

Cerebral embolism due to left atrial myxoma in a patient presenting with chest pain and ST-segment elevation: a case report

António Fontes ^{1*}, Nuno Dias-Ferreira ², Anabela Tavares ¹, and Fátima Neves³

¹Department of Cardiology, Hospital do Divino Espírito Santo de Ponta Delgada, Avenida D. Manuel I, 9500-370 Ponta Delgada, Azores, Portugal; ²Department of Cardiology, Centro Hospitalar Vila Nova de Gaia/Espinho, Rua Conceição Fernandes, 4434-502 Vila Nova de Gaia, Portugal; and ³Department of Cardiothoracic Surgery, Centro Hospitalar Vila Nova de Gaia/Espinho, Rua Conceição Fernandes, 4434-502 Vila Nova de Gaia, Portugal

Received 22 December 2019; first decision 31 January 2020; accepted 22 July 2020; online publish-ahead-of-print 30 November 2020

Background

Myocarditis is an uncommon, potentially life-threatening disease that presents with a wide range of symptoms. In acute myocarditis, chest pain (CP) may mimic typical angina and also be associated with electrocardiographic changes, including an elevation of the ST-segment. A large percentage (20–56%) of myxomas are found incidentally.

Case summary

A 62-year-old female presenting with sudden onset CP and infero-lateral ST-elevation in the electrocardiogram. The diagnosis of ST-elevation myocardial infarction was presumed and administered tenecteplase. The patient was immediately transported to a percutaneous coronary intervention centre. She complained of intermittent diplopia during transport and referred constitutional symptoms for the past 2 weeks. Coronary angiography showed normal arteries. The echocardiogram revealed moderate to severe left ventricular systolic dysfunction due to large areas of akinesia sparing most of the basal segments, and a mobile mass inside the left atrium attached to the septum. The cardiac magnetic resonance (CMR) suggested the diagnosis of myocarditis with concomitant left atrial myxoma. The patient underwent resection of the myxoma. Neurological evaluation was performed due to mild vertigo while walking and diplopia in extreme eye movements. The head magnetic resonance imaging identified multiple infracentimetric lesions throughout the cerebral parenchyma compatible with an embolization process caused by fragments of the tumour.

Discussion

Myocarditis can have various presentations may mimic acute myocardial infarction and CMR is critical to establish the diagnosis. Myxoma with embolic complications requires emergent surgery. To the best of our knowledge, this is the first case reported in the applicable literature of a myxoma diagnosed during a myocarditis episode.

Keywords

Chest pain • ST-segment elevation • Myxoma • Cardiac imaging • Case report

Learning points

- Embolic complications of an atrial myxoma are due to fragments of the myxoma itself or surface emboli.
- Thrombolytic agents can cause bleeding and embolic complications.
- Nowadays, cardiac magnetic resonance is an essential tool in the evaluation of patients with acute chest pain and patent epicardial coronary arteries.

* Corresponding author. Tel: +35 129 620 3000, Email: antoniofontes@hotmail.com

Handling Editor: Hajnalka Vágó

Peer-reviewers: Tina Khan and Luca Arcari

Compliance Editor: Stefan Simovic

Supplementary Material Editor: Ross Thomson

© The Author(s) 2020. Published by Oxford University Press on behalf of the European Society of Cardiology.

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited. For commercial re-use, please contact journals.permissions@oup.com

Introduction

The accurate diagnosis of chest pain (CP) is still challenging. Establishing the correct cause and the appropriate treatment for patients with CP is one of the biggest problems physicians face in the Emergency Department (ED).¹ Myocarditis is an uncommon, potentially life-threatening disease that presents with a wide range of symptoms. In acute myocarditis, CP may mimic typical angina and be associated with electrocardiographic changes, including ST-segment elevation.² Cardiac primary tumours are extremely rare and frequently present in the form of systemic embolism.^{3,4} We are presenting a case of a patient with acute CP and ST-segment elevation, managed initially with fibrinolysis and complicated by cerebral embolism due to fragments of a left atrial myxoma. The coronary angiogram was normal, and myocarditis was confirmed through a cardiac magnetic resonance (CMR) as the myocardial process in progress.

Timeline

Days 1 and 2	62-year-old woman presenting sudden onset chest pain and infero-lateral ST-elevation in the ECG. Administration of tenecteplase. Patient complained of intermittent diplopia during transport to percutaneous coronary intervention centre. Coronary angiography showed patent epicardial coronary arteries. Echocardiogram revealed moderate to severe left ventricular systolic dysfunction and a mobile mass (60 mm) inside the left atrium.
Day 4	Cardiac magnetic resonance suggested the diagnosis of myxoma and myocarditis.
Day 5	Patient underwent resection of left atrial myxoma with autologous pericardial patch repair of the atrial septal defect.
Day 10	Neurological evaluation due to mild vertigo whilst walking and diplopia in extreme eye movements.
Day 12	Head magnetic resonance imaging identified multiple infracentimetric lesions throughout the cerebral parenchyma compatible with lesions caused by an embolism due to the myxoma.
Day 14	Patient was discharged from the hospital.
2 months	Good recovery and normal left ventricular ejection fraction.

Case presentation

A 62-year-old Caucasian woman with cardiovascular risk factors of smoking and dyslipidaemia was evaluated in the ED of her local hospital due to sudden onset anterior chest tightness with no radiation, relief positioning nor intensification through breathing movements. The patient described throat pain, as well as fever and cough for the

preceding 2 weeks. Constant CP started 2 h before the ECG was obtained, and it showed ST-segment elevation in the inferior and lateral leads (*Figure 1A*). This clinical condition was interpreted as a ST-segment elevation myocardial infarction. Acetylsalicylic acid and clopidogrel loading doses were immediately administered and thrombolytic treatment with tenecteplase (7000 U) was initiated [time estimated to percutaneous coronary intervention (PCI) was more than 120 min]. The patient was transferred to a PCI centre. During the helicopter transportation, the patient experienced two episodes of diplopia, lasting a few minutes. At arrival, ECG was repeated showing no ST-segment deviation (*Figure 1B*) and the coronary angiogram did not identify any atherosclerotic lesions. The patient noted complete relief from CP, although during evaluation presented with tachypnoea, basal crackles upon pulmonary auscultation, a 3/6 systolic murmur in the cardiac area, blood pressure of 130/70 mmHg, heart rate of 90 b.p.m. and body temperature of 37.8°C. Bed rest, furosemide, and supplemental oxygen were initiated. The echocardiogram showed moderate to severe left ventricular systolic dysfunction, due to areas of akinesia in the apex, distal and mid segments of the walls, sparing most of the basal segments and a mobile friable mass inside the left atrium, attached to the septum with dimensions of approximately 55 mm × 42 mm (*Figure 2*). Images acquired 48 h after admission revealed a left ventricle ejection fraction slightly improved ([Supplementary material online, S1–S3](#)).

The initial laboratory study showed leucocytosis ($15.7 \times 10^3 \text{ cell/mm}^3$) with neutrophilia and C-reactive protein (3.6 mg/dL), N-terminal prohormone of brain natriuretic peptide (1431 pg/mL), and troponin I (19.4 µg/L) slightly high. Other laboratory values were within normal range. On the second day, C-reactive protein increased to 14 mg/dL and the maximum troponin I levels were 20.2 µg/L.

The head computed tomography did not present bleeding or recent ischaemic lesions but could not identify morphologic sequelae of focal ischaemic lesions in the cerebellum and left occipital lobe.

In order to clarify the characteristics of the mass and understand the cause of the cardiac dysfunction, the patient was transferred to a tertiary centre with CMR available and possibility of performing cardiac surgery.

The CMR showed a mobile left atrium mass ([Supplementary material online, S4 and S5](#)), adherent to the interatrial septum, isointense with respect to myocardium on T1 weighted images (WI), hyperintense with respect to myocardium on T2 WI, with slight enhancement during first pass perfusion, and heterogeneous enhancement in the late gadolinium enhancement (LGE) images, suggesting a possible myxoma (*Figure 3*). Regarding the myocardium evaluation, the left ventricle was non-dilated with mild systolic dysfunction and multiple subepicardial LGE foci in the inferolateral and mid-distal segments of the anterolateral wall, consistent with the diagnosis of myocarditis (*Figure 4*).

The patient underwent surgery on the 6th day with total tumour excision and autologous pericardial patch repair of the atrial septal defect. The periprocedural transoesophageal echocardiogram showed the mobility of the mass in to the left ventricle during diastole ([Supplementary material online, S6–S8](#)).

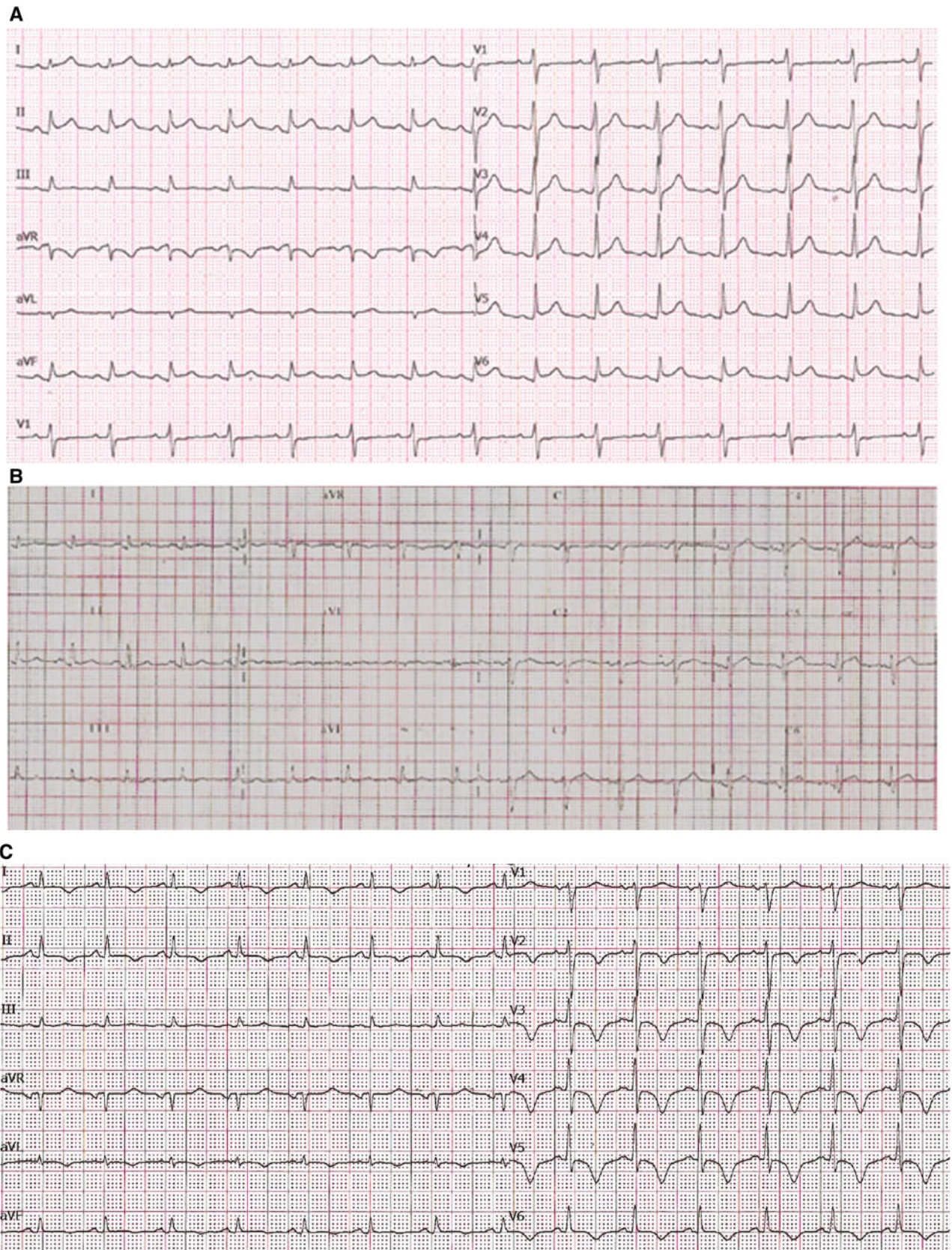


Figure 1 (A) Admission electrocardiogram with infero-lateral ST-segment elevation. (B) Electrocardiogram performed when patient arrived to the percutaneous coronary intervention centre, no ST-segment elevation present in inferior leads. (C) Electrocardiogram at Day 3, presenting T wave inversion in anterior, lateral, and inferior leads.

During recovery, the patient presented with mild vertigo while walking and diplopia in extreme eye movements. After a neurologist assessment, a cerebral magnetic resonance imaging was requested where were identified multiple infracentimetric lesions throughout the cerebral parenchyma, infra and supratentorial, affecting the cerebellar peduncles and left caudate nucleus. These lesions were moderately hypointense on T1 WI and hyperintense on T2 WI, presenting enhancement after administration of gadolinium. These features were consistent with an embolization process caused by fragments of the myxoma. The histopathological results of the operative specimen confirmed the diagnosis of myxoma.

The patient started in a rehabilitation program and at the 2 months' follow-up visit complete recovery from neurological deficits

was noted with the echocardiogram revealing normal left ventricle ejection fraction.

Discussion

This case emphasizes the importance of an adequate characterization of CP and integration of other relevant data from the medical history, essential for a correct, and definitive diagnosis.⁵ On the other hand, an early echocardiogram assessment can reveal unexpected

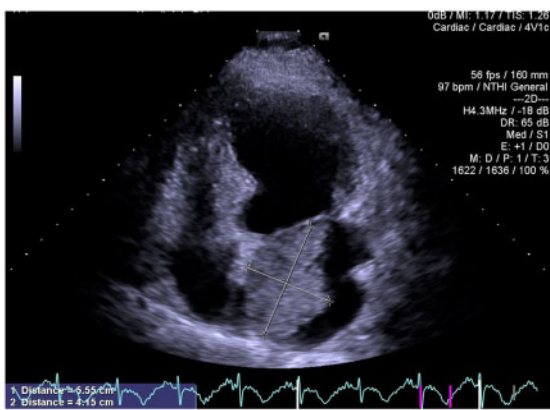


Figure 2 Left atrium myxoma with 56 mm × 24 mm dimensions.

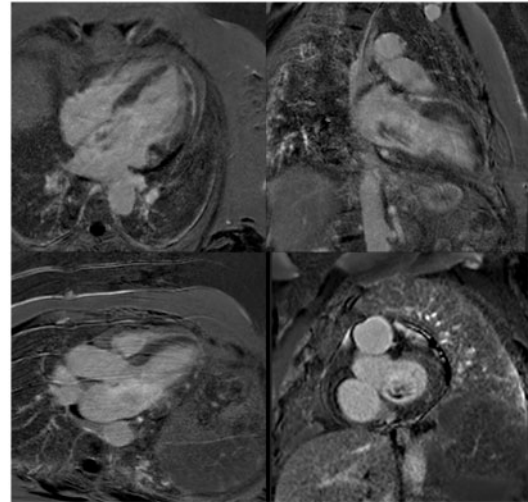


Figure 4 Images showing multiple subepicardial foci of late gadolinium enhancement in the inferolateral and mid-distal segments of the anterolateral wall, highly suggestive of myocarditis.

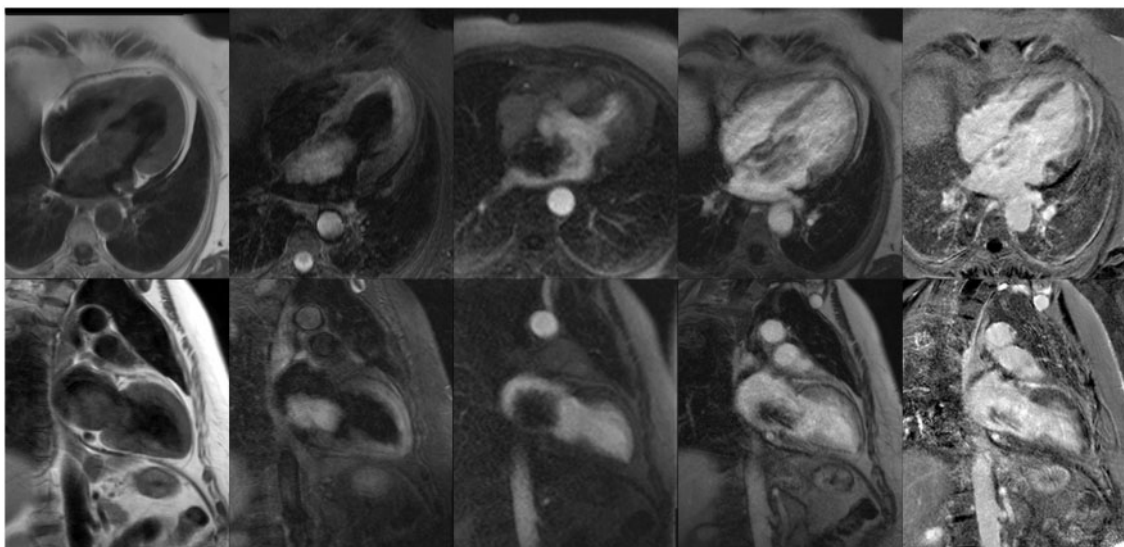


Figure 3 Cardiac magnetic resonance panel of four and two chamber planes showing a left atrium mass, adherent to the interatrial septum, isointense with respect to myocardium on T1 WI (A), hyperintense with respect to myocardium on T2 WI (B), with discrete enhancement during first pass perfusion sequence (C) and heterogeneous late gadolinium enhancement (E), in accordance with the diagnosis of a myxoma.

aetiologies. In our case, it could have raised other hypotheses, such as: embolic coronary event, stress cardiomyopathy, or myocarditis. A large percentage (20–56%) of myxomas are found incidentally.⁶

The CMR was essential to establish a definitive diagnosis of myocarditis, allowing tissue characterization of the mass and ultimately helping to guide medical and surgical management. Final diagnosis of myocarditis was supported by the LGE subepicardial pattern (not characteristic of stress cardiomyopathy or embolic myocardial infarction), the slightly elevated levels of troponin and the rise of the C-reactive protein from admission.⁷ The background of throat pain, as well as fever and cough in the preceding 2 weeks could also suggest an infection triggering the myocarditis.

The transient episodes of diplopia and the walking vertigo after surgery were probably related to embolic events. Two hypotheses may explain why patients with myxoma can present embolic events after administration of thrombolytic agents: the drug may cause lysis of an accumulated thrombus existent in the tumour surface or, in the presence of haemorrhagic areas and an abundant vascular supply, thrombolysis can increase haemorrhage and cause rupture of small fragments.⁸ In our case, the presence of cerebral microemboli suggested a high embolic potential of the mass, which prompted cardiac surgeons to perform urgent surgery.

Conclusion

The identification of the aetiology of CP remains a challenge, requiring clinical and technical expertise. The progress of cardiovascular imaging has improved diagnostic accuracy in numerous clinical scenarios. To the best of our knowledge, this is the first case reported in the applicable literature of a myxoma diagnosed during a myocarditis episode.

Lead author biography



António Fontes was born in the Azores, Portugal, in 1989. He received the MD degree from Faculty of Medicine of the University of Coimbra, Portugal in 2013 and afterwards started the specialization in cardiology. His research interest is mainly focused on arrhythmias, pathophysiology

of coronary artery disease, and multimodal cardiac imaging, in particular in ischaemic heart diseases and heart failure.

Supplementary material

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

Acknowledgements

We thank Doctor Vergilio Schneider (Hospital de Santo Espírito da Ilha Terceira).

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 image(s) and associated text has been obtained from the patient in line with COPE guidelines.

Conflict of interest: none declared.

Funding: none declared.

References

1. Rohacek M, Bertolotti A, Grutzmueller N, Simmen U, Marty H, Zimmermann H, et al. The challenge of triaging chest pain patients: the Bernese University Hospital experience. *Emerg Med Int* 2012;**2012**:975614.
2. Dec GW Jr, Waldman H, Southern J, Fallon JT, Hutter AM Jr, Palacios I. Viral myocarditis mimicking acute myocardial infarction. *J Am Coll Cardiol* 1992;**20**: 85–89.
3. Lam KY, Dickens P, Chan AC. Tumors of the heart. A 20-year experience with a review of 12,485 consecutive autopsies. *Arch Pathol Lab Med* 1993;**117**: 1027–1031.
4. Elbardissi AW, Dearani JA, Daly RC, Mullany CJ, Orszulak TA, Puga FJ, Schaff HV. Embolic potential of cardiac tumors and outcome after resection: a case-control study. *Stroke* 2009;**40**:156–162.
5. Panju AA, Hemmelgarn BR, Guyatt GH, Simel DL. Is this patient having a myocardial infarction? *JAMA* 1998;**280**:1256–1263.
6. Steger CM, Hager T, Ruttman E. Primary cardiac tumours: a single-center 41-year experience. *ISRN Cardiol* 2012;**2012**:906109.
7. Ferreira VM, Schulz-Menger J, Holmvang G, Kramer CM, Carbone I, Sechtem U, et al. Cardiovascular magnetic resonance in nonischemic myocardial inflammation: expert recommendations. *J Am Coll Cardiol* 2018;**72**:3158–3176.
8. Stefanou MI, Rath D, Stadler V, Richter H, Hennersdorf F, Lausberg HF, et al. Cardiac myxoma and cerebrovascular events: a retrospective cohort study. *Front Neurol* 2018;**9**:823.