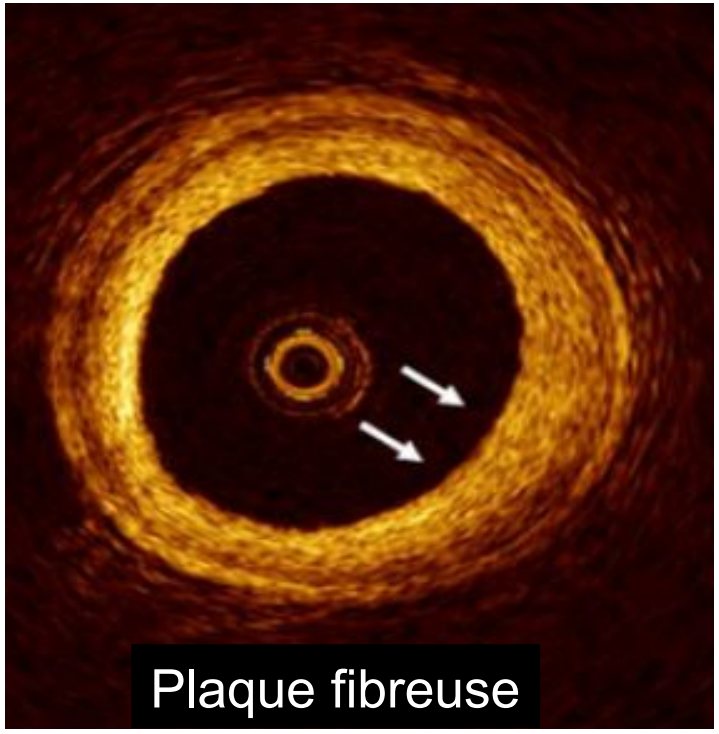
Caractéristiques OCT des Coronaropathies Stables et Instables

Nicolas Meneveau
CHU Jean Minjoz, Besançon

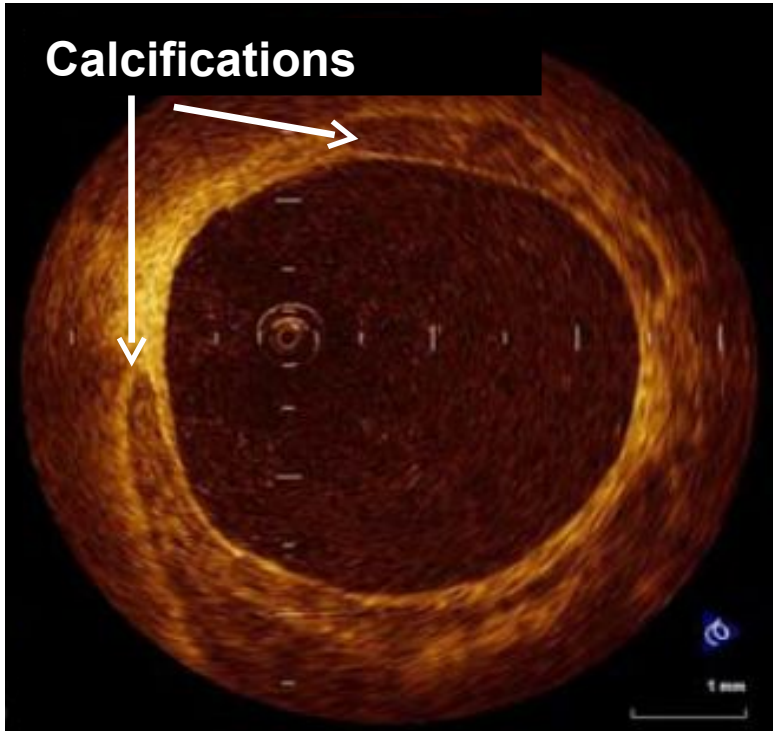
Caractérisation de la plaque stable

Identification de la composition de la plaque

Plaque stable



Haute réflectivité
(pas d'atténuation du signal)

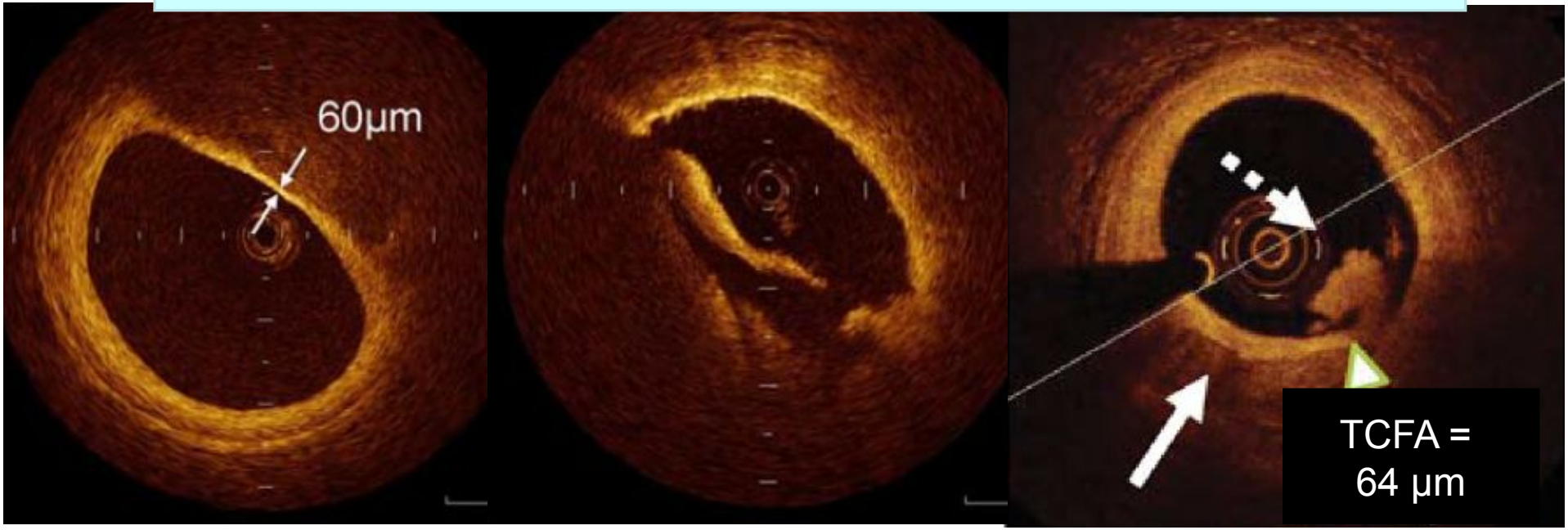


Faible réflectivité
(atténuation du signal et
bordures bien délimitées)

Identification des plaques vulnerables

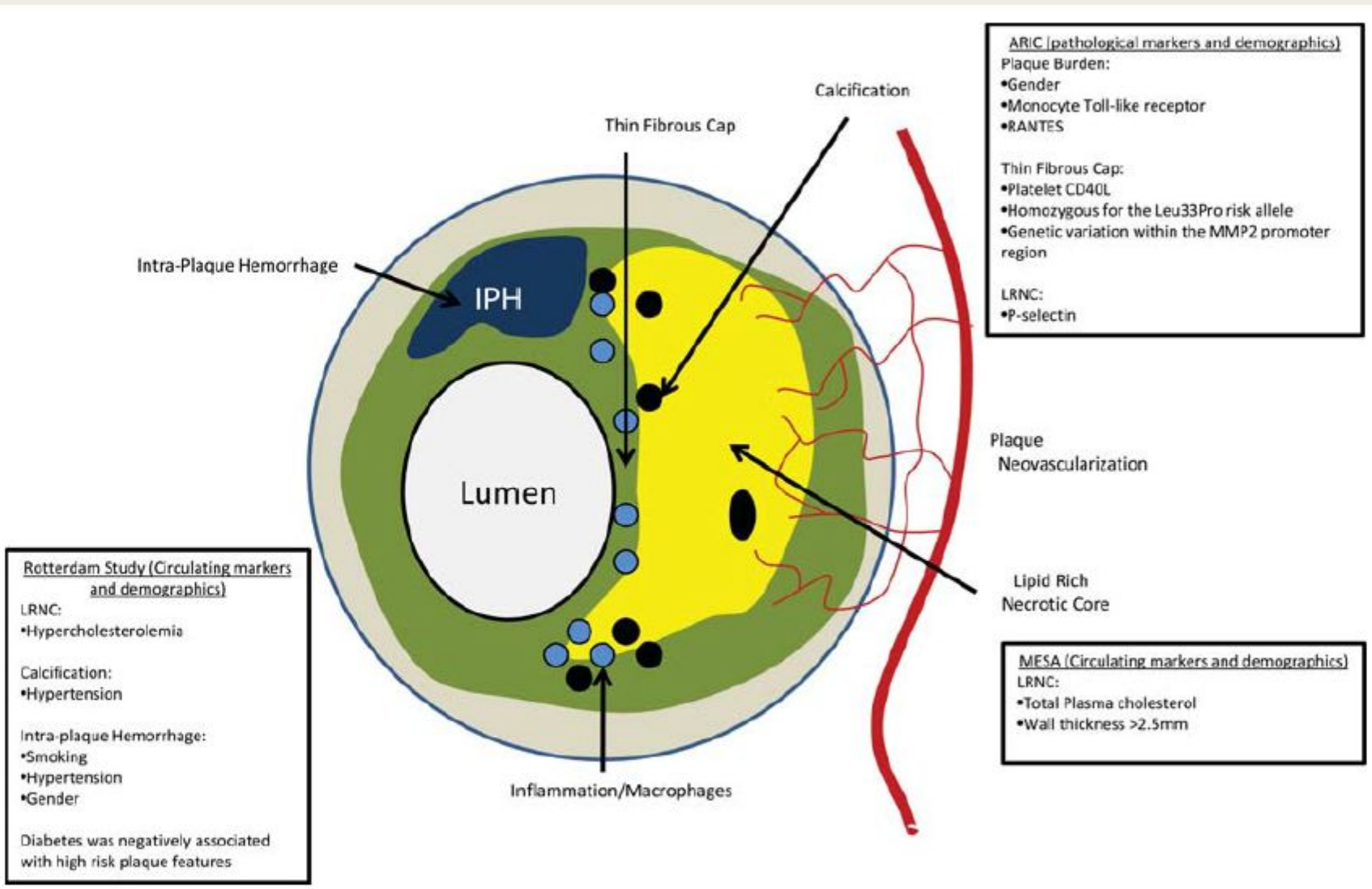
Morphological features of vulnerable plaque :

- Large lipid core,
- TCFA < 65 µm,
- Accumulation of macrophages in the fibrous cap,
- Microchannels,
- Intraluminal thrombi



Kubo T et al. Cardiovasc Interv and Ther 2010;25:2–10. Uemura S et al. Eur Heart J 2012; 33:78–85

Caractérisation de la “plaque vulnérable”



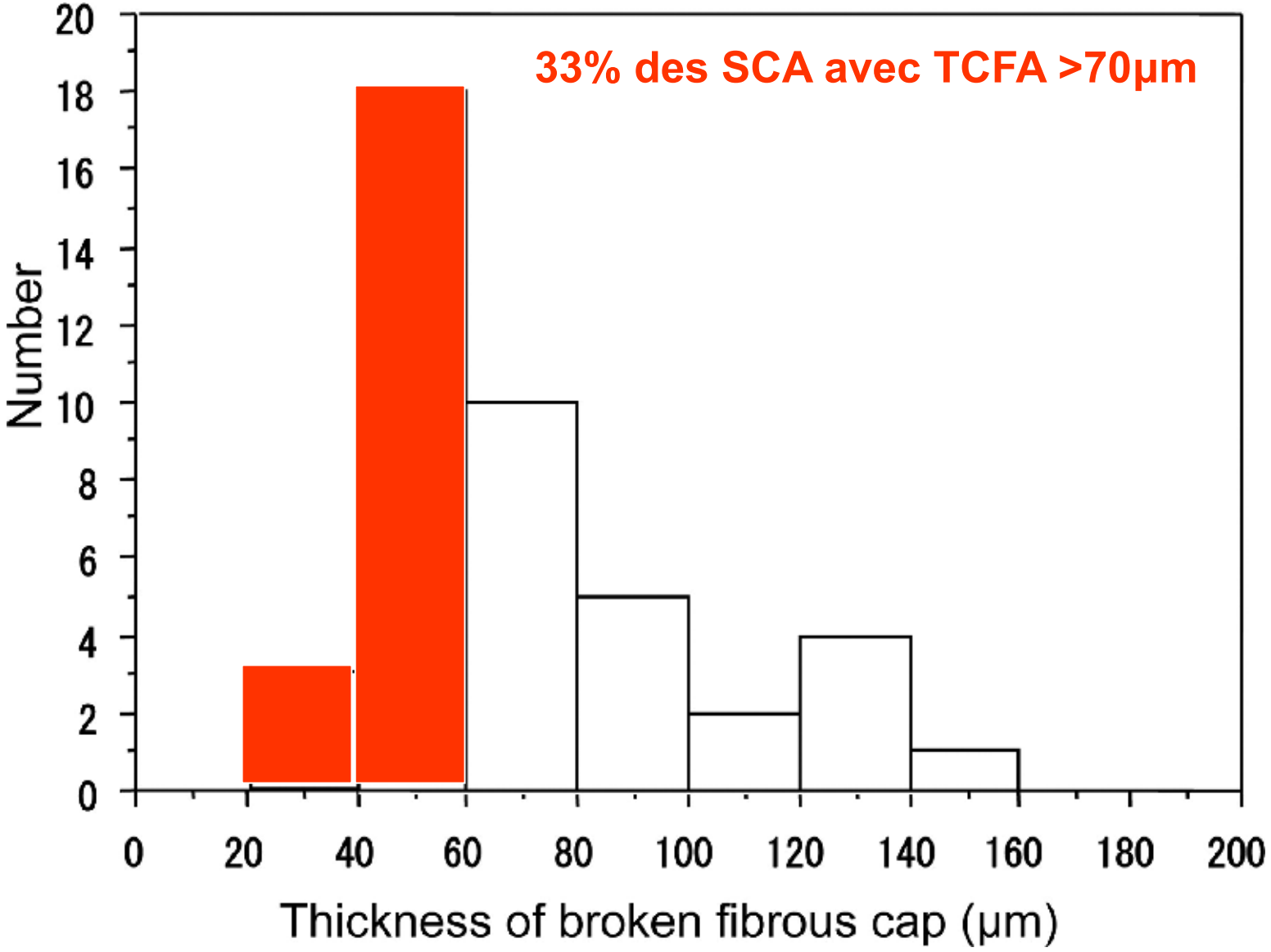
Fayad ZA et al. Eur Heart J 2012;33:160-161.

Caractéristiques associées à un risque de rupture ou de thrombose

- TCFA (<65µm)
- Plaque lipidique
- Centre nécrotique ± hémorragie intraplaque
- Néovascularisation par les vasa vasorum
- Présence de SMCs dans la chappe fibreuse
- Présence de nbses ¢ inflammatoires ds la chappe fibreuse
- Inflammation adventitielle/périvasculaire
- Positive remodeling
- “Spotty” ou microcalcifications

Fog Bentzon et al. Circ Res 2014;114:1852.

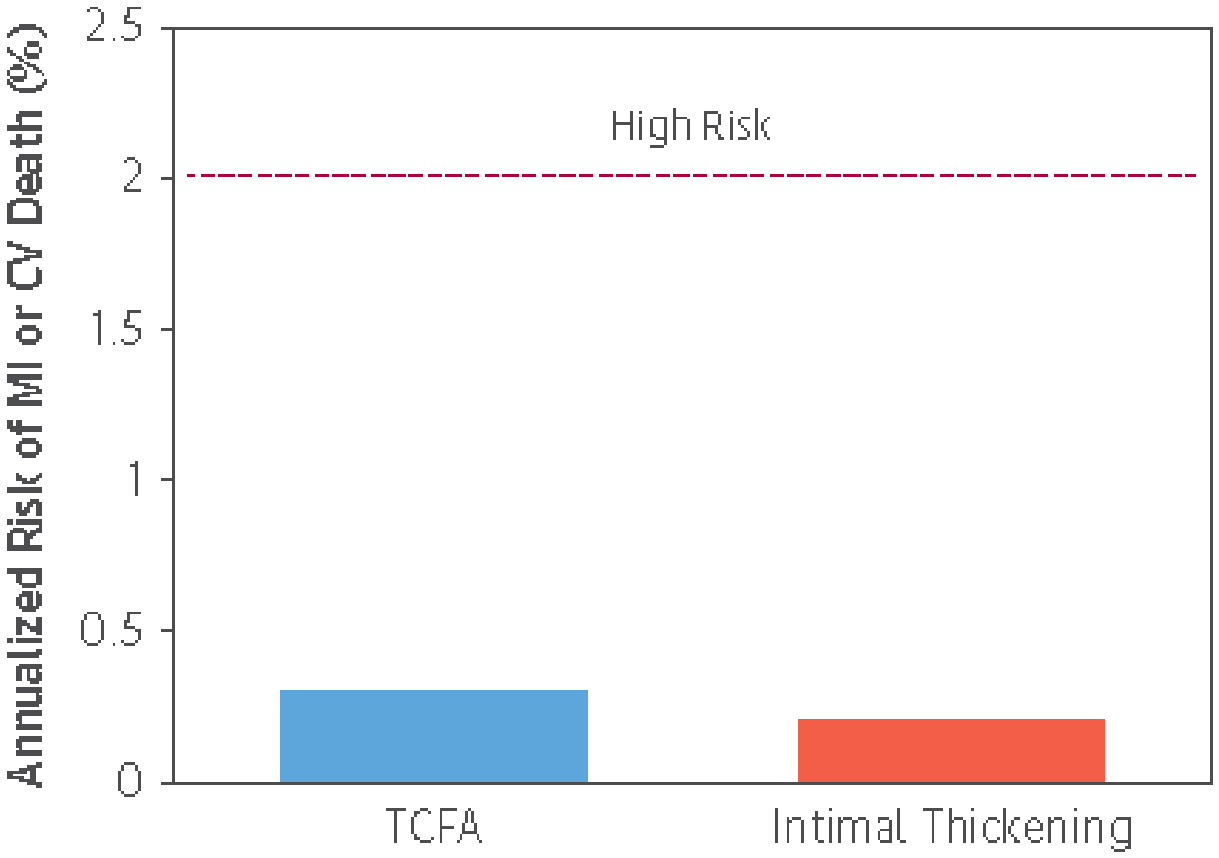
TCFA : oui, mais pas seulement….



Tanaka, A. et al. Circulation 2008;118:2368.

TCFA : oui, mais pas seulement….

Taux d'évt annuel associé à TCFA < 65 µm : 0.06%



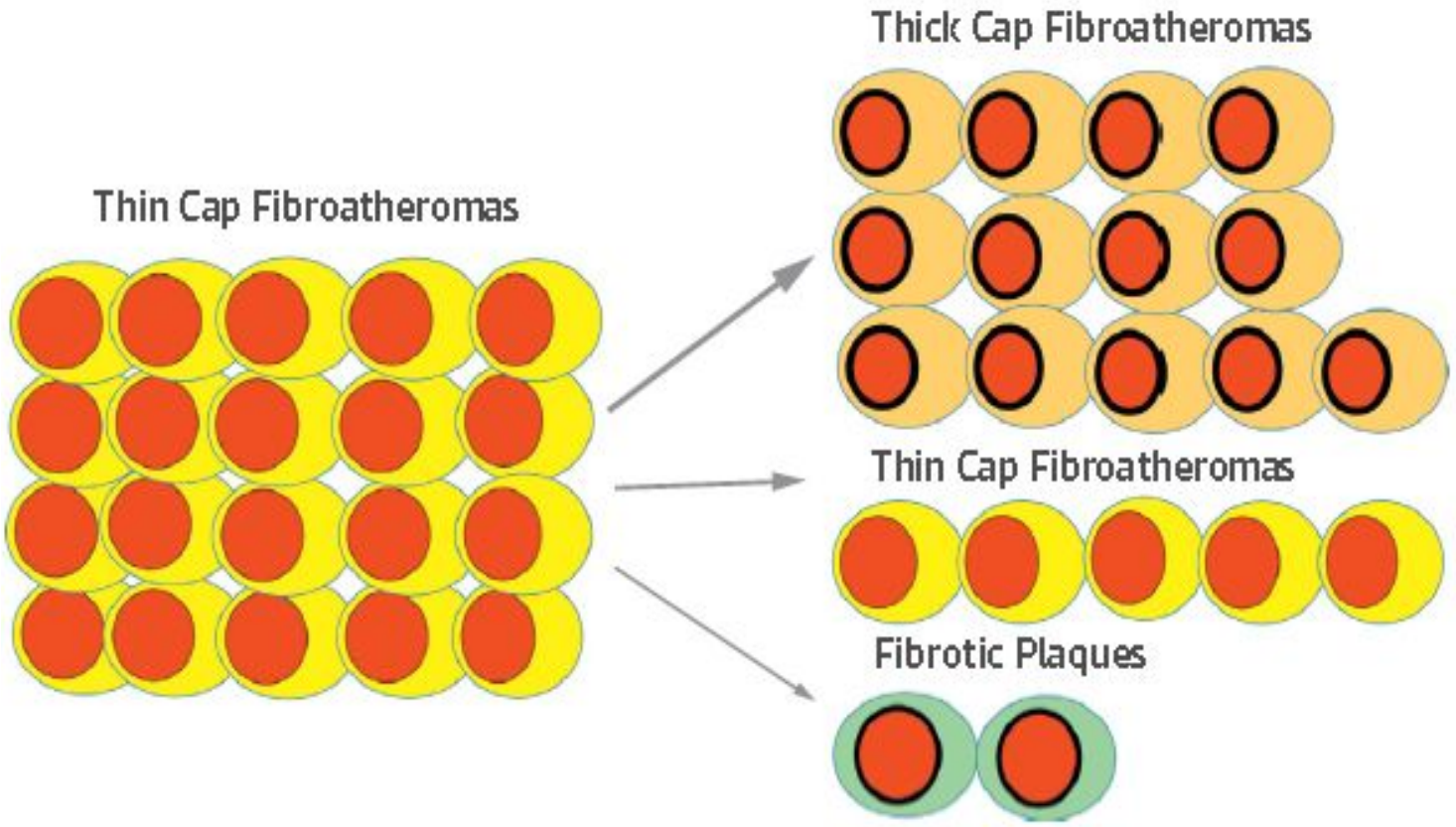
Hlatky MA et al. JACC 2012;60:569-80.

TCFA : oui, mais pas seulement….

Evolution à 12 mois des TCFA :

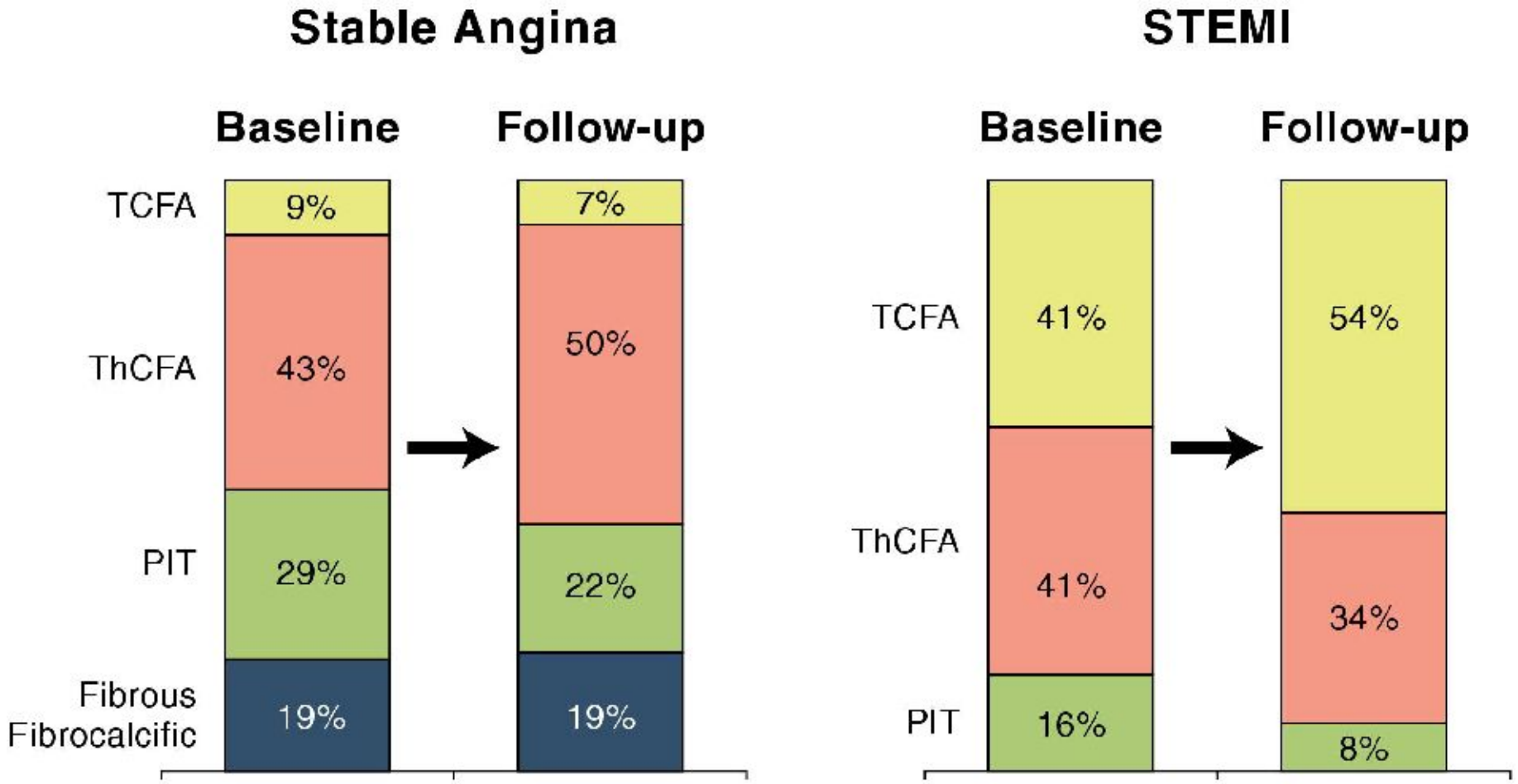
- 25% restent inchangées
- 75% des plaques perdent leur caractère vulnérable

(Épaississement du TCFA ou transformation en plaque fibreuse)



Kubo T et al. JACC 2010;55:1590-7..

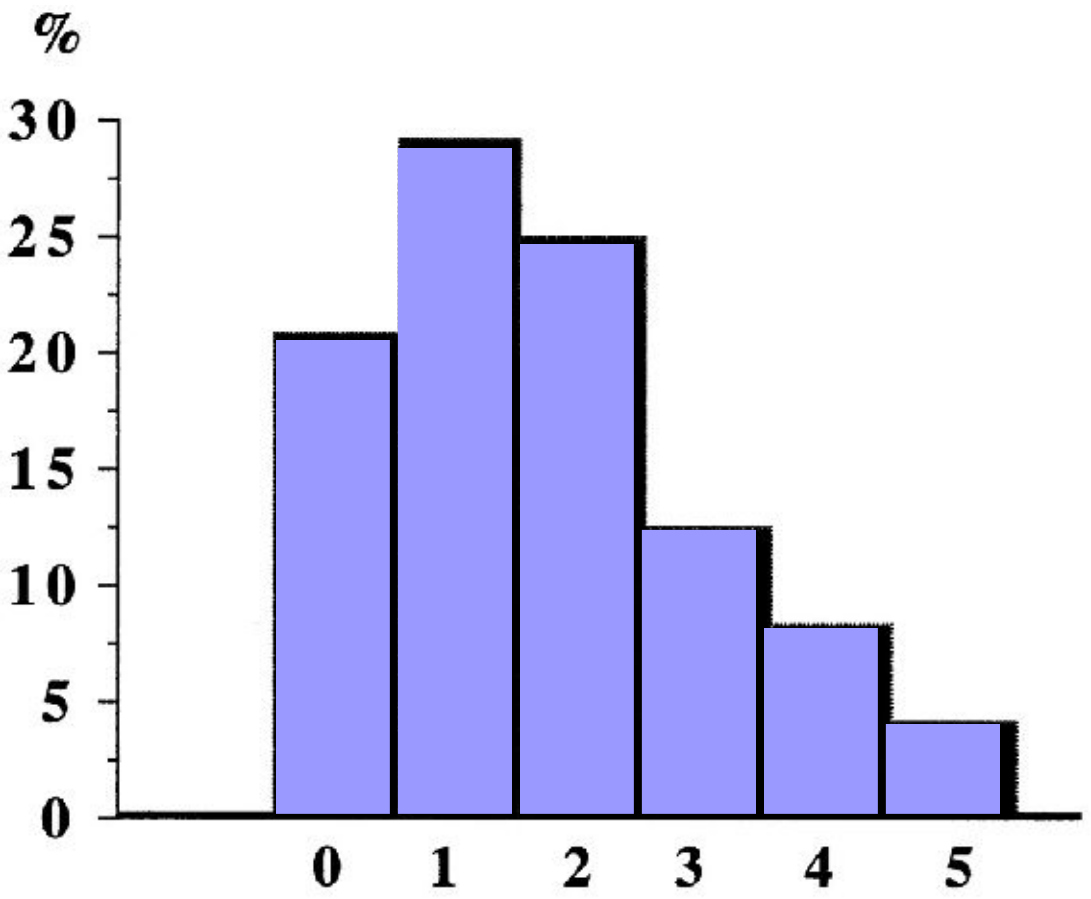
TCFA :
l'évolution varie en fonction du contexte clinique



13 month follow-up

Zhao Z et al JACC Cardiovasc Imag 2013;6:86.

Distribution of coronary plaque ruptures distinct from culprit lesion



Rioufol G, et al. Circulation. 2002;106:804

Caractéristiques associées à un risque de rupture ou de thrombose

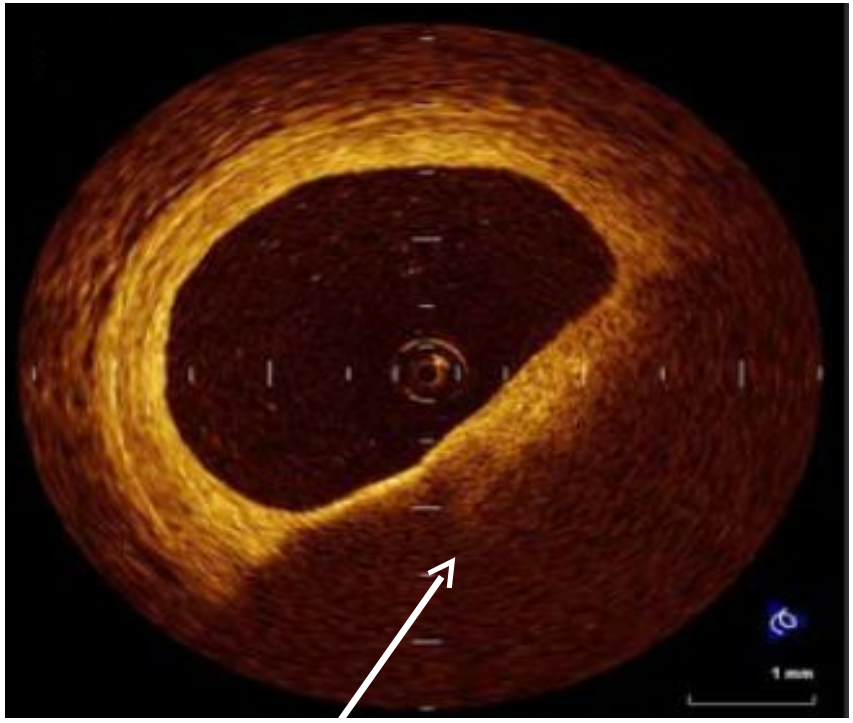
- TCFA (<65µm)
- Plaque lipidique
- Centre nécrotique ± hémorragie intraplaque
- Néovascularisation par les vasa vasorum
- Présence de SMCs dans la chappe fibreuse
- Présence de nbses ¢ inflammatoires ds la chappe fibreuse
- Inflammation adventitielle/périvasculaire
- Positive remodeling
- “Spotty” ou microcalcifications

Fog Bentzon et al. Circ Res 2014;114:1852.

Impact de la composition de la plaque

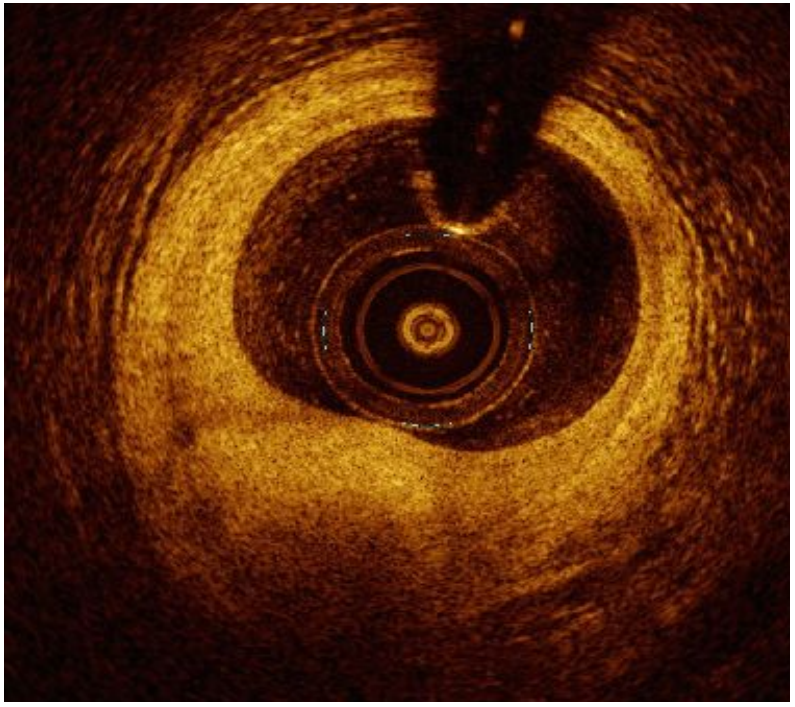
Identification de la composition de la plaque

Plaque vulnérable



Plaque lipidique : faible réflectivité (atténuation du signal et bordures mal limitées)

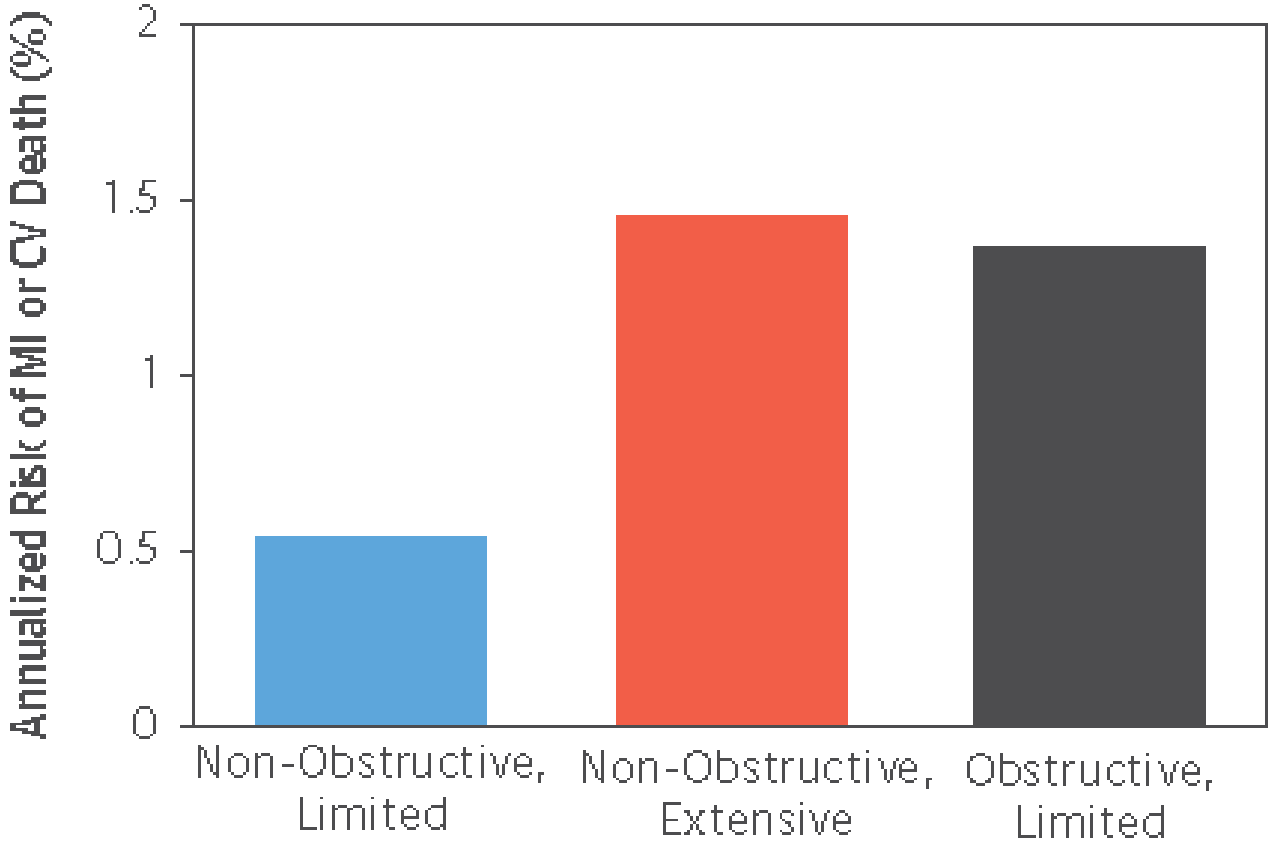
Plaque stable



Plaque fibreuse : haute réflectivité
(pas d'atténuation du signal)

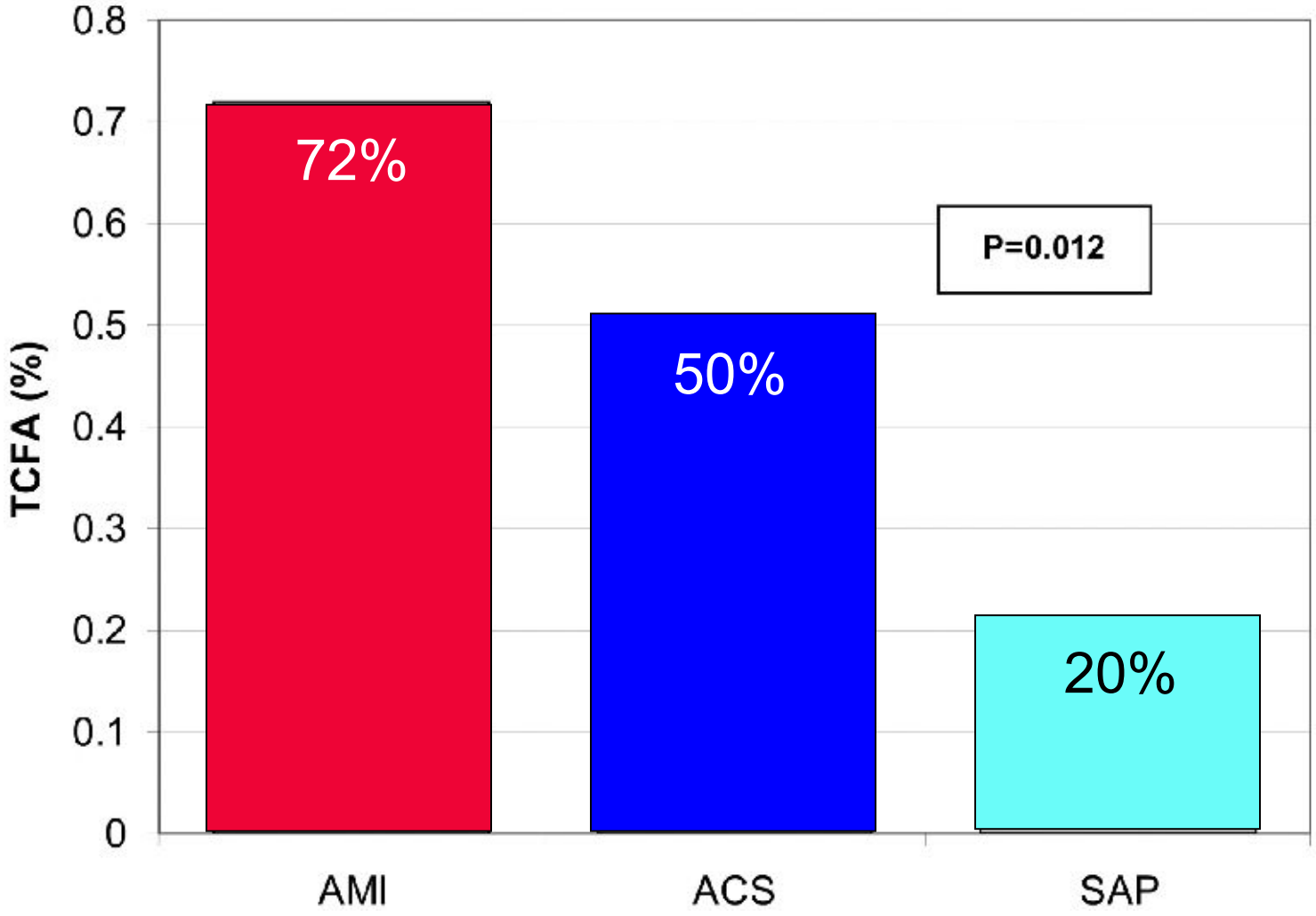
Concept de “plaque burden” (surface de la plaque / surface LEI)

Taux d’evts annuels en fonction du caractère obstructif de la plaque



Bittencourt MS et al. Circ Cardiovasc Imaging 2014;7:282-91.

Importance de l'association plaque lipidique étendue (≥ 2 quadrants) & TCFA ≤ 65 µm.



Jang I et al. Circulation. 2005;111:1551.

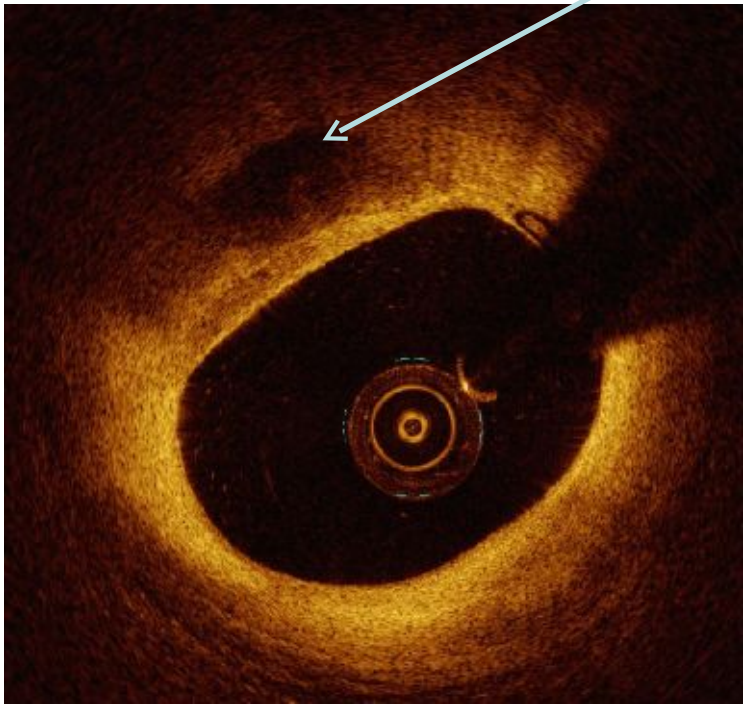
Caractéristiques associées à un risque de rupture ou de thrombose

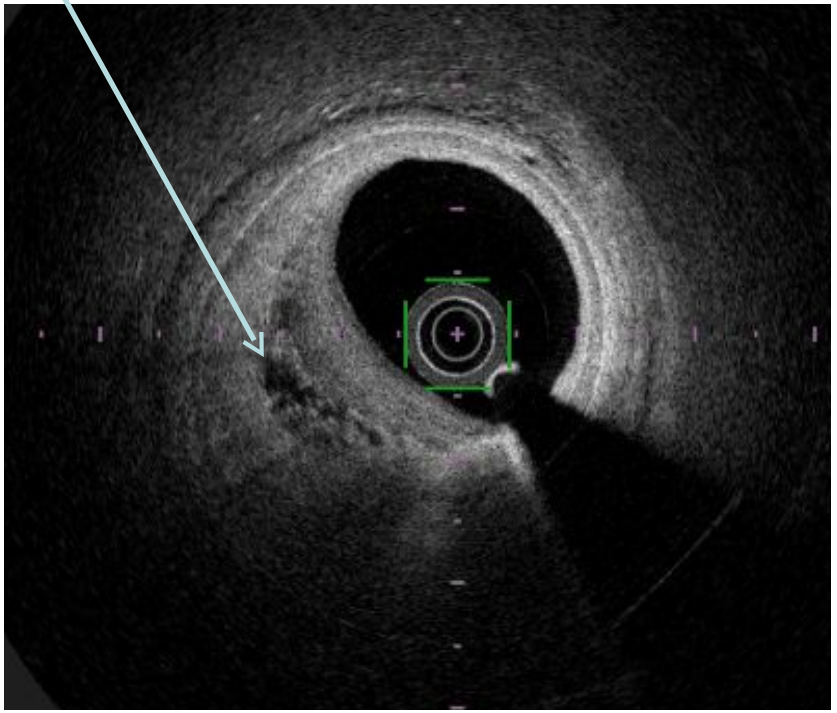
- TCFA (<65µm)
- Plaque lipidique
- Centre nécrotique ± hémorragie intraplaque
- Néovascularisation par les vasa vasorum
- Présence de SMCs dans la chappe fibreuse
- Présence de nbses ¢ inflammatoires ds la chappe fibreuse
- Inflammation adventitielle/périvasculaire
- Positive remodeling
- “Spotty” ou microcalcifications

Fog Bentzon et al. Circ Res 2014;114:1852.

Necrotic core / hémorragie intraplaque

Localisation : intimale-sous intimale

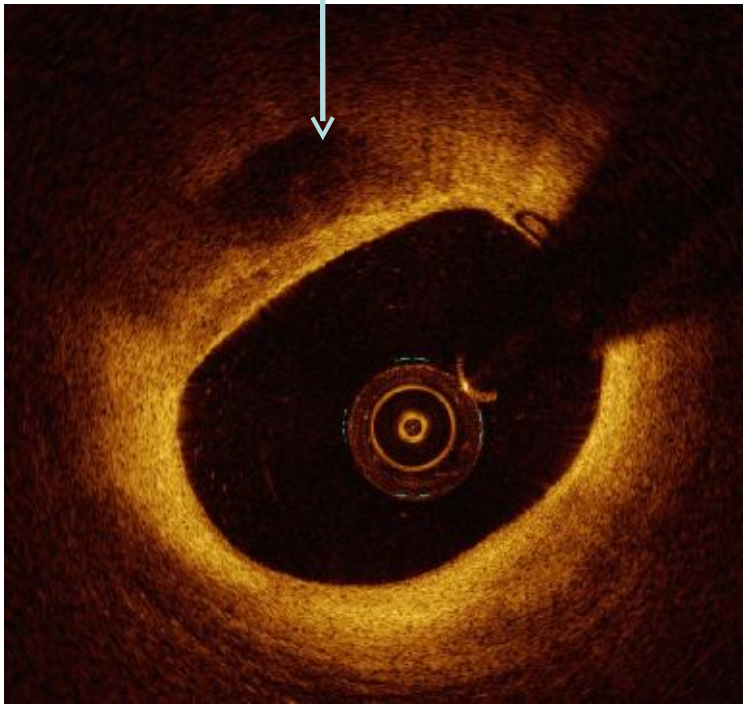




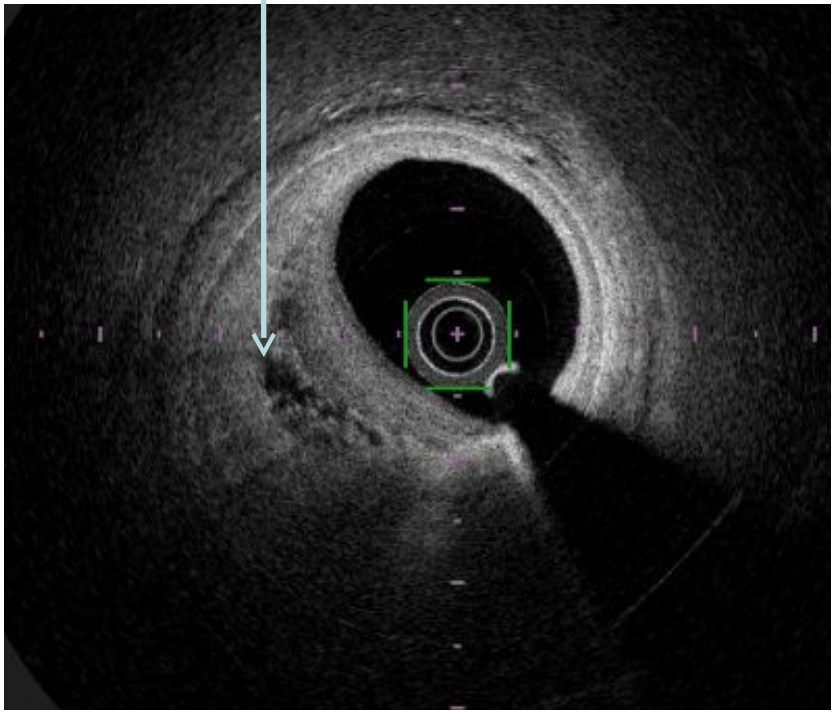
Faible réflectivité (atténuation du signal et bordures ± bien délimitées)

Necrotic core / hémorragie intraplaque

Hémorragie intraplaque?



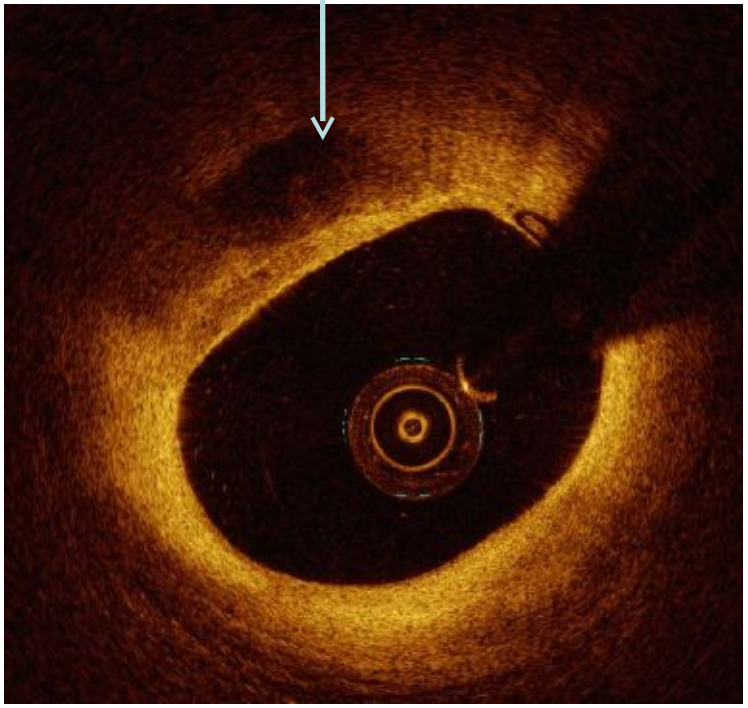
Necrotic core ?



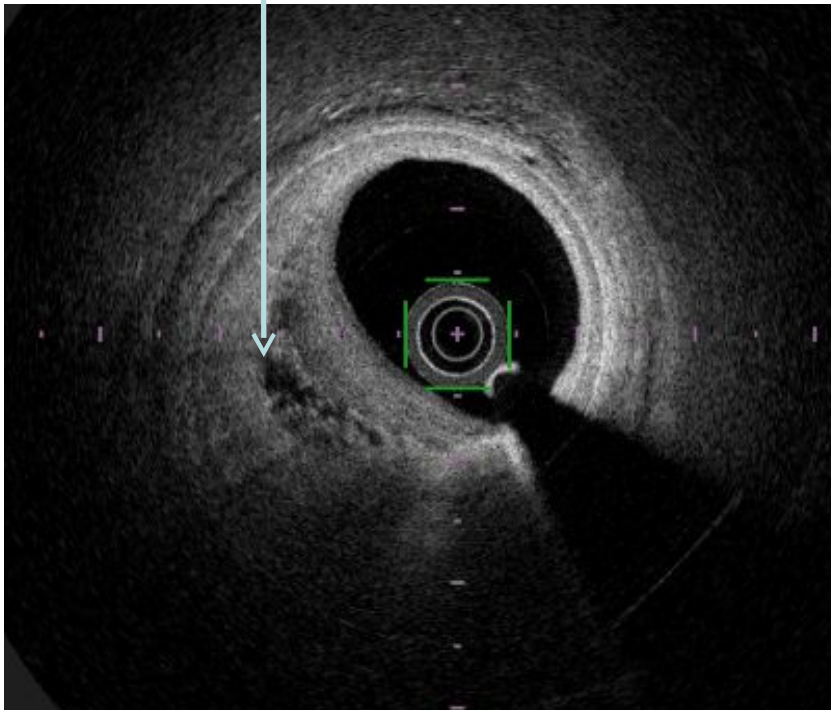
Faible réflectivité (atténuation du signal et bordures ± bien délimitées)

Necrotic core / hémorragie intraplaque

Necrotic core ?



Hémorragie intraplaque?



Faible réflectivité (atténuation du signal et bordures ± bien délimitées)

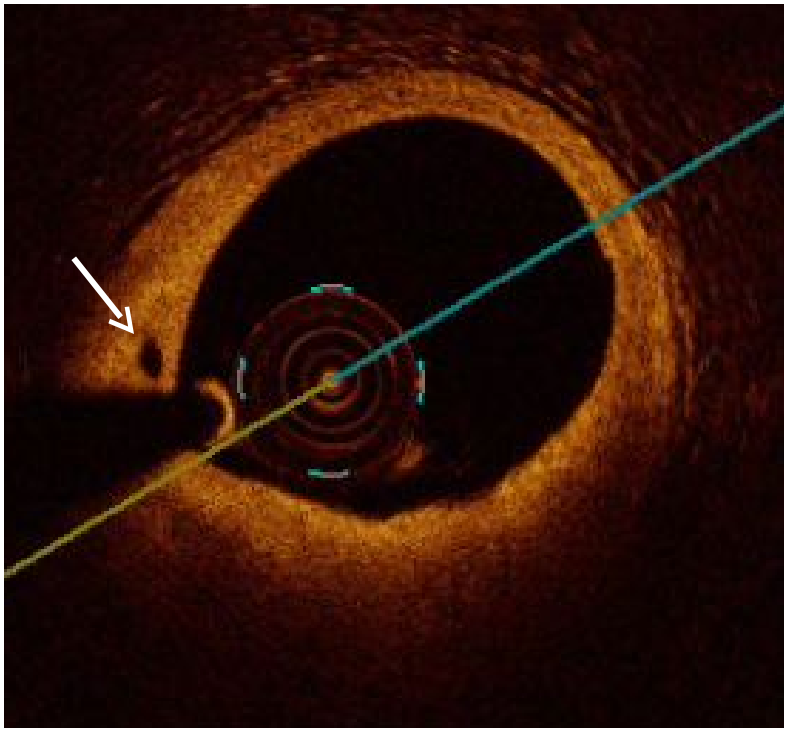
Caractéristiques associées à un risque de rupture ou de thrombose

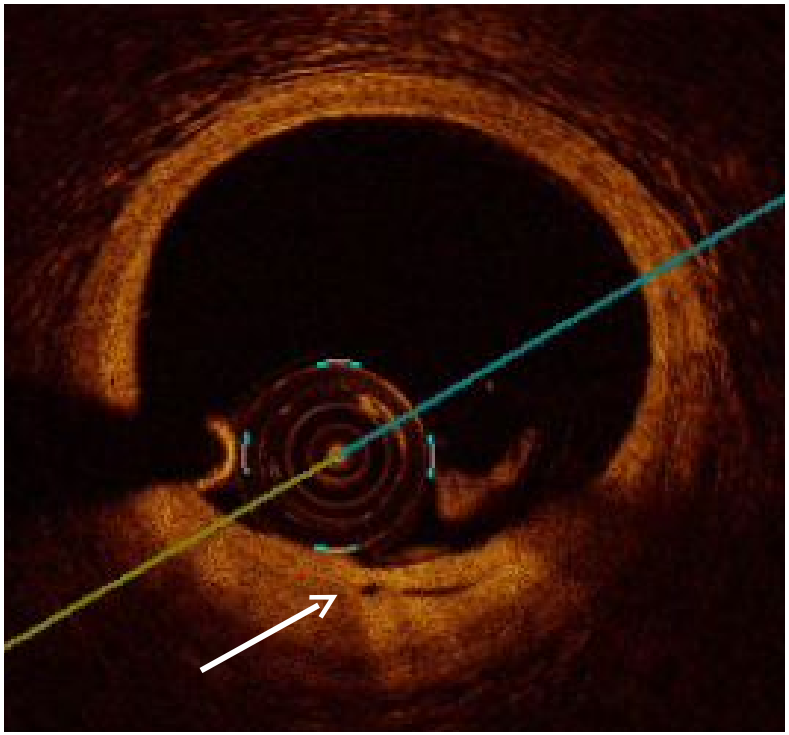
- TCFA (<65µm)
- Plaque lipidique
- Centre nécrotique ± hémorragie intraplaque
- Néovascularisation par les vasa vasorum
- Présence de SMCs dans la chappe fibreuse
- Présence de nbses ¢ inflammatoires ds la chappe fibreuse
- Inflammation adventitielle/périvasculaire
- Positive remodeling
- “Spotty” ou microcalcifications

Fog Bentzon et al. Circ Res 2014;114:1852.

Néovascularisation par les vasa vasorum

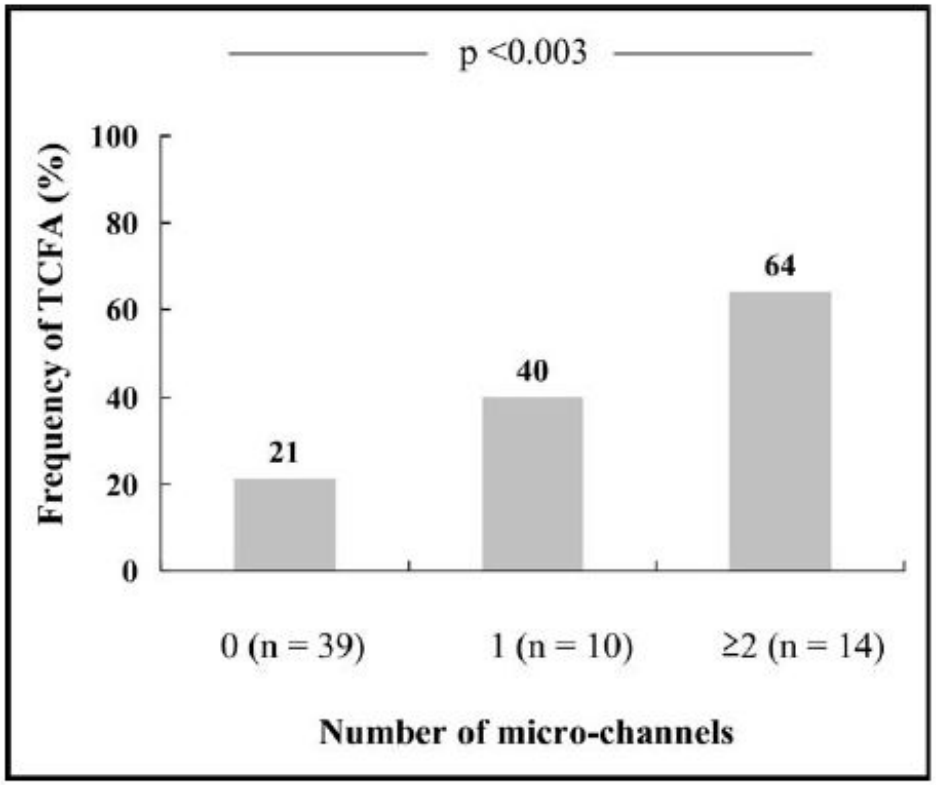
Néovaisseaux intimaux

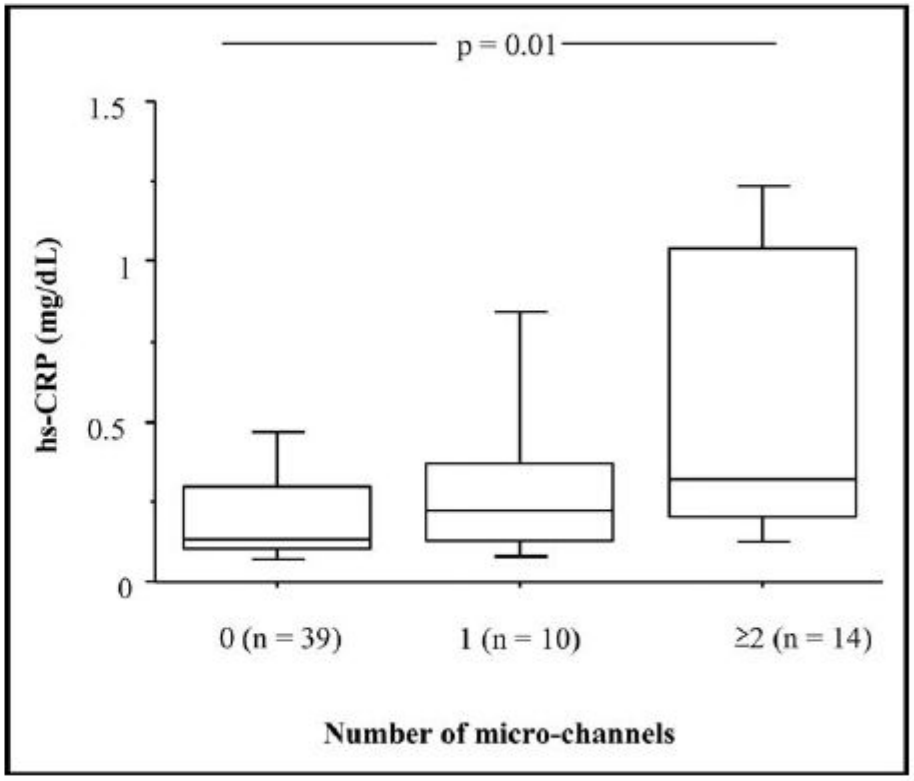




Néovascularisation et vulnérabilité

La présence de néovaisseaux intimaux est associée à un risque accru de vulnérabilité





Kitabata H et al. Am J Cardiol 2010;105:1673–1678.

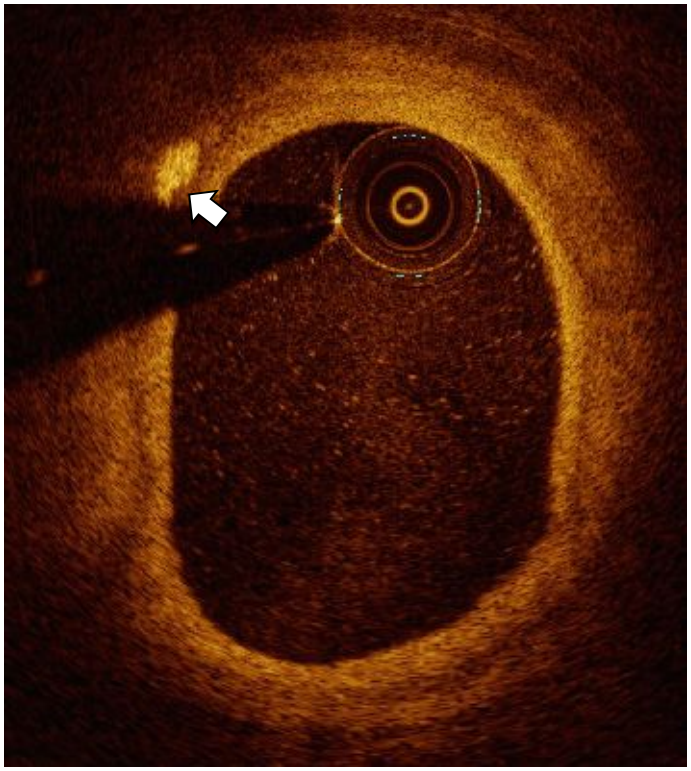
Caractéristiques associées à un risque de rupture ou de thrombose

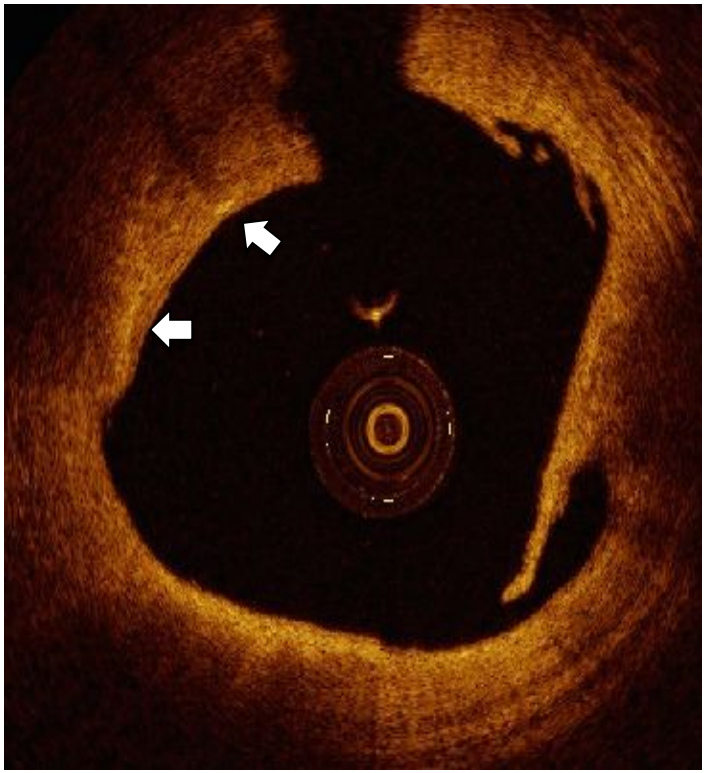
- TCFA (<65µm)
- Plaque lipidique
- Centre nécrotique ± hémorragie intraplaque
- Néovascularisation par les vasa vasorum
- Présence de SMCs dans la chappe fibreuse
- Présence de nbses ¢ inflammatoires ds la chappe fibreuse
- Inflammation adventitielle/périvasculaire
- Positive remodeling
- “Spotty” ou microcalcifications

Fog Bentzon et al. Circ Res 2014;114:1852.

Dépots de macrophages

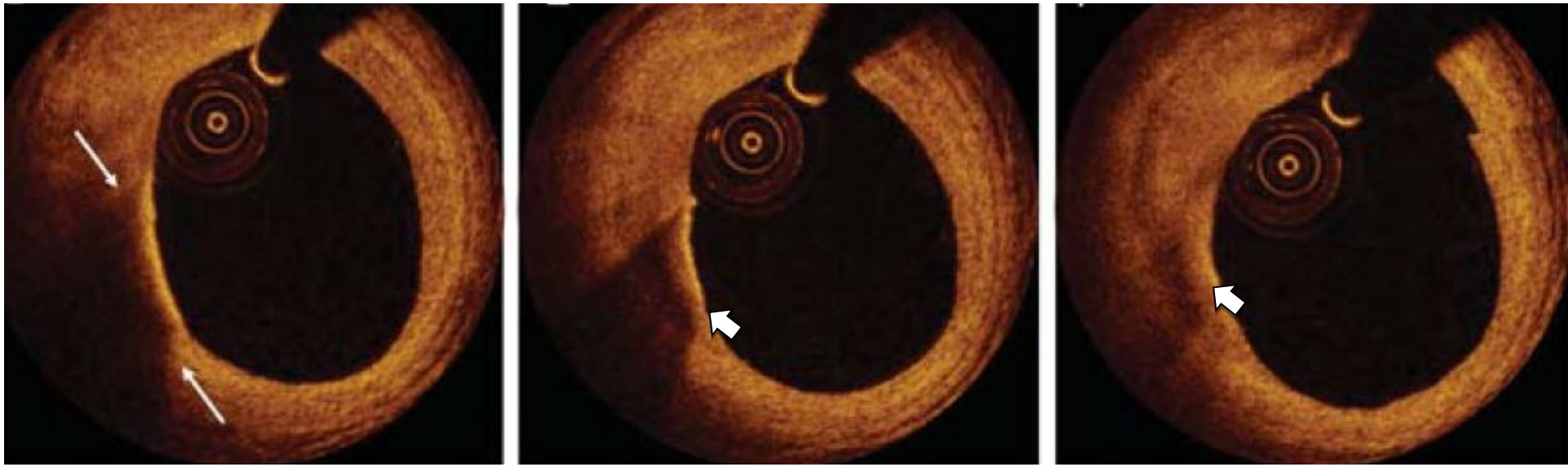
Accumulation de cellules inflammatoires intraplaques :
Hypersignal en bandes (diagnostic différentiel : dépots de cholestérol, artéfacts, LEI)





Dépots de macrophages

Accumulation de cellules inflammatoires préférentiellement dans la chape fibreuse :
Hypersignal en bandes (diagnostic différentiel : dépots de cholestérol, artéfacts, LEI)



Plaque Vulnérable

≠

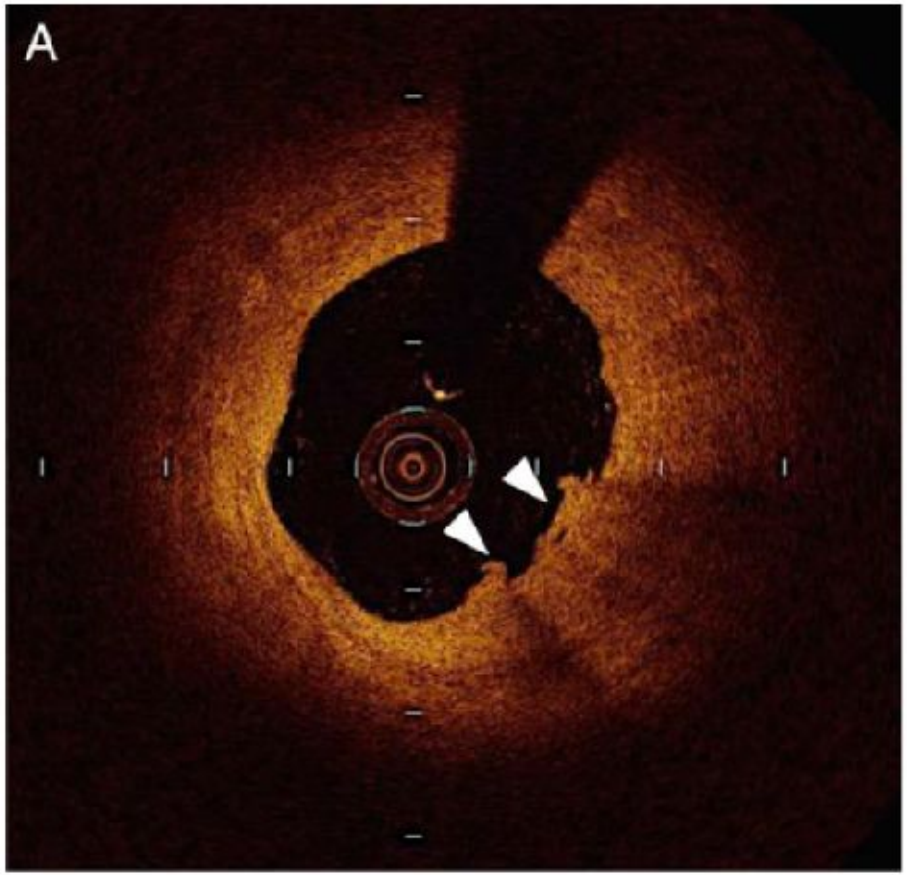
Rupture de Plaque

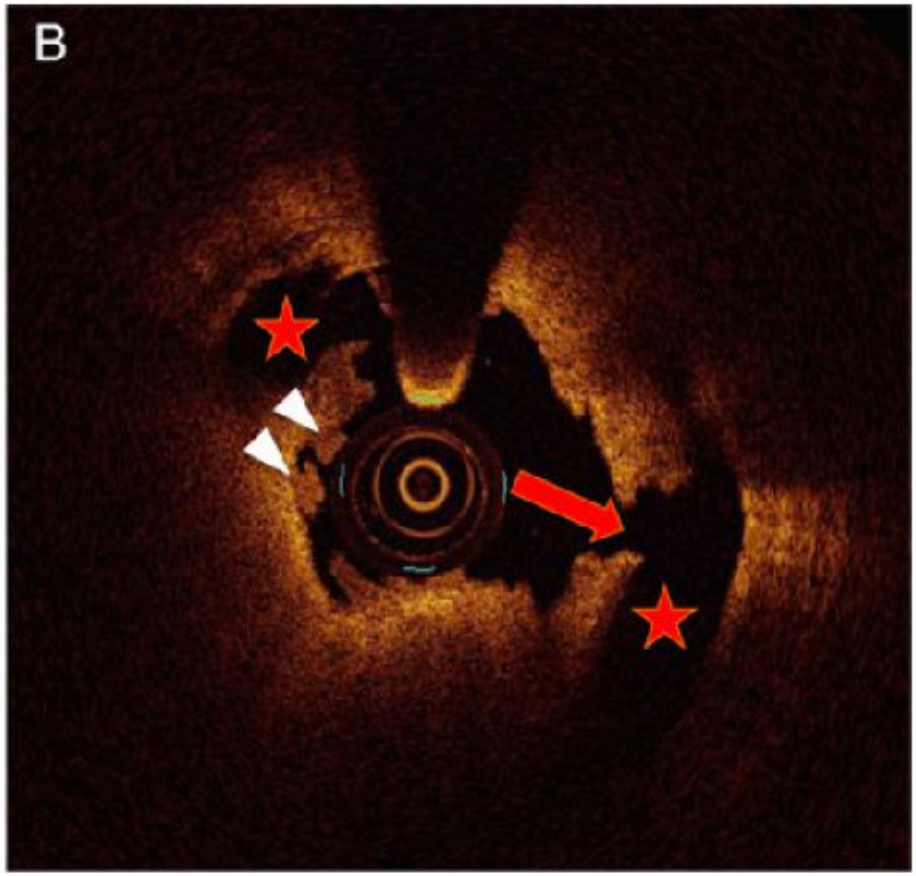
Impact Pc de la rupture de plaque vs TCFA intact

139 Pts avec SCA (66% ST- et 34% ST+)

57 Pts (41%) avec TCFA intact

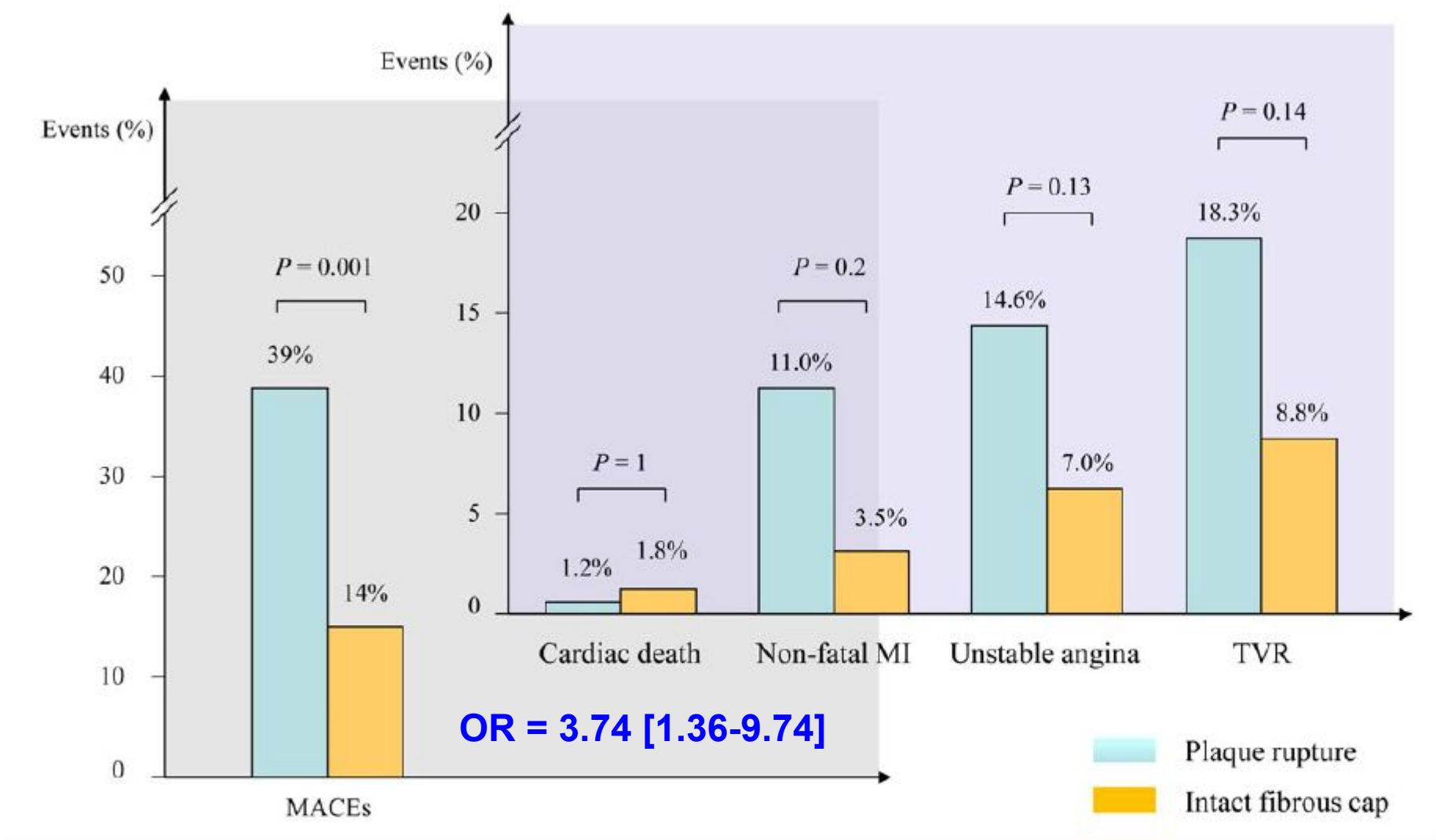
82 Pts (59%) avec RP





Niccoli G et al. Eur Heart J 2015.

Impact Pc de la rupture de plaque vs TCFA intact à 3 ans



Niccoli G et al. Eur Heart J 2015.

Identification des plaques vulnérables

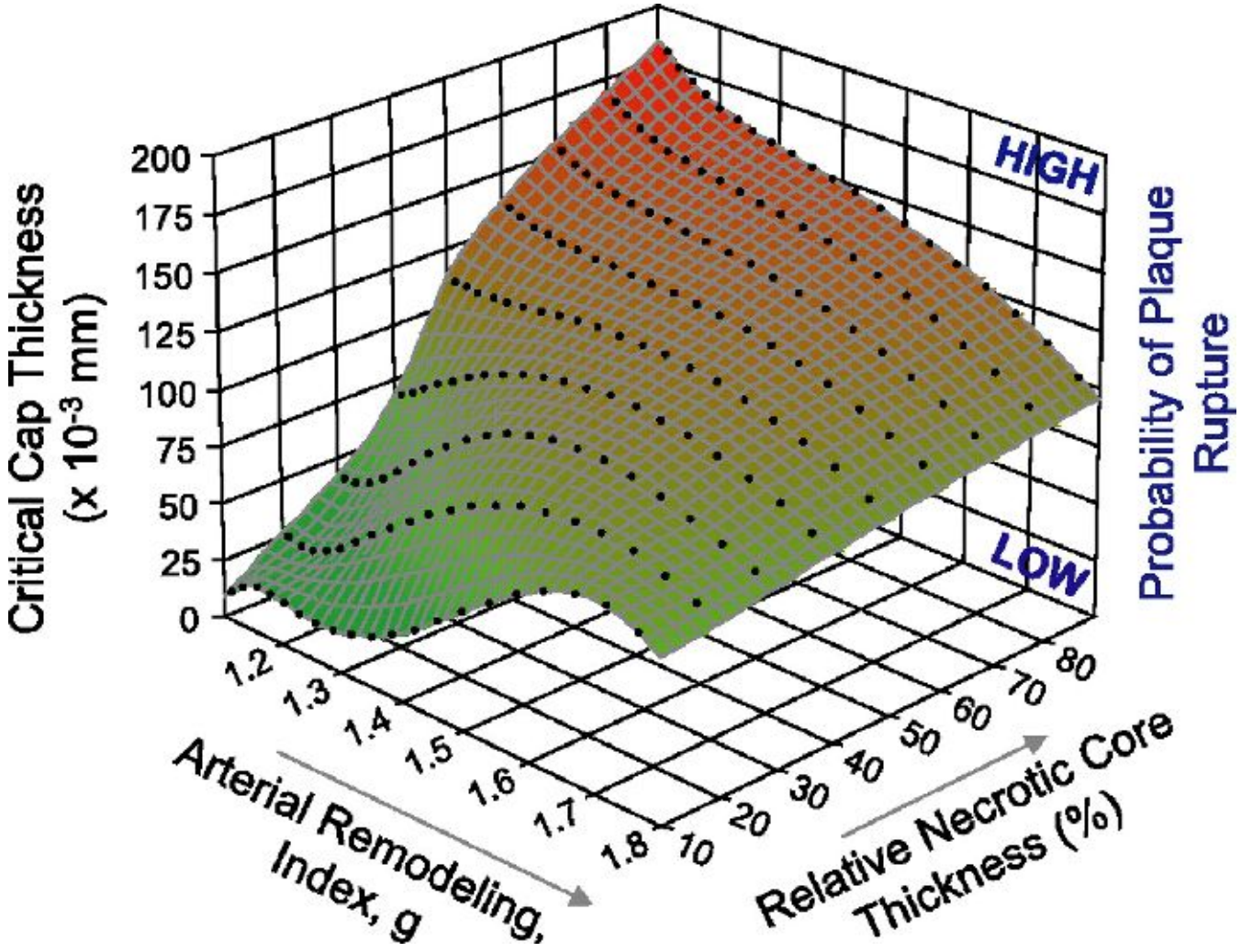
- 53 CAD patients undergoing PCI
- 69 non significant coronary plaques (%DS < 50%). 7-month FUP.

Baseline characteristics	Progression (N=13) (19%)	Non-progression	P
Intimal laceration	61.5%	8.9%	< 0.01
Microchannels	76.9%	14.3%	< 0.01
Lipid pools	100%	60.7%	0.02
TCFA	76.9%	14.3%	< 0.01
Macrophage images	61.5%	14.3%	< 0.01
Intraluminal thrombi	30.8%	1.8%	< 0.01

- Regression analysis : TCFA (OR = 20; p<0.01) & microchannel images (OR = 20; p<0.01) correlated with subsequent luminal progression

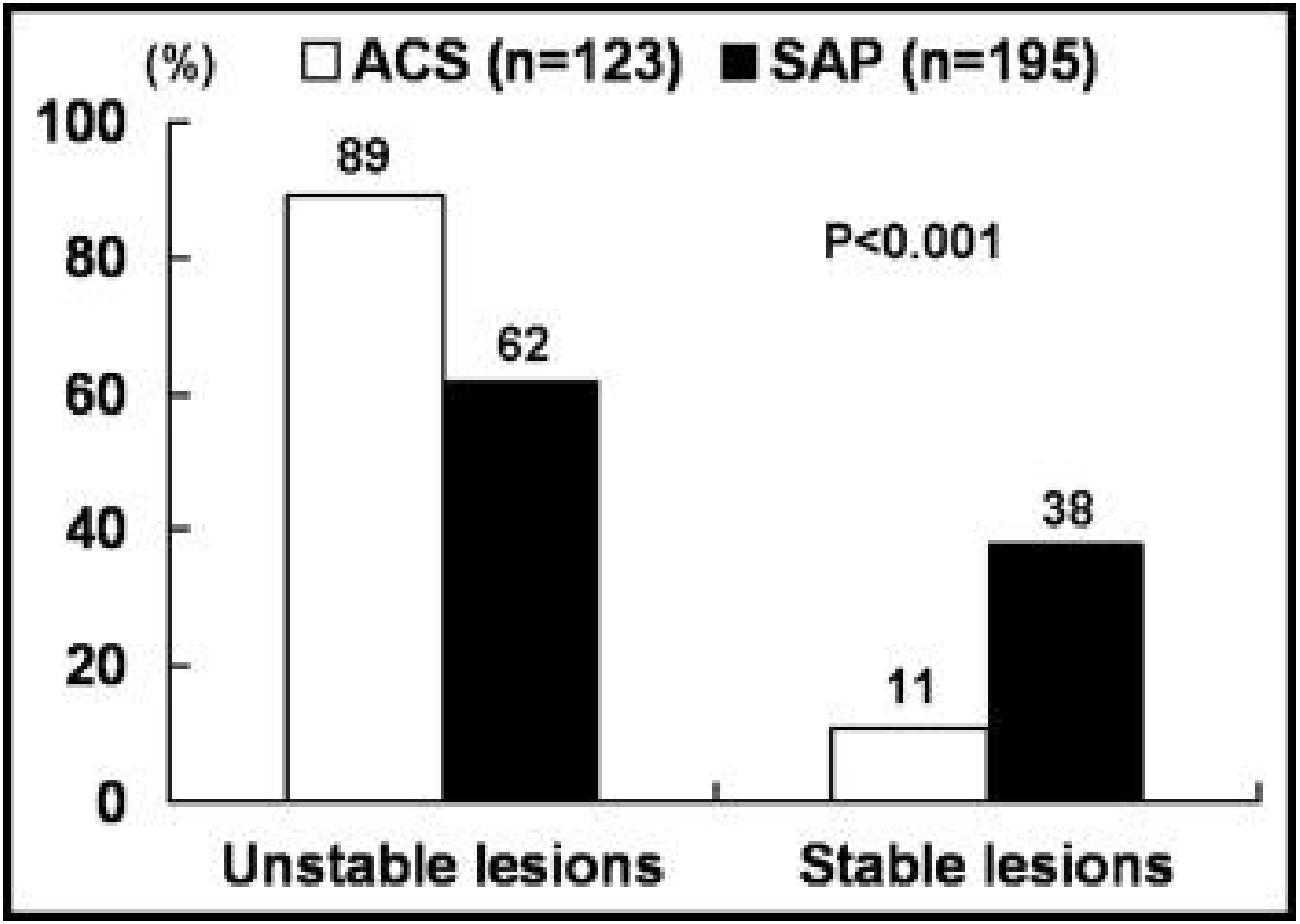
Uemura S et al. Eur Heart J 2012; 33:78–85.

Rupture de plaque : épaisseur du TCFA, surface du centre nécrotique et remodellage vasculaire



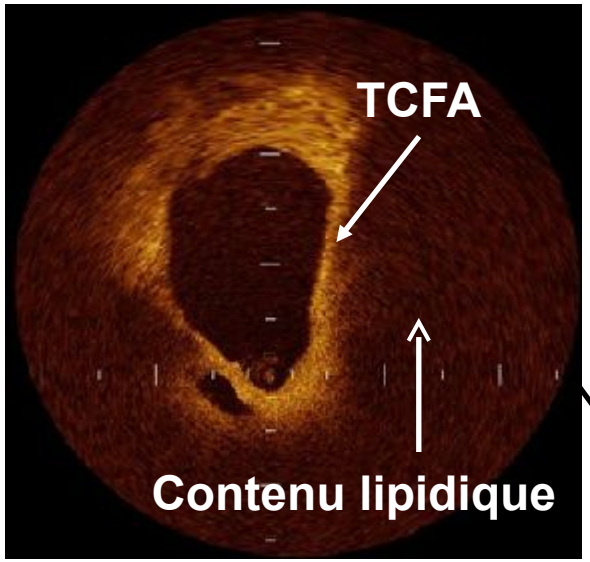
Ohayon, J. et al. Am J Physiol Heart Circ Physiol 2008:295;H717.

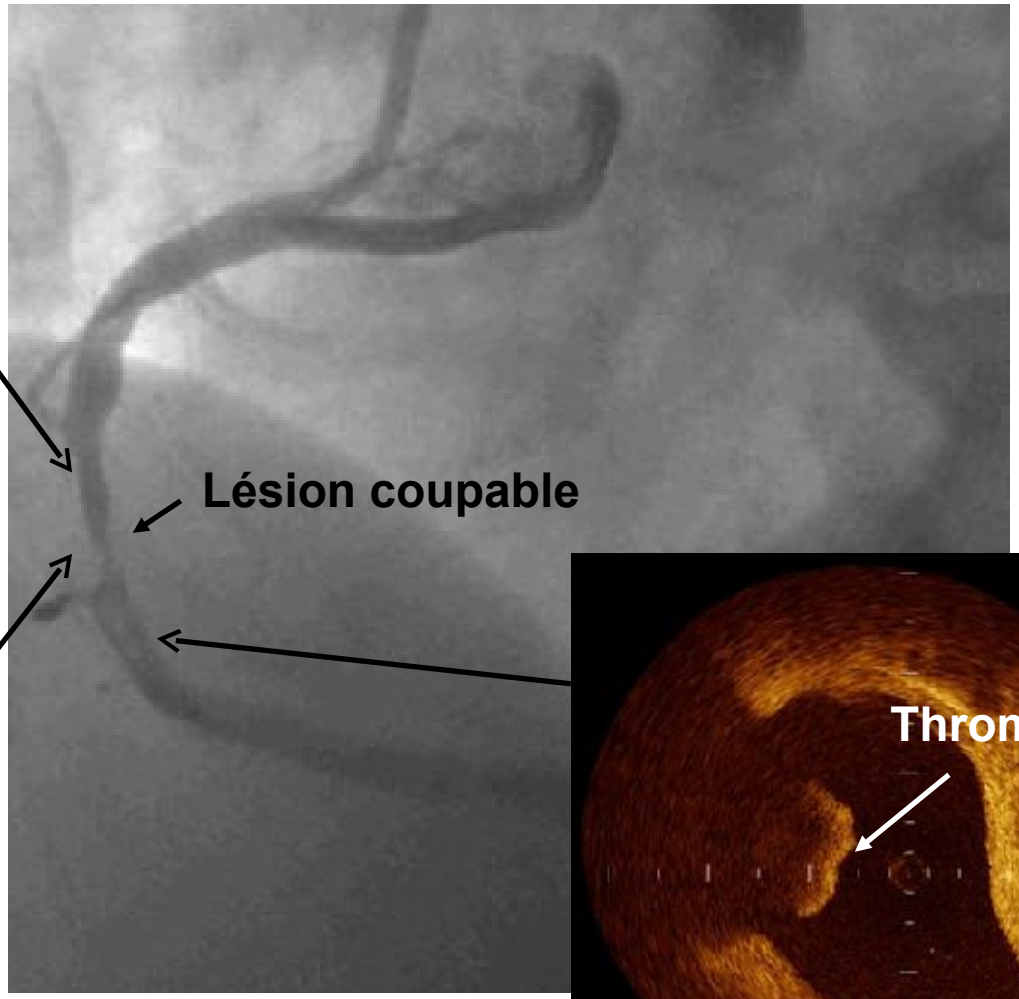
Incidence of unstable (VH-TCFA or ruptured plaque) and stable plaques in ACS vs SAP.

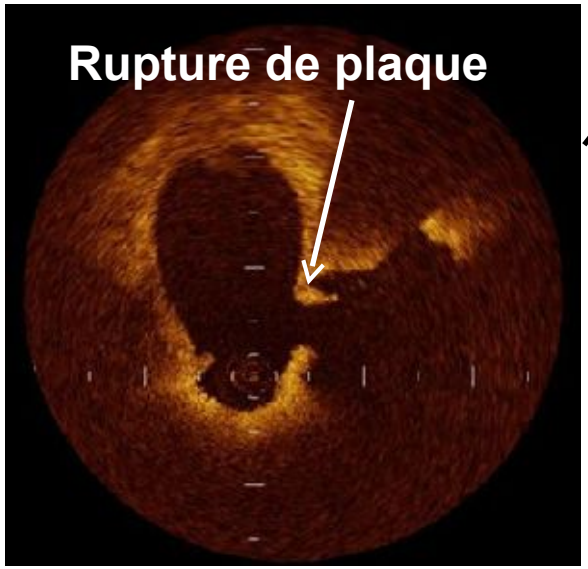


Hong MK et al Am J Cardiol 2007;100:953

TCFA + Plaque lipidique + Rupture de plaque + Thrombus = SCA



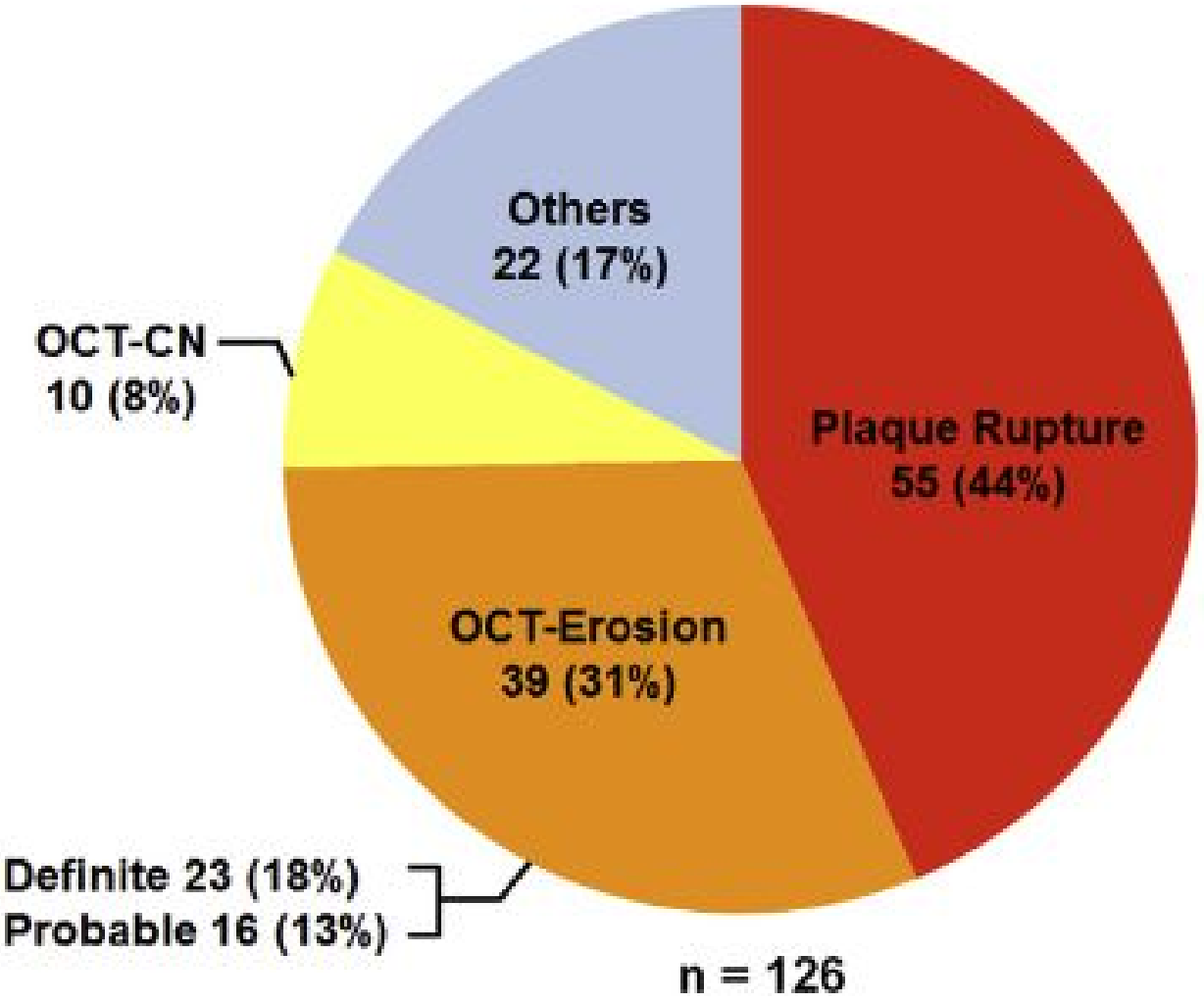




La rupture de Plaque

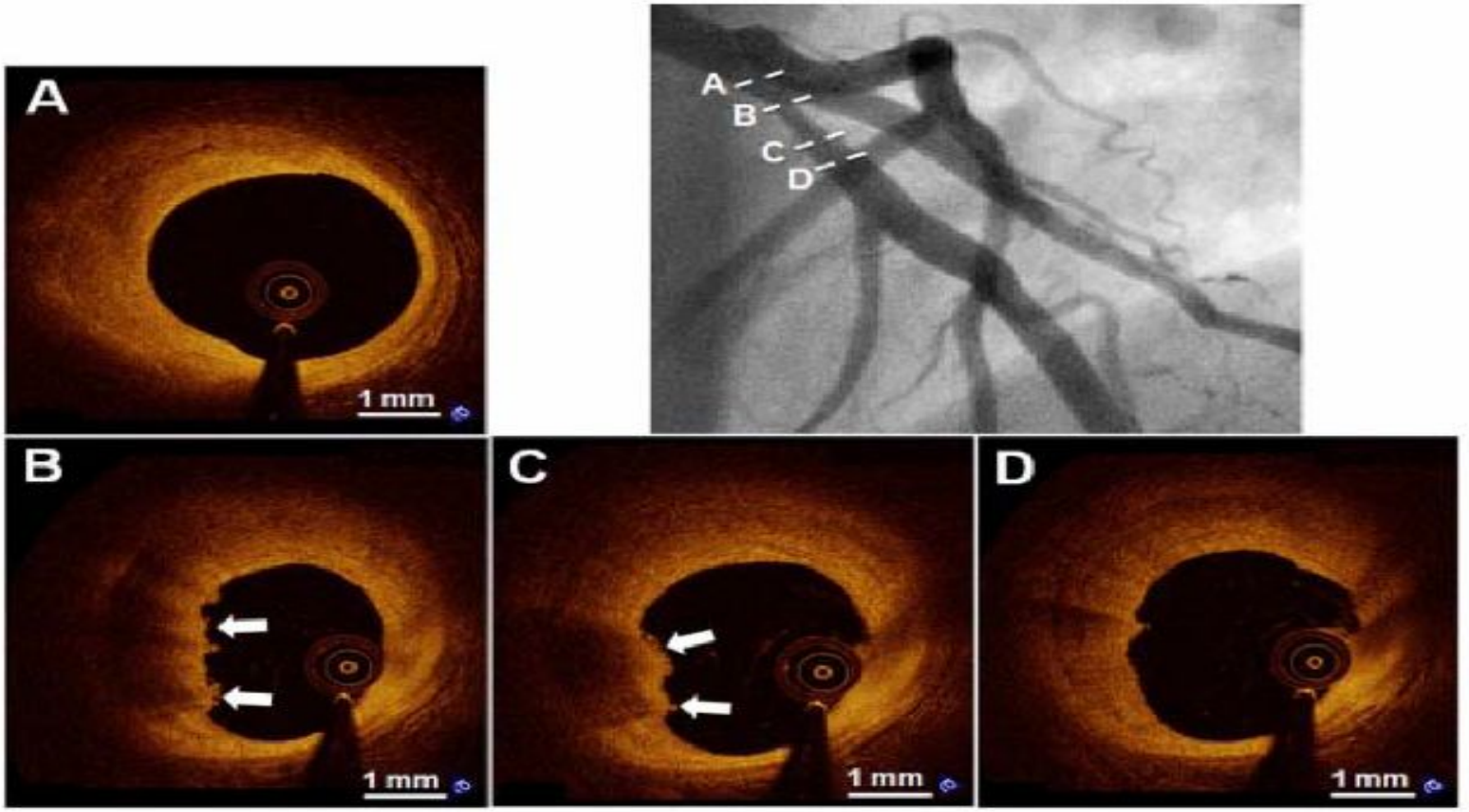
n’explique pas tout ….

Incidence des ruptures de plaque, érosions de plaques, nodules Ca++ chez les pts avec SCA



Jia H et al JACC 2013;62:1748-58.

Erosion de Plaque (certaine ?) et SCA

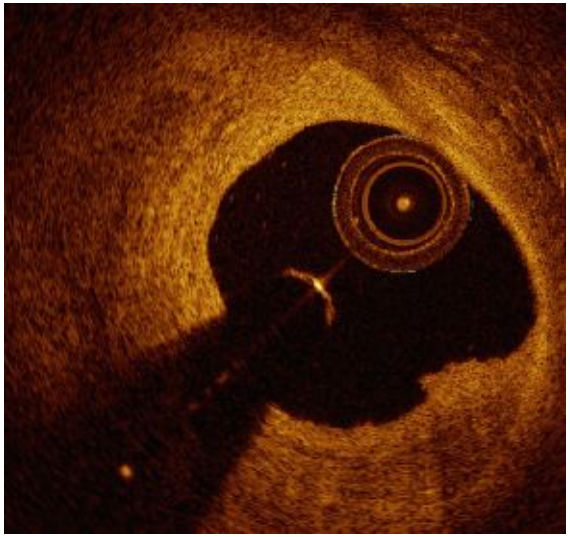


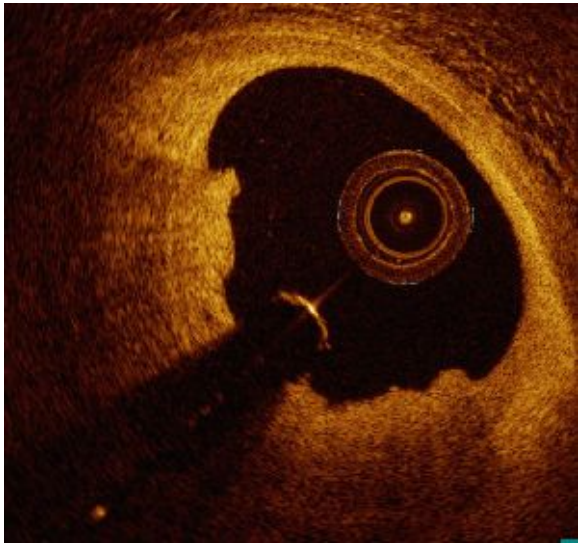
Définition : irrégularité de la surface intraluminale en l'absence de rupture de la chape fibreuse, avec fréquente présence de thrombus (Tearney et al. JACC 2012)

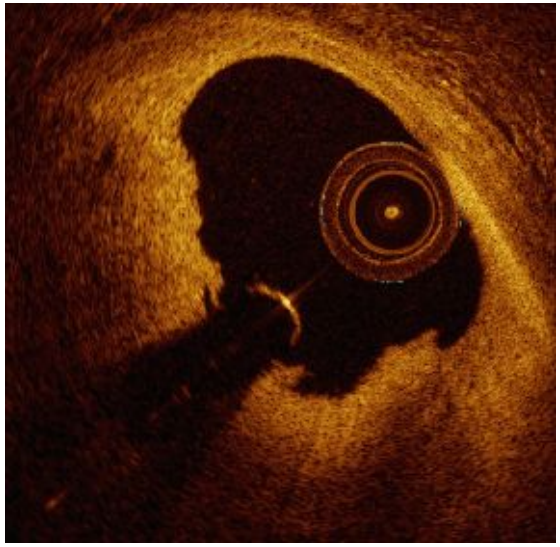
Jia H et al JACC 2013;62:1748-58.

Erosion de Plaque (certaine ?)

Définition : irrégularité de la surface intraluminale en l'absence de rupture de la chape fibreuse, avec fréquente présence de thrombus (Tearney et al. JACC 2012)

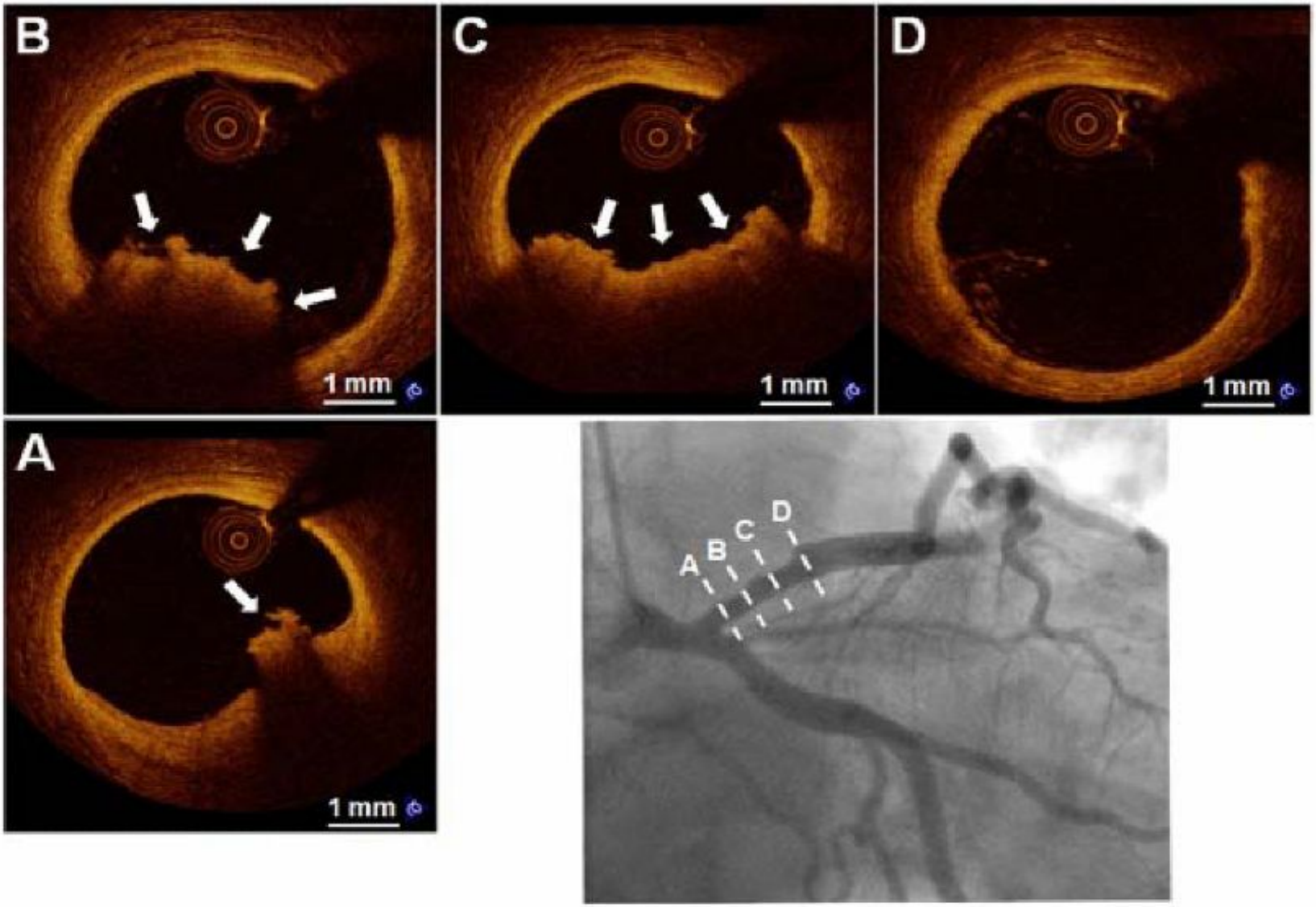






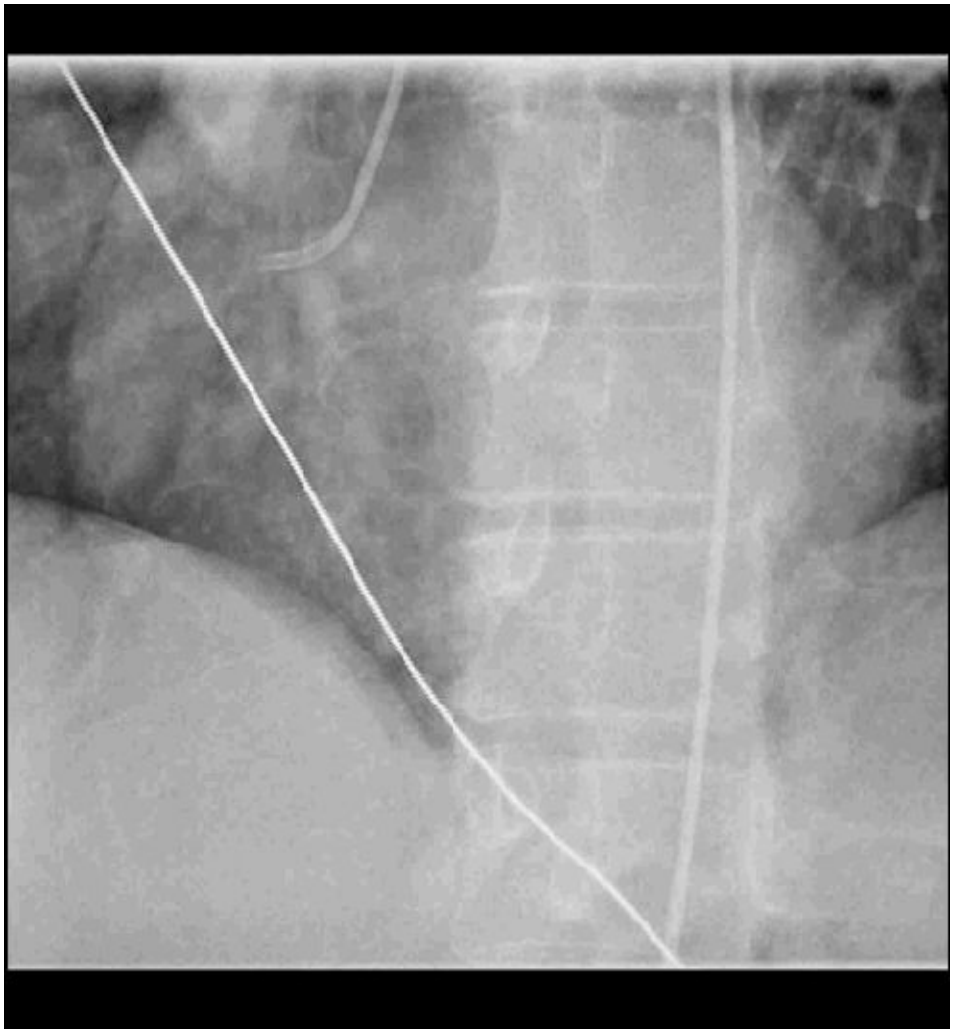
Nicolas Amabile

Erosion de Plaque (probable ?) et SCA



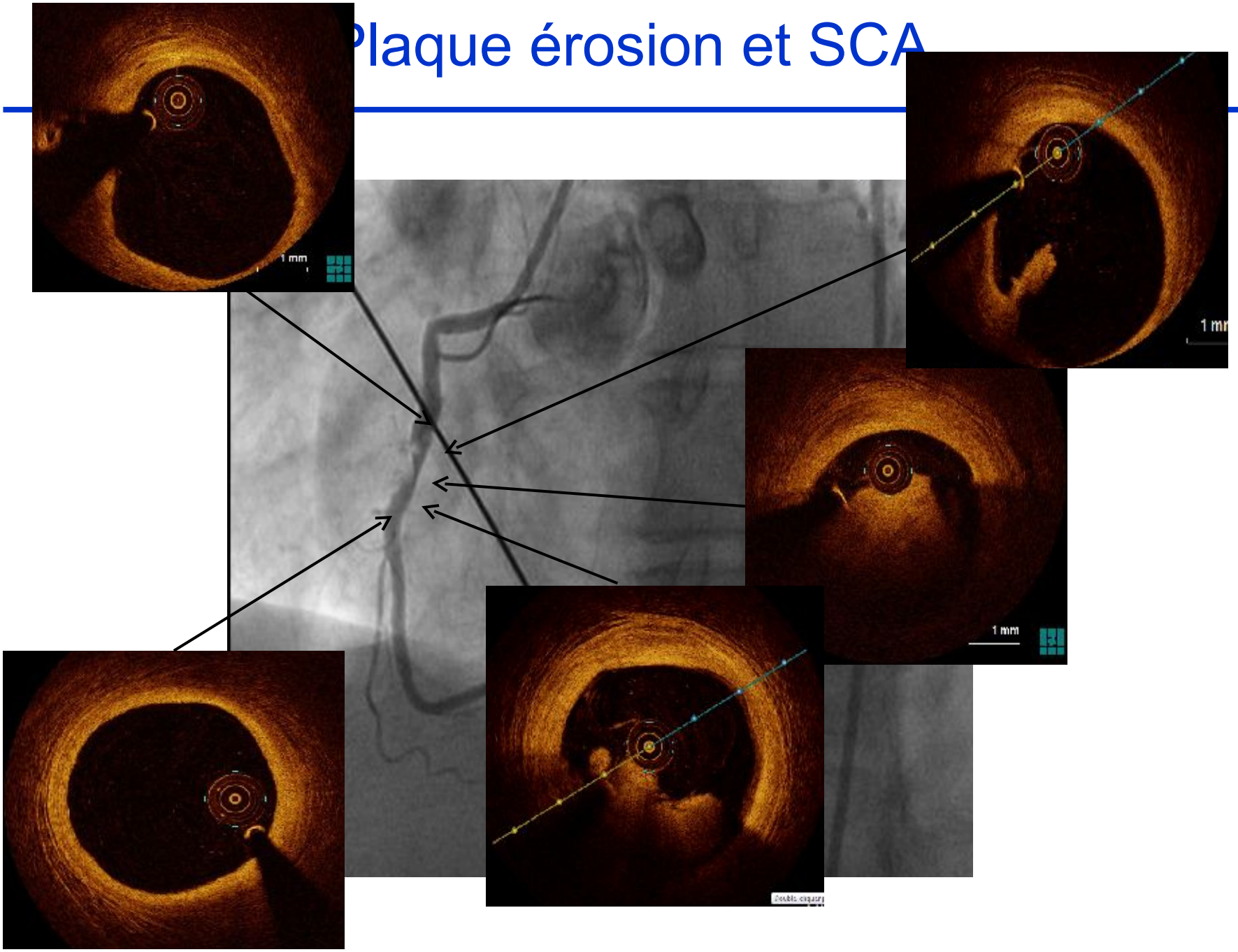
Jia H et al JACC 2013;62:1748-58.

Plaque érosion et SCA

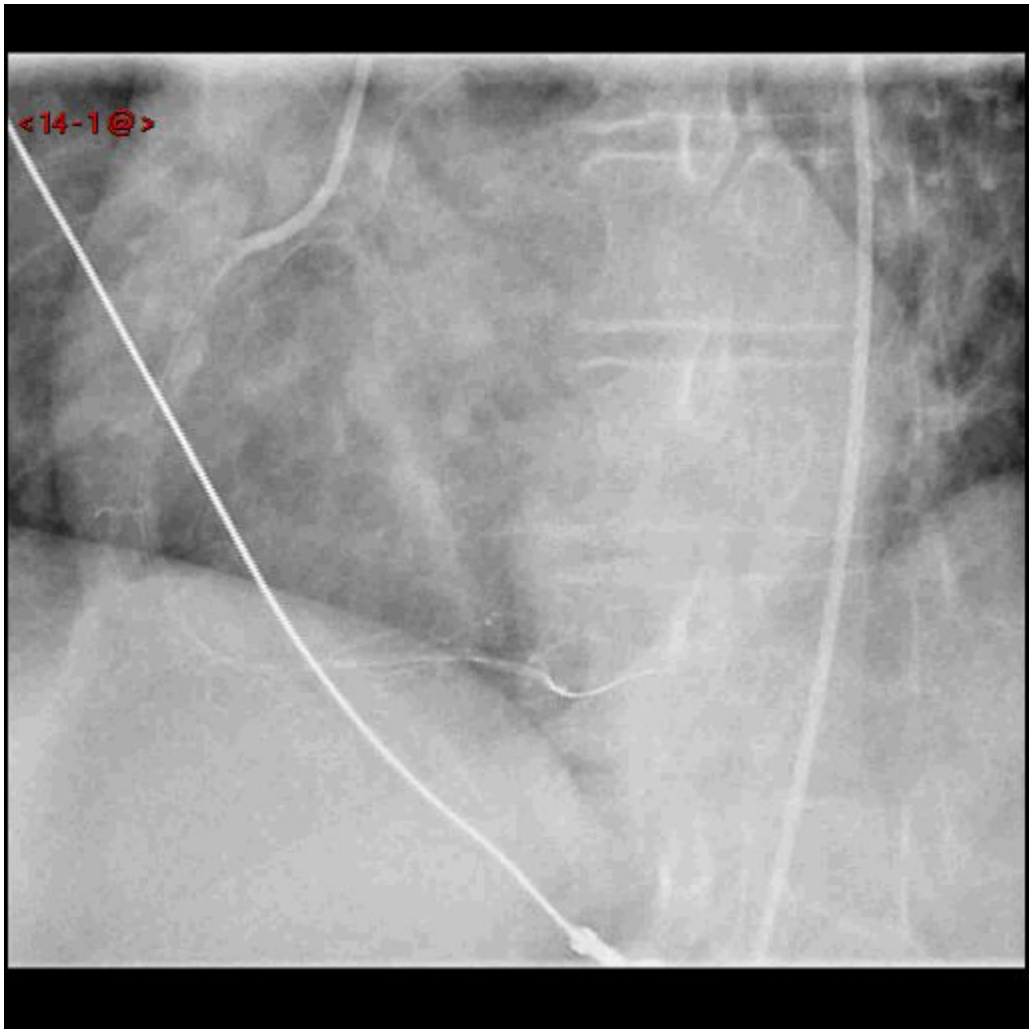


- **Pt 56 ans**
- **SCA ST-**
- **CD2**
- **Lésion intermédiaire inhomogène**
- **Fux TIMI III**

Plaque érosion et SCA

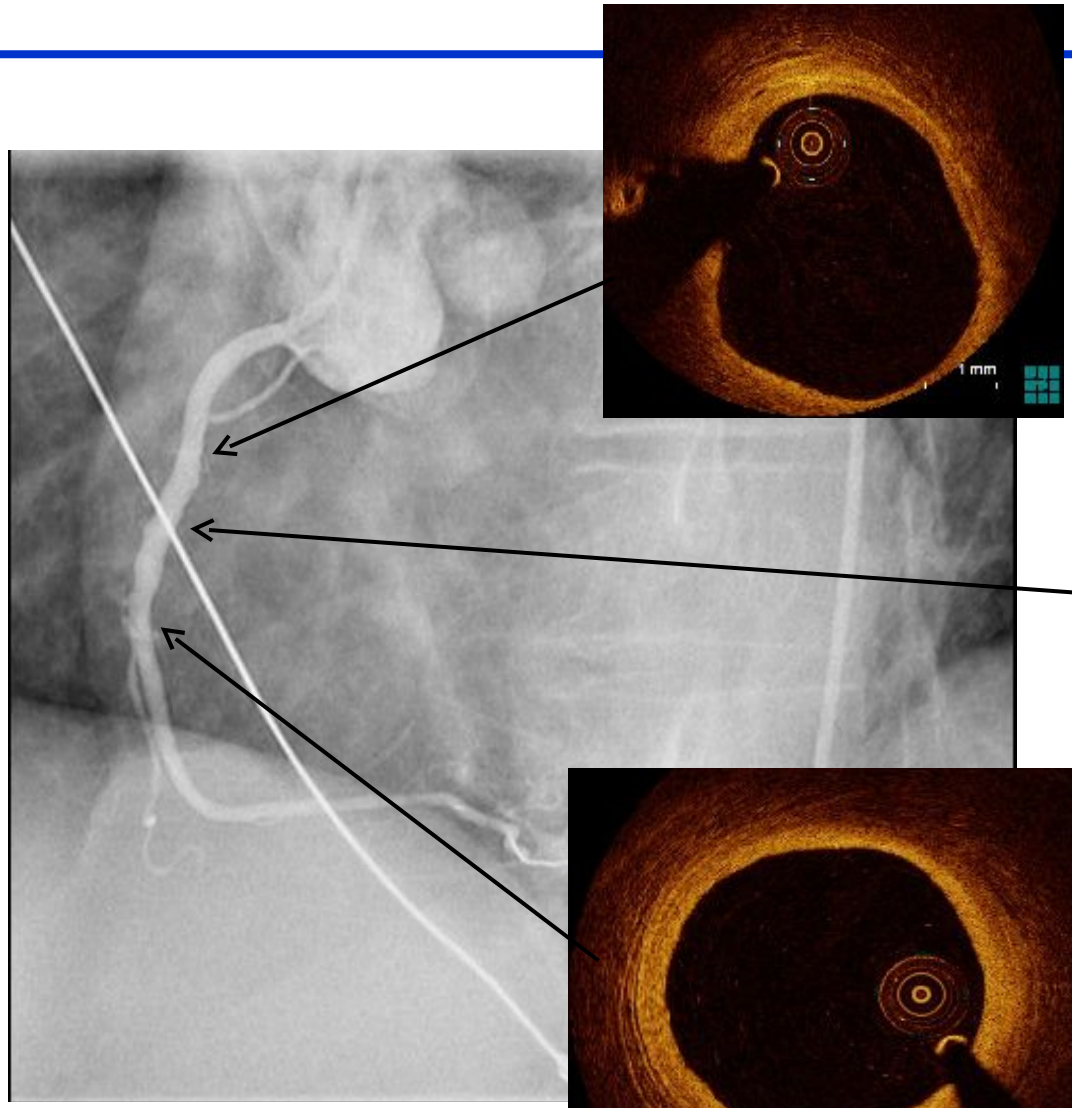


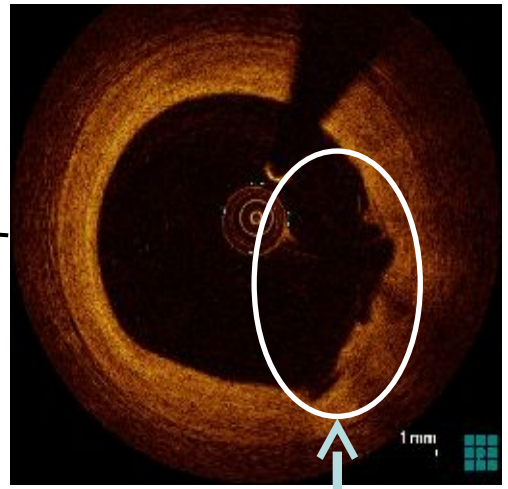
Plaque érosion et SCA



- **Post thromboaspiration**

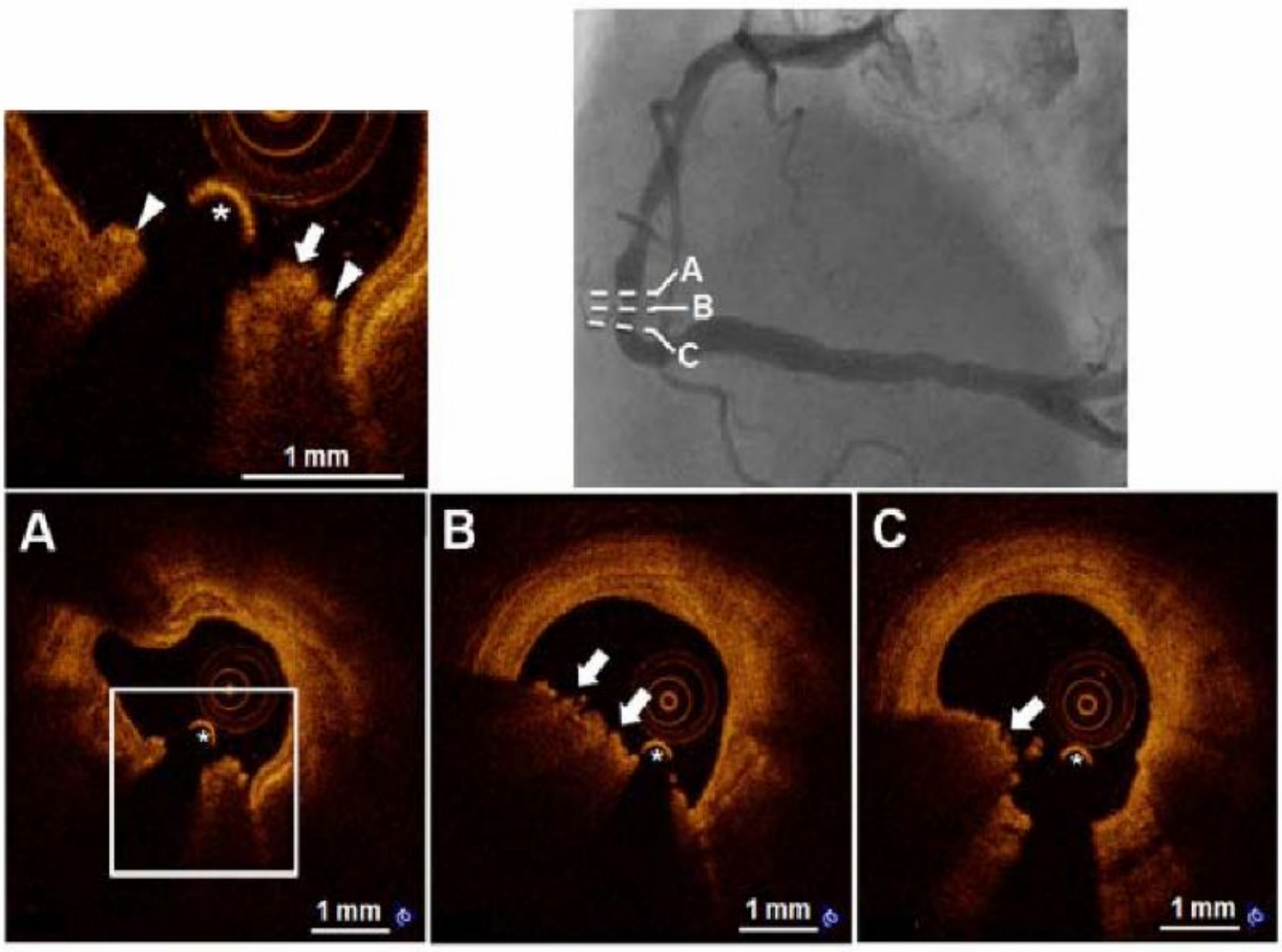
Plaque érosion et SCA





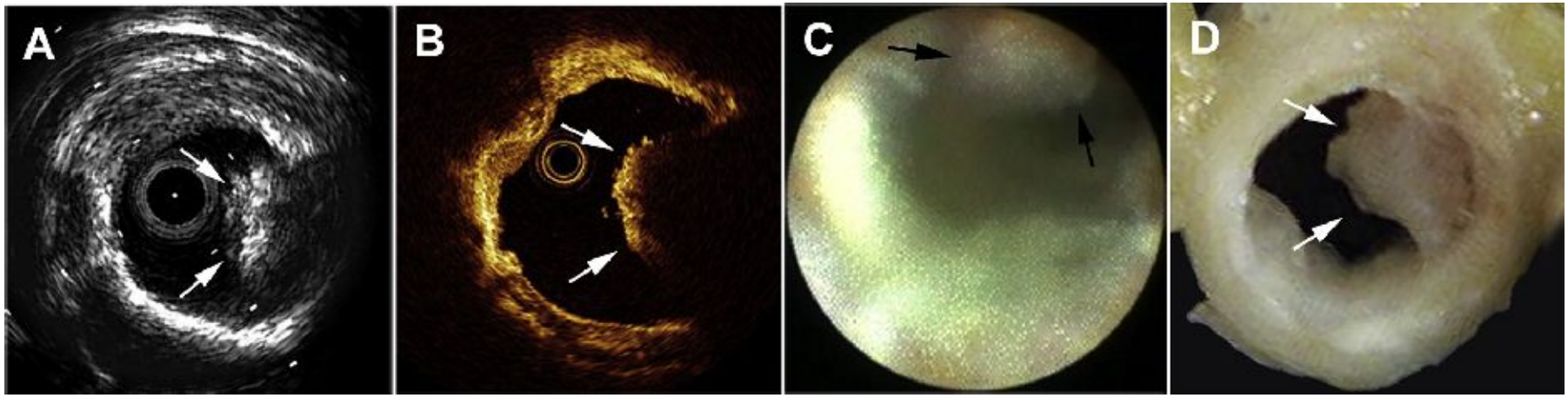
Erosion intimale & rupture plaque fibrotique

Nodules Ca++ et SCA



Jia H et al JACC 2013;62:1748-58.

Ex-vivo imaging of a calcified nodule

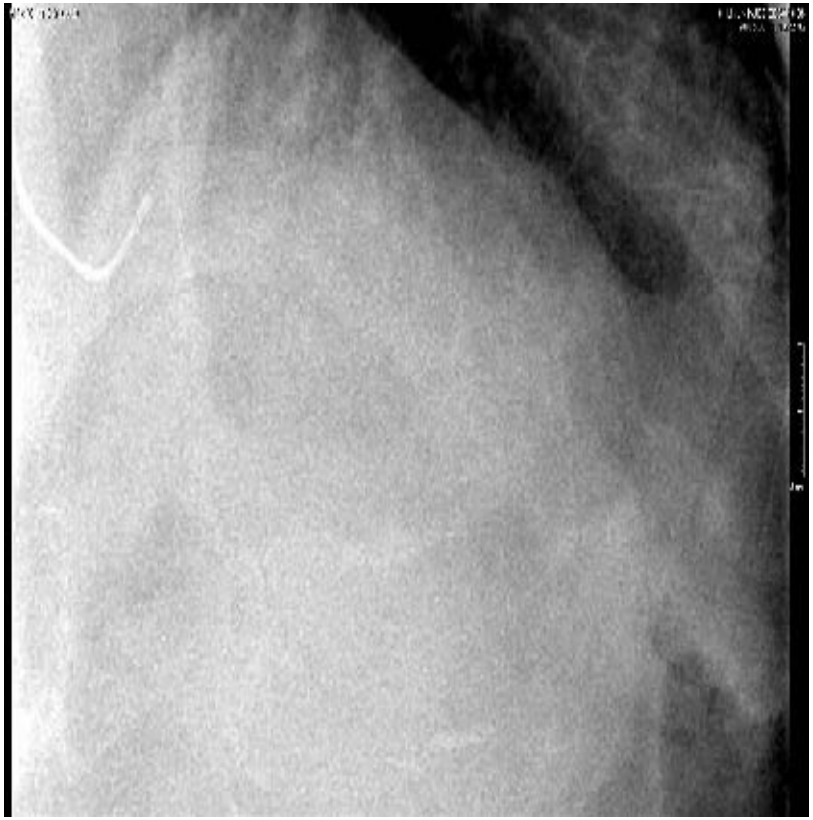


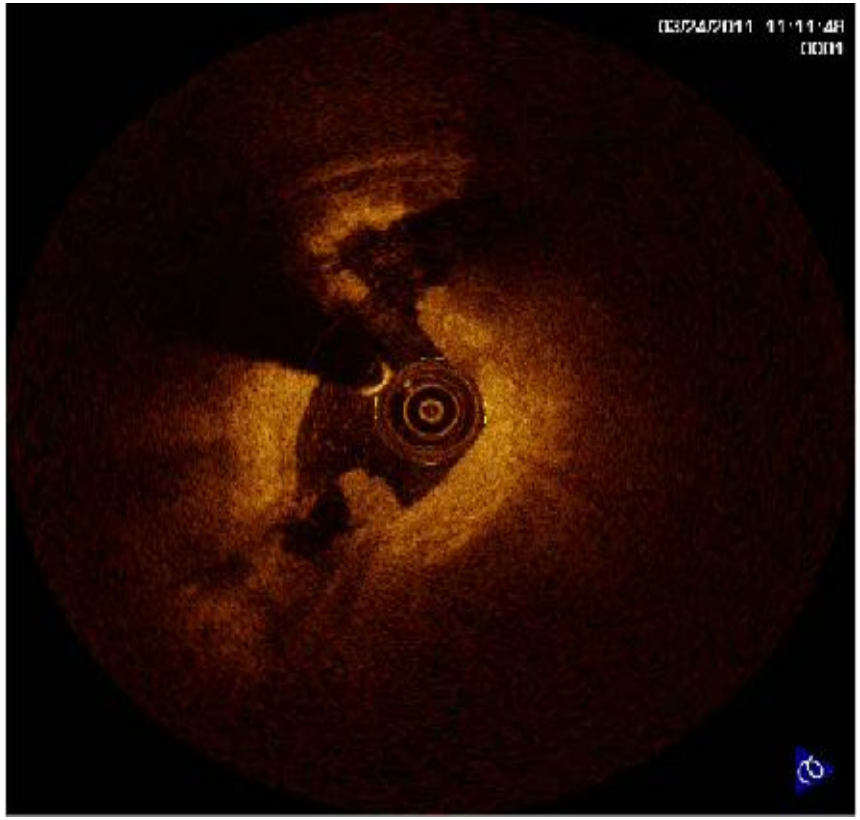
IVUS	OCT	Angioscopy	Gross appearance

Hao H et al JACC Cardiovasc Intv 2014;8:937

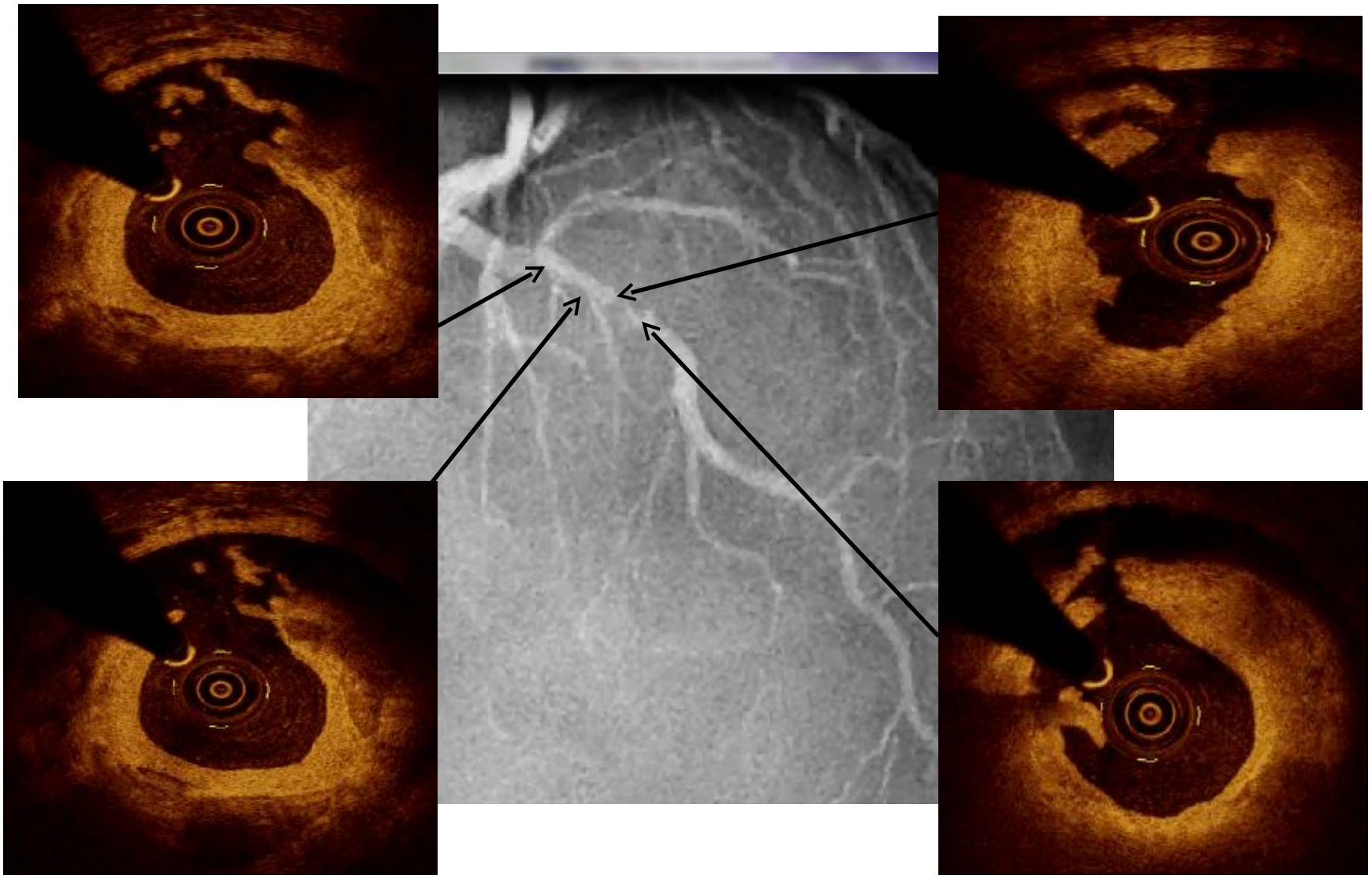
Rupture plaque et dissection sous adventicielle

36 ans sportif haut niveau, SCA ST+ transitoire au cours de la trans-jurassienne

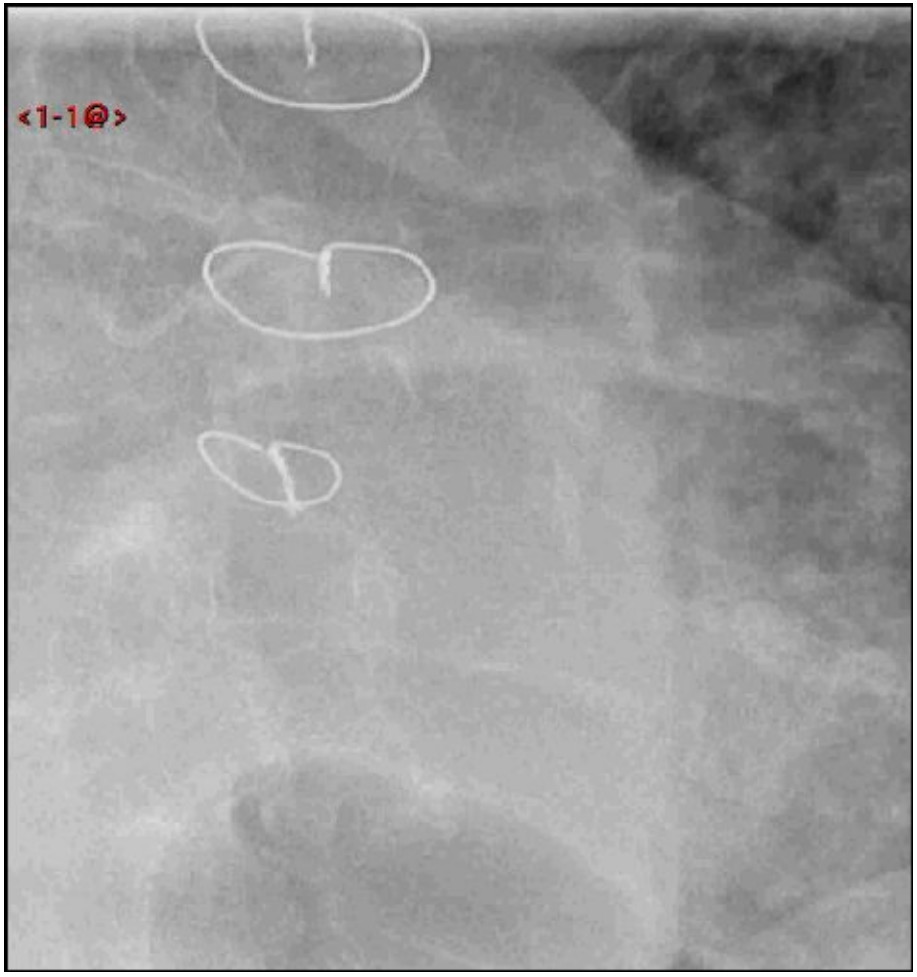




Rupture plaque et dissection sous adventicielle



Rupture plaque et dissection sous adventicielle



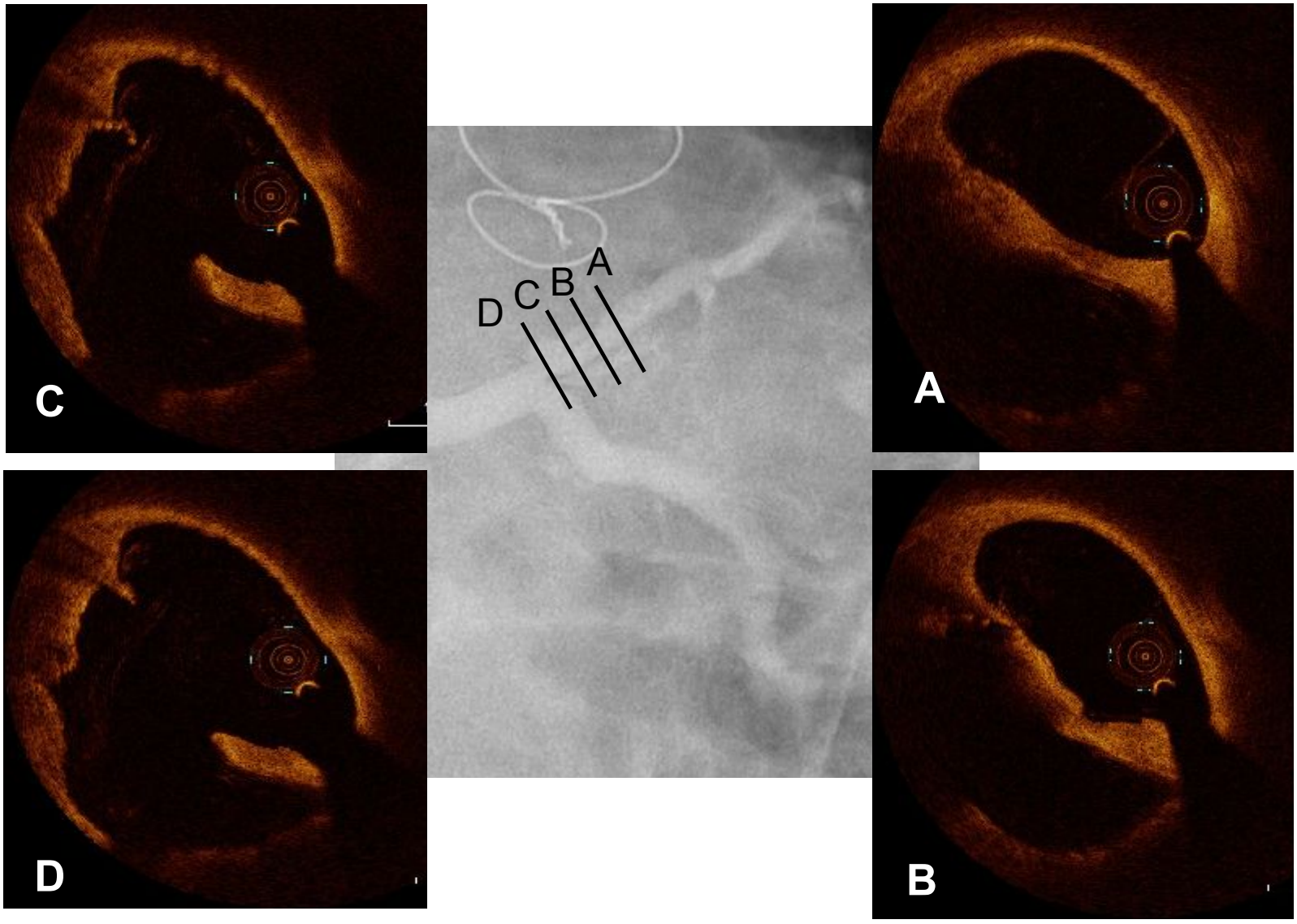
- Pt 59 ans

- ATCD de dissection aortique

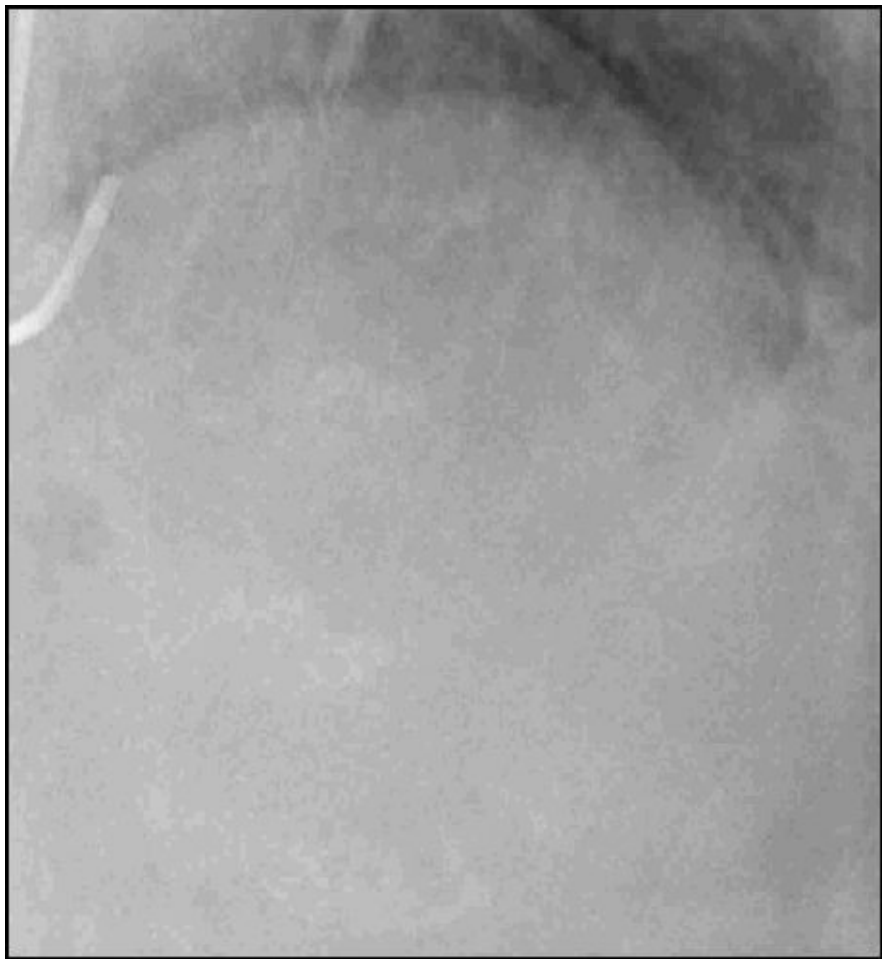
-HTA, cholestérol, BMI > 30,

-SCA ST-

Rupture plaque et dissection sous adventicielle

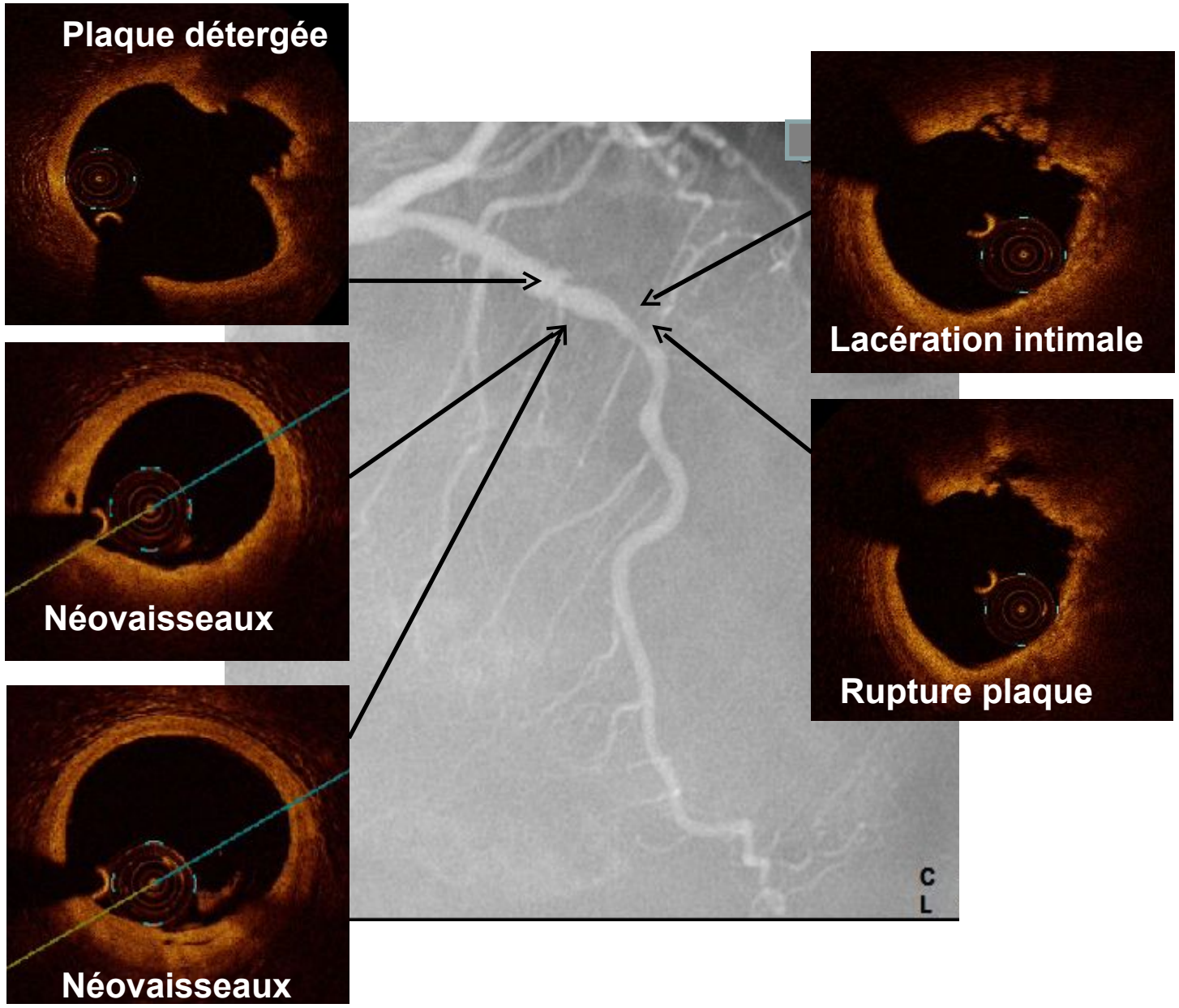


Plaque détergée



- **Pt 67 ans**
- **DNID, HTA, tabac**
- **ACFA sous AVK**
- **SCA ST- (<24h)**

Plaque détergée



Informations potentielles apportées par l'OCT dans l'identification des lésions responsables de SCA

- Les lésions à haut risque associent volume de plaque important, contenu lipidique et chape fibreuse fine.
- Aucune de ces caractéristiques ne permet d'identifier avec certitude l'évolution d'une lésion.
- SCA possible en cas de chape fibreuse épaisse ou de plaque fibreuse plutôt que lipidique.
- La rupture de plaque n'est pas le seul mécanisme physiopathologique en cause dans les SCA (érosion de la plaque, nodules Ca++,…).
- Lesions évolutives : prédiction d'evts difficile.
- Contexte inflammatoire systémique à prendre en compte.

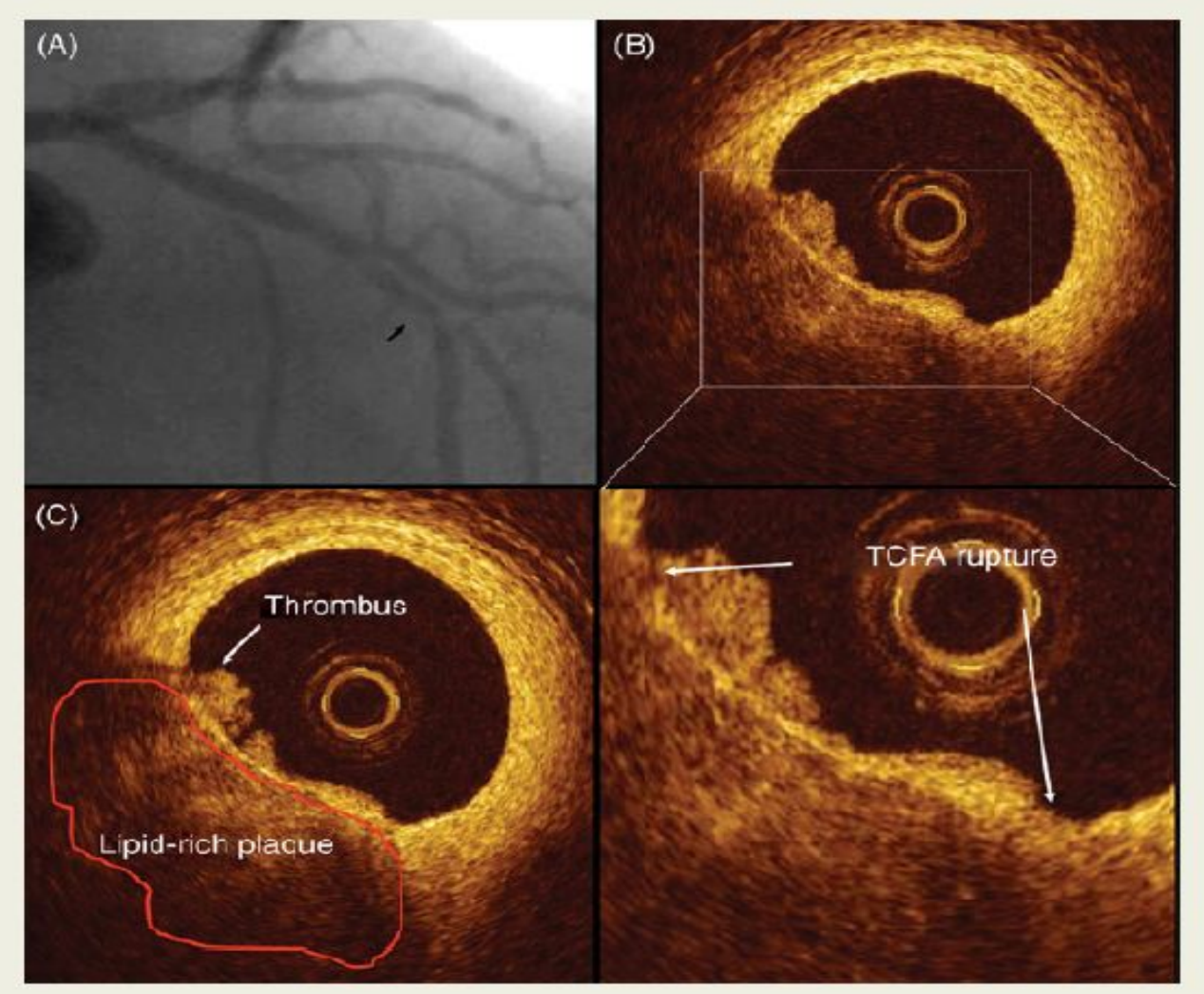
New insights in acute coronary syndromes derived from intracoronary imaging.

- In the setting of ACS coronary arteries demonstrate increased inflammation leading to a period of increased risk of subsequent events. Lipid core/necrotic core size may be correlated with severity of inflammation.

- Plaque ruptures and TCFAs are also seen in stable patients, begging the question on why some ruptured TCFAs cause ACS while others do not. Lipid core area, extent of arterial wall inflammation and reaction of platelets and other blood elements to abrupt changes in arterial wall morphology are of importance.

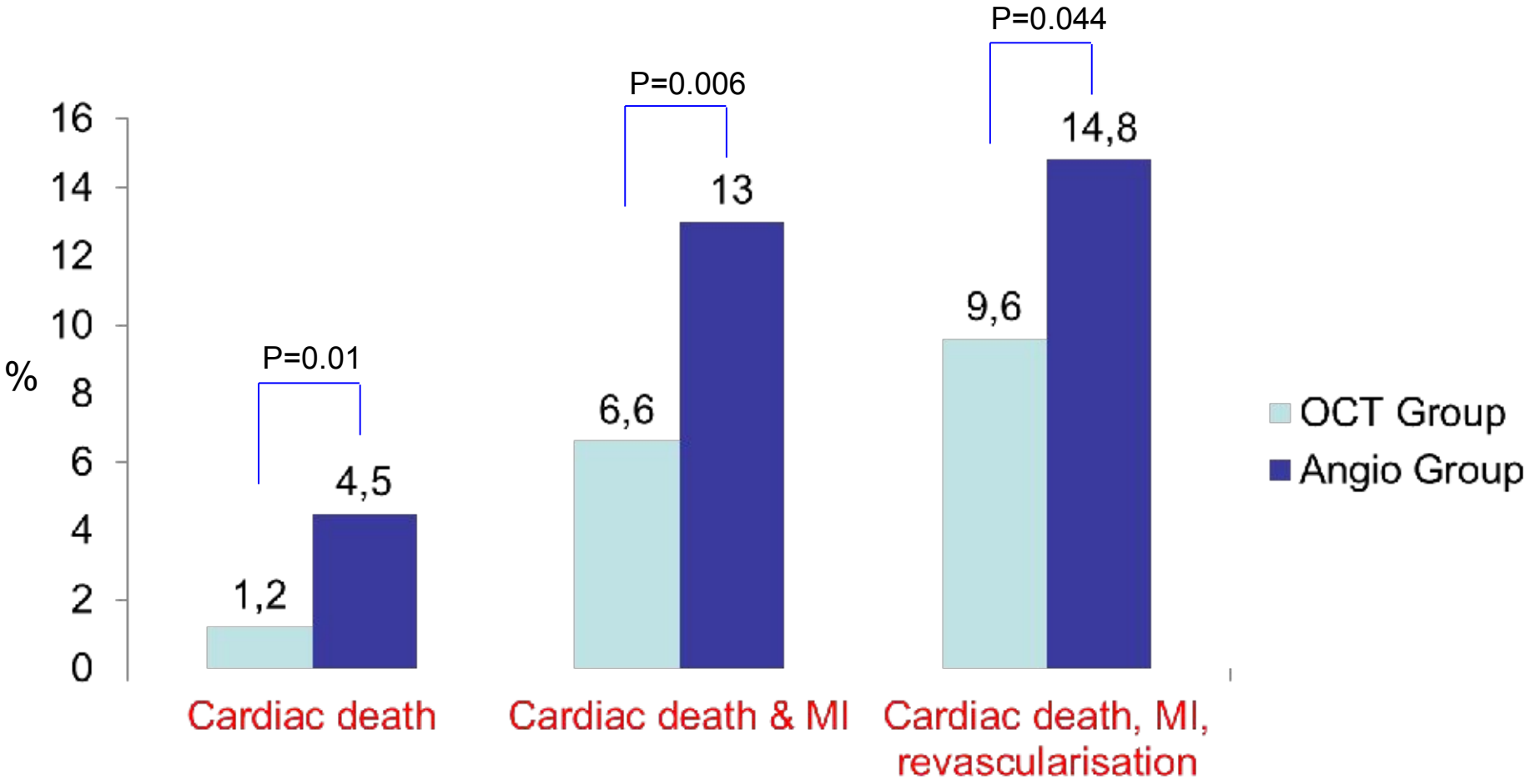
Identification des plaques vulnérables

Epaule : zone de rupture privilégiée de la chappe fibreuse



Can OCT-guided PCI improve clinical outcomes ?

Angiographic + OCT guidance was associated with a significantly lower one-year risk of MACE.



EuroIntervention 2012;8:823-29.

Etude DOCTORS

Does optical coherence tomography optimize results of stenting? Rationale and study design

Nicolas Meneveau, MD, PhD,[a] Fiona Ecarnot, MSc,[a] Geraud Souteyrand, MD,[b] Pascal Motreff, MD, PhD,[b] Christophe Caussin, MD,[c] Eric Van Belle, MD, PhD,[d] Patrick Oldmann, MD, PhD,[c] Olivier Morel, MD, PhD,[c] Alain Grentzinger, MD,[f] Michael Angioi, MD,[g] Romain Chopard, MD,[a] and François Schiele, MD, PhD[a] *Besançon, Clermont-Ferrand, Paris, Lille, Strasbourg, Belfort, and Vandœuvre les Nancy, France*

Background To date, no randomized study has investigated the value of optical coherence tomography (OCT) in optimizing the results of coronary angioplasty for non–ST-segment elevation acute coronary syndromes.

Methods DOCTORS is a randomized, prospective, multicenter, open-label clinical trial to evaluate the utility of OCT to optimize results of angioplasty of a lesion responsible for non–ST-elevation acute coronary syndromes. Patients (n = 250) will be randomized to undergo OCT-guided angioplasty (use of OCT to optimize procedural result, including change to strategy with the possibility of additional interventions) or angioplasty under fluoroscopy alone.

The primary end point is the functional result of the angioplasty procedure as assessed by fractional flow reserve (FFR) measured at the end of the procedure. Secondary end points include safety of OCT in the context of angioplasty for ACS, percentage of patients in whom OCT reveals suboptimal result of stenting, percentage of patients in whom a change in procedural strategy is decided based on OCT data, correlation between quantitative measures by OCT and FFR, determination of a threshold for quantitative OCT measure that best predicts FFR ≥0.90, and identification of OCT variables that predict postprocedure FFR. Adverse cardiac events (death, recurrent myocardial infarction, stent thrombosis, and repeat target lesion revascularization) at 6 months will be recorded.

Conclusion The DOCTORS randomized trial (ClinicalTrials.gov NCT01743274) is designed to investigate whether use of OCT yields useful additional information beyond that obtained by angiography alone and, if so, whether this information changes physician strategy and impacts on the functional result of angioplasty as assessed by FFR. (Am Heart J 2014;168:175-181.e2.)

Etude DOCTORS

Does optical coherence tomography optimize results of stenting? Rationale and study design

Nicolas Meneveau, MD, PhD,[a] Fiona Ecarnot, MSc,[a] Gerald Souteyrand, MD,[b] Pascal Motreff, MD, PhD,[b] Christophe Caussin, MD,[c] Eric Van Belle, MD, PhD,[d] Patrick Ohlmann, MD, PhD,[e] Olivier Morel, MD, PhD,[e] Alain Grentzinger, MD,[f] Michael Angioi, MD,[g] Romain Chopard, MD,[a] and François Schiele, MD, PhD[a] *Besançon, Clermont-Ferrand, Paris, Lille, Strasbourg, Belfort, and Vandoeuvre les Nancy, France*

Am Heart J 2014;168:175-181.

Pts 18-80 yrs with NSTE-ACS who meet inclusion criteria

Initial coronary angiography with a single lesion on the culprit artery without diffuse disease on the same vessel considered to be responsible for the ACS.

Randomization

OCT Group

OCT performed after initial coronary angiography :

1. Stent length and diameter chosen based on the quantitative measures by OCT.
2. Use of IIb/IIIa inhibitors and/or thromboaspiration systematically considered in case of thrombus.
3. Rotational atherectomy considered in case of circumferential calcifications.

OCT Post Stent Implantation :

1. Additional balloon inflations in case of stent malapposition or underexpansion (minimal stent area/reference lumen area ≤80%).
2. Additional stent implantation(s) in case of incomplete lesion coverage & edge dissection.

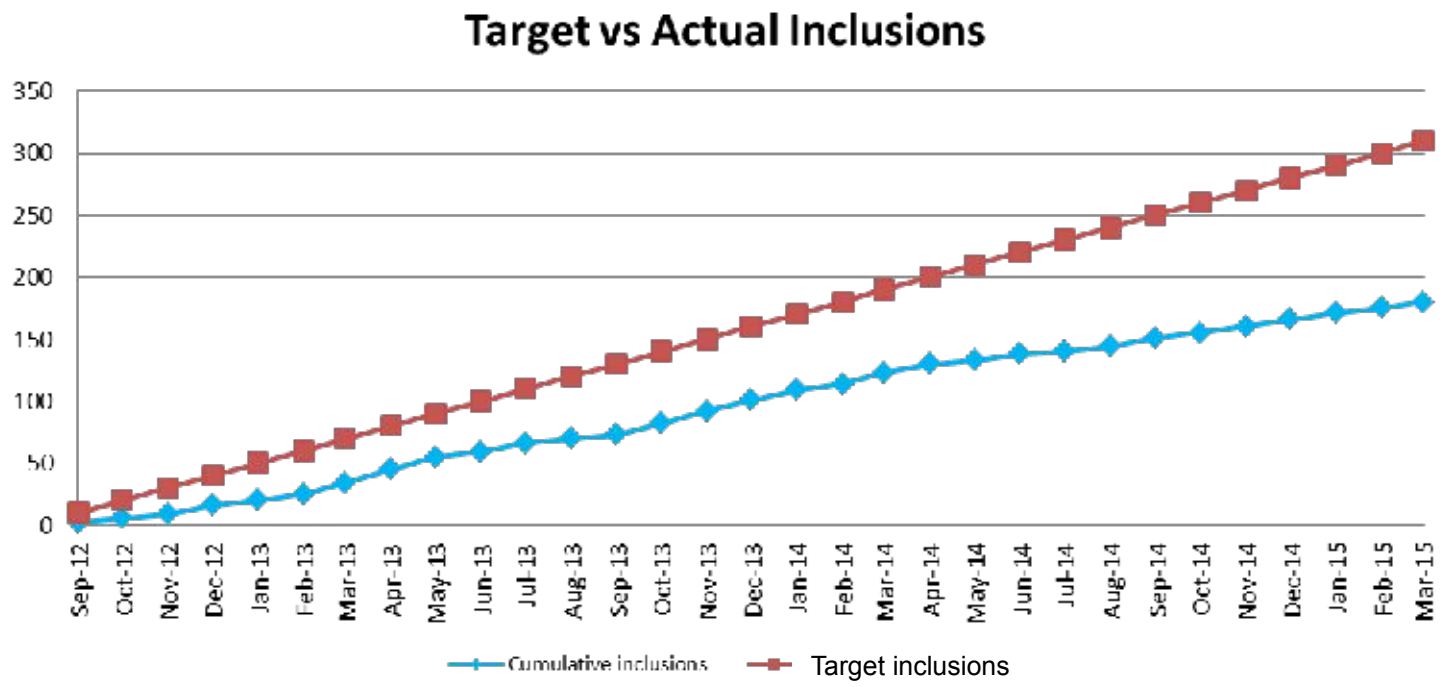
- Several OCT runs can be performed, as required
- Operator may change procedural strategy based on OCT data immediately available
- Possibility of additional interventions.

FFR measured at the end of the procedure.
No further interventions, regardless of the FFR value.

Control Group

FFR measured at the end of the procedure.
No further interventions, regardless of the FFR value.

Inclusion Status



To date, 180 patients included.
70 patients required to achieve target.