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CASE SERIES
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Case report: acute myocarditis in two patients with coronary artery disease presenting with chest pain—thinking outside the box

Nicola Amelotti[1,2]*†, Matteo Brusamolino [1,2]*†, Massimo Mapelli [1,2], Mauro Contini [1], Andrea Baggiano[1,2], Fabio Fazzari[1], Gianluca Pontone[1,3], and Piergiuseppe Agostoni[1,2]

[1]Centro Cardiologico Monzino, IRCCS, Via Carlo Parea, 4, 20138 Milan, Italy; [2]Department of Clinical Sciences and Community Health, Cardiovascular Section, University of Milan, Milan, Italy; and [3]Department of Biomedical, Surgical and Dental Sciences, University of Milan, Milan, Italy

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Background In a subset of patients, acute myocarditis (AM) may mimic acute myocardial infarction, with a similar clinical presentation characterized by chest pain, electrocardiogram (ECG) changes consistent with acute coronary syndromes (ACS), and serum markers increment.

Case summary We present two cases of infarct-like myocarditis in patients with known coronary artery disease (CAD), in which the discrepancy between transthoracic echocardiogram findings, ECG, and angiography prompted us to look beyond the simplest diagnosis. In these cases, making a prompt and correct diagnosis is pivotal to address adequate therapy and establish a correct prognosis.

Discussion The right diagnosis can avoid unnecessary coronary revascularizations and subsequent antiplatelet therapy that may be associated with an increased haemorrhagic risk. Moreover, it allows setting up guideline-directed therapy for myocarditis, proper follow-up, as well as recommending abstention from physical activity.

Keywords Infarct-like myocarditis • Acute coronary syndrome • Cardiac magnetic resonance • Chest pain • Case report

ESC curriculum 2.3 Cardiac magnetic resonance • 3.1 Coronary artery disease • 2.2 Echocardiography • 6.5 Cardiomyopathy • 3.2 Acute coronary syndrome

Learning points

- The diagnosis of acute myocarditis (AM) poses a diagnostic challenge, especially in patients with a high pre-test probability of acute coronary syndromes (ACS), in which a myocarditis can be misdiagnosed as an ACS.
- The diagnosis of AM mimicking ACS is clinically relevant because it determines treatment and prognosis. A multidisciplinary approach and a high index of suspicion are needed.

* Corresponding author. Tel: 02580021, Email: matteo.brusamolino@unimi.it (M.B.), Tel: 02580021, Email: nicola.amelotti@unimi.it (N.A.)
† These authors contributed equally to this work and share first authorship.
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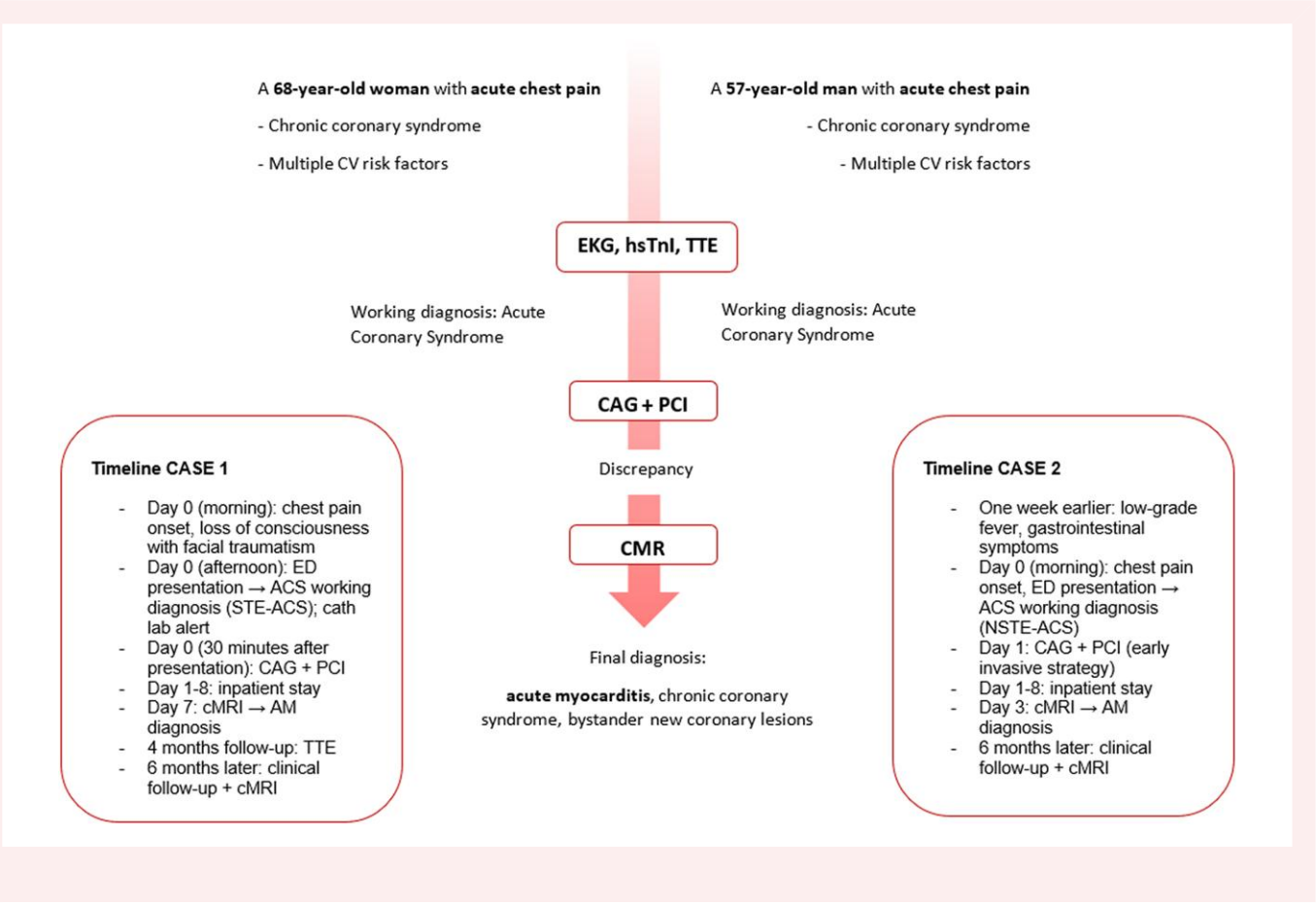
Introduction

Acute myocarditis (AM) is an inflammatory disease of the heart that can present with a wide range of symptoms.[1] In a subset of patients, AM may mimic acute myocardial infarction, with a similar clinical presentation characterized by chest pain, electrocardiographic changes consistent with acute coronary syndromes (ACS), and serum markers increment.[2] The diagnosis of AM mimicking ACS is clinically relevant because it determines treatment and prognosis. Myocarditis is a frequent final diagnosis in patients who receive an initial diagnosis of acute myocardial infarction with non-obstructive coronary arteries (MINOCA).[3] However, differential diagnosis is particularly challenging in patients with known coronary artery disease, in whom coronary stenosis can represent only a bystander of an underlying inflammatory process. We present two cases of infarct-like myocarditis in patients with known coronary artery disease, in which the discrepancy between transthoracic echocardiogram findings, electrocardiogram (ECG), and angiography prompted us to look beyond the simplest diagnosis.

Summary figure



Patient 1

A 68-year-old woman presented to the emergency department for retrosternal chest pain, not accentuated by movement or inspiration, lasting about 30 min, arose at rest, and associated with sweating and nausea. She reported, while in pain, a sudden loss of consciousness, unwitnessed, due to which she fell to the ground reporting facial trauma. She did not present any preceding viral-like symptoms. In the emergency department, she was still symptomatic. She had a history of arterial hypertension and dyslipidaemia. Six months before the presentation, she underwent percutaneous coronary intervention (PCI) of mid-distal left anterior descending coronary artery for exertional angina. Her medication included acetylsalicylic acid 100 mg od, clopidogrel 75 mg od, ramipril 5 mg bid, rosuvastatin 20 mg od, and lansoprazol 30 mg od. The patient denied taking any other medication or illicit drug.

Physical and neurological examination was unremarkable. The presenting ECG showed sinus rhythm and subtle ST-segment elevation at the J-point in the inferior leads (1 mm) and V7–9 (0.5 mm) and a reciprocal ST-segment depression in aVL (*Figure 1A and B*). Blood tests showed negative C-reactive protein (0.5 mg/L, normal value <5 mg/L), slight neutrophilic leucocytosis [white blood cell (WBC) count 10 700/uL, normal values 3900–10 500/uL; neutrophils 8650/uL, normal values 1800–7700/uL, 81%, normal values 37–73%; lymphocytes 1500/uL, normal values 1000–4800/uL, 14.2%, normal values 20–45%; monocytes 500/uL, normal values 0–800/uL, 4.2%, normal values 2.5–10%; eosinophils 0%, normal values <5%, and basophils 0%, normal values 0–2%; haemoglobin 13.3 g/dL, normal values 13.5–17.2 g/dL; platelet count 191 000/uL, normal values 140 000–450 000/uL], and an elevation of high-sensitivity troponin I (hsTnI) up to 5891 ng/L (normal values <53 ng/L). A transthoracic echocardiogram showed mid inferior, inferolateral, and inferoseptal hypokinesia, preserved ejection fraction, mild-to-moderate mitro-aortic regurgitation, and absence of pericardial effusion (Supplementary material online, *Video S1*). A computed tomography (CT) scan was performed, which excluded fractures or intracranial haemorrhage as result of the facial trauma. The coronary

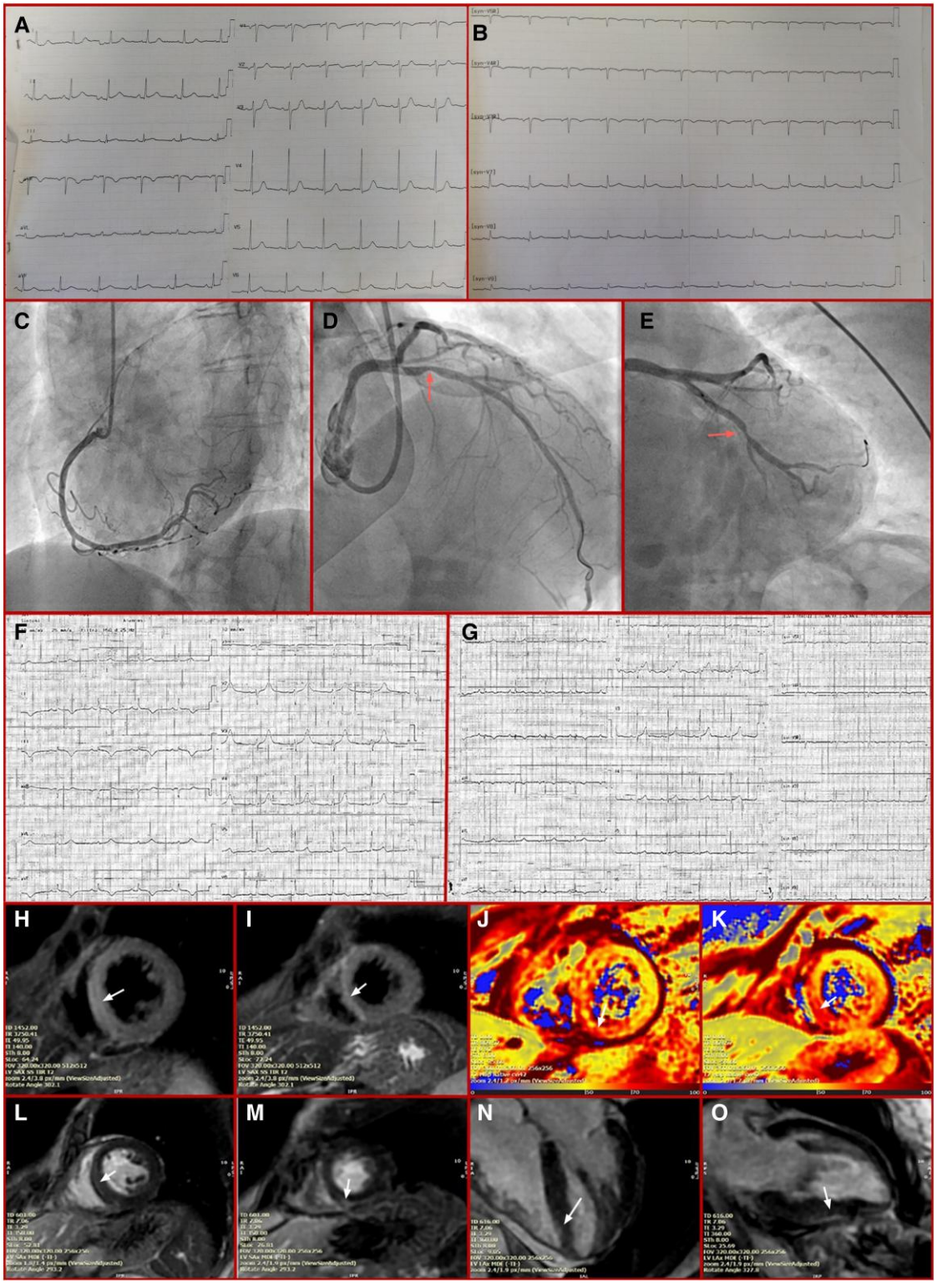


Figure 1 (*A* and *B*) Presenting ECG: sinus rhythm and ST-segment elevation in the inferolateral and posterior leads with specularity in aVL. (*C*–*E*) Invasive CAG showing a dominant RCA free from stenosis (*C*), significant stenosis of proximal LAD (*D*, arrow) and LCx (*E*, arrow). (*F* and *G*) Dynamic ECG changes showing negative T-waves in inferior leads, V6, and posterior leads (V7–9). (*H* and *I*) Mid (*H*) and apical (*I*) short-axis view T2-weighted images showing increased signal intensity at mid to apical septal and inferior walls (arrows). (*J* and *K*) Mid (*J*) and apical (*K*) short-axis view T2 mapping images showing increase in T2 values at mid to apical septal and inferior walls (arrows). (*L*–*O*) Mid (*L*) and apical (*M*) short-axis view, 4ch long-axis view (*N*), and 2ch long-axis view (*O*) showing mild enhancement and subepicardial enhancement (both of non-ischaemic pattern) at mid to apical septal and inferior walls (arrows). ECG, electrocardiogram; CAG, invasive coronary angiogram; RCA, right coronary artery; LAD, left anterior descending artery; LCx, left circumflex artery.

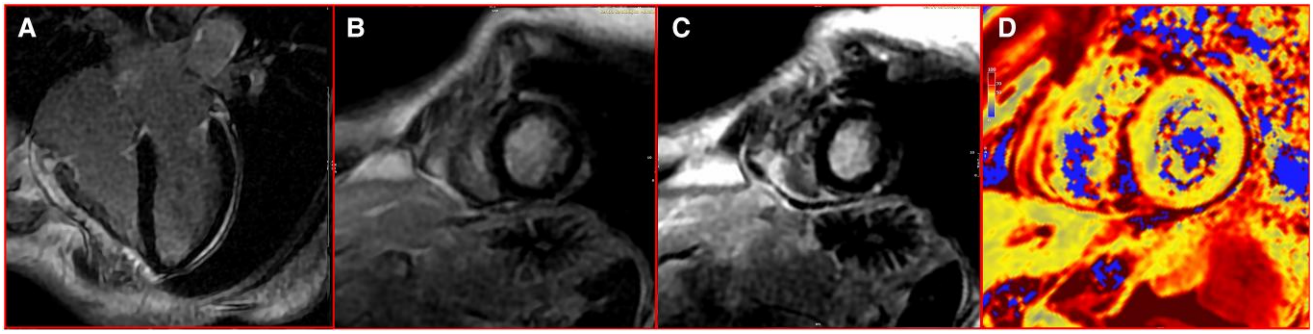


Figure 2 (*A*–*D*) Follow-up CMR. Apical four-chamber (*A*), mid to apical short-axis (*B*), and apical short-axis (*C*) showing the absence of LGE. Mid short-axis view T2 mapping image (*D*) showing normal T2 values. CMR, cardiac magnetic resonance; LGE, late gadolinium enhancement.

angiography showed a focal, eccentric, severe proximal left anterior descending coronary artery stenosis (80%); a focal, eccentric, severe mid left circumflex artery (LCx) stenosis (80%); and a dominant right coronary artery (RCA) free from stenosis (*Figure 1C–E*; Supplementary material online, *Figures S1* and *S2*). The patient was haemodynamically stable. Therefore, the patient underwent primary PCI of the hypothesized culprit (mid LCx) and subsequent complete revascularization during the index procedure (see Supplementary material online, *Figure S3*). In the following days, the ECG showed negative T-waves in inferolateral leads (*Figure 1F* and *G*). Repetitive ventricular ectopic beats were noted at continuous telemetry monitoring. Progressive reduction of troponin values was observed (peak value 5992 ng/L on Day 1). C-reactive protein remained persistently within normal values for the entire duration of hospitalization, and WBC count underwent rapid normalization (Day 2: WBC 8300/uL, N 560/uL, 67%). Given the discrepancy between ECG findings, wall motion abnormalities, and coronary angiography, we decided to perform a cardiac magnetic resonance (CMR). Indeed, the presence of a large dominant RCA free of stenosis, with a large posterolateral and posterior descending artery free of disease, was not entirely consistent with the segmental wall motion abnormalities found at transthoracic echocardiography. The CMR (performed on the 7th day of hospitalization) showed preserved biventricular size and ejection fraction with mid inferoseptal, inferior, and inferolateral hypokinesia, increase of myocardial signal intensity in T2-weighted images, increase of T2 values (69 ± 4 ms), and late gadolinium enhancement (LGE) with non-ischaemic subepicardial pattern at mid to apical septal and inferior walls (*Figure 1H–O*). These findings were in line with acute non-ischaemic myocardial damage (myocarditis). The patient was discharged on dual-antiplatelet therapy (cardioaspirin 100 mg od, ticagrelor 90 mg bid), atorvastatin 80 mg od, bisoprolol 1.25 bid, and ramipril 2.5 mg od. She was advised to avoid strenuous physical activity for 6 months. Four months later, the patient was asymptomatic. A transthoracic echocardiogram showed mid-basal inferior hypokinesia and normokinesia of the remaining segments. A 24 h ambulatory ECG monitoring showed no sustained arrhythmias. Blood exams, comprehensive of hsTnI and B-type natriuretic peptide, were within normal limits. A CMR performed 6 months later showed preserved biventricular size and ejection fraction with normal tissue characterization (*Figure 2*).

Patient 2

A 57-year-old man presented to the emergency department reporting retrosternal chest pain following a quarrel, described as a stab wound, lasted 10 min. The patient reported low-grade fever and gastrointestinal malaise, with a few episodes of poorly formed stools, the week before the admission to our hospital. He was known for multiple cardiovascular risk factors (former smoker, hypertension, dyslipidaemia, diabetes mellitus) and previous multiple non–ST-elevation (NSTE)-ACS (one 8 years before and one 1 year before, treated with percutaneous revascularization on the left circumflex coronary artery and posterolateral branch of the left circumflex, respectively). His medication included olmesartan/amlodipine 40/5 mg od, acetylsalicylic acid 100 mg od, rosuvastatin/ezetimibe 20/10 mg od, metformin 1000 mg od, and semaglutide 0.5 mg weekly subcutaneous injection. The patient denied taking any other medication or illicit drug. In the emergency department, physical examination was unremarkable, as were the presenting ECG, chest X-ray, and transthoracic echocardiography. Blood exams were within normal limits (haemoglobin 13.5 g/dL, normal values 13.5–17.2 g/dL; platelet count 208 000/uL, normal values 140 000–450 000/uL; WBC count 6700/uL, normal values 3900–10 500/uL; neutrophils 2360/uL, normal values 1800–7700/uL, 35.2%, normal values 37–73%; lymphocytes 2500/uL, normal values 1000–4800/uL, 37%, normal values 20–45%; monocytes 1700/uL, normal values 0–800/uL, 25%, normal values 2.5–10%; eosinophils 170/uL, normal values 0–450/uL, 2.5%, normal values <5%; basophils 0%, normal values 0–2%), except for elevated troponin (hsTnI up to 4659 ng/L, normal values <53 ng/L) and C-reactive protein (176 mg/L, normal values <5.0 mg/L). A coronary angiography was performed, showing a right dominant coronary circulation with a moderate-grade focal eccentric stenosis of the mid-circumflex artery. Therefore, intravascular imaging (optical coherence tomography) was performed, showing severe eccentric atheromatous disease, thin-cap fibroatheroma, critical [minimal lumen area (MLA) 4 mmq; planimetric stenosis of about 65%]. The stenosis was treated with drug eluted stent implantation (*Figure 3A* and *B*). The following days, we observed progressive electrocardiographic evolution in precordial anterolateral leads (V3–V6) and peripheral inferior leads with isodiphasic T-wave morphology (*Figure 3C* and *D*). Given the discrepancy between post-procedural electrocardiographic alterations and coronary alterations observed at angiography and considering the recent gastrointestinal syndrome, a CMR was requested and performed on the 4th day of hospitalization. It showed preserved biventricular size and function; mid to apical anteroseptal, anterior, and inferolateral increase in T1 and T2 values (T2 mapping value: 72 ± 6 ms, tissue equivalent of oedema); and apical anterior, lateral, and basal to mid inferolateral LGE with non-ischaemic pattern (intramyocardial and subepicardial). Those findings were in line with acute multifocal myocarditis (*Figure 3E–J*). Blood tests showed progressive normalization of inflammatory indices (C-reactive protein at discharge 2.7 mg/L) and myocardial necrosis biomarkers (hsTnI at discharge 25.52 ng/L). A transthoracic echocardiogram, performed before discharge, showed preserved biventricular size and function. The patient was discharged on double-antiplatelet

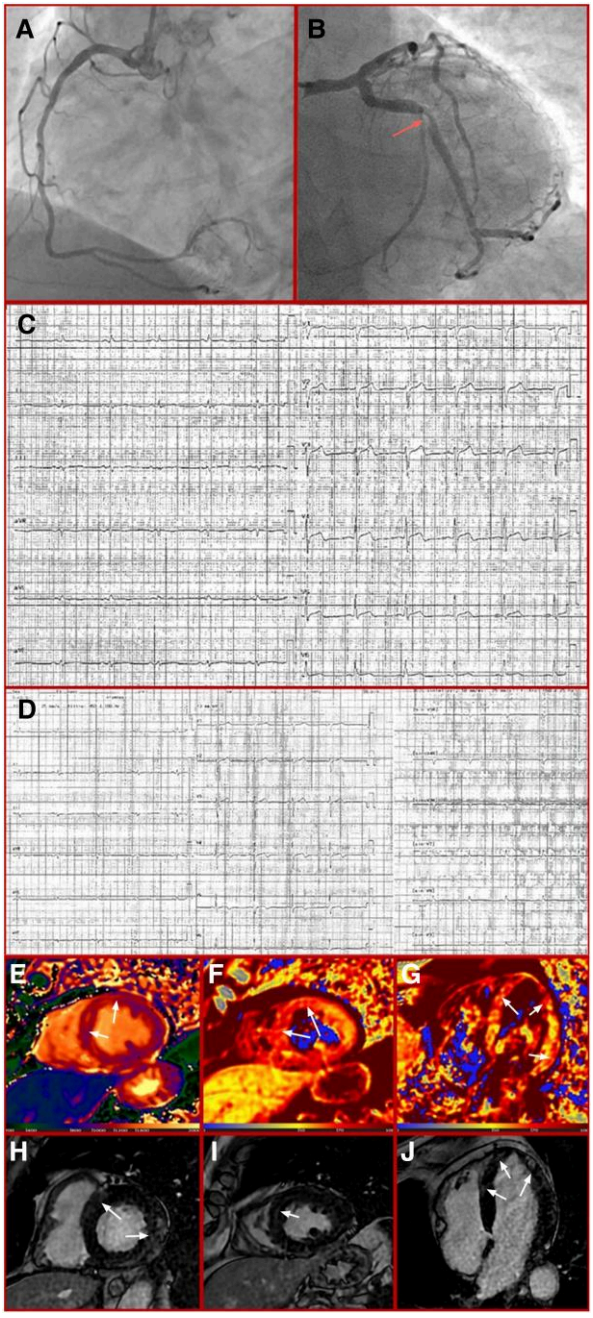


Figure 3 (*A* and *B*) Invasive CAG showing RCA free from relevant stenosis (*A*) and eccentric focal significant stenosis of mid LCx (*B*, arrow). (*C* and *D*) Post-procedural ECG showing inferolateral ischaemic evolution. (*E*) Mid to apical short-axis view T1 mapping image showing increase in native T1 values at septal and anterior levels (arrows). (*F* and *G*) Mid to apical short-axis view (*F*) and 4ch long-axis view (*G*) T2 mapping images showing increase in T2 values at mid to apical septal and anterior walls and basal lateral wall (arrows). (*H* and *J*) Basal short-axis view (*H*), mid to apical short-axis view (*I*), and 4ch long-axis view (*J*) LGE images showing subepicardial and intramyocardial enhancement (non-ischaemic pattern) at basal anteroseptum, basal inferolateral wall, and mid to apical septal and anterior walls (arrows). CAG, invasive coronary angiogram; RCA, right coronary artery; LCx, left circumflex artery; ECG, electrocardiogram.

therapy (cardioaspirin 100 mg od, ticagrelor 90 mg bid), rosuvastatin/ezetimibe 20/10 mg, and with cardioprotective therapy with bisoprolol 1.25 mg bid, ramipril 2.5 mg bid, and eplerenone 25 mg od. He was also advised to avoid strenuous physical activity for 6 months. The patient reported no significant symptoms during follow-up. A CMR performed 6 months later showed preserved biventricular size and function, normal T1 and T2 values, and basal inferolateral LGE with non-ischaemic pattern (*Figure 4*).

Discussion

Acute myocarditis is an inflammatory disease of the heart that may occur as a consequence of infections, exposure to toxic substances and drugs, and immune system activation[1] Some patients may present with chest pain, electrocardiographic changes, and elevated serum levels of cardiac biomarkers. Therefore, differential diagnosis among AM and ACS may be difficult. Current guidelines advocate coronary angiography in patients with suspected myocarditis to rule out ACS, and myocarditis is a frequent final diagnosis in MINOCA patients, defined as the absence of obstructive disease on angiography (i.e. no coronary artery stenosis ≥50%) in any major epicardial vessel.[4] Several authors described how myocarditis may mimic ACS.[5–7] However, patients discussed in these papers are typically young and had a low prevalence of coronary risk factors, and coronary angiography showed no obstructive coronary artery disease. There are, to the best of our knowledge, only a few reports of episodes of acute myocarditis in patients already known for coronary artery disease. In our patients, there was angiographic evidence of severe coronary artery stenosis, effectively excluding them from MINOCA classification; in these cases, it can be very difficult to recognize that a significant coronary artery stenosis represent only a bystander of an underlying inflammatory myocardial disease. In both our cases, the decision to treat the lesions in the cath lab was motivated by the belief that the patients had ACS.

In the first case, the patient underwent primary PCI of the hypothesized culprit infarct-related artery (mid-circumflex artery) and subsequent complete revascularization during the index procedure, due to the evidence of a high-degree left anterior descending artery stenosis with high probability of functional relevance (e.g. >70% stenosis subtended by a relatively large area of myocardium), low complexity lesion, and the low-to-moderate contrast volume load.[8] Indeed, randomized trials have demonstrated that PCI of non-infarct-related artery lesions for complete revascularization in patients with ST-elevation myocardial infarction STEMI improves clinical outcomes compared with infarct-related artery-only PCI and that fractional flow reserve-guided strategy did not have a significant benefit over an angiography-guided strategy in guiding complete revascularization.[9–11] It is well known that if a clear culprit is not detected, further invasive evaluation (using intravascular imaging) may be considered to identify the underlying cause.[12] However, our patient showed a clinical presentation consistent with ST-segment elevation-ACS and a significant coronary artery stenosis in a plausible infarct-related artery, and therefore, no further investigation was undertaken in the cath lab. In the second case, the clinical presentation was different and suggestive for NSTE-ACS. In this case, coronary intravascular imaging was performed. Even if no plaque rupture was detected on optical coherence tomography, the stenosis was judged to be amenable to PCI by the interventional cardiologist performing the procedure, given the very high-risk features of the plaque.

The decision to carry out further investigations and challenge the initial diagnosis of ACS is justified by the discrepancies between electrocardiography findings, echocardiography, and the supposed culprit lesion found at coronary angiography. Indeed, in the first case, electrocardiography was suggestive for an inferior STEMI; culprit coronary could have been either circumflex artery (absence of reciprocal ST depression in lead I) or RCA (ST-elevation in lead III > lead II and reciprocal aVL depression). At echocardiography inferior, inferolateral and

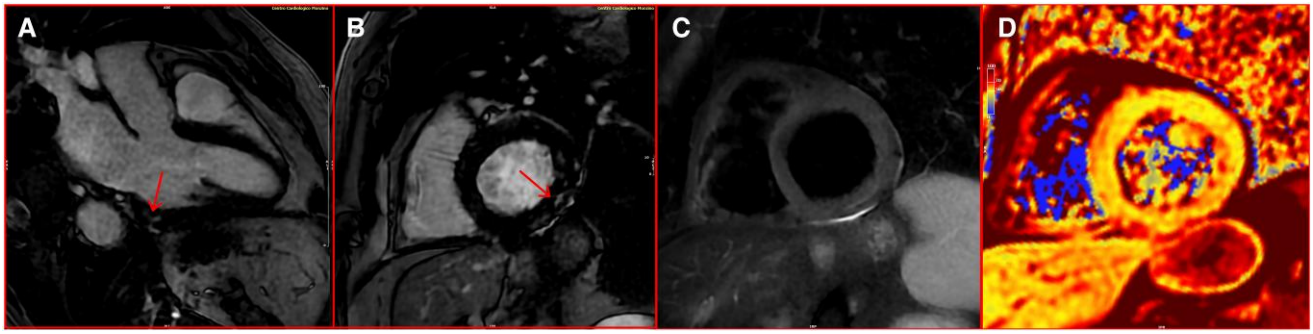


Figure 4 (*A*–*D*) Follow-up CMR. Apical three-chamber (*A*) and basal short-axis (*B*) showing subepicardial LGE (non-ischaemic pattern) at basal inferolateral segment (arrow). Basal (*C*) short-axis view T2-weighted image showing normal signal intensity at basal segments. Basal (*D*) short-axis view T2 mapping image showing normal T2 values at basal segments. CMR, cardiac magnetic resonance; LGE, late gadolinium enhancement.

inferoseptal hypokinesia was documented. However, in the cath lab, we found a multivessel disease with a large dominant RCA and its branches (posterolateral and posterior descending artery) free of stenosis (that collides with the inferior and inferoseptal hypokinesia) and a circumflex artery with severe stenosis but no evidence of obstruction. In the second case, the discrepancy was observed between dynamic electrocardiographic changes (anterolateral and inferior T-wave inversion) and coronary alterations observed at angiography (moderate mid-circumflex stenosis, absence of other epicardial stenosis in a RCA-dominant circulation).

In our cases, CMR supported the diagnosis of acute myocarditis in both patients, according to the Updated Lake Louise Criteria,[13] and ruled out ischaemic aetiology (non-ischaemic LGE pattern). Indeed, the CRM performed in the acute phase showed in both patents at least one T2-based criterion with at least one T1-based criterion. The diagnosis of certainty, as well as the aetiological diagnosis, is based only upon endomyocardial biopsy which, however, was not performed because of patients' low risk profile.[1] Polymerase chain reaction (PCR) testing and circulating antibodies of common cardiotropic viruses were not performed, due to their limited diagnostic usefulness.[4]

In retrospect, the history of recent gastrointestinal syndrome and the increase in C-reactive protein were further elements suggestive of AM in the second case. These features were absent in the first patient. However, up to 20% of acute myocarditis may have normal C-reactive protein values.[1] We did not detect, with hindsight, any other features in the first patient that could have heralded the diagnosis earlier, beyond the aforementioned discrepancies.

The cases presented represent a real-life situation that highlights the difficulty in clinical practice of differentiating ACS from myocarditis with bystander moderate coronary artery disease. Indeed, the diagnosis of AM remains a challenge because of the lack of easily accessible diagnostic methods that are both sensitive and specific.[4] Although the risk profiles and comorbidities of patients with AM and ACS differ substantially, significant overlap exists. Cardiac magnetic resonance is not routinely performed after ACS and may be not available in the acute setting of a suspicious ACS, even if some authors have highlighted a possible role for this test in the assessment of chest pain in the ED.[14] Moreover, the sensitivity of this test to detect oedema and vascular permeability decreases over time, reducing its usefulness for the differential diagnosis between ACS and AM if carried out late from the index event.[15] Endomyocardial biopsy remains the reference standard, but it is not routinely performed owing to its associated risks.[16]

Further studies are required to assess whether some of the cases of ACS in patients with high pre-test probability of CAD and evidence of ≥50% stenosis at coronary angiography constitute inflammatory processes. Indeed, we may suppose that in the absence of the discrepancies seen in our patients, a diagnosis of AM would have been missed. Making a correct differential diagnosis between the two diseases is therapeutically and prognostically relevant: it could have allowed avoidance of unnecessary PCI and subsequent antiplatelet therapy and associated increased haemorrhagic risk.

Eventually, as AM may lead to dilated cardiomyopathy in up to 20% of cases,[17] patients should be offered long-term non-invasive cardiological follow-up. Uptitration of anti-remodelling drugs to maximal tolerated doses should be pursued, and serial echocardiograms and CMR should be performed over time. In the event of prolonged documented increase of cardiac enzymes or progressive reduction in ventricular function, an endomyocardial biopsy should be considered.[4] In conclusion, the diagnosis of AM can be extremely challenging in these patients, and a high index of suspicion is needed to go beyond the simplest diagnosis.

Lead author biography

Nicola Amelotti is a cardiology resident at the University of Milan and currently working at Centro Cardiologico Monzino. He has a particular interest in cardiac imaging.

Supplementary material

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

Consent: The authors confirm that consent for submission and publication of this case report has been obtained from the patient in line with the Committee on Publication Ethics (COPE) guidance.

Conflict of interest: None declared.

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Data availability

The data underlying this article are available in the article and in its supplementary material online.

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