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CASE REPORT
Cardiovascular imaging

Calcified left ventricular apical aneurysm with intramural thrombus: a case report

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Background Left ventricular aneurysms (LVAs) are a well-appreciated complication of acute myocardial infarction. Ventricular aneurysms involving the left ventricle (LV) typically evolve as a result of anterior myocardial infarction and are associated with greater morbidity, complication rates, and hospital resource utilization. Incidence of LVA is decreasing with advent of modern reperfusion therapies; however, in the setting of excess morbidity, clinicians must maintain an appreciation for their appearance to allow timely diagnosis and individualized care.

Case summary This case report describes the clinical history, investigation, appearance, and management of a patient with calcified apical LVA with history of previous anterior ST-elevation myocardial infarction. The patient was initially admitted for elective coronary angiography in the setting of worsening exertional dyspnoea and subsequently underwent coronary artery bypass graft, aneurysm resection, and LV reconstruction.

Discussion Left ventricular aneurysms are an uncommon complication experienced in the modern era of acute myocardial infarction and current reperfusion therapies, but remain an important cause of excess morbidity and complication. Evidence-based guidelines for the diagnosis, workup, and subsequent management of LVAs are lacking. The imaging findings presented in this case serve as an important reminder of the appearance of LVAs so that timely diagnosis and individualized care considerations can be made.

Keywords Coronary angiogram • Echocardiography • Aneurysm • Case report

ESC curriculum 2.1 Imaging modalities • 2.2 Echocardiography • 3.1 Coronary artery disease • 3.4 Coronary angiography • 6.2 Heart failure with reduced ejection fraction

Learning points

- Understand calcified left ventricular aneurysm as an infrequent complication of acute myocardial infarction that may be complicated by associated thrombus.
- Observe the appearance of calcified left ventricular aneurysm on plain chest X-ray, transthoracic echocardiography, and invasive coronary angiography.

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Introduction

Left ventricular aneurysms (LVAs) are an appreciated complication of acute myocardial infarction (AMI).[1] Left ventricular aneurysms typically involve the left ventricular (LV) anterior and/or apical walls, with left anterior descending artery (LAD) occlusion and/or anterior ST-elevation myocardial infarction (STEMI) as independent determinants of aneurysm formation.[2] Left ventricular aneurysm incidence has decreased with modern reperfusion technologies, but remains associated with greater morbidity, complication rates, and hospital resource utilization.[1] Current European Society of Cardiology STEMI guidelines suggest incidence of LVAs to be <5%.[3]

This case study details the diagnosis, investigation, and management of calcified apical LVA with associated thrombus in a patient with exertional dyspnoea and ischaemic heart disease.

Summary figure

Timeline of salient events

~15 years prior to angiogram	Patient admitted to the hospital with anterior STEMI. Underwent percutaneous coronary intervention to the LAD with 1× bare metal stent.
2–3 months prior to angiogram	Patient reports subjective worsening in symptoms of exertional dyspnoea. Stress echocardiogram is negative; however, patient is unable to achieve sufficient exercise level. Referred for elective invasive coronary angiogram.
Day 0	Admission for elective coronary angiogram. Coronary angiogram demonstrating calcified LVA. Transthoracic echocardiogram (TTE) suggestive of aneurysm with intramural thrombus on contrast study. Commenced on anticoagulation and medication for heart failure with reduced ejection fraction (EF).
Day 2	Discharged home for outpatient surgical intervention.
Day 16	Admission for elective coronary artery bypass graft (CABG) + LV aneurysmectomy + ventricular repair. Uncomplicated procedure. Transferred to intensive care unit (ICU) post-procedure.
Day 19	Discharged from ICU to general cardiothoracic surgery ward.
Day 24	Discharged from coronary care unit to care of cardiologist in the community. Remained well in the community and compliant with anticoagulant/antiplatelet agents + guideline-based medical therapy for heart failure.

Case presentation

A 71-year-old male presented for elective coronary angiography for worsening exertional dyspnoea in the context of anterior STEMI with percutaneous intervention to LAD 15 years prior. Stress TTE was equivocal for ischaemia prompting referral for angiogram. Further echocardiographic findings were not provided. Cardiovascular risk factors included hypercholesterolaemia, hypertension, being an ex-smoker, and having a first-degree relative with premature coronary artery disease (CAD). Medications included aspirin, clopidogrel, isosorbide mononitrate, atenolol, candesartan, hydrochlorothiazide, and pravastatin.

The patient reported a 2–3 month decline in exercise tolerance without angina. Physical examination revealed bipedal oedema. Metabolic profile was unremarkable. Electrocardiogram (ECG) demonstrated sinus bradycardia, with narrow QRS complexes, extensive Q-waves, ST-elevation in leads V_2 andV_3, and T-wave inversion in V_3–V_6 (*Figure 1*).

Invasive coronary angiography demonstrated mild distal stenosis of the left main coronary artery, with proximally occluded LAD in keeping with prior STEMI (see Supplementary material online, *Videos S1* and *S2*). The circumflex artery was mildly diseased, while the right coronary artery was ectatic, heavily calcified with severe proximal and mid-vessel disease, and supplied collaterals to the left system. Marked apical calcification concerning for calcified aneurysm was noted (*Figure 2*). Left ventriculography was not performed given possibility of LV thrombus.

Transthoracic echocardiogram (see Supplementary material online, *Videos S3–S5*) demonstrated a mildly dilated and thickened LV, with mild-to-moderate segmental systolic dysfunction and LVEF of 40 ± 5%. Apical segments were thinned and aneurysmal, while mid-anterior and mid-septal segments were thinned and akinetic. Grade II diastolic dysfunction and elevated filling pressure was noted. Contrast study identified a filling defect near the apico-inferior/infero-septal segments favoured to be thrombus (*Figure 3*). The left atrium was moderately dilated, while the right atrium and ventricle were of normal size and function. There was no significant valvular disease.

The patient was discussed at the institutions' heart team conference, involving cardiology, cardiac surgery, and cardiac anaesthesia. Dyspnoea was attributed to chronic right coronary artery (RCA) ischaemia with concurrent mild–moderate systolic and moderate diastolic dysfunction. Contribution of LAD disease to symptomatology was also postulated. Consensus to proceed with surgical intervention of aneurysmectomy and CABG was achieved. Aneurysmectomy was aimed at improving LV function and reducing risk of stroke, and hence, CABG was favoured for revascularization. Collateral supply from RCA to the left system suggested some viability of LAD territory myocardium, and distal LAD was planned for revascularization to address potential contribution to symptomatology.

The patient commenced warfarin and continued dual antiplatelet therapy. Guideline-based therapy for heart failure was instituted. Chest X-rays (CXRs) were obtained for surgical planning (*Figure 4*). Computed tomography (CT) was not undertaken, as it would not alter surgical approach. Viability study with cardiac magnetic resonance imaging (MRI) may have assisted decision-making regarding revascularization of the LAD; however, it was not available.

Elective CABG and aneurysmectomy with LV reconstruction was undertaken. The pericardium was opened, LVA observed at the apex, and then resected completely with bovine pericardial patch repair. The left internal mammary artery and left radial artery were grafted to the LAD and posterior descending artery, respectively. Resected specimen measured 30 × 50 × 36 mm and was dome shaped with new and old friable thrombus centrally and surrounding calcification (*Figure 5*). Histological analysis demonstrated extensive replacement of myocardium by hyalinized, collagenous stroma with focal calcification and adherent thrombus.

The patient recovered uneventfully. Day 7 TTE demonstrated no intraventricular thrombus and LVEF of 40%. The patient reported improvement in exercise tolerance after a period of recovery and cardiac rehabilitation, which persists at 2 years post-op.

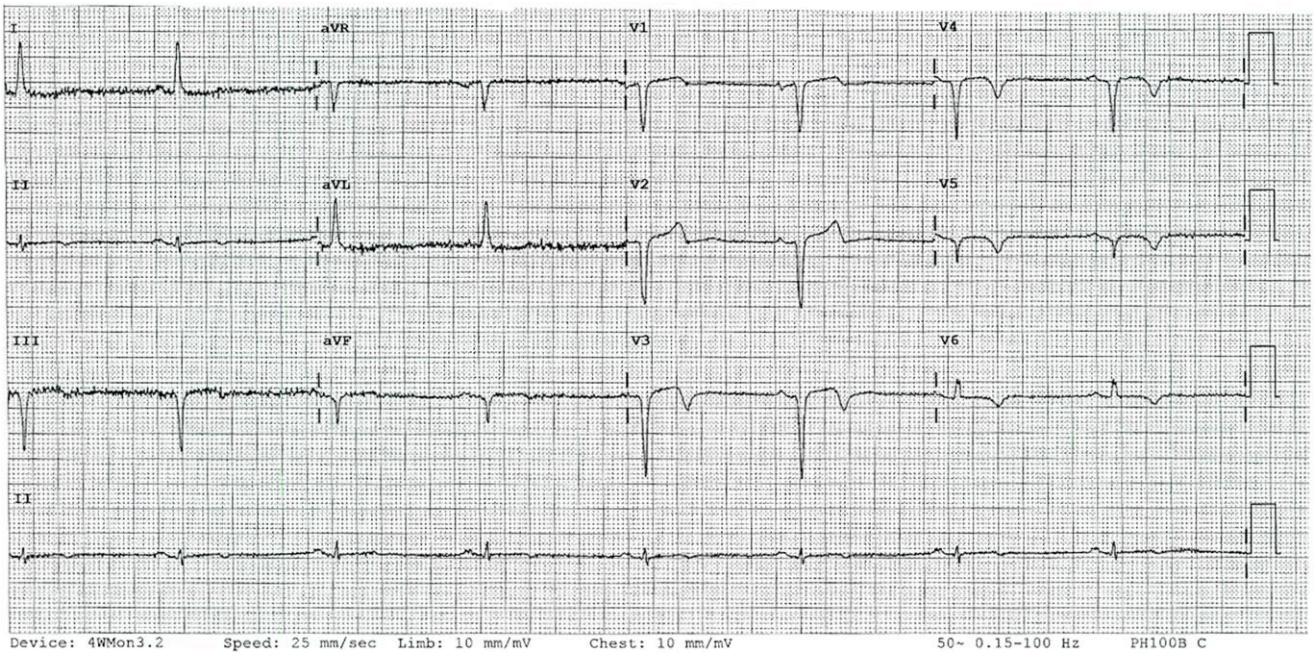

Figure 1 Baseline electrocardiogram demonstrating sinus bradycardia with a rate of 47 b.p.m. Left axis deviation, with extensive Q-waves and T-wave inversion through V_3–V_6 and small degree of ST-elevation in V_2–V_3, is also demonstrated, in keeping with previous anterior ST-elevation myocardial infarction and left ventricular aneurysm.

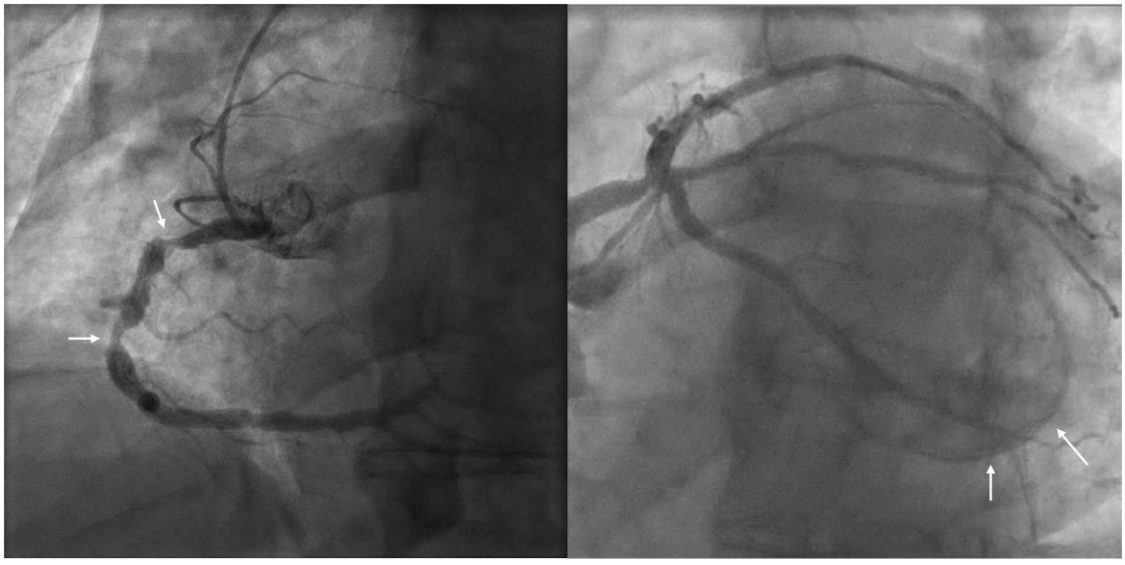

Figure 2 Invasive coronary angiography. Left panel: Coronary angiogram following injection of the right coronary system. The right coronary artery is ectatic with proximal and mid-vessel stenosis (arrows). Right panel: Coronary angiogram following injection of contrast into the left coronary system, with delineation of a calcified left ventricular aneurysm at the apex (arrows).

Discussion

Left ventricular aneurysms are an uncommon yet significant complication of AMI, increasing risk of thromboembolism, heart failure, ventricular arrhythmias, tamponade, and rupture.[1,4] Access to modern reperfusion strategies has decreased LVA incidence.[1] This case details the imaging, macroscopic, and microscopic appearance of an even rarer calcified LVA in a patient with distant history of STEMI. Classical ECG

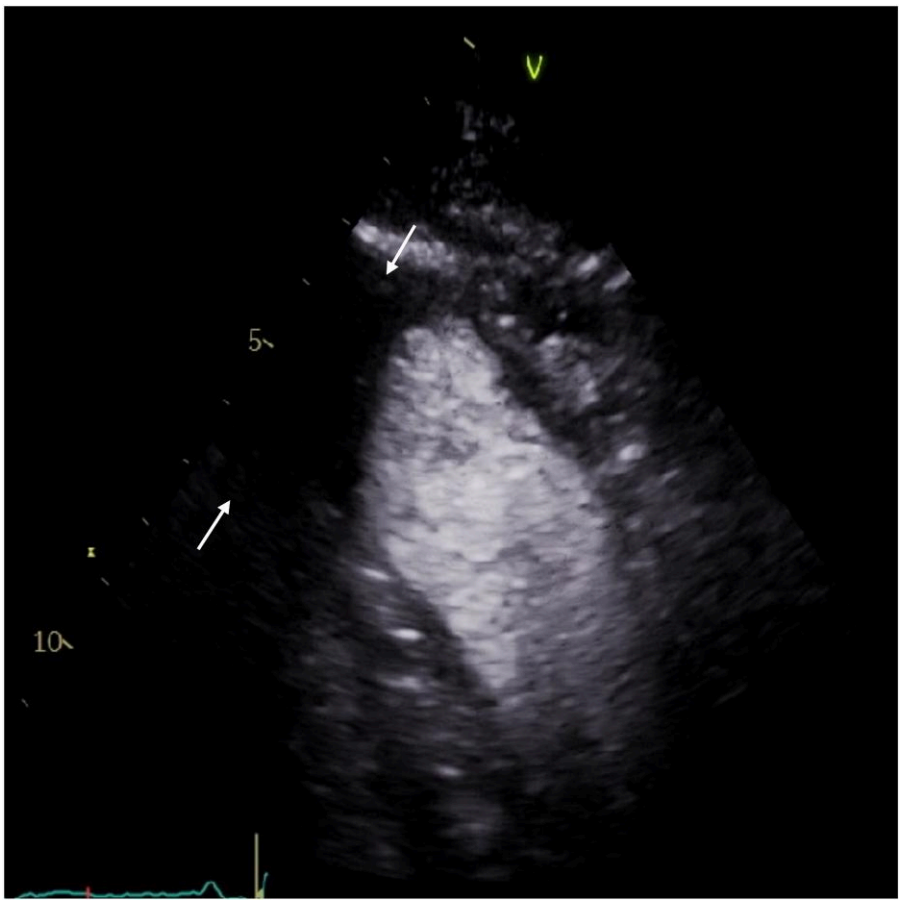

Figure 3 Transthoracic echocardiogram with intravenous contrast demonstrating apical inferior/infero-septal segment area filling defect (arrows), favoured in this context to represent mural thrombus.

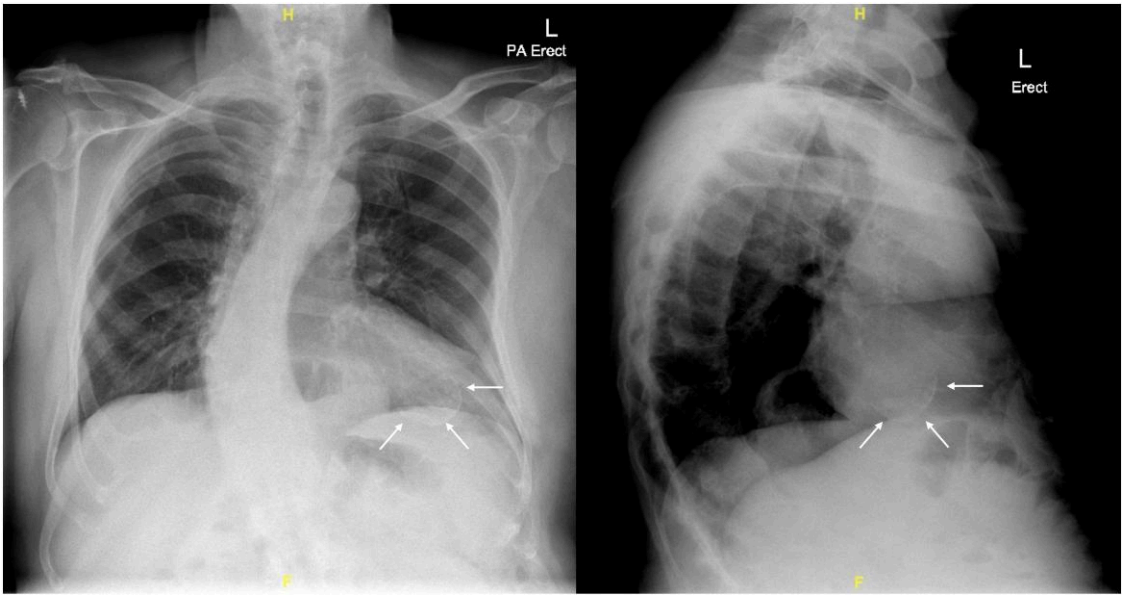

Figure 4 Posterior–anterior and lateral chest X-ray. Posterior–anterior and lateral projection chest X-ray demonstrating well-defined calcific lesion within the cardiac silhouette (arrows). Scoliosis with right-sided convexity also seen.

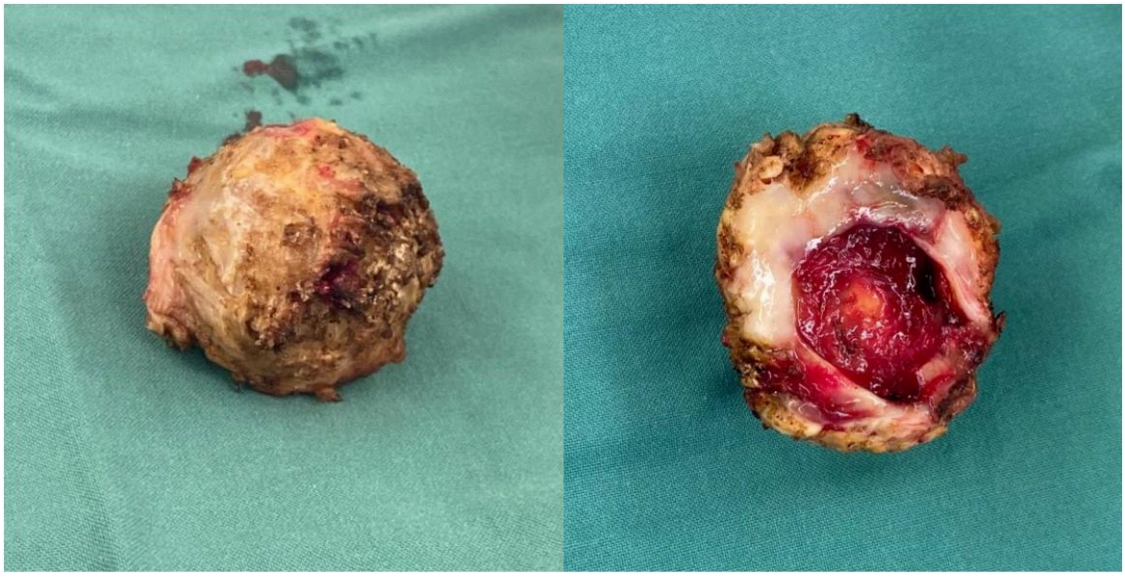

Figure 5 Macroscopic appearance of surgically resected specimen. Surgically resected left ventricular apical aneurysm, demonstrating dome-shaped appearance macroscopically with the presence of intra-aneurysmal thrombus.

changes of LVA, namely ST-elevation with T-wave inversion and Q-waves, are present.

Limited reports of calcified LVA exist. Consistent with our experience, these relate to distant anterior AMI and had associated thrombus.[5–7] Thrombus is seen in 48% of patients who undergo aneurysmectomy.[8] Clinicians should remain suspicious of the development of thrombus in LV injury, given the potential for embolic events.[4] Calcified ventricular aneurysm is suggestive of myocardial injury and aneurysm formation in the distant past, given the time required for calcification. In chronic LVAs, fibrosis and calcification lead to underlying pericardium becoming rigid, and hence, rupture rarely occurs.[9]

As incidence of LVA decreases, maintaining ability to identify aneurysm remains important. Evaluation involves multimodal imaging, including CXR (which may show cardiomegaly, aneurysmal deformation, and occasionally calcification as seen in our study) and TTE. Imaging with CT and MRI may be useful in the case of diagnostic doubt and for surgical planning.[9]

Consensus guidelines exist for STEMI[3]; however, guidelines pertaining to investigation, diagnosis, and management of LVA are lacking, and surgical management remains controversial. Early coronary reperfusion and angiotensin-converting enzyme inhibitors are central to minimizing ventricular remodelling and aneurysm development.[9] Medical therapy is likely appropriate in most cases.

In contrast, left ventricular pseudoaneurysms (LVPs) resulting from rupture of the LV secondary to myocardial injury, with containment by pericardium or scar tissue, necessitate emergency surgical repair. The use of percutaneous transcatheter LV closure devices for LVP is increasing[10]; however, they do not have an established role in aneurysms outside of this context.

Surgical resection appears favoured in case reports and small studies of LVA, particularly when CAD necessitating CABG is present. These are likely biased towards patients undergoing intervention, while majority are treated conservatively and not reported. Surgical resection eliminates fibrotic tissue substrate for ventricular arrhythmia, decreases myocardial demand, improves contractility, and reduces need for lifelong anticoagulation, thus decreasing complications and improving symptoms.[9]

Conflicting evidence of mortality/morbidity benefit exists and may be related to the degree of LV end-diastolic volume reduction achieved.[11] A retrospective analysis of patients with LVA and CAD comparing CABG with aneurysmectomy vs. percutaneous coronary intervention (PCI) vs. medical management found surgical management superior in improved LV end-diastolic dimension and LVEF.[12] Another study demonstrated association between surgical repair of LVA and improvement in functional status, with concomitant coronary revascularization not improving early mortality or functional status but decreasing risk of post-op low-output state.[13]

Five-year post-operative survival is reported as high as 91.4%,[14] with superior 5-year survival in surgical vs. medical management analysis (70–75% vs. 45%).[15] However, many studies comparing surgical and medical management predate current guidelines for heart failure and may no longer be reliable. Symptomatic improvement achieved in our case may be attributable to aneurysmectomy and CABG, heart failure therapy, or cardiac rehabilitation, but is likely cumulative of all interventions. As LVA incidence decreases and contemporary medical therapies evolve, further evaluation of impact of surgical intervention on morbidity and mortality is required.

Lead author biography

Dr Dean Nelson is a physician trainee from St Vincent's Hospital Melbourne in Victoria, Australia. During his work in the Department of Cardiology, he has enjoyed the opportunity to explore interesting case studies in the area of interventional cardiology and echocardiography with the support and mentorship of his co-authors. Dean hopes to undertake advanced training in cardiology in the future and combine his interest in clinical cardiology with his passion for research.

Supplementary material

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

Consent: The authors declare that consent has been given for publication of each of the detailed case studies in accordance with COPE guidelines.

Conflict of interest: None declared.

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Data availability

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

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