



## Case report

## Rhinovirus-associated dilated cardiomyopathy

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## ABSTRACT

Rhinovirus is the main cause of the common cold. There is little to no published literature of rhinoviral associated myocarditis. We report a rare case of rhinovirus infection in a patient with myocarditis leading to dilated cardiomyopathy. Infection is an established cause of myocarditis. Prodromal “flu-like” symptoms in a young patient with unexplained heart failure should raise concern for viral myocarditis. Diagnosis is often made by clinical presentation and not by endomyocardial biopsy due to invasiveness. Polymerase chain reaction is a rapid test that supports the diagnosis and may elucidate the role of the virus in myocarditis. Defining causes and mechanisms leading to this severe cardiovascular condition may prove critical to targeted therapy. Physician should be aware that rhinovirus as a possible pathogen for severe myocarditis and dilated cardiomyopathy.

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## Introduction

Dilated cardiomyopathy is a common cause of heart failure. The prevalence of dilated cardiomyopathy (DCM) is 40 cases per 100,000 [1]. It is characterized by the presence left ventricular or biventricular dilatation. Prolonged impairment of contractility leads to decompensated heart failure. The evaluation of heart failure starts with the clinical presentation. Symptoms include dyspnea, orthopnea and paroxysmal nocturnal dyspnea. When DCM is suspected on clinical presentation, routine etiological workup involves ruling out coronary artery disease and identifying reversible causes of cardiomyopathy.

For example, patients with alcoholic cardiomyopathy are known to have a survival benefit with alcoholic abstinence. Similarly, patients with arrhythmia-induced cardiomyopathy with rate control have improved prognosis.

Infectious agents are one of the leading causes of dilated cardiomyopathy. Cytomegalovirus, adenovirus, herpesvirus and influenza virus are common pathogens associated with myocarditis [2].

Rhinovirus is a known cause of the common cold and can exacerbate other pulmonary conditions. It is a member of the family *Picornaviridae*. Historically, rhinovirus was classified as a

separate genera with enterovirus, but recently have been merged into the *Enterovirus* genus [3]. Rhinovirus associated myocarditis has been reported to the United States Centers for Disease Control and Prevention (CDC), but the association has not been published or established in literature [4]. Here, we present a rare case of the detection of Rhinovirus in an adult presenting with viral myocarditis and progression to acute onset dilated cardiomyopathy.

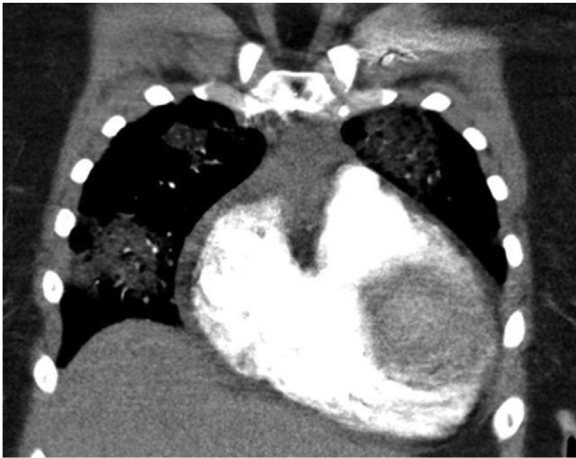
## Case report

In the winter season, a 27 year old African American female presented to the emergency department with progressive shortness of breath and associated paroxysmal nocturnal dyspnea. She had prodromal symptoms of rhinorrhea and myalgia. Past medical history revealed an ectopic pregnancy with salpingectomy 3 months prior to presentation. On physical examination, temperature was 99.1 degrees Fahrenheit, pulse of 110 beats per minute and blood pressure of 115/82 mmHg. The patient was in moderate distress, tachypneic and cardiac examination revealed a S3 heart sound. Laboratory examination revealed a white blood cell count of 9500 per cubic millimeter and lactic acid of 2.9 mmol per liter. B HCG was <1 mIU/mL. Initial troponin peaked at 0.022 ng/mL and remained flat.

An electrocardiogram revealed sinus tachycardia and nonspecific t wave inversions. Acute decompensated heart failure was suspected and echocardiogram revealed a left ventricular ejection fraction of 5–10 %.

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**Fig. 1.** Imaging of CT Chest angiogram with and without contrast: prominent dense mass-like infiltrates scattered throughout the lungs. Cardiomegaly was present.

Computed Tomography (CT) of the thorax with contrast demonstrated mass-like infiltrates throughout the lungs (Fig. 1). No pulmonary embolism was detected.

The next day, she was intubated and started on norepinephrine for hypoxic respiratory failure and severe hypotension. Vancomycin, cefepime and azithromycin, which were started in the emergency department, were discontinued when cultures from bronchoalveolar lavage were negative for bacteria.

Viral respiratory polymerase chain reaction (PCR) testing was obtained. PCR testing for rhinovirus was positive and the remaining panel was negative for adenovirus, parvovirus B19, HIV, CMV, influenza A/B, parainfluenza, RSV A/B, adenovirus, metapneumovirus and legionella pneumophila. Testing for Mycoplasma pneumoniae, Coxsackie antibody, EBV, influenza A and B were all negative.

The patient's cardiovascular status improved and she was extubated, weaned off oxygen and taken off vasopressors. She was initiated on standard heart failure medications prior to discharge and continued furosemide.

## Discussion

Infection is an established as a cause of myocarditis and dilated cardiomyopathy. It includes bacterial, viral, fungal and parasitic infections. Prodromal “flu-like” symptoms should raise the suspicion for viral myocarditis. Myocarditis is characterized by diffuse or focal inflammation of the myocardium. Echocardiography is a noninvasive imaging that assesses for wall abnormalities. The gold standard for definitive diagnosis of myocarditis is endomyocardial biopsy(EMB), but is done selectively due to the high degree of invasiveness [5]. Furthermore, false negative on biopsy can occur when the sample is not taken from the affected location.

Two common pathophysiological mechanisms that cause viral myocarditis are direct viral damage and an abnormal immune response. Dennert et al. proposes 3 phases of myocarditis leading to dilated cardiomyopathy. The first phase consists of myocyte injury due to direct destruction by virus mediated lysis. The second phase is a result of persistent viral genomic fragments that promotes a dysregulated immune response. The viral antigens share the same cardiac antigens, thus displaying molecular mimicry, leading to further cardiac myonecrosis. In the final phase, the progression to DCM is characterized by histologic findings of autoantibodies cross reacting with cardiac antigens [6].

The association of rhinovirus with dilated cardiomyopathy has rarely been reported in literature. Agend et al. reports a case of a 4 and a half year old boy who presented with acute onset of dyspnea and lower extremity swelling. Echocardiography demonstrated severe dilation of the ventricles and ejection fraction of 18 %. Molecular tests showed the presence of rhinovirus C genetic material in the nasal swab and serum while tests for other enterovirus were negative [7].

Over the past few decades, novel therapy with immunosuppressive for viral myocarditis has been explored. There have been promising results regarding the role of immunomodulation in certain patient populations. In 2001, Wojnicz et al. studied the use of prednisone in conjunction with azathioprine for patients with biopsy-proven inflammatory viral myocarditis. A clinically significant increase in left ventricular ejection fraction was seen for individuals in the treatment group [8]. Gkouziouta et al. studied the effects of pathogen driven therapy (PDT) in patients with new onset dilated cardiomyopathy and evidence of infection on EMB. All 93 patients were administered standard heart therapy. The experimental group also received PDT. The PDT regimen was selected antimicrobials based on the bacterial or viral pathogen. After 3 months, a significant improvement in left ventricular function was noted in the experimental group [9]. Not all studies administering immunotherapy have shown a favorable response. In a study reported in 2001, McNamara et al. was given intravenous immunoglobulin to the treatment group but there was no significant differences in ejection fraction compared to the control group [10]. Thus far in clinical practice, patients with dilated cardiomyopathy receive standard heart failure therapy and medications based on ejection fraction. Future research is needed to determine whether immunosuppressive or antiviral treatment should be pursued.

Our study has a few limitations. A definitive diagnosis of rhinovirus myocarditis could not be established since an EMB was not performed. However, our patient did present with classic prodromal symptoms and a positive rhinovirus test.

New onset of heart failure due to peripartum cardiomyopathy (PPCM) was considered but unlikely. The most common timing of PPCM is during the end of the second trimester, when the body is under the maximum hemodynamic load [11]. Our patient had an ectopic pregnancy 3 months prior to presentation to the emergency department, so she would not have experienced the full effects of hemodynamic stress from a normal pregnancy.

We report an atypical manifestation of Rhinovirus in an adult with acute onset DCM. Rhinovirus is well known to cause upper respiratory symptoms, but there is little literature that documents its pathogenic role in viral myocarditis or dilated cardiomyopathy. Early consideration for viral-induced dilated cardiomyopathy should be considered when a young adult with “flu like” symptoms presents with new onset heart failure. With more comprehensive diagnostic testing, endomyocardial biopsy and viral serology testing, future research can confirm the exact viral association with dilated cardiomyopathy and employ pathogen driven therapy.

## Author statement

Individuals contributing to the paper and their roles.

-Jeffrey Chow (main author): writing of original draft, patient care, reviewing and editing, visualization approval of final version of the article.

- Joey Murphy: writing of original draft, patient care, reviewing and editing, approval of final version of the article.

- Ashish Subedi: writing of original draft, reviewing and editing, patient care, supervision, visualization, approval of final version of the article.

- Amudhan Jyothidasan: reviewing and editing, approval of final version of the article.

### Ethical approval

All authors have made a significant contribution to the case report. The paper is original and ideas from other articles and authors have been appropriately cited. If there are any errors, it's the author's obligation to notify the journal editor and publisher.

### Informed consent

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

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### Declaration of Competing Interest

All authors have seen and agreed to the submitted version of the paper. The material is original and it has not been published elsewhere nor submitted for publication simultaneously. If accepted, this paper will not published elsewhere.

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- Joey Murphy: patient care, draft and revision of article, approval of final version of the article.  
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