CASE REPORT

Utility of Lower Limb Positive Pressure Test for Diagnosis of Diastolic Heart Failure : A Case Report

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Abstract : A 70-year-old woman with dyspnea on exertion was admitted to our hospital. She had a history of apical hypertrophic cardiomyopathy (HCM) and repeated hospitalization for heart failure. Results of physical examination were normal except for leg edema. Echocardiography showed apical HCM with preserved LV systolic function (LVEF=70%). Although dyspnea on exertion and leg edema improved rapidly with the use of diuretics, her symptoms soon worsened when daily activity was started again. In order to examine the effect of preload on hemodynamics, we performed a lower limb positive pressure test by compressing both legs using a household air leg massager. Echocardiography showed increases in mitral E velocity, E/A ratio and pulmonary venous D flow as well as decrease in stroke volume during the lower limb positive pressure test. Simultaneously-recorded pressure study also showed elevated LVEDP and increased v wave of pulmonary capillary wedge pressure. These results suggested that even a small increase in preload led to elevation of LVEDP and symptomatic worsening due to severe diastolic heart failure in the present case. The lower limb positive pressure test may be useful for assessing the effect of preload on hemodynamics in patients with diastolic heart failure. J. Med. Invest. 61 : 404-408, August, 2014

Keywords **:** *diastolic heart failure, lower limb positive pressure test, apical hypertrophic cardiomyopathy*

INTRODUCTION

Patients with signs and symptoms of heart failure and a normal left ventricular ejection fraction are considered to have diastolic heart failure (DHF) (1, 2). At least one third of all patients with congestive heart failure have a normal or near-normal ejection fraction. As is the case with systolic heart failure (SHF), high morbidity rate (including rate of hospitalization) associated with DHF is one of the major concerns in clinical practice (3). DHF can occur in a variety of settings and a therapeutic strategy based on the pathological condition is needed. Although various procedures for assessing DHF have been proposed, they have several limitations (4, 5). We report a case of DHF successfully diagnosed by using a novel stress test named "lower limb positive pressure test".

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CASE REPORT

A 70-year-old woman presented to our hospital with dyspnea on exertion and lower-limb edema (New York Heart Association (NYHA) class III). She had been diagnosed with apical hypertrophic cardiomyopathy (HCM) 2 years ago, and she had been repeatedly hospitalized for the same symptoms and treated with diuretics for the past 4 years. She had no history of hypertension. Arterial blood pressure was 156/84 mmHg, pulse rate was 80 bpm, and arterial saturation was 98% (room air). Results of physical examination were normal except for bilateral leg pitting edema. No murmurs, gallops, or pulmonary rales were audible. Chest X-ray showed borderline cardiac enlargement with a cardiothoracic ratio of 0.51 without pulmonary congestion. A 12-lead electrocardiogram showed ordinary sinus rhythm, a QRS axis of approximately 30 degree, and a QT interval of 0.394 sec. Her electrocardiogram showed ST segment depressions and giant negative T waves in leads I, II, aVL and V2-6. No signs of left atrial overload were seen (Figure 1). Laboratory data were normal except for elevated BNP (90 pg/ml).

Echocardiography on admission showed already-known apical HCM without basal hypertrophy or outflow tract obstruction (LVEF=70%). Doppler echocardiography revealed a restrictive transmitral flow pattern and pulmonary hypertension (estimated pulmonary artery systolic pressure : 50 mmHg). The results of cardiac magnetic resonance imaging and scintigraphic evaluation were compatible with apical HCM. Results of coronary angiography were normal. Pressure study revealed elevated pulmonary capillary wedge pressure (PCWP, mean : 29 mmHg) and elevated pulmonary arterial pressure (PAP, mean : 28 mmHg).

Based on these findings, a diagnosis of cardiac decompensation due to apical HCM was considered. Although her symptoms improved rapidly with rest and diuretics, the symptoms easily relapsed after restart of her daily life.

Considering the worse-than-expected diastolic dysfunction, we performed a "lower limb positive pressure test" (Figure 2). The test is performed by passive compression of both legs (about 90 mmHg) using a household air leg massager, DM-5000EX® (Nitto Kohki, Tochigi, Japan), which enlarges preload due to increased venous return. Echocardiography during the lower limb compression revealed that mitral E velocity and E/A ratio increased with accelerated pulmonary venous D flow. The stroke volume was decreased and peak velocity of the tricuspid regurgitation jet was increased (Figure 3). Simultaneously-recorded pressure study also showed elevated left ventricular end-diastolic pressure (LVEDP) and

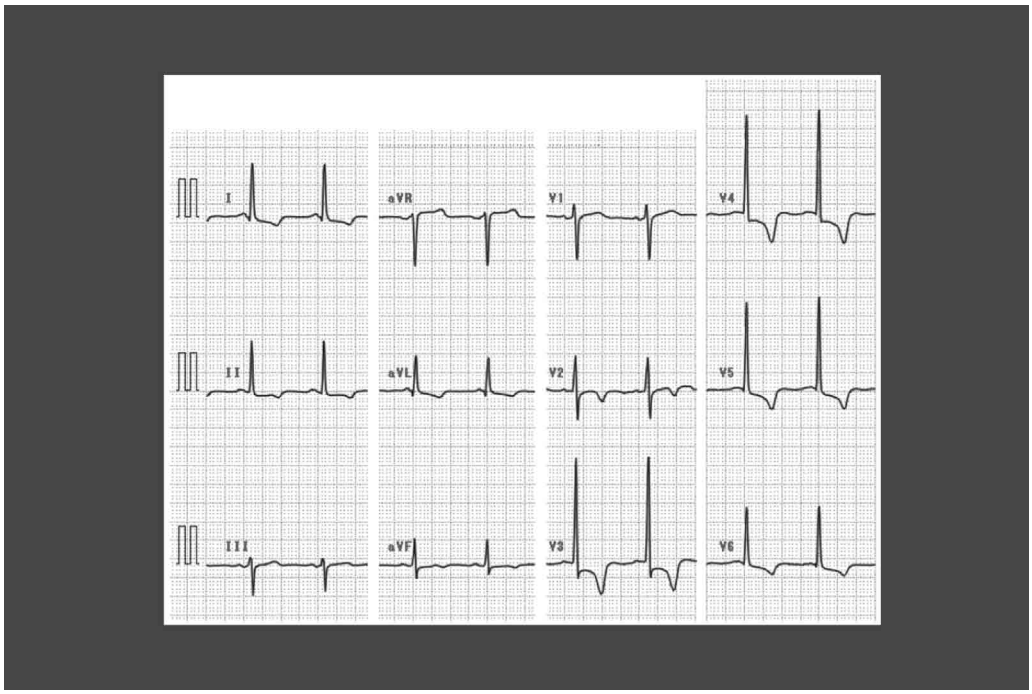

Figure 1 A 12-lead electrocardiogram showed ordinary sinus rhythm, a QRS axis of approximately 30 degree, and a QT interval of 0.394 sec. Her electrocardiogram showed ST segment depressions and giant negative T waves in leads I, II, aVL and V2-6. No signs of left atrial overload were seen.

Figure 2 Lower limb positive pressure test is performed by passive compression of both legs (about 90 mmHg) using a household air leg massager, DM-5000EX® (Nitto Kohki, Tochigi, Japan), which enlarges preload due to increased venous return.

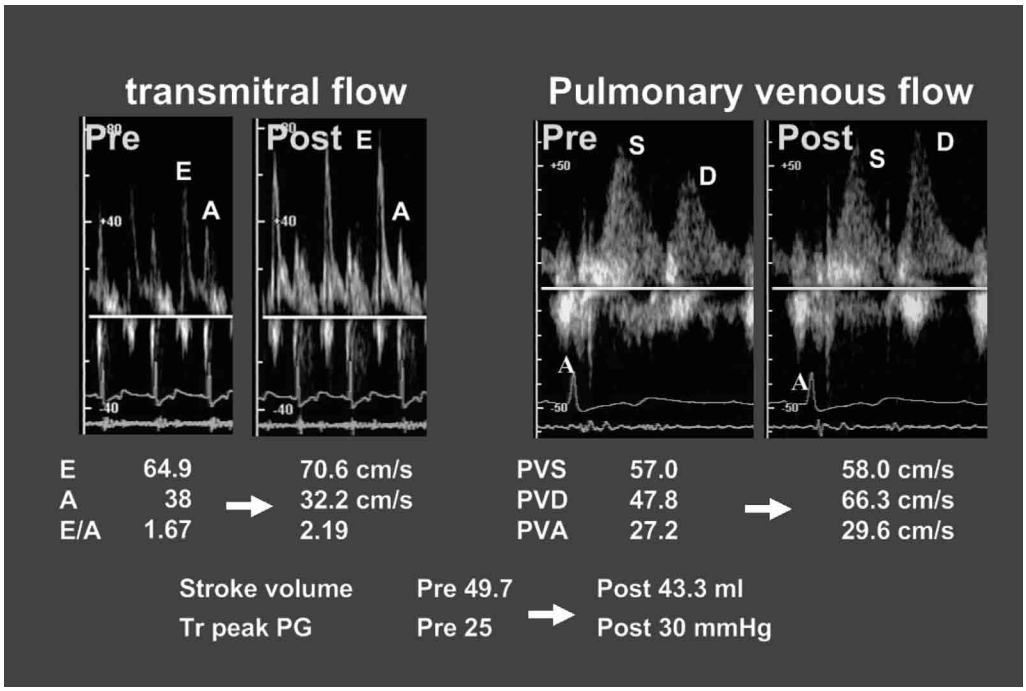

Figure 3 Echocardiography before and during the lower limb compression revealed that mitral E velocity and E/A ratio increased with accelerated pulmonary venous D flow. The stroke volume was decreased and peak velocity of the tricuspid regurgitation jet was increased. PG : pressure gradient, PVA : atrial flow reversal of pulmonary venous flow, PVD : pulmonary venous flow velocity in diastole, PVS : pulmonary venous flow velocity in systole, Tr : tricuspid regurgitation.

increased v wave of PCWP (Figure 4). These results suggested that even a small increase in preload led to elevation of LVEDP and symptomatic worsening due to severe DHF in the present case.

Although there are no data to support the use of a particular pharmacologic strategy in patients with DHF, our patient was administered diuretics, carvedilol and candesartan according to ACCF/AHA

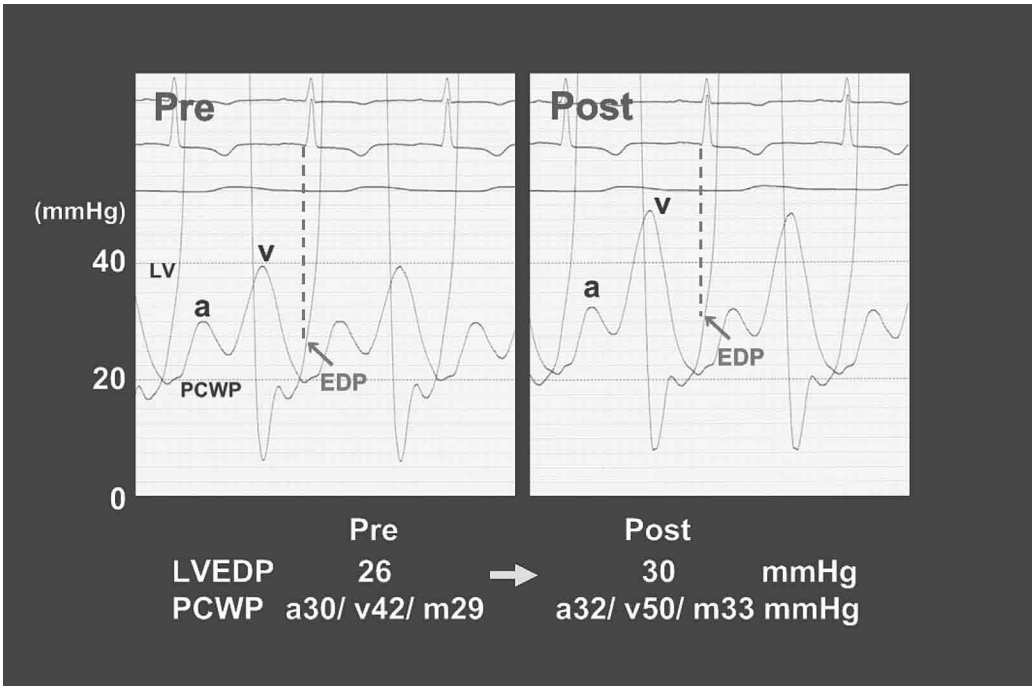

Figure 4 Cardiac catheterization before and during the lower limb compression revealed elevated left ventricular end-diastolic pressure (LVEDP) and increased v wave of pulmonary capillary wedge pressure (PCWP). EDP : end-diastolic pressure, LV : left ventricule, PCWP : pulmonary capillary wedge pressure.

guidelines (6). Her symptoms and signs of DHF gradually improved even under de-escalation of bed rest level. She was discharged and the rate of rehospitalization for recurrent DHF decreased.

DISCUSSION

We experienced a case of DHF due to apical HCM with frequent relapse of symptoms. In general, as in our case, exercise intolerance in DHF is caused by elevated PCWP and impaired stroke volume leading to dyspnea (7). Our case did not show remarkable left ventricular hypertrophy except for at the apex in image morphology. However, a diastolic stress test demonstrated noteworthy diastolic dysfunction.

DHF can occur in a variety of settings and a therapeutic strategy based on the pathological condition is needed. Echocardiography is a key component of the evaluation of patients with suspected DHF. Patients with DHF have symptoms, mainly with exertion, because of the rise in filling pressure that is needed to maintain adequate left ventricular filling and stroke volume. Therefore, it is useful to evaluate diastolic function with exercise or several hemodynamic stresses. Various stress tests for DHF have been proposed (4, 5), but they have several limitations.

The limitation of the Valsalva maneuver is that not all patients, including patients who cannot communicate, are able to perform. Nitroglycerin administration is also a useful strategy for experimental preload reduction, but it involves the risk of hypotension or inability of immediate discontinuation. In addition, these load reductive tests are relatively safe, but a load increasing test is preferable for assessment of several symptoms on exertion. Although a leg raising test increases venous return and enhances the preload, it is not standardized and requires limitation of body position.

In our case, we performed a lower limb positive pressure test using a household air leg massager (8). Leg cuffs compress both legs (about 90 mmHg) and enlarge preload by increasing venous return. We determined the 90 mmHg of pressure, which was lower than systolic blood pressure, for an intention to increase preloads without increasing afterloads. In our previous study, SHF, DHF, and control groups were studied for transmitral flow or pulmonary venous flow before and during application of lower limb positive pressure. We revealed that mitral A velocity decreased in the SHF, increased in the DHF, but did not change in the control group during increases in preload (9). In our case, transmitral flow velocity pattern changed to restrictive

pattern with the reduction of mitral A velocity and left ventricular output by leg positive pressure. These results suggested that even a small increase in preload led to elevation of LVEDP due to DHF.

The advantages of this test are that there is no necessity for long breath holding or drug dosing and that the load can be immediately interrupted at any time. We can control cuff pressure liberally and perform a quantitative examination.

In conclusion, we successfully evaluated the degree of DHF by using a novel stress test named "lower limb positive pressure test". This test may be useful for assessing the effect of preload on hemodynamics in patients with DHF.

CONFLICT OF INTERESTS

None for each author

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