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INTRAVASCULAR IMAGING (AG TRUESDELL, SECTION EDITOR)

The Role of Imaging for MINOCA (Myocardial Infarction with No Obstructive Coronary Artery Disease): a Review of Literature and Current Perspectives

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Abstract

Purpose of Review The objective of this review is to summarize scientific statements on the diagnosis and management of myocardial infarction with no obstructive coronary artery disease (MINOCA); define the diagnostic role of optical coherence tomography (OCT), intravascular ultrasound (IVUS), and cardiac magnetic resonance imaging (CMR); and provide representative case examples.

Recent Findings The majority of patients with MINOCA are evaluated by conventional coronary angiography. However, intracoronary imaging using OCT or IVUS permits more accurate understanding of the underlying pathology. These and other imaging modalities provide significant diagnostic and prognostic value.

Summary Although nonobstructive disease is the hallmark of the disease, MINOCA is associated with significant morbidity and mortality. Every effort to define the underlying pathology is necessary and requires more standardized use of imaging in clinical practice.

Keywords MINOCA · Imaging · IVUS · OCT

Introduction

The term MINOCA (myocardial infarction with no obstructive coronary artery disease) was coined to describe patients presenting with an acute myocardial infarction (AMI) without evidence of obstructive coronary artery disease (CAD) [1, 2]. In the early studies of both ST elevation (STEMI) and non-ST elevation myocardial infarction (NSTEMI), approximately 10% of patients had no significant CAD on coronary angiography, a finding that was later confirmed in several AMI registries [3–6]. Epidemiologically, MINOCA is a syndrome with a prevalence of 6–14%. Several etiologies have been recognized including, but not limited to, plaque disruption, coronary spasm, and coronary thromboembolism [7]. Further evaluation is necessary to determine the underlying diagnosis, appropriate management, and prognosis of individuals with MINOCA.

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Clinical Presentation and Outcomes

Clinically, patients with MINOCA may be differentiated from patients with a myocardial infarction (MI) secondary to atherosclerosis by certain characteristics. Several large registries reported MINOCA in predominantly young female patients (up to 40%) presenting with NSTEMI [7–9]. MINOCA patients are unique in that they have a lower prevalence of traditional atherosclerotic cardiovascular risk factors and have a lower but clinically significant annual mortality rate [5, 7]. Safdar et al. reported a 54% prevalence rate for dyslipidemia

and hypertension in those with MINOCA in the Variations in Recovery: Role of Gender on Outcomes of Young AMI Patients (VIRGO) study [10]. The VIRGO study also revealed that patients with MINOCA had a higher prevalence of hypercoagulable disorders and were more likely to be non-white [10]. This study demonstrated that the course of MINOCA was not benign. The length of hospital stay and short- and long-term outcomes in MINOCA were similar to patients with MI and CAD (MICAD). However, the 12-month mortality for MINOCA was two times higher than the 0.5% annual mortality rate observed for middle-aged women in the general population of the USA [11]. In the SWEDEHEART registry, approximately 6% of patients with MINOCA had a subsequent MI, with progression of clinically important coronary artery stenosis at the time of the second MI [12]. In this registry, 22% of patients with MINOCA who had developed a re-infarction died during the follow-up period.

In the VIRGO registry, MINOCA phenotypes were classified into 5 types: class I included patients with atherosclerotic obstructive disease who underwent revascularization; class II included those with obstructive coronary artery disease (≥ 50%) without evident plaque rupture/thrombosis; class III included nonobstructive coronary artery disease (< 50%); class IV included patients with a non-plaque mechanism such as coronary artery vasospasm (relieved by intracoronary nitroglycerin), spontaneous coronary artery dissection (SCAD), and coronary embolization; and class V included those with an unidentified pathology. Intracoronary imaging was not utilized for classification [4]. A comprehensive list of possible underlying etiologies is described in Table 1. The most common causes include plaque disruption, coronary artery spasm, thromboembolism, coronary dissection, Takotsubo cardiomyopathy, myocarditis, and other forms of type 2 myocardial infarction [13, 14]. The CRUSADE by Patel et al evaluated the prevalence, predictors and outcomes of NSTEMI with insignificant coronary disease. The investigators reported an incidence of MINOCA of 9% with a low incidence of adverse outcomes. The strongest predictors of insignificant obstructive coronary artery disease were female sex and young age. Propensity-Matched analysis From the Acute Catheterization and Urgent Intervention Triage Strategy Trial (Acuity) noted that individuals with a NSTEMI and elevated troponin levels without obstructive coronary disease have a low rate of subsequent myocardial infarction and unplanned revascularization. However, they are at risk for 1-year mortality from non-cardiac causes [15, 16].

Table 1 Underlying pathophysiological mechanisms/possible etiologies of MINOCA

Coronary causes	Non-coronary cardiac causes	Non-cardiac disorders
Vasospastic angina	Myocarditis	Pulmonary embolism
Coronary microvascular disorders 1. Microvascular angina 2. Microvascular spasm 3. Coronary slow flow	Takotsubo cardiomyopathy	Renal impairment
Coronary plaque disruption	Other cardiomyopathies	Stroke
Spontaneous coronary Thrombosis/embolism		Sepsis
Coronary artery dissection		Acute respiratory distress syndrome
Acute aortic dissection extending into the coronary arteries		

Position Statements for the Diagnosis and Management of MINOCA

In order to address the varying pathophysiologies, etiologies, disease characteristics, and outcomes associated with

Table 2 Diagnostic criteria for myocardial infarction with nonobstructive coronary arteries (MINOCA)

The diagnosis of MINOCA is made after coronary angiography in a patient presenting with an acute myocardial infarct based on the following criteria
1. AMI criteria
(a) Positive cardiac biomarker (preferably cardiac troponin) defined as a rise and/or fall in serial levels, with at least one value above the 99th percentile upper reference limit
(b) Corroborative clinical evidence of infarction including at least one of the following:
(i) Symptoms of ischemia
(ii) New or presumed new significant ST-T changes or new LBBB
(iii) Development of pathological Q waves
(iv) Imaging evidence of new loss of viable myocardium or new RWMA
(v) Intracoronary thrombus evident on angiography or at autopsy
2. Nonobstructive coronary arteries on angiography
(a) Defined as the absence of obstructive CAD on angiography (no coronary artery stenosis ≥ 50%) in the infarct-related artery
(b) This includes patients with the following:
(i) Normal coronary arteries (no stenosis > 30%)
(ii) Mild coronary atherosclerosis (stenosis > 30% but < 50%)
3. No clinically overt specific cause for the acute presentation
(a) At the time of angiography, the specific diagnosis is not apparent
(b) It is necessary to further evaluate the patient for the underlying cause of MINOCA

LBBB left bundle branch block, *RWMA* regional wall motion abnormalities, *CAD* coronary artery disease

MINOCA, position statements were developed by the European Society of Cardiology (ESC) in 2017 and the American Heart Association (AHA) in 2019. These statements described the diagnostic criteria for MINOCA based on both clinical and imaging criteria (Table 2) [2]. The Fourth Universal Definition of MI is employed in addition to the coronary angiographic finding of no coronary artery stenosis ≥ 50% in the infarct-related artery. In the diagnosis of MINOCA, it is important to exclude alternate causes for an elevated troponin such as sepsis or pulmonary embolism. It is equally important to rule out obstructive disease that may have gone unrecognized by the basic angiogram such as subtle plaque disruption or embolism as well as microvascular dysfunction and vasospasm. Finally, diseases that can mimic an MI (e.g., myocarditis) must also be appropriately diagnosed. An algorithm to evaluate the myocardium with cardiac magnetic resonance (CMR) imaging, intracoronary structure with intravascular ultrasound (IVUS), and optical coherence tomography (OCT) as well as provocative testing for spasm and vasomotor integrity is endorsed by the ESC and AHA position statements (Fig. 1). In other studies, inducible coronary artery spasm has been reported in 27% and thrombophilia disorders in 14% of patients.

Cardiac Magnetic Resonance Imaging

As mentioned earlier, the diagnosis of MINOCA may trigger multiple different diagnostic evaluations depending on the clinical presentation. For the initial work-up, echocardiography and left ventriculography may provide information about the ejection fraction and wall motion abnormalities. In studies targeting potential etiologies of MINOCA, cardiac magnetic resonance (CMR) imaging has been a useful preliminary investigation and has been cited as the key diagnostic tool [2, 17]. Late gadolinium enhancement (LGE) is useful not only in the localization of the area of myocardial damage, but it can provide insights into the mechanism. For example, subendocardial LGE suggests an ischemic cause of injury, while a non-ischemic pattern may suggest myocarditis or an infiltrative disorder. CMR may also show large areas of myocardial edema with/without necrosis among MINOCA patients with plaque disruption, suggesting temporary cessation of flow [17–19]. Some CMR-based studies have shown typical MI in about 24% of patients, myocarditis in 33%, and no significant abnormalities in 26%. CMR can determine the extent of myocardial damage, left ventricular volumes, and function, thereby providing objective data to guide treatment and

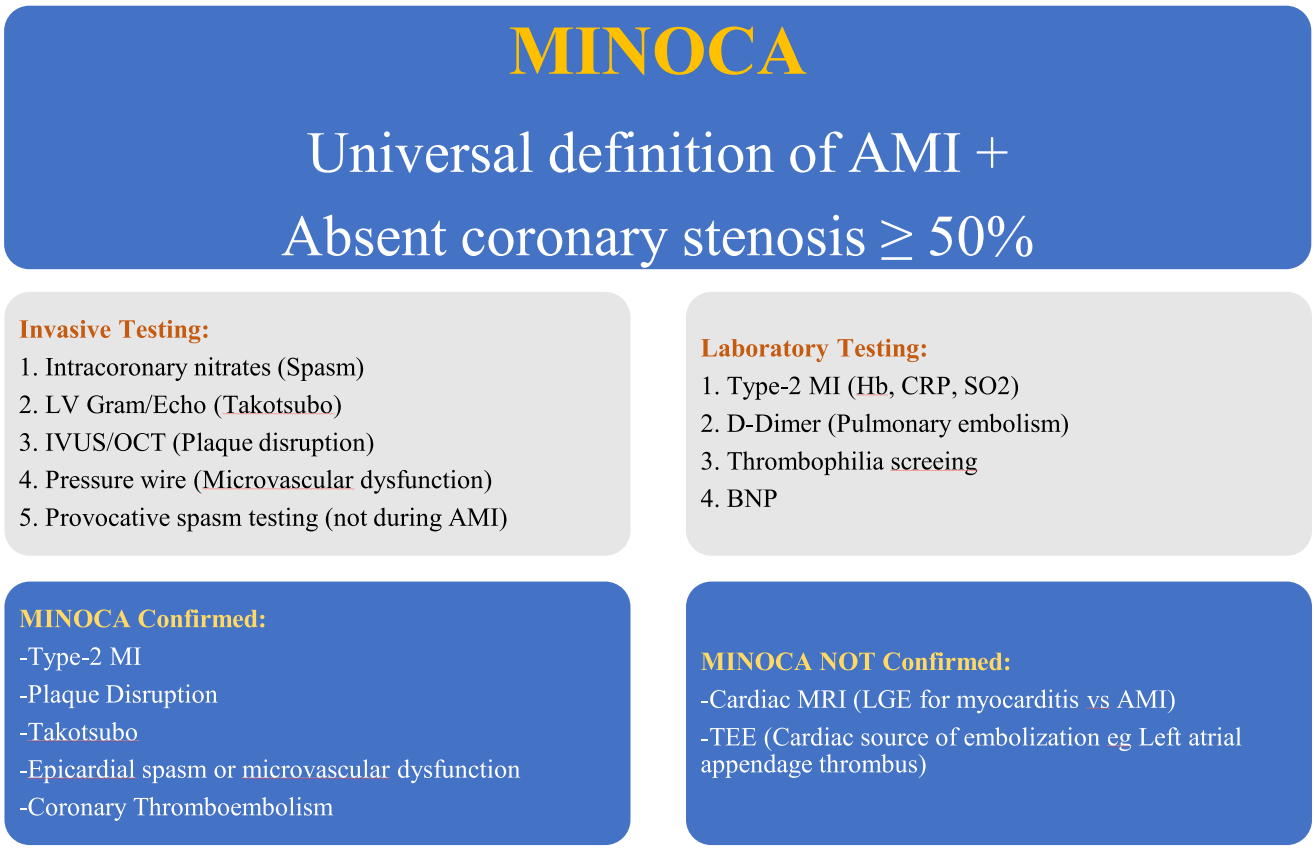


Fig. 1 Diagnostic algorithm for MINOCA. LV Gram, left ventriculogram; echo, echocardiogram; Hb, hemoglobin; CRP, C-reactive protein; SO2, oxygen saturation; BNP, brain natriuretic peptide; LGE, late gadolinium enhancement; AMI, acute myocardial infarction; TEE, transesophageal echocardiogram

prognosis. In their recent publication, Dastidar et al. reported that CMR identified the cause for the troponin rise in 74% of patients (25% myocarditis, 25% MI, and 25% cardiomyopathy). A normal CMR was reported in 26%. Over a median follow-up of 3.5 years, 5.7% patients died. Patients with cardiomyopathy had the worst prognosis (15% mortality), followed by MI (4% mortality) and myocarditis (2% mortality). The investigators concluded that a CMR diagnosis of cardiomyopathy and ST segment elevation are the most important predictors of mortality [17]. Examples highlighting the utility of CMR in diagnosing myocarditis and myocardial injury with corresponding normal coronary arteries are provided in Figs. 2, 3.

Intracoronary Imaging

The angiographic cut-off of < 50% stenosis for a diagnosis of MINOCA is limited by substantial inter- and intra-observer variability in visual estimation of stenosis. Similarly, the infrequent use of intravascular imaging often results in missed diagnoses of substantial atherosclerosis or thrombosis/spasm in suspected MINOCA cases. At the time of cardiac catheterization, intracoronary imaging, either with IVUS or OCT, is valuable in identifying plaque disruption as well as coronary dissection or thrombosis [2]. Plaque disruption is a frequent cause of MINOCA and accounts for 5–20% of all type 1 AMI cases. In two studies using IVUS, plaque disruption was found in approximately 40% of patients with MINOCA [20, 21]. The use of IVUS and OCT is still limited in spite of the well-recognized limitations of conventional coronary angiography alone. Both these modalities can guide and optimize most percutaneous revascularization procedures, as they can characterize the underlying plaque with identification of calcium burden, identify suboptimal stent apposition and expansion, define the mechanisms of stent failure (stent restenosis and stent thrombosis), and have been found to potentially lower contrast volume in dedicated low-contrast IVUS laboratories, commonly referred to as zero-contrast or low-contrast studies [22]. However, the ULTIMATE IVUS trial demonstrated greater contrast use with IVUS-guided strategy compared with an angiogram-only treatment strategy. While considered a relatively newer technique when compared with IVUS, OCT offers higher resolution and more anatomic detail [23]. For example, the CLI-OPCI II Study not only identified specific features of suboptimal stent expansion by OCT, but it also demonstrated higher adverse clinical outcomes in those with suboptimal stenting [24]. In contrast to IVUS, OCT uses infrared light for imaging. OCT catheters deliver and collect near infrared light with a wavelength of approximately 1300 nm to create cross-sectional images of the artery lumen and wall [25]. The infrared OCT machine measures echo time delay and signal intensity of the reflected or back-scattered light from the coronary wall structures during a pull-back along the coronary artery [26]. Cross sections of the coronary

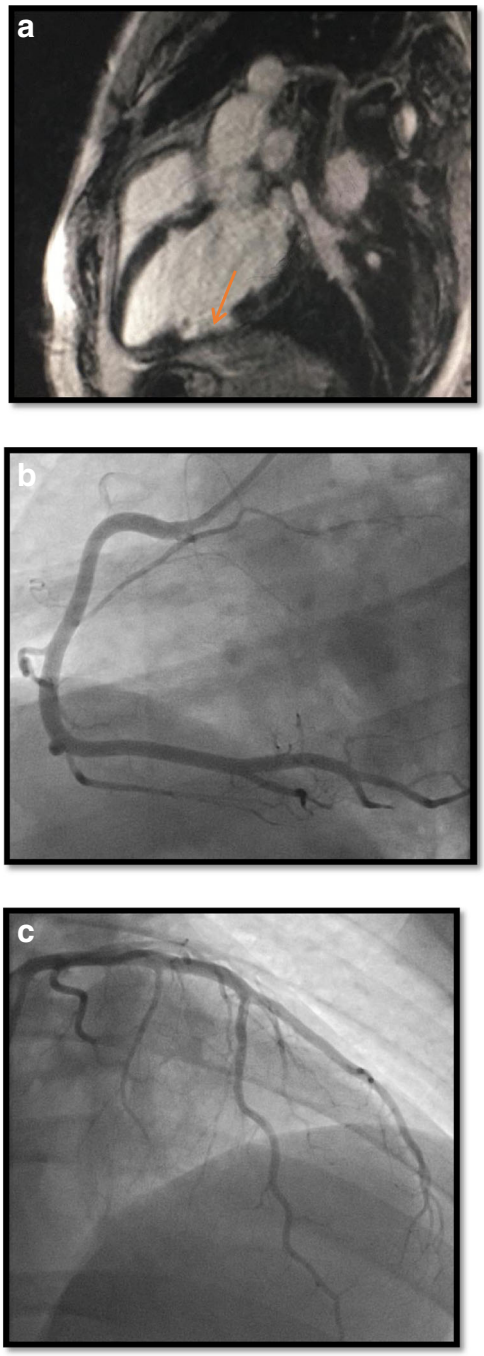
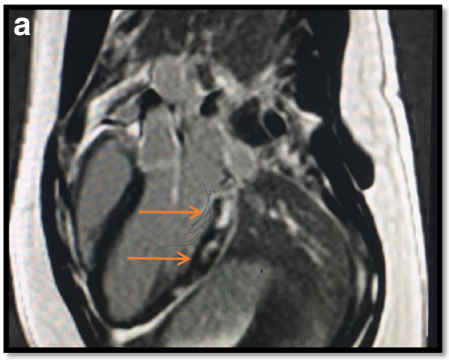


Fig. 2 **a** CMR image of MINOCA with a normal coronary angiogram. A 27-year-old gentleman with a 10-year history of smoking was admitted with atypical chest pain, dynamic EKG changes, and serum high sensitivity troponin that was elevated up to 8 ng/l. Transthoracic echocardiography shows infero-basal hypokinesia with an ejection fraction of 50%. The cardiac magnetic resonance (CMR) study demonstrated focal transmural edema of the inferolateral wall with corresponding focal transmural late gadolinium enhancement (LGE). **b** Coronary angiogram of the normal right coronary artery (left anterior oblique view). **c** Coronary angiogram of the normal left anterior descending coronary artery (right anterior oblique, cranial view)



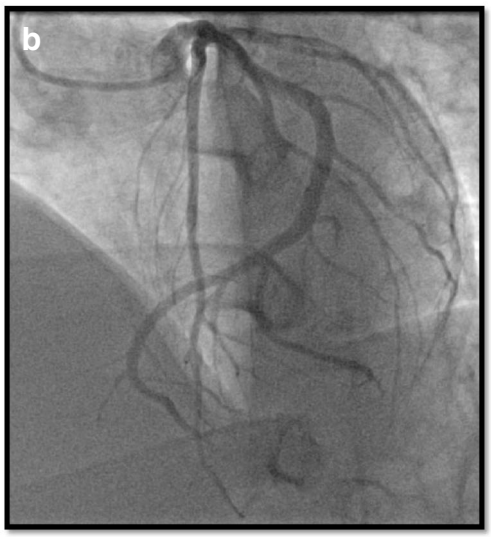


Fig. 3 **a** CMR image of myocarditis. A 19-year-old gentleman whose cardiovascular risk factors include smoking and hyperlipidemia was admitted through the ER with a NSTEMI. An echo revealed a preserved LV function with EF 50%. CMR demonstrated diffuse edema with patchy epicardial and midwall LGE. **b** Coronary angiogram of the normal left anterior descending and dominant left circumflex coronary artery (left anterior oblique, cranial view)

Table 3 Major differences between OCT and IVUS including technical and pathological characterization of coronary lesions

	OCT	IVUS
Tissue penetration	1–2 mm	6–10 mm
Technology	Near infrared	Ultrasound
Pull-back speed	20 mm/s	1 mm/s
Resolution		
Axial	10–20 μm	100–200 μm
Transverse	20–40 μm	200–300 μm
Minimum guide catheter size	5 Fr (6 Fr preferable)	5 French
Maximum frame rate	100 frames/s	30 frames/s
Lines per frame	500	256
Blood removal with contrast	Yes	No
Pathological characterization		
Necrotic core	++	+
Thin-cap fibroatheroma	+++	-
Thrombus	+++	+
Stent apposition/expansion	+++	++
Dissection	+++	++
Calcium	++	+++
Ostial lesion evaluation	+	++

OCT optical coherence tomography, *IVUS* intravascular ultrasound, *mm* millimeter, *μm* micrometer, *Fr* French

+++: excellent assessment

++: good assessment

+: average assessment

-: poor assessment

artery are then created allowing for real and offline analysis of each section. Axial resolution with OCT (100–200 μm) is much higher than that of IVUS (10–20). Another important difference is the lower depth of tissue penetration with OCT (2–3 mm) compared with IVUS (4–8 mm) [25]. Table 3 provides an overview of key differences between OCT and IVUS.

There are numerous advantages to adopting intracoronary imaging, OCT in particular, to complement coronary angiography; OCT can (1) differentiate tissue characteristics including plaque components (fibrous, calcified, versus lipid-rich plaque) [26]; (2) identify unstable plaque [27]; (3) differentiate plaque rupture vs erosion [28]; (4) identify red and white thombi [29]; (5) identify spontaneous or edge dissection during PCI^{-} [30]; and (6) detect incomplete stent apposition as well as in-stent restenosis [31–33]. Given the wide applications of intracoronary imaging, it can be useful in patients with MINOCA as it may identify the pathophysiological process and etiology. The second consensus document of the European Association of Percutaneous Cardiovascular Interventions (EAPCI) on the clinical use of intracoronary imaging focused on the utility of IVUS, OCT, and NIRS in facilitating angiographic interpretation and guidance of treatment of acute coronary syndromes beyond the mere guidance of stent selection and optimization of deployment [34]. In a study of 38 patients presenting with MI, plaque disruption was identified in 40% of patients with MINOCA [35]. Similarly, coronary thrombus was identified in 7 patients. In patients presenting with spontaneous coronary artery dissection (SCAD) [36], OCT was used to describe the presence and absence of fenestrations between the true (TL) and false lumens (FL). The authors found that in the absence of fenestrations, there was a significantly larger expansion of external elastic lamina and a larger false lumen; there were no significant differences in the density of the vasa vasorum in SCAD compared with the control subjects. Figure 4 is a representative panel of patients presenting with MINOCA and their corresponding OCT images.

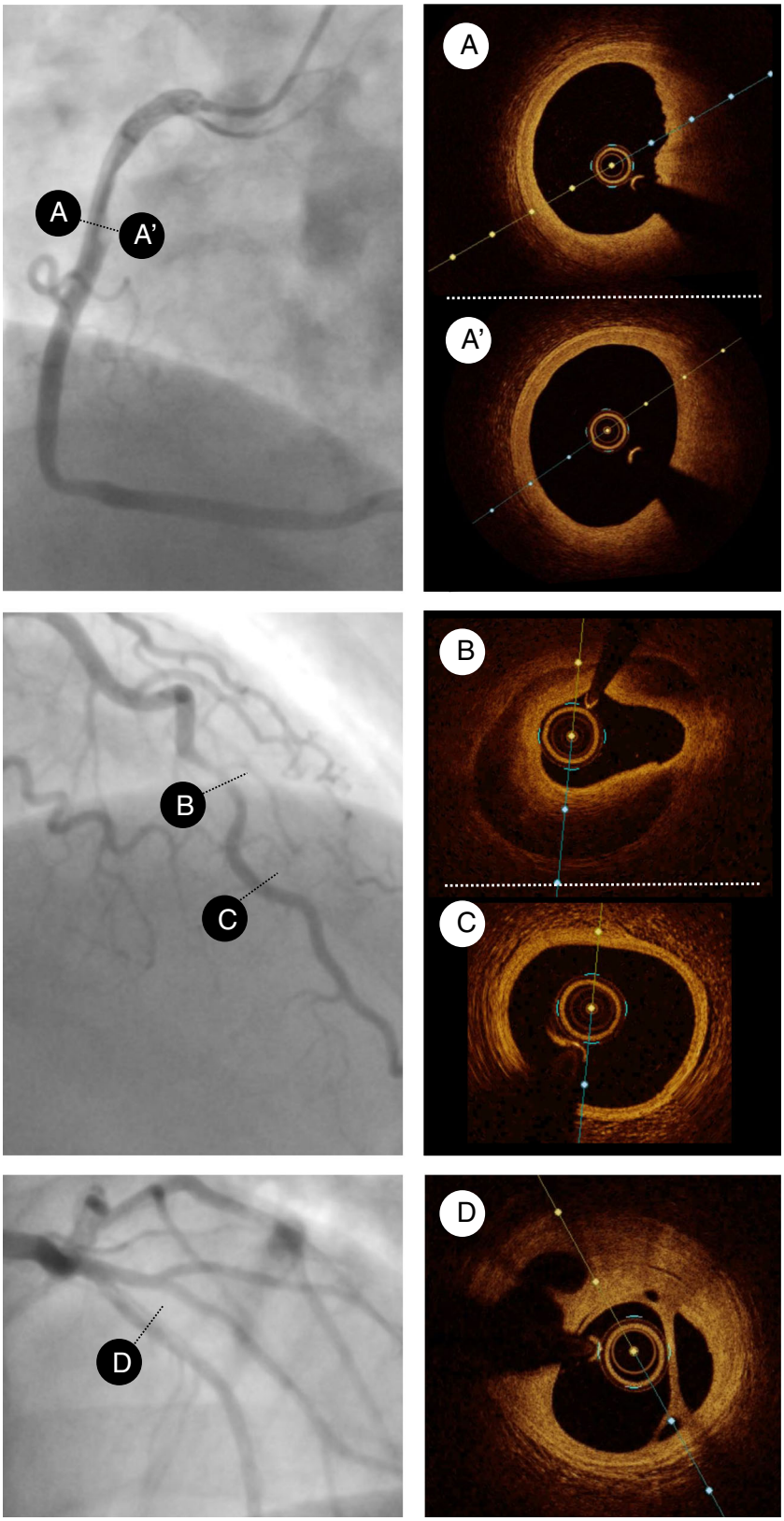


Fig. 4 OCT Panels of patients with MINOCA. Panel 1: A 52-year-old female presents with 2-h history of chest pain and inferior ST elevation on ECG. Risk factors include smoking and a family history of ischemic heart disease. Angiography demonstrates a moderate proximal stenosis with an intra-luminal filling defect likely representing thrombus (level A/A'). Immediate intracoronary imaging with OCT (panel A) reveals an irregular lumen contour and deep structure attenuation with minimal intimal thickening from 7 to 1 o'clock. Repeat OCT analysis 5 weeks later demonstrates resolution of the irregular lumen contour with evidence of an underlying thick-cap fibroatheroma (panel A'). Panel 2: A 48-year-old female presents with 24-h history of chest pain and T-wave inversion in ECG leads V2-V6. Her troponin is elevated, her cardiovascular risk factors are limited to smoking, and a family history and angiography is undertaken within 24 h on admission. A moderate stenosis is observed in the mid-LAD segment. OCT assessment reveals significant luminal restriction with evidence of dehiscence of the intimo-medial complex from the adventia (panel B), despite normal vessel architecture in the distal vessel (panel C), indicative of a type III spontaneous coronary artery dissection (a lesion mimicking coronary atherosclerosis). Panel 3: A 45-year-old intravenous drug user with a previous history of a conservatively managed myocardial infarction presented with chest pain and transient anterior T-wave changes. Angiography demonstrated a non-flow limiting abnormality in the mid-LAD with linear defects consistent with possible coronary dissection. OCT assessment demonstrated multiple lumens with fibrotic septae suggestive of a recanalized thrombus (panel D)

Conclusion

Conventional coronary angiography has been the standard method of investigation for individuals presenting with an MI whether MINOCA or MICAD. However, with advancements in both intracoronary imaging and noninvasive cardiac imaging technologies, especially CMR, a more accurate diagnosis of the underlying pathology may be achieved. Such modalities not only add diagnostic value, but they are useful in tailoring management and prognosticating long-term outcomes in MINOCA patients.

Compliance with Ethical Standards

Conflict of Interest All authors declare no conflict of interest.

Human and Animal Rights This article does not contain any studies with human or animal subjects performed by any of the authors.

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