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CASE REPORT
Coronary intervention

ST-segment elevation myocardial infarction without culprit lesion—a case report picturing the challenging interplay of epicardial atherosclerosis and coronary artery spasm

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Background Approximately 5–15% of patients with acute coronary syndrome have myocardial infarction with unobstructed coronary arteries (MINOCA). Guidelines recommend invasive assessments to identify underlying causes for MINOCA such as coronary artery spasm (CAS), spontaneous coronary dissection, or microvascular disease as well as non-invasive assessments in search of myocarditis, takotsubo syndrome, or cardiomyopathies.

Case summary A 54-year-old male patient presented with ST-segment elevation myocardial infarction (STEMI). Upon arrival, ST-segment elevation and symptoms had ceased. Emergency coronary angiography showed diffuse epicardial atherosclerosis with stenoses in the distal left anterior descending coronary artery (LAD) and second diagonal branch (D2); however, no epicardial occlusion was seen. Left ventriculography showed no clear wall motion abnormalities. Based on these findings, intracoronary acetylcholine (ACh) testing in search of CAS was performed. At 200 µg ACh intracoronary ST-segment elevation and chest pain recurred. Angiography showed occlusive epicardial spasm in the LAD and D2. Based on studies where the tendency of epicardial CAS was linked with the presence of epicardial atherosclerosis, the decision was made to perform PCI in the LAD and D2. ACh re-challenge after intracoronary nitroglycerine revealed only very mild symptoms, no demonstrable epicardial CAS, and no ST-segment elevation anymore. Cardiac enzymes reached their peak on day one [creatine kinase max 262 U/L (norm < 190 U/L), maximum of high-sensitivity troponin T 269 pg/mL (n < 14 pg/mL)].

Discussion There is a broad spectrum of patients with STEMI without culprit lesion regarding the extent of epicardial disease. In cases with an unclear culprit lesion, other causes for the acute presentation such as CAS should be investigated in an ad hoc fashion. The interplay of epicardial atherosclerosis and CAS should receive more attention in future trials.

Keywords Case report • MINOCA • Coronary angiography • Epicardial coronary artery spasm • Epicardial atherosclerosis • PCI

ESC curriculum 3.1 Coronary artery disease • 3.2 Acute coronary syndrome • 3.4 Coronary angiography

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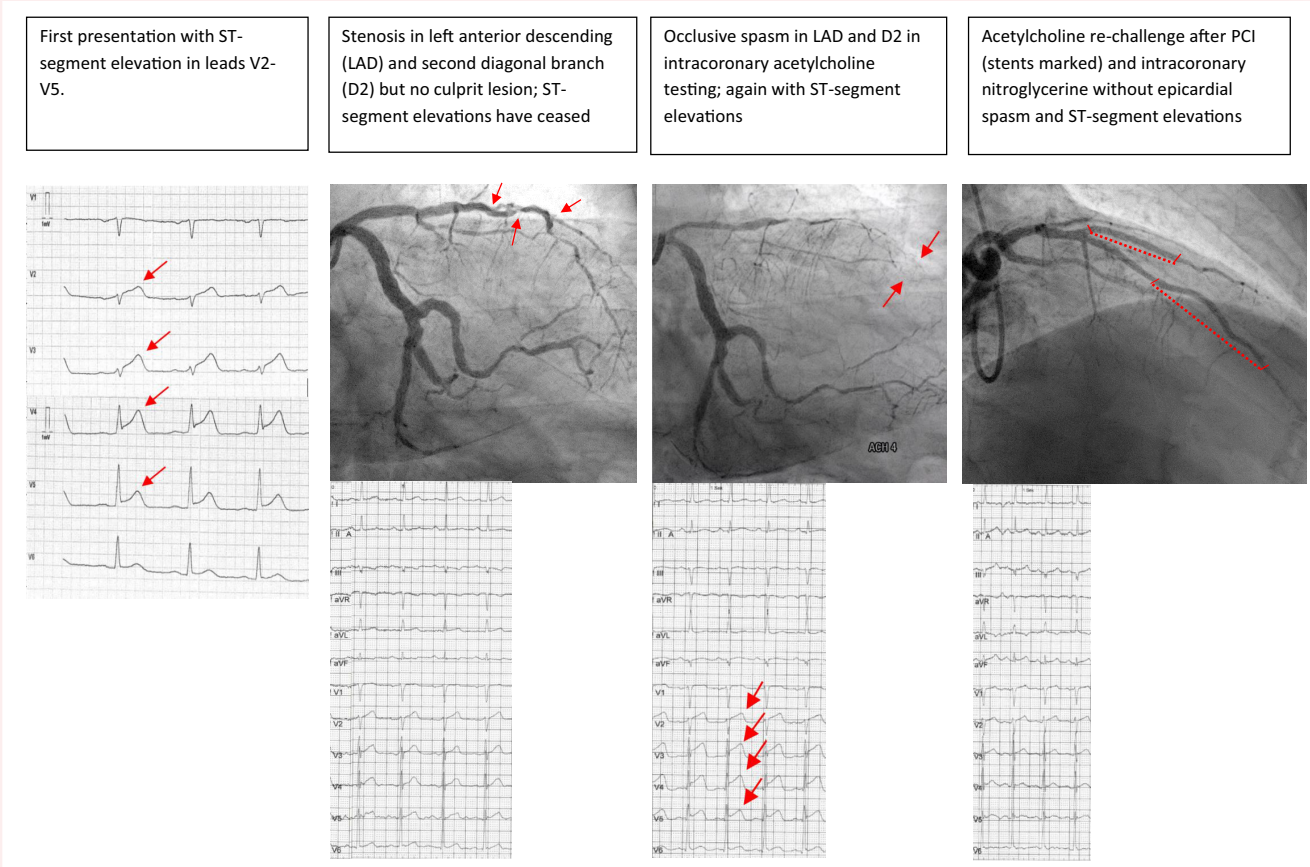
Learning points

- In patients presenting with ST-segment elevation myocardial infarction and unclear culprit lesion, other causes for acute presentation should be taken into account and acetylcholine (ACh) testing can be performed in the acute setting with an excellent safety profile.
- This case illustrates the interplay between epicardial atherosclerosis and coronary artery spasm.
- ACh re-challenge confirms the usefulness of nitrates in the prevention of epicardial spasm and can guide pharmacological management of these patients.

Introduction

In recent years, patients with acute coronary syndrome yet no culprit lesion have received more and more attention.[1] For such patients, the term myocardial infarction with unobstructed coronary arteries (MINOCA) has been introduced and criteria for the diagnosis and work-up of these patients have been proposed.[2] Depending on the clinical setting, ~5–15% of patients with acute coronary syndrome are affected by this condition.[3] The new 2023 European Society of Cardiology guidelines on the management of patients with acute coronary syndrome recommend invasive assessments to identify underlying causes for MINOCA such as spontaneous coronary dissection, coronary spasm, or microvascular disease as well as non-invasive assessments in search of myocarditis, takotsubo syndrome, or cardiomyopathies.[4] Despite these recommendations, real-world clinical cases can sometimes be challenging as shown in the present case.

Summary figure



Case presentation

A 54-year-old male patient was referred to our hospital by the emergency medical services with the diagnosis of ST-segment elevation myocardial infarction (STEMI). Sixty minutes before arrival at the hospital, the patient started having severe central chest pain for the first time. The patient was a heavy smoker with ~25 cigarettes per day and was under long-term treatment with candesartan and amlodipine for arterial hypertension. Immediate 12-lead electrocardiogram (ECG) showed

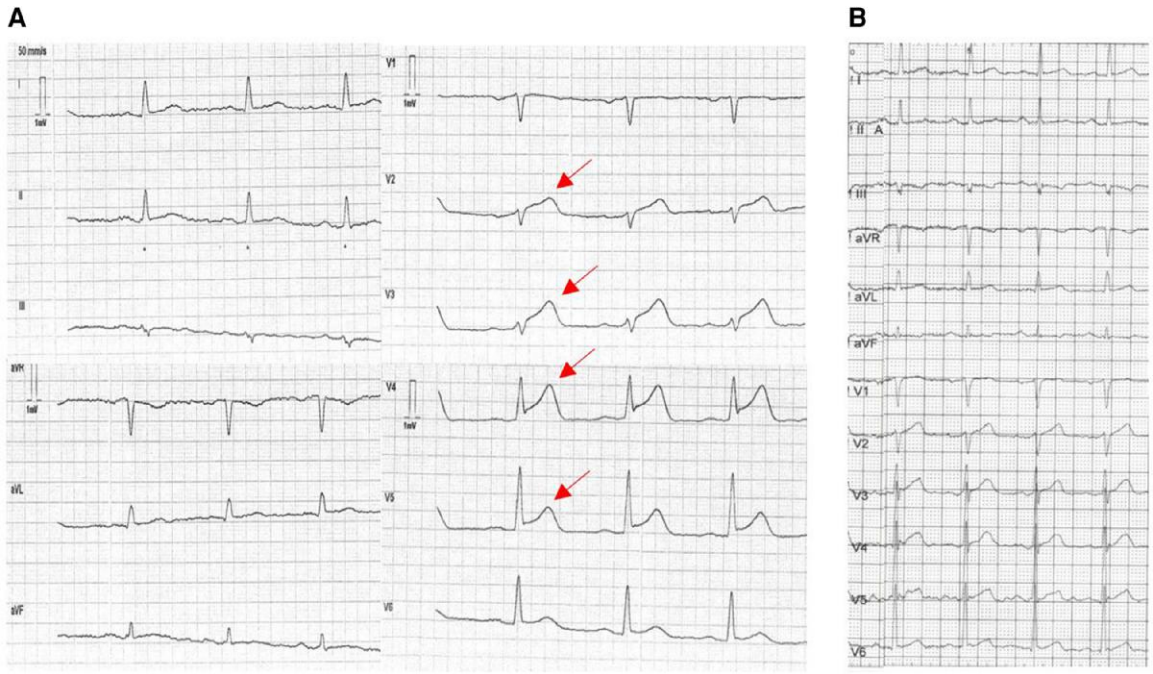


Figure 1 (*A*) Emergency 12-lead electrocardiogram with ST-segment elevation in leads V2–V5. (*B*) 12-Lead electrocardiogram upon arrival at the catheterization laboratory without ST-segment elevation.

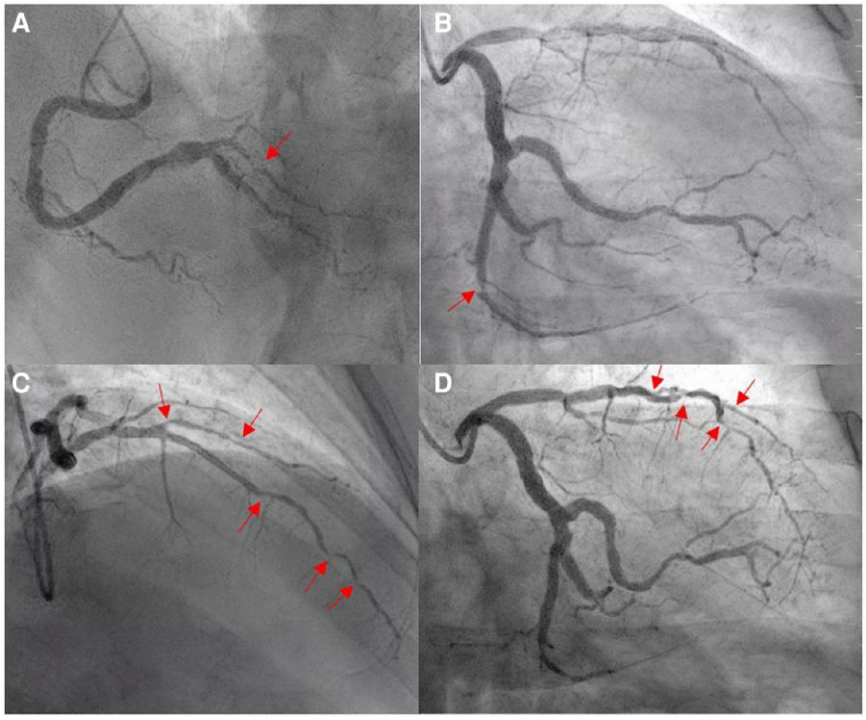


Figure 2 (*A*) Right coronary artery with stenosis, left anterior oblique cranial view. (*B*) Left coronary artery with stenosis in circumflex coronary artery, right anterior oblique caudal view. (*C*) Left coronary artery with stenosis in left anterior descending coronary artery and second diagonal branch, right anterior oblique cranial view. (*D*) Left coronary artery with stenosis in left anterior descending coronary artery and second diagonal branch, right anterior oblique caudal view.

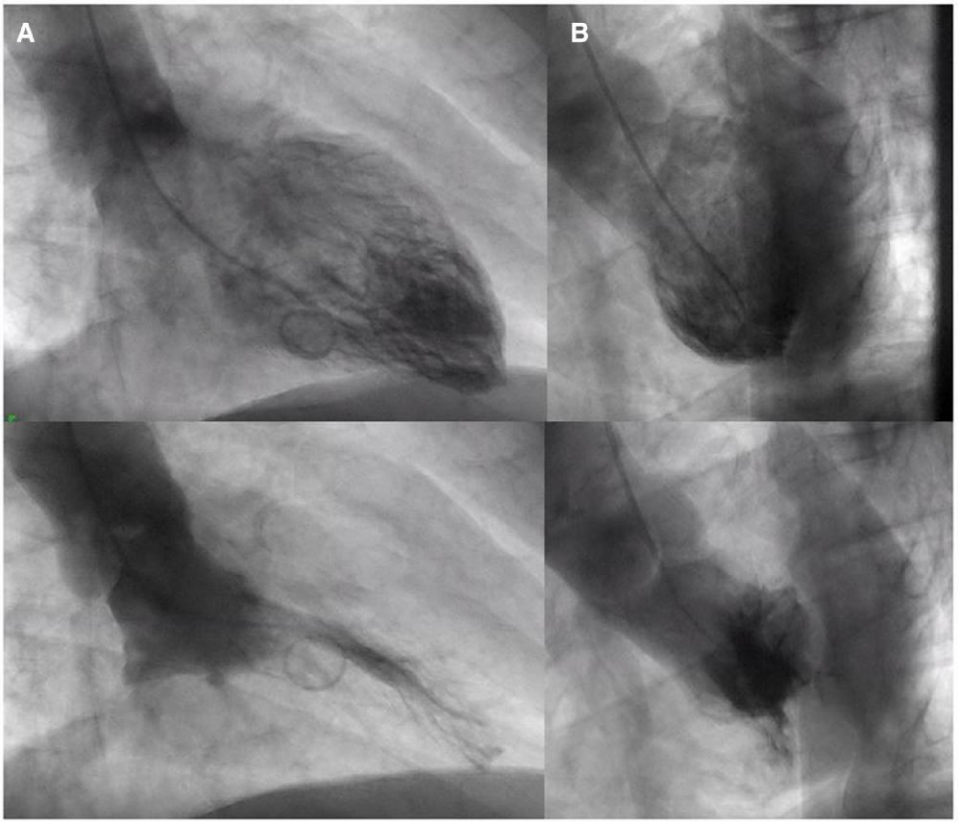


Figure 3 Left ventriculography without relevant regional wall motion abnormality and normal left ventricular function, (*A*) right anterior oblique and (*B*) left anterior oblique.

ST-segment elevation in leads V2–V5 (*Figure 1A*). The patient was treated with aspirin, heparin, ondansetron, and morphine. He was transferred directly to the catheterization laboratory bypassing the emergency department in stable conditions. Of note, upon arrival, the ST-segment elevation had ceased (*Figure 1B*) and the symptoms had disappeared. Emergency coronary angiography showed diffuse epicardial atherosclerosis with dilated segments as well as epicardial stenoses in the distal left anterior descending coronary artery (LAD), second diagonal branch (D2), distal right coronary artery, and distal circumflex coronary artery (*Figure 2*). However, no epicardial occlusion could be detected and there was thrombolysis in myocardial infarction III flow in all vessels. To better decide which myocardial territory was affected and which epicardial artery possibly needed PCI, a left ventriculography was performed. Surprisingly, it showed normal left ventricular function and only discrete hypokinesia of the left ventricular apex (*Figure 3*). Based on these findings, the clinical course, and the patient's history, intracoronary (ic) acetylcholine testing in search of coronary spasm was performed in an ad hoc fashion as reported previously.[5,6] At a dose of 200 µg ic acetylcholine, the patient developed the same severe chest pain as hours before with concomitant ST-segment elevation in the anterior leads. Angiographically, occlusive epicardial spasm in the LAD and D2 could be documented (*Figure 4A*). Intracoronary nitroglycerine led to reversion of the ECG, symptoms, and spasm (*Figure 4B*). Thus, a diagnosis of occlusive epicardial coronary spasm in the setting of STEMI with concomitant epicardial stenoses was made. Based on the ECG findings and studies where the tendency of epicardial coronary spasm was linked with the presence of epicardial atherosclerosis, the decision was made to perform PCI in the LAD and D2.[7] The D2 was treated with a 2.5 × 32 mm drug-eluting stent (DES), and the LAD was treated with two 2.5 × 24 mm DES (*Figure 4C*). Finally, after successful PCI an acetylcholine re-challenge after ic nitroglycerine was performed as reported previously.[8] The patient had only very mild symptoms, no demonstrable epicardial spasm, and no ST-segment elevation anymore (*Figure 4D*).

Discussion

This case nicely illustrates the challenges when performing emergency coronary angiography in patients with STEMI. The rate of STEMI without culprit lesion has been described to be ~6–10%.[3] However, the spectrum of epicardial disease can be variable ranging from 0% stenosis to 90% stenosis. Studies have shown that pressure wire measurements can be safely taken in the setting of intermediate stenoses.[9] The challenge in our case was that despite high-grade epicardial stenoses in the LAD and D2 none of these appeared visually with thrombus or plaque rupture. Moreover, normal left ventricular function without any clear wall motion abnormalities supported this notion. The decision to perform intracoronary acetylcholine testing was based on the clinical presentation (male smoker with sudden resting angina) and recent studies reporting an excellent safety profile of acetylcholine testing not only in the stable setting.[5,10] Interestingly, occlusive epicardial spasm could be documented in this patient, which is the likely reason for the acute clinical presentation in our opinion. However, based on studies on the interplay between epicardial atherosclerosis and coronary spasm (the concept of a spastic plaque) the decision was made

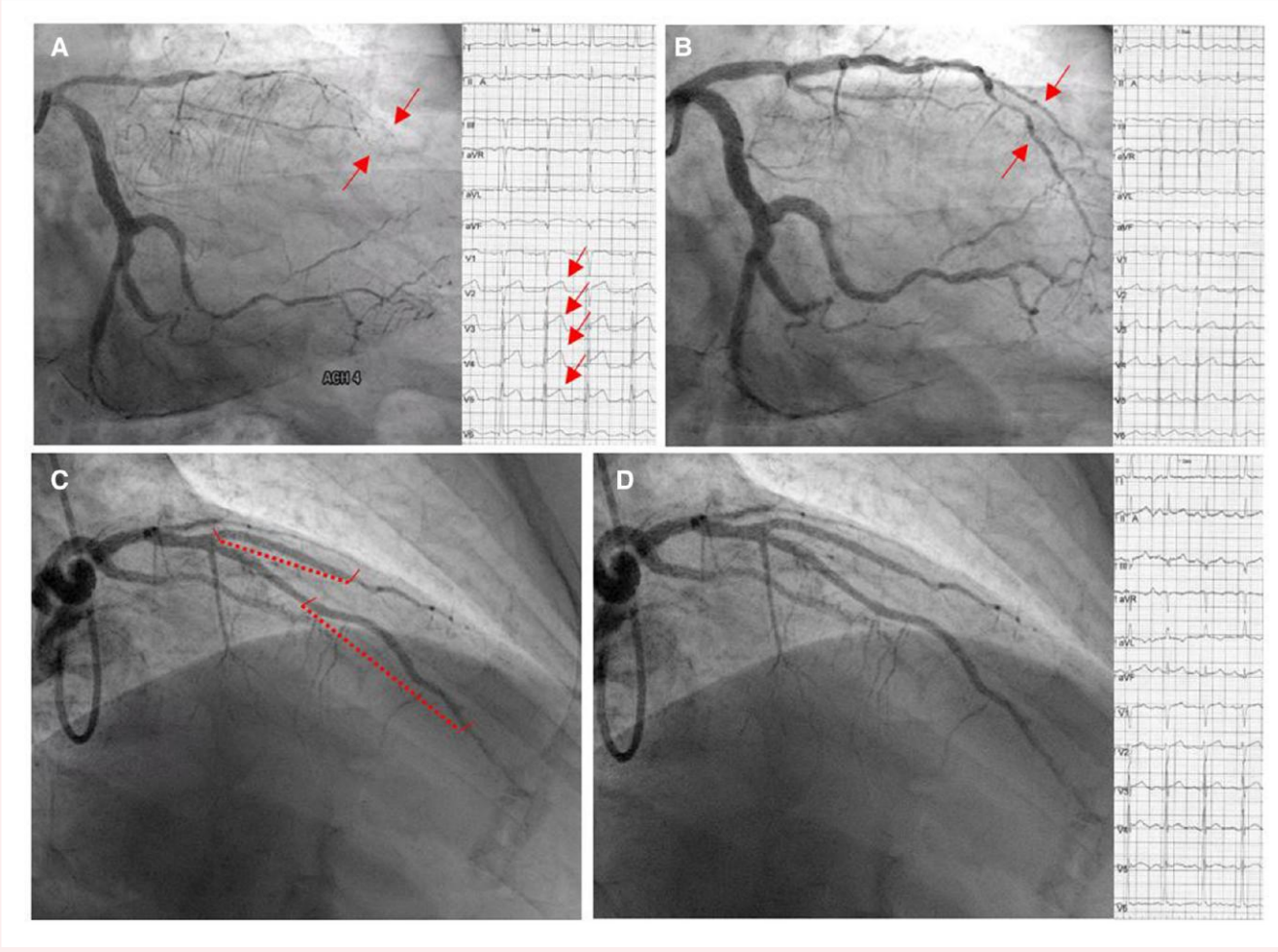


Figure 4 (*A*) Left coronary artery with occlusive spasm of left anterior descending coronary artery and second diagonal branch in intracoronary acetylcholine testing at a dose of 200 µg (right anterior oblique caudal view) and ST-segment elevation in the anterior leads. (*B*) Left coronary artery after intracoronary nitroglycerine with stenosis in left anterior descending coronary artery and second diagonal branch but with reversion of electrocardiogram and spasm (right anterior oblique caudal view). (*C*) Left coronary artery after percutaneous coronary intervention of left anterior descending coronary artery and second diagonal branch (stents marked; right anterior oblique cranial view). (*D*) Acetylcholine re-challenge after intracoronary nitroglycerine without epicardial spasm and ST-segment elevation (right anterior oblique cranial view).

to treat the epicardial stenoses in the LAD and D2.[7,11–13] Finally, ACh re-challenge confirmed the usefulness of nitrates but also the effectiveness of stenting in the prevention of epicardial spasm in this patient.[6] This also guided pharmacological management including platelet inhibition, cholesterol lowering, and antispastic drugs (diltiazem and oral nitrates). Of note, the patient reported intermittent chest pain at rest in the first days after the acute event. Up-titration of diltiazem to 120 mg twice daily and initiation of isosorbide dinitrate 20 mg twice daily led to a marked improvement in symptoms. Cardiac enzymes reached their peak on day 1 [creatine kinase max 262 U/L ($n < 190$ U/L), maximum of high-sensitivity troponin T 269 pg/mL ($n < 14$ pg/mL)], and the patient was discharged on day 4 in stable conditions. When considering differential diagnoses in this case myocarditis must be taken into account. However, as the present case had no history of infection and no demonstrable elevation of inflammatory markers in the laboratory analyses, cardiac MRI was not performed. In contrast, we believe that the heavy smoking was a major factor for the development of epicardial disease but also for coronary spasm.[14]

In summary, there is a broad spectrum of patients with STEMI regarding the extent of epicardial disease. In cases with an unclear culprit lesion, other causes for the acute presentation such as coronary spasm should be taken into account and investigated in an ad hoc fashion. This may help optimizing the management of such challenging cases. The interplay of epicardial atherosclerosis and coronary spasm should receive more attention in future trials.

Lead author biography

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Supplementary material

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

Consent: The patient consented to the use and publication of his data. All images and information have been anonymized and comply with COPE guidelines.

Conflict of interest: None declared.

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Data availability

The data underlying this article will be shared on reasonable request to the corresponding author.

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