A Case Report of Myopericarditis in a Young Female
Neda Ebrahimi, Tal Do, Juan Taveras and Oscar Munoz*
Texas Tech University Health Sciences Center, USA

Abstract
Our case presents a 25-year-old NICU nurse who developed sudden severe chest pain and shortness of breath with increased cardiac markers and ST-segment elevation in the inferior leads. She immediately underwent cardiac catheterization which was unremarkable. Her echocardiogram showed a normal left ventricular ejection fraction of 60%. She was eventually diagnosed with viral myopericarditis via cardiac MRI. Myopericarditis is characterized by pericardial inflammation with myocardial involvement. Viral infections are the most common causes of myopericarditis in developed countries. These patients usually present with nonspecific flu-like symptoms of upper respiratory or gastrointestinal tract infection. In this case, our patient has a severe chest pain that presents similarly to a myocardial infarction. Management of myopericarditis depends on the extent of cardiac involvement.

Keywords: Viral myocarditis; Myopericarditis; Pericarditis

Case Presentation
This is a 25-year-old lady with a past medical history of asthma, ovarian cysts and systemic lupus erythematous diagnosed at 16 years old and never been treated, who presented with dyspnea and severe pleuritic retrosternal chest pain that was pressure-like, non-radiating, worsened with laying down and inspiration and got better with leaning forward. She developed the symptoms when she was returning from a six-hour drive which worsened and brought her to the ED. Her blood pressure was 86/54 mmHg, and respiratory rate was 21 breaths per minute. Physical examination revealed a young well-nourished female who appeared in moderate distress secondary to pain. The Troponin I and BNP at admission were 12.43 ng/mL and 138 pg/dL. Her ESR and CRP levels were within normal limits. The initial electrocardiogram showed sinus rhythm with the 1 mm ST-segment elevation in inferior leads with ST depression in V1 and V2 (Figure 1, 2). The patient underwent cardiac catheterization which showed no high-grade coronary artery lesions or evidence of ruptured plaque. Transthoracic echocardiogram revealed no pericardial effusion, trace mitral regurgitation with preserved LVEF of 60% (Figure 3). Chest CT angiogram was done to rule out pulmonary embolism which was negative (Figure 4). Due to history of SLE, the ANA and anti-double stranded DNA antibodies were checked and the results were negative. The repeat Troponin-I was 41.60 ng/mL and repeat BNP was 510 pg/dL. The patient developed acute hypoxemic respiratory failure secondary to pulmonary edema and was put on high-flow oxygen. She went for a cardiac MRI which confirmed myopericarditis. Her serology studies were positive for Coxsackie A and B antibodies.

Introduction
Myopericarditis is an inflammatory disease of pericardial sac and myocardocytes. The presentation of myopericarditis is mainly pericarditic syndrome with minor myocardial involvement.

In developed countries, myopericarditis is usually caused by infectious pathogens, such as Coxsackie B, parvovirus B19, adenoviruses, herpes viruses, hepatitis C virus and influenza [1,2]. These viruses cause myopericardial damage via direct cytolytic or cytotoxic effects [3-5]. It can also be triggered by noninfectious etiologies, including autoimmune disorders such as systemic lupus erythematous, giant cell arteritis and Takayasu arteritis, cardio-toxins such as cocaine, alcohol and hypersensitivity reactions to medications (antibiotics, diuretics, lithium) [6].

People who have myopericarditis are typically between 20 to 50 years of age often with a history of a recent upper respiratory infection or development of a rash after being exposed to a new drug or vaccine.

Myopericarditis can present with symptoms of pericarditis, myocarditis or a combination of both. They can vary from nonspecific symptoms such as fever, muscle tenderness to more severe
conditions like arrhythmias and congestive heart failure. Some cases can manifest like acute coronary syndrome with acute chest pain, palpitations and dyspnea. The chest pain is sharp, severe, positional and pleuritic in nature, which is worsened by cough and inspiration. It is important to differentiate myopericarditis from acute coronary syndrome because of the similar presentations. Therefore, patients with a high pretest probability should undergo cardiac angiography.

The physical examination may be normal in most cases. Some can reveal friction rubs associated with pericardial involvement or signs of fluid overload (including pulmonary or peripheral edema, jugular venous distention, S3) or cardiac valve dysfunction such as functional mitral or tricuspid regurgitation.

Initial work-up generally includes complete blood count with differential, cardiac biomarkers, acute phase reactants, pro-BNP, EKG and chest radiography. Cardiac imaging such as echocardiogram, coronary angiography and cardiovascular magnetic resonance help detect impaired cardiac functionality and any evidence of inflammatory edema, myocyte necrosis or pericardial effusion. An Endomyocardial Bipsy (EMB) is histological and immunological evaluation of cardiac tissues. Although it is considered the gold standard, it is rarely performed due to the invasive nature. Cardiac Magnetic Resonance Imaging (CMRI) is a useful approach to estimate myocardial tissue injuries [7]. Recent studies have showed that CMR has a diagnostic sensitivity and specificity of 66% and 73% for acute myocarditis [8].

Myopericarditis diagnosis requires both evidence of pericarditis with myocardial involvement [9].

- Acute pericarditis (at least 2 criteria met)
- Typical chest pain
- Electrographical changes (diffuse ST segment elevation, PR-segment depression)
- Pericardial friction rub
- Pericardial effusion

Plus one of the following:
- Elevated cardiac biomarkers (Troponin, CKMB)
- Left ventricular dysfunction (decreased LVEF)
- Imaging of myocardial inflammation (CMR)
- Absence of other causes.

The course of myopericarditis varies with the clinical presentation. The majority of cases with mainly pericardial inflammation are self-limited without overt sequela. The repeat ECG and echocardiogram will normalize after a few days in most patients. However, if myocardium is severely damaged, patients can rapidly deteriorate and develop respiratory distress, myocardial failure or unexpected death. Studies have shown that people with severe risk factors...
including LVEF <45%, ventricular arrhythmias or cardiogenic failure, have increased adverse cardiac outcomes [2]. A more severe form of myocarditis is fulminant myocarditis which is characterized by a new onset of severe heart failure requiring mechanical circulatory support and bears a higher mortality risk.

Treatment of myopericarditis is usually managed similarly to pericarditis. The combination of NSAID (aspirin, ibuprofen or indomethacin) and colchicine are the preferred treatment in most cases. For those with contraindications to NSAIDs, prednisone and colchicine can be used. In case of autoimmune myocarditis, glucocorticoids may be considered.

Due to likelihood of developing late complications especially dilated cardiomyopathy, all patients with acute myocarditis are recommended to follow up with echocardiographic monitoring, cardiovascular magnetic resonance, nuclear testing or cardiac computed tomography on a regular basis to evaluate cardiac functions.

In our case, the patient has a history of SLE which was diagnosed 10 years ago and has been in remission. She started having sharp chest pain and dyspnea after the 6-h road trip. Her myopericarditis could be due to a combination of autoimmunity with a viral infection. Her symptoms are manifesting as pericarditis with an elevation of Troponin. She was discharged home with oral ibuprofen 600 mg and colchicine 0.6 mg for 1 week and a course of prednisone 40 mg daily to be tapered by 5 mg every other day. She was advised to follow up with her primary care doctor, cardiologist and rheumatologist as an outpatient.

**Conclusion**

Myopericarditis is not an uncommon condition which is mostly caused by a viral infection. If the majority of symptoms are pericarditis, the prognosis is favorable. However, it can have worse prognosis with myocardial involvement. The nature of sharp severe chest pain, elevation of cardiac biomarkers and diffuse ST segment elevation raise a concern for myocardial ischemia. It is important to initiate work-up to rule out MI at the early phase. On the other hand, we have to recognize if there is any myocardial injury since it can increase risk of ventricular arrhythmias and heart failure.

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**References**