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CASE REPORT
Coronary heart disease

Hyperacute simultaneous cardiocerebral infarction related to floating thrombus in the ascending aorta: a case report

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Background Ascending aortic thrombus has been reported in several case reports, often revealed by peripheral embolization, but very few revealed by cardiocerebral infarction. Moreover, there is no defined treatment strategy.

Case summary An 83-year-old woman was admitted to our intensive care unit for concurrent acute myocardial infarction (AMI) and acute stroke, both with the presence of an embolism. Imaging revealed a floating thrombus in the ascending aorta. The thrombus resolved after anticoagulant therapy was administered, and there was no subsequent embolism recurrence.

Discussion Floating thrombus in the ascending aorta is an unusual cause of AMI. The main mechanisms of thrombus formation include erosion of an atherosclerotic plaque, but it can also form without tissue abnormality with the probable implication of Virchow's triad. However, the precise mechanism for thrombogenesis remains unknown. In patients with a low surgical risk, we should consider surgical treatment, especially as anticoagulant therapy does not appear to reduce the risk of arterial embolization. Thrombolysis and endovascular interventions have also proven effective in certain cases. Overall, in patients with high surgical risk, decision will have to be made on a case-by-case basis.

Keywords Acute myocardial infarction • Embolic stroke • Coronary embolism • Ascending aorta thrombus • Floating thrombus • Case report

ESC Curriculum 2.1 Imaging modalities • 3.2 Acute coronary syndrome • 3.4 Coronary angiography

Learning points

- Ascending aortic thrombus should be suspected in cases of multiple systemic embolisms.
- Simultaneous acute myocardial infarction (AMI) and ischaemic stroke should lead to a search for arterial embolization, and it could be useful to perform a head and chest computed tomography scan prior to cardiac catheterization in case of neurologic symptoms in the context of AMI.

Introduction

Both acute myocardial infarction (AMI) and acute ischaemic stroke are medical emergencies which require timely diagnosis and management. Coronary artery embolism (CE) is recognized as a non-atherosclerotic cause of ST-segment-elevation MI (STEMI), with a prevalence ranging from 4% to 13% according to angiographic or autopsy studies.[1–4] Coronary artery embolism has been associated with several clinical conditions such as aortic and mitral prosthetic heart

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valves, atrial fibrillation (AF), dilated cardiomyopathy, neoplasia, infective endocarditis, atrial septal defect, intracardiac tumour, and hypercoagulable state.[3] There are some cases in the literature where AMI was caused by ascending aortic thrombus embolization but with no clear therapeutic strategy.[5,6] We report an unusual case of simultaneous acute myocardial infarction and embolic stroke in an older woman.

Timeline

Time	Event
23 January 2021 12H25	Sudden dyspnoea and chest pain
23 January 2021 13H10	Admission to emergency room
23 January 2021 13H28	Acute myocardial infarction diagnosed on electrocardiogram
23 January 2021 14H00	Cardiac catheterization: significant stenosis of the proximal left anterior descending artery (LAD) artery, distal occlusion of the LAD artery, and two distal occlusions of obtuse marginal branches.
23 January 2021 14h20	Confusion and incoherent speech
23 January 2021 15H00	Computed tomography (CT) scan: no cerebral lesion, ascending aorta floating thrombus
25 January 2021	Brain magnetic resonance imaging (MRI): multiple acute embolic strokes
8 February 2021	CT scan: decreasing in size of the thrombus, no new cerebral lesion
10 April 2021	Follow-up CT scan: total resorption of the thrombus

Case presentation

An 83-year-old woman presented to the emergency department with acute chest pain and sudden dyspnoea. The pain was not modified by breathing or position, and the clinical examination revealed crackles in the lung bases on pulmonary auscultation and no abnormality on cardiac auscultation.

She had a past medical history of chronic obstructive pulmonary disease treated with inhaled steroids, and breast cancer treated by surgery 10 years previously without recurrence. She was a current smoker.

Electrocardiogram showed ST-segment elevation in the anterior and inferior leads (*Figure 1*).

The patient was diagnosed with anterior acute myocardial infarction and subsequently underwent emergency cardiac catheterization. The intervention revealed significant stenosis of the proximal left anterior descending artery (LAD) with thrombolysis in myocardial infarction III flow, distal occlusion of the LAD, and two distal occlusions of the obtuse marginal arteries. There was no luminal stenosis on the right coronary artery (*Figure 2*).

During the procedure, she presented with confusion and incoherent speech without any focal neurological signs. Consequently, no coronary revascularization was performed (thrombolysis, thrombectomy, or angioplasty).

Aortic root dissection or embolic migration was suspected as the cause of the problem. We therefore immediately performed a CT scan, which showed no recent cerebral lesions or dissection, but a floating thrombus was identified in the ascending aorta in contact with a calcified atheromatous plaque. Otherwise, no left atrial appendage or other cardiac thrombus was found (*Figure 3*).

We then performed a brain magnetic resonance imaging (MRI), which showed ischaemic stroke in multiple territories, leading to a diagnosis of acute embolic stroke (*Figure 4*).

Blood testing revealed elevated troponin Ic levels (peak at 270 ng/mL, normal range < 0.1 ng/mL) and hyperhomocysteinaemia (50 µmol/L). The other blood parameters were within normal ranges, including haemoglobin, blood clotting, renal function, inflammatory markers, and autoimmune tests.

Transthoracic echocardiography showed severe hypokinesis of the anteroapical and apical segments (left ventricular ejection fraction = 35%), and no cardiac or aortic thrombi were observed, although the echogenicity of the aortic root was of poor.

She was monitored during the first week in the intensive care unit, and no supraventricular tachycardia was detected.

Medical treatment with unfractionated heparin 20 UI/kg/h and aspirin 100 mg was initiated, but we did not perform emergency surgery considering the frailty of the patient. Given a rapid neurological recovery with no residual impairment and small cerebral lesions without haemorrhagic component, there was no contraindication to the introduction of curative anticoagulation therapy.

Two weeks later, a thoraco-abdominopelvic CT scan showed that the ascending aorta thrombus had decreased in size, and there was no abnormality at the abdominopelvic level.

Considering this decrease and the absence of clinical deterioration, especially with a normal neurological state and considering a risk–benefit balance unfavourable to surgery, we decided to continue medical treatment with vitamin K antagonists (International normalized ratio 2–3) and aspirin 100 mg, in combination with close clinical and imaging follow-up. We did not plan additional brain imaging during the follow-up in the absence of new neurological impairment.

We also introduced cardio-protective treatment considering the post-infarction systolic cardiac dysfunction (angiotensin-converting enzyme inhibitor i.e. ramipril 5 mg, beta-blocker i.e. bisoprolol 2.5 mg, and aldosterone antagonist i.e. eplerenone 25 mg), and the patient attended a cardiac rehabilitation program before being discharged. There were no guidelines for imaging and clinical follow-up in this particular case, so we implemented imaging follow-up over a period of 3 months, during which total resorption of the thrombus was obtained (*Figure 5*), and maintained long-term anticoagulant therapy, transthoracic echocardiography at 3 months showed a significant recovery with a remaining hypokinesis of the apical segment (left ventricular ejection fraction = 45%), the patient had no residual neurological deficit nor symptoms of heart failure.

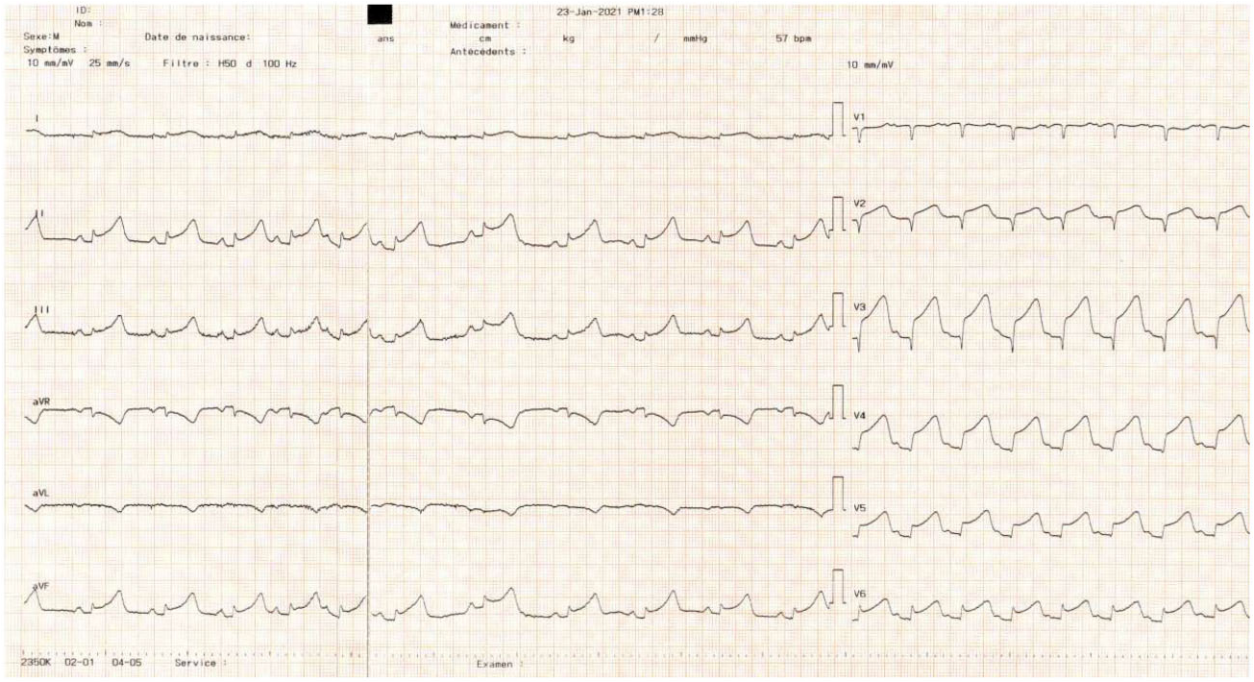

Figure 1 Electrocardiogram showing ST-segment elevation in the anterior and inferior leads.

Discussion

Epidemiology of coronary embolism

Floating thrombus in the ascending aorta is an unusual cause of AMI, but there are some cases reported in the literature. Simultaneous ascending aorta floating thrombus is a rare cause of cardiocerebral infarction, as illustrated by the large cohort study published by Popovic *et al.* including 1232 consecutive patients who presented *de novo* STEMI.[3] The case definition of CE used in this study consisted of major and minor criteria according to Shibata *et al.*[4] They identified only 53 patients with STEMI (4.3%) and CE; 12 of these patients (22.6%) had multisite CE, and 9 patients (17.0%) had another extracoronary embolic localization. Atrial fibrillation was the most frequent cardiac cause of CE (41.5%), while 15 patients had paroxysmal or permanent AF (28.3%) and 7 patients had new-onset AF (13.2%). Only two cases of thoracic aortic thrombus were observed (3.7%), and 14 patients (26.3% of cases) had no identifiable cause of CE. In addition, the cohort published by Shibata in 2015, including 1776 patients who presented with *de novo* acute MI, the prevalence of CE was 2.9% ($n = 52$), including 8 (15%) patients with multivessel CE. Atrial fibrillation was the most common cause ($n = 38$, 73%), and no cases of aortic thrombus were identified.[4]

Risk factors for ascending aorta floating thrombus

The mechanism of thrombus formation linked to atherosclerotic plaque rupture is well known, and is associated with key risk factors such as hyperlipidaemia, smoking, and diabetes mellitus.

In their review, Knoess *et al.* noted that the formation of ascending aortic thrombus is more frequently observed in young women. In addition, a significant proportion of cases presented no macroscopic or microscopic tissue anomaly at the thrombus attachment site. Although several prothrombotic risk factors may contribute to this phenomenon, the precise mechanism for thrombogenesis remains unknown.[5]

Superficial erosion of an atherosclerotic plaque leading to thrombus formation may be the main mechanism revealed by previous studies; however, it could also be formed without tissue abnormality with a probable significance of Virchow's triad.[5–7]

European Society of Cardiology guidelines emphasize the notion of complex aortic plaques (defined as plaques with mobile thrombi or ulcerations or a thickness ≥4 mm). These complex plaques are a risk factor for embolization, particularly for stroke. The guidelines therefore define two types of lesions; mobile thrombus without atherosclerosis for which the pathophysiological causes are unclear, and mobile thrombus in the context of complex plaques.[8]

The lesions presented by our patient thus fell within the definition of a complex plaque, with imaging that confirmed the presence of atheroma, and our patient also presented risk factors such as sex, age, and current smoking.

Multiple localizations, imaging, and follow-up

To our knowledge, there are only three reported cases of simultaneous coronary and cerebral embolization related to thrombus of the ascending aorta.[9–11]

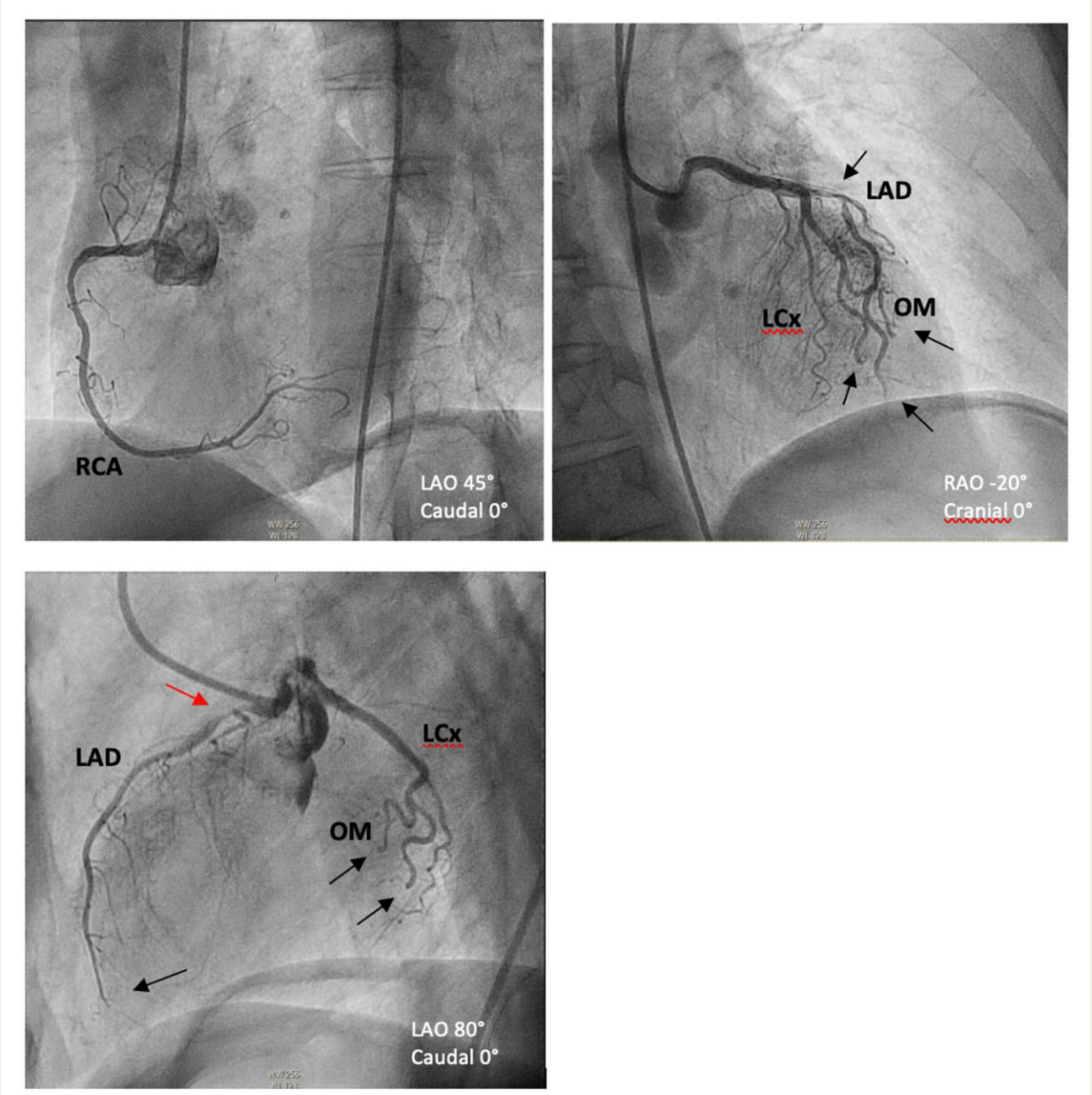

Figure 2 Coronary angiogram showing significant stenosis of the proximal left anterior descending artery, acute obstruction of the distal left anterior descending artery and of the circumflex artery. No luminal stenosis in the right coronary artery.

However, thrombus of the ascending aorta is more frequently associated with distal embolization, particularly with acute lower limb ischaemia, or discovered incidentally.

In acute coronary syndrome, it is useful to perform a rapid echocardiographic examination before coronary angiography, especially in patients with hemodynamic instability or pulmonary oedema. This may allow the medical team to detect mechanical complications such as papillary muscle rupture, ventricular septal defect, cardiac tamponade, or cardiogenic shock, which could avoid a therapeutic delay and optimize management.

Simultaneous acute coronary syndrome and ischaemic stroke should lead to a search for aortic dissection and arterial embolization prior to coronary angiography. A thrombus in the ascending aorta, a thrombus in the aortic valve, or an aortic dissection are among the injuries in which coronary angiography should be performed with parsimony as potentially harmful. In these cases, a trans-oesophageal

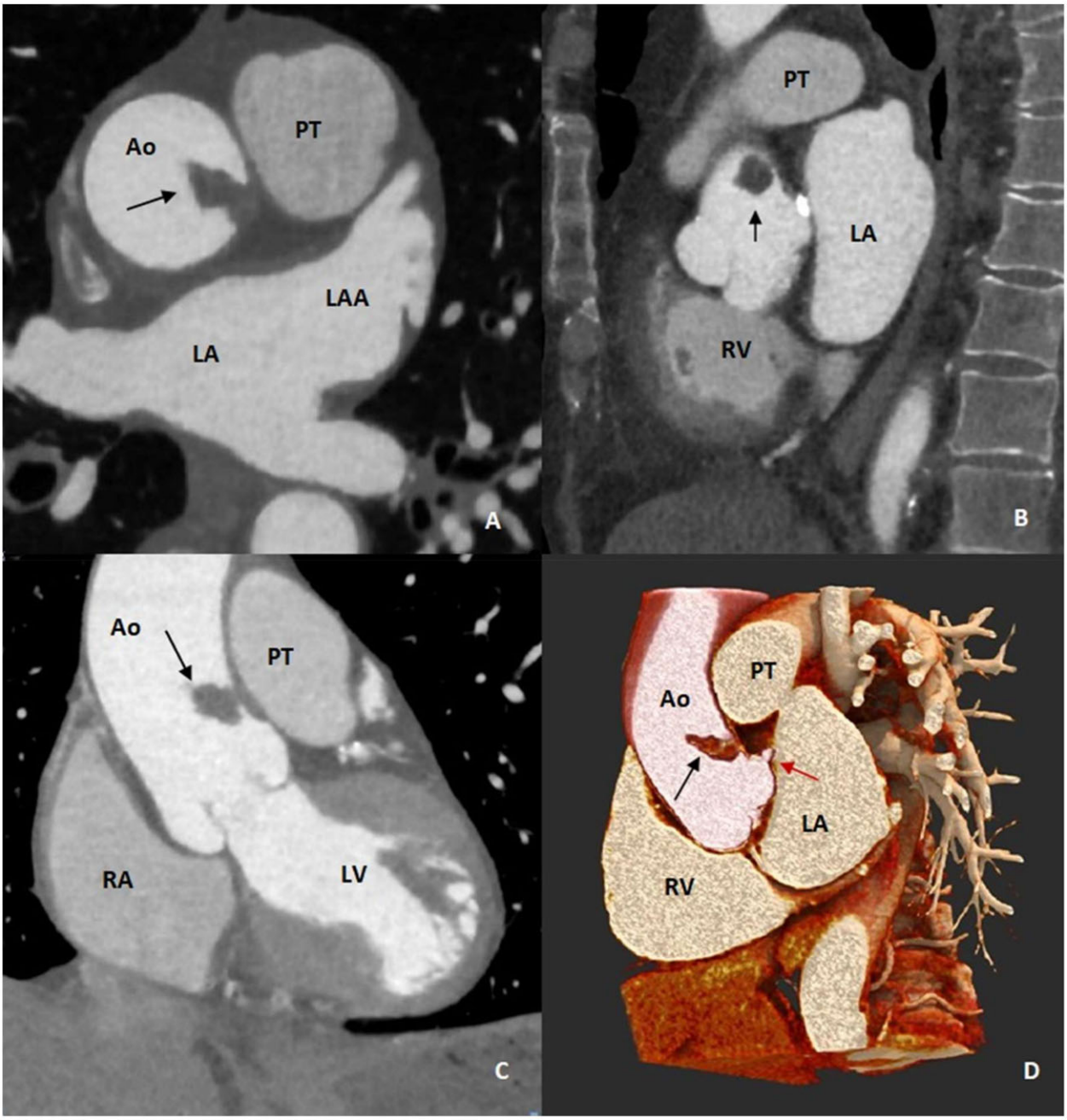

Figure 3 Computed tomography angiography of the supra-aortic arteries showing the floating thrombus in the ascending aorta (black arrow) close to the left main coronary artery ostium (red arrow) (*A*) axial plane; (*B*) sagittal plane; (*C*) coronal plane; (*D*) volume rendering reconstruction.

echography can be very useful as it has a better echogenicity than trans-thoracic echography, particularly for the aortic valve and the aorta. Furthermore, in our experience, it could be faster to perform than a CT scan, which remains the gold standard for the assessment of aortic lesions.

As for our patient, there was neither neurological impairment nor clinical symptoms of distal embolization, so we could not have suspected a thrombus in the aorta. Furthermore, given that the aortic root was poorly visualized on transthoracic ultrasound, clinical management could not have been improved. Indeed, in case of an STEMI, time to revascularization should not be delayed, thus no other complementary imaging beside a rapid echocardiographic examination could have been performed. Unfortunately, manipulations during the cardiac catheterization may have led to embolic stroke.

No clear recommendations on imaging during follow-up are available, but it seems appropriate to perform aortic CT scan until complete resolution of the thrombus, to allow analysis of the

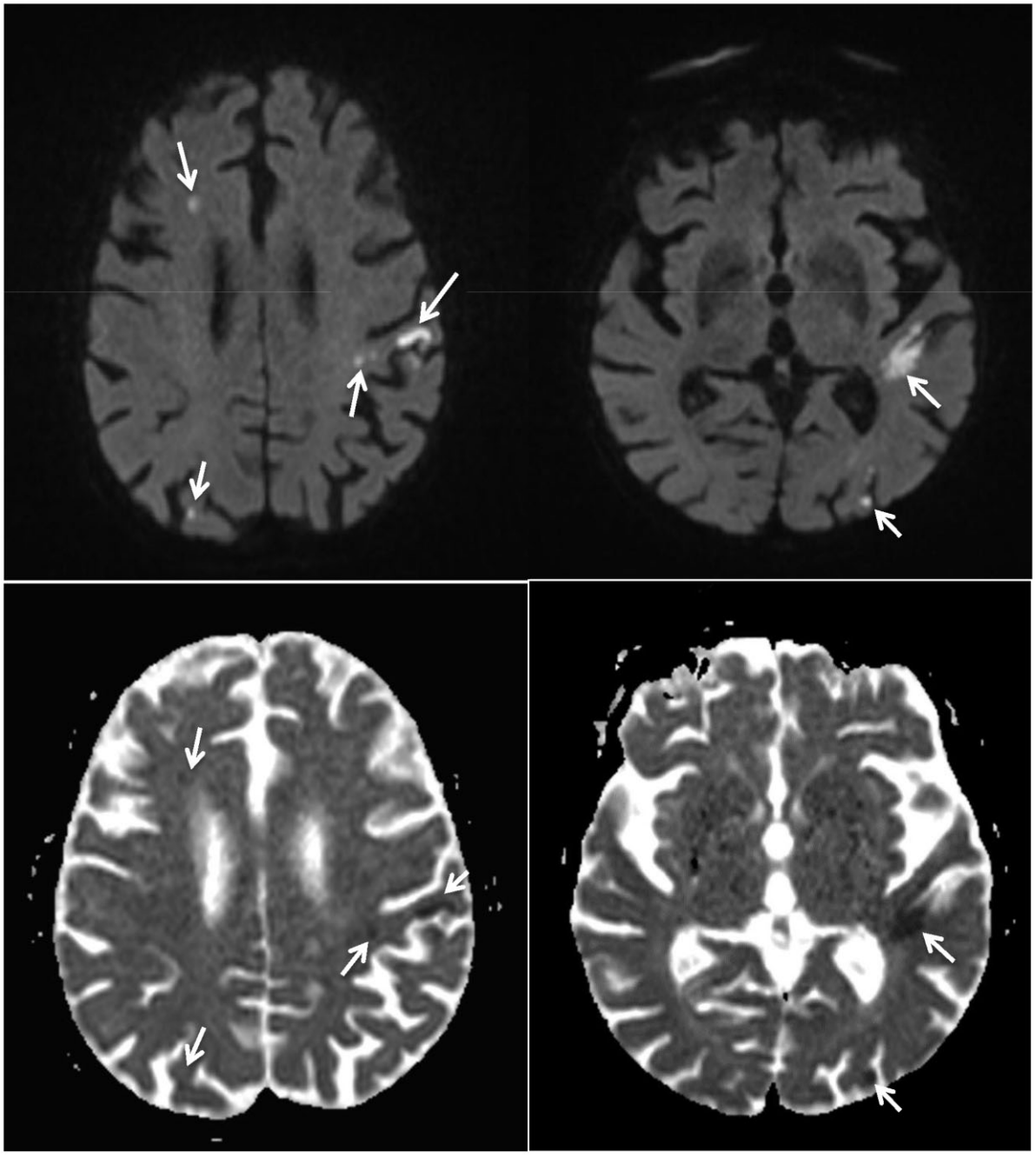

Figure 4 Diffusion-weighted magnetic resonance imaging (DWI) (above) and apparent diffusion coefficient (ADC) map (below) showing a multi-territory ischaemic stroke, suggesting an embolic aetiology.

effectiveness of the treatment, and to allow a better comparison than in follow-up with cardiac ultrasound alone. It also may be interesting to perform an aortic MRI to analyse the composition of the atheromatous plaques.

Treatment

There is debate as to whether patients should undergo treatment involving surgery or anticoagulant therapy. However, as no comparative data are available, the current medical guidelines remain unclear.[8]

A recent meta-analysis published by Chen *et al.* reviewed 107 cases of ascending aorta thrombus and compared the primary surgery strategy vs. anticoagulant therapy. They found that the thrombus persisted in approximately one-third of patients treated with initial anticoagulant therapy, and recurrent arterial embolization was observed in about 21%. No significant difference was observed in mortality rates between the two strategies.[12]

Another meta-analysis published by Fayad *et al.* reviewed 200 patients with aortic mural thrombus, of whom 112 received

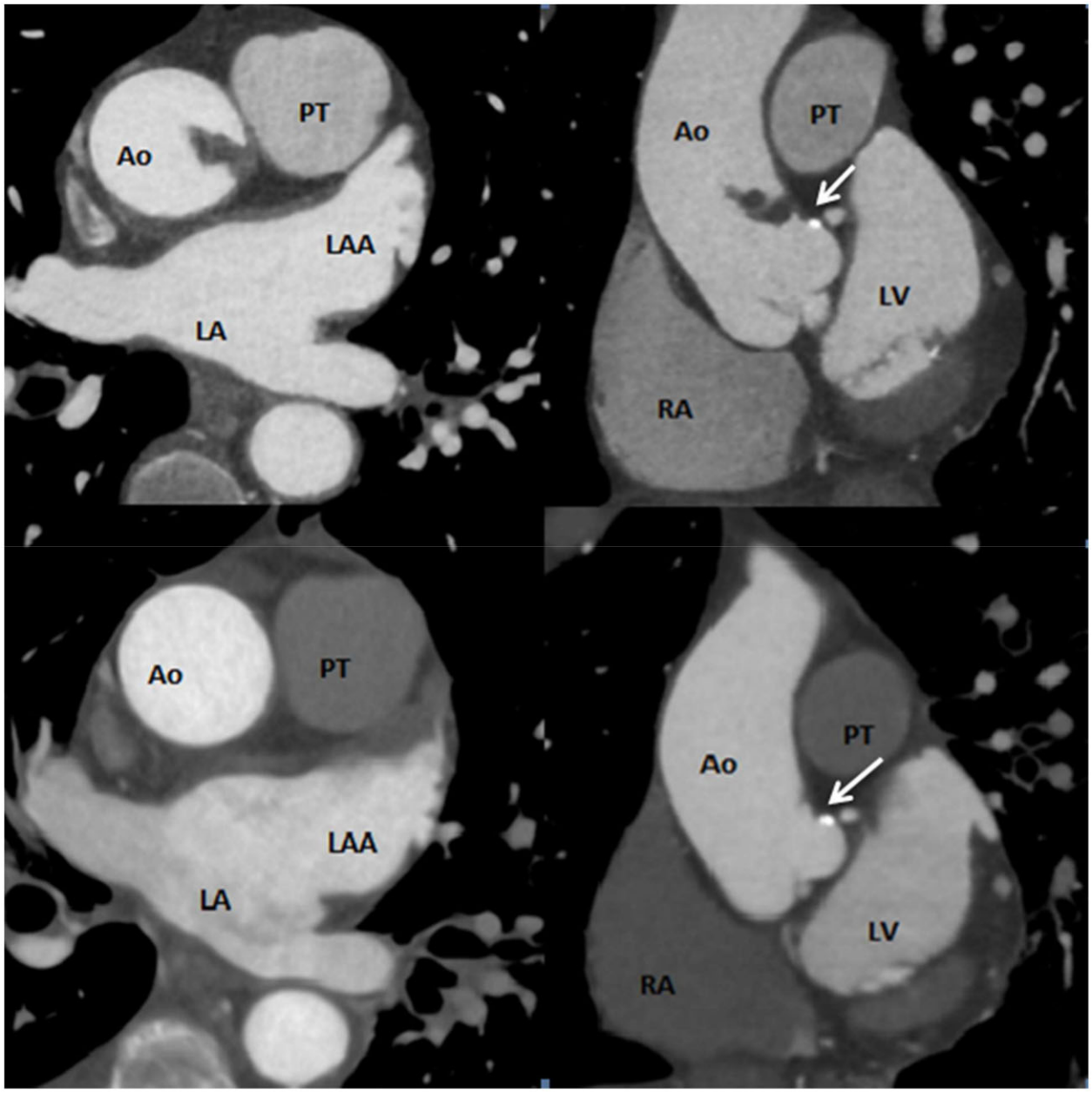

Figure 5 Initial (top) and 3-month follow-up (bottom) computed tomography angiography showing complete regression of the aortic thrombus with persistence of a partially calcified atheromatous plaque (arrow).

anticoagulation therapy and 88 received surgical treatment. 26.4% of patients treated with anticoagulation therapy experienced thrombus persistence or recurrence, compared with 5.7% of patients treated with surgery. In addition, there was a recurrence of peripheral arterial embolization in 25.7% vs. 9.1% of cases, respectively. Again, there was no significant difference in mortality rates between the two strategies.[13]

A surgical strategy seems more suitable for patients with a low surgical risk, especially as anticoagulant therapy does not appear to reduce the risk of arterial embolization, as illustrated in our case. However, in patients with high surgical risk like our patient, it was complicated to decide in the absence of clear recommendations and comparative studies, and it appears that for the time being this decision will have to be made on a case-by-case basis.

There is no current indication to use a direct oral anticoagulant therapy for the treatment of aortic thrombus. In our experience, the use of vitamin K antagonists is more widely used for valvular or left ventricular thrombi. We therefore decided to use a vitamin K antagonist for this purpose.

Regarding the use of thrombolysis, although it may allow resorption of the thrombus and may be useful in associated emergency situations such as ischaemic stroke on arterial embolization, there are

several problems. First, the risk of massive embolization, if the lysis of the thrombus base is faster than the lysis of the corpus. In addition, this technique presents a non-negligible risk of haemorrhage, especially if the origin of the thrombus is infectious.

Endovascular thrombus aspiration has also been assessed for feasibility. It has been described during thrombectomy of the descending aorta and aortic arch, allowing complete removal and subsequent analysis of the thrombus. However, there is a risk of peri-procedural embolization and arterial wall injury that can be limited by the use of intravascular ultrasound guidance.[14,15] Overall, it could be a promising technique in patients with a high surgical risk.

Lead author biography

Ranny Issa is a cardiology resident in the University Hospital of Dijon. He received a degree of in-depth training in medical sciences 2017, Dijon University and also a degree in cardiac intensive care unit 2021, Lyon University.

Supplementary material

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

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Slide sets: A fully edited slide set detailing these cases 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 image and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: Consultant or speaker for Bayer, BMS/Pfizer, Boehringer Ingelheim, Novartis, Sanofi, and Servier (Y.C.).

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