Case Report

Rapidly progressive dilated cardiomyopathy following parainfluenza type 3-related fulminant myocarditis in an adolescent: A case report

Atharva Deodhar, Shraddha Sunthwal, Sagar Lad, Preeti Lad, Priya Malhotra, Aayushi Pal, Shravan Kulkarni, Raj Ganacharya, Sahil Lad[1]

Department of Pediatric Intensive Care Unit, Jehangir Hospital, Pune, [1]Government Medical College, Dharashiv, Maharashtra, India

Abstract

Parainfluenza virus type 3 is an uncommon cause of myocarditis.[1] A previously healthy 13-year-old girl presented with a 1-week history of fever and cough followed by rapid cardiogenic shock with markedly elevated cardiac biomarkers and severe left ventricular dysfunction (ejection fraction <10%). Respiratory RT-PCR detected parainfluenza virus type 3 as the sole pathogen. Despite aggressive inotropic support, milrinone, levosimendan, and intravenous immunoglobulin, ventricular function did not recover, and she progressed to end-stage dilated cardiomyopathy and died within 1 month. Early recognition and timely access to mechanical circulatory support are critical.

Keywords: Adolescent, cardiogenic shock, dilated cardiomyopathy, fulminant myocarditis, parainfluenza virus type 3

Address for correspondence: Dr. Sagar Lad, Pediatric Intensive Care Unit, Jehangir Hospital, Pune - 411 006, Maharashtra, India.
E-mail: drsagarlad@yahoo.com

INTRODUCTION

Viral infections are the most identified cause of myocarditis in children and adolescents, producing a spectrum from subclinical injury to fulminant myocarditis with rapid hemodynamic collapse and high short-term mortality.[2,3] Parainfluenza viruses are primarily respiratory pathogens; myocardial involvement is rare but has been reported.[4] The reported incidence of pediatric myocarditis is low but likely underestimated,[2,3] and a proportion of clinically apparent cases progress to dilated cardiomyopathy over time.[5,6]

CASE REPORT

A 13-year-old girl with no prior medical history presented after 1 week of fever and 4–5 days of cough and then developed progressive dyspnea, generalized weakness, oliguria, and intermittent chest pain. On admission, she was tachycardic (130/min), tachypneic (35/min), hypotensive (90/60 mmHg), and hypoxic (SpO_2 90% on room air). Examination revealed raised jugular venous pressure, bilateral basal crepitations, pedal edema, hepatomegaly, and a gallop rhythm with a prominent third heart sound.

Laboratory tests showed systemic inflammation and marked myocardial injury: C-reactive protein – 20 mg/L, alanine aminotransferase – 1040 U/L, aspartate aminotransferase – 644 U/L, Creatinine phosphokinase-MB (CK-MB) – 38.2 ng/mL, troponin-T – 30.9 ng/L, and N-terminal pro-B-type natriuretic peptide – >35,000 pg/

Received: 14-03-2026 **Revised:** 13-05-2026
Accepted: 01-06-2026 **Published:** 29-10-2026

Access this article online

Quick Response Code:

Website: https://www.ovid.com/jnls/jpcr

DOI: 10.4103/jpcc.jpcc_34_26

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 License (CC BY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

For reprints contact: WKHLRPMedknow_reprints@wolterskluwer.com

How to cite this article: Deodhar A, Sunthwal S, Lad S, Lad P, Malhotra P, Pal A, *et al*. Rapidly progressive dilated cardiomyopathy following parainfluenza type 3-related fulminant myocarditis in an adolescent: A case report. J Pediatr Crit Care 2026;13:311-3.

mL. Chest radiograph demonstrated cardiomegaly with pleural effusion. Electrocardiogram (ECG) showed sinus tachycardia with low-voltage QRS and diffuse ST-T changes. Transthoracic echocardiography revealed a dilated left ventricle (LV) with global hypokinesia and severely reduced systolic function (ejection fraction [EF] <10%).

Respiratory Reverse Transcriptase-PCR (RT-PCR) identified parainfluenza virus type 3 as the only detected pathogen. An expanded viral panel was negative for influenza, respiratory syncytial virus, metapneumovirus, rhinovirus, and adenovirus. Cardiac magnetic resonance imaging (MRI) excluded anomalous coronary origins but could not reliably characterize myocardial edema or late gadolinium enhancement because of clinical instability; endomyocardial biopsy was not performed due to hemodynamic instability and limited pediatric biopsy expertise.

Initial management included high-flow oxygen escalating to noninvasive ventilation, aggressive inotropic and vasopressor therapy (dobutamine, noradrenaline, milrinone, and adrenaline), and levosimendan infusion. Intravenous immunoglobulin (2 g/kg over 2 days) was administered for suspected immune-mediated myocardial injury. Mechanical circulatory support (extracorporeal membrane oxygenation [ECMO] or ventricular assist devices [VAD]) was not available locally; transfer to a tertiary center was discussed, but an early transfer was deferred due to instability.

After 9 days, she transiently stabilized and was discharged on guideline-directed heart failure therapy (enalapril, carvedilol, and furosemide) and low-dose aspirin, with planned referral for advanced heart failure evaluation. One week later, echocardiography showed persistent severe LV dysfunction (EF ≈ 10%). She was readmitted with progressive heart failure, counseled for advanced therapies including transplantation, but deteriorated and died within 1 month of initial presentation.

DISCUSSION

This case documents a rare but severe temporal association between parainfluenza type 3 infection and fulminant myocarditis in an immunocompetent adolescent. The clinical presentation – respiratory prodrome followed by cardiogenic shock, markedly elevated cardiac biomarkers, diffuse ECG changes, and profound global LV dysfunction – meets criteria for probable acute myocarditis.[1] Alternative causes (stress cardiomyopathy, genetic cardiomyopathy, metabolic disorders, autoimmune disease, and toxin exposure) were considered. They were made less likely by the clinical context, biomarker profile, and absence of pathogenic variants on whole-exome sequencing.

Fulminant myocarditis is characterized by rapid hemodynamic deterioration and high short-term mortality despite aggressive supportive therapy. Outcomes correlate strongly with the degree of LV dysfunction at presentation and the availability and timing of mechanical circulatory support; an EF below 10% at presentation, as in this patient, predicts poor prognosis and often necessitates urgent ECMO or VAD support.[3,4] In this case, lack of immediate access to mechanical circulatory support and the patient's early instability limited transfer options and likely contributed to the adverse outcome.[5,6]

Pathogenesis involves direct viral cytotoxicity and immune-mediated myocardial injury; persistent immune activation or viral persistence can drive progressive remodeling and dilated cardiomyopathy.[4] Parainfluenza is not routinely regarded as cardiotropic, but prior case reports document clinically significant myocardial injury and, rarely, fulminant courses requiring transplantation.[7,8] Intravenous immunoglobulin was used for its potential immunomodulatory effects; however, systematic reviews and trials show inconsistent benefit for presumed viral myocarditis, and routine use is not uniformly supported.

Practical implications from this case include maintaining a high index of suspicion for myocarditis in children and adolescents with recent respiratory illness who develop heart failure signs; obtaining early cardiac biomarkers, ECG, and echocardiography; performing broad respiratory viral testing because uncommon pathogens may be temporally associated with severe myocardial injury; and expediting transfer to centers with mechanical circulatory support and transplantation capability when severe LV dysfunction or refractory cardiogenic shock is present.[5,6] Definitive confirmation of parainfluenza myocarditis was not possible because endomyocardial biopsy was not performed, and MRI tissue characterization could not be completed due to instability. Detection of parainfluenza in respiratory samples establishes a temporal association but does not prove direct myocardial infection.

CONCLUSION

Parainfluenza virus type 3, though primarily a respiratory pathogen, can be temporally associated with fulminant

myocarditis and rapid progression to end-stage dilated cardiomyopathy in adolescents. Early recognition, aggressive supportive care, and timely access to mechanical circulatory support and evaluation for transplantation are essential to improve survival in such fulminant presentations.

Author's contribution statement

AD contributed to the literature search, clinical studies, data acquisition, manuscript preparation; SS contributed to the concept, design, definition of intellectual content, literature search, clinical studies, experimental studies, data acquisition, data analysis, statistical analysis, manuscript editing and manuscript review; SL contributed to the concept, design, definition of intellectual content, literature search, clinical studies, experimental studies, data analysis, statistical analysis, manuscript review; PL contributed to the concept, design, definition of intellectual content, literature search, data analysis, statistical analysis, manuscript review; and PM contributed to the literature search, clinical studies, experimental studies, data acquisition, manuscript preparation, manuscript editing.

Data availability statement

The data that support the findings of this study are available from the corresponding author, [SL], upon reasonable request.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Caforio AL, Pankuweit S, Arbustini E, Basso C, Gimeno-Blanes J, Felix SB, *et al*. Current state of knowledge on aetiology, diagnosis, management, and therapy of myocarditis: A position statement of the European society of cardiology working group on myocardial and pericardial diseases. Eur Heart J 2013;34:2636-48, 2648a-d.
2. Cooper LT Jr. Myocarditis. N Engl J Med 2009;360:1526-38.
3. Kindermann I, Barth C, Mahfoud F, Ukena C, Lenski M, Yilmaz A, *et al*. Update on myocarditis. J Am Coll Cardiol 2012;59:779-92.
4. Romero-Gómez MP, Guereta L, Pareja-Grande J, Martínez-Alarcón J, Casas I, Ruiz-Carrascoso G, *et al*. Myocarditis caused by human parainfluenza virus in an immunocompetent child initially associated with 2009 influenza A (H1N1) virus. J Clin Microbiol 2011;49:2072-3.
5. Canter CE, Simpson KE. Diagnosis and treatment of myocarditis in children in the current era. Circulation 2014;129:115-28.
6. Towbin JA, Lowe AM, Colan SD, Sleeper LA, Orav EJ, Clunie S, *et al*. Incidence, causes, and outcomes of dilated cardiomyopathy in children. JAMA 2006;296:1867-76.
7. Robinson J, Hartling L, Vandermeer B, Sebastianski M, Klassen TP. Intravenous immunoglobulin for presumed viral myocarditis in children and adults. Cochrane Database Syst Rev 2020;8:CD004370.
8. Kühl U, Pauschinger M, Seeberg B, Lassner D, Noutsias M, Poller W, *et al*. Viral persistence in the myocardium is associated with progressive cardiac dysfunction. Circulation 2005;112:1965-70.