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A Case Report

A CASE REPORT ON VALVULAR HEART DISEASE WITH ATRIAL FIBRILLATION

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Abstract:

Background: Valvular heart disease can cause cardiac changes, predisposing patients to atrial fibrillation (AF). AF may worsen symptoms and increase thromboembolic risk, making diagnosis and management important.

Case Presentation: A 62-year-old man presented with three months of exertional breathlessness, two months of palpitations and fatigue, and one month of bilateral pedal edema. Examination showed an irregularly irregular pulse, elevated JVP, crepitations, and a grade 3/6 pansystolic murmur at the apex. ECG confirmed AF with rapid ventricular response at 118 bpm. Echocardiography revealed grade III mitral regurgitation, left atrial enlargement (49 mm), mildly dilated left ventricle, and LVEF of 48%. Treatment with metoprolol, apixaban, furosemide, losartan, and atorvastatin improved symptoms and ventricular rate.

Conclusion: Recognition, anticoagulation, rate control, and congestion management can stabilize patients, while mitral regurgitation requires cardiology evaluation.

Keywords: Valvular heart disease, mitral regurgitation, atrial fibrillation, anticoagulation, echocardiography.

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INTRODUCTION:

Valvular heart disease includes conditions in which one or more heart valves become narrowed, stiff, or unable to close properly. These changes disturb normal blood flow and place extra pressure on the heart chambers. Mitral valve disease matters because the mitral valve controls blood flow between the left atrium and left ventricle.^[1] Mitral regurgitation occurs when the valve does not close completely, allowing blood to flow backward into the left atrium during contraction. Mild disease may cause few symptoms, but significant regurgitation can lead to breathlessness, tiredness, reduced exercise capacity, and fluid retention. Over time, continued volume overload may cause enlargement of the left atrium and changes in ventricular function. Early recognition and regular echocardiographic assessment are especially important clinically.^[2]

Atrial fibrillation is one of the most common rhythm disturbances and is frequently associated with structural heart disease. In atrial fibrillation, the atria do not contract normally, producing an irregular rhythm. Patients may notice palpitations, weakness, dizziness, tiredness, or shortness of breath, although some individuals have very few symptoms.^[3] When atrial fibrillation occurs with mitral regurgitation, the enlarged left atrium can provide a favorable setting for development and persistence of the arrhythmia. At the same time, the irregular and often rapid ventricular rate can worsen existing symptoms. Another concern is formation of blood clots that can travel to the brain and cause ischemic stroke. Therefore, assessment of stroke risk and appropriate anticoagulation when indicated are important parts of management.^[4]

The relationship between mitral regurgitation and atrial fibrillation can become a continuing cycle. Significant mitral regurgitation increases the amount of blood entering the left atrium, which may gradually enlarge. An enlarged atrium can develop abnormal electrical activity and sustain atrial fibrillation. Once atrial fibrillation develops, loss of effective atrial contraction can reduce cardiac filling and worsen symptoms.^[5] The clinical presentation can vary from mild palpitations to marked breathlessness, fatigue, swelling of the legs, and reduced daily activity. Echocardiography is particularly useful because it can show the severity of mitral regurgitation, chamber size, ventricular function, and related cardiac changes clearly. Electrocardiography confirms the rhythm disturbance, while laboratory testing helps identify associated conditions and supports safe treatment planning and follow-up visits.^[6]

Management of patients with valvular heart disease and atrial fibrillation requires attention to both the valve and rhythm. Treatment is directed toward controlling ventricular rate, reducing fluid-related symptoms, and preventing thromboembolic complications. Beta blockers may control rate, while diuretics can help relieve congestion and peripheral edema. Anticoagulant therapy may be indicated according to stroke risk and valve type.^[7] However, medicines do not correct significant structural valve abnormalities. Patients with important mitral regurgitation may therefore require cardiology assessment to determine whether valve repair or replacement, including transcatheter approaches in selected cases, should be considered when clinically indicated in suitable patients after specialist assessment. Regular follow-up is necessary to monitor symptoms, rhythm, ventricular function, renal function, electrolytes, and treatment response over time.^[8]

CASE PRESENTATION:

Patient Demographics : A 62-year-old male patient presented to the cardiology outpatient department with complaints of progressive shortness of breath, intermittent palpitations, generalized fatigue, and swelling of both lower limbs. The patient was a retired office administrator. He had a known history of hypertension for approximately 8 years and a past history suggestive of rheumatic fever during childhood. He had no previously documented history of atrial fibrillation or chronic heart failure.

Chief Complaints

The patient presented with the following complaints:

- Shortness of breath, particularly on exertion, for approximately 3 months
- Intermittent palpitations for 2 months
- Generalized fatigue and reduced exercise tolerance for 2 months
- Bilateral lower-limb swelling for approximately 1 month

The symptoms had gradually increased in severity and were associated with difficulty performing routine physical activities.

History of Present Illness

The patient was apparently well until approximately 3 months before presentation, when he developed gradually progressive shortness of breath during routine activities. Initially, breathlessness occurred only after moderate exertion, such as climbing stairs or walking for longer distances. Over the following weeks, the symptoms progressed and began occurring with minimal physical activity. There was no history of syncope or loss of consciousness. The patient also

reported increasing fatigue and reduced exercise tolerance. During the month preceding admission, he developed swelling of both ankles, which was more prominent toward the evening and partially improved after overnight rest.

Because of the progressive symptoms, the patient was referred for cardiovascular evaluation. Clinical examination and subsequent investigations suggested significant valvular heart disease associated with newly diagnosed persistent atrial fibrillation.

Past Medical History

The patient had been diagnosed with hypertension approximately 8 years previously and had been receiving regular antihypertensive treatment. His blood pressure had generally remained controlled. He had not undergone previous valve surgery or other cardiac intervention. There was no previous history of hospitalization for decompensated heart failure.

Medication History

Before presentation, the patient was taking the following medications:

Medication	Dose	Frequency	Indication
Amlodipine	5 mg	Once daily	Hypertension
Losartan	50 mg	Once daily	Hypertension
Atorvastatin	20 mg	Once daily	Cardiovascular risk reduction

Family History

There was no significant family history of rheumatic heart disease, valvular heart disease, atrial fibrillation, cardiomyopathy, or sudden cardiac death.

Personal and Social History

The patient was a former smoker with an estimated smoking exposure of approximately 10 pack-years. He had discontinued smoking approximately 5 years earlier. He reported occasional alcohol consumption. His dietary intake was mixed, with no specific dietary restrictions.

Occupational History

The patient was a retired office administrator. During his working years, his occupation was predominantly sedentary and involved prolonged periods of desk work.

Allergic History

The patient had no known drug allergies. He also reported no known food allergies or previous history of serious allergic reactions.

General Physical Examination

On examination, the patient was conscious, alert, and oriented to time, place, and person. He appeared mildly dyspneic but was able to communicate in complete sentences.

His vital signs were:

Parameter	Finding
Blood pressure	132/82 mmHg
Heart rate	118 beats/min
Respiratory rate	22 breaths/min
Temperature	98.2°F
Oxygen saturation	95% on room air

The pulse was irregularly irregular with a rate of approximately 118 beats/min. Mild elevation of the jugular venous pressure was noted. Bilateral pitting pedal edema was present, predominantly around the ankles. There was no cyanosis, clubbing, significant pallor, or generalized lymphadenopathy.

Cardiovascular Examination

Cardiovascular examination revealed an irregularly irregular rhythm. The apical impulse was mildly displaced laterally. A grade 3/6 pansystolic murmur was heard best at the cardiac apex and radiated toward the left axillary region. The murmur was suggestive of significant mitral valve regurgitation. No pericardial rub was identified. Peripheral pulses were palpable, although their rhythm was irregular.

Respiratory Examination

On respiratory examination, the respiratory rate was mildly increased. Fine bilateral basal crepitations were present, particularly at the posterior lung bases. There was no evidence of significant wheezing or focal bronchial breathing. The findings were compatible with mild pulmonary congestion.

Abdominal Examination

The abdomen was soft and non-tender. No significant hepatomegaly or splenomegaly was detected. There was no clinically significant ascites.

Neurological Examination

The patient was conscious and oriented. Higher mental functions were normal. Cranial nerve examination was unremarkable. There was no focal motor or sensory neurological deficit. No clinical evidence of previous stroke or transient ischemic attack was identified.

Laboratory Investigations Complete Blood Count

Parameter	Patient Value	Reference Range
Hemoglobin	13.2 g/dL	13–17 g/dL
Total leukocyte count	7,600 cells/ μ L	4,000–11,000 cells/ μ L
Neutrophils	62%	40–75%
Lymphocytes	30%	20–45%
Platelet count	$238 \times 10^3/\mu$ L	$150\text{--}450 \times 10^3/\mu$ L
ESR	18 mm/hr	<20 mm/hr

Renal Function Tests

Parameter	Patient Value	Reference Range
Blood urea nitrogen	26 mg/dL	7–20 mg/dL
Serum creatinine	1.0 mg/dL	0.7–1.3 mg/dL
Sodium	138 mmol/L	135–145 mmol/L
Potassium	4.2 mmol/L	3.5–5.1 mmol/L
Chloride	102 mmol/L	98–107 mmol/L

Liver Function Tests

Parameter	Patient Value	Reference Range
AST	31 U/L	10–40 U/L
ALT	28 U/L	7–56 U/L
ALP	88 U/L	44–147 U/L
Total bilirubin	0.9 mg/dL	0.2–1.2 mg/dL
Albumin	4.1 g/dL	3.5–5.0 g/dL

Cardiac Biomarkers

Investigation	Result	Interpretation
NT-proBNP	1,850 pg/mL	Elevated
Troponin-I	Negative	No biochemical evidence of acute myocardial injury

Thyroid Function Tests

Parameter	Patient Value	Reference Range
TSH	1.86 μ IU/mL	0.4–4.0 μ IU/mL
Free T4	1.18 ng/dL	0.8–1.8 ng/dL
Free T3	3.1 pg/mL	2.3–4.2 pg/mL

Blood Glucose and Metabolic Investigations

Parameter	Patient Value	Reference Range
Fasting blood glucose	96 mg/dL	70–100 mg/dL
HbA1c	5.6%	<5.7%
Total cholesterol	168 mg/dL	<200 mg/dL
LDL cholesterol	92 mg/dL	<100 mg/dL
HDL cholesterol	44 mg/dL	>40 mg/dL
Triglycerides	132 mg/dL	<150 mg/dL

Electrocardiogram

A 12-lead electrocardiogram demonstrated atrial fibrillation with a rapid ventricular response. The ventricular rate was approximately 118 beats/min. P waves were absent and the ventricular rhythm was irregularly irregular. No acute ST-segment elevation or other electrocardiographic evidence of acute myocardial infarction was identified.

ECG Impression: Persistent atrial fibrillation with rapid ventricular response.

Chest Radiography:

Chest radiography demonstrated mild cardiomegaly with mild pulmonary vascular congestion. No focal pulmonary consolidation or pleural effusion was identified. The findings were consistent with underlying structural heart disease and early congestive cardiac changes.

Echocardiographic Examination

Echocardiographic Parameter	Finding
Left atrial diameter	49 mm
Left ventricular ejection fraction	48%
Mitral valve	Significant grade III mitral regurgitation
Left ventricle	Mildly dilated
Right ventricular function	Preserved
Pulmonary artery pressure	Mildly elevated
Pericardium	No significant effusion

Echocardiography demonstrated significant mitral regurgitation with left atrial enlargement and mild impairment of left ventricular systolic function. The enlarged left atrium provided an important structural substrate for the development and persistence of atrial fibrillation.

DIAGNOSIS:

Based on the patient's progressive exertional dyspnea, intermittent palpitations, generalized fatigue, reduced exercise tolerance, and bilateral lower-limb edema, a cardiovascular disorder was suspected. Clinical examination revealed an irregularly irregular pulse and a pansystolic murmur at the cardiac apex radiating to the left axilla, suggesting mitral valve disease with atrial fibrillation. ECG confirmed atrial fibrillation with rapid ventricular response. Echocardiography demonstrated significant grade III mitral regurgitation with left atrial enlargement and mildly reduced left ventricular systolic function, confirming valvular heart disease complicated by persistent atrial fibrillation.

TREATMENT:

On Day 1, the patient was admitted for management of symptomatic persistent atrial fibrillation with rapid ventricular response, significant grade III mitral regurgitation, mild pulmonary congestion, and bilateral pedal edema. Metoprolol tartrate 25 mg orally twice daily was initiated for ventricular rate control, with monitoring of heart rate and blood pressure. Furosemide 40 mg orally once daily was started for relief of fluid overload and peripheral edema. His pre-existing losartan 50 mg orally once daily was continued for blood-pressure control and cardiovascular protection. Atorvastatin 20 mg orally once daily was also continued. Renal function, serum potassium, blood pressure, urine output, and clinical signs of congestion were monitored.

On Day 2, the patient remained in atrial fibrillation, although his ventricular rate showed improvement following metoprolol therapy. Metoprolol tartrate 25 mg orally twice daily and furosemide 40 mg once daily were continued. Following assessment of thromboembolic risk, renal function, bleeding risk, age, and body weight, apixaban 5 mg orally twice daily was initiated for prevention of AF-related thromboembolic complications. The

patient's creatinine was 1.0 mg/dL and age was 62 years, with no documented dose-reduction criteria. Losartan 50 mg once daily and atorvastatin 20 mg once daily were continued. Blood pressure, heart rate, bleeding manifestations, renal function, and electrolytes were monitored.

On Day 3, the patient demonstrated progressive improvement in ventricular rate, palpitations, and respiratory symptoms. Metoprolol tartrate 25 mg orally twice daily was continued for ventricular rate control, with adjustment considered according to resting heart rate, blood pressure, and tolerance. Furosemide 40 mg orally once daily was continued because mild pedal edema remained. Apixaban 5 mg orally twice daily was continued for thromboembolic prevention without evidence of bleeding. Losartan 50 mg once daily was maintained for hypertension and ventricular afterload reduction, while atorvastatin 20 mg once daily was continued for lipid management. Serum creatinine, sodium, potassium, blood pressure, fluid balance, and clinical symptoms were reassessed.

On Day 4, the patient showed further clinical improvement, with reduced exertional breathlessness, fatigue, palpitations, and lower-limb edema. His ventricular rate was controlled at approximately below 100 beats/minute at rest. Metoprolol tartrate 25 mg orally twice daily was continued, with monitoring for bradycardia, hypotension, dizziness, or excessive fatigue. Furosemide 40 mg once daily was maintained because of the previous pulmonary congestion and pedal edema. Apixaban 5 mg twice daily, losartan 50 mg once daily, and atorvastatin 20 mg once daily were continued. Daily weight, urine output, blood pressure, renal function, potassium concentration, and bleeding symptoms were monitored during treatment.

On Day 5, the patient was clinically more stable, with substantial reduction in palpitations and dyspnea and almost complete resolution of bilateral pedal edema. Metoprolol tartrate 25 mg orally twice daily was continued for rate control, maintaining an acceptable resting ventricular rate

without symptomatic hypotension or bradycardia. Furosemide 40 mg orally once daily was continued with reassessment of the need for long-term diuretic therapy according to volume status. Apixaban 5 mg orally twice daily was continued for stroke prevention. Losartan 50 mg once daily and atorvastatin 20 mg once daily were maintained. Electrolytes, renal function, blood pressure, heart rate, and signs of recurrent congestion were monitored.

On Day 6, the patient remained hemodynamically stable with improved breathlessness, fatigue, exercise tolerance, and peripheral edema. His resting heart rate was approximately 80–90 beats/minute. Metoprolol tartrate 25 mg orally twice daily, apixaban 5 mg orally twice daily, furosemide 40 mg orally once daily, losartan 50 mg orally once daily, and atorvastatin 20 mg orally once daily were continued. The patient was counselled regarding strict medication adherence, anticoagulant-related bleeding precautions, avoidance of unnecessary NSAIDs, moderate dietary sodium restriction, daily weight monitoring, and recognition of worsening dyspnea or edema. Cardiology consultation was arranged for definitive evaluation of the significant mitral regurgitation.

On Day 7, the patient was considered clinically stable for discharge following improvement in dyspnea, palpitations, fatigue, pulmonary congestion, and pedal edema. The discharge medications included metoprolol tartrate 25 mg orally twice daily for ventricular rate control, apixaban 5 mg orally twice daily for prevention of AF-related thromboembolism, furosemide 40 mg orally once daily for management of congestion, losartan 50 mg orally once daily for hypertension and cardiovascular management, and atorvastatin 20 mg orally once daily for lipid control. He was advised to continue follow-up with cardiology.

DISCUSSION:

The present case demonstrates how significant mitral regurgitation can contribute to enlargement of the left atrium and development of atrial fibrillation. The patient had grade III MR, a left atrial diameter of 49 mm, persistent AF, and symptoms including breathlessness, palpitations, fatigue, and reduced exercise tolerance. These findings are clinically consistent with previous observations. Gertz et al. (2011) reported that patients with AF and significant MR had larger left atria and mitral annuli, and that restoration of sinus rhythm could reduce MR severity in selected patients. Similarly, Kato et al. (2026) found that greater left atrial enlargement was associated with more severe MR and worsening symptoms in patients with atrial functional MR. Thus, the left atrial enlargement in our patient may have played

an important role in both the development and persistence of AF.^[9,10]

The echocardiographic findings in this patient are also important because the combination of significant MR, atrial enlargement, and mildly reduced ventricular function may increase the risk of future cardiac complications. His left ventricular ejection fraction was 48%, while the left atrium was markedly enlarged. Mesi et al. (2021) found that patients with severe atrial functional MR commonly had atrial fibrillation, left atrial abnormalities, and a greater risk of heart-failure hospitalization and mortality compared with patients having primary MR. In another study, Hirji et al. (2022) demonstrated that atrial functional MR and ventricular functional MR have different clinical characteristics and outcomes, with atrial functional MR being more commonly associated with AF and relatively preserved ventricular function. These findings closely resemble the present case and emphasize the importance of careful echocardiographic assessment.^[11,12]

The treatment given in this case focused initially on controlling the rapid heart rate, reducing fluid overload, and preventing AF-related thromboembolism. Metoprolol was used for rate control, furosemide for pedal edema and pulmonary congestion, and apixaban for stroke prevention. This approach is supported by previous evidence. Dawwas GK found that the benefits of apixaban compared with warfarin were consistent in patients with mitral regurgitation. Importantly, patients with significant mitral stenosis were excluded, making the findings particularly relevant to MR rather than rheumatic mitral stenosis. Similarly, Melillo et al. (2020) studied patients with AF and MR receiving direct oral anticoagulants and found comparable thromboembolic effectiveness across different MR severities, although patients with more severe MR required careful monitoring for bleeding. These findings support the anticoagulation strategy used in this patient.^[13,14]

Although the patient's symptoms improved with medical treatment, the underlying significant MR remains an important long-term concern. Medical therapy can reduce congestion and control AF-related symptoms, but it does not directly repair the leaking mitral valve. In this case, cardiology follow-up and assessment by a valve team are therefore important. Wagner et al. (2024) reported favorable outcomes following mitral valve repair in patients with atrial functional MR, with low rates of recurrent significant MR and reoperation. Similarly, Kim et al. (2020) found that symptomatic patients with severe atrial functional MR had a higher risk of cardiac death or heart-

failure hospitalization during follow-up, particularly when severe MR and ventricular enlargement were present. These findings support continued monitoring and consideration of definitive valve intervention if significant symptoms or ventricular changes persist despite medical management.^[15,16]

CONCLUSION:

This case highlights the clinical importance of recognizing atrial fibrillation in patients with significant valvular heart disease, particularly mitral regurgitation. The patient presented with progressive breathlessness, palpitations, fatigue, reduced exercise tolerance, and lower-limb edema, with ECG confirming atrial fibrillation with rapid ventricular response and echocardiography demonstrating grade III mitral regurgitation, marked left atrial enlargement, and mildly reduced left ventricular systolic function. Initial management with rate control, anticoagulation, and treatment of fluid overload resulted in improvement in symptoms and ventricular rate. This case emphasizes the importance of early clinical assessment, echocardiographic evaluation, appropriate stroke prevention, and regular monitoring. Although medical therapy can provide symptomatic and hemodynamic stabilization, significant mitral regurgitation requires continued cardiology follow-up and multidisciplinary evaluation for definitive valve-directed management.

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