Phonoechocardiographic Identification of Chordal Snap in Hypertrophic Cardiomyopathy

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In three patients with hypertrophic cardiomyopathy, a late systolic click following mid systolic murmur was recognized. M-mode and two-dimensional echocardiography showed the presence of systolic anterior motion of the chordae tendineae in all cases. Phonoechocardiographic studies showed that the time of the click always coincided with the endpoint of mild anterior motion of the chordae tendineae. The click was considered to be a chordal snap. (Internal Medicine 31: 1094–1097, 1992)

Key words: a late systolic click, systolic anterior chordal motion, asymmetric septal hypertrophy

Introduction

A mid or late systolic click occurs in a variety of diseases and its genesis is considered largely as the result of sudden distension of the chordae tendineae or mitral valve cusps during ventricular systole (1). In hypertrophic cardiomyopathy, especially when outflow tract obstruction is severe, an early systolic sound may be found (2, 3). Sze and Shah (3) reported that the sound was produced in connection with systolic anterior motion of the anterior mitral leaflet in touch with the ventricular septum. However, there are few reports (4–6) on a mid or late systolic click in hypertrophic cardiomyopathy and the exact mechanisms for it are not clear.

We recognized a late systolic click following mid systolic ejection murmur in three patients with hypertrophic cardiomyopathy. In these patients, systolic anterior motion of the chordae tendineae out of touch with the ventricular septum was observed by echocardiographic studies. Here, we report the relationship between the motion of the chordae tendineae and the click using phonoechocardiography.

Methods

Study patients

Three patients with hypertrophic cardiomyopathy, aged 19 to 37, were studied because of the presence of a late systolic click following mid systolic murmur (Table 1). Two patients were noted to have an extra systolic sound

prospectively and one was noted retrospectively from the records of routine phonoechocardiographic examinations in fifty patients with hypertrophic cardiomyopathy at our institution. The diagnosis of hypertrophic cardiomyopathy was made by clinical and echocardiographic criteria.

Phonocardiogram

Phonocardiograms of the two prospective patients were obtained using a multichannel apparatus with microphones placed over the apex in the supine position. The carotid pulse and the electrocardiogram were simultaneously recorded as reference tracings.

Echocardiogram

In these three patients, complete two-dimensional echocardiograms were performed in all standard views with a 2.5 MHz transducer for imaging (Toshiba, SSH-60A). An M-mode echocardiogram was obtained by setting the beam direction to the chordae tendineae showing systolic anterior motion, which was observed from two-dimensional echocardiograms. M-mode echocardiograms with an electrocardiogram and phonocardiogram were recorded on strip-chart paper at 50 or 100 mm/sec. Furthermore, real-time images of M-mode and two-dimensional echocardiograms, which were simultaneously displayed with the imaging system, were recorded on videotape for further analysis. To determine the relationship of the chordal anterior motion and the timing of the click, echo images were examined frame by

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Chordal Snap in HCM

Table 1. Patient Characteristics

	Case 1	Case 2	Case 3
Age (year)	37	21	23
Gender	male	female	female
Echo. findings			
IVSth (mm)	28	22	20
PWth (mm)	8	8	8
SAM	presence	presence	presence
Intraventricular pressure gradient (mmHg)	11→15 (Infusion of NTG)	25→30 (Inhalation of amyl nitrite)	unknown
Family history of H(O)CM	absence	presence	presence

H(O)CM: hypertrophic (obstructive) cardiomyopathy, IVSth: thickness of interventricular septum, NTG: nitroglycerin, PWth: thickness of posterior wall, SAM: systolic anterior motion.

frame in slow-motion picture from the beginning of the Q wave to the click. In the two prospective patients, the intraventricular pressure gradient was examined.

Results

Phonocardiograms on the apex and the carotid pulse tracings

Figure 1 shows phonocardiograms recorded at the apex and the carotid pulse curves in cases 1 and 2. Phonocardiograms demonstrated mid systolic ejection murmur and a late systolic click following it. The high-pitched clicks were within a flat portion before the incisura of the carotid pulse curve in case 1 and within a rapid downstroke following a rapid upstroke of it in case 2.

Relationship between systolic anterior motion of the chordae tendineae and the timing of the click

Figure 2 shows the simultaneous records of phono-

Fig. 1. Phonocardiograms on the apex and carotid pulse tracings in case 1 (left panel) and case 2 (right panel). The arrows denote the late systolic high-pitched click following mid systolic ejection murmur.

Fig. 2. Case 1. Simultaneous recording of M-mode echocardiogram at the chordal level and phonocardiogram on the apex. The arrow shows the ending point of systolic anterior motion of the chordae tendineae. C: click.

Fig. 3. Case 2. Simultaneous recording of M-mode echocardiogram and phonocardiogram after Valsalva maneuver. The interval between the click (C) and the second heart sound and the interval between Q and click (between arrows) varied with spontaneous respiration. The upper arrow indicates the ending point of chordal systolic anterior motion.

cardiogram and M-mode echocardiogram of case 1. Mild systolic anterior motion of the chordae tendineae occurred from the time of the first heart sound to late systole, and the starting time of a late systolic click following mid systolic murmur coincided with the ending point of the systolic anterior motion. The interval between the Q wave and the click (Q-C) was 0.30 seconds. Figure 3 shows a spontaneous variation of the appearance of the click with a Q-C interval varying from 0.26 to 0.30 seconds immediately after Valsalva maneuver in case 2. Moreover, it was shown that the click consistently coincided with the ending point of the systolic anterior motion. In case 3, an M-mode echocardiogram demonstrated the presence of several chordae tendineae showing systolic anterior motion which ended not only at the second heart sound but also at the late systolic click (Fig. 4).

Fig. 4. Case 3. A simultaneous record of M-mode echo- and phonocardiogram. This figure demonstrates the existence of chordal echoes showing systolic anterior motion with a different duration. C: click.

Two-dimensional echocardiographic analysis of the generating source of late systolic click

Simultaneous records of phonocardiograms, M-mode and two-dimensional echocardiograms were performed in cases 1 and 2 in order to examine the genesis of the click more closely (Fig. 5). After the chordae tendineae attached to the anterior mitral leaflet projected a little into the ventricular outflow tract at the time of the first heart sound, it showed a further anterior movement at mid systole and suddenly stretched straight at the occurrence of the late systolic click accompanied by the reduction of middle and apical ventricular volume. On the other hand, mitral valve prolapse was not recognized at the time of the click in these cases.

Discussion

The idea that the mid or late systolic click may originate from abnormal chordae tendineae was proposed by Reid (7) and Barlow et al (8). This click was named *chordal snap* by Reid (7). Such a click is commonly accompanied by late systolic murmur, which denotes mild mitral regurgitation. Tucker et al (4) recognized a mid systolic click in two out of ninety patients with hypertrophic obstructive cardiomyopathy, and explained the genesis of the click by inequality of functional length of some

Fig. 5. Simultaneous display of M-mode and two-dimensional echocardiograms at the first heart sound (A), mid systole (B) and the time of late systolic click (C) in case 1 (left panel) and case 2 (right panel). Each two-dimensional echo image was obtained at the time of right end in each M-mode echocardiogram. Arrowheads on the images at the mid systole indicate systolic anterior motion of the chordae tendineae.

chordae tendineae resulting from asymmetric septal hypertrophy. They emphasized the presence of partial billowing of the mitral leaflets. However, they did not mention the presence of systolic anterior motion of the chordae tendineae and its relation to the timing of the click. Furukawa et al (5) recognized one case of hypertrophic cardiomyopathy with a late systolic click which coincided with the end of mild systolic anterior motion, but did not discuss its genesis. Luisada et al (6) also reported that one case of hypertrophic cardiomyopathy with moderate subaortic obstruction had a late systolic click which could not be explained by the alternative view of Sze and Shah (3) (impact of the anterior leaflet of mitral valve against the septum) because the anterior mitral motion had already ended at the time of the click. However, they did not mention obviously the relationship between the click and the anterior mitral or chordal motion. In the present three cases of hypertrophic (obstructive) cardiomyopathy, we observed a late systolic click following the mid systolic ejection murmur, and not accompanied by late systolic murmur. The time of the click always coincided with the endpoint of systolic anterior motion of the chordae tendineae. The anterior motion was at its maximum at the time of the mid systolic ejection murmur. Moreover, the existence of chordal echoes showing systolic anterior motion with a different duration in case 3 seemed to prove the inequality of chordal length. Accordingly, it was suggested that systolic anterior motion of the chordae tendineae may be due to inequality of its functional length resulting from asymmetric septal hypertrophy and the loose chordae may be pulled anteriorly toward the ventricular septum by the ejected blood flow. However, we could not observe mitral valve prolapse at the time of the click in all cases using two-dimensional echocardiography in all standard views. Echocardiographic observation at the time of the click suggested

that apical directed traction by the contracting papillary muscle and ventricle was more strongly transferred to the loose chordae which showed anterior motion in early and mid ejection phase than valvular directed traction. It appears that the mechanism by which the late systolic click occurs may be similar to that previously postulated (7, 8), and that mitral valve prolapse may not be always associated with the click following mid systolic murmur.

In conclusion, our observation suggested that the late systolic click in hypertrophic cardiomyopathy is a chordal snap occurring when several loose chordae tendineae are suddenly stretched straight and integrated into a whole without billowing of the mitral leaflets.

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