



Detection and Treatment of Thin Fibrous Cap, Lipid Rich and Inflammatory Vulnerable Plaque – Angioscopic Evaluation

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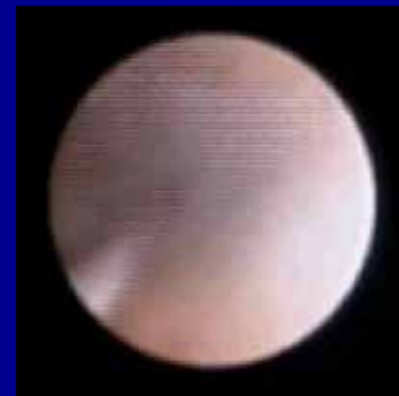
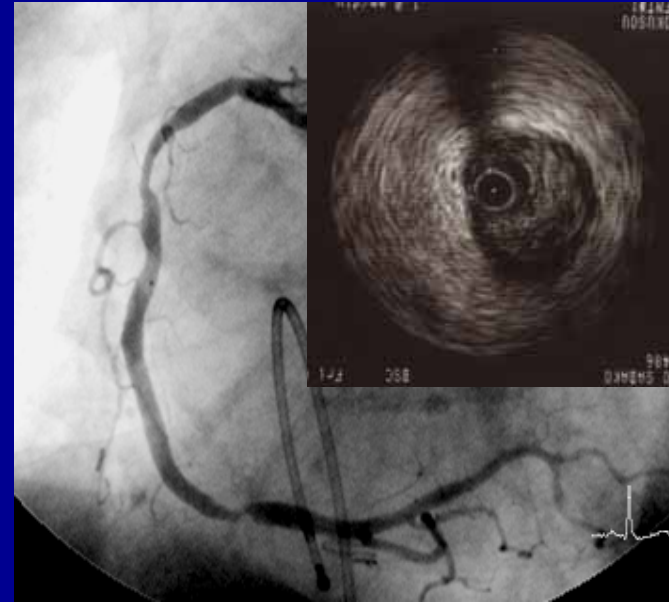
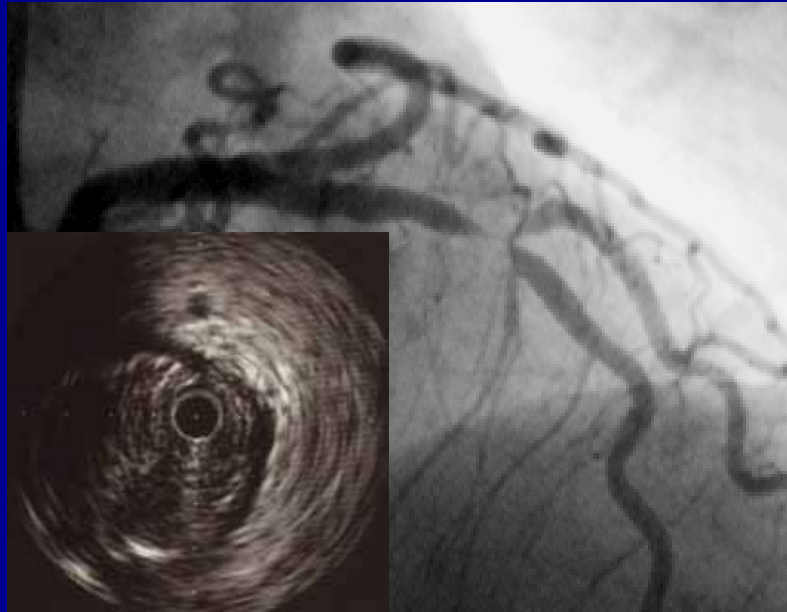
**~Characteristics of
coronary angiography~**

1. full color

2. high resolution

3. three dimension

Coronary angiography, IVUS and angioscopy



Today's headline



1. Angioscopic classification

2. Comparison between angioscopic
macromorphology and microscopic
histopathology

3. Treatment of vulnerable plaque

Statin

Coronary intervention

~Classification of angioscopic findings~

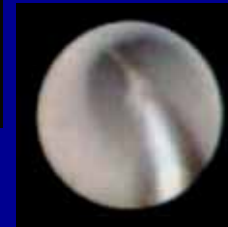
◆Plaque

color

yellow



white



shape

smooth

complex

rupture
flap,
irregularity (erosion)



◆Thrombus

color

red



white

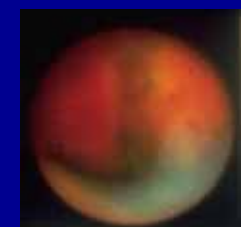


shape

occlusive

non-occlusive

mural(lining)
intraluminal



Today's headline



1. Angioscopic classification

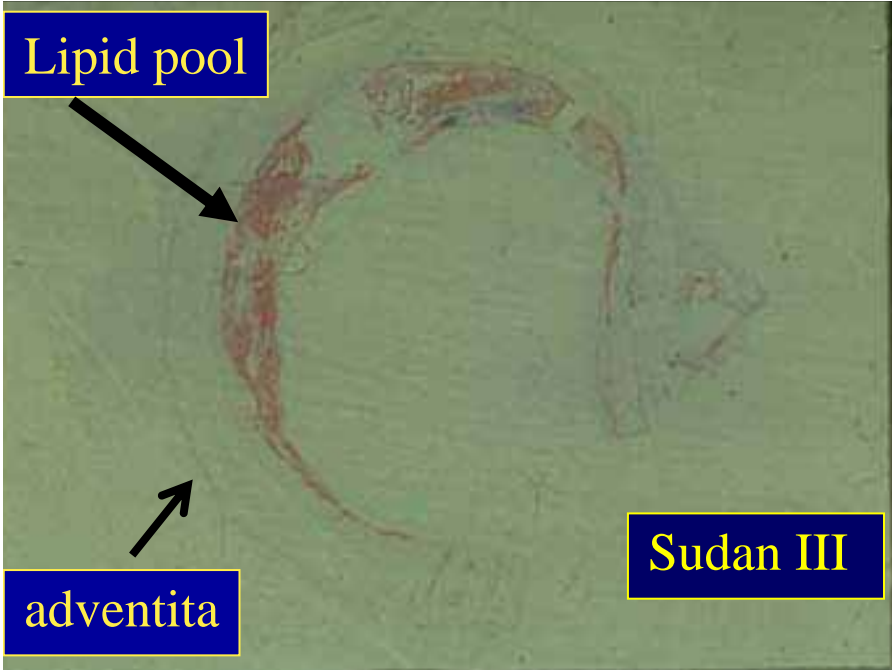
2. Comparison between angioscopic

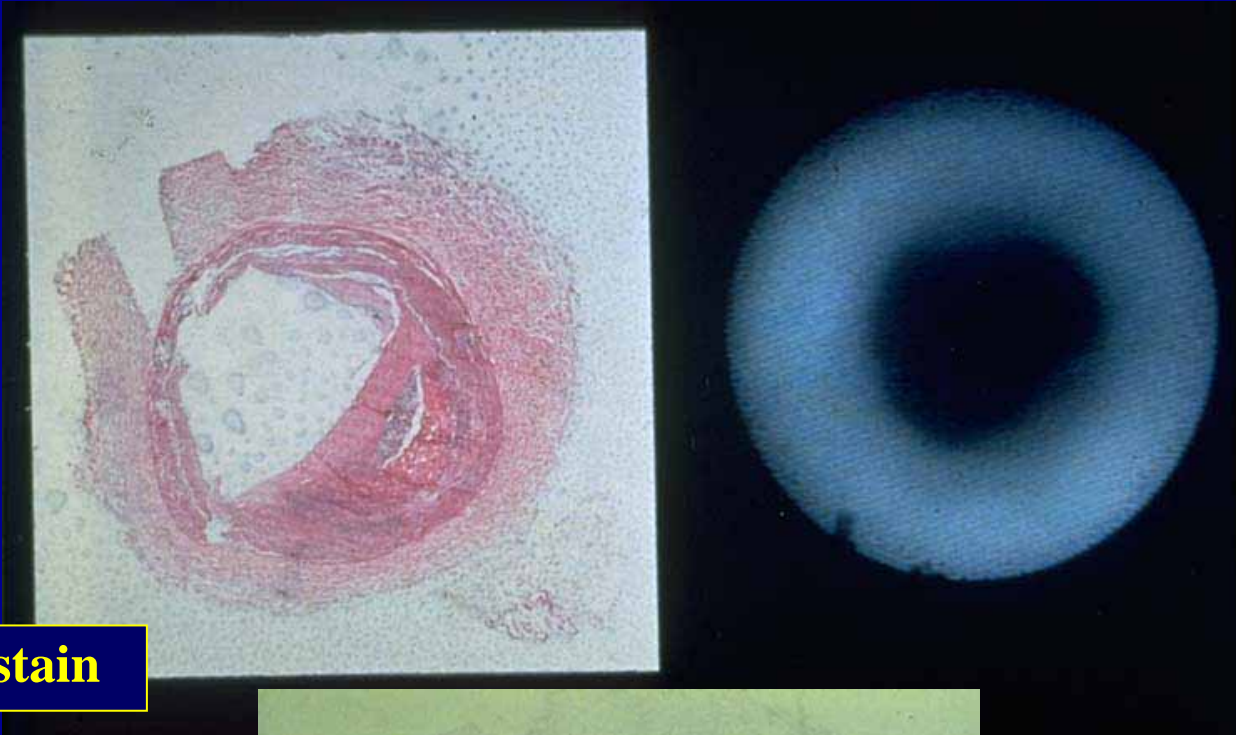
macromorphology and microscopic
histopathology

3. Treatment of vulnerable plaque

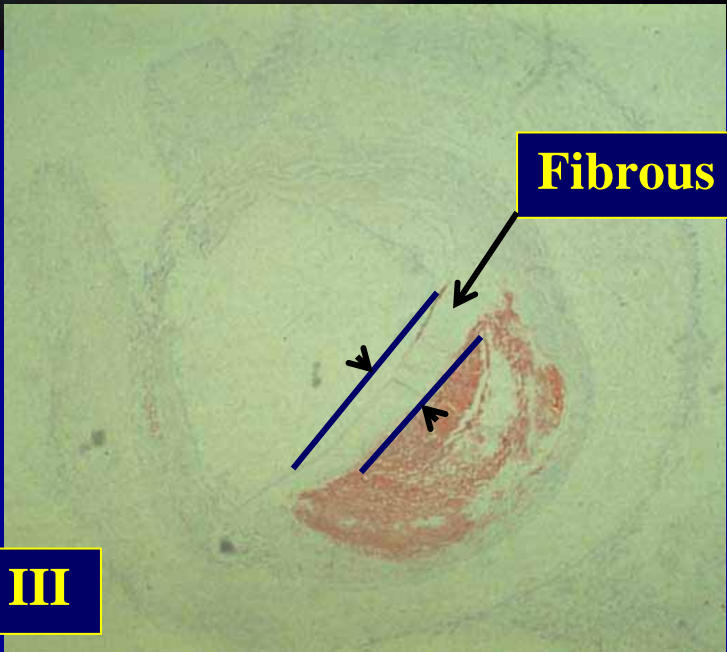
Statin

Coronary intervention





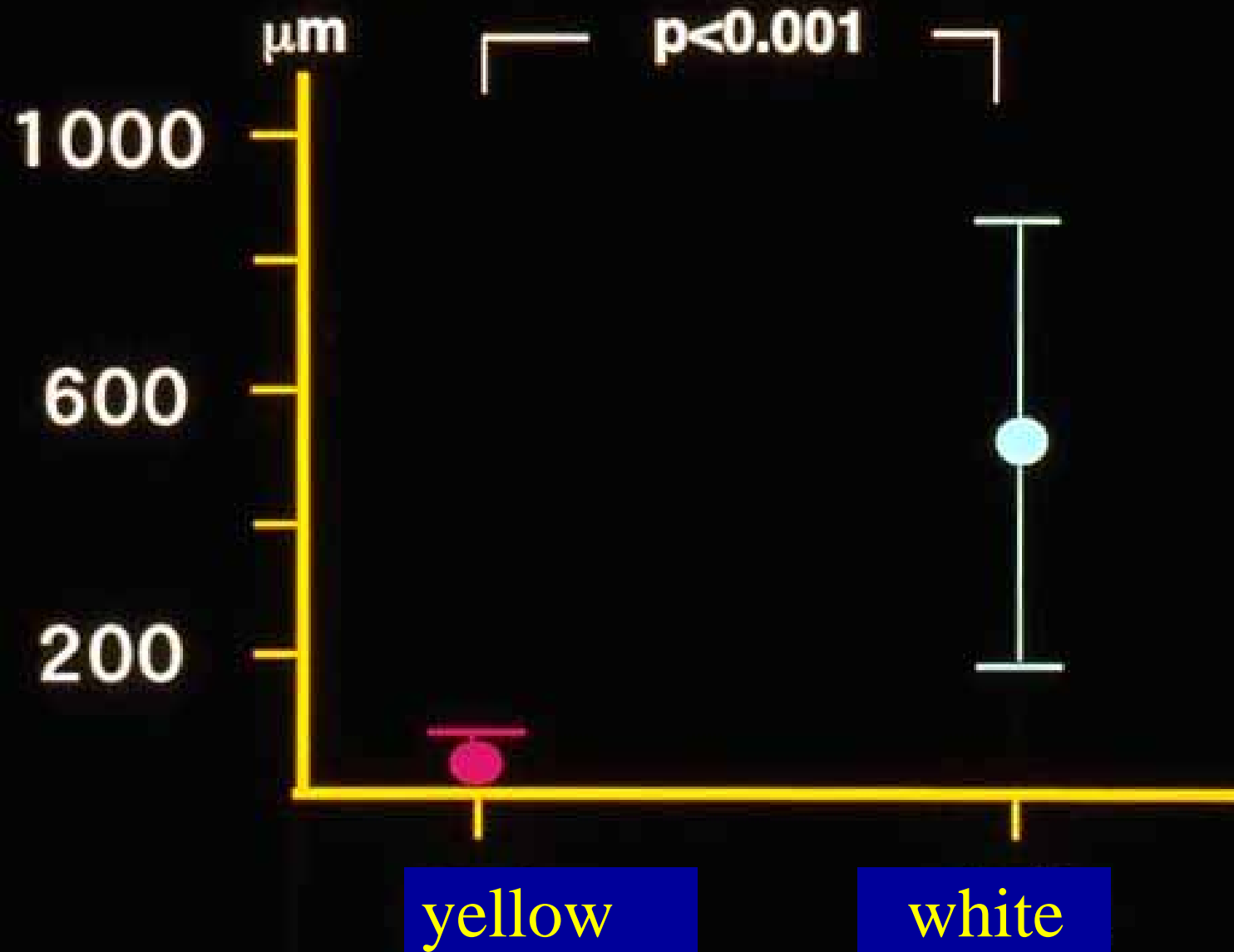
H—E stain



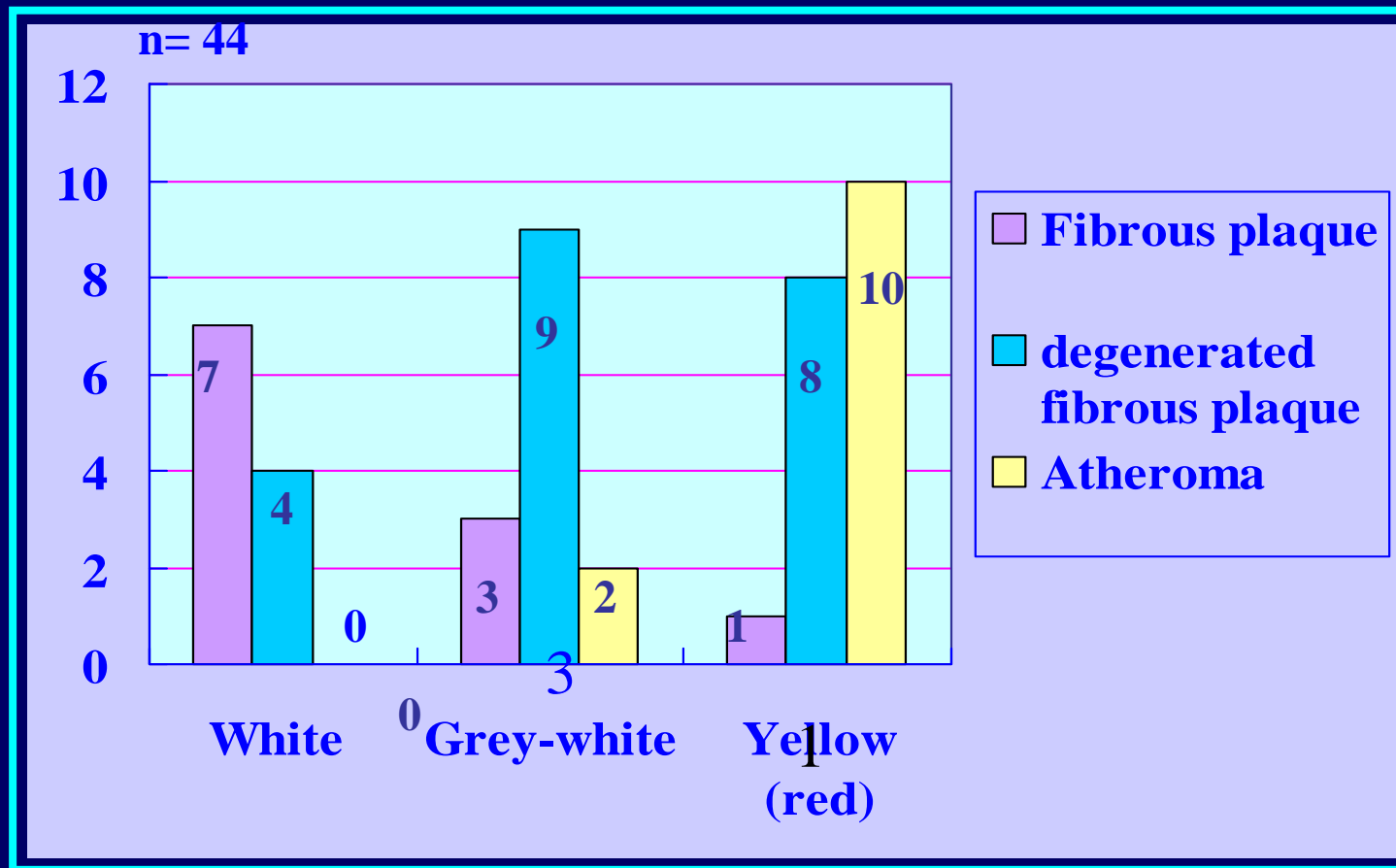
Fibrous cap

Sudan III

Thickness of fibrous cap

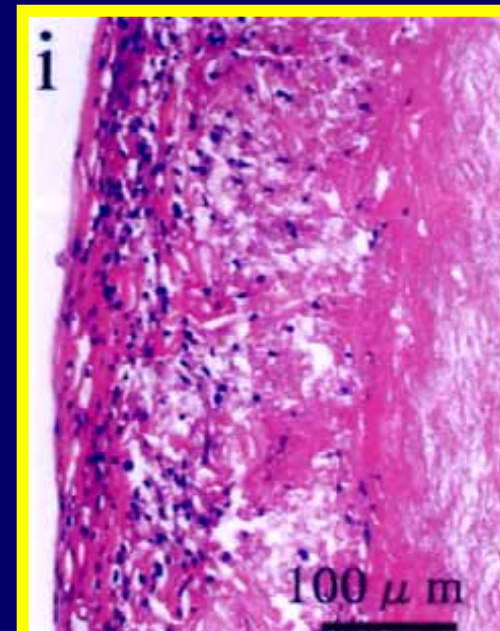
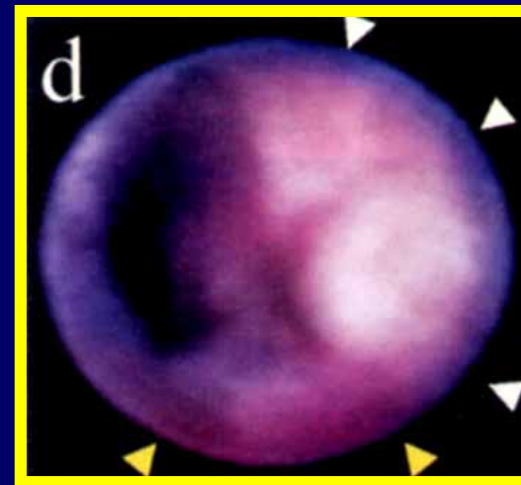


Comparison between angioscopic plaque color and histomorphology entitles of 44 atherectomy Specimens

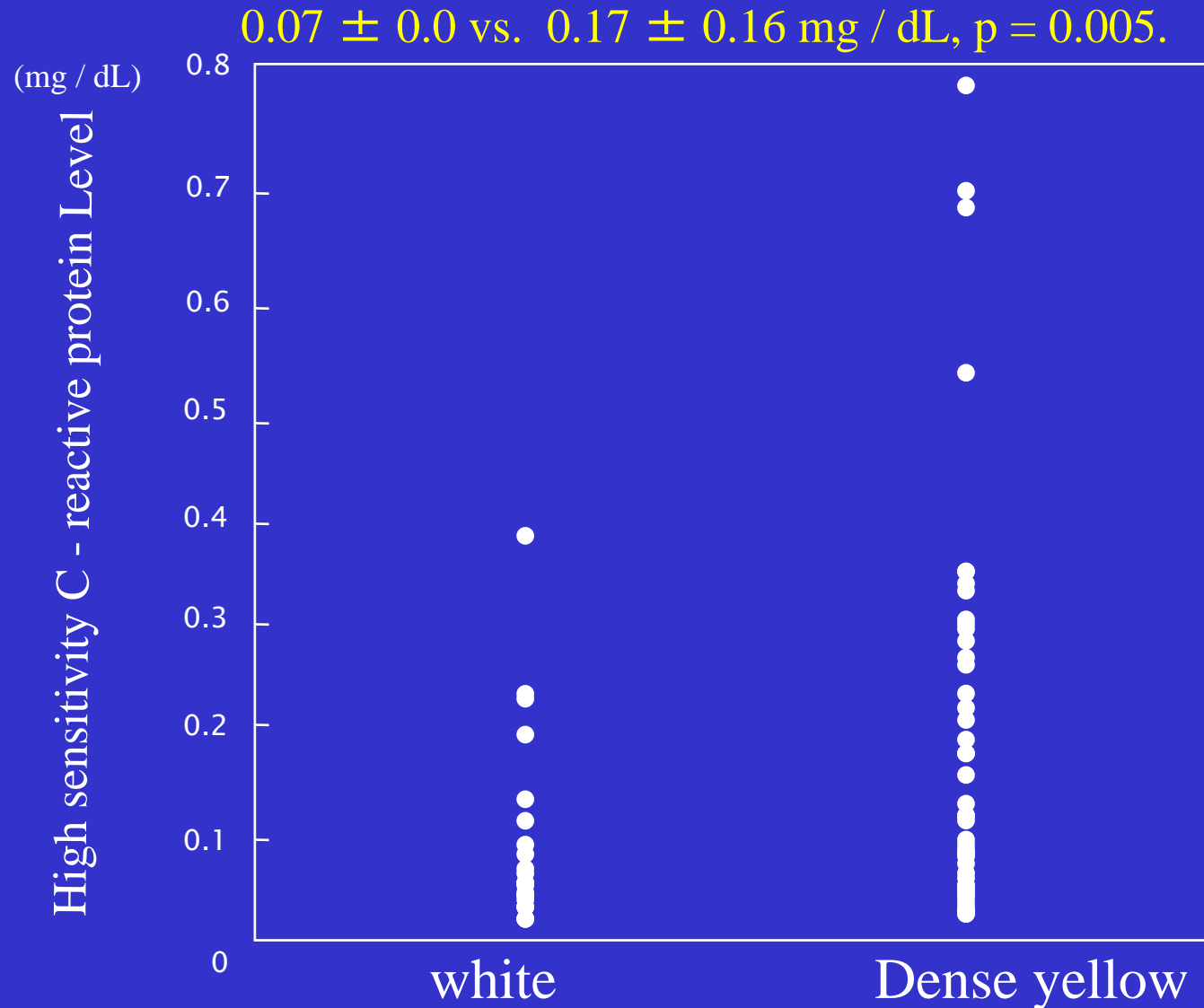


Angioscopic surface color

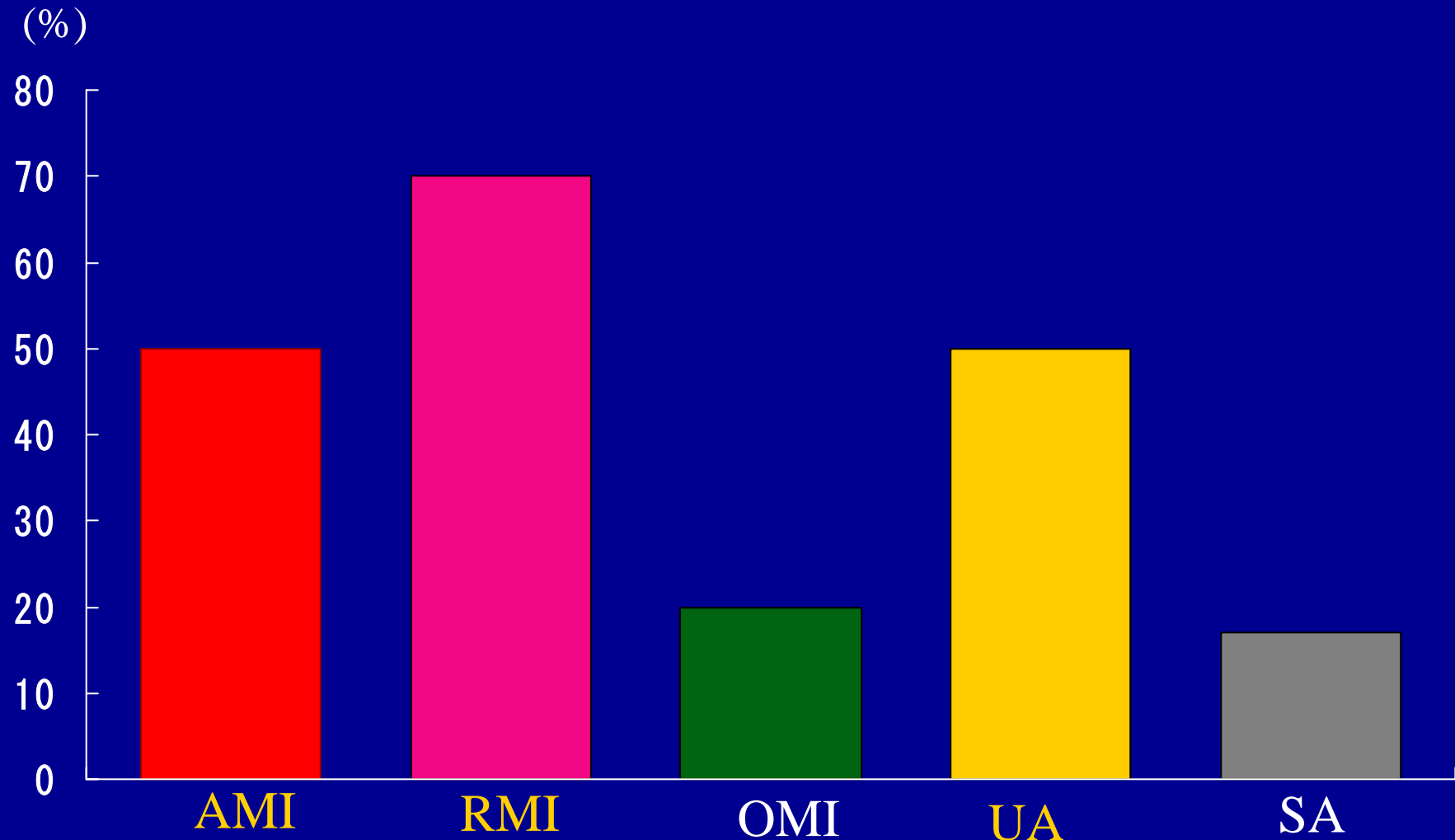
Yellow plaque and macrophage



Comparison between hs CRP levels and Plaque color



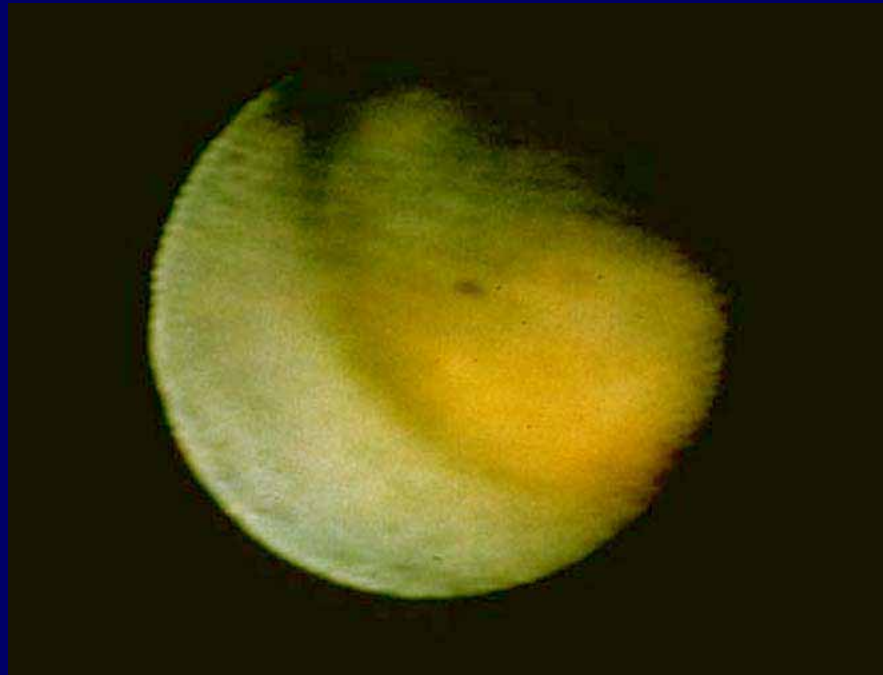
The frequency of yellow plaque



Mizuno K, et al. Lancet, 337:809,1991

yellow

white



Fibrous cap

thin

thick

Inflammation

+

—

Lipid pool

large

small

Vulnerable

Stable

Today's headline



1. Angioscopic classification
2. Comparison between angioscopic macromorphology and microscopic histopathology
3. Treatment of vulnerable plaque
 - Statin
 - Coronary intervention

Background



There have been no reliable methods to examine the plaque stabilization in living patients.

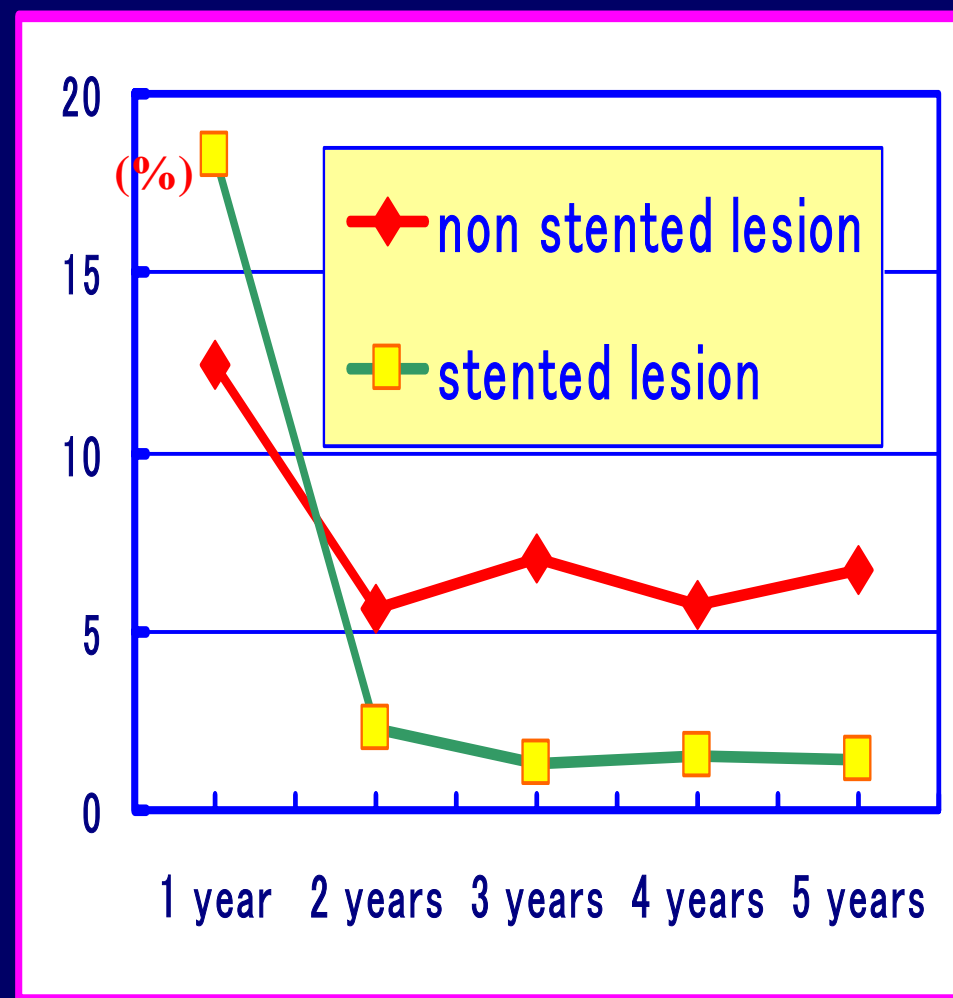
Color and morphology of coronary plaque are regarded as determinant factor of plaque stability and instability.

Landmark Intervention

Trials using Statins

	Primary	Secondary
High LDL	WOSCOPS (192, 159, -26%) (-22%, -31%)	4S (188, 122, 35%) (-30%, -34%)
Moderate ~ Average LDL	AFCAPS/EXCAPS (156, 115, 25%) (0%, -37%)	CARE (139, 98, 32%) (-9%, -24%) LIPID (150, 112,

Hazard rate per year for target (stented) lesion and non target (non stented) lesion after stenting



Hypothesis

Lipid lowering therapy and stenting

induce plaque stabilization

Aim



Investigation of the effect of statin and stenting on plaque stabilization using coronary angioscopy.

Today's headline



1. Angioscopic classification
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3. Treatment of vulnerable plaque

Statin

Coronary intervention

Objective

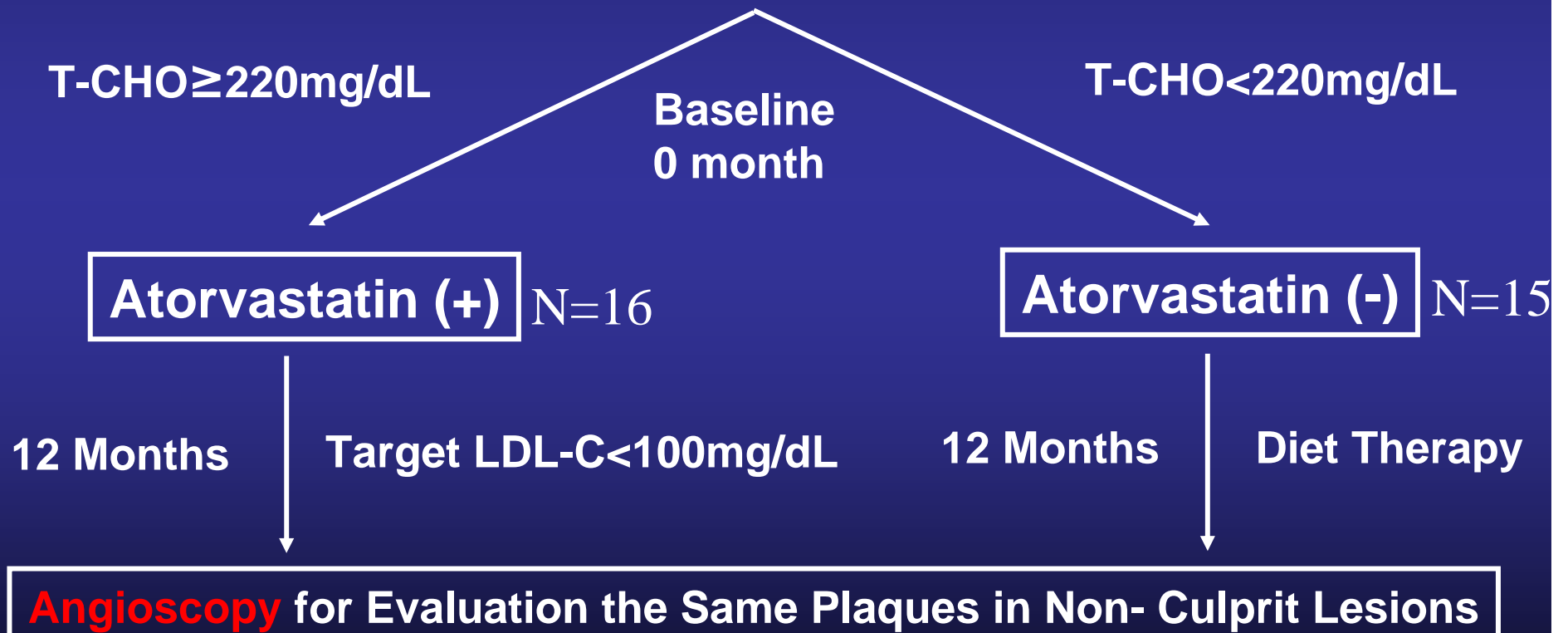


Changes in coronary plaque color and morphology by statin were evaluated using coronary Angioscopy.

Study Design



Ischemic Heart Disease, PCI for Culprit Lesions
Yellow Plaques in Non- Culprit Lesions detected by angiography



Score of the plaque color and complexity

White

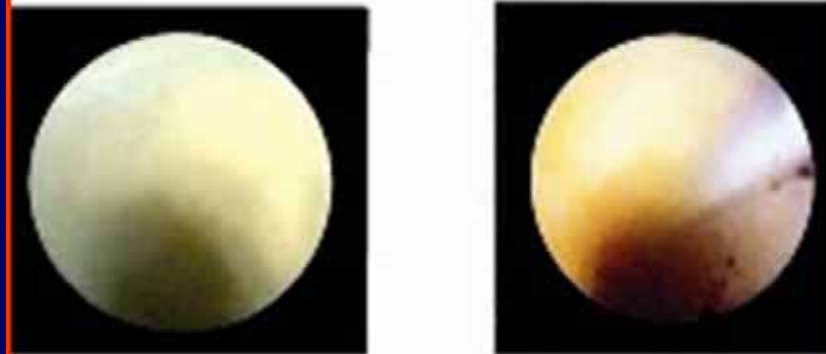
0



1 Light yellow

Yellow

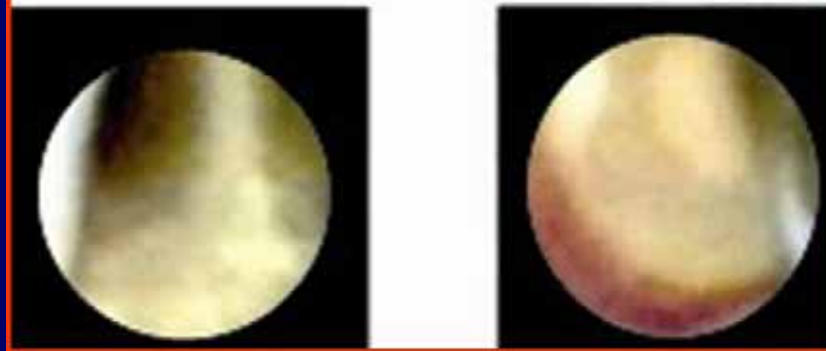
2



3 Dark yellow

irregular

1

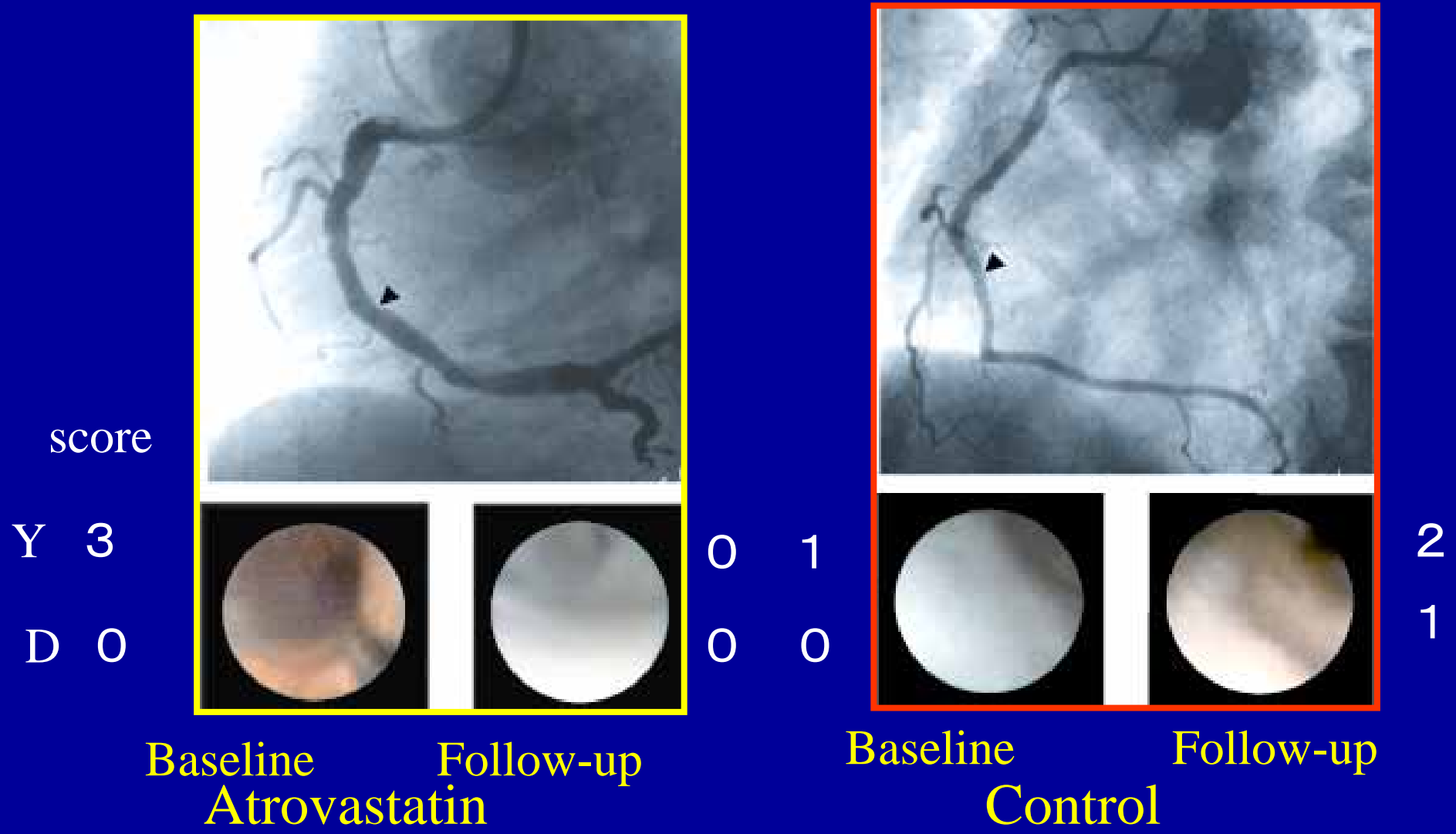


1 thrombus

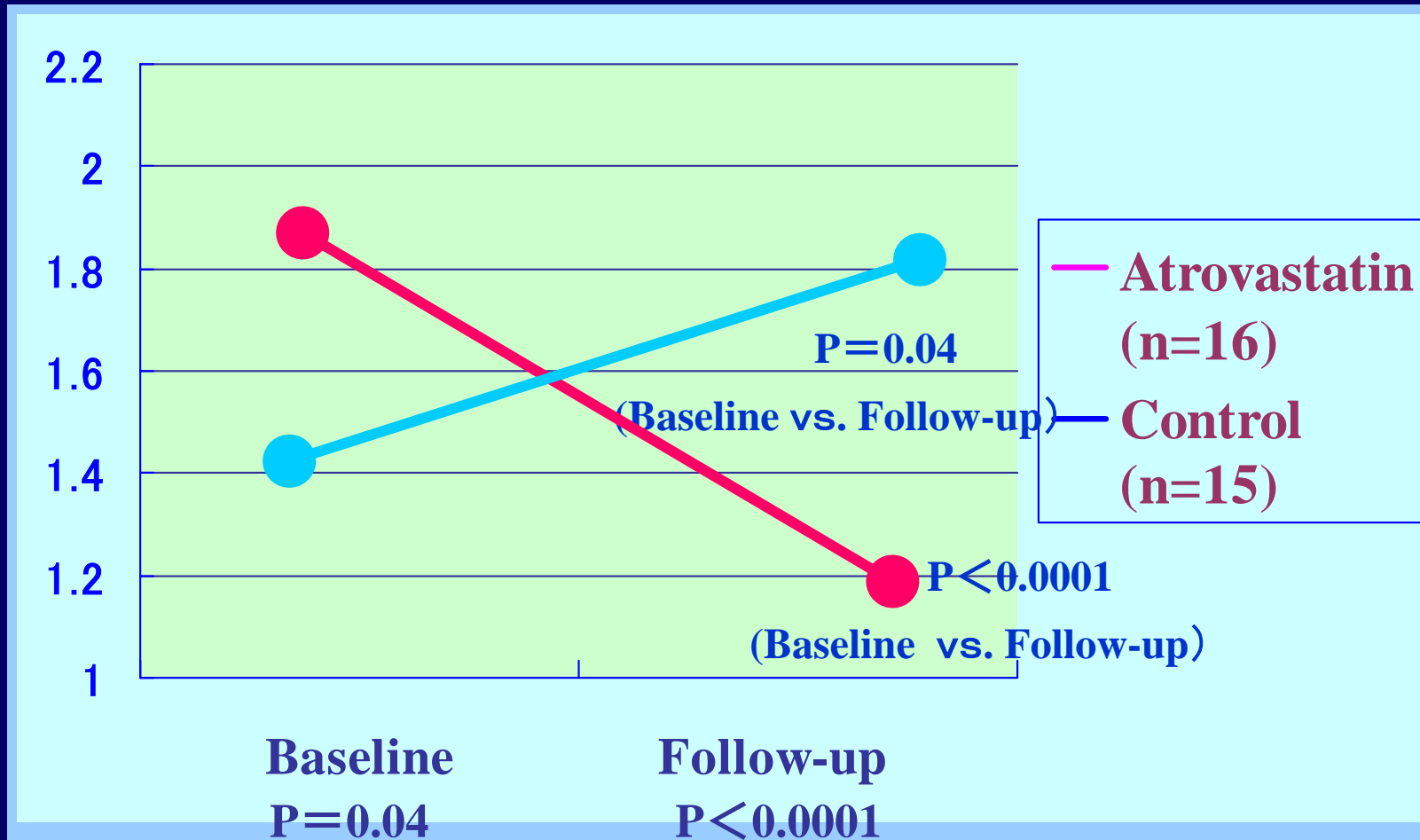
0: without irregular

0: without thrombus

Changes of angioscopic findings from baseline to follow-up

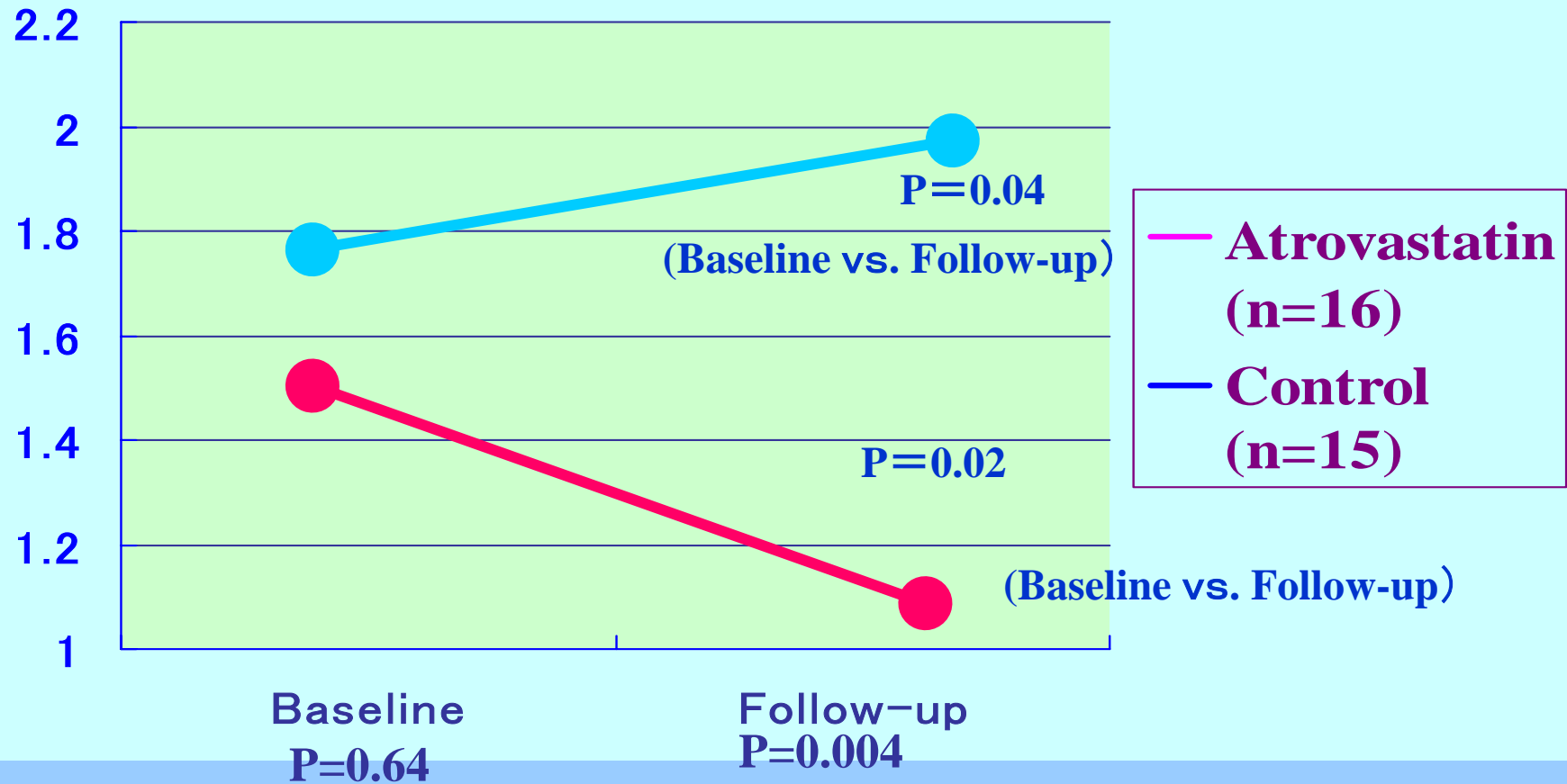


Changes in mean yellow score from baseline to follow-up



Comparison vs. Atrovastatin

Changes in mean disrupted score from baseline to follow-up



Comparison vs. Atrovastatin

Summary

LDL-C lowering by atrovastatin resulted in the reduction of angioscopic yellow grade and complexity of coronary plaque.

Lipid lowering therapy changed the quality of coronary plaque and should lead to coronary plaque stability

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Statin

Coronary intervention



Objective

The morphologic changes in infarcted-related lesions after stenting in acute myocardial infarction or recent myocardial infarction were investigated with coronary angioscopy.

Patients and methods



Coronary angiography (acute myocardial infarction N=28, post infarction angina N=15) was serially performed for the infarcted related lesions at baseline, after balloon inflation, and after stenting following balloon angioplasty and at one and six months after stenting.

Angioscopic findings



Plaque

yellow

white

smooth

irregular

Thrombus protruding

lining (mural)

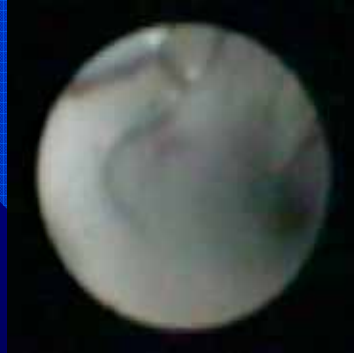
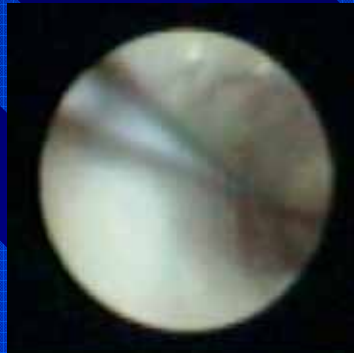


Stent coverage as Assessed by Angioscopy

Stent coverage score (semi-quantitatively visual evaluation)

- 0: complete exposure of stent struts**
- 1: exposure of stent struts with partial coverage**
- 2: > 50% coverage of stent struts**
- 3: almost complete coverage with slightly visible stent struts**
- 4: complete coverage of stent struts**

Stent coverage score



Grade 0

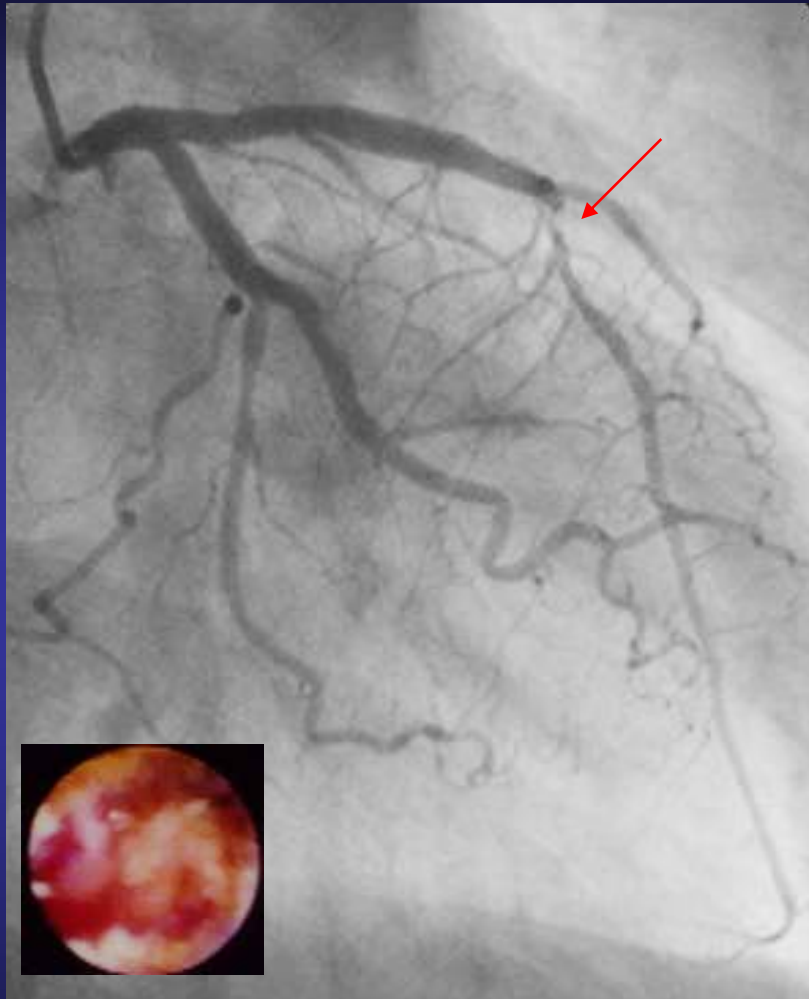
Grade 1

Grade 2

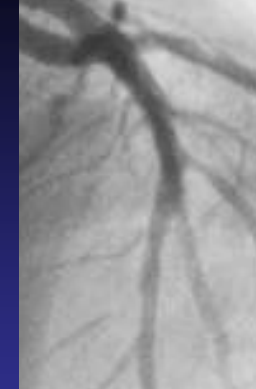
Grade 3

Grade 4

Acute STEMI



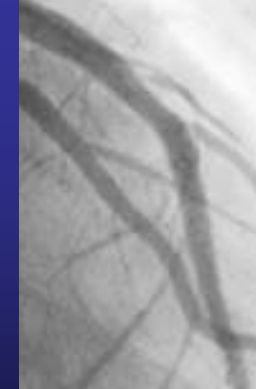
After POBA



After stenting



1-m F/U



6-M F/U



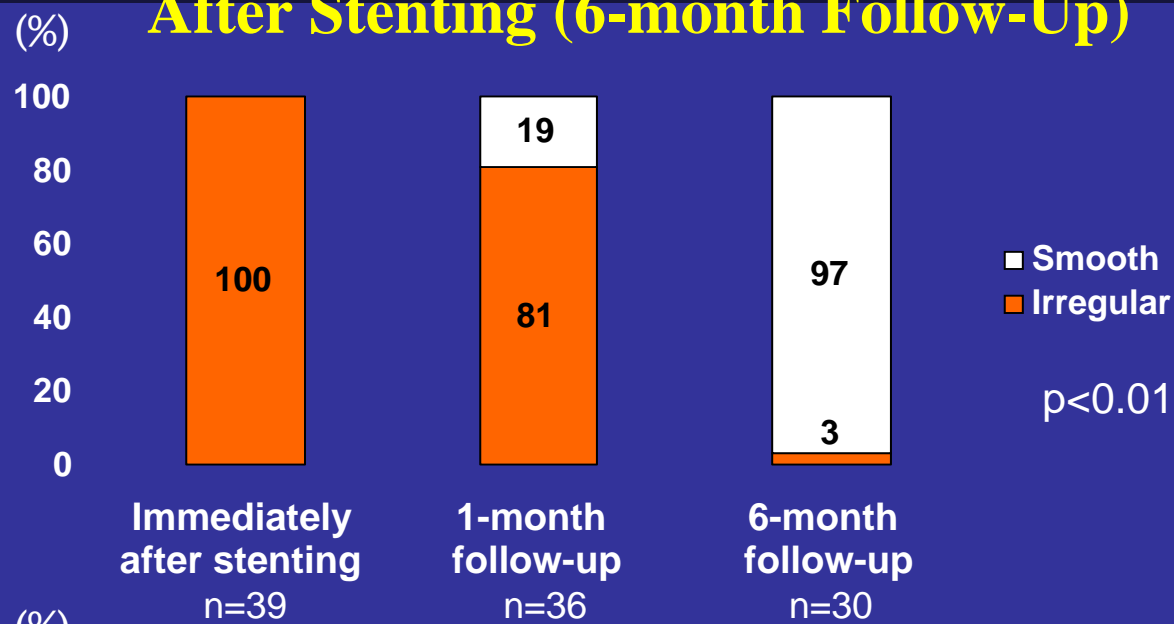
Stent coverage score =1

Stent coverage score =4

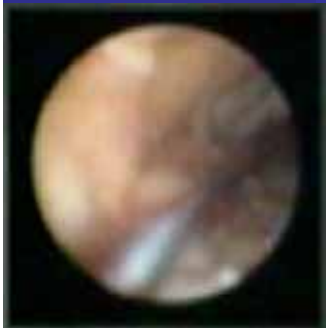
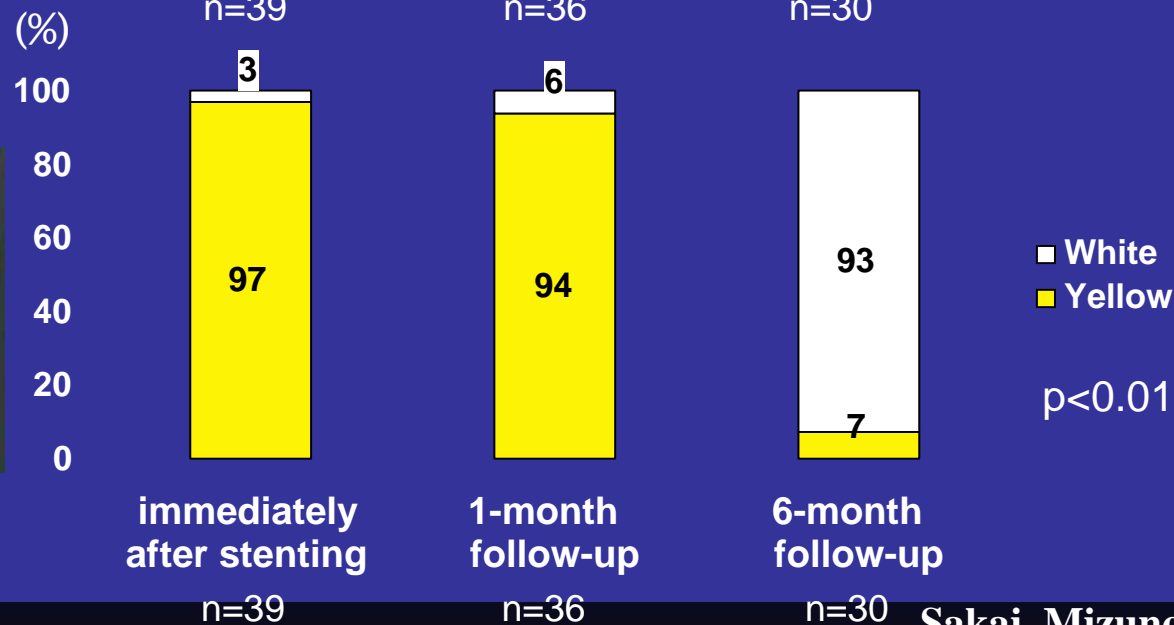


Changes in Plaque Surface (A) and Color (B) After Stenting (6-month Follow-Up)

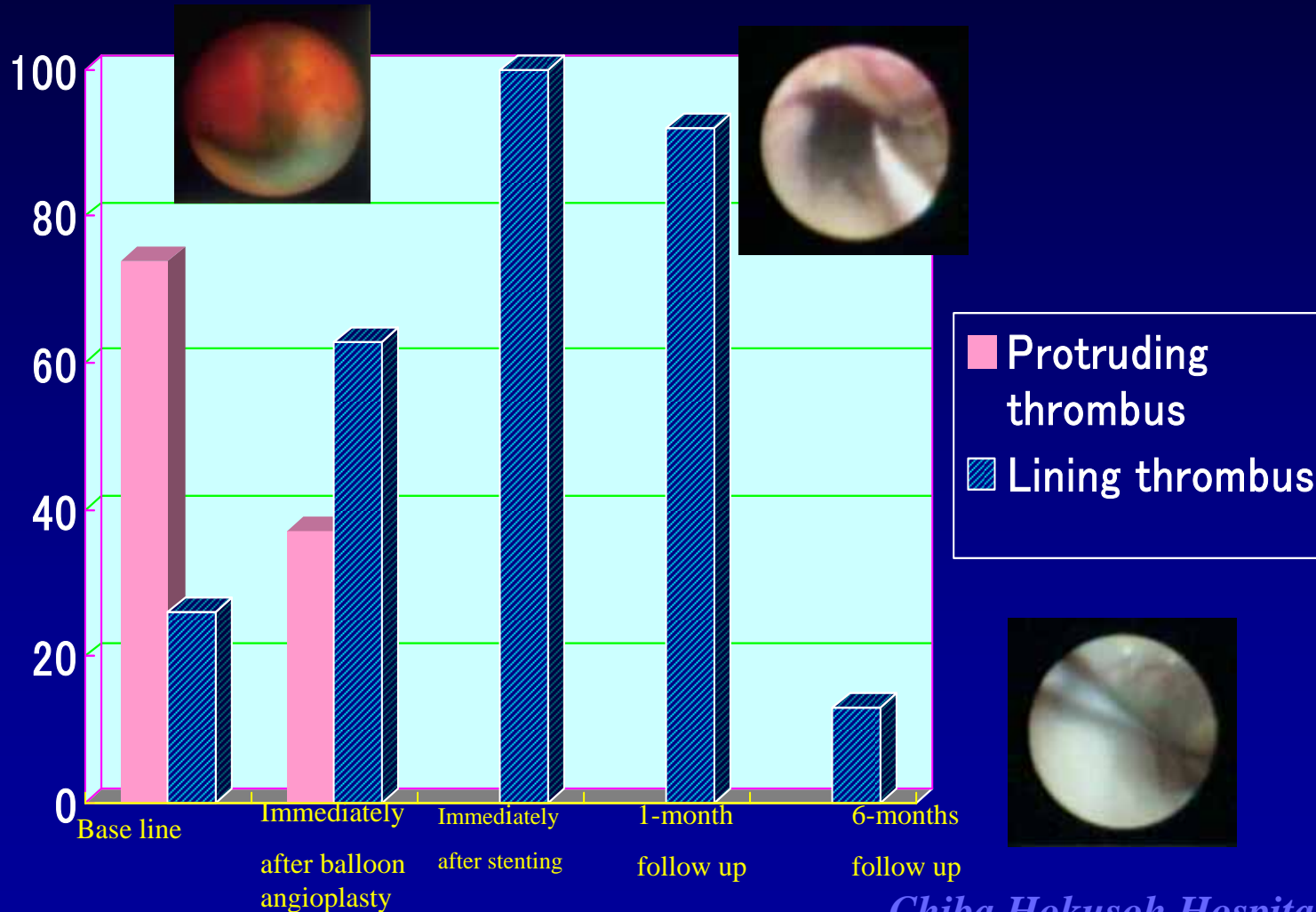
A



B



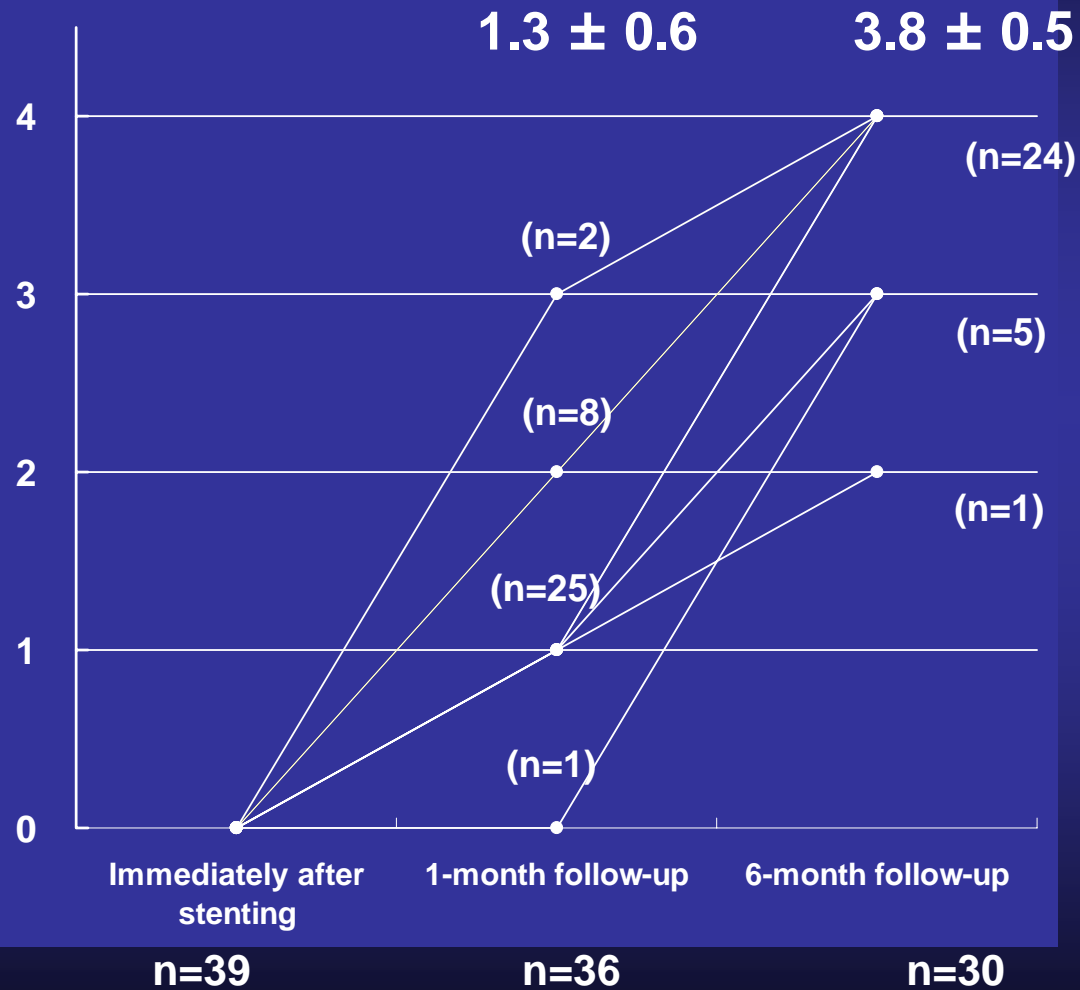
Changes in the Incidence of thrombus



Changes in Stent Coverage Score from baseline to follow-up (6 months)



Stent Coverage Score



p<0.01



Summary

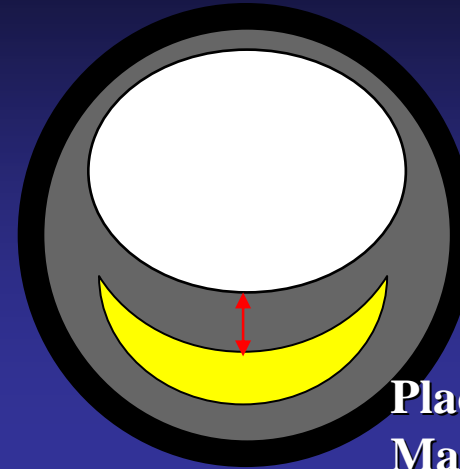


1. At baseline, most of the lesions had complex morphology, yellow plaque color, and protruding thrombus.
2. After stenting, the protruding thrombus and intimal flap disappeared.
3. At one-month-follow up, irregular and yellow surface, along with a lining thrombus, is still observed.
4. At six months follow-up, the neointima was found to have sufficiently formed over the stent.



Plaque stabilization

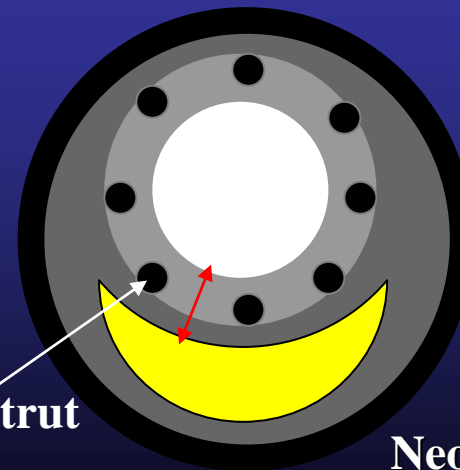
Lipid-lowering
therapy
(**statins**)



Plaque lipid decreased
Mature collagen increased

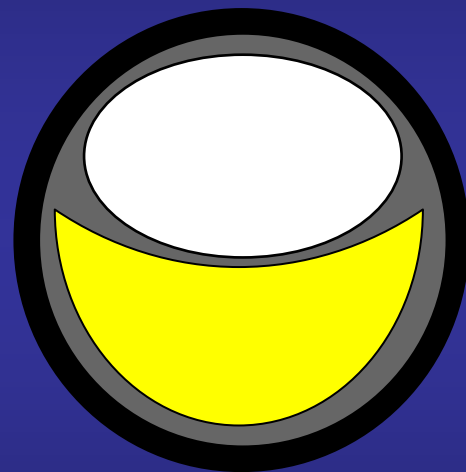
Mechanical plaque sealing

PCI
(**stenting**)



Stent strut

Neointimal hyperplasia



Vulnerable plaque

Thin fibrous cap
Large lipid core

Conclusion

Coronary angiography is an excellent tool with which to approach the unanswered question regarding plaque vulnerability and the effect of treatment **in living patients.**