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CASE REPORT
Other

ST-segment elevation myocardial infarction due to septic coronary embolism: a case report

Carlotta Mazzoni [1,2,3]*, Valentina Scheggi [3,4], Niccolò Marchionni [1,2,3] Pierluigi Stefano [2,3,5]

[1]Division of General Cardiology, Azienda Ospedaliero-Universitaria Careggi and University of Florence, Largo Brambilla 3, 50134, Florence, Italy; [2]Department of Experimental and Clinical Medicine, School of Human Health Sciences, Azienda Ospedaliero-Universitaria Careggi and University of Florence, Largo Brambilla 3, 50134, Florence, Italy; [3]Cardiothoracovascular Department, Azienda Ospedaliero-Universitaria Careggi and University of Florence, Largo Brambilla 3, 50134 Florence, Italy; [4]Division of Cardiovascular and Perioperative Medicine, Azienda Ospedaliero-Universitaria Careggi and University of Florence, Largo Brambilla 3, 50134, Florence, Italy; and [5]Division of Cardiac Surgery, Azienda Ospedaliero-Universitaria Careggi and University of Florence, Largo Brambilla 3, 50134, Florence, Italy

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Background Coronary artery embolism is an infrequent cause of type 2 myocardial infarction which can be due to arterial thromboembolism or septic embolism. While systemic embolization is one of the most acknowledged and threatened complications of infective endocarditis, coronary localization of the emboli causing acute myocardial infarction is exceedingly rare occurring in <1% of cases.

Case summary A 52-year-old man with a history of Bentall procedure and redo aortic valve replacement due to prosthetic degeneration (11 years prior to the current presentation) presented to the emergency department with high-grade fever and myalgias. Shortly after his arrival, he experienced typical chest pain and an electrocardiogram demonstrated signs of inferior ST-elevation myocardial infarction: coronary angiography showed a lesion of presumed embolic origin at the level of the mid-distal circumflex coronary artery which was treated with embolectomy. Transthoracic and transoesophageal echocardiography highlighted the presence of a periaortic abscess. The final diagnosis of infective endocarditis as the cause of septic coronary artery embolization was confirmed with a Positron Emission Tomography-Computed Tomography (PET-CT) exam and by the growth of *Staphylococcus lugdunensis* on repeated blood cultures. The patient underwent successful redo Bentall surgery the good outcome was confirmed at 1-month follow-up.

Discussion Type 2 myocardial infarction caused by coronary embolism is a rare presentation of infective endocarditis and requires a high level of suspicion for its diagnosis. Prosthetic heart valves are a predisposing factor for infective endocarditis: aortic root abscess requires surgery as it rarely regresses with antibiotic therapy.

Keywords Infective endocarditis • Periaortic abscess • Staphylococcus lugdunensis • ST-segment elevation myocardial infarction • coronary artery embolism • Case report

* Corresponding author. Tel +39 3407063422, Email: carlotta.mazzoni92@gmail.com; carlotta.mazzoni@unifi.it
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Learning points

- Septic coronary embolization secondary to infective endocarditis is a rare (<1%) but recognized cause of ST-segment elevation myocardial infarction.
- Diagnosis of septic coronary embolism in the setting of acute coronary artery disease requires a high level of suspicion for infective endocarditis (recent history of fever, previous prosthetic valve implantation).
- The diagnosis of infective endocarditis in patients with prosthetic valves is often challenging and requires multi-modality imaging, which can be time-consuming and lead to delayed surgery but could avoid overtreatment in patients who are by definition at high risk.
- Appropriate management of septic coronary embolism is not standardized, and evidence comes mainly from case reports. Long-term anti-thrombotic therapy after an acute coronary syndrome depends on the pathogenetic mechanism. Dual antiplatelet therapy is not indicated in patients with septic embolization in the absence of atherosclerotic coronary artery disease.

Introduction

Coronary artery embolism is one of the recognized causes of ST-segment elevation myocardial infarction (STEMI) in the absence of obstructive coronary artery disease. Its prevalence as a mechanism of STEMI ranges from 4% to 13% according to angiographic or autoptic studies.[1] Embolization is most often secondary to arterial thrombo-embolism, while coronary septic emboli are an extremely rare complication of infective endocarditis, occurring in <1% of patients.[1]

Timeline

2006	Bentall operation for ascending aortic aneurysm and a bicuspid aortic valve
2017	Redo surgery for bioprosthetic degeneration
12 September 2020 7:30 am	Emergency department access for fever
12 September 2020 10:50 am	Acute onset of chest pain with electrocardiogram evidence of STEMI. Primary Percutaneous Coronary Intervention (PCI) with embolectomy in the left circumflex artery
29 September 2020	Redo Bentall surgery for periaortic abscess
14 October 2020	Hospital discharge in good overall conditions
25 November 2020	Good result of the surgery at 1-month follow-up

Case presentation

A 52-year-old Caucasian man presented to the emergency department with the main complaint of high-grade fever (38.5°C) and myalgias for 1 week.

His medical history was significant for a Bentall operation for an ascending aortic aneurysm associated with a bicuspid aortic valve 14 years before the current presentation, and subsequent redo surgery with implantation of a biologic aortic valve prosthesis 11 years later, for prosthetic degeneration. The only medication he was taking was aspirin 100 mg daily for carotid artery disease.

A rapid nasopharyngeal swap for COVID-19, done as soon as the patient presented to the emergency department, excluded SARS-CoV-2 infection.

Physical examination was unremarkable except for a 2/6 systolic murmur at the upper sternal border.

Shortly after hospital arrival, the patient experienced sudden onset of typical chest pain: an electrocardiogram (ECG) showed junctional rhythm and an inferior STEMI (*Figure 1*). Bedside echocardiography revealed inferior and apical akinesia and a double lumen image in the aortic root, which initially raised the suspicion of aortic dissection, promptly excluded with an emergent aortic CT-angiogram.

The patient was then transferred to the cath-lab, where coronary angiography showed a suspected embolic occlusion of the mid-distal circumflex coronary artery, immediately re-opened with embolectomy using an Export aspiration catheter (Medtronic) (*Figure 2A and B*, *Videos 1* and 2). All other coronary branches were normal. Given the likelihood of a septic nature of the embolus according to the patient's clinical presentation, angioplasty, and stenting of the lesion were not performed. Dual antiplatelet therapy was not started for the same reason and the lack of atherosclerotic coronary artery disease. The embolic material was sent for cultural examination but did not grow any pathogen.

Repeated transthoracic echocardiography after the procedure confirmed the presence of a periaortic hypo-echogenic image suggestive of a periaortic abscess (*Figure 3A and B*, *Video 3*, Supplementary material online, *Videos S1–S3*). The aortic prosthesis was well-functioning and showed no signs of endocarditis as did the other native valves; a moderate degenerative mitral valve regurgitation was also noted; left ventricular systolic function had recovered, without residual wall motion abnormalities. A subsequent transoesophageal exam confirmed these findings. Post-procedural ECG showed normal sinus rhythm and T-wave inversion in the inferior leads.

Laboratory exams were significant for neutrophilic leucocytosis with a WBC count of 12 900/mm^3 and an elevated CRP at 108 mg/L (procalcitonin was negative), as well as a significant rise in high-sensitivity cardiac troponin T peaking at 2771 pg/mL; creatinine was 1.03 mg/dL (reference values 0.7–1.2 mg/dL), liver function tests showed mild elevation of the alanine aminotransferase (ALT) (0.88 mg/dL, reference values 10–50 mg/dL) with a normal AST (44 mg/dL, reference values 10–50 mg/dL).

Empiric antibiotic therapy with vancomycin (1 g every 12 h), gentamicin (80 mg every 8 h) and rifampin (600 mg daily) was immediately started.

Blood cultures eventually grew penicillin-sensitive *Staphylococcus lugdunensis* and antimicrobial therapy was switched from vancomycin to oxacillin (2 g every 6 h), continuing gentamicin and rifampin (at the increased dosage of 450 mg twice daily).

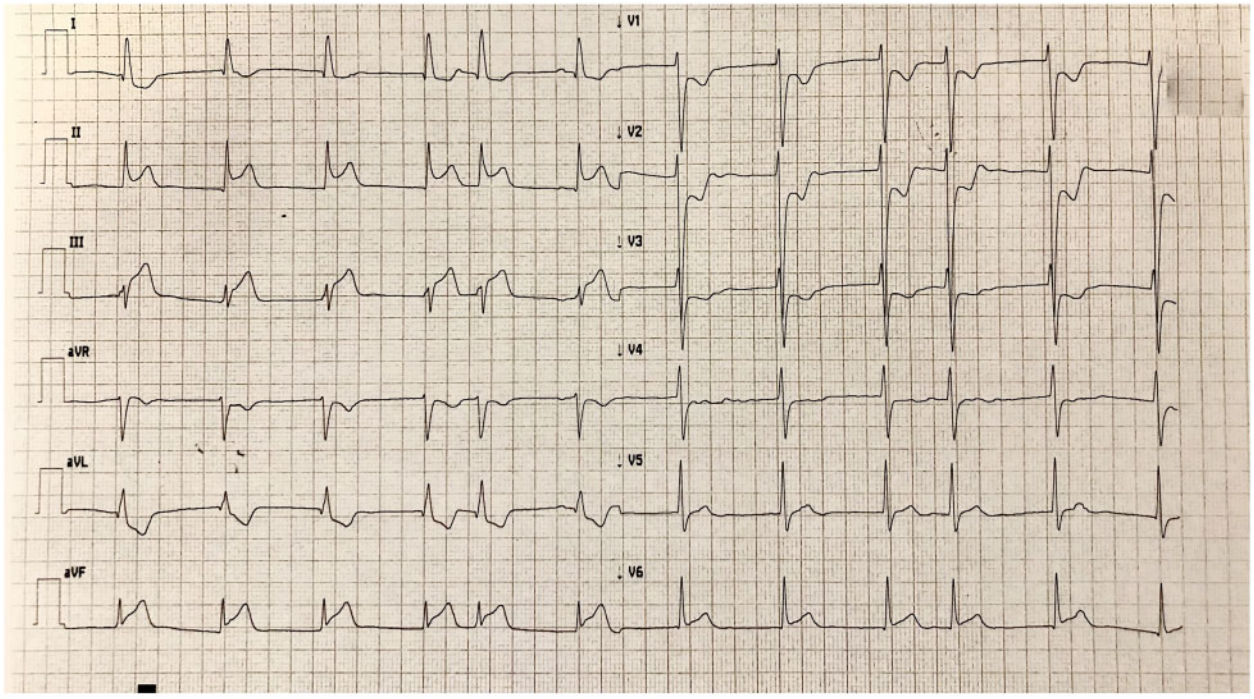


Figure 1 Electrocardiogram during chest pain at presentation in the emergency department showing junctional rhythm, ST-segment elevation in the inferior leads and ST-segment depression in leads D1-aVL and V1–V3.

After the introduction of antibiotic therapy, white blood cell (WBC) count normalized and C-reactive protein (CRP) progressively declined at 32 mg/L.

A PET-CT exam confirmed the presence of a hypermetabolic area around the aortic prosthesis compatible with a periaortic abscess (*Figure 4*).

The patient underwent redo Bentall procedure with the positioning of a mechanical prosthesis mounted on a Dacron vascular prosthesis and mitral valve repair 15 days after the initial presentation. The timing of surgery was influenced by the need for all the necessary preoperative imaging exams to achieve a definite diagnosis—considering also the patient's high surgical risk, being at his second redo operation (EuroSCORE II 13.21%). He recovered well from surgery and had no major complications apart from some initial episodes of junctional rhythm and atrial flutter. He was put on anticoagulant therapy with warfarin with the indication to re-evaluate the need for anticoagulation after 3 months, aspirin was stopped considering the patient had no atherosclerotic coronary artery disease; antibiotic therapy with gentamicin was stopped after a total of 3 weeks and the patient was discharged with the indication to continue oxacillin and rifampin for a total of at least 6 weeks of treatment. At discharge, the aortic prosthesis was well-functioning (*Figure 5*, Supplementary material online, *Videos S4* and *S5*), left ventricular systolic function was preserved and there was no residual mitral regurgitation.

At 1-month follow-up the patient was asymptomatic and in sinus rhythm, echocardiography confirmed the good result of the surgery.

Discussion

Infective endocarditis should be highly suspected in an STEMI patient who has a prosthetic heart valve and presents with fever in the absence of coronary atherosclerotic lesions on angiography. Septic coronary emboli are an infrequent, yet recognized cause of STEMI. Prosthetic valve endocarditis (PVE) represents one of the major downsides of valvular heart surgery and accounts for 20% of all infective endocarditis.[2]

Staphylococcus aureus is currently the most common cause of PVE.[3] Infections from *S. lugdunensis* affects mostly native valves and has an aggressive course; in PVE, it is usually localized in the aortic position and, when associated with abscess formation, carries a poor prognosis.[4]

Definite diagnosis of infective endocarditis requires the integration of microbiological, imaging, and laboratory findings, and is based on modified Duke criteria.

Transthoracic and transoesophageal echocardiography is the imaging modality of choice, which can detect vegetations and/or perivalvular extension in most cases. In PVE, CT-PET imaging can further guide towards a definite diagnosis, particularly in uncertain cases.[3,5]

In our patient, the diagnosis of endocarditis was rather straightforward, as he presented both the two major Duke criteria (aortic abscess on imaging and positive blood cultures for a typical pathogen) and also three minor criteria (predisposing factors, fever >38°C and embolic vascular phenomena). Perivalvular abscess formation is

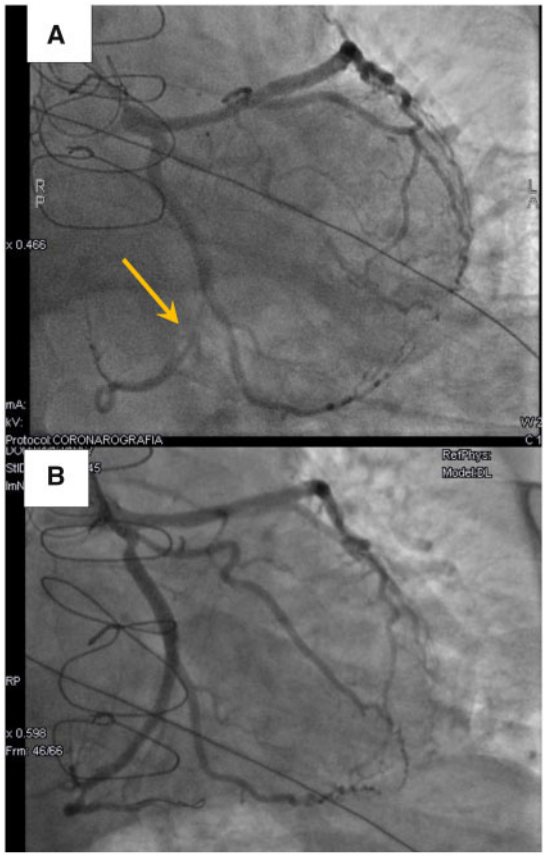
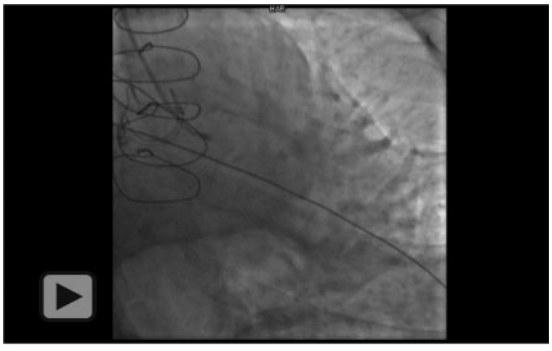
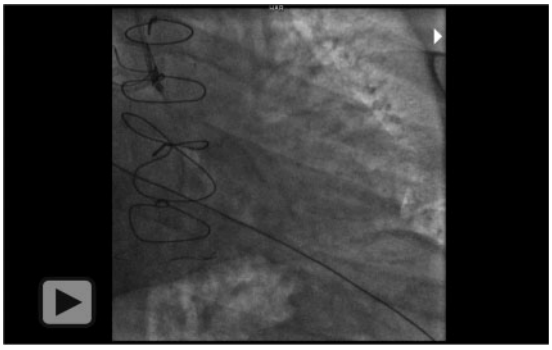


Figure 2 (*A*) Coronary angiography showing the lesion of suspected embolic origin at the level of the mid-distal circumflex coronary artery (arrow); (*B*) angiographic result after embolectomy.



Video 1 Coronary angiography showing an occlusion of suspected embolic origin in the mid-distal circumflex artery.

associated with a more aggressive disease course and worse outcomes; it is more commonly seen as a complication of PVE, especially aortic PVE, and it appears to occur more often in endocarditis caused by *S. aureus*.[6,7]



Video 2 The angiographic results after embolectomy with no evidence of atherosclerotic coronary artery disease.

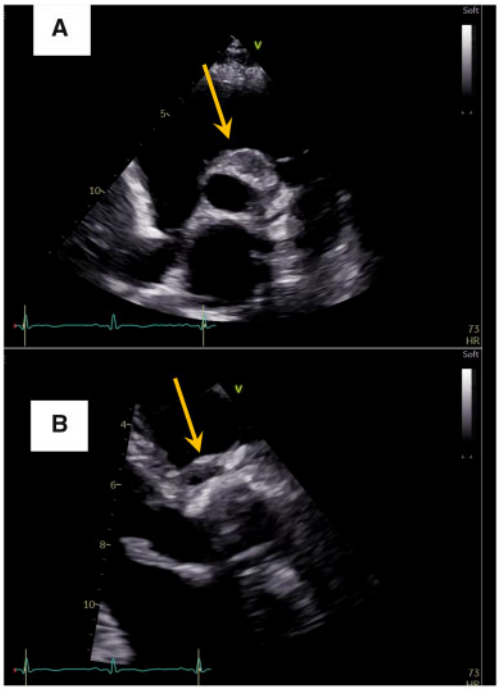


Figure 3 Echocardiographic view in the parasternal short (*A*) and long (*B*) axis of the periaortic abscess.

Despite the known association of perivalvular aortic abscess with complete heart block, our patient did not experience any significant rhythm abnormality apart from transient junctional rhythm in the context of an inferior STEMI.

Systemic embolization is one of the most dreaded complications of endocarditis, occurring in 20–50% of the patients. According to the literature, major risk factors for systemic embolization in endocarditis are *S. aureus* endocarditis, and large vegetations;[7,8] no study has yet investigated specific risk factors for coronary embolism.[1,3,5]

Coronary artery embolism is most often secondary to arterial thromboembolism, mainly in the setting of atrial fibrillation; septic

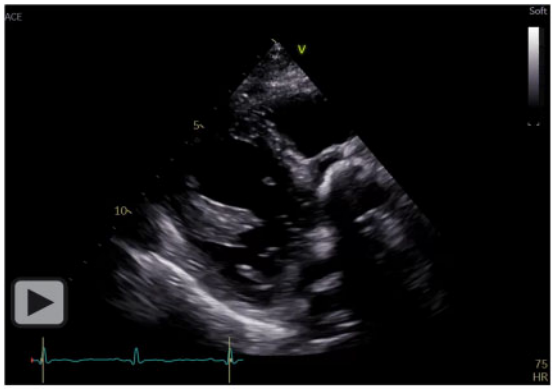
emboli are less frequent, complicating <1% of endocarditis cases.[9–11] Myocardial infarction as a complication of endocarditis can also occur due to compression of the coronary artery ostia by an aortic root abscess.[10]

Previous prosthetic valve implantation, a new heart murmur, fever on admission and leucocytosis are potential hallmarks of septic embolization as a cause of STEMI but are not sufficient to rule out 'a priori' atherosclerotic cardiovascular disease,[11] which requires a coronary angiography demonstrating normal coronary arteries, as well as an occlusion compatible with an embolic origin.

Embolic events are predominantly localized at the level of the left anterior descending coronary artery (LAD), due to preferential flow for anatomic reasons.[11,12] Hence, an inferior STEMI caused by embolization in the left circumflex artery, as in our case, is a rather atypical clinical scenario.

In a review by Nazir *et al.*[10] which included 100 patients with STEMI associated with endocarditis, embolic events occurred mainly in the left coronary artery (87% of cases: 75% involving the LAD, 10% the circumflex artery, 2% the left main), while the right coronary artery was involved in 12% of cases.

Aspiration embolectomy—the strategy used in our patient due to the high clinical suspicion for septic embolization—is advocated as a reasonable first-line choice in this context, considering the risks of angioplasty and stenting of an infected thrombus, such as mycotic aneurysm formation.[10–13] Since evidence of STEMI complicating endocarditis derives mainly from case reports though, uncertainties remain regarding the best treatment strategy. An early invasive catheter intervention appears to be a feasible option, as conservative management and thrombolysis are both associated with worse outcomes.[10–15] Given the distinct pathogenetic mechanism of this kind



Video 3 Transthoracic echocardiography clips showing an image suggestive of a periaortic abscess at the level of the aortic root in parasternal long axis.

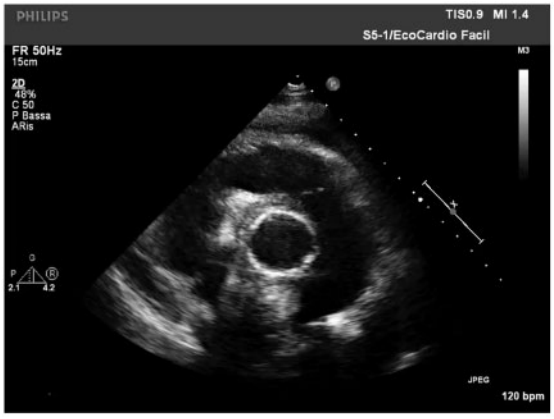


Figure 5 Echocardiographic parasternal short-axis view at the level of the aortic valve after aortic valve replacement showing the regression of the periaortic abscess.

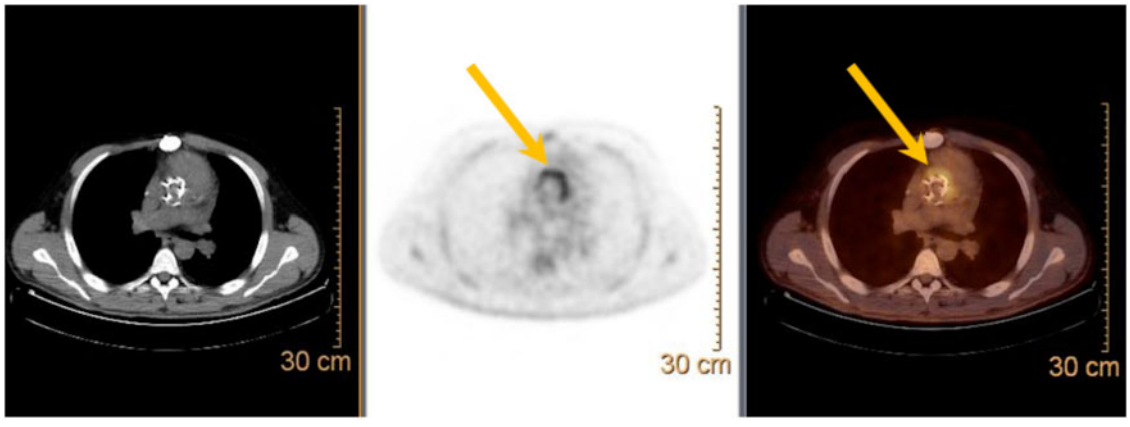


Figure 4 PET-CT scan image showing a hypermetabolic area (arrows) around the aortic prosthesis compatible with a periaortic abscess.

of myocardial infarction, in the absence of atherosclerotic coronary artery disease and plaque disruption, dual antiplatelet therapy is not recommended unless stenting of the lesion is performed.

Apart from the management of its possible complications, the mainstay of treatment for infective endocarditis remains appropriate antibiotic therapy and early surgery, which becomes mandatory in cases with locally uncontrolled infection, heart failure or lesion at high risk of embolization.[6,7]

The diagnosis of infective endocarditis in patients with prosthetic valves can be challenging and requires multi-modality imaging, which can lead to a delay in surgery but at the same time avoids overtreatment in patients who are by definition at high risk.

Perivalvular abscess is associated with a poor prognosis: it is a form of uncontrolled infection and therefore represents an indication for urgent surgery (within a few days) as antibiotic therapy can rarely eradicate the infection.[2–6]

Conclusions

Our case describes a strikingly uncommon complication of infective endocarditis, which can cause an STEMI as a result of septic coronary embolism. A high index of suspicion is necessary to achieve a prompt diagnosis and establish timely life-saving therapies.

Lead author biography

Dr Carlotta Mazzoni studied Medicine at the University of Bologna, from which she graduated in 2017. She is currently completing her residency in Cardiology at Careggi University Hospital in Florence, Italy.

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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: None declared.

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