

Optical coherence tomography and acetylcholine provocation for diagnosing coronary vasospasm in MINOCA patients

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Dear Editor,

Myocardial infarction with non-obstructive coronary artery disease (MINOCA) occurs in up to 14% of patients presenting with acute coronary syndrome (ACS).¹ Clinically, such patients present with ACS symptoms, elevation of troponin and coronary angiograms showing non-obstructive coronary arteries (<50% stenosis).² MINOCA has myriad aetiologies, which are classified according to coronary (i.e. coronary embolism, spasm or spontaneous dissection) and non-coronary pathologies (i.e. trauma or myocarditis).³

Coronary artery vasospasm is an important cause of ACS that can lead to myocardial infarction, ventricular arrhythmias and sudden death. Asians seem to have a higher propensity of vasospastic angina, but more recent studies have shown that this condition is also common among Caucasians.⁴⁻⁶ Coronary angiography alone may yield inconclusive results. Current journal consensus guidelines⁷ suggest considering acetylcholine provocation tests in MINOCA patients to induce coronary artery vasospasm and establish the underlying cause of ACS.⁸

Optical coherence tomography (OCT) is a high spatial resolution intracoronary imaging modality useful to detect subtle causes of ACS not identified by coronary angiography such as plaque rupture or recanalised thrombus.⁹

OCT identifies plaque rupture as a fibroatheroma with fibrous cap disruption over necrotic core,¹⁰ with or without cavity formation, whereas it detects thrombus as an intraluminal mass attached to the luminal surface or floating within the lumen¹¹ while plaque erosion can be seen as a plaque with thrombus and no evidence of rupture in multiple adjacent frames. Thrombosis of calcified nodules can be identified as evidence of thrombus in conjunction with calcium protruding into the lumen, frequently forming sharp, jutting angles.¹² Ruptured plaques of patients with ST elevation myocardial infarction (STEMI) are associated with a greater extent of cap disruption and a smaller minimal lumen

area compared with ruptured plaques of patients with non-STEMI.^{13,14} Ruptured culprit plaques of ACS had a lower lumen area compared with non-culprit plaques in the same patients who had undergone silent rupture.¹⁵

We present 3 cases of MINOCA, highlighting the roles of OCT and acetylcholine provocation tests to establish coronary vasospasm as the underlying cause.

Case 1

A 59-year-old male, without prior known cardiovascular risk factors, presented to the emergency department (ED) twice due to multiple episodes of chest pain over 1 month. He was scheduled for exercise stress echocardiogram for evaluation. Before stress echocardiogram was performed, he again presented with chest pain. Electrocardiogram (ECG) in ED showed acute anterolateral and inferior wall ST elevation. Emergency coronary angiogram showed luminal irregularities, TIMI 3 flow on all 3 coronary arteries with mild myocardial bridging on mid-left anterior descending (mid-LAD) artery (Fig. 1). OCT revealed no evidence of plaque rupture/recanalised thrombus. Acetylcholine challenge with 20 mcg and 100 mcg was positive for severe focal spasm in mid-LAD artery (Fig. 1) with reproduction of ST elevation and symptoms of chest pain. The spasm was reversed with intracoronary nitroglycerin. No coronary stents were implanted, and he was treated with dual antiplatelet therapy and oral calcium channel blockers (CCB).

Case 2

A 47-year-old male presented with recurrent left-sided chest pain. ECG showed Wellens' Type B pattern suggestive of LAD ischaemia with low-grade troponin rise and managed as non-ST elevation ACS. Coronary angiogram showed mid-LAD 30–40% stenosis, the rest of the coronaries have no significant obstruction (Fig. 1). OCT of mid-LAD lesion showed fibrous plaque with a plaque burden of 43%, but no signs of plaque rupture/recanalised

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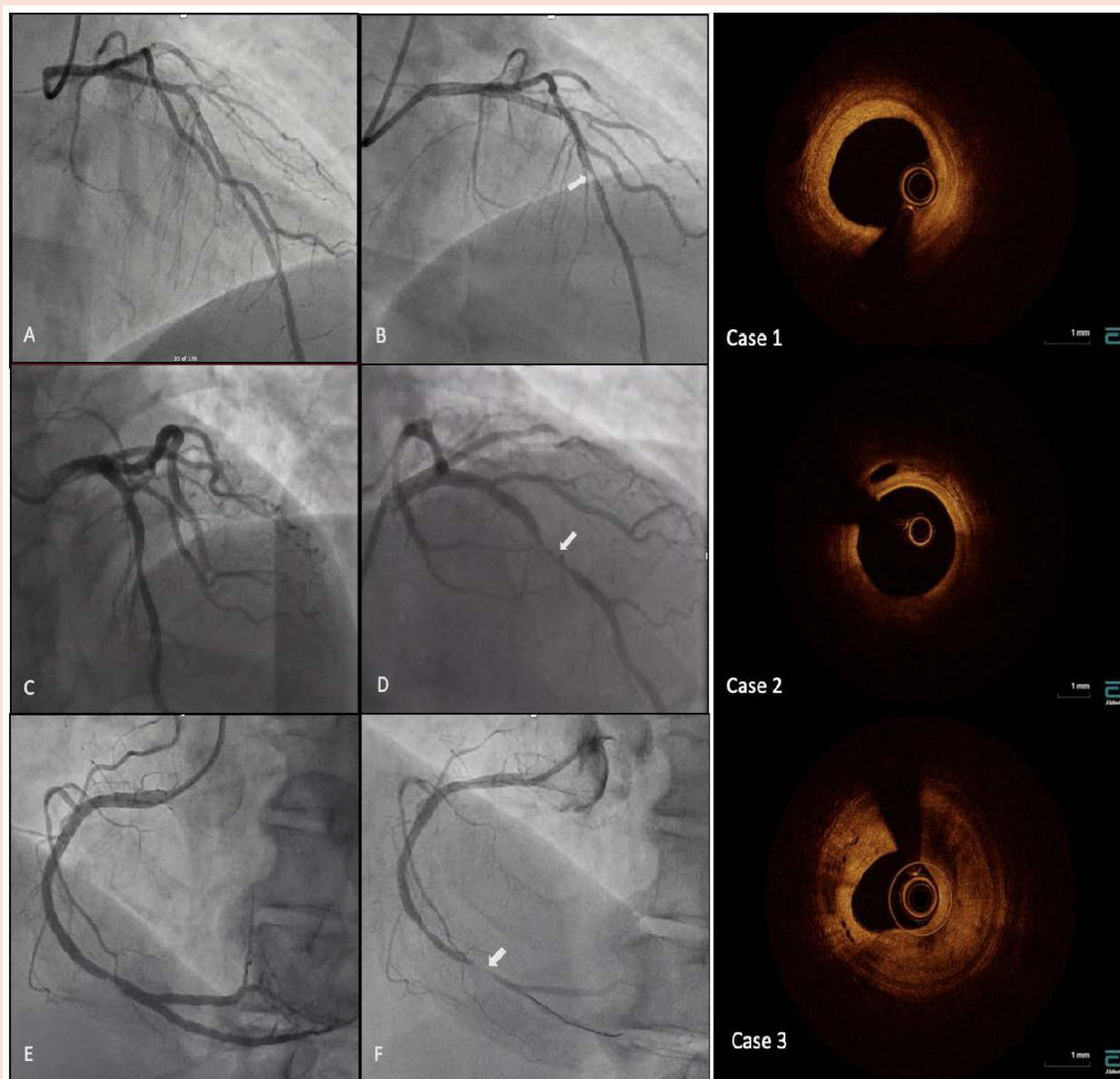
thrombus. Vasoreactivity study with intra-coronary (IC) acetylcholine 200 mcg demonstrated focal 90% coronary vasospasm of the mid-LAD (Fig. 1D) with patient developing chest pain and ECG changes. He was given CCB and nitrates as maintenance. Repeat ECG after 3 months showed resolution of deep T wave inversions.

Case 3

A 56-year-old male was admitted due to ST elevation on the inferior leads and regional wall

motion in the right coronary artery during treadmill stress echocardiogram, associated with epigastric discomfort. Coronary angiogram revealed non-obstructive lesions and minor disease in distal right coronary artery (RCA) (Fig. 1E). Fractional flow reserve measured across distal RCA with intravenous adenosine infusion was not haemodynamically significant. OCT was done and was negative for plaque rupture with no signs of recanalised thrombus. Further evaluation using vasoreactivity testing with acetylcholine 200 mcg

Fig. 1. Coronary angiography of the 3 patients with acute coronary syndrome.



(A), (C), (E) Non-obstructive lesions at baseline with (B), (D), (F) interval severe focal spasm (as shown in the arrows) noted on each patient after vasoreactivity testing with acetylcholine, respectively. Rightmost figures show optical coherence tomography images at the sites of the induced spasm of the 3 patients showing predominantly fibrous plaque with intact intimal layer and no evidence of plaque rupture, plaque erosion or thrombus.

showed severe focal spasm at the distal RCA (Fig. 1F). The spasm was reversed with intracoronary nitroglycerin. No coronary stents were implanted, and he was treated with dual antiplatelet therapy and oral CCB.

In Case 3, there was evidence of neo-vascularisation within the intima, suggesting some chronicity in this plaque.

All 3 patients had typical anginal symptoms with localising ECG features either of Wellens' syndrome or ST elevation. In these patients, coronary angiogram did not identify any culprit lesions that could account for the clinical presentations. OCT was performed in all these cases and ruled out plaque rupture and/or recanalised thrombus. Finally, vasoreactivity with acetylcholine, with maximum dose of up to 100–200 mcg given via slow intracoronary bolus demonstrated coronary artery vasospasm in all 3 patients.

This case series illustrates that a comprehensive coronary evaluation using OCT with acetylcholine provocation in addition to coronary angiography is essential to establish the diagnosis of coronary vasospasm as cause of ACS. As shown above, accurate diagnosis avoids unnecessary coronary artery stenting and is indispensable for tailored therapeutic management such as the use of CCB to avoid recurrence of further symptoms and ACS occurrence. Larger, multicentre studies are still needed to validate these findings to provide more robust and generalisable results.

Ethics statement

Not applicable

Declaration

The authors declare there are no affiliations with or involvement in any organisation or entity with any financial interest in the subject matter or materials discussed in this manuscript.

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