KISEP
ISSN 1975-4612

www.kse-jcu.org

REVIEW J CARDIOVASC ULTRASOUND 2007;15(2):37-39

ECHOCARDIOGRAPHIC EVALUATION OF CONSTRICTIVE PERICARDITIS

JONG-WON HA, MD, PHD[1] AND JAE K. OH, MD[2]
[1]CARDIOLOGY DIVISION, YONSEI UNIVERSITY COLLEGE OF MEDICINE, SEOUL, KOREA
[2]DIVISION OF CARDIOVASCULAR DISEASES, MAYO CLINIC, ROCHESTER, MINNESOTA, USA

KEY WORDS : Echocardiography · Constrictive pericarditis.

INTRODUCTION

Constrictive pericarditis (CP) is an inflammatory process of the pericardium, leading to a thickened, scarred, and often calcified pericardium that limits diastolic ventricular filling. The diagnosis of CP can be challenging because of its protean presentations and the difficulty of distinguishing it from other disease entities with similar symptoms and hemodynamics. In this review, we will focus on morphologic and hemodynamic assessment by echocardiography for the diagnosis of CP.

TWO-DIMENSIONAL AND DOPPLER ECHOCARDIOGRAPHIC FINDINGS

Most patients with CP show characteristic 2-dimensional echocardiographic abnormalities. These include abnormal ventricular septal motion with prominent respiratory septal "bounce", calcified or thickened pericardium, or dilated inferior vena cava. These abnormal 2-dimensional echocardiographic findings are the most important clue for the diagnosis of CP and the observation of these findings should raise the diagnostic possibility of CP. Further demonstration of characteristic Doppler findings such as respiratory variation in mitral inflow velocity will support the diagnosis of CP. Hatle et al.[1] described the unique feature of respiratory variation in mitral inflow (Fig. 1) and hepatic vein velocities (Fig. 2) in patients with CP, and this substantially improved the accuracy for the diagnosis of CP. However, a subset of patients with CP does not demonstrate such respiratory variation in Doppler velocities,[2-5] and mitral inflow velocities may be indistinguishable from those of other causes of heart failure. Therefore, a lack of these typical respiratory flow velocity changes should not exclude the diagnosis. There are 2 possible explanations for the absence of typical respiratory changes in Doppler velocities in patients with CP.[2] First, some patients may have a combination of restrictive cardiomyopathy (RCM) and CP. Because the ventricular filling is limited by a noncompliant restrictive myocardium as well as a constrictive pericardium, full respiratory variation in ventricular filling may not occur. Second, the normal inspiratory intrathoracic pressure decline may not produce significant variation in mitral inflow velocities if the left atrial pressure is markedly increased and results in earlier mitral valve opening at a steeper portion of the left ventricular pressure curve. In this situation, repeat Doppler examination after an attempt

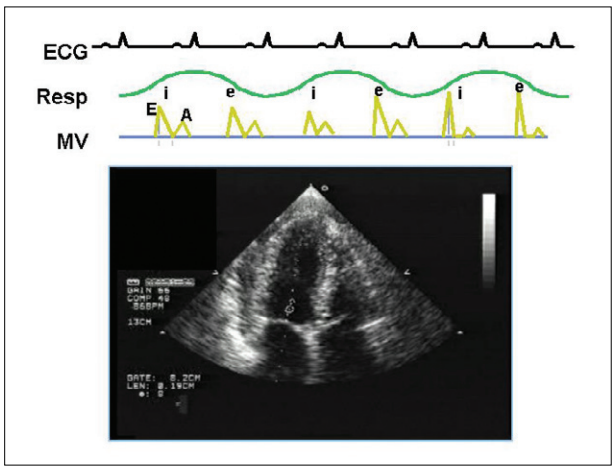

Fig. 1. Schematic drawing of Doppler velocities from mitral inflow along with electrocardiogram (ECG) and respirometer (resp) indicating inspiration (i) and expiration (e). The mitral inflow velocities decrease immediately after the onset of inspiration and increase with expiration.

• Received : April 9, 2007 • Accepted : May 7, 2007
• Address for Correspondence : Jong-Won Ha, Cardiology Division, Yonsei University College of Medicine, 250 Seongsan-no, Seodaemun-gu, Seoul 120-752, Korea Tel : +82-2-2228-8448, Fax : +82-2-2227-7943 , E-mail : jwha@yumc.yonsei.ac.kr

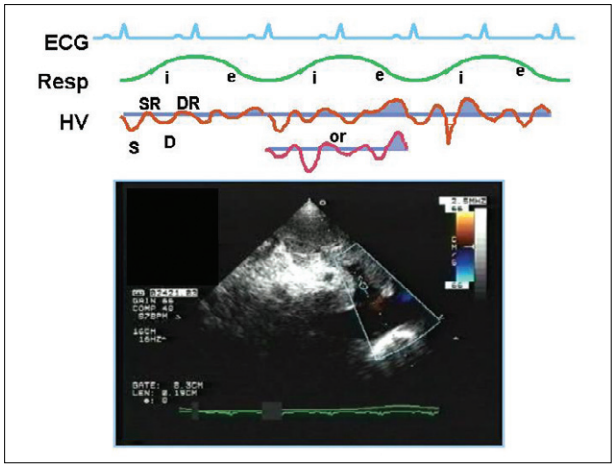

Fig. 2. Schematic drawing of Doppler velocities from hepatic vein (HV) along with electrocardiogram (ECG) and respirometer (resp) indicating inspiration (i) and expiration (e). There is exaggerated diastolic reversals and decreased diastolic forward flow in the hepatic vein with the onset of expiration. D indicates diastole, DR: flow reversal during diastole, S: systole, SR: flow reversal during systole.

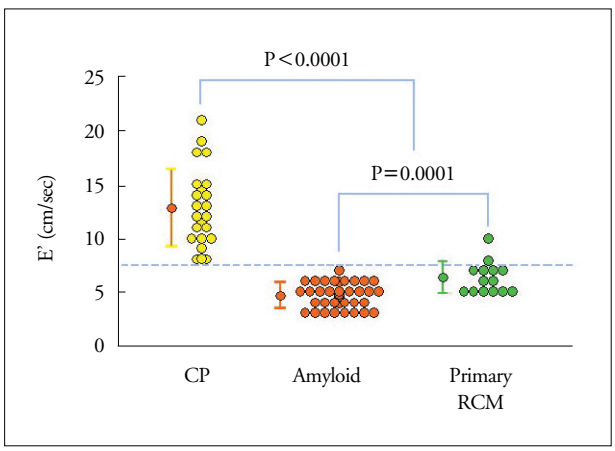

Fig. 3. Comparison of early diastolic mitral annular velocity (E') between the study groups. A value of > 8 cm/s differentiated patients with constriction with 95% sensitivity and 96% specificity (Adapted from reference #4).

to reduce preload has been shown to unmask the typical respiratory variation of mitral inflow velocity.[2] Recently, tissue Doppler echocardiography (TDE) has been used as a method to evaluate the diastolic function by measuring early diastolic mitral annular velocity (E').[6-8] The E(profile during diastole reflects shortening and lengthening of the myocardial fibers along a longitudinal plane, and it can be used to quantify longitudinal mitral annular motion. It has been shown that E (measured by TDE is reduced in patients with RCM whereas it is relatively normal or even accentuated in CP[4][9-11] (Fig. 3). Therefore, recording of E (by TDE is another useful means to diagnose CP when mitral inflow velocity reveals a restrictive filling pattern without sufficient respiratory variation. The recording of

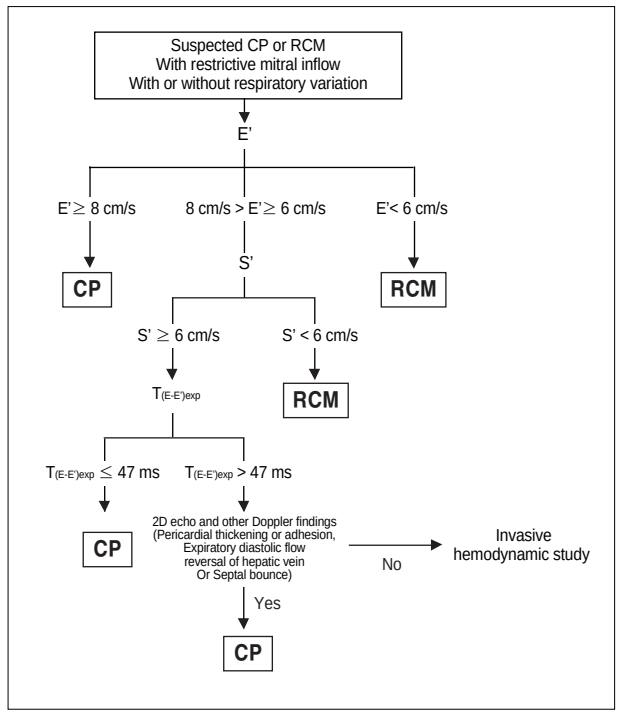

Fig. 4. A proposed diagnostic algorithm for differentiation between CP and RCM. If mitral inflow is pseudonormal or restrictive in patients who are suspected of CP or RCM, the next step should be the assessment of septal mitral annular velocity by TDE. If E' is normal or accentuated (≥ 8 cm/s), a diagnosis of CP is favored. If E' is low (< 6 cm/s), RCM or heart failure from myocardial disease should be considered. In patients with "grayzone" E' (6-8 cm/s), additional measurements of T (E'-E) and S' will be helpful. Additionally, the presence of additional two-dimensional and Doppler echocardiographic features of CP can further confirm the diagnosis.

E (by TDE should be an essential part of echocardiographic Doppler evaluation of all patients with heart failure, especially when CP is suspected.[3]

Although E' by TDE is very helpful diagnostic indicator for CP, it also has some caveats. Since previous investigations did not study patients with combined CP and RCM (constrictive pericarditis with coexisting myocardial abnormality), it is not clear whether E (would still be as diagnostic as in isolated CP. A cut off E' value of 8 cm/s has been shown to be a good discriminator between CP and RCM. However, in a subset patient with CP who had coexisting myocardial abnormalities, E' might not be high. In addition, despite excellent specificity of E' of 8 cm for differentiating CP from RCM, its sensitivity was relatively low, especially in patients with CP who had underlying myocardial abnormality.[2][6] Therefore, additional parameters will be helpful for further accurate diagnosis. In contrast to CP, LV longitudinal systolic function is abnormal in patients with RCM.[7] Garcia and colleagues demonstrated that the onset of E' occurred 7.5±3.5 ms after peak mitral inflow

velocity in patients with RCM, compared to 22±19 ms earlier in controls.[9] Subsequently, $T_{(E'-E)}$ has been shown to correlate with index of LV relaxation.[8] It has also been shown that with the worsening of heart failure by rapid pacing, E' not only progressively decreased but also delayed in onset.[12] This finding suggests that when relaxation is abnormal, not only the magnitude of E' is reduced but its onset is also delayed in reference to the onset of mitral valve opening or mitral inflow. A recent study has shown that S' and $T_{(E'-E)}$ have incremental value for differentiation between CP and RCM; S' was significantly higher and $T_{(E'-E)}$ was shorter in patients with CP compared with RCM. Although diagnostic accuracies of S' and $T_{(E'-E)}$ were lower than that of E', when combining E', S' and $T_{(E'-E)}$, sensitivity and diagnostic accuracy for differentiating CP from RCM could be improved.[12] With this robust discriminatory power, we propose a diagnostic algorithm for discrimination of CP form RCM (Fig. 4). If the mitral inflow is pseudonormal or restrictive (high E, low A, short deceleration time) in patients with suspected CP or RCM, the next step should include assessment of septal mitral annular velocity by TDE. If E' is normal or accentuated (≥8 cm/s), a diagnosis of CP is favored. If E' is low (<6 cm/s), RCM or heart failure from myocardial disease should be considered. In patients with "grayzone E' (6-8 cm/s)", additional measurements of $T_{(E'-E)}$ and S' would be helpful for further accurate diagnosis. In addition, the presence of additional two-dimensional and Doppler echocardiographic features of CP can further confirm the diagnosis.

Conclusion

Since echocardiography is capable of assessing cardiovascular structure, function, and hemodynamics noninvasively, it is well suited for the evaluation of patients with suspected constrictive pericarditis. Abnormal 2-dimensional echocardiographic findings such as abnormal ventricular septal motion with prominent respiratory septal "bounce", calcified or thickened pericardium, or dilated inferior vena cava are easily recognized by transthoracic echocardiography. Hemodynamic assessment by Doppler echocardiography provides the unique feature of respiratory variation in mitral inflow and hepatic vein velocities in patients with CP, and this substantially improved the accuracy for the diagnosis of CP. Recent developments in echo-Doppler techniques made non-invasive diagnosis of CP more accurate. Especially, the recording of E' by TDE should be an essential part of echocardiographic Doppler evaluation of all patients with heart failure, especially when CP is suspected.

References

1. Hatle LK, Appleton CP, Popp RL. *Differentiation of constrictive pericarditis and restrictive cardiomyopathy by Doppler echocardiography. Circulation 1989;79:357-70.*
2. Oh JK, Tajik AJ, Appleton CP, Hatle LK, Nishimura RA, Seward JB. *Preload reduction to unmask the characteristic Doppler features of constrictive pericarditis. Circulation 1997;95:796-9.*
3. Ha JW, Oh JK, Ommen SR, Ling LH, Tajik AJ. *Diagnostic value of mitral annular velocity for constrictive pericarditis in the absence of respiratory variation in mitral inflow velocity. J Am Soc Echocardiogr 2002;15:1468-71.*
4. Ha JW, Ommen SR, Tajik AJ, Barnes ME, Ammash NM, Gertz MA, Seward JB, Oh JK. *Differentiation of constrictive pericarditis from restrictive cardiomyopathy using mitral annular velocity by tissue Doppler echocardiography. Am J Cardiol 2004;94:316-9.*
5. Sohn DW, Kim YJ, Kim HS, Kim KB, Park YB, Choi YS. *Unique features of early diastolic mitral annulus velocity in constrictive pericarditis. J Am Soc Echocardiogr 2004;17:222-6.*
6. Nagueh SF, Middleton KJ, Kopelen HA, Zoghbi WA, Quinones MA. *Doppler tissue imaging: a noninvasive technique for evaluation of left ventricular relaxation and estimation of filling pressures. J Am Coll Cardiol 1997;30:1527-33.*
7. Nagueh SF, Sun H, Kopelen HA, Middleton KJ, Khoury DS. *Hemodynamic determinants of the mitral annulus diastolic velocities by tissue Doppler. J Am Coll Cardiol 2001;37:278-85.*
8. Sohn DW, Chai IH, Lee DJ, Kim HC, Kim HS, Oh BH, Lee MM, Park YB, Choi YS, Seo JD, Lee YW. *Assessment of mitral annulus velocity by Doppler tissue imaging in the evaluation of left ventricular diastolic function. J Am Coll Cardiol 1997;30:474-80.*
9. Garcia MJ, Rodriguez L, Ares M, Griffin BP, Thomas JD, Klein AL. *Differentiation of constrictive pericarditis from restrictive cardiomyopathy: assessment of left ventricular diastolic velocities in longitudinal axis by Doppler tissue imaging. J Am Coll Cardiol 1996;27:108-14.*
10. Rajagopalan N, Garcia MJ, Rodriguez L, Murray RD, Apperson-Hansen C, Stugaard M, Thomas JD, Klein AL. *Comparison of new Doppler echocardiographic methods to differentiate constrictive pericardial heart disease and restrictive cardiomyopathy. Am J Cardiol 2001;87:86-94.*
11. Ha JW, Oh JK, Ling LH, Nishimura RA, Seward JB, Tajik AJ. *Annulus paradoxus: transmitral flow velocity to mitral annular velocity ratio is inversely proportional to pulmonary capillary wedge pressure in patients with constrictive pericarditis. Circulation 2001;104:976-8.*
12. Choi EY, Ha JW, Kim JM, Ahn JA, Seo HS, Lee JH, Rim SJ, Chung NS. *Incremental Value of Combination of Systolic Mitral Annular Velocity and Time Difference Between Mitral Inflow and Early Diastolic Mitral Annular Velocity to mitral annular early diastolic velocity for Differentiating Constrictive Pericarditis from Restrictive Cardiomyopathy. J Am Soc Echocardiogr 2007;20:738-43.*