Optical coherence tomography: high-resolution imaging modality useful in identifying the pathophysiology of coronary vasospasm in acute coronary syndrome

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DESCRIPTION

A 39-year-old woman with gastro-oesophageal reflux disease, tobacco abuse and family history of premature coronary artery disease (CAD) presented to the hospital reporting of new onset substernal chest pain for 4 days prior to admission. She experienced multiple episodes of chest pain at rest and exertion without aggravating or alleviating factors. Each episode lasted for 15 min with radiation to the left arm. Associated symptoms included left arm tingling sensation, diaphoresis, dry mouth, blurry vision and dizziness. Physical examination was unremarkable. Initial vital signs were significant for tachycardia with heart rate in 130 s. ECG showed sinus tachycardia and ST-segment elevations in leads V1, V2 that were transient (figure 1). Serial cardiac biomarkers showed up-trending troponin levels at 2.05 ng/mL (normal range: 0.00–0.04 ng/mL). Patient was started on heparin drip, aspirin, statin, beta-blocker and scheduled for urgent cardiac catheterisation.

Transthoracic echocardiogram revealed a preserved ejection fraction of 60% without regional wall motion abnormalities. Coronary angiography showed severe (80%–90%) proximal stenosis of left anterior descending (LAD) with normal left circumflex and right coronary arteries (video 1). Our differential diagnosis included non-ST-segment elevation myocardial infarction (NSTEMI) and coronary vasospasm. An amount of 200 μg of intracoronary nitroglycerin was administered in the left coronary system. Repeated coronary angiography after adequate time was given to allow the nitroglycerin to percolate, which revealed persistence of the lesion (video 2). Given these angiographic findings, we decided to proceed with intervention of the LAD. We first inserted a 7-french XB 3.0 guide catheter into the left coronary artery. As we were inserting a run-through interventional wire

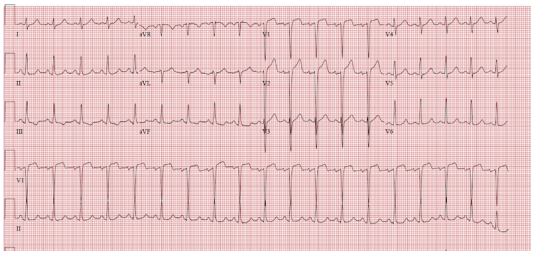
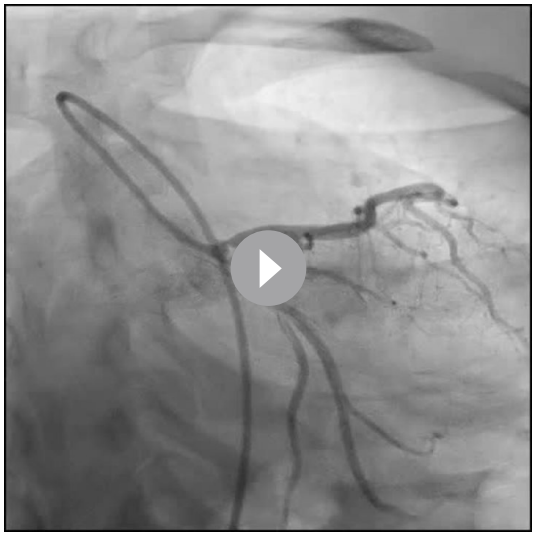
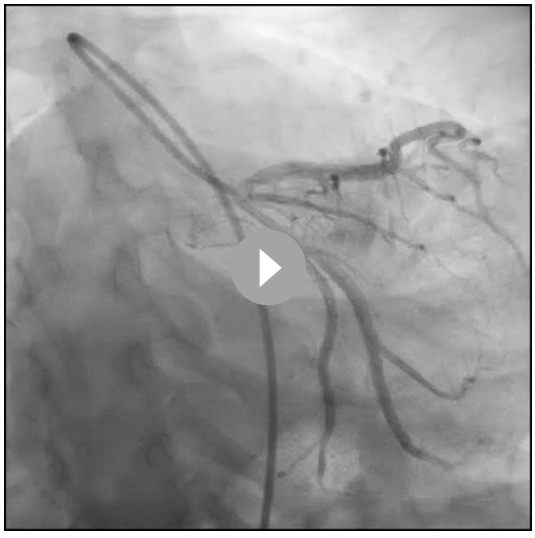


Figure 1 EKG. Transient ST-segment elevations in leads V1 and V2.



Video 1 AP caudal view. Coronary angiogram. Severe proximal stenosis of LAD. AP, Anterior/Posterior; LAD, left anterior descending.



Video 2 AP caudal view. Coronary angiogram. Persistence of the LAD lesion after intracoronary nitroglycerin. AP, Anterior/Posterior; LAD, left anterior descending.

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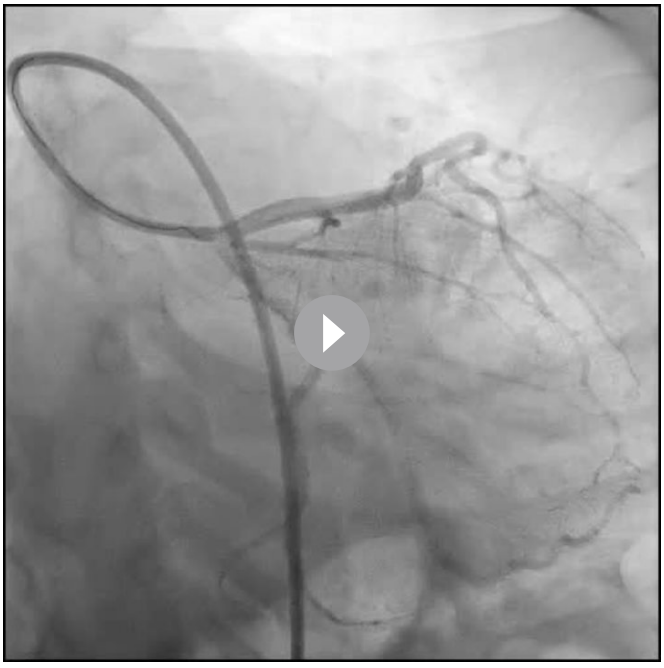


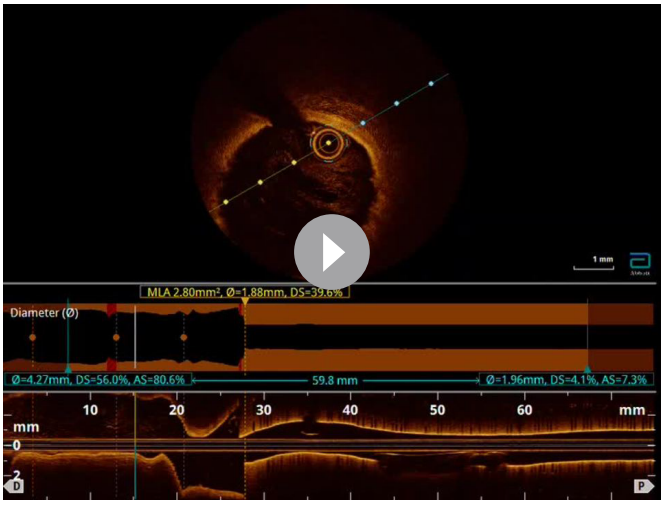
Video 3 AP caudal view. Coronary angiogram. Resolution of LAD lesion. AP, Anterior/Posterior; LAD, left anterior descending.

into the LAD, the lesion was not appreciated anymore in the proximal LAD, hence increasing the suspicion of coronary vasospasm. (video 3). The patient was asymptomatic during cardiac catheterisation. Decision was made to perform optical coherence tomography (OCT)-guided intravascular imaging of the LAD to assess intraluminal pathology in the LAD. OPTicross OCT catheter was inserted and intravascular images of the LAD were obtained, which revealed absence of CAD or plaque rupture in the LAD (video 4). The diagnosis of vasospastic angina (VSA) was, hence, confirmed.

Patient was started on calcium channel blockers. She was strongly advised to quit smoking and has been asymptomatic in outpatient follow-up visits.

OCT can help study pathology of coronary arterial wall at the vasospasm sites in patients with VSA.[1 2] Luminal irregularity, intraluminal thrombi and intimal erosion are the most common morphological features of coronary artery spasm sites visualised using OCT in patients with VSA.[3–6] VSA can occur in angiographically normal or near-normal coronary arteries.

Above case represents VSA as the cause for acute coronary syndrome confirmed with OCT, which helped in ruling out atherosclerotic CAD and plaque rupture. To evaluate culprit lesion, OCT was performed, which helped in confirming the mechanism of acute coronary syndrome in our patient.[7] Our patient had normal coronary arteries at the vasospasm sites on OCT. Troponin elevation can be seen in both coronary vasospasm and NSTEMI.[8]



Video 4 Optical coherence tomography. Absence of coronary artery disease or plaque rupture in LAD. LAD, left anterior descending.

Learning points

- Optical coherence tomography (OCT) can be used to confirm the mechanism of acute coronary syndrome.
- OCT reveals coronary artery wall microstructure at vasospastic sites.
- Coronary vasospasm and non-ST-segment elevation myocardial infarction can both cause elevation of troponin and present with typical features of chest pain.
- Coronary vasospasm is an uncommon presentation of acute coronary syndrome. It is less common than typical angina caused by atherosclerotic disease. It should be one of the leading differentials when younger age group patients present with chest pain.
- Cigarette smoking is a major risk factor in vasospastic angina and coronary artery disease.

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