APPORT DE L'IRM CARDIAQUE DANS LE MINOCA

Dr Jérôme ADDA

IMAGERIESUD NICE 07/04/2026

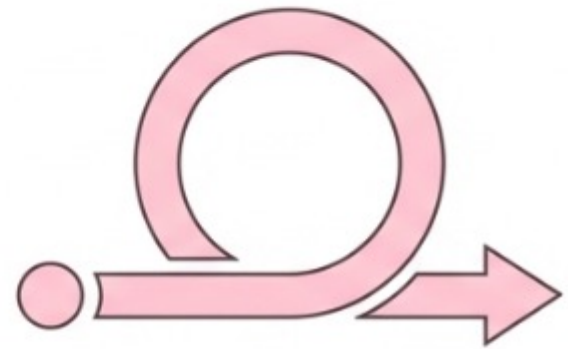
CENTRE HOSPITALIER BÉZIERS

Jérôme ADDA
MD, PhD, Cardiologist, Cardiac Imaging

Sans radiation

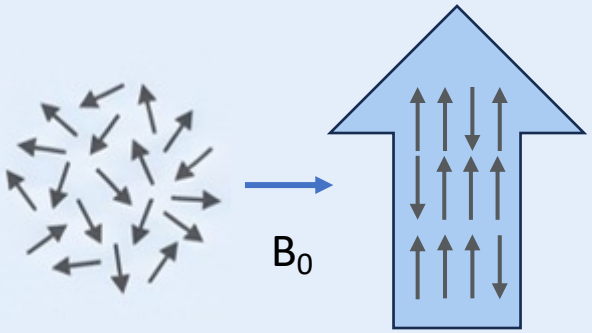
Haute précision

Évaluation combinée de la morphologie et de la fonction



Suivi longitudinal

Idéal pour évaluer la progression de la maladie et l'effet des traitements

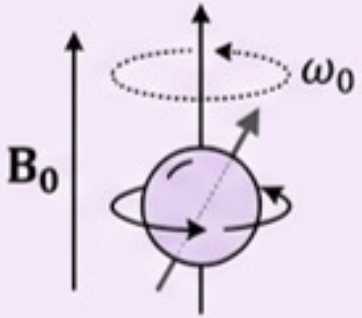


Méthode d'imagerie basée sur propriétés proton hydrogène (eau, graisse...)

Protons possèdent un moment cinétique, et un moment magnétique (petits aimants)

Les moments magnétiques s'alignent dans un champ magnétique puissant B_0 (0.5 à 3T), résultant en une

Aimantation macroscopique nette M_0

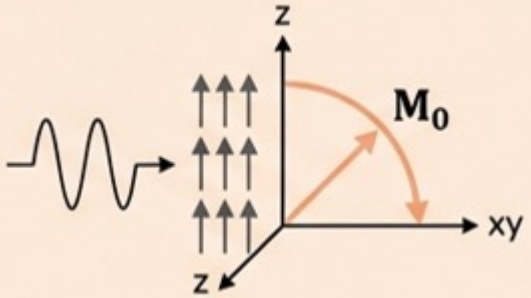


Ces moments magnétiques tournent autour de leur axe sous l'effet de B0, à une fréquence connue (**Fréquence de Larmor ω_0**)

$$\omega_0 = \gamma B_0$$

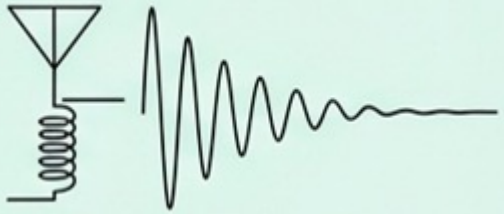
γ : rapport gyromagnétique

Toute l'IRM repose sur cette fréquence.



On applique une **onde de radiofréquence** à la fréquence de Larmor → transfert d'énergie

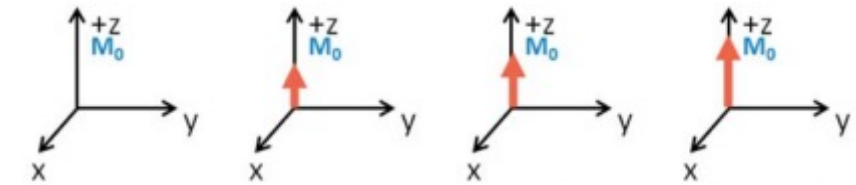
L'aimantation bascule du plan longitudinal (z) au plans transverse (xy).

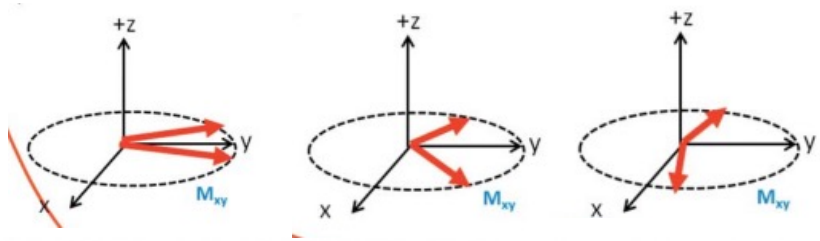


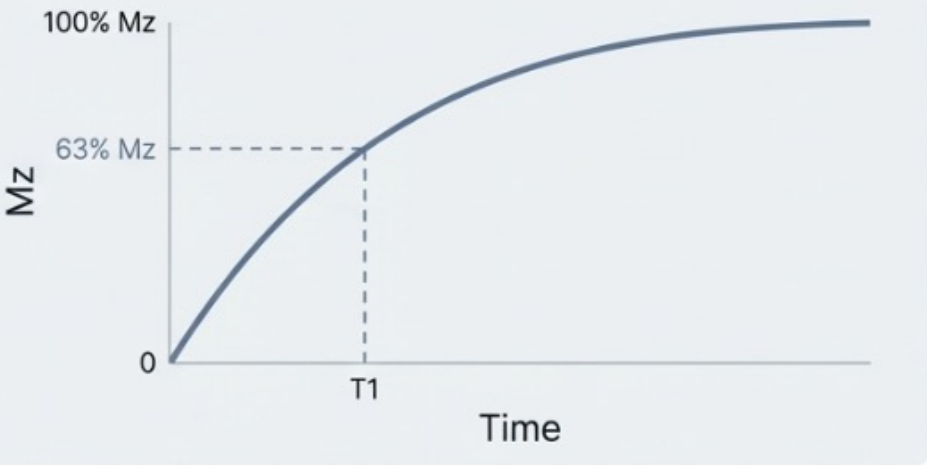
Une fois l'impulsion RF arrêtée, l'aimantation transverse précesse, et une aimantation en rotation induit un courant dans les antennes.

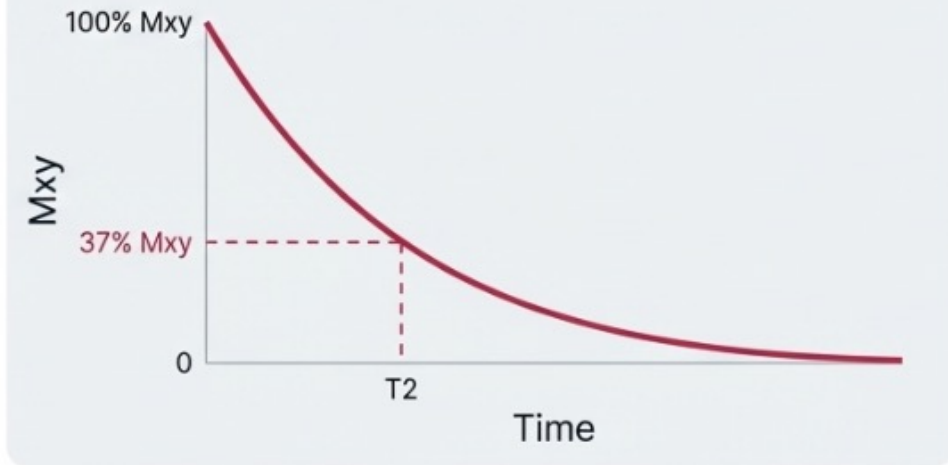
C'est le signal IRM

Ce signal décroit rapidement, c'est la relaxation.



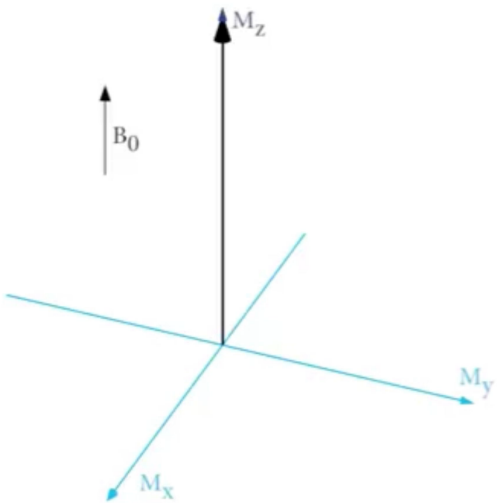


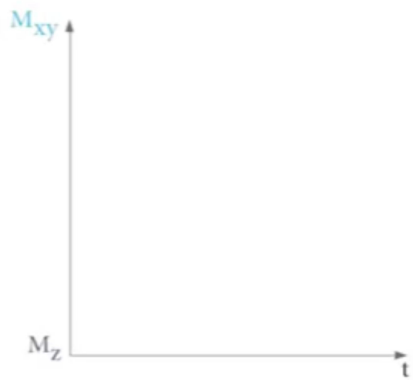


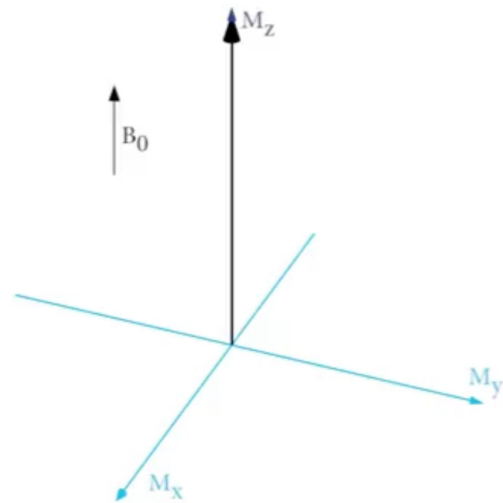


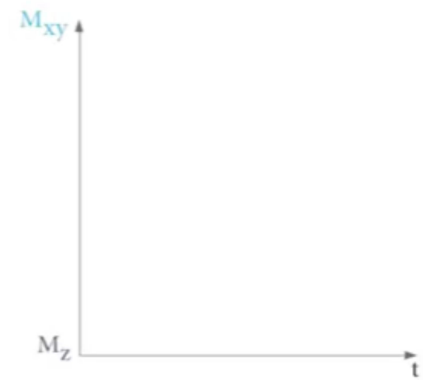
T1 (Relaxation Longitudinale)	T2 (Relaxation Transversale)
• Mécanisme : Interaction Spin-Réseau (échange d'énergie avec le milieu). • Mesure : Temps de repousse de Mz à 63%.	• Mécanisme : Interaction Spin-Spin (déphasage entre protons). • Mesure : Temps de déclin de Mxy à 37%.

Utilité Clinique : La différence intrinsèque des temps T1 et T2 entre les tissus est la source primaire du contraste en IRM.


M_z
B_0
M_y
M_x

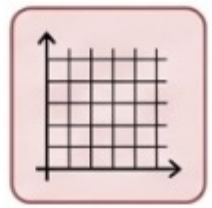

M_{xy}
M_z
t


M_z
B_0
M_y
M_x


M_{xy}
M_z
t

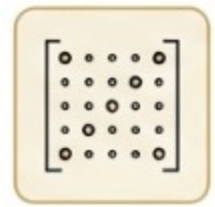
Excitation Sélective

Impulsion RF + Gradient de coupe (Gss). Isole la tranche d'intérêt.



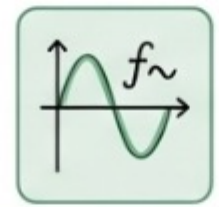
Encodage Spatial

Gradients de phase (Gpe) et de fréquence (Gfe). Assigne des coordonnées spatiales.



Espace K

Remplissage matriciel des lignes de données spatiales brutes.



Transformée de Fourier

Conversion mathématique 2D du domaine fréquentiel au domaine spatial.

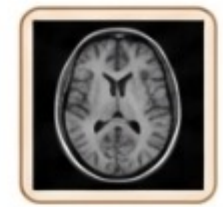
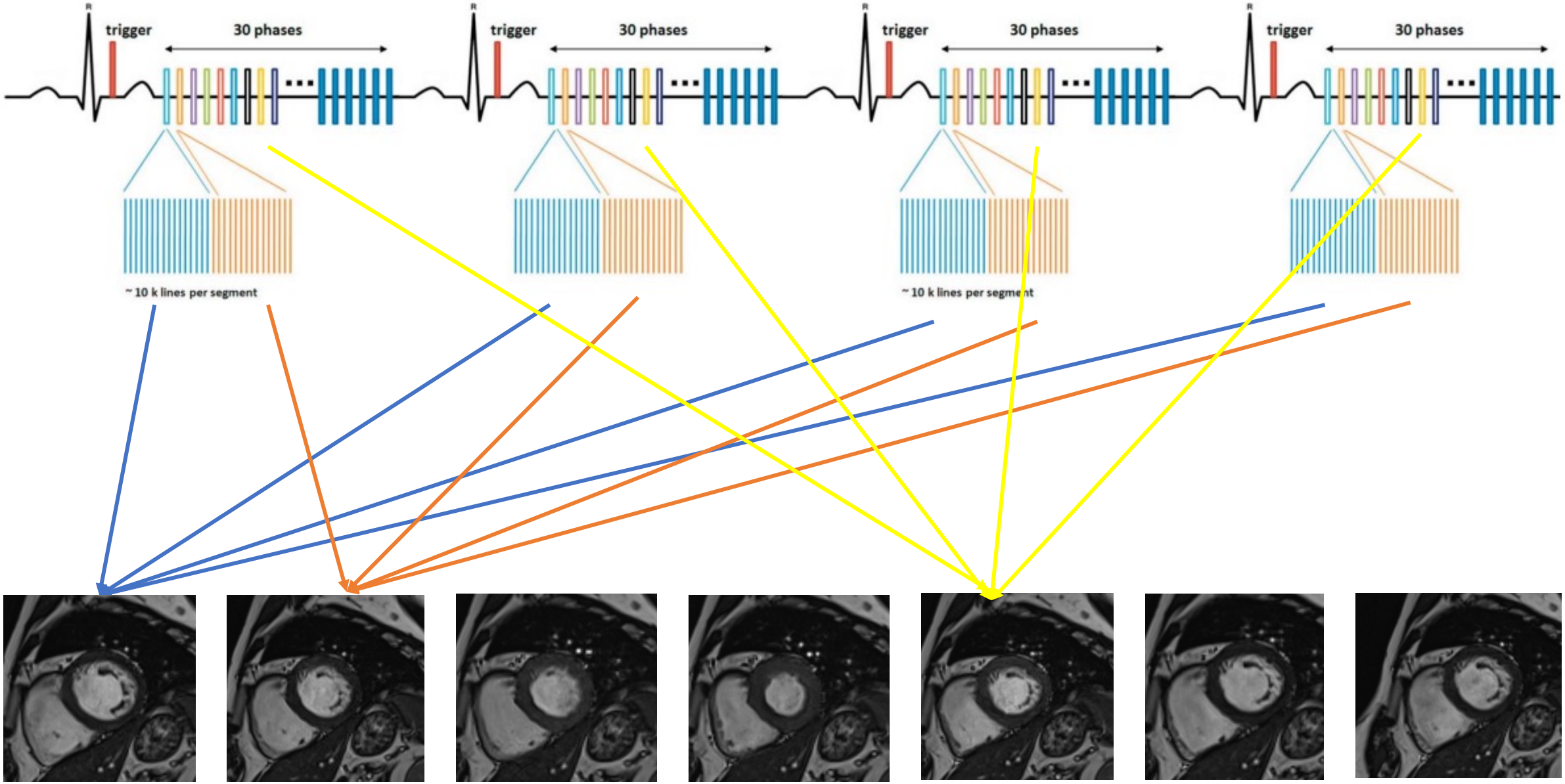


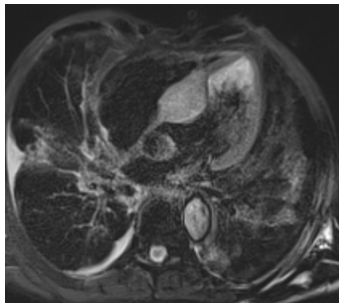
Image Finale

Reconstruction du résultat clinique.


trigger
30 phases
trigger
30 phases
trigger
30 phases
trigger
30 phases
~ 10 k lines per segment
~ 10 k lines per segment

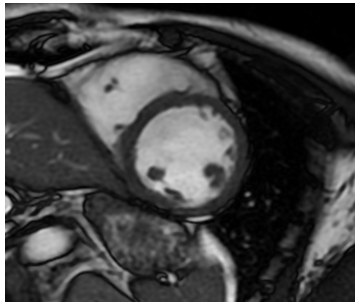
Morphologie

Anatomie statique et détection de masses tissulaires.



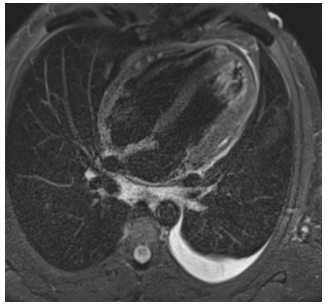
Fonction

Cinétique pariétale et volumes ventriculaires (Cine).



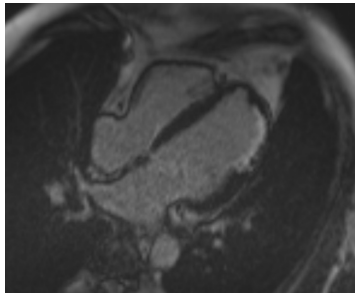
Œdème

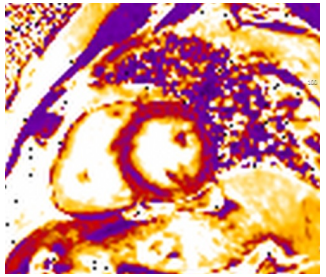
Contenu en eau
Séquences STIR et T2 mapping



Tissu

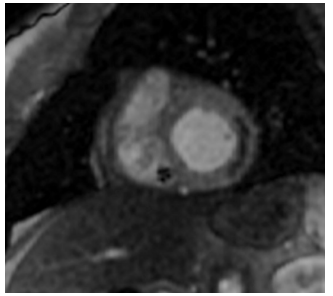
Expansion MEC
Rehaussement tardif, T1 mapping et VEC

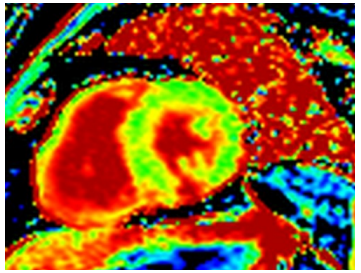




Perfusion

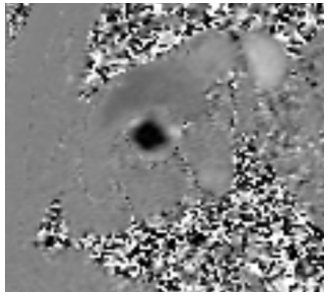
Évaluation de la microcirculation et détection de l'ischémie.

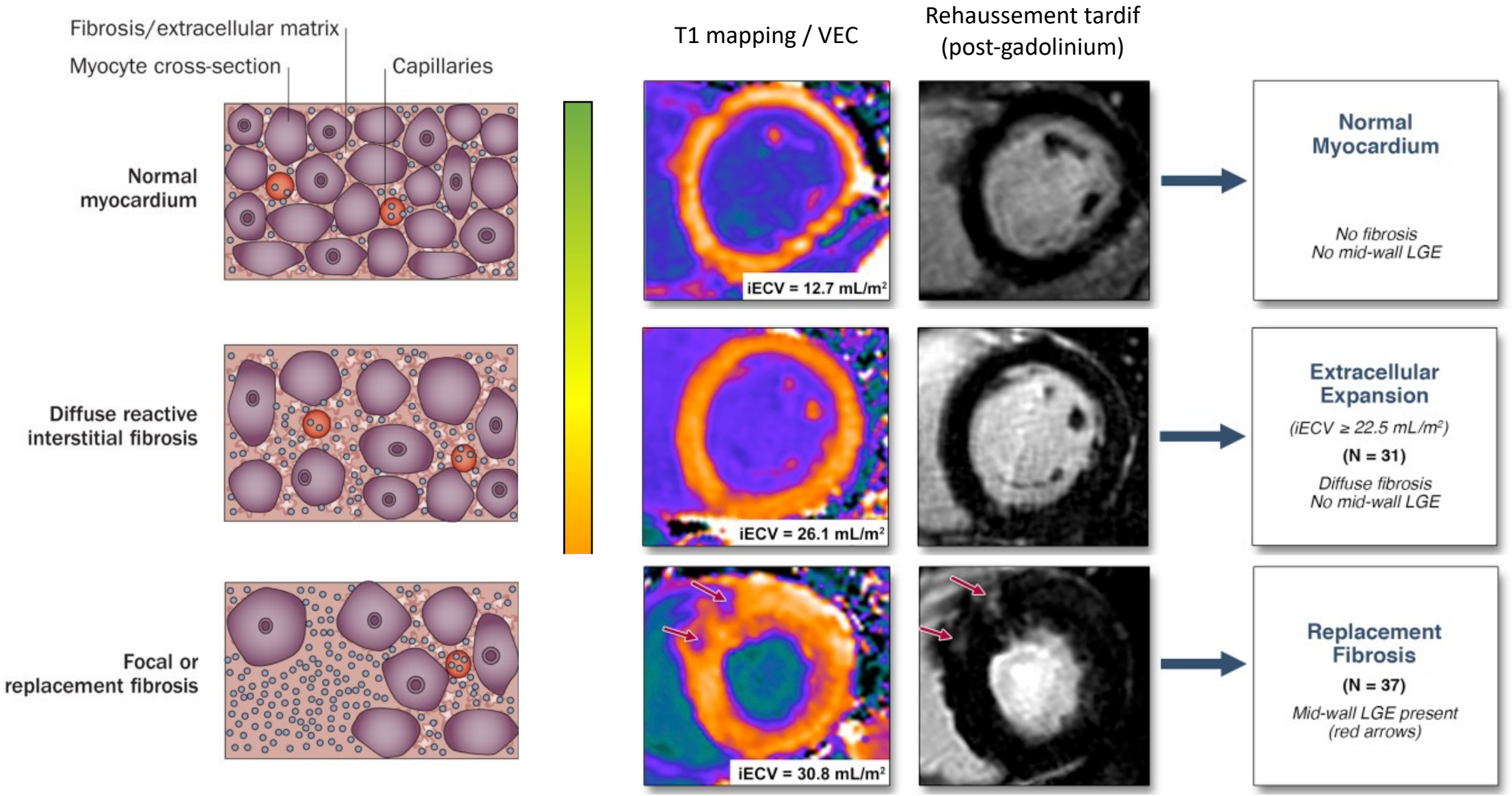




Flux

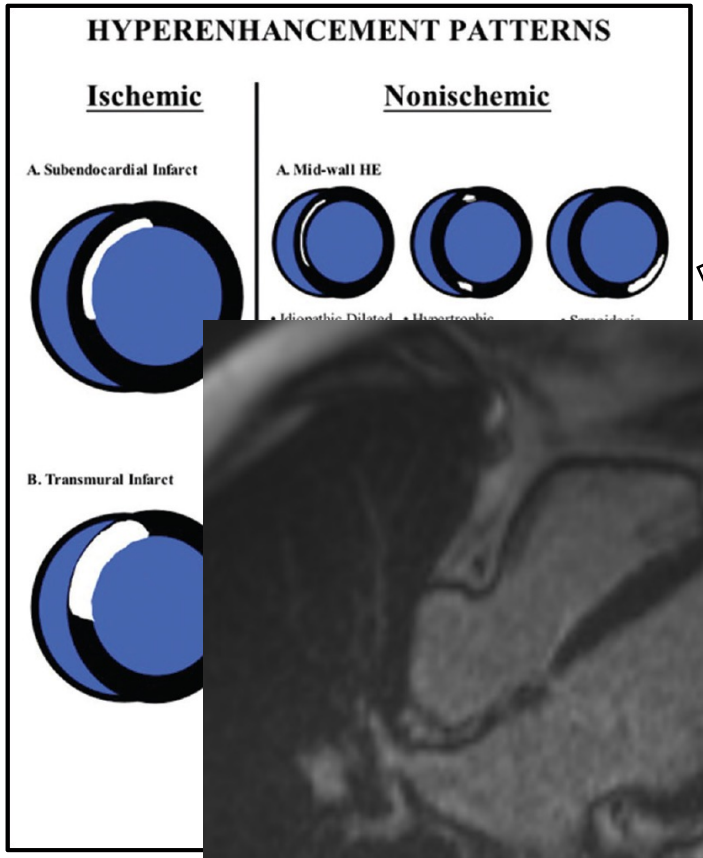
Cartographie des vitesses et hémodynamique globale.

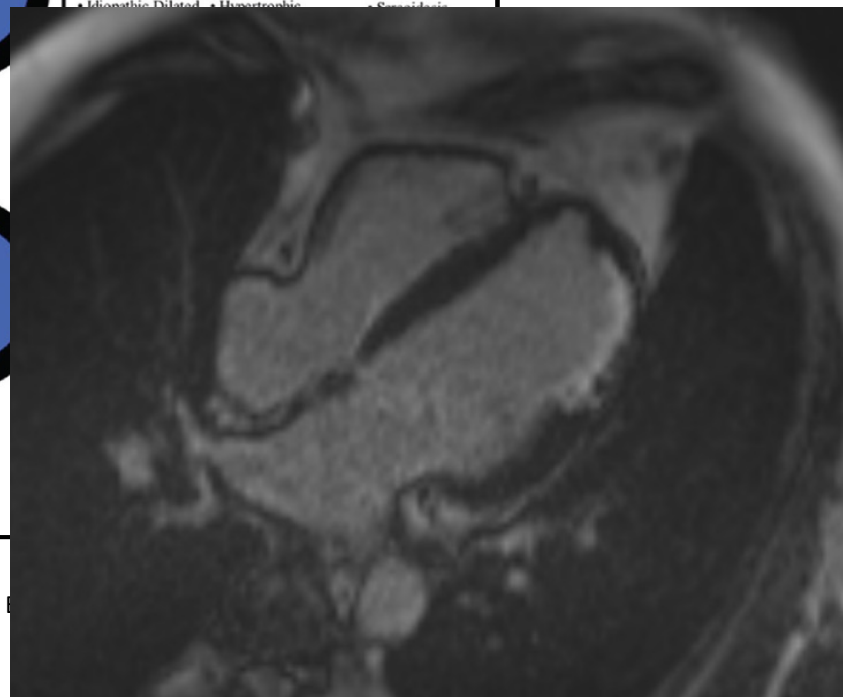




FIBROSE REHAUSSEMENT TARDIF
MUSCLE NORMAL = NOIR FIBROSE = BLANC

2005

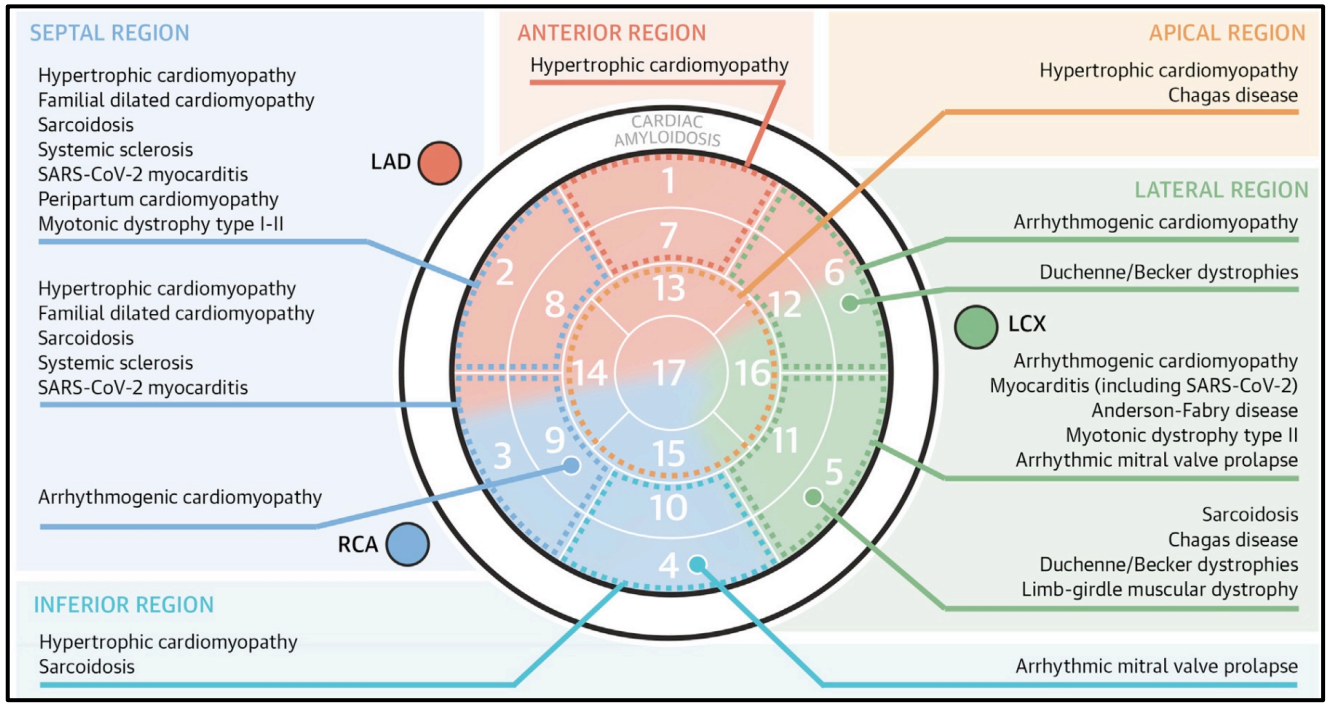


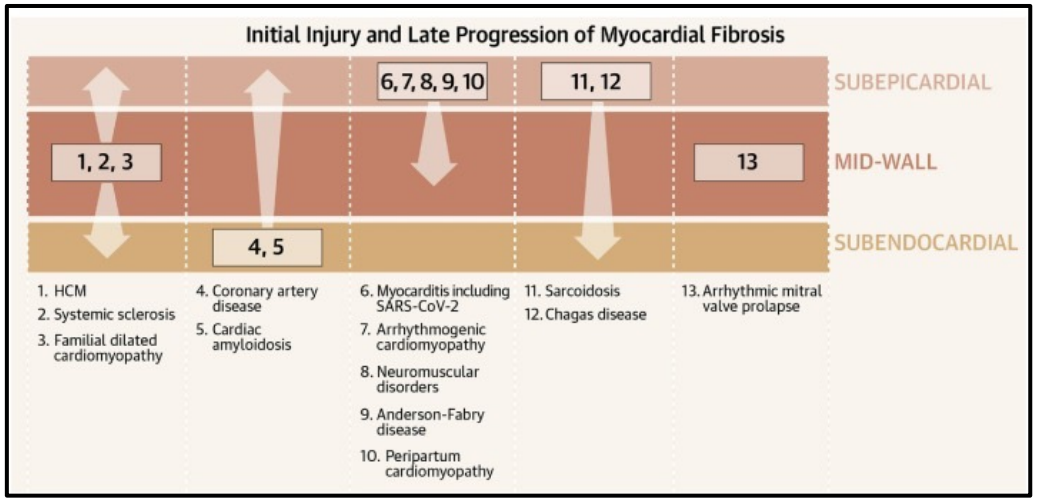


Mahrholdt et al, E

Sous-endocardique ou transmural = ischémique

2024





Del Franco et al., J Am Coll Cardiol Advances 2024;3(10):101214

MINOCA

MI

MYOCARDIAL
INFARCTION

NO

NO
OBSTRUCTIVE

CA

CORONARY
ARTERY

2017

ESC working group position paper on myocardial infarction with non-obstructive coronary arteries

Stefan Agewall[1]*, John F. Beltrame[2], Harmony R. Reynolds[3], Alexander Niessner[4], Giuseppe Rosano[5,6], Alida L. P. Caforio[7], Raffaele De Caterina[8], Marco Zimarino[8], Marco Roffi[9], Keld Kjeldsen[10], Dan Atar[1], Juan C. Kaski[6], Udo Sechtem[11], and Per Tornvall[12], on behalf of the WG on Cardiovascular Pharmacotherapy

2020 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation

The diagnosis of MINOCA is made in patients with AMI fulfilling the following criteria:

1. AMI (modified from the 'Fourth Universal Definition of Myocardial Infarction' criteria):

- Detection of a rise or fall in cardiac troponin with at least one value above the 99th percentile upper reference limit and
- Corroborative clinical evidence of infarction as shown by at least one of the following:
  a. Symptoms of myocardial ischaemia
  b. New ischaemic electrocardiographic changes
  c. Development of pathological Q waves
  d. Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in a pattern consistent with an ischaemic cause
  e. Identification of a coronary thrombus by angiography or autopsy

2. Non-obstructive coronary arteries on angiography:

- Defined as the absence of obstructive disease on angiography (i.e. no coronary artery stenosis ≥50%) in any major epicardial vessel[a]

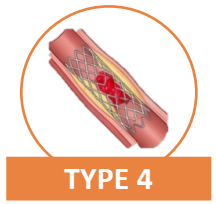
This includes patients with:

- Normal coronary arteries (no angiographic stenosis)
- Mild luminal irregularities (angiographic stenosis <30% stenoses)
- Moderate coronary atherosclerotic lesions (stenoses >30% but <50%)

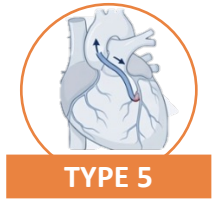
3. No specific alternate diagnosis for the clinical presentation:

- Alternate diagnoses include, but are not limited to, non-ischaemic causes such as sepsis, pulmonary embolism, and myocarditis

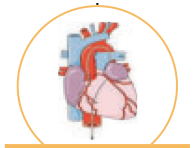
TYPE 3



TYPE 4



TYPE 5



ICA

OBSTRUCTIVE CAD

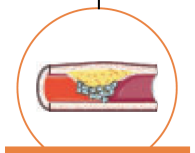
Follow Appropriate Management Guidelines

NON-OBSTRUCTIVE CAD

DIAGNOSTIC WORKUP (See Central Illustration)

ISCHEMIA

MINOCA



TYPE 1

TYPE 2

PLAQUE-INDUCED EVENTS

O_2 SUPPLY REDUCTION

O_2 SUPPLY-DEMAND IMBALANCE

Plaque Rupture

Plaque Erosion

Eruptive Nodule

Coronary Spasm

Coronary Embolism

Endothelial Dysfunction

SCAD

Severe Hypertension

Severe Anemia

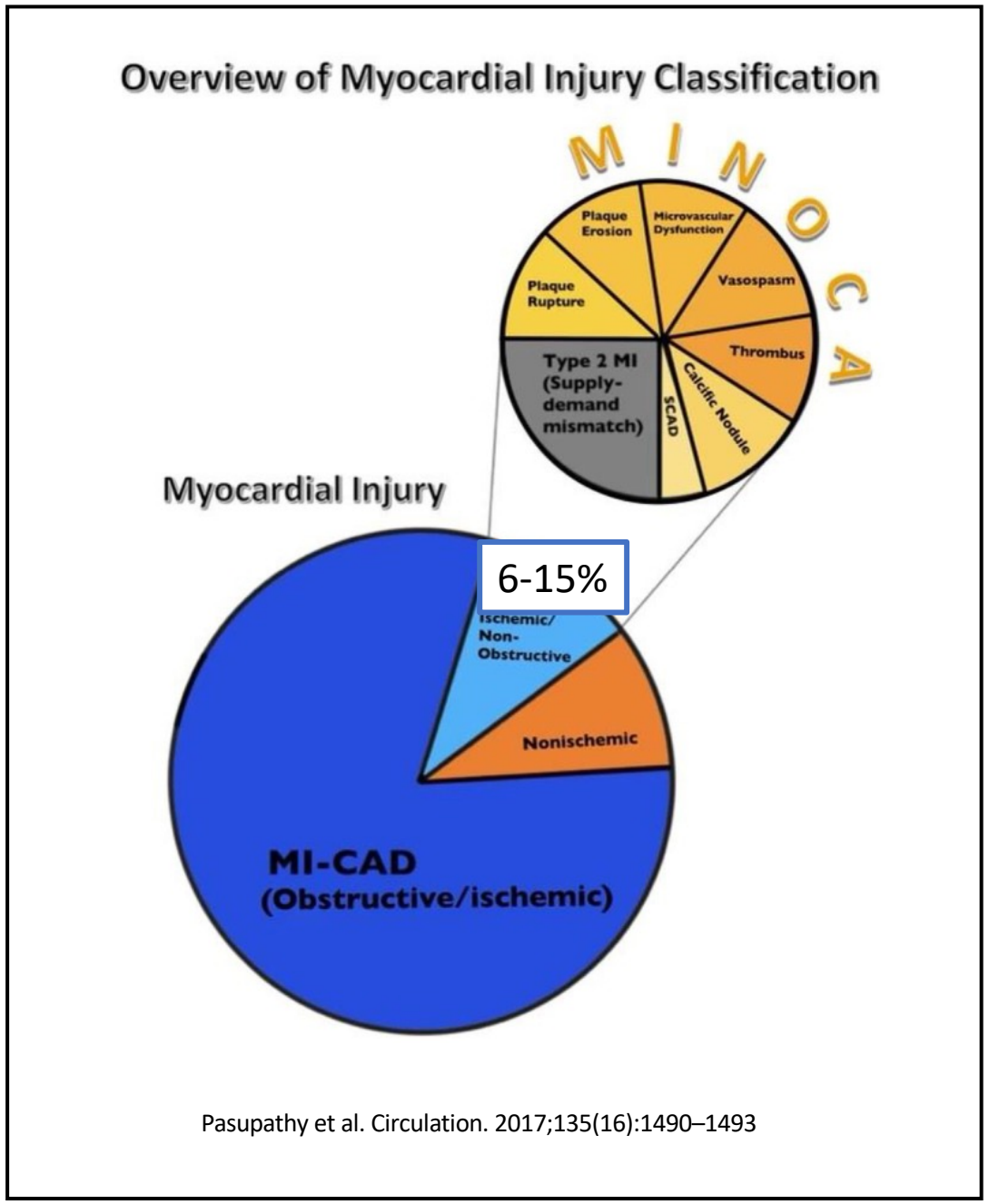
Bradyarrhythmias

Tachyarrhythmias

Severe Hypoxia - ARDS

Severe Hypotension

Occhipinti, Bucciarelli-Ducci, Capodanno. Eur Heart J Acute Cardiovasc Care. 2021 Oct 1;10(7):813-822.



Pasupathy et al. Circulation. 2017;135(16):1490–1493

Rapport F/H 1.3-2 : 1

Mortalité sans maladie CV : 2.2%
MINOCA : 4.6%
IDM avec obstruction coronaire : 14.3%

Barr et al., Heart Lung Circ. 2018; **27**:165-174

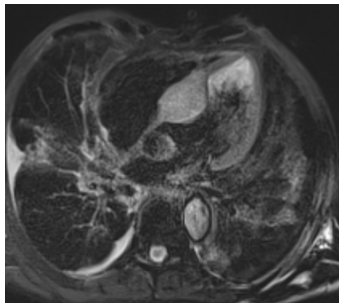
Mortalité doublée à 5 ans dans STEMI MINOCA vs STEMI

Quesada et al., JAMA Netw Open. 2023; **6**, e2343402

Le diagnostic de MINOCA est un diagnostic provisoire
(*working diagnosis*)

Morphologie

Anatomie statique et détection de masses tissulaires.



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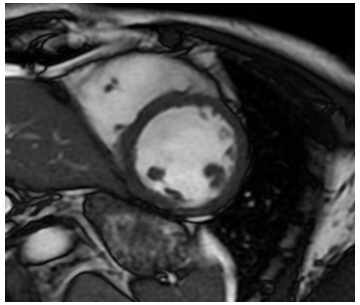

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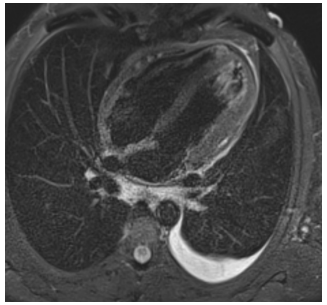

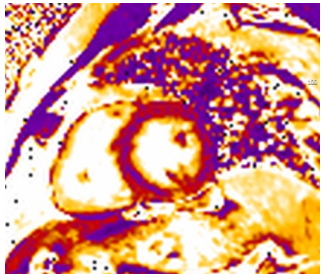

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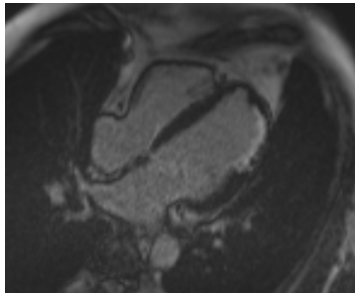

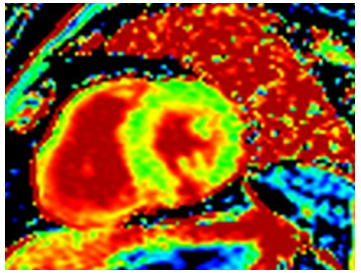

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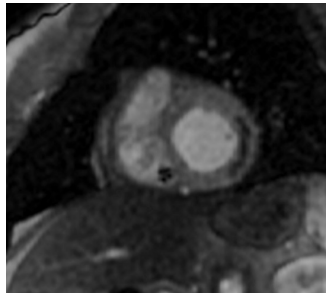

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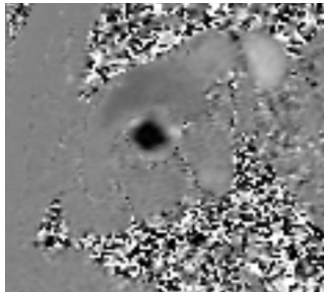


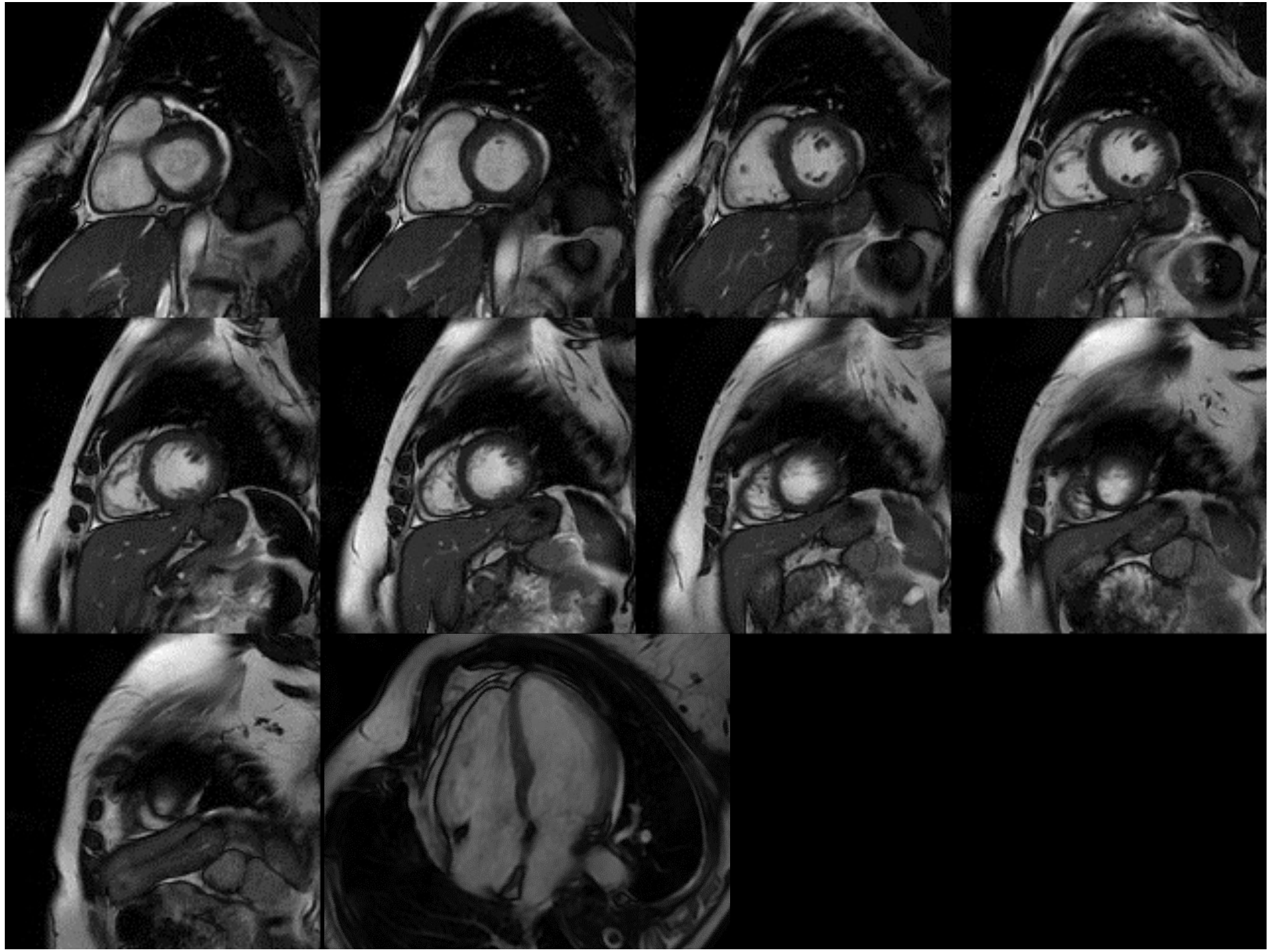
CAS CLINIQUE 1

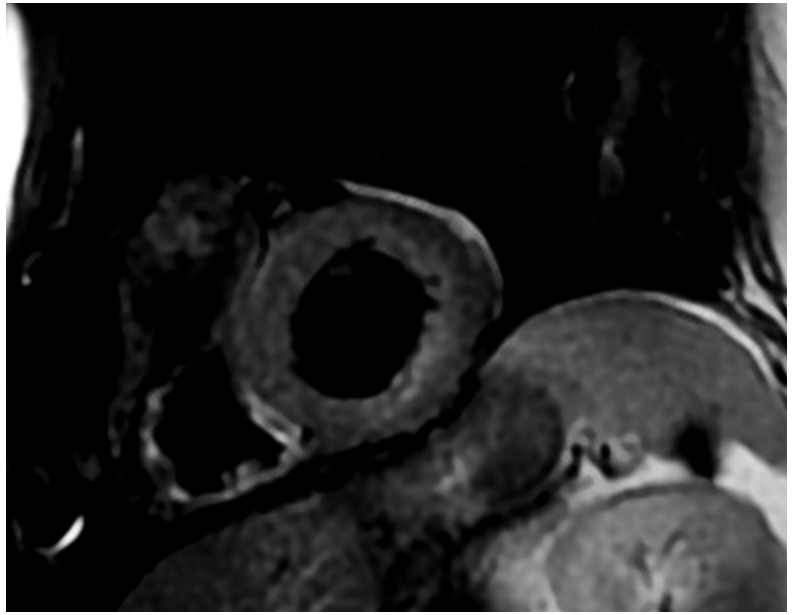
Patiente de 62 ans

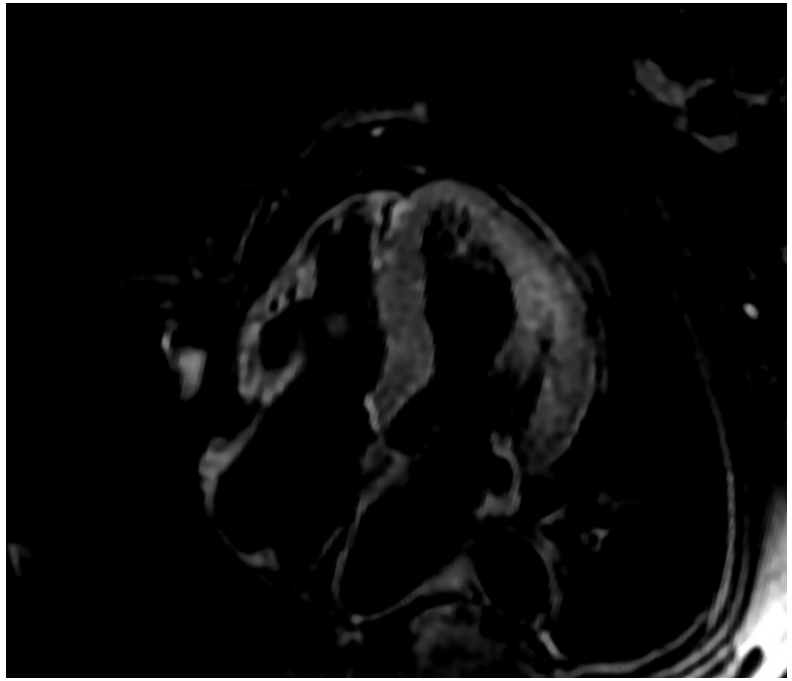
Douleur thoracique avec élévation de troponine

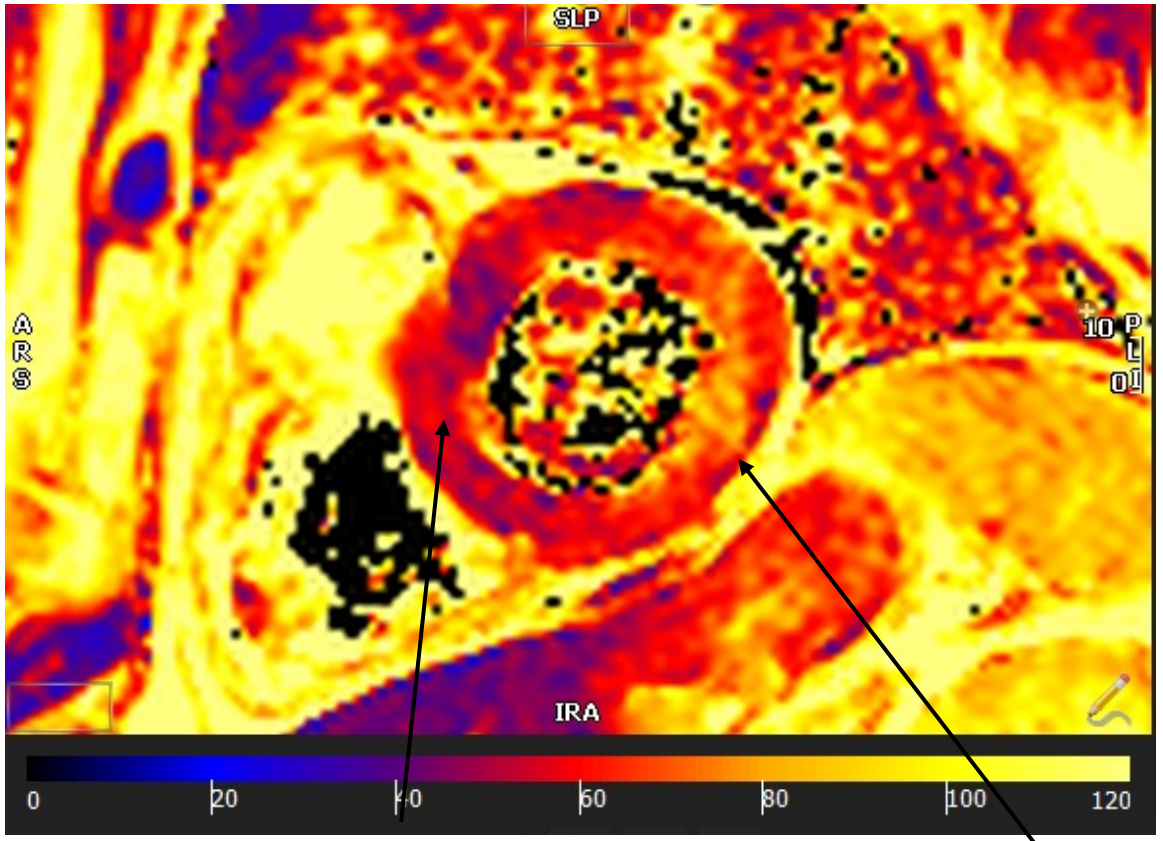
ECG normal

Coronarographie normale



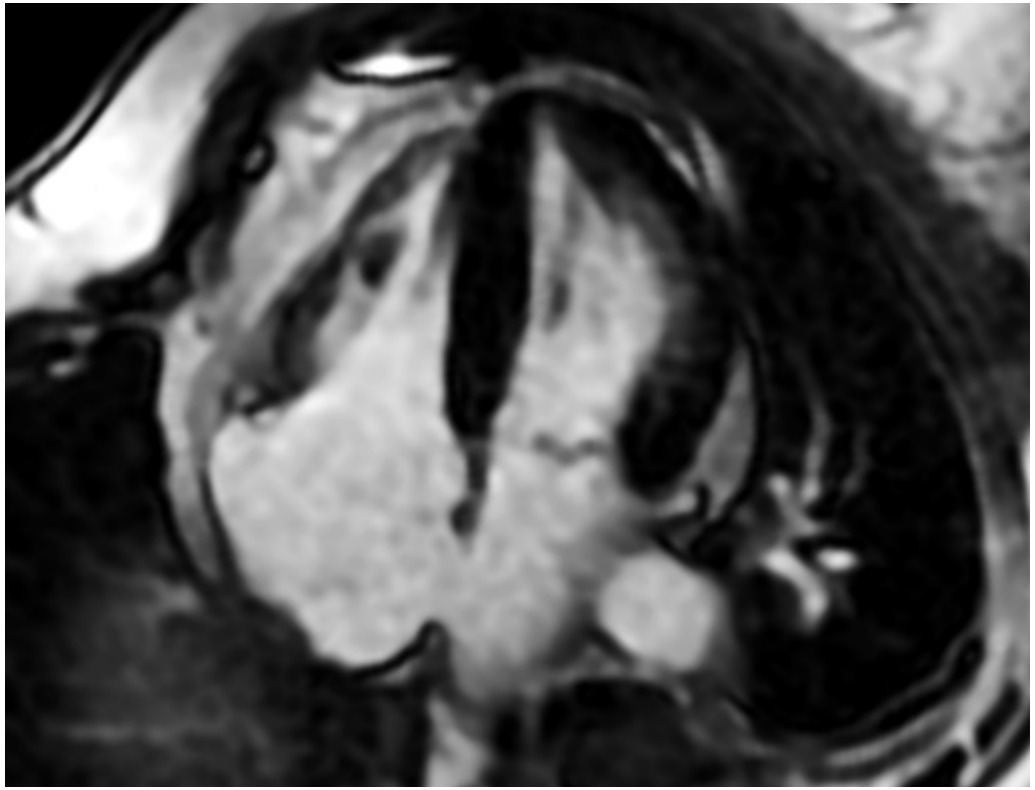


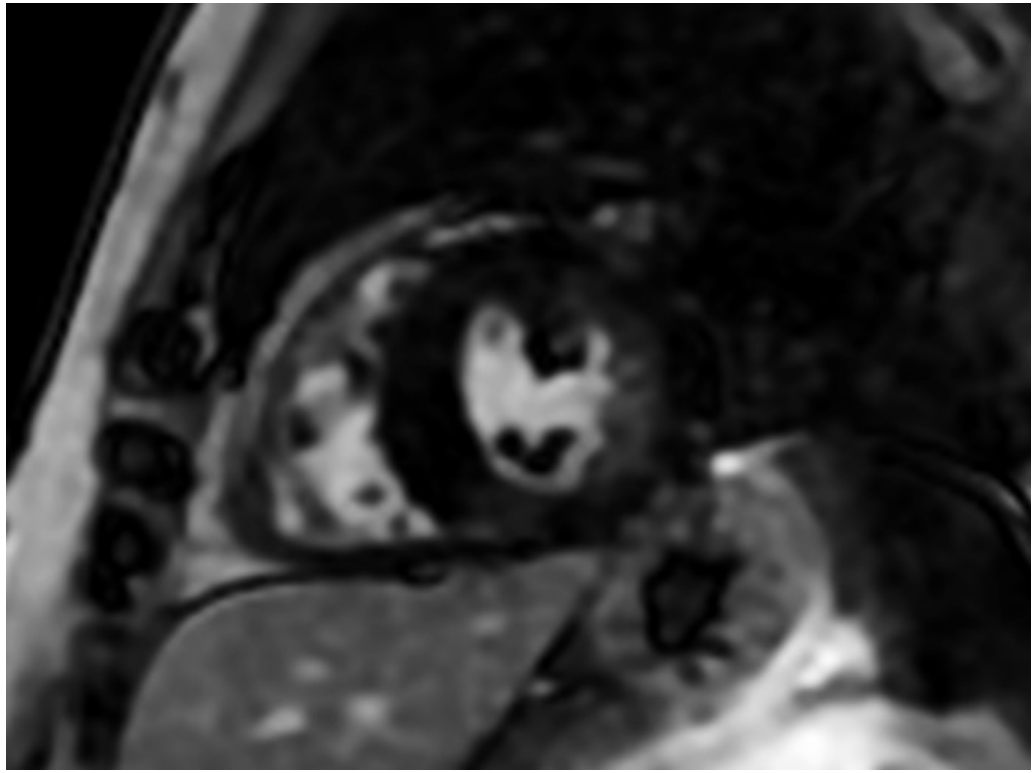




52 ms

70 ms



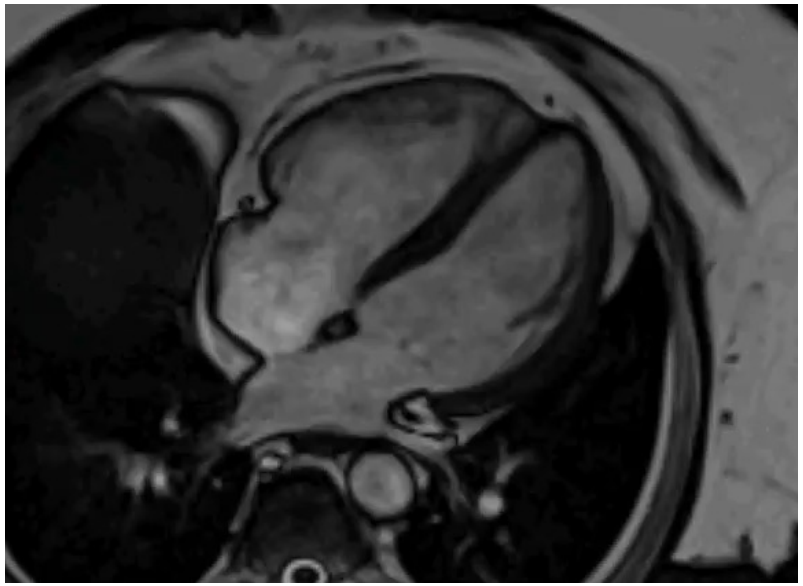


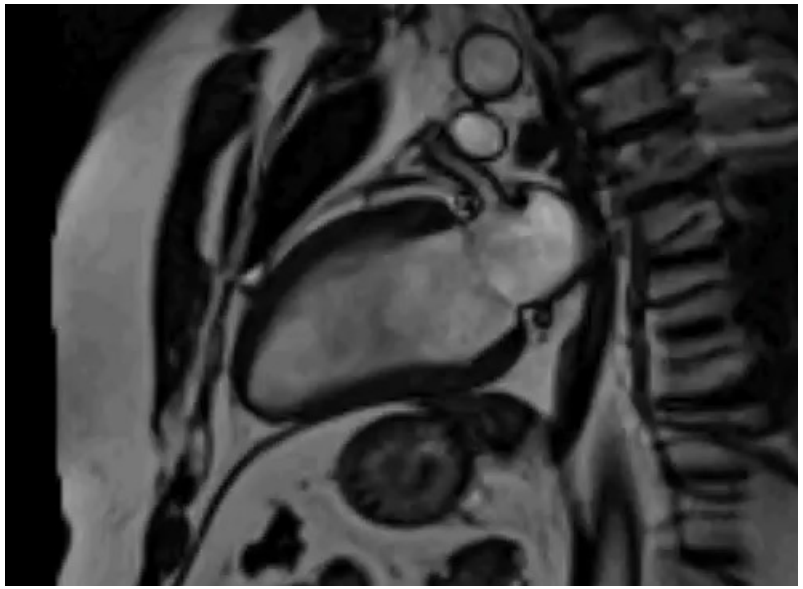
REHAUSSEMENT SOUS-ENDOCARDIQUE = ISCHEMIQUE

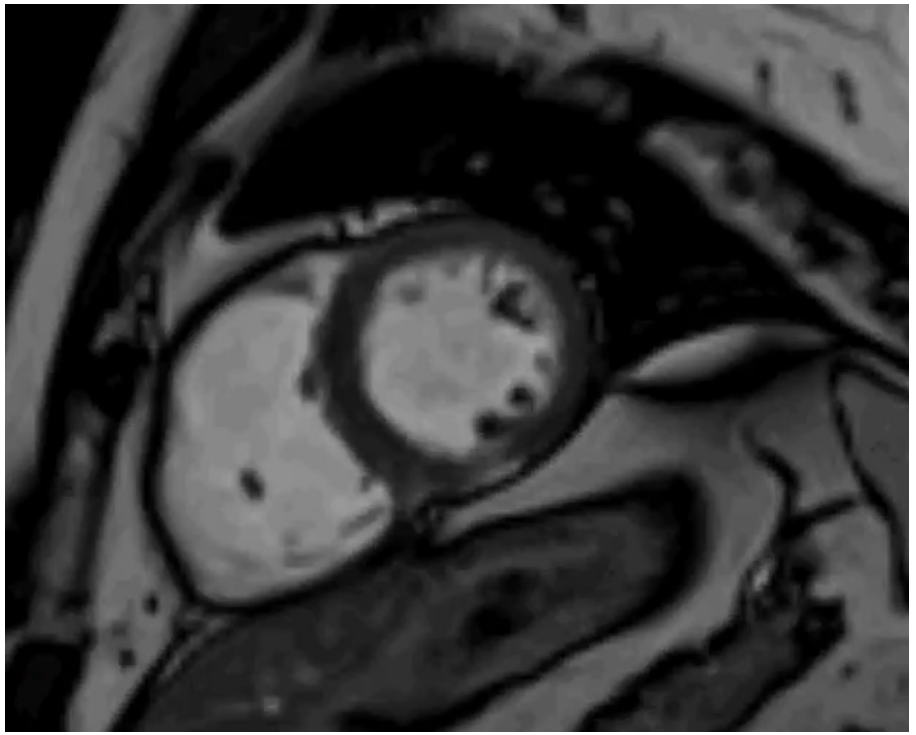
MINOCA ✓

CAS CLINIQUE 2

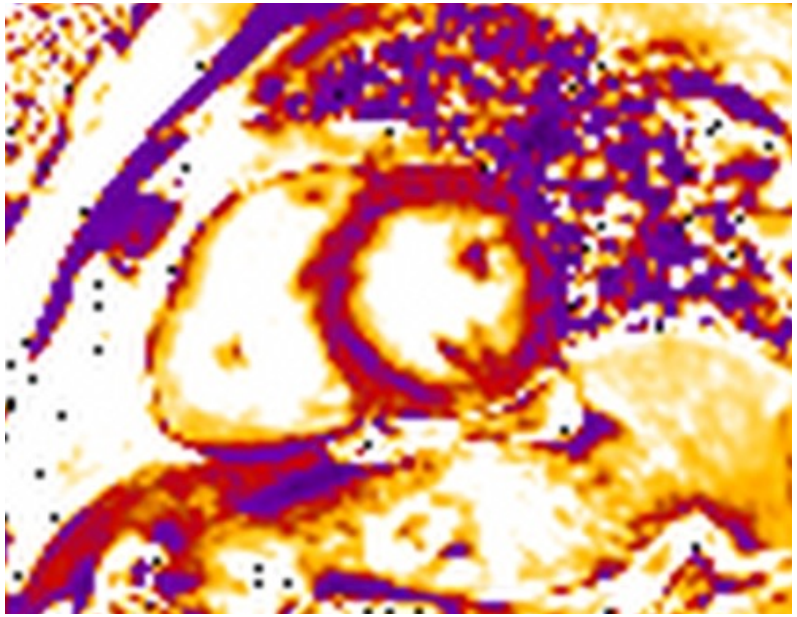
51 ans douleur thoracique
164 ng/l troponine
Coronarographie normale



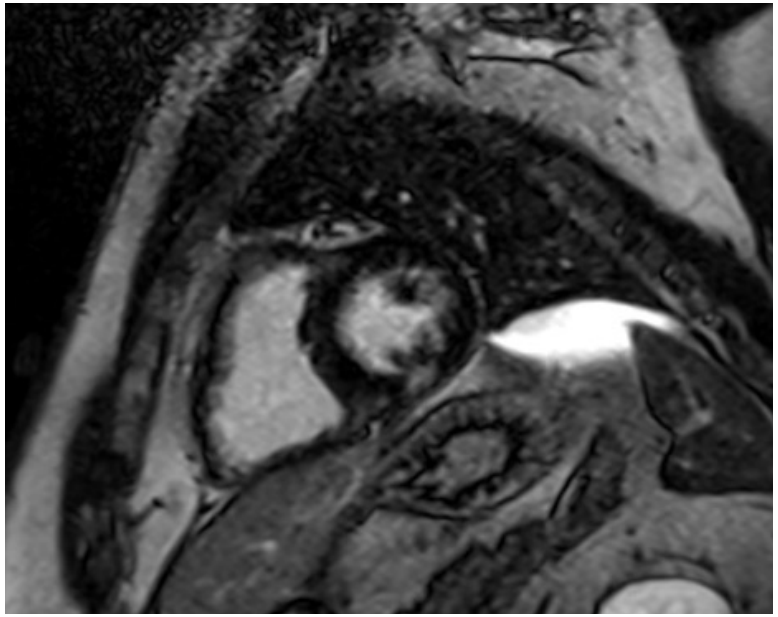


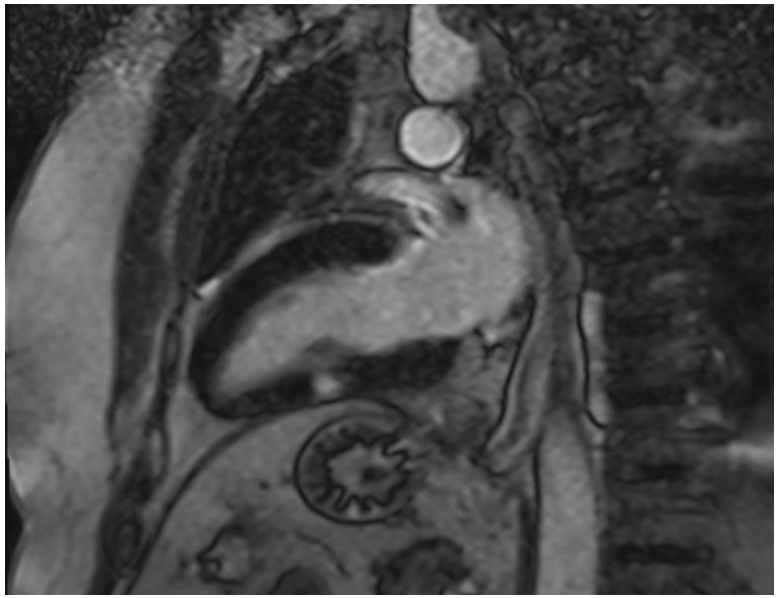


T2 mapping

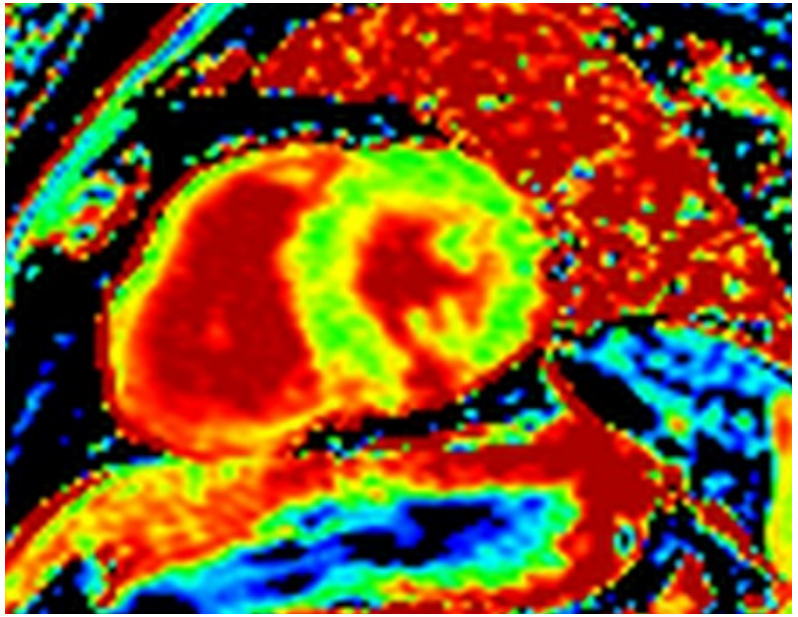


LGE





ECV mapping

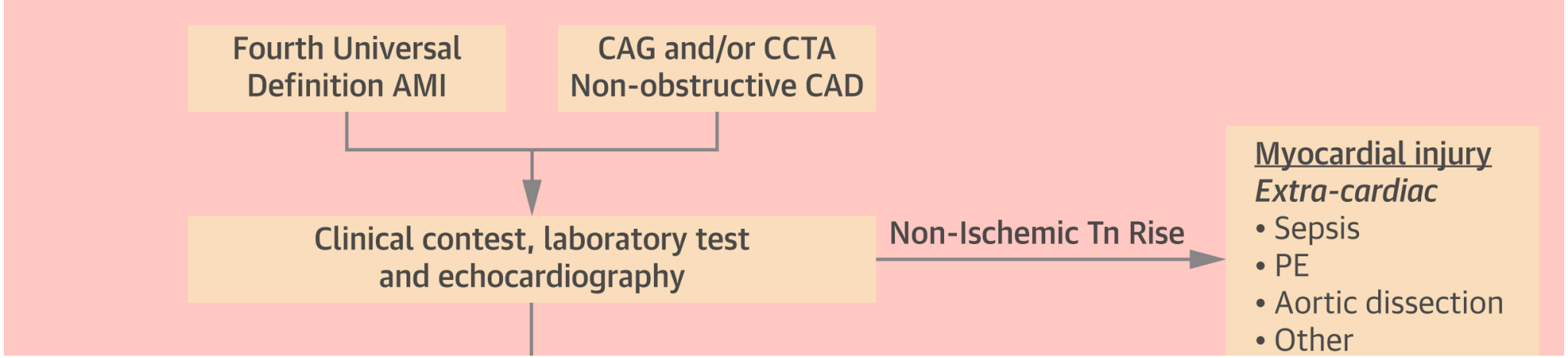


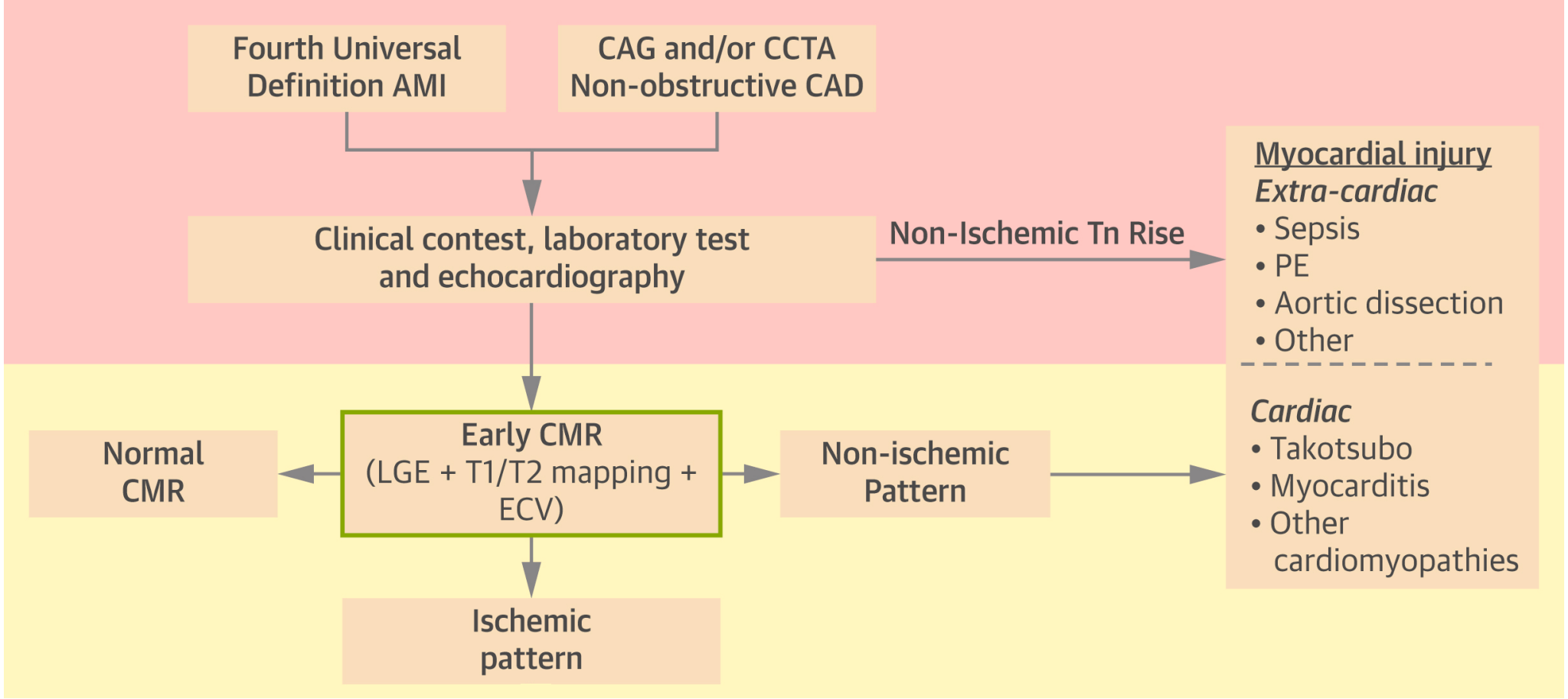
REHAUSSEMENT TRASMURAL

=

ISCHEMIQUE

MINOCA ✓


Fourth Universal Definition AMI
CAG and/or CCTA Non-obstructive CAD
Clinical contest, laboratory test and echocardiography
Non-Ischemic Tn Rise
Myocardial injury
Extra-cardiac
• Sepsis
• PE
• Aortic dissection
• Other


Fourth Universal Definition AMI
CAG and/or CCTA Non-obstructive CAD
Clinical contest, laboratory test and echocardiography
Non-Ischemic Tn Rise
Myocardial injury
Extra-cardiac
• Sepsis
• PE
• Aortic dissection
• Other
Cardiac
• Takotsubo
• Myocarditis
• Other cardiomyopathies
Normal CMR
Early CMR (LGE + T1/T2 mapping + ECV)
Non-ischemic Pattern
Ischemic pattern

CAS CLINIQUE 3

Homme de 47 ans

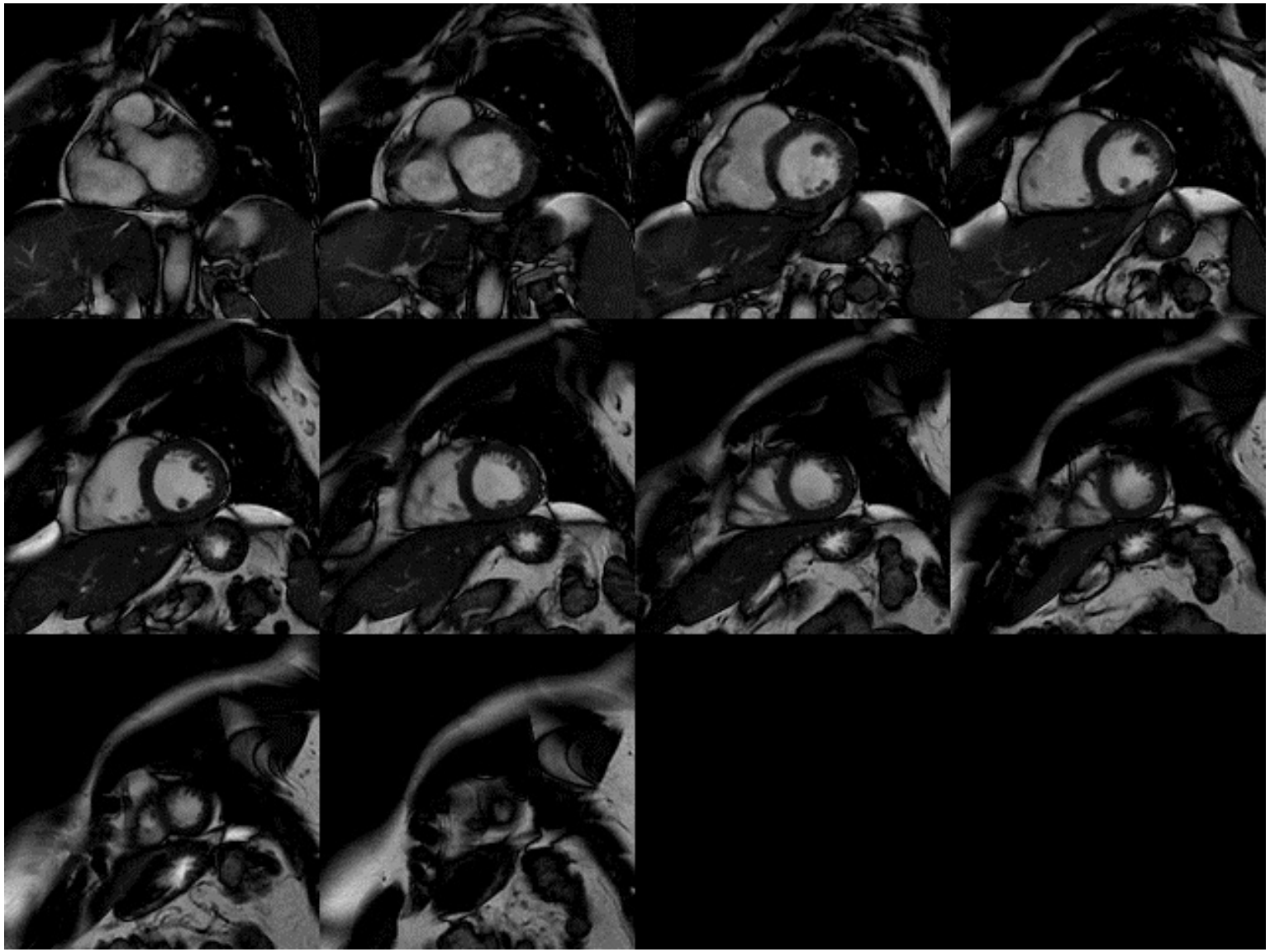
Pas de Facteur de risque cardiovasculaire

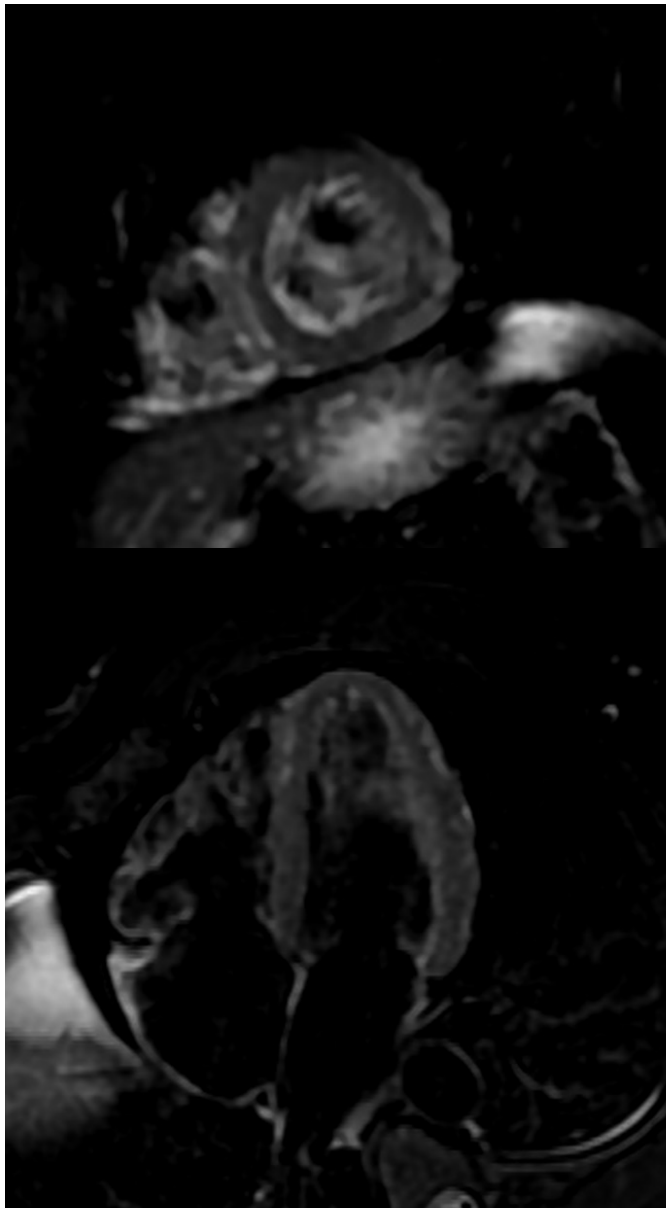
Douleur thoracique avec pic troponine 8600 ng/ml

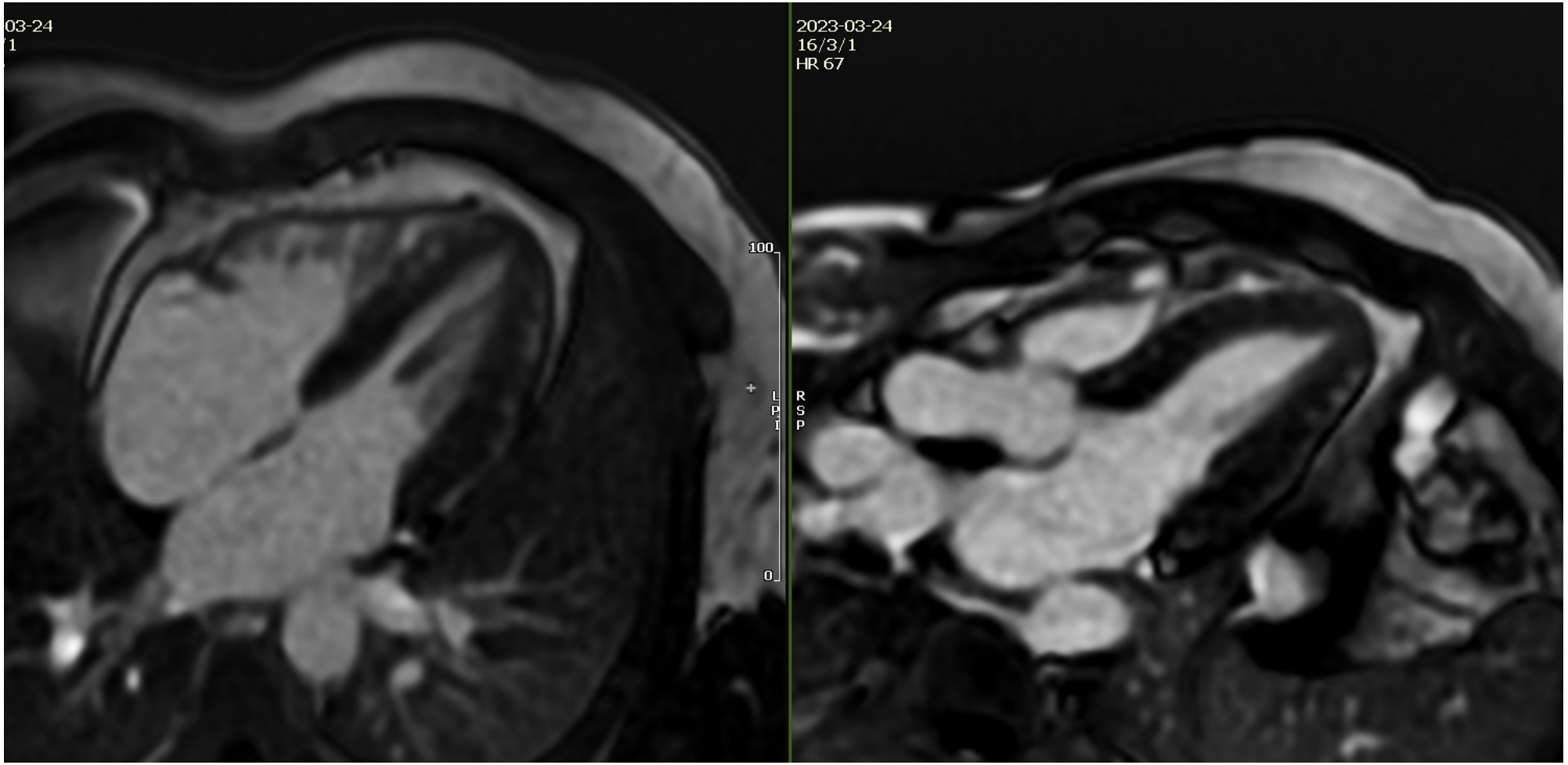
ECG : sus-décalage diffus

Coronaires normales

IRM à J11







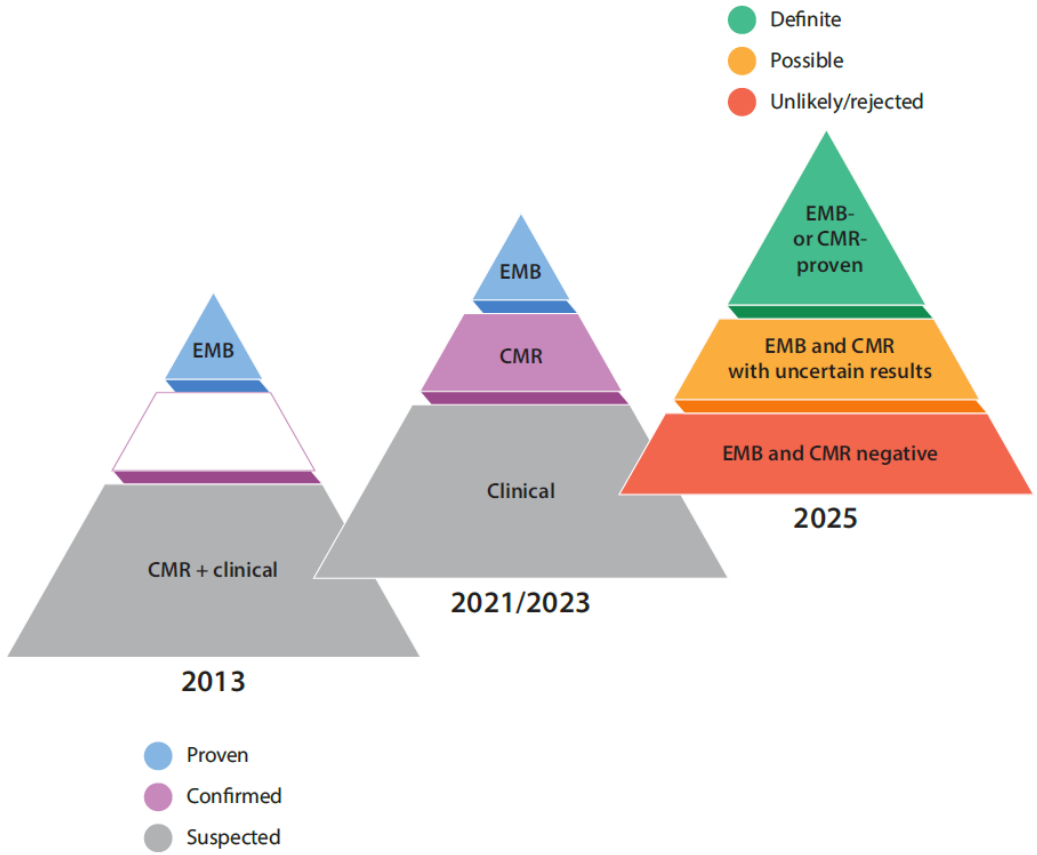
REHAUSSEMENT SOUS-EPICARDIQUE = NON ISCHEMIQUE

NON MINOCA ✗

MYOCARDITE AIGUË NON COMPLIQUEE

ESC European Society of Cardiology
European Heart Journal (2025) **46**, 3952–4041
https://doi.org/10.1093/eurheartj/ehaf192

2025 ESC Guidelines for the management of myocarditis and pericarditis



Updated Lake Louise Criteria (LLC) for myocarditis

CMR-proven myocarditis=
2 out of 2 updated LLC main criteria fulfilled

CMR-uncertain myocarditis=
only 1 out of 2 updated LLC main criteria fulfilled

Main criteria

T2-based criterion
Myocardial oedema

Abnormal T2-mapping or T2-weighted imaging

T1-based criterion
Non-ischaemic myocardial injury

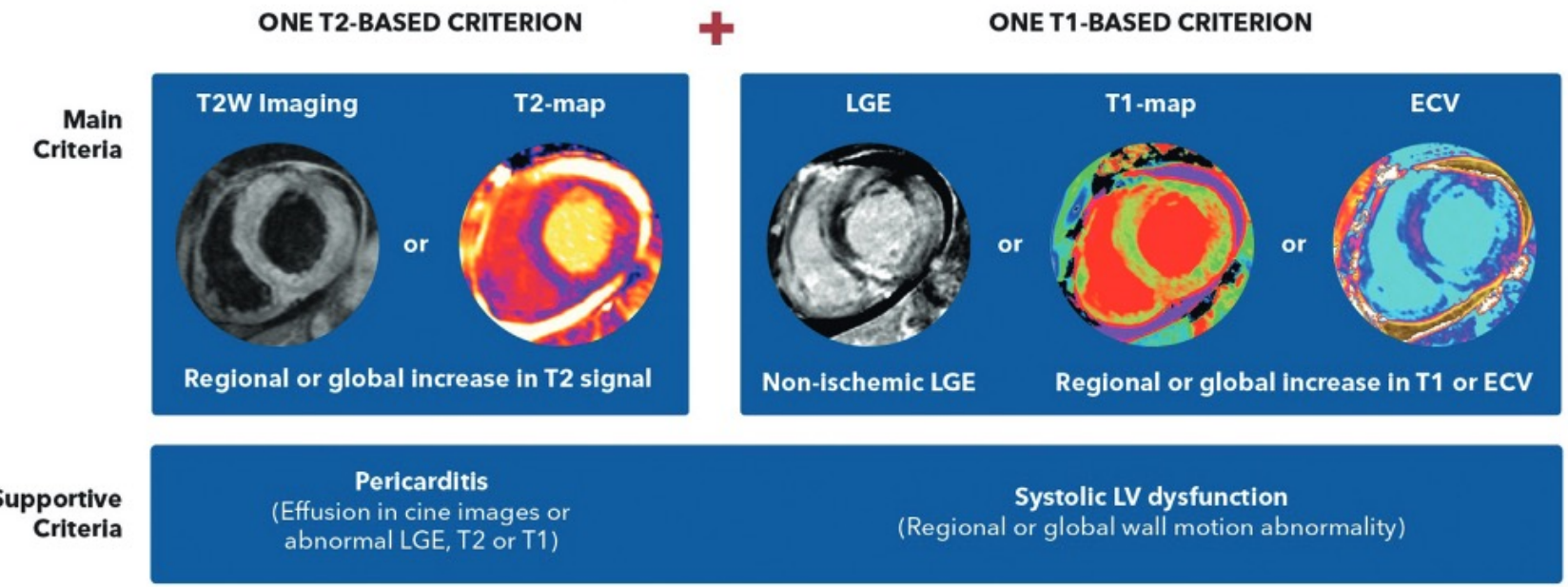
Abnormal T1-mapping, ECV or LGE

Supportive criteria

Pericardial abnormalities

Systolic LV-dysfunction

CMR Detection of Non-ischemic Inflammation
The Updated Lake Louise Criteria (2018)



CAS CLINIQUE 4

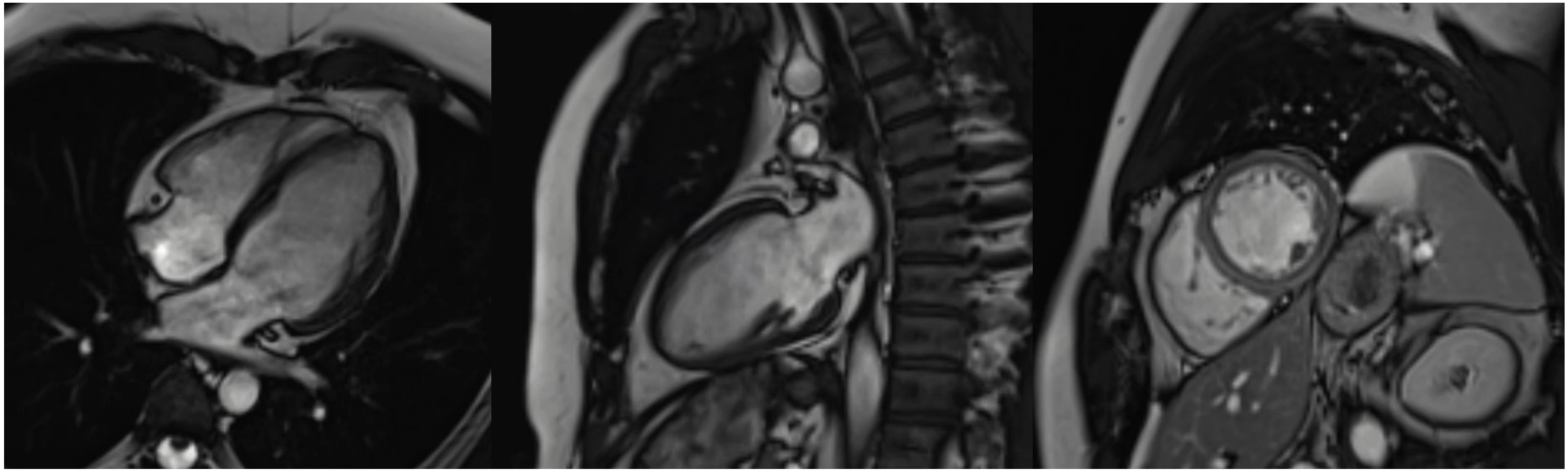
Patient de 34 ans

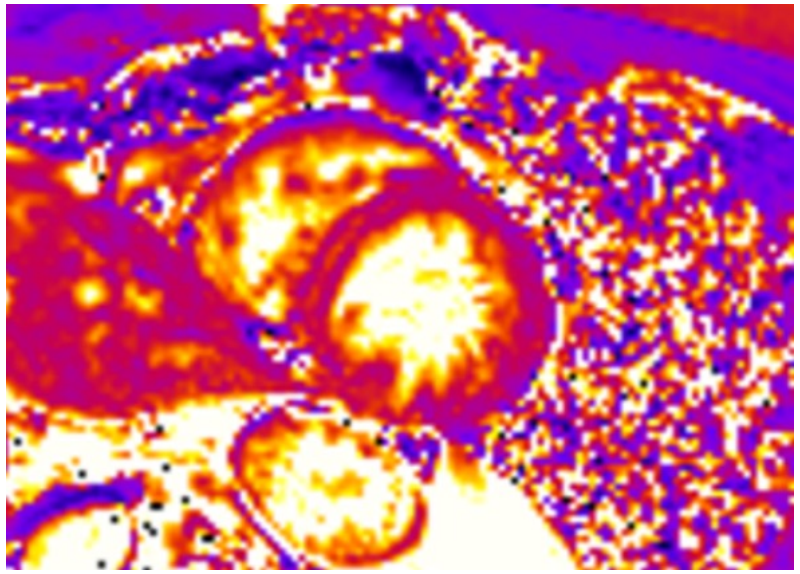
Episode de douleur thoracique avec élévation de troponine

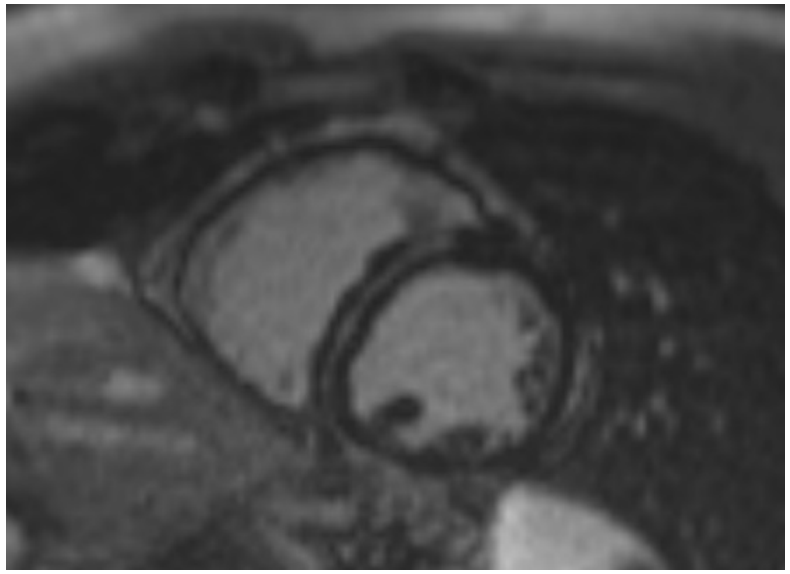
Coroscanner normal

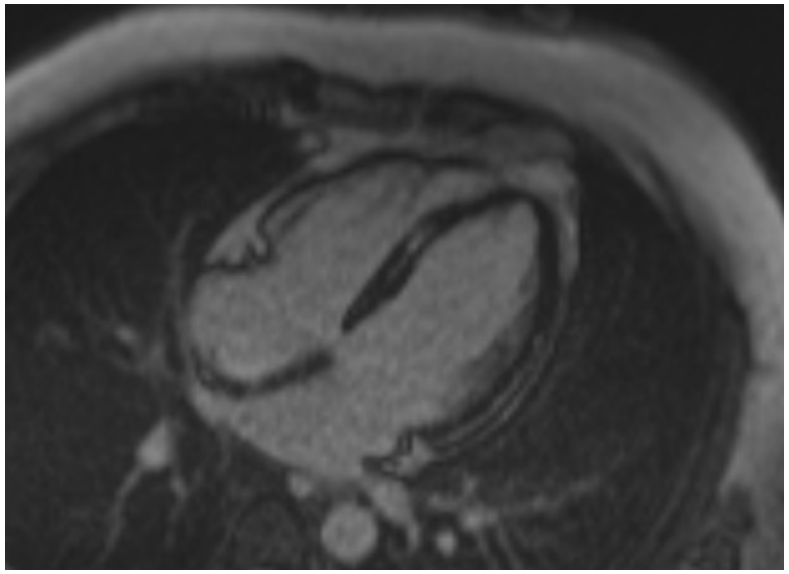
Considéré comme une myocardite, avec RDV IRM programmé la semaine suivante

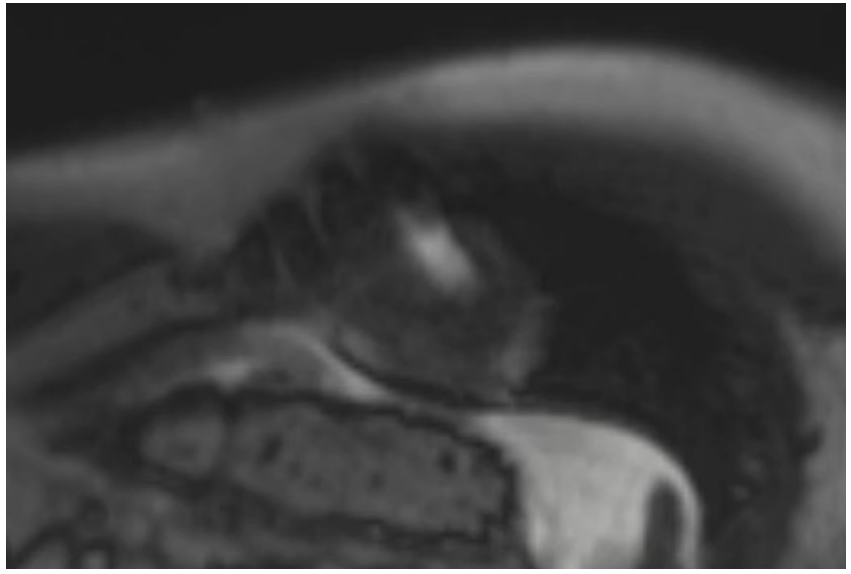
Ne s’est pas présenté à son IRM, qui a été réalisée à M3 à la demande de son cardiologue







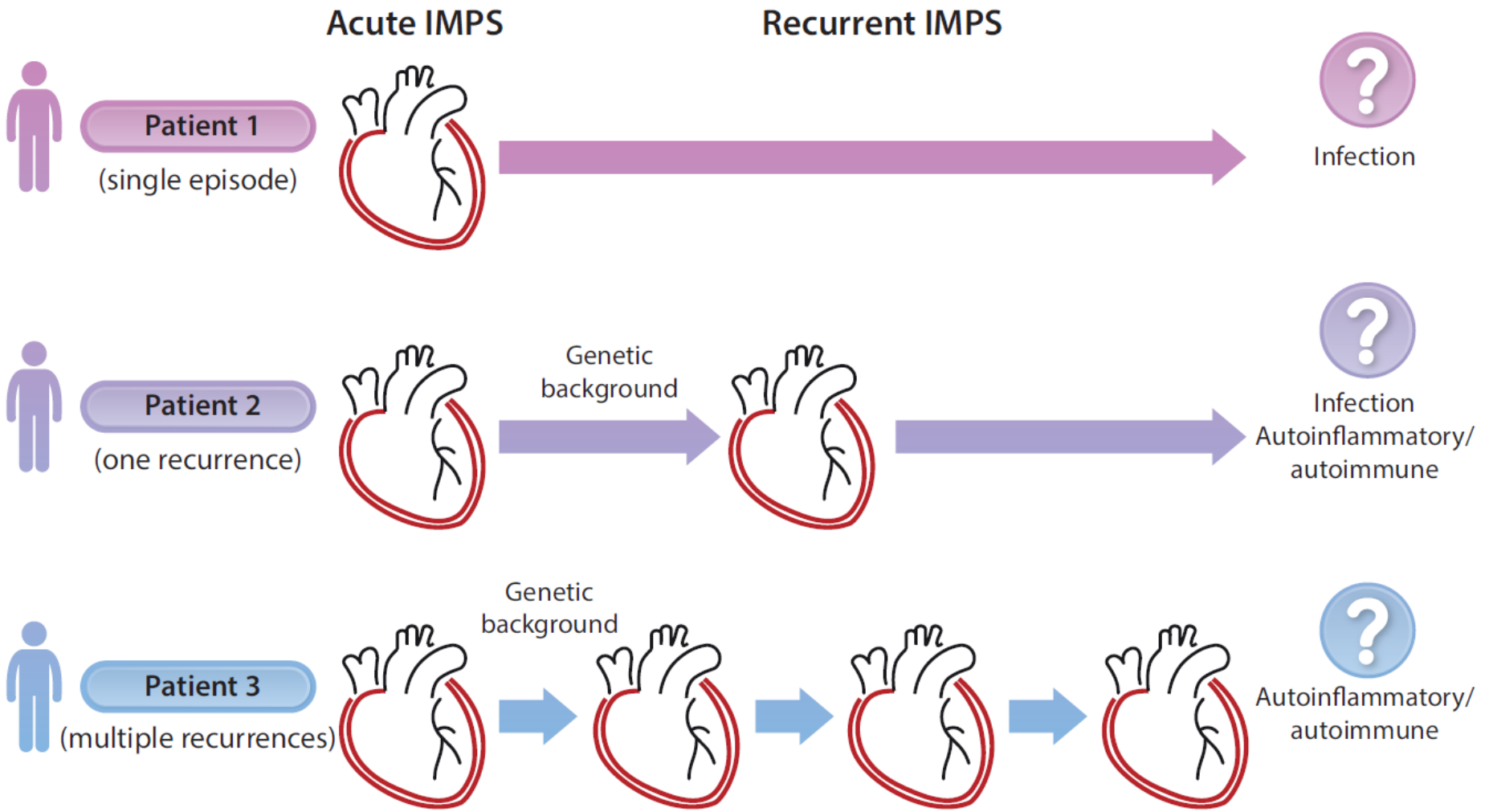




NON MINOCA ✗

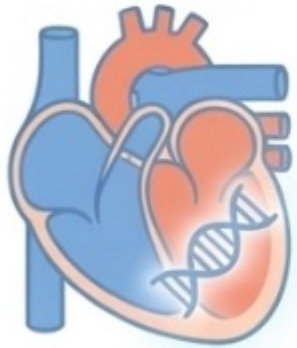
Myocardite aiguë, œdème persistant à M3

MAIS SURTOUT rehaussement tardif circonférentiel ou « ring-like »



Schulz-Menger et al., Eur Heart J 2025 Oct 22;46(40):3952-4041

Substrat Vulnérable



- **Prédisposition Génétique :** Mutations hétérozygotes (ex. *TTN*, DSP), haploinsuffisance.

Déclencheurs Environnementaux

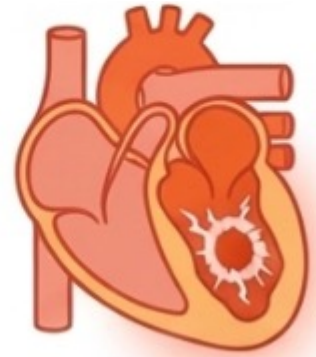
Chimiothérapie

Grossesse (Péripartum)

Consommation d'alcool

Myocardite aiguë

Troubles immunitaires systémiques



- **Démasquage :** Stress précipite l'expression.
- **Risques :** Arythmies ventriculaires, insuffisance cardiaque.

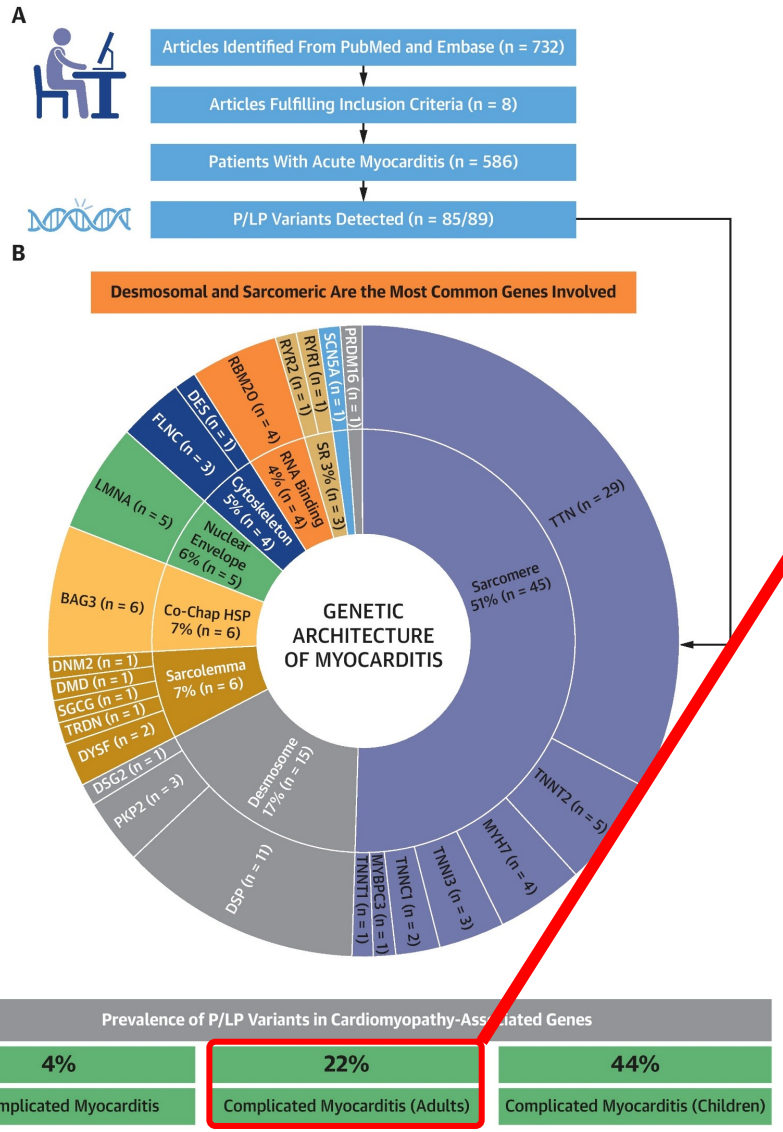
Action Clinique : Nouveau Paradigme

Diagnostic Génétique : Panels modernes pour identifier la cause fondamentale, même avec déclencheur évident.

Stratification du Risque : Surveillance ciblée et DAI (ex. calculateur DSP).

Thérapie & Dépistage : Thérapie selon recommandations, dépistage familial en cascade.

CENTRAL ILLUSTRATION: Prevalence of P/LP Variants in Cardiomyopathy-Associated Genes in Patients With Myocarditis



Monda E, et al. J Am Coll Cardiol HF. 2024;12(6):1101-1111.

Genetic testing should be considered in patients with definite myocarditis/pericarditis in cases of:[50,51,64,94,150] • family history of IMPS, inherited or suspected cardiomyopathy • severe ventricular arrhythmia[c] • significant left/right LGE (e.g. ring-like pattern or septal LGE) or persistent LVEF systolic dysfunction • recurrent myocarditis or persistent troponin elevation	IIa	B

Table 21 High-risk genotypes and associated predictors of sudden cardiac death

Gene	Annual SCD rate	Predictors of SCD
LMNA[185,186,438,541,865,878,879]	5–10%	Estimated 5-year risk of life-threatening arrhythmia using *LMNA* risk score (https://lmna-risk-vta.fr)
FLNC-truncating variants[866,867,880]	5–10%	LGE on CMR LVEF < 45%
TMEM43[868,881]	5–10%	Male Female and any of the following: LVEF <45%, NSVT, LGE on CMR, >200 VE on 24h Holter ECG
PLN[542,882,883]	3–5%	Estimated 5-year risk of life-threatening arrhythmia using *PLN* risk score (https://plnriskcalculator.shinyapps.io/final_shiny) LVEF < 45% LGE on CMR NSVT
DSP[185,186]	3–5%	LGE on CMR LVEF < 45%
RBM20[869]	3–5%	LGE on CMR LVEF < 45%

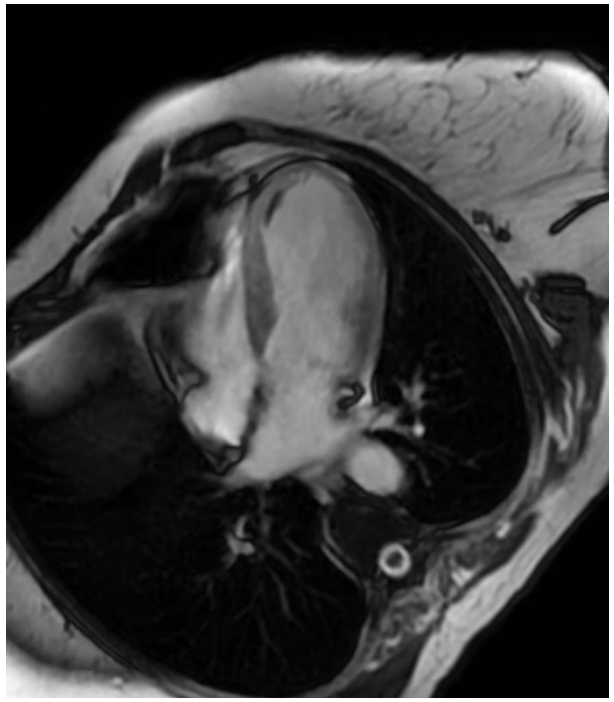
CAS CLINIQUE 5

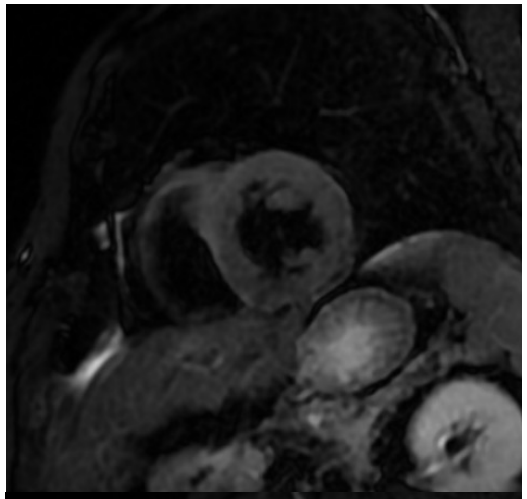
Patiente de 72 ans

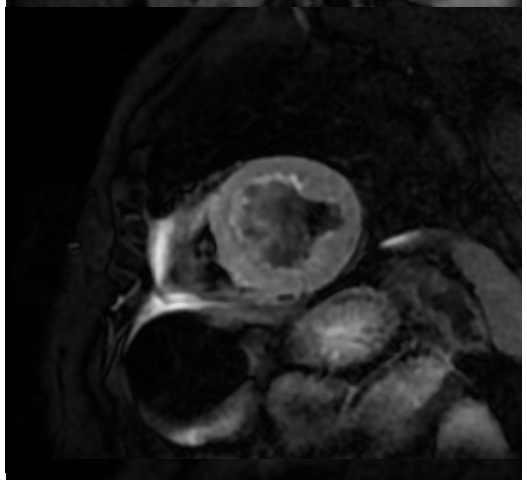
Douleur thoracique typique 1h suite à un stress émotionnel

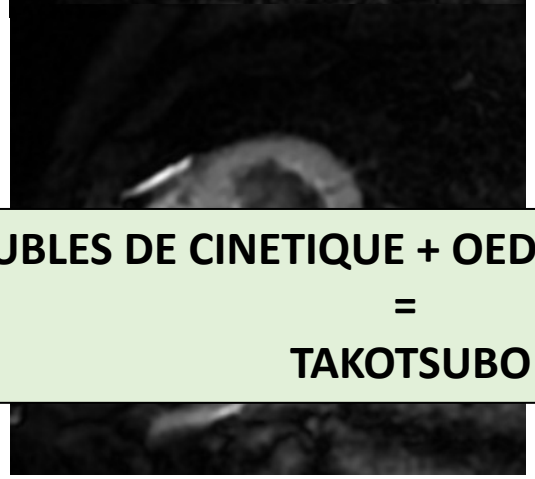
ECG onde T négative antérieure

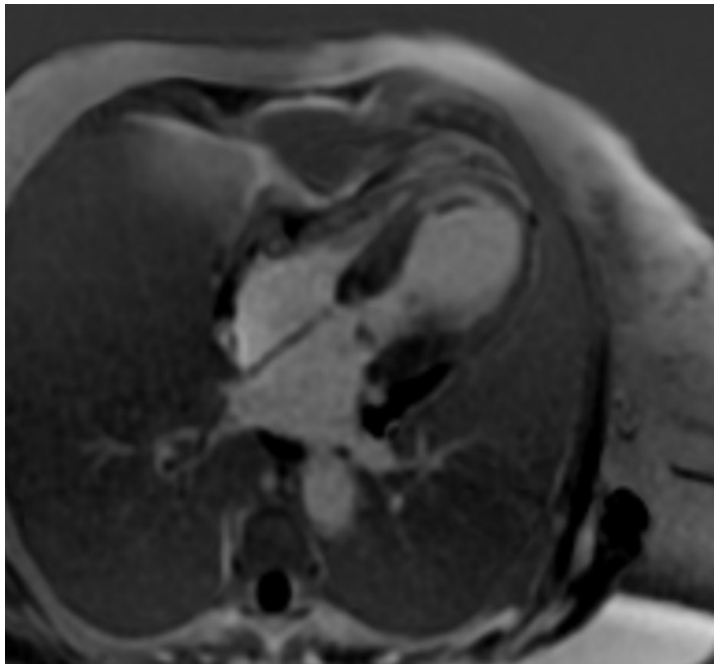
Coronarographie normale.

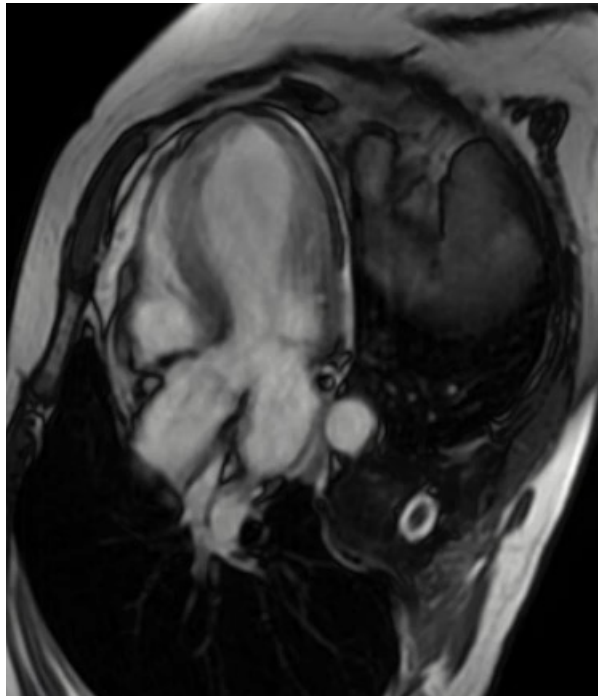


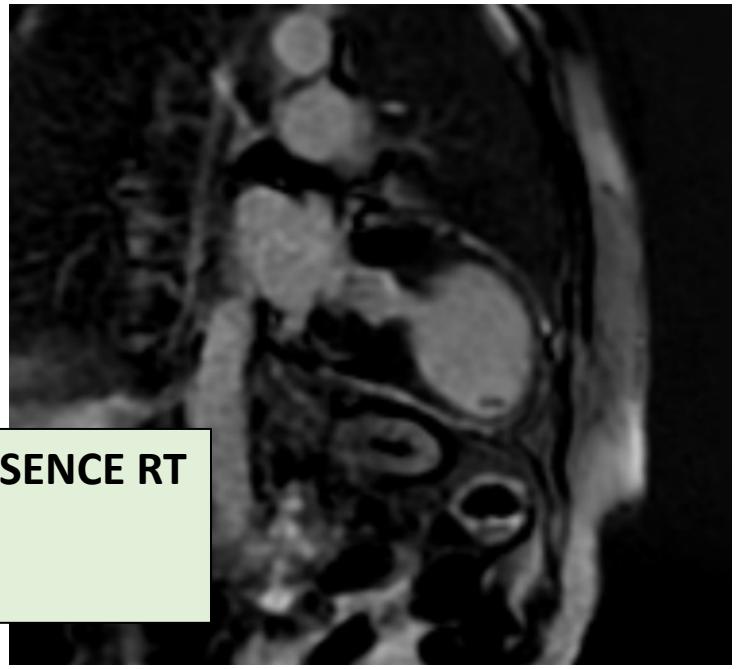












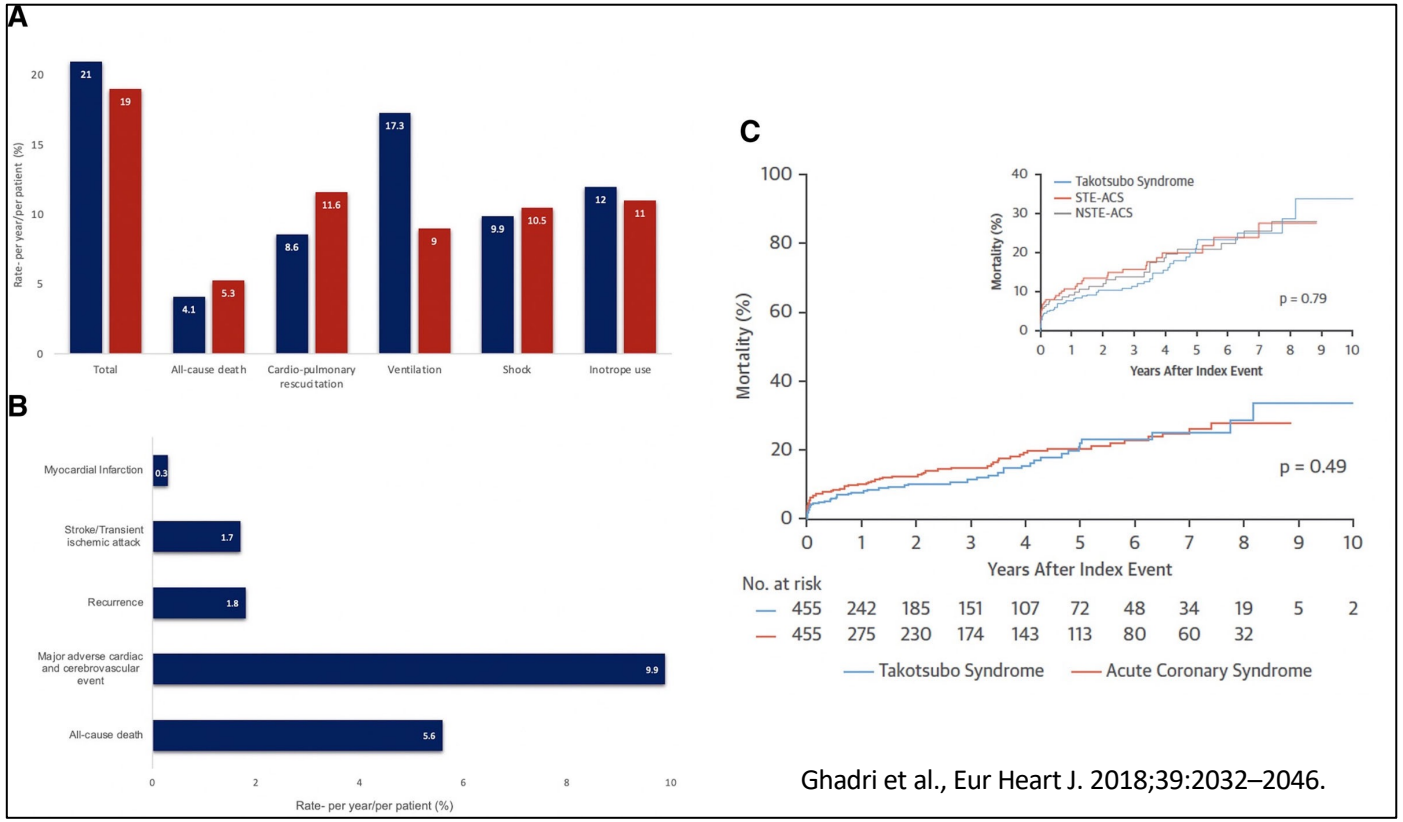
TROUBLES DE CINETIQUE + OEDEME + ABSENCE RT
=
TAKOTSUBO

Cardiomyopathie de Takotsubo ou cardiopathie de stress

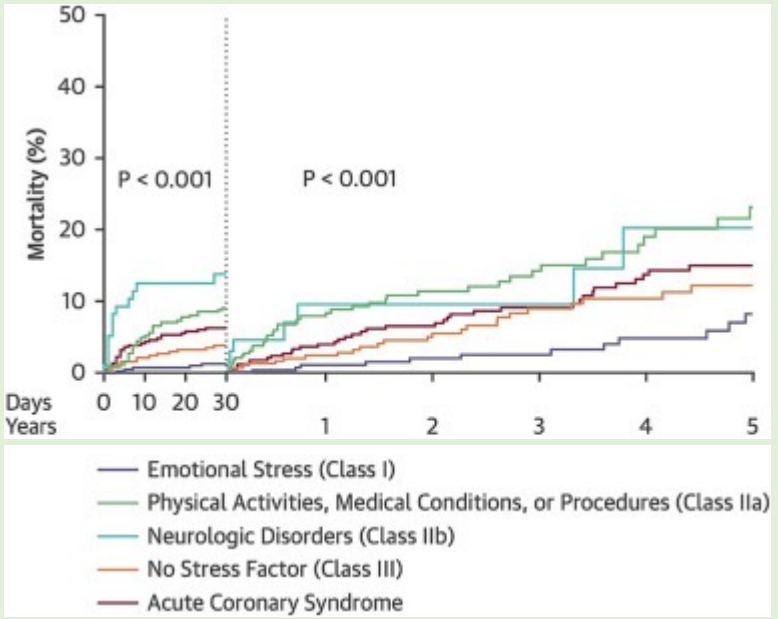
Dysfonction **ventriculaire aiguë réversible** liée à une **tempête catécholaminergique** avec :

- atteinte préférentielle apicale
- dysfonction microvasculaire
- toxicité myocardique directe

2-3% des SCA, 90% de femmes



Ghadri et al., Eur Heart J. 2018;39:2032–2046.



Différentes présentations

- Classique apicale (81.7%)
- Médioventriculaire (14.6%)
- Basale (2.2%)
- Focale (1.5%)

Kato et al., J Cardiol 2025 Apr;85(4):263-267

CAS CLINIQUE 6

Patiente de 40 ans, sans antécédents médicaux
Douleur épigastrique, suivie de fièvre et de signes d'inflammation systémique.

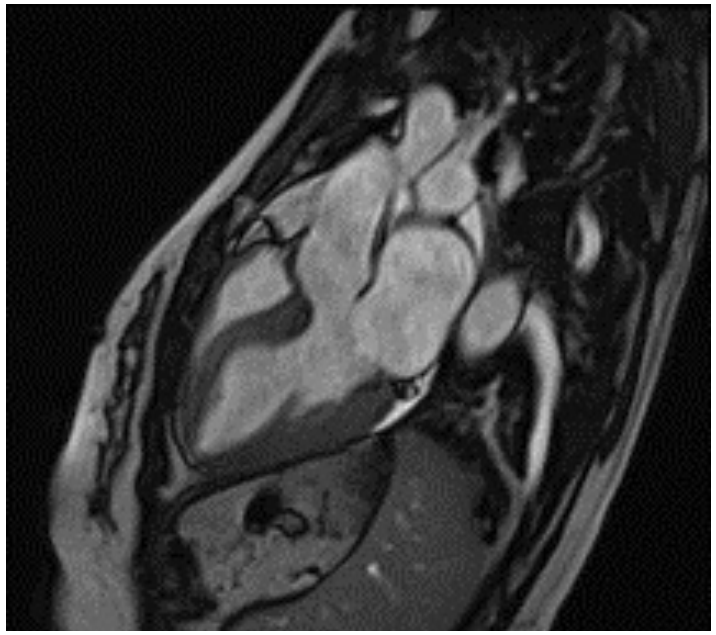
Suspicion de cholécystite → cholécystectomie.

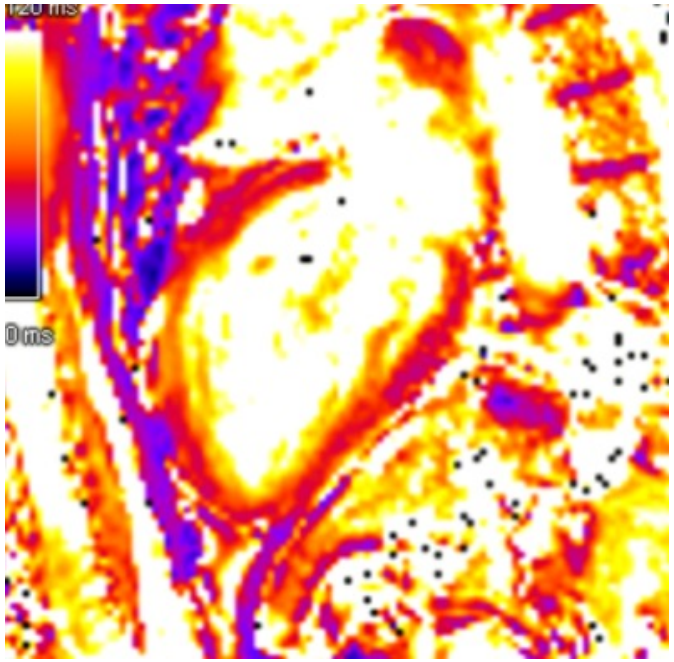
Pendant l'hospitalisation, élévation de troponine jusqu'à 800 ng/L

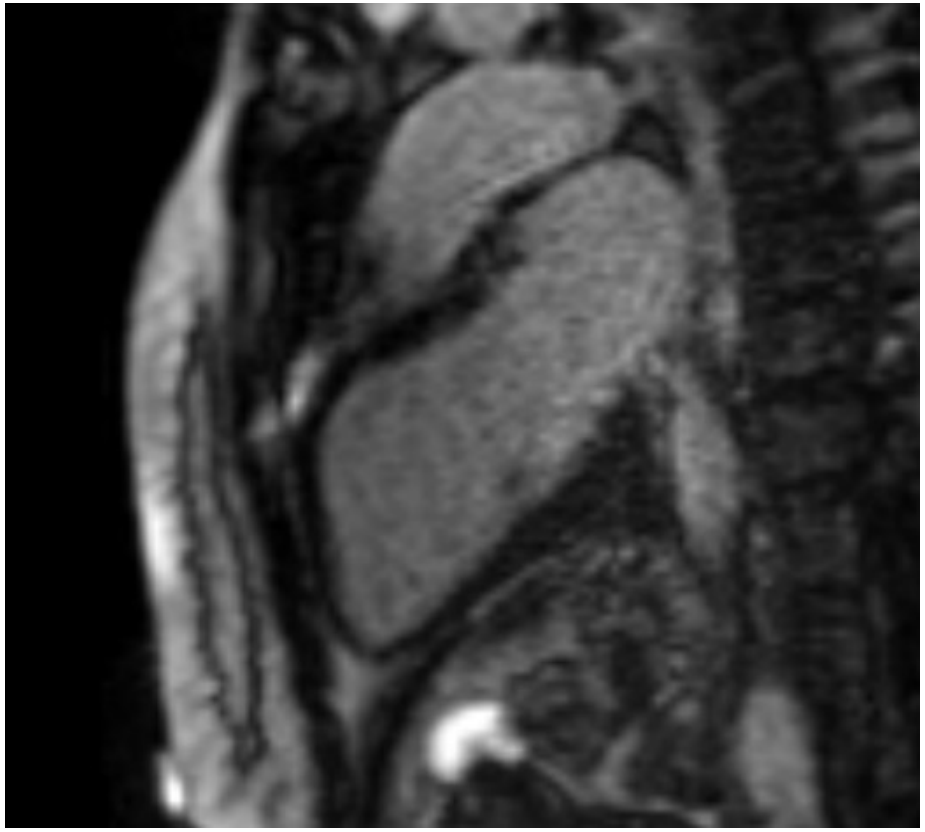
Anomalies ECG : T négatives en D1 et aVL, T biphasique en V2).

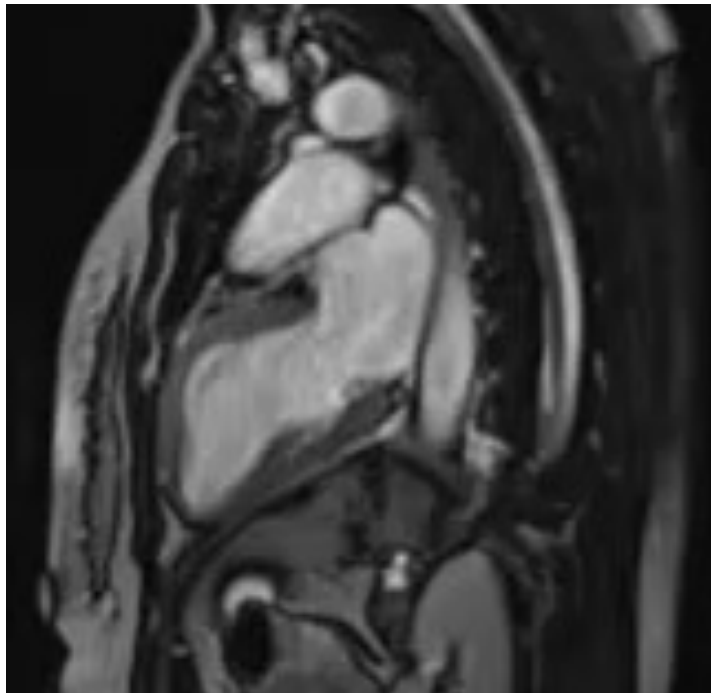
ETT étiquetée normale

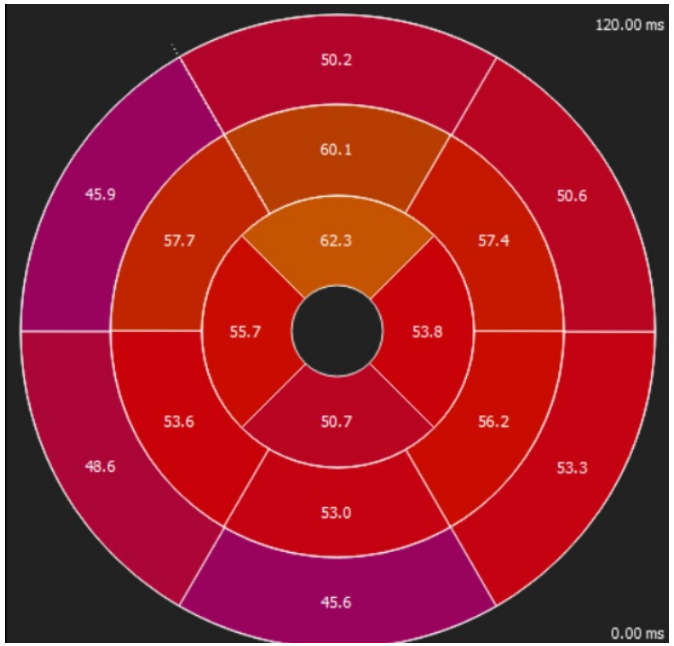
Syndrome inflammatoire (post-opératoire) : Myocardite?











FOCAL TAKOTSUBO CM

CAS CLINIQUE 7

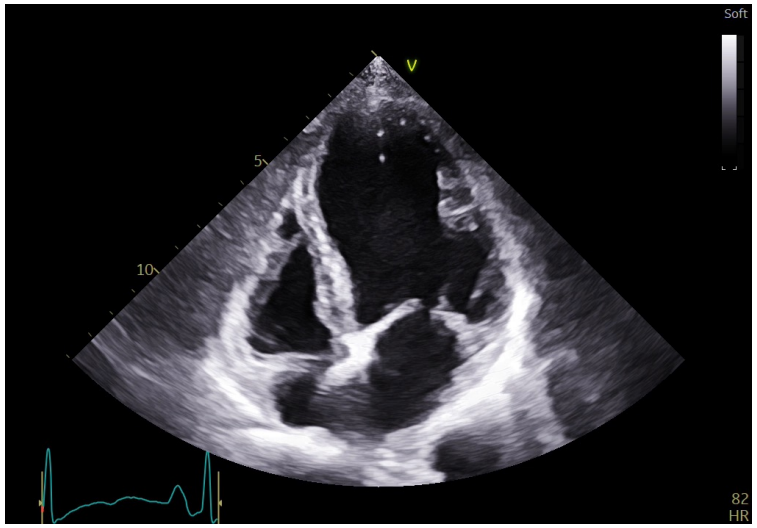
71-year-old woman

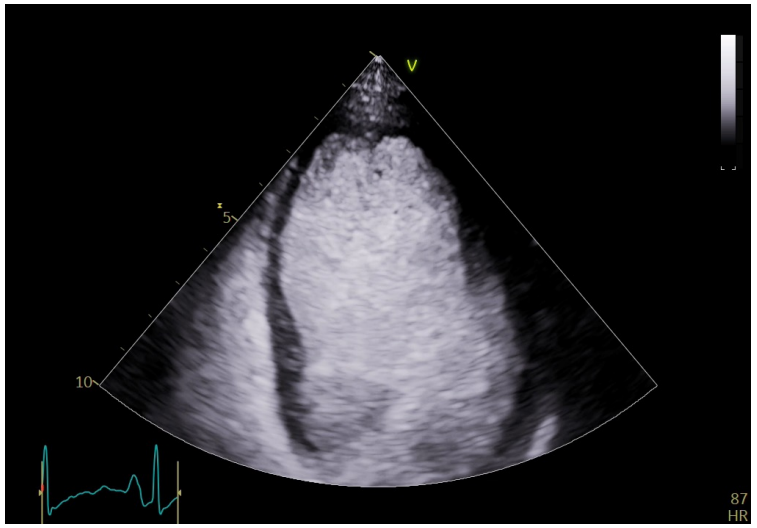
No significant history

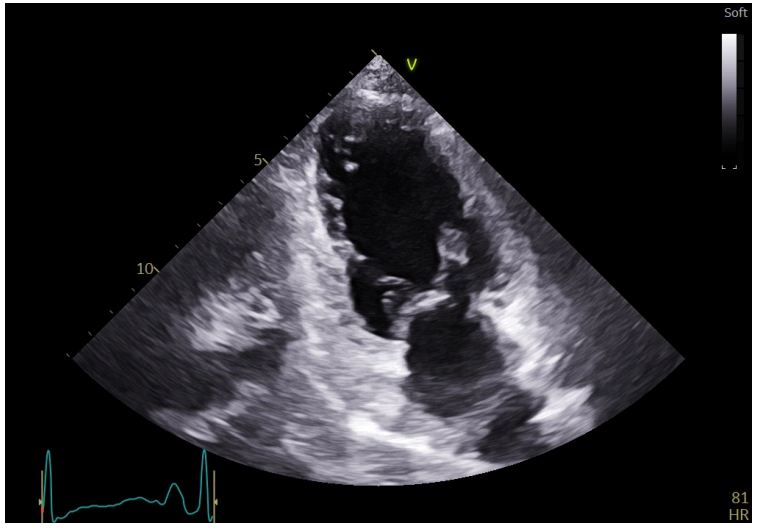
Presented to the emergency department for constrictive chest pain

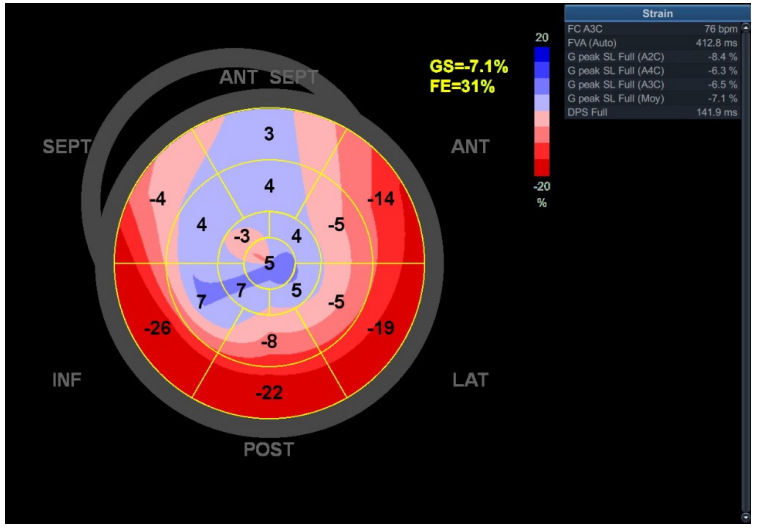
ECG: deep negative T waves in the anterior territory

Blood test : increase in troponin up to 3884 ng/ml









LVEF 30%, extensive apical akinesia, no LV thrombus

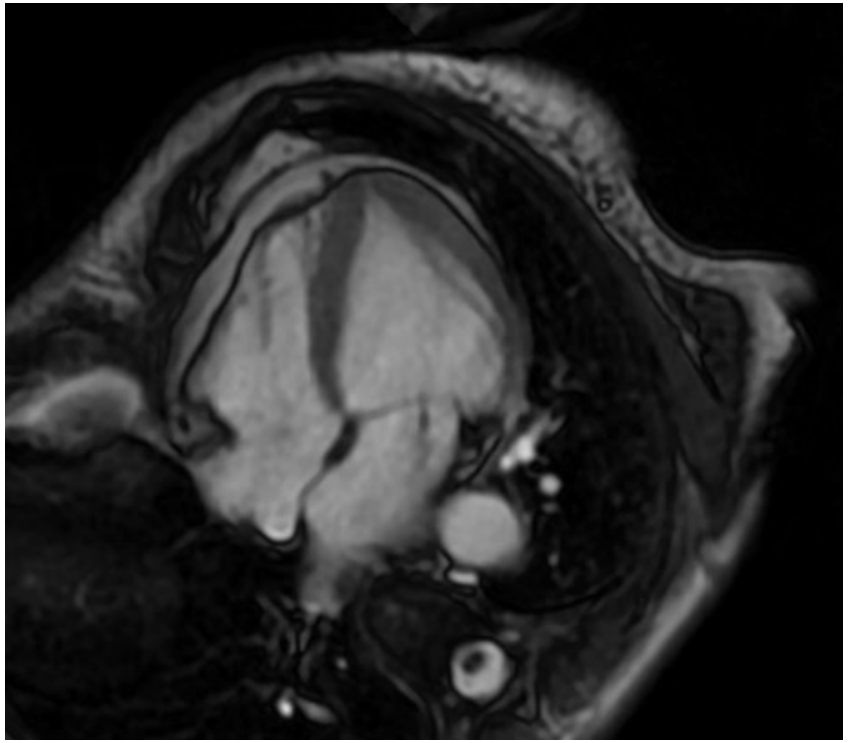
Coronary angiography : normal

Working diagnosis: TakoTsubo Cardiomyopathy

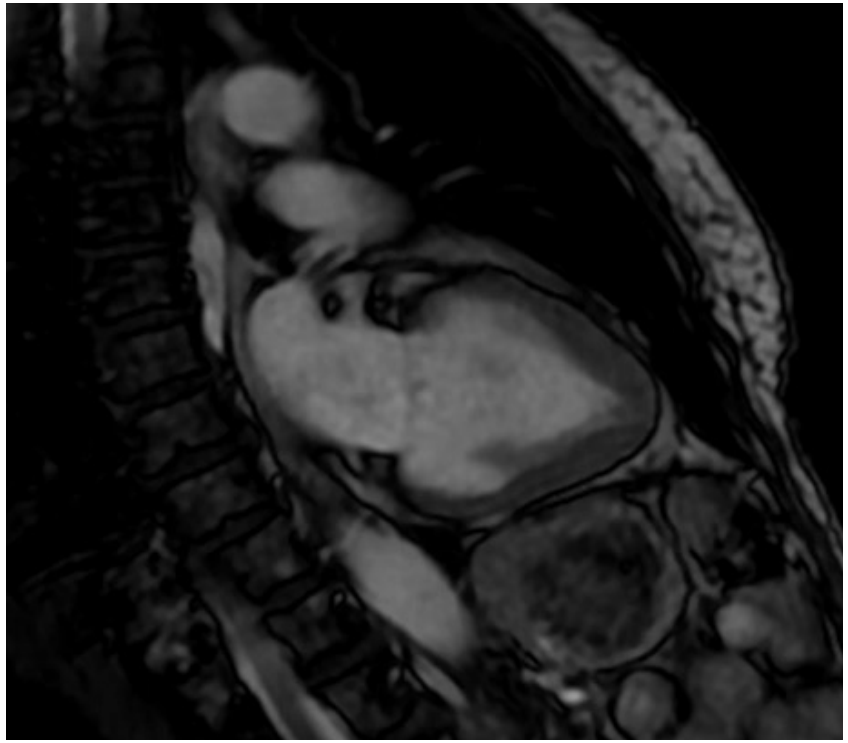
INITIAL CMR AT DAY 5

CINE SEQUENCES

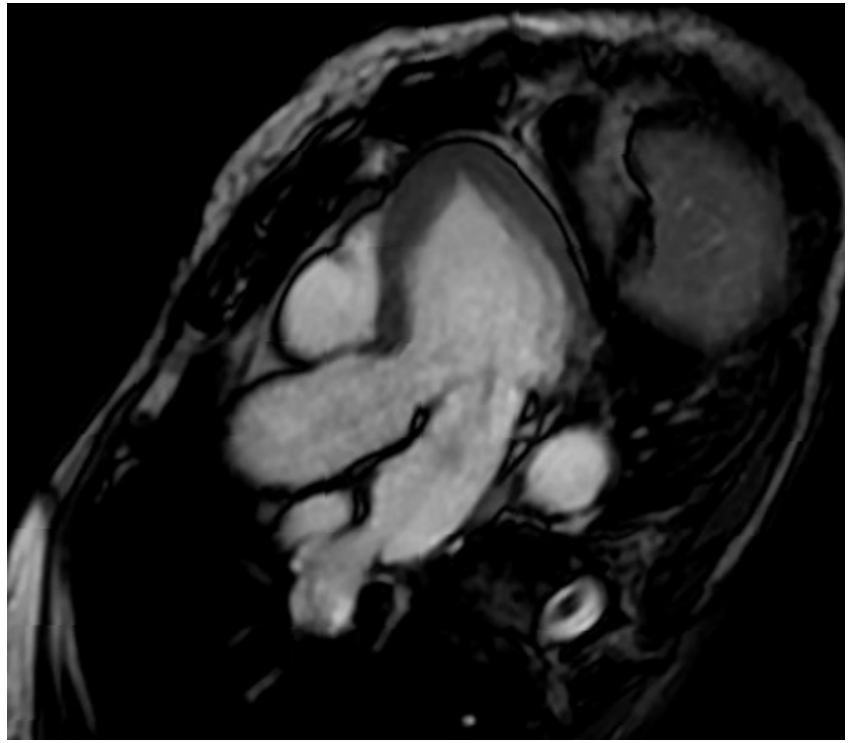
A4C



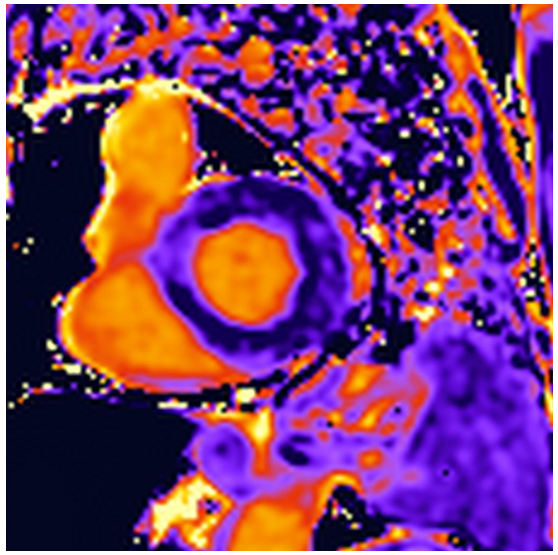
A2C

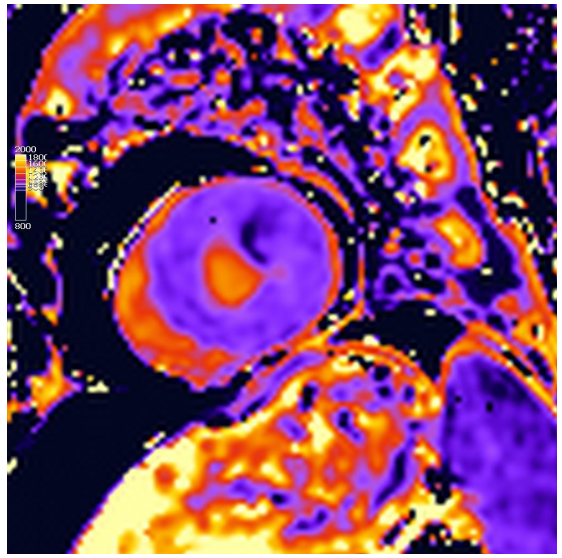


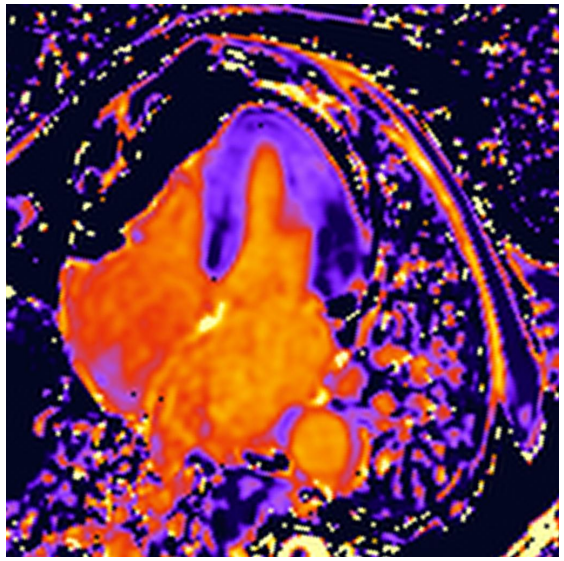
A3C

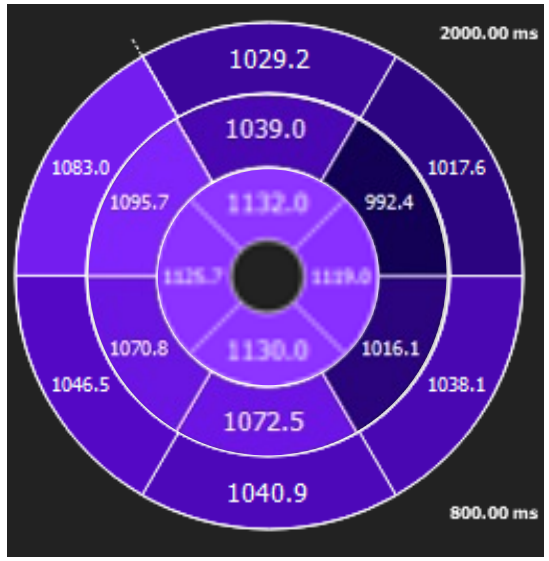


T1 MAPPING = EXTRACELLULAR COMPARTMENT



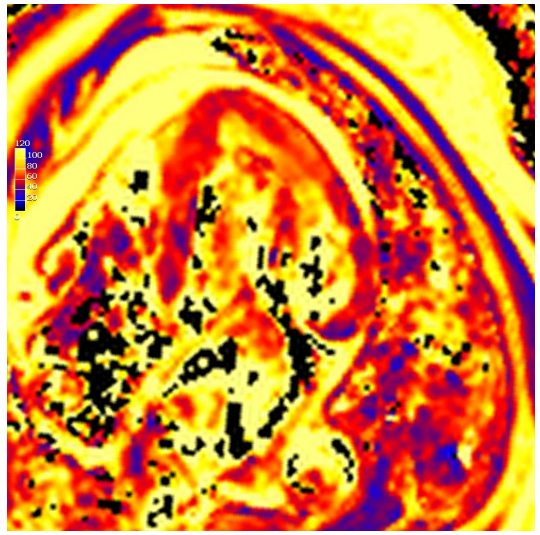


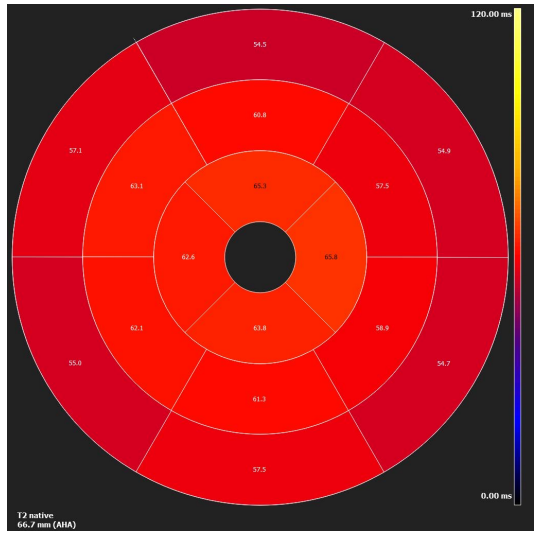




Up to 1132 ms (apex)
N<1005 ms

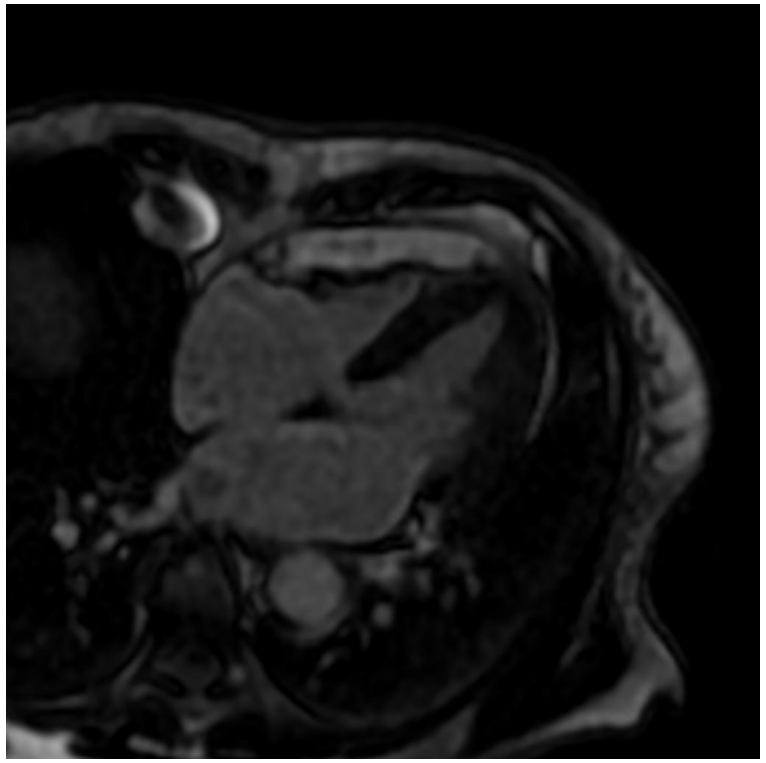
T2 MAPPING = OEDEMA



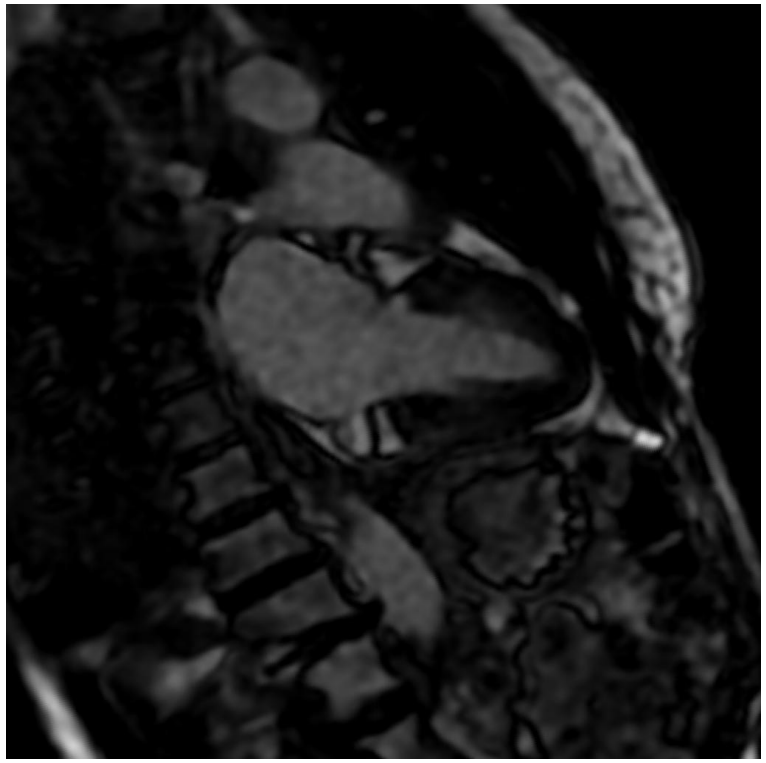


63-65 ms (N<55 ms)

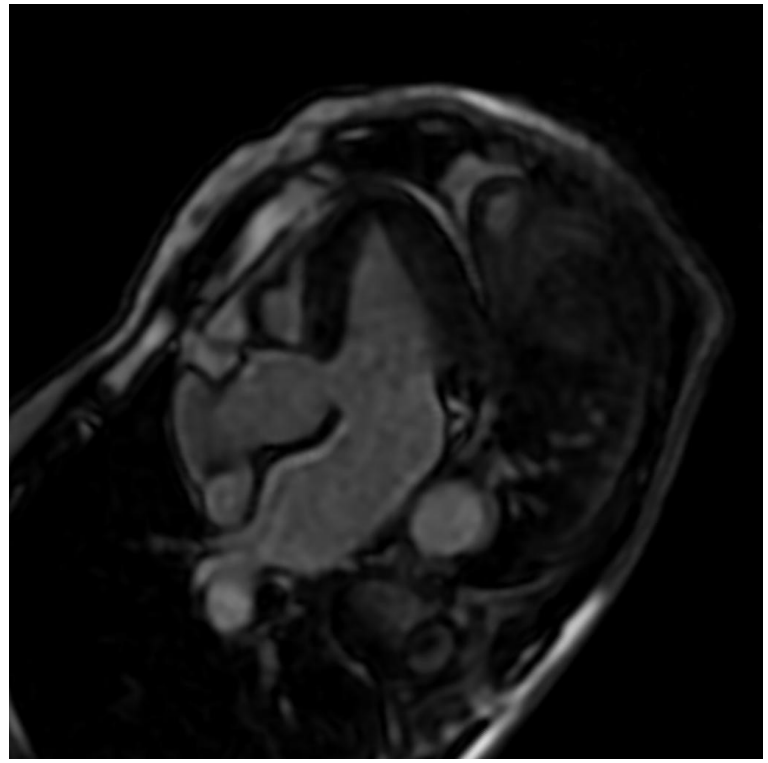
A4C



A2C



A3C



NO LATE GADOLINIUM ENHANCEMENT

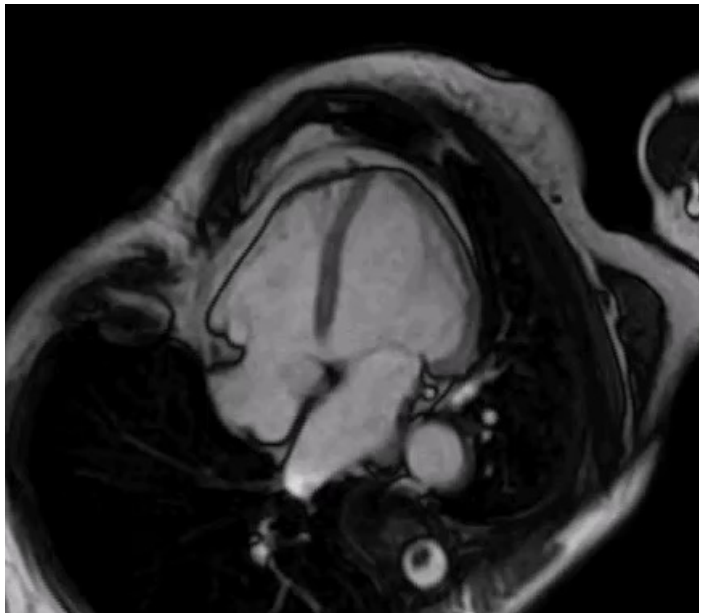
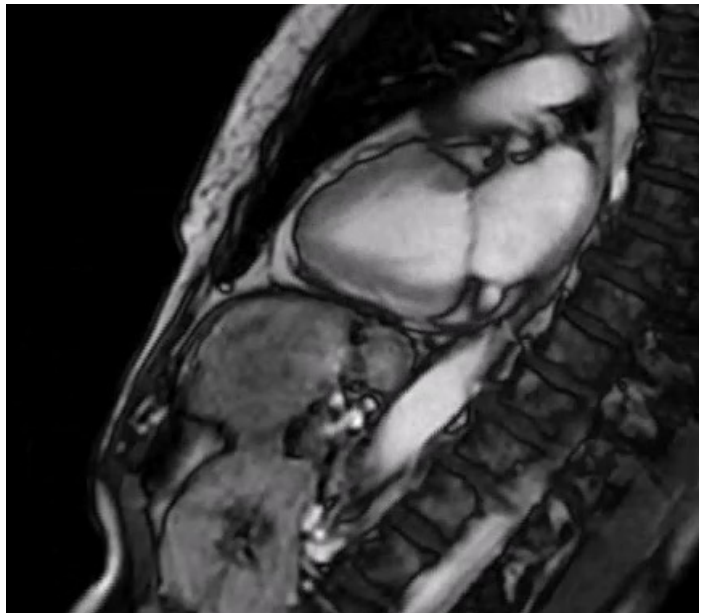
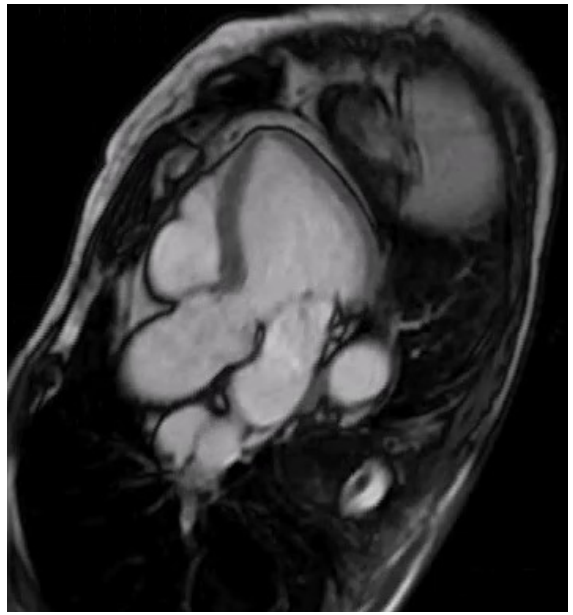
Considering the initial presentation, working diagnosis still considered as Takotsubo cardiomyopathy

Initiation of ACEi and betablocker

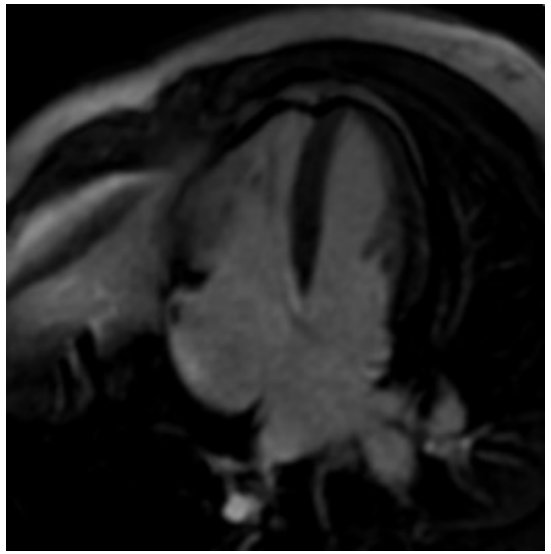
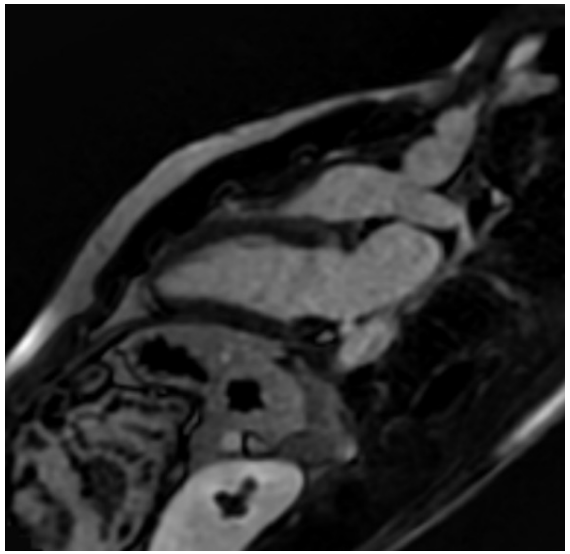
Discharged home

CMR was performed again 3 months later:

Cine: regression of hypertrophy

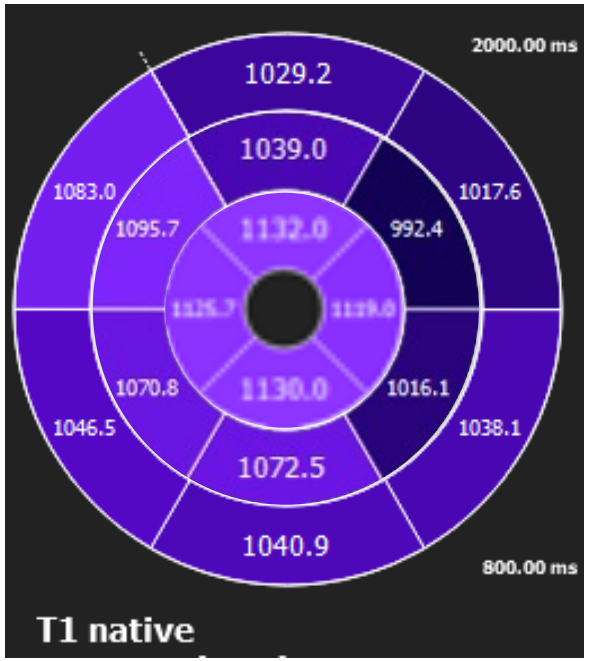
No LGE

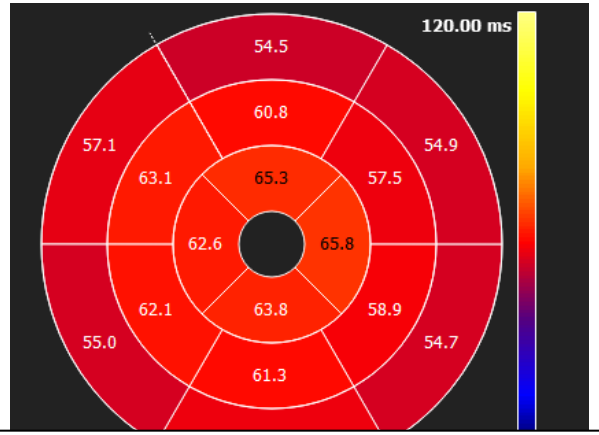



T1 MAPPING

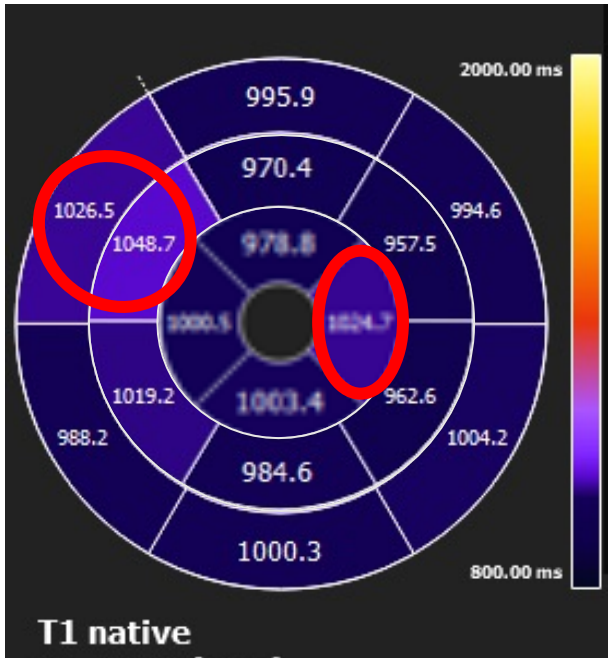
T2 MAPPING

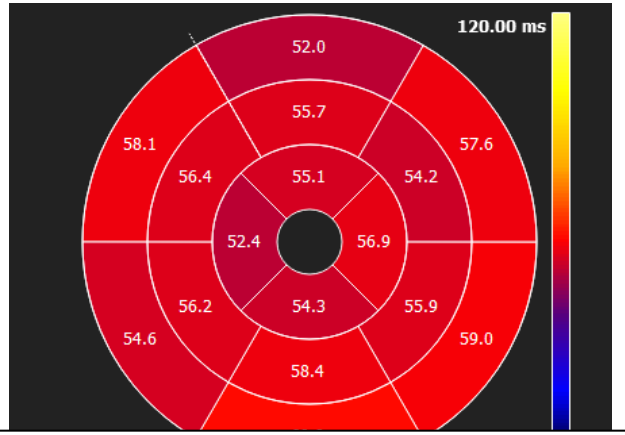
M0





M3

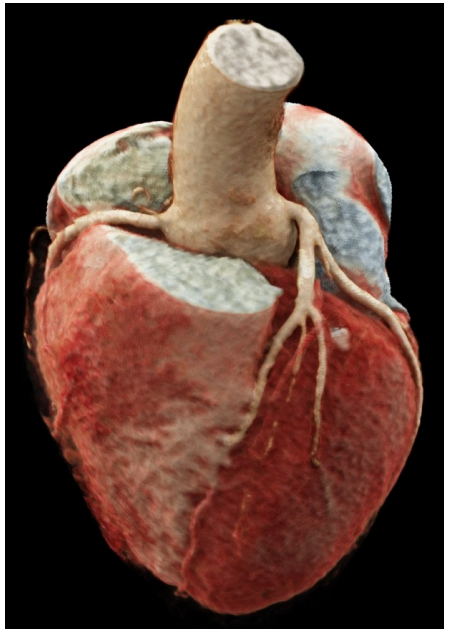




FINAL DIAGNOSIS: TAKOTSUBO CARDIOMYOPATHY WITH TRANSIENT APICAL HYPERTROPHY (OEDEMA)

CAS CLINIQUE 8

Courtesy of Dr Christina Karamarkou, Elisabeth Krankenhaus Essen, Germany



45-year-old female presented with **acute chest pain**, **palpitations**, and **SOB**.

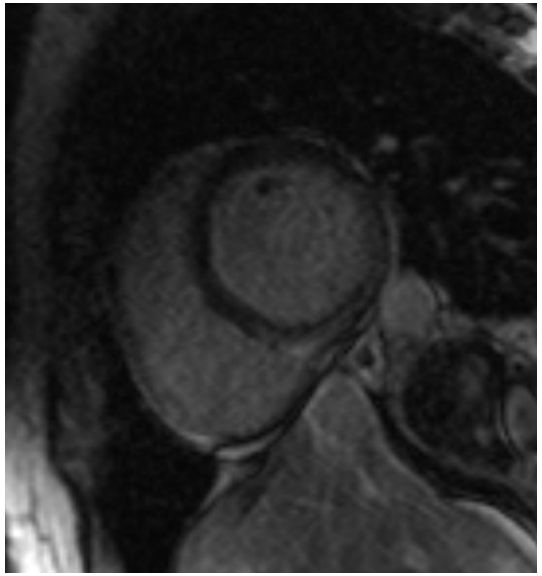
- **Social History:** High Work-Related Stress, but no specific trigger before symptom onset.
- **PMH:** Hypertension (compliant on Metoprolol and Telmisartan) and asthma.
- **Vitals:** Tachycardic (99 bpm) and hypertensive (190/110 mmHg).

2 Months prior

Chest pain
Paresthesias
Nausea
Headaches
Exertional dyspnea

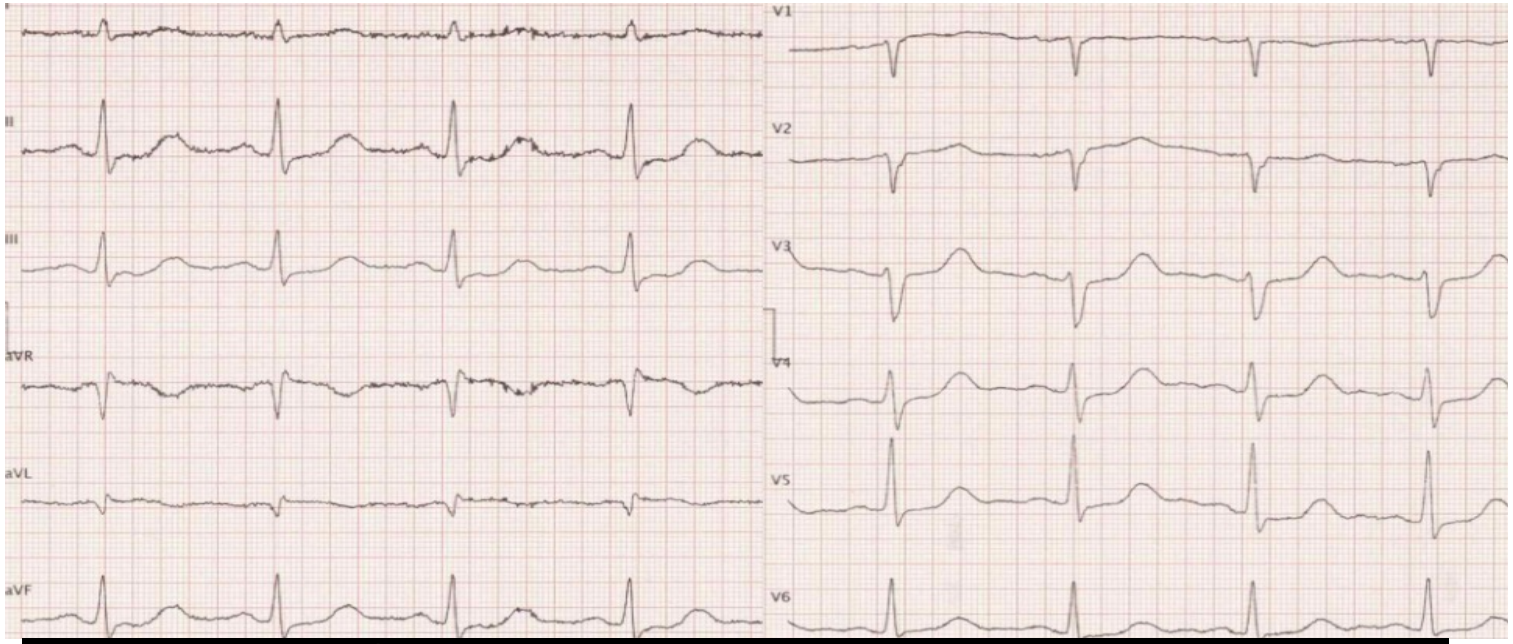
Normal ECG
Elevated cardiac biomarkers

CCTA
excluded coronary artery disease
CMR
confirmed acute myocarditis

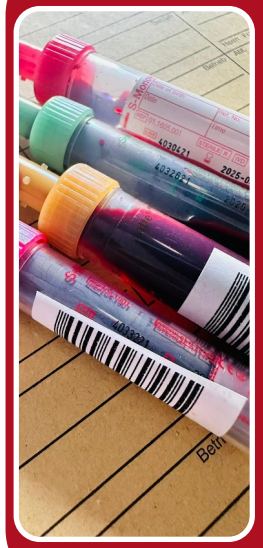


Initial Workup

- **ECG**:



- **Labs:**

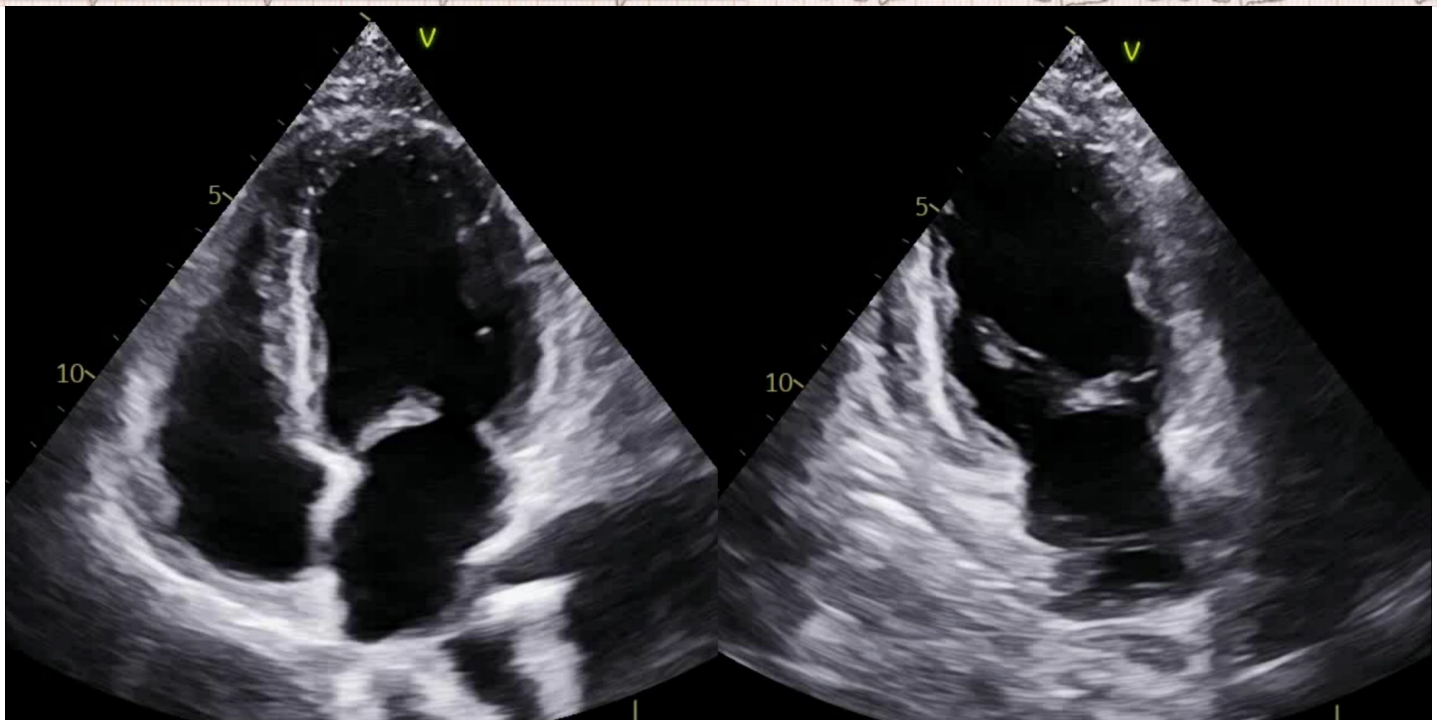


Troponin: Elevated at 638 ng/L, rising to 865 ng/L within one hour (normal: <14 ng/L)

White Blood Cell Count: Leukocytosis: 14.9K/mm³ (normal: 4.49-12.68K/mm³)

Other Labs: Electrolytes, liver function, renal function, and urinalysis all within normal limits

- **Echo**:



Diagnosis and Management

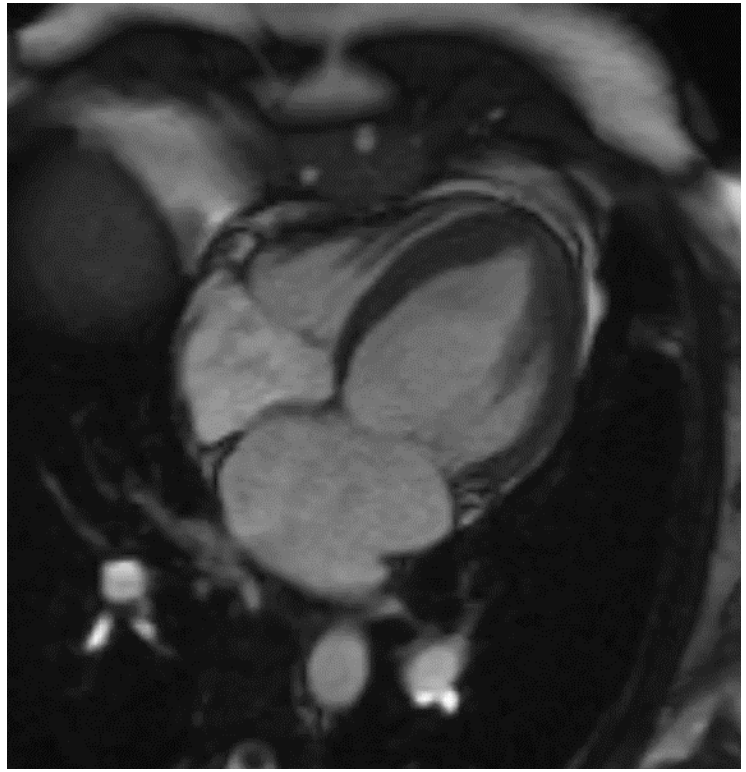
Initial Management: Admitted to Cardiac Care Unit,

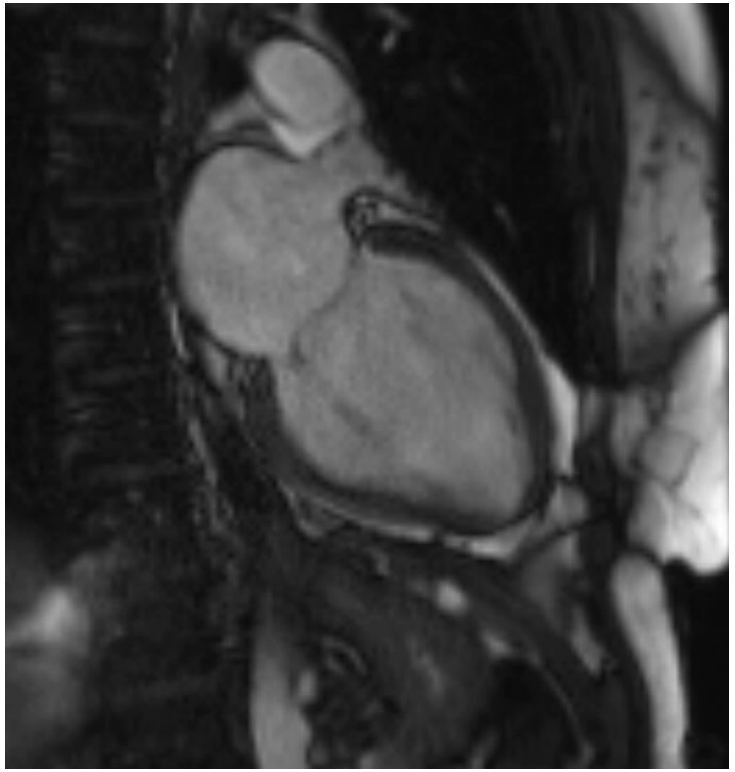
Medications: Aspirin, Heparin, Metoprolol, Morphine

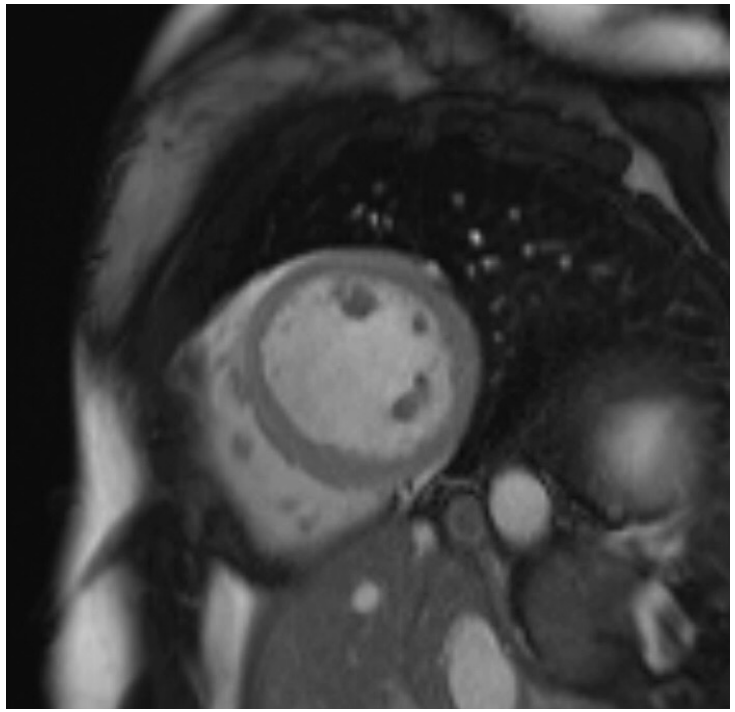
Diagnosis: Basal Type Takotsubo Syndrome (TTS) DD Recurrence of myocarditis

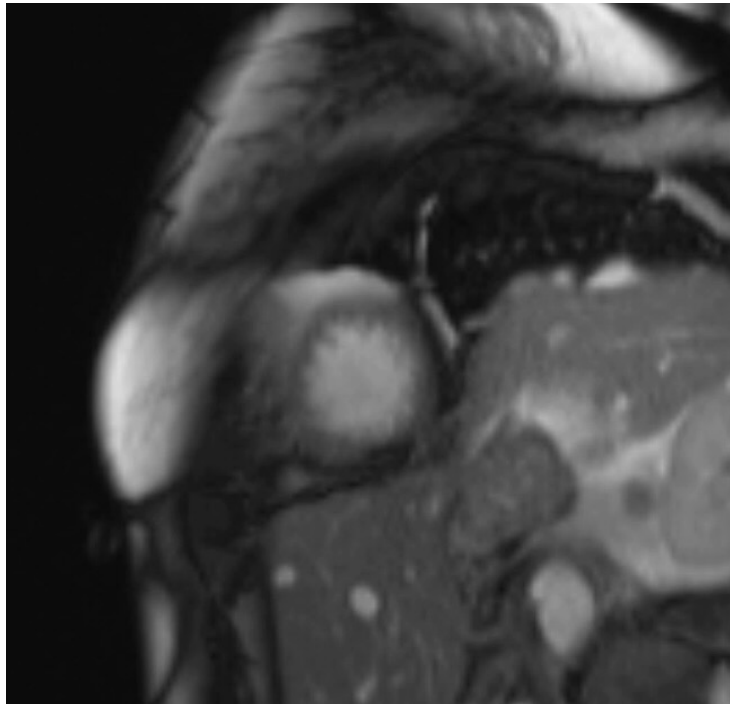
Further Investigations:

Invasive left heart catheterization: Considered but deferred (InterTAK Diagnostic Score of 55)

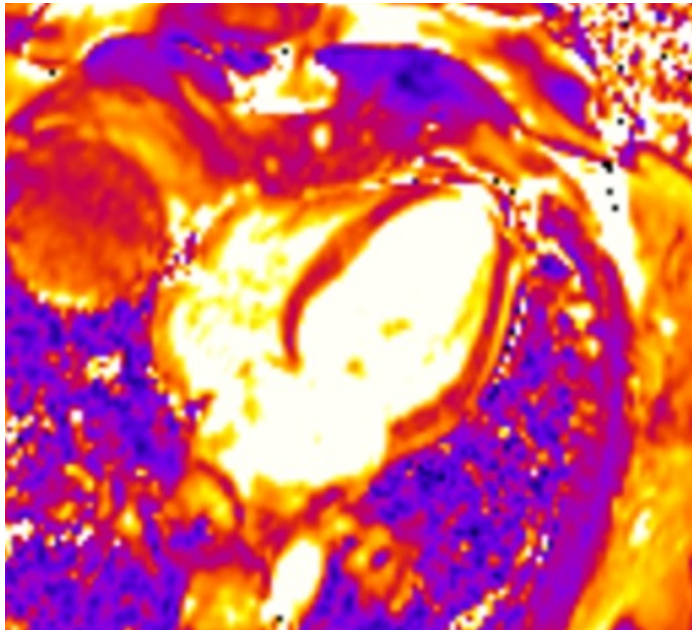


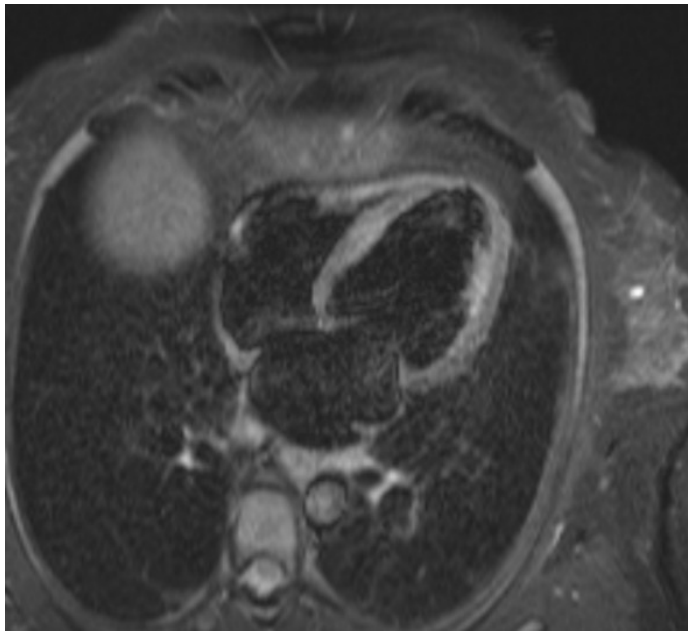




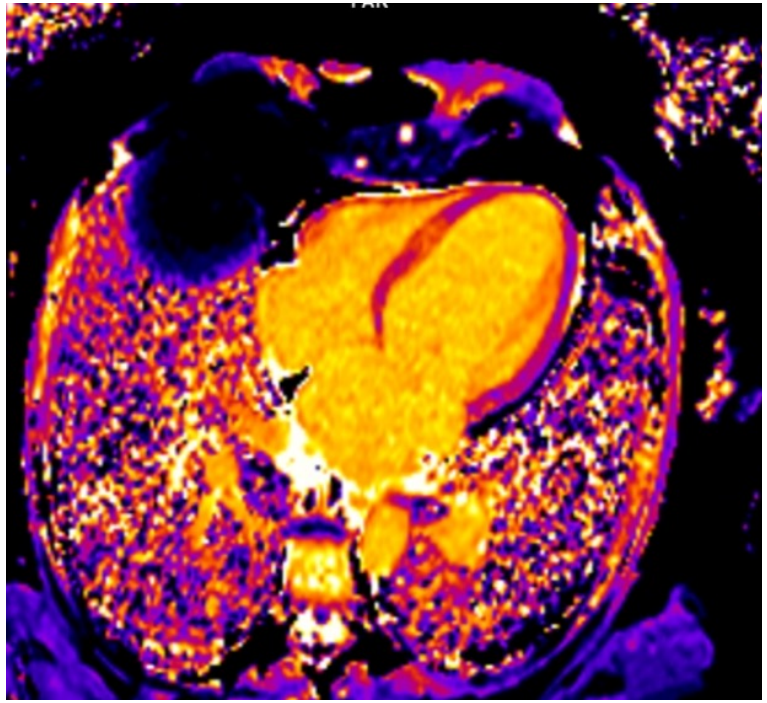


T2 mapping

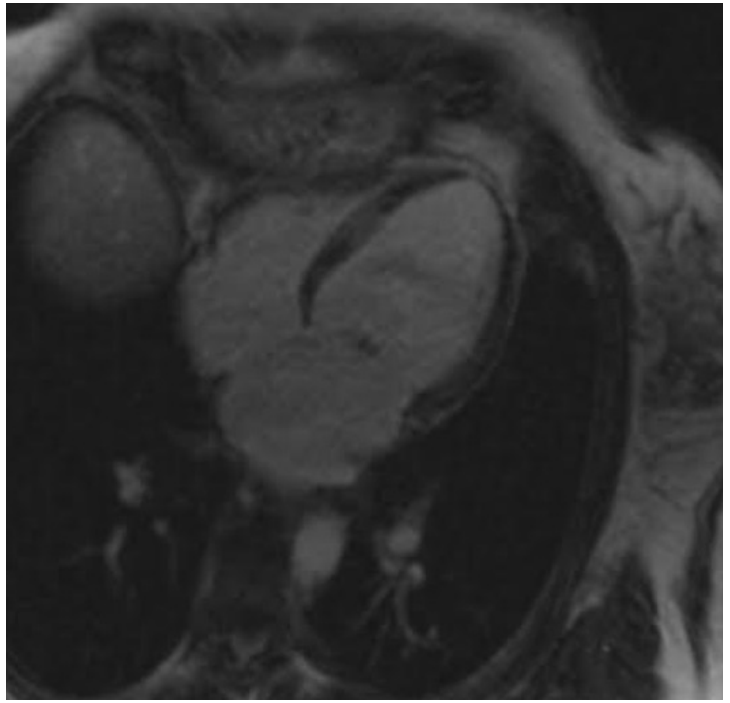




T1 mapping



LGE



Diagnosis and Management

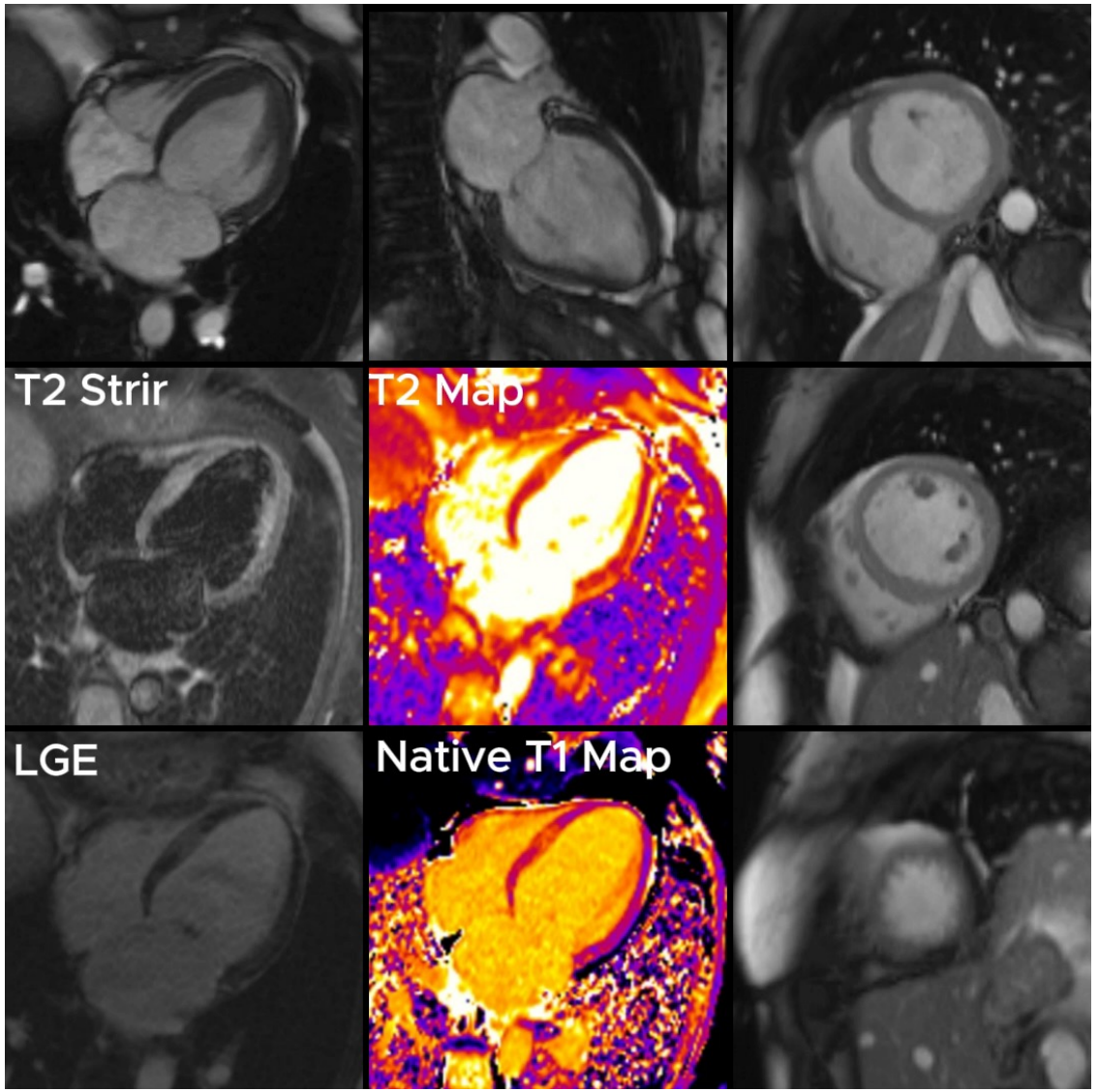
Initial Management: Admitted to Cardiac Care Unit,
Medications: Aspirin, Heparin, Metoprolol, Morphine

Diagnosis: Basal Type Takotsubo Syndrome (TTS) DD Recurrence of myocarditis

Further Investigations:

Invasive left heart catheterization: Considered but deferred (InterTAK Diagnostic Score of 55)

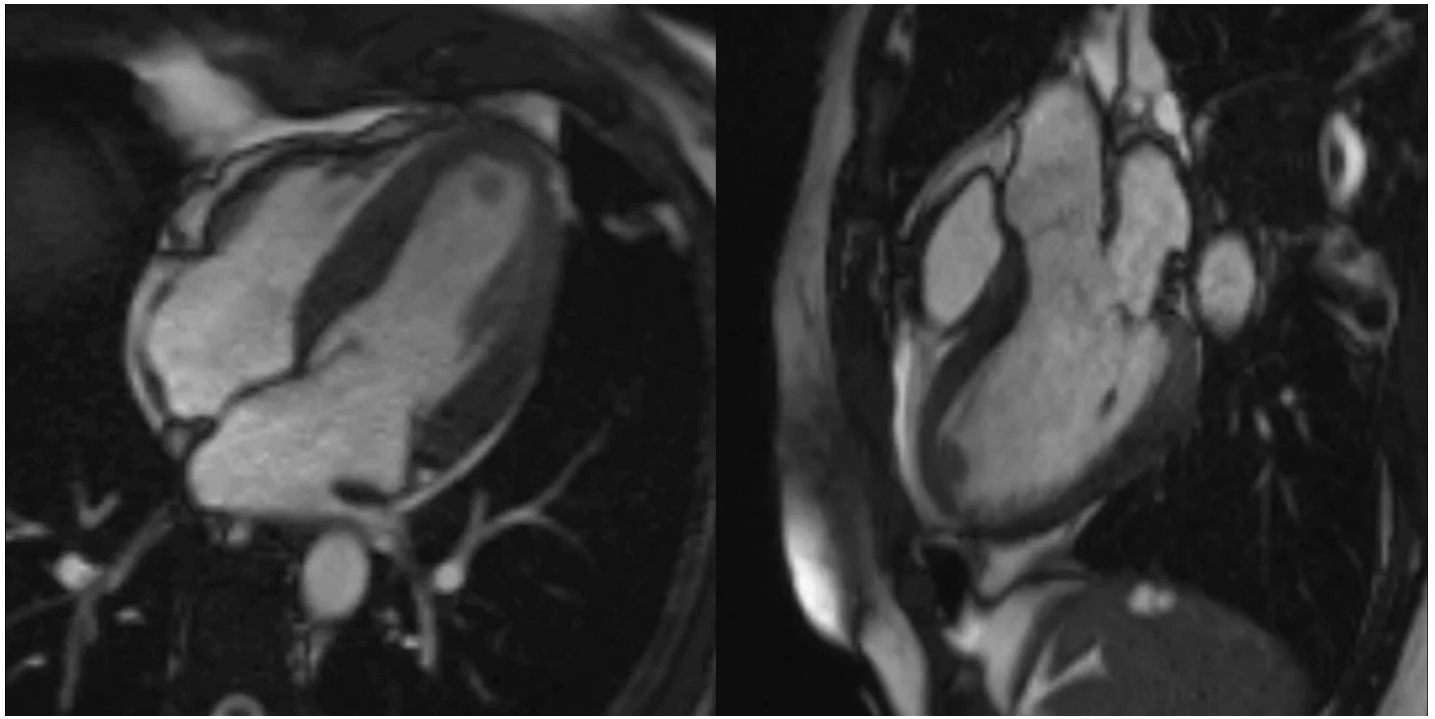
CMR: Confirmed basal TTS.



Continued Care and Discharge:

- Added Empagliflozin and Spironolactone
- Follow-up: 7 days later full recovery of LV function, discharged on Metoprolol & Telmisartan and advised to avoid stress

Follow-up



Two Months Later: Recurrent chest pain, hypertension, headaches, palpitations, and nausea, no clear trigger.

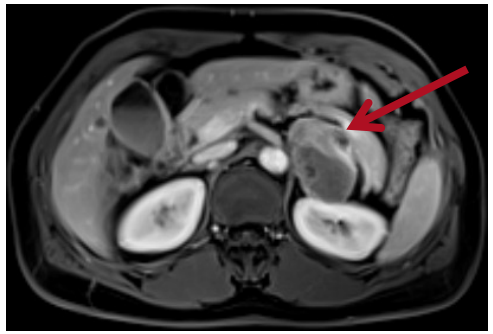
Findings:

- **ECG:** ST-segment elevation (V2 to V6).
- **Labs:** elevated cardiac biomarkers
- **TTE:** Apical ballooning, EF 42%, apical thrombus.

Further Investigations: CMR: Confirmed apical type Takotsubo Syndrome (TTS) recurrence.

New Suspicion: Pheochromocytoma- induced TTS.

Clonidine suppression test: positive → Elevated plasma metanephrine + normetanephrine → MRI of the adrenal glands



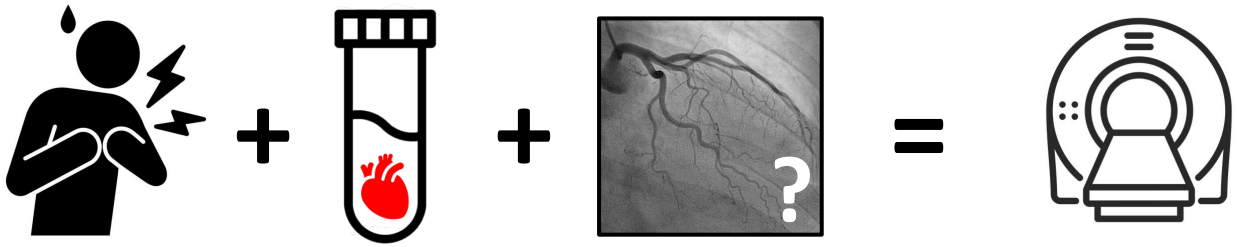
Laparoscopic resection of the adrenal mass: Histopathology confirmed **Pheochromocytoma**.

- **No Specific Stressor**: Chronic work stress without a specific trigger suggests the need to consider other TTS triggers like neurologic disorders and pheochromocytoma.

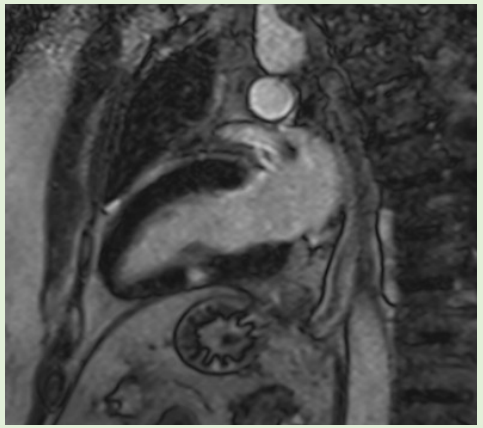
- **Imaging is Key:** CMR confirmed TTS and was preferred over coronary angiography due to its non-invasive nature, confirming myocardial edema and absence of LGE.

- **Pheochromocytoma as a Trigger:** Consider pheochromocytoma early, especially in younger or recurrent cases, to prevent recurrence and severe outcomes.

- **Re-evaluating Myocarditis:** The initial myocarditis diagnosis could represent early pheochromocytoma-induced TTS, supported by the disappearance of LGE in follow-up.

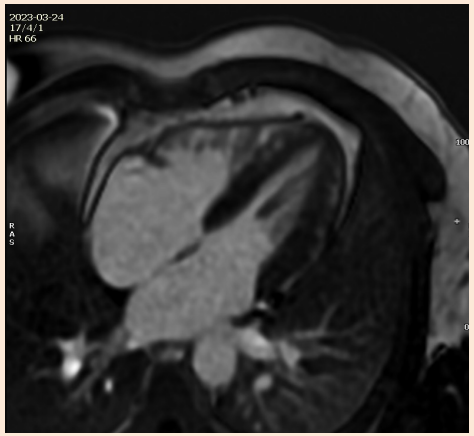


MINOCA



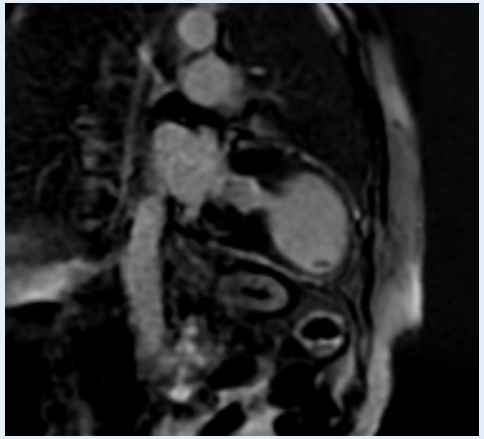
Œdème + rehaussement sous-endocardique ou transmural

MYOCARDITE



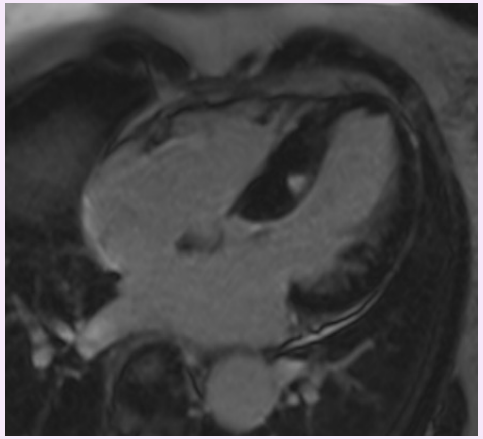
Œdème + rehaussement sous-épicardique

TAKOTSUBO



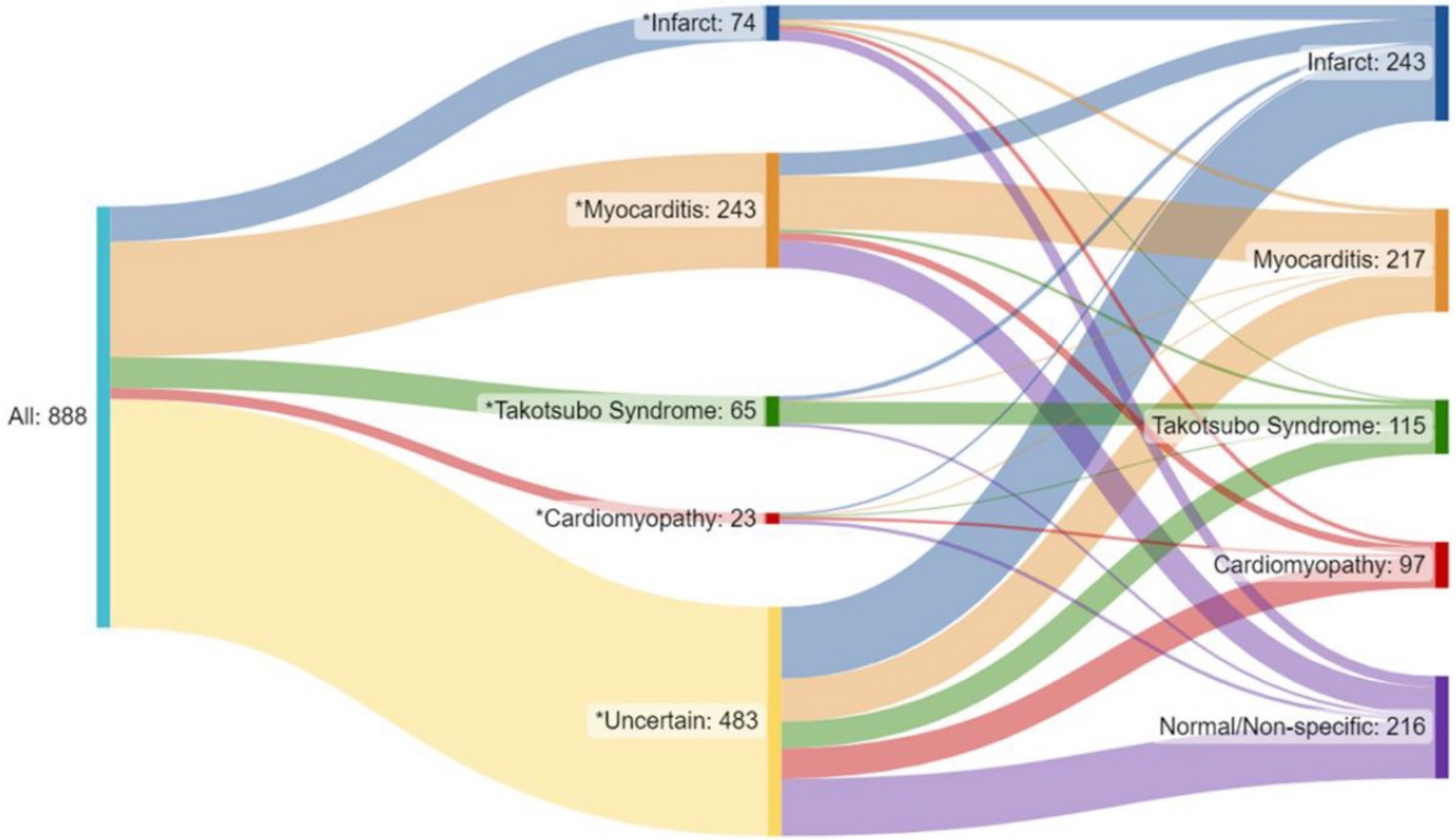
Œdème sans rehaussement

CARDIOMYOPATHIE



Œdème +/-
Rehaussement variable

Peut-on se fier aux examens de base uniquement?

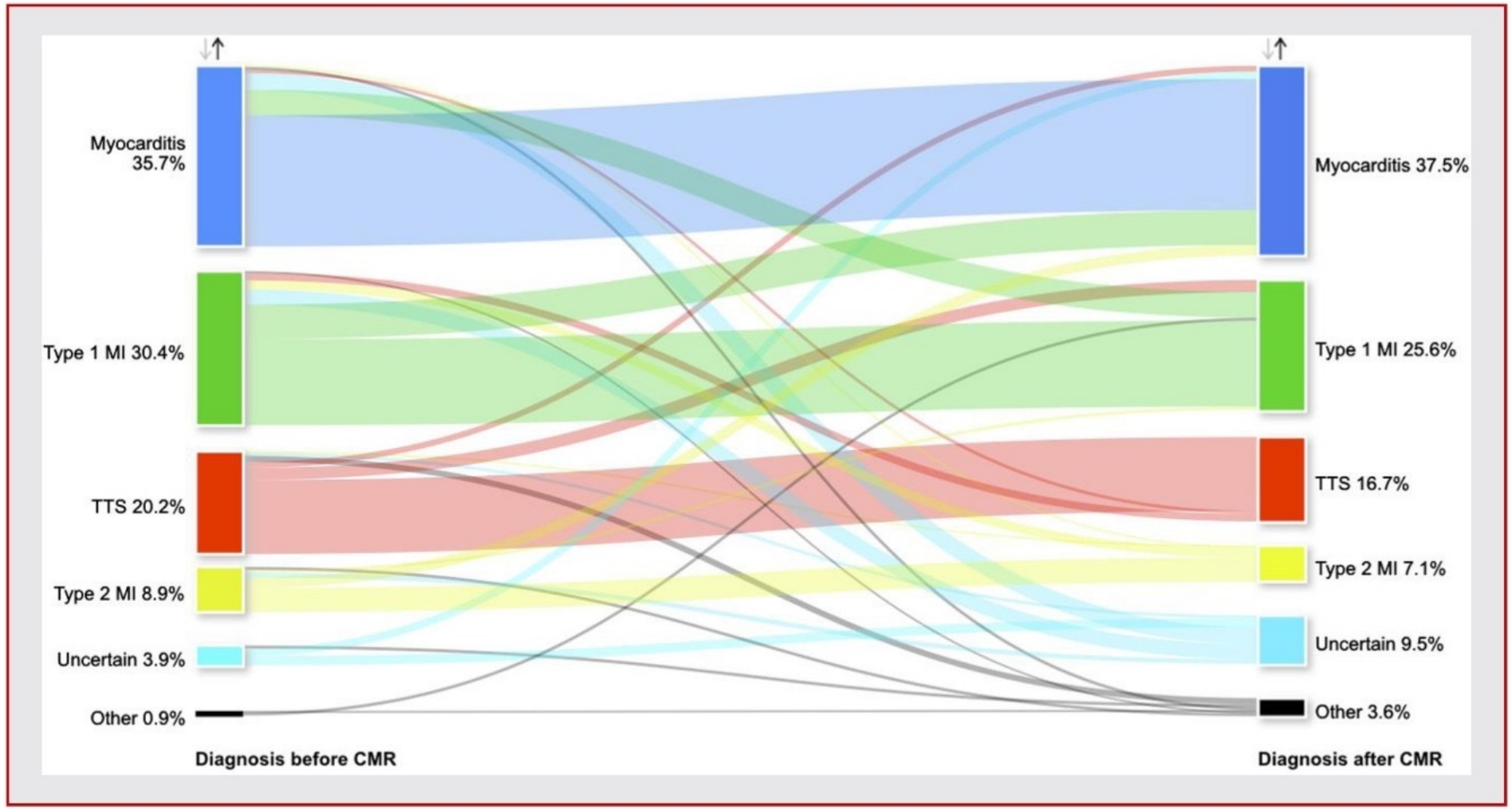


Liang et al. Eur Heart J Cardiovasc Imaging 2024 25, 8–15

Clinical Research

Diagnostic value of cardiac magnetic resonance imaging for myocardial infarction with non-obstructive coronary arteries: The CRIMINAL prospective registry

Charlotte Dagrenat [a], Amine Douair [b], Antoine Apert [b], Grégoire Range [c], Jean Louis Georges [d], Olivier Nallet [e], Nicolas Delarche [f], Nadine Ferrier [g], Jérôme Rischner [h], Jérôme Clerc [i], Edouard Naoum Nehmé [j], Antoine Boge [k], Franck Barbou [l], Christophe Jeannot [m], Regis Delaunay [n], Laurent Michel [o], Franck Goiorani [a], Philippe Couppie [a], Hende Madiot [p], Loïc Belle [b]...Pierre Leddet [a] * ✉



A

Post-CMR Diagnosis

Pre-CMR Diagnosis	Total Sample n = 204	Myocarditis	MI	TakoTsubo	Other CM	Normal CMR
	Myocarditis	34	23	5	4	7
	MI	10	6	8	6	1
	TakoTsubo	1	5	3	0	2
	Other CM	1	0	1	0	0
	Uncertain	8	19	2	8	50

B

Diagnosis			
	New Diagnosis	25%	29%
	No Change	34%	12%
	Clinical Impact Overall (n = 204)	No Change	Change in Management

Management

IRM diagnostique 70% des cas

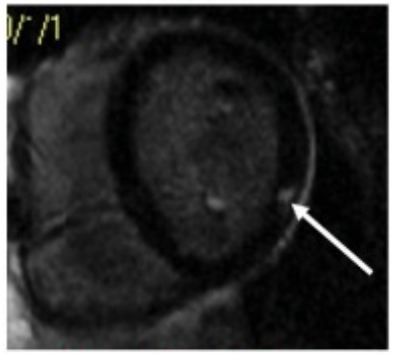
Changement diagnostic / PEC 66%

Meilleure précision < 15 jours (84% vs 67%)

Dastidar et al., JACC cardiovas Imaging 2017 Oct;10(10 Pt A):1204-1206.

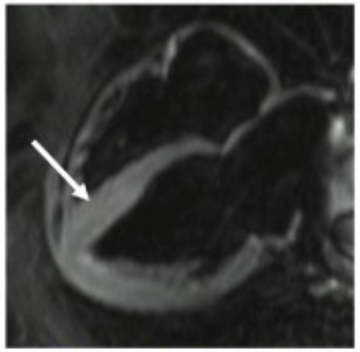
Diagnostic Yield of CMR in ACS With Non-Obstructive Coronary Arteries		
	Scan Interval <14 Days	Scan Interval ≥14 Days
Troponin <211 ng/L	76%	53%
Troponin ≥211 ng/L	94%	72%

MINOCA

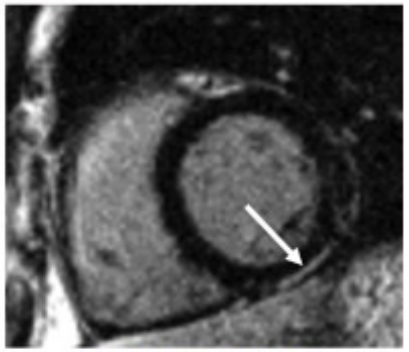


Myocardial Infarction

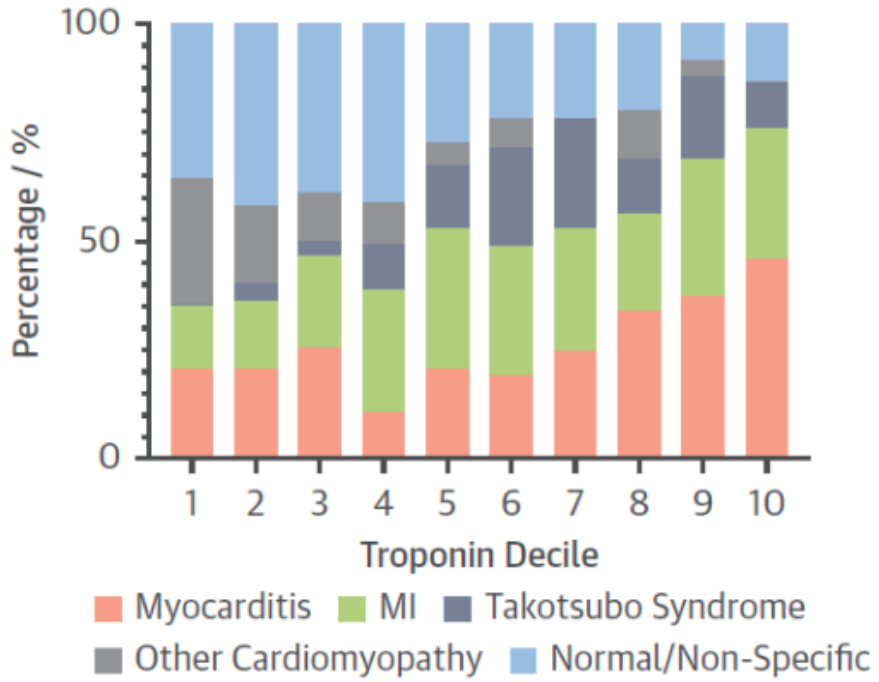
Non-MINOCA



Takotsubo Syndrome

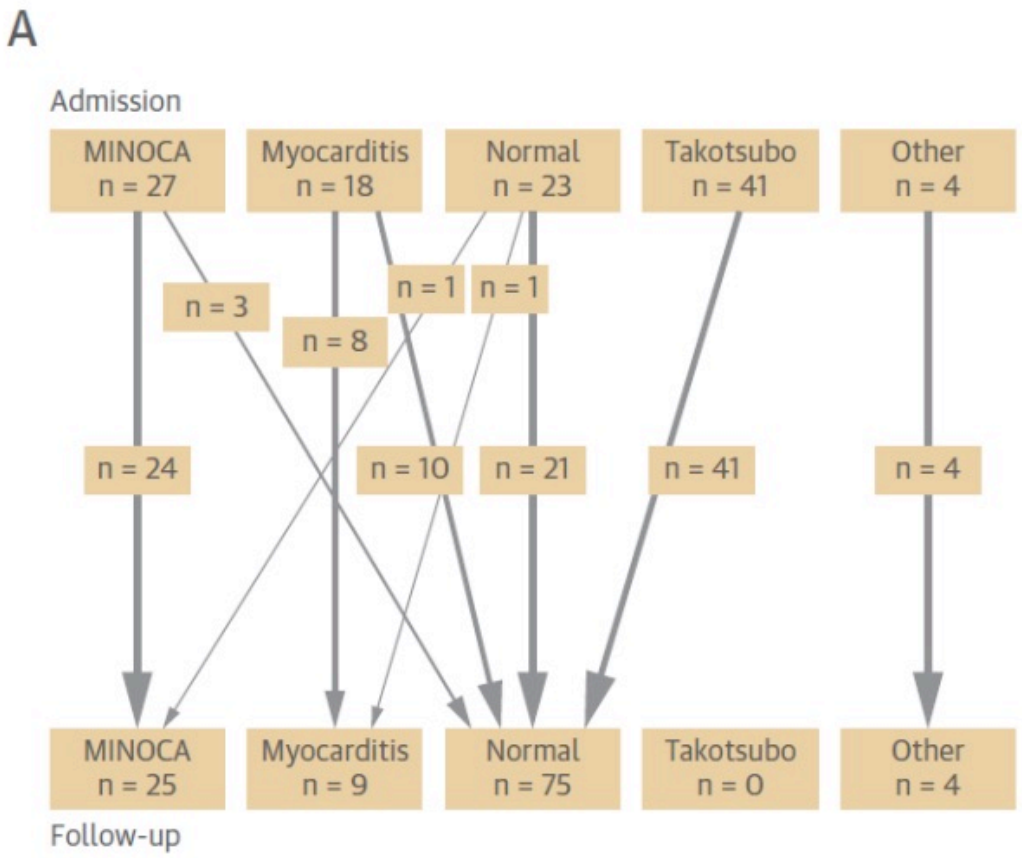


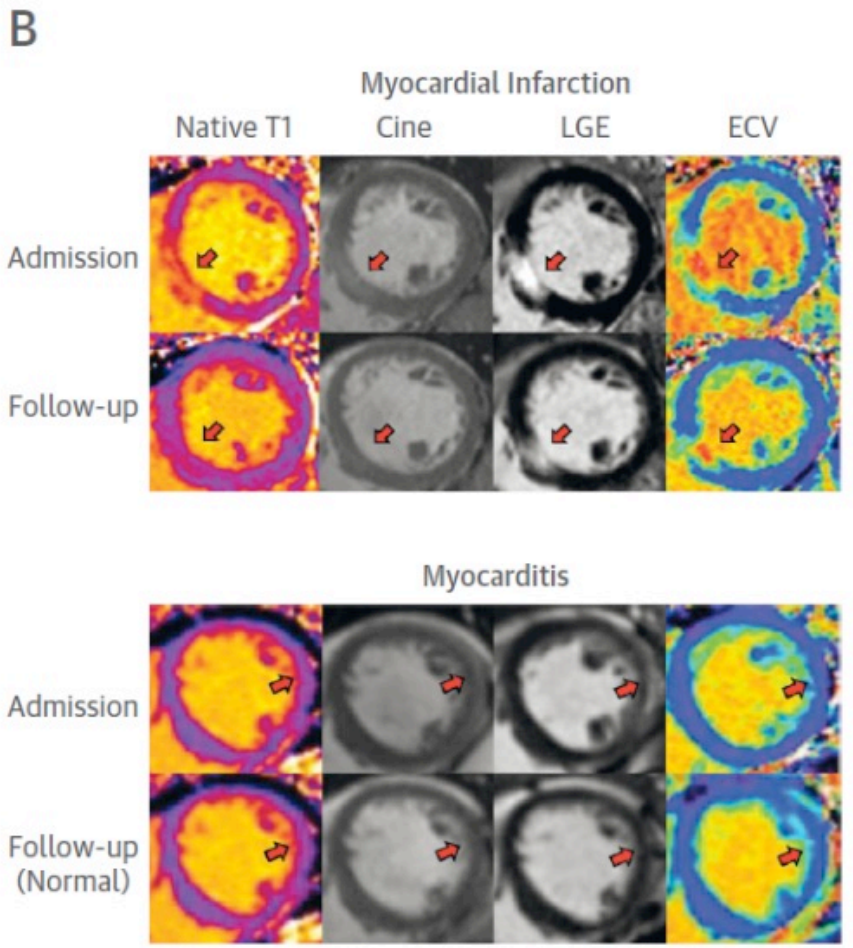
Acute myocarditis



Williams et al. J Am Coll Cardiol Img 2022;15(9):1578-1587

6 MOIS

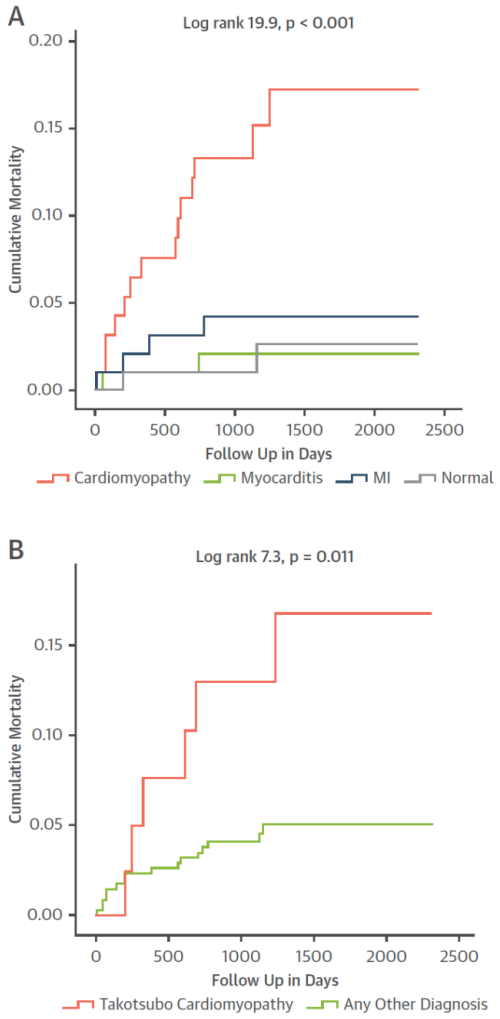


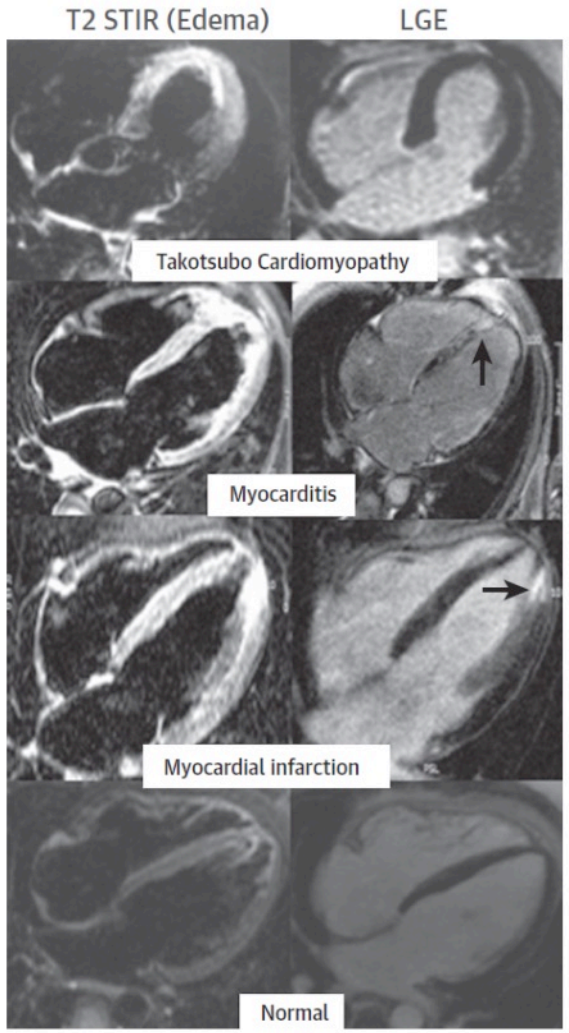


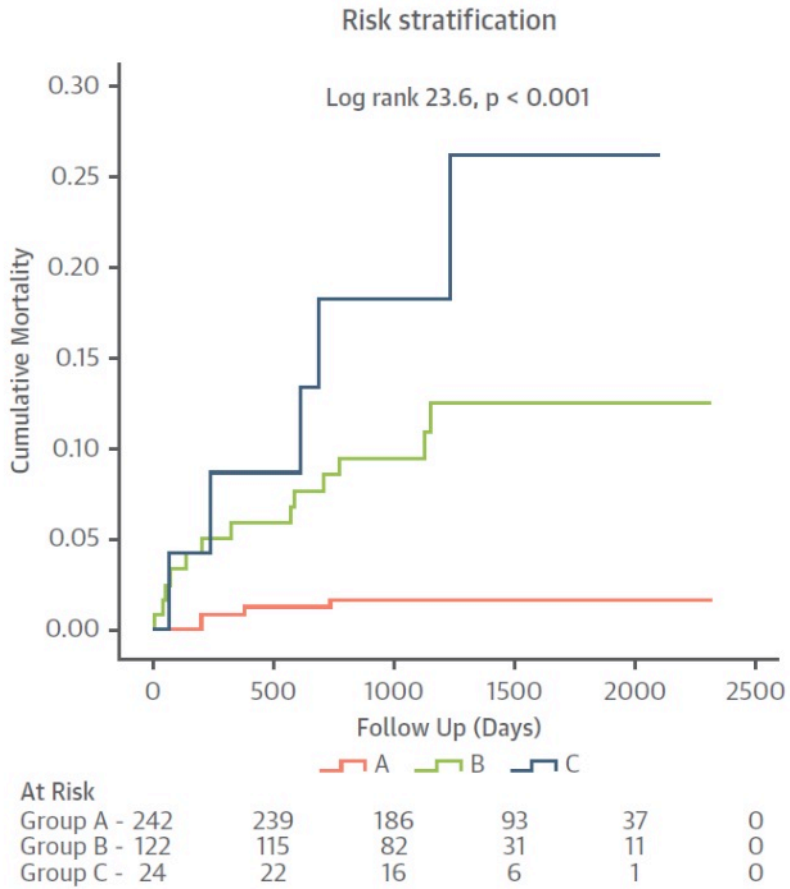
Nickander et al. JACC Cardiovasc Imaging 2023 Jan;16(1):128-129.

DELAI OPTIMAL : ENTRE 48h ET 15 JOURS

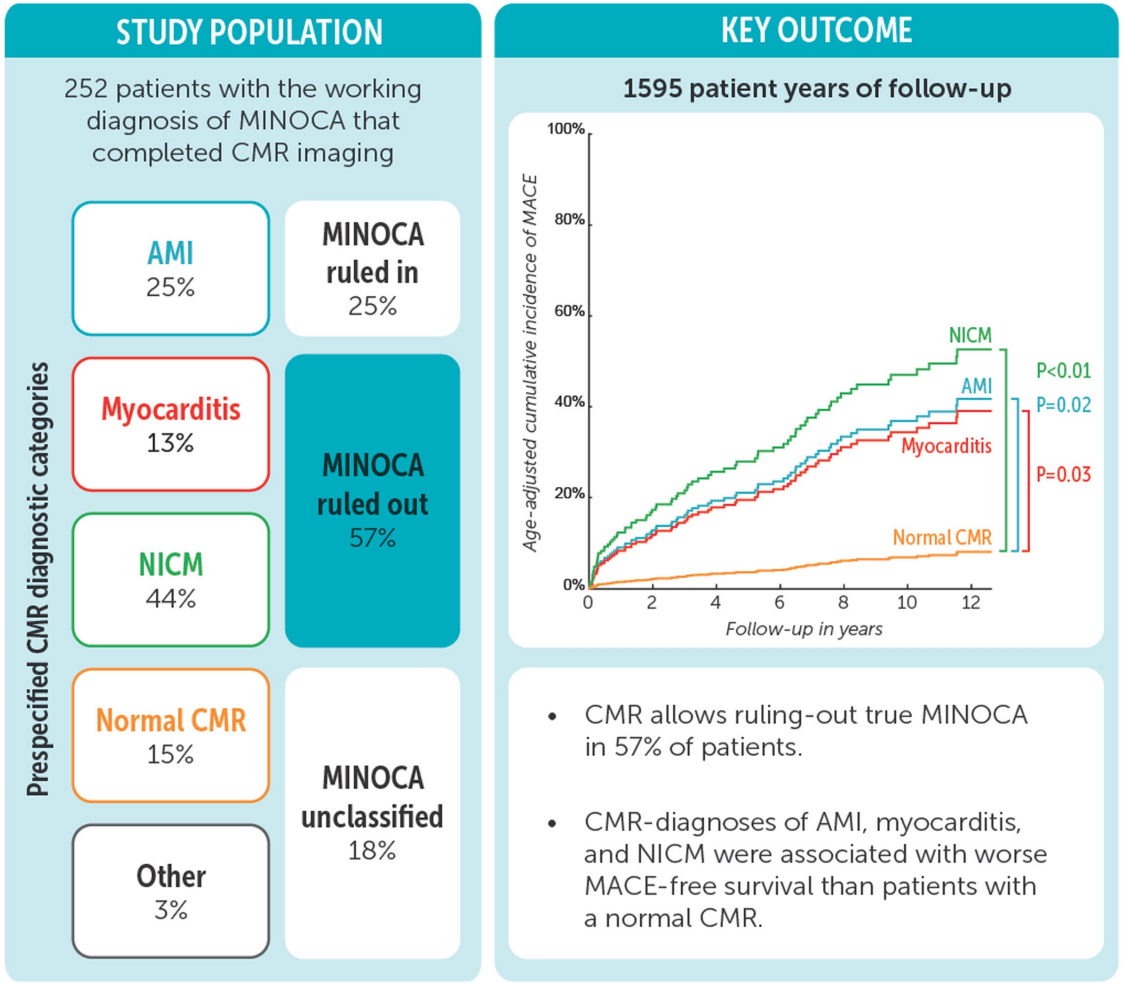
Au-delà du diagnostic, quel est l'impact pronostique ?



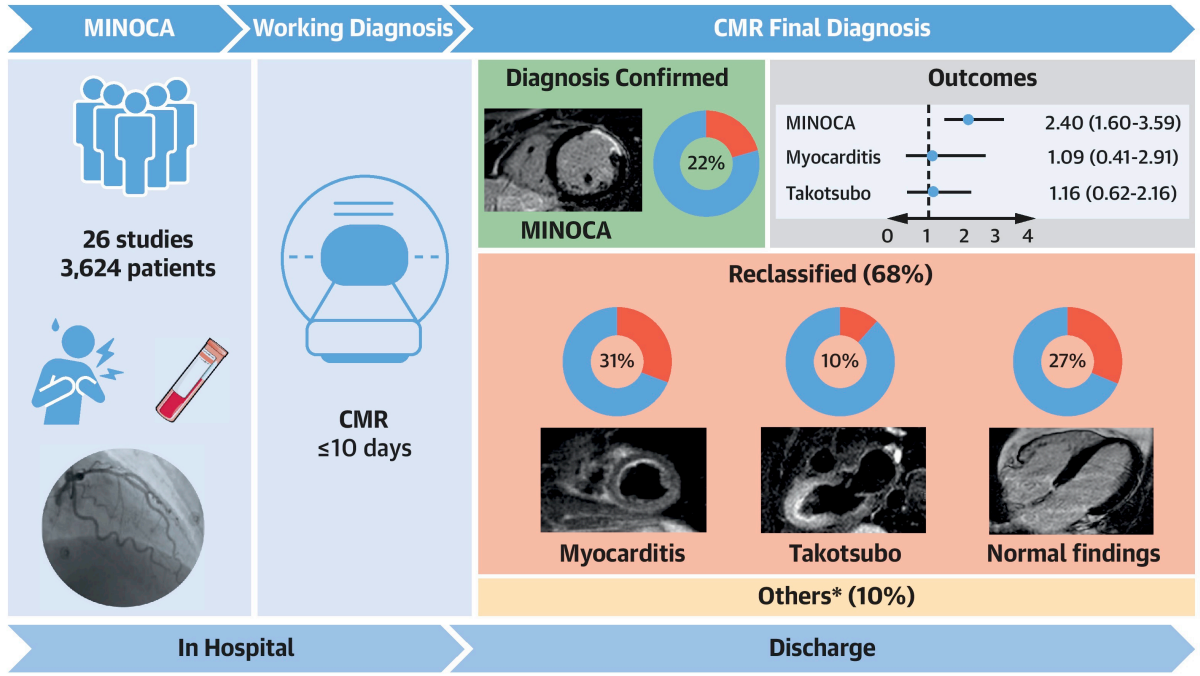




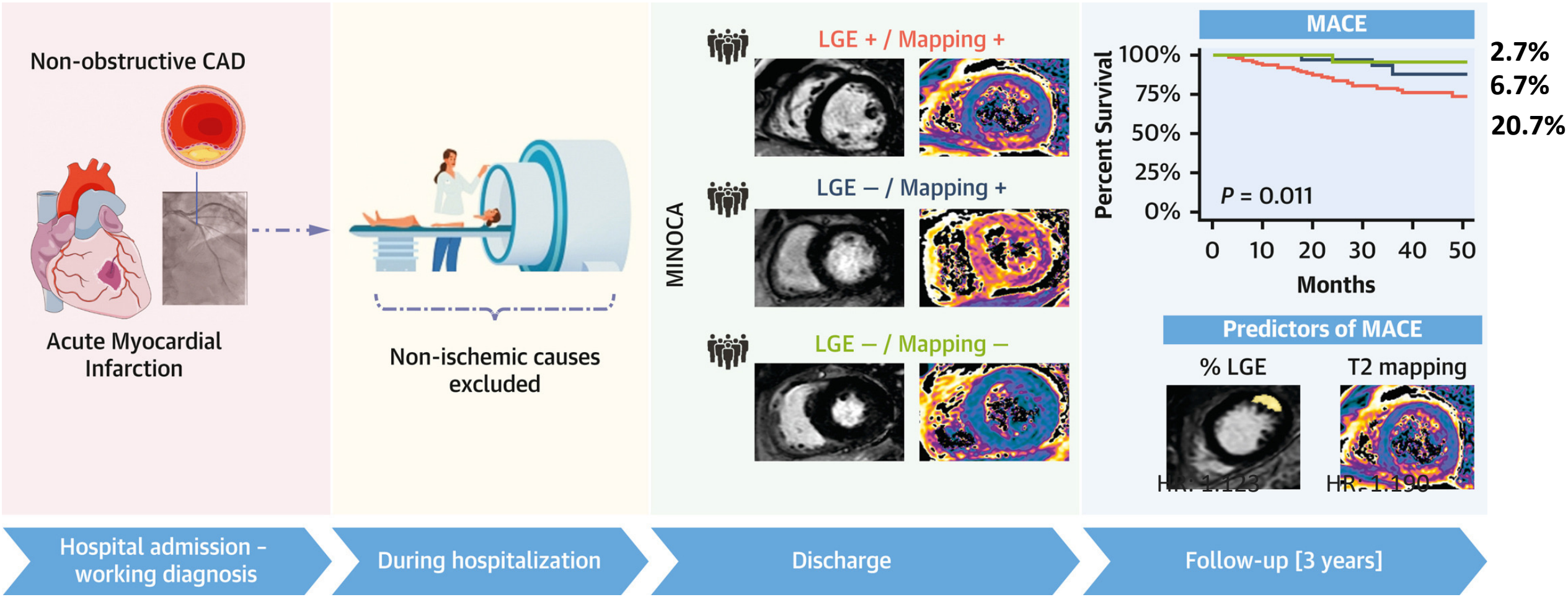
Dastidar et al., JACC cardiovas Imaging 2019 Oct;12(10):1973-1982.



Konst et al. Circ Cardiovasc Imaging 2023 Aug;16(8):e014454

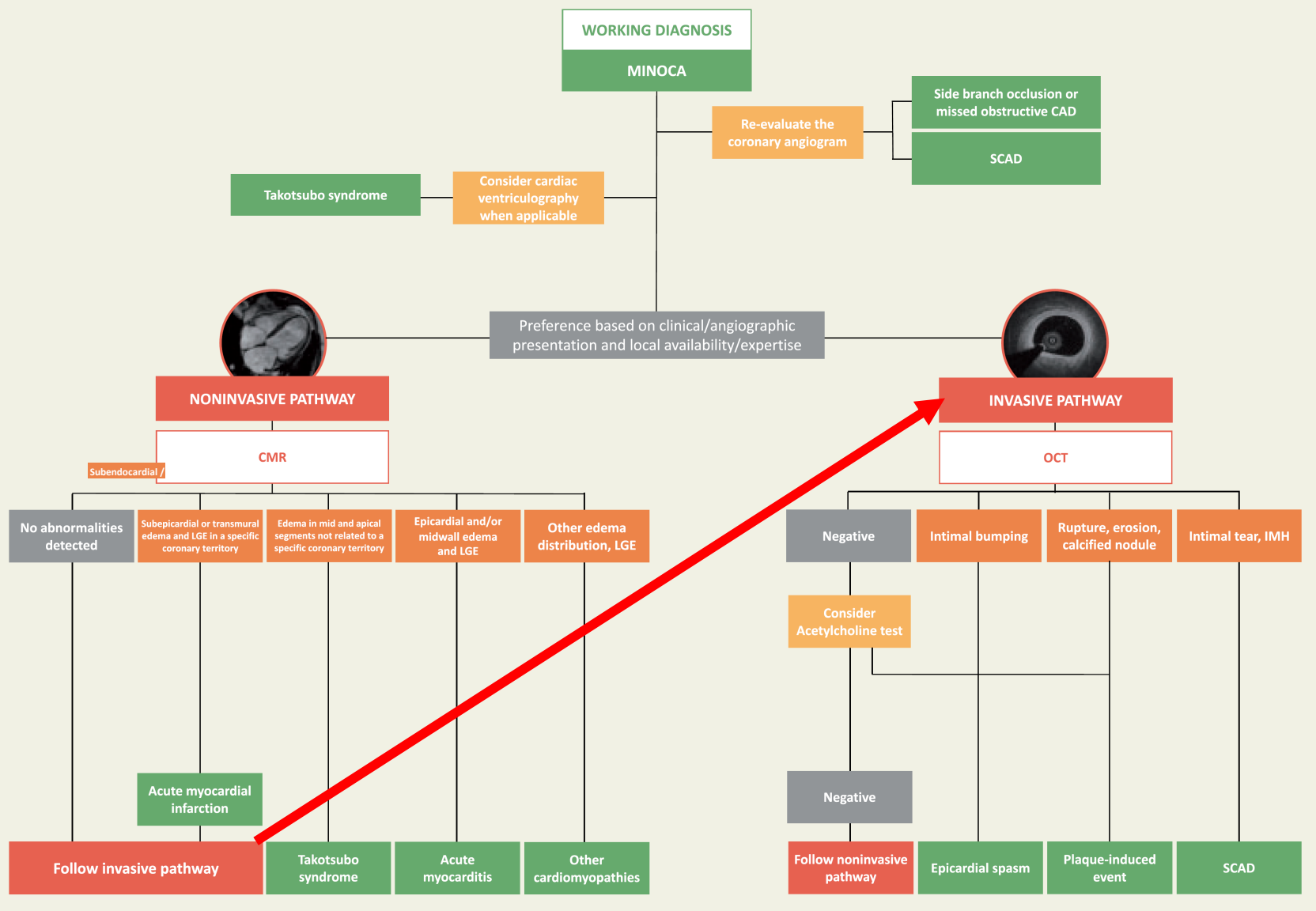


Mileva N, et al. J Am Coll Cardiol Img. 2023;16(3):376-389.



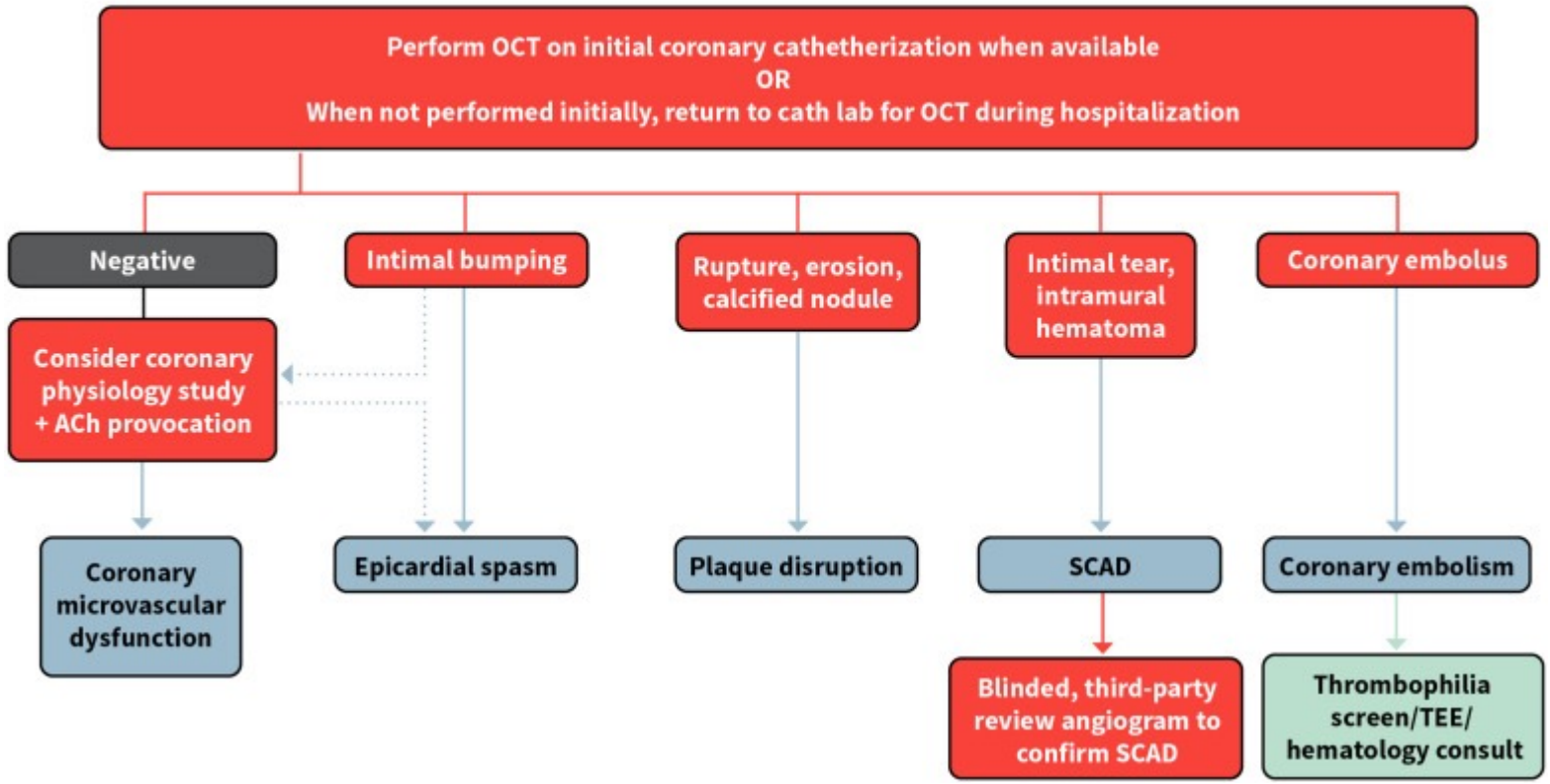
Bergamaschi et al., JACC cardiovasc Imaging 2023 Jul 10;S1936-878X(23)00242-5.

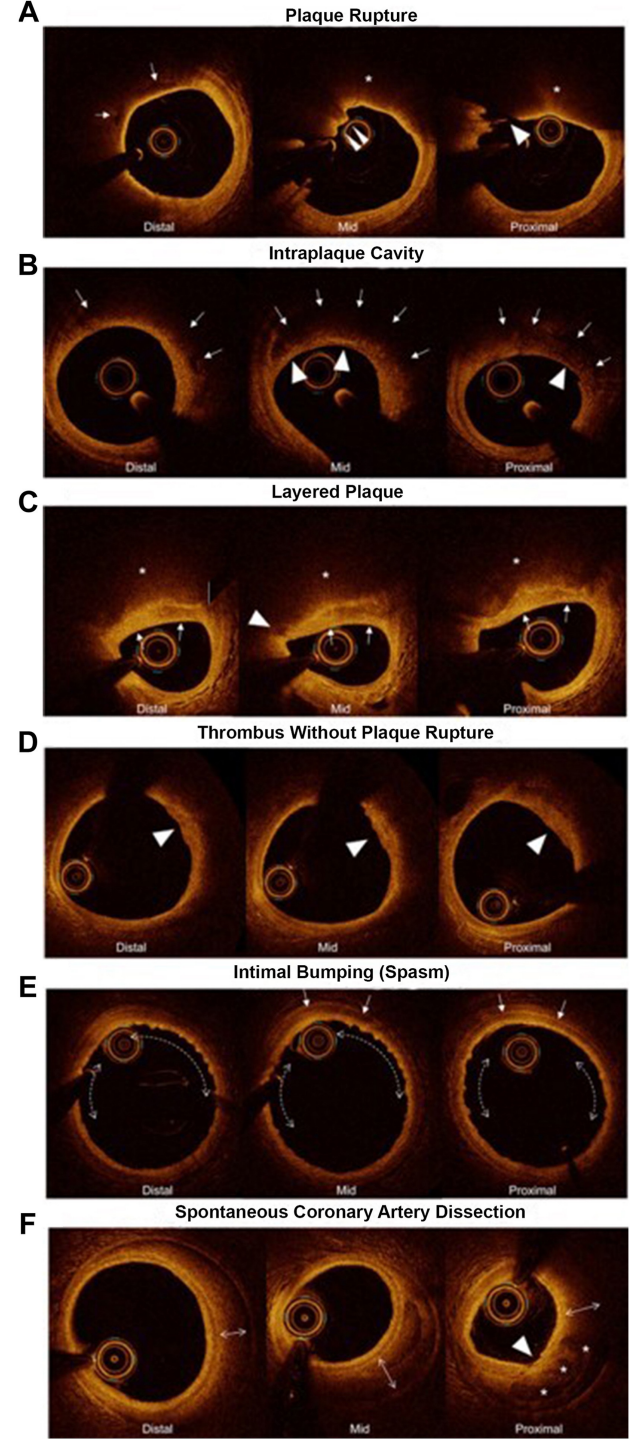
Et une fois le diagnostic de MINOCA confirmé?

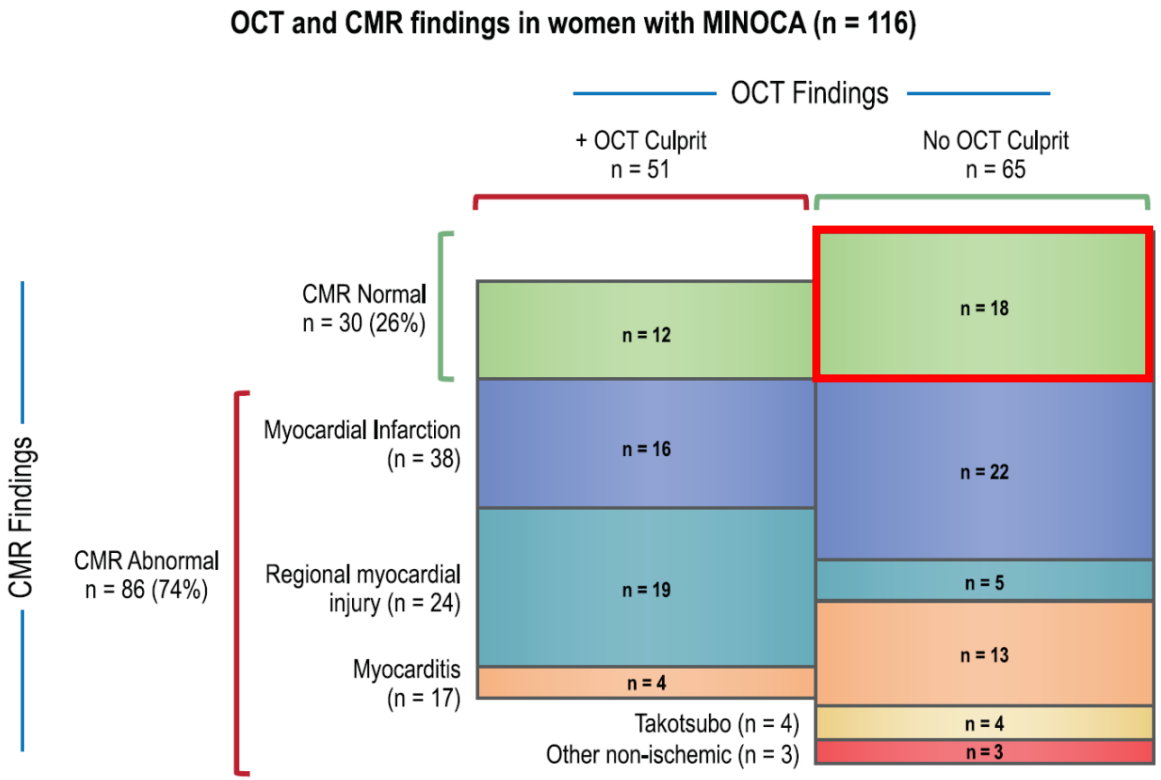


Occhipinti, Bucciarelli-Ducci, Capodanno. Eur Heart J Acute Cardiovasc Care. 2021 Oct 1;10(7):813-822.

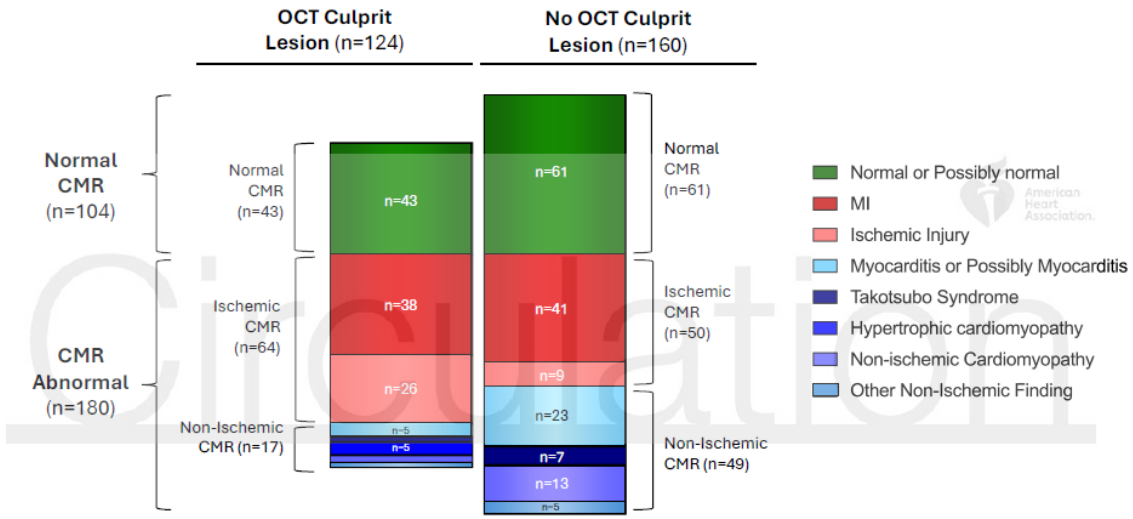
DIAGNOSTIC PATHWAY PART 2 - INVASIVE TESTING



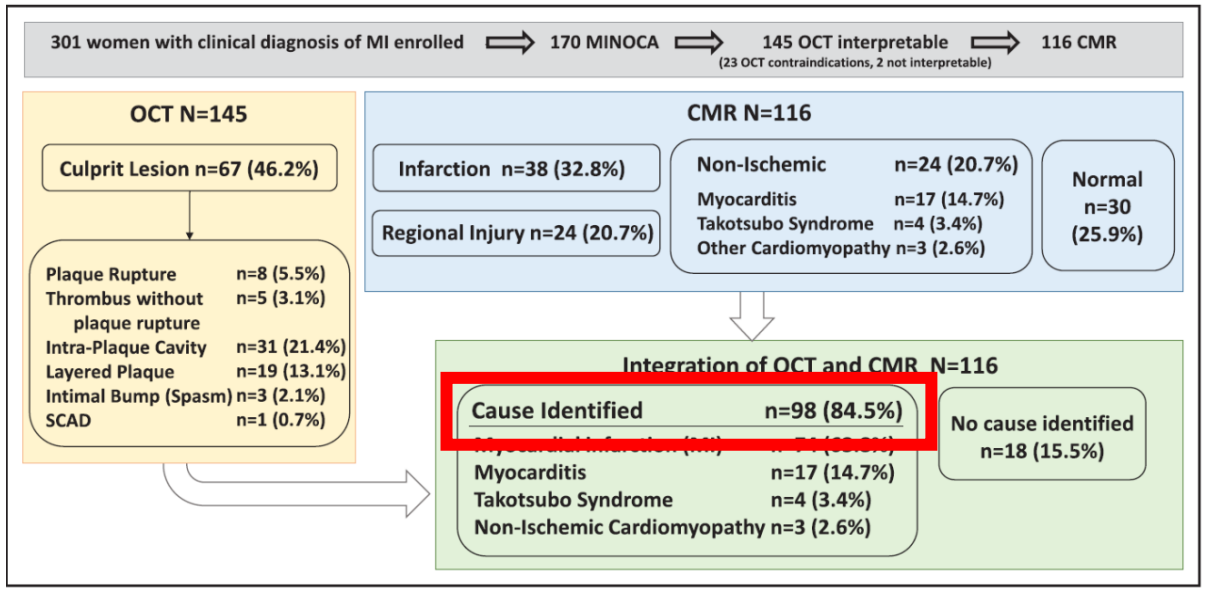




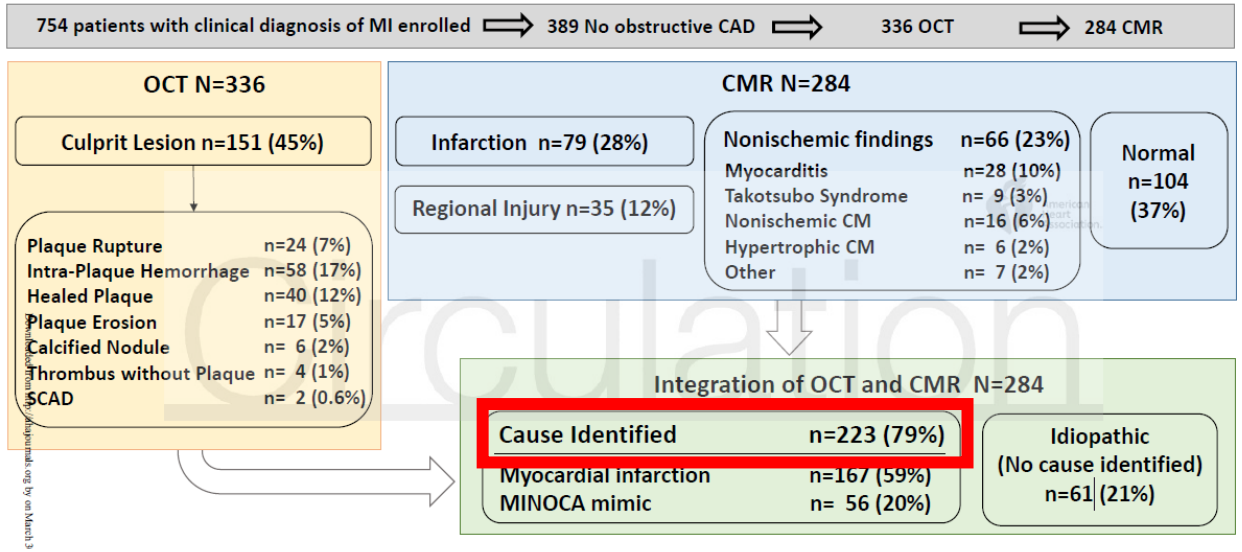
Reynolds et al., Circulation. 2021;143:624–640.



Reynolds et al., Circulation. 2026



Reynolds et al., Circulation. 2021;143:624–640.



Reynolds et al., Circulation. 2026

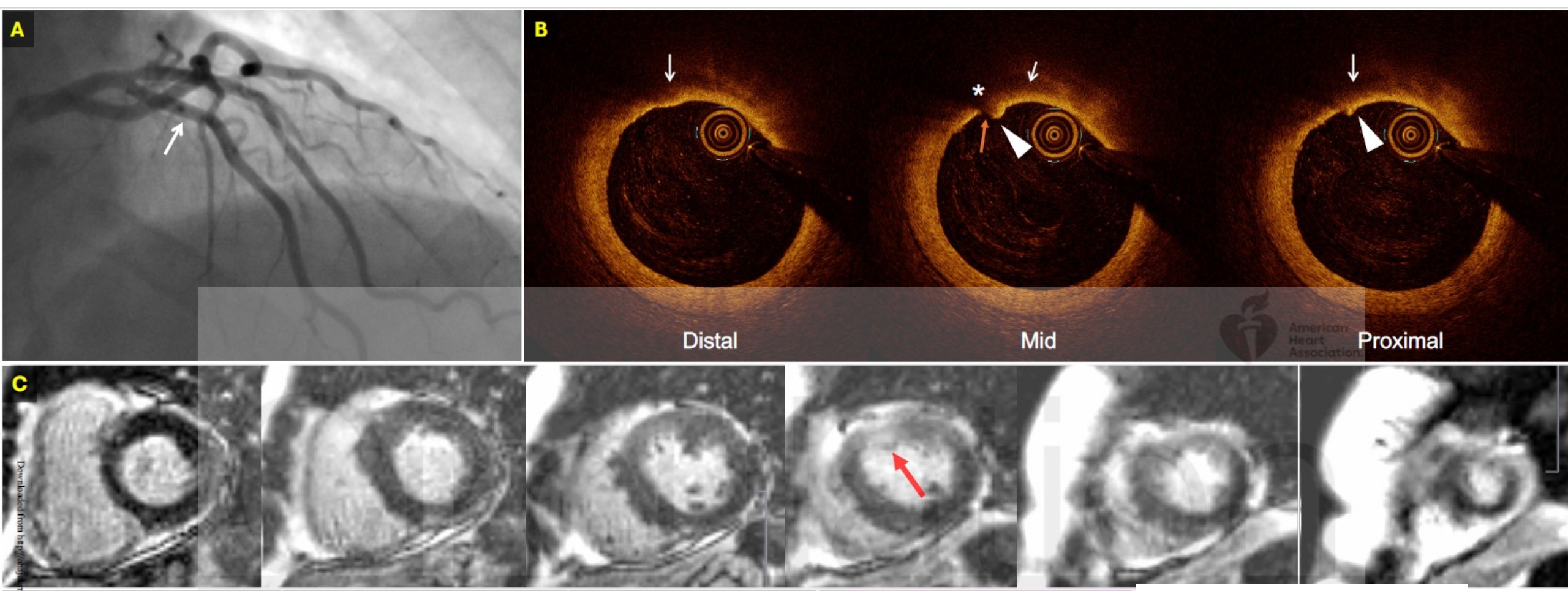


Figure 6. Plaque rupture with regional ischemia in the LAD distribution

Panel A – Coronary angiography with no obstructive CAD. The culprit lesion is located in the proximal LAD (white arrow).

Panel B - A low-intensity mural thrombus with an irregular surface is observed (white triangles) and a part of the thrombus protrudes into the lumen. The underlying plaque shows strong attenuation, consistent with the lipidic plaque (asterisk). The rupture site is marked with an orange arrow. Overall, these findings suggest plaque rupture within a relatively small amount of lipidic plaque in a large lumen, followed by mural thrombus formation.

Panel C - LV SAX LGE imaging demonstrates focal subendocardial infarct (red arrow) in mid anterior segment. The remaining segments demonstrate no infarction.

Panel D - LV short axis STIR images demonstrate increased signal within basal anterior, basal anteroseptal, basal inferoseptal, mid anterior, mid anteroseptal, mid inferoseptal, apical septal, apical anterior and apical inferior segments consistent with diffuse edema.

Panel E - AHA 16 segment native T1 bull's-eye representing corresponding increase in T1 mapping ranging from 1141 ms in basal, 1264 ms in mid and 1231 ms in the apical segments (normal T1 mapping < 1100 ms at 1.5 T scanner).

Reynolds et al., Circulation. 2026

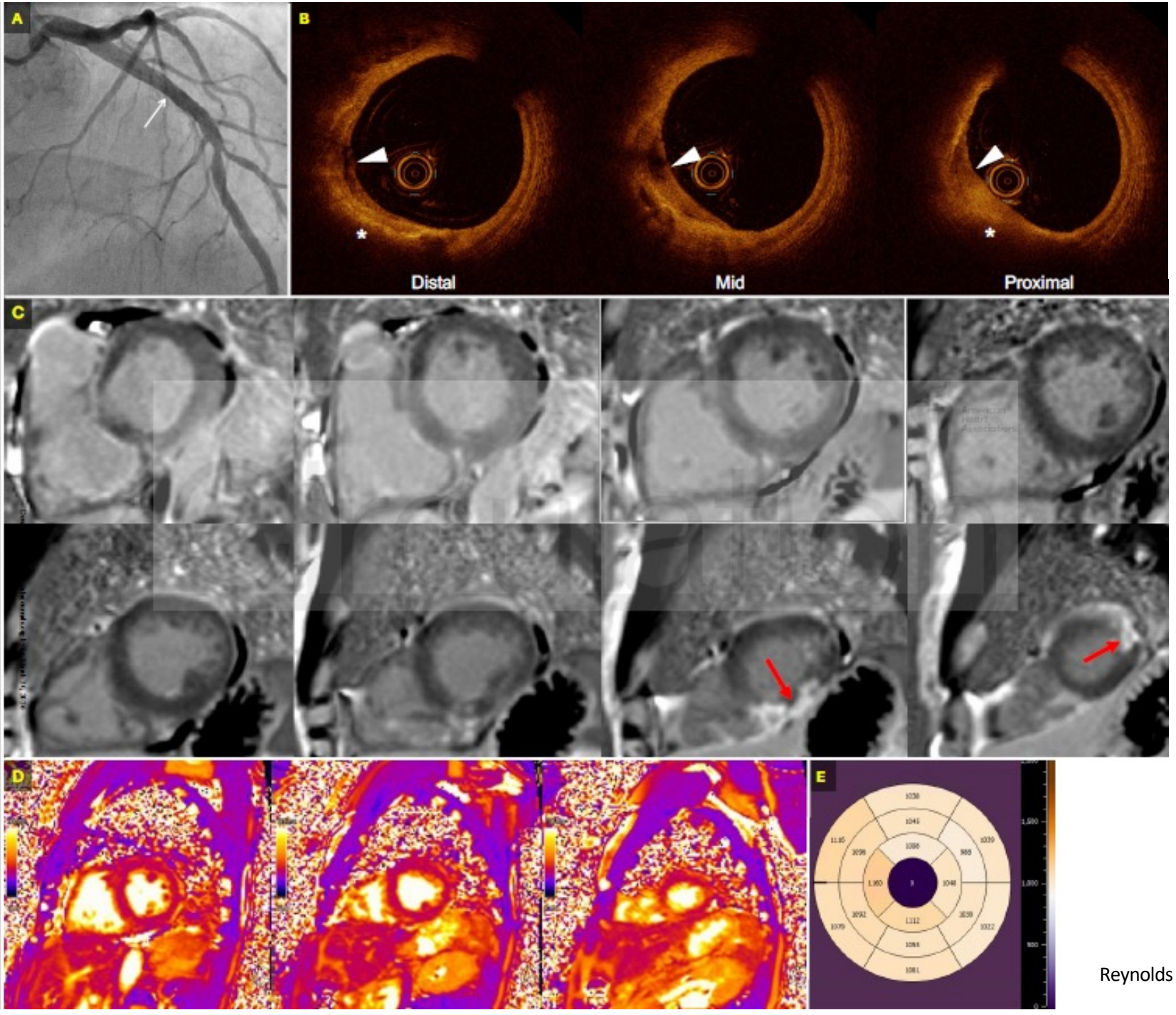


Figure 7. Healed plaque in the LAD leading to acute myocardial infarction

Panel A - Coronary angiography revealed a large LAD coronary artery that wraps around the apex to supply the inferior wall. A healed plaque culprit lesion was identified in the mid LAD.

Panel B- A layered structure with very low and heterogeneous signal intensity (white triangles) is observed overlying the lipid-rich plaque (asterisk), consistent with a thrombus in the healing stage.

Panel C: LV SAX PSIR LGE shows transmural subendocardial LGE in the apical septal, inferior and lateral segments suggestive of Acute MI.

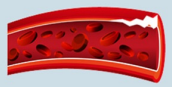
Panel D- T2 mapping shows (arrows) edema in the apical septal, inferior and lateral segments.

Panel E - T1 Native Mapping bull's eye shows corresponding high T1 signal

Reynolds et al., Circulation. 2026

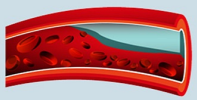
DIAGNOSTIC FINAL DE MINOCA

MINOCA CAUSES AND TREATMENTS

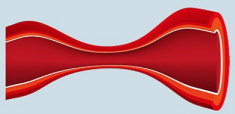


SCAD:
- ASA
- DAPT
- BB
- ACEI*

CMD:
- Angina control (BB, CCB, nitrates, ranolazine)
- ACEI and statins*



EMBOLISM:
- Anticoagulation
- Interatrial shut closure
- Specialty referral



VASOSPASM:
- CCB and/or nitrates

PLAQUE RUPTURE:
- DAPT
- Statins
- BB?

UNKNOWN CAUSE:
- ACEI
- Statins
- BB?

NONPHARMACOLOGICAL TREATMENTS

CARDIOVASCULAR REHABILITATION

CARDIOVASCULAR RISK FACTOR MANAGEMENT

PSYCHOSOCIAL SUPPORT

Pacheko et al., Canadian Journal of Cardiology, 40, 953-968

AHA/ACC CLINICAL PRACTICE GUIDELINE

2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR Guideline for the Evaluation and Diagnosis of Chest Pain: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

1	B-NR	1. In patients with acute chest pain and myocardial injury who have nonobstructive coronary arteries on anatomic testing, CMR with gadolinium contrast is effective to distinguish myopericarditis from other causes, including myocardial infarction and nonobstructive coronary arteries (MINOCA).[1-6]
1	B-NR	2. In patients with acute chest pain with suspected acute myopericarditis, CMR is useful if there is diagnostic uncertainty, or to determine the presence and extent of myocardial and pericardial inflammation and fibrosis.[7-12]

Gulati et al., Circulation 2021 Nov 30;144(22):e368-e454

ESC European Society of Cardiology
European Heart Journal (2023) **00**, 1–107
https://doi.org/10.1093/eurheartj/ehad191

ESC GUIDELINES

2023 ESC Guidelines for the management of acute coronary syndromes

Developed by the task force on the management of acute coronary syndromes of the European Society of Cardiology (ESC)

Recommendations	Class[a]	Level[b]
In patients with a working diagnosis of MINOCA, CMR imaging is recommended after invasive angiography if the final diagnosis is not clear.[544,545]	I	B
Management of MINOCA according to the final established underlying diagnosis is recommended, consistent with the appropriate disease-specific guidelines.[546,550,552]	I	B
In all patients with an initial working diagnosis of MINOCA, it is recommended to follow a diagnostic algorithm to determine the underlying final diagnosis.	I	C

Byrne et al., Eur Heart J 2023 Aug 25;ehad191

• *L’IRM cardiaque est l’examen clé pour **reclasser le diagnostic** de MINOCA, avec un rendement élevé lorsqu’elle est réalisée précocement.*

• *Elle permet une **caractérisation tissulaire précise**, distinguant infarctus, myocardite, takotsubo et autres étiologies.*

• *Au-delà du diagnostic, elle apporte une **stratification pronostique essentielle**, orientant la prise en charge et le suivi.*