

Infantile Viral Clinically Myopericarditis with Fibrinous Strands: A Challenging Case Report of Successful Management

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Abstract

Case Presentation: A 14-month-old female infant was admitted with tachypnea, tachycardia, hypotension, prolonged fever of unknown origin, lethargy, poor feeding, leukocytosis, and an elevated erythrocyte sedimentation rate. Chest radiography revealed mild cardiomegaly with bilateral pleural effusion. Owing to a lack of clinical improvement and poor response to initial treatment, a pediatric cardiology consultation was obtained, and echocardiography was performed, which revealed mild bilateral pleural effusion and pericardial effusion with fibrinous strands. Chest ultrasound and Computed Tomography (CT) angiography of the heart and mediastinal vasculature confirmed these findings. To rule out malignancy, bacterial infection, tuberculosis, and other etiologies, pericardiocentesis was performed, and the specimens were analyzed; the results were solely consistent with viral clinically myopericarditis. The patient received appropriate treatment for the condition for 14 days, during which her clinical status gradually improved, and she was subsequently discharged. Follow-up evaluations 4-8 weeks later demonstrated improved cardiac function and the absence of pericardial effusion.

Conclusion: The most important conclusion of this article is that the use of PCR in this patient prevented misdiagnosis and error in the treatment protocol.

Key Words: Acute, Clinically, Fibrinous Strand, Infant, Myopericarditis, Viral.

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1- INTRODUCTION

The World Health Organization/International Society and Federation of Cardiology (WHO/ISFC) defines myocarditis as an inflammatory disease of the myocardium, diagnosed by a combination of histologic, immunologic, and immunohistochemical criteria (1).

Over the past several decades, the diagnosis of myocarditis has remained challenging, as it may present with a wide spectrum of clinical phenotypes in both children and adults, ranging from subclinical disease to overt cardiogenic shock and sudden cardiac death.

The prevalence of myocarditis in young children and adults is estimated to be between 0.6% and 1.8% (2).

The prevalence of myopericarditis in children is lower than that of isolated pericarditis or isolated myocarditis and remains poorly defined. Furthermore, the fibrinous form of this condition is considerably rarer and has been infrequently reported (3).

Acute myocarditis and pericarditis are associated with a wide range of causative agents, including viral, bacterial, fungal, toxins, cardiac surgery, blunt trauma, hypersensitivity reactions, protozoal, helminths, rickettsial, spirochetal, and systemic diseases (3,4,5,6,7,8). However, classically, the majority of cases in children and adults have been linked to secondary viral infection with common viruses.

The mechanisms of acute myocardial injury vary depending on the causative viral agent. Enteroviruses, for example, are cytolytic viruses that enter cardiomyocytes via the same transmembrane receptor [the coxsackie virus and adenovirus receptor (CAR)] and exert severe destructive effects through various pathways, including cleavage of the host protein dystrophin by viral protease 2A (9).

The cause and pathogenesis of pericarditis and myocarditis are due to almost identical etiologic factors (3).

Acute myocarditis and pericarditis may present in infants with fever, respiratory distress, tachycardia, hypotension, gallop rhythm, murmur, cardiac arrhythmia, cardiogenic shock, and anterior chest pain, typically characterized as pressure-like or sharp in nature, which is notably exacerbated in the recumbent position. Affected patients often prefer to sit upright and lean forward. The pain is further aggravated by inspiration, coughing, and movement. However, in infants, the presentation may be atypical, manifesting as unexplained respiratory distress, tachycardia, lethargy, poor feeding, and, rarely, abdominal pain and fever (5).

The most dangerous complication of pericarditis is cardiac tamponade, which is characterized on physical examination by 1- distant heart sounds, 2- hypotension, and 3- elevated central venous pressure with jugular venous distension. Ultimately, diminished coronary perfusion pressure leads to impaired cardiac function, reduced cardiac output, and hypotension (3,10).

Cardiac Magnetic Resonance Imaging (MRI) is the best diagnostic method for myocarditis. However, in the absence of cardiac MRI, other imaging modalities-including echocardiography, cardiac Computed Tomography (CT) angiography, electrocardiography (ECG), and clinical tests-can aid in the diagnosis of disease (3,11). The term myopericarditis refers to a condition in which the clinical manifestations of pericardial syndrome are predominant and account for the majority of clinical cases. In contrast, the term perimyocarditis is also occasionally used, primarily in cases where myocardial involvement constitutes the dominant feature. Nevertheless, these terms are sometimes employed interchangeably in the medical literature (3).

The management of myopericarditis comprises a combination of therapeutic strategies directed at both myocarditis and pericarditis. No definitive cure currently exists for either condition, a limitation that likely reflects the heterogeneous nature of their underlying etiologies. However, treatment is tailored to the suspected cause and may involve a broad spectrum of agents, including inotropes, corticosteroids, NSAIDs, acetyl salicylic acid (ASA), colchicine (12,3), antibiotics, and antiviral drugs, alongside supportive measures such as supplemental oxygen and mechanical ventilation. In the current era, for patients who are refractory to medical therapy, more invasive modalities—including ventricular assist devices (VADs) (11), pericardiocentesis—

may also be employed (12), and extracorporeal membrane oxygenation (ECMO) (13).

The objective of this report is to present a rare case of clinically viral myopericarditis with fibrinous strands in a pediatric patient who achieved a favorable therapeutic response without antibiotic therapy, responding adequately to the standard management protocol for myopericarditis in children.

2- CASE PRESENTATION

A 14-month-old female infant was admitted with weight = 10.5kg, length = 77 cm and head circumference = 46cm due to tachypnea, tachycardia, fever of unknown origin, lethargy, and poor feeding. Laboratory findings are shown in Table 1.

Table-1. Key laboratory data in a patient with probably myopericarditis.

Parameter	WBC	ESR*	CRP**	Troponin I	D-dimer	Nt-pro BNP	TSH***	FT4****
Value	17.6 Count /microliter	95 mm/h	3 mg/dL	0.2 ng/mL	1150 ng/mL	1896 pg/mL	3.7 mIU/L	1.1 ng/dL
Normal Range	5000-15000	0-10	< 1.0	0.12 - 0.6	<500	Acute HF>300	1-6 Yr 0.4-6.2	0.9-1.7

*Erythrocyte sedimentation rate, ** C- reactive protein, *** Thyroid stimulating hormone, **** Free T4

Vital signs for this patient were Blood Pressure = 60/35 mmHg, Respiratory Rate = 47/minute, Heart Rate = 150 /minute, temperature = 38 °C, SpO₂ = 92%. A chest X-ray revealed mild cardiomegaly and left lateral pleural effusion (Figure 1). A 12-lead ECG revealed sinus tachycardia without ST-T changes. Due to a lack of improvement, a cardiac consult and echocardiography showed pleural effusion (PLE) and fibrinous strands of pericardial effusion (PE) size of 18mm in a 4-chamber apical view (CT angiography, PE = 16mm) and mild systolic dysfunction with EF = 48% and FS = 25% (Figure 2). Although ECG-gated cardiac MRI is considered the optimal modality option for myocarditis, it was not available in the region; therefore, this imaging was not performed, and a chest ultrasound and cardiac CT

angiography were recommended, with the report shown in Figure 3.

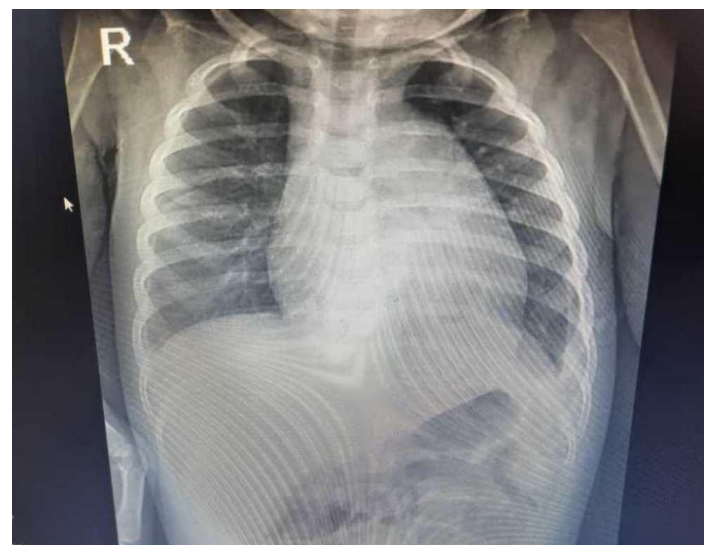


Figure-1: Chest X-Ray (AP view) showed mild cardiomegaly (water bottle sign) accompanied by a mild pericardial effusion at the left costophrenic angle (Arrow).

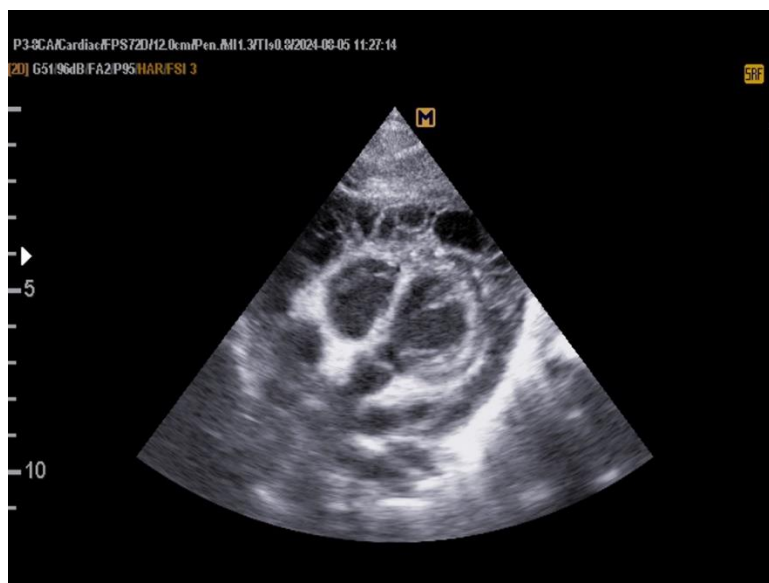


Figure-2: Transthoracic echocardiography modified four-chamber view image showed fibrinous strands with a worm-like appearance pericarditis.

To rule out malignancy (leukemia, lymphoma), bacterial infection (Staphylococcus aureus, Streptococcus pneumoniae, Haemophilus influenzae, etc.), tuberculosis, post-pericardiotomy syndrome, and other causes

(14,15,16,17,18), bacterial blood culture was negative. Pericardiocentesis was done, and viral (19) and bacterial PCR were analyzed; the results showed only viral pericarditis.

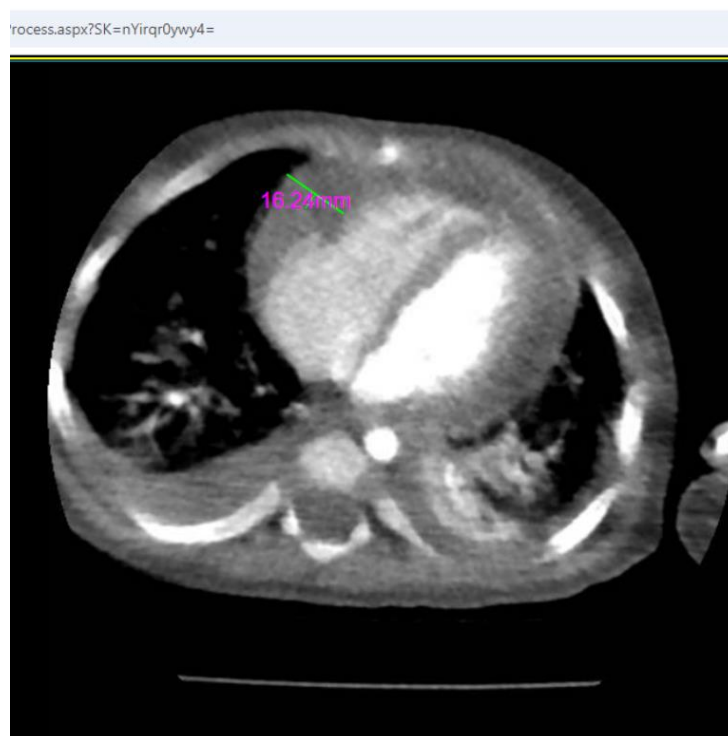


Figure-3: The cardiac CT-angiography study demonstrated a 16-mm pericardial effusion, passive atelectasis involving the basal segments of the bilateral lower lung lobes and no significant pericardial thickening. Additionally, no radiographic imaging findings suggestive of congenital heart disease and pericardial calcification were identified in this evaluation.

Other pericardial fluid findings included glucose, protein, and Lactate Dehydrogenase (LDH) levels of 120 mg/dL, 2.4g/dL, and 350 U/L, respectively. A white blood cell count of 400/ μ L was observed, with 80% lymphocytes, 20% polymorphonuclears and no blasts. Gram staining was negative for microbial pathogens and the clinical diagnosis was probable myocarditis (Figure 4). In the absence of an endomyocardial biopsy (the gold standard) and cardiac MRI, we established the clinical diagnosis of 'probable myocarditis' based on the consensus criteria of the European Society of Cardiology (ESC) (20). The diagnosis was supported by the combination of clinical symptoms (e.g. palpitation, cardiogenic shock and tachypnea), elevated cardiac biomarkers (troponin I, NT- proBNP, D-dimer, Erythrocyte sedimentation rate (ESR) and C- reactive protein (CRP)), and echocardiographic findings consistent with left ventricular systolic dysfunction, following the exclusion of acute coronary syndrome (Figure 4).

Real-time PCR was performed using a probe-based approach on a Roche LightCycler 96 Real-Time PCR (Roche, Germany) at the Shafa laboratory in Birjand, Iran.

The patient was managed symptomatically with corticosteroids [tablet Prednisolone 2.5mg/q6 hours (H) for 48 H] and intravenous immunoglobulin (IVIG) 5000mg/ slow intravenous (IV) infusion daily X 4 days due to autoimmune and viral myopericarditis. Dopamine 100 mg/IV daily infusion X 72 hours and milrinone 7.5 mg/IV infusion daily X 72 hours were initiated to address hypotension and cardiogenic shock due to myocardial involvement (myocarditis) and O₂ nasal continuous positive airway pressure (CPAP) due to hypoxia. Progressive improvement in the patient's overall clinical status was observed,

accompanied by the resolution of the pericardial effusion. The patient was discharged in stable general condition, and complete recovery was confirmed during echocardiography (EF= 63%, FS= 32%, without PE and PLE) in subsequent follow-up evaluations after 4-8 weeks. At the 4-week follow-up, laboratory values for D-Dimer, NT-proBNP, ESR and troponin I were 124, 92, 7, and negative, respectively. Ethical approval was obtained from the University Ethics Committee (IR.BUMS.REC.1400.009) for this case report.

Result of the panel:

Adenovirus	Undetectable
CMV	Undetectable
EBV	Undetectable
HSV 1	Undetectable
HSV 2	Undetectable
VZV	Undetectable
Enterovirus	Positive
Parechovirus	Undetectable
Human herpes virus 6	Undetectable
Human herpes virus 7	Undetectable
Parvovirus B 19	Undetectable

Figure-4: PCR of the patient's pericardial fluid sample, which was reported positive for enterovirus.

3- DISCUSSION

Pericarditis is often accompanied by some degree of myocarditis, in which case it is known as myopericarditis, and this was the case for our patient (3).

While our previous studies identified other viral pathogens, including Human herpesvirus 7 (HHV-7), Epstein-Barr Virus (EBV), parvovirus B19, and Varicella-zoster virus (VZV), the present study focused specifically on enterovirus.

Currently, the increased availability of PCR enables the more rapid and accurate identification of viral and bacterial

pathogens associated with this disease (15). However, in their review, Sozzi FB et al. addressed all viruses implicated in myocarditis, including enteroviruses (15).

In the present case, the infant presented with fever, poor feeding, tachypnea, lethargy, tachycardia, and respiratory distress—findings that were consistent with those reported by Imazio M. et al. and Sozzi F.B. et al (3, 15).

In contrast, in the study by Elikowski W. et al., chest pain and an infarct-like presentation were the dominant clinical features (19).

The 2026 ESC guidelines describe severe pleuritic pericardial pain that is exacerbated by deep inspiration and relieved by leaning forward. However, given the child's age, eliciting such a history is not feasible, which further underscores the critical importance of a meticulous physical examination and close attention to clinical signs to facilitate timely diagnosis (20).

In the study by Elikowski W. et al., elevated troponin levels returned to normal over time, whereas in our patient, troponin I, D-Dimer, ESR, and NT-proBNP levels normalized after 4 weeks (19).

In the study by Elikowski W. et al., serial echocardiography showed gradual normalization of left ventricular function, septal motion, and myocardial effusion, which was consistent with the findings in our patient (19).

Based on the study by Imazio M. et al., utilizing data from large registries, chest pain was the most frequently reported symptom (up to 95% of cases), followed by dyspnea (up to 49% of cases). Other typical but nonspecific symptoms, including fatigue, palpitations, and syncope, were also reported. Additionally, a prodrome of fever, influenza-like illness, or gastrointestinal symptoms was reported in 18% to 80% of patients. In our patient,

however, only the respiratory symptoms, palpitations and fever were concordant with the findings of the aforementioned study (3).

In a review article by Sozzi FB et al., transthoracic echocardiography (TTE) is generally considered the first-line imaging modality for suspected myocarditis, particularly when hemodynamic instability precludes the use of more accurate imaging techniques such as cardiac magnetic resonance (CMR) (3).

In this study, the most common findings at presentation were regional wall motion abnormalities (especially involving the inferior or inferolateral walls), diastolic dysfunction with preserved left ventricular ejection fraction (LVEF), and global left ventricular systolic dysfunction. However, in cases of fulminant myocarditis, a non-dilated left ventricle with global hypokinesis, increased wall thickness and echogenicity (resulting from myocardial interstitial edema), possibly associated with right ventricular dysfunction, was observed. In our patient, however, only mild systolic dysfunction without significant ventricular dilation was observed (15).

In the study by Imazio et al., treatment with high-dose IVIG, prednisolone, or azathioprine has been shown to improve cardiac function and reduce inflammatory markers. Furthermore, beta-blockers (carvedilol) are recommended in the management of acute myocarditis, even in uncomplicated cases, due to their antiarrhythmic effects. Accordingly, we used the first two agents in the treatment of our patient (3).

Nevertheless, as the study by Imazio M and colleagues demonstrated a lower incidence of arrhythmias in cases of concomitant pericardial and myocardial involvement (myopericarditis), we refrained from administering beta-blocker therapy to our patient (3).

In the study by Sozzi et al., the use of mechanical ventilation in conjunction with vasoactive agents was found to be beneficial in the setting of cardiogenic shock. In keeping with this, we utilized dopamine and milrinone during the treatment course; however, contrary to the article's recommendation, we found no indication for the use of ECMO or VAD (15).

Elikowski et al. reported that treatment of herpes zoster myocarditis with acyclovir resulted in a favorable response, with no sequelae and a good prognosis. The clinical course and follow-up of enteroviral myopericarditis in our patient were similarly favorable (19).

Based on the review study by Lazarou E et al., the overall prognosis of acute pericarditis is largely dependent upon the underlying etiology; however, it is generally excellent, with the majority of patients achieving complete recovery. Nevertheless, when accompanied by myocardial involvement, it portends a worse prognosis. Fortunately, in our patient, despite concurrent myocardial involvement and the development of cardiogenic shock, the prognosis remained favorable (21).

3-1. Limitation

Limitations of this report include the unavailability of cardiac MRI as a better diagnostic option, the exclusion of endomyocardial biopsy due to its invasive nature, and the inherent constraints of a single-case design.

4- CONCLUSION

The educational value of this article lies, firstly, in the life-threatening nature of myopericarditis, and secondly, in the atypical presentation of viral myopericarditis in children, which can mimic fatal bacterial forms in the differential diagnosis.

The objective of this article was to highlight the uncommon clinical patterns of certain diseases, including myopericarditis, in our pediatric patient. Given that the information in standard reference textbooks may occasionally diverge from a patient's clinical manifestations and course, the clinician must, in such situations, be capable of independently making appropriate therapeutic decisions and sharing the outcomes with colleagues. This approach ensures that, upon encountering similar cases in the future, at least a documented repository of prior analogous experiences is available and can be readily applied. Furthermore, despite extensive recent investigations, substantial advancements in therapeutic protocols, and the numerous review articles published in this field, many questions still remain unanswered.

5- ACKNOWLEDGMENT

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6- CONFLICT OF INTEREST

The authors declared no conflicts of interest.

7- REFERENCES

1. Richardson P. Report of the 1995 World Health Organization/International Society and Federation of Cardiology Task Force on the definition and classification of cardiomyopathies. *Circulation*. 1996;93:841-2.
2. Weber MA, Ashworth MT, Risdon RA, Malone M, Burch M, Sebire NJ. Clinicopathological features of paediatric deaths due to myocarditis: an autopsy series. *Archives of disease in childhood*. 2008 Jul;93(7):594-8.
3. Imazio M, Trincherò R. Myopericarditis: etiology, management,

and prognosis. *International journal of cardiology*. 2008 Jun 23;127(1):17-26.

4. Mahrholdt H, Wagner A, Deluigi CC, Kispert E, Hager S, Meinhardt G, et al. Presentation, patterns of myocardial damage, and clinical course of viral myocarditis. *Circulation*. 2006 Oct 10;114(15):1581-90.

5. Akbari A, Niksefat F, Mirshahi A, Salehi F. Toxic myocarditis following scorpion sting in a child: a case report and pediatric management approach. *BMC pediatrics*. 2025 Nov 4;25(1):900.

6. Shimizu C, Rambaud C, Cheron G, Rouzioux C, Lozinski GM, Rao A, Stanway G, Krous HF, Burns JC. Molecular identification of viruses in sudden infant death associated with myocarditis and pericarditis. *Pediatric Infectious Disease Journal*. 1995 Jul;14(7):584-7.

7. Troughton RW, Asher CR, Klein AL. Pericarditis. *The lancet*. 2004 Feb 28;363(9410):717-27.

8. Salehi F, Salehi S, Yousefi M. Diagnosis of viral myocarditis in children by multiplex real-time PCR in Vali-Asr Hospital, Birjand, eastern Iran. *Caspian Journal of Pediatrics*. 2021 Mar 10;7(1):458-64.

9. Pinkert S, Dieringer B, Klopffleisch R, Savvatis K, Van Linthout S, Pryshliak M, et al. Early treatment of Coxsackievirus B3-infected animals with soluble Coxsackievirus-Adenovirus Receptor inhibits development of chronic Coxsackievirus B3 cardiomyopathy. *Circulation: Heart Failure*. 2019 Nov 19;12(11):e005250.

10. Pérez-Casares A, Cesar S, Brunet-Garcia L, Sanchez-de-Toledo J. Echocardiographic evaluation of pericardial effusion and cardiac tamponade. *Frontiers in pediatrics*. 2017 Apr 24;5:79.

11. Wang ZJ, Reddy GP, Gotway MB, Yeh BM, Hetts SW, Higgins CB. CT and MR imaging of pericardial disease. *Radiographics*. 2003 Oct;23(suppl_1):S167-80.

12. Al-Zakhari R, Upadhya G, Galligan S, Shehaj F. The myth of colchicine in treating myopericarditis: case report and literature review. *Cureus*. 2020 Jun 30;12(6).

13. Toda K, Ako J, Hirayama A, Kinugawa K, Kobayashi Y, Ono M, et al. Outcomes of veno-arterial extracorporeal membrane oxygenation with percutaneous left ventricular unloading in fulminant myocarditis. *ASAIO Journal*. 2024 Apr 1;70(4):258-63.

14. Shaddy RE, Penny DJ, Feltes TF, Cetta F, Mital S. Moss and Adams' heart disease in infants, children, and adolescents including the fetus and young adults. 10th ed: Wolters Kluwer 2020, p. 4097-123.

15. Sozzi FB, Gherbesi E, Faggiano A, Gnan E, Maruccio A, Schiavone M, et al. Viral myocarditis: classification, diagnosis, and clinical implications. *Frontiers in cardiovascular medicine*. 2022 Jun 20;9:908663..

16. Moradian M, Alizadehasl A, editors. Atlas of echocardiography in pediatrics and congenital heart diseases. Springer; 2021 Jan 4.

17. Eidem BW, Johnson JN, Lopez L, Cetta F. Echocardiography in Pediatric and Adult Congenital Heart Disease, 3e. Lippincott Williams & Wilkins, a Wolters Kluwer business; 2021. Accessed July 07, 2026, p.475-92

18. Lee SH, Kim WH, Ko JK. Fibrinous pericardial effusion in a three-dimensional echocardiography. *QJM: An International Journal of Medicine*. 2015 Aug 1;108(8):665-7.

19. Elikowski W, Marszałek A, Małek-Elikowska M, Ganowicz-Kaatz T, Mozer-

Lisewska I. Myopericarditis in a 23-year-old male with herpes zoster. *Polski Merkurusz Lekarski: Organ Polskiego Towarzystwa Lekarskiego*. 2016 Feb 1;40(236):97-101.

20. Schulz-Menger J, Collini V, Gröschel J, Adler Y, Brucato A, Christian V, et al. 2025 ESC Guidelines for the Management

of Myocarditis and Pericarditis. *Giornale italiano di cardiologia* (2006). 2026 Jan;27(1 Suppl. 1):e1-93.

21. Lazarou E, Tsioufis P, Vlachopoulos C, Tsioufis C, Lazaros G. Acute pericarditis: update. *Current Cardiology Reports*. 2022 Aug;24(8):905-13.