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MINOCA: MI With Non-Obstructive Coronary Artery Disease

AHM Waliul Islam, Sofian Johar, Partho Pritom Saha Roy, Ezam Emran, Moncey Oomen
Department Of Cardiology, RIPAS Hospital, Brunei Darussalam

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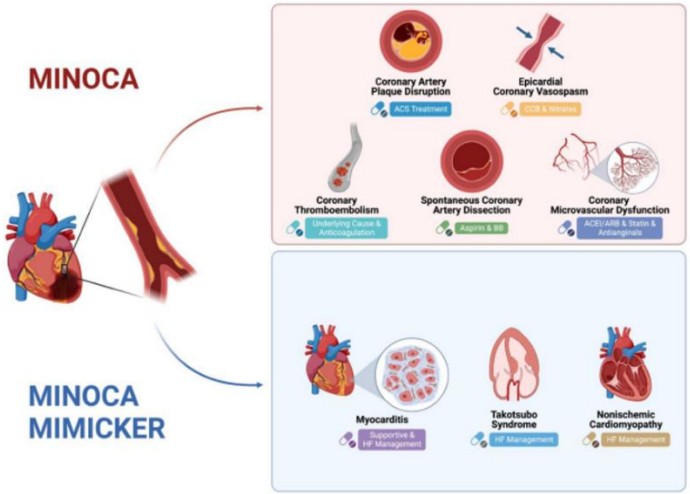
Myocardial Infarction in patient with non-obstructive coronary artery disease (MINOCA) is evident in up to 5-15% of all acute myocardial infarctions (AMI) and disproportionately affects young female. MINOCA Patients has worst prognosis than patients without cardiovascular diseases. A minority of patients with AMI are found to have MINOCA. Some physicians fail to realize that the absence of obstructive coronary artery disease does not exclude the AMI possibility. MINOCA has a number of heterogenous causes, including coronary disruption, coronary vasospasm, coronary embolism, spontaneous coronary artery dissection (SCAD) and coronary microvascular disfunction (CMD) and non- ischemic mechanism (Takotsubo cardiomyopathy, myocarditis and non-ischemic cardiomyopathy) the MINOCA mimickers. Multimodality Imaging such as intravascular imaging (IVUS, OCT), CT imaging, cardiac magnetic resonance (CMR) including provocative test for coronary vasospasm might be necessary to determine the cause and treatment. MINOCA might have a better prognosis than MI with obstructive CAD, and it is not benign.

Patients with acute MI routinely undergo coronary angiography to identify obstructive CAD. However, MI and non-obstructive coronary arteries (MINOCA) accounts for upto 5-15% of patients presenting with acute coronary syndrome (ACS) and is associated with poor cardiovascular outcomes despite the lack of epicardial obstructive atherosclerotic plaque.[1-3] Myocardial Infarction without significant obstructive coronary artery disease (CAD) has been observed for decades in patients presenting with MI without culprit artery lesion. Occurrence of Acute Myocardial Infarction (AMI) without significant coronary artery disease (CAD) reported almost 80 years before.[4] The term MINOCA has recently been used to describe these patients.[5] A reasonable number of patients with AMI are found to have MINOCA.[6-8] Unfortunately, some physicians fail to realize that the absence of obstructive CAD doesn't exclude the possibility of AMI.

In the year 2017, the European Society of Cardiology (ESC) position paper on MINOCA introduced diagnostic criteria for MINOCA based on third universal definition of MI as follows; the presence of positive cardiac biomarkers with clinical evidence of infarction, absence of stenosis (>50%) in any epicardial coronary arteries on coronary angiography.[9] The pathologic mechanism of MINOCA include; coronary artery plaque disruption, coronary vasospasm, coronary microvascular dysfunction, Spontaneous coronary dissection (SCAD). Coronary embolism/ thrombus, takotsubo cardiomyopathy, myocarditis and non-ischemic cardiomyopathy. In 2018, the universal definition of MI to exclude non-ischemic mechanism like takotsubo cardiomyopathy, myocarditis and non-ischemic cardiomyopathy.[10] American Heart Association (AHA) in 2019 excluded non-ischemic mechanism from the MINOCA definition and labelled them MINOCA-mimickers.[1]

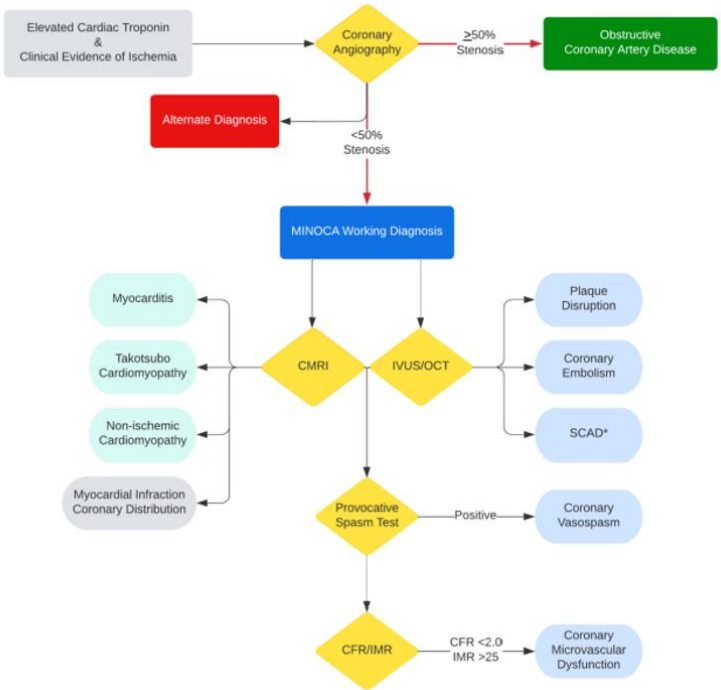
MINOCA is found in 5% of AMI patients,[1,3] it may affects upto 5- 15% in patients undergoing cardiac catheterization for MI.[1] In the early, Gibson at el., observed slower blood flow in some non-culprit arteries in MI patients.[11] This was considered a sign of microvascular dysfunction. Compare to patients with MI secondary to coronary artery disease (MI-CAD), MINOCA patients are women and younger and less likely to have dyslipidaemia.[12] Coronary plaque disruption is considered one of the most common causes of MINOCA, along with coronary vasospasm, coronary embolism, spontaneous coronary artery dissection (SCAD) and coronary microvascular dysfunction.[13]



Mechanism of MINOCA and MINOCA-MIMICKERS and Differences in Treatment

MINOCA is considered a heterogenous working diagnosis with an estimated prevalence of 5-15% among all acute myocardial infarction (AMI) patients.[14-15] In a pooled analysis of 23 studies, prevalent of MINOCA was 8.1% among 806851 consecutive AMI patient.[16] MINOCA has been reported by large national registries worldwide, including US, Japan, Poland, Sweden with the incidence of MINOCA 2.9-10.2%.[17-19] Compared to MI with obstructive CAD, MINOCA patients were younger with a median age of 61 more common in black and Hispanic.[16]

The majority MINOCA patient (70-80%) present with non-ST elevation myocardial infarction (NSTMI), limiting understanding of ST-elevation myocardial infarction (STEMI) in MINOCA.[16] ST-elevation at presentation is a predictor of all cause death in MINOCA.[14] STEMI has higher risk of mortality than those with NSTMI. A single centre retrospective study patients undergoing coronary angiogram in UK (n= 2,521), reported that only 4.4% had MINOCA based on 2017 ESC criteria. All cause mortality was 3.6 %in 30 days, and 4.5 % at one year follow up.[20] In a single centre registry in Denmark (n=4793) 11% of STEMI activations had normal (0% stenosis) or non-obstructive (1-49%) coronary arteries but only 6.5% (n=30) had elevated cardiac troponin. In a multivariate analysis, long-term mortality risk was two-fold higher in patients with normal coronary arteries and elevated cardiac troponin than those obstructive CAD.[21]



Algorithm with multimodality imaging and testing for MINOCA and MINOCA MIMICKERS. IVUS/OCT should be avoided if SCAD Suspected

The duration of therapy and optimal medical treatment is not well established for patient with MINOCA with no recurrent angina.[1] At initial presentation, management of MINOCA is similar to MI-CAD patient. Once angiography demonstrate no obstructive disease, treatment should be tailored to underlying pathophysiology of MINOCA. The *SWEDEHEART* study demonstrated a significant mortality benefit in patients with MINOCA on statins and angiotensin converting enzyme inhibitor. For secondary prevention.[17,22] Korean registry also showed that prescription of renin-angiotensin blocker and statin at discharge was associated with lower mortality in patients with MINOCA.[1,3,9] All patient with MINOCA should receive Statin, Beta-blocker and aspirin

The duration of DAPT in MINOCA is not clear. Compelling evidence for a beneficial effect of DAPT in case of small plaque rupture in non-significant stenosis and without overlying thrombus is lacking. In SCAD long term low-dose Aspirin is recommended for secondary prevention after MINOCA. A post hoc analysis of CURRENT-OASIS 7, randomized trial comparing high dose versus standard dose of clopidogrel in patient with MINOCA as opposed to in patients with MI-CAD.[23] The ESC guidelines advocate 1 year followed by indefinite single antiplatelet therapy in cases where plaque disruption is the suspected mechanism. [9] The American Heart Association recommend Aspirin monotherapy with consideration of second antiplatelt.[1]

The *SWEDEHEART* trial failed to show a significant benefit of Beta blocker at 1 year in patient with MINOCA.[17] The WARRIOR Trial focuses individual with non-obstructive CAD, statin and ACE-I/ARB improves MACE with compare to standard care.[24] *Smilowitz et al*, in a registry of 5,913 patients with MINOCA, has found considerable variable in discharge prescription of ACE-I/ARB and Beta Blockers. In a median of 45.6% patients received ACE-I/ARB, while 74.1% were discharged on Beta Blockers. [25]

In addition, those with plaque disruption not undergoing stenting may be treated with DAPT by adding ticagrelor for <1 month, based on low revascularization rates at 1 and 4 year follow up , 5.7% and 21.1%.[26] Beta blocker and renin-angiotensin system inhibitors should be considered in those with LV dysfunction.[1] Long-acting calcium channel antagonist (dihydropyridine and non-dihydropyridine) are used widely in MINOCA patients secondary to epicardial coronary vasospasm given that they relax vascular smooth muscles secondary to suppressed calcium flow.

Based on non-randomized observations, conservative management is favoured in patients with SCAD over percutaneous coronary intervention (PCI), given that majority of dissections heal with conservative management and the increased risk of interventions.[27] There is a paucity of data on the effectiveness of PCI with or without drug-eluting stents for the treatment of culprit MINOCA lesions due to plaque ulceration or erosion.[1] Interventional management of MINOCA patient with spontaneous coronary artery disease (SCAD) either by PCI or CABG is controversial. In presence of SCAD, stenting is associated with increased risk of complications.[28] Therefore, a conservative approach without angioplasty is recommended in majority of patients. PCI is suggested only in the presence of high-risk anatomical features including severe proximal locations in the left main coronary and left anterior descending artery, low TIMI grade or ongoing ischemia with hemodynamic instability.[28] CABG does not represent an appropriate solution in most patients with SCAD. In fact, long term patency of bypass grafts is poor due to the frequent healing of grafted arteries, which may lead to competitive flow and sub-sequent graft occlusion.[26]

MI and non-obstructive coronary arteries (MINOCA) are recognized as an important contributor to adverse cardiovascular outcomes in both men and women but is particularly prevalent in young women. It can be present as both STEMI and non-STEMI and multiple coronary mechanism such as coronary plaque disruption, coronary artery spasm, coronary microvascular dysfunction, spontaneous coronary artery dissection and coronary thromboembolism can trigger MINOCA. It represents 5-15% of patients undergoing cardiac catherization for MI and is associated with higher adverse outcomes compared with normal population. Patients with MINOCA should be investigated further by IVUS, OCT to rule out plaque disruption or SCAD, if required measurement by CFR and IMR to rule out CMD and provocating rule out coronary vasospasm. In addition, Cardiac Magnetic Resonance (CMR) may be useful to assess gadolinium enhancement consistent with MI to help exclude MINOCA mimic such as stress induced cardiomyopathy and myocarditis. Secondary prevention with ACE-I and statin can be used in MINOCA, but the indication for DAPT and other therapy in MINOCA management beyond 1-year is unknown.[29]

Patients with MINOCA are at higher risk of cardiovascular mortality and morbidity compared to the general populations.[16] MINOCA is not benign and associated with in hospital mortality of 3-5% at 1 year and 24% risk of MACE including composite cardiac mortality, reinfarction, heart failure and stroke at 4 years. The *VIRGO* study showed 1-year mortality in patients with MINOCA approximately 5%. Additionally, *Zeng et al* reported that 1-year composite MACE including death, MI, stroke, target lesion revascularization and angina rehospitalization was 15.3% for atherosclerotic causes versus 4.5% for non-atherosclerotic causes of MINOCA.

MINOCA is not well addressed in our patient population. With the available resources of IVUS, OCT, CMR we can address in a broader way in our patient population. CT scanning in future may become a frontline gatekeeper for non-invasive diagnosis of MINOCA. At present with the available resources and medication, can treat the patient with MINOCA. Thus, to reduce the morbidity and mortality in this group of MINOCA patients.

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