

## CASE REPORT



## Intracardiac mass presenting as acute myocardial infarction

DIANA RUXANDRA HĂDĂREANU<sup>1)</sup>, MIHAELA CORINA BERCEANU<sup>1)</sup>, ROXANA DANIELA STROESCU<sup>1)</sup>, SEBASTIAN MILITARU<sup>1-3)</sup>, CONSTANTIN MILITARU<sup>1-3)</sup>, CĂLIN DINU HĂDĂREANU<sup>4)</sup>, VICTOR CORNEL RAICEA<sup>4,5)</sup>, NELU IRINEL ȘOȘEA<sup>4)</sup>, ELIAN OCTAVIAN BOLDU<sup>4)</sup>, OANA CRISTINA MUNTEANU MIREA<sup>1,2)</sup>, RĂZVAN ILIE RADU<sup>6)</sup>, IOANA ANDREEA GHEONEA<sup>7)</sup>, MIHAI MARIUS BOTEZAT<sup>8)</sup>, CRISTIAN MILITARU<sup>1,4)</sup>

<sup>1)</sup>Department of Cardiology, Clinical Emergency County Hospital, Craiova, Romania

<sup>2)</sup>Department of Cardiology, University of Medicine and Pharmacy of Craiova, Romania

<sup>3)</sup>Cardiomed Hospital, Craiova, Romania

<sup>4)</sup>Department of Cardiovascular Surgery, Clinical Emergency County Hospital, Craiova, Romania

<sup>5)</sup>Department of Cardiovascular Surgery, University of Medicine and Pharmacy of Craiova, Romania

<sup>6)</sup>Department of Interventional Cardiology, Prof. Dr. C. C. Iliescu Emergency Institute for Cardiovascular Diseases, Bucharest, Romania

<sup>7)</sup>Department of Medical Imaging, University of Medicine and Pharmacy of Craiova, Romania

<sup>8)</sup>Department of Neurology, Clinical Emergency County Hospital, Craiova, Romania

### Abstract

Cardiac tumors, although rare, present intricate diagnostic and therapeutic challenges, necessitating timely intervention for optimal patient outcomes. This case report focuses on a 65-year-old woman admitted with chest pain and loss of consciousness, ultimately diagnosed with a left ventricular cardiac myxoma. The patient's presentation mimicked acute coronary syndrome, highlighting the diagnostic complexity associated with cardiac tumors. Advanced imaging modalities, including transthoracic echocardiography, computed tomography, and invasive coronary angiography, played a pivotal role in characterizing the intracardiac mass. Histopathological (HP) examination, utilizing immunohistochemistry, confirmed the tumor as a cardiac myxoma. The patient management involved a multidisciplinary approach, leading to surgical resection of the mass and mitral valve replacement. The case underscores the importance of the HP confirmation in patients with cardiac masses, especially when multimodality cardiac imaging suggests various tumor types, simultaneously emphasizing the need for a comprehensive diagnostic approach that includes advanced imaging and histopathology to ensure an accurate diagnosis and tailored management of cardiac tumors.

**Keywords:** cardiac tumor, myxoma, acute myocardial infarction.

### Introduction

Cardiac tumors, although rare, present unique diagnostic and therapeutic challenges, necessitating timely diagnosis and appropriate interventions [1]. The incidence of cardiac tumors varies across different studies, with estimates ranging from 0.0017% to 0.28% in autopsy series [2], and up to 90% of primary cardiac tumors are benign [1]. However, with advances in imaging techniques and increased clinical awareness, the detection of cardiac tumors has seen an increase in recent years [3]. Cardiac tumors often mimic other cardiac pathologies, their clinical presentation largely depending on the tumor's location, size, and impact on cardiac function. Common symptoms include chest pain, dyspnea, palpitation, and syncope, which are often nonspecific and can be mistaken for coronary artery disease or heart failure [1, 4]. Additionally, patients may experience systemic embolic events resulting from tumor fragmentation and embolization [1, 5], or in the case of obstructive tumors, such as myxomas, symptoms may be accentuated during postural changes or exertion. Furthermore, malignant tumors

and metastases may manifest with weight loss, fever, and night sweats [1]. The diagnosis often necessitates a combination of advanced multi-modality cardiac imaging including transthoracic and transesophageal echocardiography [6], cardiac computed tomography (CCT) [7], cardiac magnetic resonance (CMR) [8], and positron emission tomography [9] for an accurate tumor characterization and localization [3]. In cases of suspected lymphomas or infiltrative diseases, endomyocardial biopsy can aid in identifying these tumor types [10]. However, a definitive diagnosis relies on histopathological (HP) examination, typically obtained through surgical resection of the mass. The management of cardiac tumors requires a multidisciplinary approach involving cardiologists, cardiothoracic surgeons, anesthesiologists, radiologists, oncologists, and pathologists. Although treatment strategies depend on various factors, the surgical resection remains the primary treatment for many benign tumors such as myxomas [11, 12], while malignant tumors often require a combination of surgical intervention and oncological treatment [4]. Finally, regular follow-up is essential to monitor and assess treatment efficacy.

## Aim

The aim of this case report was to present a particular case of cardiac myxoma and provide a comprehensive overview of its clinical presentation, diagnostic evaluation, and management, and emphasize the importance of a multidisciplinary approach for the correct diagnosis, and management of this patient.

## Case presentation

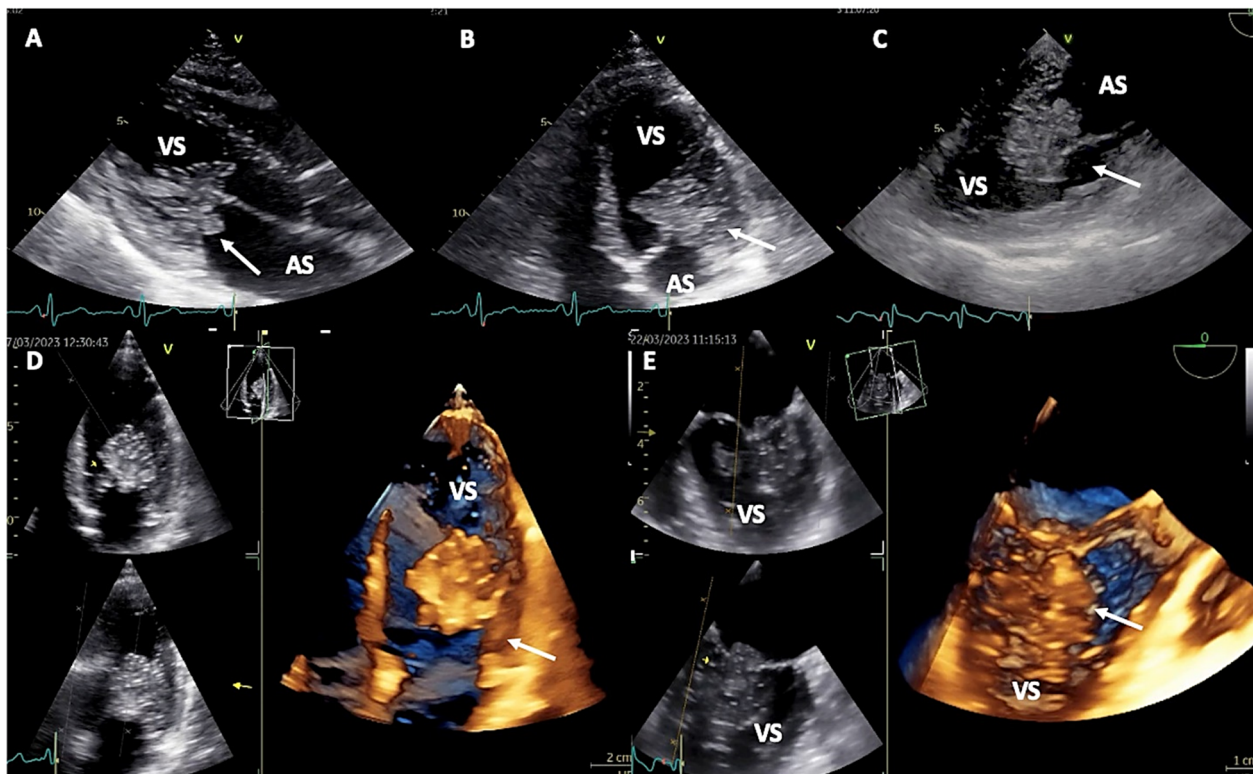
We present the case of a 65-year-old woman, who was admitted to the Emergency Department for constrictive chest pain irradiated to the jaw that lasted for approximately 20 minutes, followed by a short episode of loss of consciousness and associated craniofacial trauma. She had no known history of cardiac diseases, and she was not taking any medications at that moment.

Physical examination revealed preserved mental status and cognition, no signs of pulmonary or systemic venous congestion, and normal peripheral oxygen saturation. At cardiac auscultation she had normal heart sounds and no heart murmurs. Her heart rate was 100 beats per minute and the blood pressure 140/90 mmHg.

A 12-lead electrocardiography (ECG) was performed showing accelerated idioventricular rhythm, with a heart rate of 100 beats per minute, and ST segment depression of maximum 3 mm in leads V1–V3. Consequently, right-

sided and posterior chest leads were also recorded showing 1 mm ST segment elevation in leads V8 and V9. Cardiac enzymes were also elevated – high-sensitive cardiac troponin I levels were 1400 ng/L (upper limit of normality 24 ng/L), and creatine kinase muscle-brain fraction levels were 70 U/L (upper limit of normality 5 U/L). Accordingly, the diagnosis of acute posterior ST-elevation myocardial infarction (STEMI) was established.

At transthoracic echocardiography, the presence of a large, inhomogeneous intracardiac mass at the level of the left ventricle (LV), with irregular margins, attached at the level of the posterior mitral valve and posterior LV wall was observed (Figure 1, A–E). Due to the patient presentation with a syncopal episode preceded by chest pain that resulted in associated craniofacial trauma, and the presence of an intracardiac mass, CT was also performed in the Emergency Department showing no active intracranial bleeding and no extracardiac tumoral masses. The repeated 12-lead ECG after 15 minutes showed normal sinus rhythm and resolution of the ST segment elevation in the posterior chest leads. Accordingly, because of the presence of transient ST segment elevation corresponding to non-ST-elevation myocardial infarction (NSTEMI), and the presence of an intracardiac mass, the patient was admitted to the Cardiology Department for further investigations and treatment, and invasive coronary angiography evaluation was delayed.



**Figure 1** – Echocardiographic evaluation showing a large, hyperechoic cardiac mass (white arrows) with irregular borders, attached at the level of the left ventricular posterior wall and posterior mitral valve leaflet in transthoracic parasternal long axis view (A), and apical 4-chamber view (B), and transesophageal 2-chamber view (C). Three-dimensional rendering of the cardiac mass by transthoracic (D) and transesophageal echocardiography (E). AS: Left atrium; VS: Left ventricle.

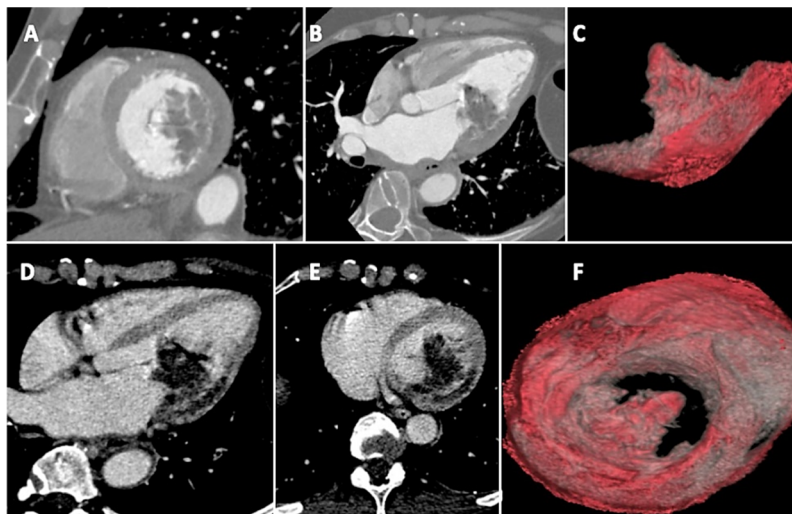
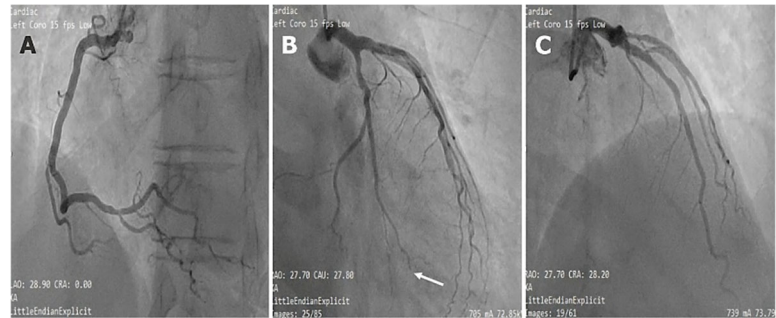
In the following days, further investigations were performed, including invasive coronary angiography that indicated an embolic occlusion of a distal branch of the obtuse marginal branch of the left circumflex coronary

artery, and no other associated coronary lesions (Figure 2, A–C). Transesophageal echocardiography (Figure 1, A–E) and CCT (Figure 3, A–F) confirmed the presence of the mass at the level of the LV, with a large base of implantation at

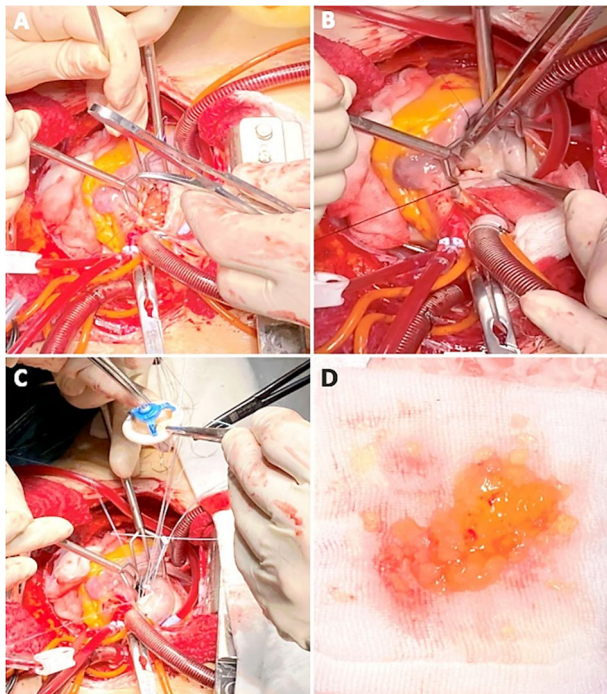
the level of the posterior LV wall, extending also towards the base of the posterior mitral valve leaflet. CMR was not available at that moment. The case was discussed in the heart team, and together with the patient we opted for

surgical resection of the cardiac mass, and surgical mitral valve replacement with a biological prosthetic valve (Figure 4, A–D) given the large dimensions of the tumor and the high embolic risk.

**Figure 2 – Invasive coronary angiography showing no significant coronary lesions at the level of the right coronary artery (A), and left anterior descending artery (C), and with an embolic occlusion of a distal branch of the obtuse marginal branch of the left circumflex coronary artery (B, white arrow).**



**Figure 3 – Cardiac computed tomography. Arterial phase in short axis (A) and long axis (B) showing the cardiac mass with irregular borders localized at the base of the left ventricle, on the lateral wall, and posterior mitral valve leaflet. Venous phase in short axis (D) and long axis (E) showing the cardiac mass with low initial contrast uptake. Three-dimensional rendering of the cardiac mass (C and F).**



**Figure 4 – Intraoperative images of the cardiac mass showing its relationship with the mitral valve (A and B), and the mitral valve replacement with a biological prosthesis (C). The macroscopic appearance of the tumor (D) with large dimensions, irregular borders, yellowish color, and areas of hemorrhage was suggestive of cardiac myxoma.**

Immediately after resection of the tumor formation, it was sent to the Laboratory of Pathological Anatomy, where it was placed in 10% formalin solution for 48 hours, for fixation and then it was embedded in paraffin, according to the classical histopathology protocol. With the microtome, sections were made through the tumor with a thickness of 4  $\mu$ m, which were then stained with Hematoxylin–Eosin (HE) and Goldner–Szekely (GS) trichrome. The microscopic study showed that the tumor was made up of elongated cells, with numerous extensions (cells like fibroblasts), collagen fibers and amorphous connective matrix. Also, rare inflammatory cells were identified (Figures 5–8).

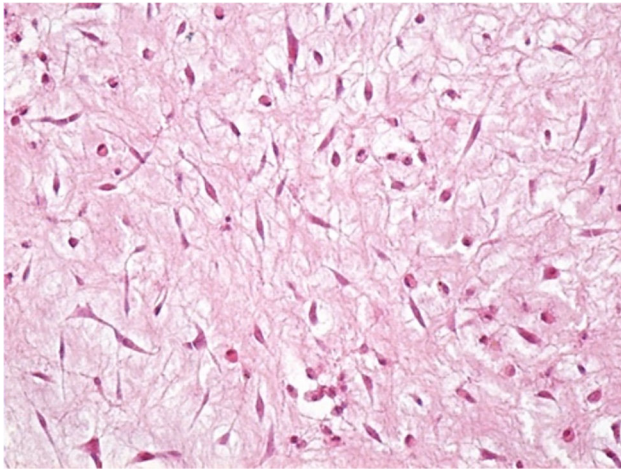
For the detailed study of the tumor cells, they used the immunohistochemistry technique with the following antibodies: anti-vimentin (monoclonal mouse anti-vimentin, clone V9, 1/50 dilution, Dako); anti-alpha-smooth muscle actin ( $\alpha$ -SMA) (monoclonal mouse anti-human SMA, clone 1A4, 1/100 dilution, Dako); anti-cluster of differentiation (CD)34 (monoclonal mouse anti-human CD34 Class II, clone QBEnd-10, 1/50 dilution, Dako); anti-CD3 (monoclonal mouse anti-human CD3, clone F7.2.38, 1/25 dilution, Dako); anti-CD20 (monoclonal mouse anti-human CD20cy, clone L26, 1/50 dilution, Dako); anti-CD68 (monoclonal anti-human CD68, clone KP1, 1/100 dilution, Dako); anti-tryptase [monoclonal mouse anti-human mast cell (MC) tryptase, clone AA1, 1/500 dilution, Dako].

The tumor cells were positive for the anti-vimentin antibodies, which proves that they are connective cells

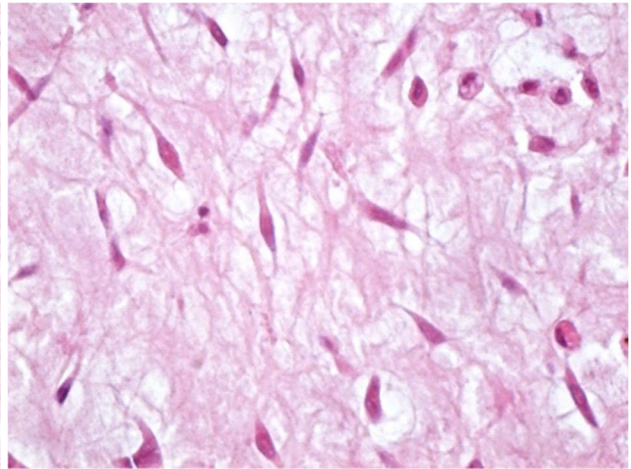
(Figure 9), positive for the anti- $\alpha$ -SMA antibody (Figure 10), aspects characteristic of activated fibroblasts (myofibroblasts) and intensely positive for the anti-CD34 antibody (Figure 11), which suggests that they originate in stem cells from the hematogenous marrow. Analysis of inflammatory cells

showed that macrophages and MCs were well represented (Figures 12 and 13), while T- and B-lymphocytes were very rare.

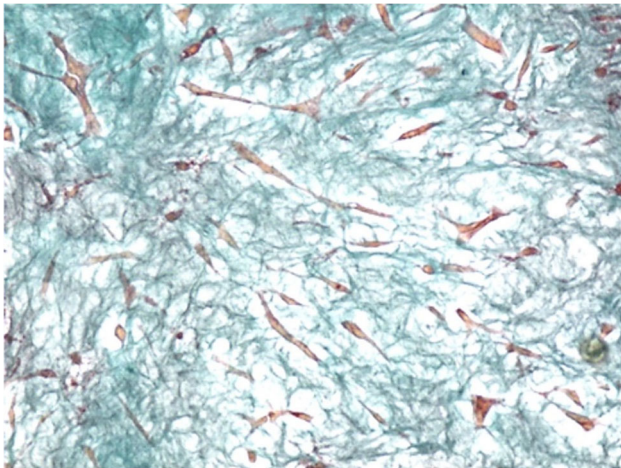
The final diagnosis of cardiac myxoma was established by the HP exam.



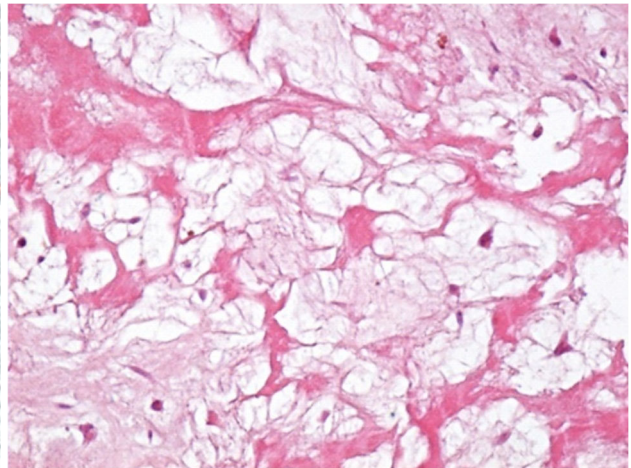
**Figure 5** – Overview of the tumor structure consisting of fusiform cells with multiple extensions, collagen fibers arranged in all directions, and amorphous conjunctival matrix. Hematoxylin–Eosin (HE) staining,  $\times 100$ .



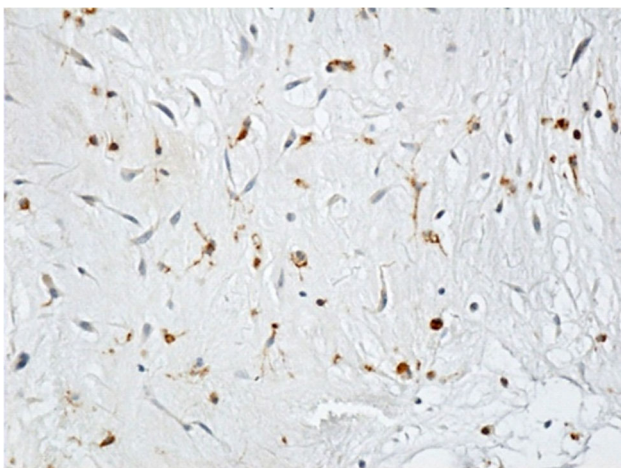
**Figure 6** – Detail image from the previous figure. Among the fusiform connective cells, there are rare round cells of inflammatory type. HE staining,  $\times 200$ .



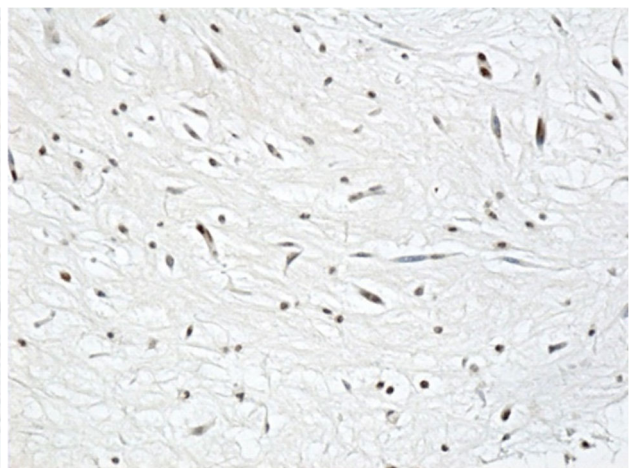
**Figure 7** – The tumor area formed by connective cells and collagen fibers with a plexiform arrangement. Goldner–Szekely (GS) trichrome staining,  $\times 100$ .



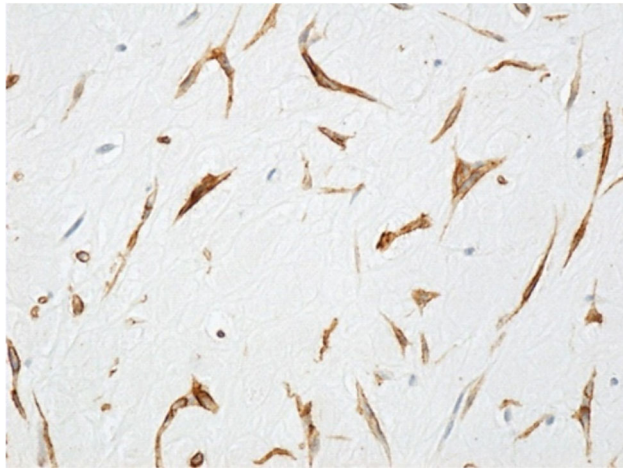
**Figure 8** – The tumor area formed mainly of amorphous conjunctival matrix. HE staining,  $\times 100$ .



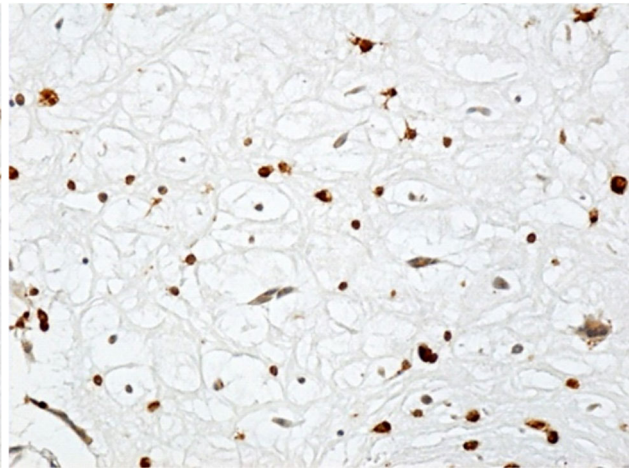
**Figure 9** – Positive reaction of tumor cells to the anti-vimentin antibody. Immunostaining with anti-vimentin antibody,  $\times 100$ .



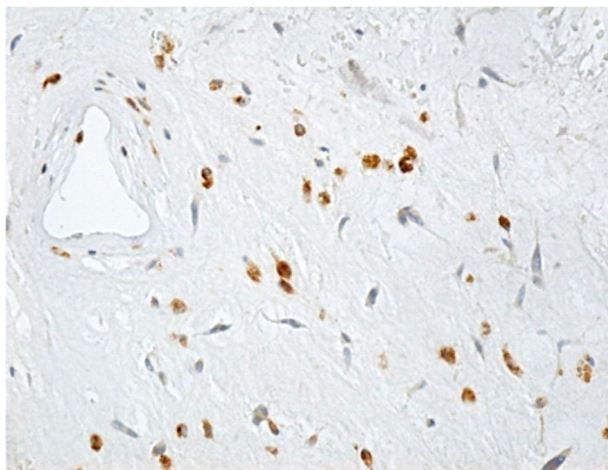
**Figure 10** – Tumor cells positive for the anti-alpha-smooth muscle actin ( $\alpha$ -SMA) antibody. Immunostaining with anti- $\alpha$ -SMA antibody,  $\times 100$ .



**Figure 11 – Tumor cells intensely positive for the anti-cluster of differentiation (CD)34 antibody. Immunostaining with anti-CD34 antibody, ×200.**



**Figure 12 – Numerous macrophages unevenly distributed in the tumor stroma. Immunostaining with anti-CD68 antibody, ×200.**



**Figure 13 – The area of the tumor infiltrated with numerous mast cells arranged mainly perivascularly. Immunostaining with anti-tryptase antibody, ×200.**

**Discussions**

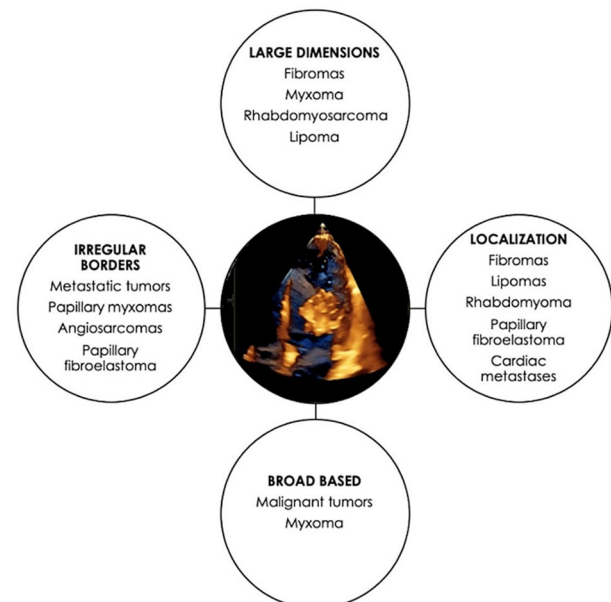
Cardiac tumors presenting as acute myocardial infarction (AMI) or acute coronary syndrome (ACS) are rare yet critical entities that pose diagnostic challenges and increased adverse outcomes. The symptoms are often indistinguishable from typical AMI or ACS, and timely diagnosis and differentiation of cardiac tumor embolism and secondary AMI from conventional atherosclerotic causes are essential for their appropriate management [13]. Advanced imaging techniques as presented in our case play a crucial role in establishing the accurate diagnosis and guiding the therapeutic decisions. On one hand, the imaging features of different types of cardiac tumors can vary significantly, and on the other hand, there are several imaging characteristics suggestive of malignancy (Table 1). The imaging features might be more indicative of a certain type of cardiac tumor. In our case, however, the imaging features of the tumor indicate several types of cardiac tumors (Figure 14). The final HP diagnosis was of cardiac myxoma.

Cardiac myxomas are the most frequent primary cardiac tumors [14], and they typically appear as well-defined,

**Table 1 – Imaging features suggestive of malignancy. Written in *italics> are the CCT characteristics suggestive of malignancy found in our patient***

Echocardiography	CCT	CMR
Localization at the level of right heart chambers	<i>Large dimensions &gt;5 cm</i>	Large dimensions
Local invasion	Intramural localization	Localization at the level of right heart chambers
Pericardial effusion	<i>Sessile</i>	Irregular borders
	<i>Irregular borders</i>	Heterogeneous appearance on T1- and T2-weighted images
	Calcifications	Pericardial effusion
	Pericardial effusion	

CCT: Cardiac computed tomography; CMR: Cardiac magnetic resonance.



**Figure 14 – Imaging features of the cardiac mass suggestive of several tumor types.**

pedunculated masses attached to the endocardium, often in the left atrium near the *fossa ovalis* [1]. At echocardiography, although having a variable appearance, ranging from solid to cystic, multilobulated or polypoid, they typically have a

characteristic swinging motion [15, 16]. At CCT, myxomas typically show low attenuation values consistent with soft tissue on non-contrast scans. Post-contrast scans can reveal heterogeneous enhancement due to the vascular nature of the tumor and might help differentiate myxomas from other cardiac masses, sometimes with calcifications [17]. However, CMR is considered the “gold standard” imaging technique for the detailed evaluation of cardiac tumors. Myxomas are usually heterogeneous on both T1- and T2-weighted images [18]. On cine sequences they are hyperintense in comparison to the myocardium and hypointense when compared to the blood [19]. On T1-weighted scans, myxomas generally appear as low-to-intermediate signal intensity masses due to their mucopolysaccharide content. On T2-weighted images, they have high signal intensity, emphasizing their gelatinous consistency. On late Gadolinium enhancement sequences, they have heterogeneous enhancement [19]. The management of cardiac myxomas involves a multidisciplinary approach, and surgical resection remains the cornerstone of therapy for cardiac myxomas. With complete excision, the surgical excision results in a low recurrence rate. This approach was adopted in our case, with the excised tumor tissue undergoing HP examination to confirm the diagnosis of cardiac myxoma and rule out malignancy. The HP evaluation also helps to identify any unusual features that may influence post-operative management [20]. Furthermore, in patients with familial or syndromic forms of cardiac myxomas, genetic counseling and screening for related genetic conditions, such as Carney complex, may be recommended to identify potential family members at risk and provide appropriate surveillance and management. Finally, although cardiac myxomas generally have excellent prognosis following complete surgical resection [11], regular follow-up is essential after the surgical removal of a cardiac myxomas, because 10–15% of cases recur, usually at the same site [21], some studies recommending lifelong follow-ups [11].

## ✉ Conclusions

The peculiarity of this case is the incidental diagnosis of a LV cardiac myxoma in a patient without known cardiac history that presents to the Emergency Department with NSTEMI. Furthermore, while an integrative approach plays a pivotal role for the diagnosis and management of cardiac tumors, surgical resection remains the cornerstone treatment in these patients. Finally, our case highlights the importance of the HP examination for the definitive diagnosis of cardiac masses, especially in patients in whom the multimodality cardiac imaging features reflect are suggestive for several different tumor types.

## Conflict of interests

The authors declare that they have no conflict of interests.

## Acknowledgments

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partnership with R&D institutes and universities, aimed at innovating processes and products in economic sectors with growth potential.

## Authors' contribution

Diana Ruxandra Hădăreanu and Mihaela Corina Berceanu equally contributed to the paper.

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### **Corresponding authors**

Sebastian Militaru, Assistant Professor, MD, PhD, Department of Cardiology, University of Medicine and Pharmacy of Craiova, 2 Petru Rareș Street, 200349 Craiova, Romania; Phone +40724–423 227, e-mail: sebastian.militaru@umfcv.ro

Victor Cornel Raicea, Lecturer, MD, PhD, Department of Cardiovascular Surgery, University of Medicine and Pharmacy of Craiova, 2 Petru Rareș Street, 200349 Craiova, Romania; Phone +40740–026 362, e-mail: dr.raicea.victor@gmail.com

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