Prevalence and significance of layered culprit plaques in ACS

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Introduction

- The majority of ACS cases are caused by plaque rupture or plaque erosion, leading to occlusive thrombosis.
- Plaque destabilization occurs silently, especially when plaque burden and thrombus volume are small.
- The repair process results in a layered plaque, characterized by distinct layers of organized thrombus and/or collagen.
- Autopsy studies have shown that healed plaques are frequent up to 61-73% in men who died from coronary event.

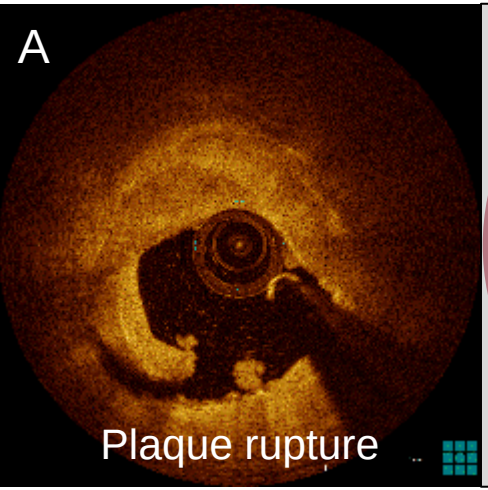
Aim of the study

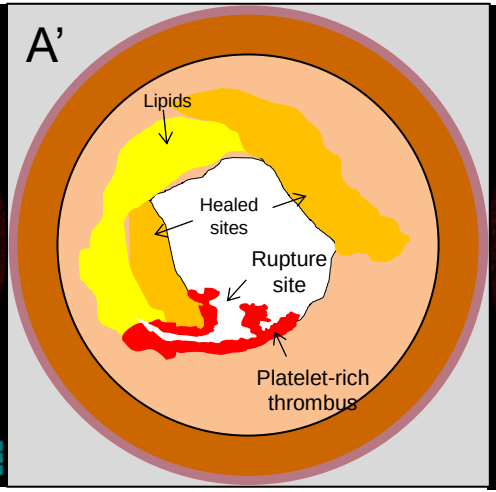
To determine the prevalence, morphologic characteristics, and clinical significance of healed culprit plaques in patients with ACS

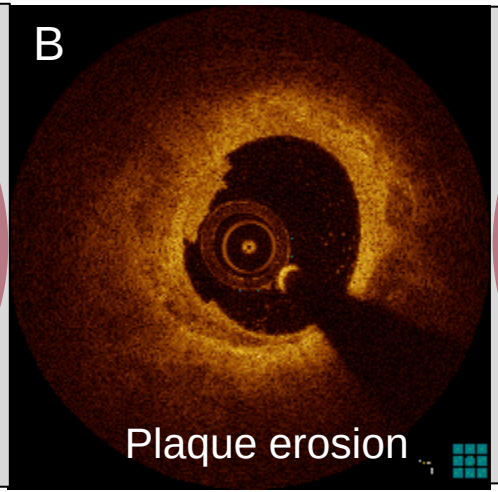
OCT definition of healed plaque: plaque with at least one layer of different optical signal intensity located close to the luminal surface and clearly demarcated from the underlying tissue

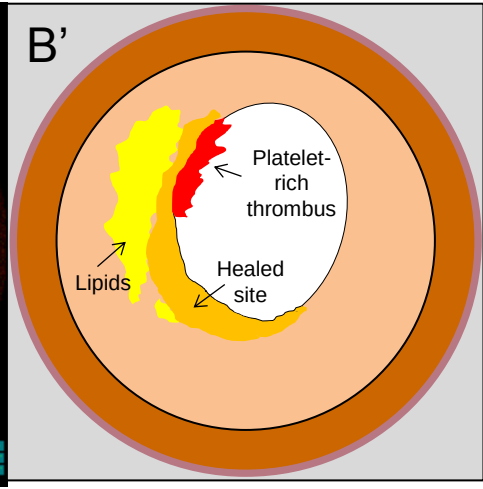
Shimokado et al. *Atherosclerosis* 2018

Representative cases









Plaque rupture

Plaque erosion

Demographics

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	Layered phenotype ***108 (28.7%)***	***Non-layered phenotype*** ***268 (71.3%)***	***p***
Age, yrs	58.5±10.7	57.1±11.8	0.285
Male gender	85 (78.7)	202 (75.4)	0.492
BMI, kg/m^2	23.9±6.9	24.2±6.2	0.764
Risk factors			
Hypertension	55 (50.9)	137 (51.1)	0.973
Hyperlipidemia	48 (44.4)	89 (33.2)	0.041
Smoking	58 (53.7)	147 (54.9)	0.840
DM	38 (35.2)	63 (23.5)	0.021
Clinical history			
Chronic kidney disease	9 (8.3)	15 (5.6)	0.353
Previous MI	17 (15.7)	17 (6.3)	0.009
Previous PCI	14 (13.0)	31 (11.6)	0.706
Previous CABG	0 (0)	2 (0.7)	1.000
Clinical presentation			0.042
STEMI	64 (59.3)	188 (70.1)	
NSTE-ACS	44 (40.7)	80 (29.9)	
Laboratory data			
LVEF, %	58.1±10.2	58.0±8.8	0.903
LDL-C, mg/dL	116.7±39.2	116.1±38.6	0.902
hs-CRP, mg/L	4.98 (1.00-11.32)	3.00 (0.30-10.15)	0.029
Peak Tn-I, µg/L	40.8 (17.0-110.9)	56.8 (23.9-120.3)	0.874

Angiographic findings

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	Layered phenotype ***108 (28.7%)***	***Non-layered phenotype*** ***268 (71.3%)***	***p***
Angiographic data			
Culprit vessel			0.814
LAD	56 (51.9)	146 (54.5)	
RCA	38 (35.2)	93 (34.7)	
LCx	14 (13.0)	29 (10.8)	
Multivessel disease	60 (55.6)	105 (39.2)	0.004
B2/C lesion	78 (72.2)	158 (59.0)	0.018
Lesion length, mm	16.7±8.5	15.9±7.6	0.460
MLD, mm	0.94±0.44	1.13±0.56	0.006
RVD, mm	3.26±0.64	3.59±0.62	0.782
DS, %	70.0±14.1	65.7±14.7	0.028

OCT findings

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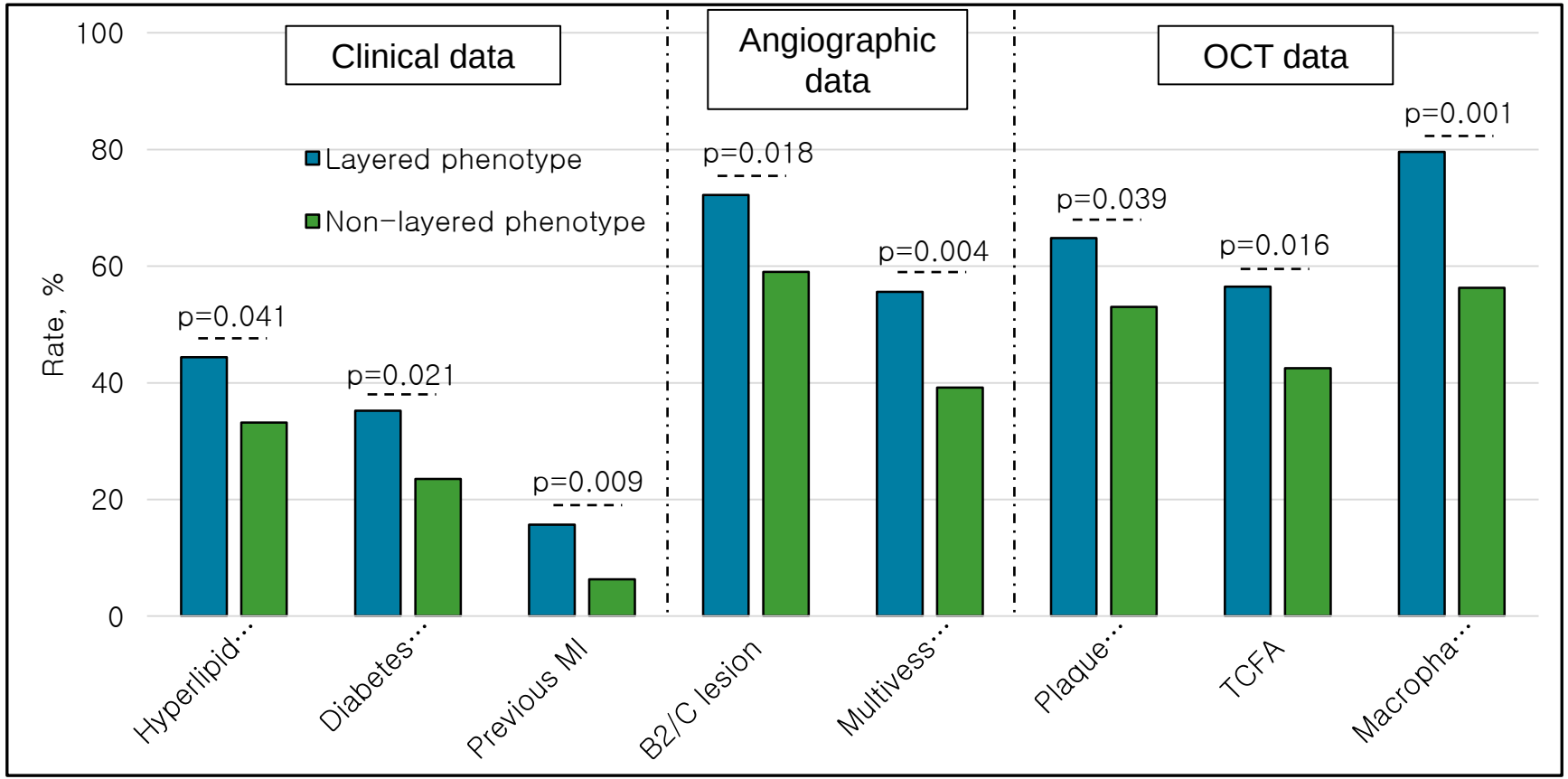
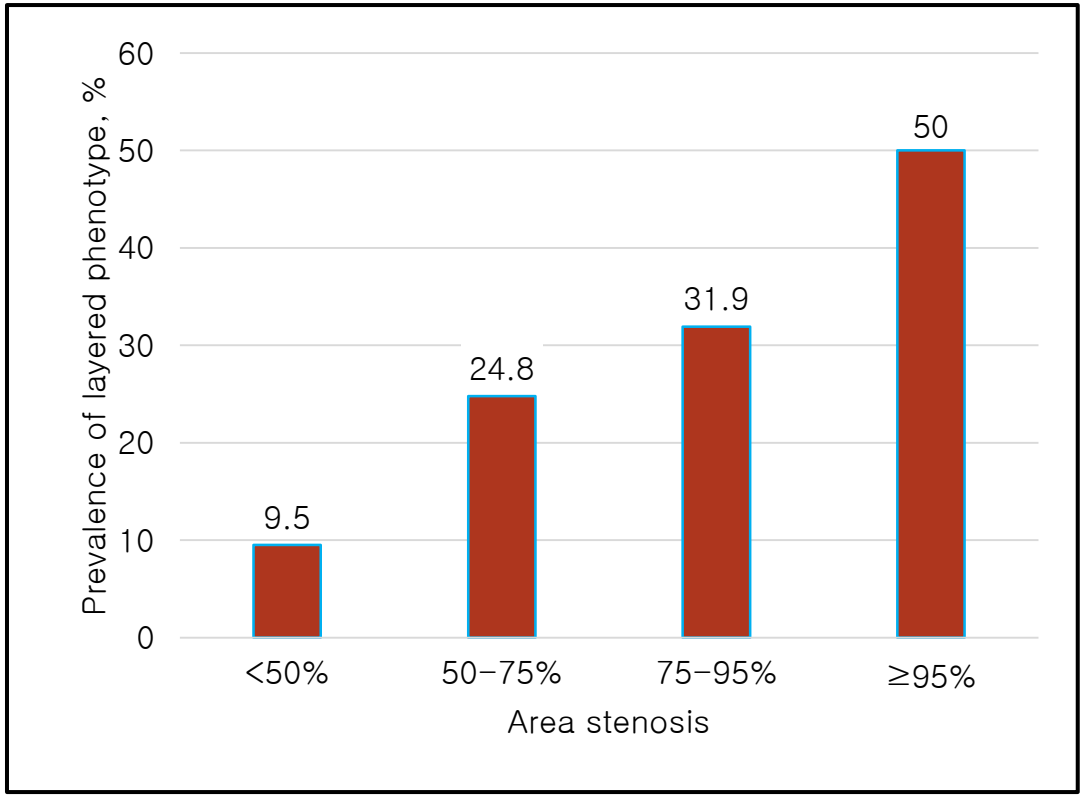
	Layered phenotype ***108 (28.7%)***	***Non-layered phenotype*** ***268 (71.3%)***	***p***
OCT data at the culprit lesion			
Underlying pathology			0.039
Plaque rupture	70 (64.8)	142 (53.0)	
Intact fibrous cap	38 (35.2)	126 (47.0)	
Plaque type			0.013
Fibrous plaque	18 (16.7)	78 (29.1)	
Lipid plaque	90 (83.3)	190 (70.9)	
FCT, µm	60 (50-80)	60 (50-81)	0.360
Lipid index, mm°	3240.0	2995.8	0.203
TCFA	61 (56.5)	114 (42.5)	0.016
Macrophage accumulation	86 (79.6)	151 (56.3)	0.001
Thrombus	103 (95.4)	242 (90.3)	0.146
Microvessels	47 (43.5)	92 (34.3)	0.095
Calcification	36 (33.6)	81 (30.2)	0.623
MLA, mm^2	1.45±0.87	1.84±1.09	0.001
AS, %	79.23±9.49	74.27±14.32	0.001

Clinical, Angio, OCT findings



Degree of stenosis and layered plaque



Clinical outcome

	Layered phenotype ***49 (21.7%)***	***Non-layered phenotype*** ***177 (78.3%)***	***p***
Death	0 (0.0)	1 (0.6)	1.0
AMI	1 (2.0)	1 (0.6)	0.387
TVR	3 (6.1)	5 (2.8)	0.375
Hospitalization	16 (32.7)	28 (16.5)	0.013

Summary

- Healed plaques, a signature of previous subclinical thrombosis, at the culprit lesion are:

1) found in 29% of patients with ACS, and one-third of them have a multi-layered pattern;
2) more frequent in ACS patients with hyperlipidemia, DM, and a history of MI;
3) complex on angiogram and more frequently multi-vessel disease;
4) frequently associated with plaque rupture, TCFA and macrophage infiltration;
5) multi-layered plaques present more severe luminal narrowing as compared to single-layered or non-layered plaques.

Conclusions

The combination of plaque vulnerability, local inflammation, and greater plaque burden in addition to systemic inflammation may outweigh the protective mechanism of plaque healing and predispose those plaques to develop occlusive thrombus.

JACC 2019 In press.

Thank you