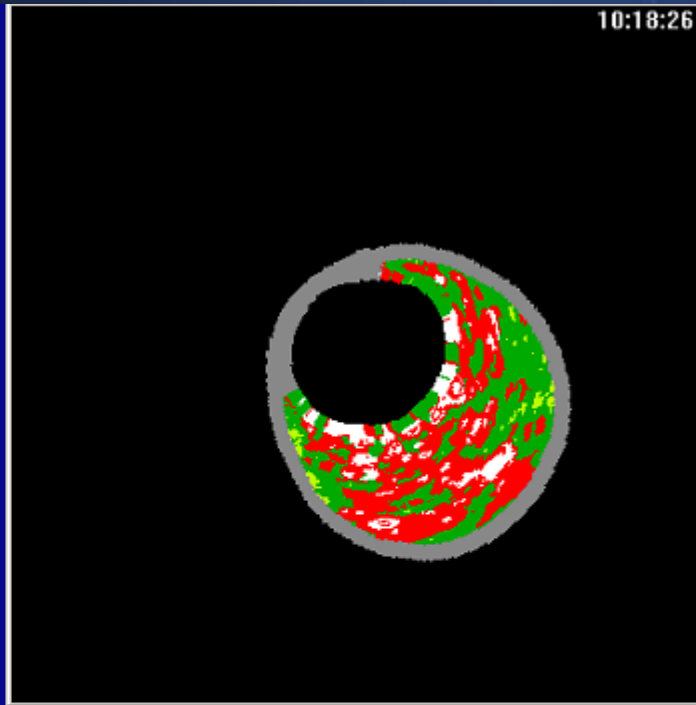
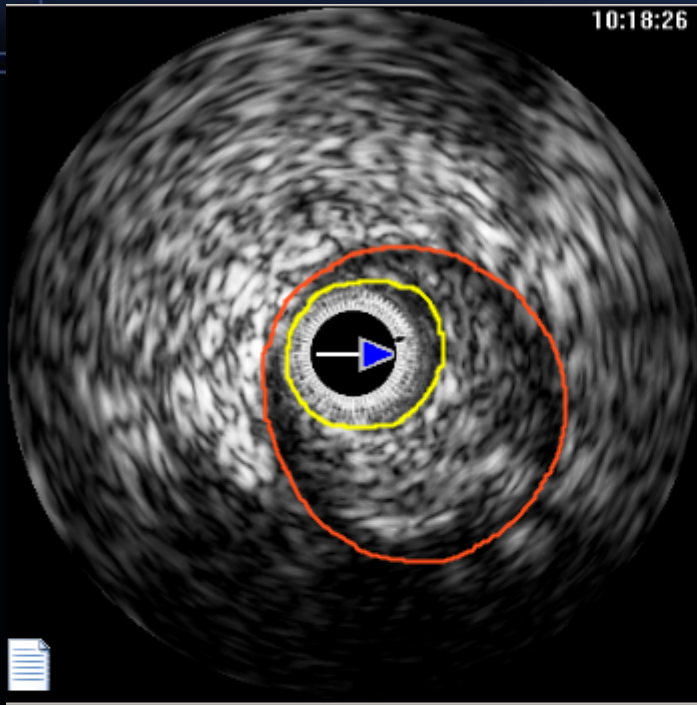


08/09/2007

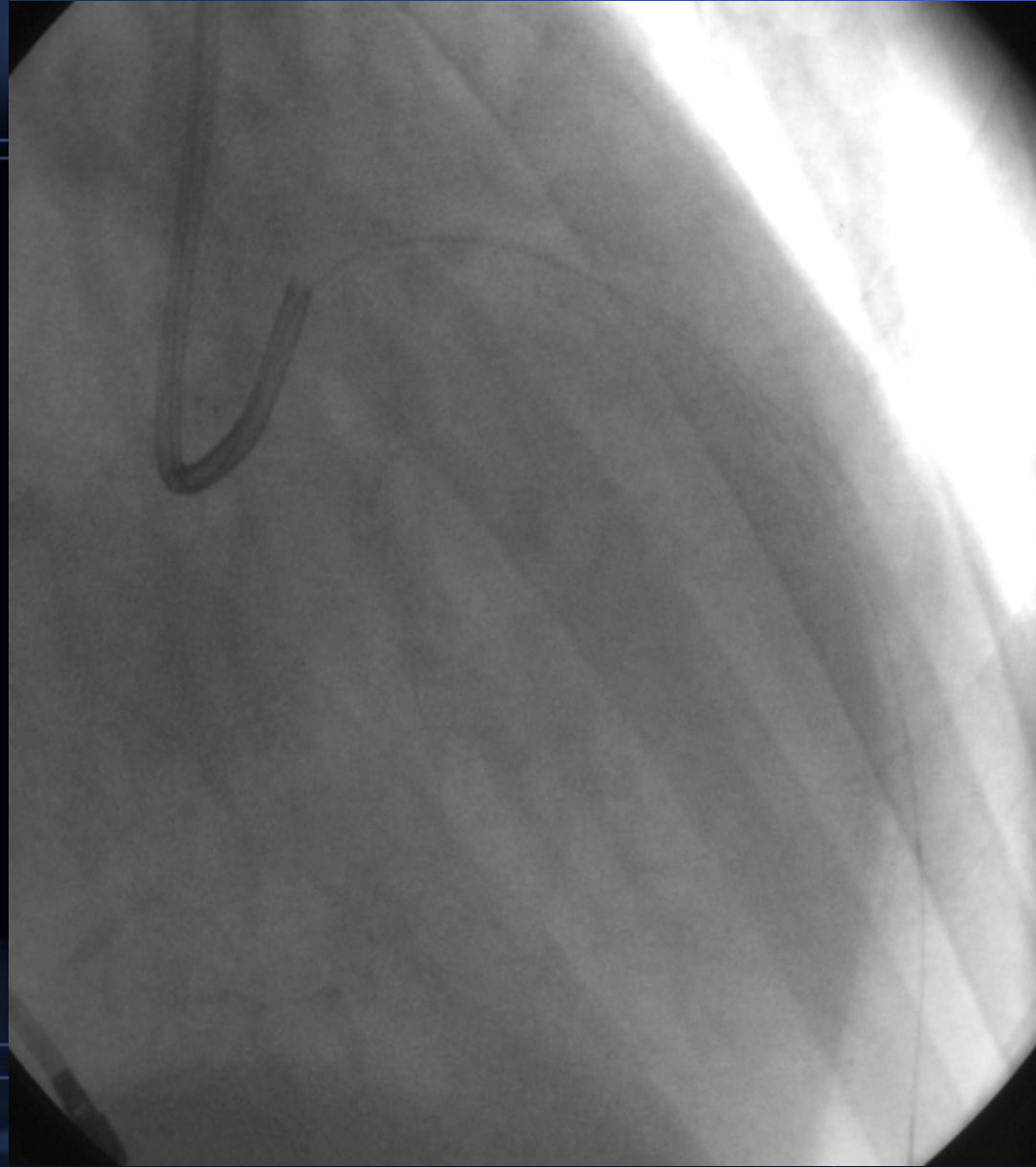
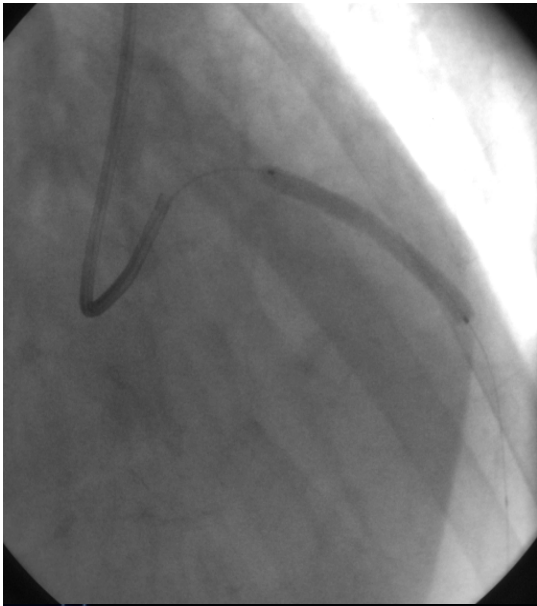
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8 MM



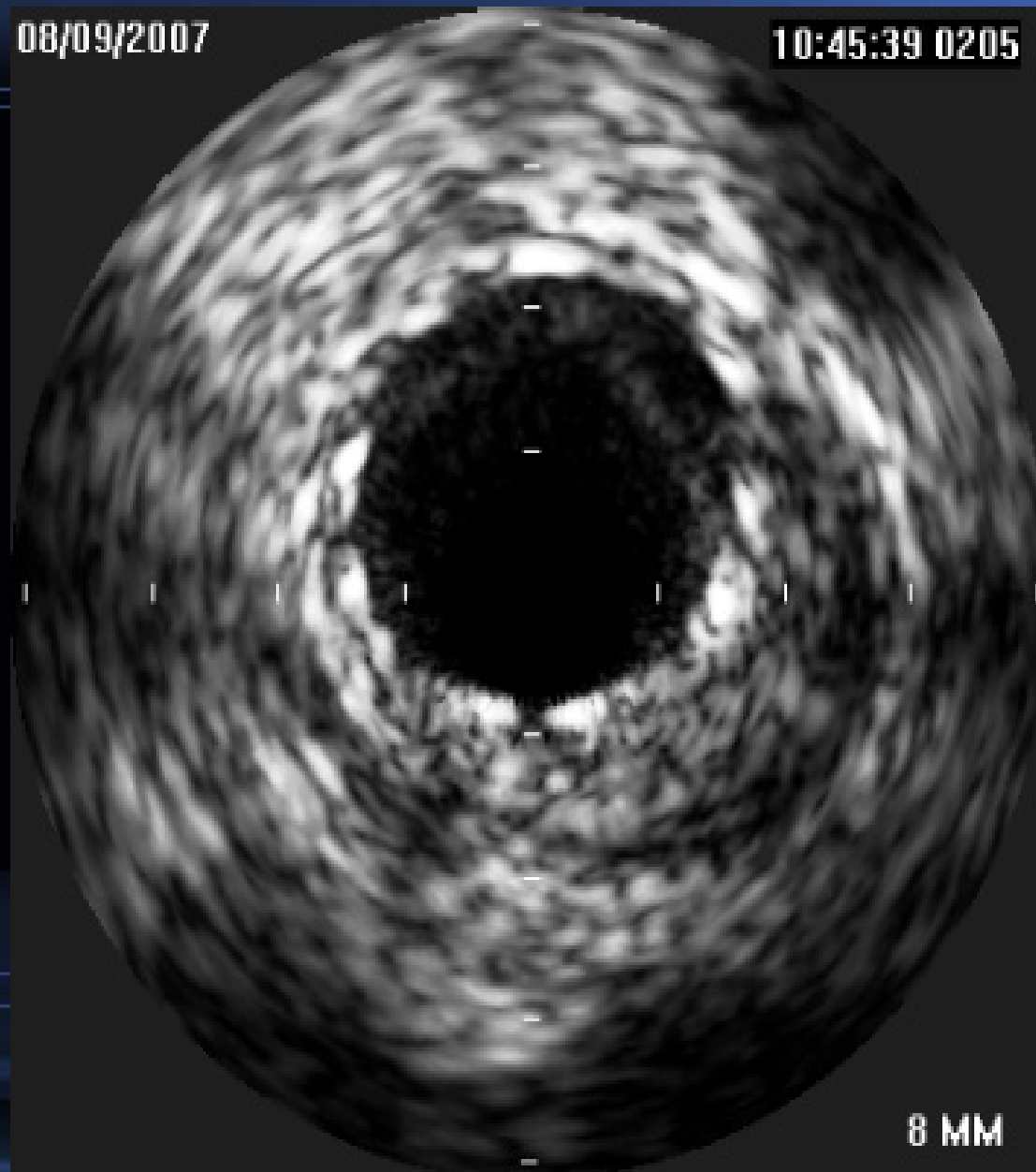
Lumen Area	3.9 mm		More ...
Vessel Area	16.0 mm		
Plaque Area	12.1 mm		
% Plaque Burden	76 %		
FI Green Area	3.7 mm	42 %	
FF Light Green Area	0.2 mm	2 %	
DC White Area	1.0 mm	12 %	
NC Red Area	3.8 mm	44 %	



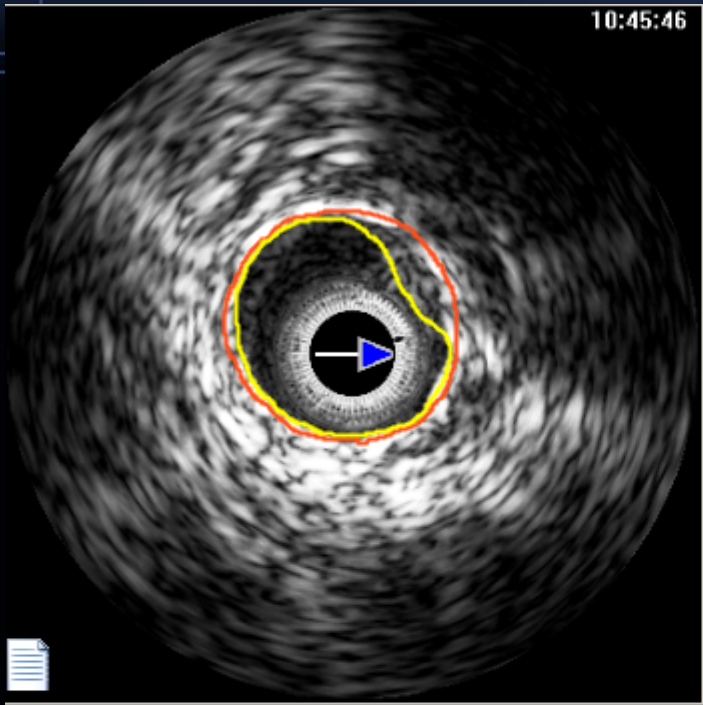
3.5*38mm stent for mLAD at 8atm

08/09/2007

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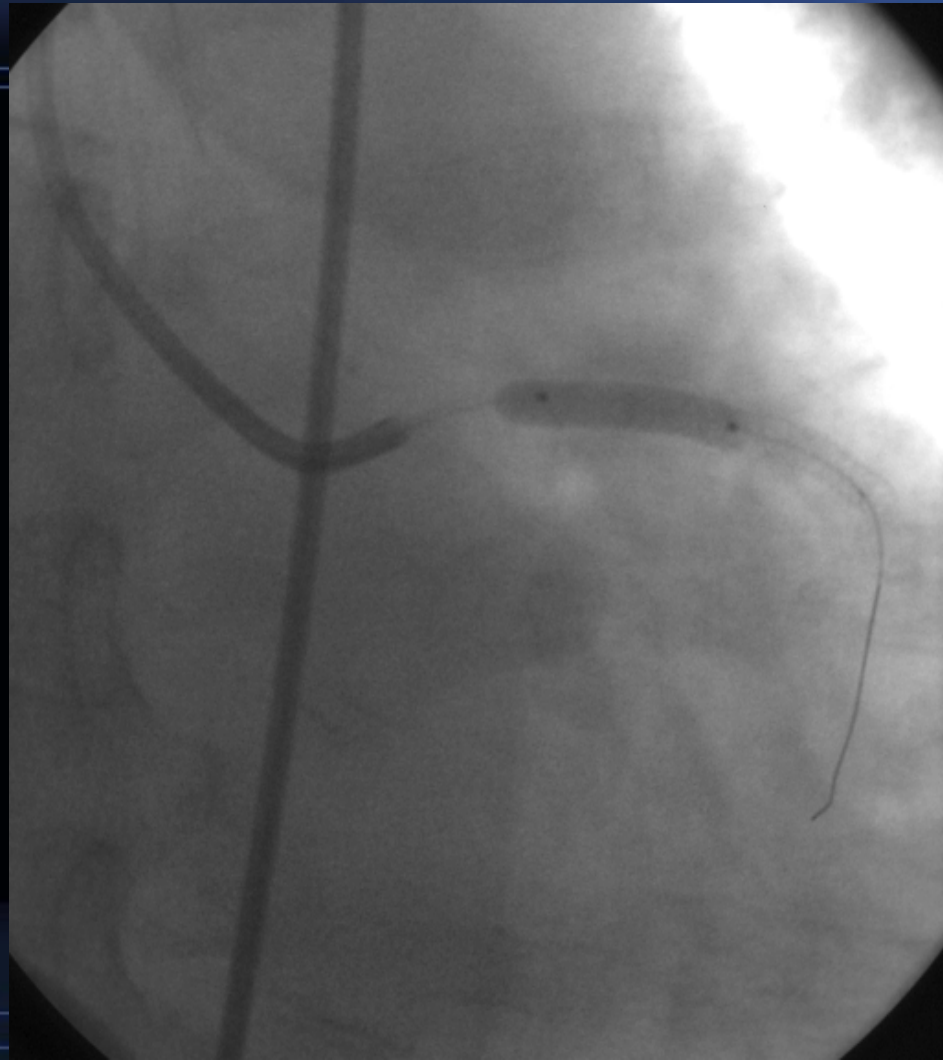


8 MM



Lumen Area	7.2 mm		
Vessel Area	9.2 mm		
Plaque Area	2.1 mm		
% Plaque Burden	22 %		
FI Green Area	0.2 mm	55 %	
FF Light Green Area	0.0 mm	0 %	
DC White Area	0.0 mm	6 %	
NC Red Area	0.2 mm	40 %	

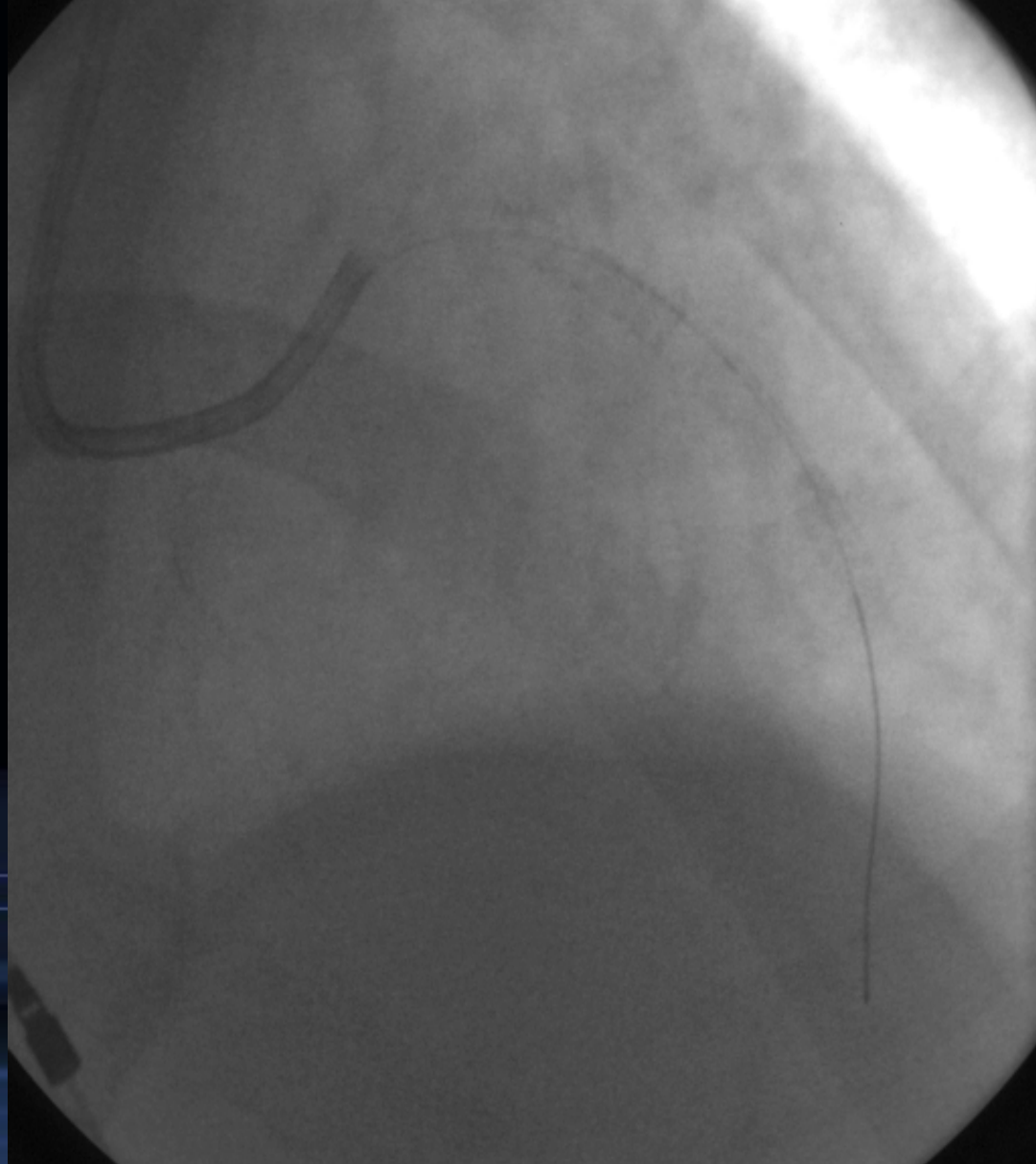
[More ...](#)



3.5*18mm stent for pLAD at 14atm

3.5*18, 3.5*38mm Stent Implantation

No-reflow



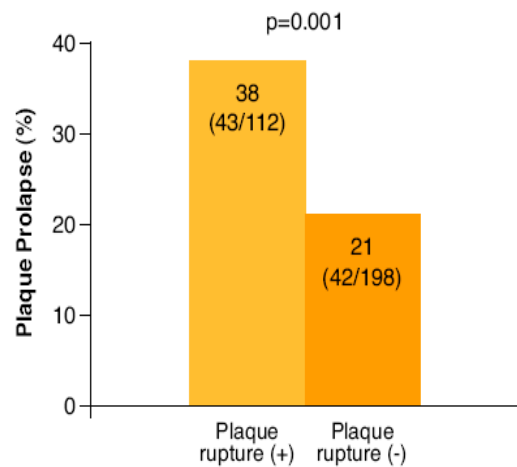


Figure 2. Incidence of PP in Relation to the Presence or Absence of the Plaque Rupture

A ruptured plaque contained a cavity that communicated with the lumen with an overlying residual fibrous cap fragment. A fragmented and loosely adherent plaque without a distinct cavity and without a fibrous cap fragment was not considered a plaque rupture. The PP was observed more frequently in lesions with plaque rupture compared with lesions without plaque rupture.

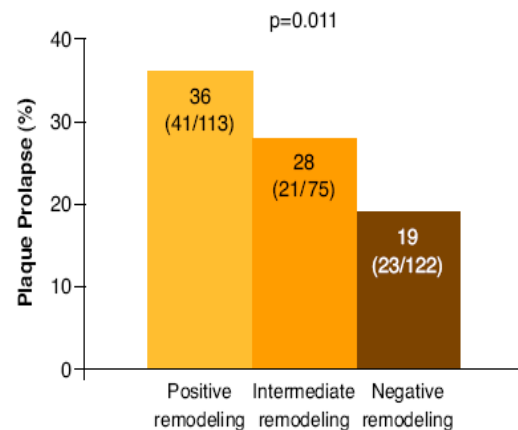


Figure 3. Incidence of PP in Relation to the Remodeling Pattern

Remodeling index was defined as the lesion site external elastic membrane (EEM) cross-sectional area (CSA) divided by the average of the proximal and distal reference EEM CSA. Positive remodeling (n = 113) was defined as a remodeling index >1.05, intermediate remodeling (n = 75) as a remodeling index between 0.95 and 1.05, and negative remodeling (n = 122) as a remodeling index <0.95. The PP was most frequently observed in the group showing positive remodeling compared with the groups showing intermediate or negative remodeling.

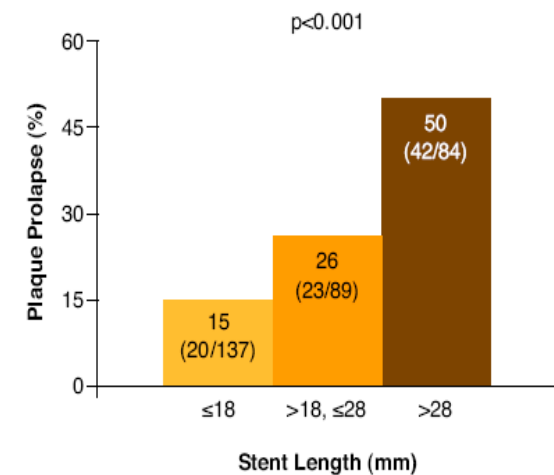


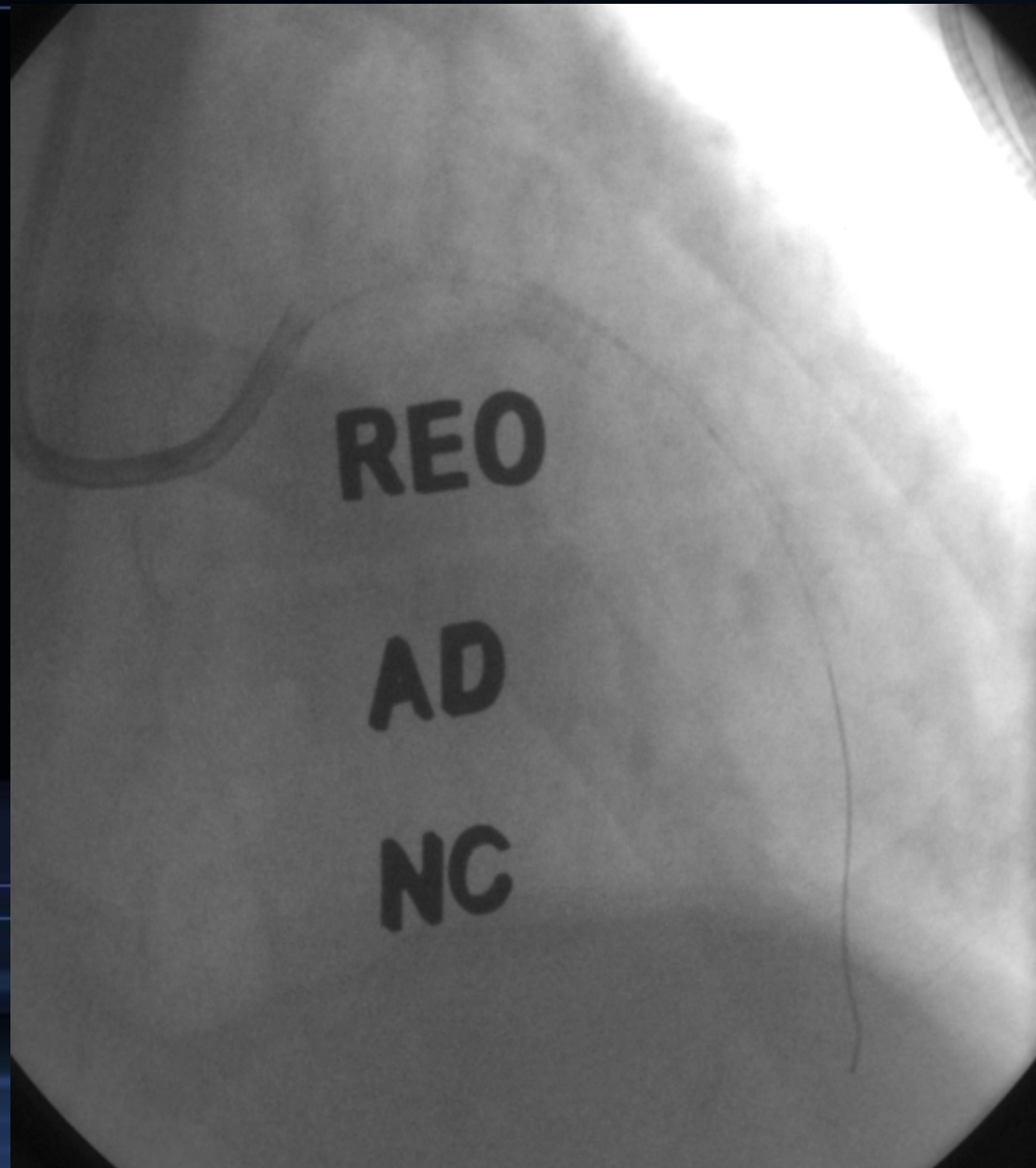
Figure 1. Incidence of PP in Relation to the Stent Length

Stent length was divided into 3 groups such as short stent length group (less than 18 mm, n = 137), intermediate stent length group (between 18 and 28 mm, n = 89), and long stent length group (more than 28 mm, n = 84). Plaque prolapse (PP) was most frequently observed in the group with the long stents (>28 mm) compared with the groups with short- (≤18 mm) or intermediate-length (>18 and ≤28 mm) stents.

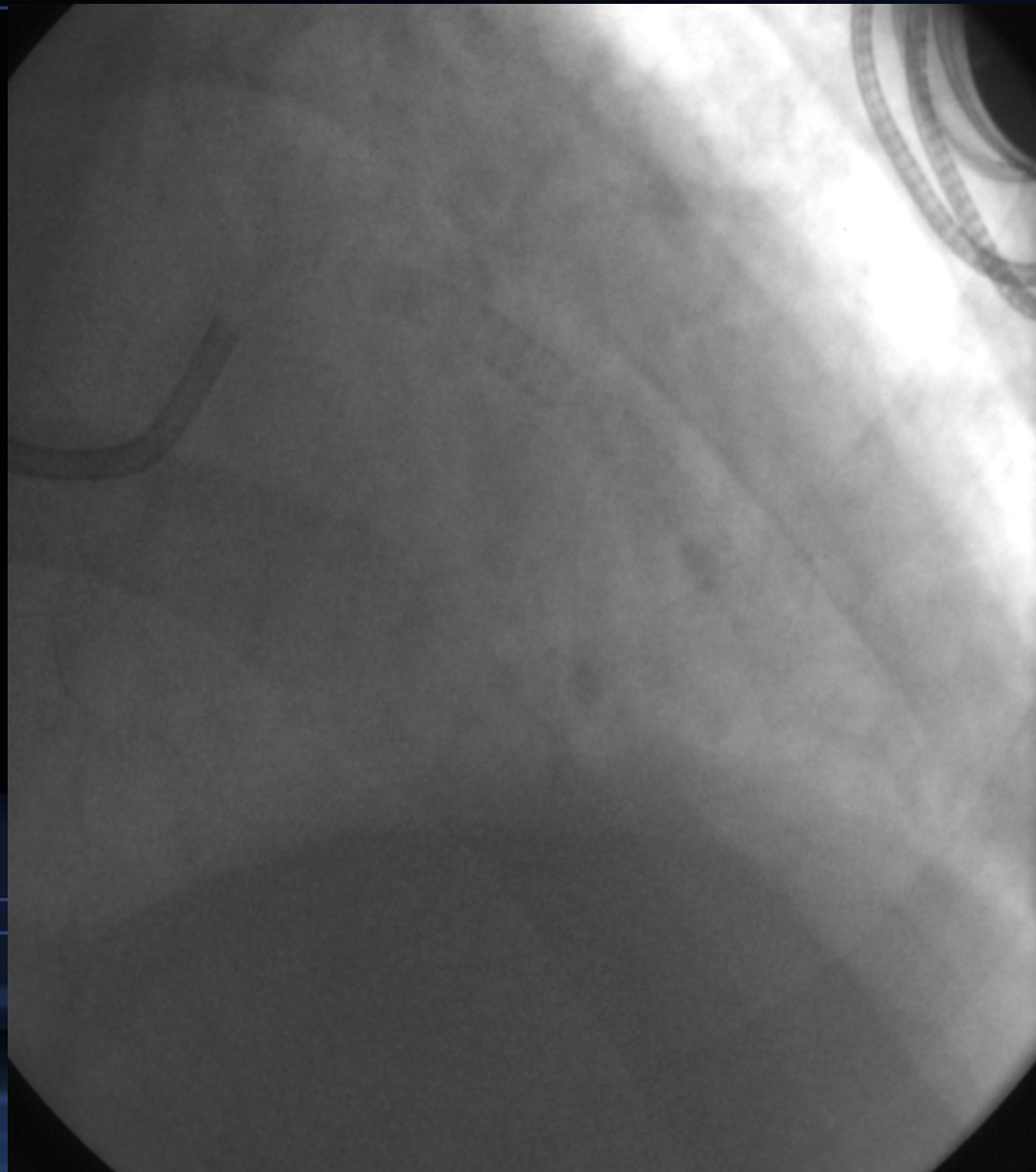
METHODS Intravascular ultrasound (IVUS) imaging was performed in 310 patients immediately following stenting for their first acute myocardial infarction. Multiple clinical, angiographic and IVUS derived variables were compared among patients with and without intrastent PP.

CONCLUSIONS PP occurs in one-fourth of infarct-related arteries after stent implantation. Lesion characteristics such as plaque rupture and positive remodeling, together with longer stent predict PP. Although long-term follow-up is pending, PP is associated with more myonecrosis after stenting in patients with acute myocardial infarction. (J Am Coll Cardiol Img 2008;1:489-97) © 2008 by the American College of Cardiology Foundation

ReoPro, Adenosin, Nicorandil



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European Heart Journal
doi:10.1093/eurheartj/ehp034

CLINICAL RESEARCH

Impact of plaque components on no-reflow phenomenon after stent deployment in patients with acute coronary syndrome: a virtual histology-intravascular ultrasound analysis

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Received 18 October 2008; revised 23 December 2008; accepted 12 January 2009

Aims

We used virtual histology-intravascular ultrasound (VH-IVUS) to evaluate the relation between coronary plaque characteristics and no-reflow in acute coronary syndrome (ACS) patients.

Methods and results

A total of 190 consecutive ACS patients were imaged using VH-IVUS and analysed retrospectively. Angiographic no-reflow was defined as TIMI flow grade 0, 1, and 2 after stenting. Virtual histology-intravascular ultrasound classified the colour-coded tissue into four major components: fibrotic, fibro-fatty, dense calcium, and necrotic core (NC). Thin-cap fibroatheroma (TCFA) was defined as focal, NC-rich ($\geq 10\%$ of the cross-sectional area) plaques being in contact with the lumen in a plaque burden $\geq 40\%$. Of the 190 patients studied at pre-stenting, no-reflow was observed in 24 patients (12.6%) at post-stenting. The absolute and %NC areas at the minimum lumen sites (1.6 ± 1.2 vs. 0.9 ± 0.8 mm², $P < 0.001$, and 24.5 ± 14.3 vs. $16.1 \pm 10.6\%$, $P = 0.001$, respectively) and the absolute and %NC volumes (30 ± 24 vs. 16 ± 17 mm³, $P = 0.001$, and 22 ± 11 vs. $14 \pm 8\%$, $P < 0.001$, respectively) were significantly greater, and the presence of at least one TCFA and multiple TCFA within culprit lesions (71 vs. 36%, $P = 0.001$, and 38 vs. 15%, $P = 0.005$, respectively) was significantly more common in the no-reflow group compared with the normal-reflow group. In the multivariable analysis, %NC volume was the only independent predictor of no-reflow (odds ratio = 1.126; 95% CI 1.045–1.214, $P = 0.002$).

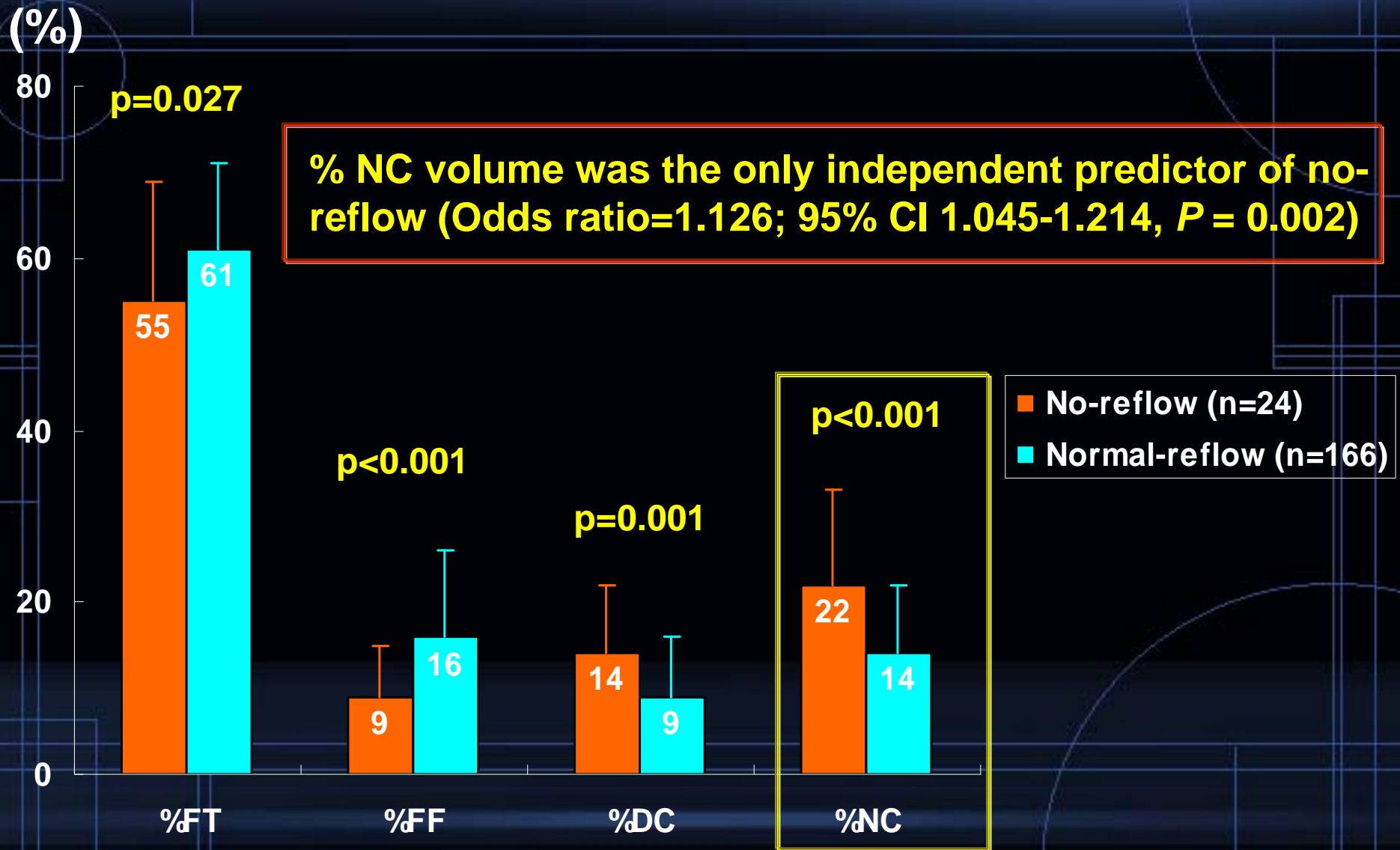
Conclusion

In ACS patients, post-stenting no-reflow is associated with plaque components defined by VH-IVUS analysis with larger NC and more TCFA.

Keywords

Coronary disease • Stents • Plaque • Ultrasonics

Plaque Component and No-Reflow



[JACC Cardiovasc Imaging. 2009 Apr;2\(4\):458-68.](#)

Impact of plaque composition on cardiac troponin elevation after percutaneous coronary intervention: an ultrasound analysis.

[Hong YJ](#), [Mintz GS](#), [Kim SW](#), [Lee SY](#), [Okabe T](#), [Pichard AD](#), [Sattler LF](#), [Waksman R](#), [Kent KM](#), [Suddath WO](#), [Weissman NJ](#).

Cardiovascular Research Institute/Medstar Research Institute, Washington Hospital Center, Washington, DC 20010, USA.

Comment in:

[JACC Cardiovasc Imaging. 2009 Apr;2\(4\):469-72.](#)

Abstract

OBJECTIVES: We used virtual histology-intravascular ultrasound (VH-IVUS) to study the relationship between pre-percutaneous coronary intervention (PCI) coronary plaque characteristics and post-PCI cardiac troponin I (cTnI) elevation.

BACKGROUND: Percutaneous coronary intervention is often complicated by post-procedural myocardial necrosis as manifested by elevated cardiac markers.

METHODS: Eighty consecutive patients (29 stable and 51 unstable angina) with normal pre-PCI cTnI levels were imaged before PCI using VH-IVUS. Patients were divided into 2 groups according to the presence (Group I, n = 38) or absence (Group II, n = 42) of post-PCI cTnI elevation \geq or =3x the upper limit of normal (0.08 ng/ml).

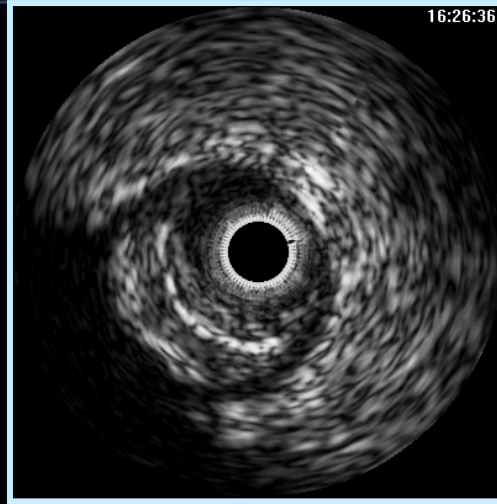
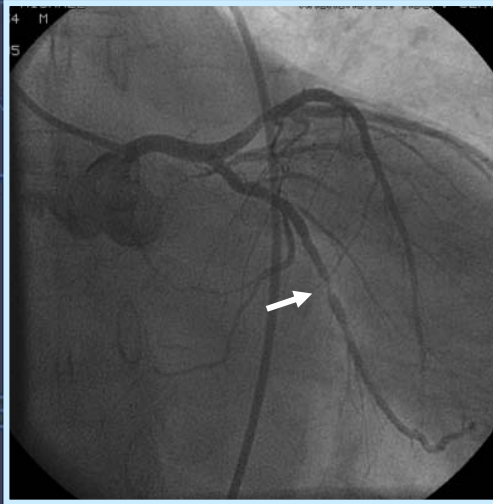
RESULTS: The absolute and percent necrotic core volumes were significantly greater in Group I than in Group II (13.6 \pm 6.4 mm³ vs. 7.9 \pm 4.4 mm³, p < 0.001, and 19.8 \pm 10.4% vs. 12.8 \pm 8.4%, p = 0.015, respectively). The absolute and percent necrotic core areas were significantly greater in Group I than in Group II at the minimum lumen site (1.70 \pm 0.91 mm² vs. 0.61 \pm 0.39 mm², p < 0.001, and 22.9 \pm 11.7% vs. 10.4 \pm 6.6%, p < 0.001, respectively) and at the largest necrotic core site (2.00 \pm 0.86 mm² vs. 0.81 \pm 0.78 mm², p < 0.001, and 24.0 \pm 11.7% vs. 12.9 \pm 6.6%, p < 0.001, respectively). The Δ cTnI correlated with: 1) absolute and percent necrotic core area at the minimum lumen site and at the largest necrotic core site; 2) absolute necrotic core volume; 3) percent fibrofatty area at the minimum lumen site; and 4) lesion site plaque burden. In the multivariate analysis, absolute necrotic core area at the minimum lumen site was the only independent predictor of post-PCI cTnI elevation \geq or =3x the upper limit of normal (odds ratio: 1.318; 95% confidence interval: 1.090 to 1.594, p = 0.004).

CONCLUSIONS: The VH-IVUS analysis shows that post-PCI cTnI elevation occurs in lesions with a large necrotic core area. The VH-IVUS may play an important role in detecting which lesions are high risks for myocardial necrosis after PCI.

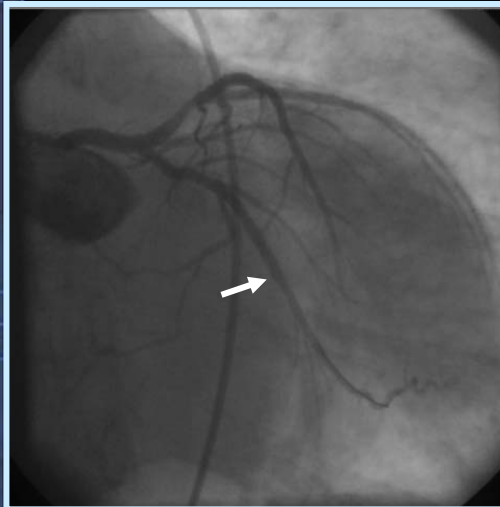
PMID: 19580729 [PubMed - indexed for MEDLINE]

Plaque Component and Tnl Elevation

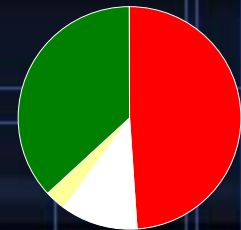
Pre-PCI (cTnl=0ng/ml)



Cypher (cTnl=3.24ng/ml)

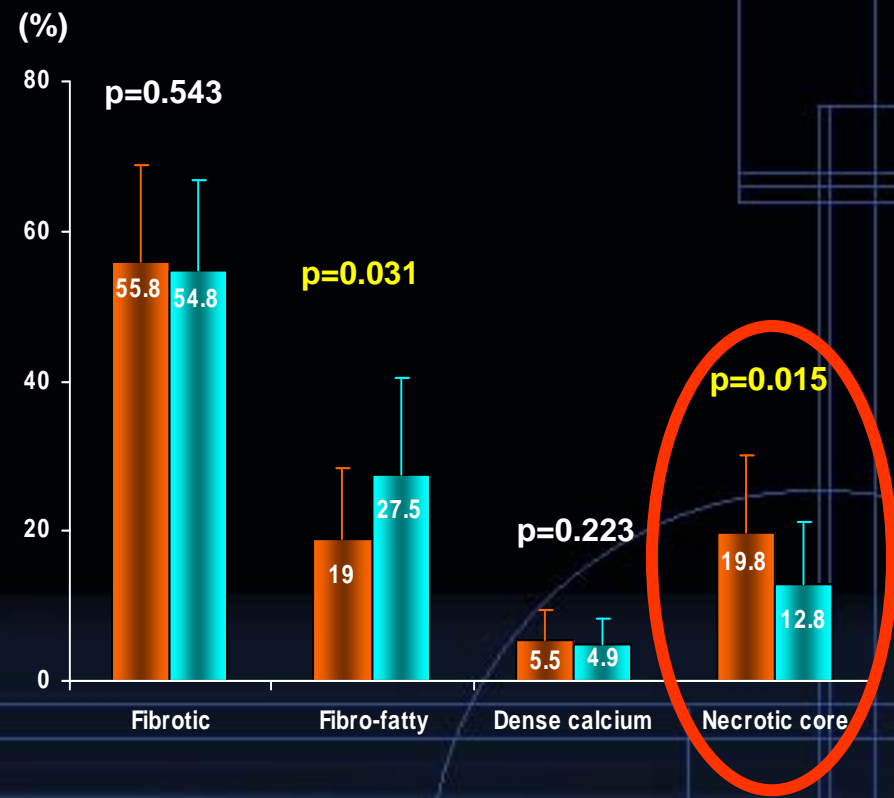
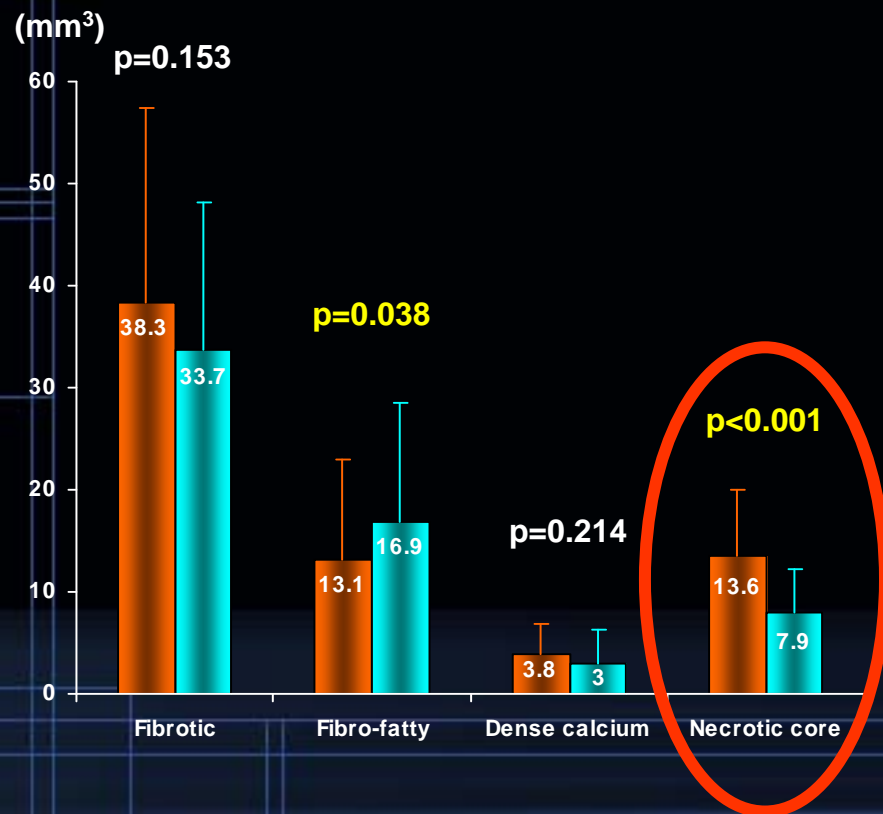


Lumen Area	3.8 mm ²	
EEL Area	16.0 mm ²	
Plaque Area	12.2 mm ²	
% Plaque Burden	76%	
Fibrous Area	3.7 mm²	37%
Fibro-Fatty Area	0.3 mm²	3%
Dense Calcium Area	1.1 mm ²	11%
Necrotic Core Area	5.0 mm²	49%



Plaque Component and Tnl Elevation

■ cTnl elevation $\geq 3X$
■ cTnl elevation $< 3X$





Relation Between Plaque Components and Plaque Prolapse After Drug-Eluting Stent Implantation – Virtual Histology–Intravascular Ultrasound –

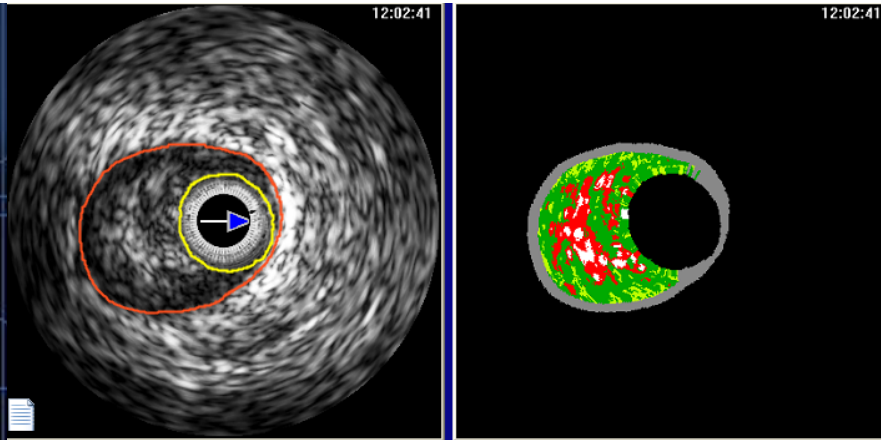
Young Joon Hong, MD; Myung Ho Jeong, MD; Sang Wook Kim, MD*; Yun Ha Choi;
Eun Hae Ma; Jum Suk Ko, MD; Min Goo Lee, MD; Keun Ho Park, MD; Doo Sun Sim, MD;
Nam Sik Yoon, MD; Hyun Ju Yoon, MD; Kye Hun Kim, MD; Hyung Wook Park, MD;
Ju Han Kim, MD; Youngkeun Ahn, MD; Jeong Gwan Cho, MD;
Jong Chun Park, MD; Jung Chae Kang, MD

Background: It is not well known which plaque components are associated with the development of plaque prolapse (PP) and what are the major components in prolapsed plaque. The relationship between pre-stenting plaque components and post-stenting PP was assessed and the plaque components of prolapsed plaque were evaluated in patients who underwent drug-eluting stent (DES) implantation using virtual histology–intravascular ultrasound (VH-IVUS).

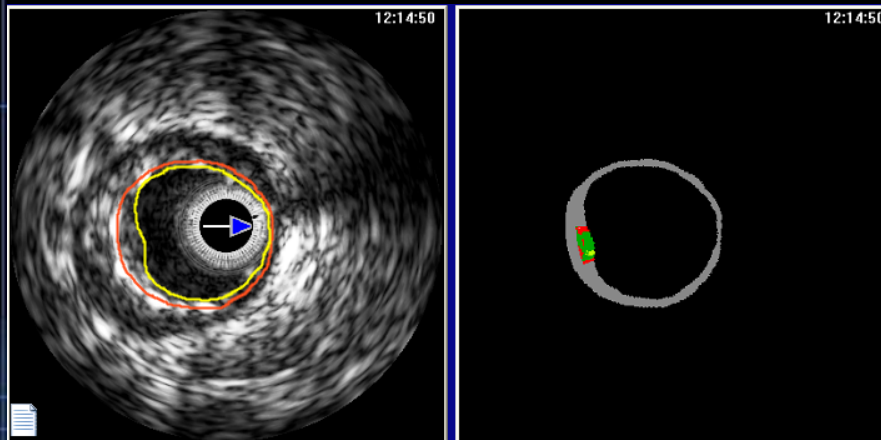
Methods and Results: The study group consisted of 132 patients who underwent DES implantation and pre- and post-stenting VH-IVUS. Of these patients, 68 patients had 76 PP lesions and 64 patients had 76 non-PP lesions. Intra-stent PP volume was $3.6 \pm 1.5 \text{ mm}^3$. Plaque volume was significantly greater and absolute fibrotic (FT) and necrotic core (NC) volumes were significantly greater in PP lesions compared with non-PP lesions. On multivariate analysis, absolute NC (odds ratios [OR]=1.14, $P < 0.001$) and FT volume (OR=1.09, $P < 0.001$) were independently associated with the development of PP. In intra-stent prolapsed plaque the FT component was greatest, but the NC component was also large, and %NC volume correlated positively with Δ creatinine kinase-MB ($r=0.489$, $P < 0.001$) and Δ troponin-I ($r=0.679$, $P < 0.001$), and %FT volume correlated negatively with Δ CK-MB ($r=-0.539$, $P < 0.001$) and Δ troponin-I.

Conclusions: NC and FT components were associated with development of PP; and NC and FT components in prolapsed plaque were associated with cardiac enzyme elevation after DES implantation. (*Circ J* 2010; 74: 1142–1151)

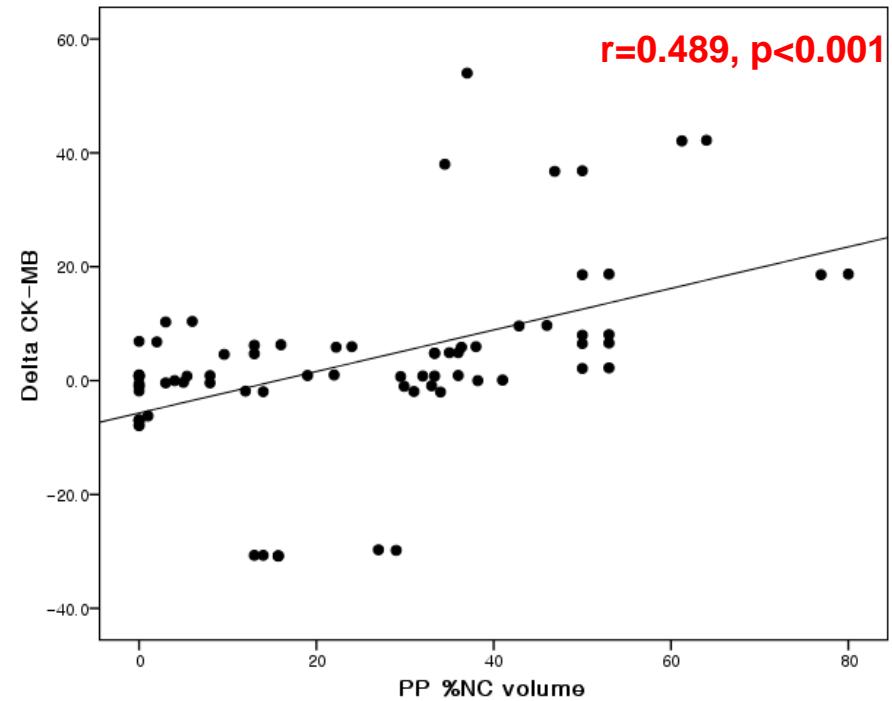
Key Words: Atherosclerosis; Coronary disease; Intravascular ultrasound; Stent



Lumen Area	3.9 mm ²		
Vessel Area	15.0 mm ²		
Plaque Area	11.1 mm ²		
% Plaque Burden	74 %		
FI Green Area	4.6 mm ²	58 %	
FF Light Green Area	0.9 mm ²	11 %	
DC White Area	0.5 mm ²	7 %	
NC Red Area	1.9 mm ²	24 %	



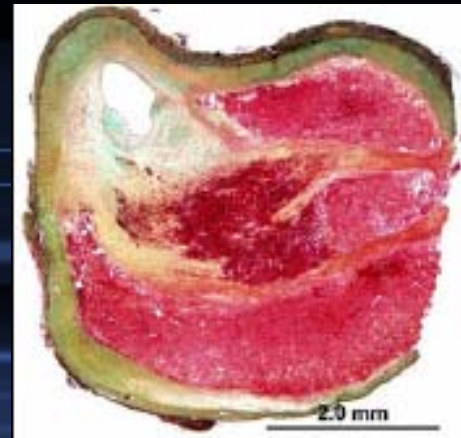
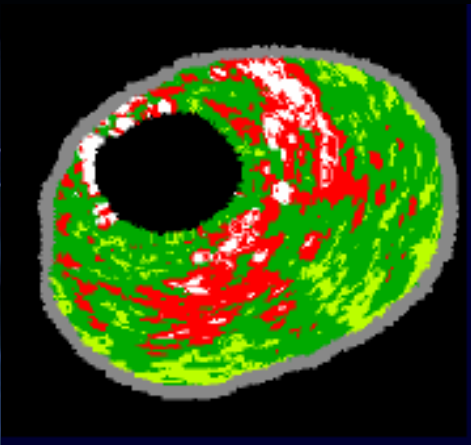
Lumen Area	7.7 mm ²		
Vessel Area	10.2 mm ²		
Plaque Area	2.6 mm ²		
% Plaque Burden	25 %		
FI Green Area	0.2 mm ²	66 %	
FF Light Green Area	0.0 mm ²	7 %	
DC White Area	0.0 mm ²	0 %	
NC Red Area	0.1 mm ²	27 %	



Plaque Component, Plaque Prolapse, Post-PCI CK-MB Elevation

Could Necrotic Core be the Determinant Factor We Should Target Our Attention?

- What is **necrotic core**?
 - It is plaque composition filled with intracellular lipid, often inflammatory cells, and has no matrix
 - It is the predominant composition with or without Ca found in plaques which cause a SCD and a thrombotic event



Conclusions

Plaque rupture

Multiple Plaque rupture

Thrombus

Positive remodeling

Necrotic core

Thin-cap fibroatheroma



Plaque prolapse

No-reflow

Cardiac enzyme





2010 GICS

8th Gwangju Interventional Cardiology Symposium

Program 1. Pre - Live Symposium Program

Date: June 11th (Fri) 2:00 PM, 2010
Venue: Kinnadaeung Convention Center
Moderator: Doo Sun Sim, MD

14:00 - 14:40	Session I New Cardiovascular Imaging Chairman: Young Joon Hong, MD Panelist: Jeong Woo Park, MD; Joong Woo Chung, MD; Dong Goo Kang, MD; Sang Rok Lee, MD; Won Yu Kang, MD; Keun Ho Park, MD Lecture: Microemboli and Microvascular Obstruction in Acute Coronary Thrombolysis and Sudden Coronary Death: Rupture of Non-Thin Cap Fibroatheroma in Color Lesions of Acute ST Elevation Myocardial Infarction: A Virtual Histology Intravascular Ultrasound Analysis Moderator: Sang Wook Kim, MD Panelist: Sung Yun Lee, MD
16:00 - 16:30	Coffee Break
16:30 - 17:30	Session II One stage vs. staged procedure in STEMI Chairman: Young Joon Hong, MD Panelist: Hun Sik Park, MD; Young Youp Koh, MD; Jang Hyun Cho, MD; Jei Keon Chae, MD; Younkeun Ahn, MD Lecture: One stage procedure Moderator: Young Joon Hong, MD Panelist: Young Sun Park, MD
17:30 - 18:30	Session III Transfemoral vs. Transradial approach in STEMI Chairman: Ki Bae Seung, MD; Young Ho Jeong, MD Panelist: Jang Cheol Park, MD; Jae Young Rhew, MD; Seung Uk Lee, MD; Seok Kyu Oh, MD; Ju Han Kim, MD Lecture: Transfemoral approach Moderator: Myoung Ki Hong, MD Panelist: Kwang Soo Cha, MD
18:30 -	Dinner

Program 2. Live Demonstration

Date: June 12th (Sat), 2010
Venue: Myung Hak Grand Auditorium of Chonnam National University Medical School Heart Center of Chonnam National University Hospital
Moderator: Doo Sun Sim, MD

08:00-08:30	Registration	Opening Remark: Jung Chae Kang, MD (Chonnam National University Hospital)
08:30-09:30	Case #1-2 Chairman: Dong Ju Oh, MD; Myoung Ho Jeong, MD Operator #1: Ki Bae Seung, MD; Younkeun Ahn, MD; Ju Han Kim, MD Operator #2: Tae Hoon Ahn, MD; Jang Hyun Cho, MD; Sun Ho Hwang, MD Panelist: Sang Wook Kim, MD; Joon Woo Kim, MD; Jang Cheol Park, MD; Taek Keun Ohn, MD; Chaeil Woong Chai, MD; Myung Woo Park, MD IVUS commentator: Young Joon Hong, MD	
09:30-10:00	Lecture 1	Left Atrial Appendage Closure: Local Interventional Therapy for Atrial Fibrillation and Stroke Robert S Schwartz, MD
10:00-11:00	Case #3-4 Chairman: Chung Jen Wu, MD; Sang Uk Lee, MD Operator #3: Won Heum Sim, MD; Woon Kim, MD; Kyung Hee Yun, MD Operator #4: Seung Jae Tahk, MD; Seok Kyu Oh, MD; Sang Rok Lee, MD Panelist: Young Youp Koh, MD; Jei Keon Chae, MD; Jang Ho Bae, MD; Woong Chol Kang, MD; Young Hoon Jeong, MD; Sun Ho Hwang, MD IVUS commentator: Sung Yun Lee, MD	
11:00-11:30	Lecture 2	Ta Be antistomat Naoto Inoue, MD
11:30-12:30	Case #5-7 Chairman: Myoung Ki Hong, MD; Jae Young Rhew, MD; Soo Hyun Kim, MD Operator #5: Naoto Inoue, MD; Jei Keon Chae, MD; Dong Goo Kang, MD Operator #6: Sung Ho Kim, MD; Ju Han Kim, MD; Young Kuk Cho, MD Operator #7: Dong Haeon Choi, MD; Kwang Soo Cha, MD; Jeong Woo Chung, MD; Jung Sun Kim, MD; Sang Yup Lim, MD; Jae Yeon Moon, MD Panelist: Jang Ho Bae, MD	
12:30-13:30	Luncheon Symposium Chairman: Jung Chae Kang, MD; Jee Ki Ahn, MD Lecture: Ta Be antistomat Real world experience of SES in complex cases (Japan Experience) Sung Tae Yoon, MD Resolute-ALL Covered Trial at EuroPCR 2010 Takashi Shiono, MD Chairman: Chung Jen Wu, MD	
13:30-14:30	Case #8-10 Chairman: Young Joon Hong, MD; Myoung Ho Jeong, MD Operator #8: Jung Han Yoon, MD; Young Youp Koh, MD; Won Yu Kang, MD Operator #9: Takashi Shiono, MD; Seung Uk Lee, MD; Shin Eun Lee, MD Operator #10: Naoto Inoue, MD; Youngkrun Ahn, MD; Young Joon Hong, MD Panelist: Hun Sik Park, MD; Young Hoon Jeong, MD; Chaeil Woong Chai, MD; Sung Yun Lee, MD; Young Hoon Jeong, MD; Kwang Soo Cha, MD; Young Sun Park, MD IVUS commentator: Sung Yun Lee, MD	
14:00-14:30	Video case presentation Chairman: Young Joon Hong, MD Presenter: Anterior wall STEMI: Sang Rok Lee, MD; Inferior wall STEMI: Dong Goo Kang, MD Panelist: Jung Sun Park, MD; Kwang Soo Cha, MD; Sang Yup Lim, MD; Woong Chol Kang, MD; Jang Cheol Park, MD; Young Hoon Jeong, MD; Young Sun Park, MD	
14:30-	Closing remark	Myoung Ho Jeong, MD

Thank You For Your Attention!