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When fever masks heart failure: A case report of ischemic cardiomyopathy in an elderly woman

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Abstract
Background: Heart failure is a leading cause of morbidity and mortality in the elderly, but its diagnosis can be challenging due to overlapping symptoms with other common conditions, such as pneumonia. Fever and elevated inflammatory markers can mask the underlying cardiac pathology, leading to delayed recognition and treatment.
Case: We report the case of a 73-year-old woman with no prior comorbidities who presented with fever, chest pain, and fatigue. Initial evaluation revealed leukocytosis and elevated C-reactive protein, leading to a presumptive diagnosis of bacterial pneumonia and antibiotic treatment. However, the patient's symptoms persisted, and her clinical condition deteriorated with worsening dyspnea and orthopnea. Further investigations, including echocardiography and cardiac imaging, revealed a dilated left ventricle, reduced ejection fraction (40%), and chronic total occlusion of the left anterior descending artery. A cardiac PET scan confirmed extensive myocardial scarring, consistent with ischemic cardiomyopathy. The patient was initiated on guideline-directed medical therapy with significant clinical improvement at follow-up.
Conclusion: This case highlights the diagnostic difficulty in differentiating heart failure from infections in elderly patients presenting with nonspecific symptoms and inflammatory markers. It underscores the importance of a high index of suspicion and the role of multimodal imaging in diagnosing ischemic cardiomyopathy, even in the absence of classic cardiac risk factors.

Keywords: Ischemic cardiomyopathy, heart failure, elderly patient, misdiagnosis, multimodal imaging

Introduction

Case Presentation

A 73-year-old female patient with no known comorbidities presented to the outpatient department with fever, chest pain, and fatigue for one week. The chest pain was more localized on the left side radiating to the left shoulder. She had mild difficulty breathing that was aggravated by walking short distances. She was able to lie flat, and no air hunger was reported during the night. There was no lower limb swelling. On examination, she was tachypneic with a respiratory rate of 30 breaths/minute, a respiratory exam revealed bilateral basal crepitations, and lower limb pitting edema up to the level of the ankles. At this point, a complete blood count and CRP were done, which are depicted in Table 1.

Table 1: Laboratory tests

Parameter	Result Unit	Reference range
WBC	18.56 x 10^9/L ↑	4.00 - 10.00
Neutrophils #	14.34 x 10^9/L ↑	2.00 - 7.00
Lymphocytes #	3.12 x 10^9/L	0.80 - 4.00
Neu%	77.3% ↑	50.0 - 70.0
Lymph%	16.8% ↓	20.0 - 40.0
Hemoglobin	14.5g/dl	11.0 - 16.0
RBC	4.70 x 10^12/L	3.50 - 5.50
Plt	398 x 10^9/L ↑	100 - 300
CRP	200mg/mL ↑	0.5 - 10

The patient was treated for bacterial pneumonia and was given IV cefepime 1g 12 hourly for 5 days. On day 3 of the antibiotics, she still had a persistent fever and chest pain, and the difficulty in breathing worsened with activity and she was unable to lie flat. A CBC was repeated and a chest X-ray was done as shown below in Table 2. A blood culture was done, which showed no bacterial growth.

Table 2: Complete blood count (Table 2 should appear where it is currently)

Parameter	Result Unit	Reference range
WBC	14.58 x 10^9/L ↑	4.00 - 10.00
Neutrophils #	11.18 x 10^9/L ↑	2.00 - 7.00
Lymphocytes #	1.63 x 10^9/L	0.80 - 4.00
Neu%	76.7% ↑	50.0 - 70.0
Lymph%	11.2% ↓	20.0 - 40.0
Hemoglobin	12.6g/dl	11.0 - 16.0
RBC	4.14 x 10^12/L	3.50 - 5.50
Plt	316 x 10^9/L ↑	100 - 300

Chest X-ray showed:
Mild left basal pleural effusion and overlying lower zone parenchymal haziness-likely inflammatory. Right lower zone lung markings are accentuated. (Figure 1)

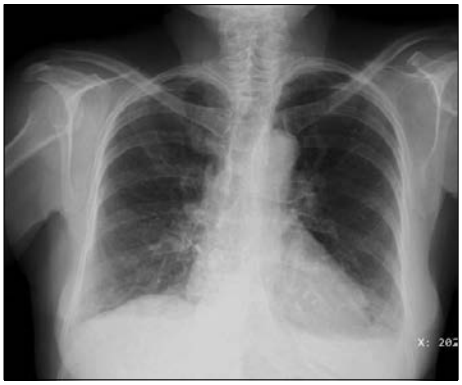

Fig 1: PA view, chest X-ray

Antibiotics were changed and she was given IV ceftriaxone + Salbactam 1.5g 12 hourly and IM Diclofenac 75mg prn for five days. She had no improvement and was then given oral doxycycline 100mg bd and cefixime 400mg bd for 5 days. There was no clinical improvement. A CBC and CRP were repeatead (Table 3).

Table 3: Laboratory tests

Parameter	Result Unit	Reference range
WBC	17.61 x 10^9/L ↑	4.00 - 10.00
Neutrophils #	13.21 x 10^9/L ↑	2.00 - 7.00
Lymphocytes #	2.63 x 10^9/L	0.80 - 4.00
Neu%	75.0% ↑	50.0 - 70.0
Lymph%	14.9% ↓	20.0 - 40.0
Hemoglobin	12.5g/dl	11.0 - 16.0
RBC	4.20 x 10^12/L	3.50 - 5.50
Plt	467 x 10^9/L ↑	100 - 300
CRP	58.0mg/L ↑	0.5 - 10
ESR	60mm/hr ↑	0 - 20

At this point, the patient had difficulty breathing at rest and was unable to lie flat. Her lower limb swelling progressed to the pretibial level. She had easy fatigability and loss of appetite. On physical examination, she was an elderly woman, ill-looking, not pale, and tachypneic, vitals; PR 109/min, RR 34/min, BP 107/70mmHg, SPO2 96% on room air. Strong regular pulse, rate 109/min, synchronous with radial and femoral pulses, JVP raised, precordial findings showed normal findings. The liver was tender on deep palpation, with smooth borders, Hepatojugular reflux was positive, and lung bases had bilateral basal crepitations.
She was then referred to a higher center where CBC, CRP, serum creatinine, serum electrolytes, serum albumin, lipid profile, liver function tests, and urine analysis were done and were within the normal range. Serological testing for HIV, HepBsAg, and Anti-HCV were negative. NT proBNP was 10625pg/ml. additionally, ECHO, CT scan of the chest, and abdominopelvic USS were done.
The ECHO showed dilated left ventricle, moderate left ventricular impairment, anterior and anteroseptal severe hypokinesia, no left ventricular hypertrophy, moderate mitral regurgitation, aortic valve mildly calcific, trivial AR, moderate to severe TR, right heart normal in size RVSP 34 + 10mmHG, pleural effusion. Ejection fraction of 40% (figure 2)

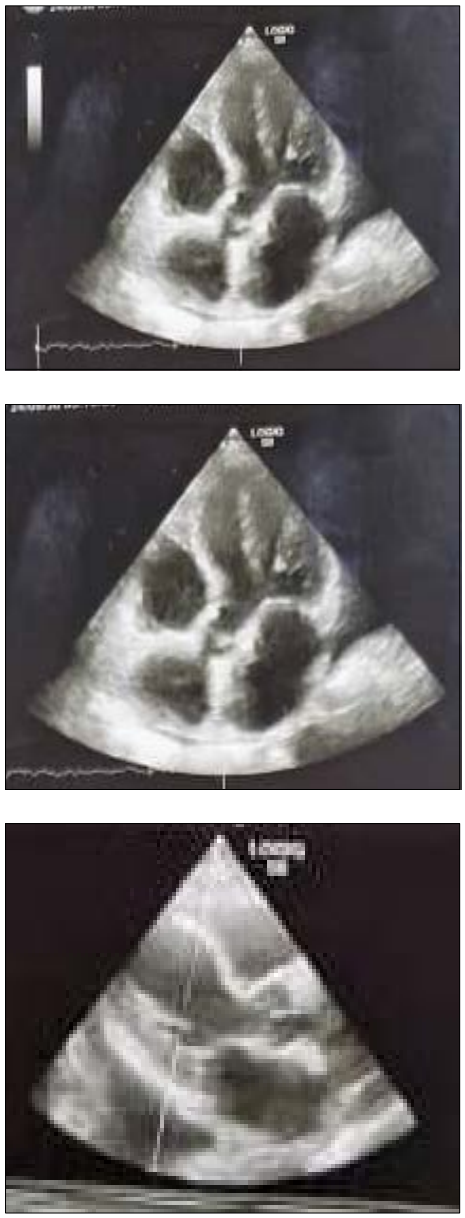

Fig 2: ECHO, Impression: ischemic cardiomyopathy, likely of a recent ACS.

CT SCAN showed patchy consolidation of the right apical region and mixed-type perihilar vasculature mimicking bat-wing appearance. There was also right basal patchy posterior segmental consolidation with a deposit of calcification and left lower lobe atelectatic consolidation. Suggestive of pulmonary congestion due to heart failure. Cardiomegaly and bilateral moderate pleural effusions.
Abdominal pelvic ultrasound showed prominent hepatic and IVC, venocongestion. Bilateral significant pleural effusions.
Furthermore, a 2D ECHO with color Doppler, a coronary angiography, and a cardiac PET viability scan were done and the results were as follows;
2D ECHO with color Doppler; EF 40%. Sclerosed aortic valve, with mildly dilated left atrium. The left ventricle showed a heart rate of 106 bpm. Minimally thinned out anterolateral wall, hypokinetic. Otherwise normal LV size and moderate dysfunction. Mild to moderate MR, mild TR.
A coronary angiography was done (figure 3);

- **LMCA:** Normal, bifurcates into LAD and LCX.
- **LAD:** Proximal segment has chronic total occlusion with a faint anterograde filling of mid-Distal LAD and D1
- **LCX:** Non-dominant. Proximal LCX has mild irregularities. Distal LCX and OM's - normal
- **RCA:** right dominant system. Mild RCA has mild irregularities. The rest of RCA, PDA, and PLB are normal. RCA giving collaterals to mid and distal LAD.
- Conclusion**:** right dominant system CTO of proximal LAD.

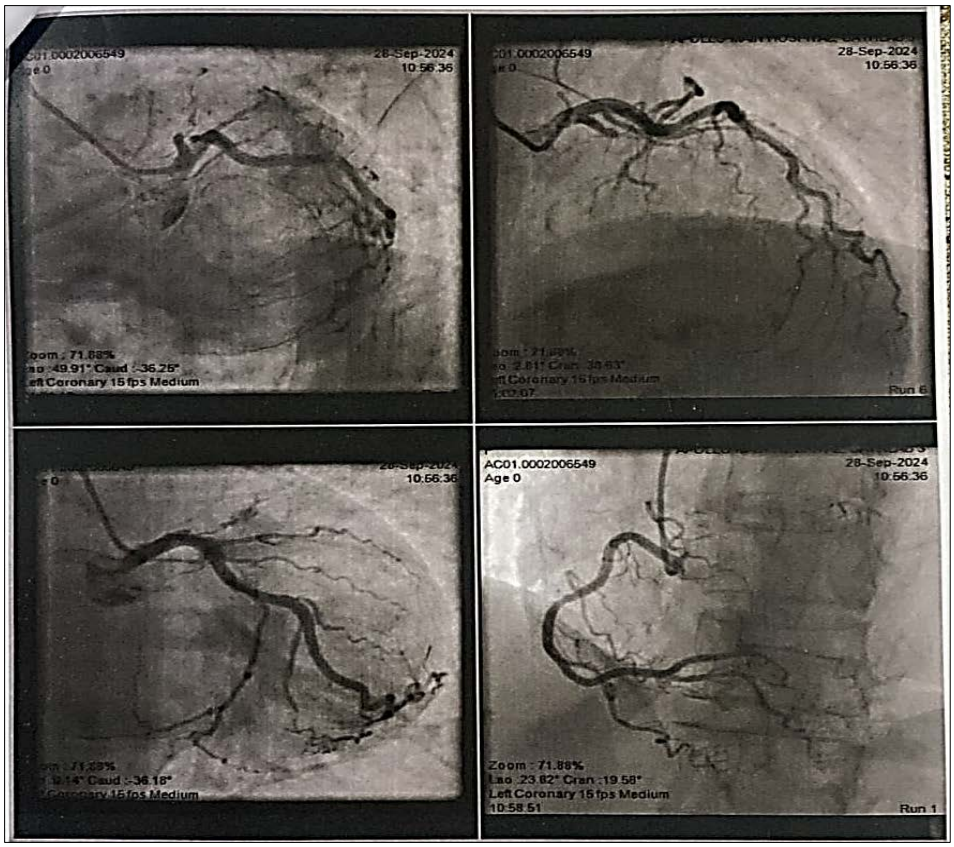

Fig 3: CT angiography

A cardiac PET VIABILITY SCAN showed perfusion defects in the apex, adjoining apical segments, mid anterior and part of the basal anterior, almost the entire septum, and part of the anterolateral segments of the left ventricle. Perfusion defect is approximately 50% of the total LV myocardium. In conclusion, matched areas with perfusion and metabolic defects involving the apex, adjoining apical segments, mid-anterior part of the basal anterior, almost entire septum, and part of anterolateral segments of the LV myocardium are suggestive of scarred (non-viable) myocardium.
The patient was then put on antifailure medication; Oral furosemide 20mg once a day, oral clopidogrel 75mg once a day, Sacubitril and Valsartan 50mg once a day, Eplerenone 25mg once a day, bisoprolol 2.5mg once a day and atorvastatin 20mg nocte. There was significant improvement 4 weeks later in the lower limb edema and breathing pattern.

Discussion

This case presents a 73-year-old female with initially vague symptoms of fever, chest pain, and fatigue, which led to a diagnostic delay in identifying her underlying heart failure. Despite initial treatment for pneumonia, her condition worsened, revealing heart failure with ischemic cardiomyopathy. This case highlights the diagnostic challenges clinicians face, particularly in elderly patients with atypical presentations, and underscores the importance of early recognition and multimodal diagnostic approaches.

Diagnostic Challenges and the Role of Multimodal Imaging

Heart failure often presents with nonspecific symptoms such as dyspnea, fatigue, and chest pain, making it challenging to diagnose, especially when these symptoms overlap with other conditions like pneumonia. A recent study by Vogel *et al.* emphasized the difficulty in distinguishing between

infectious causes and heart failure, particularly in patients with concurrent respiratory symptoms [1]. The patient's initial presentation of fever and chest pain, coupled with abnormal inflammatory markers (WBC, CRP), could easily have been attributed to bacterial pneumonia. However, the lack of response to antibiotics and the persistence of symptoms prompted further investigation, ultimately revealing a cardiac cause.

The role of imaging in the diagnostic work-up of heart failure is well established. A study done by Wong *et al*. concluded that echocardiography, along with imaging techniques like CT and PET scans, is critical in diagnosing the etiology of heart failure, especially in cases where the cause is not immediately apparent [2]. In this case, the patient's echocardiogram, which revealed left ventricular dilatation, hypokinesia, and a reduced ejection fraction of 40%, coupled with coronary angiography showing a chronic total occlusion (CTO) of the proximal LAD, confirmed the diagnosis of ischemic cardiomyopathy. Additionally, the cardiac PET scan's identification of scarred, non-viable myocardium provided valuable information for understanding the extent of myocardial damage.

Heart Failure Pathophysiology: Ischemic Cardiomyopathy and Its Implications

Ischemic heart disease is the most common cause of heart failure, and it remains a major global health challenge[3]. A recent meta-analysis by He X *et al*. found that ischemic cardiomyopathy accounts for approximately 40-60% of all heart failure cases, particularly in patients with risk factors such as hypertension, diabetes, and a history of coronary artery disease [3]. In this case, the patient's coronary angiogram revealed a chronic total occlusion of the LAD artery, consistent with the ischemic etiology of her heart failure. The findings of the cardiac PET scan supported this, showing areas of myocardial scar, which were indicative of previous infarction.

The pathophysiology of ischemic cardiomyopathy involves the loss of myocardial tissue due to sustained ischemia, leading to ventricular remodeling and impaired systolic function. Another study by Kubo *et al*. reviewed the long-term prognosis of patients with ischemic cardiomyopathy and emphasized the role of interventions such as revascularization, pharmacotherapy, and heart failure management in improving survival [4]. In this case, the patient's coronary artery disease was not amenable to revascularization, due to an irreversible risk of death on the operating table; highlighting the importance of optimal medical management to prevent further progression of heart failure.

Management of Heart Failure in the Elderly

The management of heart failure in elderly patients presents unique challenges due to comorbidities, polypharmacy, and frailty. Guidelines from the American College of Cardiology [5] recommend guideline-directed medical therapy (GDMT) for all patients with heart failure with reduced ejection fraction (HFrEF), including angiotensin-converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs), beta-blockers, and mineralocorticoid receptor antagonists (MRAs) [6]. In this case, the patient was started on Sacubitril/Valsartan, a combination drug shown to reduce hospitalization and mortality in heart failure [6]. Additionally, diuretics like furosemide were used to manage fluid overload, and antiplatelet therapy with clopidogrel was initiated to prevent thrombotic complications due to her coronary artery disease.

The role of Sacubitril/Valsartan in heart failure management is well supported by studies such as McMurray et.al, who demonstrated that Sacubitril/Valsartan reduced the risk of death from cardiovascular causes and hospitalization for heart failure in patients with HFrEF [6]. Furthermore, Gheorghiade *et al*. found that beta-blockers and MRAs significantly improve outcomes in heart failure patients, even in elderly populations [7]. This case exemplifies how the use of these therapies can improve symptoms and prevent further cardiac damage, even in advanced stages of heart failure.

Prognosis and Long-Term Management

The long-term prognosis of patients with ischemic cardiomyopathy is generally poor without appropriate intervention. Anker *et al*. reviewed the survival outcomes of patients with ischemic heart failure and noted that timely medical intervention, including the use of ACEIs, beta-blockers, and MRAs, significantly improves long-term survival [8]. The patient in this case was started on a comprehensive heart failure regimen, which will require ongoing monitoring to assess response to therapy, manage complications, and adjust medications as necessary.

Conclusion

This case underscores the importance of considering heart failure, particularly ischemic cardiomyopathy, in the differential diagnosis of elderly patients with nonspecific symptoms. The role of multimodal imaging in diagnosing and managing heart failure is critical, as it allows for a comprehensive evaluation of myocardial function, coronary anatomy, and the extent of damage. While the initial diagnosis was delayed due to the presentation of respiratory symptoms, the subsequent diagnostic work-up provided crucial information for targeted therapy, which included guideline-directed medical therapy and symptom management. Early recognition and appropriate management of heart failure can significantly improve outcomes and reduce the morbidity associated with this condition.

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Author Contributions

S.A.M.: Conceptualization, case investigation, writing—original draft preparation.

W.C.L.: Supervision, writing-review and editing.

All authors reviewed and approved the final manuscript.

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Competing interests

The authors declare no competing interests.

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Not applicable.

Clinical trial number
Not applicable.

Consent for publication
Consent for publication was obtained from the patient. This included clinical details, laboratory and radiology reports.

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