The 28th TCTAP 2023

Hot Topics: Vulnerable Plaque, Imaging & Physiology
2023/5/8 (Mon), 2:15 PM ~ 2:23 PM
Presentation Theater 1, Vista 3, B2

Physiology or Imaging for Identification of Vulnerable Plaque; Physiology May Miss?

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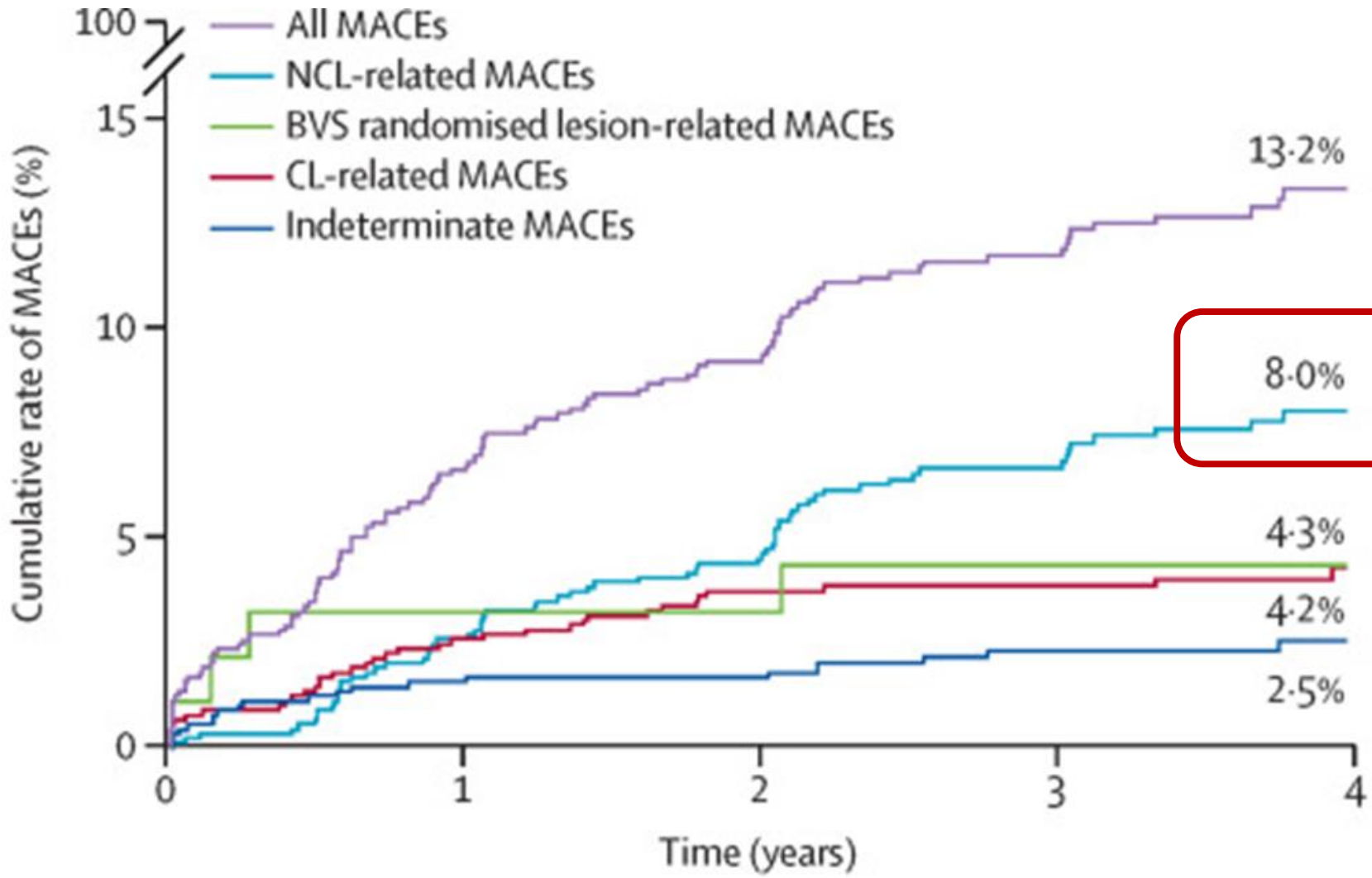
Disclosure statement of financial interest

Within the past 12 months, I or my spouse/partner have had a financial interest/arrangement or affiliation with the organization(s) listed below.

Affiliation/Financial Relationship	Company
Grant/Research Support	No
Consulting Fees/Honoraria	No
Major Stock Shareholder/Equity	No
Royalty Income	No
Ownership/Founder	No
Intellectual Property Rights	No
Other Financial Benefit	No

PROSPECT II: follow-up NCL-related MACE in ACS



All MACEs

NCL-related MACEs

BVS randomized lesion-related MACEs

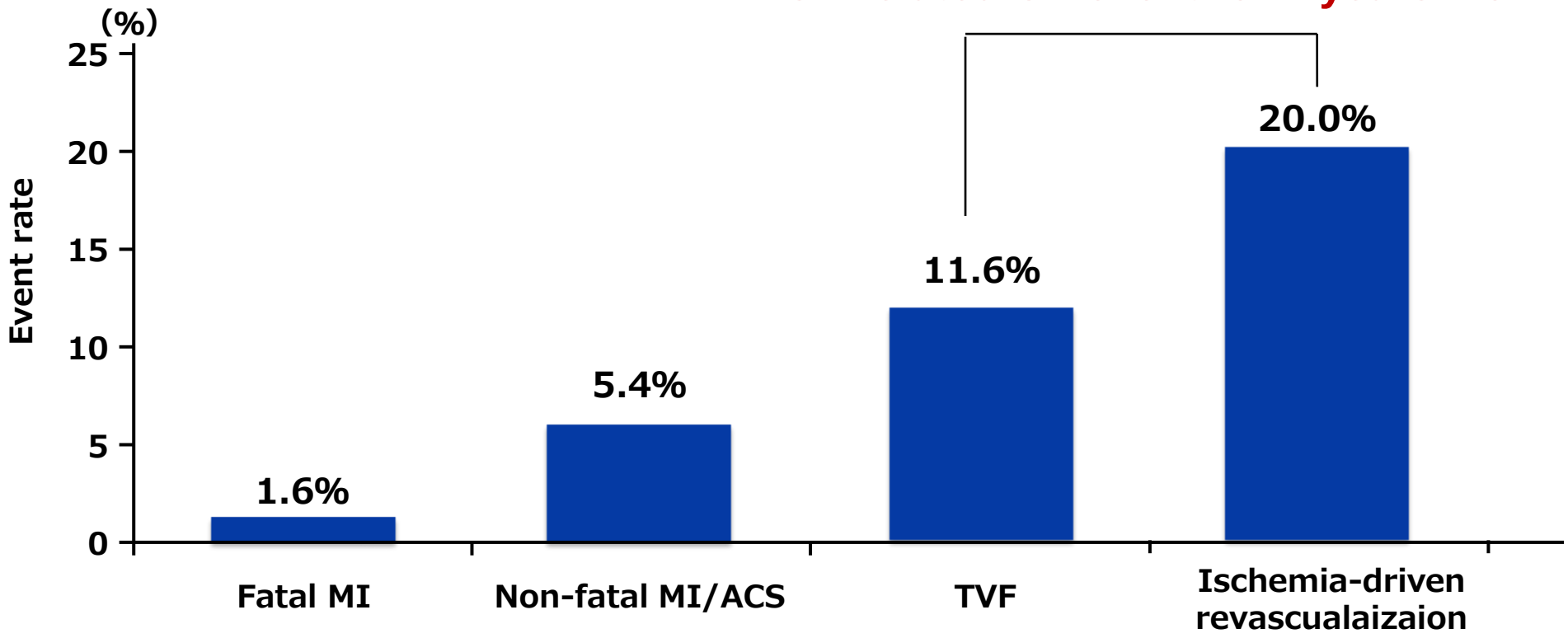
CL-related MACEs

Indeterminate MACEs

PACIFIC: follow-up NCV-related MACE in ACS

The PACIFIC Registry is a multicenter, prospective study in JAPAN that enrolled 3,597 ACS patients and followed them for 2 years.

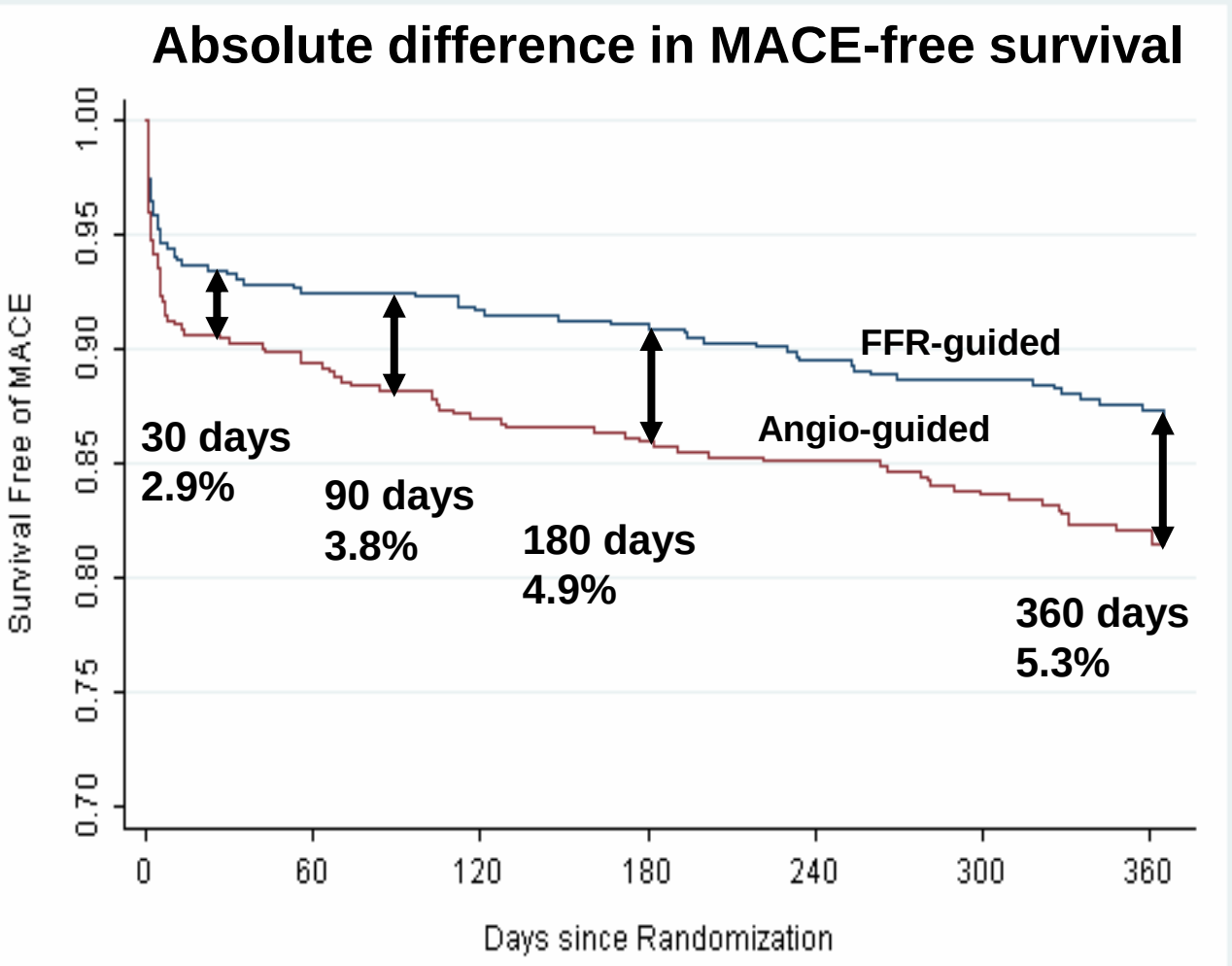
NCV-related CV event for 2 years ≈ 8%



The incidence of fetal MI, non-fatal MI, TVF, and ischemia-driven revasuclarization was 1.6%, 5.4%, 11.6% and 20.0%, respectively.

Physiology

FAME: FFR-based PCI deferral



ESC guidelines 2018

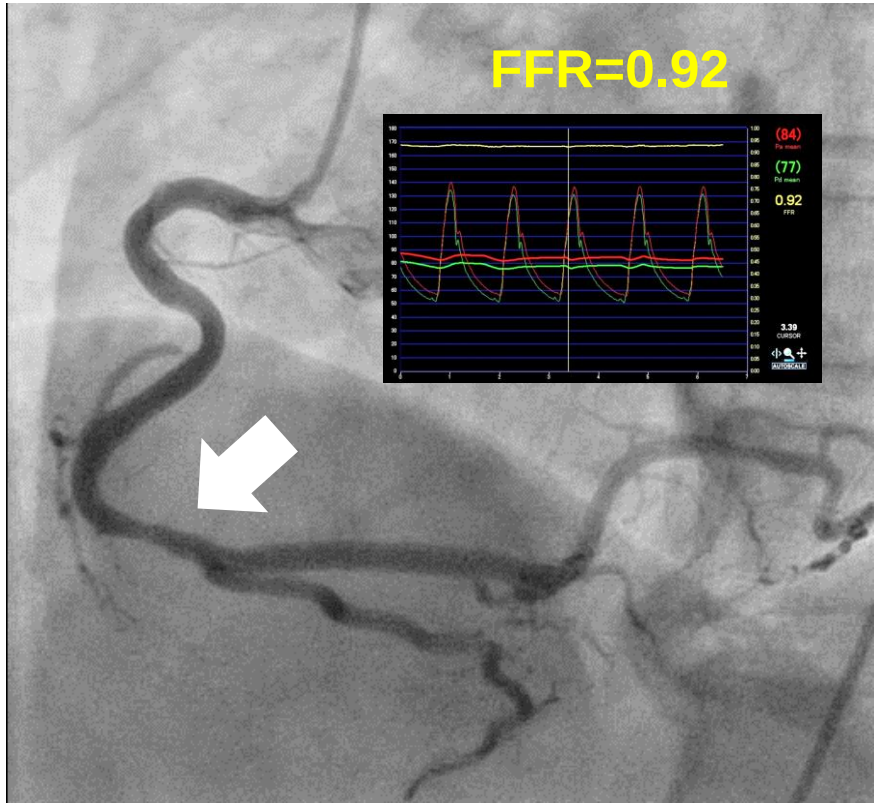
Recommendations on functional testing for lesion assessment

Recommendations	Class[a]	Level[b]
When evidence of ischaemia is not available, FFR or iwFR are recommended to assess the haemodynamic relevance of intermediate-grade stenosis.[15,17,18,39]	I	A
FFR-guided PCI should be considered in patients with multivessel disease undergoing PCI.[29,31]	IIa	B

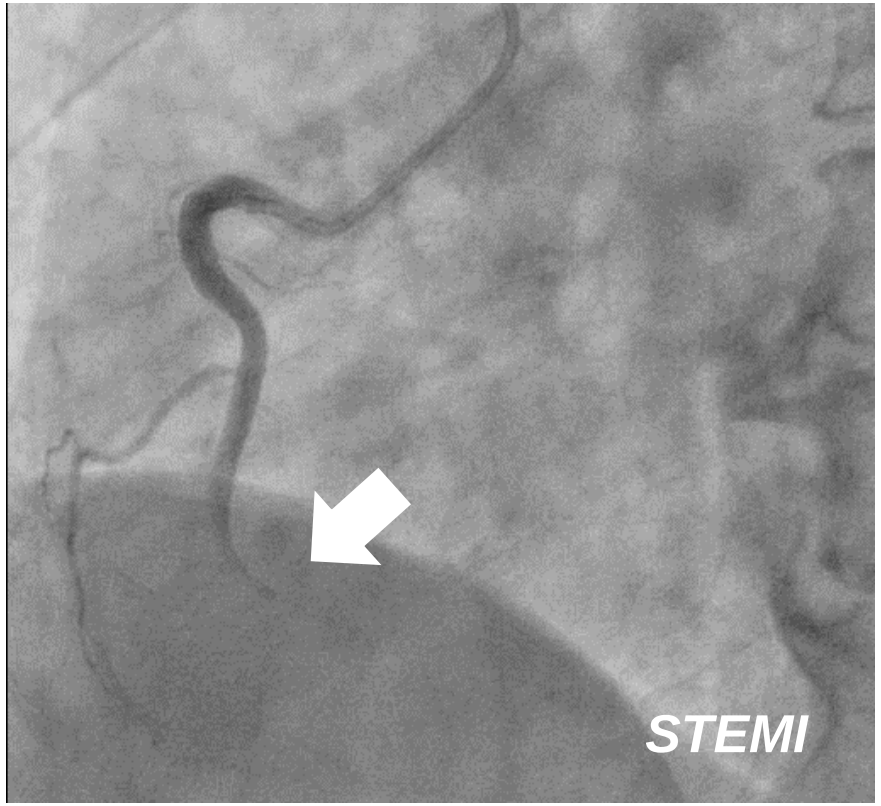
FFR = fractional flow reserve; iwFR = instantaneous wave-free ratio; PCI = percutaneous coronary intervention.
a Class of recommendation.
b Level of evidence.

ACS arising from FFR-negative NCL

Baseline



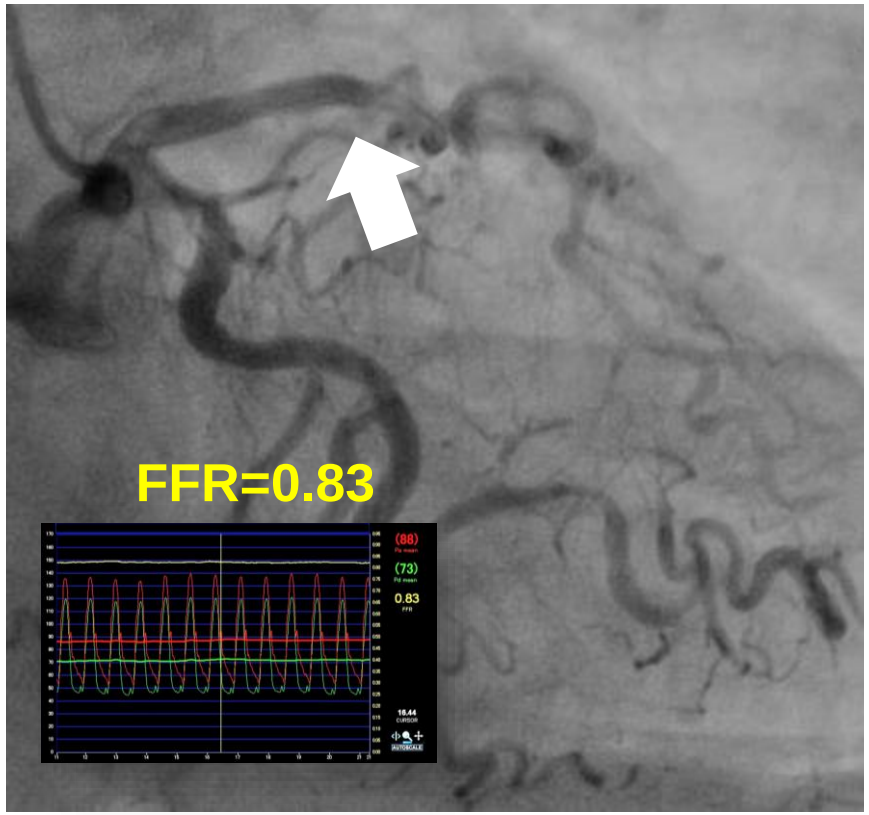
16 months



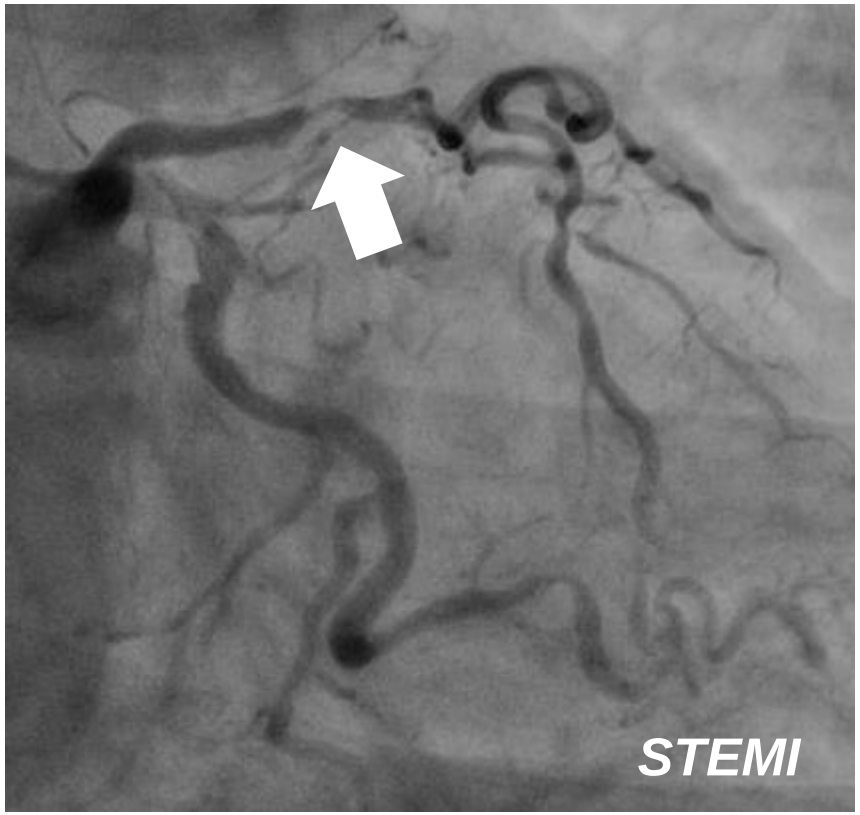
64-year-old male. Baseline angiography showed a mild stenosis in mid RCA, but FFR was negative for ischemia. This patient misunderstood that he has no disease and discontinued the medication. Sixteen months later, he developed STEMI with cardiogenic shock. Coronary angiogram revealed occlusion in mid RCA.

ACS arising from FFR-negative NCL

Baseline



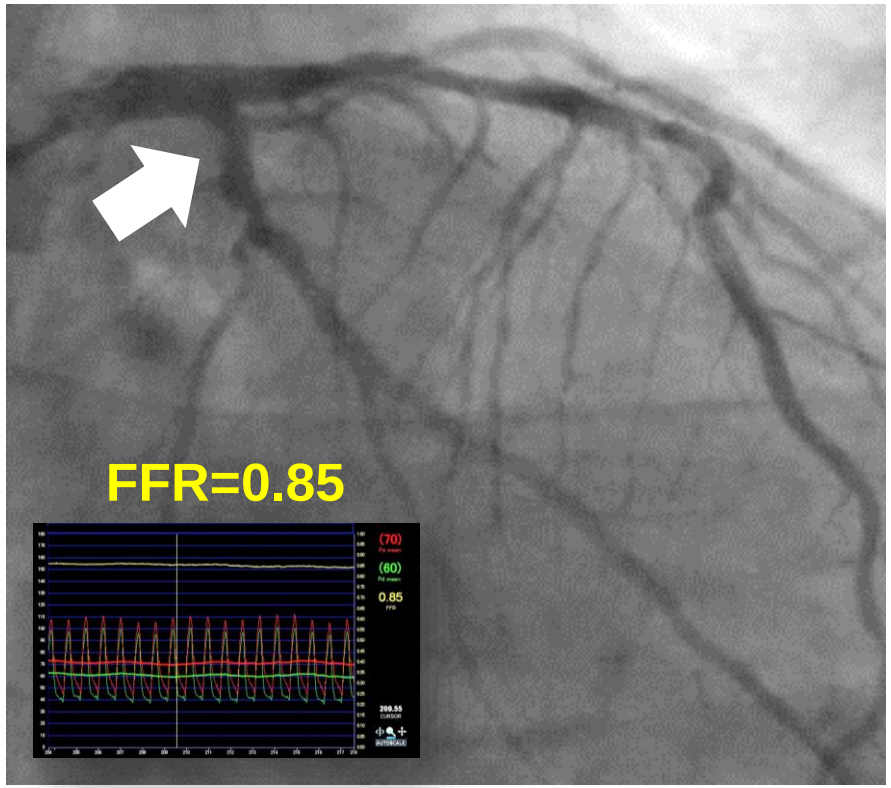
13 months



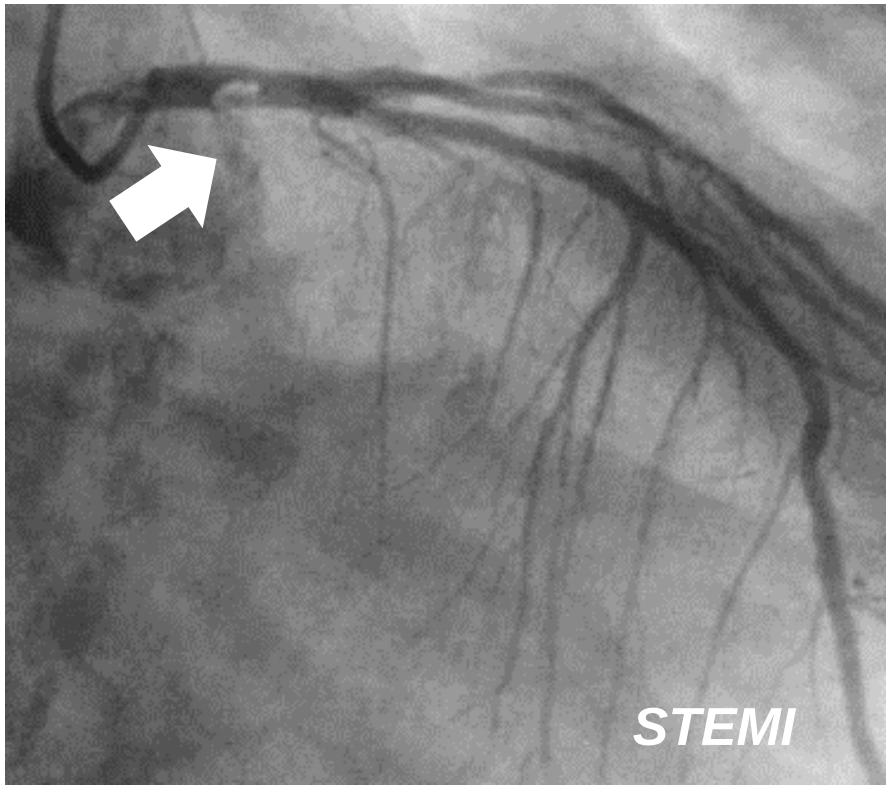
72-year-old man. At baseline he had STEMI and underwent PCI for RCA lesion. Angiography showed a mild stenosis in proximal LAD and FFR was negative for ischemia. He had hypertension, diabetes, and Peripheral arterial disease. Aspirin 100 mg, Valsartan 80 mg, and Vildagliptin 50 mg were administered. Blood tests showed LDL-C=91, HDL-C=35, HbA1C=6.5. Thirteen months later, he developed STEMI arising from the proximal LAD lesion.

ACS arising from FFR-negative NCL

Baseline



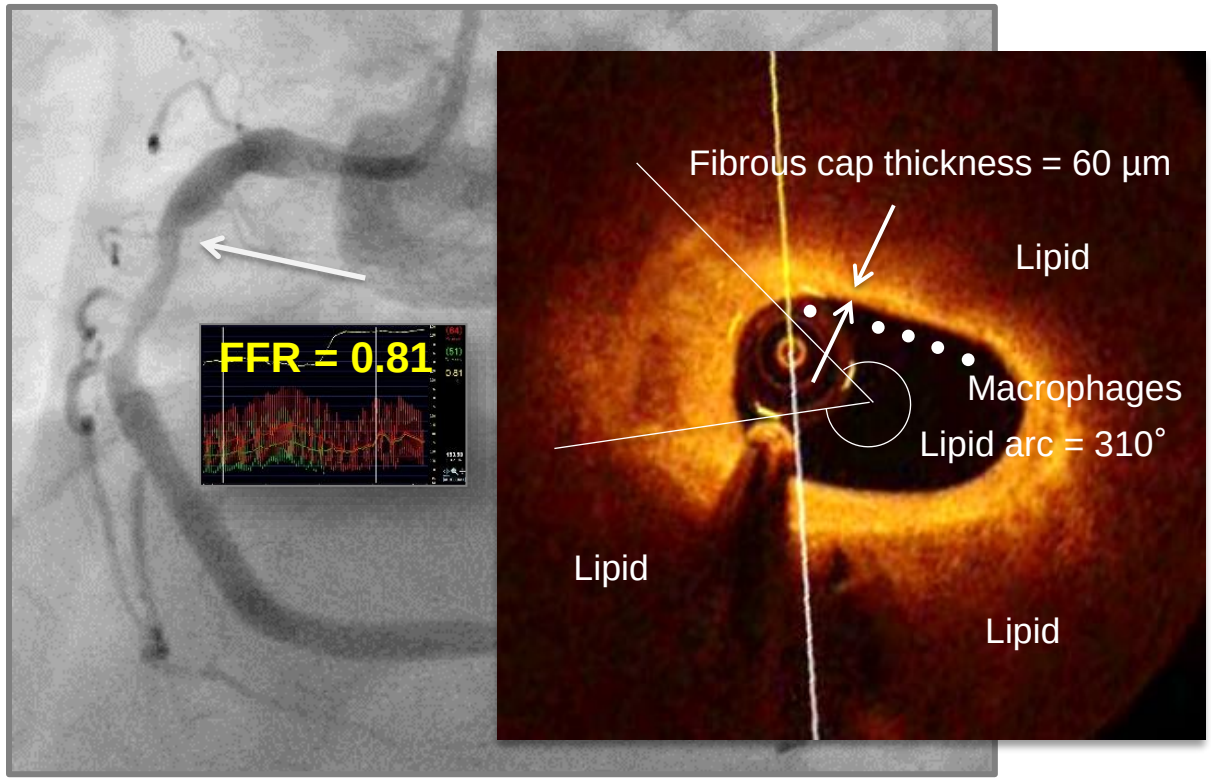
7 months



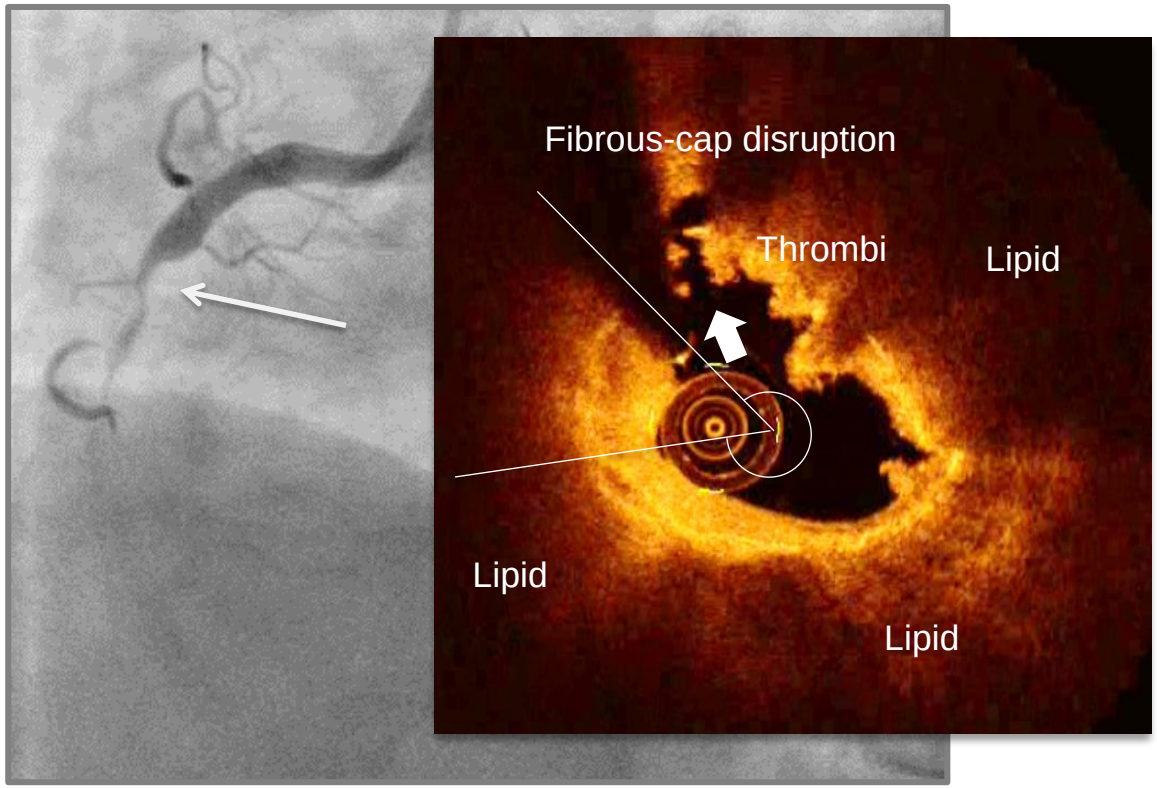
68-year-old man. At baseline he had NSTEMI and underwent PCI to RCA lesion. Angiography showed a mild stenosis in proximal LCX and FFR was negative for ischemia. He had HT, DLP, CKD (hemodialysis), and smoking habit. Aspirin 100 mg, Imidapril 5 mg, Carvedilol 3.75 mg, Atorvastatin 10 mg, and Ethyl icosapentate 1800 mg were administered. Blood tests showed LDL-C=47 and HDL-C=27. Seven months later, he developed STEMI arising from the proximal LCX lesion.

AMI arising from LRP+TCFA

Baseline



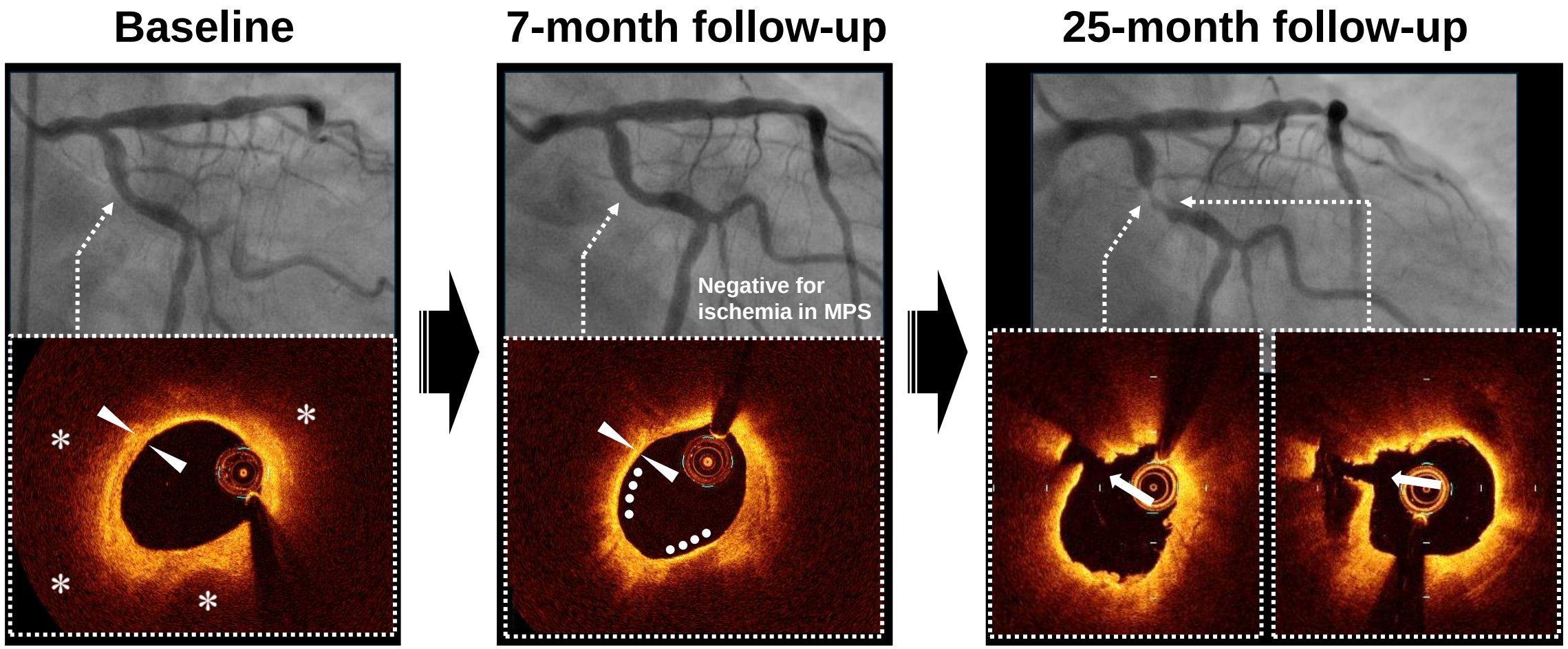
10 months follow-up



Baseline angiogram showed moderate stenosis in the proximal RCA. Baseline OCT characterized the plaque as both LRP and TCFA (maximum lipid arc 310˚ [asterisks], minimum fibrous cap thickness 60μm [arrow heads], minimum lumen area 2.27mm^2, and presence of OCT-derived macrophage infiltration [dots]). This plaque was associated with AMI 10 months after baseline imaging. Follow-up angiography showed that the stenosis developed into an occlusion. Follow-up OCT showed rupture (arrow) of the plaque that was imaged in the baseline OCT.

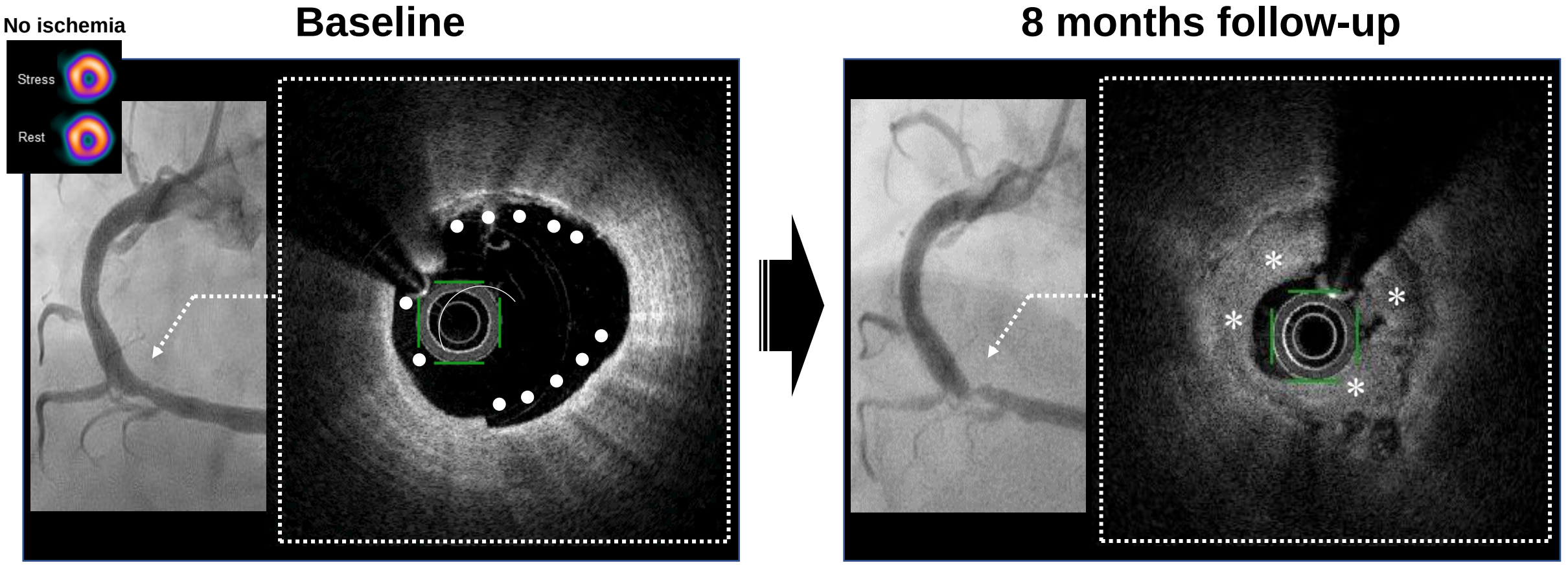
Kubo T, Mintz GS, Akasaka T, et al. EHJ img doi: 10.1093/ehjci/jeab028.

AMI due to plaque rupture arising from TCFA



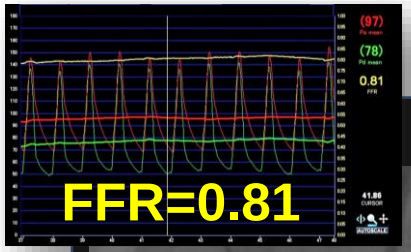
Angiogram at baseline showed mild stenosis in the proximal left circumflex artery. OCT at baseline characterized the plaque as LRP (maximum lipid arc = 360˚ [asterisks] and minimum fibrous cap thickness = 140μm [arrowheads]). Angiogram at 7-month follow-up showed no progression in the stenosis. OCT at 7-month follow-up showed the decrease of the fibrous cap thickness and the presence of macrophages accumulation [dots], characterizing this plaque as both LRP and TCFA (minimum fibrous cap thickness = 60μm [arrowheads]). This plaque was associated with myocardial infarction 25 months after baseline imaging. Angiography at 25-month follow-up showed that the stenosis developed into a subtotal occlusion. OCT at 25-month follow-up showed rupture [arrows] of the plaque that was imaged during OCT at baseline and 7-month follow-up. ACS = acute coronary syndrome, LRP = lipid-rich plaque, OCT = optical coherence tomography, TCFA = thin-cap fibroatheroma.

AMI due to plaque erosion from fibrous plaque



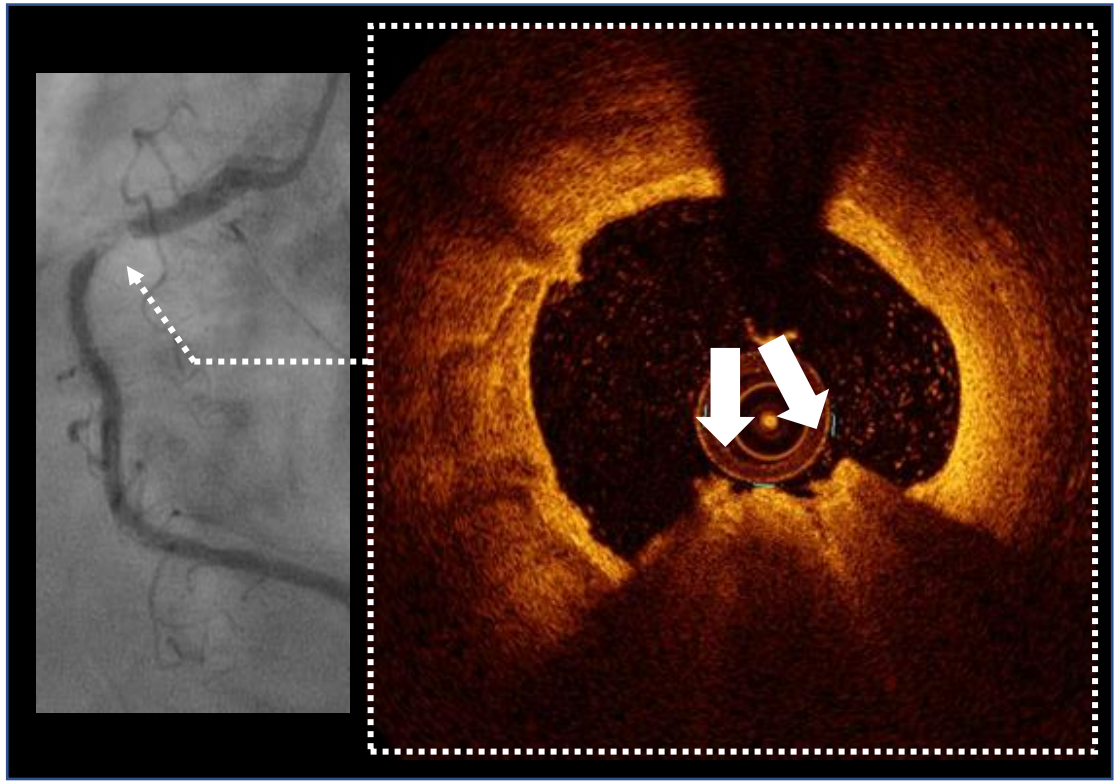
Baseline angiogram showed mild stenosis in the mid right coronary artery. Baseline OCT identified a fibrous plaque with macrophages accumulation [dots]. This plaque was associated with myocardial infarction 8 months after baseline imaging. Follow-up angiography showed that the stenosis developed into a subtotal occlusion. Follow-up OCT detected thrombus [asterisks] overlying intact fibrous cap of the plaque (i.e. OCT-erosion) that was imaged during baseline OCT. ACS = acute coronary syndrome; OCT = optical coherence tomography.

AMI due to calcified nodule from fibrocalcific plaque



Baseline

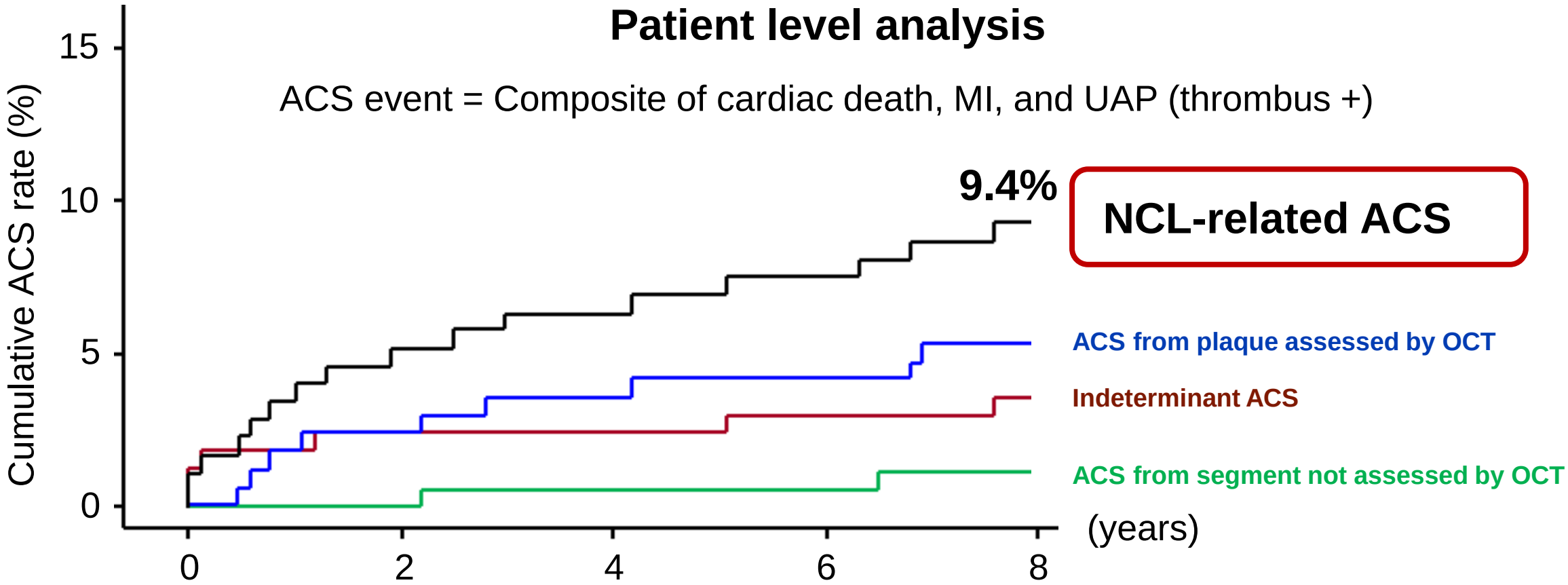
9 months follow-up



Baseline angiogram showed mild stenosis in the proximal right coronary artery. Baseline OCT identified a plaque with superficial calcium sheet [asterisks]. This plaque was associated with myocardial infarction 9 months after baseline imaging. Follow-up angiography showed that the stenosis developed into a subtotal occlusion. Follow-up OCT detected calcified nodule [arrows] in the plaque that was imaged during baseline OCT. AMI = acute myocardial infarction; OCT = optical coherence tomography.

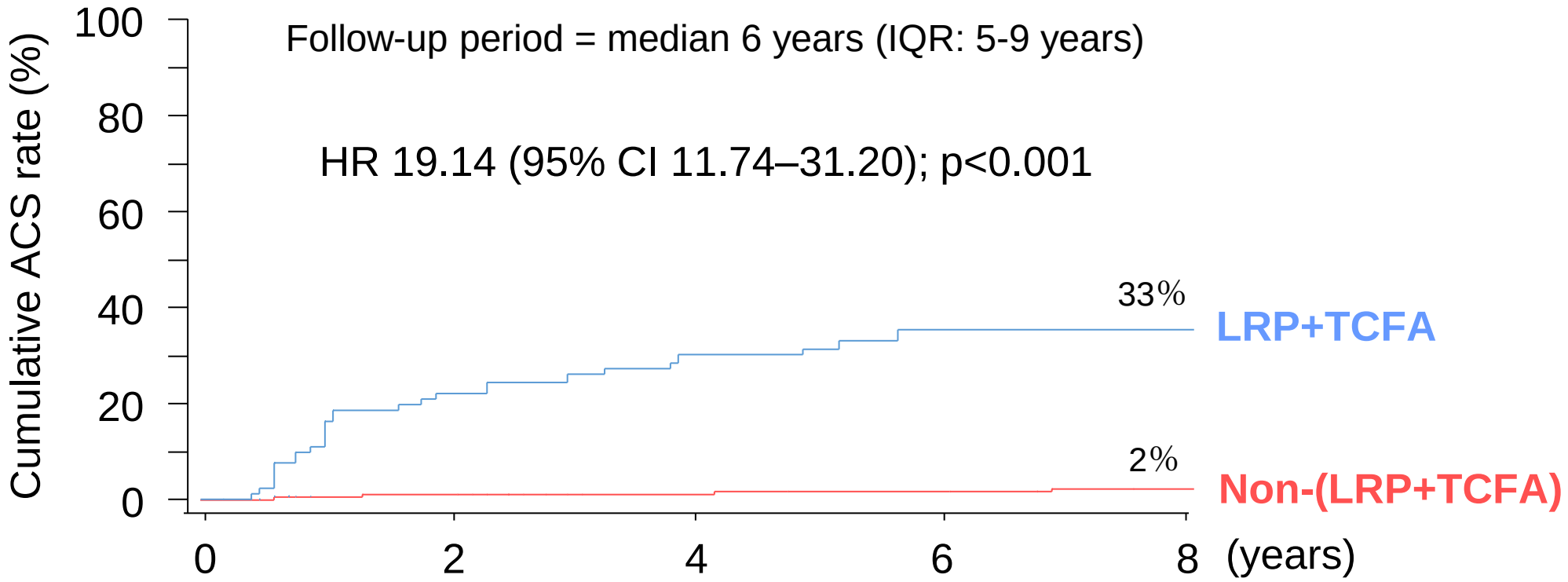
ACS arising from physiology-negative NCLs

An OCT registry included 1,378 patients with 3,578 non-culprit plaques that did **not induce ischemia on physiological testing (such as FFR, iFR, or MPS) at baseline**. We investigated the incidence of follow-up ACS arising from these non-culprit plaques during a median follow-up period of 6 years.



OCT-derived TCFA and follow-up ACS

The association between non-culprit plaques characterized as both lipid-rich plaque (LRP) and TCFA by OCT and the risk of follow-up ACS was evaluated at the lesion level (n=3,533).

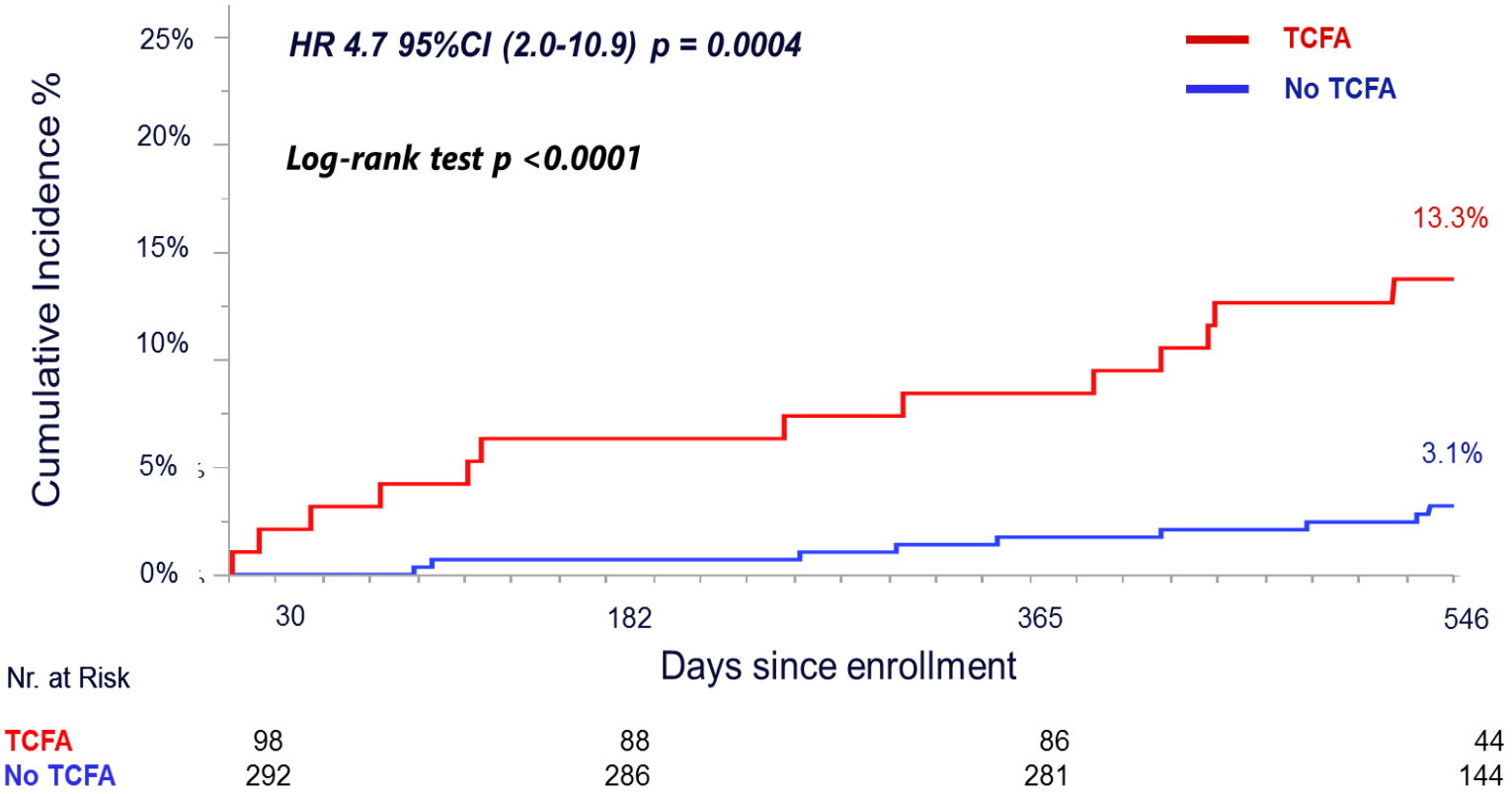


Non-culprit plaques characterized as both LRP and TCFA were associated with ≈17-fold increase in subsequent ACS events during a median follow-up period of 6 years.

TCFA in diabetics with FFR-negative lesions

The COMBINE study enrolled 334 DM patients with at least 1 coronary lesions with angiographic diameter stenosis 40-80% and FFR >0.80 (defined as target lesions).

Primary endpoint
Target lesion related MACE
Composite of
- Cardiac death
- Target vessel MI,
- Clinically-driven TLR
- Hospitalisation due to unstable or progressive angina



In DM patients, OCT-detected TCFA was associated with a five-fold higher rate of adverse events despite the absence of ischemia.

Summary

- In the physiology-negative NCLs, imaging-determined vulnerable plaques are at increased risk for future coronary event.
- Clinical demonstration of the discordant impact of vulnerable plaque and ischemia on future adverse events represents a paradigm shift in risk stratification for coronary artery disease and paves the way for novel therapeutic strategies.