Published in final edited form as:

Echocardiography. 2013 September ; 30(8): E227–E230. doi:10.1111/echo.12278.

Assessment of Mitral Annular and Velocity Vector Imaging in Acute Myopericarditis

Matthew Lynch, M.D.^{*}, Robert O'Donnell, M.D.[†], Neal L. Weintraub, M.D.[†], and Angel López-Candales, M.D., F.A.C.C., F.A.S.E.[†]

^{*}Department of Internal Medicine, University of Cincinnati College of Medicine, Cincinnati, Ohio

[†]Cardiovascular Division, University of Cincinnati College of Medicine, Cincinnati, Ohio

Abstract

A 25-year-old student presenting with pleuritic chest pain, elevated troponin levels and subtle electrocardiogram abnormalities was treated for presumptive myopericarditis. On echocardiography, a lower than expected mitral annular displacement and systolic velocity were identified along with abnormalities in left ventricular strain generation that matched areas of edema and necrosis by cardiac magnetic resonance imaging. The patient was treated with nonsteroidal antiinflammatory drugs and colchicine, and both the symptoms and echocardiographic abnormalities rapidly resolved. These novel findings suggest that changes in mitral annular displacement and systolic velocity occur in acute myopericarditis and may be useful in following the course of the disease.

Keywords

cardiac magnetic resonance imaging; echocardiography; mitral annular systolic velocity; mitral annular systolic displacement; myopericarditis; speckle tracking; velocity vector imaging

Case Report

A 25-year-old Ecuadorian male exchange student presented to student health with a 4 days history of pleuritic chest pain, sore throat, and flu-like symptoms. He was initially treated with ibuprofen and azithromycin, but developed worsening symptoms including chest pain radiating to his left arm, persistent fevers, chills, arthralgias, nausea, and anorexia. Two days later, he presented to the Emergency Department for evaluation. The patient denied shortness of breath, and physical examination showed no signs of fluid overload or pericardial friction rub. Electrocardiogram showed subtle, diffuse ST segment elevation and nonspecific T changes. Troponin I was elevated to 12.40 ng/mL on admission and peaked at 20.30 ng/mL within 48 hours. Based on the patient's presentation and exam, myopericarditis was suspected. An extensive battery of infectious and inflammatory markers was obtained

which demonstrated no definitive underlying etiology. Figure 1 shows standard short-axis and four-chamber echocardiographic images obtained at the time of admission and on follow-up testing.

Myocardial involvement was first suggested on echocardiography by noticeable reductions in both mitral annular systolic displacement and velocity based on the patient's age (Fig. 2). This presumptive diagnosis of myopericarditis was further supported by the use of velocity vector imaging (VVI) (Research-Arena program, TomTec Imaging Systems, Munich, Germany) (Fig. 3). Offline VVI not only demonstrated reduction in strain generation but also the presence of dyssynchronous contractility. Based on the clinical suspicion of myocarditis, supported by echocardiographic data, cardiac magnetic resonance imaging (MRI) (GE, Milwaukee, WI, USA) was performed. Inversion recovery delayed gadolinium enhancement (DGE) and T₂-weighted triple inversion recovery (TIR) black blood imaging demonstrated evidence of necrosis and myocardial edema, respectively, involving the basal inferior, infero-lateral, and anterolateral segments (Fig. 4). Involvement of the pericardium and the inferior right ventricle was also identified. The cardiac MRI findings were consistent with the presence of myopericarditis. The patient responded to therapy with toradol, ibuprofen, and colchicine, with marked improvement in the pleuritic chest pain. He was discharged and advised to avoid alcohol and refrain from participating in competitive athletics. He has done remarkably well with full resolution of his symptoms and improvement of the functional abnormalities noted on echocardiography and cardiac MRI.

Discussion

Myopericarditis is a clinical term commonly used to describe involvement of both pericardium and myocardium mainly as a result of infectious, inflammatory, postradiation or drug induced etiologies.^{1,2} The clinical presentation is quite variable and mainly depends on the extent of myocardial involvement, with most cases exhibiting a subclinical cardiac expression largely overshadowed by systemic manifestations; dyspnea, chest pain, and arrhythmias are noted in a minority of cases.³⁻⁵ Electrocardiogram frequently shows nonspecific changes or diffuse ST elevations; however, it can occasionally be suggestive of an acute myocardial infarction.^{1,2} In a minority of patients, elevation of cardiac biomarkers is detected.^{1,2} Most studies of acute myocarditis report a slight preponderance in men, suggesting a protective effect of hormonal influences on immune responses in women.⁶⁻⁸ Thus, in routine clinical practice, a suspicious cluster of symptoms is usually highly suggestive of myopericarditis. Gadolinium-enhanced cardiac MRI is currently considered to be the preferred diagnostic test of choice to confirm the diagnosis, demonstrating myocardial enhancement consistent with inflammation.^{1,2,9} However, cardiac MRI is not readily available in most centers; moreover, contraindications such as implanted metal objects and claustrophobia further limit the broad application of cardiac magnetic resonance (CMR) in these patients.

Echocardiography is widely available and easily applicable in patients with presumptive acute myocarditis. While it is frequently used to exclude other causes of heart disease in these patients, no diagnostic echocardiographic features of myocarditis have been identified.^{1,2} However, the recent introduction of mitral annular plane systolic excursion

(MAPSE) and VVI have been shown to be sensitive echocardiographic parameters to assess left ventricular function. Specifically, longitudinal displacement of the mitral annulus has been found to be a highly accurate predictor of left ventricular ejection fraction.¹⁰ Furthermore, VVI is a unique technique with complex signal processing but a straightforward user interface requiring only a single frame tracing of the endocardial border to extract quantitative time-motion data that not only facilitates regional myocardial velocity and strain measurement but also rapid visual assessment of synchronicity.¹¹

To our knowledge, we report for the first time how MAPSE and VVI were useful during the acute phase and resolution of myopericarditis, as both methods showed abnormalities of global and regional involvement that matched cardiac MRI findings. Even though standard echocardiography cannot surpass the diagnostic prowess of cardiac MRI, introduction of these novel echocardiographic methods might provide readily available objective information that can help in the early management and follow-up of these patients. Moreover, performing serial echocardiography is inexpensive and feasible in the office, making it an optimal technique to follow the evolution of myopericarditis. Idiopathic myopericarditis is usually self-limited. However, some patients with this disorder develop irreversible cardiac dysfunction, which even if asymptomatic may alter the treatment course and lead to prolonged activity restrictions. Additional studies are now required to prospectively determine if these echocardiographic measures correlate with the extent of myocardial involvement by CMR and can be useful in the identification, follow-up, and prognosis of patients with myopericarditis.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

References

1. Imazio M, Trinchero R. Myopericarditis: Etiology, management, and prognosis. *Int J Cardiol.* 2008; 127:17–26. [PubMed: 18221804]
2. Cooper LT Jr. Myocarditis. *N Engl J Med.* 2009; 360:1526–1538. [PubMed: 19357408]
3. Magnani JW, Dec GW. Myocarditis: Current trends in diagnosis and treatment. *Circulation.* 2006; 113:876–890. [PubMed: 16476862]
4. Imazio M, Cecchi E, Demichelis B, et al. Myopericarditis versus viral or idiopathic acute pericarditis. *Heart.* 2008; 94:498–501. [PubMed: 17575329]
5. Hufnagel G, Pankuweit S, Richter A, et al. The European Study of Epidemiology and Treatment of Cardiac Inflammatory Diseases (ESETCID): First epidemiological results. *Herz.* 2000; 25:279–285. [PubMed: 10904853]
6. Caforio A, Calabrese F, Angelini A, et al. A prospective study of biopsy-proven myocarditis: Prognostic relevance of clinical and aetiopathogenetic features at diagnosis. *Eur Heart J.* 2007; 28:1326–1333. [PubMed: 17493945]
7. Magnani JW, Danik HJ, Dec GW Jr, et al. Survival in biopsy-proven myocarditis: A long-term retrospective analysis of the histopathologic, clinical, and hemodynamic predictors. *Am Heart J.* 2006; 151:463–470. [PubMed: 16442915]
8. Schwartz J, Sartini D, Huber S. Myocarditis susceptibility in female mice depends upon ovarian cycle phase at infection. *Virology.* 2004; 330:16–23. [PubMed: 15527830]

9. Mahrholdt H, Goedecke C, Wagner A, et al. Cardiovascular magnetic resonance assessment of human myocarditis: A comparison to histology and molecular pathology. *Circulation*. 2004; 109:1250–1258. [PubMed: 14993139]
10. Matos J, Kronzon I, Panagopoulos G, et al. Mitral annular plane systolic excursion as a surrogate for left ventricular ejection fraction. *J Am Soc Echocardiogr*. 2012; 25:969–974. [PubMed: 22795199]
11. Kim DH, Kim HK, Kim MK, et al. Velocity vector imaging in the measurement of left ventricular twist mechanics: Head-to-head one way comparison between speckle tracking echocardiography and velocity vector imaging. *J Am Soc Echocardiogr*. 2009; 22:1344–1352. [PubMed: 19828287]

Figure 1.

A. Standard end-systolic short-axis views of the left ventricle on presentation and on **B** follow-up echocardiogram performed a week later. **C.** Standard end-systolic apical four-chamber views on admission and on **D.** follow-up echocardiogram performed a week later.

Figure 3.

A. Radial and **B.** circumferential strain on the admission echocardiogram using velocity vector imaging (VVI). Solid arrows denote dyssynchrony by radial strain inferoseptal, inferior, and inferolateral walls while the broken arrows demonstrate dyssynchrony of the same wall segments when measuring circumferential strain. **C.** Radial and **D.** circumferential strain on the follow-up echocardiogram using VVI. Please note synchronicity of the previously dyssynchronous segments. In addition, both peak radial and circumferential strain values are higher in the follow-up than on the admission echocardiogram VVI images.

Figure 4. Short-axis views of the base of the heart by inversion recovery delayed gadolinium enhancement (DGE) and T₂-weighted black blood imaging. The admission magnetic resonance imaging (MRI) demonstrates an increase in signal intensity of the epicardium and mid myocardium in the inferolateral segment on DGE and T₂-weighted black blood images (panels **A.** and **B.**, white arrows). These findings are consistent with the presence of myocardial necrosis and edema, respectively. There is also evidence of RV and pericardial involvement (**A.**, black arrow). The follow-up MRI demonstrates fibrotic remodeling of the involved myocardium **C.** with complete resolution of myocardial edema **D.** Please note that myocardial edema, a transient phenomenon, has resolved; however, as seen on the images, the fibrotic remodeling or scar persists.