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Case Report

Acute Decompensated Heart Failure with Severe Left Ventricular Systolic Dysfunction of Undetermined Etiology in a 26-Year-Old Male: A Case Report

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ARTICLE INFO

Article history:

Received 23 Apr. 2026

Received in revised form 8 Jun. 2026

Accepted 30 Jun. 2026

Published 18 Aug. 2026

Keywords:
Heart failure with reduced ejection fraction

Undetermined etiology cardiomyopathy

Ischemic cardiomyopathy

Acute pulmonary edema

Hypertension

ABSTRACT

Heart failure with reduced ejection fraction (HFrEF) is rare in young adults but may develop when multiple uncontrolled cardiovascular risk factors are present. The diagnosis of an underlying cause of heart failure among younger patients is difficult due to multiple possible etiologies of LV systolic dysfunction, which might have similar clinical manifestations and imaging features. A 26-year-old man with a past medical history of uncontrolled hypertension and smoking, who came to hospital with complaints of sudden onset of dyspnea and chest pain on the left side, was investigated further to have sinus tachycardia with abnormal R-wave progression on electrocardiogram, cardiomegaly with pulmonary congestion on chest X-ray, and significantly impaired global systolic function of the left ventricle with ejection fraction of 25% on transthoracic echocardiogram. Troponins showed no dynamic change in their rise-and-fall pattern, ruling out acute coronary syndrome. He was stabilized on CPAP breathing and IV nitroglycerine and loop diuretics, with marked relief in symptoms. Following hemodynamic stabilization, guideline-directed medical therapy for HFrEF was initiated. In this case, there is acute decompensated heart failure with reduced ejection fraction (HFrEF) characterized by severe systolic dysfunction occurring in a relatively young individual suffering from numerous risk factors for cardiovascular disorders. The etiology of the condition could not be precisely determined due to the unavailability of coronary angiography and other advanced diagnostic methods. Ischemic cardiomyopathy is just one of the potential causes that also include hypertensive cardiomyopathy, idiopathic dilated cardiomyopathy, toxic cardiomyopathy, and inflammatory cardiomyopathy. The present report highlights the need for careful etiologic assessment of patients presenting with recently developed ventricular dysfunction, appropriate diagnosis in the absence of evidence of coronary disease, and prompt initiation of appropriate therapy.

1. Introduction

The number of people affected by heart failure globally continues to increase significantly, leading to morbidity, disability, and deaths [1]. This has been attributed to an increasing prevalence of risk factors for cardiovascular disease that can be modified [2]. Among the types of heart failure, heart failure with reduced ejection fraction (HFrEF) is very prevalent. HFrEF occurs when there is a decrease in ventricular systolic function. Ischemic cardiomyopathy results from long-term myocardial ischemia and infarction due to coronary artery disease [3]. Ischemic cardiomyopathy is one of the most prevalent causes of HFrEF among older populations [4, 5]. Myocardial injury caused by ischemia eventually leads to ventricular remodeling, resulting in systolic dysfunction. While ischemic cardiomyopathy mostly affects the middle-aged and elderly population, early-onset coronary artery disease is becoming more frequent among younger people, owing to the high prevalence of risk factors such as smoking, high blood pressure, and diabetes [6, 7].

The INTERHEART study revealed that a limited number of reversible risk factors, such as smoking, high blood pressure, abnormal lipids, and psychosocial stress, are responsible for the majority of myocardial infarction risk among populations from 52 different countries and various demographics [8]. The accumulation of these risk factors early on can contribute to heart damage and the development of heart failure among younger patients. According to the literature, acute decompensated heart failure occurs when there is sudden onset or progression of symptoms like dyspnea, orthopnea, paroxysmal nocturnal dyspnea, and pulmonary congestion [9]. Radiographic findings of cardiogenic pulmonary edema include cardiomegaly, Kerley B lines, and the appearance of a "bat-wing" perihilar pattern of alveolar edema [10]. Transthoracic echocardiography remains the mainstay for assessing cardiac morphology and ventricular function [11].

Determination of the cause of the left ventricular systolic dysfunction in younger patients poses a considerable diagnostic difficulty as various conditions such as hypertensive, ischemic, idiopathic dilated, inflammatory, and toxic cardiomyopathy may present with identical symptoms. The important consideration here is that the absence of imaging test results, infarction, and abnormal motion of the affected

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Citation: Asif AE, Butt AM, Hussain ST, Ibrahim A, Javaid MH. Acute Decompensated Heart Failure with Severe Left Ventricular Systolic Dysfunction of Undetermined Etiology in a 26-Year-Old Male: A Case Report. ASIDE Cardiovasc. 2026;1(2):19-25, doi:10.71079/ASIDE.CV.081826789

area makes it impossible to confirm ischemic cardiomyopathy; it can only be termed suspected or possible. Here we consider the case of a 26-year-old man with acute decompensation of HFrEF as a consequence of significant left ventricular systolic dysfunction of unknown origin, and illustrate the clinical, diagnostic, and therapeutic approach in a resource-limited setting.

2. Case Presentation

A 26-year-old male was brought to the emergency department on December 20, 2025, due to acute onset of shortness of breath associated with orthopnea and paroxysmal nocturnal dyspnoea, with concomitant left-sided chest pain of eight hours' duration. No symptoms like nausea, vomiting, palpitations, syncope, or diaphoresis were seen.

History of hypertension of two years' duration, managed by an amlodipine/valsartan combination 5 mg/80 mg taken intermittently. He reported having chest pain twice in the past that he termed a "heart attack" due to self-management of medication when there is relief from symptoms. Documentation for any such episode, including ECG tracing, troponin levels at different time points, angiographic findings, any interventions, and discharge prescriptions, in support of an acute coronary syndrome (ACS) diagnosis, was not present; hence, these are considered self-reported suspected ACS cases. There was a history of admission for acute pulmonary edema twice and meningoencephalitis once during the year 2025 in his report. Family history was positive for hypertension in both parents and one sibling. He had been smoking cigarettes and snuff for four years.

Findings from physical examination showed that the patient was suffering from acute breathlessness and distress. The following findings were noted regarding his vital signs: blood pressure 150/100 mmHg, pulse 110 beats/min, respiratory rate 24 breaths/min, and afebrile. Bilateral crackles were noted in the mid lung zone. No jugular venous distension was noted, nor was any evidence of lower limb edema. No extra heart sounds or murmurs were present at the first assessment. The abdomen was soft and non-tender.

The chest X-ray (**Figure 1**) showed cardiomegaly with bilateral hilar haziness, Kerley B lines, and batwing appearance of alveolar infiltrates suggestive of cardiogenic pulmonary edema. Electrocardiography (**Figure 2**) showed sinus tachycardia with non-progressing R waves in anterior precordial leads (V1 to V4). Acute ST-segment elevation, pathologic Q waves, or hemodynamically significant arrhythmia was not noted.

2.1. Investigations

Serial troponin-I was performed to rule out acute myocardial injury. The initial result was 0.02 ng/mL (reference lab value: 0.16 ng/mL). Another test was performed 6 hours after admission and yielded 0.03 ng/mL, still below the cutoff level. Another test, twelve hours after admission, showed a level of 0.02 ng/mL. In all these results, there was no evidence of rise and fall of levels characteristic of STEMI or NSTEMI. Therefore, there was no diagnosis of acute type 1 myocardial infarction based on the kinetics of troponin. It may be considered that there was a completed myocardial infarction with normalized troponin, especially with consideration of the medical history given by the patient; however, no objective documentation of prior ischemic events was available.

The basic metabolic panel showed no electrolyte abnormalities and intact renal function (normal serum creatinine), allowing initiation of medically indicated therapy. No anemia or leukocytosis was found in the complete blood count. Liver function tests were normal. Importantly, however, several critical laboratory studies were not performed due to financial limitations, namely: fasting lipids, hemoglobin A1c levels, thyroid hormones, markers for viral infections (coxsackievirus, parvovirus B19, and adenovirus), autoimmune antibodies, and a urine toxicology screen. Testing for brain natriuretic peptide (BNP)/NT-proBNP was not done. The lack of these tests is a major disadvantage in diagnosing the cause of cardiomyopathy.

2.2. Echocardiographic Findings

Transthoracic echocardiography revealed a slight enlargement of the left ventricle along with mild concentric hypertrophy of the left ventricle. There was severe impairment of the systolic function of the left ventricle, with a biventricular Simpson estimation of the left ventricle's ejection fraction of 25%. Importantly, there were no regional abnormalities in left ventricular wall motion within the coronary artery territories; instead, there was diffuse, global hypokinesis. This presentation is less indicative of ischemic cardiomyopathy, which causes regional wall abnormalities. It rather points towards a possible diagnosis of hypertensive, idiopathic dilated, inflammatory, or toxic cardiomyopathy, although this does not rule out an ischemic process. Mild dilation of the left atrium was seen. Normal dimensions and functions were observed for both the right atrium and ventricle. Mitral valve insufficiency and mild tricuspid regurgitation were present. There was no pericardial effusion.

2.3. Differential Diagnosis

In view of the clinical and echocardiographic findings, it was impossible to make an etiological diagnosis. Below are some differential diagnoses to be considered:

Hypertrophic Cardiomyopathy: A strong possibility due to a two-year history of poorly controlled hypertension, with mild concentric LV hypertrophy and LV global dysfunction. This is the most plausible diagnosis in light of the information available. Dilated Cardiomyopathy: A possibility due to LV global hypokinesia without regional involvement. Idiopathic and genetic causes are still possible. Ischemic Cardiomyopathy: Although premature coronary artery disease may be seen in patients with risk factors, there were no imaging studies performed for the coronary arteries.

The lack of regional wall motion abnormalities and the indeterminate nature of troponin kinetics make this a probable, rather than a confirmed, diagnosis. Acute severe left ventricular dysfunction associated with myocarditis is well documented in young adults. No viral antibody tests were performed; a cardiac MRI, needed to evaluate for myocardial inflammation or late gadolinium enhancement, was unavailable. Toxic cardiomyopathy: There was no systematic history-taking regarding alcohol consumption.

2.4. Management

As soon as the patient arrived in the emergency room, CPAP ventilation therapy and urinary catheter insertion were initiated to ensure proper fluid balance. Nitroglycerine intravenously, along with furosemide 80 mg IV, was given to the patient. These measures helped in achieving immediate and impressive improvement in symptoms. Dyspnea resolved, and there was no sign of lung congestion. The patient was transferred to the coronary care unit.

After hemodynamic stability was achieved and there was evidence of adequate renal function, guideline-directed medical therapy for HFrEF was initiated: sacubitril/valsartan 50 mg BD, metoprolol succinate 25 mg OD, spironolactone 25 mg OD, dapagliflozin 10 mg OD, and rosuvastatin 20 mg OD. Co-administration of

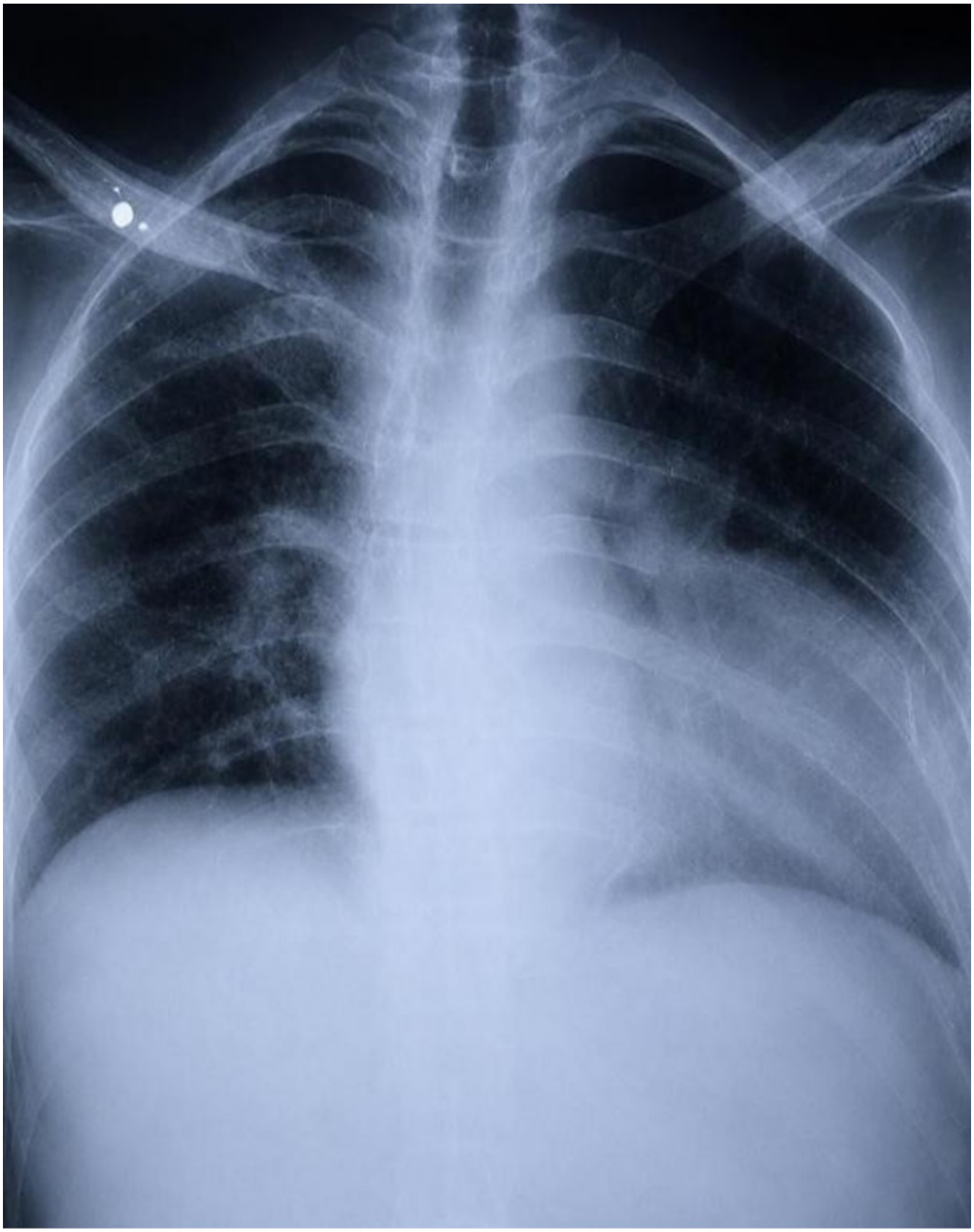

Figure 1: Posteroanterior chest radiograph obtained at admission. The image demonstrates cardiomegaly (cardiothoracic ratio >0.5), bilateral perihilar alveolar infiltrates in a "bat-wing" pattern, and horizontal linear opacities at the lung bases consistent with Kerley B lines, in keeping with cardiogenic pulmonary edema. All patient and institutional identifiers have been removed from the final image before submission.

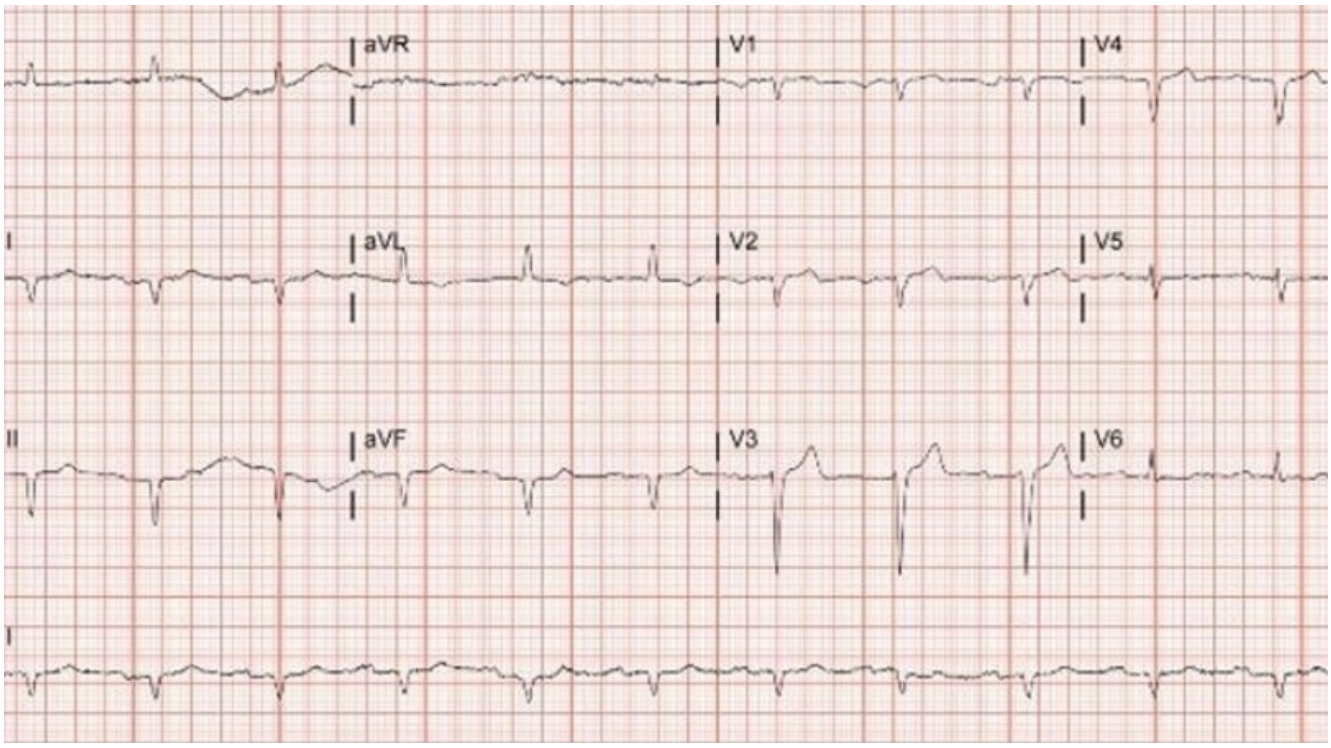

Figure 2: Twelve-lead electrocardiogram obtained at admission. The tracing demonstrates sinus tachycardia (rate approximately 110 bpm) with poor R-wave progression in the anterior precordial leads (V1–V4), suggesting anterior inactivation or prior anterior injury. No acute ST-segment elevation, pathological Q waves, or significant arrhythmia are identified. The ECG pattern is consistent with left ventricular dysfunction but is nonspecific and does not establish a diagnosis of acute myocardial infarction or confirm prior ischemic events. All patient and institutional identifiers have been removed from the final image before submission.

pantoprazole 40 mg OD was considered necessary to minimize gastrointestinal adverse effects. Regarding antiplatelet therapy: aspirin 75 mg daily and clopidogrel 75 mg daily were administered based on the presumed history of past acute coronary syndromes and considering the high cardiovascular risk profile of the patient. It is important to consider that, since the presence of confirmed ACS or obstructive coronary disease is unconfirmed at this admission, the treating team should review the use of dual antiplatelet therapy (DAPT), with documentation of the indication for such therapy, its intended duration, and a risk-benefit analysis. Where ACS cannot be confirmed, a review of the indication for dual antiplatelet therapy will be necessary during outpatient follow-up based on coronary assessment. The duration of dual antiplatelet therapy was not predefined at discharge and was planned to be reassessed following outpatient coronary evaluation.

It is important to differentiate between acute stabilization and treatment of disease processes. Acute recovery from the symptoms that led to the admission to the hospital can be traced to decongestion using positive pressure ventilation, nitrates, and intravenous diuretics. Sacubitril/valsartan, beta-blockers, mineralocorticoid receptor antagonists, and SGLT-2 inhibitors have been associated with improved outcomes in HFrEF but do not confer symptomatic benefit.

2.5. Discharge Status

Improvement was observed in the patient's clinical course during his three-day hospital stay. On discharge, the patient was able to walk around without any dyspnea. His blood pressure was 130/85 mmHg, and his resting heart rate was 82 beats per minute while on medication. The results of his tests for kidney function and potassium levels before starting sacubitril/valsartan and spironolactone were normal (the numerical readings could not be quantified, which is a limitation of this report).

The patient had been successfully weaned off oxygen supplementation and had been tolerating ambient air saturation levels adequately before his discharge from the hospital. His urine output had been satisfactory while he was receiving his diuretics, but no precise figures on his fluid balance were recorded. Unfortunately, this information was not fully reflected in the original case notes, and its absence constitutes a known limitation of this case study.

Table 1 presents a timeline of the case, based on the patient's self-reports during the index admission. There were no records other than those from the patient himself to verify the sequence of events. The prior history of chest pain described by the patient as "heart attacks" will be considered cases of self-reported suspected acute coronary syndrome.

3. Discussion

In this particular scenario, an example of acute heart failure has been provided in the form of a case where a 26-year-old male had presented with acute decompensation of HFrEF resulting from significant systolic dysfunction in the left ventricle. However, it could not be determined whether there was a specific cause of such heart failure. In other words, the condition can be considered as heart failure of unknown cause, as opposed to the term ischemic cardiomyopathy that may have also applied to this patient.

Table 1: Self-Reported Timeline of Clinical Events

Date / Period	Clinical Event	Symptoms / Findings	Evaluation / Documentation	Treatment / Outcome
Approximately 2023	Diagnosis of hypertension	Recurrent headaches and elevated blood pressure	Diagnosed locally; exact records unavailable	Started on amlodipine/valsartan (5 mg/80 mg); poor medication adherence reported
July 2023	First self-reported suspected ACS episode	Severe chest pain with shortness of breath	No ECG, troponin, or angiographic documentation available; event classification based on patient self-report only.	Symptomatic treatment at local clinic; discharged without documented cardiac workup; no revascularization or ACS-consistent discharge prescriptions on record
2024	Hospital admission for meningoencephalitis	Fever and neurological symptoms	Details unavailable; no cardiac evaluation documented	Treated conservatively; full recovery reported
During 2025 (before current admission)	Two admissions for acute pulmonary edema	Acute dyspnea and respiratory distress	No echocardiographic or angiographic records available; events based on patient self-report	Managed with supportive and decongestive therapy; outcomes and discharge medications unknown.
May 2025	Second self-reported suspected ACS episode	Chest pain requiring hospital visit	No catheterization or coronary imaging records available; no ECG or troponin documentation; event classification based on patient self-report only	Medical management reported; discharge medications and revascularization status unknown.
December 20, 2025	Current presentation with acute decompensated heart failure	Sudden severe breathlessness, orthopnea, paroxysmal nocturnal dyspnea, and left-sided chest pain	Chest X-ray: cardiomegaly, perihilar haziness, Kerley B lines. ECG: sinus tachycardia, poor R-wave progression, no acute ST elevation. Serial troponin-I: 0.02, 0.03, 0.02 ng/mL (non-diagnostic). Echo: global LV hypokinesia, EF 25%, no regional wall-motion abnormality	CPAP ventilation, IV nitroglycerine, IV furosemide 80 mg; rapid symptomatic improvement achieved
Hospital stay (3 days)	Stabilization and initiation of GDMT	Progressive improvement in dyspnea and pulmonary congestion; ambulatory at discharge	Severe global LV systolic dysfunction confirmed (EF 25%); renal function and electrolytes within acceptable limits at discharge.	Sacubitril/valsartan 50 mg BD, metoprolol succinate 25 mg OD, spironolactone 25 mg OD, dapagliflozin 10 mg OD, rosuvastatin 20 mg OD, aspirin 75 mg OD, clopidogrel 75 mg OD, pantoprazole 40 mg OD
Post-discharge	Follow-up unavailable	—	No outpatient records available	Long-term outcome unknown

Abbreviations: ACS, acute coronary syndrome; BD, twice daily; CPAP, continuous positive airway pressure; ECG, electrocardiogram; EF, ejection fraction; GDMT, guideline-directed medical therapy; IV, intravenous; LV, left ventricular; OD, once daily.

Notably, the echocardiogram results, which demonstrate generalized hypokinesia without regional wall motion abnormalities, are more suggestive of non-ischemic cardiomyopathy. Traditionally, in ischemic cardiomyopathy, wall motion abnormalities can be mapped to the territories supplied by the coronary arteries. Global dysfunction is suggestive of hypertensive, dilated, inflammatory, or toxic cardiomyopathy. In this case, the troponin results were negative for any diagnosis, and an acute coronary syndrome could not be confirmed. Previous ischemic episodes cannot be ruled out, but there is no evidence to document their occurrence.

Self-reported history of any previous episodes of chest pain needs proper interpretation. Although these were attributed to "heart attacks" by the patient, lack of supporting evidence from ECG during the episode(s), serial troponins, coronary angiogram findings, evidence of revascularization, and discharge medications in line with ACS would preclude classification as documented coronary artery disease. Any misclassification would have significant implications for diagnosis, dual antiplatelet therapy, and subsequent management in this particular case.

Limited resources constitute another important contextual consideration within the current scenario. Advanced heart scanning techniques such as coronary angiography, coronary CT angiography, and cardiac magnetic resonance imaging may not be available or affordable in low- and middle-income nations. Consequently, a provisional diagnosis of heart disease must be based on clinical examination, bedside echocardiography, and risk factor assessment, and must guide the initiation of appropriate treatment.

Where cardiac MRI is available, it remains the gold standard for differentiating ischemic from non-ischemic cardiomyopathy based on late gadolinium enhancement patterns in the myocardium. In ischemic cardiomyopathy, there is subendocardial or transmural late enhancement in a coronary territory pattern, whereas in inflammatory cardiomyopathy, the enhancement occurs at the level of midwall or epicardium. The investigation to rule out/confirm coronary artery disease would include either a coronary angiogram or a coronary CTA. These are an essential part of this patient's follow-up.

Several etiologies require individual discussion. The possibility of hypertensive cardiomyopathy is substantiated by the presence of two years of uncontrolled hypertension, mild concentric LVH, and recurrent episodes of acute pulmonary edema, one of the well-known phenotypes of pressure-overload cardiomyopathy that develops into dilated-phase hypertensive heart disease. As far as idiopathic dilated cardiomyopathy that presents with similar symptoms and global hypokinesia, its differential diagnosis is needed with exclusion of any other etiology and, possibly, genetic testing due to the positive family history of cardiovascular disorders. The diagnosis of myocarditis needs to be considered in young patients with recent onset of LV dysfunction because viral cardiomyopathy could result from previous viral infections, even though the previously experienced meningoencephalitis does not mean that it was caused by it. The role of toxic cardiomyopathy due to the abuse of stimulants and illicit drugs, as well as that of alcohol cardiomyopathy, was also not ruled out in this particular patient.

This particular case further emphasizes the importance of the association of lack of adherence with early cardiovascular disease. Young patients suffering from diseases such as hypertension show very low levels of adherence to their medications, as they stop taking their medications once the symptoms abate, something that has been observed in this particular patient, and might contribute to myocardial damage. Registry data have confirmed increasing rates of cardiovascular disease in younger adults globally [6].

HFrEF treatment strategies today rely on four evidence-based medication classes: angiotensin receptor–neprilysin inhibitors, beta-blockers, mineralocorticoid receptor antagonists, and sodium glucose cotransporter 2 inhibitors [3, 12]. Sacubitril/valsartan was shown to be superior to enalapril with respect to cardiovascular mortality and HF admissions, as shown by the PARADIGM-HF trial [13]. Similarly, empagliflozin was found to be effective in reducing cardiovascular mortality and HF hospitalizations among HFrEF patients in the EMPEROR-Reduced trial [14]. All four medications have been prescribed to the patient after hemodynamic stabilization, consistent with current guidelines.

Follow-up studies among young patients with severe left ventricular systolic dysfunction are critical in determining the effectiveness of their medications and whether they are following their medical advice, performing their evaluation, and assessing them for eligibility for device therapy. It is currently recommended that an assessment of the LV ejection fraction be performed three to six months after optimizing their medical management regimen if there is still severe systolic dysfunction (LVEF $\leq$35%) despite optimal therapy, which may warrant consideration of an implantable cardioverter-defibrillator for primary prevention of sudden cardiac death.

4. Conclusion

This is an example of acute decompensation of HFrEF associated with severe impairment of the function of the left ventricle in the context of multiple cardiovascular risk factors in a young male patient, namely poorly controlled hypertension and active smoking. The underlying etiology is unknown in this case, primarily due to the lack of coronary imaging and other sophisticated diagnostic tests. Ischemic cardiomyopathy is a possibility, but without coronary angiography, we cannot confirm that diagnosis; hypertensive cardiomyopathy and idiopathic dilated cardiomyopathy are at least equally likely. Reported episodes of chest pain in the past are not documented objectively and thus cannot serve as evidence for coronary artery disease. This case illustrates several key principles of management in patients like this, such as: (1) correct diagnosis, recognizing unconfirmed ischemic cardiomyopathy as suspicious or possible but not confirmed; (2) systematic etiological investigation; (3) prompt initiation of medical treatment according to recommendations regardless of the underlying pathology; and (4) appropriate follow-up with coronary and cardiac MRI examination as soon as possible.

Conflicts of Interest

The authors have no conflicts of interest to declare.

Funding Source

No funding sources to declare.

Acknowledgments

Not applicable.

Institutional Review Board (IRB)

Ethical approval is not required for case reports at our institution.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report.

Large Language Model

No generative artificial intelligence (AI) tools or large language models were used in the preparation, writing, editing, analysis, or revision of this manuscript. The authors take full responsibility for the content of the published work.

Author Contributions

AEA, STH, and MHJ contributed to the conceptualization of the study, while AEA, AMB, STH, and AI were responsible for investigation and data collection. AEA, AMB, STH, AI, and MHJ contributed to the methodology, and AI performed the literature review. AEA, AMB, and AI assisted in drafting the manuscript, while STH and MHJ supervised the study and MHJ was responsible for project administration. All authors reviewed and approved the final manuscript.

Data Availability

Data are available from the corresponding author on request.

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