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CASE REPORT
Coronary heart disease

Coronary embolism due to possible thrombosis of prosthetic aortic valve - the role of optical coherence tomography: case report

Raminta Kavaliauskaite , Tatsuhiko Otsuka , Yasushi Ueki , and Lorenz Räber*

Department of Cardiology, Swiss Cardiovascular Center Bern, Bern University Hospital, Freiburgstrasse 18, 3010 Bern, Switzerland

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Background Coronary embolism is an important non-atherosclerotic cause of acute myocardial infarction (AMI) that requires an individualized diagnostic and therapeutic approach. Although certain angiographic criteria exist that render an embolic origin likely, uncertainty remains. Optical coherence tomography (OCT) is a high-resolution intracoronary imaging technology that enables visualization of thrombus and the underlying coronary vessel wall, which may be helpful to distinguish between an atherosclerotic and non-atherosclerotic origin of AMI.

Case summary A 50-year-old male was admitted with ongoing chest pain. Eleven years ago, he underwent implantation of a mechanical aortic valve prosthesis due to degenerated bicuspid valve with normal coronaries on preoperative angiography. The electrocardiogram showed anterior ST-segment elevation. Emergent angiography revealed total occlusion of the proximal left anterior descending artery (LAD). Thrombus was aspirated along with administration of intravenous glycoprotein IIbIIIa inhibitor. Except the apical part of the LAD showing distal embolization, coronary flow was completely re-established with no evidence of significant atherosclerosis. Stents were not implanted on the basis of the OCT finding, which demonstrated at the site of occlusion a normal vessel wall without atherosclerosis that could explain an erosion or plaque rupture event. Transoesophageal echocardiography confirmed a floating structure in the left ventricular outflow tract, suggesting that an embolus originating from the prosthetic aortic valve obstructed the LAD. The international normalized ratio 2 days prior to presentation measured 1.9.

Discussion This case illustrates the utility of OCT to rule out the atherosclerotic aetiology of myocardial infarction and to avoid unnecessary stenting.

Keywords Coronary embolism • Prosthetic valve thrombosis • ST-elevation myocardial infarction • Optical coherence tomography • Case report

Learning points

- Optical coherence tomography is an intravascular imaging modality based on infrared light emission that provides high-resolution arterial wall images.
- Optical coherence tomography is a valuable tool to confirm the presence of an acute coronary syndrome plaque event and to determine the underlying aetiology (plaque rupture, erosion, calcific nodule, spasm, embolic event, spontaneous coronary artery dissections).

* Corresponding author. Tel: +41 31 632 09 29, Fax: +41 31 632 11 31, Email: lorenz.raeber@insel.ch
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Introduction

The leading cause of acute coronary syndrome (ACS) is atherosclerotic plaque rupture and subsequent coronary thrombosis. Nevertheless, coronary embolism (CE) is an important non-atherosclerotic cause of acute myocardial infarction (AMI), identified as an ACS aetiology in 2.9% of diagnosed cases.[1] Since downstream treatment strategies differ significantly depending on the cause of AMI, it is important to differentiate the underlying aetiologies. Coronary angiography provides a two-dimensional vessel assessment i.e. 'lumenogram', that is unable to depict vessel wall pathology and thus evaluate vessel geometry and plaque characteristics.[2] Intracoronary imaging such as optical coherence tomography (OCT) overcomes these limitations and can be highly valuable to delineate differential diagnosis of ACS.[3] Our report shows the utility of OCT in diagnostic of an ST-elevation myocardial infarction (STEMI) resulting from CE possibly as a complication of prosthetic aortic valve thrombosis.

Timeline

Eleven years ago	Implantation of a mechanical aortic valve prosthesis due to degenerated bicuspid valve.
Day 1	ST-elevation myocardial infarction because of thrombotic occlusion of the left anterior descending artery (LAD). Thrombus was aspirated along with administration of glycoprotein IIbIIIa inhibitor. Optical coherence tomography revealed a normal vessel wall without relevant atherosclerotic plaque at the site of occlusion. Accordingly, no stent was implanted.
Day 2	Transoesophageal echocardiography shows a floating structure in the left ventricular outflow tract (LVOT), suggesting that an embolus originating from the prosthetic aortic valve occluded the LAD.
Day 25	Computed tomography of the thorax reveals neither a floating structure in the LVOT nor any other abnormalities.
Day 33	The floating structure in the transoesophageal echocardiography is no longer detectable.

Case presentation

A 50-year-old male was admitted to the hospital with ongoing chest pain. Eleven years ago, he underwent implantation of a mechanical aortic valve prosthesis due to degenerated bicuspid valve with normal coronaries on preoperative angiography. His medical history included treated hypercholesterolaemia and current smoking. There were no previous records on atrial fibrillation, left ventricular dysfunction, coagulation disorders, or recent cancer. At admission, the cardiopulmonary exam was normal, the blood pressure was 95/65 mmHg, heart rate 79 b.p.m., temperature 36.2°C, and oxygen saturation of 99% on room air.

The electrocardiogram showed sinus rhythm with anterior ST-segment elevation (*Figure 1*). Emergent angiography revealed total occlusion of the proximal left anterior descending artery (LAD, *Figure 2*, *Videos 1* and 2). Thrombus was aspirated; however, no histopathological analysis of the extracted material was undertaken. Due to high thrombus burden and absence of the blood clotting status at the time of angiography, bolus of intravenous glycoprotein IIbIIIa inhibitor was administered. Except the apical part of the LAD showing distal embolization, coronary flow was completely re-established with no evidence of stenosis or significant atherosclerosis (*Figure 3*, *Video 3*). As an additional finding, first diagonal branch showed a filling defect possibly resulting from embolization or minimal iatrogenic dissection. An OCT of the LAD was performed, which revealed at the site of occlusion a normal vessel wall without relevant atherosclerotic plaque that could explain an erosion or plaque rupture, the middle part of the vessel showed minimal intimal thickening s(Supplementary material online, *Video S1*). Thus, an embolic event was the only reasonable explanation for the coronary obstruction. Accordingly, no stent was implanted. Transoesophageal echocardiography was performed and confirmed a floating structure measuring 3.5 mm in the left ventricular outflow tract (LVOT, *Figure 4*, Supplementary material online, *Videos S2* and *S3*), suggesting that an embolus originating from the prosthetic aortic valve obstructed the LAD. Except the moderate dysfunction of the left ventricle [left ventricular ejection fraction (LVEF) 40%], resulting from apical septal hypokinesia, no other abnormalities were observed, especially no dysfunction of the prosthetic aortic valve. Left atrium appendix thrombi and persistent foramen ovale were ruled out by transoesophageal echocardiography including a bubble study with Valsalva manoeuver. Left ventricular thrombus was not shown in the transthoracic echocardiography (TTE); however, it cannot be definitely ruled out since no contrast ultrasound agent was used. Although, according to Duke Criteria the diagnosis of infective endocarditis (IE) was possible (one major and two minor criteria, respectively, positive echocardiographic diagnosis, predisposing cardiac condition, and vascular phenomena embolism), in the absence of fever and laboratory inflammation parameters, we considered IE as unlikely, therefore neither blood cultures were obtained nor antibiotics were given. Additionally, the further course of the patient including 3 months of follow-up revealed no events suggesting of an endocarditis.

The international normalized ratio (INR), gained after angiography, measured 4.3, however, 2 days prior to presentation, the INR was 1.9 and the patient administered 5 mg warfarin. Target INR was increased to 2.5–3.5. Due to previously subtherapeutic INR suggesting suboptimal anticoagulation control and possible minimal non-flow limiting dissection in the diagonal branch aspirin 100 mg was given additionally only for 1 month. A month later, the floating structure in the LVOT was no longer detectable in the transoesophageal echocardiography, prosthetic aortic valve functioned normally and left ventricular function normalized (LVEF 55%). Computed tomography thorax likewise showed neither evidence of thrombus in the LVOT nor any other intrathoracic pathology. The patient was advised to take part in the INR self-testing training.

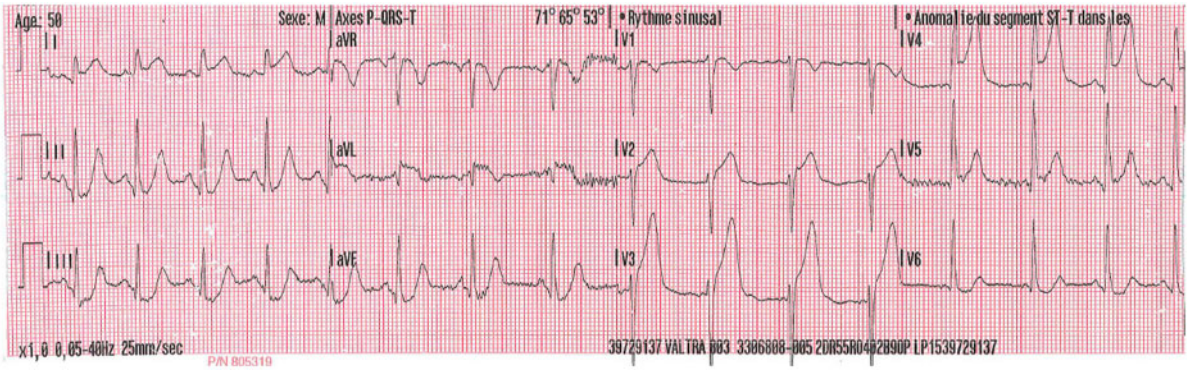


Figure 1 Electrocardiogram showing ST-elevation myocardial infarction.

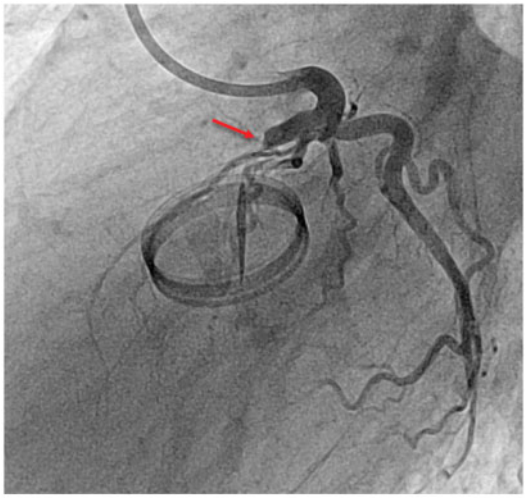


Figure 2 Coronary angiogram of the left coronary artery, red arrow pointing at the occluded left anterior descending artery, projection left antrior oblique (LAO) 87° caudal (CAU) 8°.

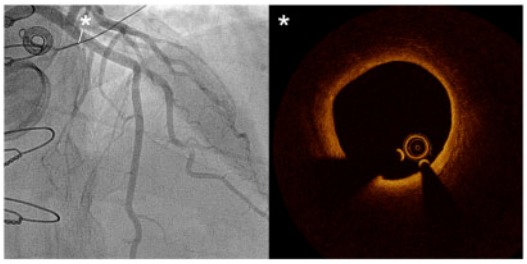


Figure 3 Coronary angiogram of the left coronary artery after thrombus aspiration showing no evidence of stenosis in the left anterior descending artery, projection right anterior oblique (RAO) 5° cranial (CRA) 37°. Right panel shows OCT still frame at the location of the coronary occlusion. The vessel wall showed no significant plaque and no signs of plaque rupture, erosion or hematoma.

Discussion

The causality of CE is multifactorial, recent retrospective study suggests that among STEMI patients the main cause of CE is atrial fibrillation—28.3%, embolus originating from valvular prosthesis is identified in 3.8%.[4] Due to its rarity, CE requires awareness and should be suspected in case of high thrombus burden without clear atherosclerotic evidence in the coronary artery or recurrent thrombus in patients with mechanical prosthesis.[5]

To date, there is no consensus on treatment of CE. The focus of management of patients presenting with acute chest pain and ST-elevation is immediate angiographic assessment.[6] Thrombus aspiration may be considered in cases of large residual thrombus after opening the vessel, while the routine thrombus aspiration is not recommended (Class III Level A).[6] Recent meta-analysis has demonstrated mortality benefits in patients with a high thrombus burden undergoing thrombectomy.[7] Coronary angiography providing a two-dimensional assessment has substantial limitations in estimating the vessel wall pathology.[8] Therefore, if diagnostic or angiographic uncertainty exists in the setting of ACS, intracoronary imaging is proposed to aid diagnosis and guide treatment.[3] Optical coherence tomography is an established intravascular imaging modality, based on infrared light emission that provides a rapid acquisition of high-resolution arterial wall images, in the range of 10–20 μm.[9] The superior resolution of OCT not only can delineate the hallmarks of the culprit lesion such as luminal discontinuity/plaque disruption and associated thrombus but also has a potential to detect a thromboembolic or vasospastic aetiology, if thrombus is observed in the absence of atherosclerosis or luminal irregularity.[3] Optical coherence tomography is further useful to detect non-atherosclerotic aetiologies of ACS (i.e. spontaneous coronary artery dissections/hematomas) or exclude the presence of an ACS plaque event e.g. in the context of myocardial infarction with non-obstructive coronary arteries, to identify vulnerable plaques, and to guide the percutaneous treatment of ACS.[2,3] Since acute and long-term patient management differs significantly depending on the cause of AMI, in patients, where CE is suspected,

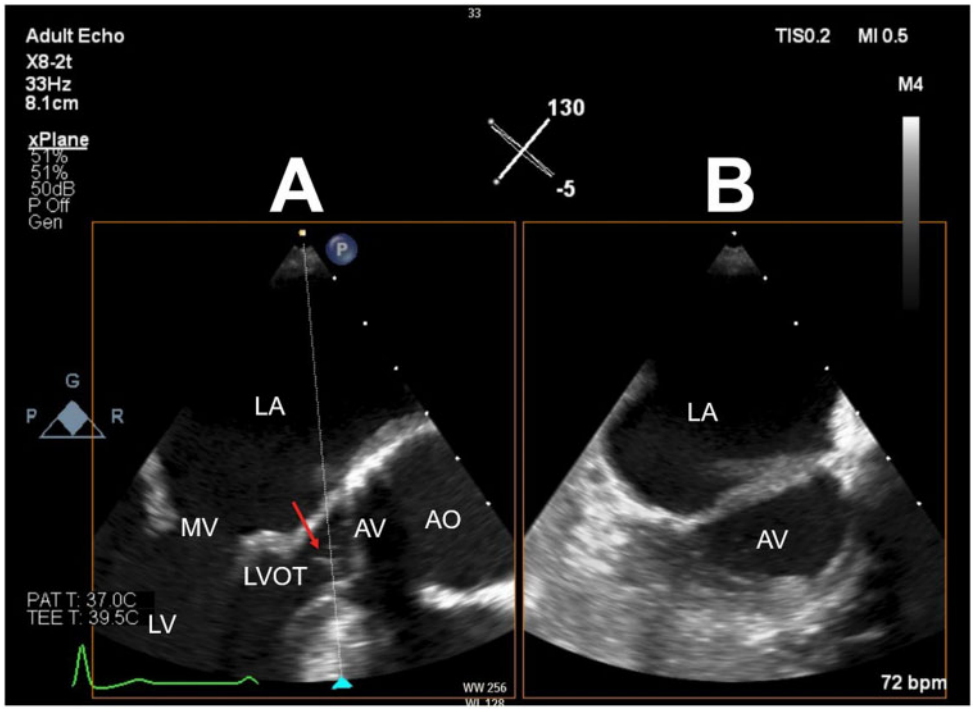
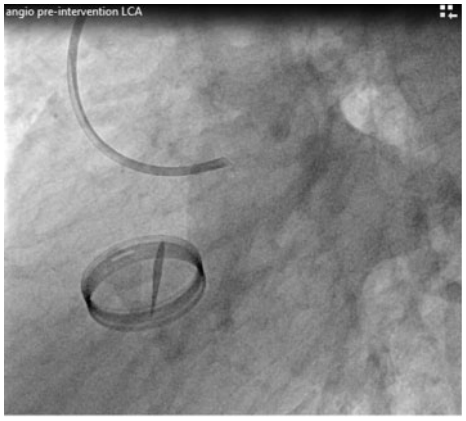


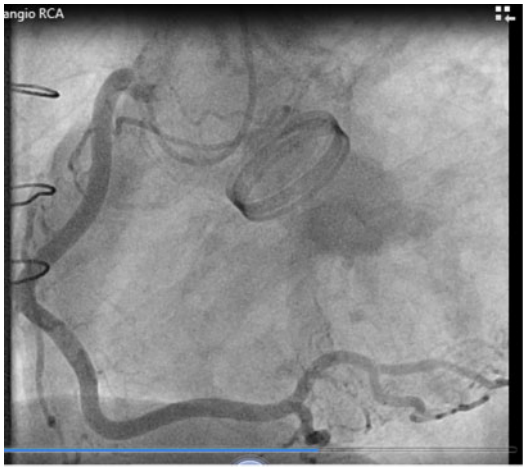
Figure 4 Transoesophageal echocardiography, *A*—longitudinal view, *B*—short-axis view. Red arrow shows floating structure in the left ventricular outflow tract. AO, aorta; AV, aortic valve; LA, left atrium ; LV, left ventricle; LVOT, left ventricular outflow tract; MV, mitral valve.

OCT may be helpful to differentiate between a plaque-induced event and the presence of embolus to determine if percutaneous coronary intervention or balloon angioplasty is required. Considering the risks of in-stent restenosis, thrombosis and the need for prolonged dual antiplatelet therapy that entails an increased risk of bleeding avoiding unnecessary stenting and an erroneous diagnosis may improve long-term outcomes of patients with CE. Our experience indicates that in patients where CE is suspected, after thrombus aspiration OCT can overcome some of the limitations posed by invasive coronary angiography and provide a valuable additional vessel assessment, therefore, it could be considered in the diagnostic algorithm of CE.

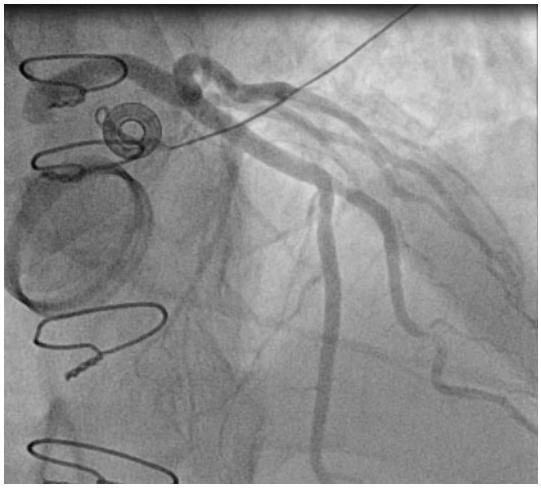
In conclusion, our case illustrates the utility of OCT to rule out the atherosclerotic aetiology of myocardial infarction and to avoid unnecessary stenting. Subsequently, no additional dual antiplatelet



Video 1 Coronary angiogram showing occlusion of the left anterior descending artery.



Video 2 Coronary angiogram of the right coronary artery.



Video 3 Coronary angiogram of the left coronary artery after thrombus aspiration.

therapy is needed resulting in lower risk of bleeding, especially by anticoagulated patients.

Limitations

It is debatable whether blood cultures should have been obtained in the presence of valve thrombosis and no fever or inflammatory markers. Since it was not done, we cannot definitely rule out the possibility of endocarditis as a source of emboli, although this is highly unlikely. Furthermore, left ventricular apex often cannot be clearly defined in the TTE, therefore to establish the presence or absence of left ventricular thrombus TTE with contrast ultrasound agent is recommended.[10] As contrast agent was not used, left ventricular thrombus was not definitely ruled out and therefore could have been an aetiology of CE. However, we believe that in the context of the patient history valve thrombosis is the most likely underlying cause of CE.

Lead author biography

Raminta Kavaliauskaite was born in 1992 in Lithuania. After finishing her medical training at the University of Vilnius in 2016, she worked at Departments of Cardiovascular Surgery and Internal Medicine. Currently, she is following her residency in cardiology at the University Hospital of Bern in Switzerland.

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: R.K. and T.O.: none declared, Y.U. reports personal fees from Infraredex, outside the submitted work. L.R. reports grants and personal fees from Abbott Vascular, Biotronik, Sanofi, and Regeneron; personal fees from Amgen, Astra Zeneca, Bayer, CSL Behring, and Occlutech; and grants from Heartflow, outside the submitted work.

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