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Original article

Clinical features of the systolic anterior motion of the mitral valve among patients without hypertrophic cardiomyopathy

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ABSTRACT

Background: The prevalence and clinical features of the systolic anterior motion of the mitral valve (SAM) without hypertrophic cardiomyopathy (HCM) have not been studied well.
Methods: Records of 9180 sequential patients who underwent echocardiography at Tokyo Women's Medical University Hospital were reviewed. SAM patients were divided into those with HCM (HCM; $n = 60$, 68%) and those without HCM (non-HCM; $n = 28$, 32%). To assess SAM morphology, non-HCM patients were divided into the valvular and chordal groups.
Results: The prevalence of non-HCM SAM was 0.3%. Non-HCM patients showed older age (65.7 ± 15.0 years vs. 56.9 ± 16.8 years, $p = 0.02$), higher prevalence of sigmoid septum (75% vs. 50%, $p = 0.03$), and lower left ventricular outflow tract pressure gradient (LVOT-PG) (27 ± 31 mmHg vs. 43 ± 41 mmHg, $p = 0.03$) than HCM patients. However, 8 of 28 non-HCM patients showed a LVOT-PG >30 mmHg. Valvular SAM showed higher dyspnea prevalence (29% vs. 0%, $p = 0.04$), higher LVOT-PG (39 ± 36 mmHg vs. 6 ± 2 mmHg, $p < 0.001$), longer anterior mitral leaflet (28 ± 2 mm vs. 26 ± 3 mm, $p = 0.04$) and more pronounced mitral regurgitation than chordal SAM.
Conclusion: Non-HCM SAM prevalence was 0.3% in the Japanese population. Non-HCM SAM correlated with older age, sigmoid septum, and a lower LVOT-PG compared with HCM SAM. Among non-HCM SAM, valvular SAM showed a significant symptom, higher LVOT-PG, and more pronounced mitral regurgitation than chordal SAM.

Introduction

The systolic anterior motion of the mitral valve (SAM) was initially considered a typical feature of hypertrophic cardiomyopathy (HCM) with left ventricular outflow tract (LVOT) obstruction [1,2]. SAM was also found to occur among patients without the echocardiographic features of HCM [3–19]. However, the clinical significance of SAM among patients without HCM has not been studied well. Recently, some reports demonstrated that exercise or dobutamine stress caused SAM and LVOT obstruction in patients without HCM [20–22]. Conversely, several reports described that SAM was rarely associated with LVOT obstruction among patients without HCM at rest [8,11].

Therefore, we aimed to clarify the prevalence of SAM without HCM and to describe the physiological and morphological features of SAM among patients without HCM in the Japanese population.

Materials and methods

Study population

The records of 9180 sequential patients who underwent echocardiography between January 2012 and March 2013 at Tokyo Women's Medical University Hospital were reviewed.

Echocardiography

Ultrasound examinations were performed using commercially available echocardiography machines (IE33™, SONOS 4500™ and SONOS 5500™, Philips Healthcare, Bothell, WA, USA. VIVID E9™, GE Vingmed, Horton, Norway. Aprio Artida™, TOSHIBA Medical

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Systems Corporation, Tochigi, Japan. SSD 5500™, Hitachi Aloka Medical, Ltd. Tokyo, Japan) with a 1–5 MHz transducer. Patients were examined while in the left lateral decubitus position, and standard views were obtained from the left parasternal and apical position [23]. Left ventricular (LV) cavity dimensions, LV wall thickness, LV ejection fraction, left atrial volume index, peak early diastolic filling (E) and late diastolic filling (A) velocities, E deceleration time, and early diastolic septal mitral annular velocity (e') were measured using previously published criteria. LV wall motion was evaluated as normal, hypokinetic, akinetic, dyskinetic, and aneurysm [23,24]. The sigmoid septum was defined by an angle between the ascending aorta and the basal portion of inter ventricular septum of less than 120° in the parasternal long-axis view at diastole [25]. The clinical diagnosis of HCM was made using 2-dimensional echocardiography. HCM was defined by a wall thickness ≥15 mm in one or more LV myocardial segments that is not explained solely by loading conditions [26]. A total of 88 patients demonstrated SAM including 60 patients with HCM (HCM) and 28 patients without HCM (non-HCM). SAM was evaluated by both the parasternal long-axis and the apical long-axis views using 2-dimensional echocardiography. SAM was defined as the systolic anterior motion of the mitral valve into the LVOT resulting in turbulent flow, visualized as a mosaic pattern by color flow Doppler imaging [27]. The LVOT pressure gradient was calculated from the velocity using the modified Bernoulli equation.

The length of the anterior and posterior mitral leaflets was measured at diastole in the parasternal long-axis view. To distinguish the structures causing SAM, the mitral leaflet was defined as a consecutive structure with similar echo reflectance and thickness as the coapted part between the annulus and the coaptation point [28,29]. The chordae was defined as a continuous structure located between the mitral leaflet and the papillary muscle.

To assess the morphology of SAM, 6 of the 28 non-HCM patients, including 1 patient with severe aortic valve stenosis and 5 patients with a history of beta-blocker use, were excluded, since it was difficult to distinguish the LVOT peak pressure gradient from the aortic valve pressure gradient in the patient with severe aortic valve stenosis and the beta-blocker might reduce the LVOT pressure gradient [30].

The remaining 22 patients were divided into the valvular and chordal SAM groups (Fig. 1). Valvular SAM was defined as systolic anterior motion involving the body of the anterior leaflet of the mitral valve. Chordal SAM was defined as systolic anterior motion involving only the chordae tendineae (Fig. 2) [31]. The LVOT area was calculated from the length of the LOVT in the parasternal long-axis view at early systole. Septal contact was defined as mitral valve or chordae tendineae touched with septum in the parasternal or apical long-axis view at early systole.

Statistical analysis

The results were expressed as the mean ± SD. Categorical variables were compared using χ^2 analysis, and continuous variables were analyzed using a Wilcoxon analysis. A p-value <0.05 was considered statistically significant. JMP software (version 10.0.02, SAS Institute Inc., Cary, NC, USA) was used.

Results

Prevalence of SAM

Among the 9180 patients who underwent transthoracic echocardiography, 88 presented with SAM, including 28 non-HCM patients. The prevalence of all SAM was 1.0% among all cases, while the prevalence of SAM without HCM was 0.3% in the Japanese population.

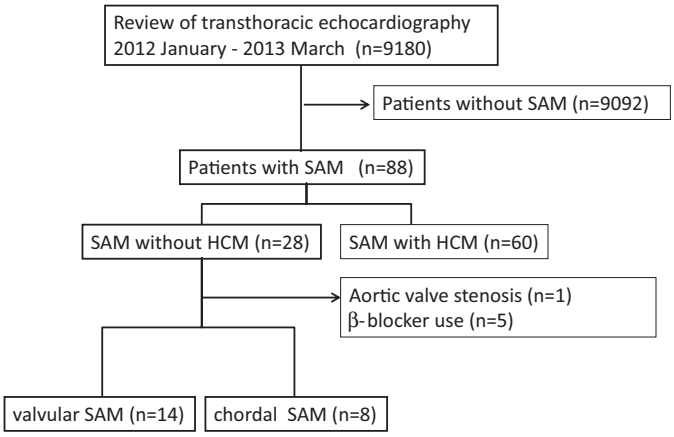

Fig. 1. Flow chart of patients. SAM, systolic anterior motion of the mitral valve; HCM, hypertrophic cardiomyopathy.

Comparison between SAM with and without HCM

Table 1 shows the clinical characteristics of patients with SAM. Non-HCM patients were significantly older than HCM patients (65.7 ± 15.0 years vs. 56.9 ± 16.8 years, $p = 0.02$). The use of beta-blockers was higher in HCM patients than in non-HCM patients (25% vs. 18%, $p < 0.01$). There was no difference in symptoms or prior history between the two groups (Table 1).

Echocardiographic findings for the patients with SAM are shown in Table 2. Intraventricular septal wall thickness (15 ± 4 mm vs. 8 ± 2 mm, $p < 0.001$) and LV mass index (125 ± 64 g/m^2 vs. 77 ± 26 g/m^2, $p < 0.001$) were larger in HCM patients than in non-HCM patients. The LV ejection fraction was similar in both groups. Left atrial volume index was larger in HCM than in non-HCM patients (48 ± 15 ml/m^2 vs. 35 ± 11 ml/m^2, $p = 0.003$). Although there were no differences in E and A between the groups, e' was significantly larger (6 ± 2 cm/s vs. 4 ± 2 cm/s, $p < 0.001$) and E/e' was significantly smaller (13 ± 6 vs. 20 ± 14, $p = 0.004$) in non-HCM than in HCM patients. HCM patients showed more frequently LV wall motion abnormalities than non-HCM patients (40% vs. 7%, $p = 0.04$). The prevalence of sigmoid septum was higher in the non-HCM than in the HCM patients (75% vs. 50%, $p = 0.03$). Non-HCM patients showed more aortic regurgitation than HCM patients. The prevalence of mild aortic regurgitation was 11/60 pts in HCM patients and 14/28 pts in non-HCM ($p = 0.008$). The grades of mitral regurgitation were equivalent for both groups. Non-HCM patients had lower LVOT pressure gradient (27 ± 31 mmHg vs. 43 ± 41 mmHg, $p = 0.03$). However, 8 non-HCM patients presented with a high LVOT pressure gradient exceeding 30 mmHg (Table 2).

Asymmetrical septal hypertrophy and diffuse hypertrophy were the most common types of morphologic pattern among HCM patients with SAM. Thirty-six asymmetrical septal hypertrophy, 9 apical and asymmetrical septal hypertrophy, 14 diffuse hypertrophy and 1 apical hypertrophy were observed among the HCM patients with SAM. There were no mid ventricular obstruction nor dilated HCM among HCM patients.

Twenty-six HCM patients with SAM showed hypertrophic obstructive cardiomyopathy (LVOT pressure gradient >30 mmHg). Three HCM patients had chordal SAM and all of them showed low LVOT pressure gradient.

Morphology of SAM without HCM

Without considering the factors possibly influencing the LVOT pressure gradient, a total of 14 out of 22 non-HCM patients were found to have valvular SAM while 8 patients had chordal SAM.

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