



MINOCA (MYOCARDIAL INFARCTION WITH NON- OBSTRUCTIVE CORONARY ARTERIES)

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Abstract:

MINOCA is defined as myocardial infarction with mild or no obstructive coronary artery disease (CAD) on angiography. It is a heterogeneous syndrome with multiple underlying causes, including coronary artery spasm (vasospastic angina), coronary embolism, spontaneous coronary artery dissection (SCAD), plaque disruption and coronary microvascular disruption (CMD). Additionally, non-ischemic conditions such as Myocarditis and Takotsubo cardiomyopathy may mimic its presence. It is characterized by evidence of acute myocardial infarction in the absence of obstructive coronary artery disease, conventionally characterized as the presence of coronary stenosis of 50% or greater on Angiography. Epidemiologically, approx 5-15% of all MI cases globally and is more common in younger individuals and females featuring as recurrent adverse cardiovascular events, persistent anginal syndromes and deminished quality of life.

Keywords: MINOCA, myocardial infarction, emotional stress, coronary vasospasm, vasospastic angina, left ventricular systolic dysfunction, ischemia with non obstructive coronary arteries (INOCA), thrombophilia, plaque erosion, troponin elevation

Introduction:

Myocardial infarction Which is associated with obstructive coronary artery disease; however, contrasts with a distinct clinical entity known as MINOCA which is associated with non- obstructive coronary arteries. The syndrome is not a single disease, it presents with typical hallmarks of acute myocardial infarction, including chest pain and ECG changes. Understanding MINOCA helps in accurate diagnosis.

What is MINOCA?

An uncommon type of heart attack mostly affecting women due to emotional stress causing fatal myocardial infarction with non-obstructed coronary arteries.

1 in 10 heart attacks occur without a blockage to the heart's arteries known as MINOCA (Myocardial Infarction with Non-Obstructive Coronary Arteries).

It is 3 times more frequent among women and can be triggered by intense emotions.

Women presenting with AMI are more than twice as likely as men to have MINOCA whereas men presenting with AMI are more likely than women to have AMI-CAD.

Compared to heart attacks with obstructive coronary artery disease, MINOCA patients are more likely to be female and younger, and they're also more likely to be Black, Maori or Pacific race, or Hispanic/Latino. Prior studies have shown that Black or Hispanic patients in particular, young Black women have a lower prevalence of obstructive coronary artery disease than white/non- Hispanic patients when they come with chest pain or are being evaluated for heart attack. It's unclear if this difference is due to discrepancies in healthcare referral patterns for testing or racial/ethnic differences.



What causes MINOCA?

Coronary artery spasms, plaque rupture, blood clots (embolism), microvascular dysfunction, spontaneous coronary artery dissection (SCAD); these are more common in women than in men causing poor blood flow to the heart.

Clinical Spectrum:

The sign and symptoms of MINOCA is same as heart attack in someone who has-

- Blocked arteries
- Chest discomfort (pain, pressure, tightness)
- shortness of breath
- Nausea and diaphoresis

Table 1: Risk Factors

Risk Factor	Clinical Relevance
Female sex	Higher prevalence due to vascular differences
Younger age	Less atherosclerosis, more functional causes
Stress triggers	Associated with Takotsubo and vasospasm
Hypercoagulable states	Risk of coronary embolism

- Syncope (in severe cases)

The quality of pain in MINOCA, maybe atypically described as pleuritic, positional, or sharp in a subset of patients particularly those with myocarditis and pericarditis also as dyspnea or preceding chest discomfort is seen in approximately 30-50% of MINOCA patients.

Physical examination findings in MINOCA are frequently unremarkable outside the acute ischemic event but may reveal tachycardia, mild hypertension or hypotension depending on the severity of ischemia and autonomic response.

Etiology:

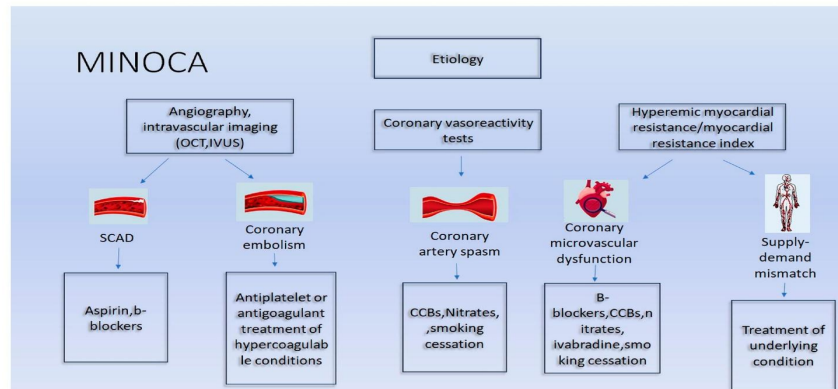
The etiological architecture of MINOCA does not have single cause, several distinct mechanisms can produce the same clinical picture, myocardial injury with clean looking coronary Arteries and a single patient may harbor multiple co-existing causative mechanism simultaneously.

- CORONARY ARTERY SPASMS (VASOSPASTIC ANGINA)
- SPONTANEOUS CORONARY ARTERY DISSECTION (SCAD)
- CORONARY THROMBOEMBOLISM
- CORONARY MICROVASCULAR DYSFUNCTION
- TAKOTSUBO CARDIOMYOPATHY

Table 2: Etiology

Category	Examples
Coronary	Plaque disruption, vasospasm, SCAD
Cardiac	Myocarditis, Takotsubo
Systemic	Sepsis, anemia, hypoxia

- PLAQUE EROSION



•MYOCARDIATIS AND INFLAMMATORY ETIOLOGIES

Pathophysiology:

There is no single explanation for how MINOCA causes myocardial injury. The mechanisms are layered – vascular, cellular, hormonal, and systemic – and they often interact. Understanding these factors guides treatment decisions for individual patients.

Patients with MINOCA present with biochemical evidence of Myocardial necrosis– typically elevated troponin levels– alongside ischemic electrocardiographic changes and anginal symptoms yet demonstrate angiographically patent or near patent coronary vessels.

- ENDOTHELIAL VASOMOTOR DYSFUNCTION
- Rho-KINASE PATHWAY ACTIVATION AND SMOOTH MUSCLE HYPER REACTIVITY
- PLATELET HYPERREACTIVITY AND THROMBOGENIC MILLIEU
- CATECHOLAMINE – MEDIATED MYOCARDIAL INJURY

Risk Factors and Epidemiology:

MINOCA predominantly affects younger women with unique risk factors including high stress, auto-immune disease, and smoking. MINOCA patients tend to have a less atherogenic risk profile than those with obstructive CAD, fewer diabetics, but traditional risk factors are still present. Hypertension is found roughly 40-50 percent of cases, and smoking which directly associated with vasospasm, also demographics as women are more frequently affected typically at a younger age than commonly heart attack patients, mental health conditions as high prevalence of anxiety and depression which may contribute to stress induced coronary artery spasms.

Other traditional CAD risk factors such as hypertension, diabetes mellitus, tobacco abuse and a family history of MI, are less frequent in MINOCA patients.

Diagnosis:

A MINOCA diagnosis is not a conclusion – it is a starting point. The angiogram has ruled out obstructive disease, but that only narrows the field.

It requires 3 step process confirming an acute heart attack (MI) ensuring coronary arteries have no blockage (<50%), and investigating the underlying cause.

The diagnosis of MINOCA with AMI fulfills the following criteria:

1. Acute Myocardial Infarction (modified from the “Fourth Universal Definition of MI” criteria)
 - Detection of rise or fall of cTn with atleast 1 value above 99th percentile upper reference and corroborative clinical evidence of infarction evidenced by atleast 1 of the following:
 - Symptoms of Myocardial ischemia



- New ischemic electrocardiographic changes
 - Development of pathological Q waves
 - 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 in any major epicardial vessel.
 3. Alternate diagnoses include but are not limited to non ischemic causes such as sepsis, pulmonary embolism, and Myocarditis.

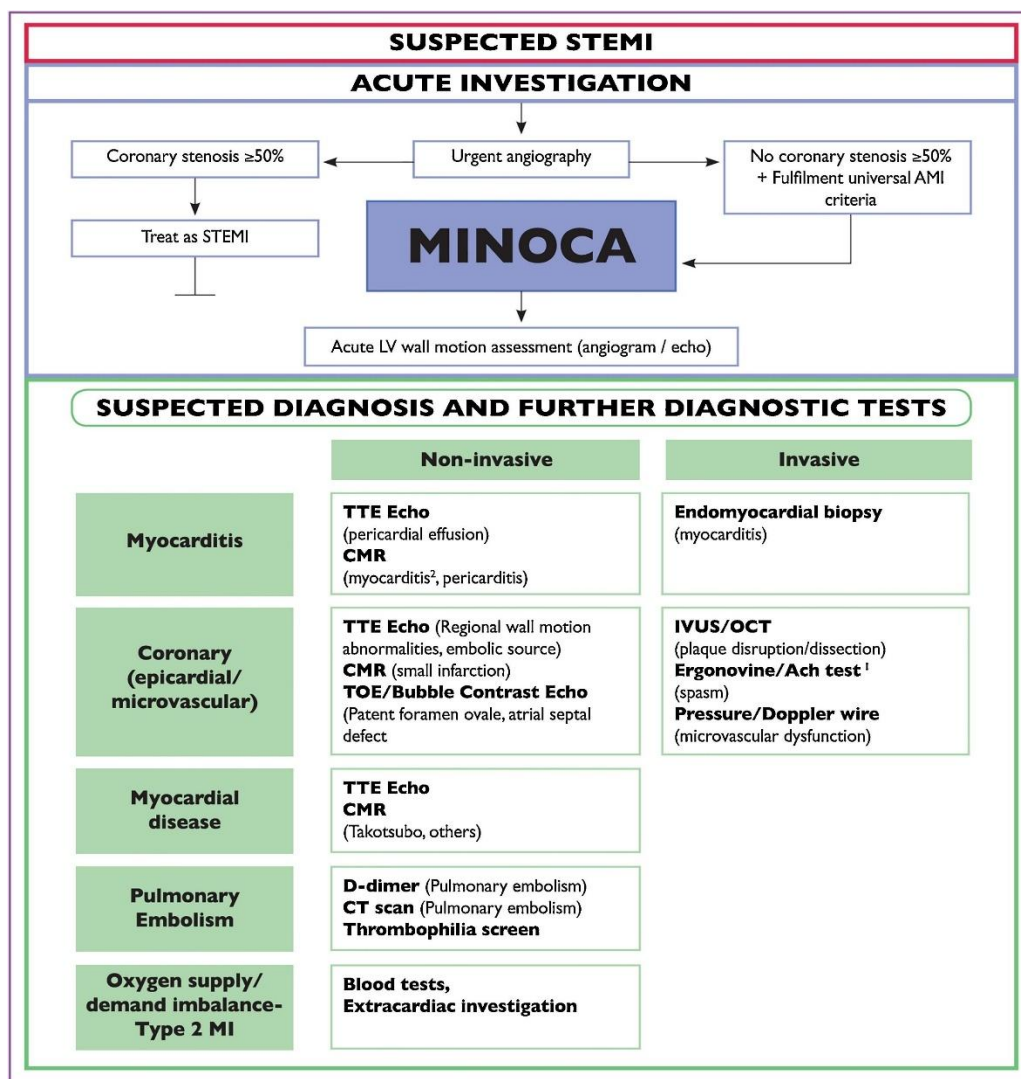
Treatment:

Treating MINOCA without knowing the mechanism is like prescribing antibiotics without knowing the organism.

MINOCA is treated the same way as other heart attacks: Antiplatelet therapy, statins, ACE-inhibitors/ angiotensin receptor blockers and beta- blockers.

For vasospastic MINOCA, calcium channel blockers are the cornerstone of long term management.

Depression, anxiety, and PTSD affect 30-50% of MINOCA survivors and independently worsen cardiovascular prognosis.





Complications and Prevention:

Complications:

Despite normal looking coronary arteries, the acute phase of MINOCA carries genuine risks, as it is not a benign condition and can lead to serious complications, including major adverse cardiovascular events (MACE) like heart failure, recurrent infarction and sudden cardiac death.

Although the main arteries are not blocked, complications arise from the underlying, often hidden, causes of the attack, which includes coronary spasm, plaque disruption, or spontaneous coronary artery dissection (SCAD).

Prevention:

Prevention in MINOCA requires addressing both the specific culprit mechanisms and the broader cardiovascular risk landscape. Mechanism- specific prevention is equally important as vasospasm patients need indefinite calcium channel blockers therapy and strict avoidance of triggers like cold, emotional stress, cocaine and non-selective beta-blockers.

Women with prior SCAD require individualized counselling about pregnancy risk, given 4-5% estimated recurrence rate per pregnancy

Perhaps most importantly, MINOCA patients actually receive in routine practice remains one of the field's most pressing challenges.

Conclusion:

MINOCA challenges one of cardiology's most deeply held assumptions, that a heart attack without a blocked artery is somehow less serious, less real, or less deserving of the same investigative intensity applied to obstructive disease.

What remains is the harder work of changing practice at the clinical level. MINOCA patients, disproportionately young women, often psychologically devastated by an event they were not supposed to have and deserve complete answers, not a discharge letter that says their arteries looked fine. That means investing in awareness, diagnostic infrastructure, and the kind of cross- functional follow-up that this complex and still not fully understood condition genuinely requires.

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