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Integrating SGLT2 Inhibitors in the Treatment Strategy for Acute Myocarditis: A Case Report

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Abstract

Background: Myocarditis is a challenging polymorphic disease with extensive variability in clinical presentation. The etiology of myocarditis often remains undetermined without an endomyocardial biopsy. Case presentation: A 25-year-old male with no prior cardiovascular history presented with acute severe retrosternal chest pain, dyspnea, fatigability, and diaphoresis, preceded by mild respiratory symptoms. Physical exam revealed stability, and ECG showed inferolateral ST-segment elevation. Laboratory tests indicated significant myocardial injury with elevated troponin and CK levels, alongside inflammatory markers. Coronary angiography excluded acute coronary syndrome, and cardiac MRI confirmed active myocarditis with characteristic late gadolinium enhancement and myocardial edema. The patient remained hemodynamically stable during hospitalization. Guideline-directed medical therapy was initiated, including a beta-blocker, an ACE inhibitor, and dapagliflozin as an SGLT2 inhibitor to support ventricular recovery and prevent remodeling. At one-month follow-up, the patient was asymptomatic with normalized biomarkers and unremarkable imaging, indicating clinical recovery. Conclusion: This case illustrates that acute myocarditis may lead to early cardiac dysfunction even in young individuals and highlights the potential benefit of adding an SGLT2 inhibitor to standard therapy to support recovery.

Keywords: *myocarditis; heart failure; SGLT-2 inhibitors*

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1. Highlights

- **Acute myocarditis may mimic acute coronary syndrome**, requiring rapid exclusion of obstructive coronary disease through multimodal evaluation.
- **Cardiac MRI remains the most sensitive non-invasive tool** for detecting myocardial edema and non-ischemic LGE patterns.
- **Young adults can develop significant myocardial injury and early functional impairment**, even in the absence of cardiovascular risk factors.
- **Initiation of guideline-directed therapy with ACE inhibitors and beta-blockers is essential** to limit adverse ventricular remodeling.
- **Adjunctive use of the SGLT2 inhibitor dapagliflozin contributed to early clinical and biomarker improvement**, suggesting a potential role in myocarditis-associated ventricular dysfunction.
- **This case supports further investigation into SGLT2 inhibitors as emerging therapeutic agents** in the management of acute myocarditis.

2. Introduction

Myocarditis is an inflammatory disease of the myocardium with a broad variability in clinical presentation, ranging from an asymptomatic course, palpitations, severe chest pain, to sudden cardiac death. There are typically three patterns

of myocarditis presentation: pseudo-infarctual chest pain, heart failure, and arrhythmias [1]. Based on these different patterns of presentation, a risk stratification model has been proposed that categorizes patients with myocarditis into three different groups: low-risk, intermediate-risk, and high-risk. Each group requires different follow-up and has a distinct prognosis [2, 3]. The etiology of myocarditis often remains undetermined. However, viral infections are responsible for the majority of myocarditis cases, but other infectious agents, drugs, toxins, and systemic diseases can also be incriminated [3, 4]. Although endomyocardial biopsy remains the gold standard for diagnosing myocarditis, its limited routine use has led to an increased reliance on advanced cardiac imaging techniques, particularly cardiac magnetic resonance. Early and accurate identification is clinically important, as approximately 20% of patients with acute myocarditis progress to dilated cardiomyopathy, in which heart failure becomes the predominant clinical presentation [1, 4]. Moreover, the risk of developing heart failure during follow-up appears to be influenced by patient age, with a higher incidence reported among individuals over 50 years compared to younger patients [5]. Given these considerations, the treatment approach for myocarditis should be guided by the pattern and severity of the clinical presentation, the immediate response to standard therapies, and any spontaneous or treatment-induced improvements [3]. In this context, the present paper reports the case of a 25-year-old patient with myocarditis who exhibited two characteristic clinical patterns of myocarditis, namely acute severe chest pain and subsequent heart failure, managed with standard therapy complemented by an SGLT2 inhibitor to support cardiac recovery.

3. Materials and Methods

This case report was prepared in adherence to institutional ethical regulations and the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from the patient for diagnostic procedures, treatment, and the use of anonymized clinical information and imaging for publication. Clinical information was obtained throughout the patient's admission to the Cardiology Department of Pius Brînzeu County Clinical Emergency Hospital, Timişoara. The diagnostic work-up comprised a comprehensive clinical assessment, including detailed medical history, physical examination, 12-lead electrocardiography (ECG), transthoracic echocardiography (TTE), serial quantification of cardiac biomarkers, invasive coronary angiography, and cardiac magnetic resonance imaging (CMR). Quantified biomarkers included high-sensitivity cardiac troponin I (hs-cTnI), creatine kinase (CK), CK-MB isoenzyme, C-reactive protein (CRP), and N-terminal pro-B-type natriuretic peptide (NT-proBNP), all processed using standardized laboratory methodologies. Echocardiographic evaluation was performed to assess global and regional ventricular systolic function, detect focal wall motion abnormalities, and exclude pericardial effusion. A coronary angiography was performed via radial access, employing standard contrast protocols, in order to exclude obstructive coronary artery disease. CMR was conducted using a 1.5-Tesla system with dedicated sequences for myocardial edema and late gadolinium enhancement. The imaging diagnosis of myocarditis was established according to the updated Lake Louise Criteria, integrating both T2-based edema markers and non-ischemic LGE distribution. Management strategies were aligned with contemporary European Society of Cardiology (ESC) clinical practice guidelines for myocarditis and heart failure, including evidence-based pharmacologic therapy aimed at optimizing cardiac function and preventing adverse remodeling.

4. Case Presentation

A 25-year-old male with no prior history of cardiovascular disease or identifiable risk factors presented to the Emergency Department of Pius Brînzeu County Clinical Emergency Hospital, Timişoara, with acute-onset, severe retrosternal chest pain that had progressively worsened over the previous 12 h. The pain was associated with dyspnea, fatigability, and profuse diaphoresis. He reported mild, nonspecific symptoms suggestive of an upper respiratory tract infection approximately three days prior to presentation. On physical examination, the patient was diaphoretic and afebrile, with stable hemodynamics and no evidence of respiratory insufficiency. Cardiac auscultation revealed regular heart sounds without murmurs. Lung examination demonstrated normal, symmetrical vesicular breath sounds without adventitious sounds. All the other findings were within normal limits.

The electrocardiography showed sinus rhythm with ST-segment elevation in the inferolateral leads without arrhythmias (**Figure 1**). Transthoracic echocardiography revealed a left ventricle with preserved ejection fraction (55%), a discrete inferior wall motion abnormality, no significant valvular disease, and no pericardial effusion.

Laboratory investigations demonstrated marked elevation of cardiac biomarkers: hs-cTnI 9312 ng/L (reference < 30 ng/L), CK 529 U/L (reference < 170 U/L), and CK-MB 62 U/L (reference < 16 U/L). Inflammatory markers were elevated, with CRP at 80 mg/L. NT-proBNP was elevated to 923 pg/mL.

Due to the dynamic rise in myocardial injury biomarkers (**Table 1**) and localized ST-segment elevations as observed in **Figure 1**, a coronary angiography was performed to exclude acute coronary syndrome. The coronary arteries were normal, with no obstructive or structural lesions (**Figure 2**).

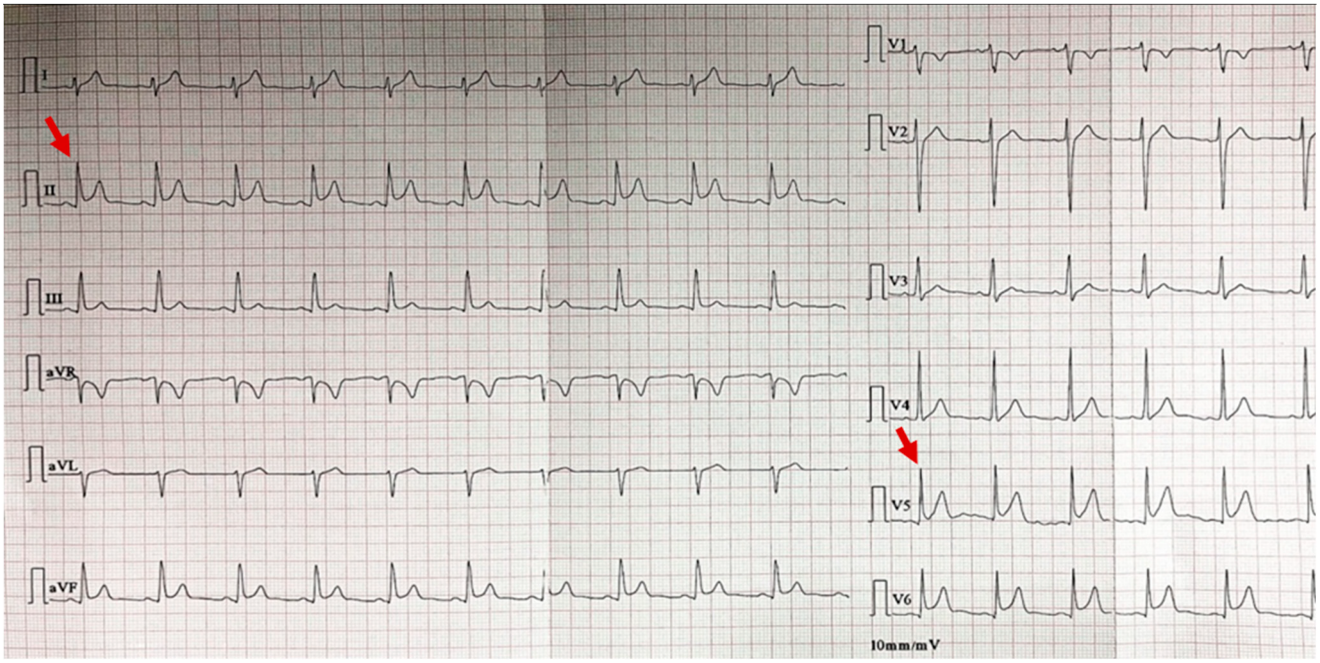


Figure 1. Electrocardiogram shows 0.5–1 mm ST-segment elevations in leads II, III, aVF, V5, and V6. The red arrows indicate ST-segment elevation.

Table 1. Serial changes in myocardial injury biomarkers.

	On Admission	At 1 h	At 3 h
hs-CTni (ng/L)	9123	12,102	21,135
CK (U/L)	529		729
CK-MB (U/L)	62		81

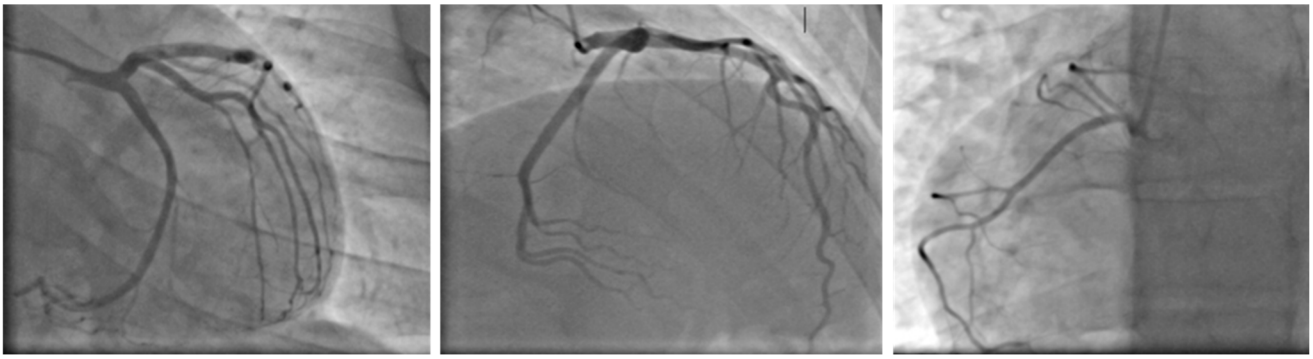


Figure 2. Coronary angiography demonstrated a predominantly left-dominant coronary system with no evidence of obstructive lesions.

Cardiac MRI demonstrated positive late gadolinium enhancement with a non-ischemic intramyocardial and subepicardial pattern affecting the lateral, inferior, and septal walls. Increased T2 relaxation times (>60 ms) confirmed persistent myocardial edema, indicative of ongoing inflammation rather than fibrotic scar, consistent with active or subacute myocarditis—**Figure 3**.

The patient remained hemodynamically stable throughout hospitalization. At discharge, a six-month therapeutic regimen was prescribed, including dapagliflozin 10 mg once daily to alleviate heart failure symptoms, particularly in the context of elevated NT-proBNP, which reflects increased ventricular wall stress; Metoprolol 25 mg twice daily, a cardioselective beta-blocker, to improve heart failure–related symptoms and reduce the risk of potential post-myocarditis arrhythmias; and Ramipril 2.5 mg once daily, an ACE inhibitor to prevent adverse ventricular remodeling.

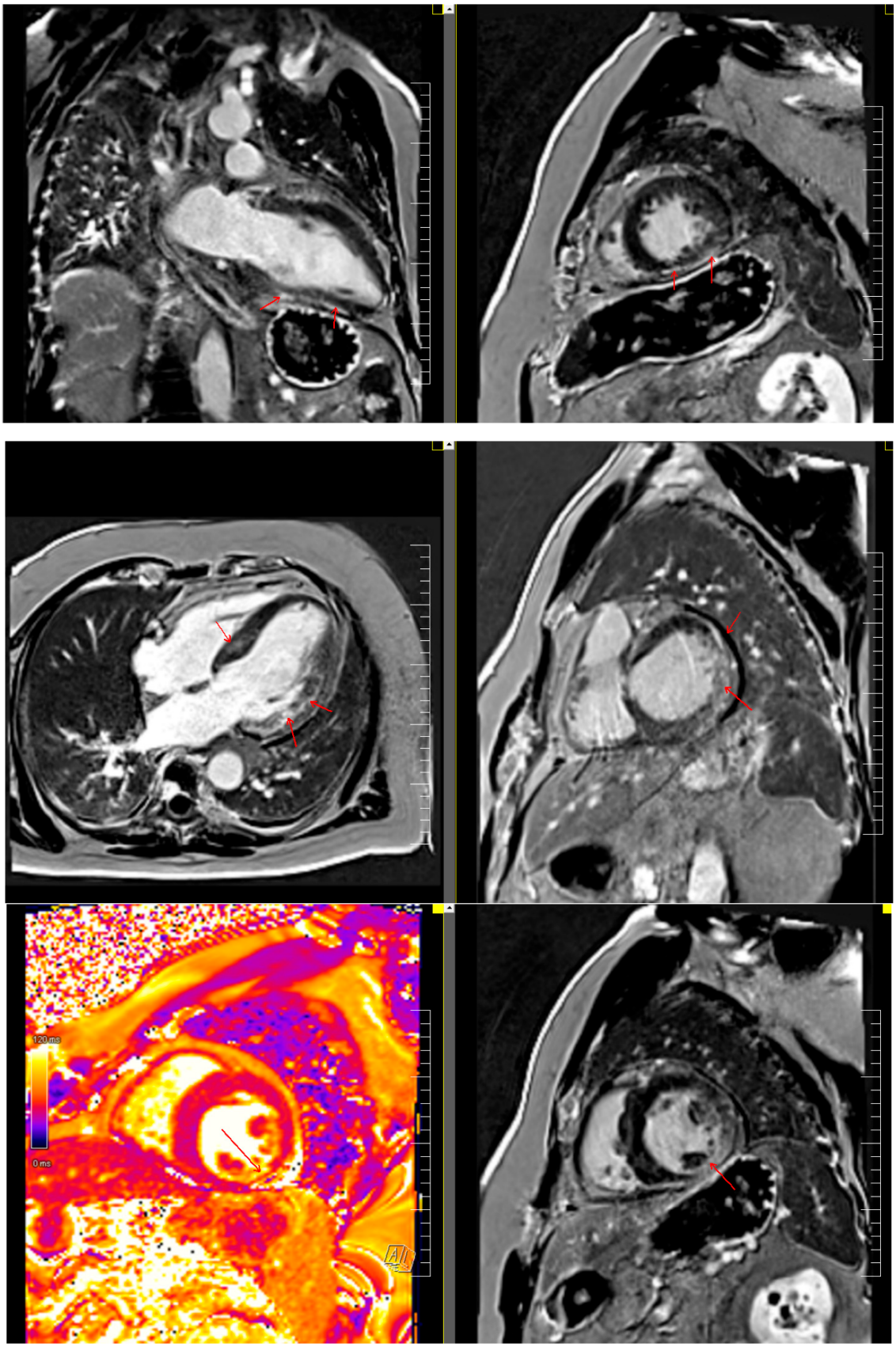


Figure 3. Cardiac magnetic resonance imaging (MRI): Late gadolinium enhancement (LGE) is positive, demonstrating intramyocardial and subepicardial contrast uptake involving the lateral, inferior, and septal walls. T2 mapping: At the site indicated by the arrow (red arrow), T2 relaxation times are increased (>60 ms), consistent with the persistence of myocardial edema. The scale bar indicates 120 ms.

At one-month follow-up, the patient was asymptomatic and hemodynamically stable. Electrocardiography (**Figure 4**) and echocardiography were unremarkable, and NT-proBNP had decreased to 355 pg/mL. The clinical timeline of the present case was as showed in **Table 2**.

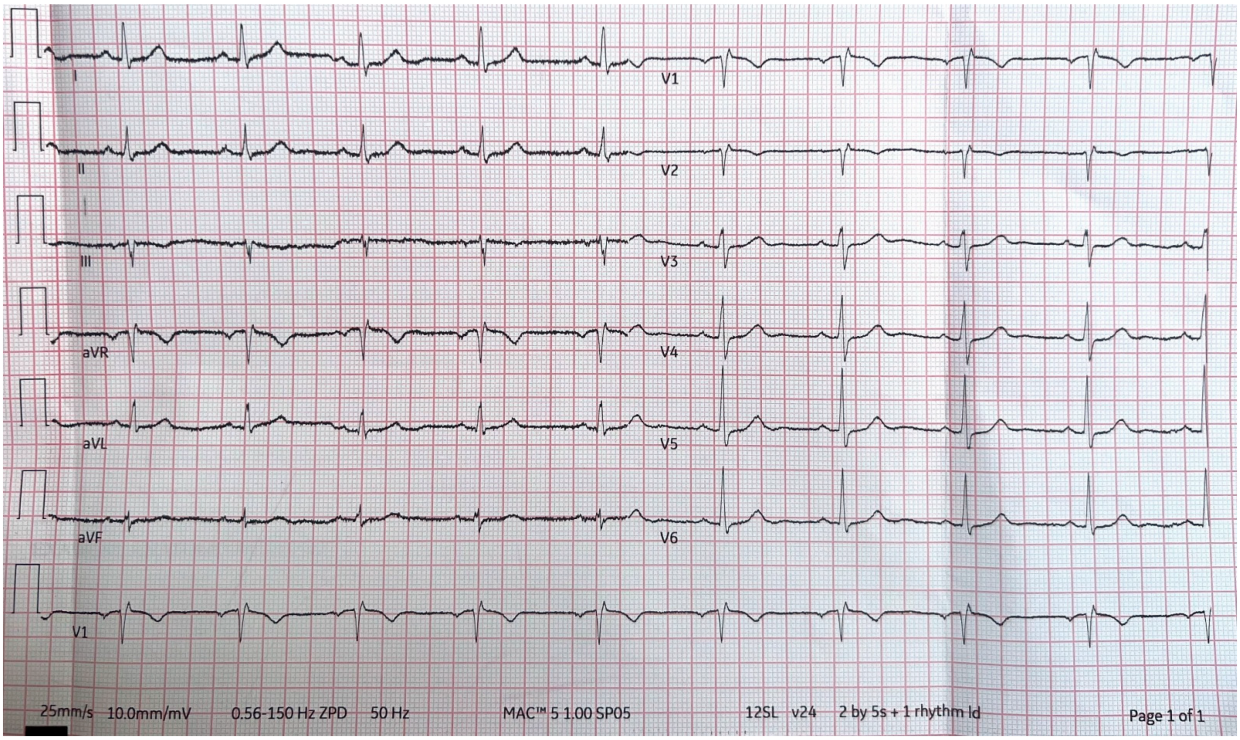


Figure 4. Electrocardiogram at one-month follow-up.

Table 2. Clinical timeline.

Timepoint	Clinical Event/Investigation	Findings/Intervention
Day-3	Mild upper respiratory symptoms	
Admission	Acute severe retrosternal chest pain, dyspnea, diaphoresis	Physical exam: hemodynamically stable, no murmurs; **ECG:** inferolateral ST-segment elevation
Hour 0–3	Laboratory assessment	**hs-cTnI:** 9123 → 21,135 ng/L; CK: 529 → 729 U/L; CK-MB: 62 → 81 U/L; CRP: 80 mg/L; NT-proBNP: 923 pg/mL
Day 0	Transthoracic echocardiography	Preserved LV EF 55%, discrete inferior wall motion abnormality, no pericardial effusion
Day 0	Coronary angiography	Normal coronary arteries; acute coronary syndrome excluded
Day 0	Initiation of therapy	Metoprolol 2 × 25 mg/day; Ramipril 2.5 mg/day;
Day 1	Cardiac MRI	Positive late gadolinium enhancement (LGE) with intramyocardial and subepicardial pattern; increased T2 relaxation times (>60 ms) indicating edema and active myocarditis
Day 2	Addition of therapy	Dapagliflozin 10 mg/day initiated, patient hemodynamically and biologically stable
Day 0–3	Analgesic support	Metamizol administered for symptom control
Day 0–5	Supportive therapy	IV fluids (2 × 500 mL 0.9% NaCl/day)
Day 7 (Discharge)	Outpatient prescription	Metoprolol 2 × 25 mg/day, Ramipril 2.5 mg/day, dapagliflozin 10 mg/day
Day 30 (Follow-up)	Clinical reassessment	Asymptomatic; ECG and echocardiography unremarkable; NT-proBNP decreased to 355 pg/mL

4.1. Differential Diagnosis

The differential diagnosis of this presentation primarily included acute coronary syndrome, given the patient's localized ST-segment elevation and markedly elevated cardiac biomarkers. Immediate coronary angiography was, therefore, essential to exclude obstructive coronary artery disease. Myopericarditis was also considered; however, the absence of pericardial effusion and the non-diffuse ECG changes made this less likely. Takotsubo cardiomyopathy was ruled out based on the preserved global systolic function and the lack of typical apical ballooning on echocardiography. Autoimmune, toxic, or hypersensitivity-related myocarditis was deemed unlikely due to the absence of exposure history, systemic symptoms, or eosinophilia. Cardiac MRI ultimately established the diagnosis by demonstrating non-ischemic, subepicardial late gadolinium enhancement and myocardial edema consistent with acute myocarditis.

4.2. Limitations

This case presents several limitations. The most significant is the absence of endomyocardial biopsy, as the patient declined the procedure, preventing definitive histopathological confirmation and etiological classification of myocarditis. Furthermore, an extended viral and autoimmune panel was not obtained, limiting the ability to identify a specific underlying cause. As this is a single-case observation, the clinical improvement associated with the use of an SGLT2 inhibitor cannot be extrapolated to broader patient populations, and no causal relationship can be established.

4.3. Patient Perspective

The patient, a previously healthy young adult, experienced acute severe chest pain and uncertainty regarding his diagnosis. The following prompt evaluation and initiation of guideline-directed therapy, including a beta-blocker, ACE inhibitor, and SGLT2 inhibitor, achieved rapid clinical and biochemical recovery and recognized the importance of ongoing cardiac surveillance.

5. Discussion

Acute myocarditis has a wide range of symptoms among patients. The most common symptom is chest pain, followed by dyspnea and syncope. Many patients diagnosed with myocarditis also report fever or prior respiratory or gastrointestinal infections [1]. Patients usually present with one of the three main patterns: severe chest pain, arrhythmias, or heart failure. Myocarditis should be suspected in any patient with these types of symptoms after exclusion of coronary artery disease, valvular heart disease, congenital heart disease, or hypertensive cardiomyopathy. It is crucial to rule out other possible causes, especially acute myocardial infarction, in these patients [3].

The etiology of myocarditis can vary, ranging from viral, bacterial, fungal, or parasite infections to drugs, toxic substances, hypersensitivity, and high catecholamine states. In many situations, the etiology of the disease cannot be determined, especially in the advanced stage—3 to 4 weeks after the infection—when the immune system already cleared out the pathogen that caused the infection [3]. Diagnosis is supported by clinical presentation and symptoms, widespread ST-segment elevation, and elevated cardiac biomarkers such as high-sensitivity Troponin I. If the pericardium is involved, there could be some PR depression on the electrocardiogram along with the widespread ST-segment changes. Echocardiography plays a crucial role in assessing the presence of other causes for the symptoms, such as valvular disease or hypertensive cardiomyopathy, and also to assess biventricular size and function [1, 4]. Histopathological analysis of myocardial tissue through endomyocardial biopsy is the gold standard for the definite diagnosis of myocarditis, but it is associated with a low rate of major complications (such as cardiac perforation, tamponade, valvular trauma, etc.) even if performed by experienced operators [6]. The current guidelines of the European Society of Cardiology recommend endomyocardial biopsy in high-risk patients and in some intermediate cases. Due to the advancement in technology and imaging techniques, cardiac magnetic resonance has been widely used to diagnose myocarditis successfully [7]. One large-scale epidemiological study reports that 12.2% of myocarditis patients aged 50 or older develop heart failure or dilated cardiomyopathy (DCM) within the first year after hospitalization compared to just 3.1% among those under 50 years [5]. Among children, the incidence rate of heart failure after myocarditis is relatively low, but the disease burden and risk for severe outcomes are high because of their immature immune systems [8]. Even though myocarditis can present in various forms, each form (chest pain, arrhythmias, and heart failure) should be treated accordingly. Angiotensin-converting enzyme (ACE) inhibitors are used routinely in patients with heart failure to reduce morbidity and mortality and to improve symptoms [9]. Discontinuation of ACE inhibitors after left ventricular improvement in patients with myocarditis could cause another heart failure episode in up to one-third of these patients [10]. Because sympathetic activation can aid in the progression of left ventricular dysfunction, beta-blockers have been extremely useful in patients with heart failure, improving morbidity and mortality with better clinical outcomes [11]. Sodium-glucose cotransporter 2 (SGLT2)

inhibitors are one of the pillars of heart failure treatment. Across reduced, mildly reduced, and preserved ejection fraction, SGLT2 inhibitors have a strong indication for use in patients with heart failure [9, 12]. Data from this retrospective cohort study indicate that treatment with SGLT2 inhibitors is associated with lower all-cause mortality and a reduced risk of progression to end-stage kidney disease in patients with heart failure with preserved ejection fraction, independent of diabetic status. These findings support the use of SGLT2 inhibitors as a beneficial therapeutic option in HFpEF, suggesting clinically meaningful cardioprotective and renoprotective effects in this population [13]. An experimental study demonstrated that SGLT2 inhibitors, such as canagliflozin, ameliorate cardiac inflammation in animal models of myocarditis, indicating a possible therapeutic benefit for myocarditis through anti-inflammatory mechanisms [14]. In a murine model of Coxsackievirus B3-induced myocarditis, dapagliflozin reduced disease severity, improved survival, and enhanced cardiac function. Its effects were linked to Stat3 pathway activation, promoting macrophage polarization toward the anti-inflammatory M2 phenotype over the pro-inflammatory M1 type. Treatment also lowered levels of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α [15]. In a myocardial ischemia–reperfusion model, dapagliflozin inhibited NLRP3 inflammasome activation, lowering IL-1β levels and caspase-1 activity. It also restored autophagic flux, enabling selective degradation of inflammasome components and improving lysosomal function. Additionally, by influencing Na^+ and Ca^{2+} handling via NHE1 and NCX, dapagliflozin may directly modulate cardiomyocyte stress and autophagy [16]. Overall, these data indicate that dapagliflozin may mitigate myocardial injury by modulating innate immune responses. Multiple systematic reviews and meta-analyses confirm significant reductions in hospitalization for heart failure and cardiovascular death with SGLT2 inhibitors in patients with heart failure of diverse etiologies, suggesting possible benefit if extended to myocarditis populations [17, 18]. Although clinical data in young patients with myocarditis remain limited, preclinical and mechanistic studies indicate that SGLT2 inhibitors may offer a promising therapeutic approach. These agents appear to exert anti-inflammatory, anti-apoptotic, and cardioprotective effects, in part by modulating NLRP3 inflammasome activity, suppressing pro-inflammatory cytokine production, and preserving cardiomyocyte viability. Further clinical trials are warranted to confirm these benefits in humans [13]. Arrhythmias are a recognized complication of myocarditis, especially in cases accompanied by signs of heart failure. Cytokines modify myocardial ion channel function, prolonging action potentials and disrupting repolarization, thereby creating a proarrhythmic environment, especially in patients with underlying cardiovascular issues or severe viral infections [19]. Recent studies suggest that SGLT2 inhibitors possess antiarrhythmic properties, though the precise mechanisms have yet to be fully clarified. Direct effects seem to involve changes in myocardial function, circulation, ion regulation, and cardiac electrical activity, while indirect effects include improvements in blood pressure, body weight, sympathetic tone, and cardiac loading conditions in patients with heart failure [20]. The positive impact of SGLT2 inhibitors on cardiac remodeling and inflammation found in preclinical studies supports further research in the context of myocarditis.

6. Conclusions

This case illustrates the variable presentation of acute myocarditis in a young, previously healthy patient, manifesting with severe chest pain and early heart failure. Multimodal assessment, including cardiac biomarkers, electrocardiography, echocardiography, coronary angiography, and cardiac MRI, was essential for diagnosis, differential diagnosis with acute coronary syndrome, and evaluation of myocardial inflammation. Early initiation of guideline-directed therapy with an SGLT2 inhibitor, beta-blocker, and angiotensin-converting enzyme (ACE) inhibitor led to symptomatic improvement and prevention of adverse ventricular remodeling, underscoring the importance of prompt and comprehensive management in myocarditis.

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Author contributions

Conceptualization, L.S. and L.H.A.; methodology, L.S. and A.A.; software, L.S. and L.H.A.; validation, A.A. and V.I.; formal analysis, L.S. and L.H.A.; investigation, L.S., A.A. and L.H.A.; resources, V.I. and L.H.A.; data curation, L.S. and L.H.A.; writing—original draft preparation, L.S. and L.H.A.; writing—review and editing, all authors.; visualization, A.A. and V.I.; supervision, A.A. and V.I.; project administration, L.H.A. and V.I. All the authors have read and agreed to the published version of the manuscript.

Conflicts of interest

The authors declare no conflicts of interest.

Data availability statement

Data are contained within the article.

Institutional review board statement

This case report was conducted in accordance with the Declaration of Helsinki, and approved by the Institutional Review Board (or Ethics Committee) of County Emergency Hospital from Timisoara, with approval document no. 23/28.02.2022.

Informed consent statement

Informed consent was obtained from the patient involved in the study.

Additional information

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