MAYO CLINIC

Gender and Spontaneous Coronary Artery Dissection

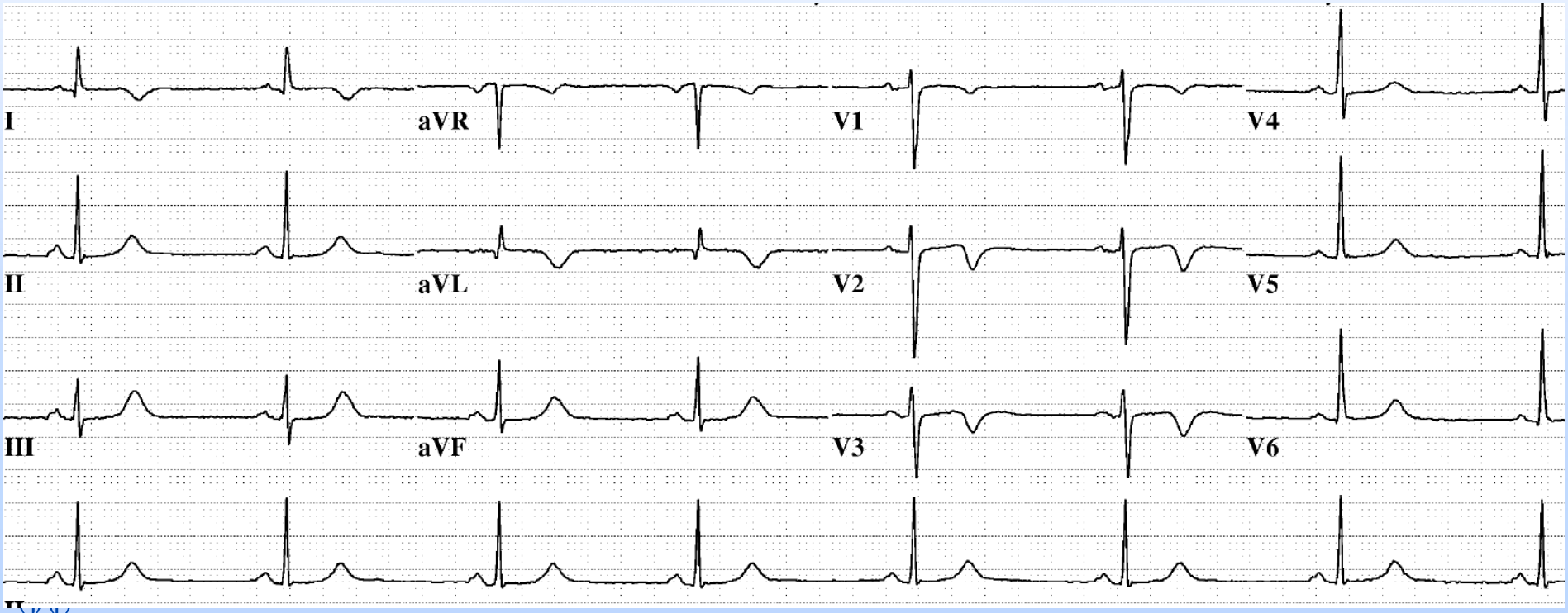
Rajiv Gulati, MD PhD

Advances in Cardiac Arrhythmias and Great Innovations in Cardiology

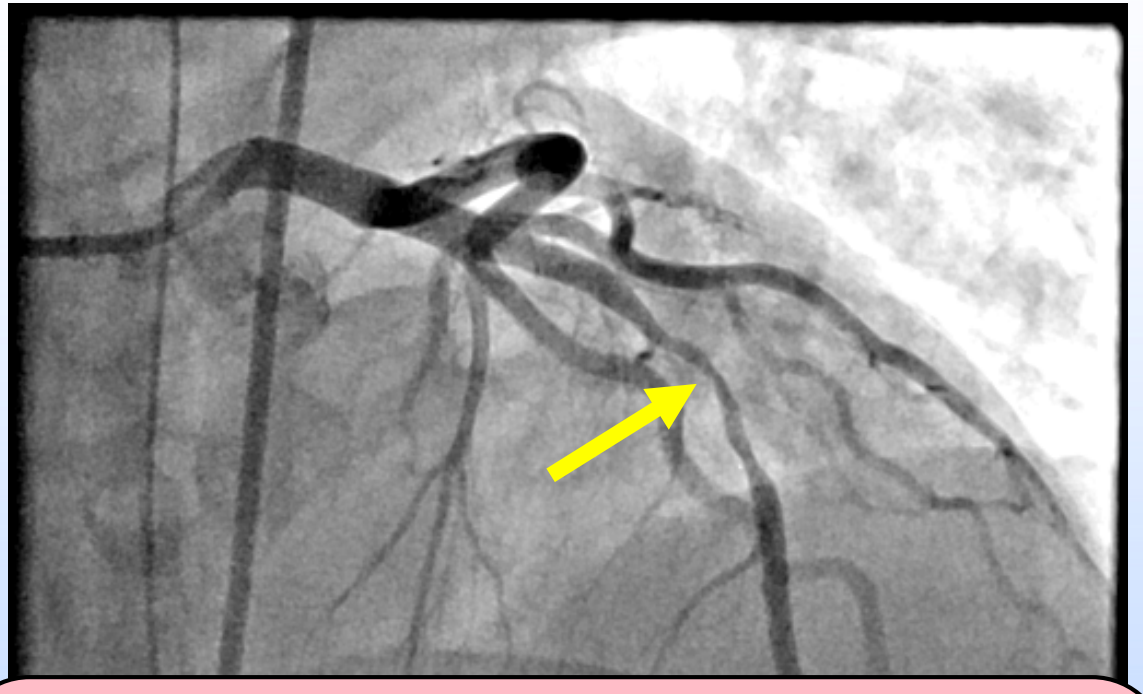
Torino, October 2015

Case: 42 year old female

- Fit with no risk factors
- V Fib arrest, full recovery, troponin+



Case: 42 year old female with ACS

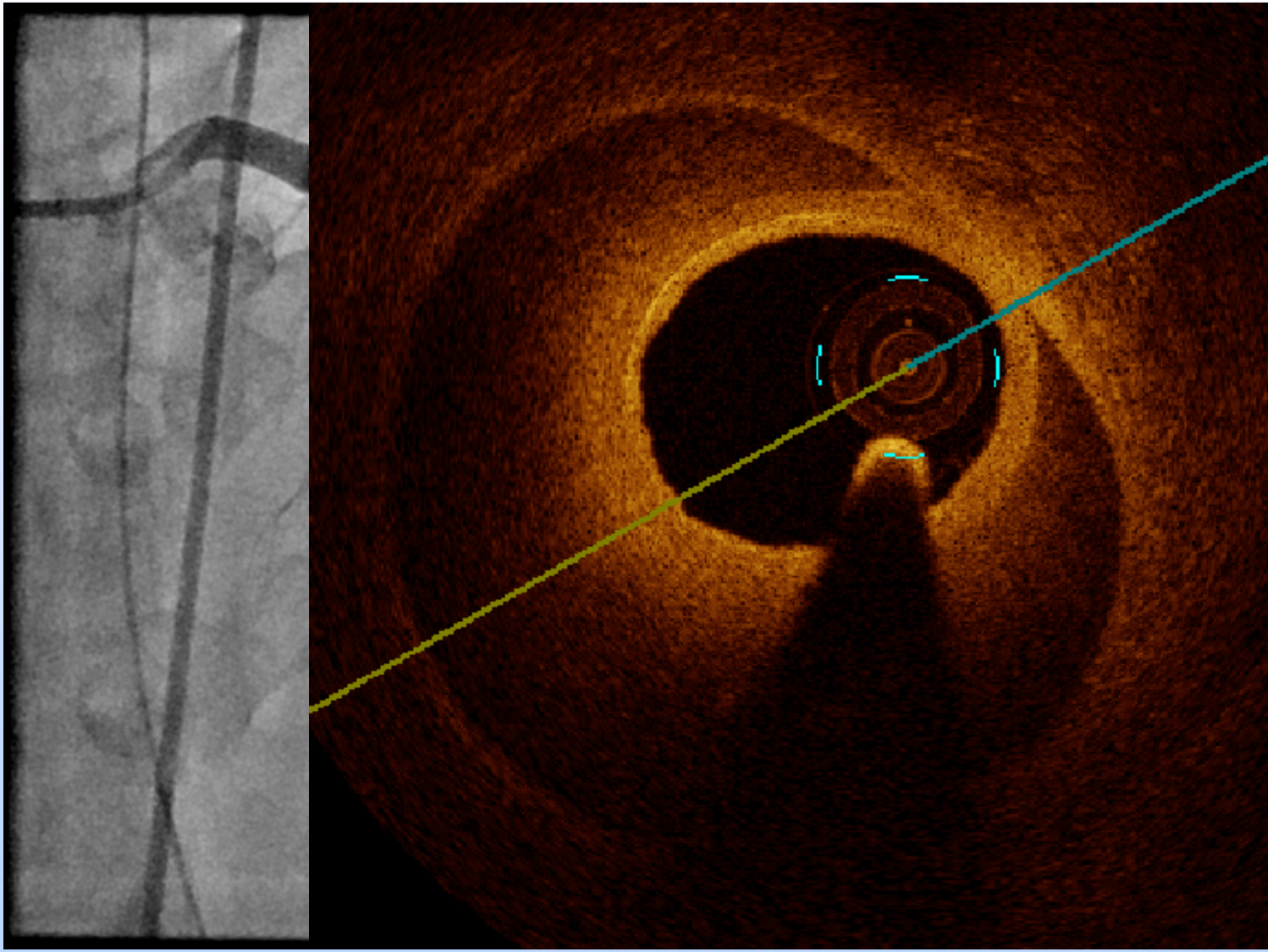


Class I, LoE A

- DAPT
- Statin
- Stent

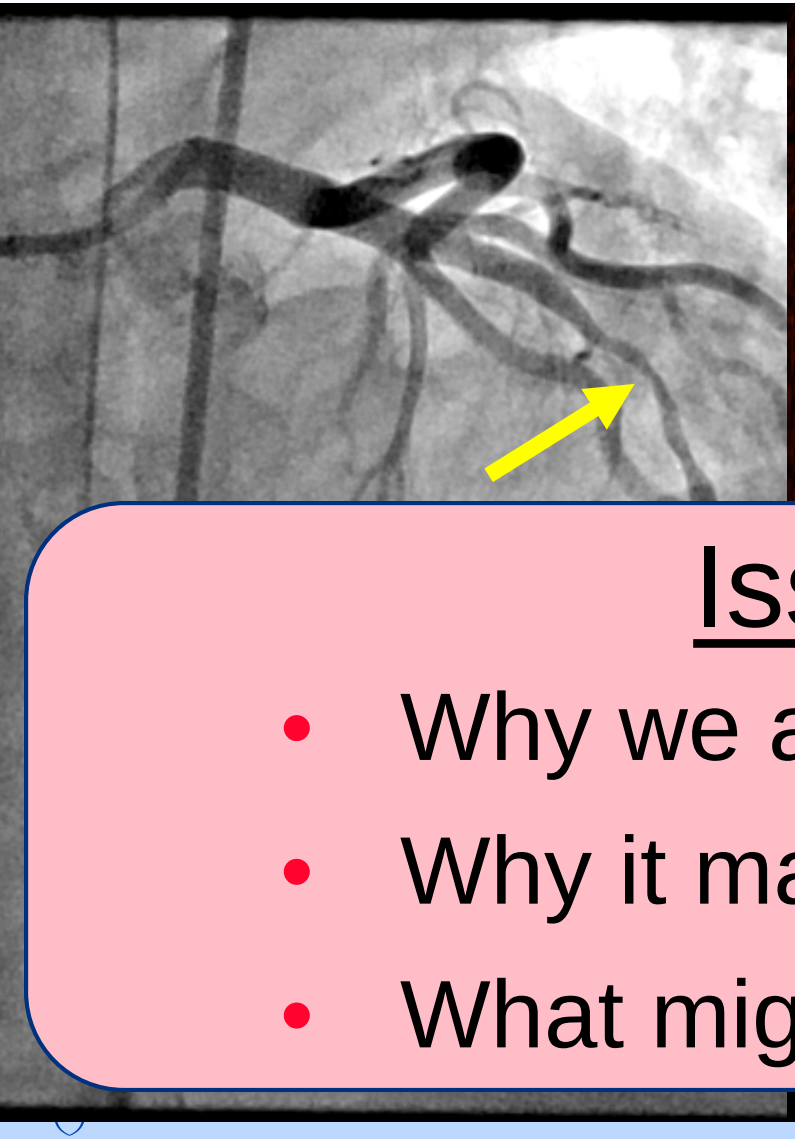
MAYO CLINIC

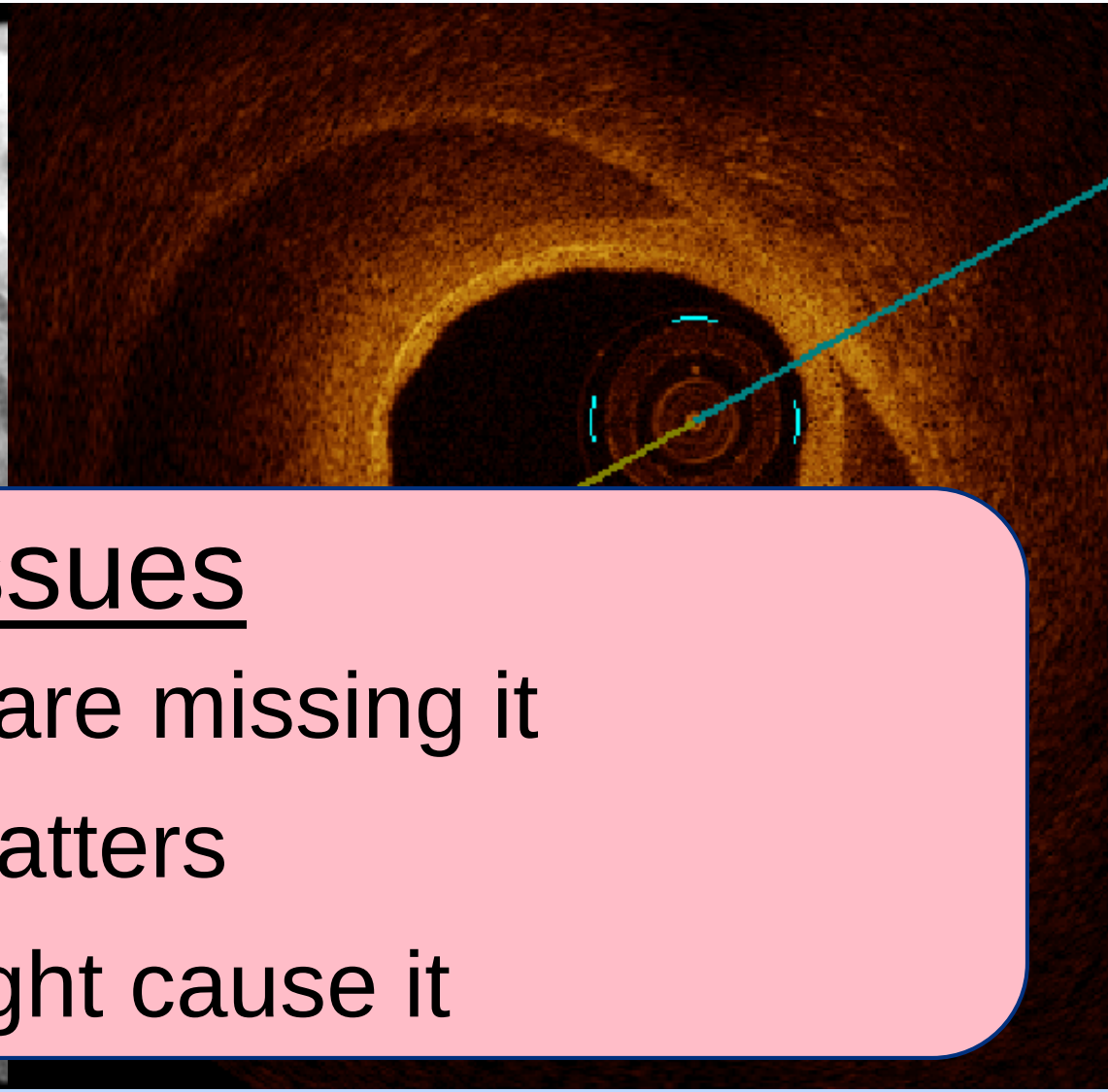
Case: 42 year old female with ACS



MAYO CLINIC

Spontaneous Coronary Dissection (and Hematoma)





Issues

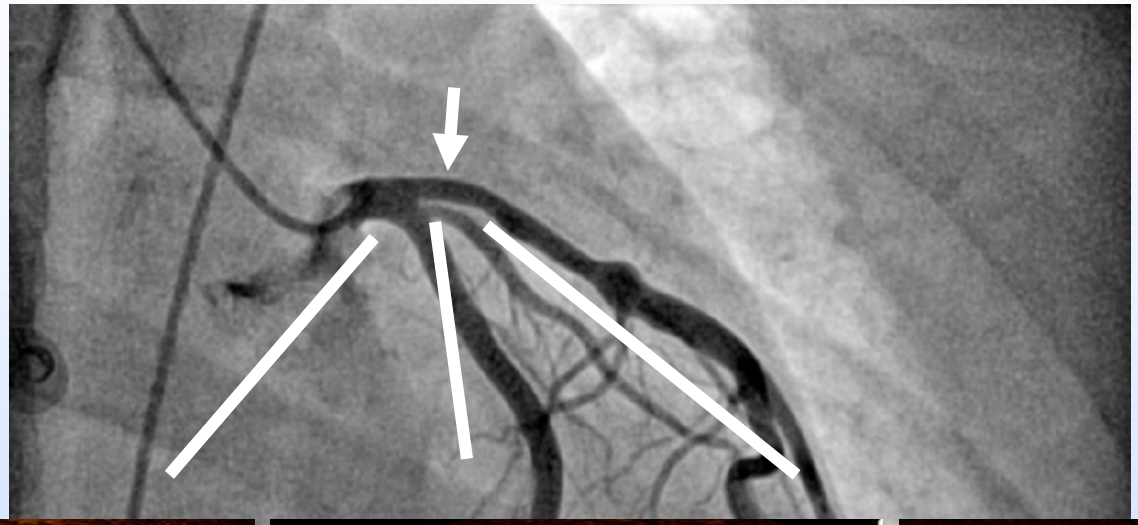
- Why we are missing it
- Why it matters
- What might cause it

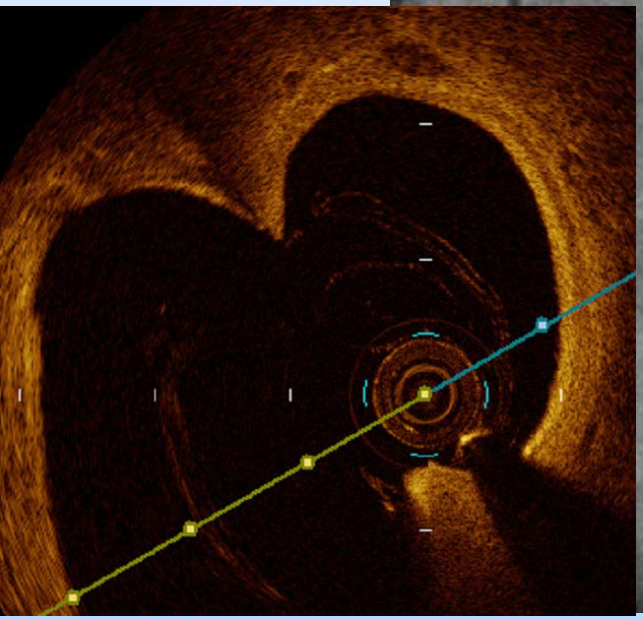
Why are we missing SCAD

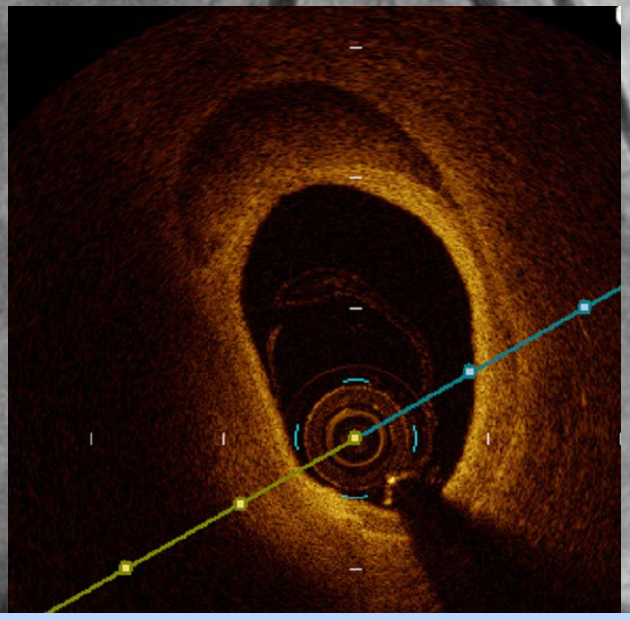
In the cath lab, we assume every stenosis is atherosclerosis…

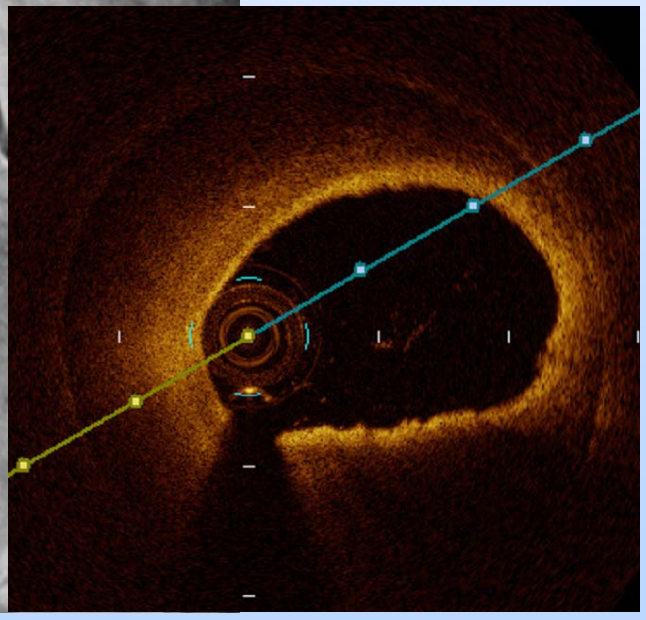
MAYO CLINIC

50 yr F with ACS









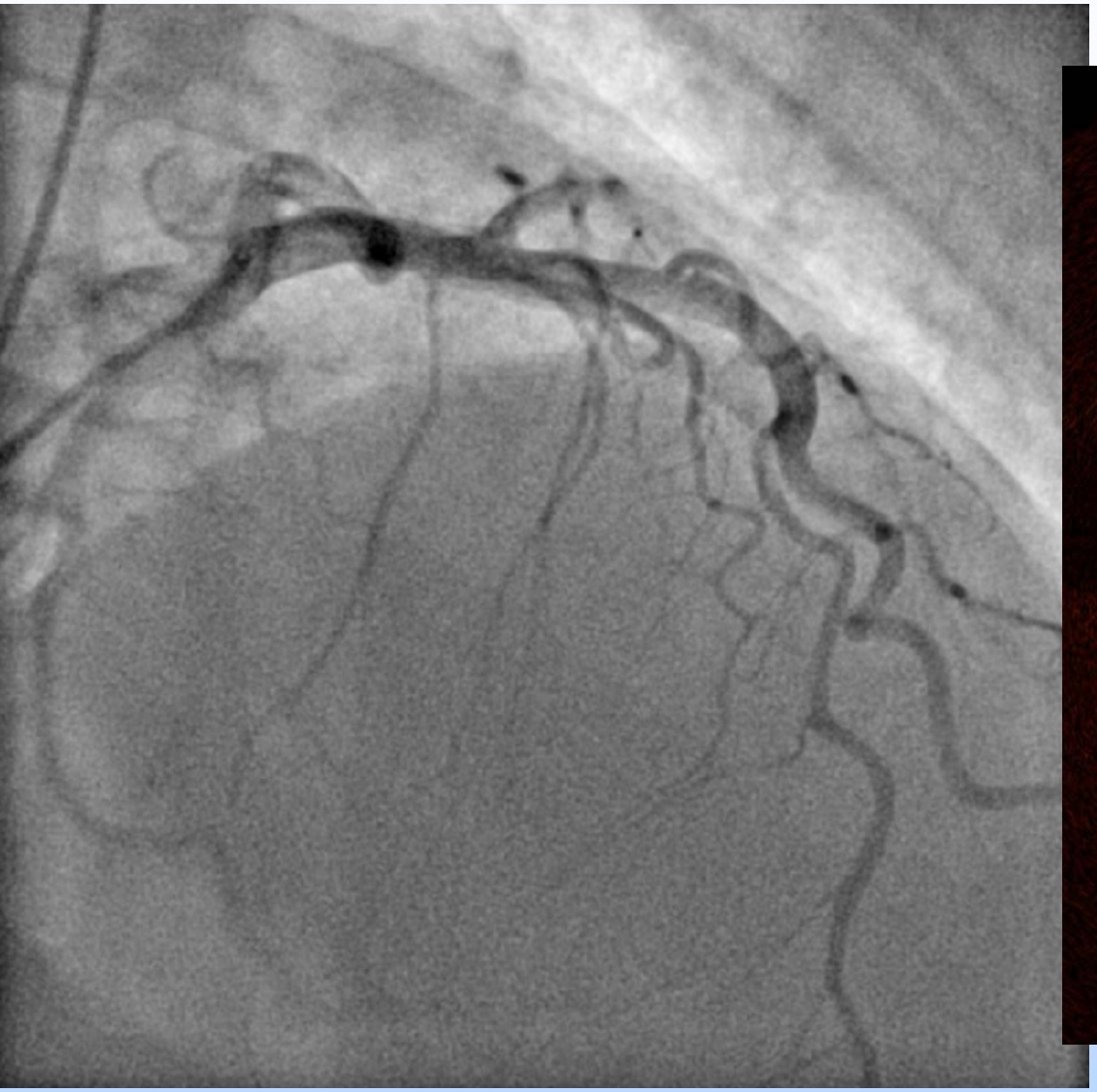
Why are we missing SCAD

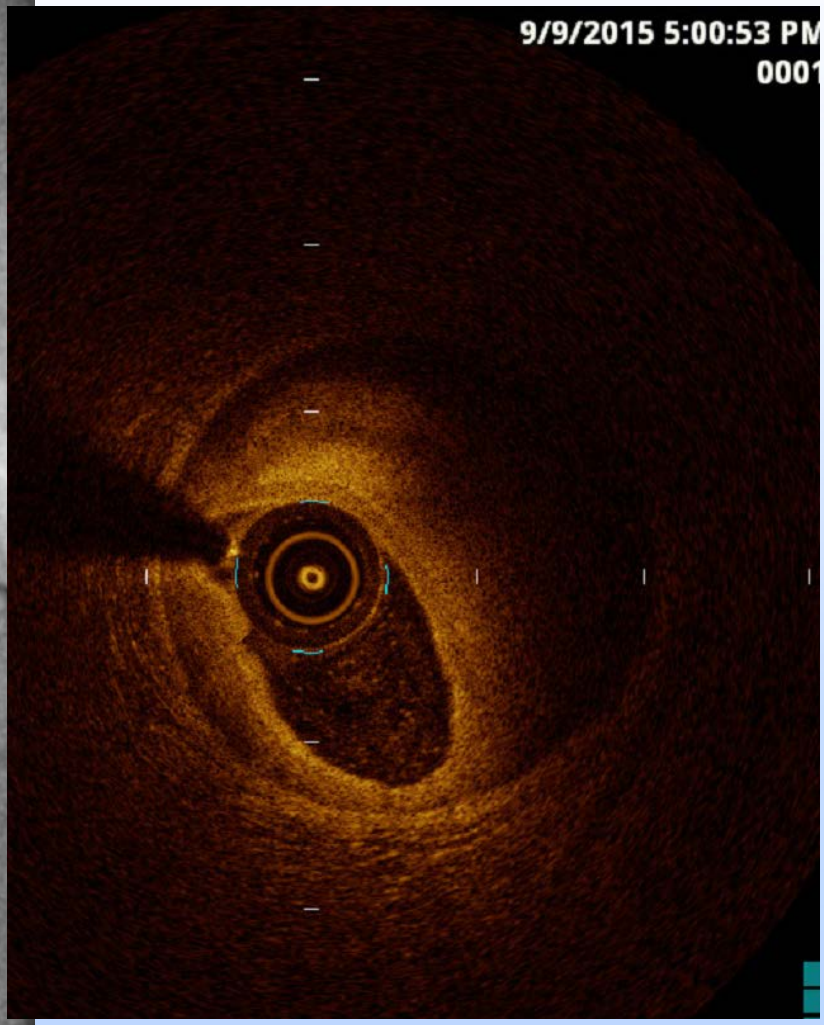
We assume atherosclerosis…

…or spasm…

MAYO CLINIC

49 yr F with Torsades transient anterior ST-T changes





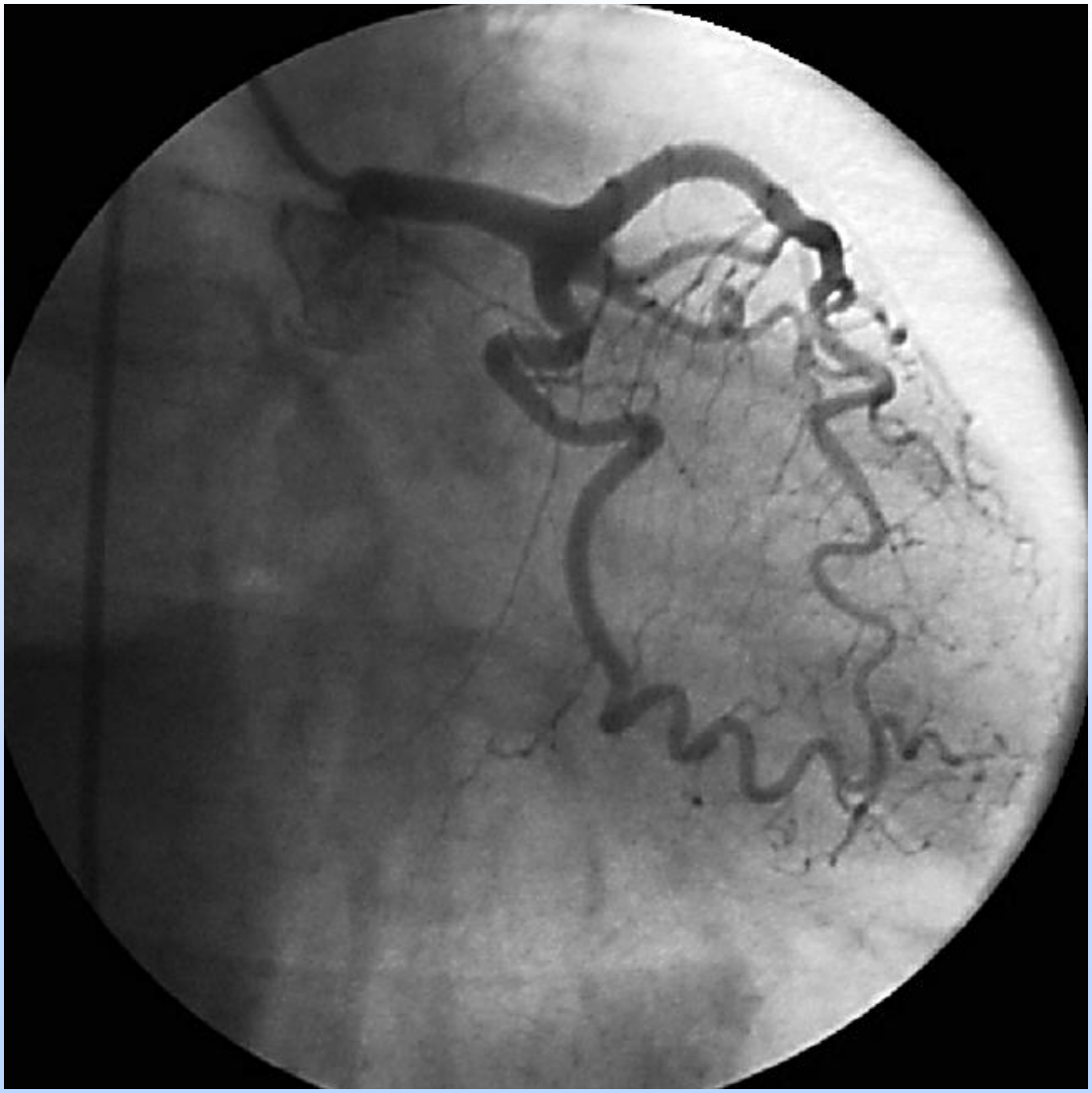
Why are we missing SCAD

We assume its atherosclerosis…

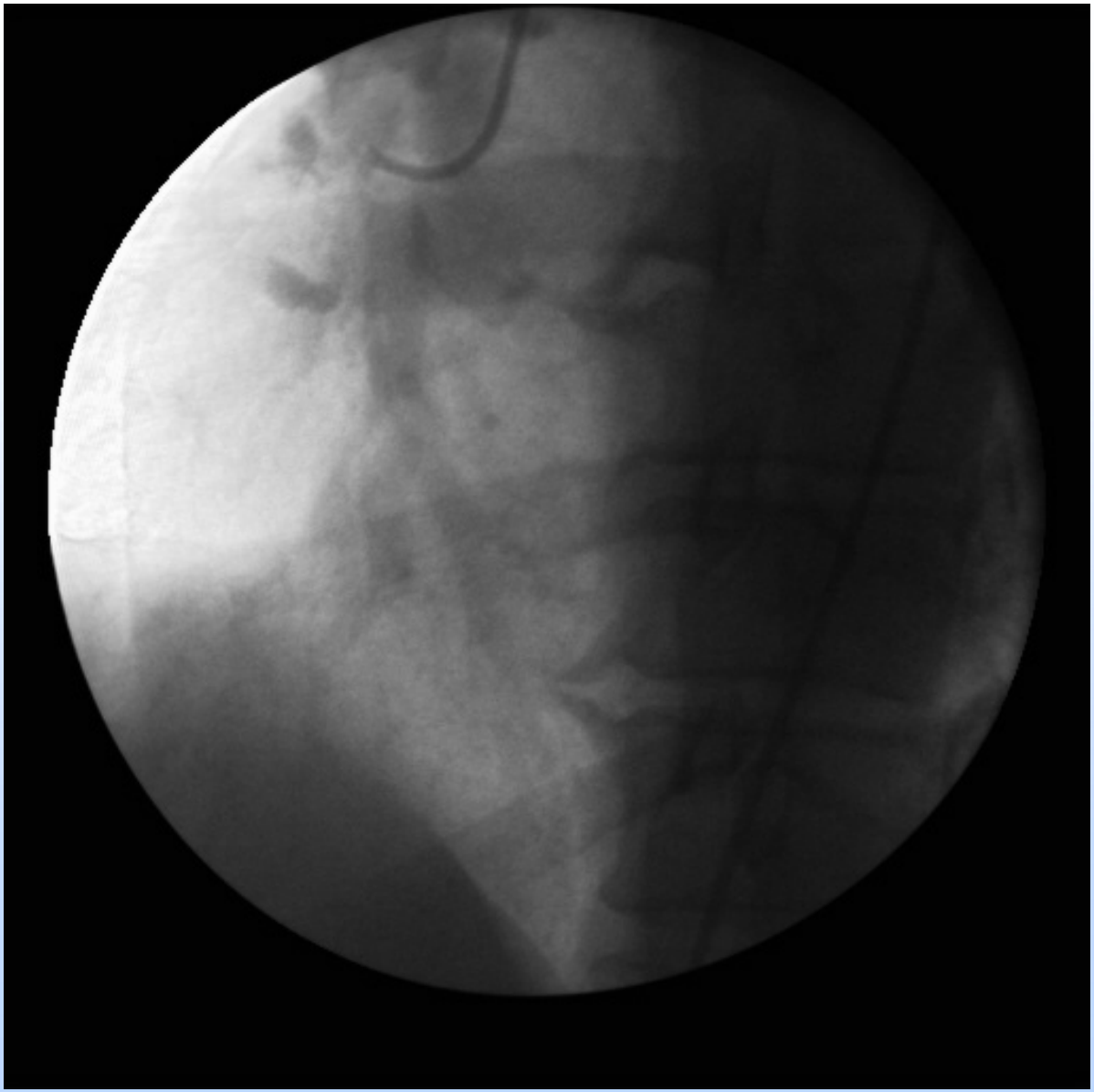
…or spasm…or we “diagnose” normal coronaries

MAYO CLINIC

Different Case - 48 yr F
ACS with “normal coronaries”

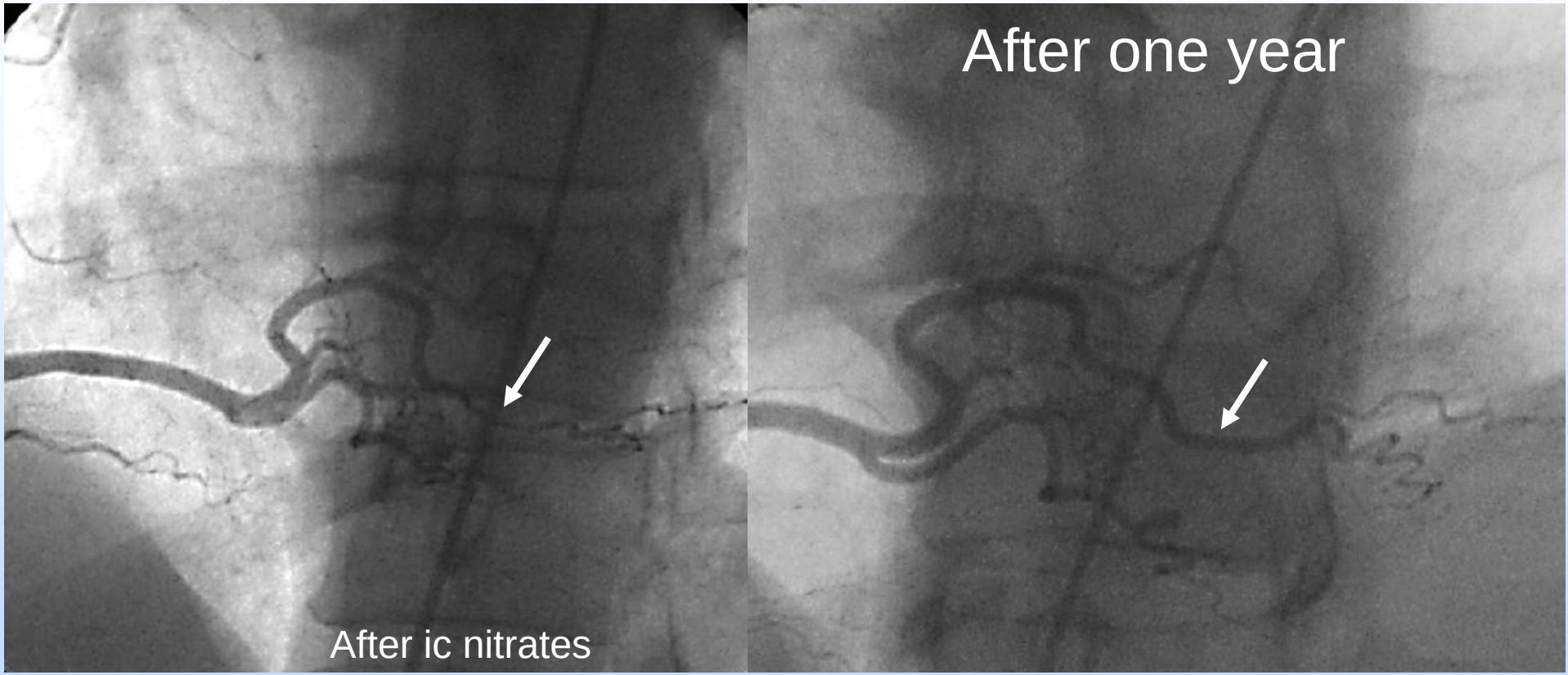


MAYO CLINIC



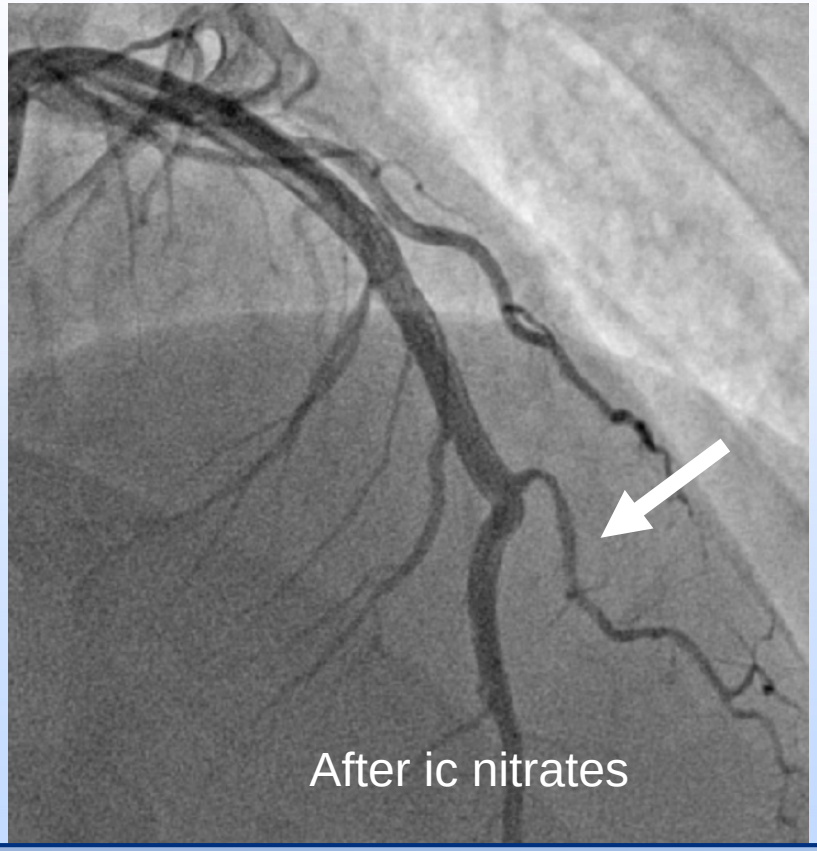
MAYO CLINIC

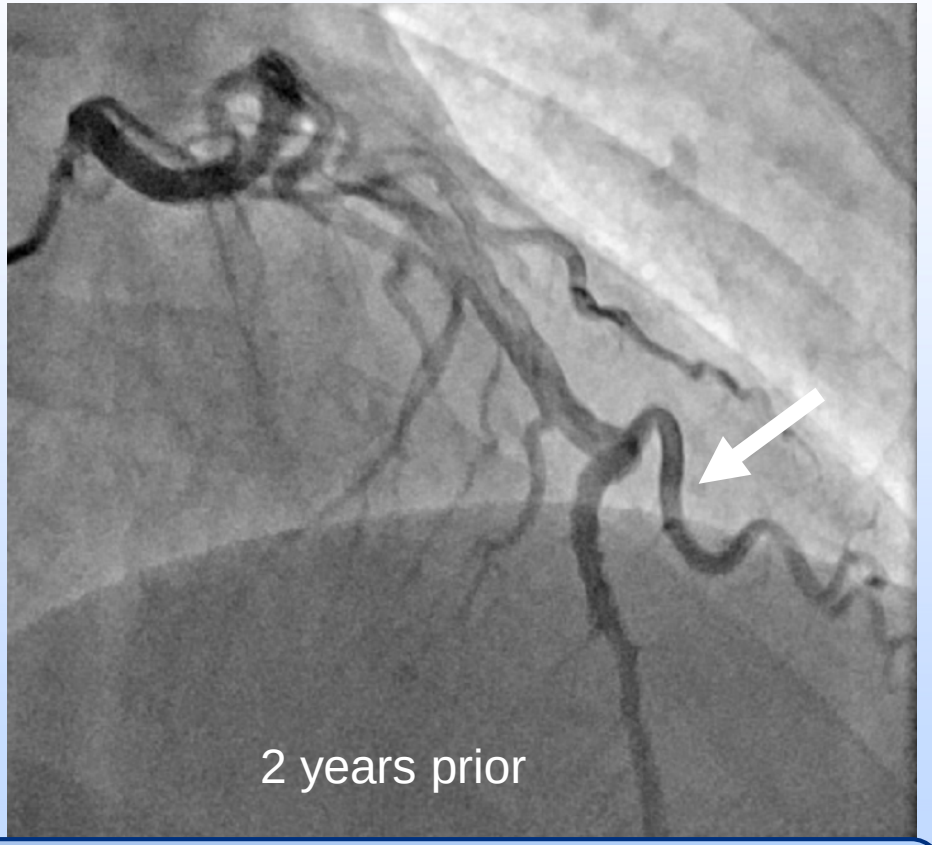
ACS with “normal coronaries”



MAYO CLINIC

Another ACS with "normal coronaries"





= SCAD, not myocarditis/spasm etc

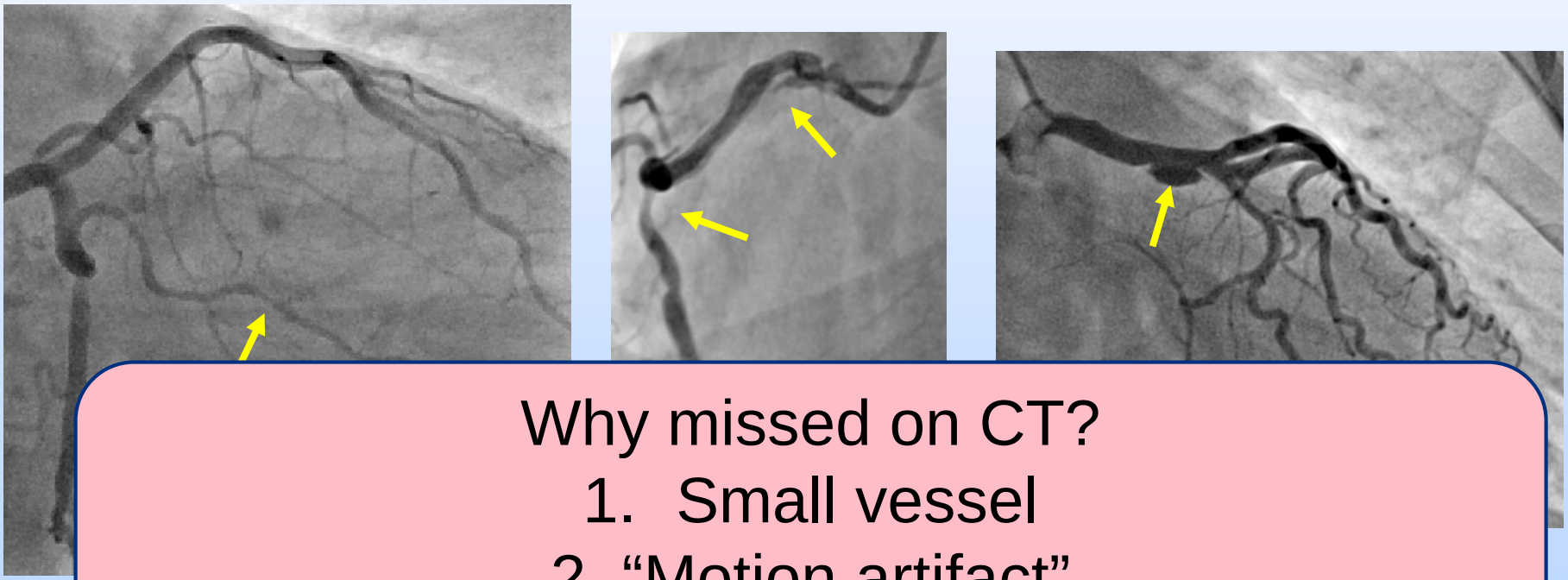
Why are we missing SCAD

We assume atherosclerosis, spasm, normal coronaries… only if the patient actually gets to the cath lab

MAYO CLINIC

Three consecutive women, atypical pain, troponin+, Mayo ED last 9 mths

Triple r/o CT → All negative



Why missed on CT?

1. Small vessel
2. “Motion artifact”
3. No athero, no interest

MAYO CLINIC

How common is SCAD?

Missed in ER

Missed in cath lab

Normal, athero, spasm, ABS, focal myocarditis,1ºVF

Missed at autopsy

Desai S et al, Am J Pathol 2012

MAYO CLINIC

Who gets SCAD, what causes it?

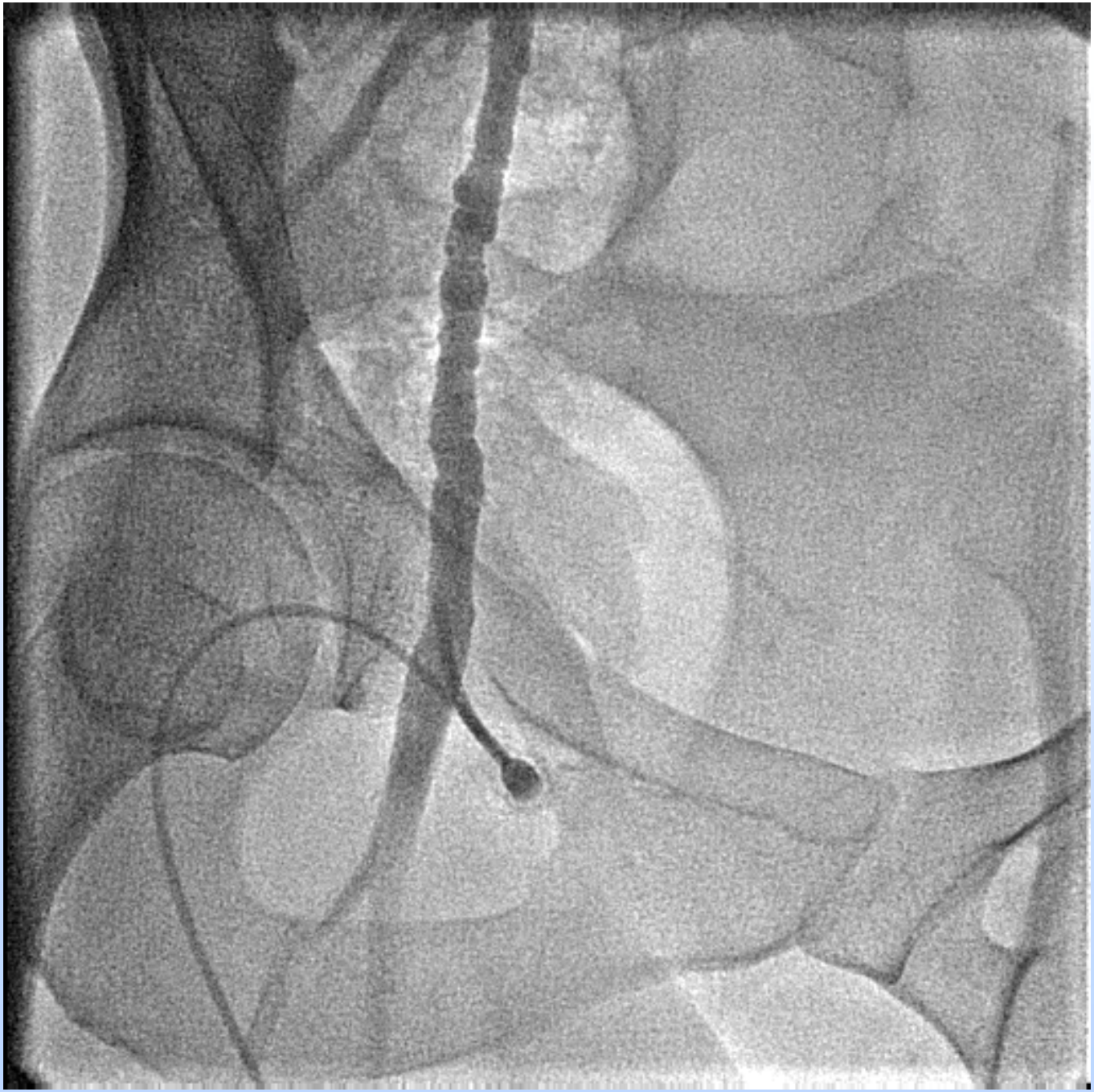
MAYO CLINIC

Spontaneous Coronary Dissection
N=87 retrospective series

- Mean age 42.6, Female 82%
- High-risk presentation (STEMI 49%, VF 14%)
- Associations
  - V low prevalence of atheroscl risk factors
  - Post-partum 18% (now 8%)
  - Physical/emotional stressors
  - …

MAYO CLINIC

Tweet MS et al, Circulation 2012



MAYO CLINIC

Fibromuscular Dysplasia
The principal association

N=50 SCAD Angio/CTA/MRA Saw J et al, JACC Intv 2013	FMD 86%	• Renal 58% • Cervical 47% • Iliac 49%
N=102 SCAD CTA neck-pelvis Prasad M et al, AJC 2015	FMD or dissection 68%	• Renal 46% • Cervical 49% • Iliac 49%

MAYO CLINIC

SCAD

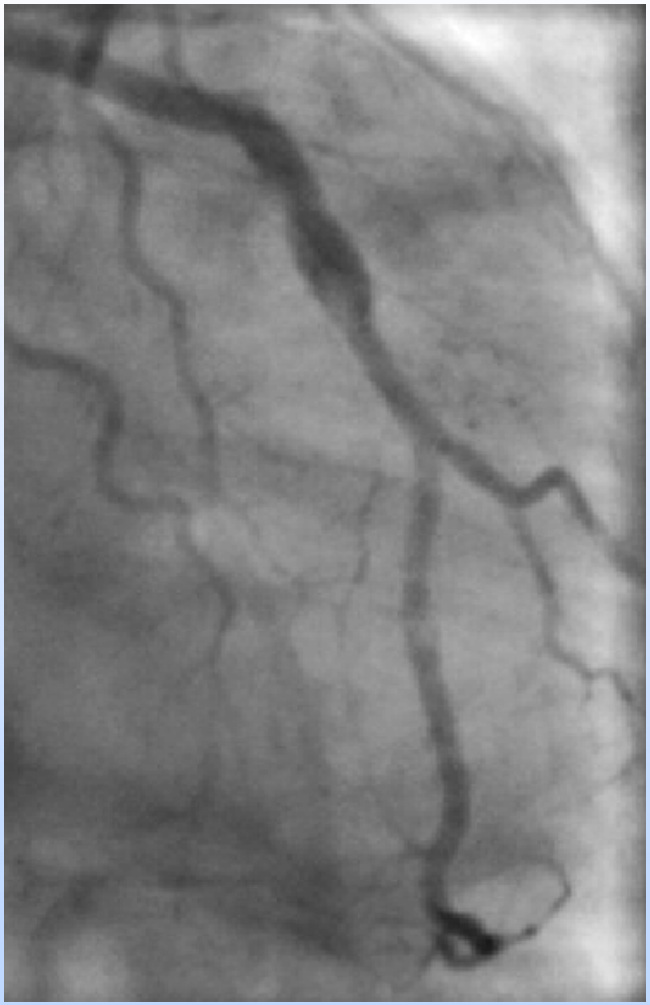
- Part of a systemic vasculopathy
- Linked with non-coronary FMD

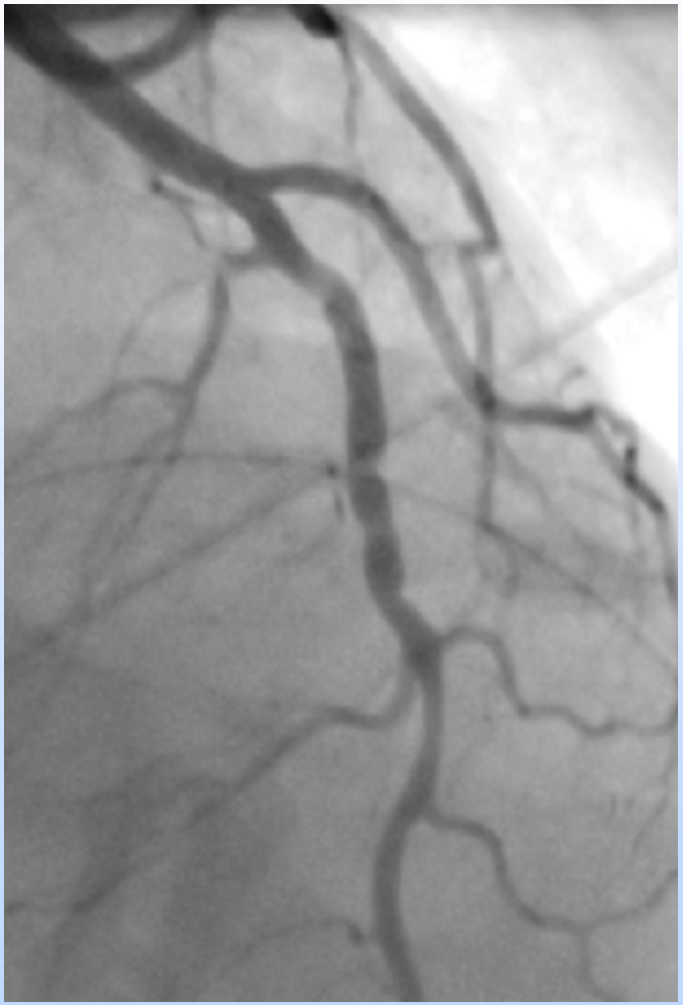
What is the coronary abnormality that predisposes to SCAD?

Is it Coronary FMD?

MAYO CLINIC

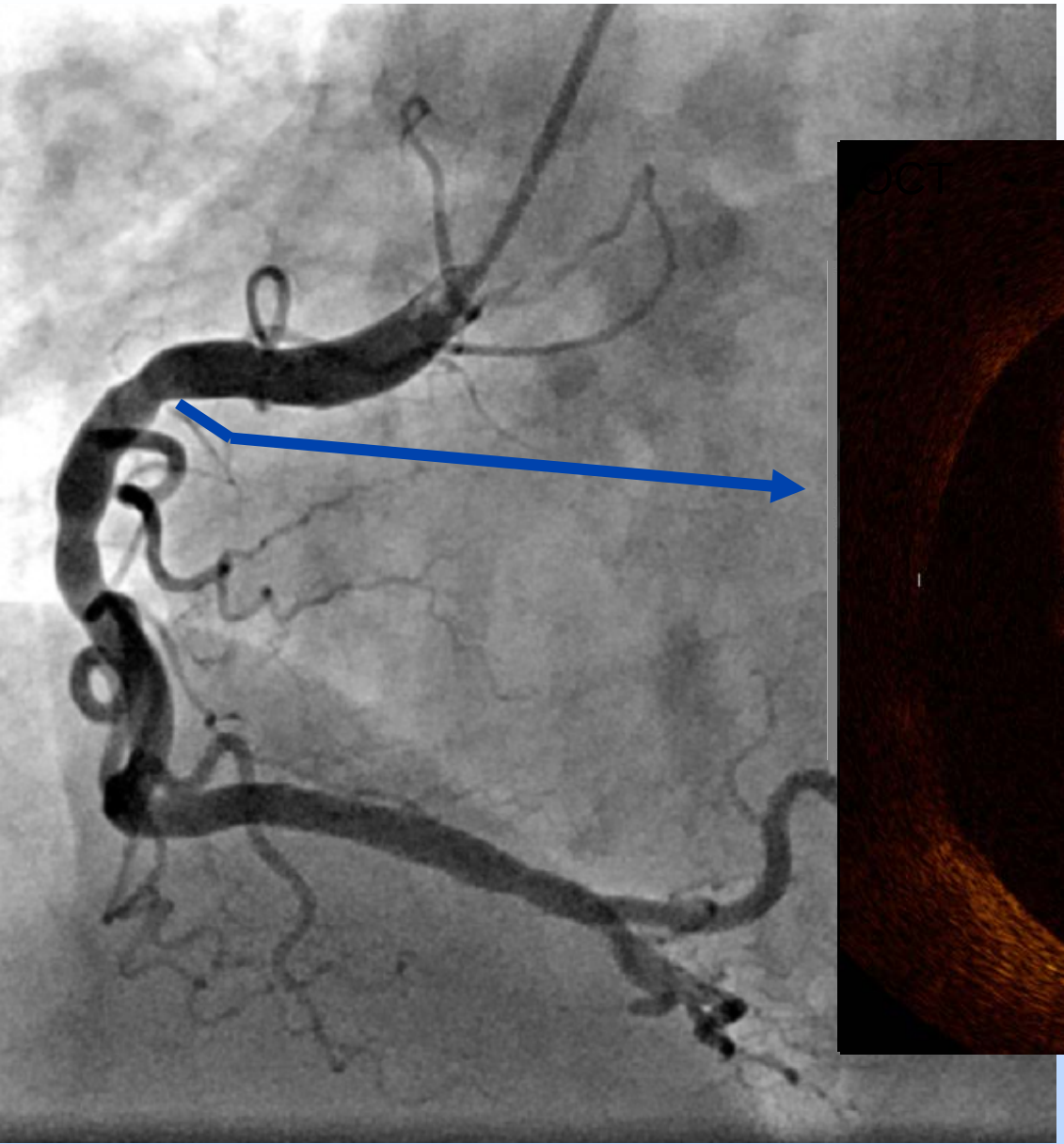
Coronary FMD?

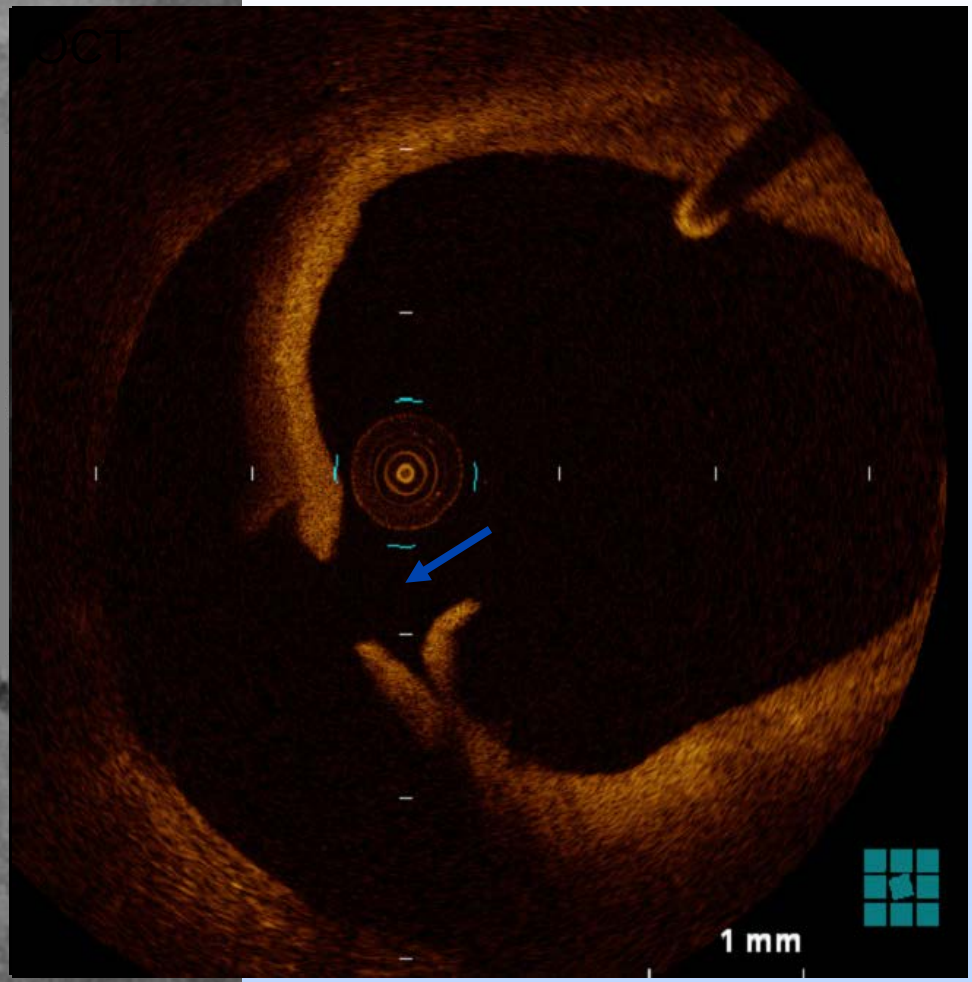




MAYO CLINIC

Coronary FMD?





Angiographic patterns in SCAD

First SCAD
N = 246

Vs
age gender HTN

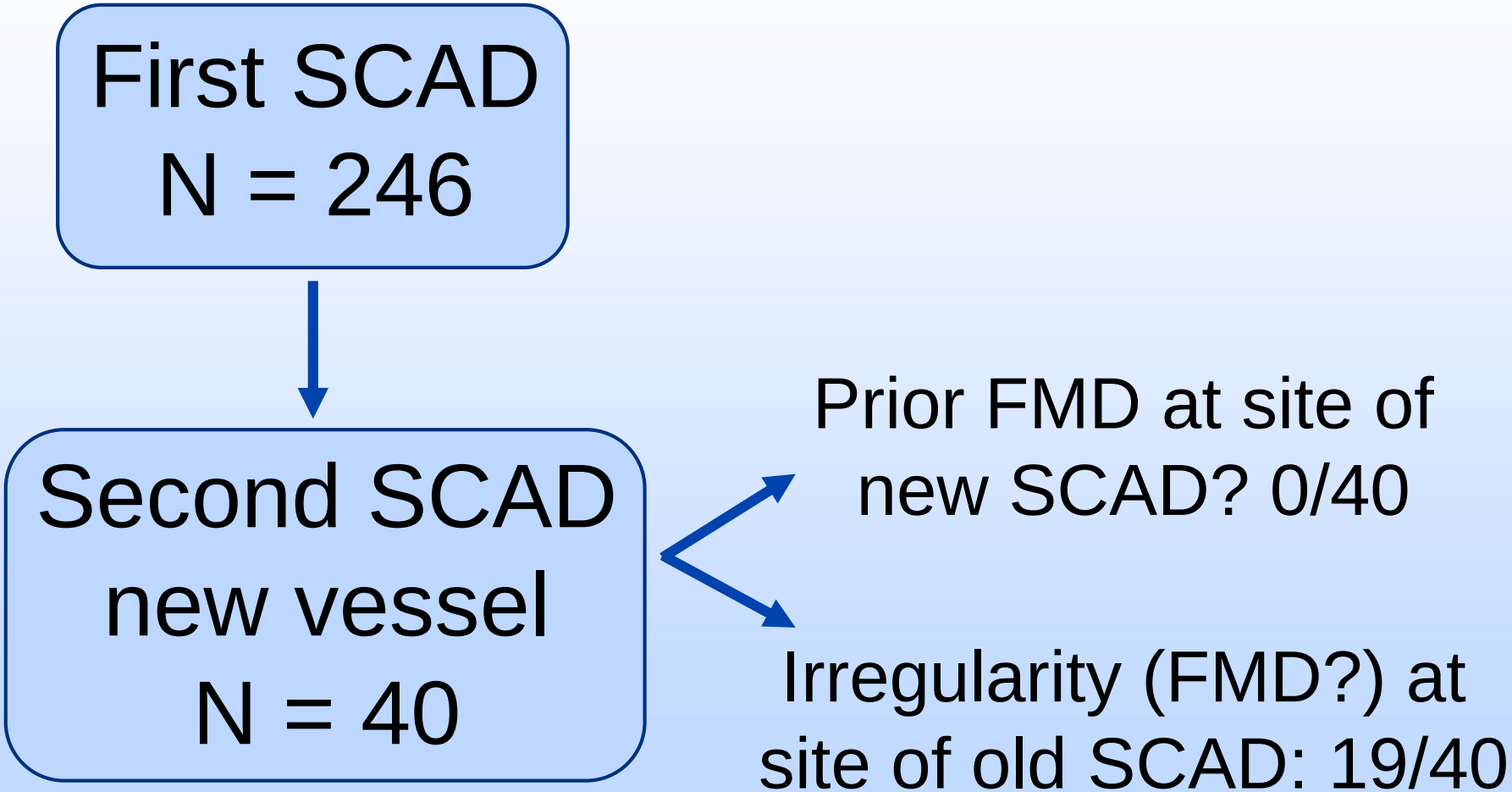
Controls
N = 313

Angiogram consistent with coronary FMD
2% vs 0% p=0.02

MAYO CLINIC

Eleid MF et al. Circ Intv 2014

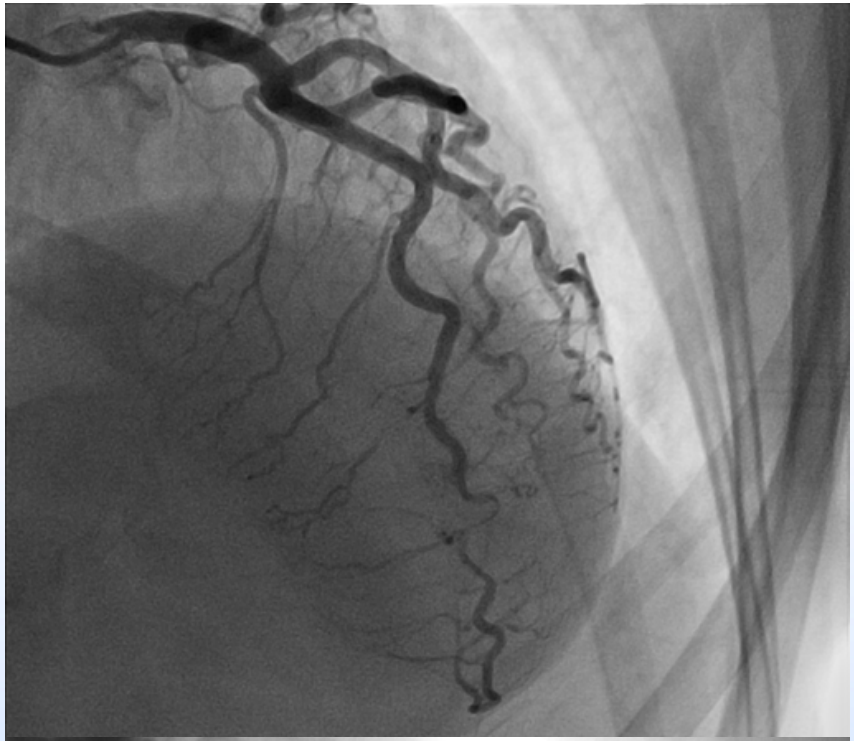
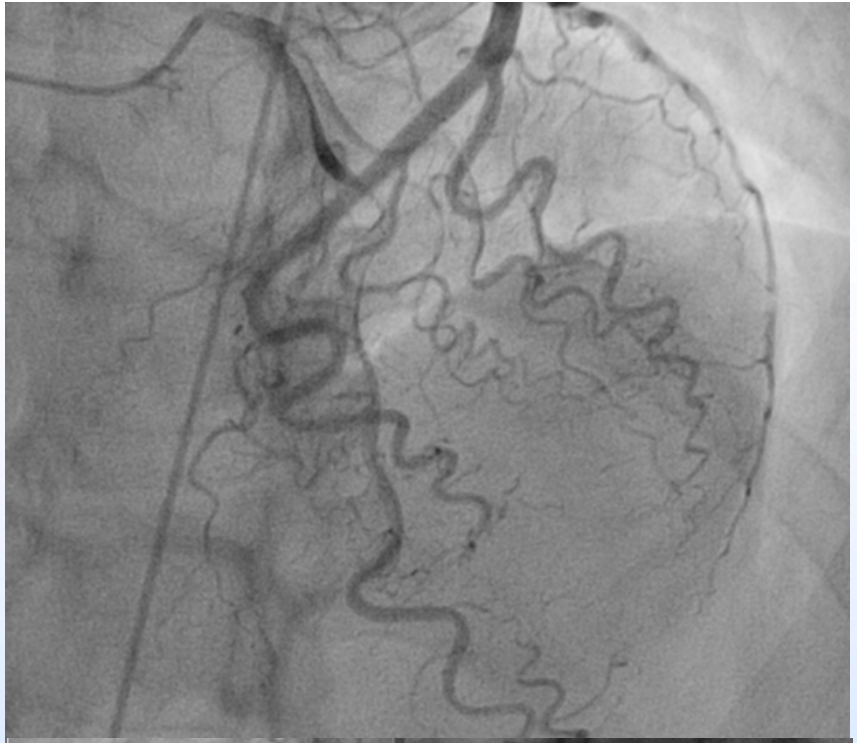
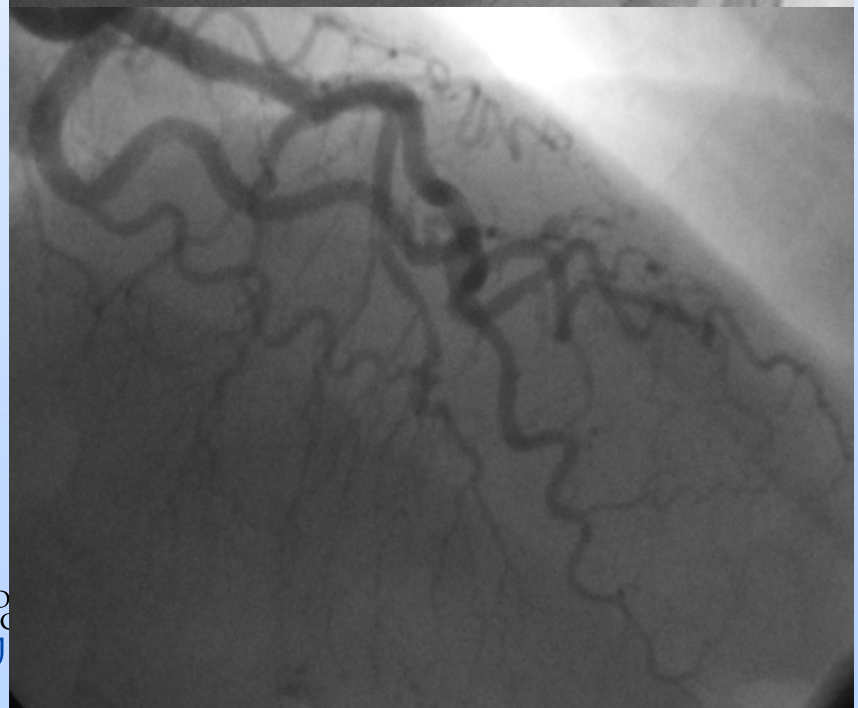
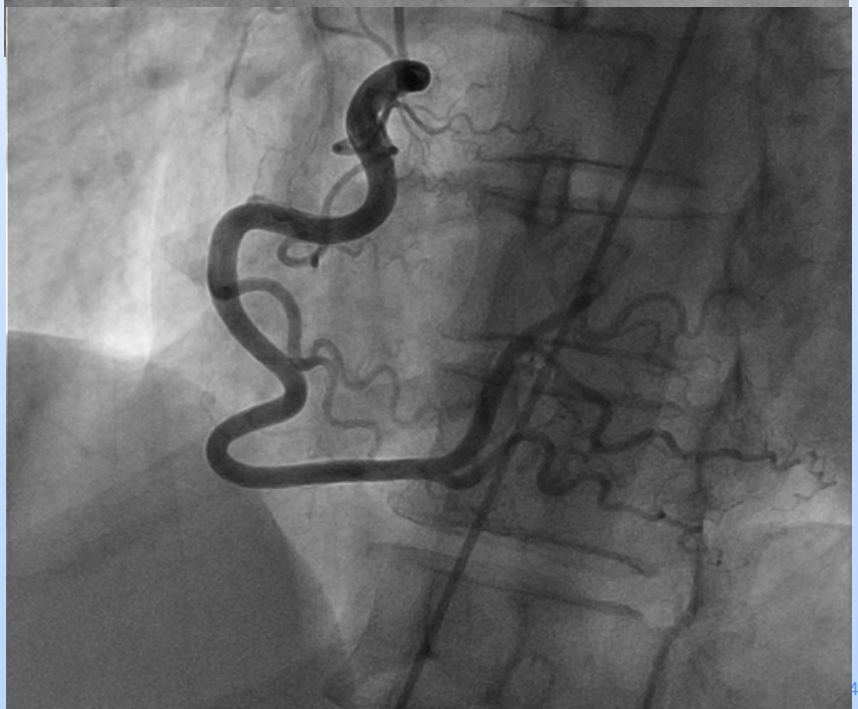
Angiographic patterns in SCAD



Eleid et al. Circ Intv 2014

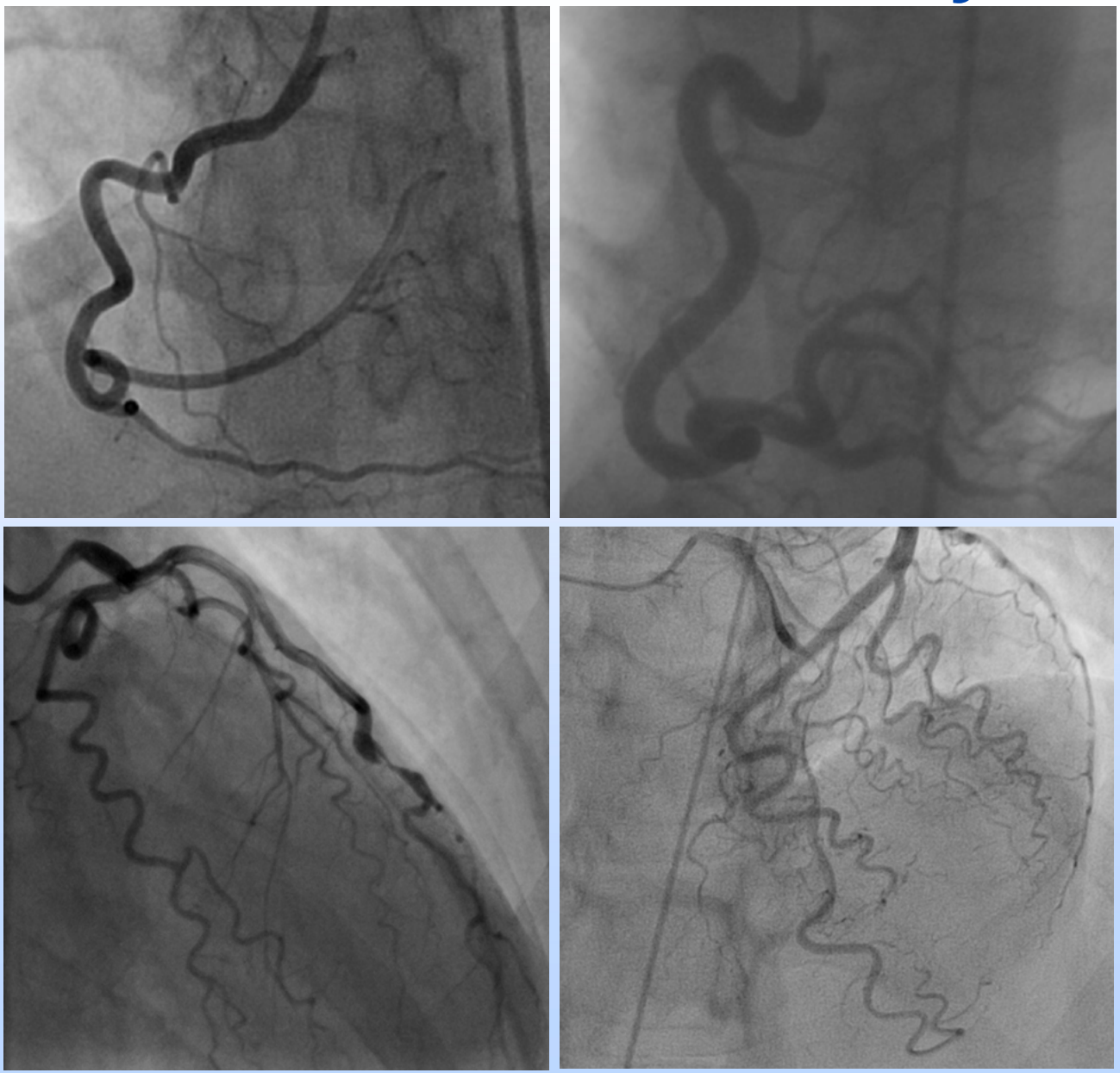
Other angiographic observations ?

MAYO CLINIC

MAYO CLINIC

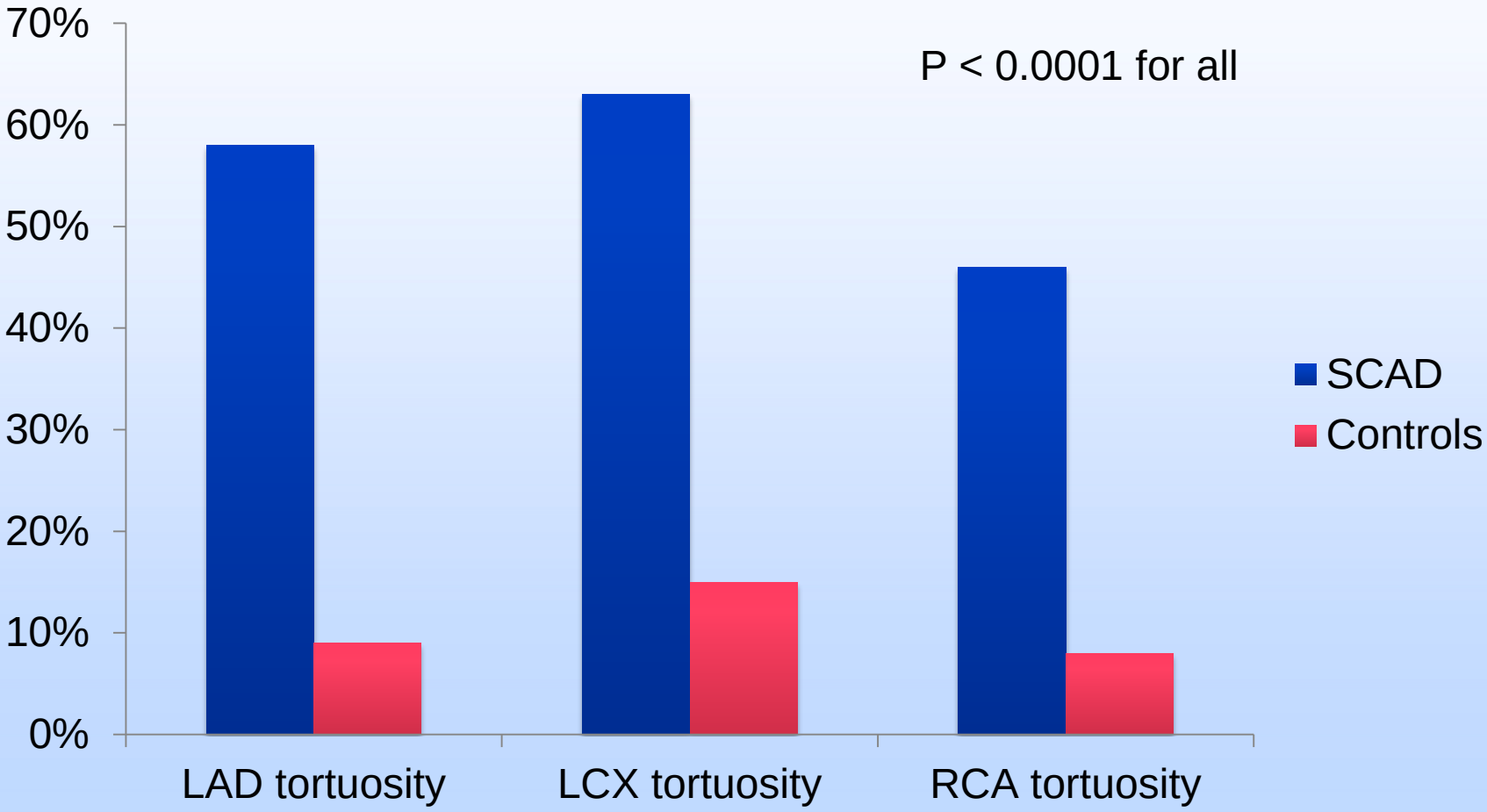
Corkscrew, multivessel symmetry



MAYO CLINIC

Prevalence of tortuosity

SCAD (n=246) vs Controls (n=313)



MAYO CLINIC

Eleid MF et al. Circ Intv 2014

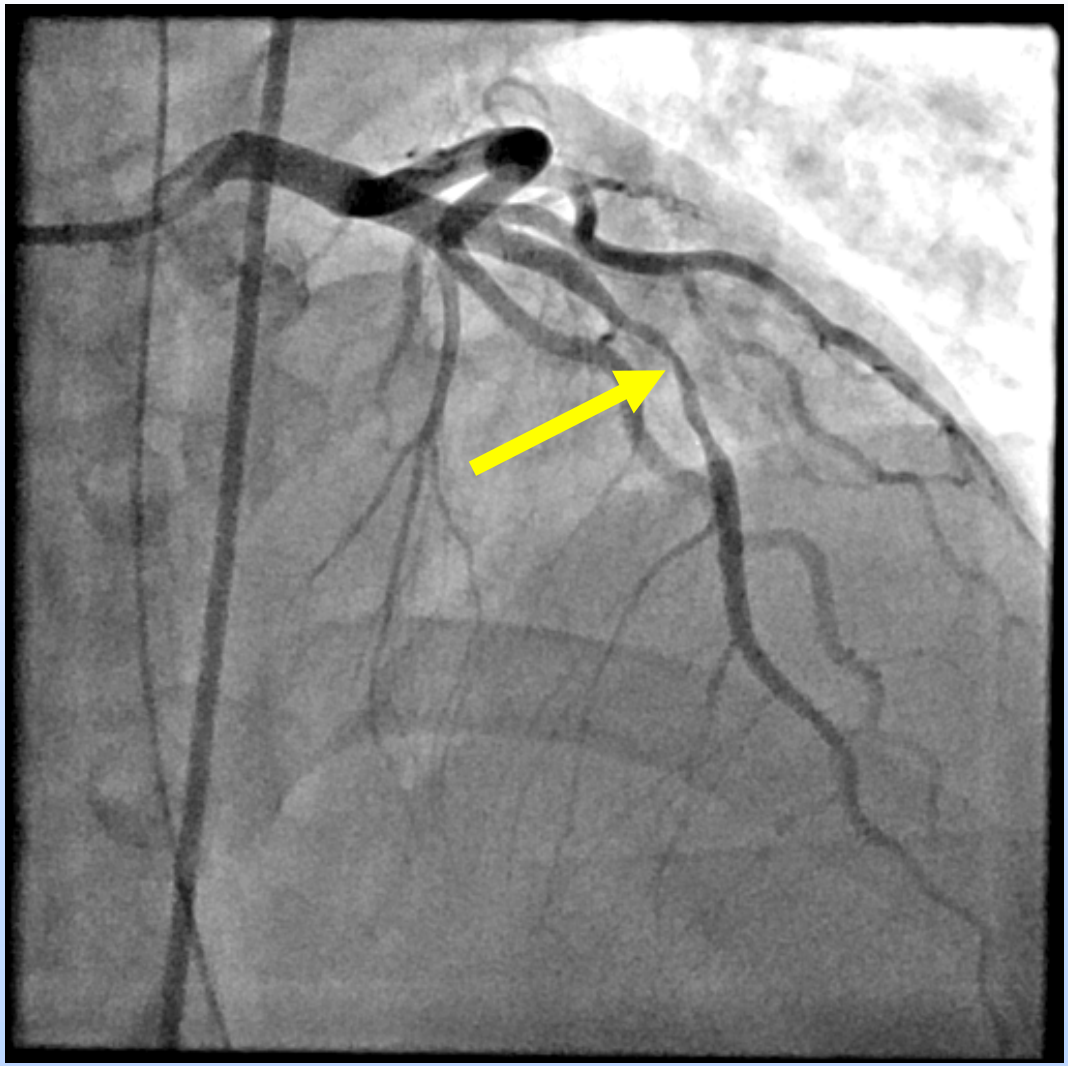
Coronary tortuosity in SCAD

- Highly prevalent. Except P-partum SCAD
- No more prevalent in hypertensive SCAD
- Much more prevalent in FMD+ vs screen negative CTA ($p<0.001$)
- Tort. Index > 5 predicts recurrent SCAD
- 80% of second SCAD (new vessel, n=40) occurred in segments of prior tortuosity

MAYO CLINIC

Eleid MF et al. Circ Intv 2014

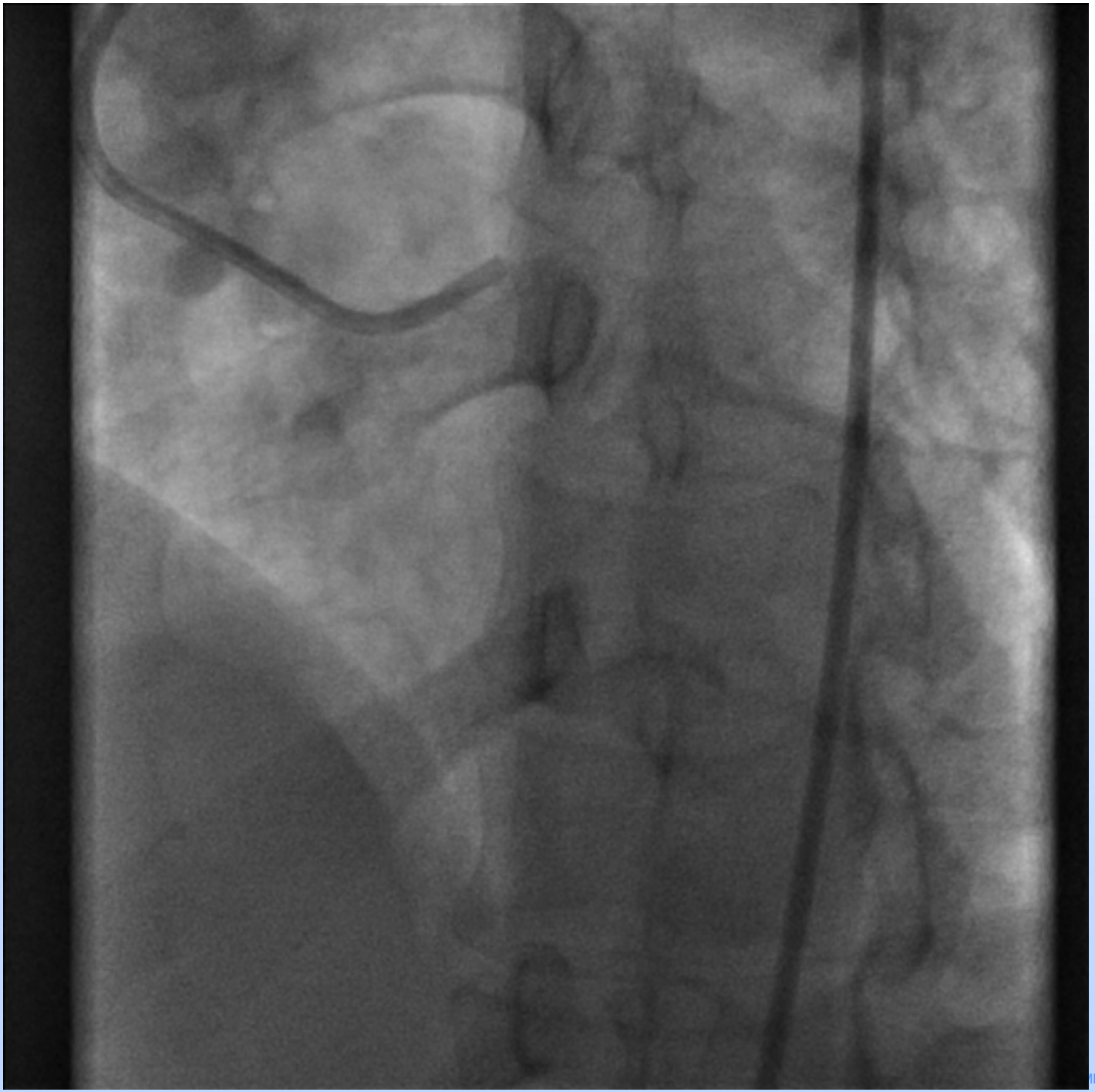
First case 42 yrs Female
So should we stent the LAD?

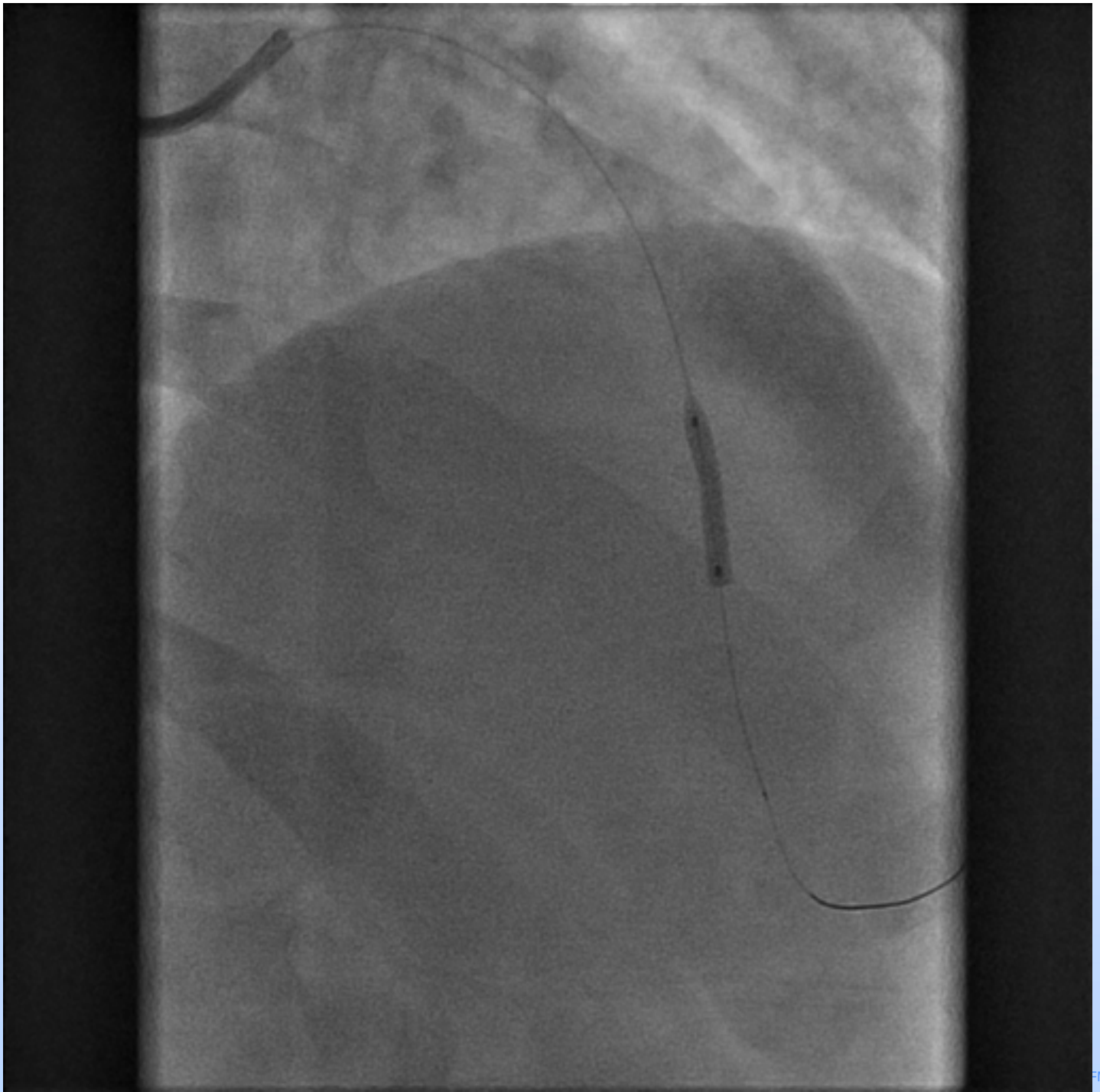


MAYO CLINIC

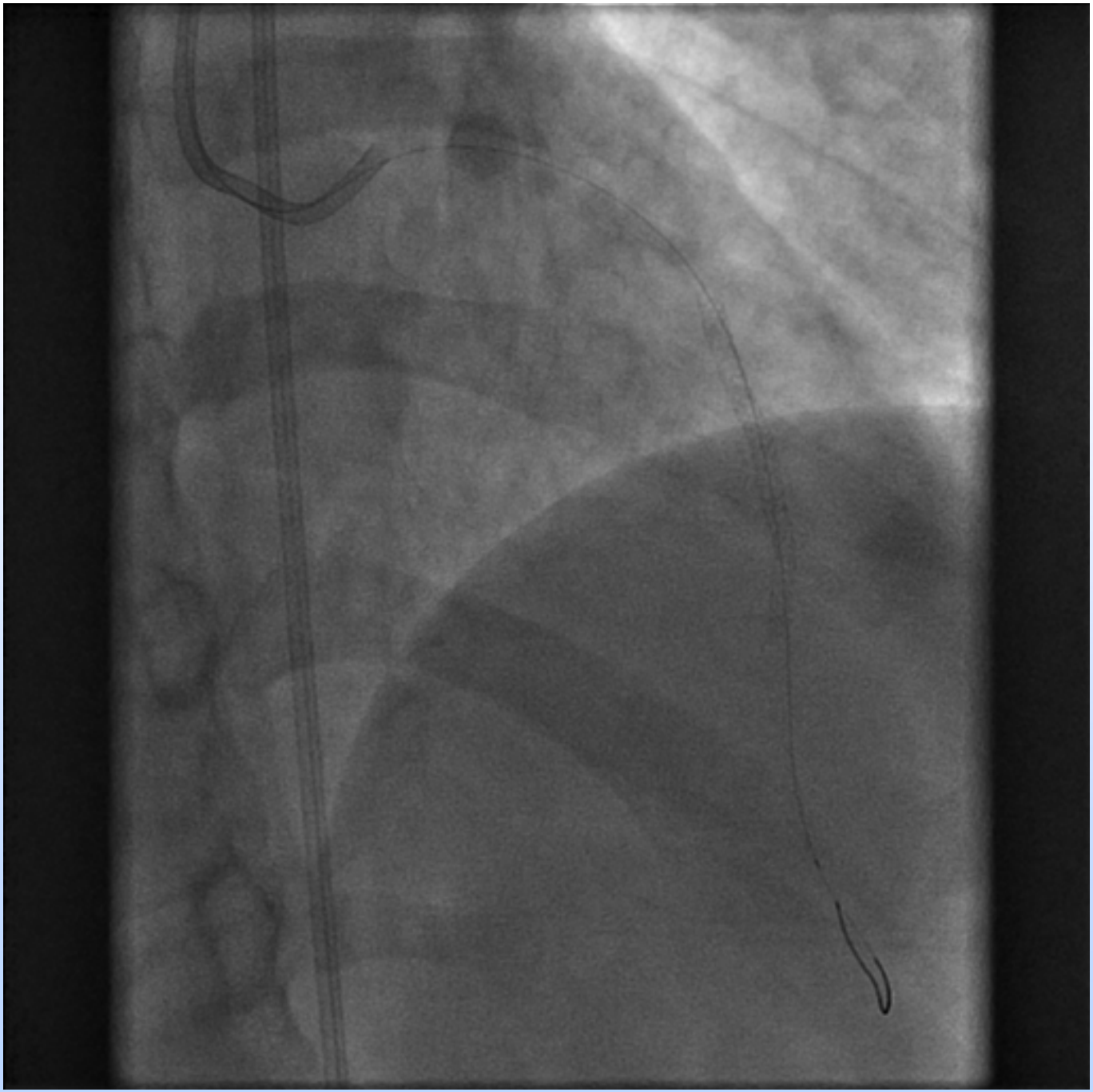
How does SCAD respond to PCI?

MAYO CLINIC

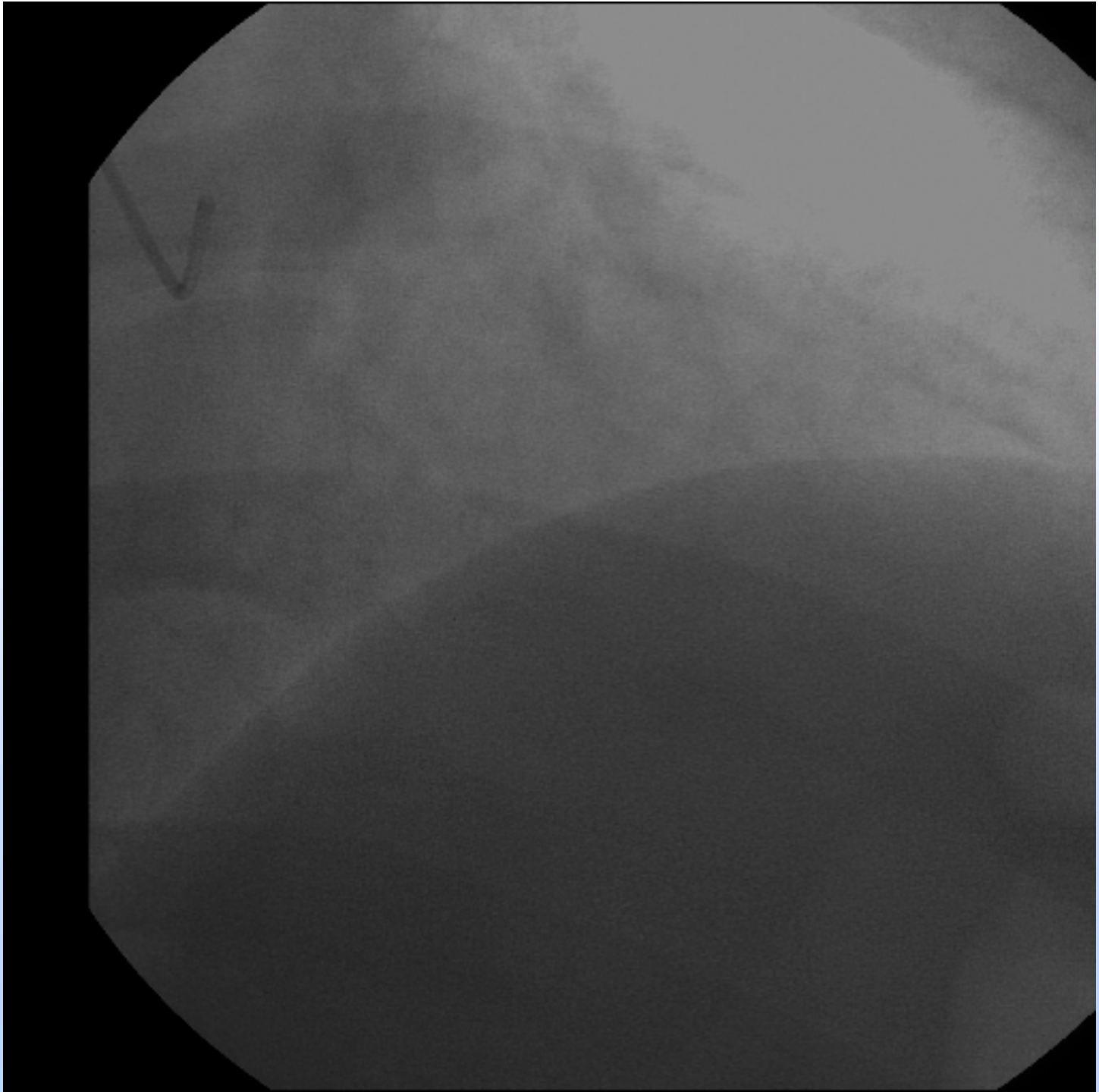


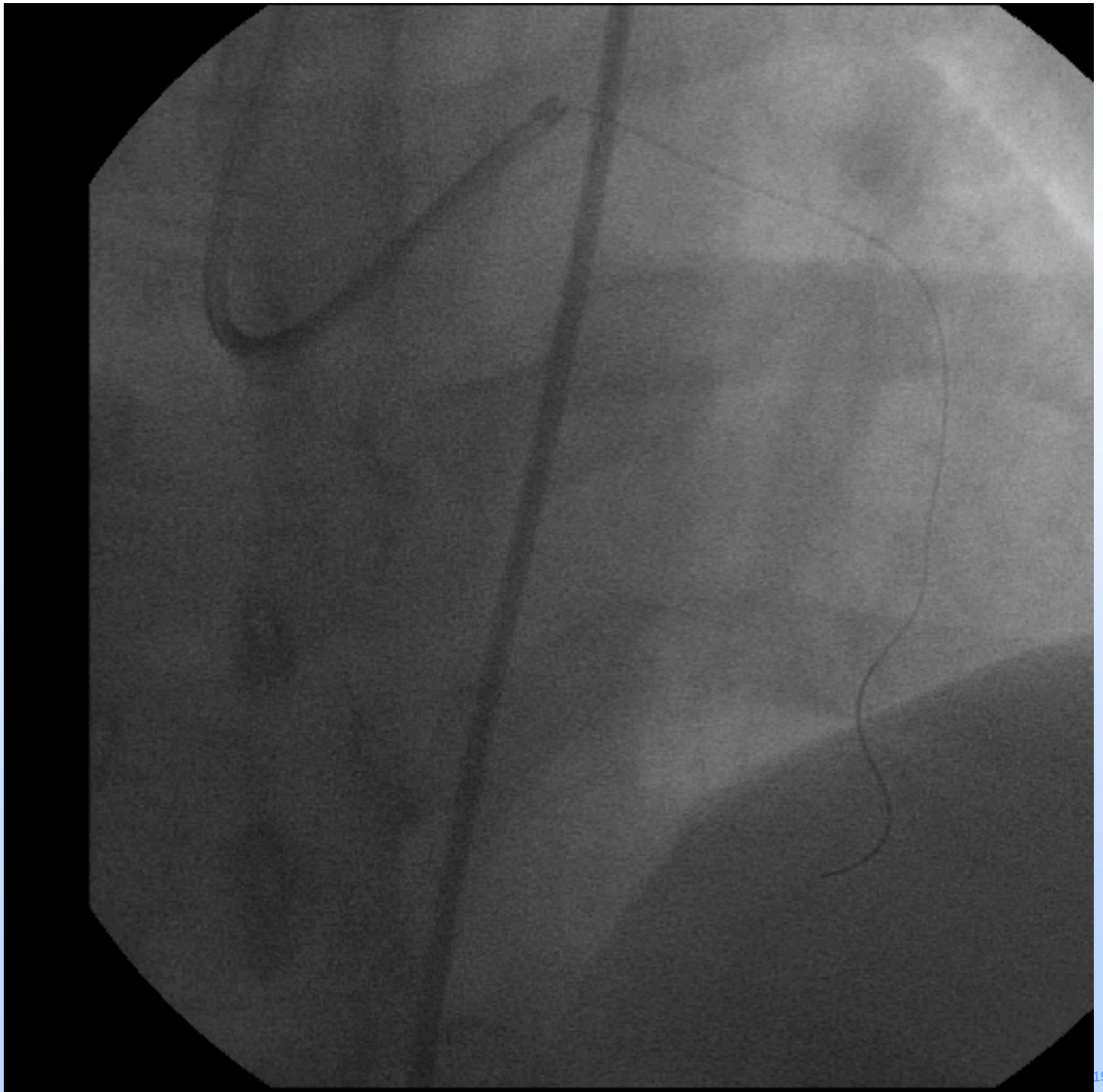


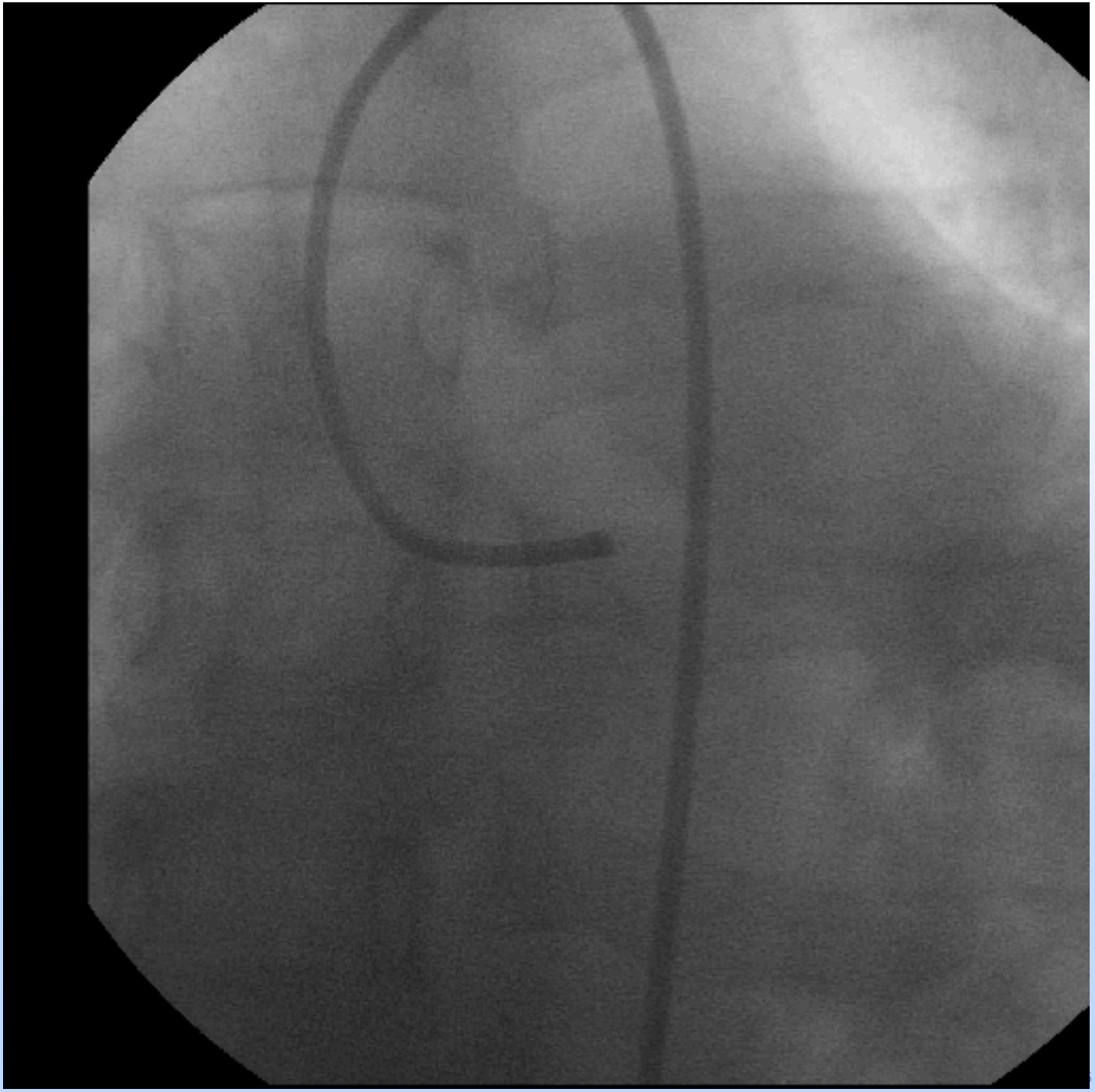
MAYO CLINIC



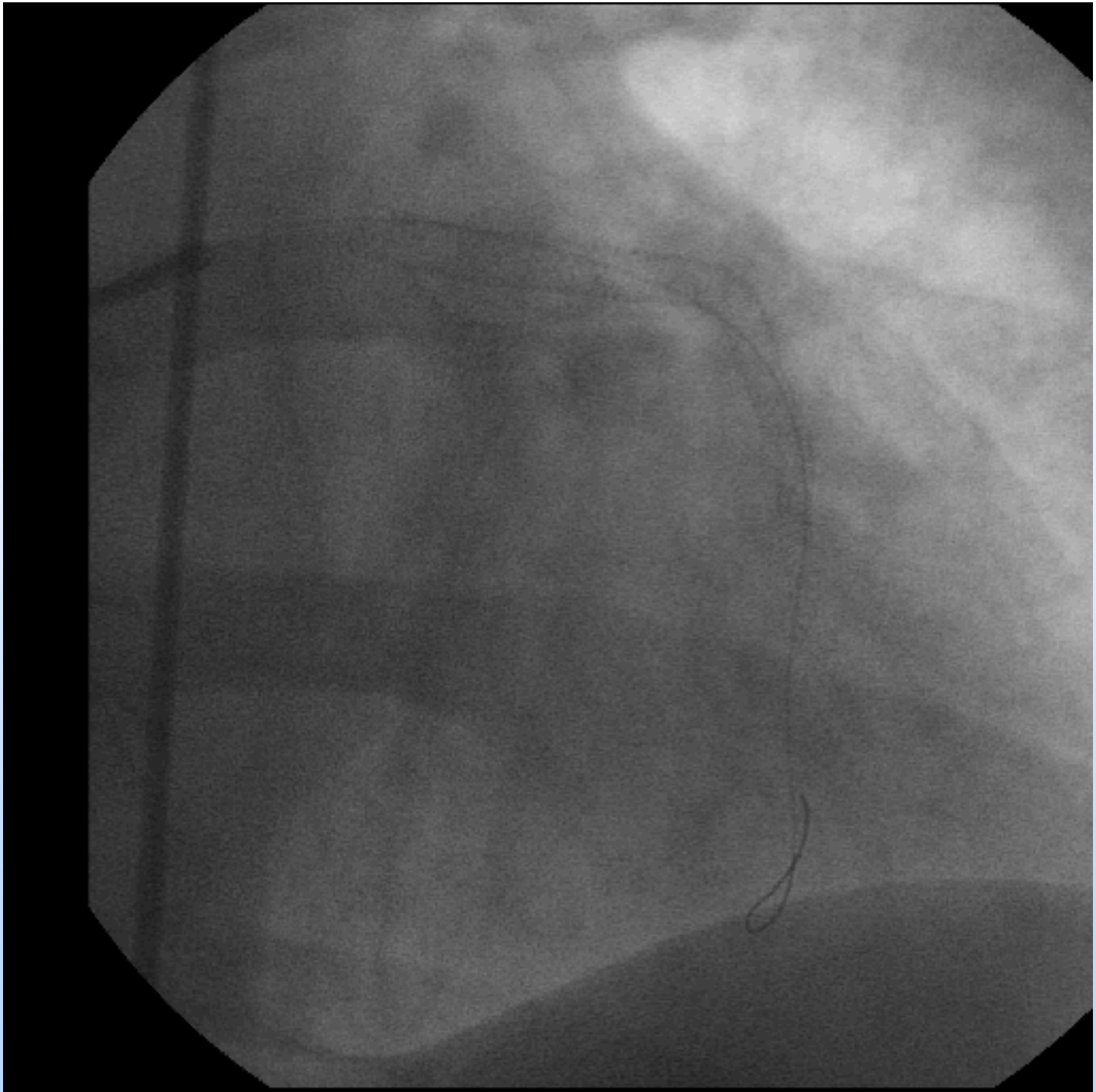
MAYO CLINIC



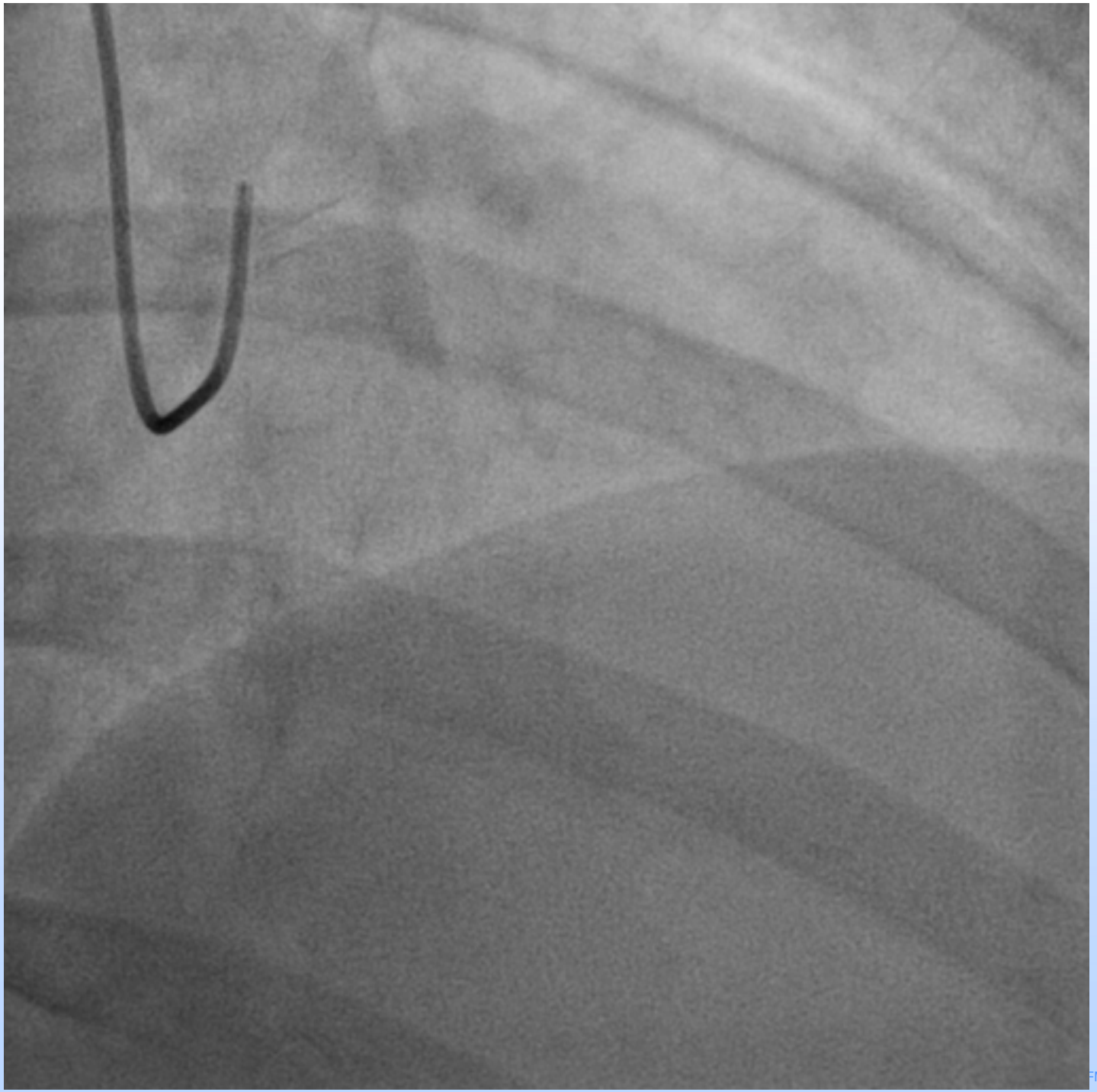




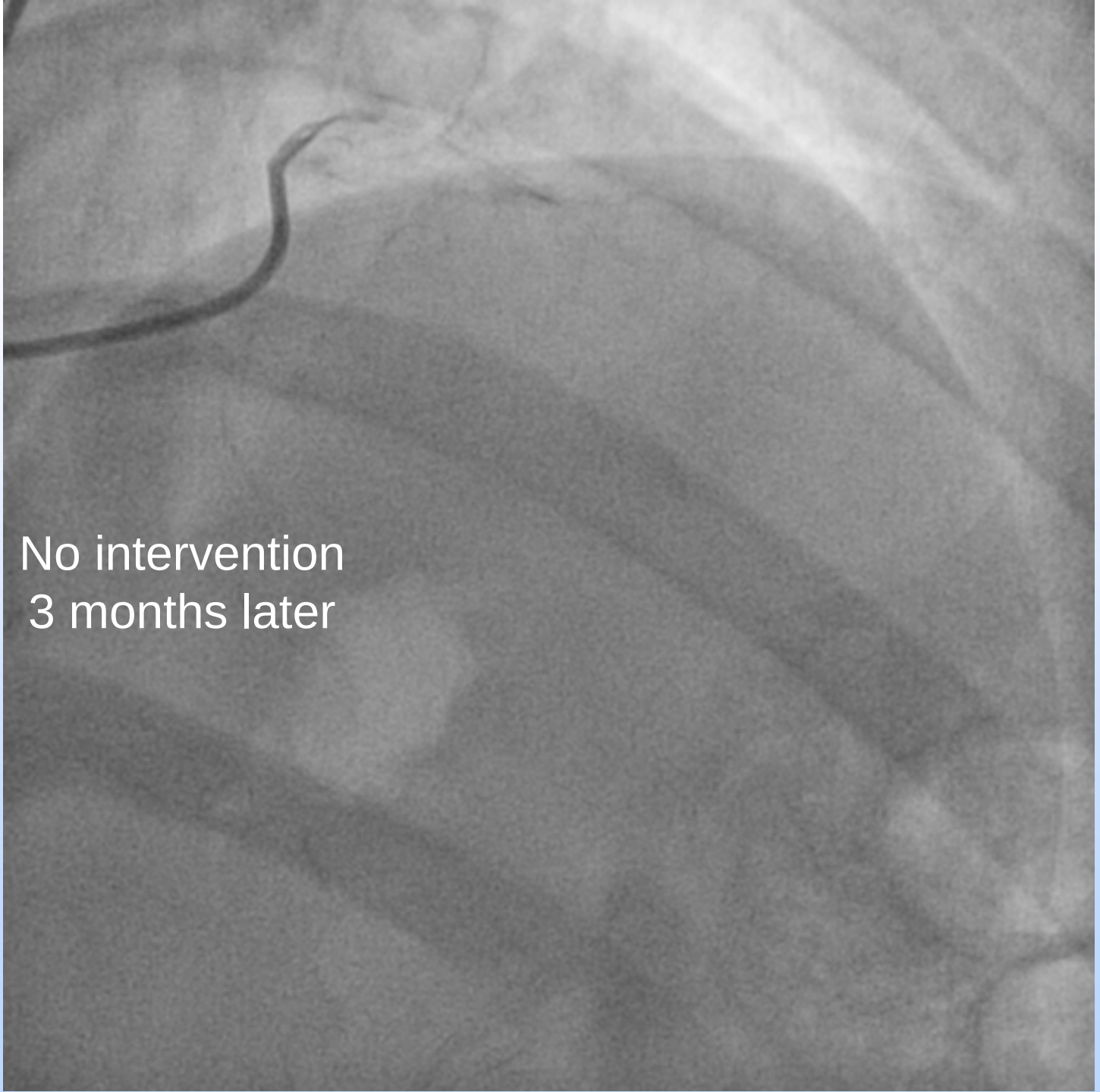
MAYO CLINIC



MAYO CLINIC



MAYO CLINIC



MAYO CLINIC

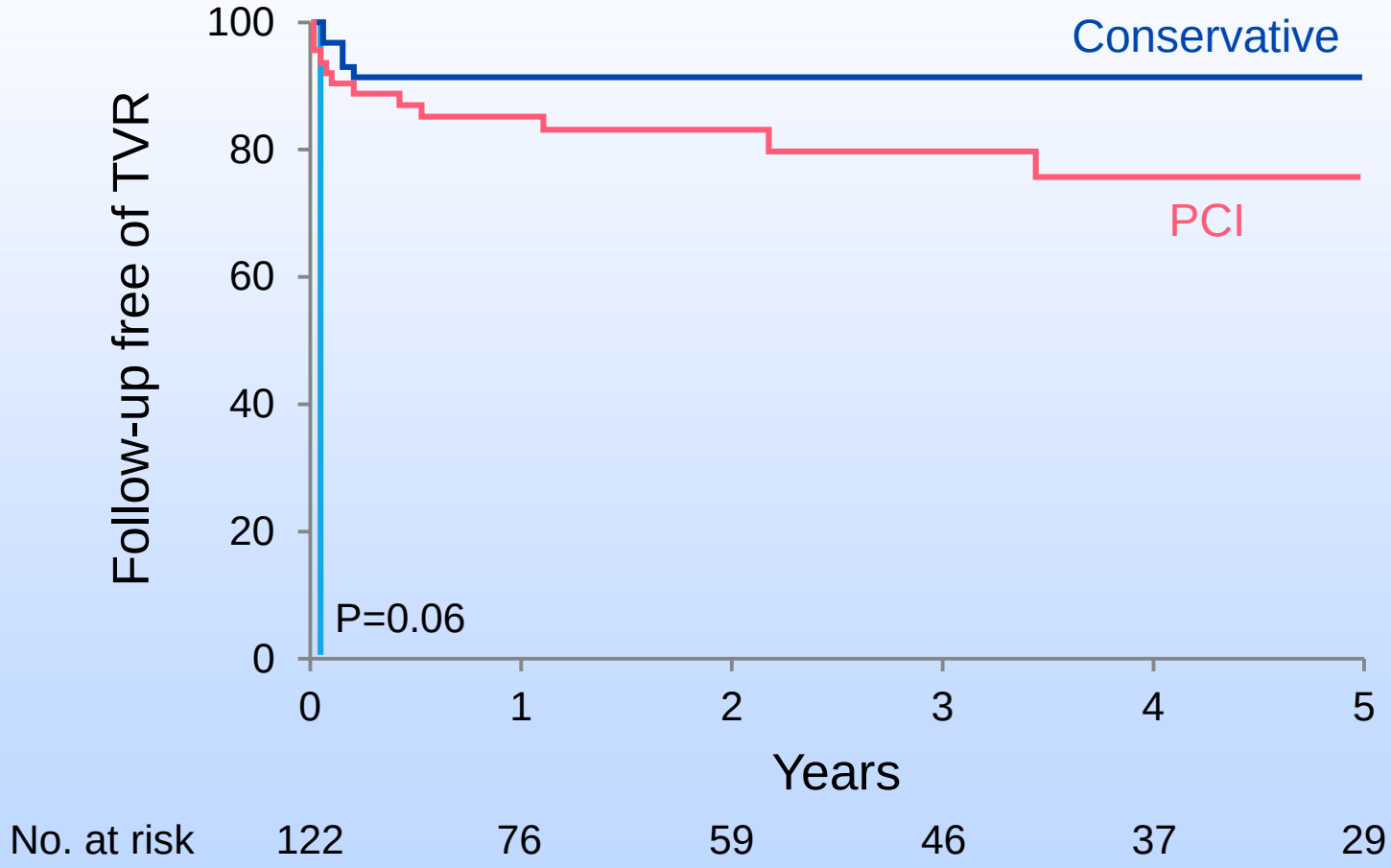
PCI vs Conservative Rx for SCAD
A retrospective, biased study

	PCI vessel occlusion (n=46)	PCI normal flow (n=41)	Conservative (n=94)
In-hospital outcomes			
Death	2%	0	0
Emergent CABG	17%	15%	4%
Extension Rx PCI	0	0	6%
Extension Rx consv	3%	3%	2%
PCI technical failure	27%	47%	NA

Failures: - Unable to enter TL with wire
- Loss of flow after stent
No angiographic predictors of PCI success

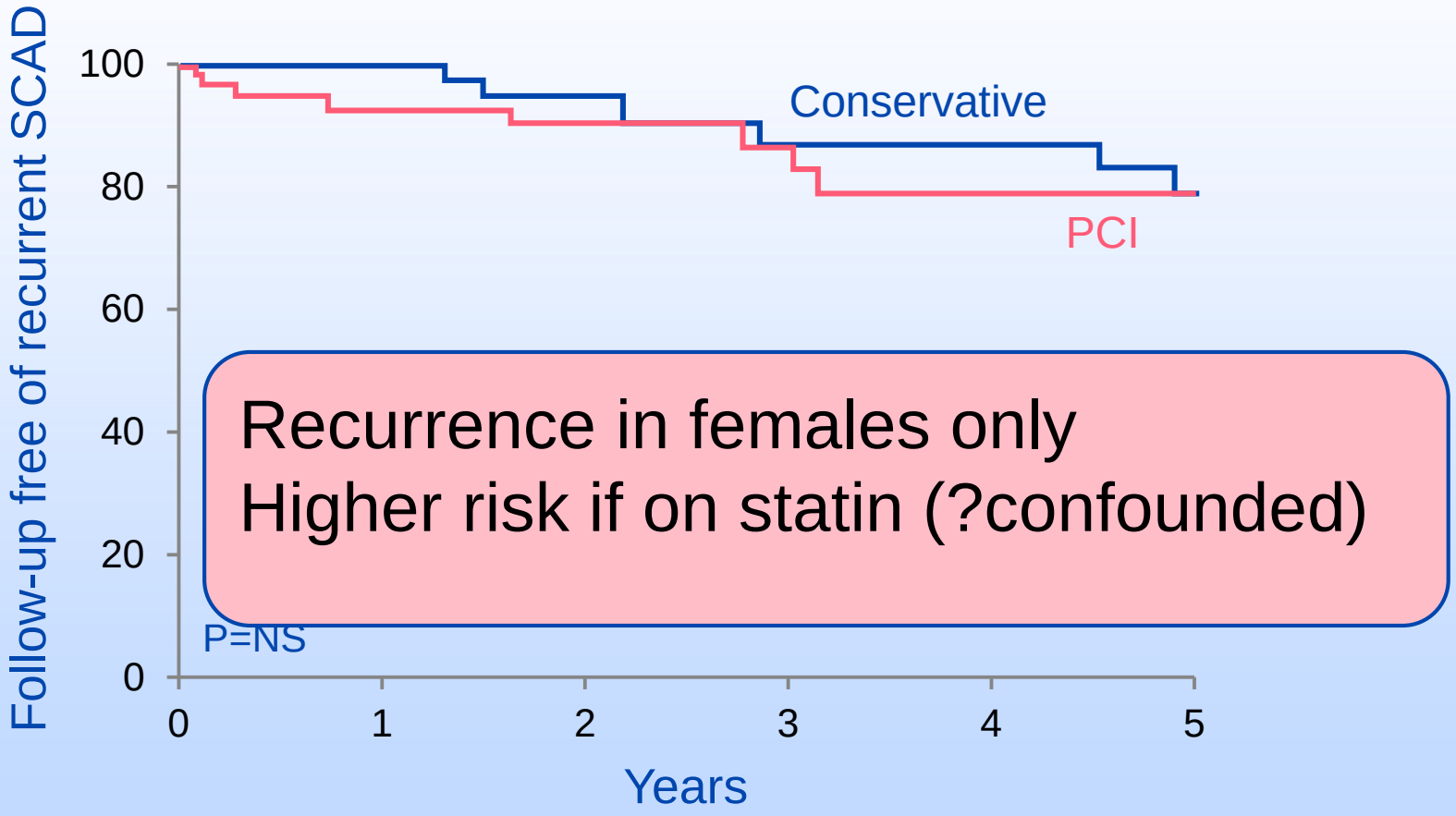
Tweet MS et al, Circ Intv 2015

Target Vessel Revascularization



Tweet MS et al, Circ CV Intv 2014

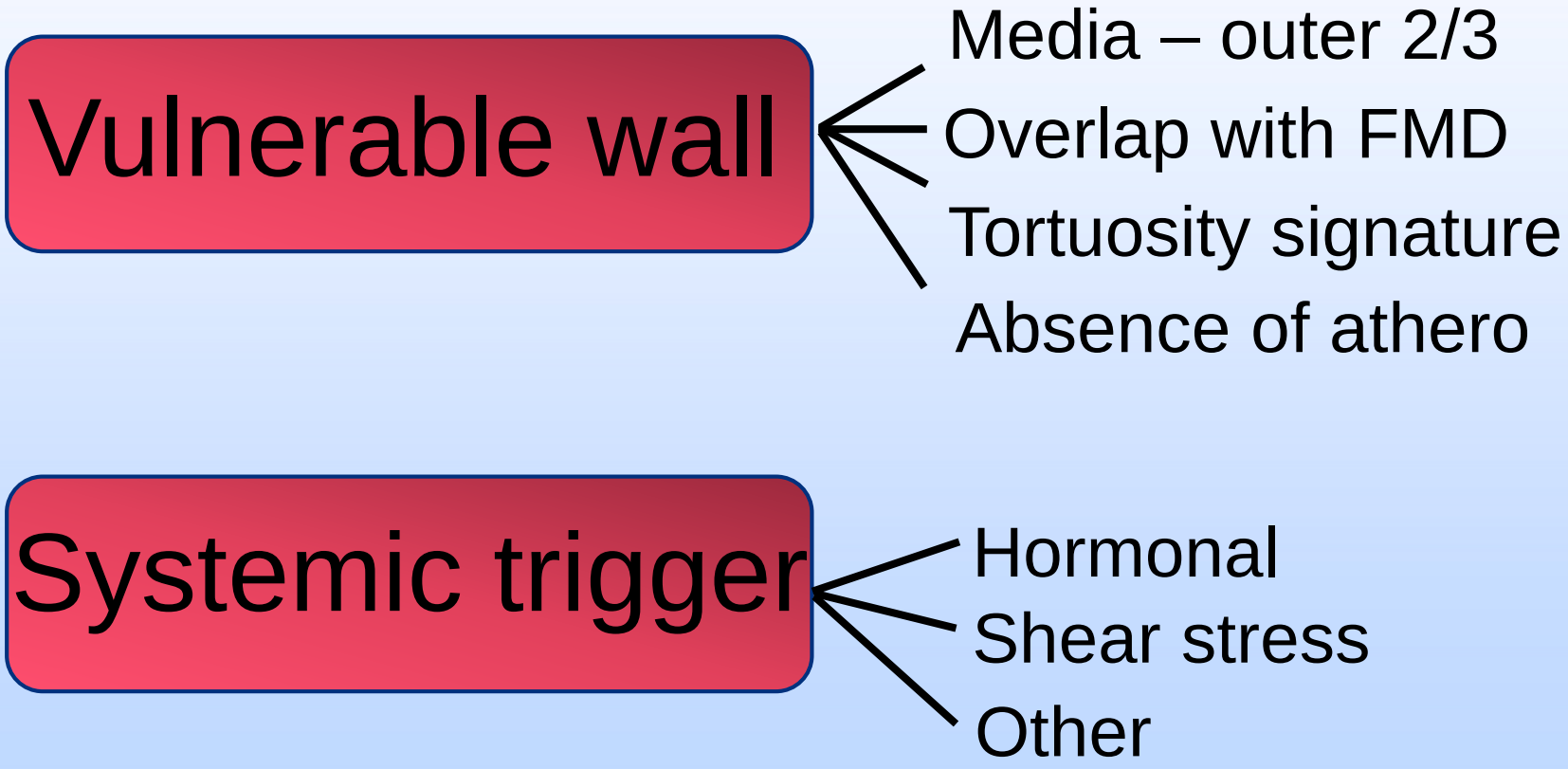
Recurrent SCAD in 18%



Recurrence in females only
Higher risk if on statin (?confounded)

Tweet MS et al, Circ CV Intv 2014

SCAD pathogenesis
Hypothesis, from clinical observations



MAYO CLINIC

Spontaneous Coronary Artery Dissection in 2015

- Under-recognized cause of MI in females
- Coronary tortuosity
- Vascular FMD
- Manage conservatively if possible
- Follow closely, 1:6 risk of recurrence
- Vulnerable wall+ trigger hypothesis: studies underway

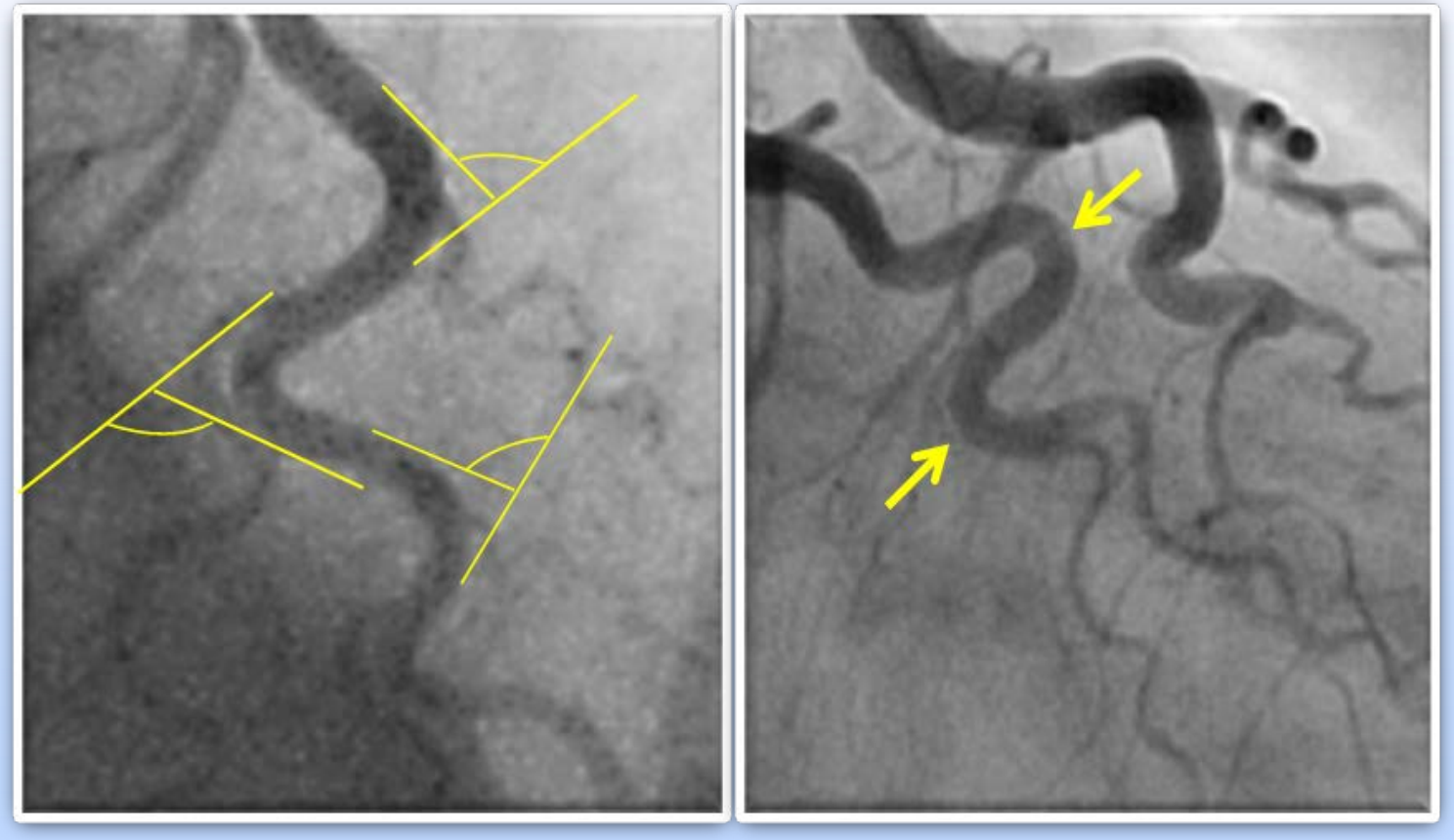
MAYO CLINIC

Thank you for your time

gulati.rajiv@mayo.edu

Tortuosity quantification and index

- **End-diastole**
- **Vessels ≥ 2 mm diameter**



MAYO CLINIC

Eleid MF et al. Circ Intv 2014

SCAD pathophysiology

- Part of a systemic vasculopathy
- Coronary architectural abn is unclear
- Characterized by coronary redundancy
  - Diagnostic clue
  - Prognostic marker
  - May be involved in pathogenesis
- Characterized by angiographic healing if left alone vs adverse response to PCI

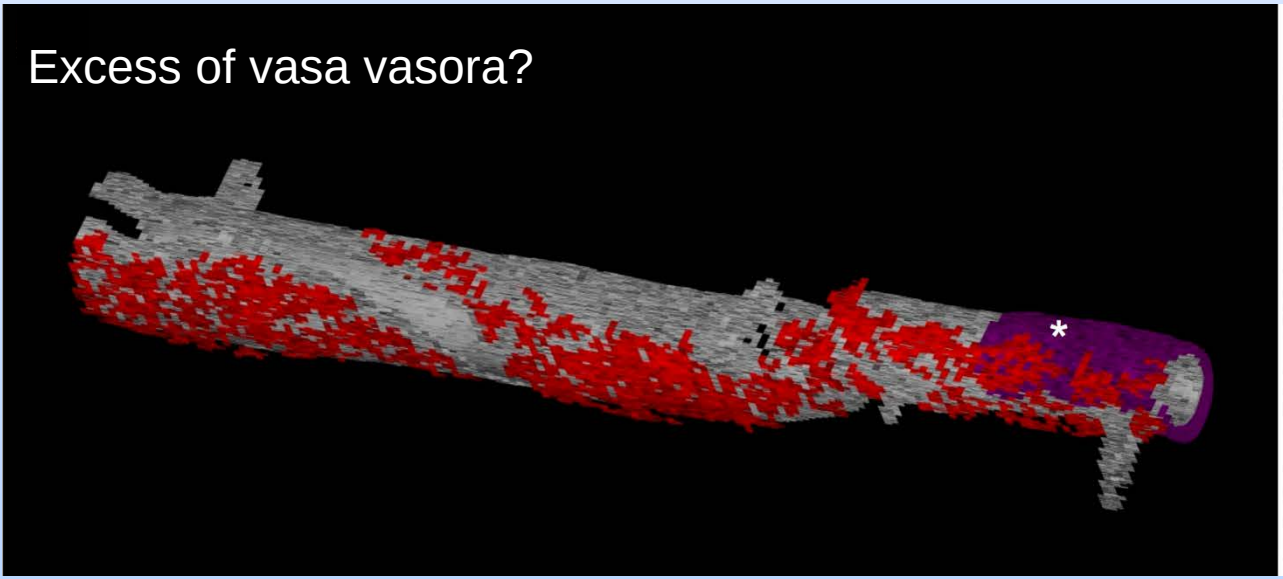
MAYO CLINIC

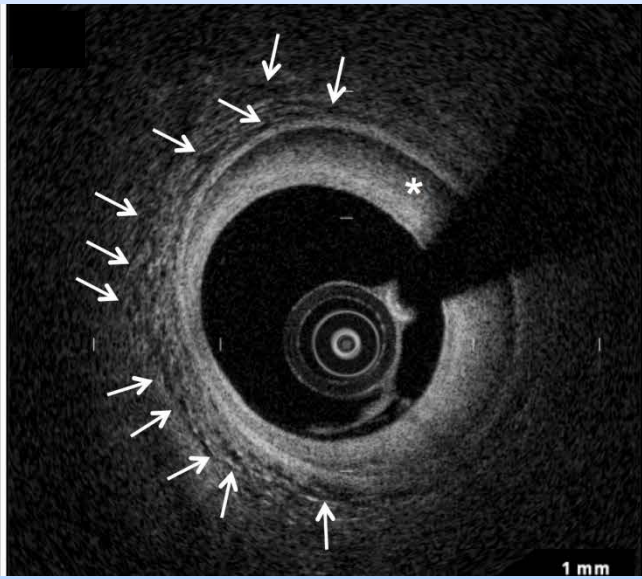
Any other clues on mechanism?

Vulnerable wall

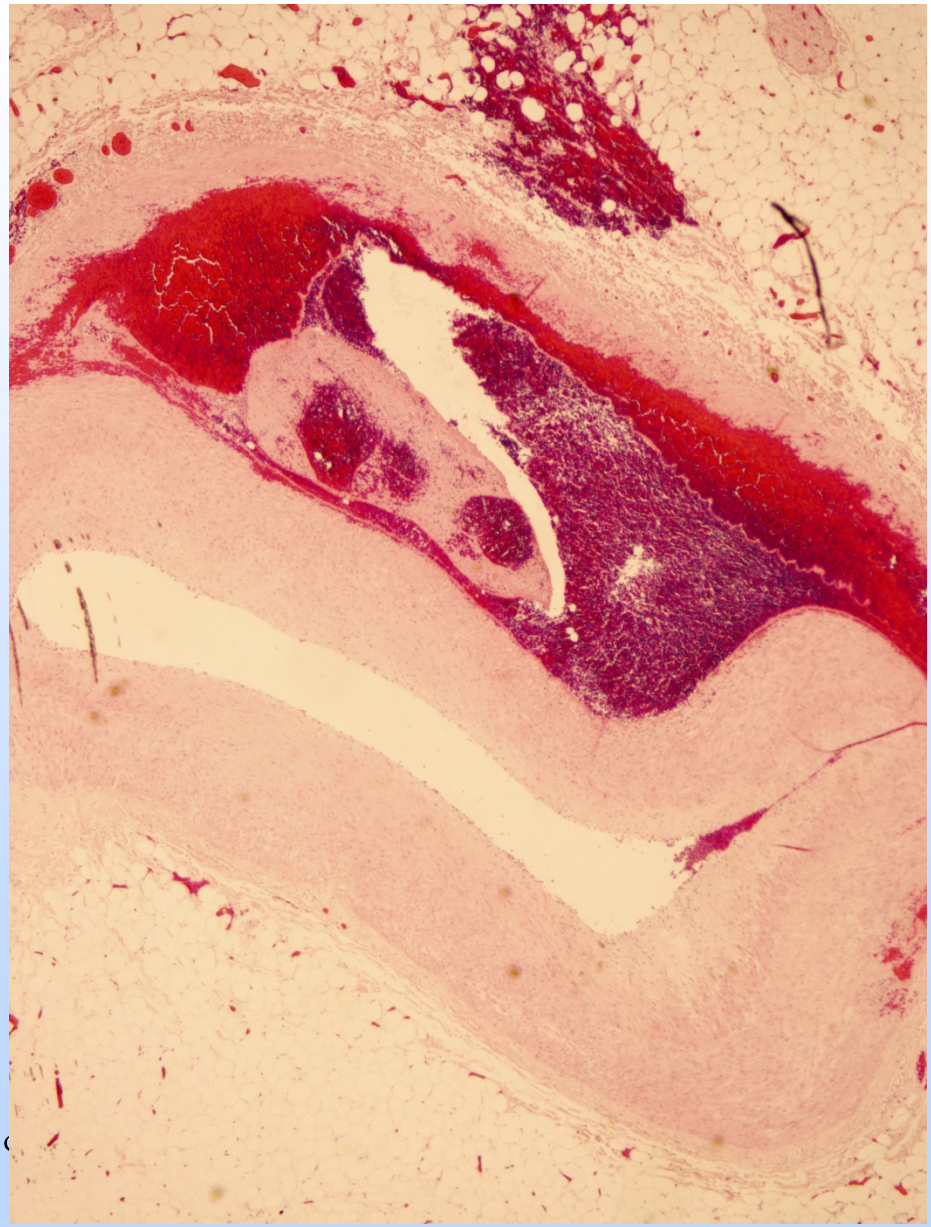
Medial hematoma ⟷ Intimal dissection

- N=8/9 near-normal endothelial function
- N=46 recurrent SCAD: 80% occur in segments of prior tortuosity

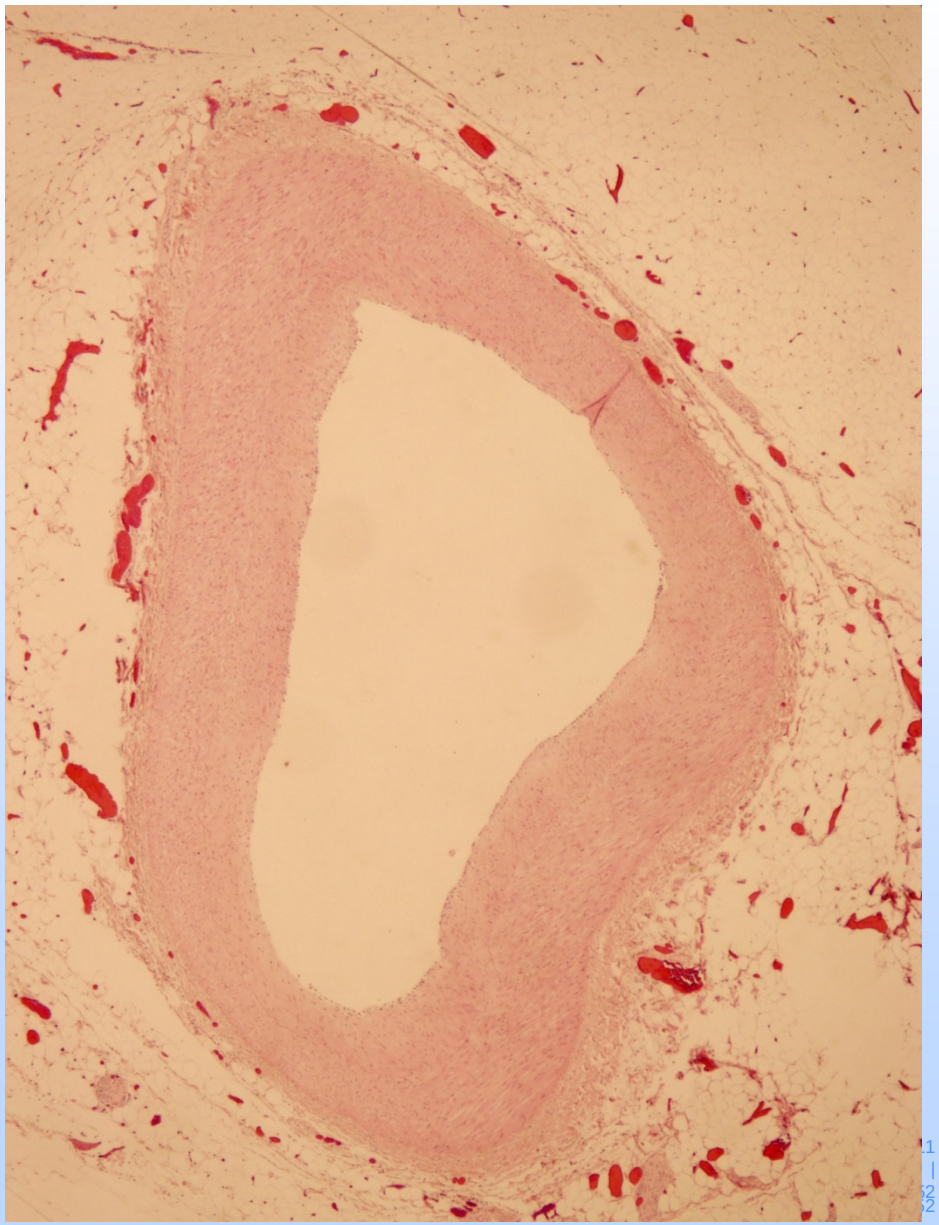


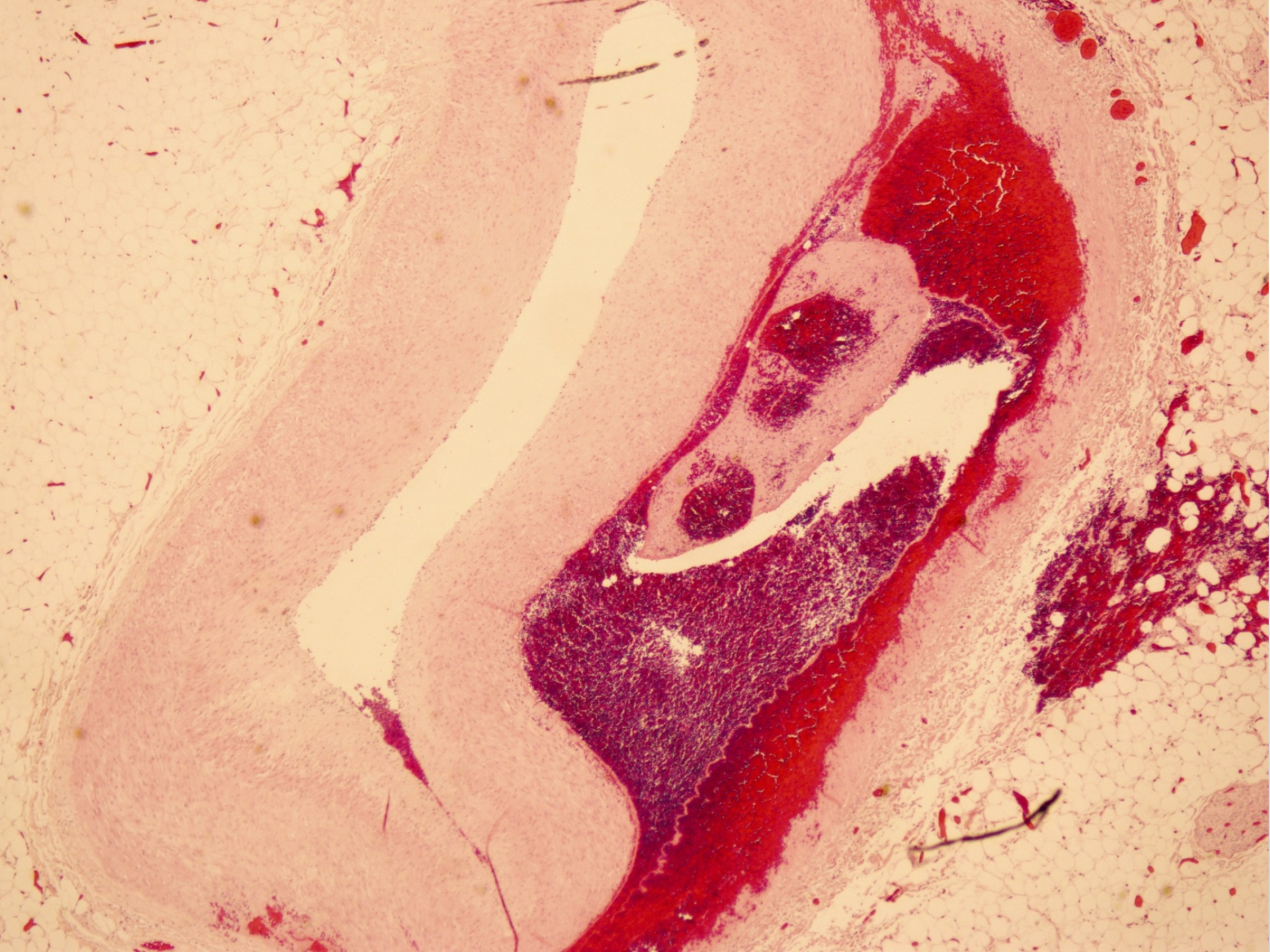


SCAD Left main



“Normal” Cx





How common is SCAD as a cause of sudden death?

N=150 sudden death, age < 35

- Coronary disease N = 48
- Non-atherosclerotic N = 16

Corrado D et al, BHJ 1992

N= 50 non-athero coronary disease

- Coronary anomaly N = 24
- Spontaneous dissection N = 10

Hill SF et al, Heart 2010

N= 9 sudden death from SCAD

- Initial pathology “normal” in N=4

“The macroscopic changes…can be difficult to detect and mistaken for postmortem thrombus or athero…”

MAYO CLINIC

Any role at all for PCI in SCAD with preserved flow?

Can we learn from natural history of SCAD?

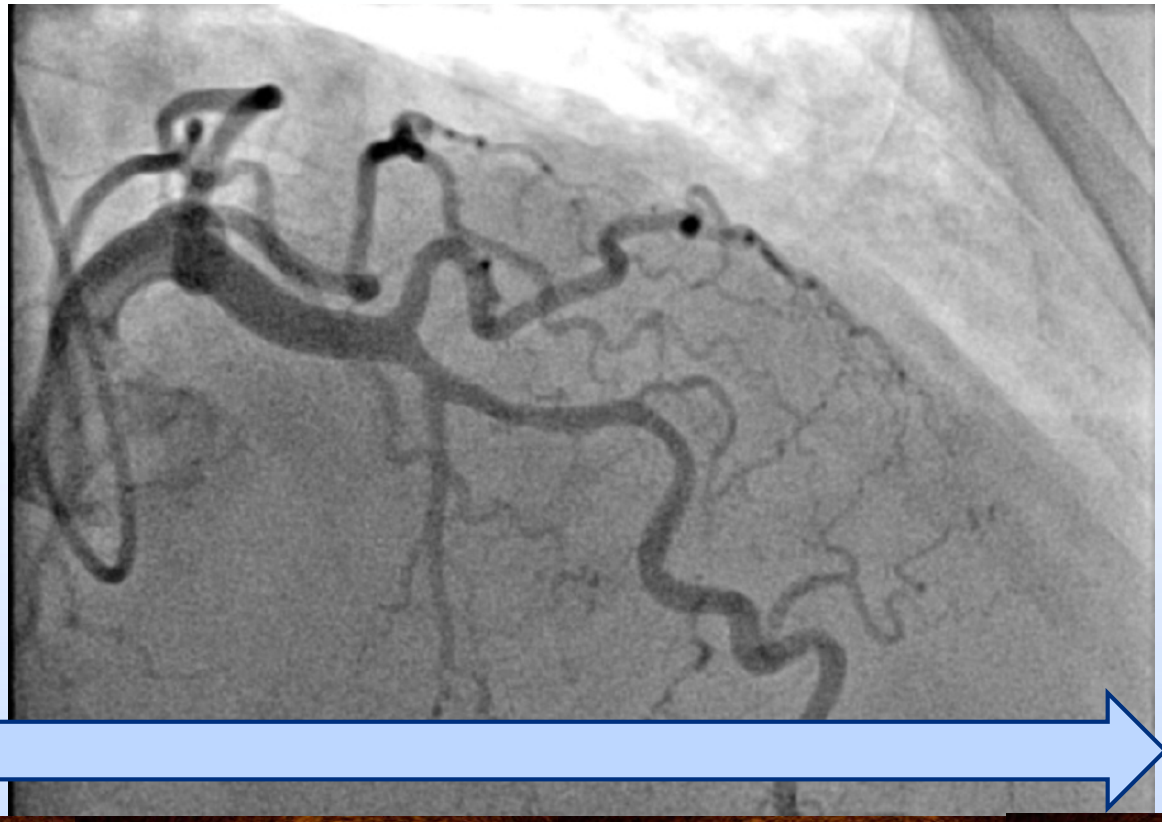
MAYO CLINIC

How to manage acute SCAD

Based on retrospective evaluation of selection-biased and survivor-biased data, plus anecdotes, last-case recall and mechanistic speculation:

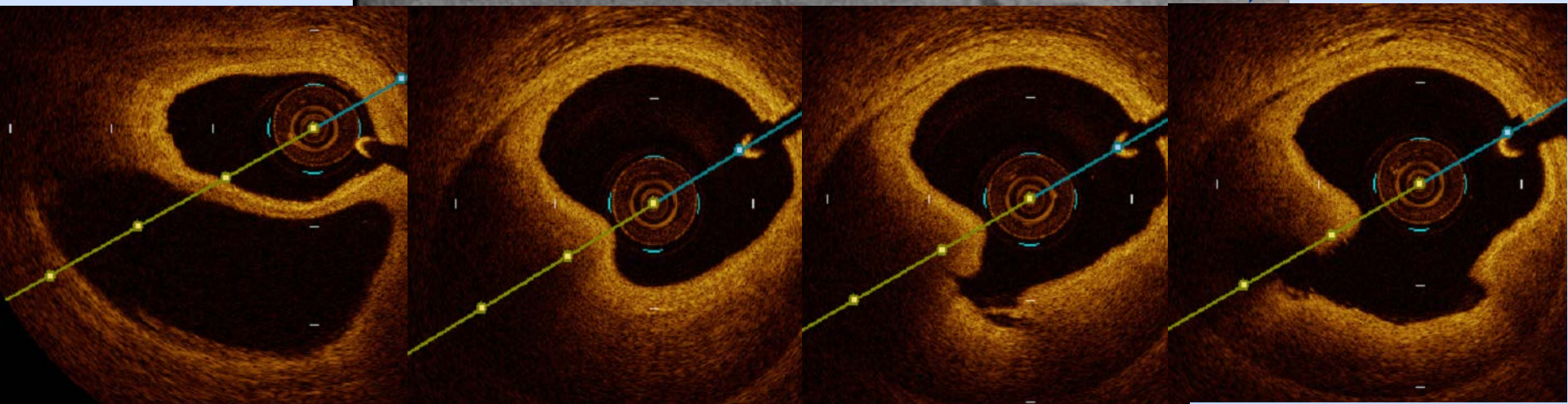
- Single antiplatelet, beta-blocker, avoid statin
- Conservative, but observe for 4-5d
- Recognize that pain often $\neq$ ischemia
- Restore flow & don't worry how it looks
- Role for decompression PCI?
- Index CABG: Good acute outcomes, but v high rate of late graft occlusion

MAYO CLINIC

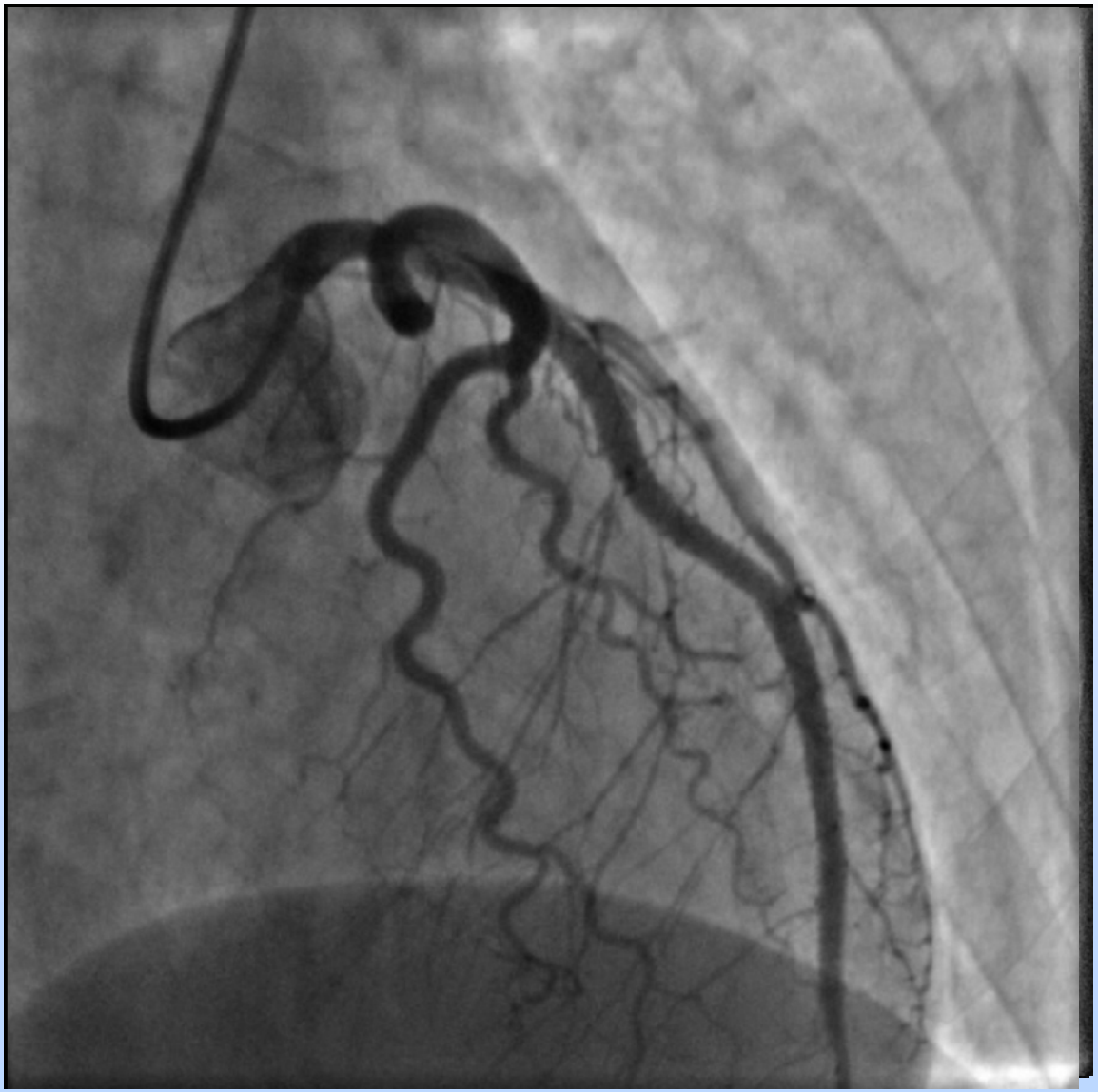


Prox

Dist

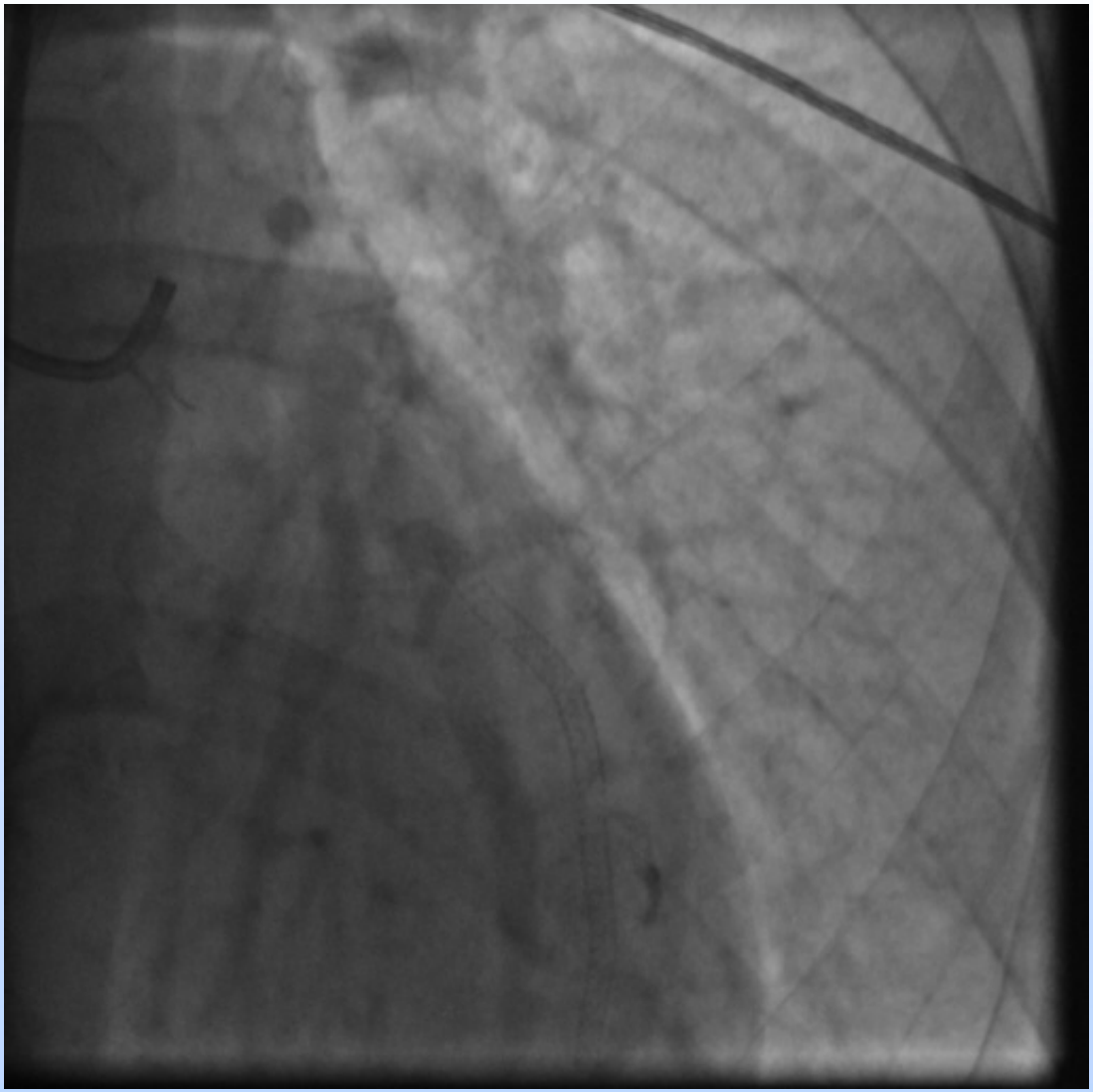


52 year old female



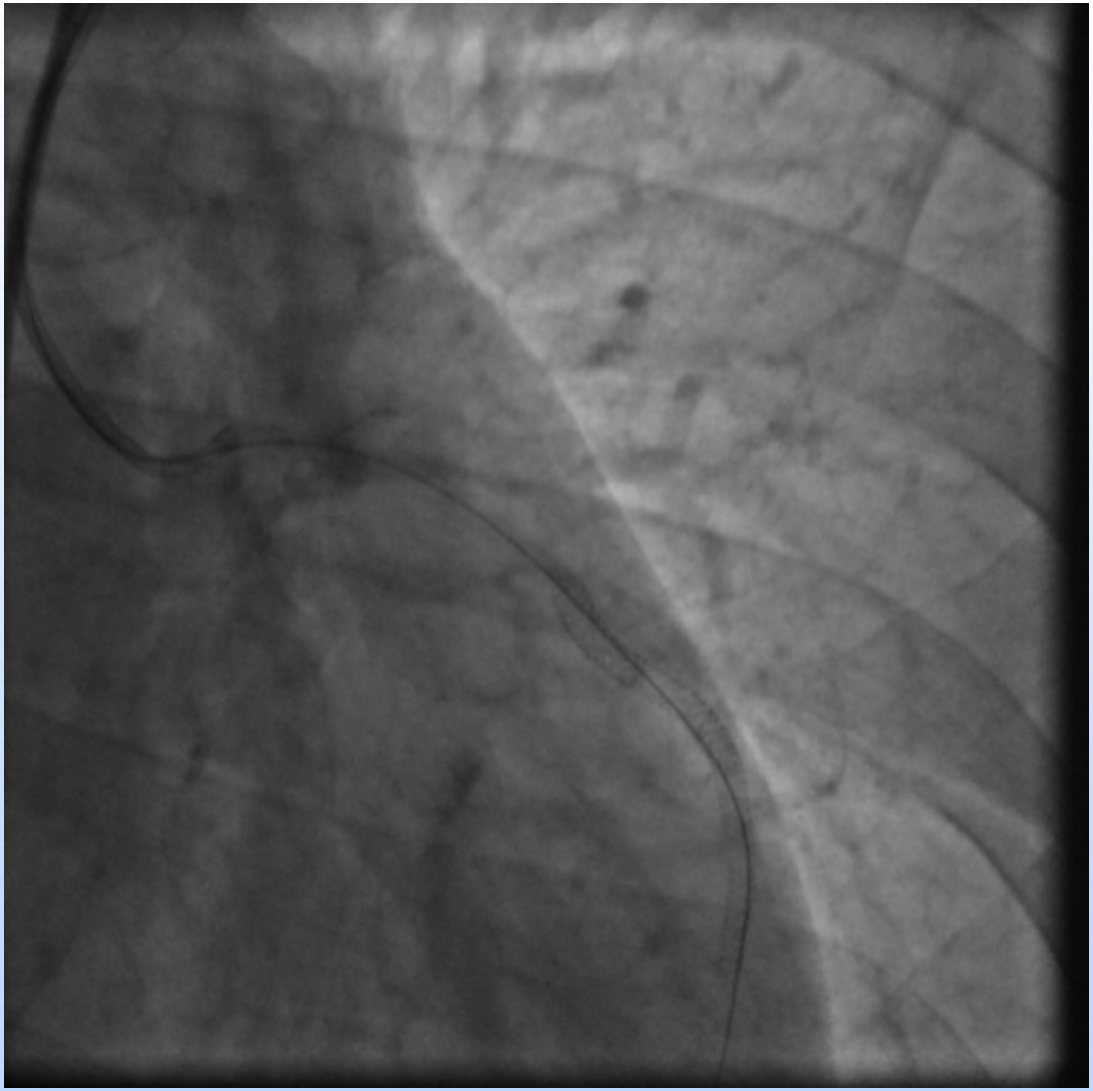
MAYO CLINIC

52 year old female



MAYO CLINIC

52 year old female



MAYO CLINIC