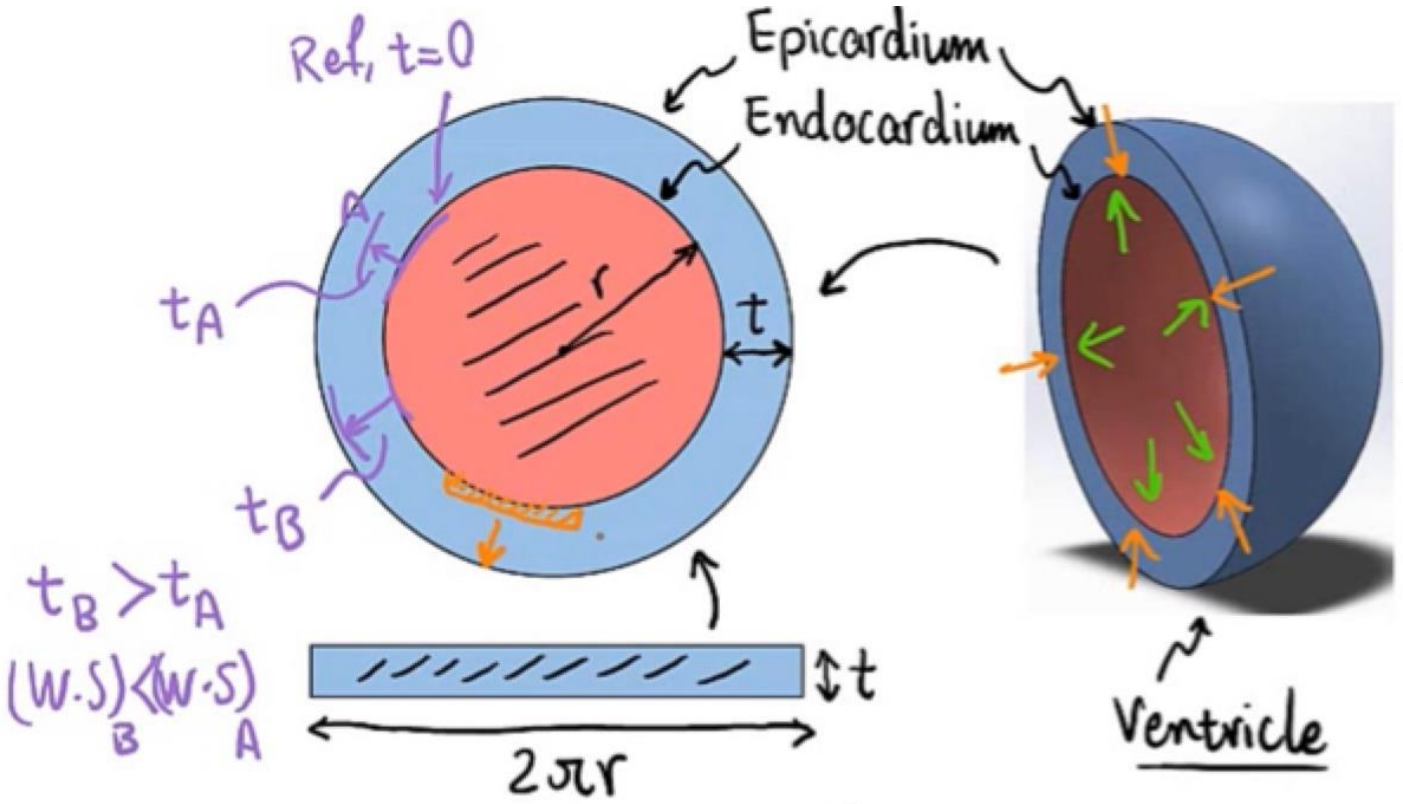


Hirmand

→ Wall Stress

→ Ischemia

$$F_B = F_W$$

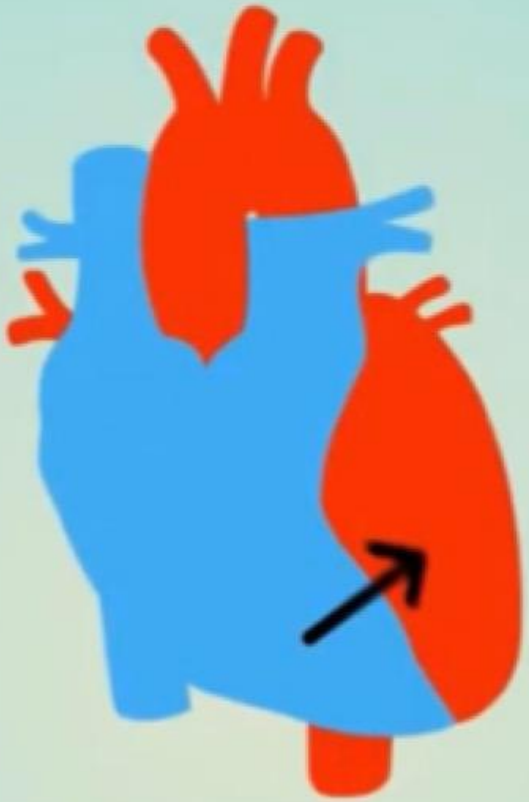
$$P \times A = (W.S)(A_W)$$

$$P \times \pi r^2 = (W.S)(2\pi r)t$$

$$P \cdot r = (W.S)(2t)$$

→ ex 1 → r↑ → A↑ → F_B↑ ⇒ W.S↑

↘ ex 2 → t↑ → A_W↑ → W.S↓



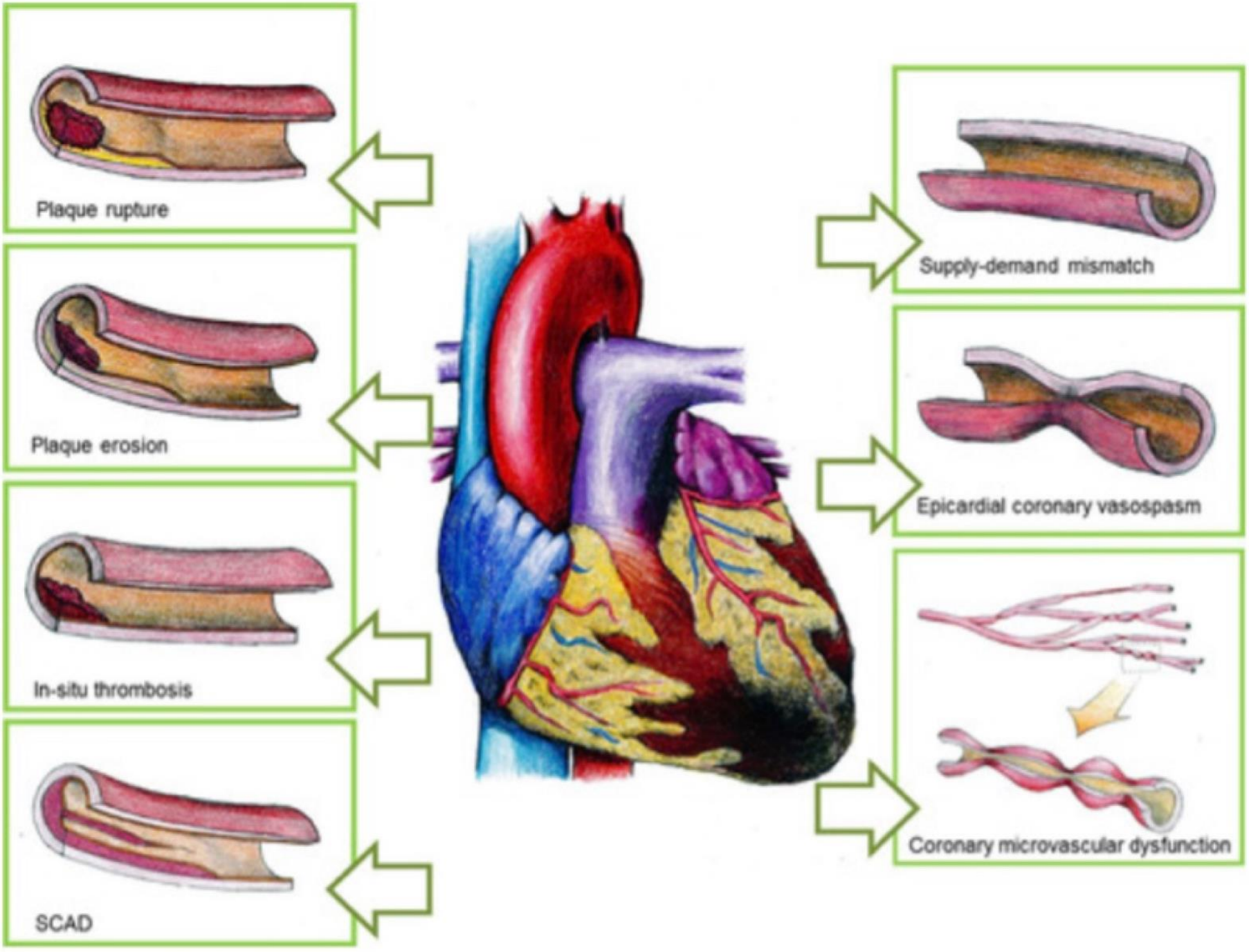
$$\text{Stress} = \frac{\text{Pressure} \times \text{Radius}}{\text{Thickness}}$$

Epidemiology

Clinical studies have reported a prevalence of MINOCA of 5% to 6% of AMI cases,[6] with a range between 5% and 15% depending on the population examined.[5,6,14–16] Although MINOCA can present with or without ST-segment elevation on the ECG, patients with MINOCA are less likely to have electrocardiographic ST-segment deviations and have smaller degrees of troponin elevation than their AMI counterparts with obstructive CAD (AMI-CAD).[14,16]

The demographic and clinical characteristics of MINOCA patients differ from other patients with AMI. MINOCA patients are usually younger[6,14–16] than patients with AMI-CAD. In a large systematic review, the average age of patients with MINOCA was 58 years, compared with 61 years among those with AMI-CAD.[6] Women are disproportionately represented among individuals with MINOCA[5,6,14–18]; they make up close to 50% of the MINOCA population but only 25% of the population with AMI-CAD.[6] Women presenting with AMI are more than twice as likely as men to have MINOCA, whereas men presenting with AMI are more likely than women to have AMI-CAD.[5,6,14,15,17,18] MINOCA is also more likely to occur in patients of black, Maori, or Pacific

Table 1. MINOCA Diagnostic Criteria
The diagnosis of MINOCA is made in patients with acute myocardial infarction that fulfills the following criteria:
1. Acute myocardial infarction (modified from the “Fourth Universal Definition of Myocardial Infarction” Criteria)
Detection of a rise or fall of cTn with at least 1 value above the 99th percentile upper reference limit
and
Corroborative clinical evidence of infarction evidenced by at least 1 of the following:
Symptoms of myocardial ischemia
New ischemic electrocardiographic changes
Development of pathological Q waves
Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischemic cause
Identification of a coronary thrombus by angiography or autopsy
2. Nonobstructive coronary arteries on angiography:
Defined as the absence of obstructive disease on angiography (ie, no coronary artery stenosis ≥50%) in any major epicardial vessel*
This includes patients with:
Normal coronary arteries (no angiographic stenosis)
Mild luminal irregularities (angiographic stenosis <30% stenoses)
Moderate coronary atherosclerotic lesions (stenoses >30% but <50%)
3. No specific alternate diagnosis for the clinical presentation:
Alternate diagnoses include but are not limited to nonischemic causes such as sepsis, pulmonary embolism, and myocarditis


Plaque rupture
Plaque erosion
In-situ thrombosis
SCAD
Supply-demand mismatch
Epicardial coronary vasospasm
Coronary microvascular dysfunction

TIPO 1	**Sindrome coronarica acuta: evento coronarico primario** • Rottura di placca/erosione/ulcerazione; dissezione coronarica
TIPO 2	**Infarto secondario a squilibrio fra richiesta e offerta di O_2** • Anemia; ipossiemia; aritmia; IPT; ipotensione; cocaina; spasmo coronarico; disfunzione endoteliale; embolia coronarica
TIPO 3	**Arresto cardiaco/Morte improvvisa** • Sintomi suggestivi di ischemia + • Δ Alterazioni ECG compatibili con ischemia o BBS *de novo* + • Nessun dosaggio per ricerca di biomarcatori
TIPO 4a	**Infarto secondario a PCI** • Sintomi suggestivi di ischemia o alterazioni ischemiche all'ECG o reperti angiografici compatibili con complicanza procedurale o imaging compatibile con perdita di miocardio vitale o RWMA *de novo* + • Biomarcatori positivi a) ↗ cTn **≥ 5 x limite superiore della norma** (> 99° percentile) con cTn basale normale (< 99° percentile) o b) ↗ cTn **> 20%** con valore basale elevato (> 99° percentile) ma stabile +
TIPO 4b	**Infarto secondario a trombosi da stent**
	Infarto secondario a CABG • ↗ cTn **≥ 10 x limite superiore della norma** (> 99° percentile) con cTn basale normale (< 99° percentile) +
IPO 5	• Onde Q *de novo* o BBS *de novo* o nuova occlusione coronarica (o CABG) all'angiografia coronarica o imaging compatibile con perdita di miocardio vitale o RWMA *de novo*

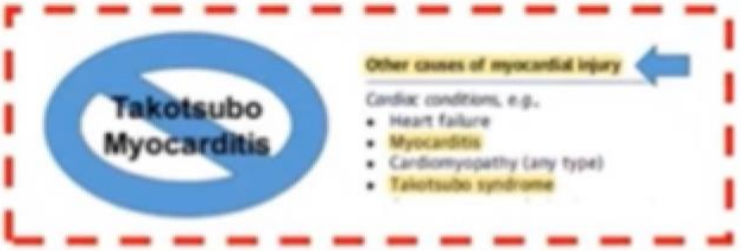
Etiologies

Coronary

- Type 1 MI (atherothrombotic)
  - Plaque rupture/erosion
- 'Coronary' Type 2 MI (non-atherothrombotic)
  - Vasospasm
  - SCAD
  - Coronary microvascular dysfunction
  - In-situ thrombosis
  - Coronary thromboembolism

Non-coronary

- 'Non-coronary' Type 2 MI
  - Supply-demand mismatch secondary to alternative etiology/trigger.
    - Anemia
    - Hypo/hyper-tension
    - Respiratory failure
    - Tachy/brady-dysrhythmias



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Sandoval. Algorithm for the evaluation of MINOCA.

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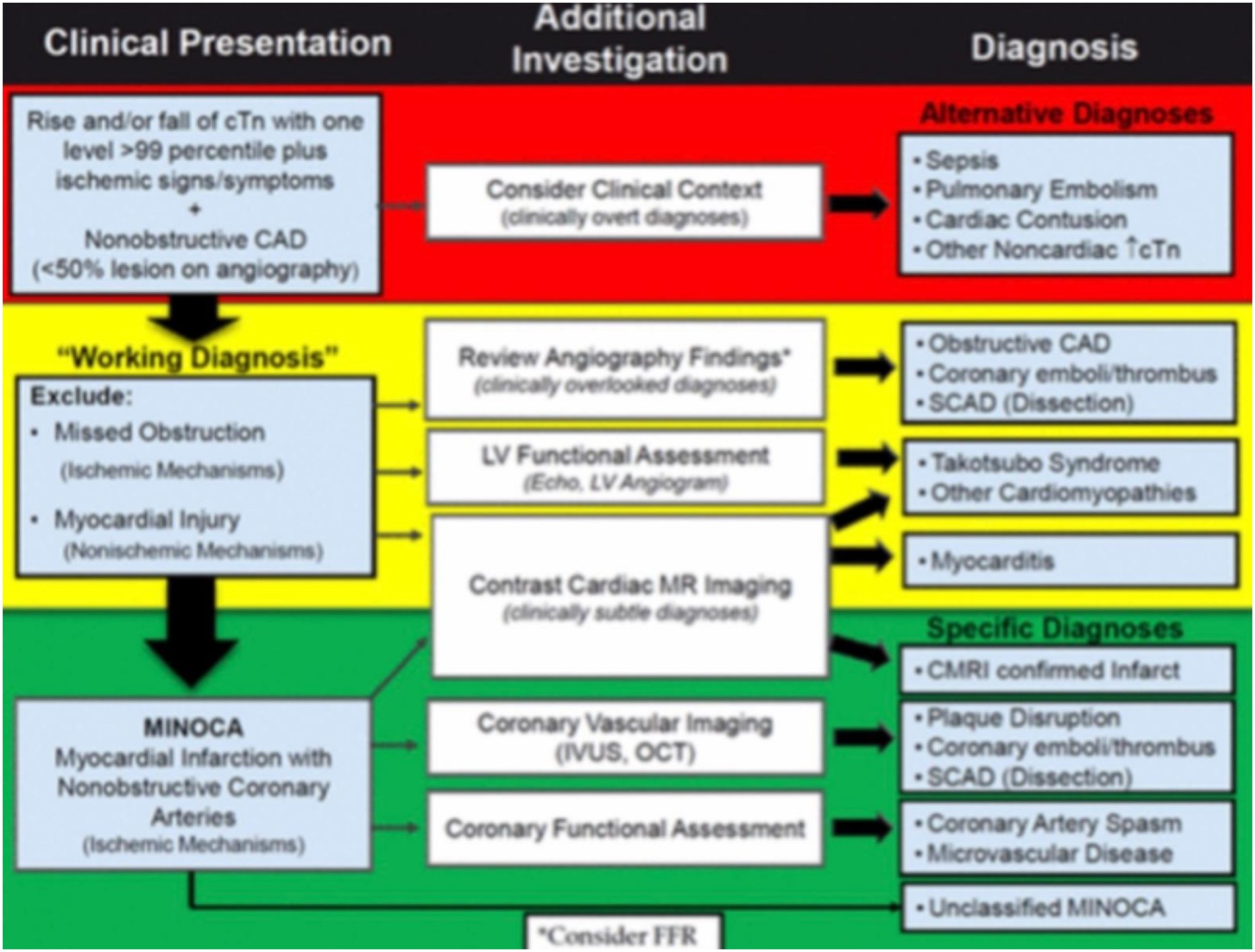
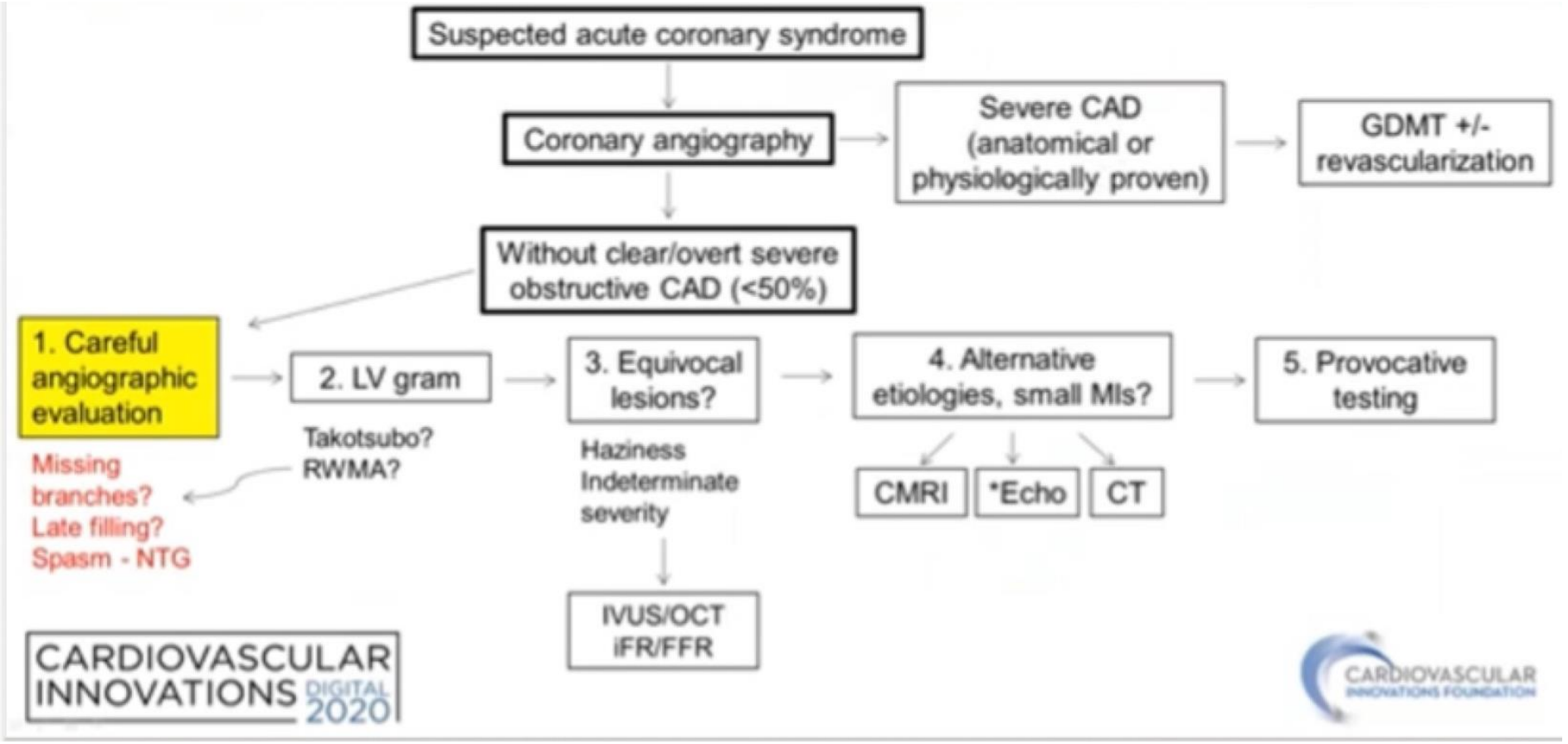
Algorithm for evaluation of the MINOCA patient - Yader Sandoval, MD


Clinical Presentation
Additional Investigation
Diagnosis
Rise and/or fall of cTn with one level >99 percentile plus ischemic signs/symptoms
+
Nonobstructive CAD
(<50% lesion on angiography)
Consider Clinical Context
(clinically overt diagnoses)
Alternative Diagnoses
• Sepsis
• Pulmonary Embolism
• Cardiac Contusion
• Other Noncardiac ↑cTn
"Working Diagnosis"
Exclude:
• Missed Obstruction
(Ischemic Mechanisms)
• Myocardial Injury
(Nonischemic Mechanisms)
Review Angiography Findings*
(clinically overlooked diagnoses)
• Obstructive CAD
• Coronary emboli/thrombus
• SCAD (Dissection)
LV Functional Assessment
(Echo, LV Angiogram)
• Takotsubo Syndrome
• Other Cardiomyopathies
Contrast Cardiac MR Imaging
(clinically subtle diagnoses)
• Myocarditis
Specific Diagnoses
• CMRI confirmed Infarct
MINOCA
Myocardial Infarction with Nonobstructive Coronary Arteries
(Ischemic Mechanisms)
Coronary Vascular Imaging
(IVUS, OCT)
• Plaque Disruption
• Coronary emboli/thrombus
• SCAD (Dissection)
Coronary Functional Assessment
• Coronary Artery Spasm
• Microvascular Disease
• Unclassified MINOCA
*Consider FFR


Suspected acute coronary syndrome
Coronary angiography
Severe CAD (anatomical or physiologically proven)
GDMT +/- revascularization
Without clear/overt severe obstructive CAD (<50%)
1. Careful angiographic evaluation
Missing branches?
Late filling?
Spasm - NTG
2. LV gram
Takotsubo?
RWMA?
3. Equivocal lesions?
Haziness
Indeterminate severity
IVUS/OCT
iFR/FFR
4. Alternative etiologies, small MIs?
CMRI
*Echo
CT
5. Provocative testing
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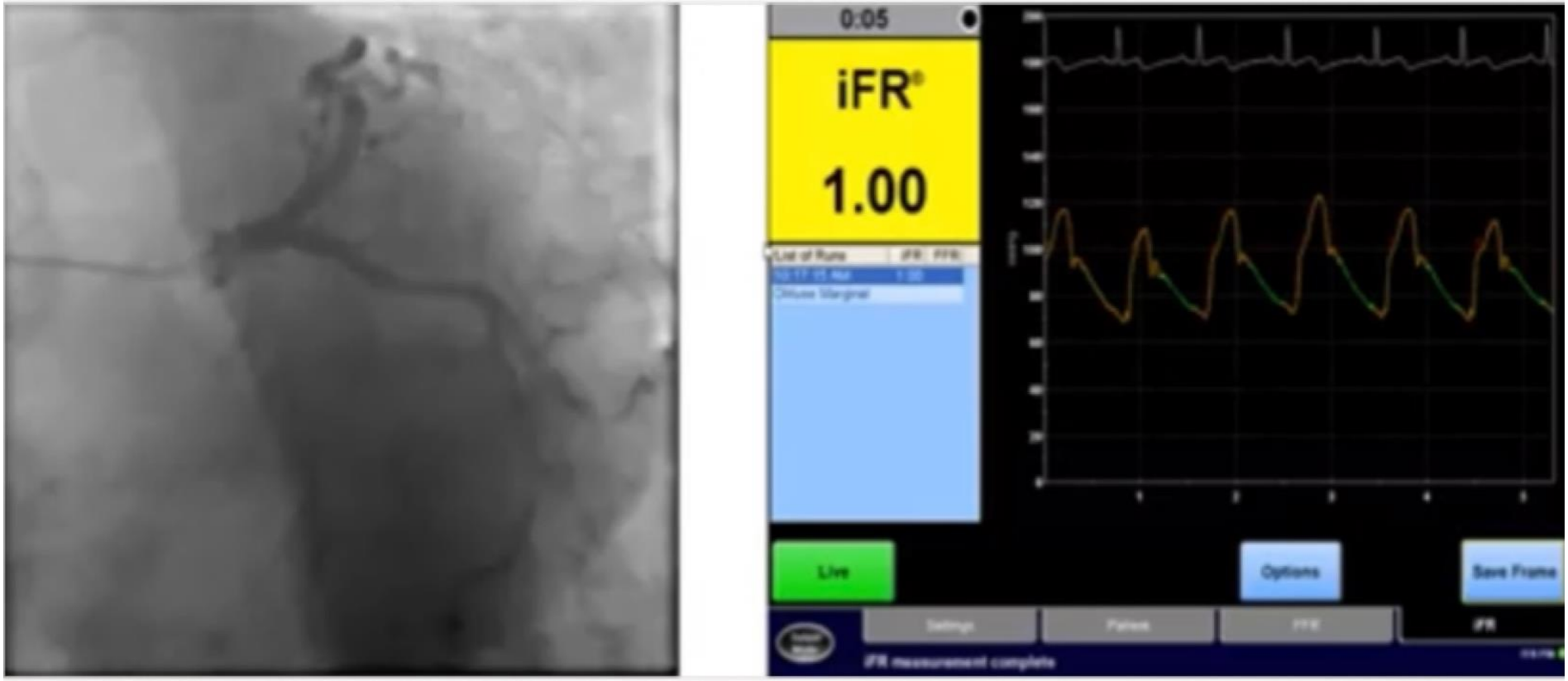
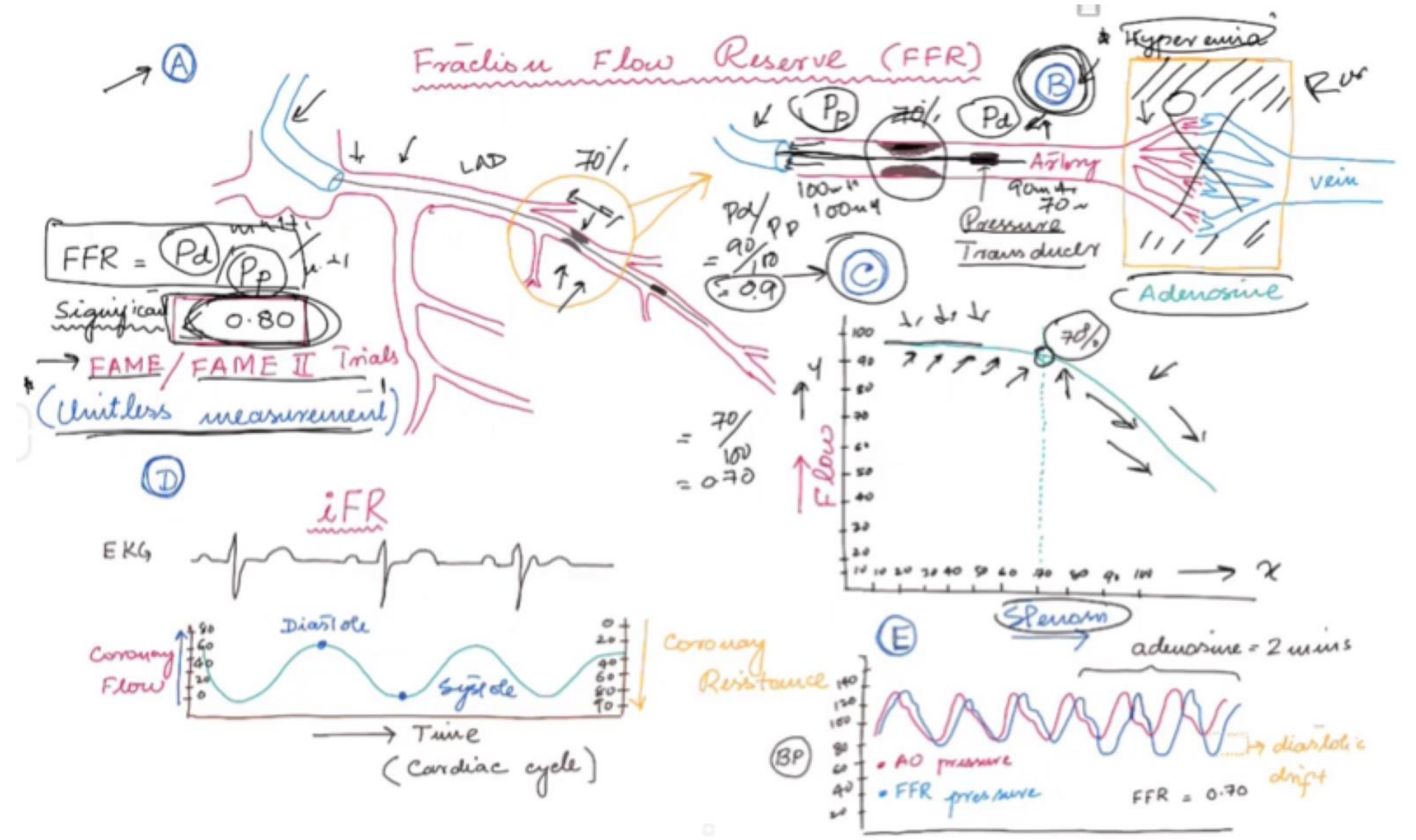
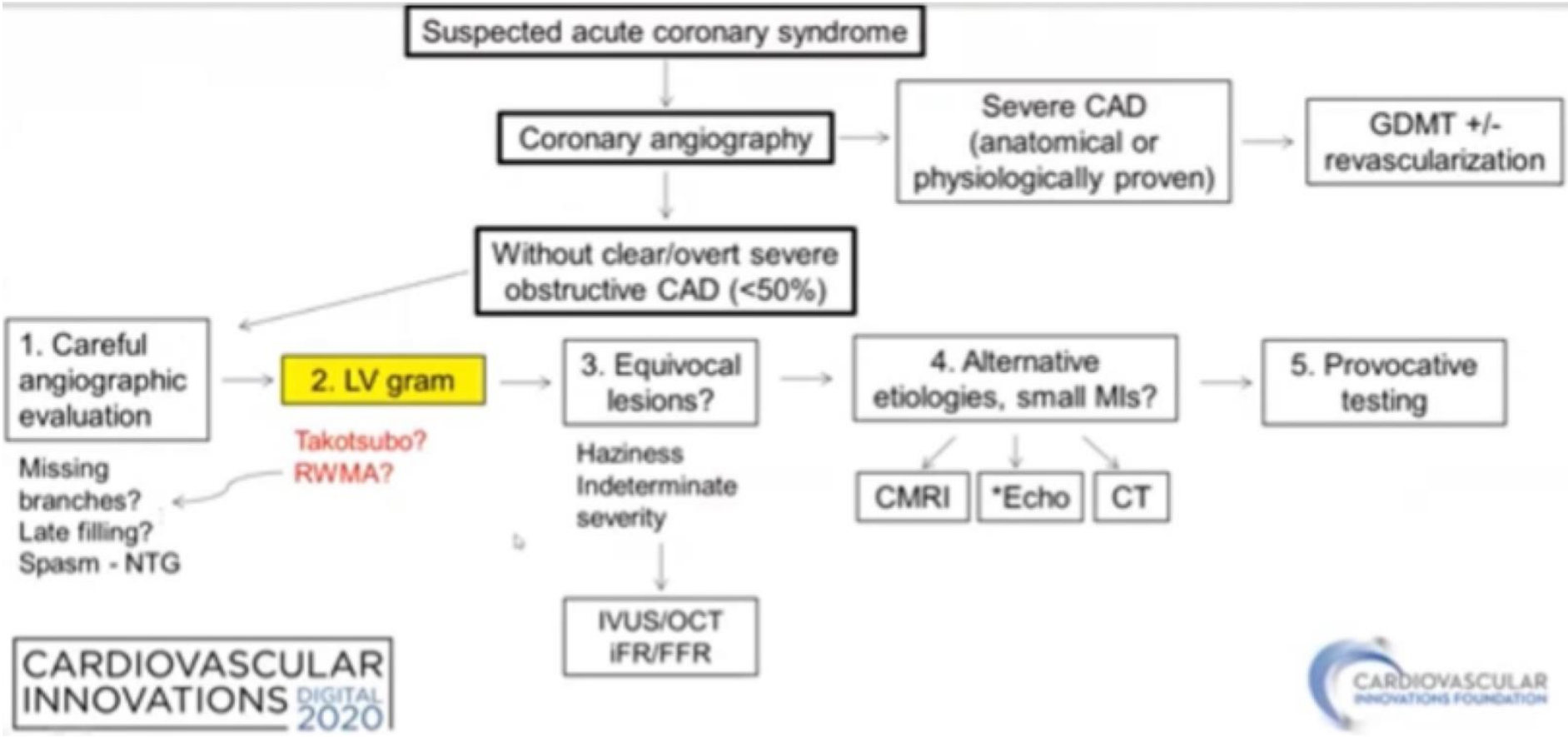

0:05
iFR®
1.00
List of Runs
iFR
FFR
Live
Options
Save Frame
Settings
Patient
FFR
iFR
iFR measurement complete


Fractional Flow Reserve (FFR)
A
LAD
70%
FFR = Pd / Pp
Significant
0.80
FAME / FAME II Trials
(Unitless measurement)
B
Pp
Pd
70%
Hyperemia
RCA
Artery
vein
100 mmHg
90 mmHg
70 mmHg
Pressure Transducer
Pd/Pp = 90/10 = 0.9
= 70/100 = 0.70
C
Adenosine
70%
Flow
Spearman
D
iFR
EKG
Diastole
Systole
Coronary Flow
Coronary Resistance
Time
(Cardiac cycle)
E
adenosine = 2 mins
BP
AO pressure
FFR pressure
FFR = 0.70
diastolic drift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5. Provocative testing
Takotsubo?
RWMA?
Missing branches?
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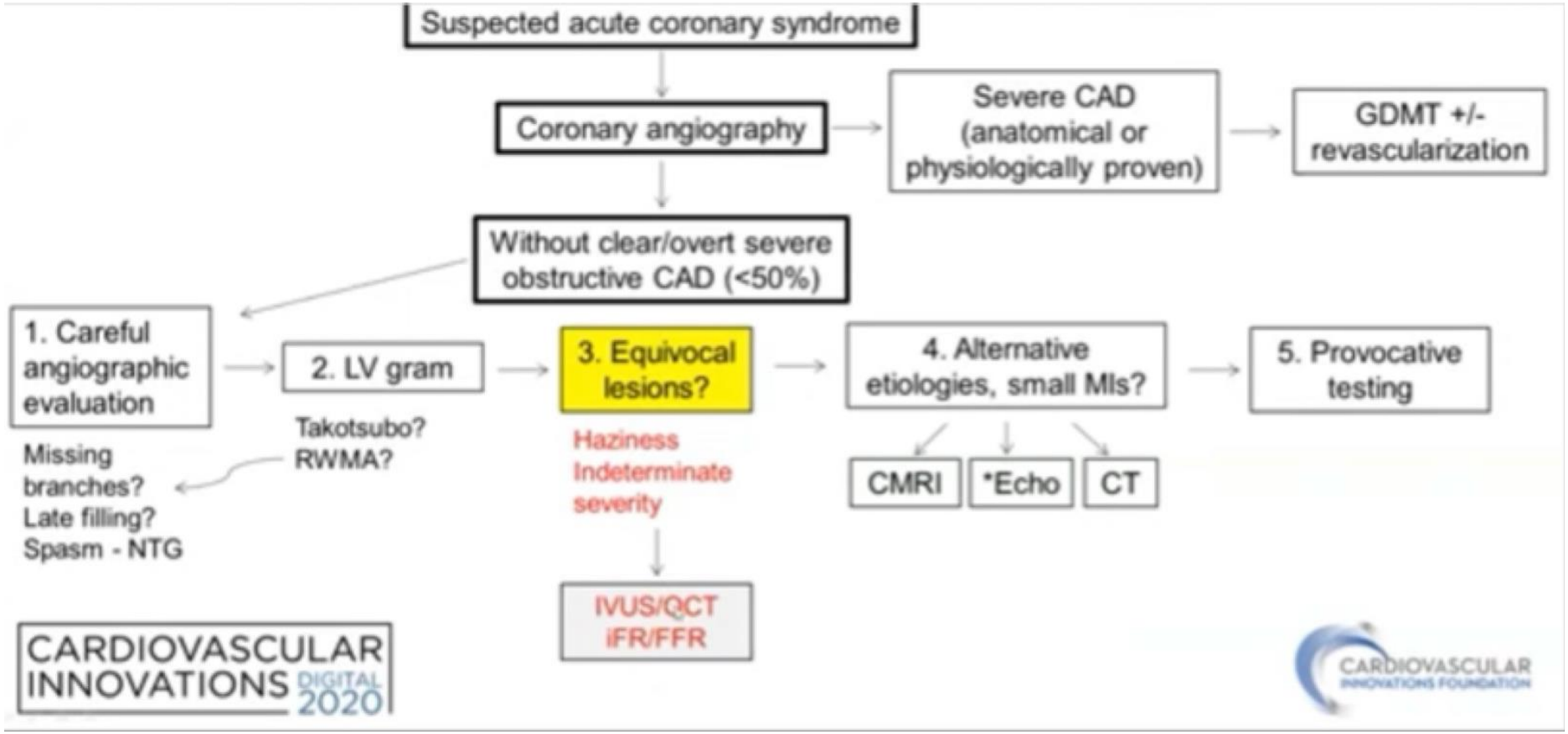
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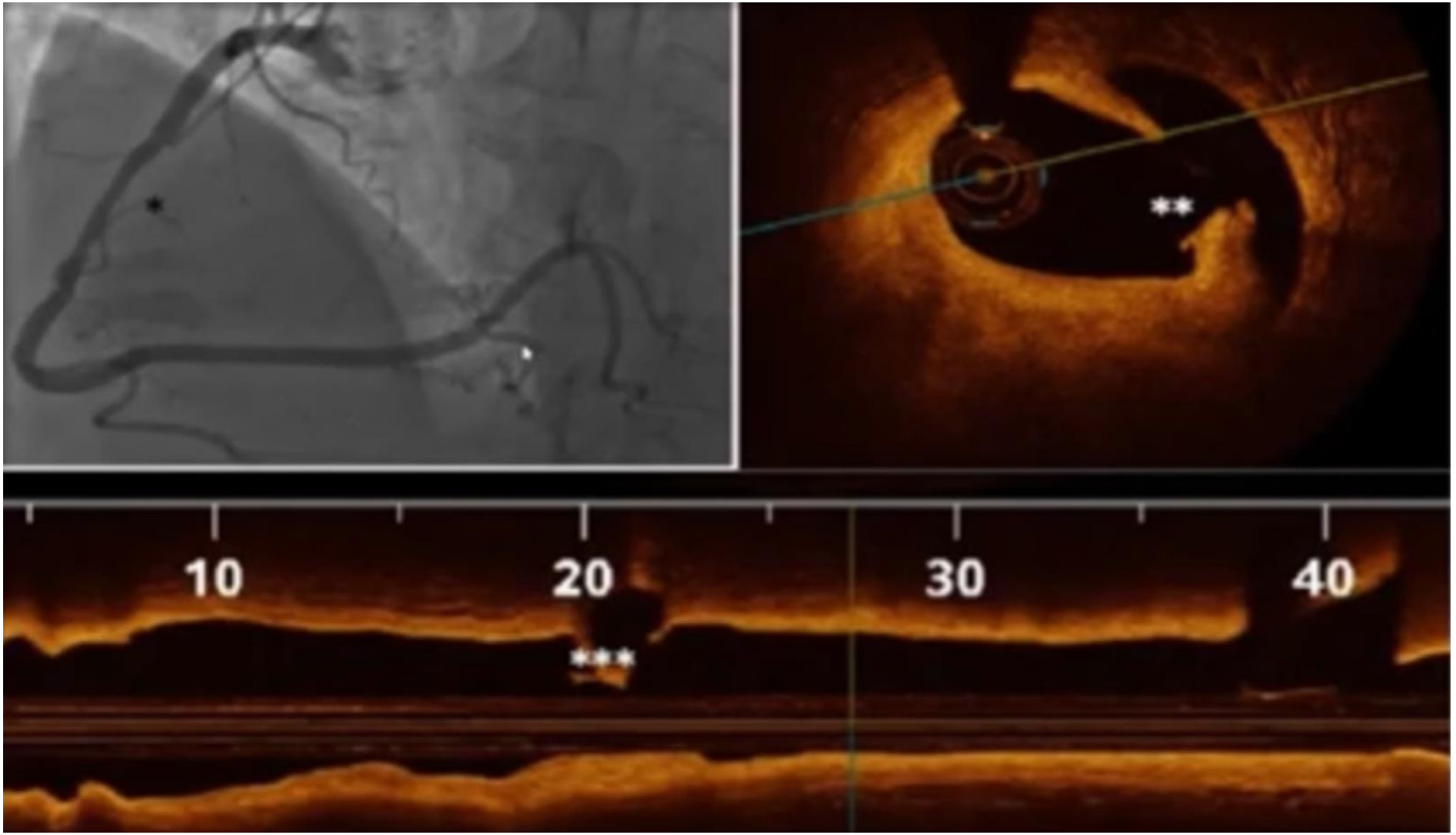
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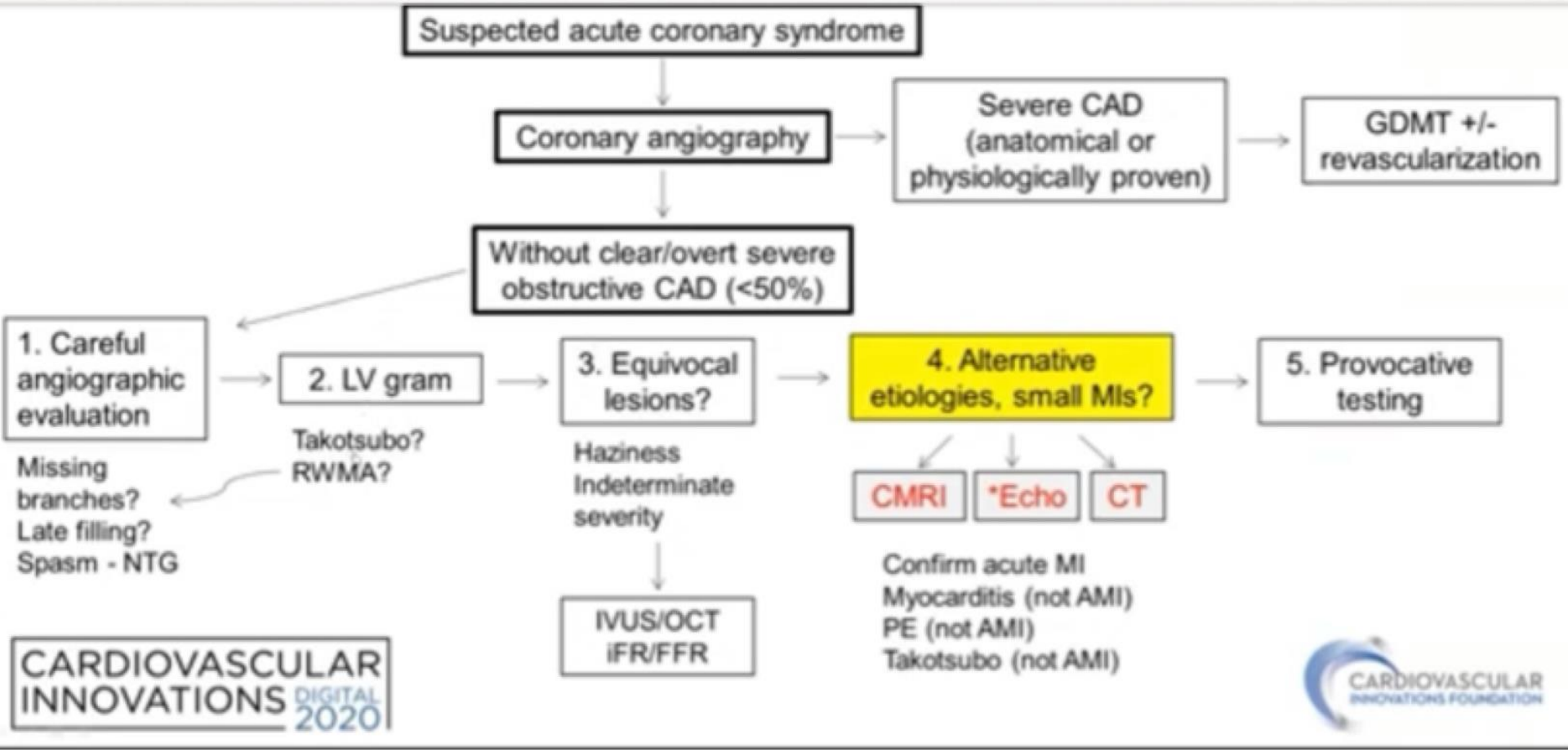
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iFR/FFR
4. Alternative etiologies, small MIs?
CMRI
*Echo
CT
Confirm acute MI
Myocarditis (not AMI)
PE (not AMI)
Takotsubo (not AMI)
5. Provocative testing
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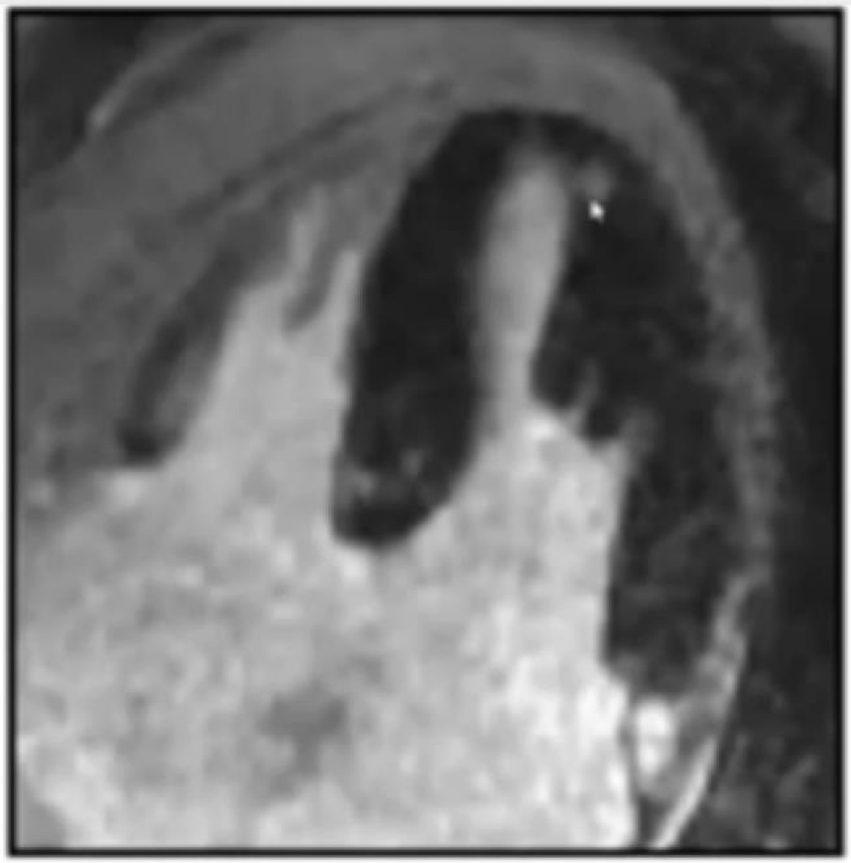
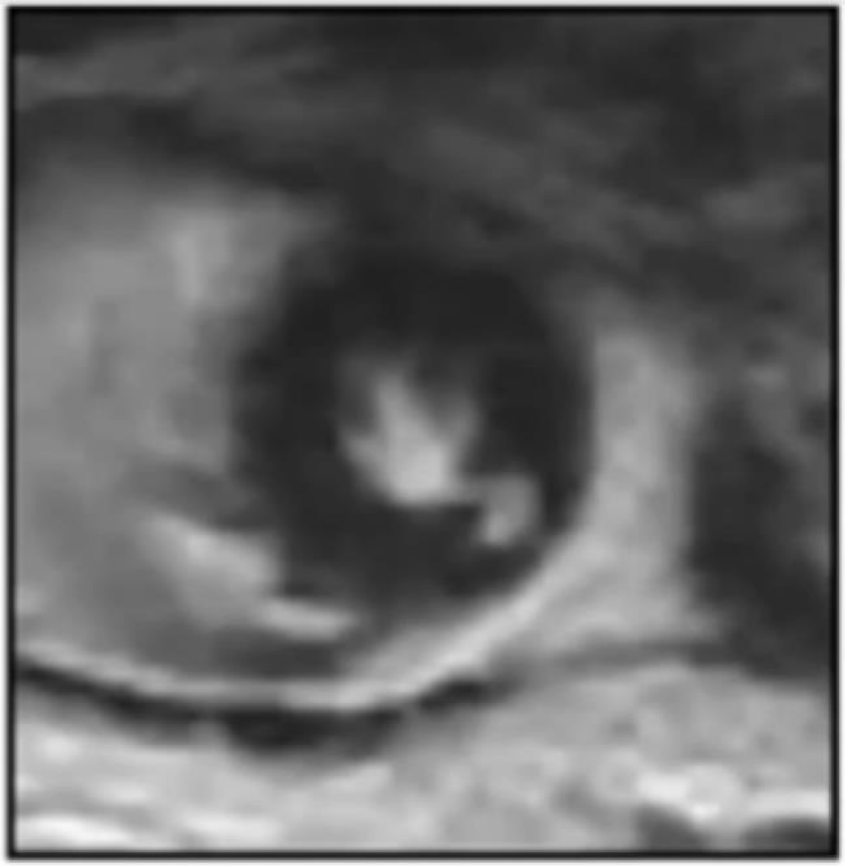
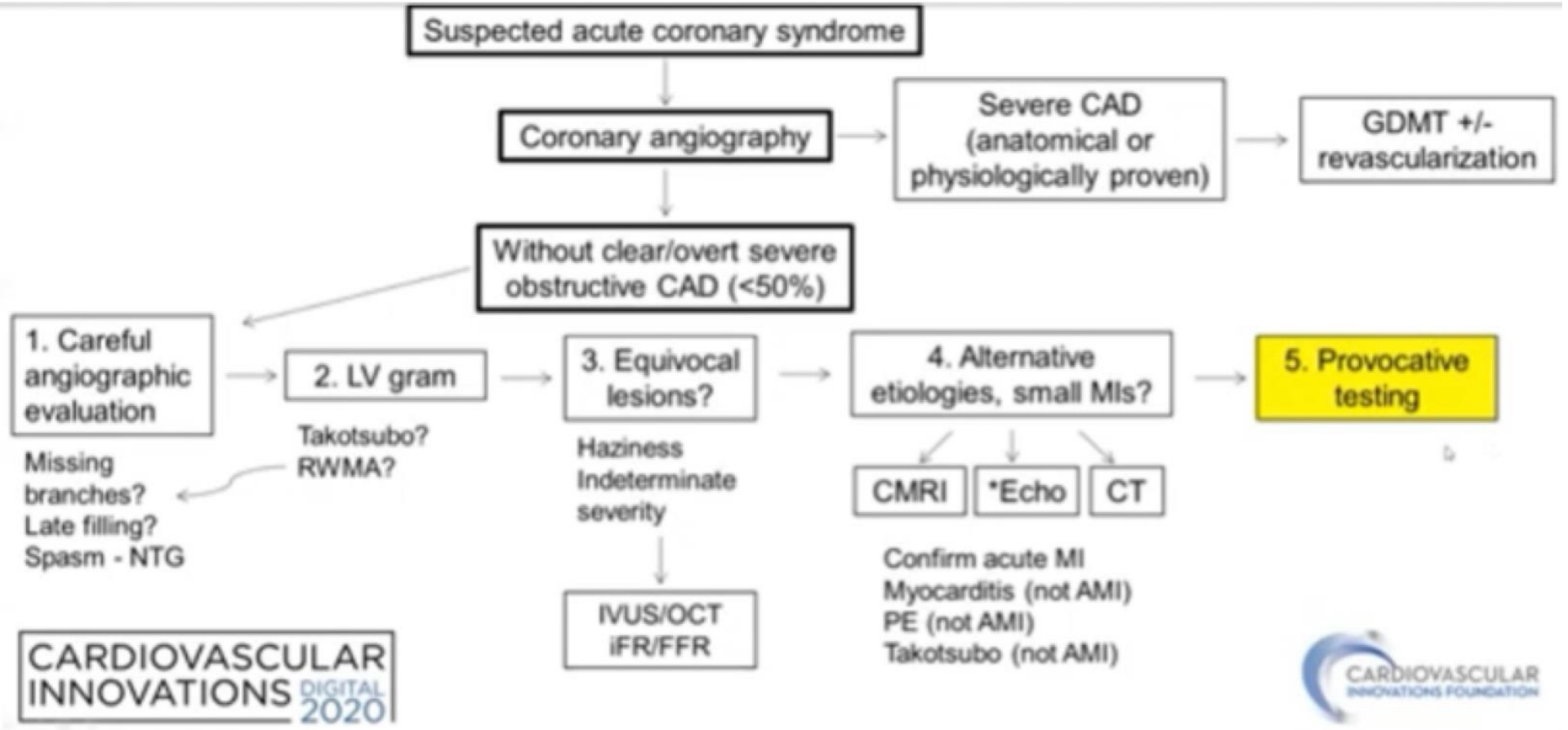




Very small focus of subendocardial delayed enhancement in the inferolateral apex could represent a small embolic infarct. Preserved left ventricular systolic function without focal wall motion abnormalities.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Takotsubo (not AMI)
5. Provocative testing
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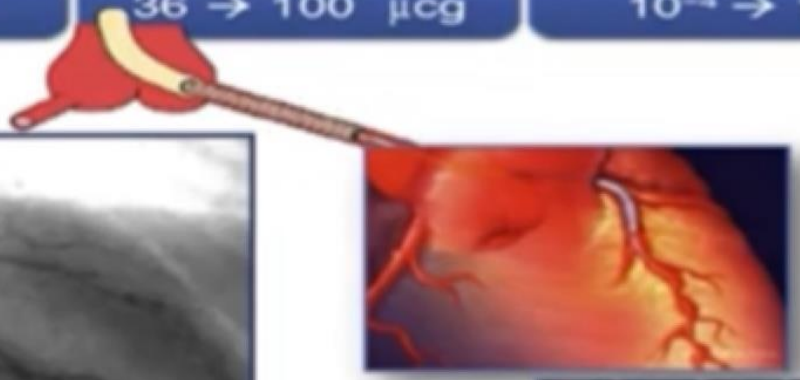
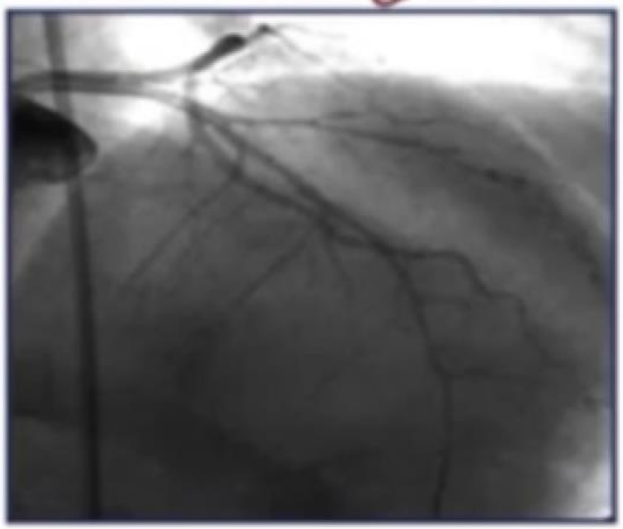
Functional angiogram protocol

Diagnostic angiography

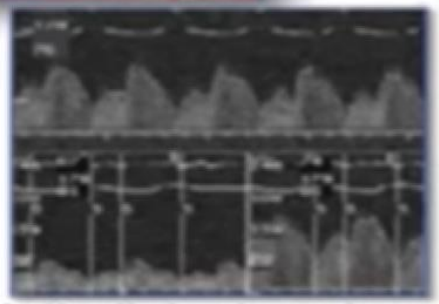
Adenosine IC 36 → 100 µcg

Acetylcholine IC 10^{-4} → 10^{-6}



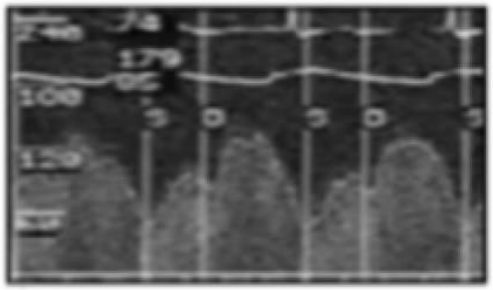


Post-Ach vasospasm

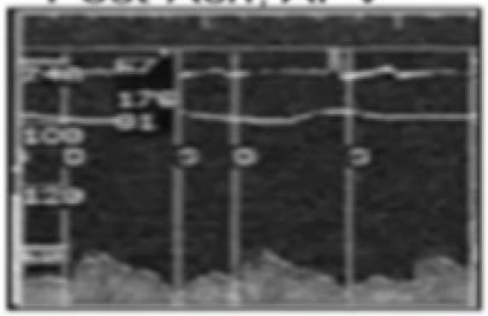


CFRne: Nonendothelial microcirc

CFRe: Microcirc, pre-ACh, APV



Post-Ach, APV



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1. In patients with ischemia and/or infarction, the absence of obstructive CAD should not lead to dismissal, but rather to interrogation of potential causes of myocardial injury, in particular if it will lead to actionable measures that modify outcomes.
2. Intracoronary imaging, IVUS or OCT, can facilitate diagnosis.
3. For those with uncertain diagnosis following comprehensive evaluation, can consider functional angiogram protocol for microvascular dysfunction.
4. Cardiac MRI can help establish diagnosis, including various other non-MINOCA causes such as myocarditis.

Table 2. Management of Patients With a Working Diagnosis of MINOCA

Underlying Mechanism/Clinical Disorder	Selective Diagnostic Investigations*	Selective/Empirical Therapies†
Clinically overlooked ischemic or nonischemic presentations (mimicking MINOCA)		
Branch "flush occlusion" or severe branch stenosis (from coronary embolism/thrombus or ruptured plaque)	Angiographic review Consider intracoronary imaging to identify plaque rupture or dissection, or de novo thrombus echocardiography review (screen valves for endocarditis; left atrium and left ventricle for thrombus source and tumor; the possibility of a PFO should also be evaluated)	Antiplatelet or anticoagulant (depending on cause) Statin β-blockers ACE inhibitors/ARBs (in presence of left ventricular dysfunction, and possibly with preserved EF)

Spontaneous coronary artery dissection	Angiographic review	Aspirin β-blocker Consider clopidogrel
Takotsubo syndrome	Left ventricular angiogram Contrast CMRI	ACE inhibition Medical or device therapies for heart failure/left ventricular dysfunction Consider β-blockers
Cardiomyopathies	Contrast CMRI	Medical or device therapies for heart failure/left ventricular dysfunction
Myocarditis	Contrast CMRI	Medical or device therapies for heart failure/left ventricular dysfunction. Consider immunomodulatory and immunosuppressive therapies

Ischemic presentation (MINOCA)		
Plaque disruption	Angiographic review Intravascular imaging (IVUS or OCT)	Aspirin High-intensity statin β-blockers (in presence of left ventricular dysfunction, and possibly with preserved EF) ACE inhibitors/ARBs (in presence of left ventricular dysfunction, and possibly with preserved EF) Consider clopidogrel/ticagrelor

Coronary artery spasm	Resolution with coronary vasodilators (eg, intracoronary nitroglycerin) Provocative spasm testing Blood toxicology testing Review of medication and nonprescription drug use (eg, migraine medications, cocaine)	Calcium channel blockers Other antispastic agents (nitrates, nicorandil, cilostazol) Consider statin
Coronary microvascular dysfunction	Angiographic review Coronary microvascular functional testing	Conventional antianginal therapies (eg, calcium channel blocker, β-blocker) Unconventional antianginal therapies (eg, L-arginine, ranolazine, dipyridamole, aminophylline, imipramine, α-blockers)

Coronary embolism/thrombus	Angiographic review Intravascular imaging (IVUS or OCT) Thrombophilia screen	Antiplatelet or anticoagulant therapy Other targeted therapies for hypercoagulable condition
Spontaneous coronary artery dissection	Angiographic review Intravascular imaging (IVUS or OCT)	Aspirin β-blocker Consider clopidogrel
Supply-demand mismatch	Review history for potential stressors	Treatment for underlying condition

Prognosis

The prognosis of patients presenting with MINOCA depends on the underlying cause and is currently under active investigation. Most studies have shown that MINOCA patients have better outcomes than their AMI-CAD counterparts.[5,6,10] However, this finding is not consistent among all reports. In the VIRGO study (Variation in Recovery: Role of Gender on Outcomes of Young AMI Patients),[16] patients with MINOCA had similar 1-month and 1-year mortality rates and comparable quality-of-life measures as patients with AMI-CAD. The Korean Infarct Registry showed that