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CASE REPORT

Cardiac Myxoma Post-Transseptal Ablation: Coincidence or Causation?

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Abstract

Background: Cardiac myxomas are benign cardiac neoplasms usually found solitarily located within a single cardiac chamber, most commonly in the left atrium. With no established cause, they are often thought to occur spontaneously with no particular genetic cause or external incitement. While it has been explored, there is no universally accepted correlation with myxoma formation following a cardiac ablation procedure. We propose the theory of an iatrogenic causation of cardiac myxoma formation, likely incited by the local tissue injury during the transseptal puncture of the interatrial wall. **Case Summary:** We present the case of a 62-year-old male with a medical history significant for Atrial Fibrillation for which he underwent a transseptal ablation procedure, during which a puncture is made through the interatrial septum. Transthoracic and transesophageal echocardiograms obtained prior to the procedure did not reveal any intracardiac masses or abnormalities. Two-years post-procedure, during an inpatient hospitalization for a suspected asthma exacerbation, a new transthoracic echocardiogram demonstrated a pedunculated mass within the left atrium.

Take Home Message(s)

- Cardiac myxomas are the most common benign cardiac neoplasms, often thought to be of sporadic origin, however are in part influenced by familial genetic disorders
- Cardiac myxomas may iatrogenically develop through a metaplastic process triggered by tissue injury during transseptal ablation procedures

Keywords: Coronary Artery Disease (CAD).

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Introduction:

Cardiac myxomas are benign neoplasms often found in persons aged thirty to sixty years age, often solitarily discovered, and less commonly multi-chambered. The primary thought process through which myxomas are understood is of a sporadic and unpredictable nature. Hereditary familial disorders with cardiac myxoma involvement are less frequently encountered, but are otherwise medically understood. We present a case of an adult male in whom a left atrial mass consistent with a myxoma was discovered during an inpatient hospitalization, when only approximately four years prior he had no presence of an intracardiac mass. The acknowledged intervention the patient had received was a percutaneous radiofrequency ablation for atrial fibrillation, which we propose was an iatrogenic consequence.

Case Presentation:

History of Presentation

A 62-year-old male presented to the emergency department complaining of shortness of breath and worsening dry cough for a 2-week duration. The dyspnea was reportedly worsened on exertion and relieved with rest. He had four prior visits to the ER within the past 12-months with similar symptoms, during which he was treated with inhaled bronchodilators and oral steroid medications. He denied any recent illness, fevers, chest pain, or palpitations. On this visit, physical examination revealed mild generalized wheezing in all lung zones. There were no signs of vascular congestion, lower extremity edema, or jugular venous distention on physical examination.

Past Medical History

He reported a long-standing history of essential hypertension, asthma diagnosed in childhood, a history of tobacco use (quit in 2011), as well as persistent Atrial Fibrillation for which he received an interatrial trans-septal ablation. He was subsequently prescribed Metoprolol Succinate 100 mg oral daily and Apixaban 5 mg oral twice daily for maintenance therapy.

Investigations

Initial Inpatient Work-up: Vital Signs: BP 132/74 mmHg, pulse 73 bpm, a febrile, SpO_2 96% on RA. High sensitivity Troponin <2.5 (x3), BNP was 78 (normal), Arterial blood gas: normal.

Serial EKGs: No ST-segment or T wave abnormalities (Figure 1) Chest X-ray: Small right-sided pleural effusion, with an increased enhancement of the pulmonary vasculature (Figure 2).

A transthoracic echocardiogram (TTE) revealed an estimated ejection fraction (EF) of 60%, with no diastolic dysfunction and no associated valvular abnormalities. There was also a 1.7 x 1.7 cm mass visualized in the left atrium (Figure 3).

A transesophageal echocardiogram (TEE) revealed a similarly estimated ejection fraction, and confirmed the presence of an approximately 1.7 x 1.9cm mass, seemingly attached to the interatrial septum at its base (Figure 4A & B). Along with the standard TEE, 3-dimensional (3D) imaging was obtained (Figure 5).

The patient then underwent right and left heart catheterization. Angiography revealed non-obstructive coronary artery disease, a normal estimated EF. Interestingly, small branching vessels were visualized, likely vascularizing a structure (Figure 6).

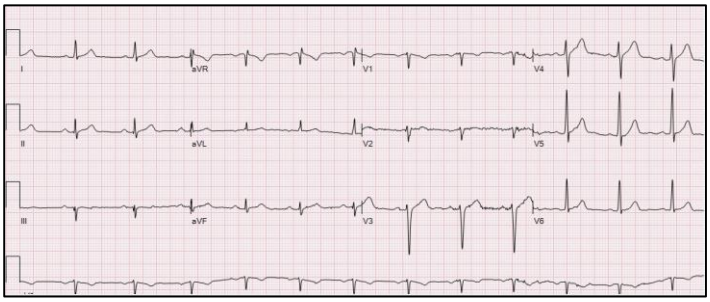

Figure 1: This EKG demonstrates a normal sinus rhythm with no significant ST abnormalities.

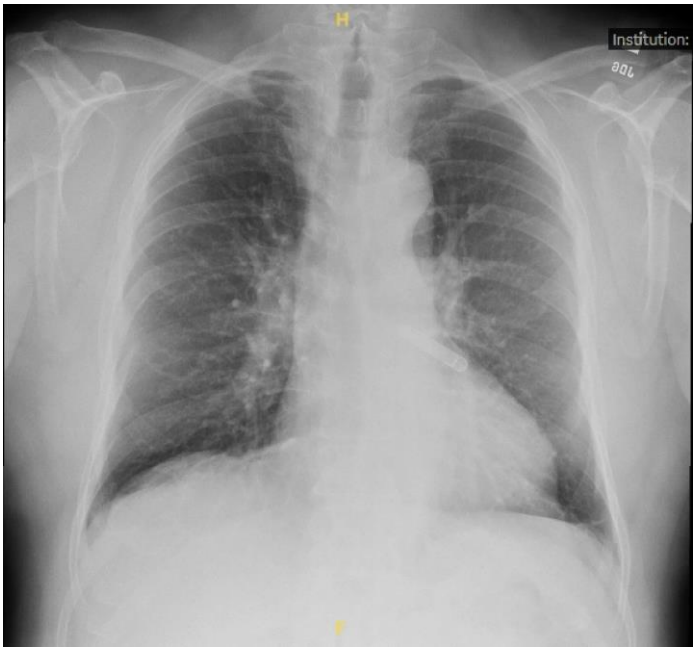

Figure 2: This chest radiograph depicts a small right-sided pleural effusion, and an increased prominence of the pulmonary vasculature. A loop recorder is visible.

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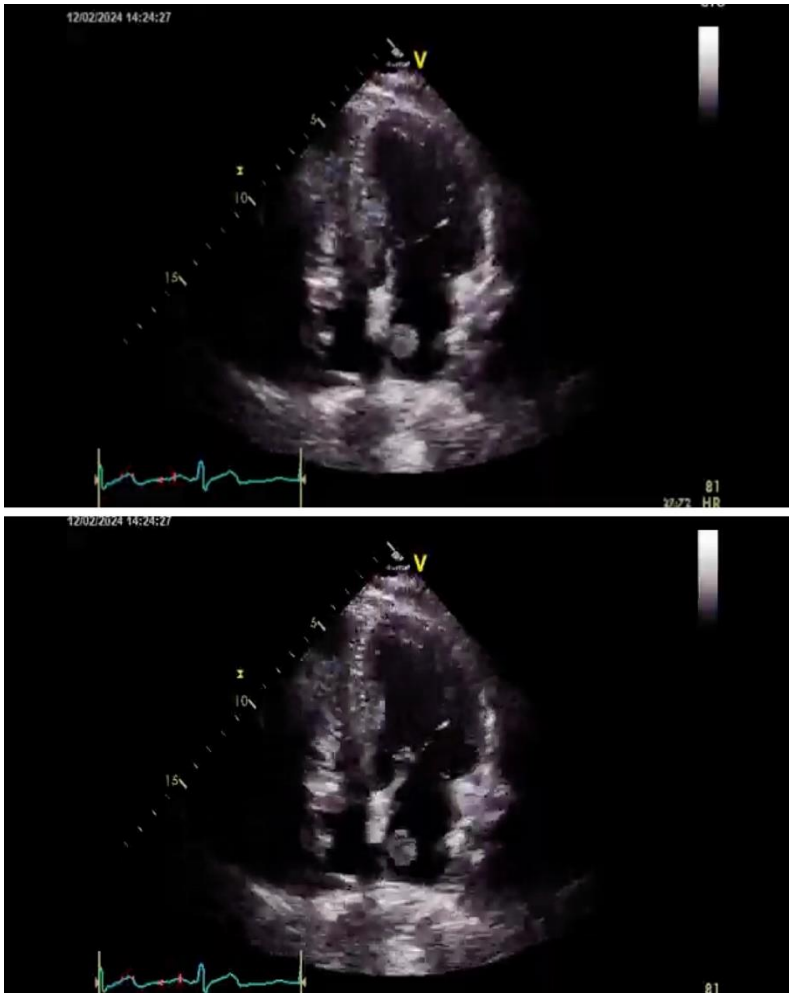

Figure 3: This is a transthoracic image of an apical 4-chamber view, depicting the left atrial mass measuring approximately 1.9 x 1.9cm.

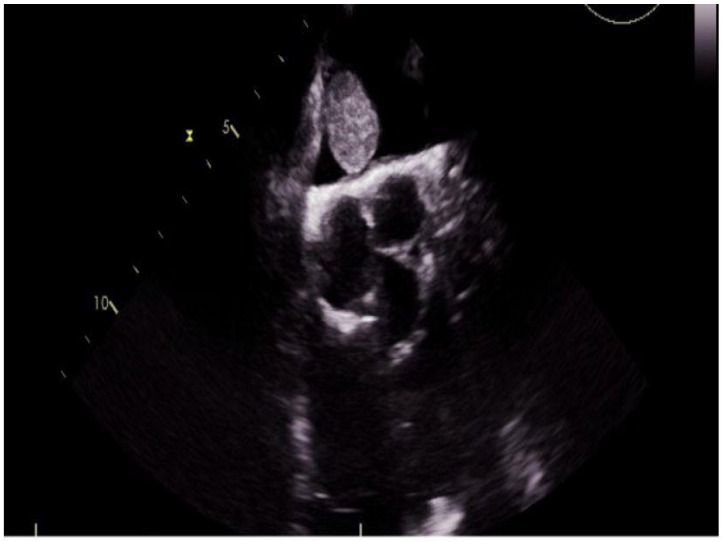

Figure 4A

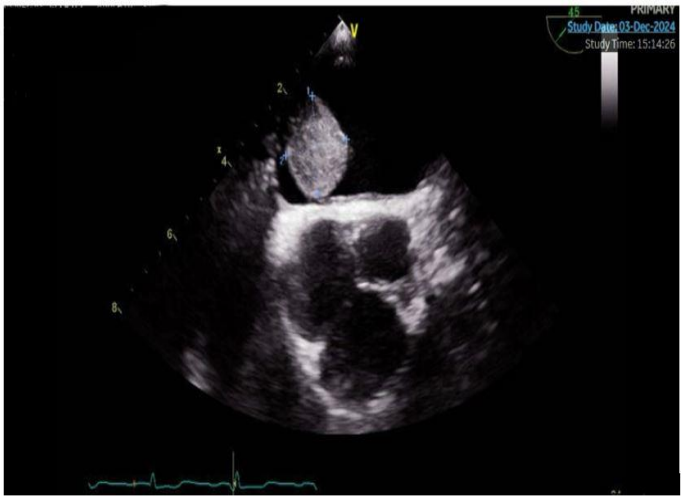

Figure 4B: This transesophageal view depicts the left atrial mass, measuring approximately 1.7 x 1.9cm.

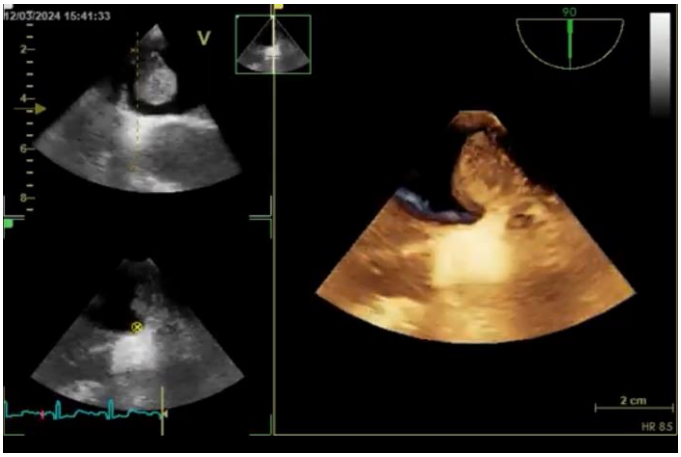

Figure 5: This is a 3-D image obtained during TEE, depicting the left atrial mass, once again measuring approximately 1.7 x 1.9cm.

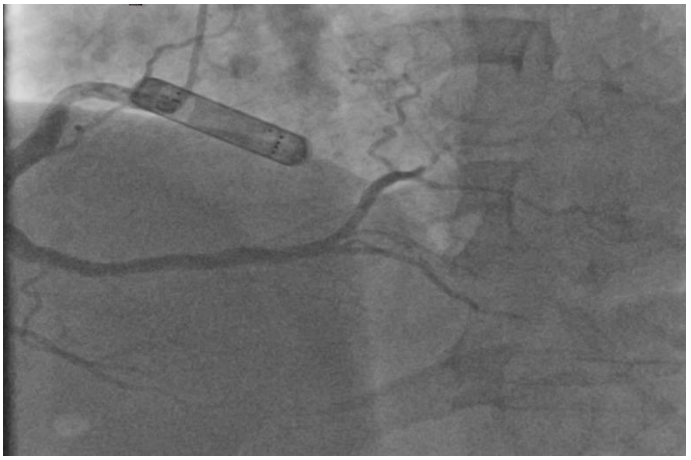

Figure 6: This image was obtained on angiography displaying the small branching vessels stemming from the Left Anterior Descending (LAD) artery, seemingly vascularizing a structure.

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Prior to admission, the patient had obtained a standard TTE in 2019. This revealed an EF of 60%, with no presence of a cardiac mass. He had also obtained a computed tomography (CT) imaging of the chest as part of a lung cancer screening method in September of 2021. This imaging revealed calcified coronary arteries, with no visible intracardiac mass.

Management:

The patient was admitted to the inpatient telemetry unit for treatment of a likely asthma exacerbation, as well as for further cardiac evaluation. Given that all echocardiographic findings thus far were consistent with a cardiac mass, most likely a myxoma, the patient was scheduled to receive a cardiac catheterization to evaluate for possible concomitant coronary artery disease (CAD).

Angiography showed non-obstructive CAD, with a normal estimated EF, with visualization of small branching vessels seemingly supplying a structure, further supporting the diagnosis of a cardiac myxoma. The patient was referred to a capable facility for outpatient evaluation for cardiothoracic surgery.

Discussion:

Cardiac myxomas are rarely occurring benign intracardiac masses with an estimated incidence of 0.5 per million per year, and an estimated prevalence of 0.03% of the general population [5,11]. Despite its relative rarity, cardiac myxomas account for up to 50-80% of all diagnosed cardiac tumors [5]. They are most predominantly observed in the left atrium, often with involvement of the interatrial septum, but are very dynamic in that they may potentially develop in any cardiac chamber, or even originate from the valves [10,11]. Myxomas are encountered in the right atrium at a rate of 20%, in the right or left ventricles at approximately 5%, or remarkably rarely discovered bi-atrially [11,17].

Myxomas are most frequently seen in persons aged thirty to sixty years of age, and appear to be far more common in women than men at an estimated ratio of 3:1 [17-19]. They are thought to originate in a sporadic or familial pattern, with the sporadic type at a proposed 95% of all diagnosed cases [5,18]. Sporadically formed myxomas are often isolated to the left atrium, while familial myxomas are inherited in an autosomal dominant fashion and have the potential to occur in clusters [18]. Carney Complex (CNC), a familial disorder, are shown to produce myxomas at a younger age, more commonly in men, and have a high recurrence rate in variable locations [18,21].

Histology & Gross Examination

The true histopathologic nature of myxoma formation remains poorly understood, however a widely accepted theory is the reactivation of genes encoding for primitive pluripotent mesenchymal cells of cardiac precursor tissue [5]. These are often referred to in literature as ‘lepidic’ or ‘myxoma’ cells, adherent to the endocardial surface without local tissue invasion, cells of which mimic the appearance of immature mesenchymal cells [5]. Histological examination of a typical cardiac myxoma is likely to reveal irregularly nucleated spindle and stellate cells in a loose myxoid stroma, or less commonly, acinar glands lined with columnar and goblet cells [7,12]. Cells are typically structured in nests around capillaries owing to the highly vascularized character of myxomas [5].

Often found solitarily located and suspended by a stalk, myxomas are varied in their appearance. Masses can have a smooth and uniform surface encircled in a layer of endothelium, or alternatively be villiform and papillary in nature. The latter type of myxomas are more likely to display more thromboembolic tendencies, likely due to its friable, adherent surface [9,10]. Masses are soft and gelatinous, with a potential friability if manipulated. A cross-sectional view is likely to reveal a pale grey, with some masses displaying areas of calcification, hemorrhage, or necrosis [14].

Physical Examination

Due to the unique and heterogeneous nature of cardiac myxomas, abnormal physical findings may not always be appreciated, and may go largely undetected unless further workup is undergone. When discovered, the most commonly encountered physical finding is a systolic murmur, followed by a diastolic murmur, likely dependent on body position and the specific valvular obstruction produced [14]. A highly characteristic low-pitched tumor ‘plop’ is an early diastolic murmur that may be appreciated after the S2, produced by the force of the myxoma against the endocardial wall [5]. Jugular venous distention may occur in combination with a prominent ‘A wave’ on EKG with right atrial outflow obstruction [5].

Clinical Presentation

The clinical manifestations of a typical cardiac myxoma can be defined by a classic triad, of which intracardiac outflow obstruction, constitutional symptoms, and embolization are defined [5,9,20]. Although benign on a cellular level, myxomas are considered to be ‘functionally malignant’ in the complications that they may present as. Embolic phenomena are particularly hazardous adverse effects of myxomas that occur in approximately 30-40% of the affected population [5,18]. This is due to direct fragmentation of tumor cells, or thrombi that form on the surface of the mass. Papillary type myxomas are more vulnerable to embolization due to the fragility and friability of their surfaces [5,17]. Embolisation stemming from left-sided myxomas are likely to result in stroke, while right

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atrial emboli may strain pulmonary perfusion and ultimately cause pulmonary hypertension [17,25].

Cardiac symptoms are frequently due to intracardiac outflow obstruction, and predominantly determined by size and location of the myxoma, experienced in up to 70% of cases [26]. Mitral or tricuspid valve pseudo-obstruction is a common complication of large pedunculated myxomas when located in the atria [5,9]. Myxomas located in the right atrium may present symptoms of right heart failure with peripheral edema, hepatomegaly, or ascites [17]. Left-sided myxomas may manifest with dyspnea, orthopnea, syncope, or dizziness [17,25]. Ventricular myxomas may reduce cardiac output by essentially mimicking pulmonary or aortic stenosis, and may similarly result in palpitations, syncope, or even sudden death [8,26].

Constitutional symptoms such as fevers, malaise, arthralgias, or generalized weight loss are commonly associated with cardiac myxomas, attributed to the release of cytokines such as Interleukin 6 and Endothelin [5,17]. This is frequently seen with right-sided rather than left, and larger sized myxomas [5]. Laboratory findings may reflect an inflammatory state with anemia, elevated white cell count, platelet count, ESR, or globulin levels [17].

Diagnosis:

Diagnostic Methods

Echocardiography is the crucial modality of which myxomas are to be evaluated. Transthoracic echocardiography (TTE) is often primarily performed in order to detect the presence of a mass, and is relatively simple and practical to perform [5,9].

Transesophageal echocardiography (TEE) is often performed after and has at times proven superior to standard TTE imaging, with an increased sensitivity and specificity for identifying the mass [5,8,9]. Both modalities are useful in visualizing the location, size, shape, and surrounding structures of the myxoma, with a TEE more likely to capture smaller or multi-chambered myxomas [9,17].

Echocardiography with contrast may be performed in order to distinguish between a thrombus and a mass if the diagnosis is unclear, with a myxoma likely to display enhancement with contrast administration due to its vascularity [5,9]. 3-dimensional (3D) imaging may be used as an adjunct to regular TTE or TEE imaging to enhance tissue characteristics [9,13].

Cardiac Computed Tomography (CT) may illustrate the presence of an intracardiac mass, however is less reliable in its ability to differentiate between a myxoma and a thrombus, even with the utilization of intravenous contrast [5,9,17]. CT may be used to identify the presence of calcification within the myxoma [5,9].

Cardiac magnetic resonance imaging (MRI) is superior in that it produces more detailed imaging, with a higher probability of detecting myxoma size, shape, and morphology. Tissue-like characteristics of the myxoma may also aid in distinguishing between a myxoma and a thrombus [5,9,13].

Chest radiography is an adjunctive method in evaluating myxomas, more so for the supplementary information that it may provide. Evidence of pulmonary hypertension with an increase in pulmonary prominence of vascularity may be present [9,13]. Radiographs may also depict relative cardiomegaly with or without specific chamber enlargement [17].

Electrocardiogram (EKG) findings may be normal, or display non-specific S or T wave abnormalities. Evidence of atrial or ventricular enlargement may be present, depending on the location and degree of associated complications of which the myxoma causes [5,15,19].

Treatment:

Definitive treatment and diagnosis of a cardiac myxoma would be with surgical excision and histological analysis of the offending mass [5,9]. Excision of the tumor with a median sternotomy followed by simple excision and pedicle removal is the common technique with which surgery is performed [27,28]. This entails the complete dissection and removal of the mass, with precision not to leave residual tissue that may be retained within the endocardium, or be disseminated into the system [9,27]. Iatrogenic valvular complications, more typically valvular regurgitation, may occur as a result of tumor manipulation and removal [27,28,29]. Atrial arrhythmias such as atrial fibrillation are also frequently known to occur following surgery [27,29].

Pathogenesis:

There are multiple proposed theories for cardiac myxoma formation. The neoplastic theory proposes a hereditary explanation for myxoma formation, often as a result of a genetic malformation leading to Carney's Complex (CNC) [18,21]. This malformation occurs in an X-linked dominant fashion due to a mutation in the protein kinase A Type 1-alpha regulatory subunit gene (PRKAR1A). This dysregulated pathway triggers unchecked cellular proliferation and promotes tumor formation. Similarly, to sporadic myxoma formation, dysregulation of the Cyclic-AMP (CAMP) pathway has been isolated in CNC myxoma growth [18,21]. Myxomas that develop under this circumstance are more likely to recur if removed, and may

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develop in multiparities when compared to sporadic formation [18].

The inflammatory theory proposes that individuals with comorbid illness or autoimmune conditions that produce a chronic systemic inflammatory state may trigger myxoma growth [22]. The inflammatory environment may stimulate localized cellular proliferation and angiogenesis, prompting growth of and sustaining a myxoma [22]. This conjecture may be supported by the fact that cardiac myxomas are often found to produce proinflammatory markers and cytokines such as IL-6, leading to the constitutional symptoms individuals may experience [23].

The thrombotic theory posits that myxomas originate from a blood clot or thrombus formed in a pocket of a cardiac chamber that ultimately undergoes dysplastic changes [23]. Endocardial tissue may develop over a thrombus that remains in situ over time. Prolonged mechanical stress, strained perfusion, and inflammatory characteristics may induce cellular transformation of the endothelial cells, and abnormal tissue proliferation [23]. That myxomas are often discovered in lowly perfused areas, such as near the interatrial septum, is a potential supporting factor toward this theory. The thromboembolic tendencies of cardiac myxomas may offer additional credence to a thrombotic origination of cardiac myxomas [23].

The dysembryoplastic theory postulates that remnants of dormant embryonic tissue, more specifically pluripotent mesenchymal cells retained within the endocardium, undergo differentiation into what eventually becomes a cardiac myxoma [5,9,23]. A potential supporting factor toward this theory is that myxomas are often found within the left atrium near the fossa ovalis, an embryonic remnant of the foramen ovale [22]. This theory may also offer an explanation for the dynamicity of myxoma tissue that can be gelatinous in nature, as the retained primordial cells possess the capacity to differentiate into multiple different cellular types [9,23].

The germ-cell line theory reinforces the idea of a primitive origin of cardiac myxomas, supported by the various genetic markers expressed by myxoma cells. Stem cell markers such as C-kit and CD34, or growth factors such as TGF-beta and PDGF may stimulate myofibroblast proliferation and subsequent myxoma formation [23,30]. A subset of myxomas have also expressed oncogenes, chemokines, interleukins, and various other growth related proteins such as MMP-1 and VEGF, all of which may play a role in tumorigenesis [23,24].

Of the multiple proposed theories of cardiac myxoma formation, the metaplastic theory is most consistent with origination following transseptal puncture, as exemplified by our case presentation. This theory posits that a metaplastic process is triggered by the tissue injury, resulting in the localized transformation of endocardial tissue into myxoid tissue of which the myxoma is composed of [22]. This dysregulated tissue proliferation could theoretically be incited by mechanical stress or inflammation at the site of which the myxoma originates [22]. The similarity of myxoma cells to endothelial or mesenchymal cells give credence to a metaplastic hypothesis [9]. This theory can be applied to the posited premise of this paper, of which the puncture of the interatrial septum induces tissue proliferation and metaplasia.

Supporting Evidence

Supporting the hypothesis in this paper, that is the incited development of a cardiac myxoma following a transseptal puncture, are the multiple other similarly reported cases. Alvarez, et Al. reported the incidental discovery of a left atrial myxoma approximately 1-year following radiofrequency ablation (RFA) for atrial fibrillation [1]. Similarly discovered, Wada et. Al. reported the discovery of a giant right atrial myxoma 3-years after obtaining a laser catheter ablation [2]. The development of a large left atrial myxoma was detected 6-months following a RFA procedure for atrial flutter in a 51-year-old male by Santillo, et Al [3]. A 55-year-old female who received a percutaneous RFA for recurrent atrial fibrillation was found to have developed a giant left atrial mass 8-months following the intervention, as documented by Kahraman, *et al*. [4]. In the aforementioned cases, a similar consensus was drawn to the one extrapolated from our documented case; of which the percutaneous tissue interruption stimulated the development of the cardiac mass.

Conclusion:

Cardiac myxomas are hypothesized to be of sporadic genesis, however have been linked in familial genetic disorders. While still not established, the proposition of a metaplastic process through which myxoma growth is incited by tissue injury can be reasoned by the multiple documented cases wherein they are discovered to have developed following radiofrequency ablation procedures. We report the interesting and thought-provoking case of a likely cardiac myxoma that was discovered in an adult male approximately 4-years after he had received a transseptal radiofrequency ablation for atrial fibrillation. Direct causation cannot be established, and further prospective and retrospective studies should be performed in order to thoroughly understand this thought process, and distinguish between coincidence or causation.

Abbreviations:

ACS: Acute coronary syndrome
CAD: Coronary artery disease
CNC: Carney Complex
CT: Computed tomography

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EKG: Electrocardiogram
ESR: Erythrocyte sedimentation rate
MACE: Major adverse cardiovascular event
MRI: Magnetic resonance imaging
NSTEMI: Non ST-segment elevation myocardial infarction
PCI: Percutaneous coronary intervention
PDA: Posterior descending artery
RFA: Radiofrequency ablation
TTE: Transthoracic echocardiography
TEE: Transesophageal echocardiography

Author Contributions:

RG: Conceptualization, writing of original draft and editing of manuscript
ES: Review and editing
AG: Review and editing
FR: Supervision and final approval of manuscript
NI: Supervision and final approval of manuscript
GU: Supervision and final approval of manuscript

Disclosures/Declaration of Interest:

The authors declare that they have no disclosures or conflict of interest.

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Conflict of Interest:

The authors have no conflict of interest. Also, the authors declare that we have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Consent:

Obtained.

Ethics Approval:

Not applicable.

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