ESC European Society of Cardiology

European Heart Journal - Case Reports (2023) **7**, 1–5
https://doi.org/10.1093/ehjcr/ytad492

CASE REPORT
Cardiovascular imaging

Case Report: concurrent myocardial and cerebral infarction due to aortic thrombus

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Received 21 December 2022; revised 18 September 2023; accepted 5 October 2023; online publish-ahead-of-print 10 October 2023

Background Aortic mural thrombus is a rare acute aortic syndrome that can present with embolism to a distal organ. No guidelines or randomized evidence exist to guide therapy for patients with aortic mural thrombus. Cardiac and cerebral embolism is a particularly unusual presentation of aortic thrombus but has significant implications for patient management.

Case summary We present an unusual case of a young patient with simultaneous embolization of aortic thrombus to the coronary and cerebral vasculature, causing cerebral infarcts and a myocardial infarction. He presented with chest pain, slurred speech, right homonymous hemianopia, and inferior ST-elevation on electrocardiogram (ECG). Bedside echocardiography identified an inferoseptal regional wall motion abnormality. Emergent computerised tomography (CT) brain and aorta showed acute cerebral infarcts and aortic mural thrombus. He was managed medically with anticoagulation and discharged without disability after a period of rehabilitation.

Discussion This case demonstrates the value of careful clinical assessment in the setting of ST-elevation prior to transferring a patient for invasive angiography, as well as highlighting the role of echocardiography and CT imaging in the diagnosis of acute aortic syndromes. We describe the various management options for aortic mural thrombus, the role of multi-disciplinary decision-making, and our rationale for selecting a strategy of anticoagulation.

Keywords ST-elevation myocardial infarction (STEMI) • Stroke • Aorta • Thrombus • CT • Case report

ESC curriculum 2.4 Cardiac computed tomography • 2.1 Imaging modalities • 3.2 Acute coronary syndrome • 9.1 Aortic disease

Learning points

- Consider emergent cross-sectional imaging in patients with ST-segment elevation on the electrocardiogram prior to invasive angiography where aortic pathology is a possibility.
- Outline investigation and management strategies for patients with aortic mural thrombus.

Introduction

The combination of ST-segment elevation on the electrocardiogram and symptoms of myocardial ischaemia is commonly associated with occlusive myocardial infarction (MI). In such cases, timely revascularization is imperative, for which there are well-established pathways to convey patients directly to the cardiac catheterization laboratory for primary percutaneous coronary intervention (PCI).[1] However, ST-elevation can be observed in other pathologies, including acute aortic syndromes. In such cases, invasive angiography may lead to iatrogenic complications.[2]

We present a case of myocardial infarction and stroke due to embolism from aortic mural thrombus, which highlights the importance of careful clinical assessment and judicious use of imaging when selecting patients for emergency PCI. The management of this rare condition

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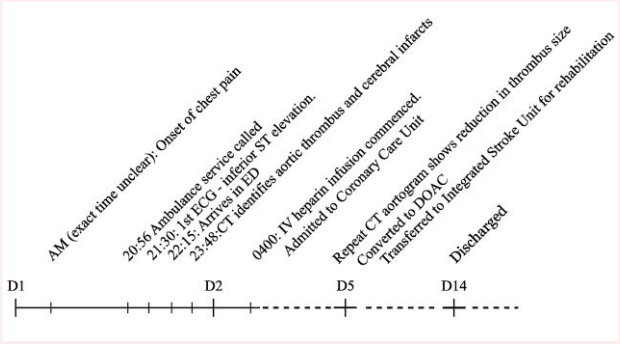
Handling Editor: Livia Gheorghe
Peer-reviewers: David Blusztein; Magdy Abdelhamid
Compliance Editor: Marta Peverelli

is not standardized, and we describe a conservative approach with multi-disciplinary input.

Summary figure



Case presentation

A 38-year-old man presented after developing severe central chest pain shortly after taking sildenafil. His past medical history included intellectual disability and medically unexplained abdominal pain, for which he took amitriptyline, metoclopramide, and lansoprazole. He smoked five cigarettes per day and consumed caffeinated energy beverages frequently. He had no symptoms or known history of COVID-19.

His initial symptoms lasted 10 minutes, recurring throughout the day. He did not have sexual intercourse. Approximately 6 hours later, he called his brother who noticed that his speech was slurred and called the ambulance service. A pre-hospital electrocardiogram (ECG) showed inferolateral ST-elevation and was transmitted to our regional primary PCI service (*Figure 1*). Due to the history of dysarthria, the patient was diverted to the Emergency Department for clinical assessment. On arrival, he was pain-free but hypertensive (blood pressure: 166/115 mmHg). Neurological examination identified dysarthria and a right homonymous hemianopia with no other deficit (NIHSS score 3). He had bilateral equal and simultaneous peripheral pulses, no murmurs, and no signs of cardiac failure. Respiratory and abdominal examinations were unremarkable. Repeat ECG showed persistent inferolateral ST-elevation. Transthoracic echocardiography identified inferoseptal hypokinesis with no other abnormalities. The ejection fraction was 56%.

Given the two simultaneous clinical syndromes of myocardial infarction and stroke, the main unifying differential diagnoses were acute aortic dissection or cardioembolic phenomena. Primary PCI was deferred pending urgent imaging, and a computerised tomography (CT) brain demonstrated acute left occipital and right superior cerebellar infarcts (*Figure 2*). CT angiography of the aorta identified a $15 \times 12 \times 10$ mm aortic thrombus adjacent to the right coronary artery (RCA) ostium, with smaller aortic thrombi adjacent to the origin of the left subclavian artery and at the level of the diaphragm (*Figure 3*). ECG gating was not available overnight, so the RCA was not well visualized.

After discussion with the Stroke and Cardiothoracic services, the risk of further emboli was felt to outweigh the risk of haemorrhagic transformation and the patient was anticoagulated with intravenous unfractionated heparin to a target anti-Xa activity level of 0.3–0.7 units/mL. Antiplatelet therapy was deferred to mitigate risk of haemorrhage. The patient did not meet criteria for thrombolysis. The initial high-sensitivity cardiac troponin-T concentration was 2406 ng/L (Roche Diagnostics, 99th centile upper reference limit ≤ 16 ng/L).

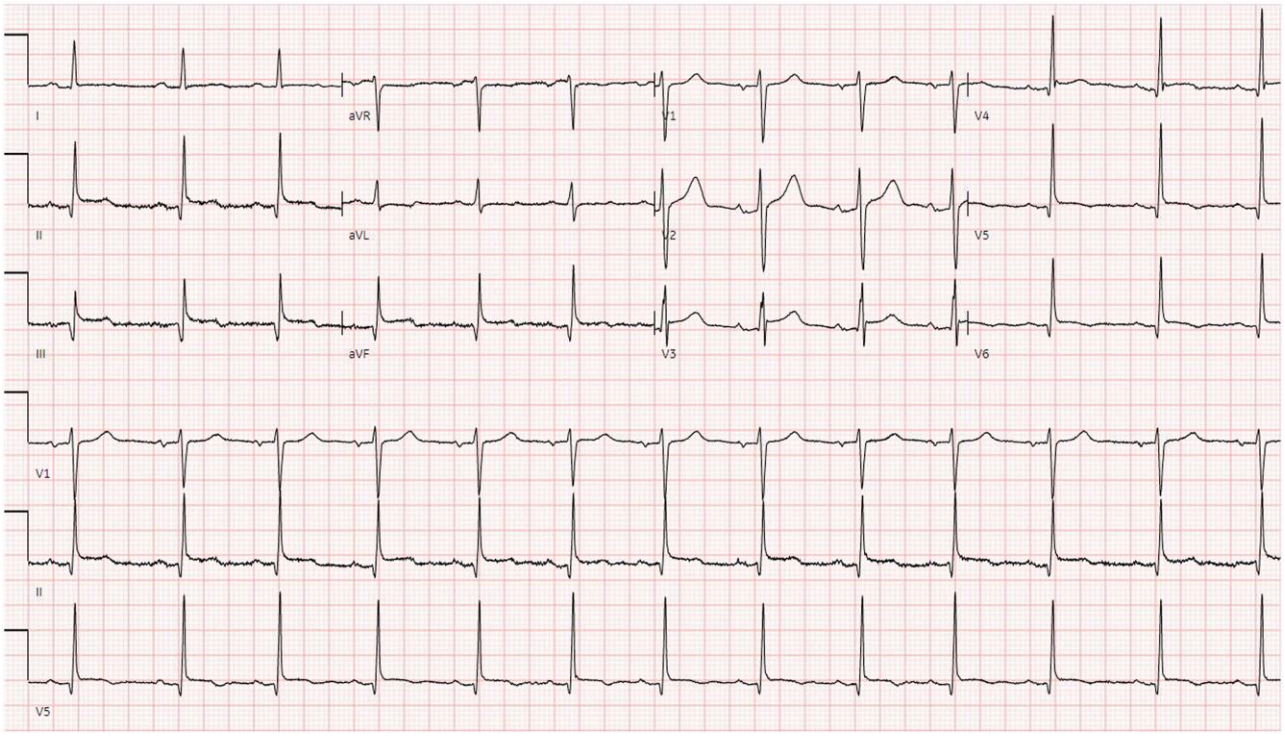


Figure 1 Initial 12-lead ECG. ST-elevation in leads II, III, and aVF.

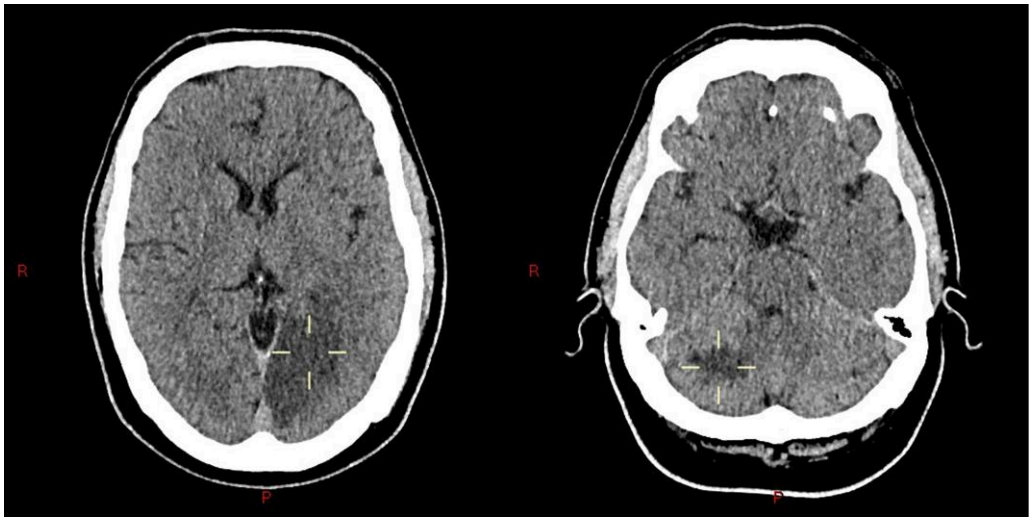


Figure 2 CT brain. Markers demonstrate acute left occipital lobe and right cerebellar infarcts.

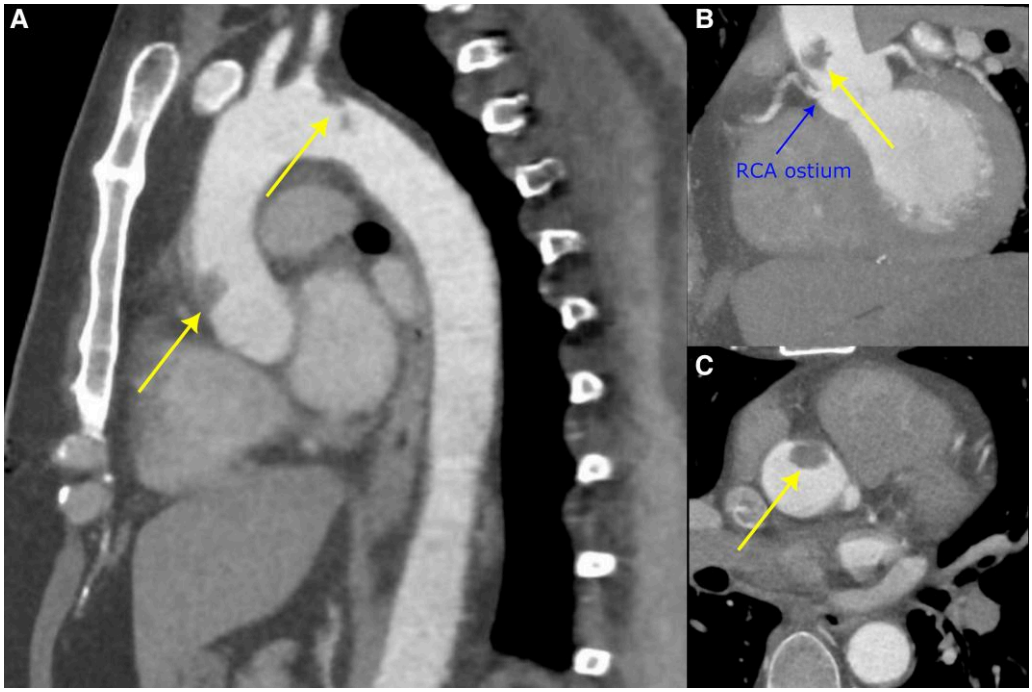


Figure 3 CT aorta. Arrows highlight thrombus. *A*) Oblique sagittal view of aorta. *B*) ECG-gated oblique view demonstrating proximity of thrombus to RCA ostium. *C*) Axial view.

Total cholesterol was 6.6 mmol/L (3.5–5.18 mmol/L) and LDL cholesterol 4.6 mmol/L (2.0–3.5 mmol/L). Serial ECGs demonstrated evolving inferior T-wave inversion with preservation of the R-wave. The patient remained stable and pain-free overnight.

The following morning, ECG-gated CT coronary angiography was undertaken. There was low attenuation plaque in the left system. There was contrast opacification of the distal RCA, which was a dominant vessel, although the mid-vessel was afflicted by motion artefact. There was no left ventricular or left atrial appendage thrombus.

De novo formation of aortic mural thrombus with coronary and cerebral embolization seems the likely diagnosis, as the symptom timing and preserved ventricular systolic function were not consistent with cardioembolism. However, the aetiology of the aortic mural thrombus was unclear.

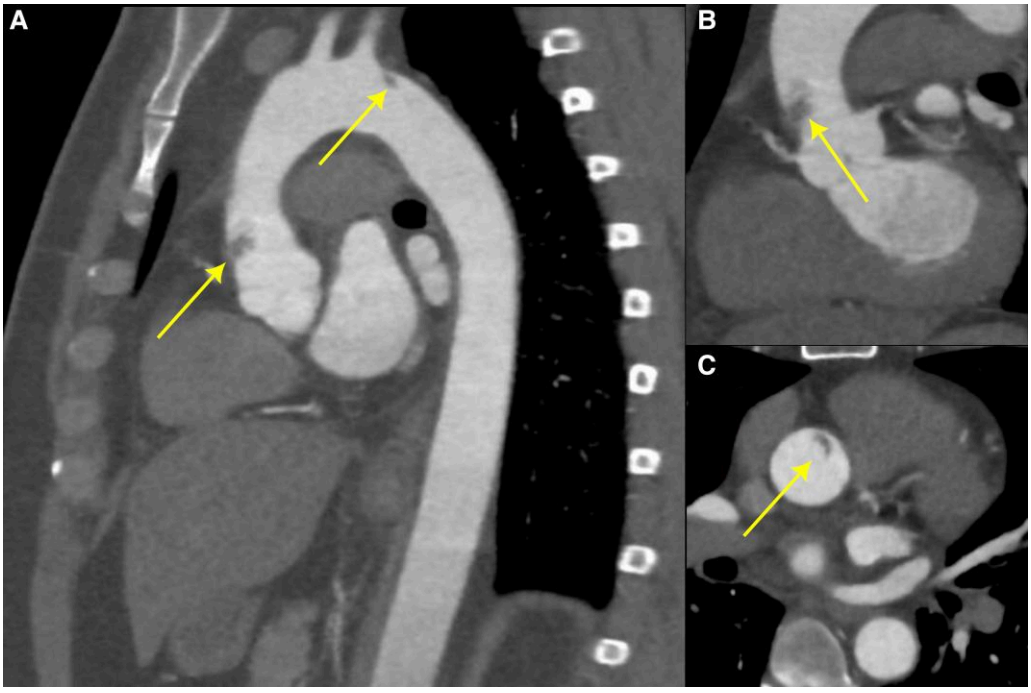


Figure 4 Repeat CT aorta showing reduction in thrombus size after 5 days of anticoagulation. Arrows highlight thrombus. *A*) Sagittal oblique view. *B*) ECG-gated oblique view. *C*) Axial view.

The patient remained on intravenous unfractionated heparin until Day 5. A repeat CT aortogram demonstrated reduction in size of the thrombi (*Figure 4*). He was switched to apixaban 5 mg b.i.d., commenced on secondary prevention (atorvastatin 80 mg, bisoprolol 5 mg, ramipril 7.5 mg), and transferred to the Integrated Stroke Unit for rehabilitation. A thrombophilia screen, including anticardiolipin antibodies, lupus anticoagulant, and Factor V Leiden, was normal. There were no arrhythmias on telemetry.

Both vision and speech improved, and the patient was discharged on Day 14 post-admission with no functional sequelae. Lifelong anticoagulation is planned though he has defaulted his planned Cardiology follow-up appointments.

Discussion

Aortic mural thrombus is a rare condition, with an estimated prevalence of 0.45% recorded in a series of over 10 000 autopsies.[3] Simultaneous coronary and cerebral embolization has occasionally been described,[2,4] but visceral or limb ischaemia are more typical manifestations. Mural thrombus is associated with underlying aortic atheroma in ~50% of cases[3]; other causes include aortic dissection, aortitis, and aneurysm,[5] which were not apparent in this case. Mural thrombus is most commonly found in the descending or abdominal aorta, with a lower incidence in the arch and, most rarely, the ascending aorta.[3,6] It is often linked with a hypercoaguable state, as observed in patients with malignancy, haematological disorders, and exogenous steroids or oestrogen[7]; again, these were absent in this case. Observational data suggest that COVID-19 may be linked to arterial thrombus formation,[8] but our patient had no evidence of SARS-CoV-2 infection. Aortic atheroma disruption leading to acute thrombus formation appears the most likely underlying pathology, particularly given his smoking habit and evidence of other arterial atheroma.

Direct transfer for primary PCI is an effective strategy for expediting revascularization in MI with ST-elevation, with significant mortality benefit.[1] However, activation of the pathway generates diagnostic momentum favouring ACS, with the potential for iatrogenic harm. The discovery of acute aortic pathology during invasive angiography is suboptimal and can precipitate further embolic events.[2] Astute clinical assessment leading to deferral of emergent invasive angiography was crucial to avoiding such harm in our patient. Alongside bedside echocardiography, ECG-gated CT angiography is clearly the imaging modality of choice for excluding acute aortic syndromes and is recommended by European Society of Cardiology guidelines in the setting of ST-elevation if aortic dissection or pulmonary embolism is suspected.[1]

Due to the rarity of aortic mural thrombus, there is a paucity of robust evidence to guide therapies. Options include anticoagulation with warfarin[9] or DOAC,[10,11] thrombolysis, or surgical thrombectomy.[6,12] The choice of optimal therapy is dictated by the individual clinical milieu. In our patient, anticoagulation rather than thrombolysis or surgery was favoured due to (i) clinical stability without ongoing myocardial ischaemia or pump failure, and (ii) acute ischaemic stroke with only mild neurological disability not meeting criteria for thrombolysis. Thrombolysis might also carry a theoretical risk of releasing pedunculated aortic thrombi, precipitating further systemic embolism.[7] We chose an intravenous unfractionated heparin infusion to enable rapid reversal of anticoagulation if haemorrhagic transformation occurred. Recurrent thrombus formation and embolization has been documented, suggesting a rationale for lifelong anticoagulation.[13]

Conclusions

Acute aortic syndromes may present with ST-segment elevation, leading to transfer for emergent primary PCI. This is associated with risk of

dislodging thrombus or propagating dissection. Careful clinical assessment is required before committing a patient with symptoms suggestive of aortic pathology to invasive coronary angiography. Whilst the management of aortic mural thrombus with concomitant coronary and cerebral emboli is not standardized, a conservative approach with multi-disciplinary team input led to a favourable outcome in our case.

Lead author biography

Alexander Thurston trained at the University of Oxford Medical School, before completing Foundation Training in South East Scotland. He is now an Internal Medicine Trainee in Edinburgh, hoping to specialize in Cardiology. His research interests focus on data science in acute cardiac care.

Consent: The authors confirm that written consent for submission and publication of the case report has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

Funding: This work did not receive funding.

Data availability

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

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