

# MYOCARDIAL INFARCTION IN THE ABSENCE OF OBSTRUCTIVE CORONARY ARTERY DISEASE

Wiren, M – OMS III, Reynolds, T – DO, Escobar, J - MD
University of New England College of Osteopathic Medicine, Biddeford, ME
Maine-Dartmouth Family Medicine Residency, MaineGeneral Medical Center, Augusta, ME



### INTRODUCTION

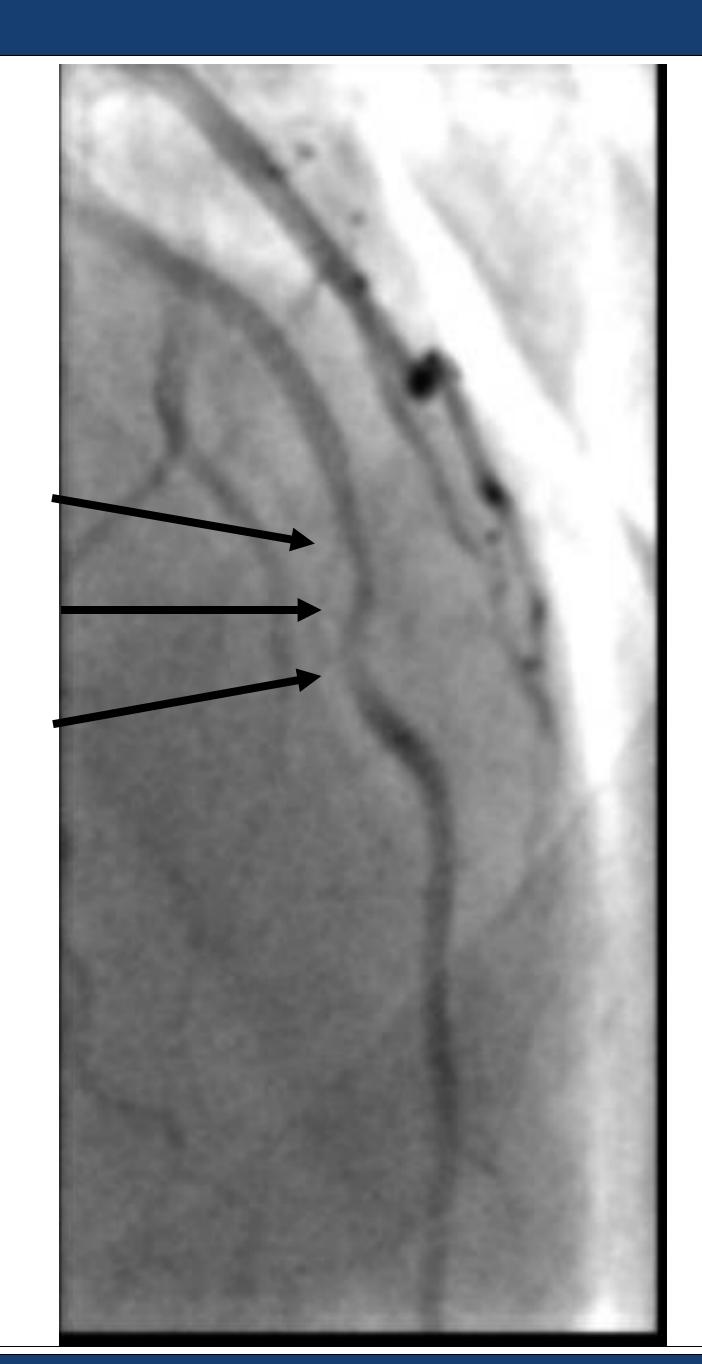
Myocardial infarction in the absence of obstructive coronary artery disease (MINOCA) is a heterogenous grouping of etiologies that cause acute myocardial infarction (AMI) in a population with little to no CAD risk factors.<sup>1,2</sup>

These patients are usually young, female, and non-white.<sup>3</sup> It is important to recognize that the demographic and clinical characteristics of MINOCA patients differ from other patients with acute MI. MINOCA patients are younger, 58 years compared with 61, and disproportionately female making up close to 50% of MINOCA population. MINOCA patients do not have the same risk factors compared to AMI-CAD, with lower prevalence of dyslipidemia, hypertension, DM, tobacco use and family history of MI. MINOCA makes up to 5% to 6% of AMI cases.<sup>1</sup>.

Specific causes of MINOCA include spontaneous coronary artery dissection (SCAD), plaque disruption, coronary emboli/thrombus, coronary artery spasm, and microvascular disease.<sup>4</sup>

We present an otherwise healthy young female patient who presented to the ED with typical MI symptoms found to have MINOCA secondary to SCAD and may indicate an underlying vasculopathy such as fibromusclar dysplasia (FMD) that was likely exacerbated by vigorous physical activity on the day of admission. It is essential for healthcare providers to become familiar with this syndrome to appropriately and identify patients who may otherwise go treated.

# Figure 1



# Figure Legend

Fig 1. Angiographic image of the patients left anterior descending coronary artery with at 50-60% narrowing at its mid-vessel consistent with SCAD. Fig 2. Graph of cTN showing an initial vale of 63 and peaking at 635. EKG on admission showing T-wave inversions in leads V1 and V2.

#### CASE

**History:** A 40-year-old previously healthy white female presented to the ED with acute onset of chest pain. At approximately 5:45 PM she stated she was working on her horse farm and was picking up a 40lbs bucket when she felt acute onset of bilateral jaw pain that radiated down to her chest and back. As the jaw pain resolved, she began to feel exclusively chest pain that radiated to her back that she described as a pressure-like sensation. She is extremely healthy and exercises daily and reports doing a kickboxing workout earlier that morning. ROS was notably negative for shortness of breath, nausea, vomiting, or diaphoresis. The rest of the ROS was negative.

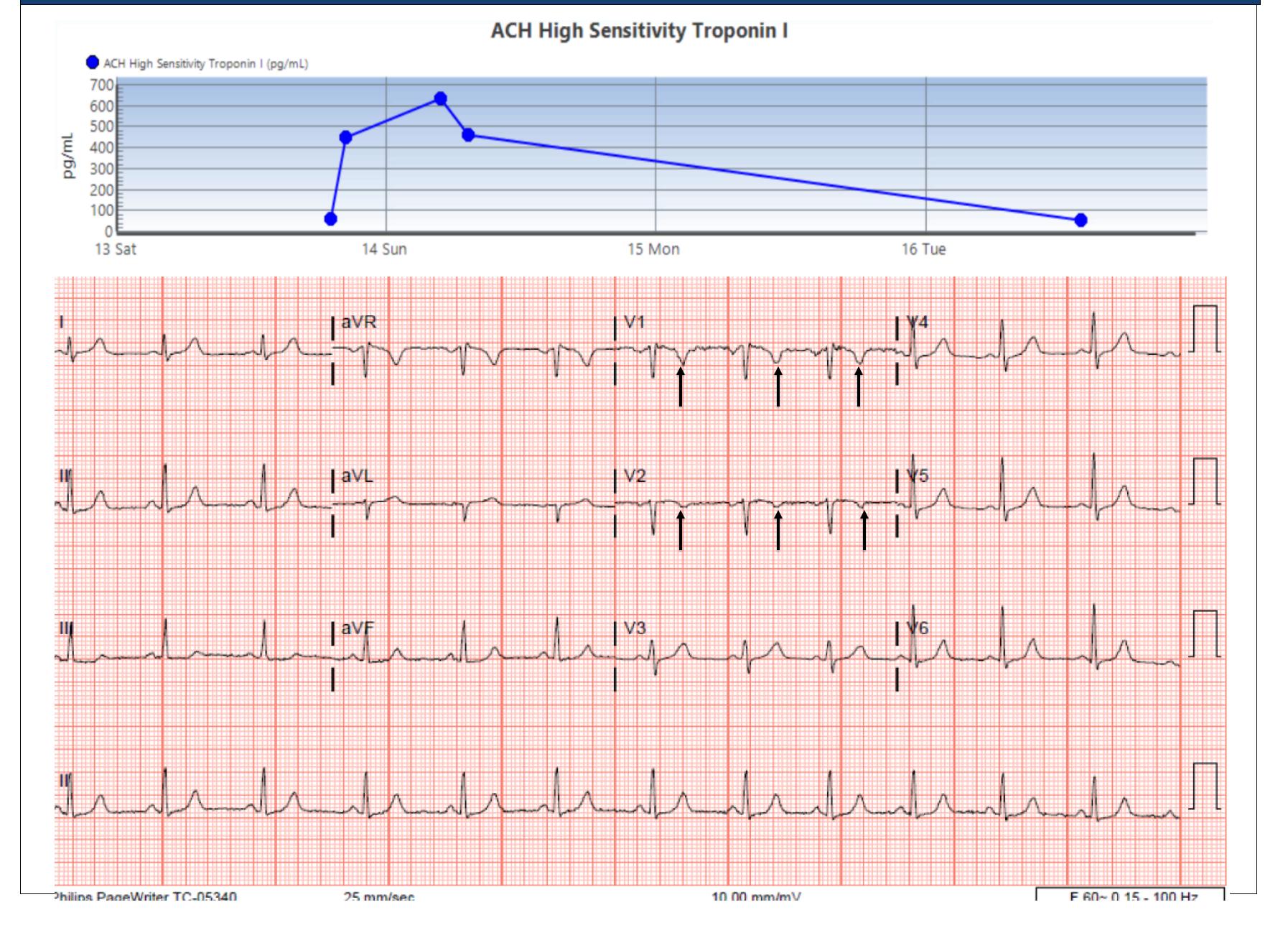
Her past medical history includes anxiety but does not take any medications for it and she doesn't take any other medications daily. No prior surgeries. No known drug allergies. Her social history was negative for tobacco or recreational drugs. She drink an occasional glass of wine. Her family history was notable for a father with CAD and stent placement at age 53 and later 4-vessel CABG and T2DM.

Her vitals were: temperature 98.1, heart rate 97, blood pressure 126/76, respiratory rate 18, O2 saturation 100% on room air. Her physical exam was notably negative for JVD, her heart sounds were normal without murmurs, rubs, or gallops and she had no edema in her lower extremities.

**Pertinent Labs and Imaging:** Her EKG on admission showed T-wave inversions in the septal leads V1 and V2 and her initial troponin level was 63 with peak level of 635 and her LDL was 94. Echocardiogram showed normal biventricular size and systolic function at 60% to 65% without regional wall motion abnormalities. CT angiogram chest showed no evidence of aneurysm or no infiltrate or parenchymal lung abnormality. She underwent a nuclear stress test which showed no clear evidence of ischemia or prior infarct but showed some T wave inversions on the lateral leads. Coronary CTA was normal without evidence of high-grade stenosis, filing defect, aneurysm, dissection or other acute abnormality of the coronary arterial system. She then underwent coronary catherization and angiography which revealed a left anterior descending artery that had a narrowing to approximately 50% to 60% at the mid vessel.

Assessment/Plan: The cause of the narrowing was determined to be a spontaneous coronary artery dissection resulting in an intramural hematoma that was compressing the lumen without clear signs of a double lumen seen in other types of dissections. She was given the diagnosis of SCAD and was discharged from the hospital on aspirin 81mg, Clopidogrel 75mg daily for two weeks, and metoprolol 25mg daily with instructions to undergo cardiac rehab for 30 to 90 days, restrict activity and reduce lifting loads, and to follow up for a cardiac MRI with LGE to better characterize degree of scar and a CT angiogram of the abdomen, pelvis, and intracranial vessels to look for signs of fibromuscular dysplasia, a known association of SCAD.

# Figure 2



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

#### **DISCUSSION**

Establishing the diagnosis of MINOCA can be challenging as patients presenting with MINOCA are usually younger, female, and without the usual risk factors for AMI, as seen with our patient, a 40y-old otherwise healthy female. Additionally, there is a persistent misconception among clinicians that the absence of obstructive CAD excludes the possibility of an AMI<sup>4</sup>. We arrived at the working diagnosis of MINOCA by utilizing the clinical algorithm above. Our patient met the following criteria:

- 1. Rise of cardiac troponin (cTn) plus ischemic signs/symptoms: The patient presentation of acute onset chest pain plus elevated cTn, peaking at 635, and ischemic changes on EKG with T-wave inversions in V1 and V2 with no overt causes of non-ischemic disease including sepsis or pulmonary embolism found on CBC or chest CTA. Clinically subtle non-ischemic mechanisms including myocarditis, vasospastic angina, or Takotsubo syndrome were ruled out due to negative inflammatory markers, serial EKGs showing no additional ischemia, and a normal echocardiogram showing no LV dysfunction.
- 2. Nonobstructive CAD: Nuclear stress test and coronary CTA showed no evidence of high-grade stenosis. Cardiac catherization with angiography showed a 50% narrowing in the LAD. However, given the patient's lack of risk factors, spontaneous coronary artery dissection (SCAD) was a more likely etiology than ruptured atherosclerotic plaque.

SCAD produces a hematoma within the tunica media, possibly by disruption traversing microvessels,<sup>5</sup> leading to separation of the intima and compression of the true lumen. SCAD can mimic atherosclerotic stenosis, and imaging with OCT or IVUS can help distinguish this lesion from other types of SCAD or etiologies. There is a high prevalence of fibromuscular dysplasia (FMD) among individuals with SCAD. If our patient is found to have co-occurring FMD, SCAD is a more likely diagnosis than atherosclerotic plaque.

### REFERENCES

- Pasupathy S, Air T, Dreyer RP, Tavella R, Beltrame JF. Systematic review of patients presenting with suspected myocardial infarction and nonobstructive coronary arteries [published correction appears in Circulation. 2015;131:e475]. Circulation. 2015;131:861–870. doi: 10.1161/CIRCULATIONAHA.114.011201
- Barr PR, Harrison W, Smyth D, Flynn C, Lee M, Kerr AJ. Myocardial infarction without obstructive coronary artery disease is not a benign condition (ANZACS-QI 10). Heart Lung Circ. 2018;27:165–174. doi:10.1016/j.hlc.2017.02.023
- Smilowitz NR, Mahajan AM, Roe MT, Hellkamp AS, Chiswell K, Gulati M, Reynolds HR. Mortality of myocardial infarction by sex, age, and obstructive coronary artery disease status in the ACTION Registry-GWTG (Acute Coronary Treatment and Intervention Outcomes Network Registry-Get With the Guidelines). Circ Cardiovasc Qual Outcomes. 2017;10:e003443.doi: 10.1161/CIRCOUTCOMES.116.003443
- Hayes SN, Tweet MS, Adlam D, Kim ESH, Gulati R, Price JE, Rose CH. Spontaneous Coronary Artery Dissection: JACC State-of-the-Art Review. J Am Coll Cardiol. 2020 Aug 25;76(8):961-984. doi: 10.1016/j.jacc.2020.05.084. PMID: 32819471.

## Acknowledgements

The University of New England College of Osteopathic and Maine-Dartmouth Family Medicine Residency at MaineGeneral Medical Center, with thanks to Dr. Kelley Harmon, DO.