

**MINOCA:  
Comment je fais le diagnostic?**

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Plateau de Cardiologie Interventionnelle**

# DÉCLARATION DE LIENS D'INTÉRÊT AVEC LA PRÉSENTATION

**Intervenant : Gerbaud, Edouard, Bordeaux**

- Je déclare les liens d'intérêt suivants :
- Consultant: Terumo France SAS, AstraZeneca
- Bourse de recherche: Fédération Française de Cardiologie

# MINOCA DEFINITION

## **Syndrome coronarien aigu**

Douleur thoracique

Modifications ECG

Elévation de la troponine

Troubles de la cinétique segmentaire: ventriculographie, ETT, IRM

**Coronaires angiographiquement normales ou sub-normales (QCA < 30% ou QCA entre 30 et 50%)**

**Absence d'étiologie évidente** lors de la présentation clinique

# Causes potentielles d'élévation de la troponine

- (1) Coronary causes
  - Plaque rupture or erosion
  - Coronary artery spasm
  - Spontaneous coronary dissection
  - Acute aortic dissection with coronary extension
  - Coronary microvascular disorders
  - Spontaneous coronary thrombosis–thrombophilia disorders
  - Coronary emboli
  - Sympathomimetic agents—cocaine, methamphetamines
- (2) Non-coronary causes
  - (a) Associated with cardiac disorders
    - Myocarditis
    - Takotsubo cardiomyopathy
    - Cardiomyopathies
    - Cardiac trauma
    - Strenuous exercise
    - Tachyarrhythmias
    - Cardiotoxins—chemotherapeutic agents
  - (b) Associated with extra-cardiac disorders
    - Stroke
    - Pulmonary embolism
    - Sepsis
    - Adult respiratory distress syndrome
    - End-stage renal failure

# ALGORITHME PRISE EN CHARGE

## MINOCA

(Universal AMI Criteria + no angiographic stenosis  $\geq 50\%$  + no overt causes at presentation)

### Invasive investigations

- Review for subtle 'missed' angiography findings (dissection, emboli or plaque disruption)
- Intracoronary nitrates (coronary spasm)

Consider:

- LV Gram or echocardiography (Takotsubo/other cardiomyopathies\*)
- IVUS/OCT (plaque rupture/erosion, dissection\*\*)
- Pressure/Doppler Wire (microvascular dysfunction)
- Provocative spasm testing (coronary spasm; preferably not in the acute phase of AMI\*\*\*)

### Laboratory assays

Consider:

- Type-2 MI (Hb, CRP, WBC, SO<sub>2</sub>)
- D-dimer (pulmonary embolism)
- Thrombophilia screen
- BNP

### MINOCA aetiologic diagnosis confirmed

Type-2 MI  
Plaque disruption  
Dissection  
Takotsubo  
Epicardial or microvascular spasm  
Coronary thromboembolism

### Diagnosis not confirmed

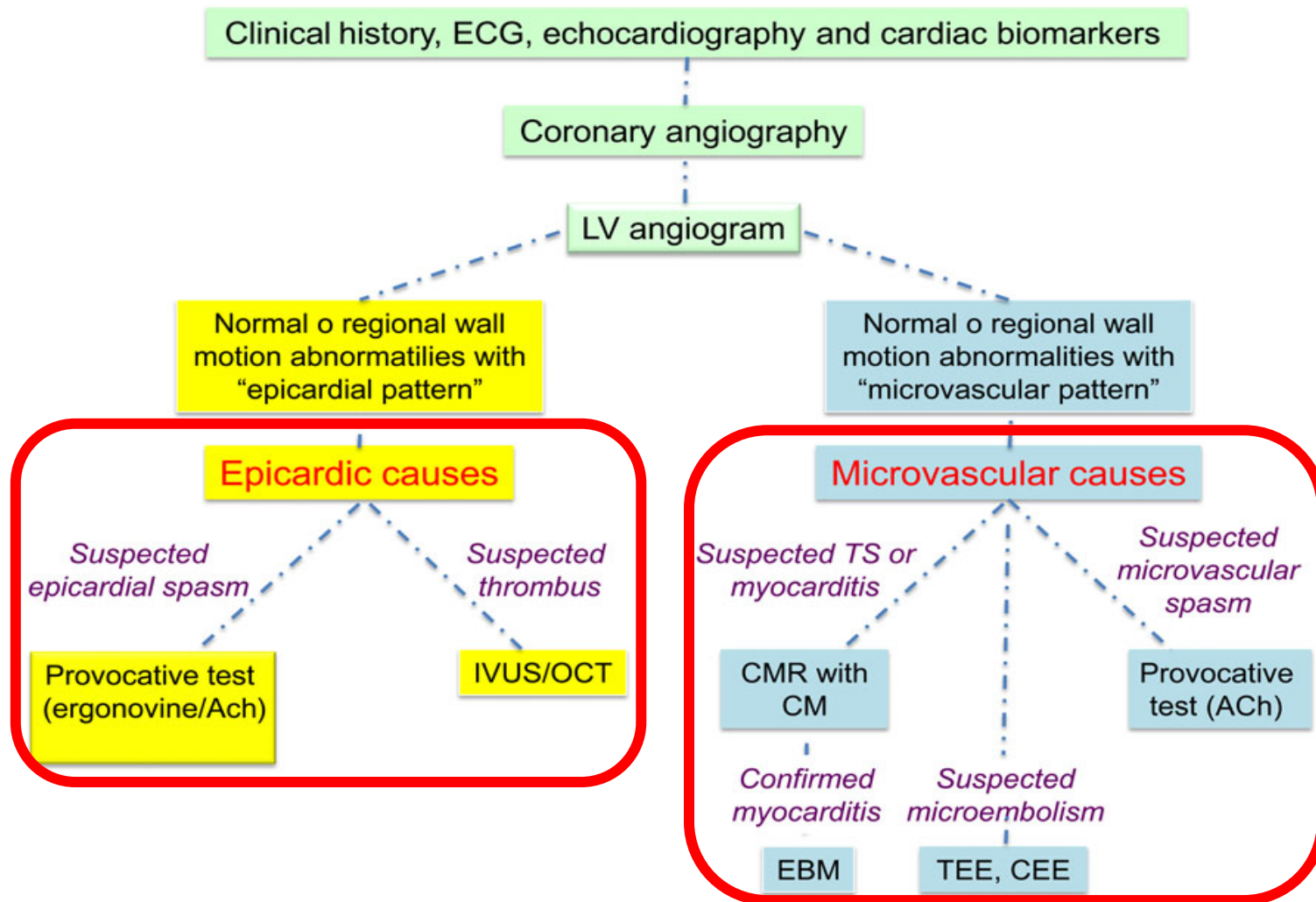
#### Cardiac MRI

- LGE (myocarditis\*\*\*\*)
- AMI

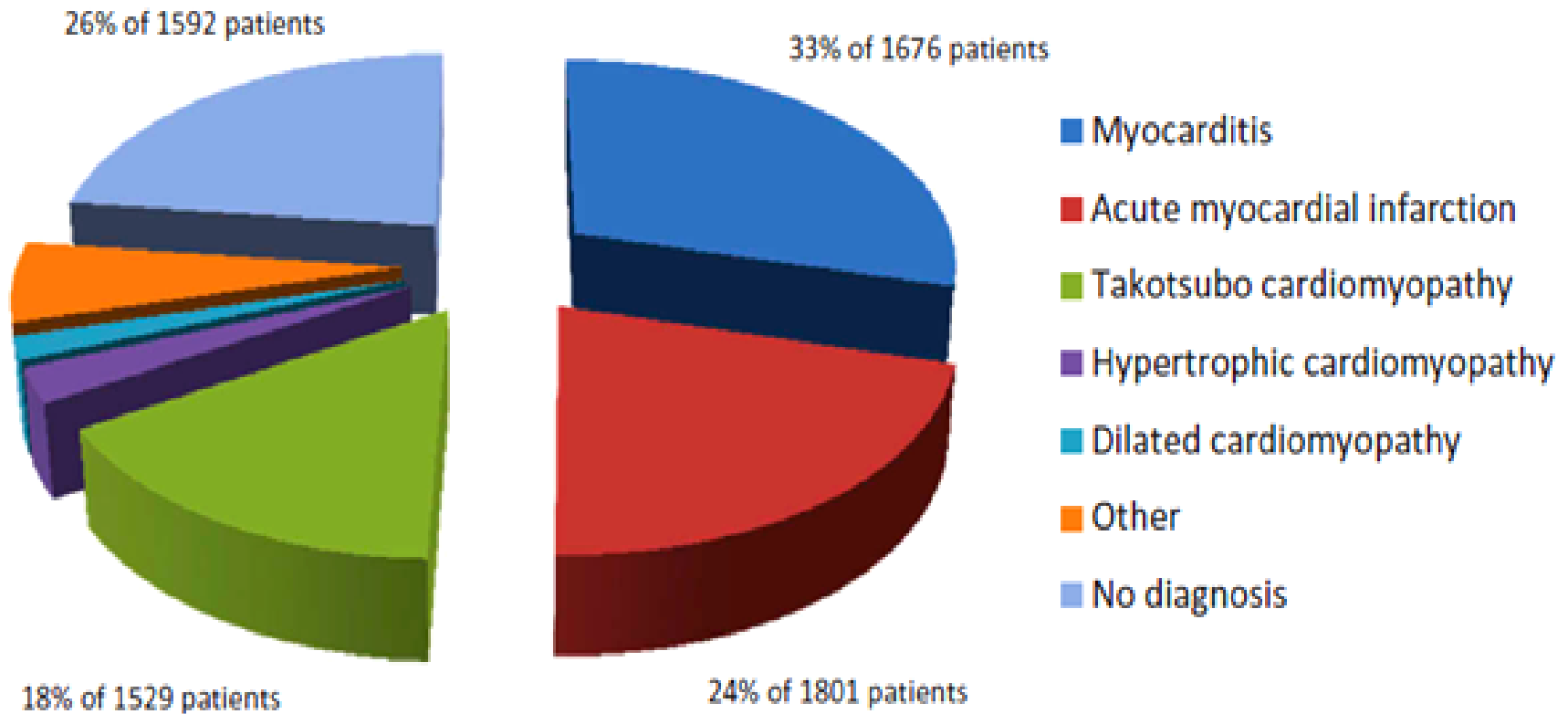
#### TEE

- Cardioembolism

# ALGORITHME PRISE EN CHARGE



# ETIOLOGIES



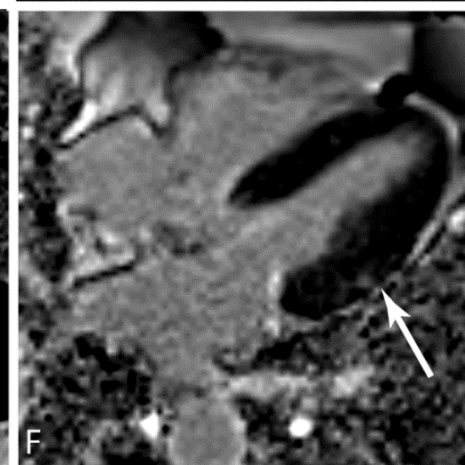
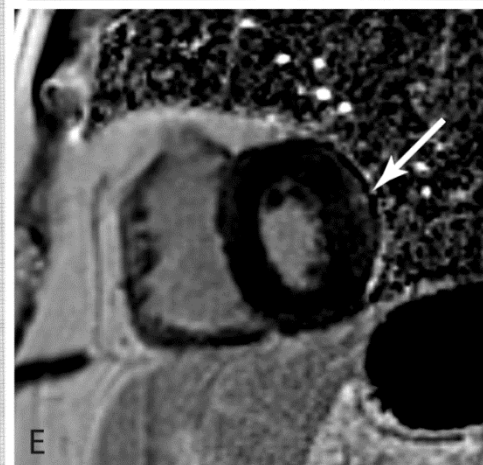
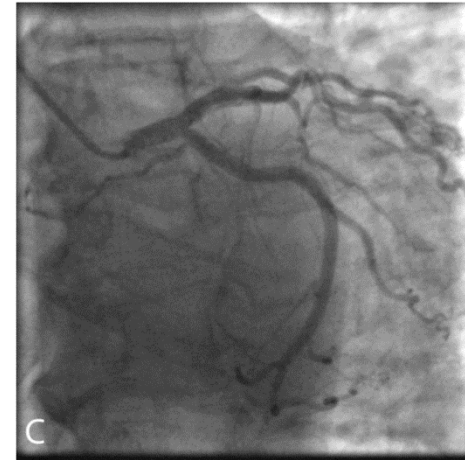
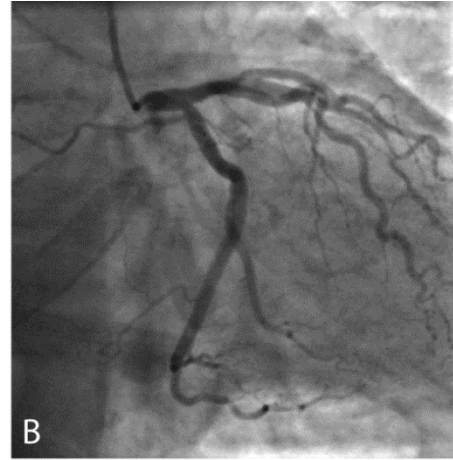
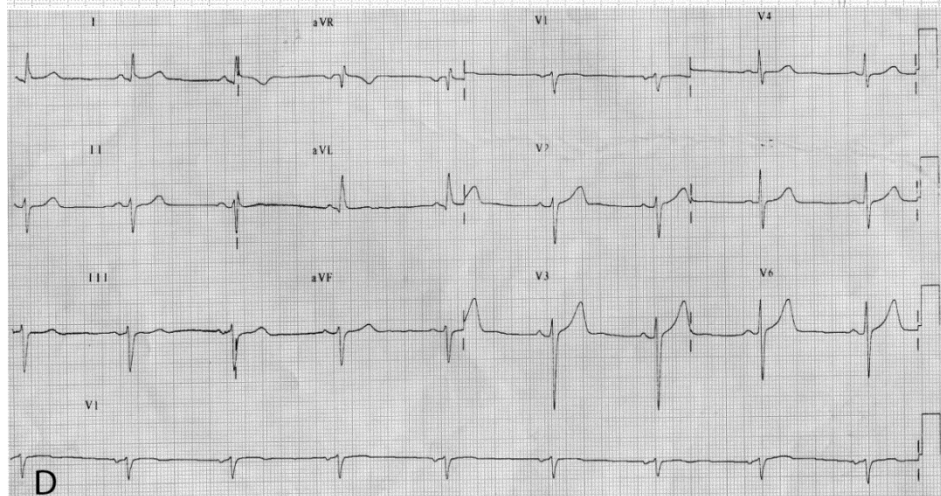
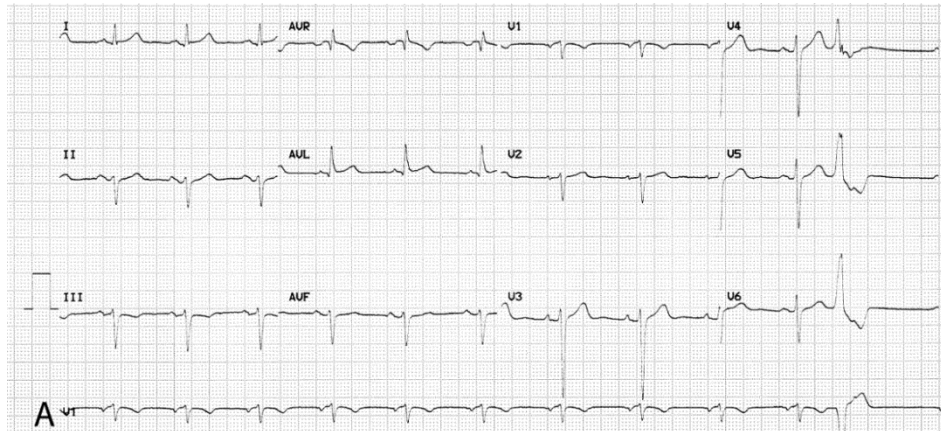
# IRM Cardiaque

	All patients (N=130)	AMI (N=37)	Myocarditis (N=34)	ABS (N=28)	Normal CMR (N=30)
Focal or regional edema	65 (50)	21 (56.8)	28 (82.4)	16 (57.1)	0 (0)
Patchy	12	0	12	0	0
Subendocardial	14	14	0	0	0
Subepicardial or septal	10	0	10	0	0
Transmural	29	7	6	16	0
MVO (1)	11 (8.5)	11 (29.7)	0 (0)	0 (0)	0 (0)
Abnormal late gadolinium enhancement					
None	58 (44.6)	0 (0)	0 (0)	28 (100)	30 (100)
Subendocardial	20 (15.4)	20 (54.1)	0 (0)	0 (0)	0 (0)
Transmural	12 (9.2)	12 (32.4)	0 (0)	0 (0)	0 (0)
Subepicardial	39 (30)	5 (13.5)*	34 (100)	0 (0)	0 (0)
Centromyocardial	1 (0.8)	0 (0)	0 (0)	0 (0)	0 (0)
Number of segments with LGE (2)	1 (0-3)	2 (1-2.5)	4 (2.75-5)	0	0
Disease location					
Anterior	39 (30)	20 (54.1)	19 (55.9)	0 (0)	0 (0)
Inferior	20 (15.4)	8 (21.6)	12 (35.3)	0 (0)	0 (0)
Lateral and inferior	31 (23.8)	9 (24.3)	22 (64.7)	0 (0)	0 (0)
One disease location	57 (43.8)	37 (100)	20 (58.8)	0 (0)	0 (0)
Multiple disease location	42 (32.3)	0 (0)	14 (41.2)	28 (100)	0 (0)
Segmental functional abnormalities					
Number of patients	56 (43.1)	14 (37.8)	14 (41.2)	28 (100)	0 (0)
Located in area of LGE	22 (16.9)	14 (37.8)	8 (23.5)	0 (0)	0 (0)
Located in remote lesions	34 (26.2)	0 (0)	6 (17.6)	28 (100)	0 (0)
Pericardial effusion	8 (6.1)	1(2.7)	7 (20.6)	0 (0)	0 (0)

Gerbaud E. et coll. Cardiac magnetic resonance imaging for the diagnosis of patients presenting with chest pain, raised troponin and unobstructed coronary arteries. *Int J Cardiovasc Imaging*. 2012;28(4):783-94.



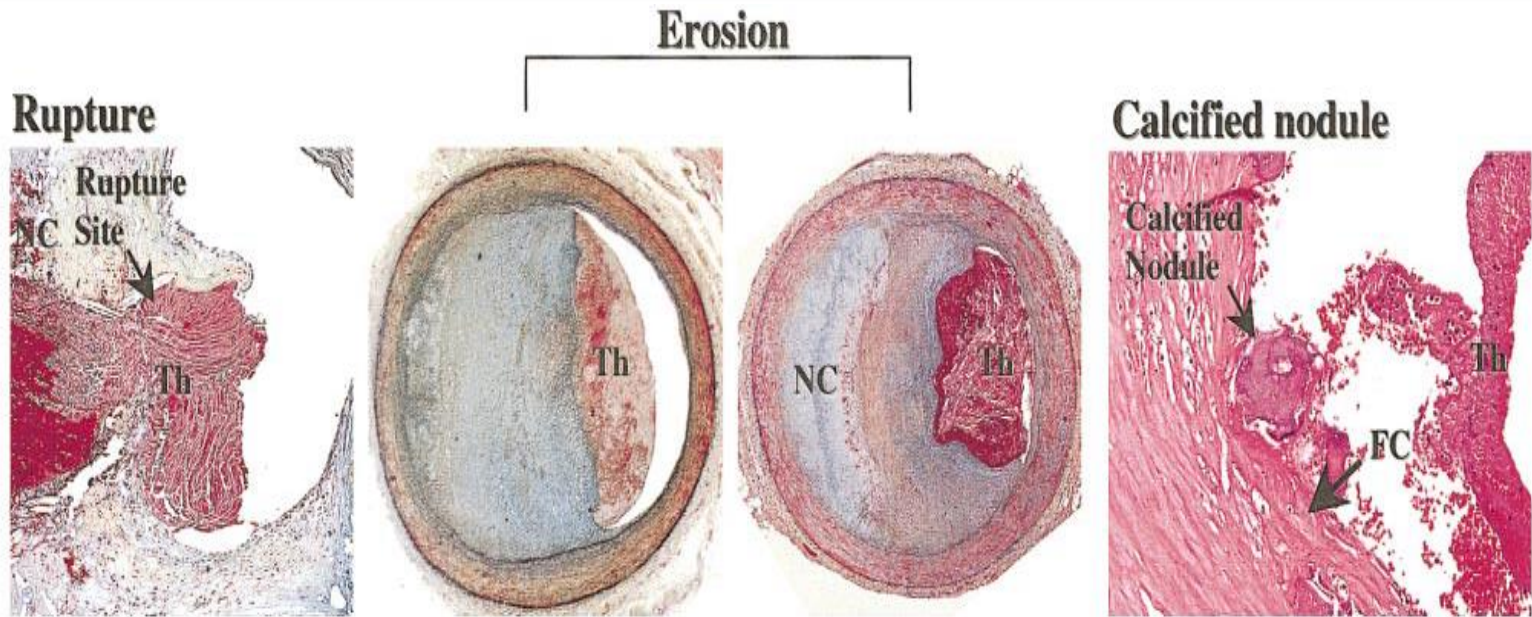
# IRM Cardiaque



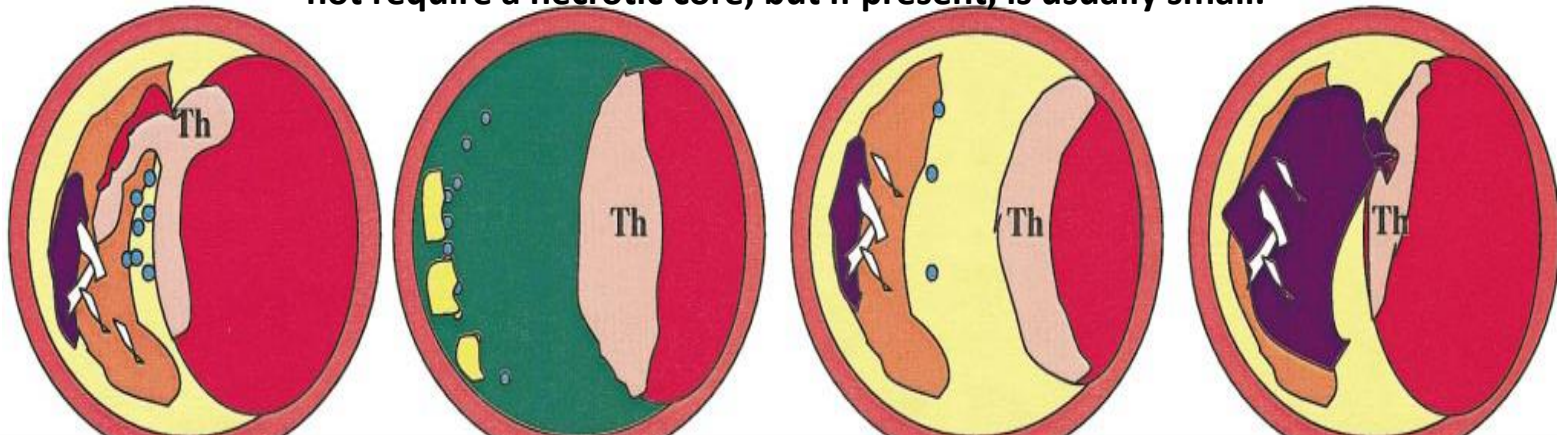
Gerbaud E. et coll. Cardiac magnetic resonance imaging for the diagnosis of patients presenting with chest pain, raised troponin and unobstructed coronary arteries. *Int J Cardiovasc Imaging*. 2012;28(4):783–94.



# Lessons from sudden coronary death

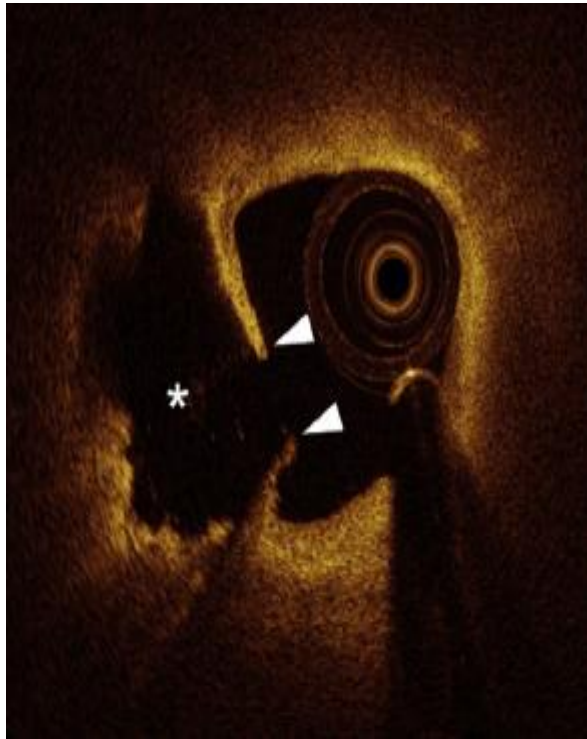


**Erosion** can occur on pathological intimal thickening or on a fibroatheroma. This form of thrombotic occlusion does not require a necrotic core, but if present, is usually small.

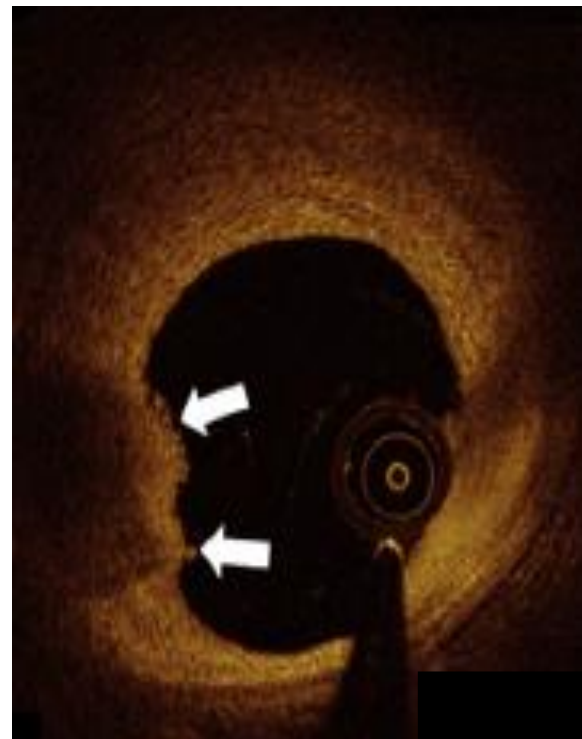


Virmani R. et coll. Lessons from sudden coronary death. A comprehensive morphological classification scheme for atherosclerotic lesions. *Arterioscler Thromb Vasc Biol.* 2010;20:1262–75.

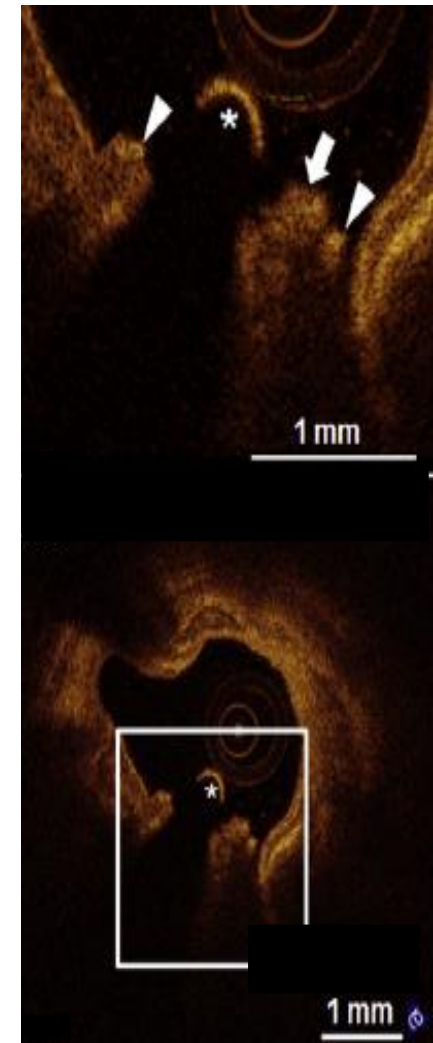
# Images correspondantes en OCT



Rupture de plaque



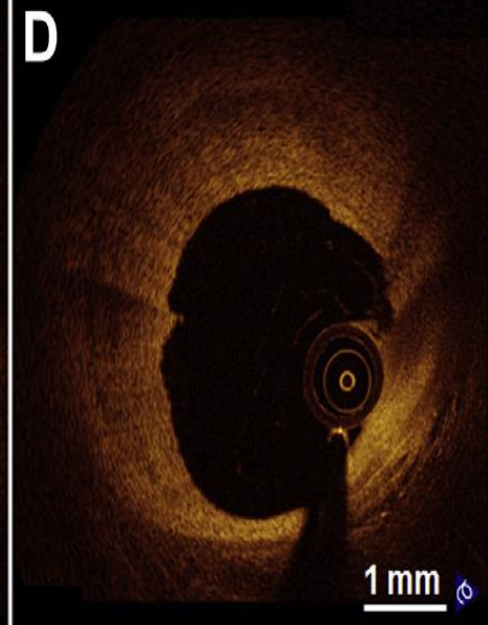
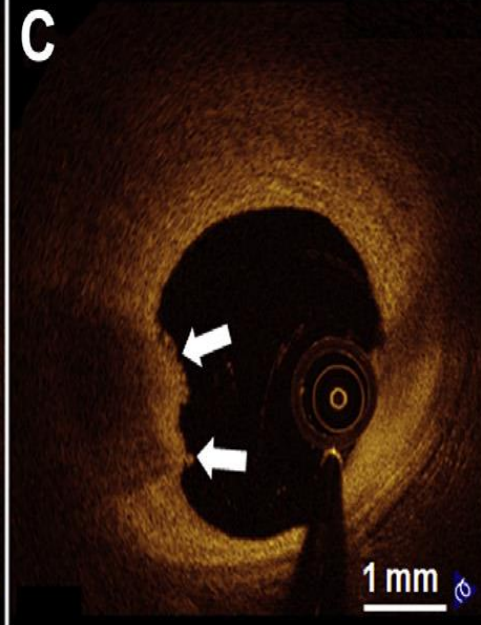
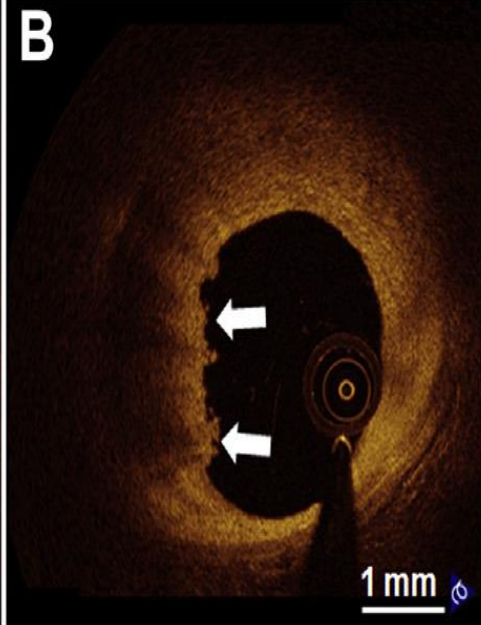
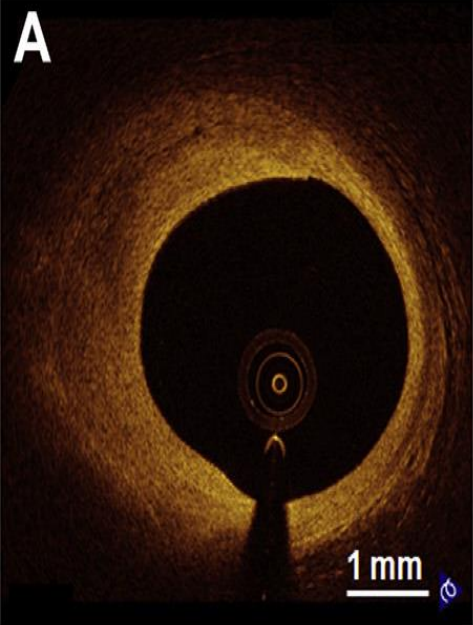
Definite OCT-  
erosion



Nodule calcifié

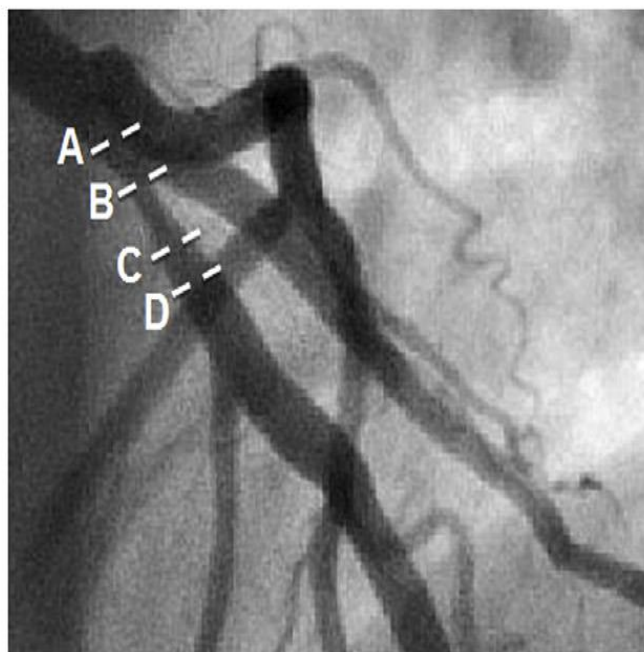
Jia H. et coll. In vivo diagnosis of plaque erosion and calcified nodule in patients with acute coronary syndrome by intravascular optical coherence tomography. J Am Coll Cardiol. 2013;62(19):1748–58.





Corresponding  
OFDI images in  
ACS

**Definite OCT-Erosion**



Presence of attached  
thrombus overlying  
an intact and  
visualized plaque

Jia H. et coll. In vivo diagnosis of plaque erosion and calcified nodule in patients with acute coronary syndrome by intravascular optical coherence tomography. J Am Coll Cardiol. 2013;62(19):1748–58.

# Test au Méthergin

- .Après arrêt des calcium-bloqueurs et nitrés
- .Méthergin: Methylene d'ergométrine, ampoules de 0.20 mg
- .Déroulement du test en 2 temps:
  - Injection IV lente (15 sec) d'une demi-ampoule (0.1 mg) puis 3 minutes après
  - Injection IV lente (30 sec) de 1.5 ampoule (0.3 mg)
- .Test positif si réduction de calibre de l'artère d'au moins 50 % de diamètre, de manière focale ou diffuse, éventuellement associée à la reproduction de la douleur et à des modifications de l'électrocardiogramme.
- .Réversibilité sous dérivés nitrés en intracoronaire
- .Déconseillé en phase aiguë de NSTEMI ou STEMI

# EXEMPLE 1

.Homme

.39 ans

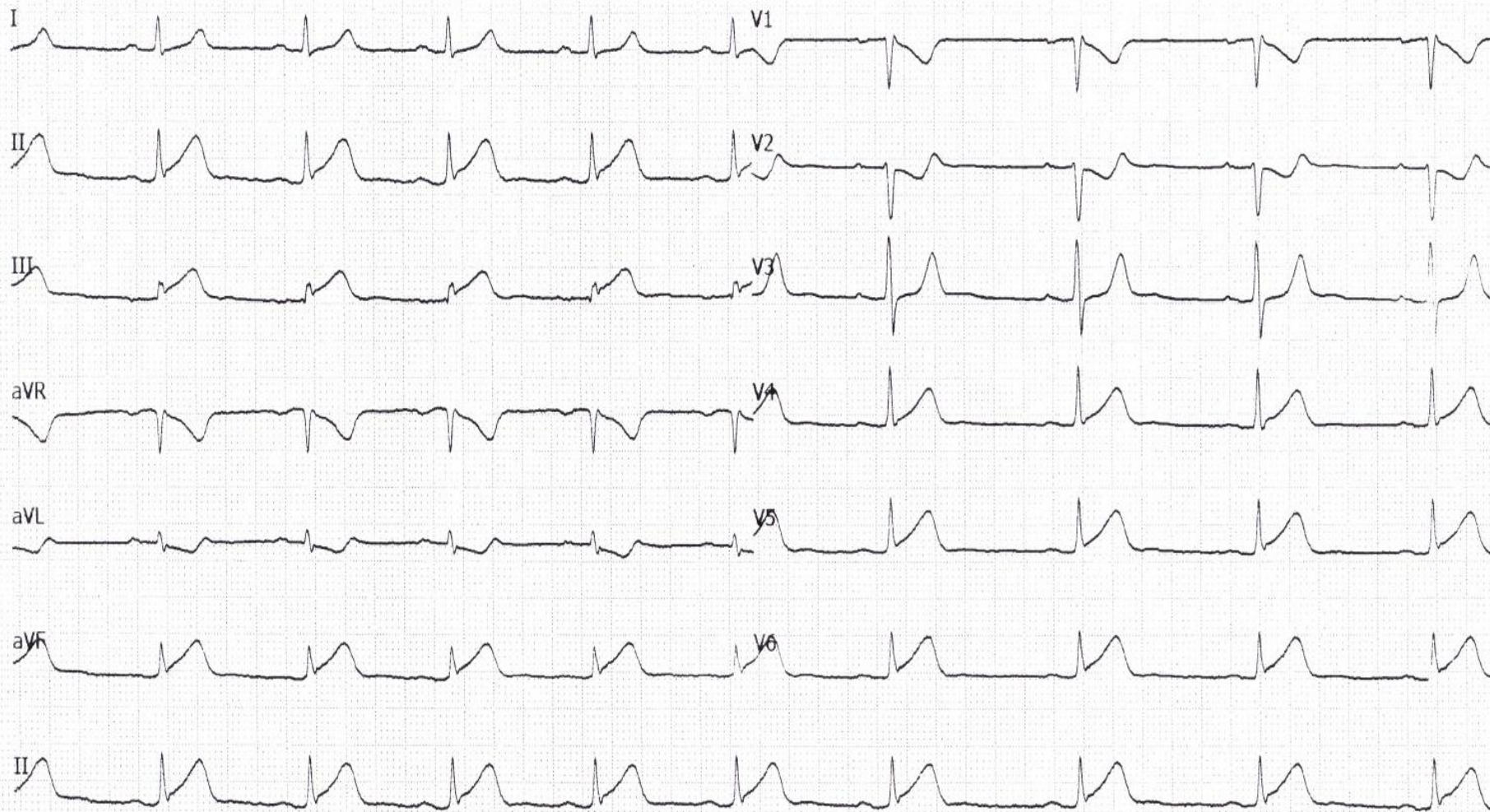
.Tabagisme actif

.Infarctus inférieur en voie de constitution

.Thrombolyse à H+1

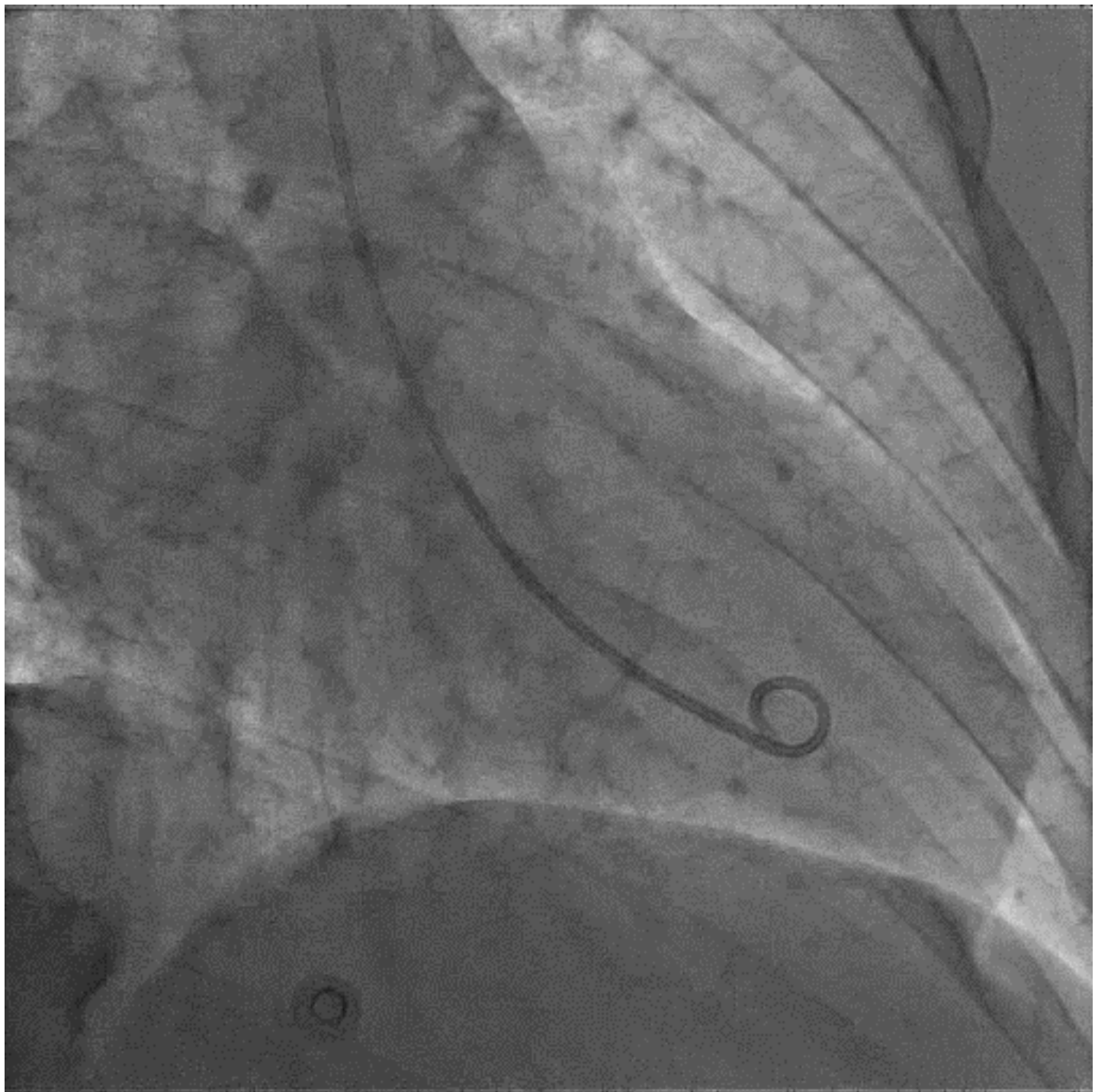


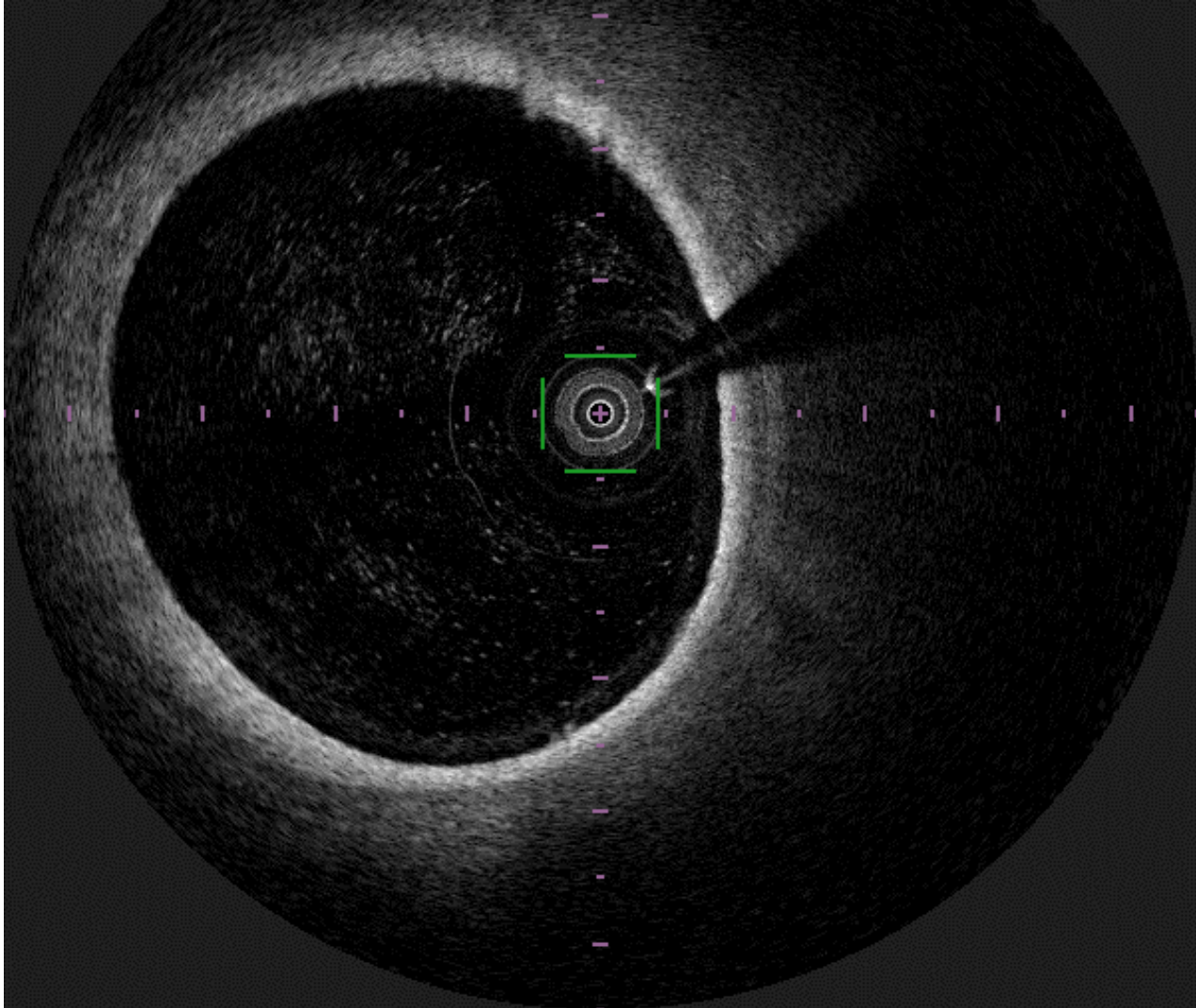
# EXAMPLE 1



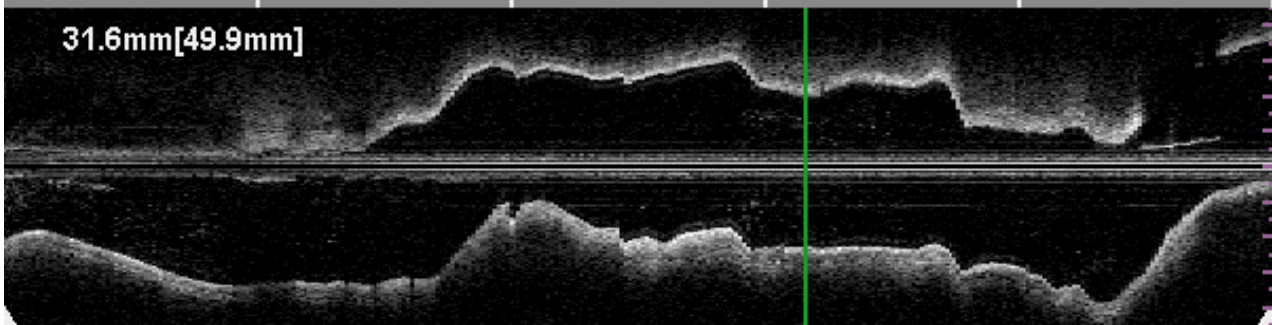




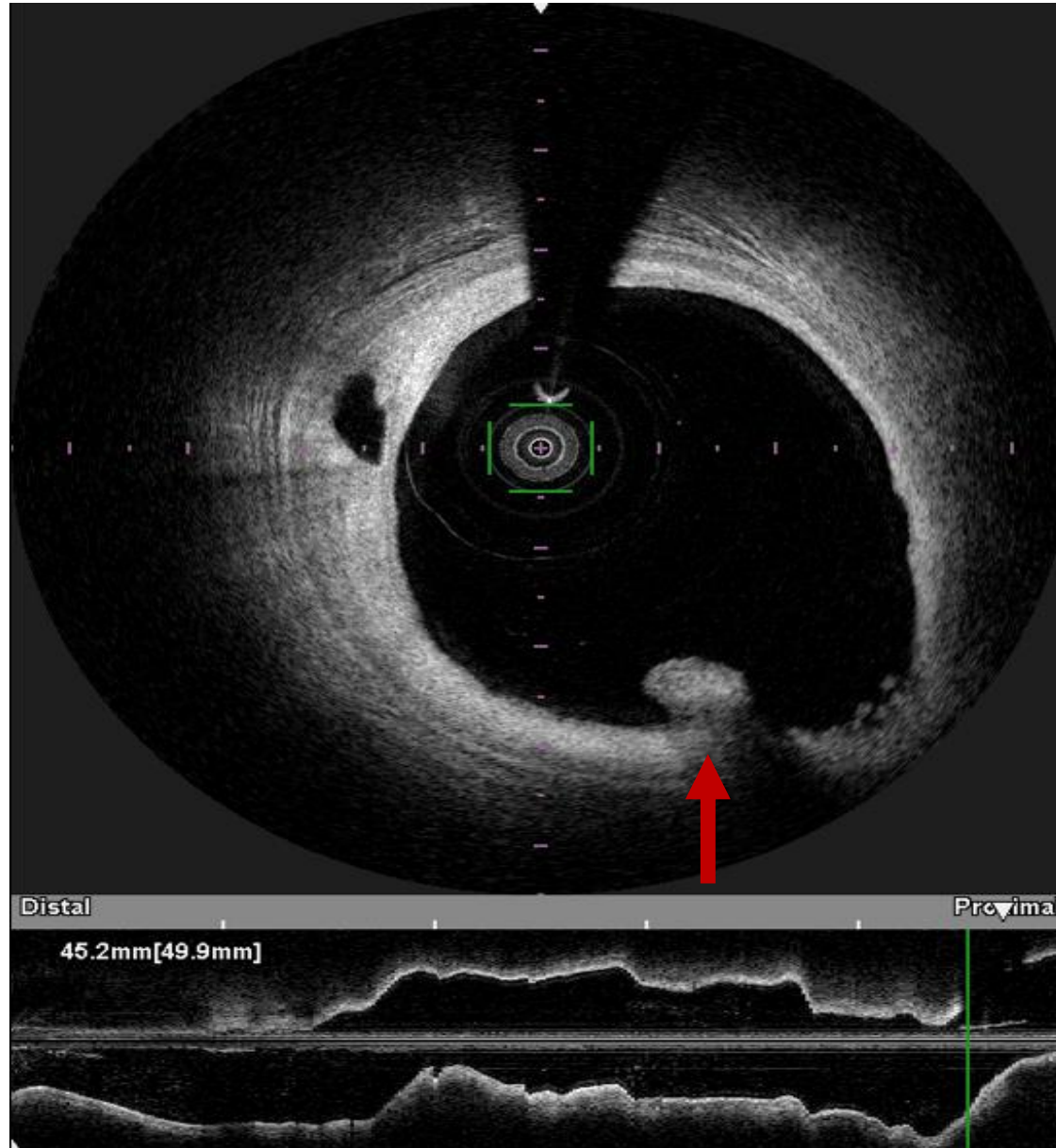




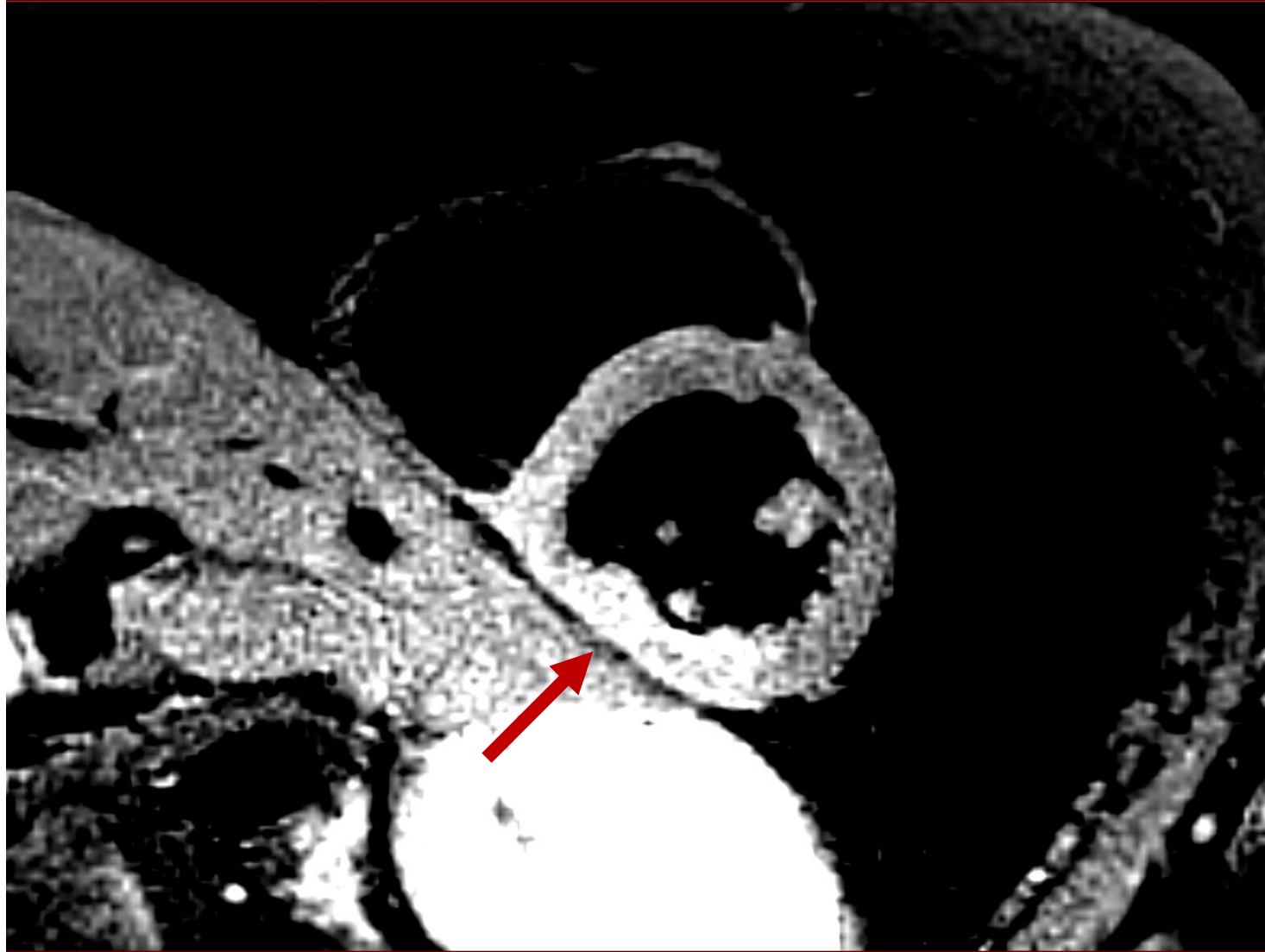
Distal Proximal



# EXAMPLE 1 - OCT

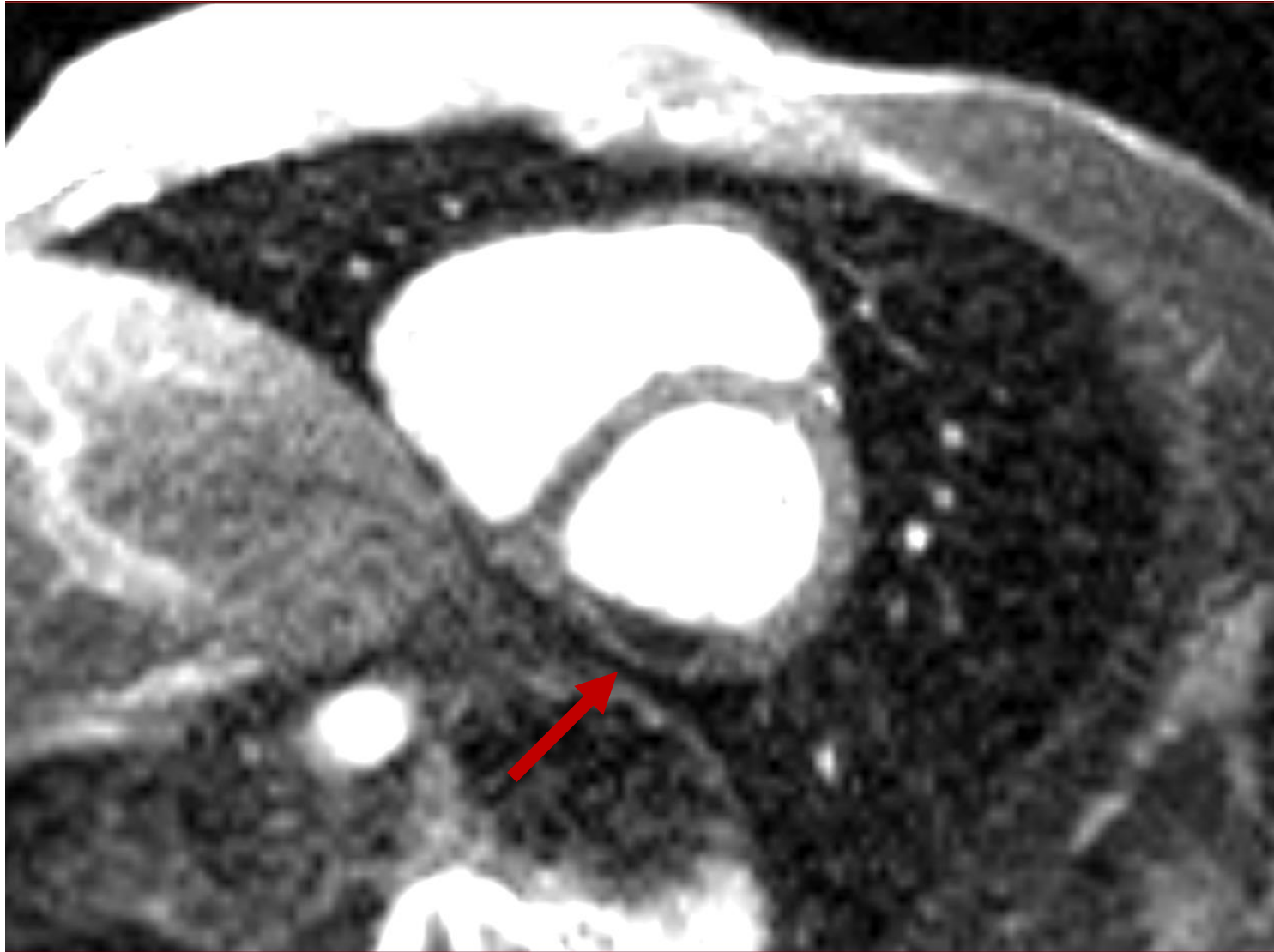


# EXAMPLE 1 – IRM T2 SPAIR





# EXAMPLE 1 – Perfusion 1<sup>er</sup> passage



# EXEMPLE 1 – IRM

## Réhaussement tardif



## EXEMPLE 2

.Femme

.47 ans

.Tabagisme actif

.Epilepsie

.Phlébite suites chirurgie orthopédique en 2018

.Stress et trouble de l'humeur

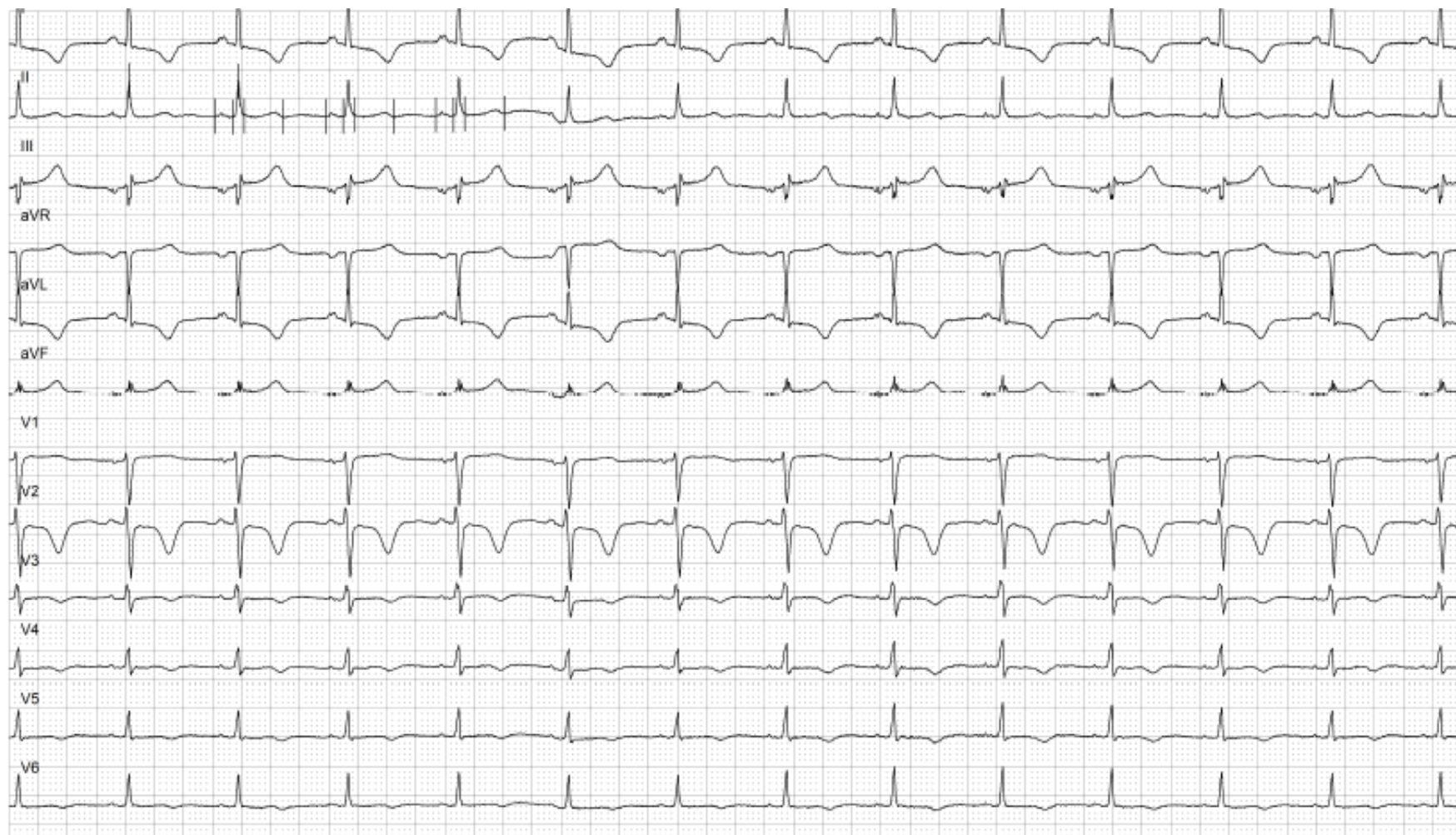
.Traitement: Keppra et Seresta

.Douleur thoracique constrictive, rétro sternale, d'apparition brutale postprandiale vers midi, irradiant dans les bras et la nuque, d'une durée de 45 minutes

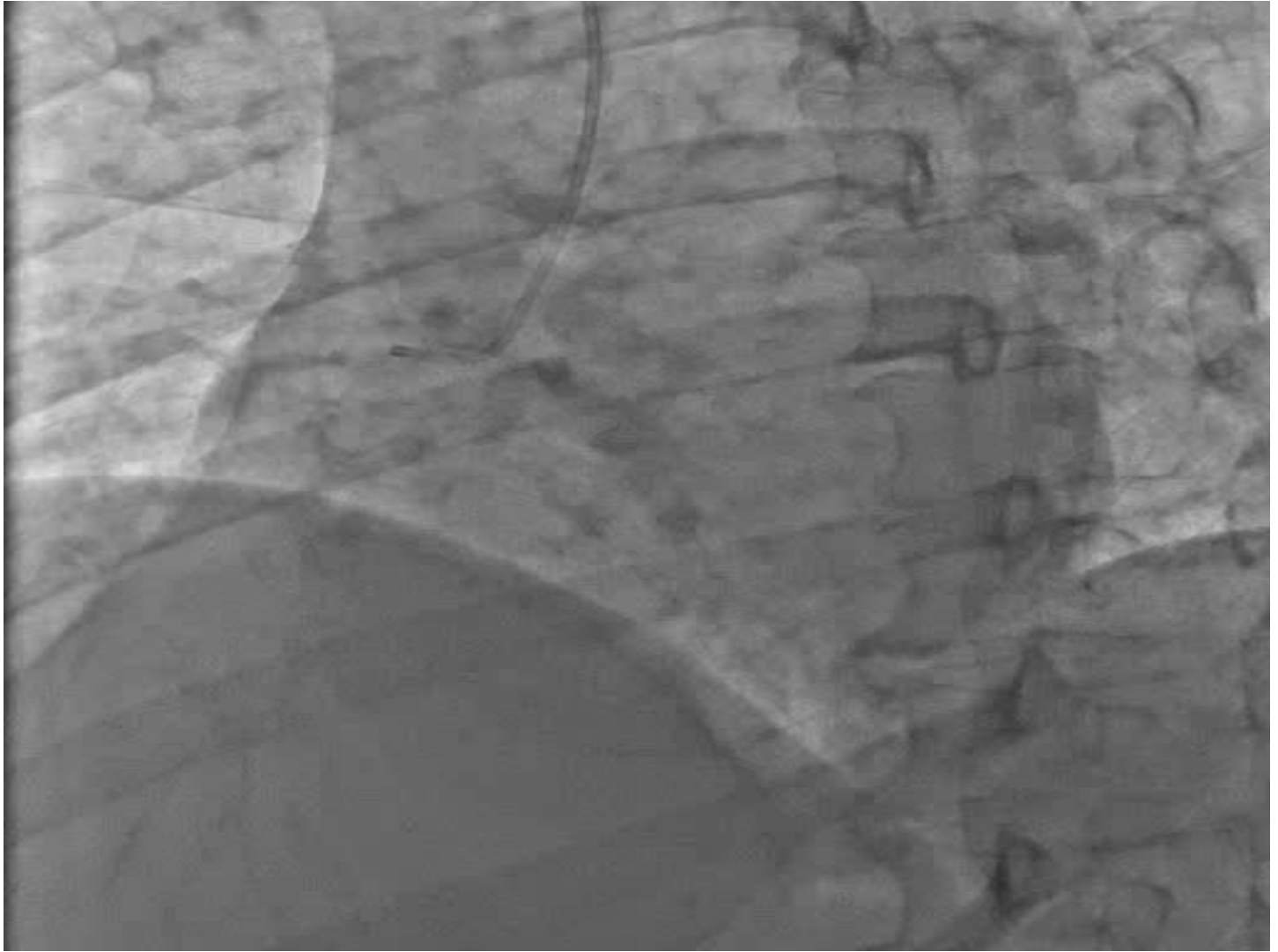
.Avait présenté une douleur thoracique similaire il y a 8 mois pour laquelle elle n'avait pas consulté

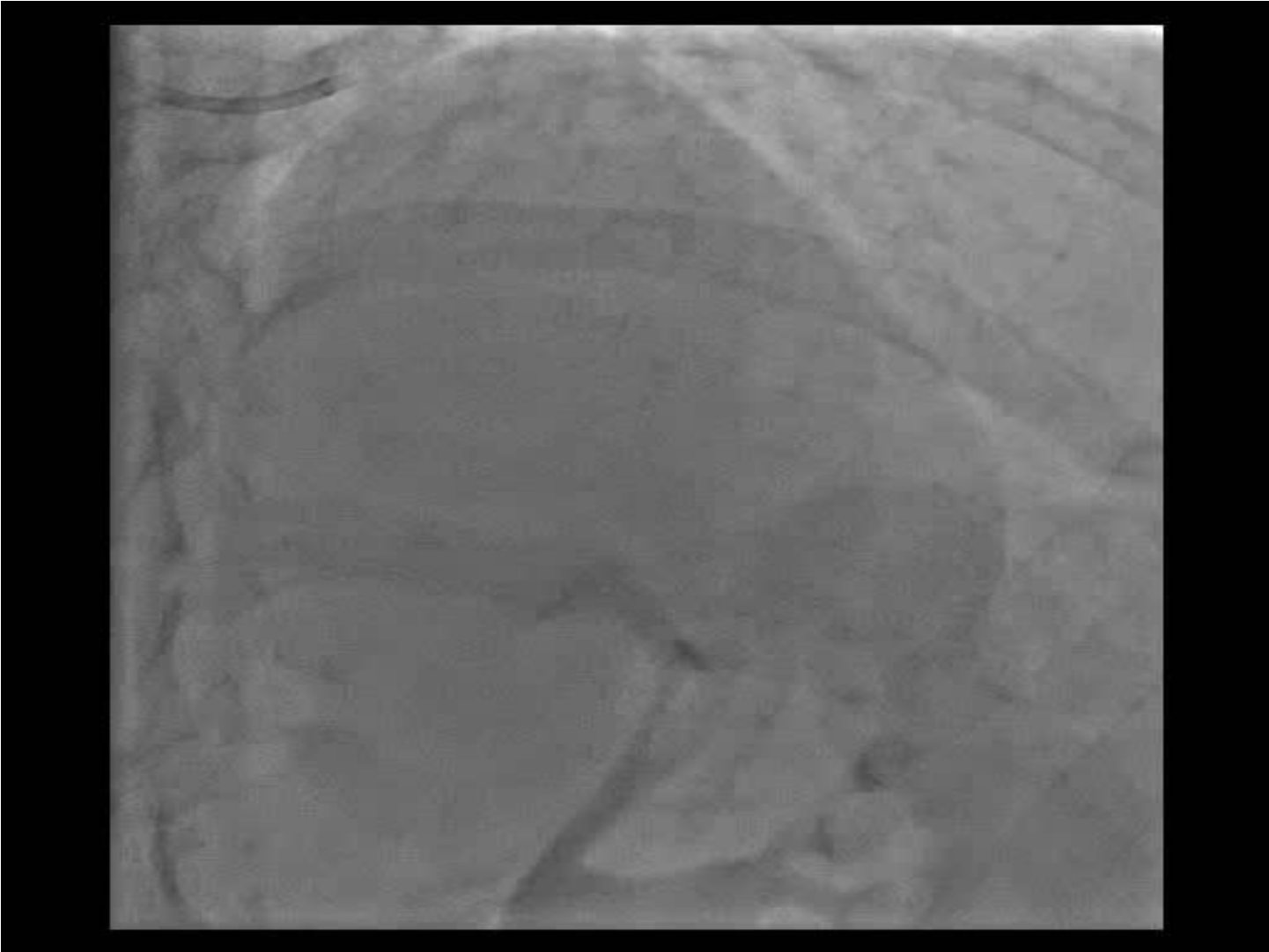
.Elévation de la Troponine HS à 148 (N<16) ng/L

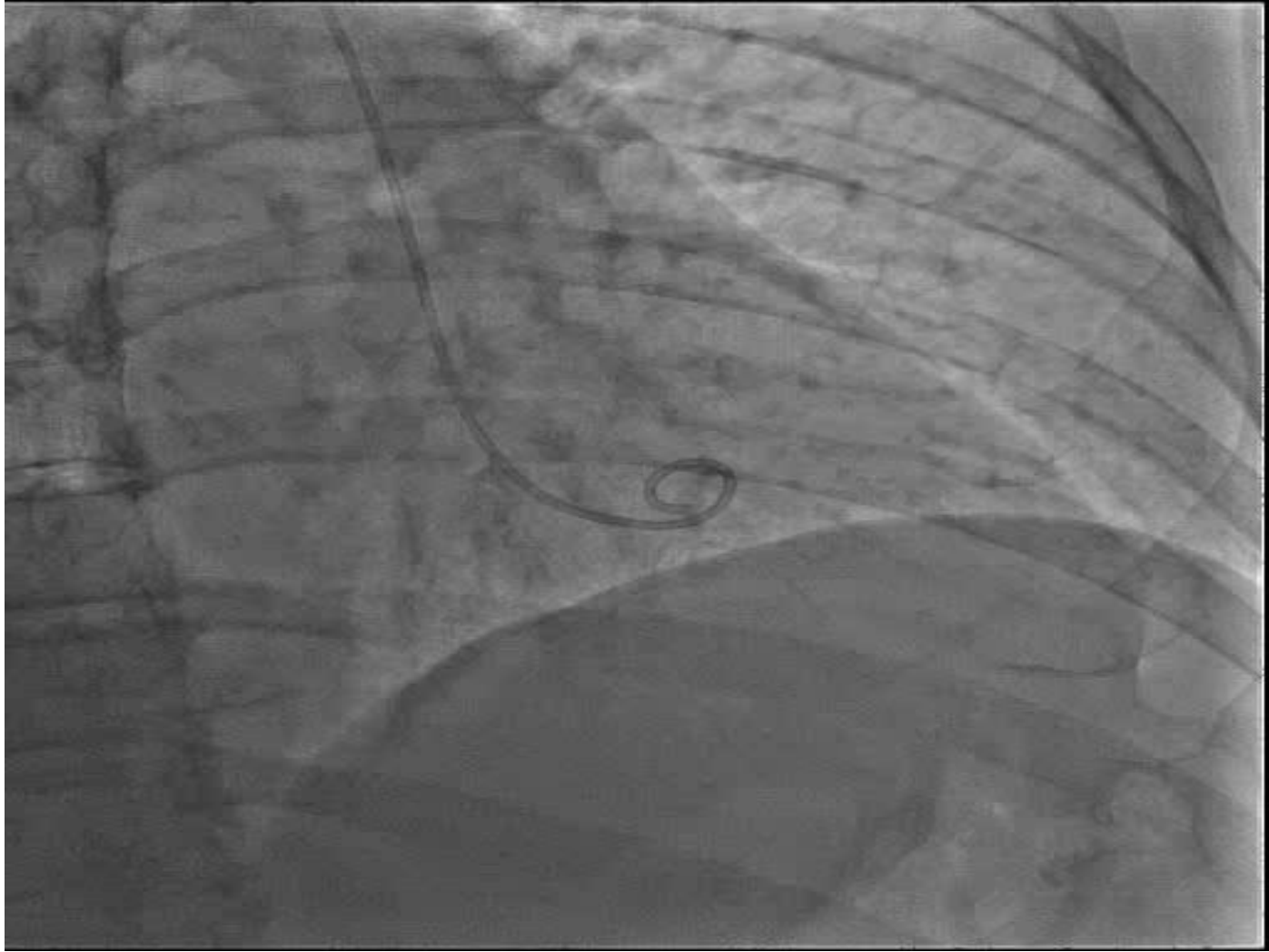
# EXAMPLE 2

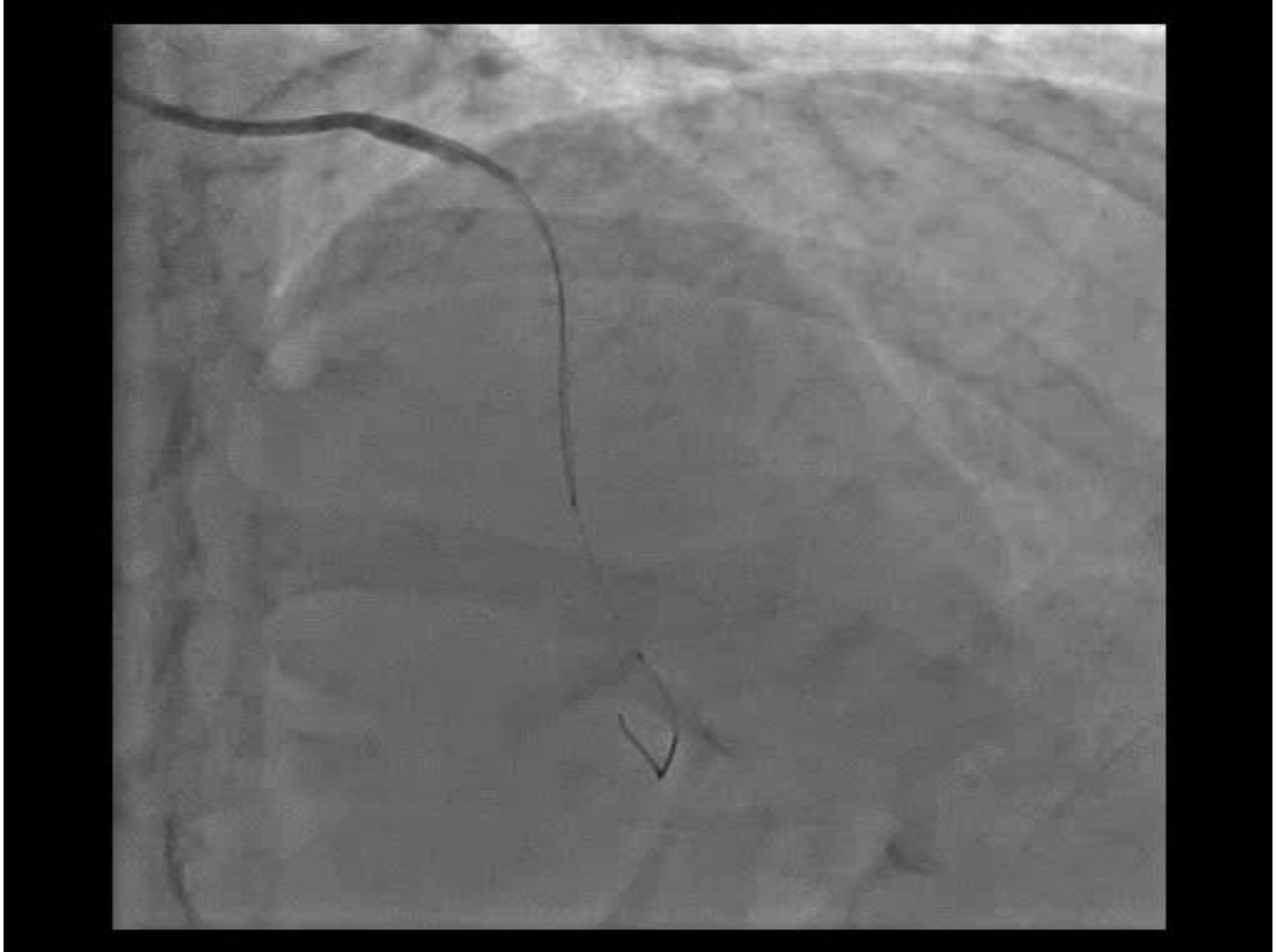


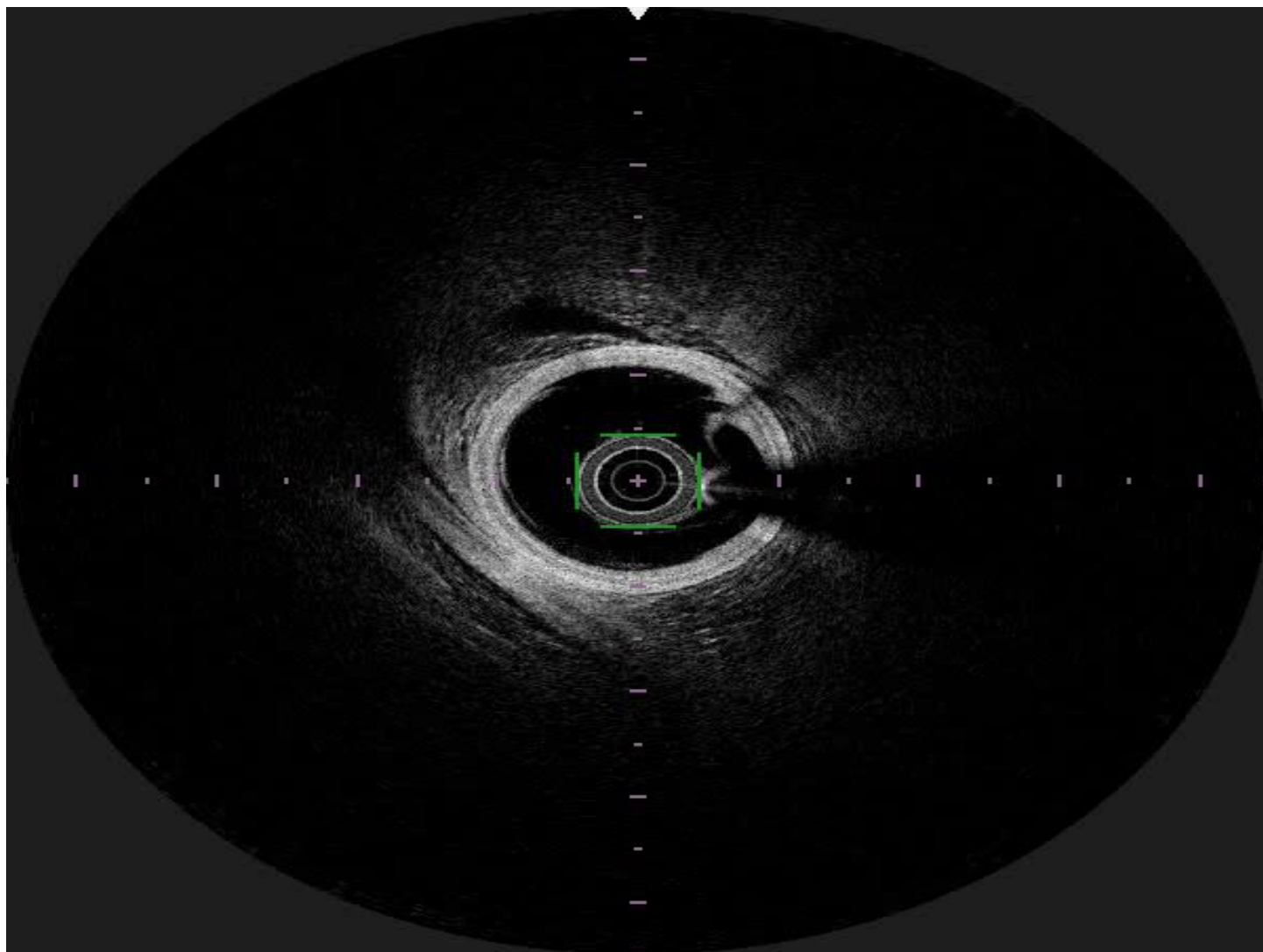




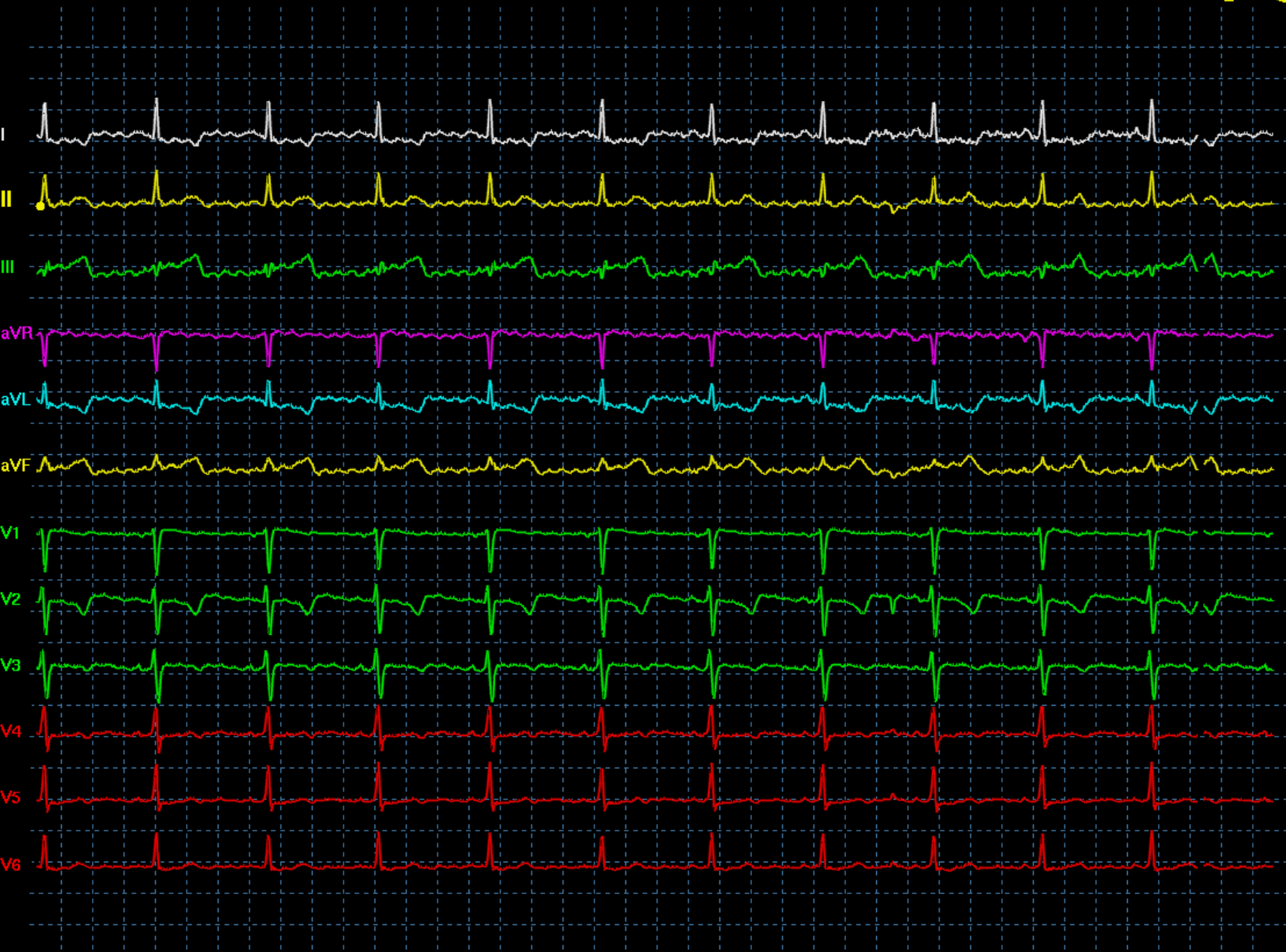






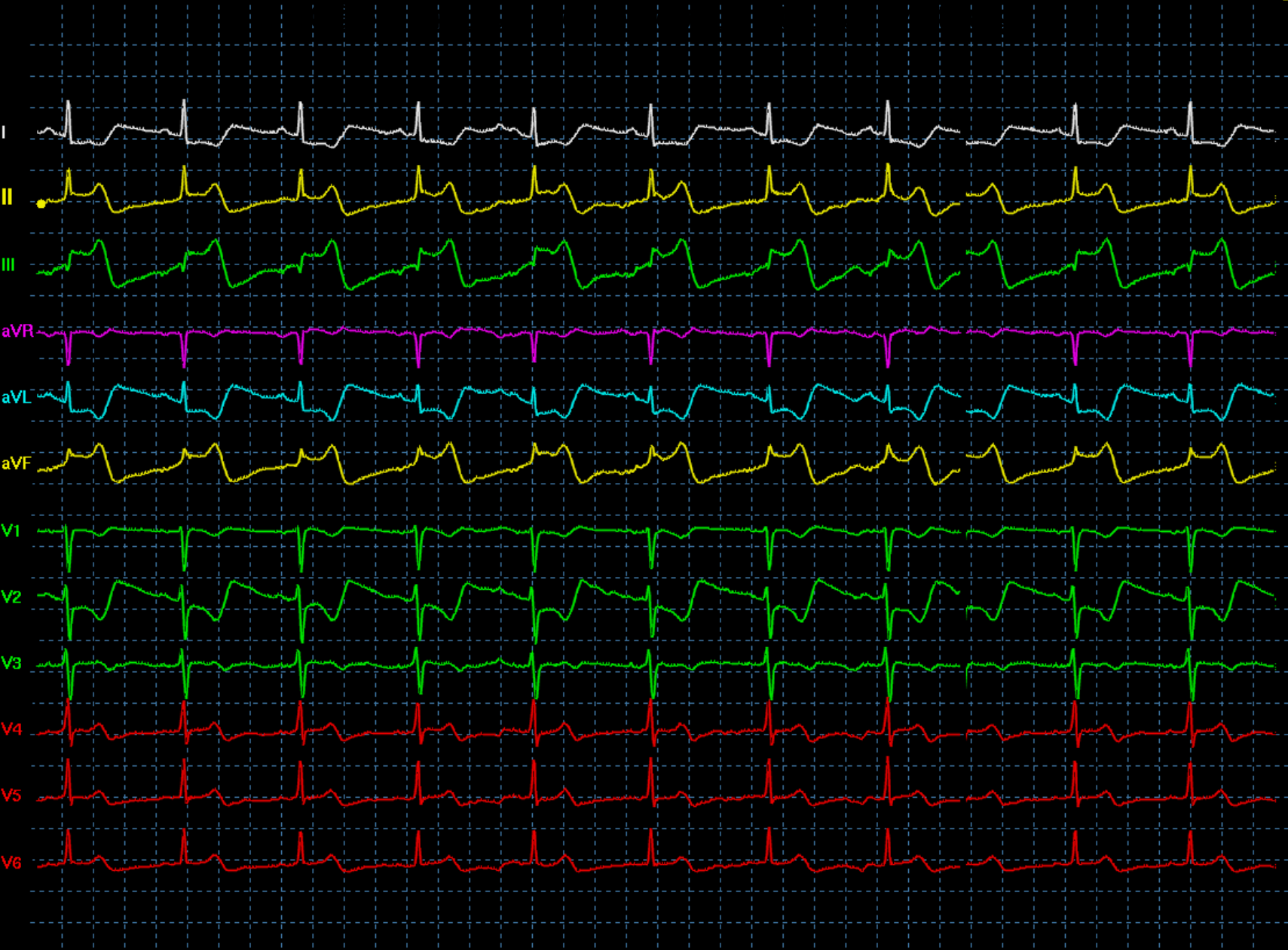


FC 75



25 mm/sec

Moniteur



METHERGIN > 6 min







METHERGIN > 6 mm

# Combinaison d'examens?

**OBJECTIVES** This study sought to assess the presence and morphological features of coronary plaques on optical coherence tomography (OCT) as the causes of myocardial infarction with nonobstructive coronary arteries (MINOCA).

**BACKGROUND** Although coronary atherosclerosis has been postulated as a potential mechanism of MINOCA, the interaction between disrupted coronary plaques and myocardial injury remains unknown.

**METHODS** In a prospective study, consecutive patients with MI but without significant coronary stenosis ( $\geq 50\%$ ) at angiography underwent OCT and cardiac magnetic resonance (CMR) with late gadolinium-enhancement (LGE). The infarct-related artery (IRA) was identified by localization of ischemic-type LGE.

**RESULTS** Thirty-eight MINOCA patients (mean age  $62 \pm 13$  years, 55% female, 39% with ST-segment elevation) were enrolled. Maximal diameter stenosis was 35% by angiography, and 5 patients (13%) had normal angiogram results. Plaque disruption and coronary thrombus were observed in 9 patients (24%) and 7 patients (18%), respectively. Sixteen of 31 patients (52%) undergoing CMR showed LGE. Ischemic-type LGE was present in 7 patients (23%) and was more common in patients with than without plaque disruption (50% vs. 13%, respectively;  $p = 0.053$ ) and coronary thrombus (67% vs. 12%, respectively;  $p = 0.014$ ). In the per-lesion analysis, the IRA showed significantly more plaque disruption (40% vs. 6%;  $p = 0.02$ ), thrombus (50% vs. 4%;  $p = 0.014$ ), and thin-cap fibroatheroma (70% vs. 30%;  $p = 0.03$ ) than the non-IRA.

**CONCLUSIONS** Plaque disruption and thrombus are not uncommon in MI without obstructive coronary stenoses at angiography and may be associated with the presence and location of ischemic-type myocardial injury on CMR. OCT may be valuable in identifying atherosclerotic etiology in individuals with MINOCA. (Optical Coherence Tomography in Patients With Acute Myocardial Infarction and Nonobstructive Coronary Artery Disease [SOFT-MI]; NCT02783963) (J Am Coll Cardiol Img 2018;■■■■) © 2018 by the American College of Cardiology Foundation.

Opolski MP. et coll. Mechanisms of myocardial infarction in patients with non-obstructive coronary artery disease. Results from the optical coherence tomography study. J Am Coll Cardiol Img 2018; In press

# CONCLUSION

- . Eliminer les causes « non cardiaques »
- . Examens complémentaires à orienter en fonction du contexte +++
  - . IRM Cardiaque
  - . Imagerie intracoronaire
  - . Test au Méthergin
  - . Bilan de thrombophilie
  - . Echographie trans-oesophagienne

