ST-Segment Elevation Myocardial Infarction and Right Atrial Myxoma

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Abstract

Background   Cardiac myxoma is the most common primary cardiac tumor. Although benign, it can cause life-threatening complications due to embolization.

Case Presentation   We describe an ST-elevation myocardial infarction (STEMI) involving a giant right atrial myxoma and persisting foramen ovale (PFO) in a 64-year-old male patient and report on emergency percutaneous interventional therapy and subsequent cardiac surgery to remove the right atrial myxoma.

Conclusion   A right atrial myxoma, combined with a PFO, can cause a STEMI. Therefore, every acute coronary syndrome patient should undergo ultrafast exploratory emergency echocardiography to protect the physician from unpleasant surprises.

Background

Primary cardiac tumors are relatively rare and fortunately, 80% are benign. Of the latter, myxoma is the most common type (50% thereof).[1] Its incidence is under 0.2%; two-thirds of them are located in the left atrium (LA), and less than a third in the right atrium (RA).[2] There is evidence that myxomas on the heart's left side can embolize and cause serious complications. Myxomas on the heart's right side can also embolize, but they usually cause a pulmonary embolism.[2] However, a paradoxical embolism is only possible in the presence of a cardiac-septum defect. It can also cause embolization complications in the left circulation.

With this case, we describe an ST-segment elevation myocardial infarction (STEMI) possibly caused by embolism fragments from a giant RA myxoma.

Case Presentation

A 64-year-old male patient suffering from a new onset of angina and nausea since the previous day's evening was admitted to the emergency room at a primary-care hospital not equipped to perform a percutaneous coronary intervention (PCI). After initial noninvasive diagnostics (electrocardiogram, cardiac biomarkers), the patient was immediately transferred to a clinic with a cardiac catheter laboratory
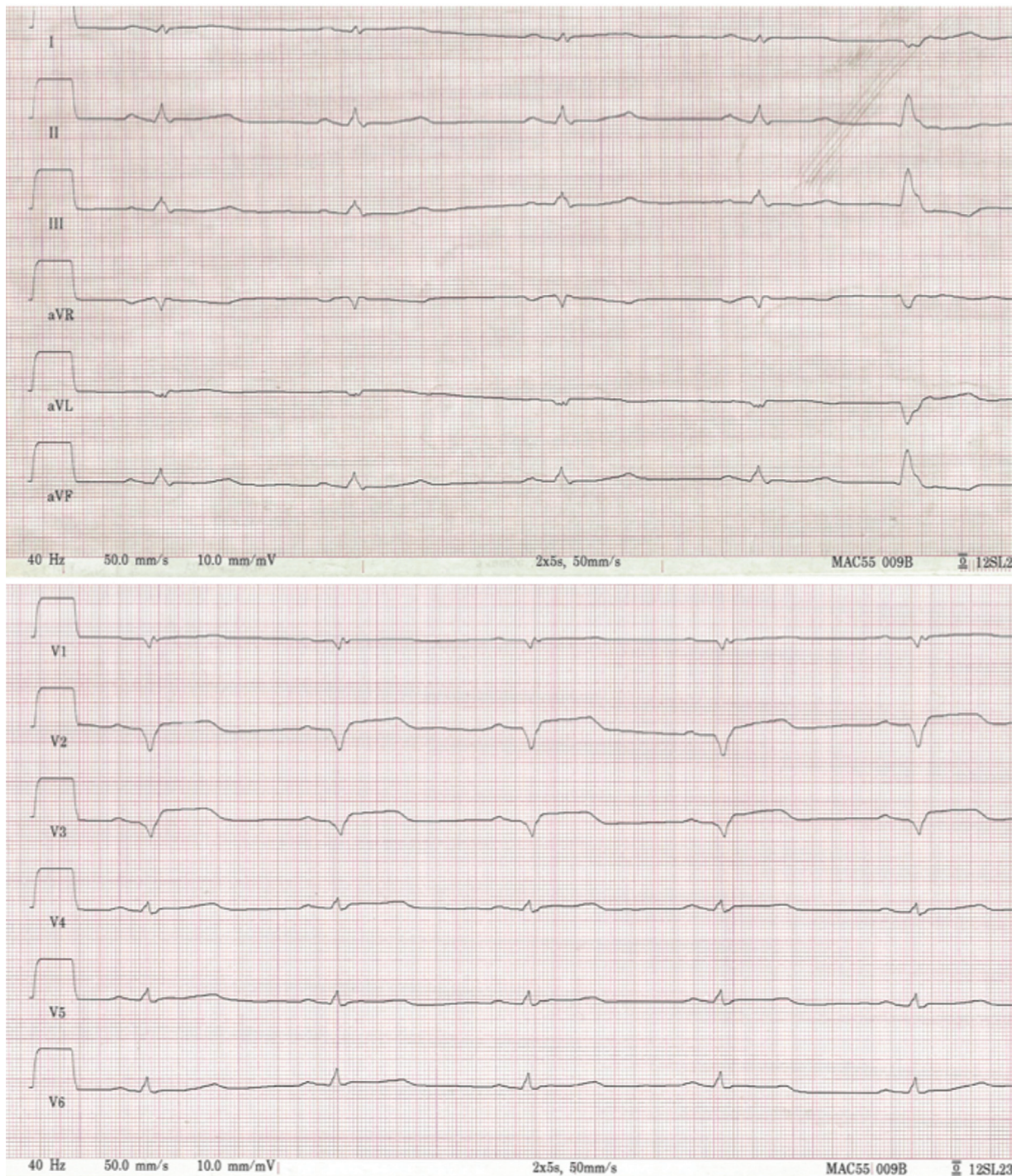
Fig. 1 Electrocardiogram.

because of ST-segment elevations (►**Fig. 1**) and highly elevated cardiac biomarkers (CK max. 61.22 µmol/s.l., CK-MB max. 4.82 µmol/s.l.). Because of the delay since his first symptoms and their persistence, the patient was immediately transferred from the stretcher to the catheter laboratory table. Coronary angiography revealed an occluded proximal left anterior descending (LAD) artery in segment 7 (►**Fig. 2A, B**) and moderate-to-severe stenosis of the right coronary artery in segment 3.

After the guidewire was first passed through the completely occluded LAD where the culprit lesion was located, the vessel was reopened almost completely and two drug-eluting stents (DESs) were implanted. The patient was then given dual antiplatelet therapy following guidelines.[3] Immediately after the emergency PCI, he underwent transthoracic echocardiography, which surprisingly revealed a giant RA mass measuring 40×50 mm prolapsing into the right ventricle (►**Fig. 3**). His left ventricular ejection fraction
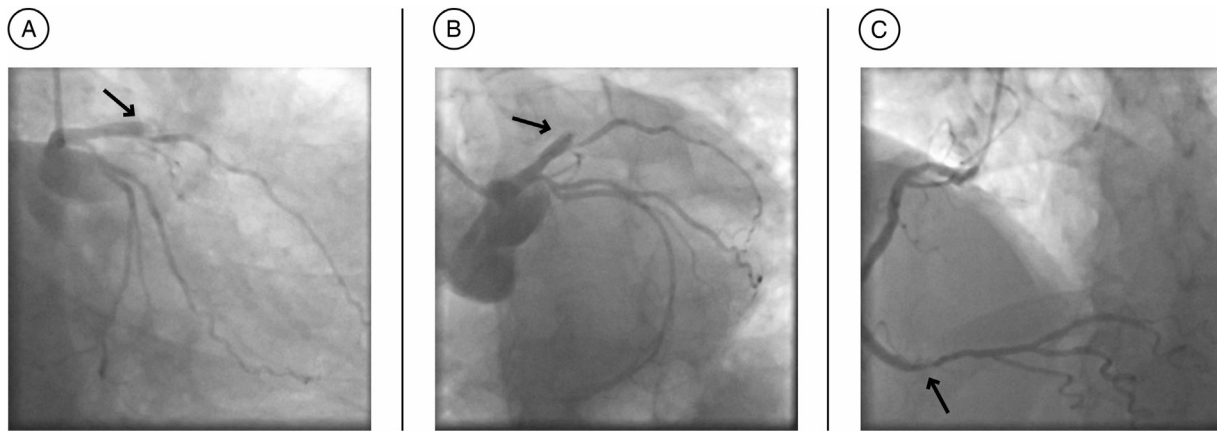
Fig. 2 Primary emergency percutaneous coronary intervention. (**A and B**) Left coronary artery with the arrow marking the total occlusion of the left anterior descending artery. (**C**) Right coronary artery with the arrow marking the high-grade stenosis in segment 3.
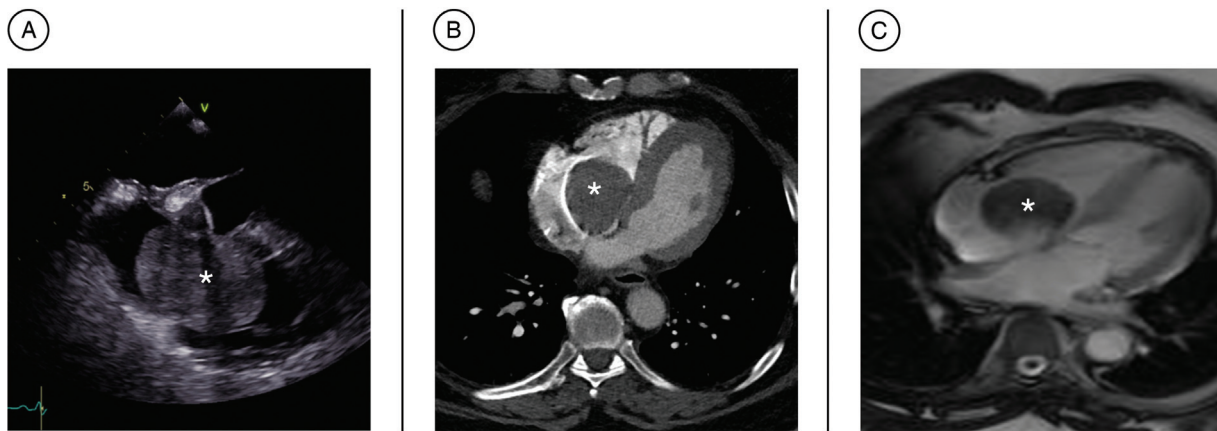

Fig. 3 Giant right atrial myxoma (*). (**A**) Transesophageal echocardiography. (**B**) Computed tomography scan. (**C**) Magnetic resonance tomography scan.

was severely reduced after the STEMI with apico- and ante-roseptal hypokinesia. He underwent transesophageal echo-cardiography to identify this intracardiac mass, confirming prior findings. Four days after the culprit lesion's primary PCI, implanting a DES eliminated the remaining stenosis in the right coronary artery's segment 3 (►**Fig. 4C**). Eleven days after the STEMI, the patient was transferred to our department for the resection of the RA's giant mass (occluding the tricuspid valve almost completely).

Our department took additional computed and magnetic resonance tomographs to improve surgical planning (►**Fig. 3A, B**). The following day, the patient underwent a minimally invasive technique in an usual manner via a right lateral mini-thoracotomy in the fifth intercostal space. The heart was accessed via the RA, where the giant tumor became immediately visible. The mass obstructed nearly the entire cavity. The tumor's macroscopic appearance revealed an ir-regular, gelatinous exterior of friable, soft consistency corre-sponding to a papillary myxoma confirmed later histopathologically (►**Fig. 5**). It was broad-based, anchored to the septum primum, and approximately 50 mm in diameter. In addition to the tumor, the patient also had a persistent

foramen ovale (PFO). The tumor was removed from the atrial myocardium, and then, the PFO could be closed.

A rethoracotomy complicated the patient's in-hospital postoperative course on the first postoperative day due to diffuse postoperative bleeding, postoperative cognitive dys-function, and pneumonia. He was discharged on postopera-tive day 23 from the regular ward to a rehabilitation facility. At the 1-year follow-up after surgery, he was clinically free of complaints. His echocardiographs revealed good left ventri-cular function (55%) with trivial mitral valve and mild tricus-pid valve regurgitation.

Discussion

Wherever it is located, embolization from a myxoma is a life-threatening complication. When located on the heart's right side, these tumors usually cause embolization in the lungs. However, a paradoxical embolization is possible in the presence of a PFO. In our case, fragments of a giant RA myxoma may have embolized and caused STEMI. To the best of our knowledge, there are no published case reports on such a scenario.
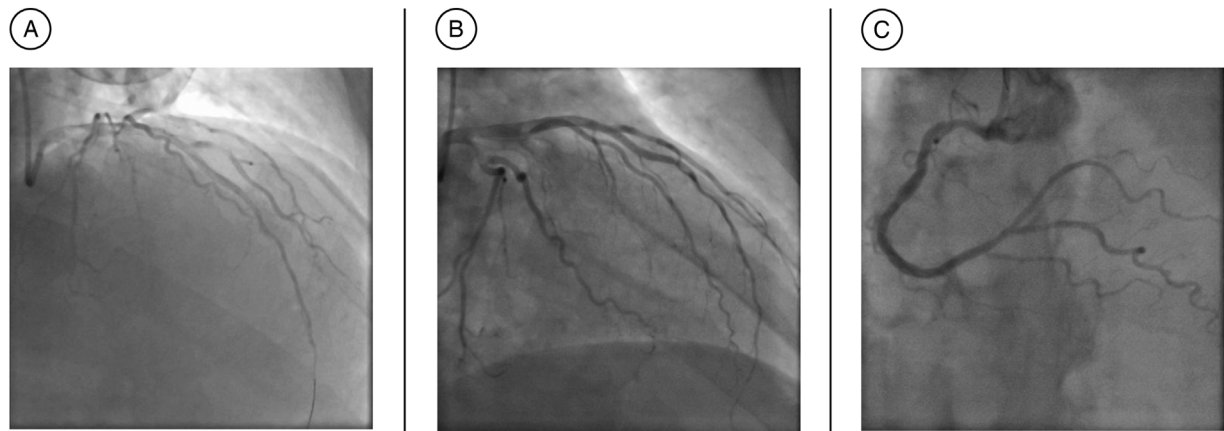
Fig. 4 (**A**) Initial left anterior descending artery's result after the first passage with the guidewire through the total occlusion and finally (**B**) after implanting two drug-eluting stents. (**C**) Final result of the second inspection of the right coronary artery's percutaneous coronary intervention.
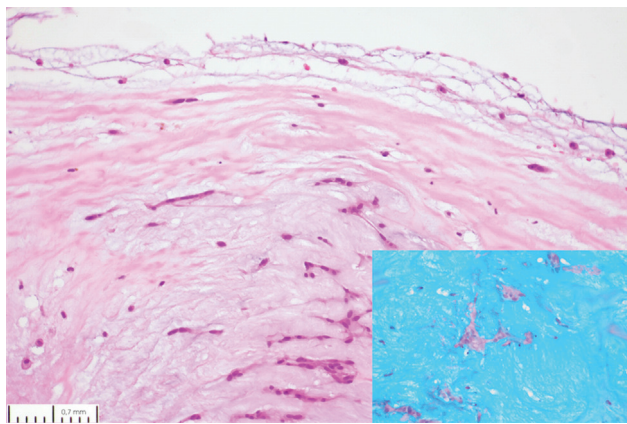

Fig. 5 Histopathological examination using light microscopy. Hematoxylin–eosin staining—inlay Alcian blue staining with periodic acid-Schiff reaction. Tumor with few cells and elements located subendothelial in loose, elongated structures, dominated by a myxoid matrix.

The probability that a myxoma will embolize is 30 to 40%.[4] Other reports have described embolization rates that differ markedly between an LA myxoma (45–60%) and RA myxoma (8–10%).[5] Therefore, we can assume that the numbers of undetected embolisms are certainly higher than those described. Furthermore, embolization from an LA myxoma into the coronary arteries has a reported probability of just 0.06%,[4] while various authors assume a more probable rate between 10 and 30% in patients with myxoma embolisms.[6–8] While all the published reports so far consistently refer to LA myxomas, paradoxical embolisms in the coronary arteries from an RA myxoma have not been described.[2,9] Similar to the paradoxical embolism of thrombotic material in deep vein thrombosis, a slight increase in right atrial pressure (e.g., Valsalva maneuvers) suffices to produce a short-term shunt reversal that can transport tumor tissue into the LA. Our patient's RA myxoma of papillary type (known as a predictor for embolization itself[10] with its irregular, gelatinous exterior and friable, soft consistency) was immediately adjacent to the concomitant PFO. The myxoma's immense size is poten-

tially why the PFO remained undetected on transesophageal echocardiography done immediately after the STEMI.

Myxoma-related myocardial infarctions (MRMIs) are extremely difficult to discriminate without echocardiographic evidence. However, there is often a substantial discrepancy between the extent of the infarction and angiographic images because stenoses and occlusions can only be documented angiographically in approximately 30% of cases or they disappear in early follow-up.[2,10] A stenosis from an acute MRMI can naturally resemble an ordinary total or subtotal occlusion of a coronary vessel. A recent literature search on the topic of myocardial infarction as a complication of an LA myxoma revealed that only in approximately 15% of cases were myxoma fragments obtainable for histopathological examination to support the diagnosis.[2] Braun et al. assume that myxoma fragments may be subject to further fragmentation, followed by distal dispersion of the myxomatous material. The histological composition of myxomas, consisting of an amorphous tumor mass containing glycosaminoglycans with no structural cellular organization, promotes the emboli's subsequent fragmentation.[2]

Additionally, substantial increases in cardiac biomarkers, also after spontaneous recanalization, suggest that reperfusion often occurs too late to preserve the heart's integrity.[2] In retrospect, our patient's culprit lesion behaved precisely like that of an MRMI.[2,10,11] The angiograph clearly showed how the total LAD occlusion disappeared after the guidewire's passage, leaving no residual stenosis. In addition, we observed no typical intracoronary "slow flow"—phenomenon, which is usually generally present after the fragmentation of a thrombotic lesion with the visible, fragmented material entering microvascular structures. We, therefore, believe that there is sufficient evidence in the patient we describe here (e.g., high cardiac enzymes, severely increased ST elevations, and strongly reduced left ventricular pump function despite the rapid reopening of the occluded artery) that this was an acute coronary syndrome (ACS) possibly caused by a paradoxical embolism from an RA myxoma.

If our patient had undergone echocardiography once before his catheter examination, his treatment course might

have been somewhat different. For the sake of fairness, we should mention that PCI is unquestionably the method of choice in STEMI, even when caused by a myxoma.[3] Although the guidelines no longer recommend thrombus aspiration,[3] the latter might have been another option under these circumstances, even knowing echocardiographically that these were myxoma fragments. Thus, without dual platelet therapy via acetylsalicylic acid and ticagrelor and without foreign material in the coronary arteries, our patient could have undergone a myxoma resection and coronary artery bypass surgery and would have carried a significantly lower risk of postoperative bleeding.

Conclusion

An STEMI resulting from paradoxical embolization from a giant RA myxoma into the coronary arteries is possible. Successful treatment entailing primary PCI followed by surgical tumor resection via minimally invasive cardiac surgery is feasible, although if the patient has already been on dual platelet therapy, their postoperative bleeding risk is high. An ultrafast emergency transthoracic echocardiograph taken for orientation before primary PCI in ACS may save the physician from unpleasant surprises before primary PCI.

Ethics Approval
Not applicable.

Consent for Publication
Oral informed consent was obtained from the patient to publish this case report and accompanying images.

Availability of Data and Materials
The anonymized data used to support the findings of this case report are available from the corresponding author upon request.

Authors' Contributions
M.V., T.G., T.B.A., and A.J.R. contributed to concept/design; M.V. and T.G. contributed to article drafting; A.J.R. and T.G. performed the surgery; M.V., T.G., T.B.A., and A.J.R. performed the critical revision of the article; M.V., T.G., T.B.A., and A.J.R. contributed to the approval of article; M.V. contributed to data collection.

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