COMPLEX CARDIOVASCULAR CATHETER THERAPEUTICS

Advanced Endovascular and Coronary Intervention Global Summit

JUNE 23-26, 2019

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INTERVENTIONAL ACADEMY

COMPLEX CARDIOVASCULAR CATHETER THERAPEUTICS

Advanced Endovascular and Coronary Intervention Global Summit

CELEBRATING 15 YEARS

PCI without Opening the Vessel: Acute Treatment of MINOCA

"The Essential is Invisible to the Eye…"
Antoine de Saint-Exupery

Gustavo Samaja, MD, FSCAI.

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CACI
COLEGIO ARGENTINO DE CARDIOANGIÓLOGOS INTERVENCIONISTAS

Nothing to disclose related to this presentation

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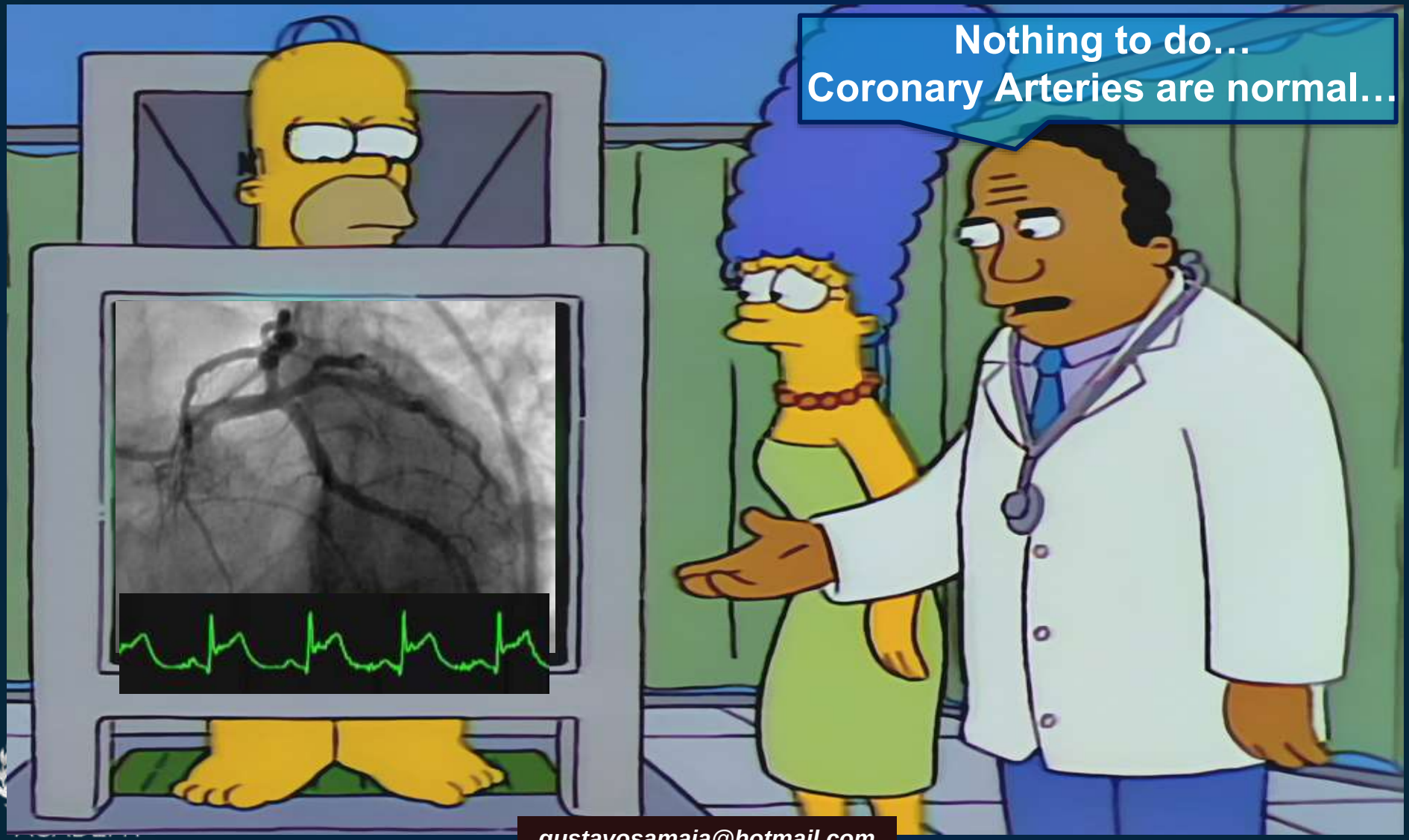
Myocardial Infarction with No Obstructive Coronary Arteries

"The Essential is Invisible to the Eye…"



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MINOCA

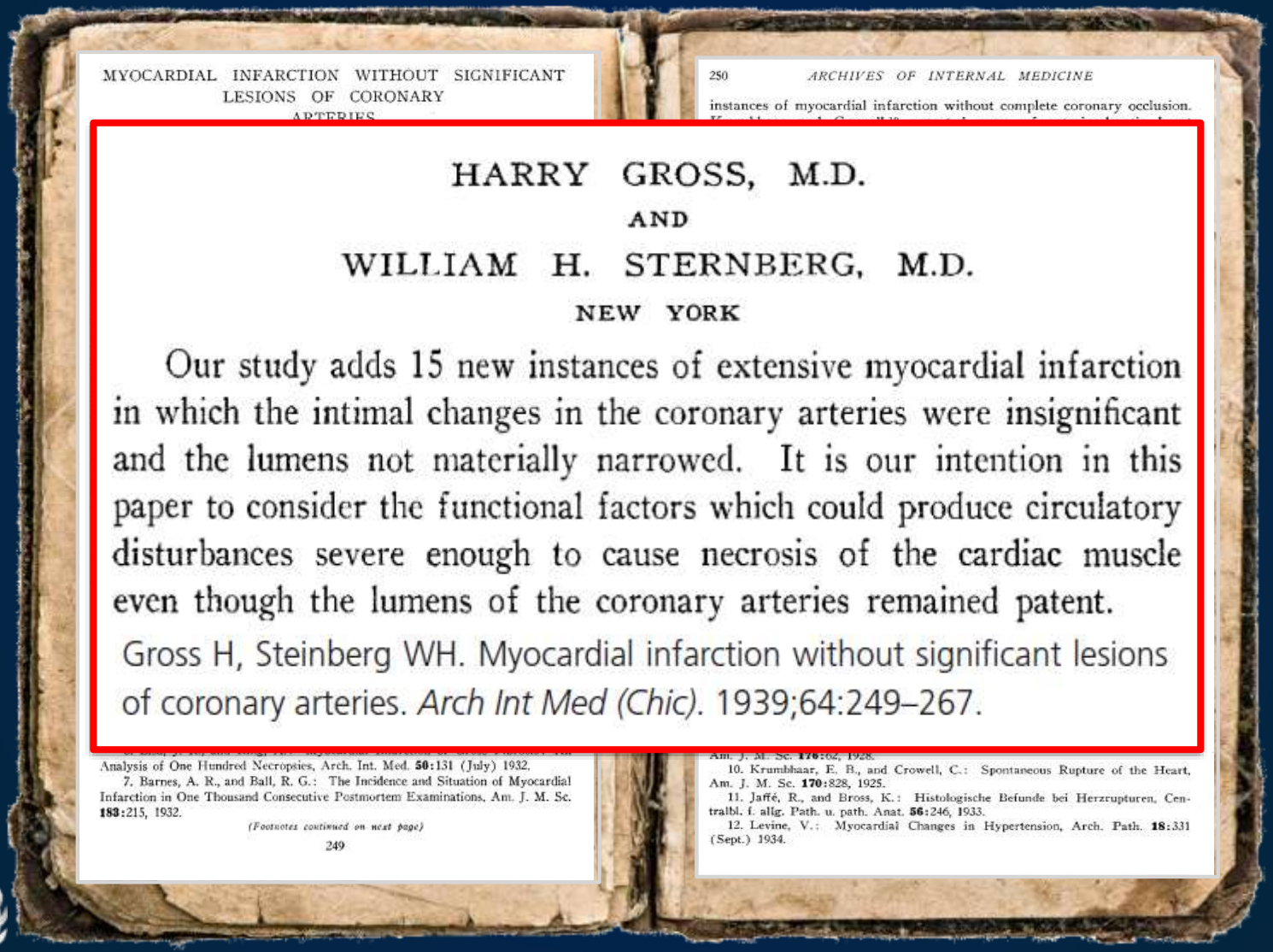
"Myocardial Infarction with No Obstructive Coronary Disease"

MINOCA is a broadly labeled term: a variety of causes can result in this clinical condition.

We are treating an AMI, despite the absence of obstructive CAD.

MINOCA patients seems to have better outcomes than their AMI-CAD counterparts, but there are controversial data about this issue…

MINOCA
Nothing new under the sun:
Coined in 2013, It was described back in the 30's…



MYOCARDIAL INFARCTION WITHOUT SIGNIFICANT LESIONS OF CORONARY ARTERIES

250 ARCHIVES OF INTERNAL MEDICINE

instances of myocardial infarction without complete coronary occlusion.

HARRY GROSS, M.D.

AND

WILLIAM H. STERNBERG, M.D.

NEW YORK

Our study adds 15 new instances of extensive myocardial infarction in which the intimal changes in the coronary arteries were insignificant and the lumens not materially narrowed. It is our intention in this paper to consider the functional factors which could produce circulatory disturbances severe enough to cause necrosis of the cardiac muscle even though the lumens of the coronary arteries remained patent.

Gross H, Steinberg WH. Myocardial infarction without significant lesions of coronary arteries. *Arch Int Med (Chic)*. 1939;64:249–267.

Analysis of One Hundred Necropsies, Arch. Int. Med. **50**:131 (July) 1932.

7. Barnes, A. R., and Ball, R. G.: The Incidence and Situation of Myocardial Infarction in One Thousand Consecutive Postmortem Examinations, Am. J. M. Sc. **183**:215, 1932.

(Footnotes continued on next page)

249

10. Krumbhaar, E. B., and Crowell, C.: Spontaneous Rupture of the Heart, Am. J. M. Sc. **170**:828, 1925.

11. Jaffé, R., and Bross, K.: Histologische Befunde bei Herzrupturen, Centralbl. f. allg. Path. u. path. Anat. **56**:246, 1933.

12. Levine, V.: Myocardial Changes in Hypertension, Arch. Path. **18**:331 (Sept.) 1934.

MINOCA Diagnostic Criteria

AHA SCIENTIFIC STATEMENT

Contemporary Diagnosis and Management of Patients With Myocardial Infarction in the Absence of Obstructive Coronary Artery Disease

A Scientific Statement From the American Heart Association

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

Criteria to be fulfilled:

- ***AMI:*** *"Fourth Universal Definition of Myocardial Infarction Criteria*

- ***Nonobstructive Coronary Arteries on Angio:*** *<50% stenosis in major epicardial vessels.*

- ***No specific Alternate Diagnosis for the Clinical Presentation***

Circulation. 2019;139:e891–e908. DOI: 10.1161/CIR.0000000000000670

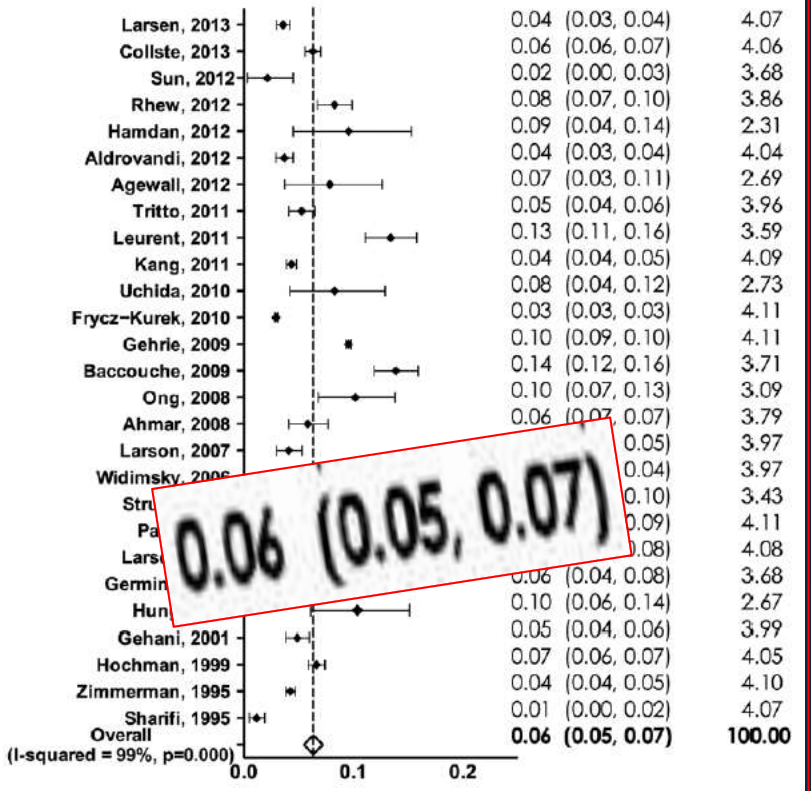
MINOCA
Prevalence and Prognosis

Systematic Review of Patients Presenting With Suspected Myocardial Infarction and Nonobstructive Coronary Arteries

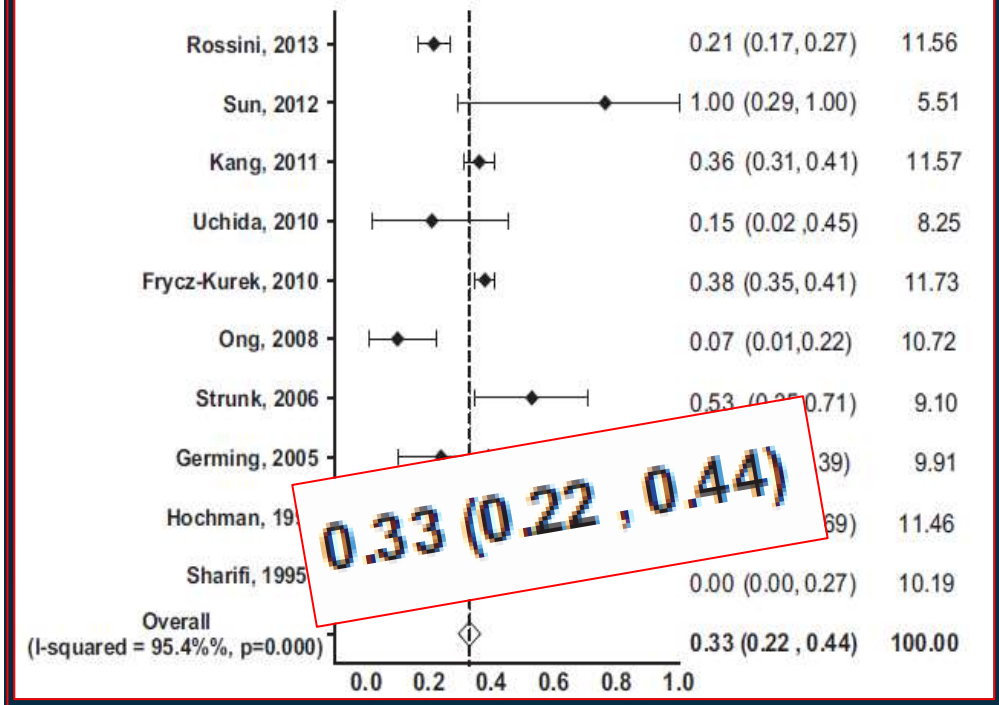
Sivabaskari Pasupathy, BSc(Hons); Tracy Air, BA (Hons), M.Biostatistics; Rachel P. Dreyer, BSc(Hons), PhD; Rosanna Tavella, BSc(Hons), PhD; John F. Beltrame, BSc, BMBS, PhD

- Prevalence: from 27 studies.
- STEMI in MINOCA from 10 studies

Prevalence 6%



STEMI in MINOCA 33%



Circulation. 2015;131:861-870.
DOI: 10.1161/CIRCULATIONAHA.114.011201

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One-Year Prognosis: from 46 studies.

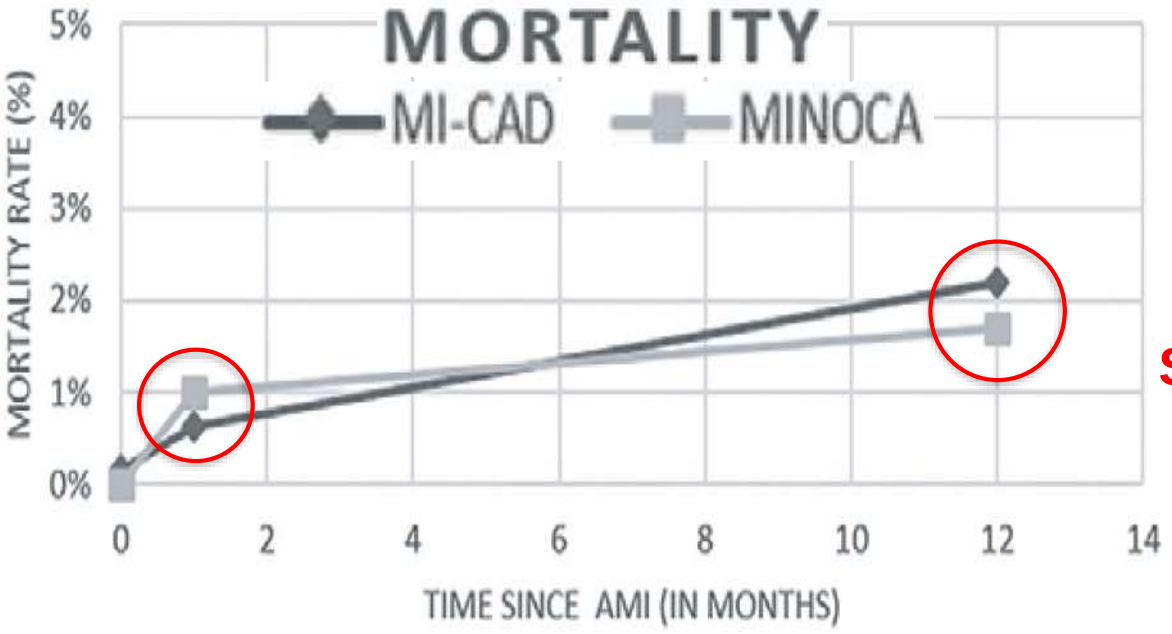
	Comparative Studies			
All-Cause Mortality	MI-CAD % (95% CI)	MINOCA % (95% CI)	OR (95% CI) *P* Value	All MINOCA Studies
In-hospital	3.2% (1.8%, 4.6%)	1.1% (-0.1%, 2.2%)	0.37 (0.2–0.67) *P*=0.001	0.9% (0.5%, 1.3%)
12-month	6.7% (4.3%, 9.0%)	3.5% (2.2%, 4.7%)	0.59 (0.41–0.83) *P*=0.003	4.7% (2.6%, 6.9%)

Circulation. 2015;131:861-870. DOI: 10.1161/CIRCULATIONAHA.114.011201

MINOCA
Prevalence and Prognosis

Presentation, Clinical Profile, and Prognosis of Young Patients With Myocardial Infarction With Nonobstructive Coronary Arteries (MINOCA): Results From the VIRGO Study

Basmah Safdar, MD, MSc; Erica S. Spatz, MD, MS; Rachel P. Dreyer, PhD; John F. Beltrame, MD, MPH; Judith H. Lichtman, PhD, MPH; John A. Spertus, MD; Harmony R. Reynolds, MD; Mary Geda, MSN; Héctor Bueno, MD, PhD; James D. Dziura, PhD, MPH; Harlan M. Krumholz, MD, SM; Gail D'Onofrio, MD, MS



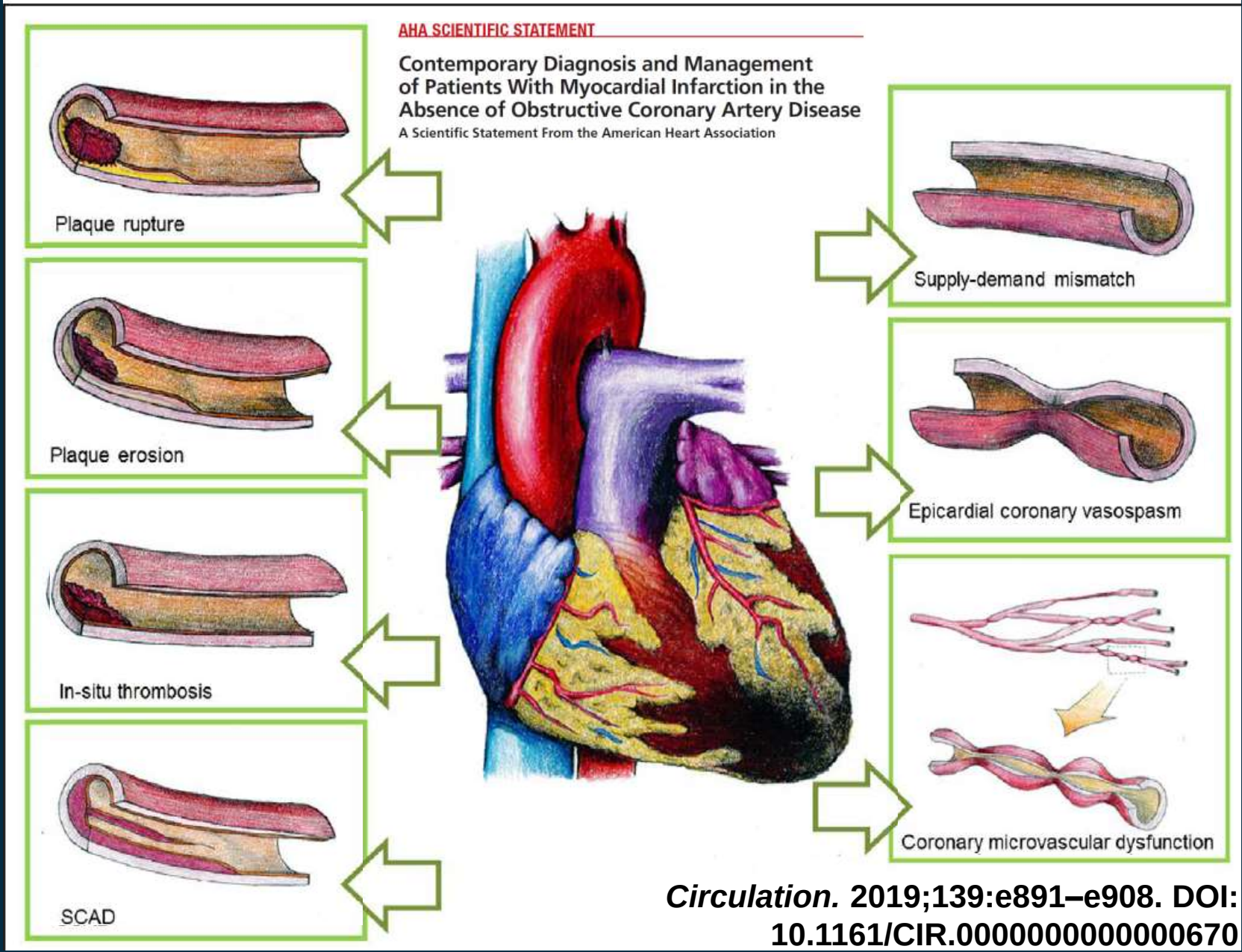
MI aged 18 to 55 years: nonobstructive vs obstructive coronary disease. N: 2690.

PREVALENCE MINOCA: 11,2%.
STEMI: 21% in MINOCA and 52% in MI-CAD.

One and 12-Months Mortality similar in MINOCA vs MI-CAD.

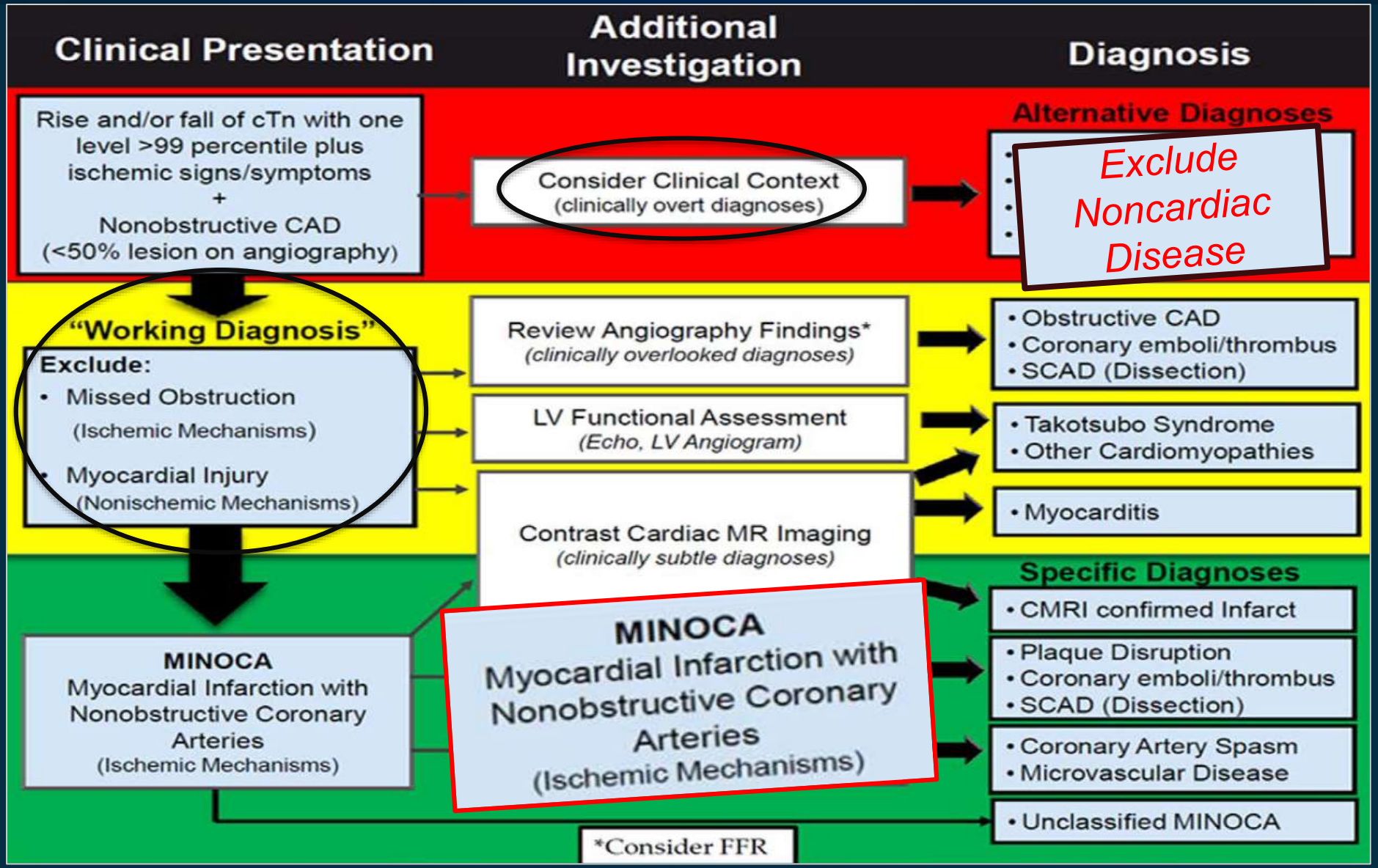
J Am Heart Assoc. 2018;7:e009174. doi:10.1161/JAHA.118.009174

MINOCA: ETHIOLOGY



Circulation. 2019;139:e891–e908. DOI: 10.1161/CIR.0000000000000670

MINOCA
A Working Diagnosis



Circulation. 2019;139:e891–e908. DOI: 10.1161/CIR.0000000000000670

MINOCA Treatment

AHA SCIENTIFIC STATEMENT

Contemporary Diagnosis and Management of Patients With Myocardial Infarction in the Absence of Obstructive Coronary Artery Disease

A Scientific Statement From the American Heart Association

Patients with acute myocardial infarction who are ruled out for obstructive coronary artery disease should undergo additional testing to elucidate the underlying cause of ischemia and to initiate appropriate treatment.

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-blocker ACE inhibitors/ARBs (in presence of left ventricular dysfunction, and possibly with preserved EF)
Spontaneous coronary artery dissection	Angiographic review	Aspirin
Takotsubo syndrome		
Cardiomyopathies		
Myocarditis		
Ischemic presentation (MINOCA)		
Plaque disruption		
Coronary artery spasm		
Coronary microvascular dysfunction		
Coronary embolism/thrombus		
Spontaneous coronary artery dissec		
Supply-demand mismatch		

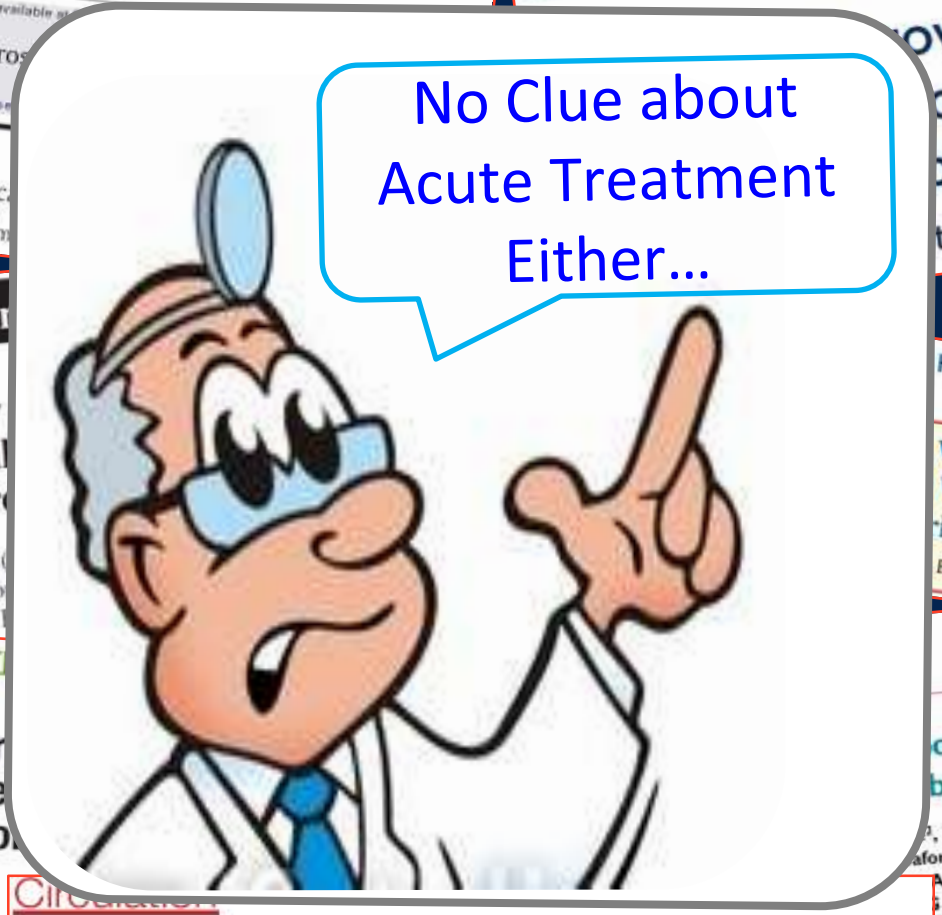
ACE indicates angiotensin-converting; intravascular ultrasound; MINOCA, myo; patent foramen ovale.
*These are selective and targeted in; infarction, including clinical assessment,
†These are selective and targeted th

Selective/Empirical Therapies†

- AAS, Clopidogrel…
- Statin
- Beta Blockers, ACE Inhibitors, Calcium Channel Blockers
- Nitrates, Nicorandil, Cilostazol…
- Unconventional Antianginal Therapies…
- Anticoagulant Therapy…
- Treatment of Underlying Condition in Supply Demand Mismatch….
- Medical or Devices Therapies in Heart Failure…

OI: 10.1161/CIR.0000000000000670

MINOCA: Acute Treatment?



Atherosclerosis 219 (2011) 10–14

Contents lists available

Atheros

journal homepage: www.else

ELSEVIER

Review

Myocardial infarction with angiographic

S. Agewall[a], L. Eurenius[b], C. Hofman-Bang[c], K. Malm

JACC STATE-OF-THE-ART REVIEW

ovascular Disease

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t Review

Coronar

Systematic Review

Suspected Myocardial

Cor

Sivabaskari Pasupathy, BSc

Rachel P. Dreyer, BSc(Ho

John F.

ORIGINAL RESEARCH ART

Medical Therapy for

Long-Term Outcome

Infarction With Nono

Disease

FOCUS REVIEWS ON CORONARY ARTERY DISEASE

Why, How and Where of

With Non-Obstructive

ries (MINOCA)

BSc, PhD; John F. Beltra

CURRENT OPINION

osition paper on myocardial

bstructive coronary arteries

, Harmony R. Reynolds[3], Alexander Niessner[4],

aforio[7], Raffaele De Caterina[8], Marco Zimarino[8],

Atar[1], Juan C. Kaski[6], Udo Sechtem[11], and

on Cardiovascular Pharmacotherapy

Circulation

AHA SCIENTIFIC STATEMENT

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A Scientific Statement From the American Heart Association

C3XV INTERVENTIONAL ACADEMY CELEBRATING 15 YEARS

Myocardial Infarction with No Obstructive Coronary Arteries

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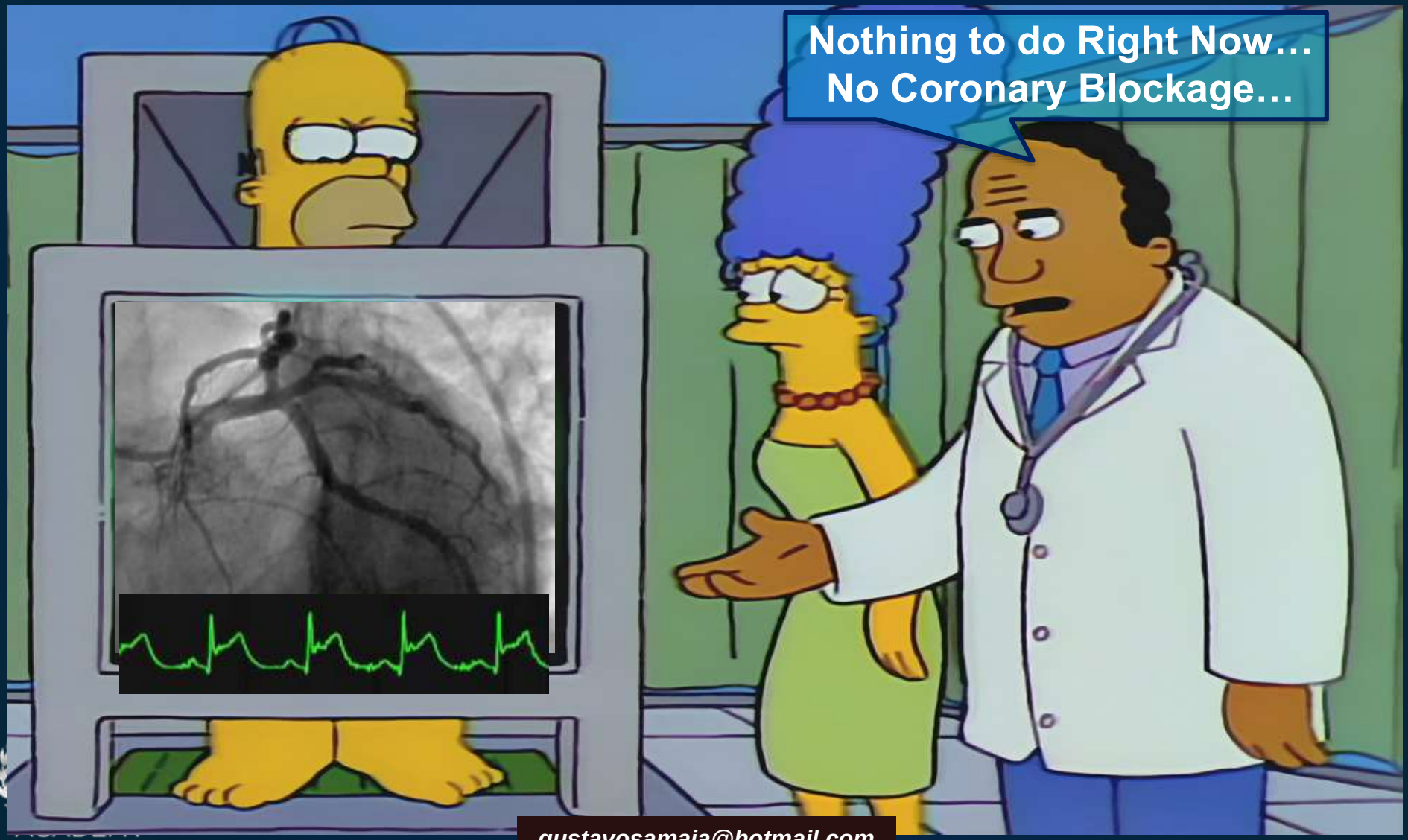

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MINOCA = AMI = Ischemia Revascularization?

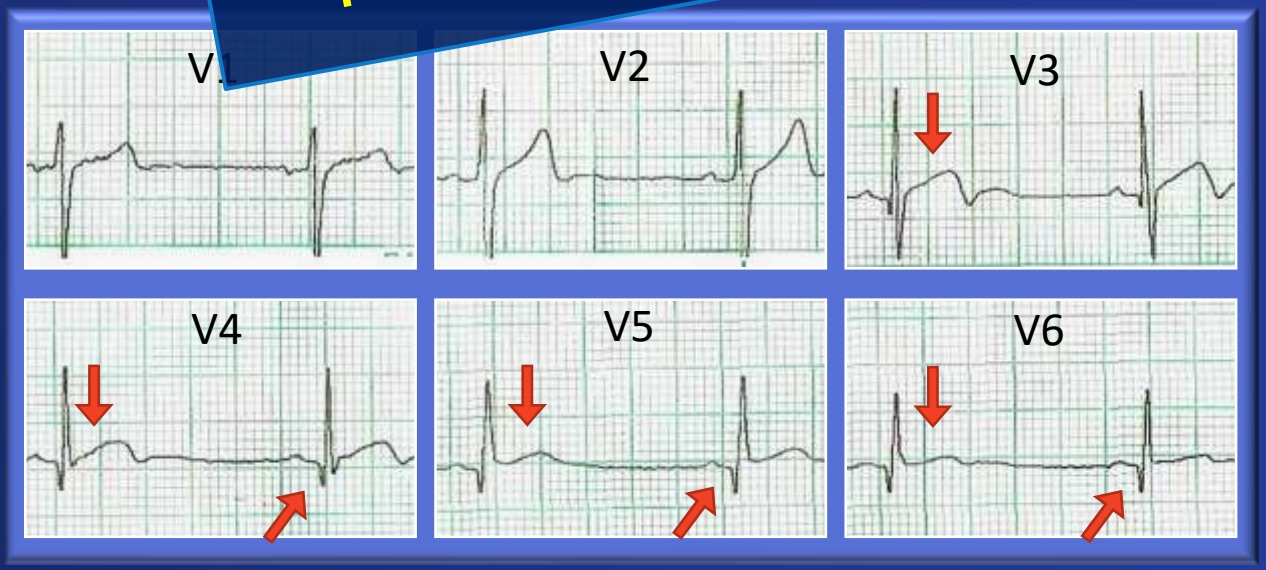
Revascularizat… is the restoration of perfusion to a ischemic … chieved by unblocking o… blood vessels or by sur… acements.

But if we have ischemia and no blockages?

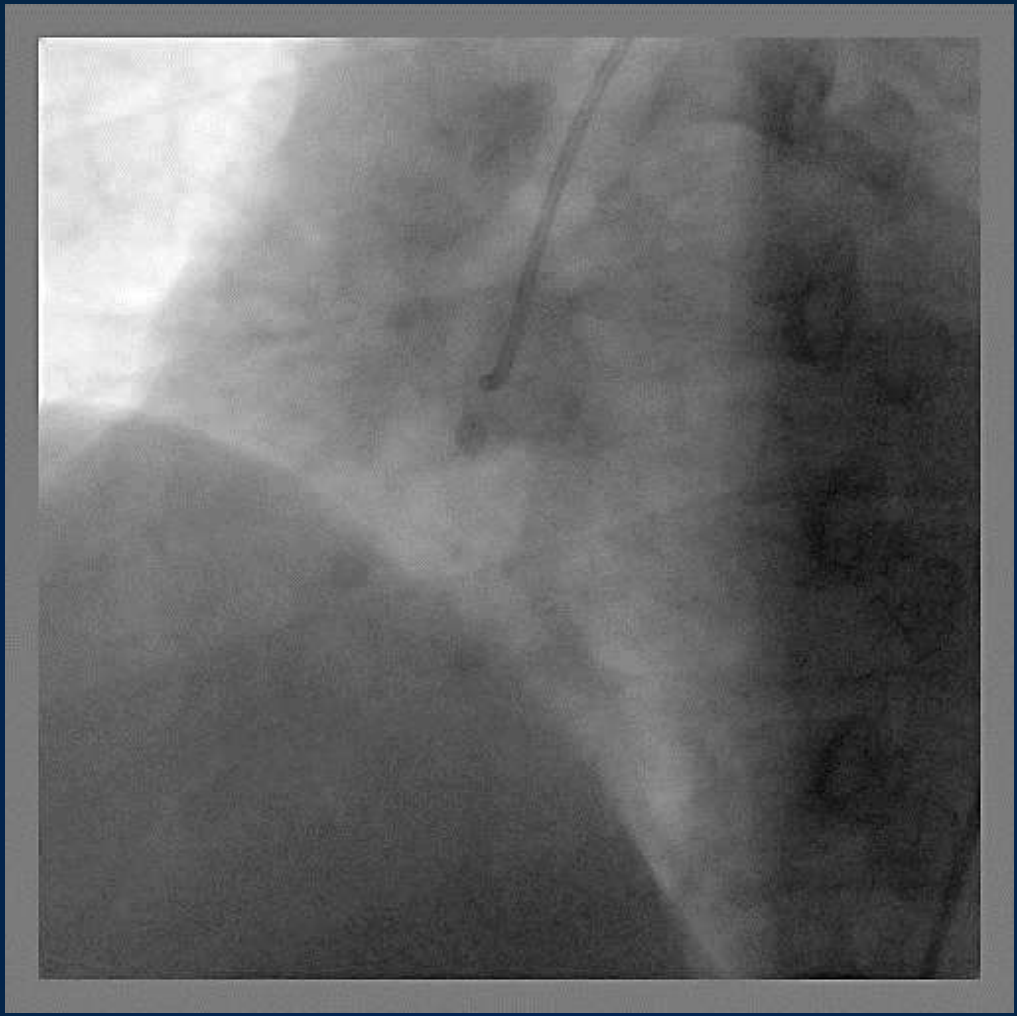
Case Presentation

- 39 years old male HT, obesity and metabolic syndrome.
- Ongoing oppressive 9/10 chest pain, associated with shortness of breath and diaphoresis.
- Mild heart failure on physical examination.
- Laboratory findings unremarkable, cardiac markers negative.
- Admission EKG showed abnormal Q waves and slight ST segment elevation in anterior and lateral leads.
- No pain relief or EKG changes with iv nitroglycerin.

Urgent angiography for immediate revascularization



Angiography: RCA.



The RCA is dominant, with no obstruction.

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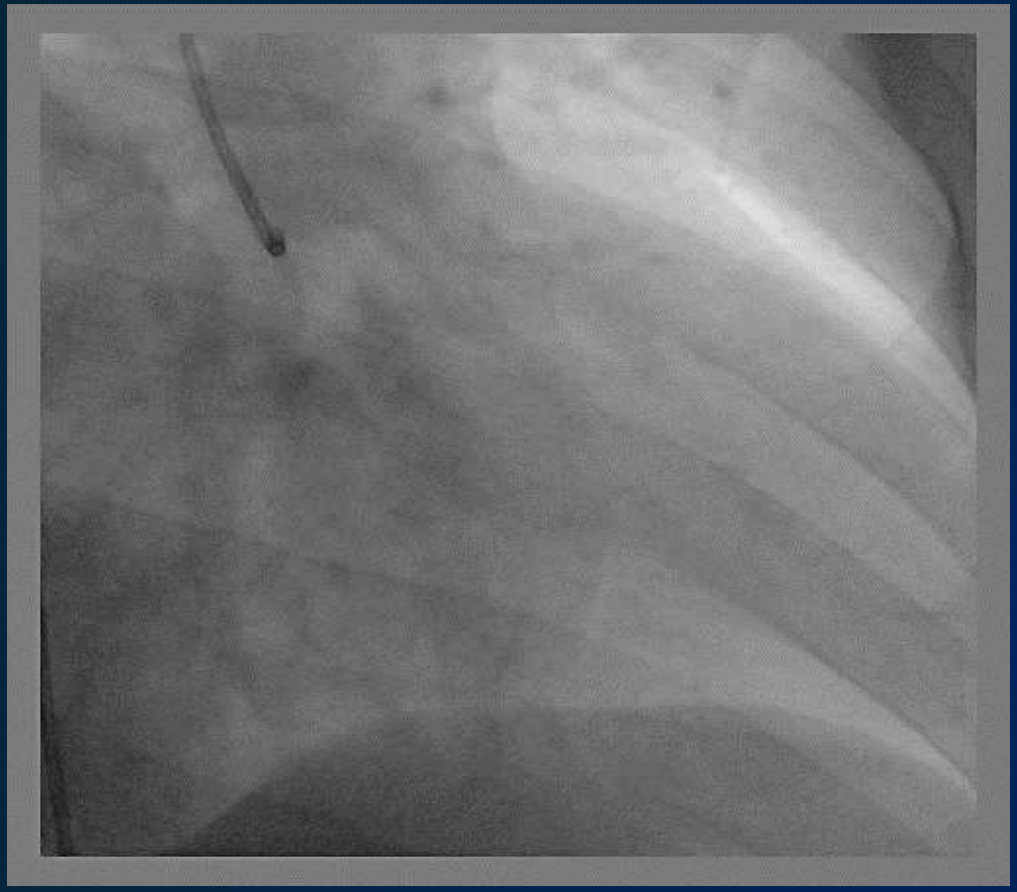
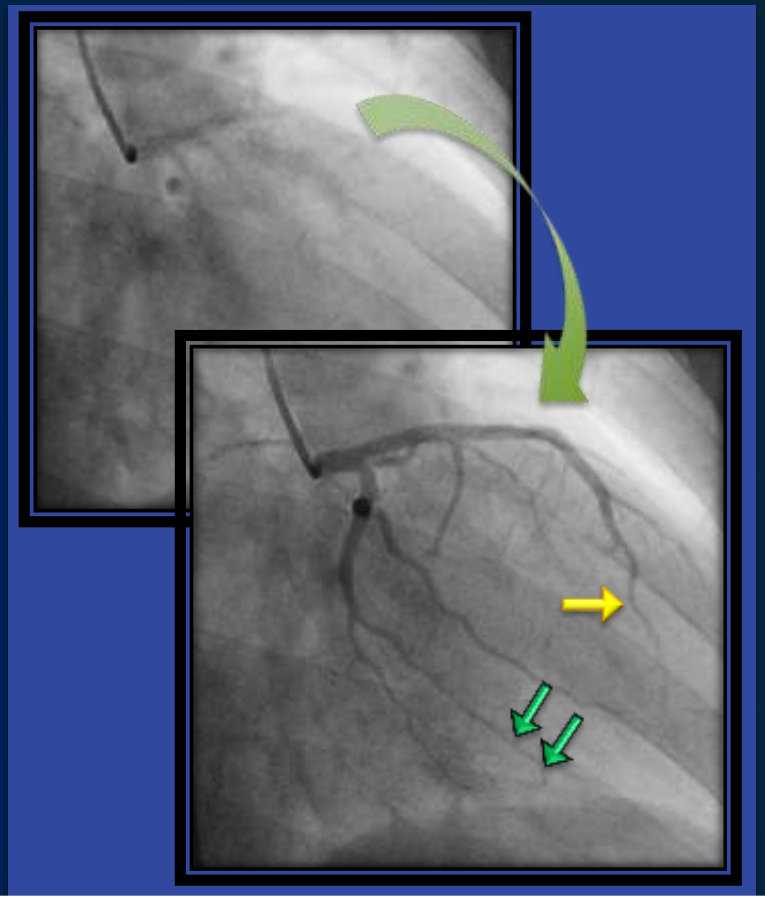
Angiography: Left Coronary Artery



No obstructions, but notorious slow flow in LAD: >3 Beats in filling the Vessel.

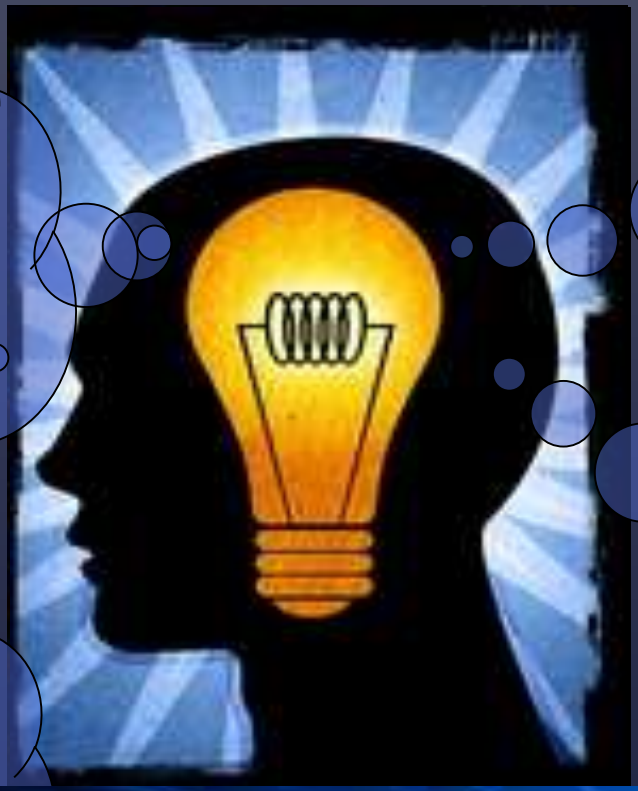


1.Contrast injection starts.
2.The CX is completely filled, the LAD is only partially filled.

We have a young male with ongoing chest pain, mild heart failure and ST changes in anterior leads… Urgent catheterization seems to be appropriate!

The patient doesn't have obstructive CAD. However, the slow flow in LAD is notorious.

Is the LAD slow flow related to the clinical situation?



Let's perform a Left Ventriculography!

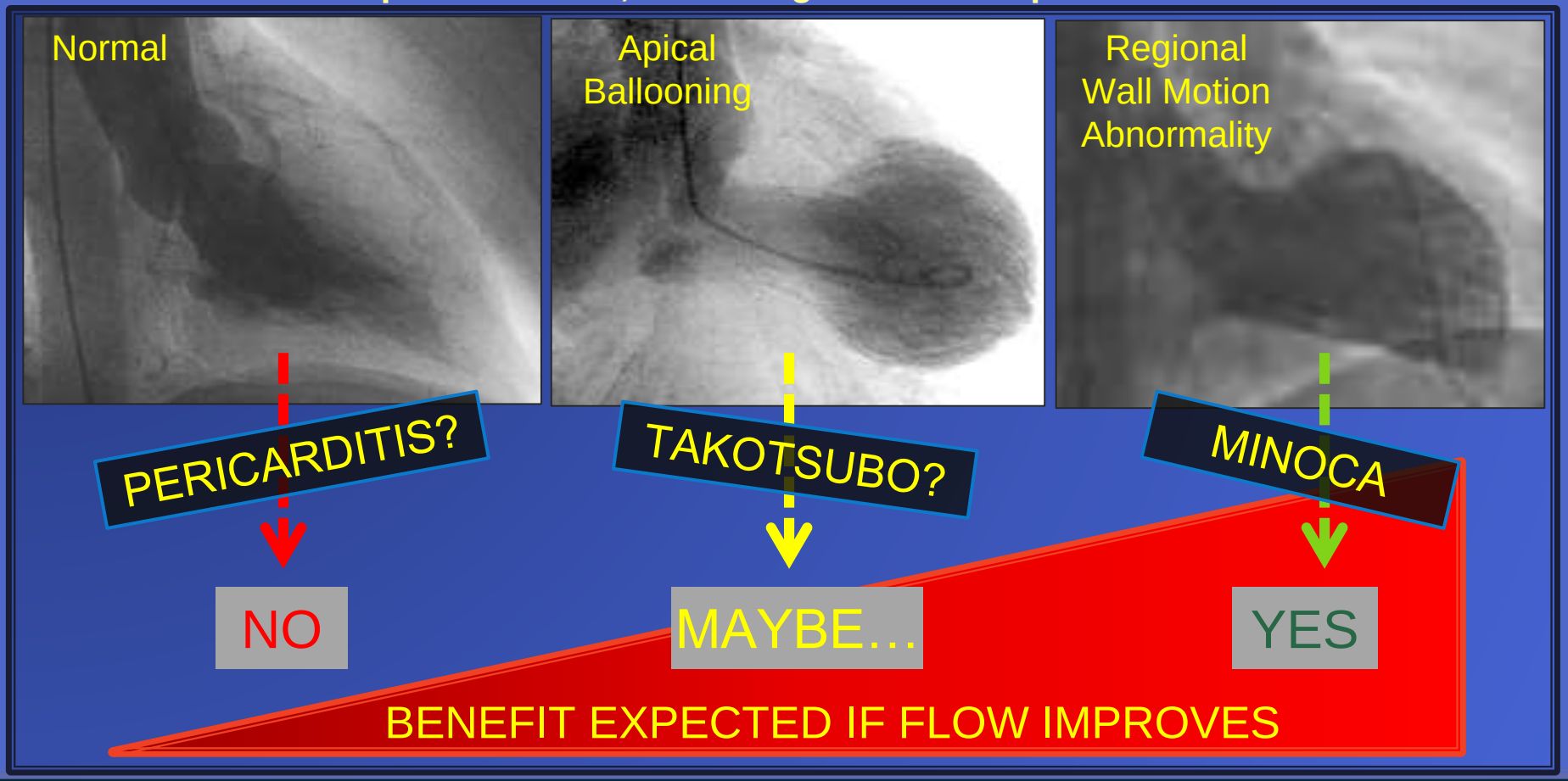
If slow flow is causing myocardial ischemia: **MINOCA**

1. How can we determine that?
2. Is there any appropriate treatment?

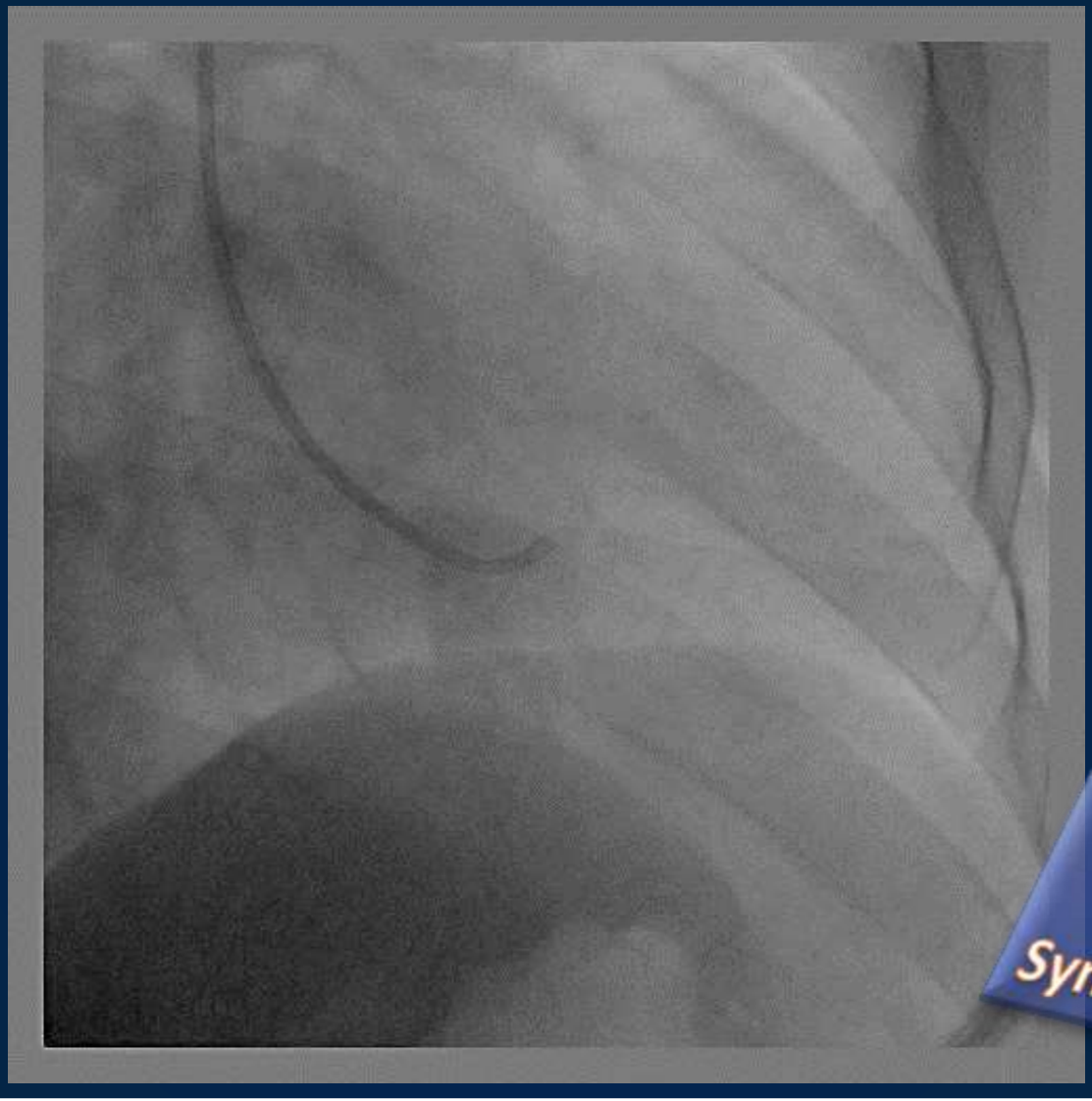
Left Ventriculography

We do not perform left ventriculography routinely.
In this case we wondered if the slow flow in LAD had impact on ventricular function, besides the chest pain and EKC changes compatible with myocardial ischemia.

In order to optimize D2B time, the patient was sent to the Cath Lab without previous ECO, according to our PPCI protocol.



Left Ventriculography



MINOCA
¿Cardiac
Syndrome Y?

Anterior hypokinesis with mild to moderate function impairment

gustavosamaja@hotmail.com

And what about treatment?

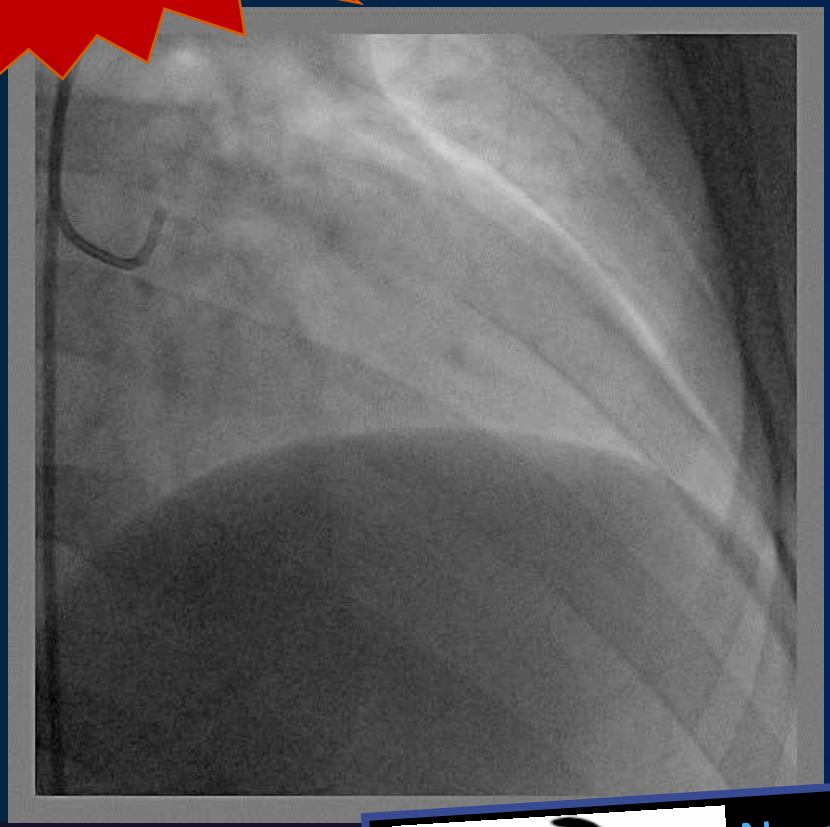
We homologated the treatment of this “unexpected” Slow Flow to our standard protocol for No Reflow.
We administered distal IC Adenosine for ensuring drug delivery to the distal vascular bed.

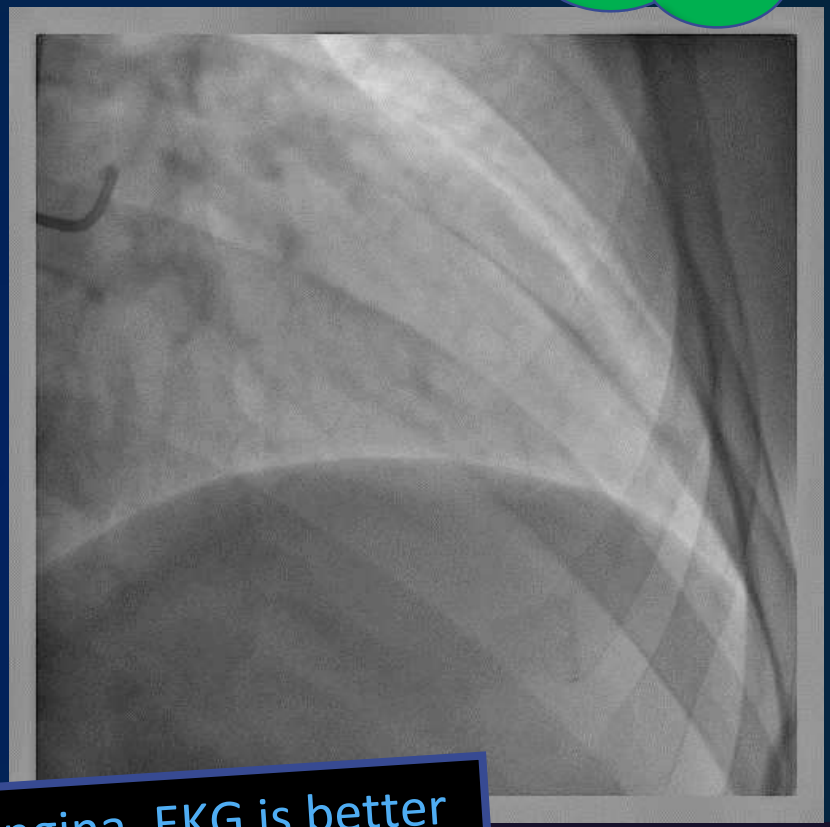
Ongoing chest-pain

Pre-adenosine

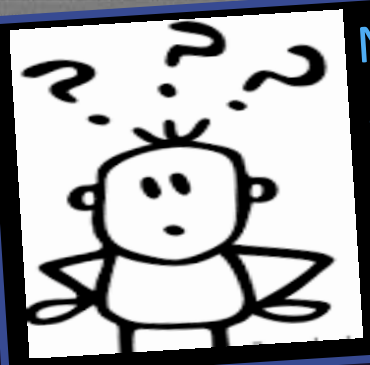
Post-adenosine

Pain relief





We excluded vasospasm: intracoronary NTG failed to restore flow



No angina, EKG is better and flow seems to have improved.

But… Can we show more objective data?

LAD flow improved and symptoms disappeared

Trying to show more objective data…
TIMI FRAME COUNT

TIMI Frame Count
A Quantitative Method of Assessing Coronary Artery Flow

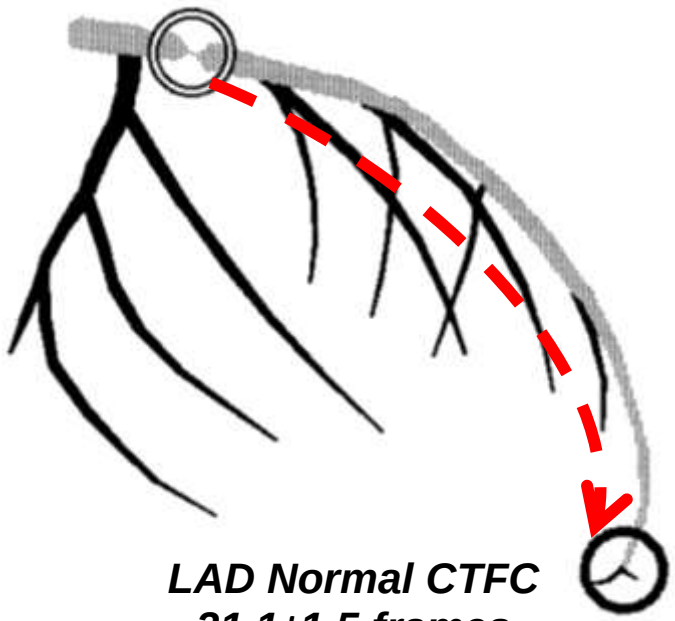
C. Michael Gibson, Christopher P. Cannon, William L. Daley, J. Theodore Dodge, Barbara Alexander, Susan J. Marble, Carolyn H. McCabe, Lori Raymond, Terry Fortin, W. Kenneth Poole, and Eugene Braunwald

Circulation. Volume 93(5):879-888. March 1, 1996.

The number of cineframes needed for dye to reach standardized distal landmarks was counted to objectively assess an index of coronary blood flow as a continuous variable.

The TIMI FRAME COUNT is longer in LAD, so the CORRECTED TIMI FRAME COUNT (CTFC) is calculated dividing by 1,7.

The index is simple, objective, reproducible and quantitative, besides all the potential factors that introduce variability.

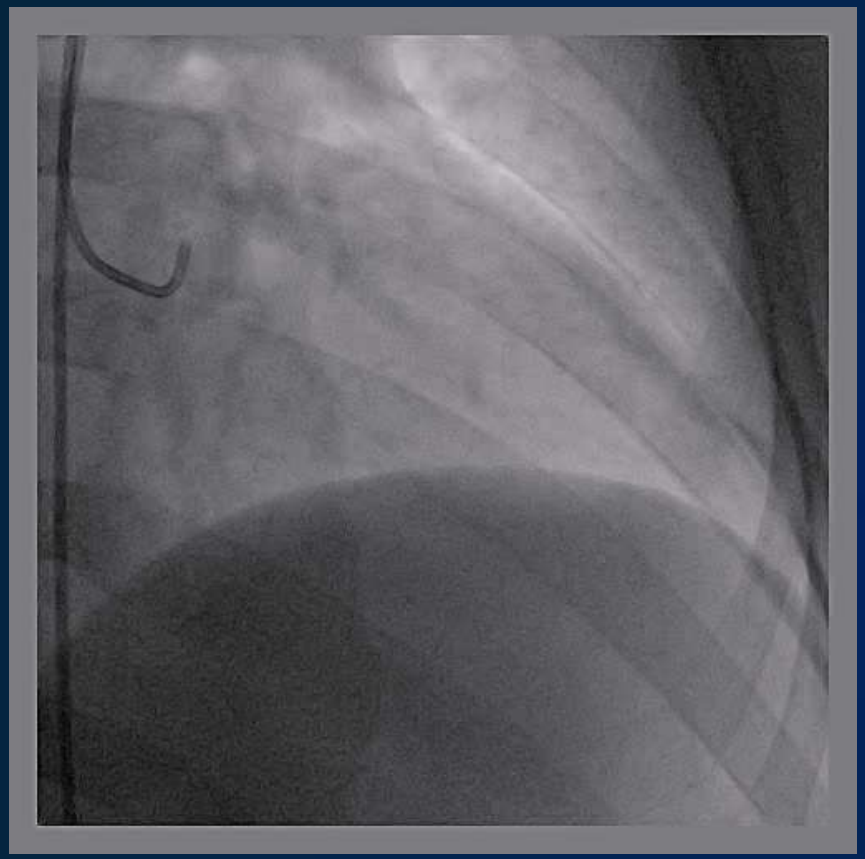


LAD Normal CTFC
21.1±1.5 frames

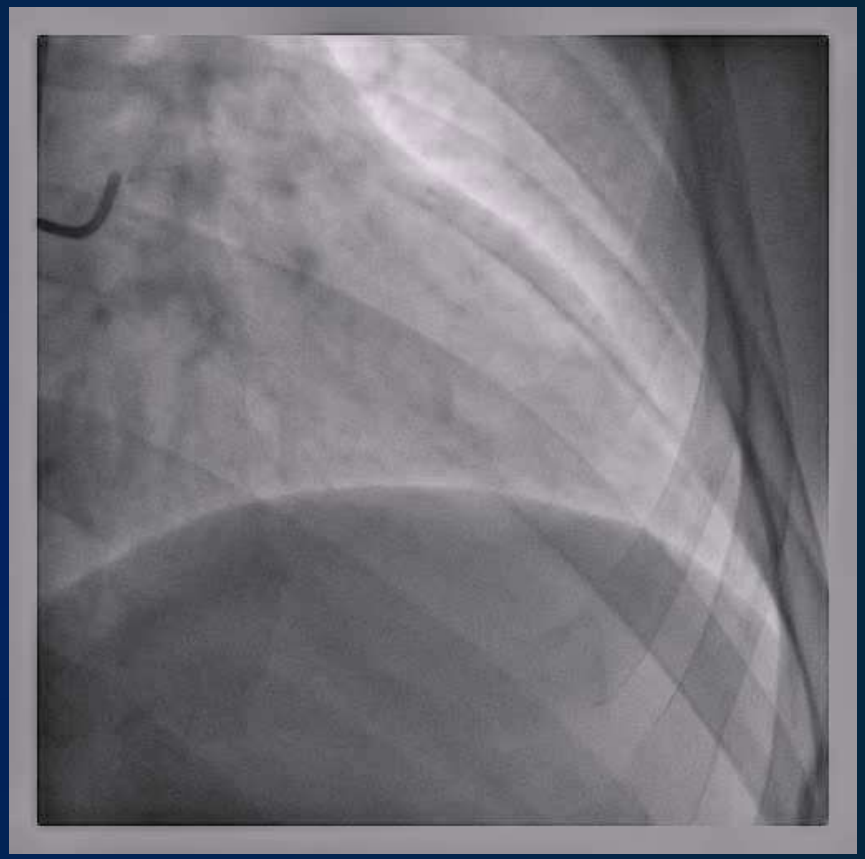
Normal flow is ≥15 to ≤27 frames for 95% Confidence Interval.

Treatment: IC Adenosine

Pre-adenosine CTFC 44,7



Post-adenosine CTFC 28,2



The original paper analyzed angiograms filmed at 30 frames/second.

Our angiograms were taken at 15 frames/second.

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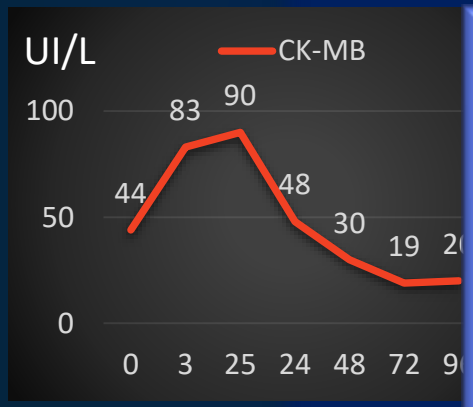
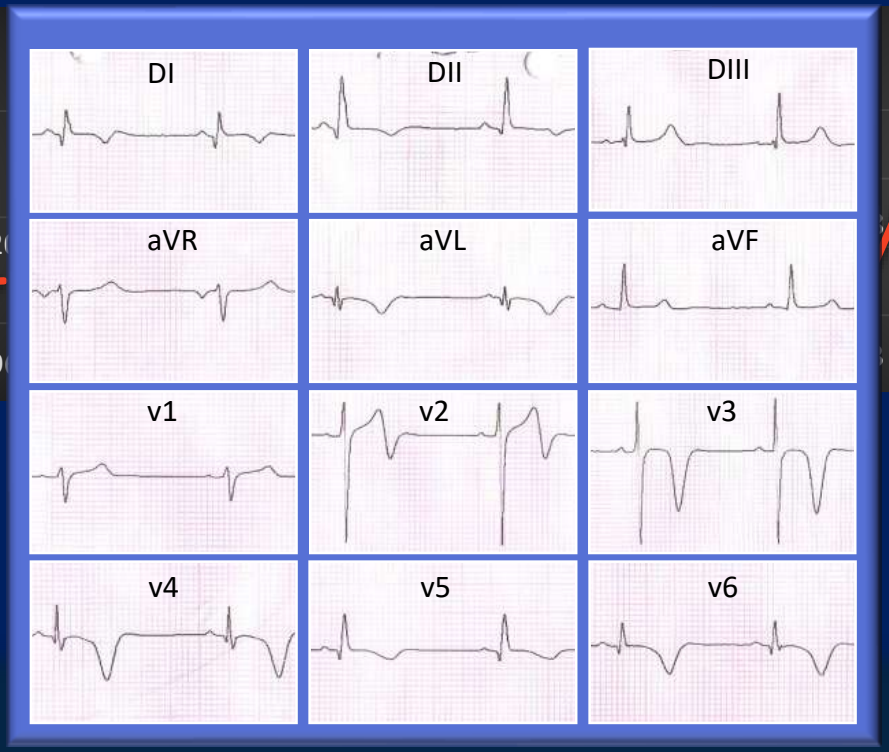
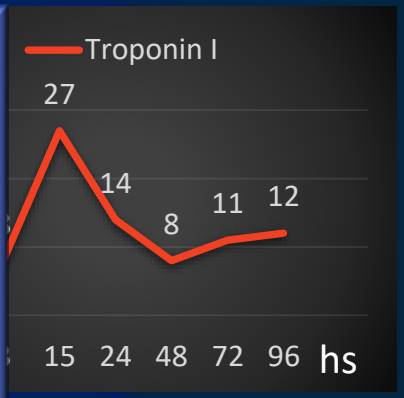
Follow Up

In Hospital

- Clinically uneventful.
-Cardiac biomarkers elevation and pronounced anterior T wave inversion.
-Discharged on aspirin, atorvastatin, enalapril and diltiazem.

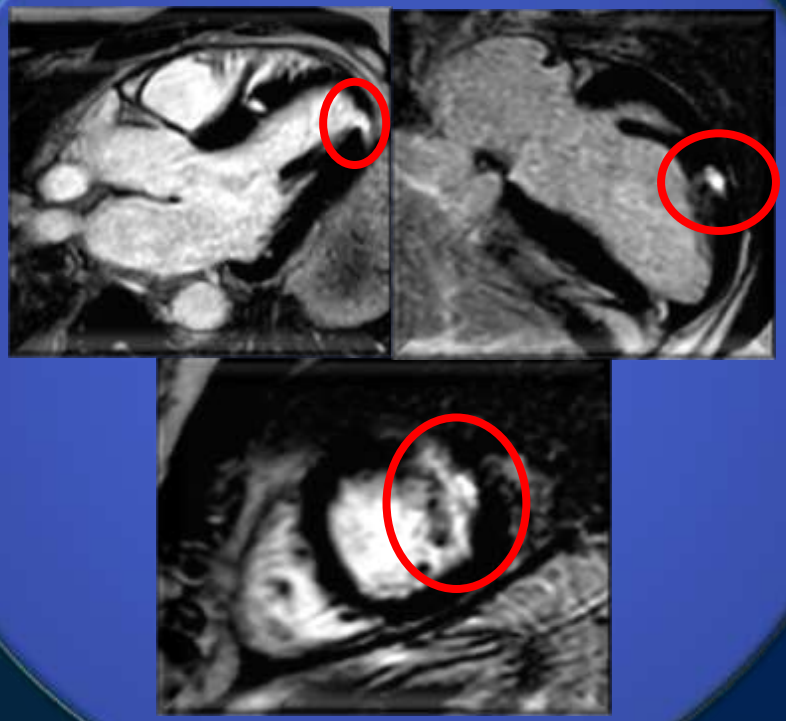






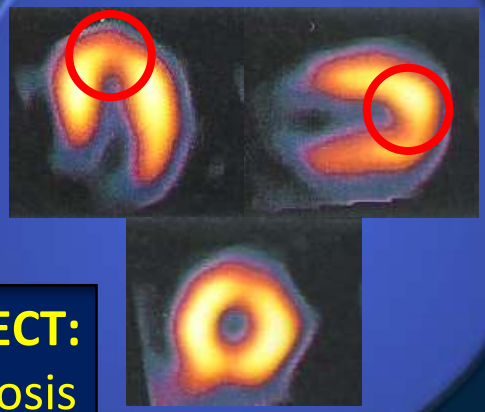
Was it a MI? It was a MI

MRI: LGE



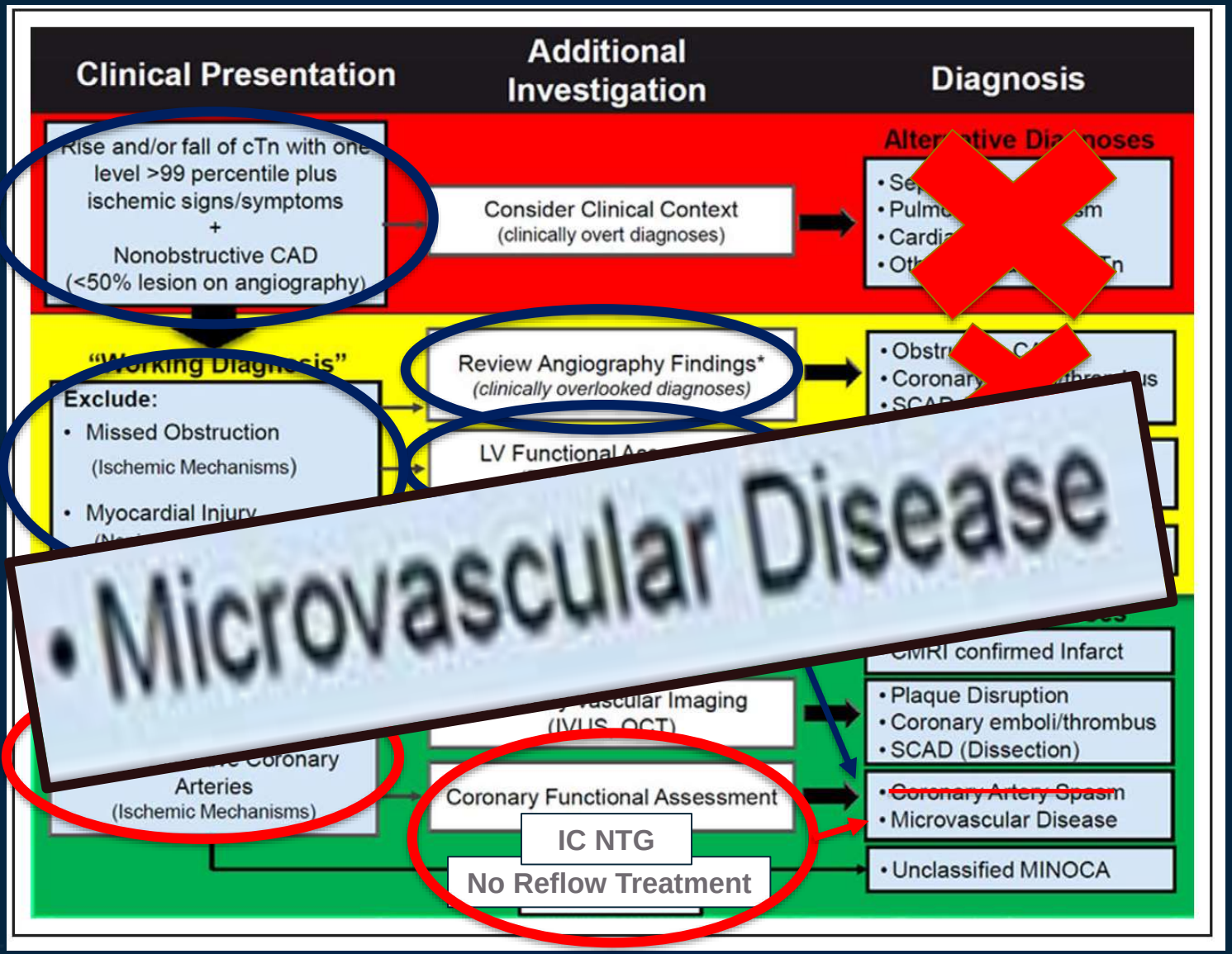
Late Gadolinium Enhancement: Anterior subendocardial necrosis with transmural progression on the apical anterior and inferior segments associated with hypokinesis with a mild impairment of LVEF.

SPECT



Cardiac SPECT: apical necrosis pattern.

The Case of our MINOCA Patient



Circulation. 2019;139:e891–e908. DOI: 10.1161/CIR.0000000000000670

The case of our MINOCA Patient

Summary of Coronary Artery Vasomotor Disorders

	Coronary Slow Flow
Chest Pain Angiography	Prolonged – at Rest
Pathophysiology	Normal with delayed opacification ✓
Risk Factors	Unclear/ Increased microvascular resistance ✓
Coronary flow reserve	Male gender, Obesity
Nitrates response	
Exercise stress test	Normal/ debatable ✓
Diagnostic test	
Management	variable
Patient Population most affected	Usually Negative / debatable
	None
	Dipyridamol, Mibefradil, others are investigative
	Young Males

Coronary Slow Flow
It is related to increased microvascular resistance and stress test is usually negative. It occurs in obese young males, with prolonged rest pain.

Angiography is normal but with delayed opacification.

Coronary [illegible] group of disorder with the main pathology being at the level of the microvascular coronary bed.

Adapted from Yakan et al. ReviewsinVascularMedicine2 (2014) 118–122

The case of our MINOCA Patient related to Coronary Slow Flow

Coronary Slow Flow is defined angiographicaly as the slow antegrade passage of contrast material in the coronary arterial tree in the absence of epicardial coronary artery stenosis or spasm.

The prevalence of the Coronary Slow Flow is between 1% and 5% of all coronary angiograms performed in ACS. True myocardial infarction is uncommon and occurs in about of 8% of those patients.

The pathophysiology of CSFP is not yet fully understood, resting resistance of the microvascular coronary arteries is thought to play a central role.

The name “Cardiac Syndrome Y” has been suggested due to the possible role of “neuropeptide Y” in the pathophysiology.

- There are not guidelines nor recommendations about acute or chronic treatment of Syndrome Y.
- We homologated the acute treatment to our protocol for No Reflow management.

The case of our MINOCA Patient related to Coronary Slow Flow

And What About Secondary Prevention in those Cases?

ORIGINAL RESEARCH ARTICLE

Medical Therapy for Secondary Prevention and Long-Term Outcome in Patients With Myocardial Infarction With Nonobstructive Coronary Artery Disease

Circulation. 2017;135:1481–1489. DOI: 10.1161/CIRCULATIONAHA.116.026336

MINOCA in 9136 p. 4.1 years f[illegible]up.

- Benefici[illegible]tins and angiotensin-c[illegible]ngiotensin

IN SUMMARY…
NO SPECIFIC RECOMMENDATIONS…

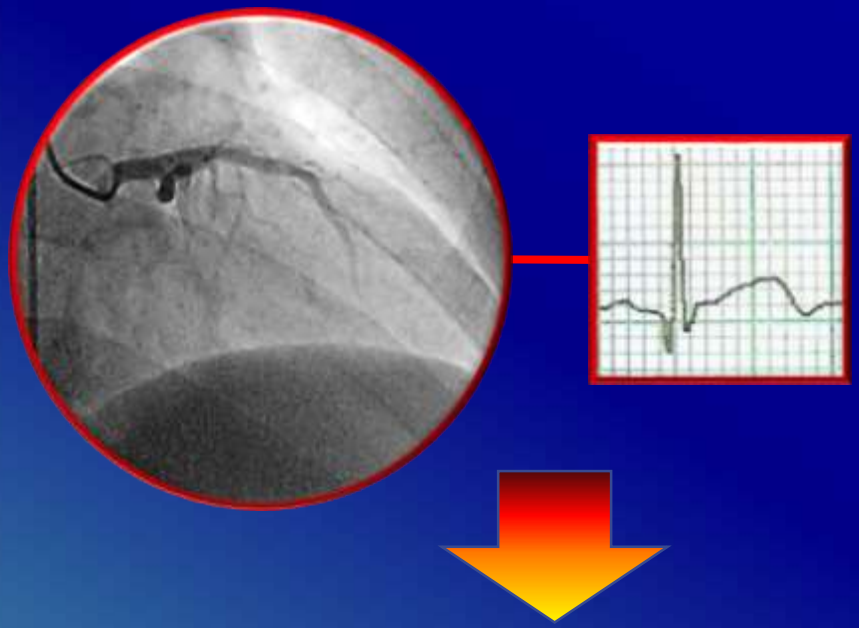
- Trend t[illegible]ects of β-blockers.

- No benefit of DAPT.

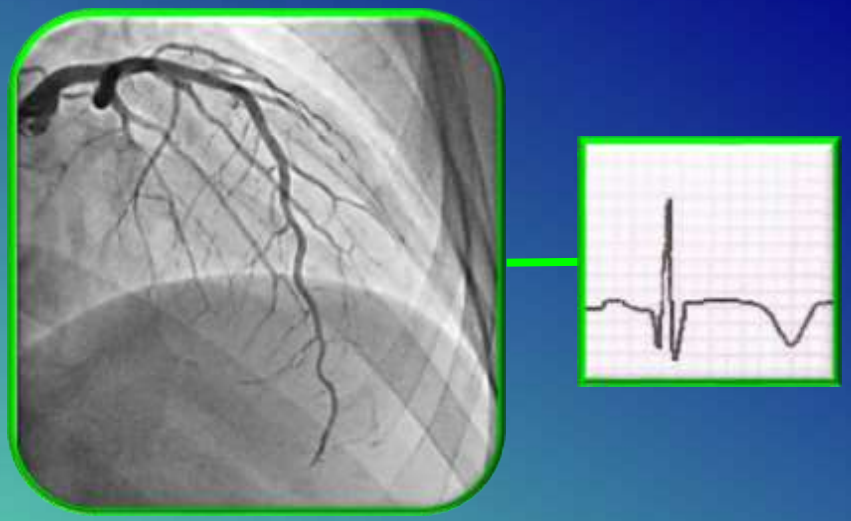
Berthil Lindal et Al. Circulation 2017

C3XV INTERVENTIONAL ACADEMY CELEBRATING 15 YEARS

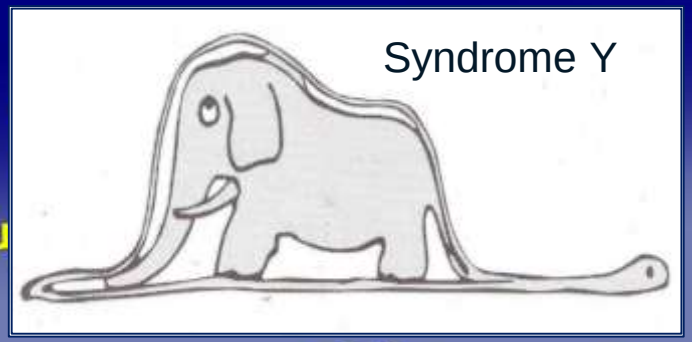
TAKE HOME MESSAGE



- Coronary arteries with no obstruction doesn't always mean "Normal Coronary Arteries": **KEEP AN EYE ON THE FLOW!**
- Improving flow with IC drugs can be beneficial for these patients, especially if "*Syndrome Y*" is causing an AMI.



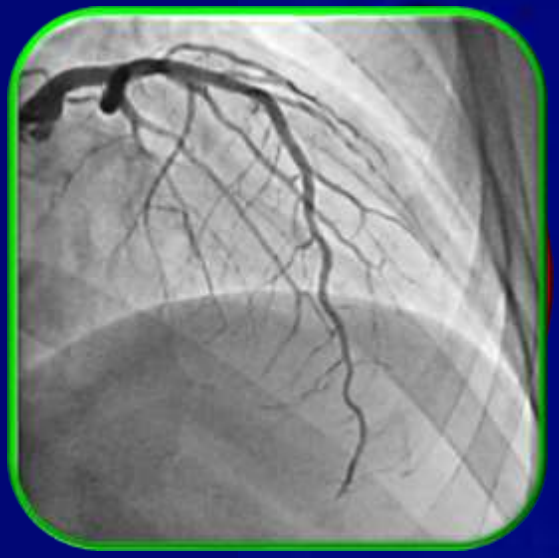
The essential can be invisible to the eye…



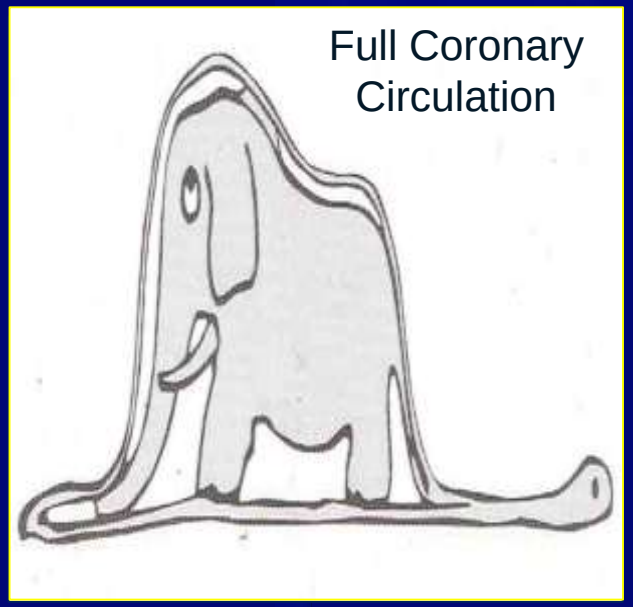
gustavosamaja@hotmail.com

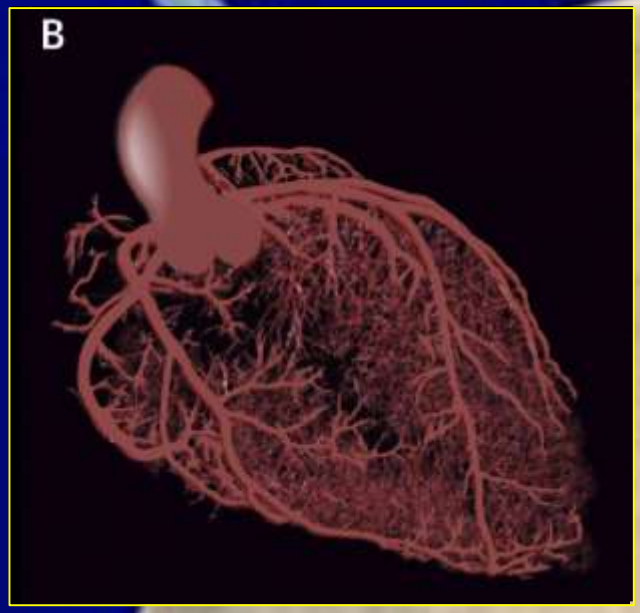
Take Home Message II

Microcirculation is Invisible to the Angio…



Be Aware of the Flow:
- MINOCA
- Syndrome Y
- No Reflow
- Blush in PPCI





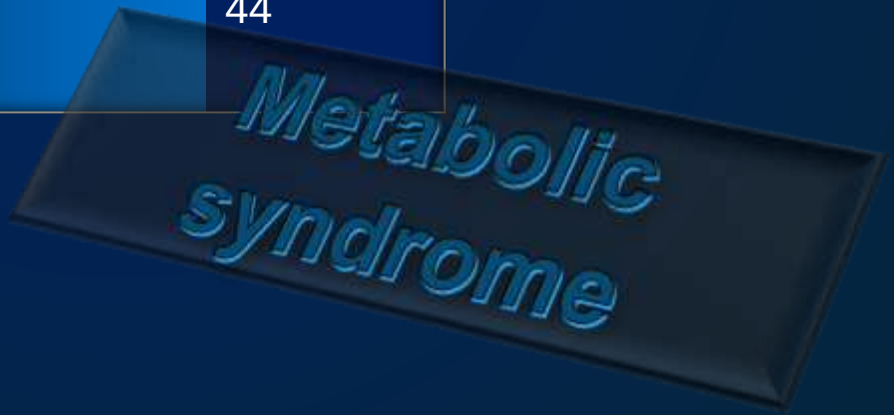

Thank You
gustavosamaja@hotmail.com

BACK UP

Laboratory results

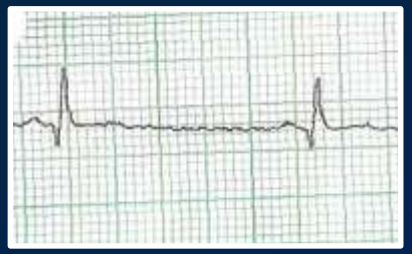
Hematocrit	38%	Hemoglobin	13mg/dl
Leukocites	11700	Platelet	230000
Urea	33 mg/dl	Creatinine	1.02 mg/dl
Sodium	139 mEq/l	Potasium	4.7 mEq/l
Glucose	125	Hepatogram	Normal
Total Cholesterol	152	Triglycerides	251
LDL Cholesterol	63	HDL Cholesterol	33
CPK	183	CK-mB	44
Troponin I	Negative		

+ Hypertension and central obesity



EKG

Admission



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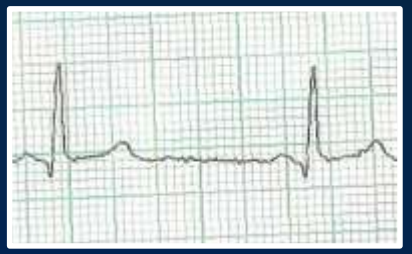
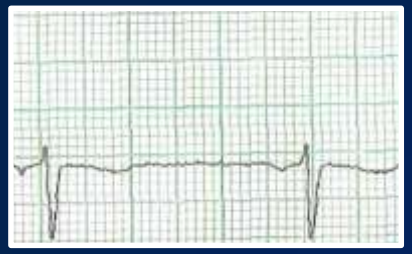
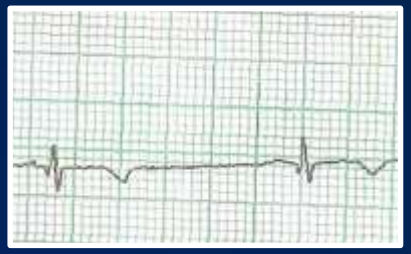


DII

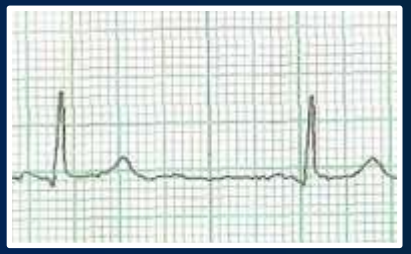
DIII



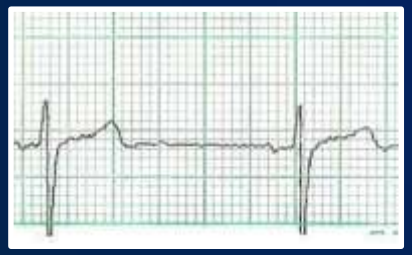
aVR



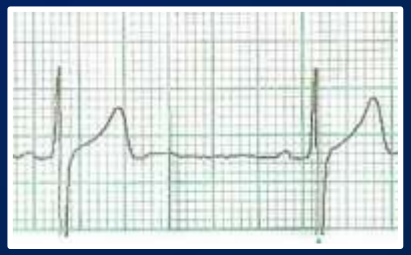
aVL



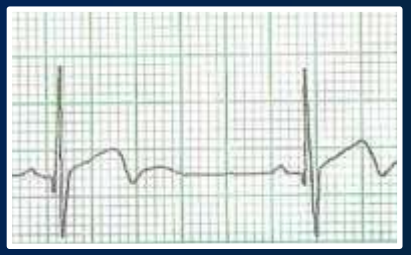
aVF



v1



v2



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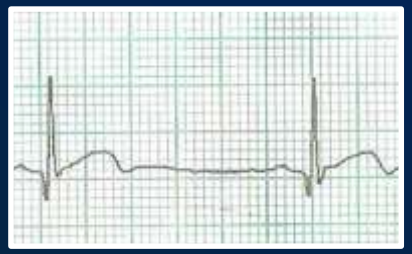
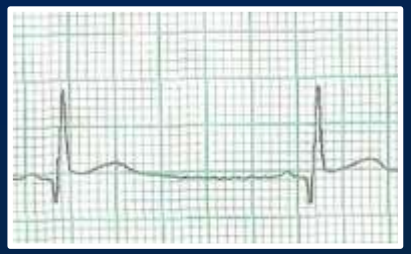
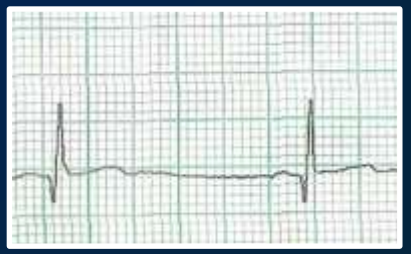


v4



v5

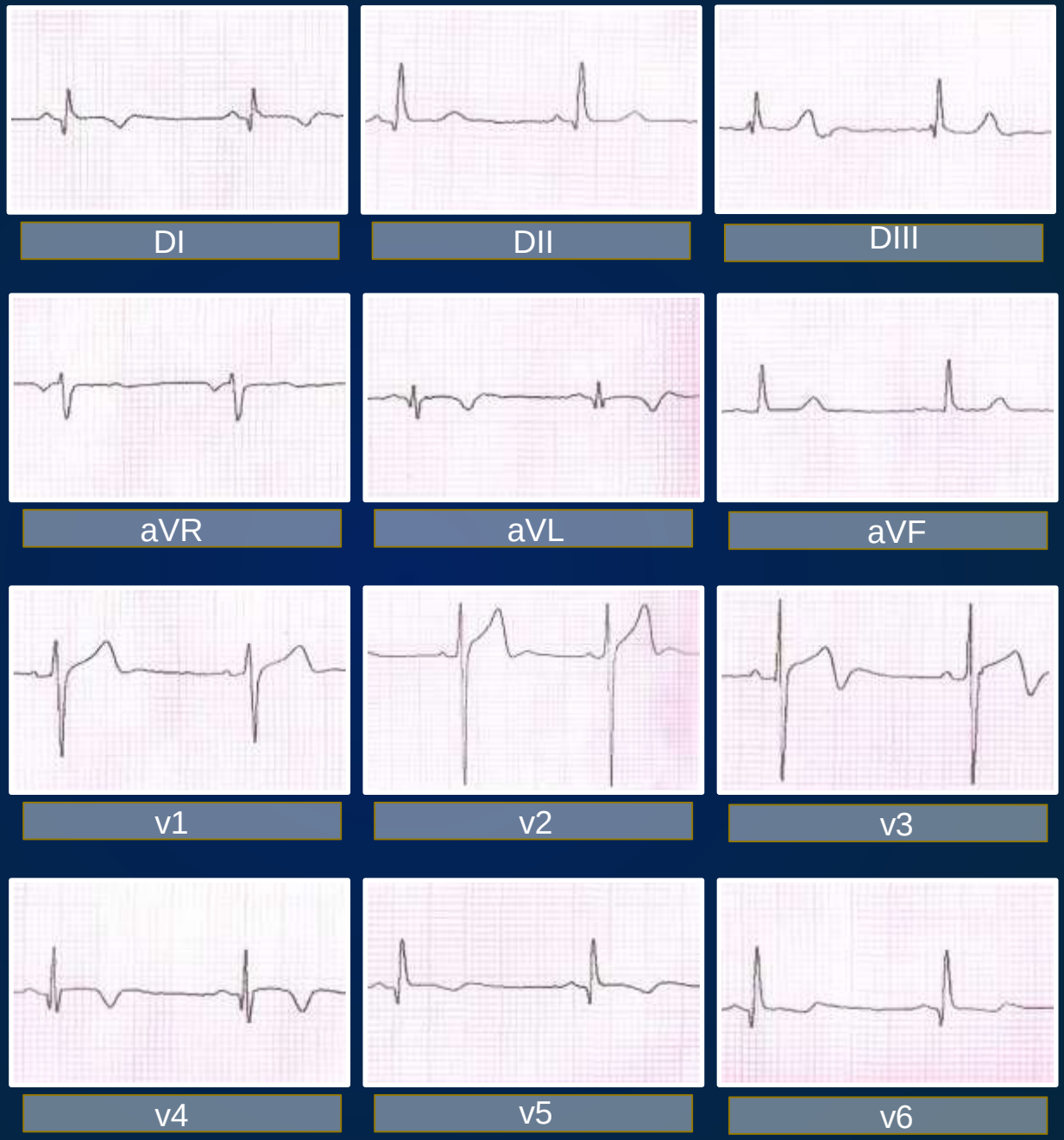


v6

C3XV INTERVENTIONAL ACADEMY CELEBRATING 15 YEARS

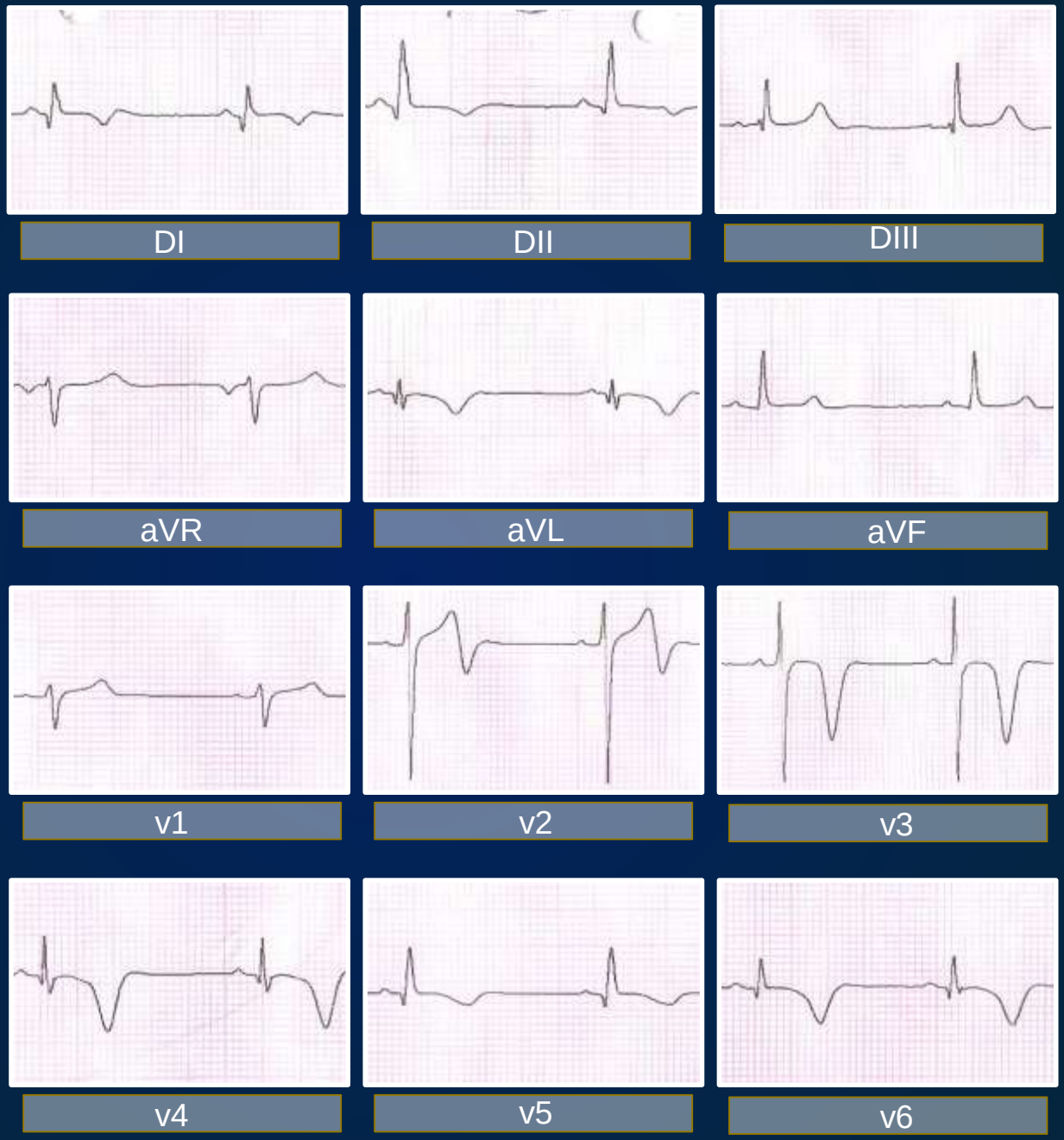
EKG

Post Adenosin



EKG

Evolution



ORIGINAL CONTRIBUTION

The Spontaneous Coronary Slow-Flow Phenomenon: Reversal by Intracoronary Nicardipine

Hetal H. Mehta, MD[1]; Mackenzie Morris, MD[1,2]; David L. Fischman, MD[1]; John J. Finley, IV, MD[3]; Nicholas Ruggiero, MD[1]; Paul Walinsky, MD[1]; Melissa McCarey, MPH[1]; Michael P. Savage, MD[1]

ABSTRACT: Objective. An under-recognized cause of chest pain, the coronary slow-flow (CSF) phenomenon is characterized by delayed coronary opacification during diagnostic angiography in the absence of epicardial coronary artery disease (CAD). Given its angiographic resemblance to no-reflow during percutaneous coronary intervention, a condition associated with microvascular spasm responsive to calcium-channel blockers, we hypothesized that spontaneous CSF may similarly be reversed by intracoronary (IC) nicardipine. **Methods.** The effect of IC nicardipine was evaluated in 30 patients. CSF was defined as spontaneously delayed flow (<TIMI 3) during diagnostic coronary angiography in the absence of obstructive epicardial CAD or other conditions associated with impaired flow. Nicardipine was administered as a 200 µg IC bolus, after which repeat angiography was performed. Coronary flow before and after nicardipine was evaluated by TIMI flow grade and corrected TIMI frame count (TFC) assessments. **Results.** The study population consisted of 22 men and 8 women (mean age, 54 ± 11 years). Clinical presentation was rest angina in 21 patients (70%). At baseline, CSF with <TIMI 3 flow was observed in 49 vessels. TFC was prolonged (>27) in 68/90 vessels (76%). IC nicardipine produced markedly accelerated coronary filling, which was corroborated by TFC analysis. TFC was 47 ± 17 before vs 15 ± 5 after nicardipine (*P*<.001). All vessels demonstrated TIMI 3 flow and TFC <28 after nicardipine treatment. **Conclusions.** IC nicardipine appears highly effective in reversing spontaneous CSF. These findings implicate microvascular spasm in the pathogenesis of CSF. Future studies of oral calcium-channel blockers in the long-term management of CSF are needed.

J INVASIVE CARDIOL 2018 December 15 (Epub Ahead of Print).
KEY WORDS: coronary microcirculation, coronary slow flow phenomenon, acute coronary syndromes, microvascular spasm

Chest pain contributes to frequent emergency room visits and is a leading cause of increasing health-care costs. An under-recognized cause of chest pain, the coronary slow flow (CSF) phenomenon is characterized by delayed coronary opacification during diagnostic angiography in the absence of obstructive epicardial coronary artery disease (CAD) or other conditions that are known to impair coronary flow.[1,2] First described in 1972, CSF may be more common than generally appreciated. CSF has been described in up to 7% of patients undergoing diagnostic angiography and may account for up to 4% of unstable angina admissions.[1,3] The clinical course can be challenging, with recurrence of chest pain occurring in up to 80% of patients and hospital readmission in 20% within a 2-year follow-up period.[4] While the pathophysiology of CSF is not fully understood, abnormally elevated microvascular resistance appears to be an important feature.[5] Because of the angiographic similarity of CSF to no-reflow observed during percutaneous coronary intervention (PCI) – a condition associated with microvascular spasm – we hypothesized that CSF may be reversed by intracoronary (IC) administration of nicardipine. Nicardipine, a dihydropyridine calcium-channel blocker with selective vasodilatory effects, has been shown to increase coronary blood flow and reverse no-reflow complicating PCI.[6-9] Accordingly, the goal of this study was to assess the effect of IC nicardipine on the CSF phenomenon.

Methods

The effect of IC nicardipine on CSF was assessed in 30 patients. CSF was defined as the presence of angiographically normal or near-normal coronary arteries and Thrombolysis in Myocardial Infarction (TIMI) 2 flow (requiring >3 beats to opacify prespecified branch points in the distal vasculature of at least one of the three major epicardial coronary vessels).[1,3] Patients were excluded if there were coronary artery stenoses >40% or other conditions that can be associated with impaired coronary flow (recent PCI, coronary artery ectasia, epicardial coronary arterial spasm, cardiomyopathy, or significant valvular heart disease).

Nicardipine was prepared in a diluted solution of normal saline with a concentration of 100 µg/1 mL. Nicardipine was administered as a 200 µg intracoronary bolus, after which repeat angiography was performed. Coronary flow before and after nicardipine was assessed by TIMI flow grade and by the corrected TIMI frame count (TFC) based on previously established criteria by Gibson et al.[10] TIMI flow grade <3 and TFC ≥28 were considered abnormal.

Demographic data are reported as mean ± standard deviation or number (percentage). Differences in TFC before and after IC nicardipine were evaluated by paired *t*-tests. A *P*-value <.05 was considered statistically significant. This retrospective study was approved by the Institutional Review Board of the participating hospitals.

Objective evidence of ischemia	
Abnormal electrocardiogram	9 (30%)
Abnormal stress test	9 (30%)
Elevated troponin	4 (13%)
Any of the above	22 (73%)

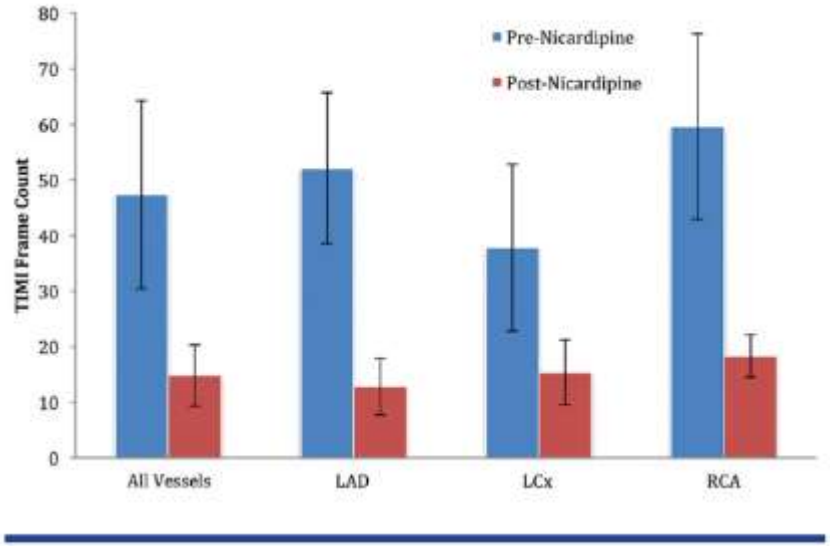
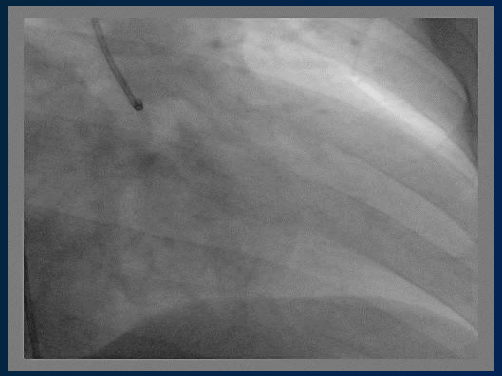
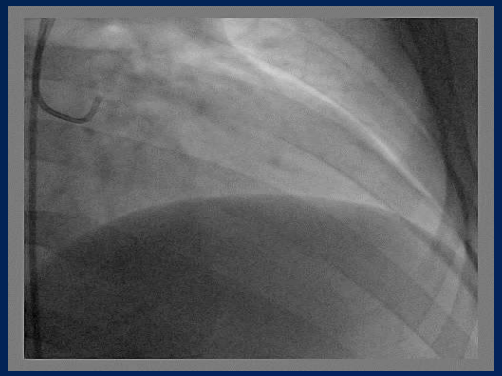
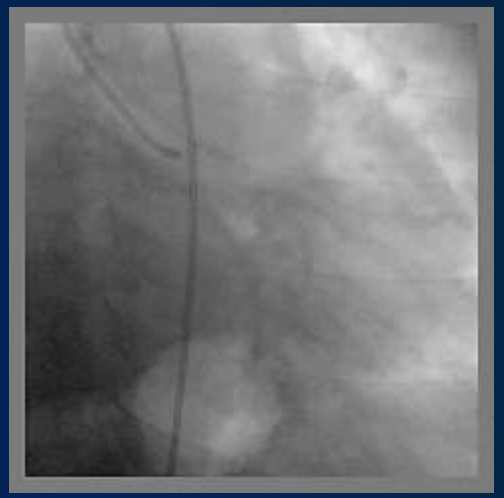
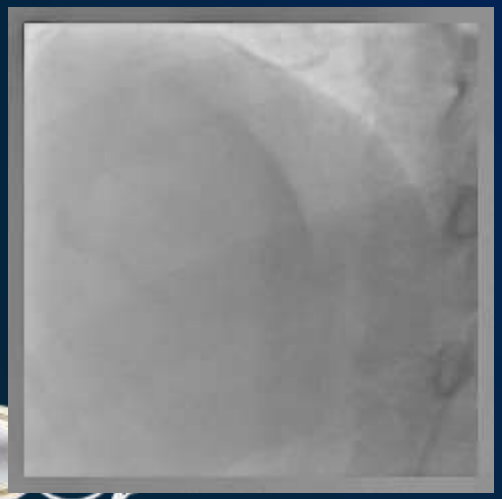
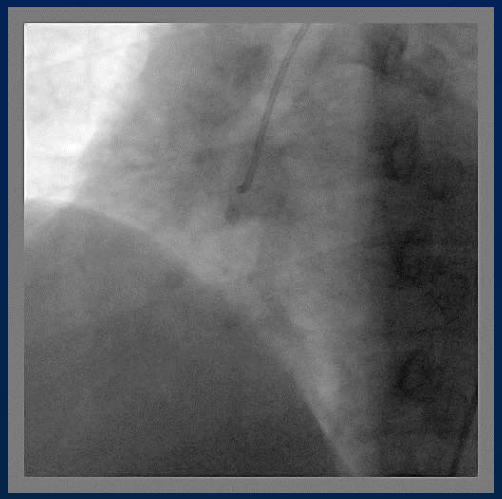
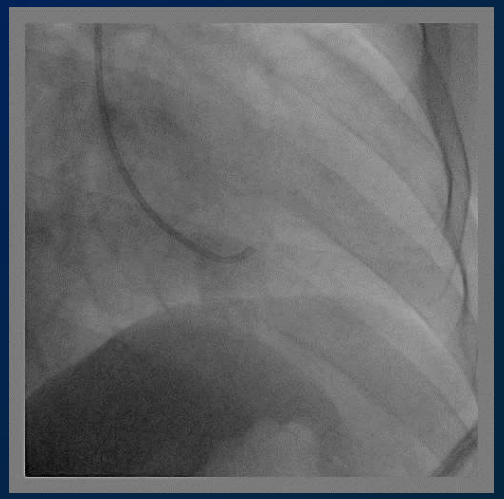
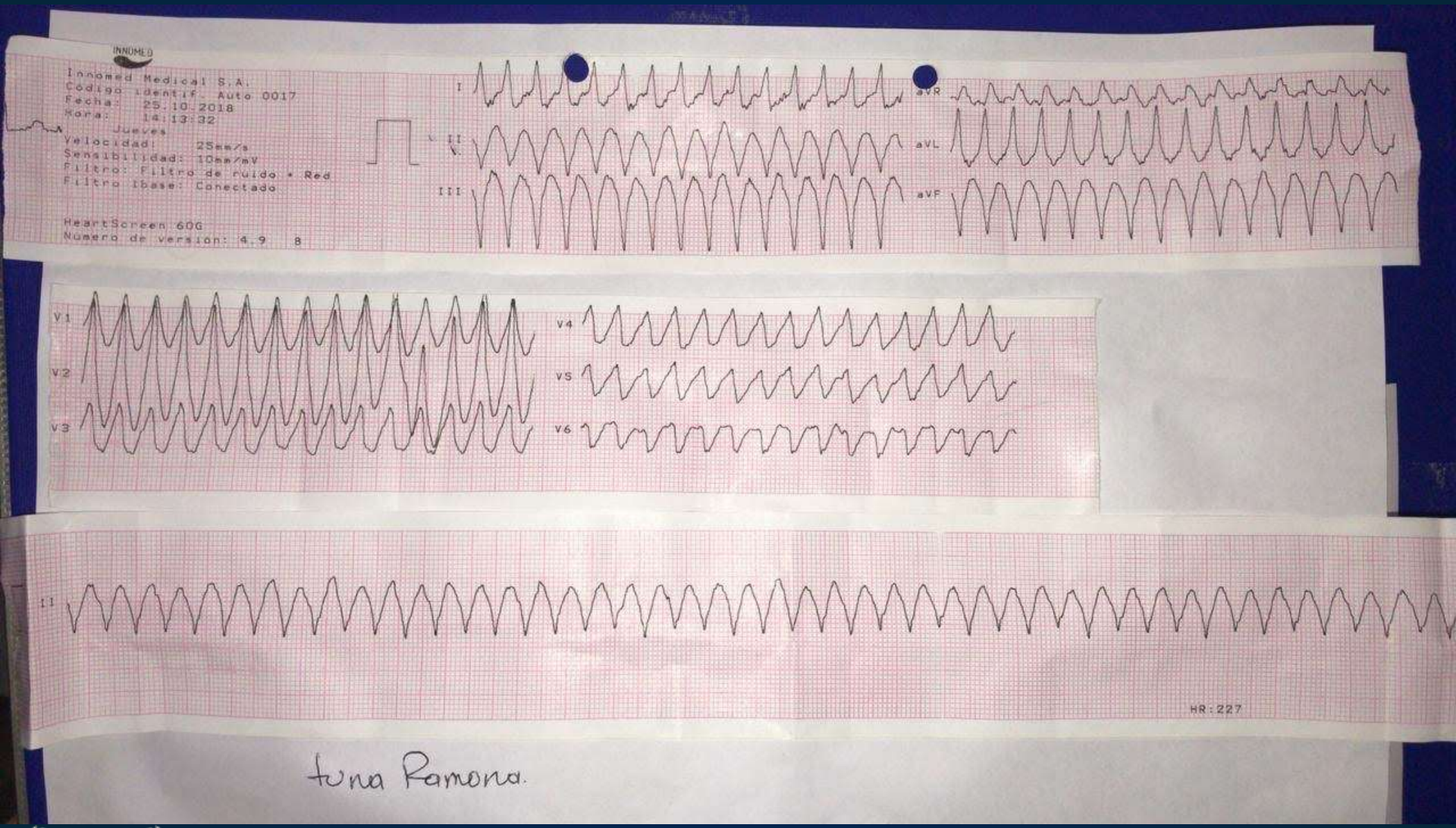


FIGURE 4. Corrected TIMI frame counts before and after intracoronary nicardipine by vascular territory. Significant improvement in TIMI frame count after nicardipine was seen for all vascular territories (all *P*<.001). LAD = left anterior descending; LCx = left circumflex; RCA = right coronary artery.

No Reference to Clinical Improvement or AMI

Angiography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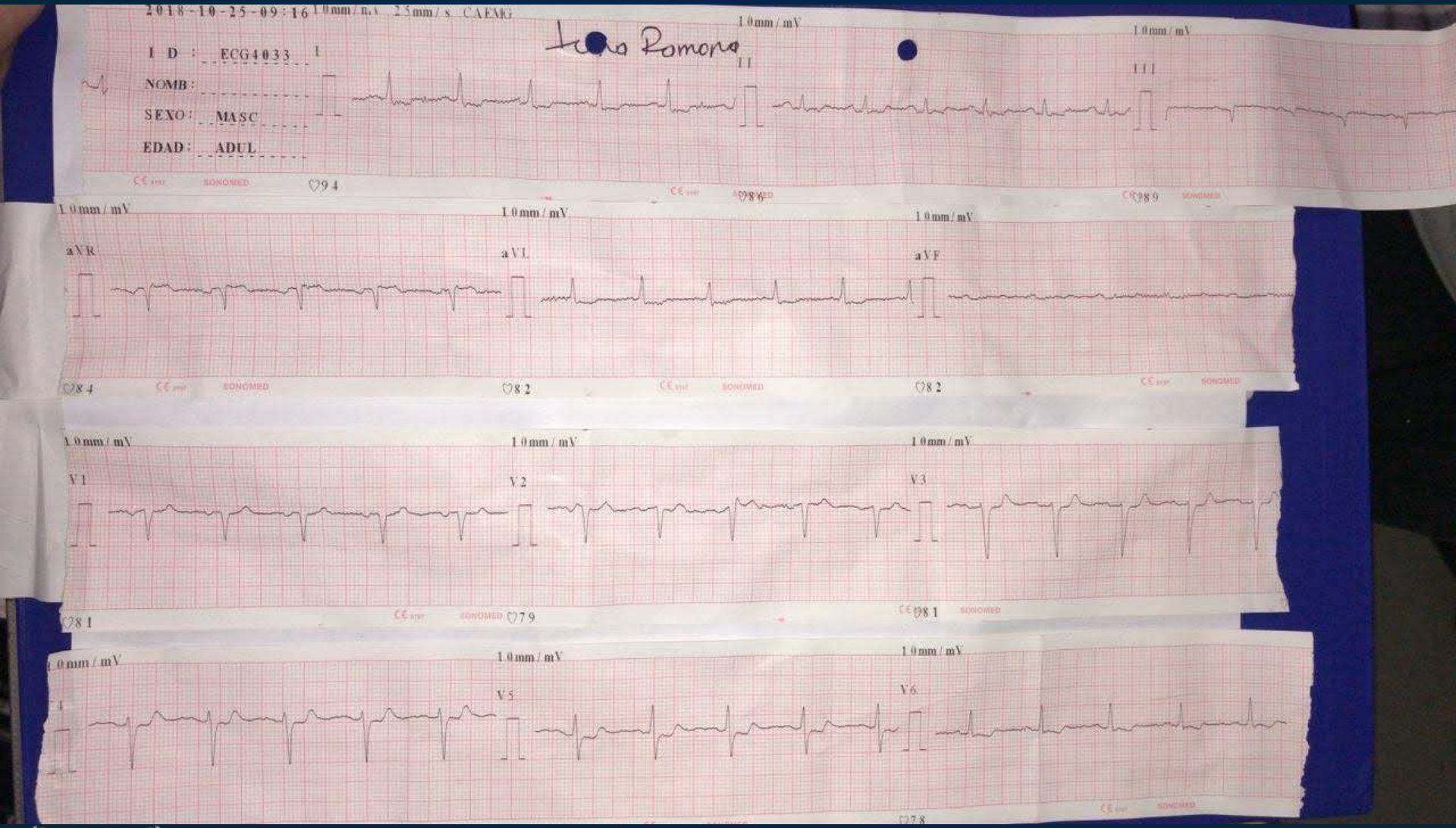
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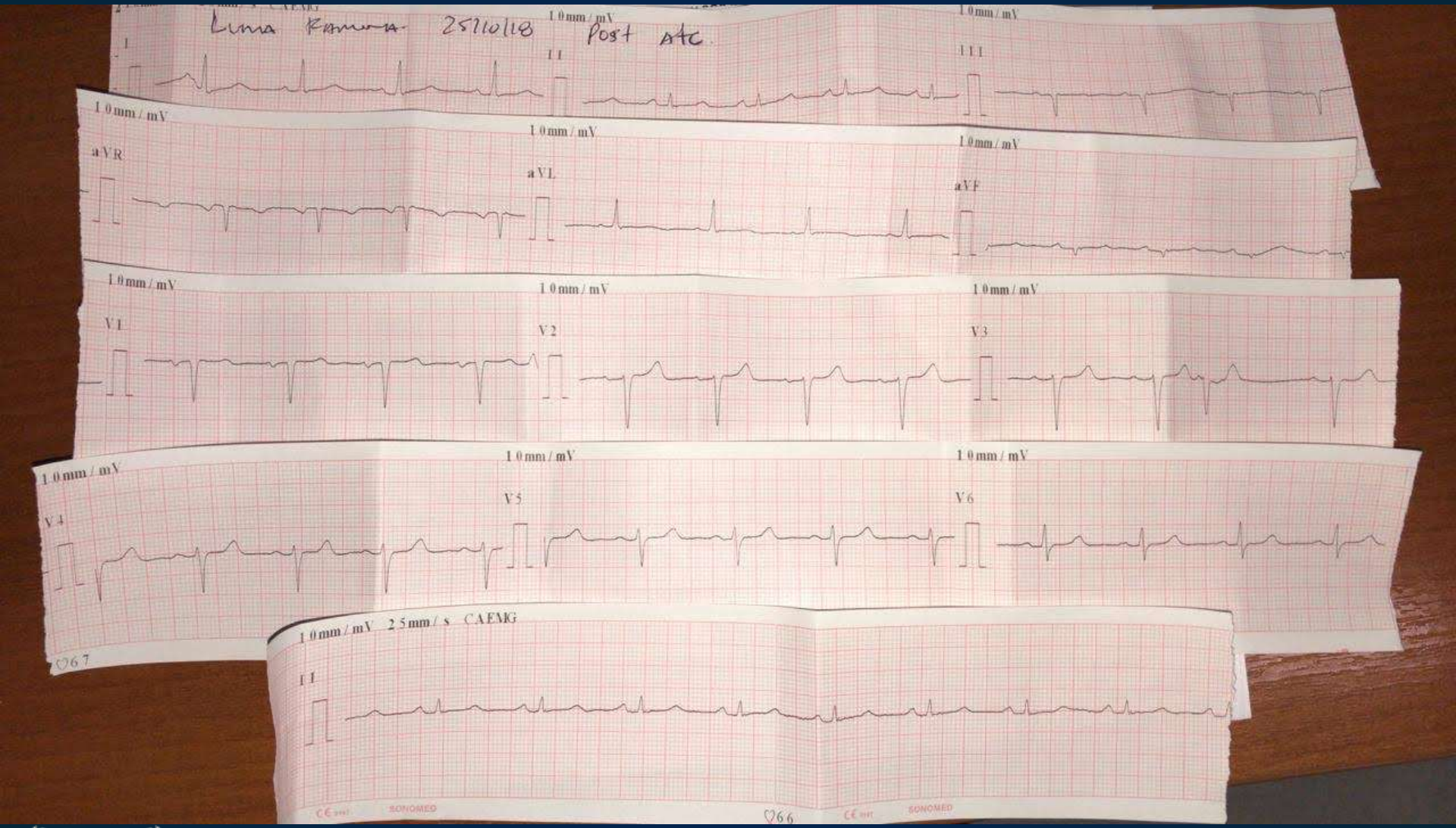
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