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CASE REPORT

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Rheumatic heart disease of the mitral valve alongside the papillary fibroelastoma of the aortic valve: A case report

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Key Clinical Message

Besides thromboses, it's crucial to also consider the rare possibility of tumors like papillary fibroelastomas when evaluating worsening cardiopulmonary symptoms in patients with severe rheumatic mitral stenosis and atrial fibrillation.

Abstract

Cardiac papillary fibroelastoma is a rare and benign endocardial tumor typically found on the aortic valve. The simultaneous occurrence of rheumatic heart disease affecting the mitral valve and papillary fibroelastoma on the aortic valve is infrequent, with limited documented instances. This unique case can enhance our understanding of the clinical presentation, diagnostic approaches, management options, and implications for patient outcomes in these two conditions. We present the case of a 47-year-old woman who was admitted to the hospital due to worsening dyspnea and fatigue, during which time she discovered an aortic valve papillary fibroelastoma. Further investigations revealed two thrombi in her left atrium and left atrial appendage, along with significant rheumatic mitral valve stenosis. The patient underwent thrombectomy, mitral valve replacement, and valve-sparing aortic valve tumor resection. Following surgery, the patient's recovery was unremarkable. This case report emphasizes the need for a comprehensive evaluation in patients with rheumatic mitral stenosis, considering all possible etiologies. While thrombi are typical in mitral stenosis and atrial fibrillation, the rare presence of tumors like papillary fibroelastomas should be recognized, underscoring the importance of further assessment when suspicion arises. Importantly, individuals with asymptomatic cardiac papillary fibroelastomas should undergo surgical treatment to minimize the potential risk of tumoral embolization.

KEYWORDS

cardiac papillary fibroelastoma, cardiac tumors, case report, oncology, rheumatic heart disease

1 | BACKGROUND

Primary cardiac tumors are rare, accounting for only 0.0017%–0.33% of all cardiac tumors, as reported by autopsy studies.[1] Among these, angiosarcoma, myxoma, and papillary fibroelastomas (PFE) were found to be the most common, with an incidence rate of 7.3%–8.5%, 24%–37%, and 7.9%–8.0%, respectively.[2] PFEs are a subtype of benign primary cardiac tumors that frequently occur on the heart valves–approximately 75% of all valvular tumors can be attributed to this type alone; their incidence in autopsy series has been reported up to as high as 0.33%.[3] While they often present no symptoms initially, they may cause embolism or stroke if left untreated. Notably, right-sided PFEs usually do not produce noticeable symptoms. At the same time, those on the left side may lead to complications like stroke or embolization due to obstruction of blood flow through affected valves.[4] Moreover, rheumatic heart disease (RHD) is another commonly associated pathology resulting from untreated streptococcal infections whereby severe damage occurs over time, leading to mitral stenosis (MS), mitral regurgitation, or other valve abnormalities.[5]

The coexistence of RHD involving the mitral valve and PFE located on the aortic valve is extremely rare, with only a case reported in the literature.[6] Therefore, reporting this unique case could contribute to our understanding of the clinical presentation, diagnosis, management options, and implications for patient outcomes of these two conditions. Therefore, we present a case of deteriorating dyspnea and fatigue associated with severe rheumatic MS and the incidental findings of an aortic valve PFE.

2 | CASE PRESENTATION

2.1 | Patient information

A 47-year-old woman with a chief complaint of deteriorating dyspnea for the past 2 weeks was referred to an internist and then to our clinic due to new mid-diastolic murmur with an intensity of 4/6 in the apex, pitting edema in both lower legs and expiratory wheezing. She complained of shortness of breath, intermittent dizziness, and palpitations in our clinic. The patient explicitly denied chest pain, abdominal pain in the right upper quadrant, hoarseness, hemoptysis, paroxysmal nocturnal dyspnea, or orthopnea. There was no history of coronary artery disease, cardiac interventions, pulmonary disease, cardiac tumors, cancer, smoking, alcohol consumption, illicit drug use, or psychological disorders. Moreover, her family history revealed no cardiopulmonary disease or oncologic disorders.

2.2 | Clinical findings

The patient's initial vital signs showed blood pressure of 135/85 mmHg, heart rate of 82 beats/min, and temperature of 36.5°C. Physical examination revealed a mid-diastolic murmur with an intensity of 4/6 in the apex, pitting edema in both lower legs, and wheezing. Additionally, the New York Heart Association (NYHA) functional class of the patient was III.

2.3 | Diagnostic assessment

The initial laboratory analysis, encompassing CBC with differential, blood culture, and urine culture, revealed no evidence of systemic infection. The laboratory tests also revealed no abnormalities in the cardiac enzyme profile. An abnormal chest X-ray showed displacement and widening of the carina, the elevation of the left main bronchus due to an enlarged left atrium, Kerley B-lines, cardiomegaly with the straightened left border of the heart, and prominent pulmonary vasculature (Figure 1). An electrocardiogram revealed atrial fibrillation (AF) and right axis deviation (Figure 2). The important findings of three-dimensional transesophageal echocardiography (TEE) revealed (Figure 3):

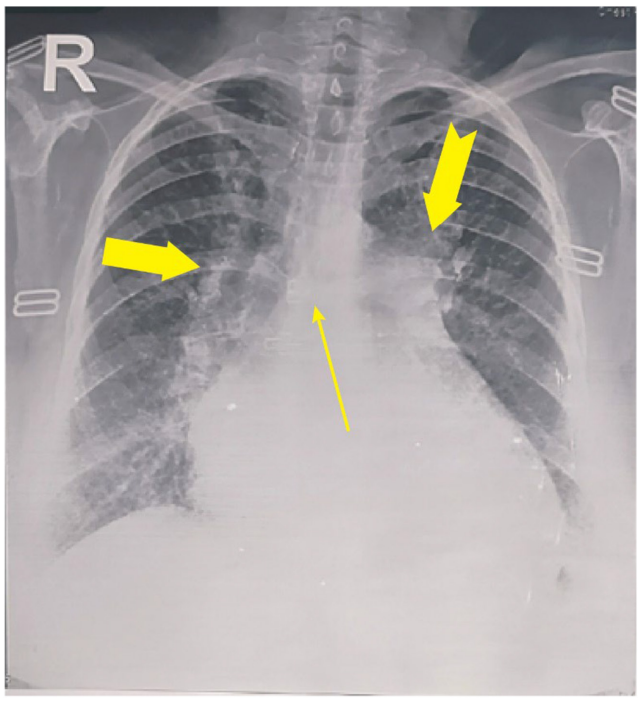

FIGURE 1 Chest X-ray shows cardiomegaly, displacement and widening of the carina (thin arrow), straightened left border of the heart (notched arrow), prominent pulmonary vasculature (thick arrow).

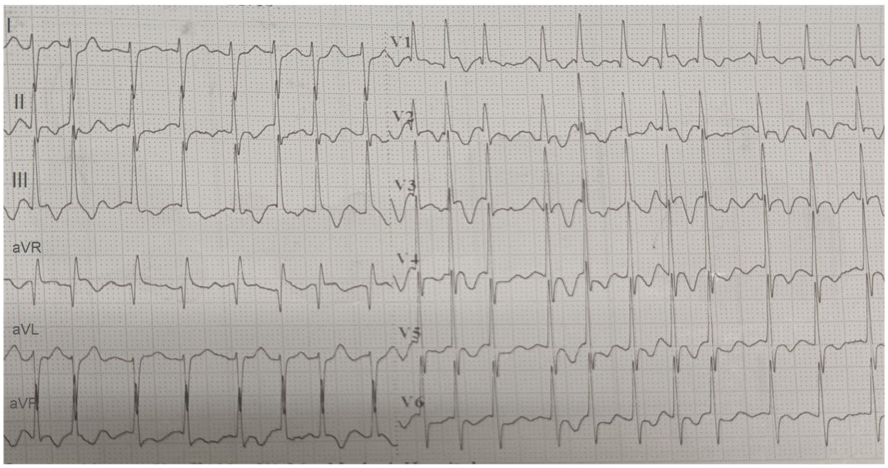

FIGURE 2 Electrocardiogram shows atrial fibrillation and right axis deviation.

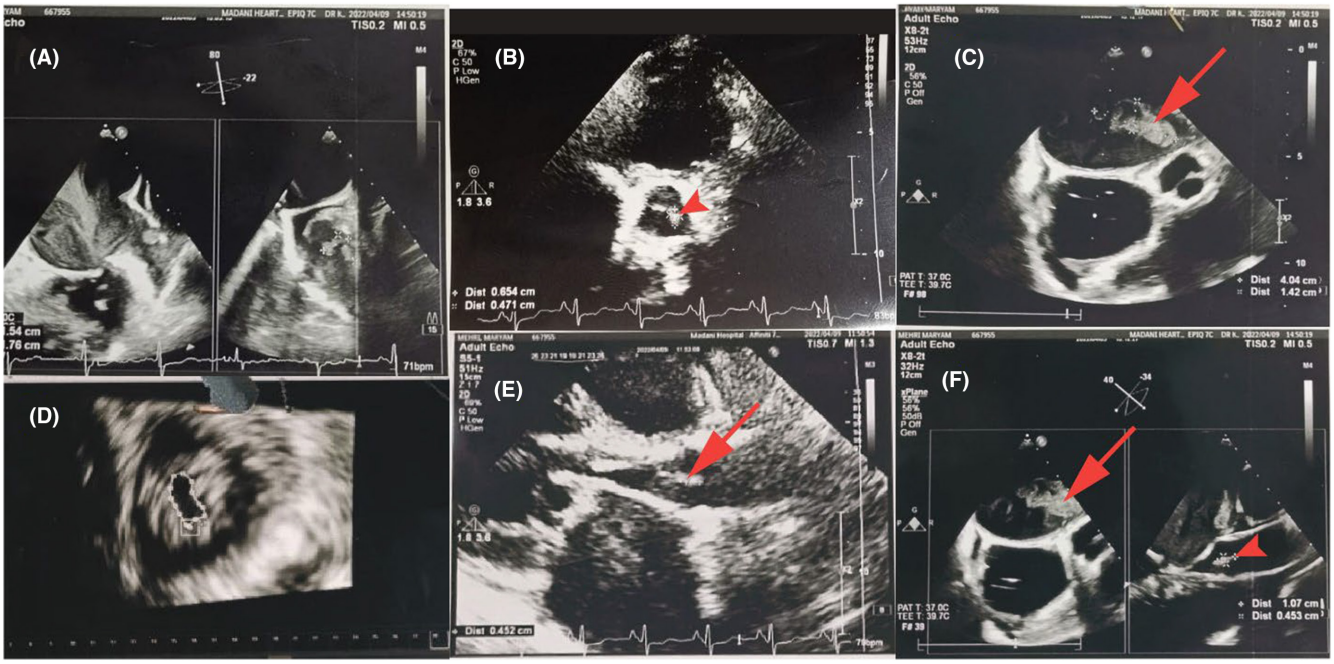

FIGURE 3 (A) Trans-esophagus echocardiography reveals thickened spontaneous echo-contrast in the left atrium and left atrial appendage, along with a blood clot in the left atrial appendage. (B) In the parasternal short-axis view, a tricuspid aortic valve is depicted, with a small mass attached to the arterial side of the aortic valve (arrowhead). (C) A visible blood clot in the left atrium (arrow). (D) Severe mitral stenosis is evident in this view. (E) A small, hypermobile, worm-like mass (10 × 4 mm) is observed attached to the arterial side of the aortic valve in this parasternal long-axis view (arrow). (F) This view showcases both the blood clot in the left atrium (arrow) and a small mass attached to the aortic valve (arrowhead).

1. Left ventricular ejection fraction: 50%.
2. The left ventricular (LV) size was small with preserved systolic function, and the LV was D-shaped.
3. The right ventricular (RV) size was enlarged with reduced systolic function, right ventricular diastolic diameter = 42 mm, and tricuspid annular plane systolic eruption = 16 mm.
4. Left atrium (LA) size was huge, with Left Atrial Volume Index = 131 cc/m2, and there was a large heterogeneous semi-mobile thrombus (size = 40 × 14 mm) with hypoechoic segments attached to the anterior wall of LA.
5. Right atrium (RA) size was enlarged, right atrial area = 18.1 cm^2.
6. The left atrial appendage (LAA) had thick spontaneous echo contrast and thrombus (size = 15 × 17 mm). Left atrial appendage emptying velocity = 20 cm/s
7. The mitral valve was thick and dome-shaped, indicative of rheumatic valve disease with severe mitral stenosis. Mild mitral regurgitation was also present. Mitral valve area by pressure half-time = 0.6 cm^2, by planimetry = 0.77 cm^2, and by Proximal Iso-velocity surface area = 0.67 cm^2, peak velocity = 2.3 m/s, pressure

gradient = 22 mmHg, mean gradient = 15 mmHg, velocity time integral = 97.5 cm.

8. The aortic valve showed a rheumatic appearance with a small hypermobile worm-like mass (size = 104 mm) attached to the arterial side of AV, AV annulus = 14 mm.
9. The tricuspid valve had moderate regurgitation, tricuspid regurgitation gradient = 94 mmHg, right ventricular systolic pressure = 101 mmHg, and severe pulmonary hypertension was present.
10. The pulmonic valve had mild regurgitation, pulmonary regurgitation gradient = 20 mmHg, acceleration time = 70 ms

The diagnosis of RHD was confirmed based on the findings observed during TEE. A workup for infective endocarditis was ordered for the patient following vegetation detection on the aortic valve. However, despite undergoing three separate blood samples using typical culture techniques, all secondary laboratory results, such as blood cultures, were normal, leading to limited differential diagnoses, including cases of sterile vegetation or endocarditis with negative cultures. Surgical excision and histopathology were deemed necessary diagnostic approaches without meeting Duke's criteria and without indications for antibiotic therapy. As such, tumorectomy coupled with aortic valve repair (AVR) and mitral valve replacement (MVR) surgery were recommended as viable options for the patient. Ultimately, diagnostic procedures were completed within 4 days following the initial visit.

2.4 | Therapeutic intervention

Three days following the diagnosis, the patient underwent a tumorectomy with AVR and MVR at Shahid Madani's Hospital in Tabriz, Iran. The procedure began after administering general anesthesia and preparation, followed by chest opening via mid-sternotomy. Subsequently, cannulation of the aorta, inferior vena cava, and superior vena cava was performed to establish the pump. An aortotomy was then carried out, which involved removing a mass measuring approximately 2 × 1 mm based on the left coronary valve under cardioplegia. Despite rheumatic changes, the aortic valve was considered suitable for repair. Subsequently, the team chose aortic valve repair as a viable and effective alternative to replacement—this decision aimed to address the specific characteristics of the valve pathology while preserving its integrity.

Additionally, the procedure involved the replacement of mitral valve leaflets with a mechanical valve (St. Jude). Subsequently, the left atrium and left atrial appendage were opened to remove a blood clot measuring approximately 2 × 4 cm and 15 × 17 mm. Following the clot removal, the left atrial appendage was encircled and tied off.

The visual examination of the mitral valve and aortic valve suggested the presence of healed rheumatic valve disease. The excised mass appeared grayish-white during the visual inspection. A day post-surgery, microscopic analysis revealed paucicellular fibroelastic tissue covered by tiny endocardial layer confirming PFE (Figure 4). Two more specimens were extracted during the procedure: one from the left atrium, which only revealed a blood clot, and another from the mitral valve, which exhibited degenerative alterations. Postoperative TEE was conducted to evaluate the heart's valve functions. The results indicated that there were no signs of stenosis or insufficiency on any part of the heart's valves, suggesting the function of the bi-leaflet mechanical prosthetic mitral valve was satisfactory after the surgery. Finally, life-long anticoagulants and aspirin treatments commenced immediately after surgery.

2.5 | Follow-up and outcomes

In evaluating outcomes, the patient's condition was precisely monitored for a month, focusing on the remission of symptoms and postoperative warning signs. Unfortunately, the patient did not attend the scheduled follow-up echocardiography; however, she did not report any adverse events during the 1 month. Additionally, meticulous observation and patient reports assessed adherence to prescribed medications and tolerability. Finally, the absence of symptoms for the entire month following admission into postoperative care facilities further underscored the favorable influence of the intervention on the patient's health.

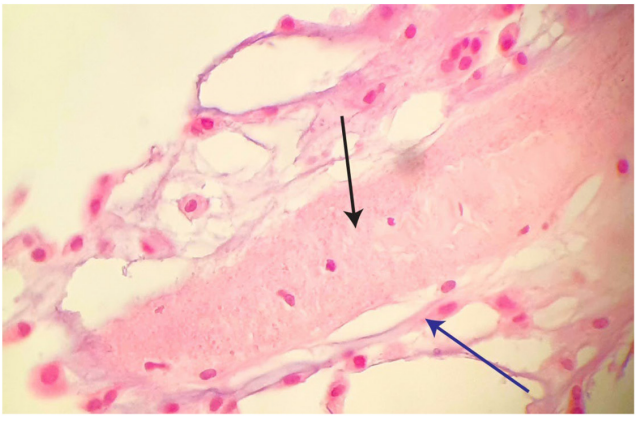

FIGURE 4 Histological section of aortic valve tissue, stained with Hematoxylin and Eosin (H&E) at an original magnification of ×40, revealing paucicellular fibroelastic tissue (black arrow) covered by a tiny layer of endocardium (blue arrow).

3 | DISCUSSION

The concomitant occurrence of severe rheumatic MS, aortic valve PFE, and thrombi in the left atrium and left atrium appendage is exceedingly rare, emphasizing the necessity for a comprehensive diagnostic approach, given the rarity and intricacy associated with such cases in clinical practice.

It is important to consider a broad range of potential causes when evaluating patients with dyspnea and incidental thrombi or valvular masses. One possible cause for these symptoms is AF, which can lead to intracardiac thrombus formation due to increased blood stagnation inside the atria that promotes clotting factors activity therein.[7] RHD may also be considered a potential underlying cause.[8] Infective endocarditis (IE) is another possibility, especially in cases where large vegetation is observed on cardiac valves. IE results from bacterial or fungal infections within the bloodstream that colonize damaged areas of heart valves.[9]

Furthermore, non-infectious valvular vegetations should be included in the differential diagnosis of patients with incidental thrombi or valvular masses and negative blood culture results.[10] Some examples include antiphospholipid antibody syndrome and organized thrombus in Libman-Sachs endocarditis, non-bacterial thrombotic endocarditis, vasculitis, giant cell arteritis, connective tissue disease, cholesterol embolization syndrome, mural thrombi associated with cardiomyopathy or heart failure, Lambl's excrescences as well as vasculitis among others.[11–13] Ultimately, it is crucial to consider atypical valvular lesions such as organized thrombus, valvular calcification, and valvular abscess in the differential diagnosis of incidental thrombi or valvular masses. Intracardiac masses, including benign tumors like PFEs, myxomas, and lipomas, and malignant tumors like sarcomas, should also be considered.[14,15] According to Kurup et al., the exact cause of PFE remains unknown; however, it may be linked with cardiovascular intimal damage. PFE can result from a history of chest radiotherapy or cardiac surgery.[16]

Intracardiac masses may present symptoms such as dyspnea, chest pain, or palpitations but are often discovered incidentally during diagnostic imaging for unrelated reasons. The clinical presentation depends on variables including location, mobility size, and inclination for tumor embolization, among others. Thrombosis is a significant cause of symptoms that could lead to cerebrovascular accidents or coronary artery occlusion, resulting in chest discomfort, myocardial infarction, and even sudden death in severe cases.[17]

After the initial assessment, standard diagnostic procedures such as electrocardiography and echocardiography are crucial for establishing a diagnosis. Echocardiography is an essential tool for identifying structural changes associated with RHD and IE, while an electrocardiogram confirms the presence of AF. In cases where thrombi are suspected due to clinical presentation or previous history of AF, TEE is superior to transthoracic echocardiography (TTE) in detecting intracardiac clots.[18] In addition, cardiac magnetic resonance imaging, cardiac computed tomography scanning, 18F-fluorodeoxyglucose positron emission tomography (18F FDG-PET), and invasive procedures like coronary angiograms may have complementary roles in evaluating cardiac masses.[19]

The choice of management strategy for patients with incidental thrombi or valvular masses depends on the results of diagnostic tests.[20] Asymptomatic individuals diagnosed with non-mobile tumors are typically treated conservatively before surgery to remove the tumor and prevent subsequent complications.[20] High-risk patients may receive anticoagulant medication as a treatment option.[21] However, surgical resection is highly recommended in cases where the mass is highly mobile or pedunculated due to its predictive role in cerebrovascular events and death.[11,22,23] In most tumoral cases, tumorectomy without valve repair or replacement is performed. However, if there is a risk of valve malfunction and high embolism, extensive tumorectomies may require additional interventions, such as valve repair or replacement, to prevent further complications.[24] According to studies, complete excision of PFE has significantly improved long-term prognosis while reducing stroke risks. Patients without surgery have a higher risk of cerebrovascular events and mortality than those who undergo surgery. So, surgery is advised for asymptomatic patients diagnosed with cardiac PFE incidentally.[25,26]

In a case report, a 55-year-old woman manifested dyspnea, palpitation, and dizziness and was diagnosed with RHD and PFE of the aortic valve initially declined surgery due to economic reasons. However, after 5 years, a successful surgical intervention involving mass removal and valve replacement was performed.[6] Similarly, a 60-year-old man with a history of recurrent respiratory infections presented symptoms of face puffiness, swelling of bilateral limbs, palpitation, and shortness of breath. Subsequent diagnostic testing revealed RHD with poorly managed AF, severely enlarged left atrium, and severe MS, for which successful open-heart surgery with mitral valve replacement addressed condition.[27] In another case, a 54-year-old woman with chest pain revealed a suspicious PFE on the aortic valve in echocardiography which obstructed the ostium of the left main AV leaflet. She ultimately underwent shave excision during surgery.[28]

Moreover, a 48-year-old man presented with vertigo, nausea, vomiting and ataxia, revealing right middle cerebral artery occlusion and leading to the discovery of a

mitral valve echogenic mass, for which open-heart mitral valvuloplasty successfully resolved the symptoms.[29] Similarly, a 51-year-old woman with slurred speech and a right frontal lobe infarct displayed a myxomatous-appearing sessile echogenicity on the mitral valve. She underwent open-heart mitral valvuloplasty without valve replacement, resulting in a stable discharge.[29] These instances highlight the importance of recognizing PFE's broader impact, extending beyond the cardiopulmonary system to manifest as neurologic symptoms.

This case report describes a patient with worsening dyspnea and fatigue, where an aortic valve PFE, severe rheumatic MS and two LA thrombi were incidentally discovered leading to thrombectomy, MVR, and AV tumor resection. While our patient exhibited similarities with the case described in the literature,[6] such as advanced age, palpitations and edema, manifestations such as chest pain and recurrent respiratory infections observed in the previous patients were not present in our case.[27,28] Furthermore, due to the timely referral of the patient to our clinic, thromboembolic complications of the cardiac mass were not observed. Comparing our case with similar cases in the literature highlights both commonalities and differences, contributing to the comprehensive understanding of disease entities and their clinical manifestations.

Our case report's educational and scientific significance lies in the uncommon convergence of these two medical conditions. This unique clinical scenario underscores how unexpected findings can emerge while investigating seemingly straightforward medical issues, highlighting the importance of exploring all possible causes when evaluating patients presenting with cardiovascular complaints. It contributes to an enhanced understanding of diagnostic complexities and potential implications for patient care. The insights derived from this case are valuable for healthcare professionals and researchers, potentially influencing clinical approaches and motivating further studies in cardiac pathology.

This case report is limited by its single-case focus and the absence of comparative analyses. Future research should consider multifaceted case studies, extended follow-up periods, diverse therapeutic approaches, and comprehensive examinations of genetics, environmental influences, and socioeconomic variables.

4 | CONCLUSION

This case report highlights the significance of comprehensive assessments in suspected cases, emphasizing that although thrombi are commonly observed as cardiac masses in patients with both MS and AF, a rare incidence of tumorus such as PFEs exists. It underscores the necessity for prompt surgical intervention, particularly for left-sided PFEs, as untreated left-sided cardiac PFEs pose an elevated risk of stroke that cannot be mitigated by warfarin or antiplatelet therapy.

AUTHOR CONTRIBUTIONS

Naser Khezerlouy Aghdam: Conceptualization; project administration; resources. **Mitra Delkhah:** Investigation; validation; visualization. **Saeid Danayi:** Data curation; resources; supervision. **Navid Sobhi:** Conceptualization; validation; writing – original draft; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

No author states to have any conflicts of interest.

DATA AVAILABILITY STATEMENT

The datasets supporting the conclusions of this article are included within the article and its additional files.

ETHICS STATEMENT

All procedures performed in this study were in accordance with the ethical standards of the Tabriz University Committee on Ethics in Medical Sciences Research and with the 1964 Helsinki declaration and its later amendments or comparable ethical standard.

CONSENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

DECLARATIONS

During the preparation of this work the authors used ChatGPT 3.5 in order to facilitate paraphrasing and grammar checking. After using this tool, the authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

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