

Acute coronary syndrome of the left main coronary artery caused by a huge floating thrombus in the ascending aorta: a case report of intravascular ultrasound effectiveness

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Background	Left main coronary artery (LMCA)–acute coronary syndrome (ACS) is a rare complication of a floating thrombus in the ascending aorta. However, diagnosing the aetiology of LMCA–ACS during an emergency situation is challeng- ing. We present a rare case of LMCA–ACS caused by a large thrombus in the ascending aorta, confirmed by intra- vascular ultrasound (IVUS).
Case summary	A 90-year-old woman presented to the emergency department complaining of chest pain and syncope. On admis- sion, her electrocardiogram showed normal sinus rhythm and a complete right bundle branch block with significant ST depression in the V3–V6 leads; hence, ACS was suspected. The first emergency angiogram of the left coronary artery showed filling defect in the proximal ascending aorta. IVUS revealed a large thrombus in the ascending aorta. The thrombus extended from the ascending aorta to the proximal left anterior descending coronary artery. IVUS confirmed that there was no dissection of the coronary artery or the proximal ascending aorta. Based on the IVUS findings, this case was diagnosed as ACS of the LMCA caused by a floating thrombus in the ascending aorta.
Discussion	This rare case of LMCA–ACS caused by a thrombus in the ascending aorta was confirmed by IVUS, which can be a useful imaging tool for diagnosing morphological abnormalities during emergencies.
Keywords	Intravascular ultrasound • Acute coronary syndrome • Floating thrombus • Ascending aorta • Left main coronary artery • Case report

Learning points

- A floating thrombus in the ascending aorta is a rare cause of left main coronary artery (LMCA)-acute coronary syndrome.
- Emergency use of intravascular ultrasound can assist in the immediate diagnosis of the aetiology of LMCA syndrome in an unstable haemodynamic condition.

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Introduction

It is well known that acute coronary syndrome (ACS) occurs as a ruptured plaque resulting in a thrombus; however, in rare cases, it might be caused without ruptured plaque such as dissection or other embolic sources.^{1–9} On the other hand, the haemodynamic status of ACS is often dramatically compromised prior to recanalization. Intravascular ultrasound (IVUS) is an imaging tool that can be utilized together with emergency percutaneous intervention for recanalization of ACS with or without unstable vital signs. Here, we present a rare case of left main coronary artery (LMCA)–ACS caused by a large thrombus in the ascending aorta, confirmed by IVUS, while the patient was in cardiogenic shock.

coronary angiography was scheduled for suspected ACS. Before the procedure, 200 mg aspirin and 300 mg clopidogrel were administered, in addition, 8000 units of unfractionated heparin (\sim 200 units per kg) were administered by intravenous bolus.

An intra-aortic balloon pump was inserted via the left common femoral artery in the catheter laboratory. The first angiogram of the left coronary artery showed filling defect in the proximal ascending aorta (*Figure 1* and *Video 1*). A guiding catheter was inserted into the left coronary artery, and the angiogram revealed no enhanced area at the LMCA or the left anterior descending coronary artery (LAD) (*Figure 2*). IVUS revealed a large thrombus (*Figure 3* and *Video 2*), extending from the ascending aorta to the proximal LAD, without dissection of the coronary artery or the proximal ascending aorta (*Figure 4* and *Video 3*). Thus, the patient was diagnosed with ACS of the LMCA caused by a floating thrombus in the ascending aorta. Although we performed thrombectomy several times, the

Timeline

Time	Event
Day1 Emergency department	Complaining of chest pain and syncope
	Electrocardiogram showed significant ST depression in the V3 to V6 leads.
	Subsequently, her blood pressure gradually decreased. Emergency coronary angiography was scheduled for suspected ACS.
Day1 Catheter laboratory	The first angiogram showed filling defect at the proximal ascending aorta.
	IVUS revealed a huge thrombus in the ascending aorta. The thrombus extended from the ascending aorta to the proximal LAD, and IVUS confirmed there was no dissection of the coronary artery or the proximal ascending aorta.
	We deployed a drug-eluting stent, and a final TIMI flow grade of 3 was obtained.
Day7	Unfortunately, she died.

Case presentation

A 90-year-old woman presented to the emergency department complaining of chest pain and syncope. Her medical history included chronic kidney disease (stage G4) and heart failure with preserved ejection fraction. Arrhythmia, atrial fibrillation, or atrial flutter had not been previously detected. On admission, her blood pressure (149/77 mmHg) and heart rate (68 b.p.m.) were stable, physical examination revealed no heart murmur. Electrocardiogram showed normal sinus rhythm and a complete right bundle branch block, with significant ST depression in the V3–V6 leads. Bedside transthoracic echocardiogram showed diffuse hypokinesis of the left ventricular wall with an ejection fraction of 20%; no evidence of apical thrombi, severe mitral regurgitation, and ventricular septal rupture were observed. Troponin I level was slightly elevated, at 0.039 ng/mL (normal, <0.034 ng/mL). Subsequently, the patient's blood pressure gradually decreased, and, as cardiogenic shock was expected, catecholamines (noradrenaline 0.2 γ) were initiated, and emergency

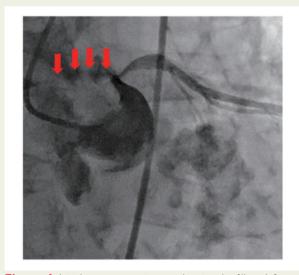


Figure I Initial coronary angiogram showing the filling defect at the proximal ascending aorta.



Video I The first angiogram of the left coronary artery showing filling defect in the proximal ascending aorta.



Figure 3 Intravascular ultrasound showing a large thrombus in the ascending aorta.

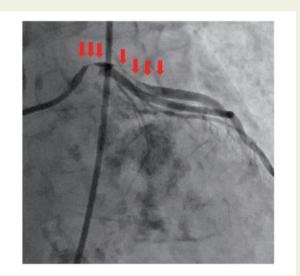


Figure 2 Angiogram using a guide catheter showing the filling defect at the left main coronary artery and the left anterior descending coronary artery.

thrombus could not be completely removed. Consequently, we deployed a drug-eluting stent, and a final Thrombolysis in Myocardial Infarction flow grade of 3 was obtained. The patient's vital signs were stable after the intervention. Contrast-enhanced computed tomography (CT) confirmed a large abnormal low-density area; thus, a large thrombus in the left coronary cusp without aneurysm was suspected. Additionally, contrast-enhanced CT confirmed that the ascending aorta was not dissected (*Figure 5*). We suggested surgical repair to remove the thrombus; however, the patient and her family opted for palliative care without invasive surgical intervention, as she was a nonagenarian. Therefore, antithrombotic (warfarin) and dual antiplatelet therapy (aspirin

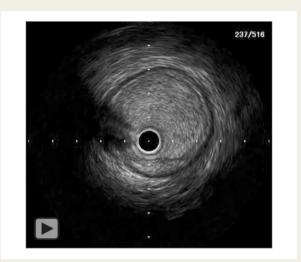


Video 2 Intravascular ultrasound showing a large thrombus in the ascending aorta.

and clopidogrel) were initiated, together with unfractionated heparin by continuous intravenous infusion (therapeutic dosing targeting 2.0–3.0 times the activated partial thromboplastin time). Unfortunately, her general condition gradually worsened, and she died of heart failure and multiple organ failure on Day 7 of hospitalization after the percutaneous intervention. The family denied an autopsy request.

Discussion

Here, we present a rare case of LMCA–ACS caused by a large thrombus in the ascending aorta, as confirmed by IVUS.



Video 3 Intravascular ultrasound showing the thrombus extended from the ascending aorta to the proximal left anterior descending artery.

A floating thrombus in the aorta can be caused by many factors, such as age-related atherosclerosis, collagen disease, trauma, malignancy, and hypercoagulable conditions.^{3,7,10,11} These conditions constitute a very rare cause of embolic events in the coronary, cerebral, and peripheral arteries.¹¹ In the present case, concerning an elder woman, we considered that the large thrombus was caused by age-related atherosclerosis, although we could not further investigate as her family refused additional examination. The patient survived until the age of 90 years without any history of significant systemic illnesses that might have led to the development of a hypercoagulable condition. In addition, contrast-enhanced CT did not reveal a malignant tumour.

Most cases of LMCA-ACS involve complicated, life-threatening haemodynamic status and recanalization should be performed immediately to prevent cardiac death. IVUS is a useful imaging tool for diagnosing morphological abnormalities, such as intramural haematoma and dissection. Moreover, IVUS can immediately assist in the diagnosis of unstable haemodynamic conditions.

LMCA-ACS is sometimes caused by diseases that require surgical treatment. It is well known that acute aortic dissection (AAD) extending to the LMCA constitutes a rare case of LMCA-

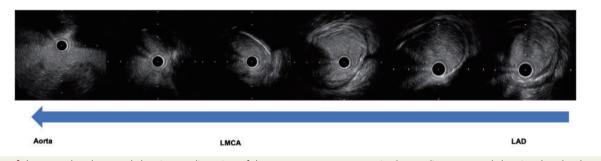


Figure 4 Intravascular ultrasound showing no dissection of the coronary artery or proximal ascending aorta, and showing that the thrombus extended from the ascending aorta to the proximal left anterior descending artery. LAD, left anterior descending coronary artery; LMCA, left main coronary artery.

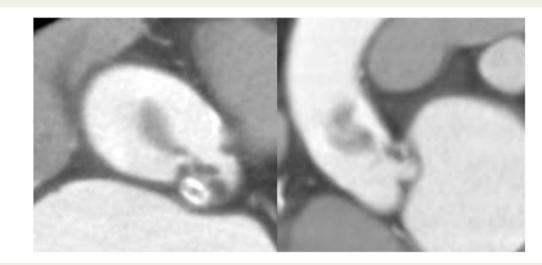


Figure 5 Contrast-enhanced computed tomography showing a large, abnormal, low-density area suspected to be a large thrombus in the left coronary cusp without an aneurysm in the ascending aorta without a dissection.

ACS.^{5,6,9} Previous reports demonstrated that IVUS was useful to confirm haematoma and false lumen between the LMCA and the aorta.^{5,6} In the present case, we first suspected dissection of the aorta in the first angiogram; however, an additional IVUS procedure did not detect haematoma or dissection of the LMCA into the aorta. Instead, a massive thrombus in the left coronary cusp and ascending aorta was found. Consequently, we were able to diagnose the cause of the LMCA–ACS.

In conclusion, we encountered a case of a large floating thrombus in the ascending aorta complicated by LMCA-ACS. IVUS assisted in the diagnosis of LMCA-ACS, showing that it can be a valuable tool, particularly in unstable haemodynamic conditions during an emergency.

Lead author biography



Hiroya Takafuji, MD, is a chief physician of cardiology at Tokyo Bay Urayasu Ichikawa Medical Center in Japan. His main interests are coronary intervention, peripheral intervention, and structural heart disease intervention (especially, congenital heart disease such as atrial septal defect and patent foramen ovale).

Supplementary material

Supplementary material is available at European Heart Journal - Case Reports online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: T.N. is a consultant for Boston Scientific, Medtronic, and Century Medical. All other authors declared no conflict of interest.

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